SIMULATION STUDIES OF THE BIOLOGICAL CONTROL OF *APHIS FABAE*

by

MICHAEL JOHN CRAWLEY B.Sc. (Edinb)

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Imperial College
Silwood Park
Ascot
Berkshire

ABSTRACT
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The use and usefulness of computer simulation models in applied ecology are discussed; there appear to be two distinct types of model, one of which seeks to provide precise, quantitative answers to economic problems, and the other to give qualitative understanding of the ecological behaviour of managed ecosystems. This distinction has not been made explicit in the past, but it is of fundamental importance.

Using the modelling techniques described, I have analysed the complex problem of determining the biological attributes of a predator species which would maintain populations of Aphis fabae Scopoli at a level which causes no significant economic loss of beans in a crop of Vicia faba L. To do this, three models are discussed; a model of the growth of the bean plant under aphid attack: a model of the growth and feeding of an aphid population under various predation regimes: and a model relating the biological attributes of a predator species to its performance in reducing and stabilizing aphid numbers. The attributes of this optimal predator are listed, and some of the known

predators of Aphis fabae are evaluated by comparison.

Finally I discuss the relationship between the complexity of a model (how much biology there is in it) and how useful it is (how well it answers the question).

CONTENTS
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GENERAL INTRODUCTION

The specific task of this thesis will be to investigate the use of biological simulation models in problem-solving. First of all, however, I will make clear what is meant by 'biological simulation models', and precisely which types of problem I shall be attempting to solve.

Models are simply reconstructions of nature for the purposes of study (Levins, 1968); they are simplified and abstract by necessity, but presume to represent the fundamental aspects of reality in an unmodified form. Of the numerous types of model (see Chapter I), biological simulation models are those which consider the detailed operation of biological processes by means of a set of equations amenable to numerical (rather than analytical) solution.

The class of problems to which such models are best applied concern the effects of manipulations or disturbances in ecological systems. Such problems typically occur in a resource management context, with the need to trace the direct effects and secondary repercussions of novel management policies. This is not to say that simulation models are valueless in theoretical studies, but rather that their benefits are greater in tackling applied problems (Chapter VI).

A problem has been chosen from the field of biological control to serve as an example of the use and usefulness of biological simulation models. This choice is based on the fact that biological control is typical of many management practices in that it involves the purposeful alteration of the distribution and abundance of species populations, and second, that the problems raised in biological control are often sufficiently complex as to defy solution by other means (mental reasoning or algebraic analysis).

Specifically, I have chosen to tackle the problem of determining the optimal biological attributes of a predator species which could maintain populations of Aphis fabae below the level at which significant economic loss of broad beans (Vicia faba L.) would occur. This problem is particularly interesting since it involves three species (a predator, an aphid and a plant) and three different spatial scales. Yield in the crop plant is determined essentially by physiological processes operating at particular nodes (determining the number of flower buds, flower survival, pollination, seed-fill, and so on), whereas the aphid population acting to reduce bean yield is distributed over whole plants. The set of processes affecting the pest population must therefore be broader than, but inclusive of, those acting on the plant. Finally, the predator population is distributed over rather large areas of the crop between points of different prey density, and will, in consequence, be affected by a still wider set of processes. This relationship is shown in Fig. 1.

The body of the thesis is divided into six chapters. In the first, I examine the types of model which are in current use in various branches of applied ecology, and detail the

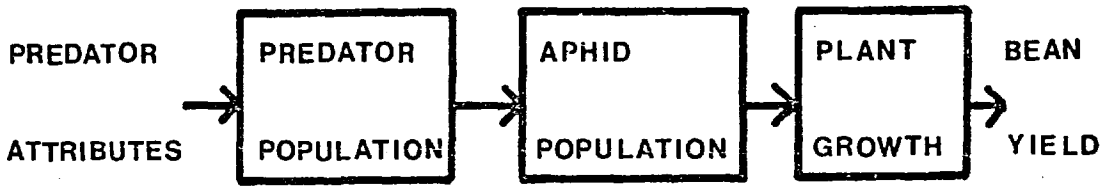


Figure 1. The three components of the problem of determining the attributes of the ideal predator for Aphis fabae on beans

procedure by which biological simulation models are designed and constructed. The pest control problem is then investigated to assess the variables which influence bean yield and the effectiveness of biological control, and to estimate the type and amount of data which will be necessary to quantify the model.

In the following chapters I shall consider the question in three broad sub-sections. First, I shall deal with the effects of Aphis fabae upon its host plant; as an agency of drain on the net production of dry matter, and as an influence upon the pattern of distribution of this dry matter between the plant organs, especially into the fruit. This section comprises chapter II, an experimental investigation, and chapter III which considers a simulation model of the growth of Vicia faba under an aphid infestation.

The second section consists of an analysis of the growth and feeding of an aphid population, with particular reference to the effects of age-selective predation on these parameters. Again, a simulation model is presented which has been built to consider the question "which strategy of predation results in the most efficient reduction in aphid feeding (and hence minimises damage to the host plant) ?" The efficiency of predation has been defined as the grams of plant dry matter saved per gram of aphid eaten; in other words, the extent to which unit feeding by the predator benefits plant yield.

Chapter V deals with the relationship between the biological attributes of a predator species (its fecundity, sex-ratio, searching efficiency, voracity, dispersive abilities, and so on) and its efficiency as an agent of biological

control against aphids. A model of predation is presented which incorporates the functional responses of the species to prey density, and the numerical responses to changes in this density. The numerical response is considered as affecting, on the one hand, the survival rate and fecundity of the predator and, on the other, the pattern of dispersal of the species over the area occupied by the prey.

The final chapter investigates the relationship between the complexity of a simulation model and its utility, and discusses the merits and limitations of modelling in assessing problems in resource management and in theoretical studies of population behaviour. Criteria are suggested for deciding the degree of complexity necessary to solve a given problem, and the use of models for purposes other than direct prediction are discussed.

CHAPTER I BIOLOGICAL SIMULATION MODELS IN PROBLEM SOLVING

Introduction.

There is currently a rather wide discrepancy between the complexity of information which is available concerning the behaviour of ecological systems, and the highly simplified abstractions from this body of knowledge which are used as a basis for answering practical questions of resource management. As the pressure on renewable resources increases, it becomes progressively more important that we understand both the extent to which proposed management practices will have effect, and the secondary repercussions which they might instigate. It is clear that in order to fulfil these requirements we must give attention to the complexities of the interaction between the resource and its environment. The conceptual models which for so long have acted as the backbone of decision-making have only a limited scope for such development. The need is for mathematical models which permit the inclusion of relatively complex biological information, and facilitate the derivation of

realistic solutions to particular problems of management.

Modelling

A mathematical model is built by observing the real system to discover those aspects of its structure which are measurable. Looking at a forest, we can measure the weight of the trees, the depth of the soil, the number of pigeons, and the daily rate of precipitation. These measurable entities we call the parameters of the system (or the 'state variables' in systems analysis terminology). From this list of parameters it is then possible to define which of them are, for particular purposes, constants, and which are variables. Next, it is necessary to consider each variable in turn, and to suggest the processes which act to change its value; photosynthesis is the process acting to increase the weight of the trees, while birth and death rates affect the number of pigeons.

Each process which can be recognised as causing important changes in the listed variables is then examined, and the factors which affect its rate or intensity are noted. Light intensity and air temperature affect the rate of tree photosynthesis, while the availability of food affects the birth rate of the pigeons. The relationship between a given factor and the process which it affects is then quantified by supplying a data-set to describe it. The data set can be in the form of a graph or a table, or an equation which summarizes either of these; it is obtained by experiment or from the literature. We can picture the chain of events in model-building as a flow-diagram representing the sequential definition of the different aspects (Fig. 2).

The variables are now represented by symbols, which can

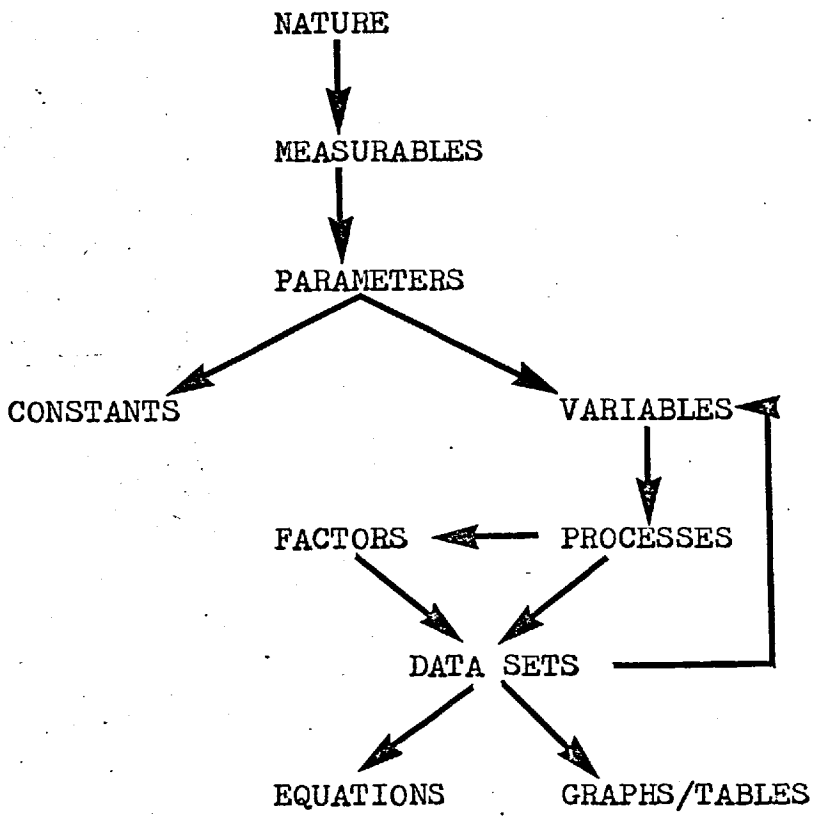


Figure 2. The chain of events in modelling. The choice of which parameters to measure, and the number of parameters which are necessary, is made in relation to the needs of the problem being tackled.

be verbal (e.g. "the weight of trees"), written abbreviations (e.g. WTTR as used in many computer languages), or single algebraic symbols (e.g. 'w'). These symbols then represent the numerical value of the variable at any time. Relationships between the factors operating in the system and the variables we have isolated (the processes) are then expressed as functions. A function describes the way in which a variable X maps onto a new value under the operation of the process; we say that $X \longmapsto f(X)$, where $f(X)$ - read as f of X - is the function describing the effects of the process on X. We can write functions without specifying their precise form, as

$$Y = f(X)$$

or we can state the shape of the function, and write, for example,

$$Y = 1 + 2X^2$$

With the variables and functions so specified, it is possible to build the mathematical model. We express the change in each variable as a function of those factors which affect it, and so, if we supply starting values for each variable, it should be possible to trace the course of the system components through time. The equations of change can be written as differentials; e.g.

$$\frac{dW}{dt} = (1 - b)W$$

or as differences, which express the values of the variable at a number of discrete times; for example, today's weight is a function of yesterday's

$$W_t = W_{t-1} . e^r$$

We shall examine these aspects in more detail later.

There are two main reasons for building a model of a system. The most common usage is in assessing the extent to which the synthesis of hitherto fragmentary information on the behaviour of individual variables and processes gives a realistic picture of the dynamics of the whole system. We can call such models 'comparative' because the behaviour of the real system is known and the model strives to mimic this behaviour (see, for example, many of the papers in modelling symposia like Jeffers, 1972; Patil et al., 1970). Second, models can be built to investigate the effects of different types of intervention on the behaviour of a system. These models are concerned with the response of the system to stress, and with the types of stress to which systems are most sensitive; we can call these 'manipulative' models (e.g. Forrester, 1961; Goodall, 1967; Crawley and Westoby, 1970). Here, the outcome is not generally known in advance, and the model is used to investigate the effects of the manipulation as best it can with the information currently available. The fundamental distinction between the two types of model is that the former tends to deal with systems at or near 'equilibrium', while the latter (because it considers the effects of stress) tends to deal with extremes.

Examples of manipulative models are fewer than examples of comparative models, but it is not possible to say whether this reflects a real imbalance of effort, or rather a feeling that manipulative models are somehow less scientific (either

because they are directly useful in management - too applied - or based on the synthesis of processes and data sets which are not fully understood - too speculative).

The structure of a problem-oriented, manipulative model should be related to the type of solution required; a problem demanding accurate numerical predictions will be modelled differently from one aiming at a qualitative or synthetic understanding of the problem. This distinction is expanded in Chapter VI.

Types of Model

Having made the decision to investigate a problem by modelling, the biologist is faced with numerous types of model from which to choose. Every model has four basic attributes: namely its form, its time-base, its units, and its determinism. Each attribute can be tackled in a variety of ways, and one model can be described in terms of the method used in each of these classes.

Model form refers to the way in which the structure of the real system has been represented. In ecology the most common forms are algebra and digital simulation. Analogue models have been used, but their applicability is rather limited (Denmead, 1972).

The majority of ecological problems are concerned with changes through time. In nature some changes are continuous (e.g. the variations in air temperature), and others are discrete (like the occurrence of rain). A model can simulate the passage of time as a continuous process by using differential equations, or as a discrete process (such as the succession of weeks in a year) by employing difference equations.

The elements which make up an ecological system can be modelled in two ways. They can be considered as individuals or as assemblages, and we might build a model to consider the behaviour of each animal in a population, or consider the population as a whole. The former would be a 'one-to-one' model and the latter 'statistical', in the sense that it considers population-means rather than individual values.

Finally, a model can be built in a deterministic fashion, so that each cause has a unique, invariant effect, or it can be made stochastic or probablistic, in which case a particular cause only produces a given effect with a certain probability. This distinction is very important when small populations are considered. The types of model attribute are summarized in Table 1.

a) Analytic and Simulation Models

Analytic, algebraic models possess a number of advantages over simulation models both with regard to their construction, and the analysis of their behaviour. Terms are grouped and expressions simplified, so that the model is more simple in appearance than its simulation counterpart. From the simplified structure the effects of a given variable on the output can often be seen at a glance, and the conditions which exist at the limits will be easily determined. The cost of this simplification lies in the necessity of algebraic specification of the shapes of the included functions. To keep the algebra at a tractable level, it is usual to fit only the simplest curves; linear, exponential and logistic are the most commonly used. This means that the resulting conclusions depend for their validity on the extent to which the curves employed faithfully represent the data sets from which they

PROPERTIES			
FORM	TIME BASE	UNITS	DETERMINISM
Algebraic	Continuous	One-to-one	Deterministic
Digital simulation	Discrete	Statistical	Stochastic
Analogue simulation			

Table 1. The attributes of model structure. A model consists of one attribute from each property; for example we can have a discrete-time, one-to-one, stochastic simulation, and a continuous, statistical, deterministic analytical model.

were drawn. With few variables the errors involved may be completely negligible for all reasonable purposes, and in such a case it should be unnecessary to resort to simulation.

As the logical structure of the problem increases, so does the difficulty of treating it compehensively by analytical means. Again, it may be that the type of solution required allows such a treatment, by demanding relatively little in the way of qualitative understanding, and seeking only straightforward quantitative predictions. If, on the other hand, the solution demands the consideration of large amounts of biological detail in the form of functional relationships and complex data sets, then the algebra may become completely daunting if not insoluble. In this case, simulation models with their almost limitless capacity for logical structuring, and their highly flexible facilities for dealing with functions, limits, discontinuities and time-lags become more appealing (Holling, 1965a; de Wit, 1970).

Added to this is the ease with which field biologists can master the basics of simulation, even without a formal mathematical training. Fundamental biological ideas in the form of causal mechanisms and graphs can be synthesized into models quite readily. The main disadvantage of simulation models is that once constructed, they are singularly difficult to solve. Thet generate what Holling and Ewing (1970) have called a 'response-space', which has as many axes as there are variables in the model. To understand the shape of this response space (how the model output behaves under a whole range of conditions) necessitates many runs of the model, each contributing a single point to the map of the response surface. Points of equal output value can then be joined to

give a contoured outline to the surface, but in practice of course, this is only possible with one or two of the variables at a time (since we can olny visualize three-dimensional spaces). It is far more difficult to obtain general results with a simulation model because there are so many potential runs which could be made (see below); similarly, the behaviour of the model at the limits of the different variables must be determined by trial and error. The choice to work with an analytical or a simulation form should be made in relation to the extent to which the advantages of one approach outweigh its disadvantages.

Ecological systems posses a set of properties which act in addition to the sheer number of factors and processes involved to complicate both our intuitive understanding and our modelling efforts. Holling (1966b) lists these system properties as spatial and historical effects coupled with the existence of thresholds, limits and discontinuities in the operation of many of the consituent processes. His thesis is that the existence of these complexities argues for the use of simulation, rather than analytical models of system structure. This assertion is not particularly robust, however, since many of the best models of spatial and historical effects in ecology are analytical (see, for example, Skellam, 1951, on dispersal and Bailey, 1964, on historical effects in epidemics). It is certainly true that simulation models can deal with almost any type of systemic complexity, but it is far more realistic to suggest the use of one particular type of model in relation to the problem in hand, rather than to some preconceived idea that some models can handle complexity and others can not.

b) Continuous and Discrete-Time Models

Within an ecological system, the chain of events which constitutes its temporal behaviour shows both continuous and discrete elements. For example, the generations of many insect species are discrete, but within each generation the animals behave continuously from birth to death. In such a situation, it is clear that neither a wholly discrete, nor a wholly continuous model will perfectly describe the time-course of the system.

Models which use a continuous time scale are typically constructed from sets of differential equations; each equation describes the rate of change in one variable as a function of the current levels of a number of other variables. The equations are then integrated, and can be solved to give the levels of each variable at a given time. Examples of models of this kind can be found in Volterra (1926, 1931) and Lotka (1923, 1925).

If the differential equations can not be integrated then it may be possible to solve the model by resorting to difference equations, which specify a sequence of discrete numbers, and from which the nth term of the sequence may be computed once the first term (u_o) has been specified; e.g. Jeffrey, 1969 writes

$$u_n = 2u_{n-1} + 1$$

which can be solved to express u_n in terms of u_o by

$$u_n = 2^n u_o + (2^n - 1)$$

Analogous to this type of solution is the pseudo-continuous evaluation of a mixture of differential and difference equations by means of one of the special 'continuous' computer compilers like DYNAMO or CSMP. These systems give the appearance of producing continuous solutions without the modeller having to solve the differential equations himself, by employing numerical approximations (see, for example, IBM, 1969).

Despite the advantages of these languages (de Wit, 1970) the discrete time-base is the most frequently used in ecological studies. A time unit is chosen applicable to the problem under consideration, and each parameter is updated once per interval. If the output changes in response to the manipulation over a period of several decades, then a year might be an appropriate interval, while if the changes are observed after a few hours, a minute might be the best unit. It is necessary to bear in mind the assumptions which are implicit in discrete-time simulation, since, under certain circumstances, they will affect the way in which model behaviour must be interpreted.

First, it is assumed that those processes which operate quickly relative to the chosen time-unit are in an equilibrium state, the level of which during any given time period being determined by the factors in operation. Second, those processes which change slowly are assumed to be constant (Levins, 1970). Third, and of considerable interest, is May's observation (1973) that a continuous and a discrete time model of the same process may have completely different stability properties. Care must be taken, therefore, that the stability properties of the real system are not masked by the effects of the time-base.

c) One-to-one and Statistical Models

A population of animals can be represented in a model in two ways. First, one can store the information relating to each animal separately (its age, sex, weight, physiological condition, position in space, etc.); this is the one-to-one type of model, and each individual animal is represented by a set of variables within the computer.

The alternative is to consider the population as a unit, with a given abundance (the total number of individuals), and to supply mean values for the parameters of interest. This approach is much more economical in terms of computer storage space and ease of computation. It is, in addition, more theoretically appealing in many cases, because one-to-one simulations tend to represent specific, unique instances of a general theory which could be more lucidly stated in a statistical model.

As a general rule, it can be said that in those circumstances where there exists a credible, well-tried general theory for some aspect of population behaviour then this should be included, and the population modelled as a unit. If, on the other hand, we have several independently derived hypotheses concerning the behaviour of individuals within the population, and the theoretical implications of their synthesis are not immediately obvious, it may be useful to incorporate these hypotheses into a one-to-one model. After a series of runs with the simulation, it may be possible to generalize the results of the individual cases into a single regression, and, from this, to suggest a theory relating the new hypotheses (theory in the sense of meta-hypothesis). In later runs the one-to-one situation can be replaced by the newly discovered statistical relationship.

There are several cases, however, where one-to-one models are the more useful form. The commonest of these concerns situations in which some form of heterogeneity is being considered. We might have similar animals experiencing different conditions at the same time, by virtue of their spatial separation in an heterogeneous environment, or alternatively, dissimilar animals which behave in a qualitatively different way under the same conditions (i.e. where there is no 'mean behaviour').

d) Stochastic and Deterministic Models

Variability is one of the most characteristic aspects of biological data. The variation observed in most experiments and field trials originates from the genetic differences between individual organisms, and from the effects of environmental heterogeneity on their developmental processes. The decision which must be made in instigating a modelling effort is whether this variability should be included as part of the model structure, or whether the mean values of the parameters will suffice. The first choice will lead to the development of a stochastic or probability model, while the second involves the production of a deterministic set of equations.

The mathematics of stochastic theory are singularly complex (see, for example, Doob, 1953), but an introduction to the subject for the reader interested in its applications can be found in Bailey (1964) and Feller (Vol. II, 1957). In general, the need to employ a probability treatment of population behaviour only becomes critical either when the population is small, or one must deal with queues or the initiation of epidemics (Bailey, 1964). In most cases when the population is relatively large, the mean of the stochastic

output will approximate the result gained from a deterministic treatment.

Deterministic models are much simpler to construct, because one event inevitably produces the same result under given conditions. It is a philosophical point whether any events in an ecosystem are truly random (i.e. causeless), and it depends entirely on the problem under consideration whether we decide to model certain events as 'causeless', or model the mechanisms by which they are caused. Typically one treats sources of variation as random if they originate beyond the bounds of the system involved in the model; in ecology these are often the climatic inputs of radiation, air temperature and precipitation. While the main body of the model might be deterministic, it is quite common that a stochastic system of climatic driving variables is employed. The advantage of this approach is that the effects of the random inputs - the noise in the system as they are sometimes known - can be readily appreciated in terms of their quantitative and qualitative influence on the output.

This type of stochastic element is relatively simple to include in a model. One first specifies the shape of the probability function associated with a particular variable at a given time. The two most commonly used shapes are the rectangular and normal distributions shown in Fig. 3. Next, the range of variation is stated, either in absolute units or as some fraction of the supplied mean. The probability distribution is then superimposed on the degree of variation, to give a graphical representation of the likelihood of appearance of any value of the parameter. A random number between 0 and 1 is then used to point out a particular value

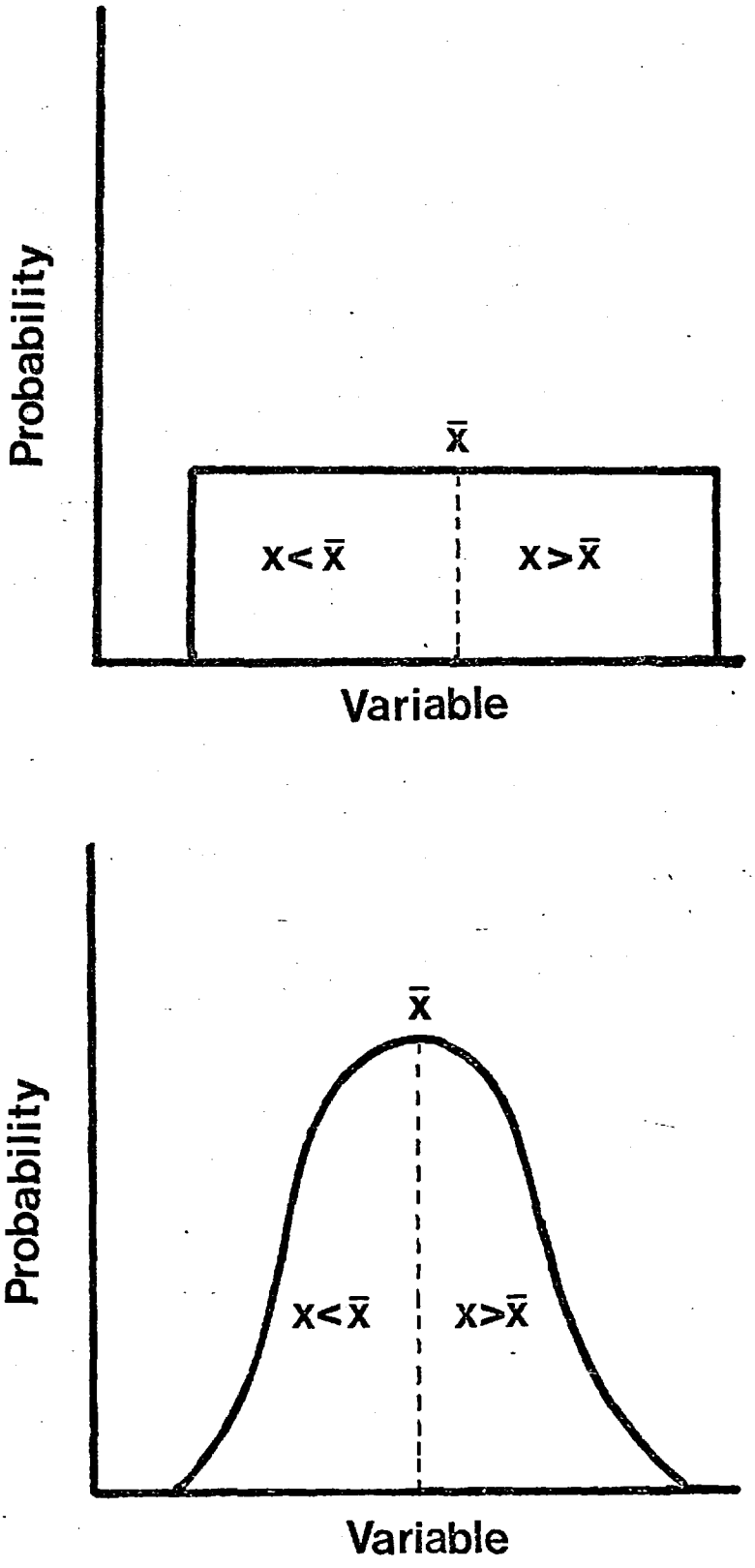


Figure 3. The rectangular and normal probability distributions. The probability of observing a particular value is proportional to the height of the curve above $\bar{X}$.

from the range of possibilities so that with the rectangular distribution all values are equally likely, and with the normal distribution a particular will be chosen with a likelihood of

$$\sigma^{-1}\ 2\pi^{-\frac{1}{2}} \exp\left[-\frac{[\gamma-\mu]^2}{2\sigma^2} \right]$$

from Kingman and Taylor (1966), where σ and μ are the standard deviation and mean respectively. Most computer systems have built-in functions for evaluating the level of Y when supplied with the mean and standard deviation; for IBM machines these are RANDU and GAUSS, and for CDC systems, RANF and GAUSS.

In much the same way, it is possible to build structurally stochastic simulation models, by overlaying probability distributions on the included data sets. Instead of picking random variables to 'drive' the model, each function is given a random element, so that instead of writing

$$Y = M * X + C$$

as in the deterministic case, we specify the mean of the function by this equation, and select an actual value by using a random number to point to a particular deviation. In Fig. 4 we see the curve Y = M*X+C with the probability distribution superimposed; the random number R points to a deviation of +12% so

$$Y = \bar{Y} * 1.12$$

or, in general,

$$Y = \bar{Y} * (1 + Dev)$$

where Dev is the deviation indicated by the random number R.

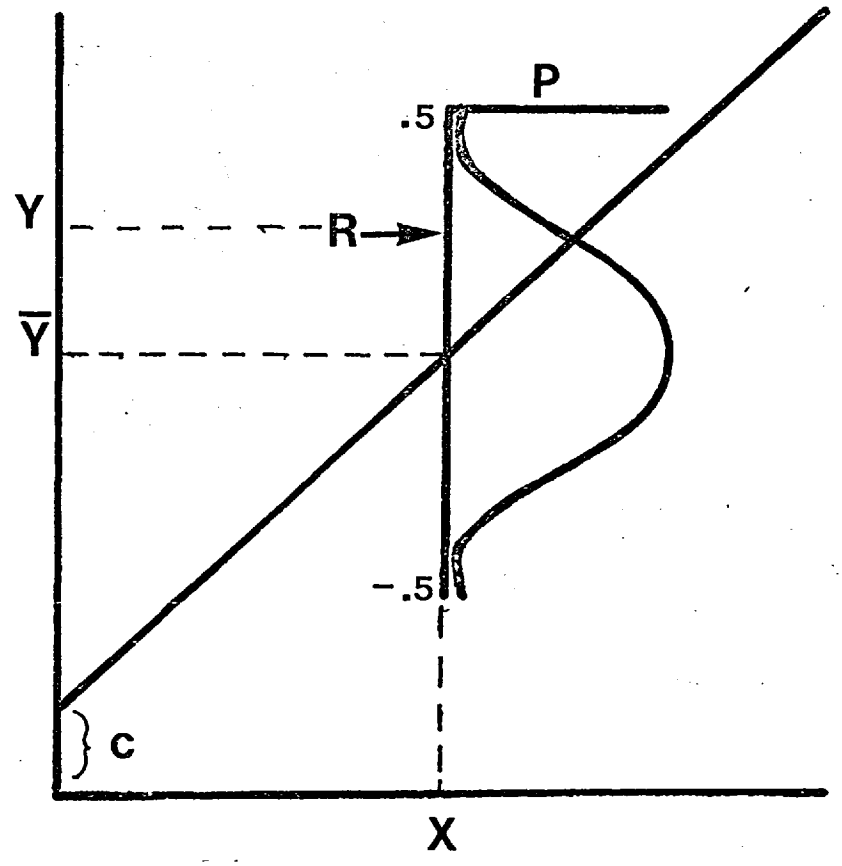


Figure 4. The probability distribution P is superimposed on the function Y=M*X+C to account for variability in the data. A value is selected at a given X by computing mean Y then using a random number R to select a deviation. Then $Y = \bar{Y} * (1 + Dev)$

Stochastic simulations are quite straightforward in their construction, but again, they only produce particular solutions for given values of the random numbers used. To obtain a picture of the behaviour of the model under a range of conditions it is necessary to run the simulation with several sets of random numbers. Fig. 5 shows an hypothetical case, with the deterministic output compared with a number of stochastic runs. The output from analytical stochastic models are 'expectations' for the value of the variable, while simulated stochastic systems produce a range of numerical values which could, if sufficient runs were made, be considered as a graphical representation of the expectation function.

Biological Simulation Models

A biological simulation model consists of a set of difference equations (or, less commonly, differential equations) which are amenable to numerical solution, and which deal with the mechanisms of the biological processes operating in the system to some degree of detail. We shall now investigate their structural properties and how they are built, and their quantitative properties and how the behaviour of the model can be assessed and applied.

A. Structural Aspects

1 Problem Definition

Probably the most important aspect of biological simulation modelling is the definition of the problem to be solved (or investigated) by the model. A model built to tackle a precise problem will have the inputs and outputs specified, and the range of conditions under which its

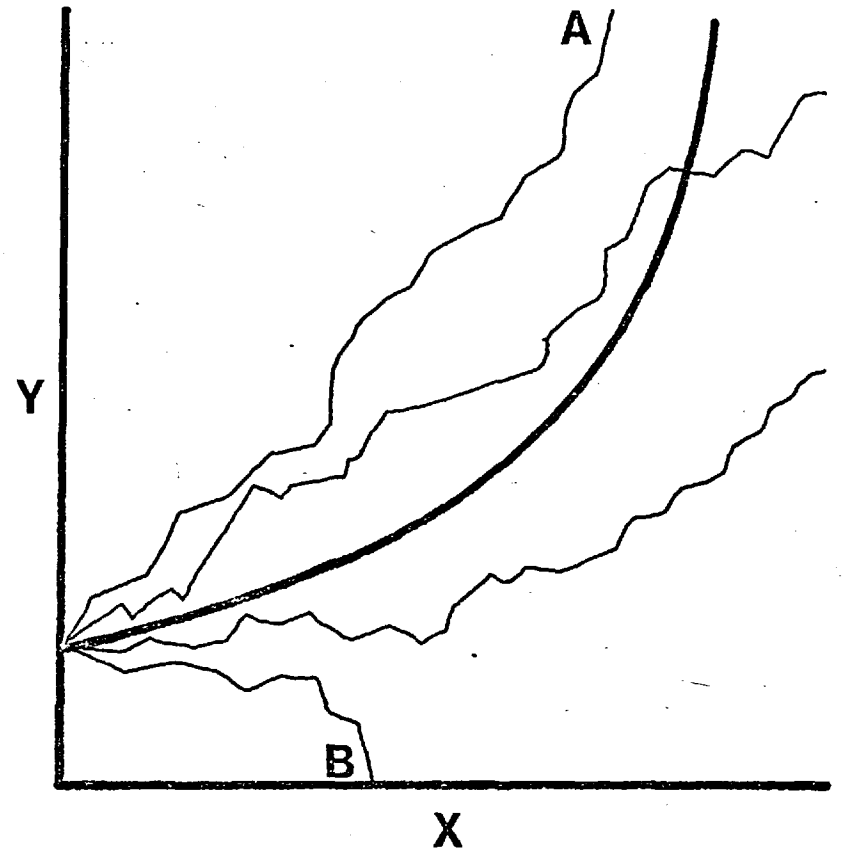


Figure 5. The outcome of an hypothetical deterministic model (thick line) compared with a number of stochastic outputs. Curves A and B show the extremes of the range of possible stochastic effects.

behaviour might be expected to apply defined. In contrast, a model defined to tackle a rather loosely defined problem will be vulnerable to misuse both with respect to input changes for which no suitable mechanisms have been included, and to use under conditions which it is not structured to consider.

A precise problem definition must, therefore, specify the inputs and outputs of the model, and the range of conditions under which the inputs bring about changes in the outputs. In this type of model, the inputs are generally the manipulations involved in a particular management practice, and the outputs are the aspects of the system of economic or social importance which the manipulation is designed to alter. The range of conditions specify the spatial, temporal and climatic constraints on the applicability of the model, and are therefore a measure of its generality.

In general, the problem can be stated as

What is the effect of X on Y when (constraints) ?

or, more broadly,

What are the effects of $\{X_i\}$ on $\{Y_j\}$ when (constraints) ?

where $\{X_i\}$ is a set of manipulations and $\{Y_j\}$ is a set of output variables. The list of constraints can be omitted from the question and included with the list of assumptions under which the model will be built; this will then serve as a frame of reference in interpreting model behaviour and applicability.

In the following chapters I shall consider the problem of determining the biological attributes of a predator species which is capable of reducing and maintaining populations of Aphis fabae at a low level so that no significant economic

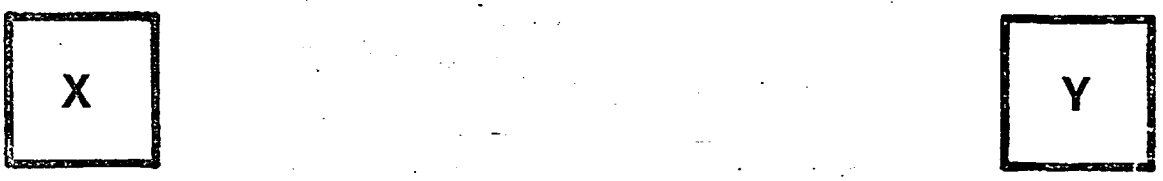
loss of broad beans (Vicia faba L.) occurs. Here, the manipulations are the biological attributes of the predator species (its fecundity, dispersive abilities, voracity and so on), and the output is bean yield. Implicit in the question is the constraint that any conclusions emerging from the model should be applicable to the field-crop situation.

There are two ways in which we could tackle this problem. We could be completely empirical, and perform field trials with all the available species which predate upon Aphis fabae to determine the most effective. This would be precise, but the solution would lack generality, and it would give us rather little insight into why the optimal predator species was the best. The alternative is to consider the biological components of the problem and to deduce from the information available those attributes which might be expected to be most important under most circumstances. This method lacks precision but it is general in the sense that it forms the basis of a synthetic understanding of the processes of yield maintenance by predatory natural enemies. I have adopted the latter approach not only in an attempt to solve the particular problem, but also to investigate the potential of this type of model-building exercise in producing suggestions and explanations which are of direct value in applied ecology. The rationale is that field experiments will tend to be more effective in situations where the synthetic nature of the system is appreciated.

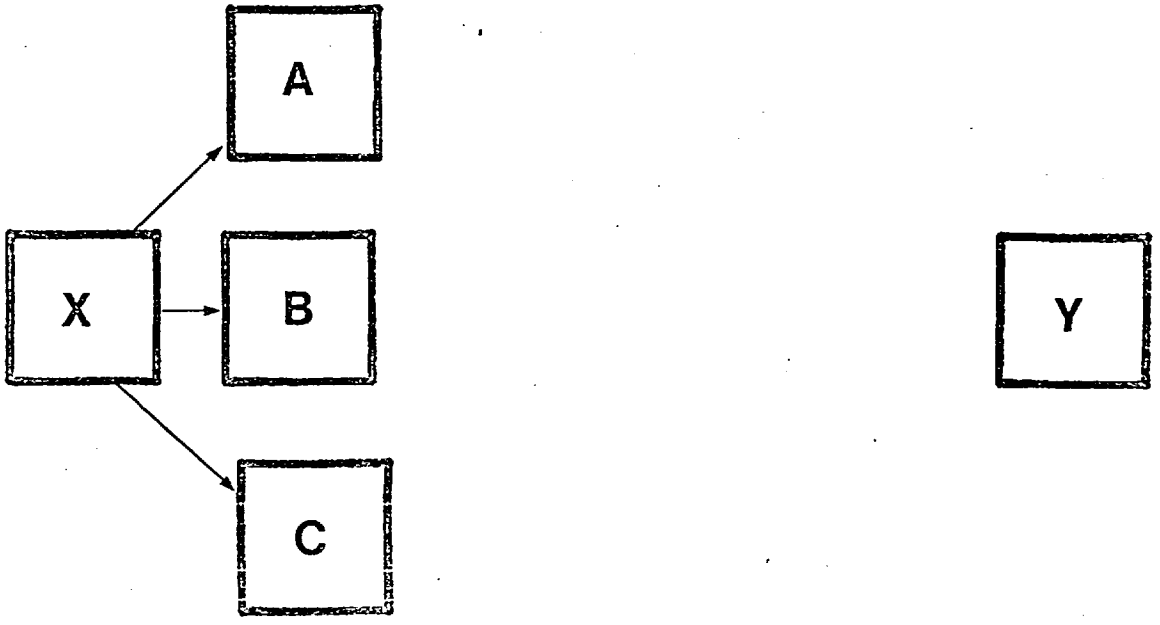
The second aspect of problem definition involves the selection of the variables to be included in the model. As we shall see, this process is of critical importance to the fulfilment of the stated objectives. The set of variables

should be sufficiently large that all important sources of variation in the output are considered, and yet small enough that analysis and interpretation are not hampered. Clearly the success of the exercise will depend upon the definition of what constitutes 'important variation' in the output; this in turn depends upon the clarity and precision of the question. One of the most powerful tools in deciding which variables to exclude from consideration in any particular question is the flow-diagram of cause and effect.

To produce a flow-diagram, we draw the manipulation(s) to the left of a page and the output(s) to the right; e.g.

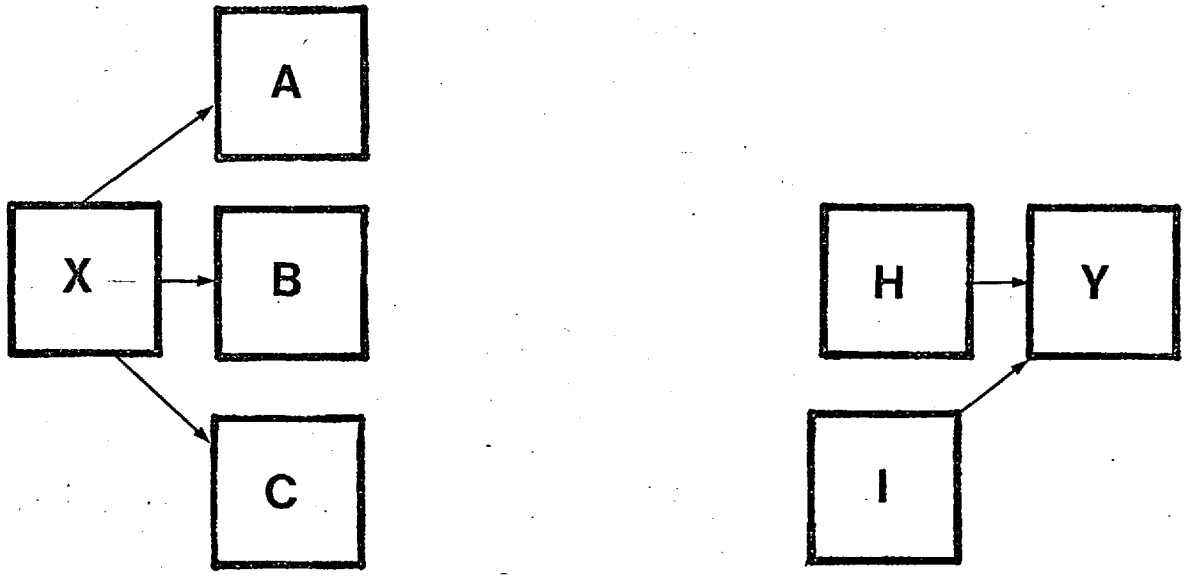


Now consider each of the manipulations (X) in turn and write those variables which are directly affected by changes in the manipulation to its right, joining them to it by arrows.



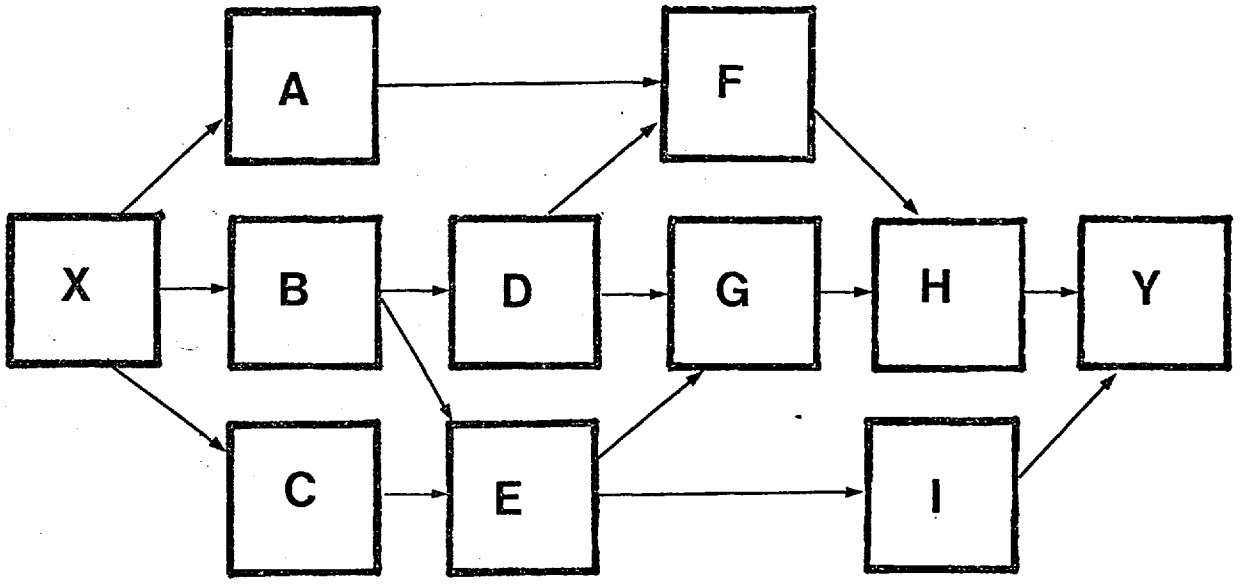
The arrow should point from affecting to affected, so the

arrow can be read as "X affects A" or "A is a function of X" (i.e. A = f(X)). Then, moving to the other side, we draw in those variables which directly affect the output to its left, and join them to it by arrows.



Taking each of the newly defined variables on the left, the procedure is repeated; those variables which they affect directly are drawn adjacent to them. If any of the variables affect oneanother, arrows are drawn between them, indicating by the direction the affecting and the affected. After a time, the variables from the left (in which changes will be induced by the manipulation) will link directly to variables which have ramified from the right (which, when altered, will affect the final outcome Y).

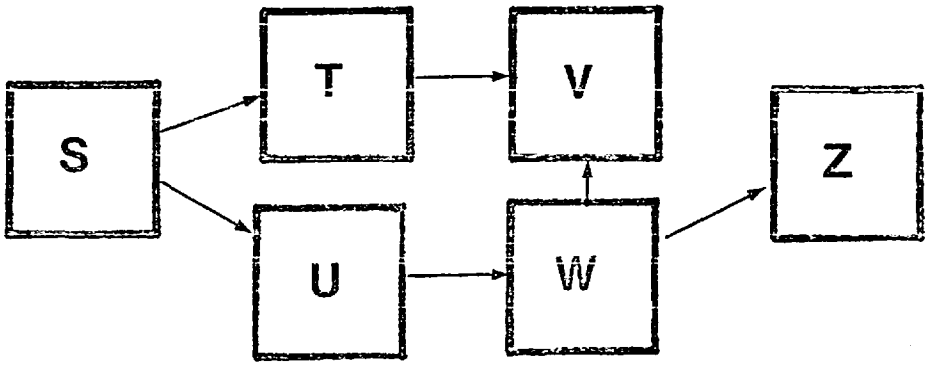
This procedure requires an understanding of the variables which are, or are likely to be, important in a given context. It is carried out by combining the experience of all interested parties; variables are included which are known to be affected by the manipulation, as are variables which are suspected as being important. The flow diagram at this stage



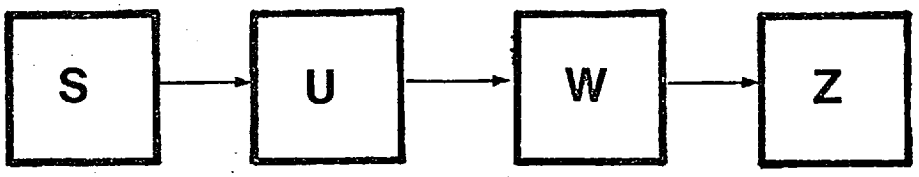
therefore represents a broad, qualitative synthesis of all the analytical understanding of the system relevant to the problem, irrespective of whether the relationships are proven.

Usually, there will be more inputs to the circumscribed system than the manipulation alone, and these must be added to the flow-diagram and connected to those variables which they affect. In ecological systems these driving variables (above) are typically meteorological; their variation is independent of the manipulation, but their effects may be profound.

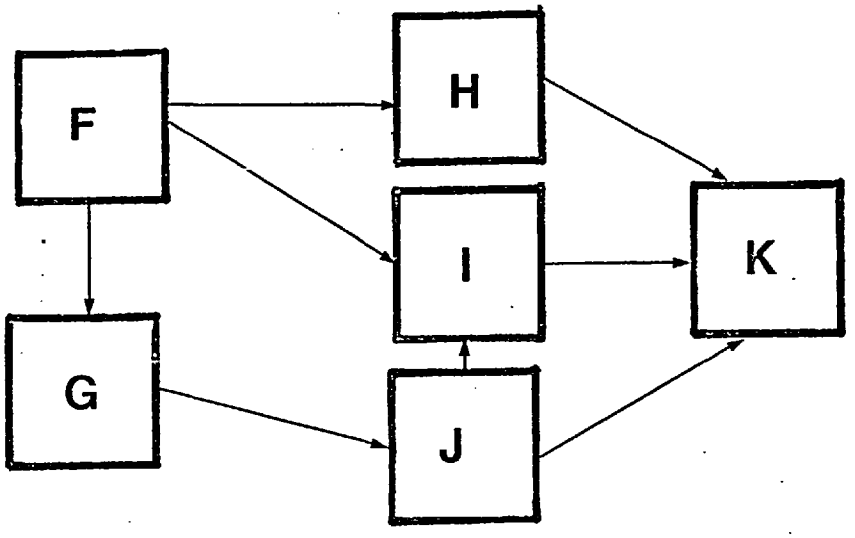
The next step in the simplification procedure consists of isolating those limbs of the flow-chart which are 'dead-ends'; finding those variables which affect none of the included compartments. These variables, and the processes by which they are changed (the arrows leading to them) can be ignored in future considerations. For example, if we have drawn



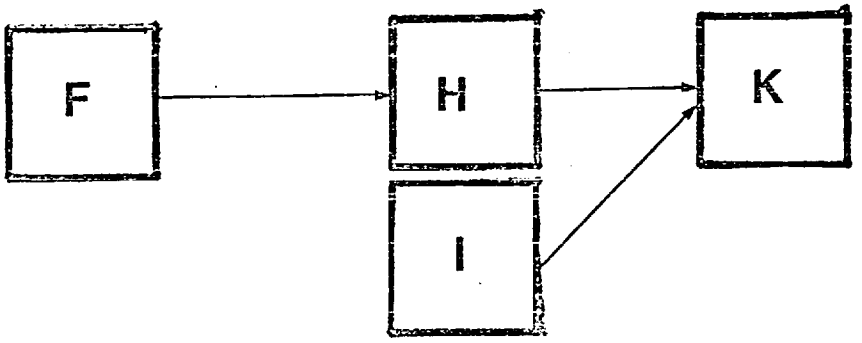
then because no arrows originate from V (it affects no other variables of interest), it can be struck out. We now notice that T need no longer be considered, and so it too is removed, leaving the simplified scheme



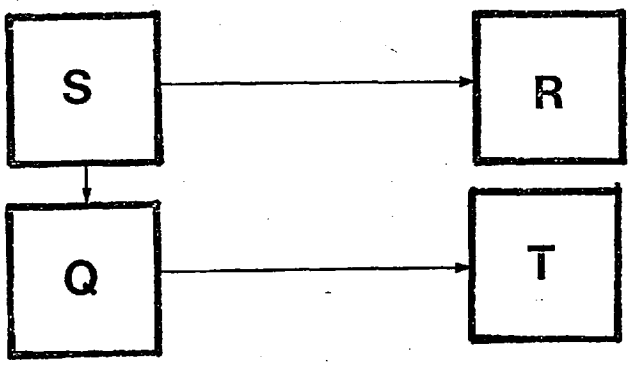
Further simplifications can be made by considering the time scale of the problem. Suppose that the question involves process which occur in the course of one year (say the growth of an annual crop). Then if one of the variables changes over a period of decades (or longer) we can consider it to be constant during any one year, and ignore the processes which change it. In the following flow-diagram we notice that I changes only very slowly, and so remove the arrow leading to it. Having done this, G and J become redundant (as above), so



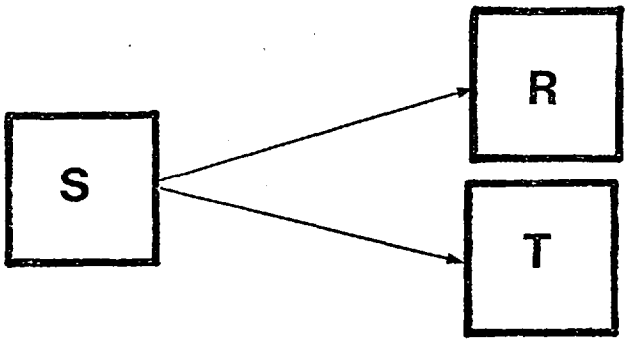
the flow chart simplifies to



Similarly, some variables in the sub-system may change very rapidly in relation to the time scale of the question. In this case we can consider that the variable is at an equilibrium level determined by its inputs, and remove the variable from the flow chart (instead of removing the process as above). The arrows leaving the compartment are joined to those entering it to produce the final flow-diagram. Suppose that Q changes in a cycle of an hour or so, depending on the level of S;



then we assume that Q is in equilibrium and imply that T is determined directly by S.



These time-scale simplifications should reduce the system to manageable proportions, and the question can be said to be defined. It will be clear, however, that the omission of processes and variables on the grounds of their rates of change limits the use to which the problem definition can be put. If, to return to the example, we were interested in the long-term effects on the ecosystem of repeated manipulations,

then it would not be valid to omit those processes which affected I (or any other slowly operating process). Equally, if we required to know the detailed day-to-day dynamics of the system under the manipulation, then we should have to include the variable Q (and all other rapidly changing variables).

The complete flow-diagram for the question considered in the following chapters is shown in Fig. 6. Its spatial scale ranges from the whole field to the node of the plant, and its time-base is the day. Changes within a day are not considered, and the model describes the system in terms of between-day variations.

This whole procedure operates by the erection of a set of hypotheses concerning each compartment; we isolate the variables which affect it, and those which are affected by it. In ecology, as compared to industrial dynamics (Forrester, 1961) for example, it is unusual that all the causes and effects are known, and very unlikely that they are all understood. Therefore the hypotheses incorporated into the problem-definition (which compartments are included and which arrows drawn) will only be as sound as the breadth of experience of the investigators. One of the main reasons for the discrepancy between the amount of information which is known and the amount which is used in solving management problems, centres about the difficulty of using material which is 'patchy' in relation to the question as a whole. The need is for a means of including, and recognising the effects of including, tentative and speculative information in the model so that the network of cause and effect is maintained intact, and along with this, the integrity of its behaviour as a system (Weiss, 1969).

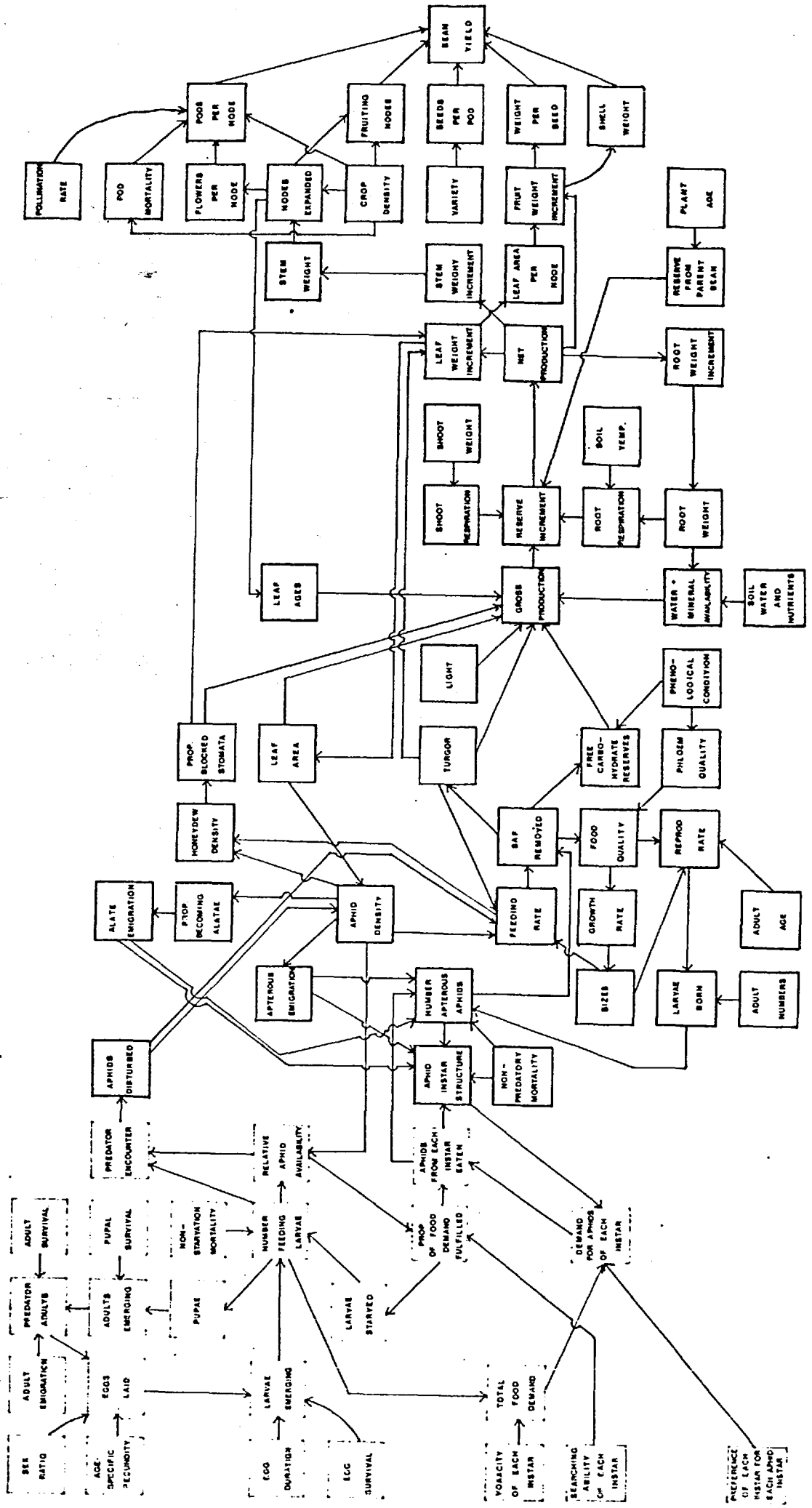
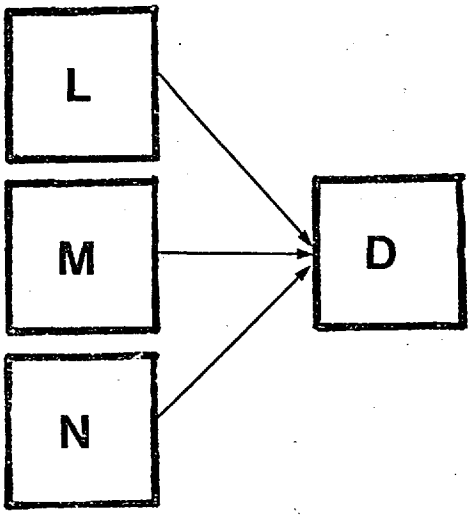


Figure 6. Flow diagram of the entire problem

In addition to isolating the processes which occur in the model system, it is necessary to consider the way in which they interact with oneanother in those cases where one variable is seen to be a function of two or more factors. The flow-diagram must be scrutinized to assess the number of such interactions, and each examined in turn. If the diagram shows



we can write

$$D = f(L,M,N)$$

In this general form it is not necessary to specify the degree to which the levels of L, M, or N affect the value of D, but neither is it possible to predict a value for D. To do this we require two more operations. First, we must suggest the effects of each variable upon the level of D by writing

$$D = f(L),\ g(M),\ h(N)$$

Then it must be stated how these three functions interact, so that given three values for f(L), g(M), and h(N) we can calculate D. This is where the principal difficulty lies. The simplest procedure is to define the maximum level for

the response of D for just one of the functions, say,

$$D_{max} = f(L)$$

and then plot g(M) and h(N) on relative scales, so that when M and N are optimal for D we put g(M) and h(N) = 1, and when M and N are at levels which are minimal with respect to change in D we put g(M) and h(N) = 0. For example, if L is such that D_{max} will be 6.0, we might have levels of M and N of 20.6 and 0.007 respectively, so that when M has this value D can achieve 76% of its maximum defined by f(L) when h(N) = 1, and when N = 0.007, D can reach only 98% of its maximum when g(M) = 1.

Clearly, since the levels of M and N are both sub-optimal for D the actual value of D will be less than D_{max}; i.e. less than 6.0. The problem which must be confronted is whether D responds to M and N such that only the least optimal factor is important (Liebig's 'law of the minimum'; see Odum, 1959);

$$D = f(L) * \min(g(M), h(N))$$
$$D = 6.0 * 0.76 = 4.56$$

or whether D is reduced to an extent determined by the mean sub-optimality of the two factors;

$$D = f(L) * (g(M) + h(N))/2$$
$$D = 6.0 * (0.76 + 0.98) /2 = 5.22$$

or, finally, whether both factors interact so that D is reduced proportionately by both g(M) and h(N);

$$D = f(L) * g(M) * h(N)$$
$$D = 6.0 * 0.76 * 0.98 = 4.46$$

The only way of choosing one procedure over the others

is to examine the data rather carefully; as the factors become increasingly sub-optimal so does the difference between the three treatments.

In some circumstances it may be possible to employ the results of a multiple regression directly in the model, so we could write

$$D = a + bL + cM + dN$$

where a, b, c and d are the constants fitted to the data in a linear regression, or

$$D = a + bL + b'L^2 + b''L^3 + cN + \ldots + d'''N^4$$

in the case of a polynomial multiple regression. If this method is employed, the limitations which apply to multiple regression models must be borne in mind (Mead, 1971; Watt, 1968; Yarranton, 1971).

2 Coding

To make the flow-diagram of system structure comprehensible to a computer it must be translated into a set of instructions. Typically, these instructions or 'statements' are written in one of the modern 'high-level' computer languages, which allow the programmer to indicate quite complex computational procedures very simply. Two types of computer language are currently available. The first, represented by FORTRAN, ALGOL and PL/I are discrete-time computer languages which operate by a stepwise updating of variables. The second type of language is less widely used (see de Wit, 1970), and treats time in a pseudo-continuous fashion. CSMP (Continuous System Modeling Program) and DYNAMO have the built-in ability to solve integral and

differential equations, as well as a very simple scheme of input and output. I shall restrict further discussion to the discrete-time languages, and to FORTRAN in particular, as this is by far the most commonly used computer code in biological work.

a) Variables

Each variable in the system to be modelled is represented by a box in the flow-diagram. To each we must now assign a unique name; in an algebraic model this would usually be a single, lower case Latin or Greek letter, but in FORTRAN we are allowed to use words of up to seven letters in length. It is useful if the variable name can be made to spell the variable it represents, or to act as a mnemonic for its name, because the comprehensibility of the computer code will be greatly enhanced. Some examples are shown in Table 2.

VARIABLE	ALGEBRA	FORTRAN
Aphid dry wt.	w	APHWT
Day-length	d	DAYL
Probability of rain	p	PRAIN

Table 2. A comparison of variable names and their intelligibility in algebra and FORTRAN.

b) Vectors

It is often found that several variables can be grouped into a vector. For example, the number of aphids of one day old, two days, three days, and so on, can be grouped into a vector a_i being the number of aphids in the ith day-class.

In FORTRAN vectors are represented by arrays; each array has a unique name of the same type as the scalar variables, but it also has a specified length - the size of the array. The names of arrays and their specific dimensions are declared at the beginning of the model as

```
DIMENSION APHID(25), DENSITY(5,5)
```

This dimension statement sets aside storage within the computer for 25 age classes of APHIDS, and creates a matrix (a two-dimensional array) of 5 rows and 5 columns to store information on aphid DENSITY.

Arrays are used by making specific reference to their individual elements in turn. If I is an integer whose value lies in the prescribed range of the array we can write

```
APHID(I) = 100.
B  =  APHID(I) * FR
```

In other words, we can perform arithmetic with or upon any element of an array, as long as we specify the subscript I.

c) Processes

By supplying names for the variables and vectors employed in the flow-diagram we have specified the bones of the model. It remains to inform the computer of the nature of the processes by which changes in the variables come about. We know from the preliminary analysis those variables which influence the behaviour of others, and we can formalize this somewhat by expressing each variable as a function of the names of its affecting factors. We can translate the flow-diagram quite literally, then, into an ordered set of equations,

in which all factors appearing to the right of the equals sign (=) have a known value. For example,

```
AGE = 10
FECUND = f(AGE)
EGGS  =  f(FECUND)
BIRTHS  =  APHIDS(AGE) * EGGS
```

is such a string of descriptive ordered equations leading to the calculation of the number of births from the tenth age class of aphids. The processes are expressed here in a purely descriptive way; we are saying that fecundity is a function of age, but not whether it increases or decreases, nor what the actual numerical values will be. This aspect is covered in part B of this section.

d) Timing

Most models dealing with ecological problems are concerned to trace the effects of a manipulation through time. It is necessary, therefore, to decide upon a standard time interval in which to code the model. Generally, the nature of the problem itself will guide this choice; for instance, the effects of shading a leaf on its rate of photosynthesis will be manifest within a minute, whilst the effects of building a dam upon the avifauna of adjacent areas may not be felt for decades.

In choosing a particular time interval we must bear in mind two points. First, that too large an interval will lead to considerable errors, because the model will make great leaps in its state from one time unit to the next, and second, that choosing too small a time scale will involve the consideration of processes whose operation is essentially irrelevant to our problem because they operate so quickly

relative to the processes of primary concern.

For many management problems, especially those to do with annual crops, a time interval of one day or one week is ideal. If important changes occur within each week, then a day will be the best unit, while in those cases where changes are most obvious between weeks, time intervals of one week should be used. The processes occurring within the model system can then be assigned units for their rates of operation; we express mortality as the fraction of the population dying per day, or in another model, precipitation as mm. per week. Once this has been done it will be clear at what frequency to collect any data which are required.

B. Quantitative Aspects

1 Data

Central to the ideal of simulation modelling is the relationship between the qualitative assessment of system structure, and the quantitative understanding of its behaviour. The qualitative information consists of an understanding of what affects what within the system, and by what means; it is the recognition of the processes which operate, and those factors which affect their rate of operation. The quantitative information, on the other hand, consists of an understanding of the extent to which changes in a factor affect the rate of the processes it controls. Normally, it is possible to produce a model structure which incorporates far more qualitative information than can be backed up with real data; more simply, our understanding of ecological systems is largely intuitive. In addition, it is unlikely that even our qualitative information is complete; we do not recognise

all the factors which can affect a specific process, and we may be unaware of certain interactions between compartments of the system. The benefit of using a computer to tackle complex problems lies in the ability of the machine to assess the consequences of a particular manipulation through many hundreds of simultaneous assumptions, a feat which is not possible in the human brain. It is reasonable to assert, therefore, that a computer model could take more aspects of the problem into account in arriving at a solution strategy than could a man faced with the same information. The fact that the information is patchy for a computer model does not make it any the less so for a mental deductive model.

Probably the most severe draw-back to the useful implementation of simulation models at the moment, seems to rest in our inability to make objective decisions as to the nature of information which it is fruitful to include, and that which tends to be superfluous.

Amount of Data

As we have seen, there are at least as many data sets necessary to quantify the model as there are processes (arrows in the flow-diagram). For some of these, information will already be available in the literature, or in the unpublished files of workers in the subject. The data will exist in one of two forms. Raw data, presented in graphical or tabular form, display the results of experiments or observations unmodified. These can be used in a model directly when there is no replication (i.e. when there is simply a sequence of observations at different times, or at different levels of the independent variable). The process then reduces

to a string of values for the dependent variable, and the process can be coded by writing

$$Y = X(I)$$

where X is an array of data points, and I indicates the level of the independent variable. If the data represent replicated results (and hence we observe different values of Y at the same X) we can either summarize the data by some statistical technique (regression or other means of curve fitting), or by fitting a curve by eye. The curve is then expressed as an equation relating X and Y, and the process is coded as

$$Y = a + bX + cX^2$$

where a, b and c are the constants of a polynomial regression. Alternatively, the data may have already been refined into an equation which can be incorporated directly into the model (this should only be done after careful consideration of the implications of the equation; see chapter V).

The procedure of statistical curve fitting is often time-consuming, and can, if high order polynomials are employed, be quite misleading (Watt, 1968). It is usually much quicker, and just as precise, to fit a curve to the raw data by eye and, instead of trying to find an equation to describe the curve, to calculate the value of Y by interpolation. This can be achieved most simply by drawing the curve as a number of straight lines, and specifying the X and Y co-ordinates of each change in slope. The more straight lines employed, the better the fit of the curve will be: Fig. 7 illustrates the procedure.

The value of Y is then calculated for any X by finding the straight line below which the X value falls.

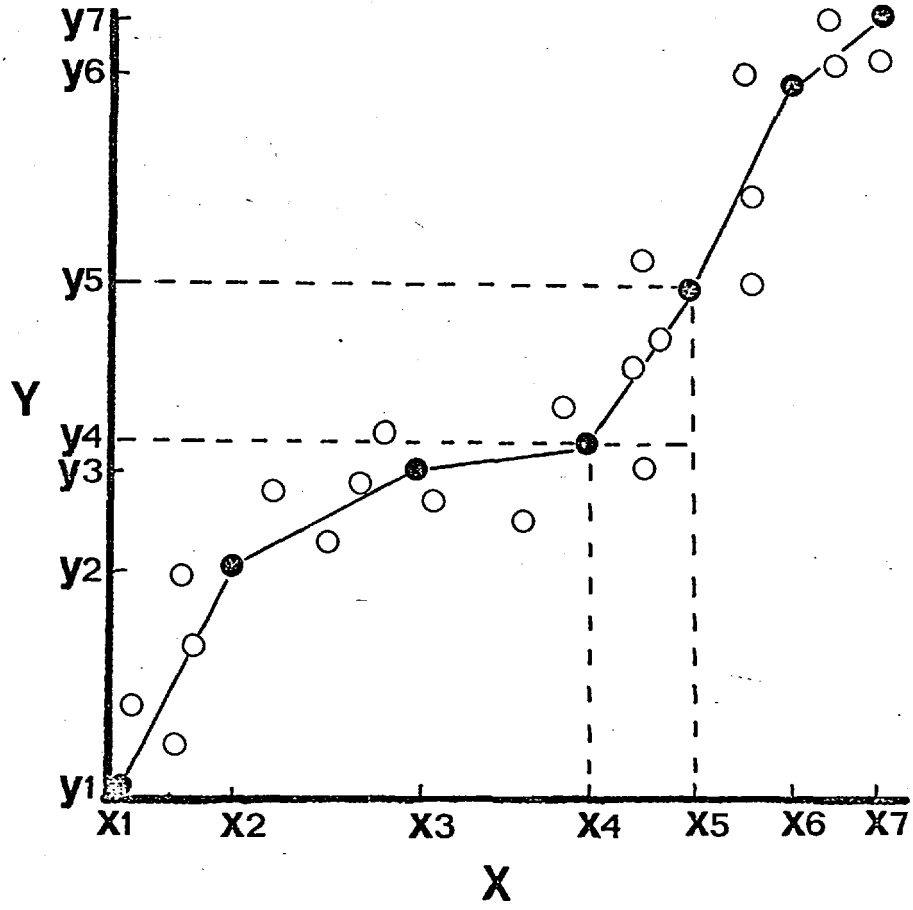


Figure 7. A scheme of curve fitting for linear interpolation. The open circles represent the raw data, and the solid circles the points selected to indicate gradient changes in the fitted curve.

Having done this, we have X_N as the greatest specified X value less than X, and X_{N+1} as the smallest X value greater than X. We then look up Y_N and Y_{N+1} associated with these points, and determine the gradient of the curve at this point by writing

$$m = \frac{Y_{N+1} - Y_N}{X_{N+1} - X_N}$$

The intercept of this curve on the Y axis (c) is given by

$$c = Y_N - m.X_N$$

so we can calculate the actual value of Y at X from

$$Y = m.X + c$$

For the remaining chapters I have assumed that this procedure is specified as a computer routine called 'F'. With this we can describe the quantitative behaviour of the processes in the model quite simply by writing

$$Y = F (X, XVAL, YVAL, NP)$$

Here, F is the name of the routine to perform linear interpolation, X is the value of the independent variable, XVAL and YVAL are arrays containing the points of gradient change on X and Y respectively, and NP is the number of points supplied. This is just a formal statement of our initial descriptive assessment of the process Y = f(X). The biology is quite unambiguous, and the precision of the representation can be judged by comparing the curve given by XVAL and YVAL with the original data. We do not have the problem (often associated with polynomial multiple regressions) of attempting

to supply some biological meaning to an X^4 term appearing in a regression equation.

There will be some processes in the flow-diagram, however, for which no data exist. Before initiating experiments to collect these missing data, it is often useful to build the model with intuitive data sets, and to rank the priorities of data collection in relation to the sensitivity of the output to changes in the values supplied. If small changes in a particular data set produce large changes in the output, it will be clear that not only should these data be collected by experimentation, but also that a high degree of precision should be associated with the estimation. For example, we may know that the generation survival of an insect from egg to adult is 20%. If the duration of this period is 50 days we can write

$$0.20 = \prod_{i=1}^{50} S_i$$

or, assuming that the daily survival S_i is constant from day to day,

$$0.20 = S^{50}$$

We can calculate the value of the daily survival rate which would give this result from

$$S = \text{antilog} \left(\log_{10} 0.20 / 50 \right)$$

which gives us S = 0.968324. We can now examine the effects of the precision of our experimental estimate of S on the final overall survival we would achieve. Suppose we could only

estimate S to two places of decimals; we should then have S = 0.97 which gives a generation survival of 21.8%; i.e. 9% out. The errors associated with each decimal place are shown in Table 4, and can be seen to increase exponentially as the precision decreases.

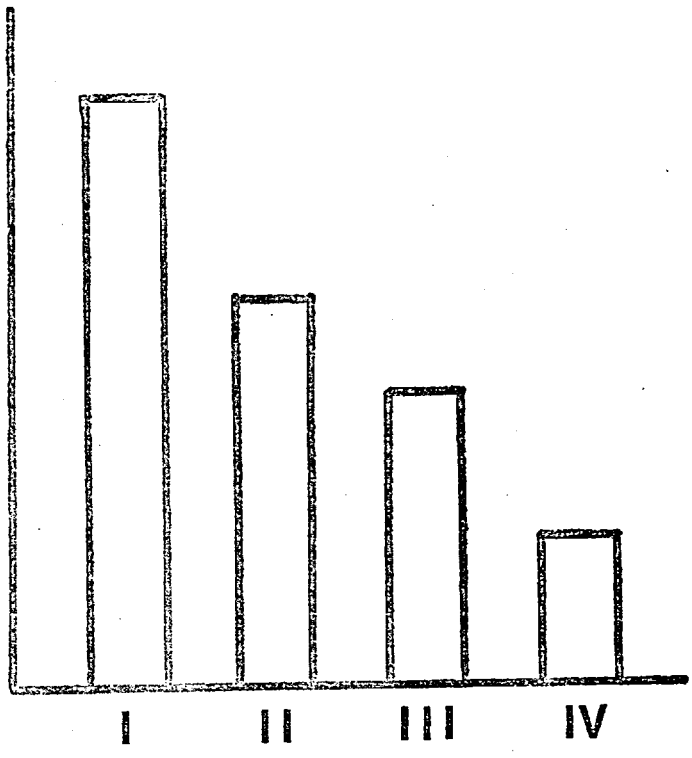
It is clear from Table 4 that intuitive data sets must be used with great care, especially if they are involved in multiplicative or power series, because very small changes in such data produce large changes in output.

In those cases in which the flow diagram contains many interactive processes, we need to know not only how each factor affects the end result, but also the extent to which the level of one factor affects the way in which the dependent variable responds to another. This type of data will require more sophisticated experimental and statistical techniques for its collection (see Section 2.d below).

An examination of the processes involved in the model will tend, therefore, to bring to light a continuum of data quality; some processes will be well understood, and there will be accurate quantitative information on their behaviour under a number of factors, while others will have information on only a limited array of parameters. To categorise the patchiness of information in the model we can draw a histogram showing the relative frequencies of a number of data types; let us say 'well understood', 'passably understood', 'poorly understood', and 'guessed' (see Fig. 8). Intuitively we could suggest that the more left-skewed the distribution in Fig. 8, the more confidence we might place in the predictions from the completed model. We are, however, interested in more than just the quantitative accuracy of the output, and

Estimate of S	+ 1 Place - 1 Place	Estimate of G	Error of 1 place (%)
	.968325	.200012	+ .006
.968324		.200002	+ .001
	.968323	.199992	- .004
	.96833	.200064	+ .032
.96832		.199961	- .019
	.96831	.199858	- .071
	.9684	.20079	+ .105
.9683		.19975	- .125
	.9682	.19873	- .635
	.969	.20170	+ 3.550
.968		.19668	- 1.660
	.967	.18678	- 6.610
	.98	.36417	+ 82.085
.97		.21800	+ 9.0
	.96	.12988	- 35.060
	1.0	1.00000	+ 400
.9		.00515	- 97.425
	.8	.000014	- 99.993

Table 4. The effects of 1 decimal place of error in the estimate of the daily survival rate S on the estimate of the generation survival G, at different precisions of the daily estimates (from six to one place of decimals). The true value of G is .2000000.



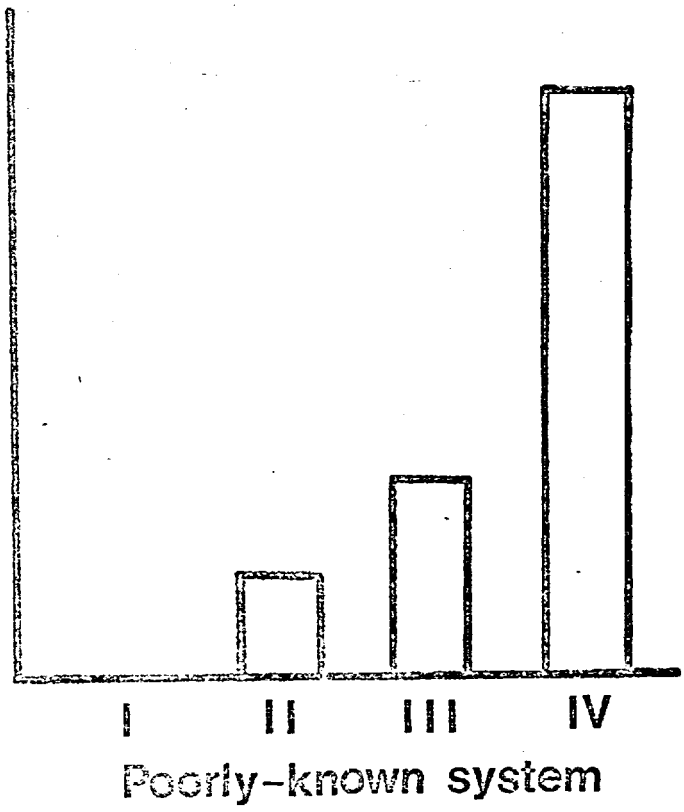


Figure 8. Hypothetical histograms for well-known (top) and poorly known (lower) systems. I=good; IV=guessed.

understanding the overall behaviour of the system in its response to certain classes of manipulation may be our principal aim. In this case it may not be the number of well-known parameters which is most important, but rather which specific relationships have been guessed. Here again, we need some guide-lines as to the classes of parameters which are most important in allowing realistic simulation of system behaviour.

2 Data Quality

We can define the quality of a data set in terms of four properties.

a) Fit

Fit is the attribute of a data set describing the degree of scatter of the raw data points about some fitted curve. If the curve has been determined by regression methods, then the fit can be said to increase as the sum of squares of the deviations of the individual datum points from the curve decreases. With an eye-fitted curve the estimate will be subjectibe; e.g. good, passable, poor.

In the model, the better the fit of a data set, the greater the confidence which can be placed in the prediction of Y from X. Similarly, a good fit suggests that the behaviour of Y in response to changes in X is well described by the equation, and hence our confidence in the qualitative behaviour of the model is enhanced. It is possible, for example, to draw many shapes of curve through a data set with a high degree of scatter, and although statistical tests may show these curves to account equally for the observed

variation, the model might behave quite differently depending upon the particular shape chosen.

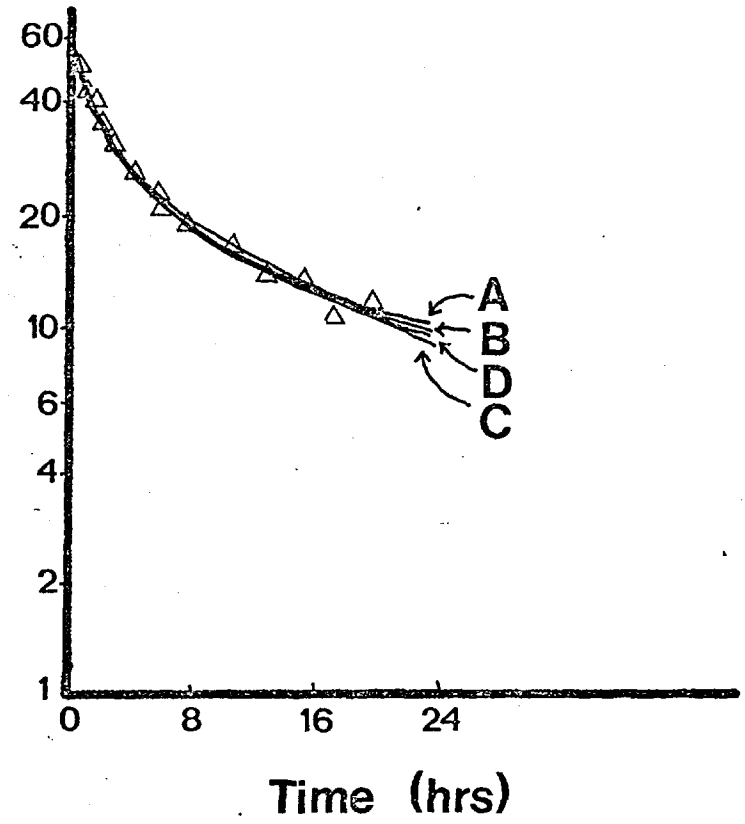
If it is thought that the variation in the data set is attributable to more than sampling error (i.e. there is another causal process in operation), we can either perform an experiment to determine the effects of the suspected factor on Y, or simply superimpose a suitable distribution on the fitted curve, and select a particular Y value by using a random number to indicate a deviation from the expected value (Stochastic Models, above).

b) Range

The range of a data set describes the fraction of the X axis which is covered by observed values. Many data sets obtained from the literature cover too limited a range to be used directly in the model, and we must decide whether it is permissible to extrapolate beyond the measured range, and, if so, to what extent.

Clearly, the precision of extrapolated graphs is determined in part by their fit. If the fit is good, then the curve is more likely to represent the true relationship, and therefore extrapolation will be more justified. Extreme caution must be exercised, however, as can be seen from Fig 9 in which all four curves fit the existing data well, but lead to widely divergent predictions when extrapolated (from Riggs, 1963).

Occasionally, we have information on a data set from beyond the specified range. In Fig 10, for example, we might know that the curve must pass through the origin. Do we join the lowest value of the curve to the origin directly with a straight line (A), or do we draw in some smoothed (though



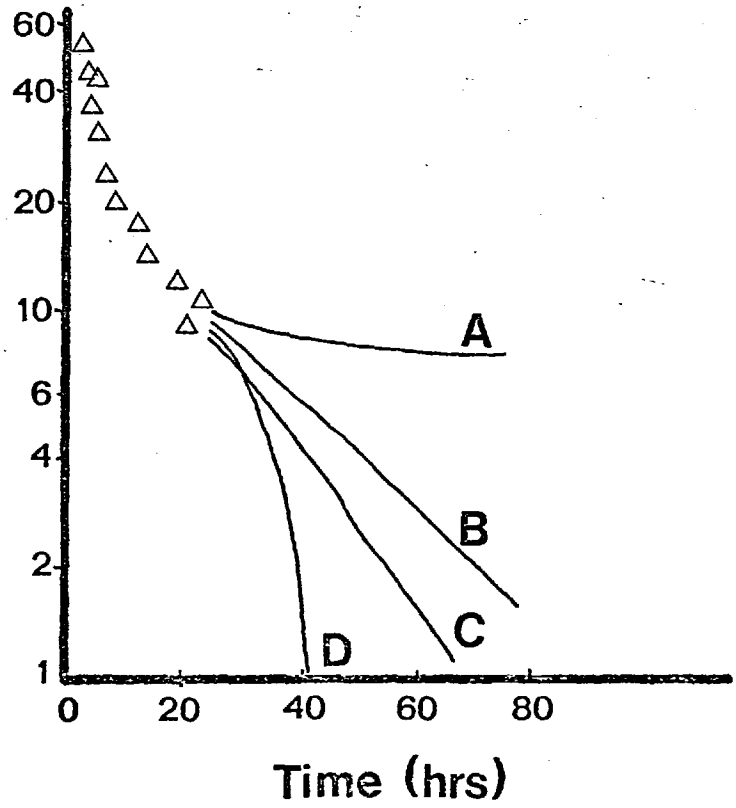


Figure 9. The dangers of extrapolation; all curves fit existing data well but lead to divergent extrapolations.

equally artificial) curve (B) ?

Again, we might know that Y has some maximum value which holds for all high values of X, but are not certain at what X value this maximum is first reached. We can not omit this information from the model, otherwise the extrapolated values of Y would become absurdly high, so some decision must be taken. The least dangerous approach is to extend the curve until the maximum value is reached, and then make Y independent of further increases in X (C).

In any event, all these alterations will tend to reduce the quality of the data set. The more constraints which we have to add artificially, and the further beyond the range we must extrapolate, the lower can be our confidence in the behaviour of the process within the model.

c) Conditions of Applicability

In many cases the data which are available to describe a process have been collected under different conditions from those in which the model will apply them. Perhaps they refer to a different species of the same genus, or to the same species in another part of the world. Certain processes, however, are rather robust, in the sense that most species in most areas follow similar trends. The general shape of the data set might well be applicable under almost all conditions, but could be x-shifted or y-shifted from one particular instance to another. We can define an x-shift as a variability in the position of a curve of fixed shape relative to the x axis, and a y-shift of a curve relative to the y axis. This is shown in Fig. 11.

When there is no indication whether the available data

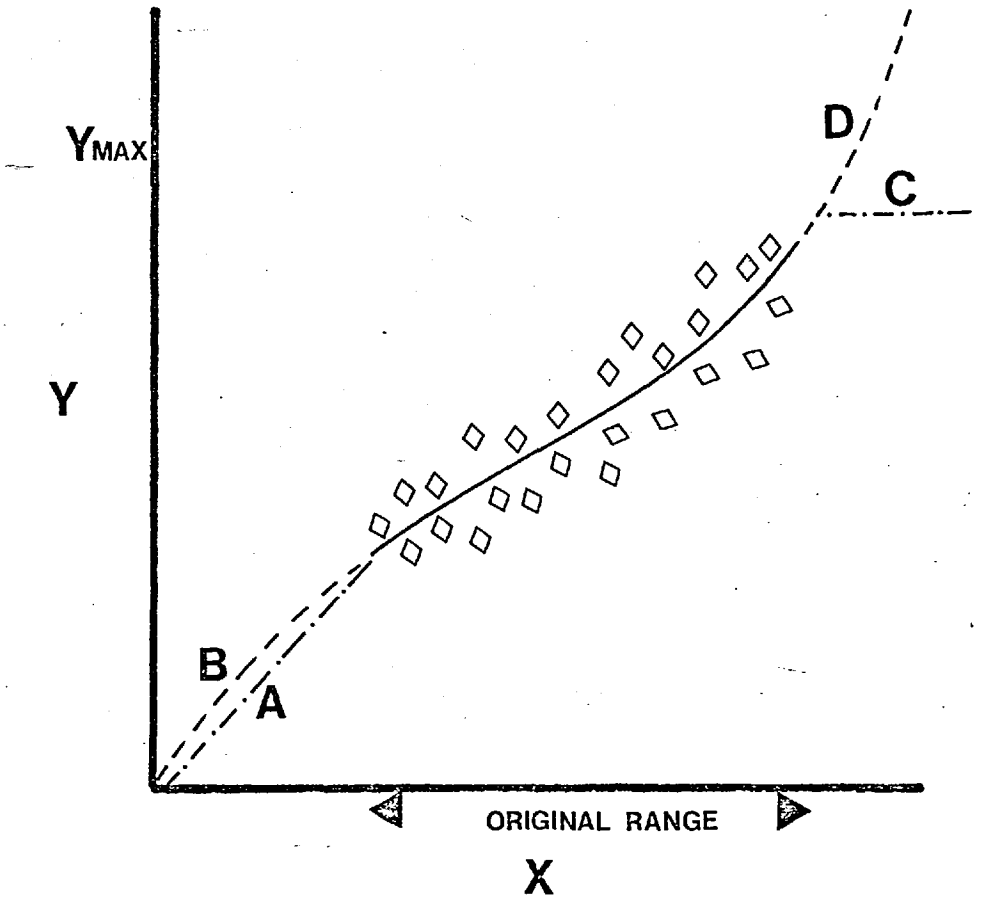


Figure 10. An example of a data set of limited range. The curve is known to pass through the origin (options A and B), and to reach a constant maximum value (D and C).

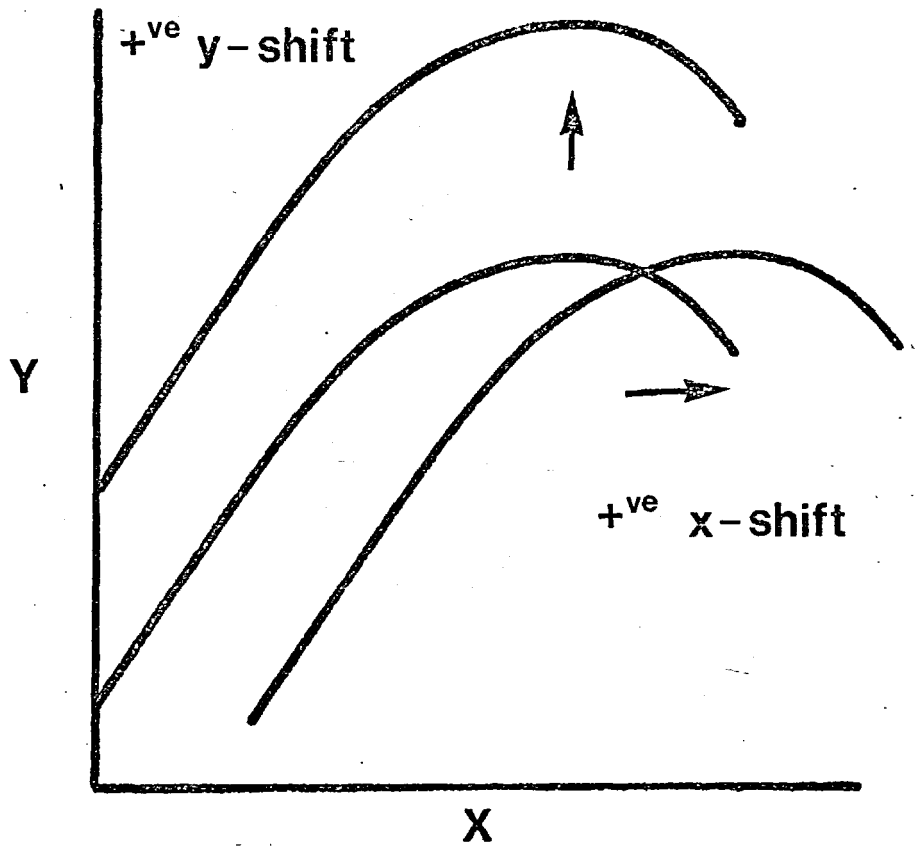


Figure 11. The idea of x-shift and y-shift in data sets of fixed shape. Different mean temperatures might bring about a y-shift, while different species in the same genus might show x-shifts.

set should be x- or y-shifted to bring it into line with our specific conidtion requirements, the data set should be left alone. In this case, its quality declines in relation to the difference between the conditions of collection and application.

d) Number of Interactions

Much of the information available in the literature comes from controlled experiments; all factors of the environment are held as constant as possible while X is varied, and the response in Y noted. The very fact that the experiments must be controlled shows how many other environmental parameters affect the level of Y.

I have already discussed the problem of representing the interaction of several factors in affecting the rate of a single process (Problem Definition, above). If we know that

$$Y = f(X), g(W), h(Z)$$

and we have controlled experiment data for each of these three effects on Y, then we can build some sort of model. The quality of the data, however, would be greatly increased if factorial experiments had been performed so that we could see the extent to which X, W and Z interact with oneanother. We can state that, in general, the quality of interactive data sets increases with the number of factorial combinations for which data have been gathered.

With a factorial data set of Y on X, W and Z our problems of modelling the interaction are significantly reduced. It should be possible in this instance to set up a four-dimensional array from the tabular data, and to perform multiple interpolation to obtain a value for Y, just as we did by one-

dimensional interpolation in the earlier example (Fig. 7).

These ideas about data quality can be synthesized in tabular form (Table 5). Here I have assigned a subjective rating to each aspect of data quality on a scale 3=good, 2= passable, and 1=poor. Summing over the four attributes, and dividing by four gives us the mean quality of a given data set. For example, if we have a data set with a fit (i.e. significance) of 90%, a range of 75% including the lower limit, collected for a different species in a different environment, but having no interaction associated with it, we score

$$\text{Quality} = (2 + 2 + 1 + 3)/4 = 2.0$$

While this classification is completely arbitrary, it may help us decide which data are contributing to the solution of the problem, and which are simply getting in the way.

Taking the model as a whole, we require a means of discovering the implications of patchy data quality on the way the model behaves, and on the measures we adopt to improve its behaviour. This involves some form of sensitivity analysis on the model. In the next section the topic of numerical sensitivity analysis will be discussed, but there is an important aspect of this procedure which can be carried out with the flow-diagram and the data sets alone.

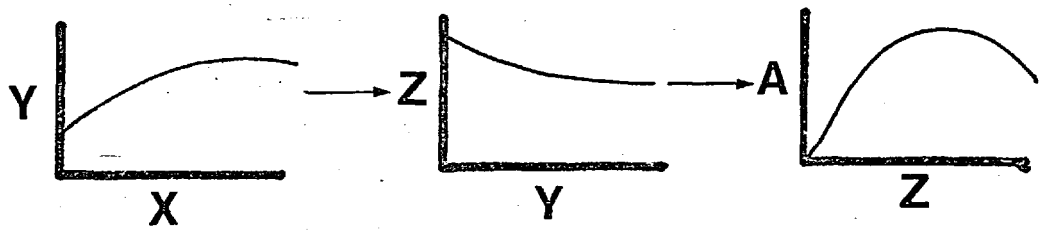
Once the model has been read into a computer, the quality of a data set is no longer taken into account, since all data are treaten in exactly the same way. This is unimportant as long as the data set is correctly descriptive of the process represented, but we must know at the outset which data are more likely to be both important and incorrect, as

ASPECT	QUALITY		
	HIGH (3)	PASSABLE (2)	POOR (1)
FIT	More than 95% confidence	More than 90% confidence	Less than 90%
RANGE	100%	80% including one extreme	Less than 70%; neither limit
CONDITIONS	Same species, same environment	Same species different environment	Different species and environment
INTERACTIONS	None	One	More than 1

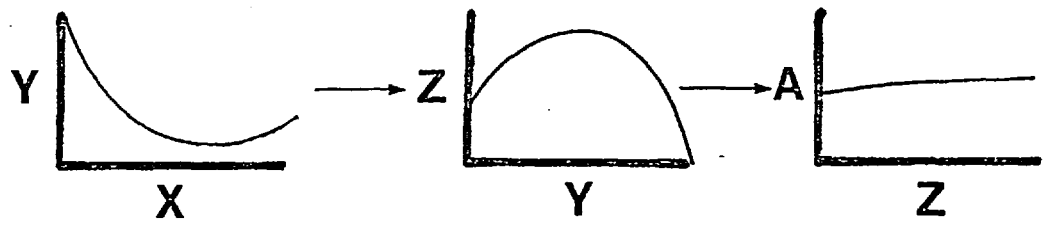
Table 5. A summary of the different aspects of data quality. For an example see text (page 67).

these may influence our entire assessment of model behaviour.

The initial sensitivity analysis consists of drawing out more or less discrete sections of the flow-diagram in a new form. Instead of drawing a chain of variables, we draw a chain of graphs, so that the dependent variable on one graph is the independent variable on the next.



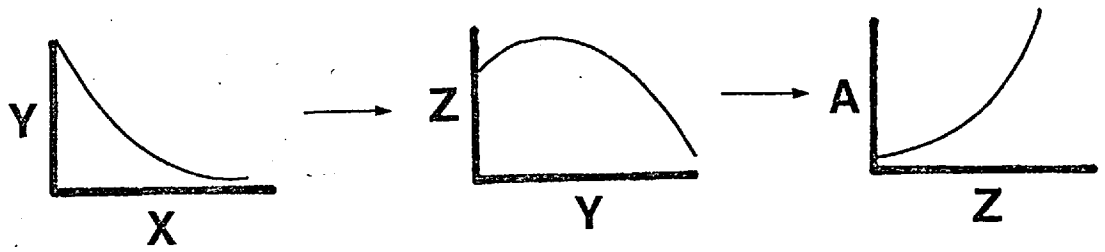
It is then possible to make certain observations on the required precision and quality of the data sets graphed. First, the data sets making up a chain which ends in a rather flat relationship need be of relatively low precision, since large changes in any of the other factors will affect the output variable only slightly. We can only have confidence in



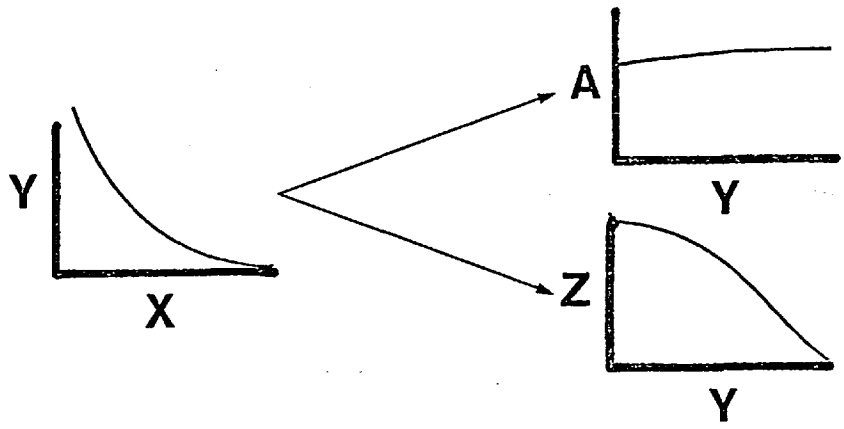
this assessment, however, when the quality of the terminal data set is relatively high (i.e. we know it to be flat, rather than merely suspecting this). This constraint applies throughout, but can be minimized by choosing higher quality data sets to act as the end-point of chains.

The more likely observation is that the terminal data set is steep, or rather steep, and that the output will respond to changes in the shape of other graphs in the chain. When

this is the case, the data sets can be ranked in terms of their maximum gradients, so that effort should be expended in increasing the quality of a graph in relation to its importance ranking.

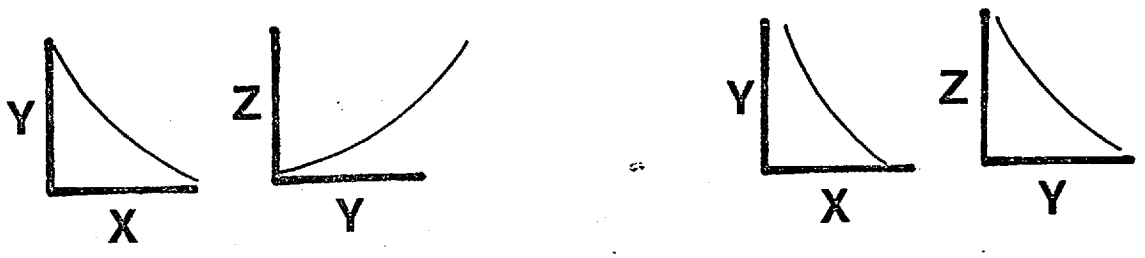


The analysis is complicated by the fact that many of the data sets in the model are not linked together as chains, but as networks. It is possible, therefore, that the quality of a data set may be unimportant in one context but have considerable influence in another. Care must be taken that all the



effects of a particular variable are considered in assessing the sensitivity of the output to changes in its affecting data set.

When a number of high-gradient relationships appear adjacent to oneanother in a chain or network it will be more important to have high confidence in all of them than would be the case if any of them were followed by a flatter relationship. This will be particularly important when the data sets are additive rather than opposed (compare (a) and (b) below).



In summary, we can make three points;

a) data sets appearing at the termination of sub-chains should be of the highest possible quality in relation to their gradients;

b) data sets appearing on the causal side (as compared to the caused side) of low gradient relationships need be of relatively low quality;

c) it is more important to expend effort in improving the quality of those relationships which are steep and have many ramifications, than those which are flat and have few effects.

3) Running and Interpretation

Synthesis of the structural code and the available data gives us a complete simulation model. This can now be run on the computer, and errors in typing and program logic gradually ironed out - a procedure affectionately known as de-bugging. When the computer finally accepts the model as being written correctly we must determine whether it still represents the system in the desired way. It is often found that after all the programming errors have been corrected some logical inconsistencies remain. These errors are not recognised by the machine, which obeys our instructions to the letter, however absurd they may be in biological terms. It is essential to check the translation of the flow-diagram into computer code to ensure that the computer has been programmed to do

exactly what is required of it.

Following on the debugging procedure, we can begin to explore the quantitative behaviour of the modelled system. This should be approached methodically because there is an almost limitless number of runs which could be performed. For instance, if there are 30 independent variables in the model, and we wish to test the effects of 5 levels in each on the behaviour of the output, there will be 5^{30} possible different runs.

For comparative purposes, we should first make some kind of control run by specifying average or standard values for all the input parameters. The manipulation is then set to zero and the model run over the requisite time period, to see the extent to which it mimics the behaviour of the real system. If the model gives a reasonable representation of reality we can proceed to investigate the effects of the manipulation under these average or 'normal' conditions. Clearly, to get this far we must have some set of criteria by which to judge when the behaviour of the model under control conditions is good enough to allow further investigation; we must, in other words, be able to validate the behaviour of the model.

The idea of validation with regard to computer simulations has been widely discussed (Goodall, 1971; Naylor and Finger, 1967; McKenney, 1967). The general points which emerge are that first, an independently collected set of data must exist with which to compare the model output, and second that numerical agreement between the modelled and real systems under one set of conditions does not imply that the model is valid for all conditions. There is consequently a risk that even though our model behaves well in the control runs it

may not respond to the manipulation in the same way as would the real system. It is obviously impractical to collect validation data for all 5^{30} possible states of the model, and so some aspects of model behaviour must be taken on trust. The important decision in designing a large-scale data collection project is which data to collect. Here again, the model can be of use, in showing those data sets to which the output is most sensitive, and hence those runs which can be most profitably validated.

The primary behaviour of the model is determined by running the simulation with increasing levels or intensities of the manipulation (unless, of course, it is an either/or decision, in which case one run will suffice). The output can be graphed simply as numbers (Fig. 12) or converted into economic units and plotted as return against expenditure. From Fig. 13 we can see at what level of manipulation the profit is maximised (the difference between the cost and return curves), and the level up to which a profit is possible (the point at which the curves intersect).

In many problems, it is of interest to know not only how the manipulation affects the specified output, but also the way in which other aspects of the system are affected. We can gain a limited view of these secondary effects by observing the values of a number of other variables which are internal to the model structure. The limitation to this part of the analysis stems from the fact that the model was built to tackle the one specific question, and hence variables which affect the dynamics of the secondary outputs may have been omitted. Predictions about the dynamics of secondary variables should therefore be interpreted with caution.

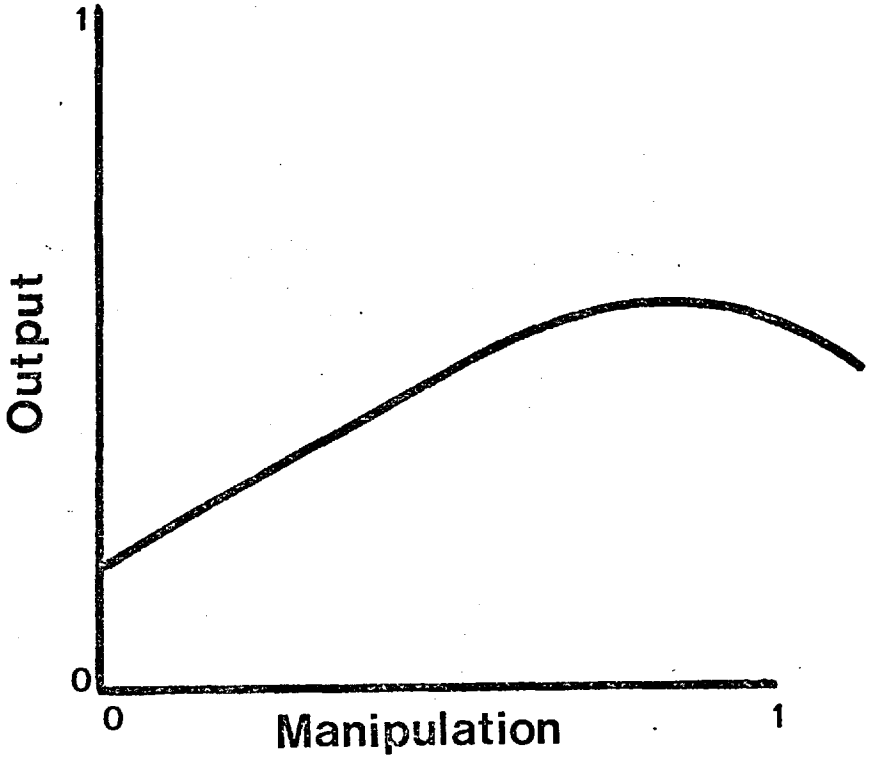


Figure 12. The output of a model plotted as a function of the intensity of the manipulation.

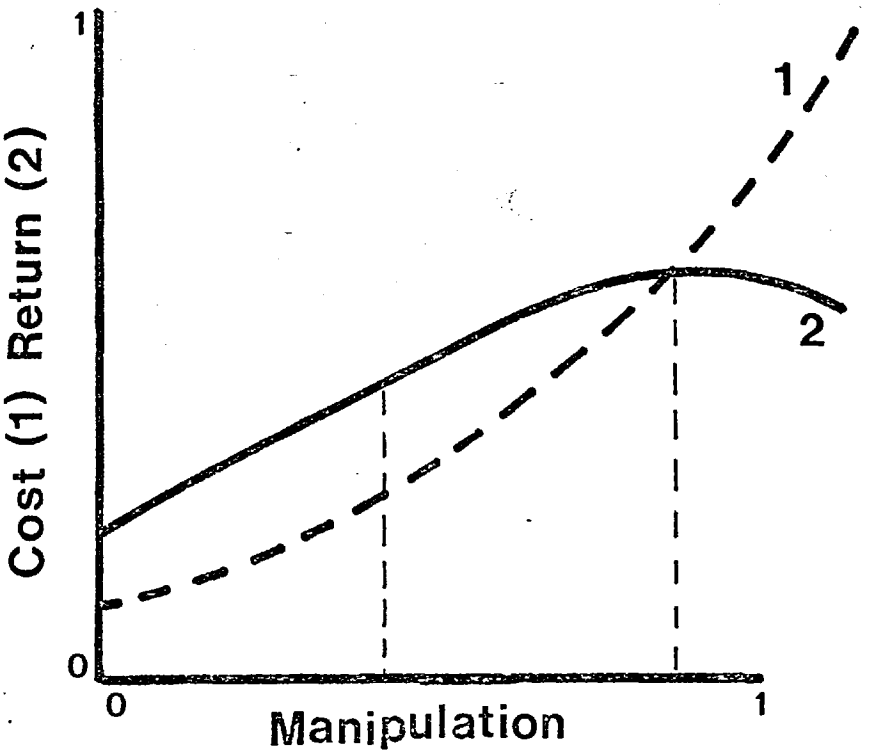


Figure 13. Model output plotted in economic terms as a function of the intensity of the manipulation. The cost of the manipulation is compared with the return produced.

The second phase of model running consists of a determination of the effects of different (i.e. non-average) conditions on the response of the output. Models tend to work rather well near their average values, because the quality of their component data sets is highest in this region. When the system is under some kind of stress (extreme shortages of some components, or extreme abundance of others), the behaviour of the model tends to be less representative. In a problem-oriented model of the type described here, those extremes which result directly from, or interact strongly with, the manipulation should have been considered in detail, and so the model should still behave realistically. It is not possible under most circumstances, however, to gather data on the effects of extremes in all the variables alone and in combination, and so here again, the output must be interpreted with caution.

Generally, the effects of climatic variables are assessed first. We might run the model with high and low yearly average temperatures, and then under conditions of drought and deluge, and assess what effects these variables have on the effectiveness of the proposed management strategy. If it is robust (it works in the same way, and to more or less the same extent under all conditions), then we can move on to test its sensitivity to other variables. When the output appears very sensitive to some particular condition (say, for example, the manipulation did not increase yield in very wet years), then we should go back to the flow-diagram and look for the cause of this behaviour. We may find, quite simply, that we have made a mistake, and left out a process which with hindsight we can see to be important (soil drainage, in the example).

Alternatively, the result could be quite appealing, and again with hindsight, we might wonder why we had not thought of it before. It is here, in the production of what Forrester (1961) has called counter-intuitive results, that one of the great values of simulation modelling lies. The model highlights effects which are implicit in the interaction of our assumptions, but which we have never considered. Before these results appear, however, we must ask questions of the model, and unless the model is structured to consider precisely those questions asked of it, the results which emerge will be in doubt.

When we are satisfied that sufficient runs have been made, and that we understand the behaviour of the system under a range of conditions, the initial question can be evaluated. The evaluation should be made in terms of both the numerical and the economic behaviour of the system, and it should be possible to state those conditions under which the proposed management policy would be effective. Care must be taken to stress that this is the way that the model behaves, and not necessarily the way in which the real system would respond to the manipulation. Since, however, the model represents the synthesis of all our qualitative and quantitative understanding of the resource system, the output is the best possible guess we can make. It remains for the management policy to be tried in pilot field experiments before any specific recommendations can be made to resource managers.

There are a number of problems which, while they can be modelled, can not be validated under field conditions. These may be purely speculative (e.g. how would a particular area look after a nuclear blast ?), or involve such a drastic change in the environment that the validation test becomes

infeasible (what will be the effects of building the next London airport at Foulness ?). In the first case, validation and field tests are irrelevant as the manipulation is speculative. In the latter, however, the model can take on immense significance, because it is the only means of prediction available to us.

4 Sensitivity Analysis

In addition to determining the effects of the manipulation on the output, and upon certain intermediate variables, it is possible to investigate the overall sensitivity of the model to a selected number of its data sets. The procedure acts to substantiate the qualitative assessment of sensitivity made earlier, and also to search for interactions which were previously overlooked.

We define an insensitive data set as one which has no appreciable affect upon the output, irrespective of changes which might be made to its shape. This aspect of sensitivity is rather different from our earlier definition, since it rests upon the position of the data set within the model structure, and on the shape of the data sets adjacent to it. We should look for sensitivity in three aspects of the data set; sensitivity to changes in gradient (discussed earlier), to changes in base position (x-shift), and to changes in height (y-shift).

To carry out these tests, we remove the original data set from the model and replace it by a sequence of test attributes. First, we make Y independent of X (a completely flat relationship), and observe the output at different constant levels of Y. This will show the response of the system to the level of Y, and also the effects of uncoupling X and Y. Next, we

can take the shape of the original data set and test x- and y-shifts. Fig. 14 shows some different curves we might try. The sensitivity of the data set in this case can be stated as the proportional change in the output arising from a given proportional change in x- or y-shift (i.e. d/Rx and d/Ry in the figure). From these tests it will be possible to suggest those aspects of the data set which must be given precedence in an experimental study.

There are limitations to this sort of sensitivity analysis. The method works well for mathematical models in which the structure is simple and unequivocally defined (see, for example, Chapter IV), but it can be misleading when applied to structurally complex, data-patchy biological simulations. This is because we can test the sensitivity of only one relationship at a time, and the incorrect shapes of other low quality data sets may completely obscure the true behaviour of the tested parameters. Similarly, unless the model has been validated under a number of conditions, it is not possible to state with any conviction that all the important processes have been included. In this event, we are not testing the sensitivity of the data set in the context of a complete model system, and may run into considerable interpretive errors.

5 Data Collection

If the models forms part of a large-scale investigation into the problem in hand, it can be employed as a means of directing data aquisition. From the runs and sensitivity analyses it will have become plain at which points high quality data are required, and where experimentation will be most rewarding. To improve the problem-solving potential of the model we should aim to collect data which meet a number

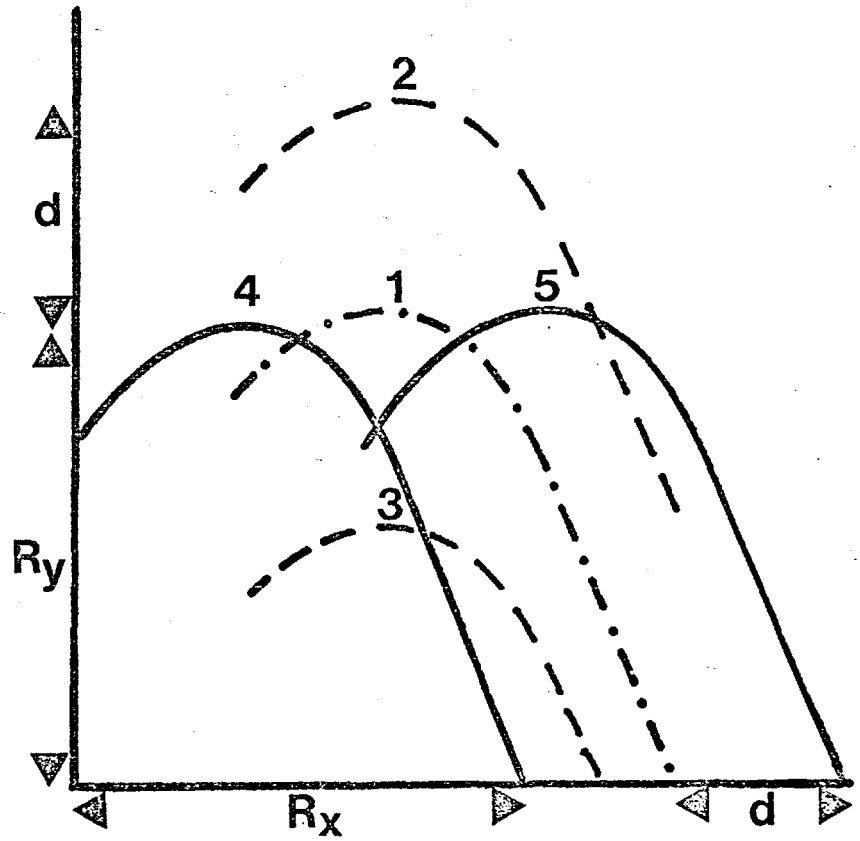


Figure 14. The family of curves which can be used to test the sensitivity of model output to this data set. 1 control; 2 +ve y-shift; 3 -ve y-shift; 4 -ve x-shift; 5 +ve x-shift. Proportional change in the data set is defined as the shift (d) divided by the range of the data; i.e. as d/Rx or d/Ry.

of requisites.

a) They should replace low quality data;

b) they should be of high gradient and show high sensitivity;

c) they should be at key positions within the model structure (at the end of causal chains, or at nodes in a network);

d) they should be involved in 'interesting' causal chains (this will increase their value in purely scientific terms).

C. Criticism of Models

The criticism of simulation models is rarely constructive, and generally consists of an assessment of whether the model is good or poor based solely upon the agreement between the output and real system behaviour. This, as we have seen, is only a part (and in many studies only a small part) of the value of a simulation model.

Analytical, algebraic models, in contrast, are criticised far more on their structure, and there seems to be more tolerance of disagreement between the output and validation data. A typical criticism of an algebraic model (and one which has frequently been leveled against the models of Lotka and Volterra - see, for example, Watt, 1968) is that its structure is over-simplified, or that the assumptions made are more or less non-biological. It is interesting to examine the possible reasons for this discrepancy in critical assessment.

Simulation models are usually written up in the literature in a format which gives the impression that the modelling itself can be taken for granted (see for instance Hughes and Gilbert, 1968; Gilbert and Hughes, 1971; Gilbert and Gutierrez, 1973). There is a statement of the biology which is important

in system structure, but little consideration given to the parameters which have been omitted, nor to the reasons why those which have been included were so. Again, it is assumed that this aspect is obvious. In effect, the results of the model runs are presented as a fait accompli, and the only basis open to the reader is to criticise the model with regard to its mimicking real system behaviour. Since the model structure is not elucidated, it is rarely clear why the model behaved in a particular way to a given manipulation, and this means that the model (while the builder himself may feel it to be biologically sophisticated) appears to the reader as a black-box; he can see the inputs and outputs, but can only guess at the internal structure.

The presentation of analytical models is markedly different. At the outset each variable is defined, and the model is presented in a step-wise fashion, so each equation is accompanied by an explanation of its implicit assumptions and limitations. The attention of the reader is therefore focussed squarely on the biology of the system and the way in which it has been interpreted. Similarly, because the model structure is laid bare, it is possible to check the algebra for one's self, and, in the process, either enhance one's understanding of the model, or highlight the errors which make it behave in the way it does. Having seen the bones of the model, the critic is likely to be far less interested in the agreement between observed and predicted results.

For these reasons, I shall adopt the method of presentation used in algebraic models even though, because there tend to be more variables in simulation models, this might become space-consuming. If a model does nothing else, it can at least focus attention on biological processes which are thought to

be understood, but which, on quantitative scrutiny, appear in a different light (see Chapters III, IV and V).

Discussion

I have briefly reviewed the types of model which are available in ecology, and explained in detail the approach which I have adopted to biological simulation modelling. The ideas and methods outlined here will be used in the remaining chapters to construct a set of models to investigate the ideal predator for Aphis fabae on beans.

One of the most important points which must be borne in mind when building and evaluating simulation models is that they are not capable of producing general solutions. They rely entirely upon numerical methods for the evaluation of the functions and expressions incorporated. It is never possible under these circumstances to say that one completely understands a simulation model (unless it is very simple indeed), but only that one knows why it behaved in the way it did for those runs which were performed. When we remember that the number of possible runs is given by

$$N = (\text{Number of states})^{(\text{Number of Variables})}$$

it is clear how little we can actually discover.

If generality is required, then the model must be stated in algebraic terms so that the response of the system to each variable can be seen for all conditions, and the state of the system at its limits can be determined. Unfortunately it is rarely possible to combine the degree of biological realism required of many management problems with the mathematical tractability demanded for analytical solution.

The lack of generality in simulation models is compensated by the fact that in many algebraic models the degree of simplification required for their construction has made the generality of their form meaningless because it is unrealistic. Holling (1966b) suggests that models should be built to maximise simultaneously precision, realism and generality, but, since simulation models can not be general, and analytical models are rarely realistic, this would not seem to be possible.

Not only is it important to choose the type of model in relation to the question under scrutiny, but also to gear the study to the type of answer required. Models which require accurate numerical predictions should be built with as simple a structure as possible commensurate with the precision required. The ultimately simple model is clearly a regression of the output on the intensity of the manipulation; if the fit of this relationship is good over a wide range of conditions, then this would be the best model for predictive purposes. If, on the other hand, the main concern of the model is to make qualitative predictions about the behaviour of the system under the manipulation, then more detail will be required in model structure. The more variables which are considered, the more complete will be the understanding which could be gained, but the more difficult will be the interpretation. A model with too few variables will tend to behave unrealistically, while a model with too many may obscure the interesting behaviour of the system in a cloud of detail.

The problem I have chosen to tackle in the forthcoming chapters requires a qualitative solution; the aim is not to predict how any particular predator species will affect bean

yield in a particular case, but rather to understand those aspects of the biology of the plant, its pest, and the potential predator which influence the degree of control achieved. To do this we must determine first that pattern of aphid feeding which can be tolerated by the plant without significant crop loss; second, the pattern of predation which will keep the damage caused by an aphid population below this specified limit; and third, that combination of behavioural and physiological attributes in a predator species which will bring about the necessary pattern of aphid consumption. The initial problem is therefore to assess the effects of feeding by Aphis fabae on the growth and fruit development of Vicia faba.

CHAPTER II THE EFFECTS OF INFESTATION BY *APHIS FABAE* SCOPOLI ON THE GROWTH PATTERN OF *VICIA FABA* L.

Introduction.

The quantitative aspects of growth and fruit production in the broad bean, *Vicia faba*, have been described by Ishag (1969), and the population dynamics of its principal insect pest (*Aphis fabae*) are well documented (Kennedy and Booth, 1954; Way and Banks, 1967, 1968; Way, 1967, 1968). Rather less attention has been given to the precise ways in which aphid infestation affects the growth of the host plant, or to the effects of various plant attributes on the performance of the aphid population. While there do exist bodies of data describing in broad terms the relationship between the number of bean stalks infested and the ultimate yield of beans (e.g. Rothamsted, 1971), and between the varietal resistance of the host plant to aphid attack and the morphological changes occurring under infestation (Tambs-Lyche and Kennedy, 1958), there is little information relating the size of the aphid

infestation to the rate of dry matter production in the crop, or to the pattern of dry matter distribution between the plant organs. A destructive sampling experiment has been performed to investigate these two effects, and to provide data for a simulation model of the growth of Vicia faba under aphid attack (Chapter III).

Materials and Methods

Fifty Garton's Spring Tick field beans were weighed fresh, and then oven dried at 70°C for 48 hours to obtain a regression of seed dry weight on fresh weight (Fig. 15). Two hundred more seeds for use in the experiment were then weighed fresh, and their dry weights interpolated from the regression. These seeds were placed in numbered positions on damp blotting paper, and allowed to germinate in a warm dark cupboard. After one week the radicles were fully emerged and plumule development had just begun. At this stage the plants were placed individually in six inch plant pots at a depth of 2 cm below the surface of John Innes No. 1 potting mixture. The pots were then set out in galvanized trays in a greenhouse under a 16 hour photoperiod at 15°C (+/- 3°C). Each week the position of the pots was randomized within the trays to minimise the effects of differences in the aerial environment between different parts of the greenhouse. After two weeks the very large and very small plants were discarded, leaving a relatively homogeneous population of 144 plants.

Each week two plants were selected at random from the population and analysed as controls. After seven weeks, when the average height of the plants was 11 cm and they had four leaves expanded (the lowest two nodes in V. faba do not

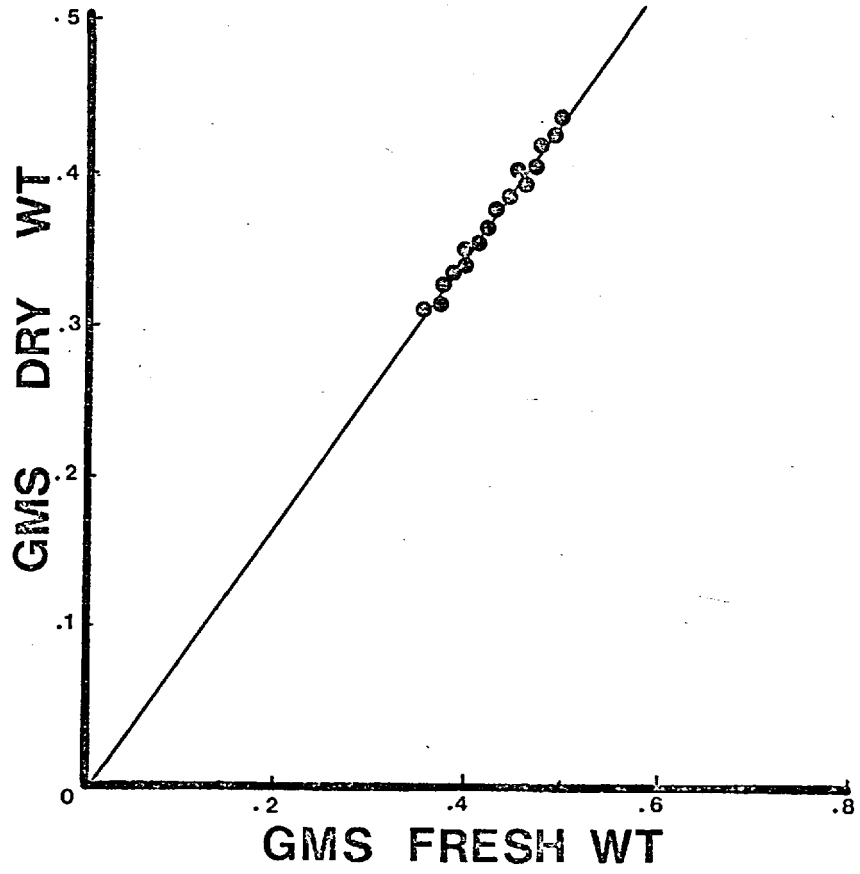


Figure 15. The regression of bean dry weight on bean fresh weight. r = 0.9995; b = 0.871. Mean bean weight is 0.377 gms dry weight and mean water content is 12.9%

generally bear leaves), the plant population was divided between two greenhouses, again choosing individuals at random. Each plant in the treatment greenhouse was infested by placing six apterous, fourth instar larvae of A. fabae in the terminal cluster of leaves. This procedure is known to produce rapid population build-up (Way and Banks, 1967). From the eighth week onwards, two plants were harvested from both the treatment and the control greenhouses. The plants were watered as regularly as necessary to keep the soil surface damp to the touch.

The plant seeds were obtained commericially, and the aphids were taken from a long-standing clonal laboratory population bred in 20°C controlled temperature rooms (Milne, 1971).

Analysis

Each plant was analysed as follows. Starting with the lowest leaf-bearing node (the third), each leaflet was cut from the petiole and placed under a 3D microscope. The number of alate and apterous aphids was determined, and the leaflet then wiped clean of insects and honeydew and weighed. Its perimeter was then traced so that leaflet area could later be determined with a planimeter, after which the leaf was placed in a numbered paper bag for oven drying. All the leaves were treated in this way until only the tight-knit group of leaves around the terminal meristem remained. This was treated as a single unit (hereafter called the terminal cluster): the aphids living within the curled leaves were counted and removed before the fresh weight was determined.

Next, the stem was cut just above the third node, in such a way that the stipule and the petiole remained attached to the top of the internode (see Fig. 16). The aphids

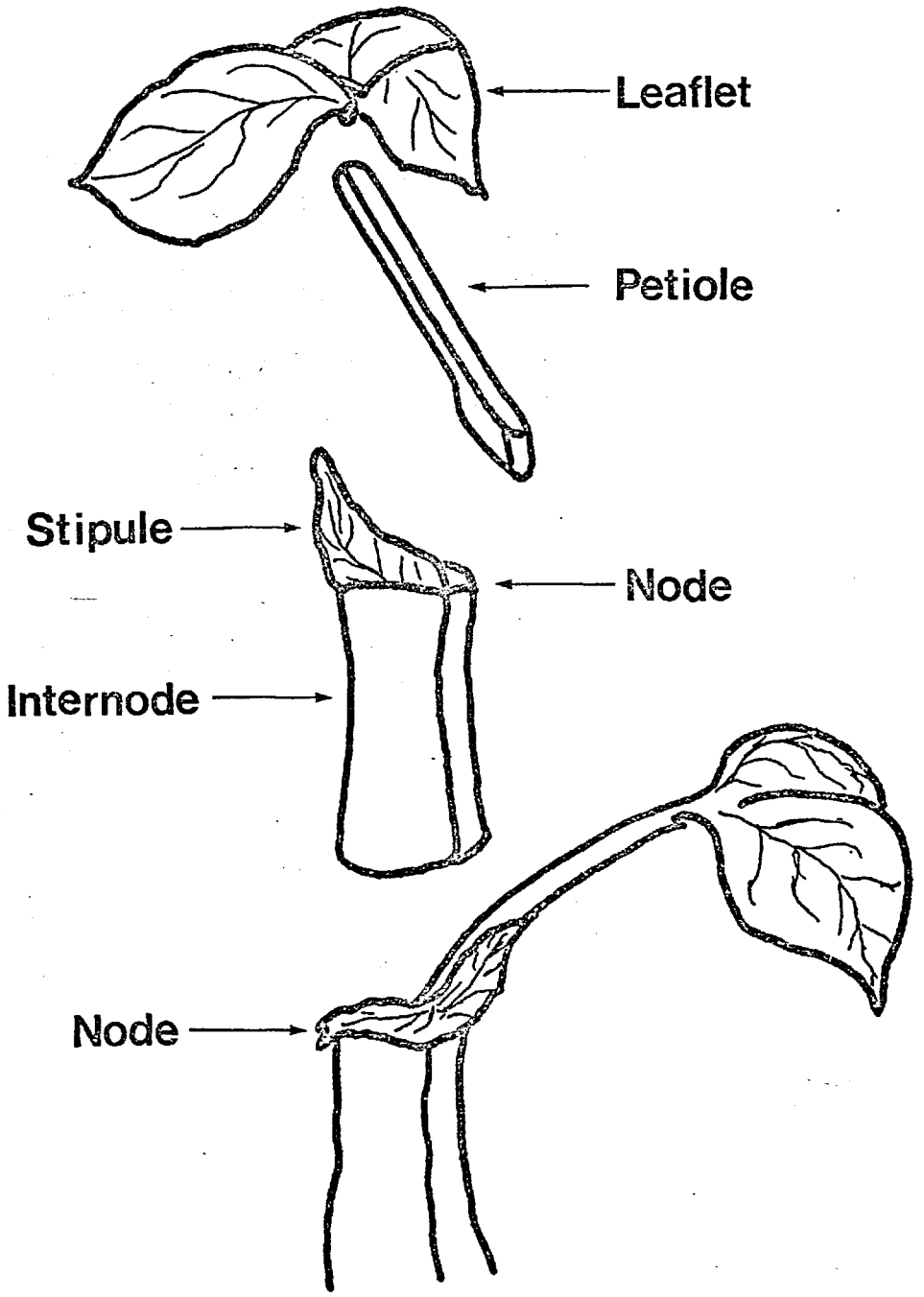


Figure 16. The parts into which the stem was divided. One node is assumed to consist of an amount of internode, petiole, leaf and reproductive tissue (flowers or fruits). The root is taken as consisting of a tap section with a certian weight of laterals, and the stem is completed by a terminal cluster.

feeding or walking along each petiole and stem section were counted and the stalk then washed clean of insects and honeydew. Each stem section was cut just above the stipule, and the petiole severed where it joined the stem. The lengths of the petiole and stem sections were then determined to the nearest mm and each part placed in an individual bag for drying.

The root system was collected by inverting the pot over a fine sieve and gently crumbling away the soil. The fine root fragments were collected with forceps and placed in a beaker of water. Once the bulk of the soil was removed, the root system was washed in a larger sieve under a running tap and dried on blotting paper. Each lateral root was cut from the tap-root and cleaned of any remaining sand or leaf mould. The smaller root sections were removed from the beaker, dried, and cleaned in the same way. All the lateral roots were placed in one bag, and the tap-root in another. Finally, the remains of the seed were cut from the base of the lowest two stem sections, and each part bagged and committed to the oven.

The analysis of control plants differed only in that they did not need to be examined for aphids or washed before being cut up. The data collected are summarized in Table 5.

Results

1 Qualitative Effects

a) Aphid Infestation

The infestation was initiated by placing six apterous fourth instar virginoparae within the curled leaves of the terminal cluster. This differs from the field situation in that natural infestations are typically started by alate adults,

Organ	Dry Wt.	Fresh Wt.	Length	Area	Aphids
Lateral Roots	*				
Tap Root	*		*		
Internodes[3]	*		*		*
Petioles[3]	*		*		*
Leaflets[3]	*	*		*	*
Flowers[3]	*				*
Pods[3]	*	*	*		*
Fruits[3]	*	*			
Parent Seed	*[1]	*[2]			

Table 5. The data collected from each plant harvested. The dry weight of the parent seed (1) is determined after the plant is cut down, and its fresh weight (2) at planting. The dry weight at planting was interpolated from Fig. 15. Those data marked (3) were collected at each node of the plant.

which fly into the crop from their winter host plant (the spindle bush, Euonymus europaeus L.). The experiment assumes that only six of the progeny of the immigrant alatae survive to adulthood, and that they remain in the terminal cluster for this period (this is borne out by field observation Kennedy and Stroyan, 1959; Way, 1967).

The progeny of these initial aphids colonized lower leaves on the plant by walking down the stem and out along the petiole. After settling on a new leaf an adult aphid would produce young over an extended period; these juveniles would not move far away from their mother, and so dense aggregates of aphids developed after a time. Aggregates were first observed on progressively lower leaves after the times shown in table 6; these data were obtained from different plants.

Weeks after infestation to first observation	Nodes from the top of the plant
1	0
2	1
3	-
4	5
5	7
6	8 (all leaves)

Table 6. The appearance of aphid aggregates on progressively lower leaves of the plant.

Initially, the aphids clustered in the folded upper surfaces of very young leaves. As these leaves opened and expanded, the aphids moved to their preferred position on the

underside, while some moved up the stem to infest the fresh leaves in the terminal cluster. Once established on a leaf, the aggregates grew rapidly in size (the number of aphids), but only gradually in density (the number of aphids per cm of leaf vein), spreading slowly along the main leaf veins. The rate at which apterae walked off the leaf to establish new aggregates elsewhere is probably related to the density and the extent of the leaf colony, but could not be measured in this experiment (see Chapter IV).

A continuous redispersal within the plant was therefore observed, some apterae climbing to reinfest the leaves above their aggregate, including those which had recently opened and may have escaped attack, and some walking down the stem, either to infest lower leaves (which might be nutritionally less desirable (Kennedy and Stroyan, 1959; Ibbotson and Kennedy, 1951), but more expedient in terms of current aphid density), or to emigrate from the plant completely and search out a new host plant.

Aphid aggregates also developed on stems and, to a lesser extent, on petioles. Here the density of animals per unit area tended to be even higher than on the leaves, upon which the aphids are confined to the rather widely spaced veins. The striking feature of the distribution of stem aggregates was the consistency of their position relative to the top of the plant. There appears to be a well-defined threshold of stem age above which few aphids are found feeding. This effect may well be due to the unavailability of phloem vessels in the outer layers of the older stem tissues within feeding range of the aphid stylets (Kennedy and Mittler, 1953). Fig. 17 shows the frequency of feeding alatae on stems

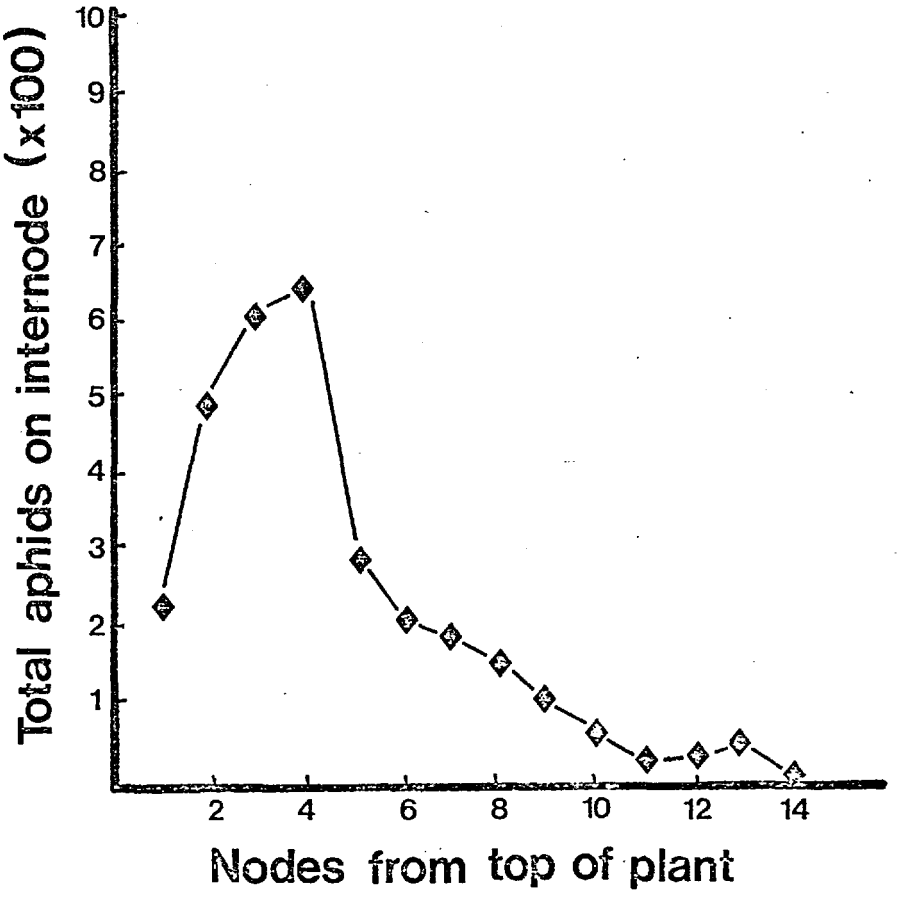


Figure 17. The total number of aphids observed to be feeding at different heights on the plant stem from all harvested plants. There is a distinct threshold between 4 and 5 nodes from the top.

of different ages; there is a distinct preference for younger internodes (i.e. stem sections closer to the top of the plant).

Six weeks after aphid infestation all the leaves were, or had been attacked (Table 6). At this stage there was very little upward dispersal; the aphids were restless, and few were feeding. The rate of emigration from the plant was very high as evidenced by the number of apterae on the rims of the plant pots. As the infestation aged, the proportion of adults with wings increased (Lees, 1966; Hille Ris Lambers, 1966). On emergence from the fourth instar the majority of the adult alatae flew off, and left the greenhouse through the ventilation system. Some, however, remained on the plants (as Shaw, 1968, noticed in his experiments); indeed the last aphids observed on some of the dying plants were adult alatae. Clearly in this case reproduction had ceased, and all the remaining apterae had walked away.

b) Plant Phenology and Damage

Vicia faba usually develops a single, unbranched stem unlike the wild British species of this genus, V. cracca and V. sativa. In those cases where branches do develop, they tend to arise at the first and second (leafless) nodes. Leaves appear in an opposite pattern as the stem elongates, and consist of two leaflets at the lower nodes, increasing to five or six leaflets per leaf above the sixteenth node. The plants never achieved more than twenty two nodes in height at maturity.

Flower buds appear in the leaf axils above the ninth node, but only a fraction of the buds produced develop into flowers (Ishag, 1969). The flowers are zygomorphic, fragrant, and require cross pollination by insects (usually by bees in the

crop situation); pollination was performed with a fine paint brush in the experiment. Some of the pollinated flowers then develop into pods which contain three or four seeds on average.

Visible effects of the aphid infestation on the well-being of the bean plants were first observed three weeks after the apterae were introduced. There was a light covering of honeydew and cast exuvia on the upper surfaces of the fully opened leaves, and several of the younger leaves were curled upward, and a little yellow. After four weeks the plants were very sticky with honeydew and the four topmost leaves were curled and wilted; two weeks later, many of the leaves showed a characteristic 'burn' on their upper surfaces caused by the germination of the chocolate spot fungus, Botrytis fabae in the honeydew substrate (M. J. Way, pers. com), and all but the lowest leaves were curled and wilted.

The stem too was scarred and blackened, particularly at the upper internodes where aphid aggregates had been dense. There was very little increase in height by the plants after this time (6 weeks), and no new leaves were formed. Nine weeks after aphid infestation, the majority of the treatment plants were dead. Very few had flowered, and none had set any pods.

2 Quantitative Effects

a) Dry Matter Production

The pattern of dry matter accumulation in the control plants over the course of the experiment is shown in Fig. 18. Had the experiment continued up to senescence, then the curve of dry weight would have been more sigmoidal in appearance, as dry matter production slowed and eventually ceased.

The aphid population acts to change this pattern of

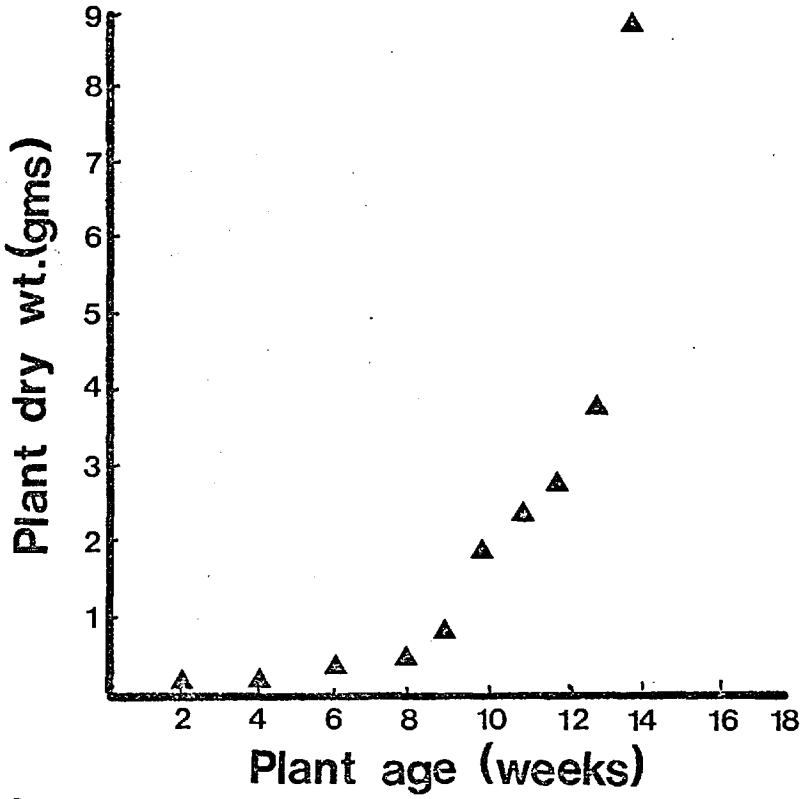


Figure 18. The pattern of dry weight increase in control plants. Plant age is in weeks since germination.

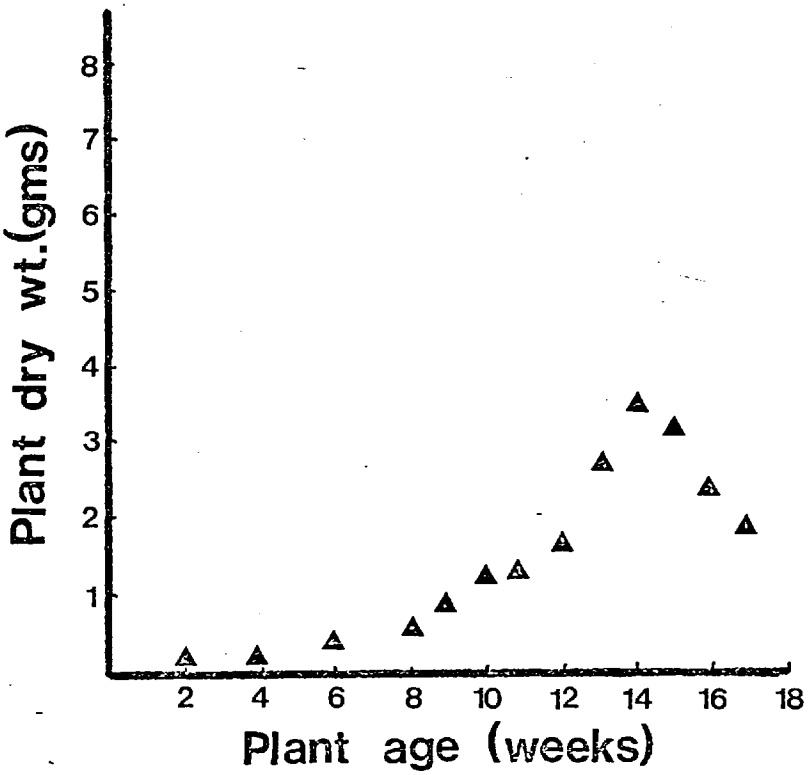


Figure 19. The pattern of dry weight increase in infested plants. No growth occurs after the 14th week; aphids arrived at the beginning of the 8th week.

growth in three ways. First, by removing photosynthates from the phloem vessels the amount of dry matter available for growth is reduced. Second, by reducing this growth, the rate of appearance of new leaves, and the rate of expansion of existing leaves are both decreased, and so the plant's potential for subsequent photosynthesis is reduced. Finally, the aphids damage the plant in a number of ways over and above tapping off a fraction of its photosynthates. The epidermis is punctured by stylets (which are often left inseted after the aphid dies), and covered with honeydew. This latter forms an ideal substrate for the germination of parasitic fungi like Botrytis fabae, and may also inhibit gas exchange by blocking the stomata. In addition, chemicals injected into the plant in aphid saliva may interfere with the normal physiological processes of the leaf (Miles, 1968). Fig. 19 demonstrates the pattern of dry matter increase in infested plants, and shows the cessation of growth at 14 weeks.

b) Production of New Leaves

The most obvious effect of the aphid infestation was in slowing the rate of leaf production. Reserves channelled to the apical meristem are removed by the aphids feeding in the terminal cluster at a sufficiently high rate to affect the incorporation of photosynthates into new tissues. The side effects of aphid presence (tissue damage and saliva injections) may also act to reduce the potential of the meristematic tissues to produce new leaves. Figure 20 compares the number of leaves expanded in treatment and control plants of the same age.

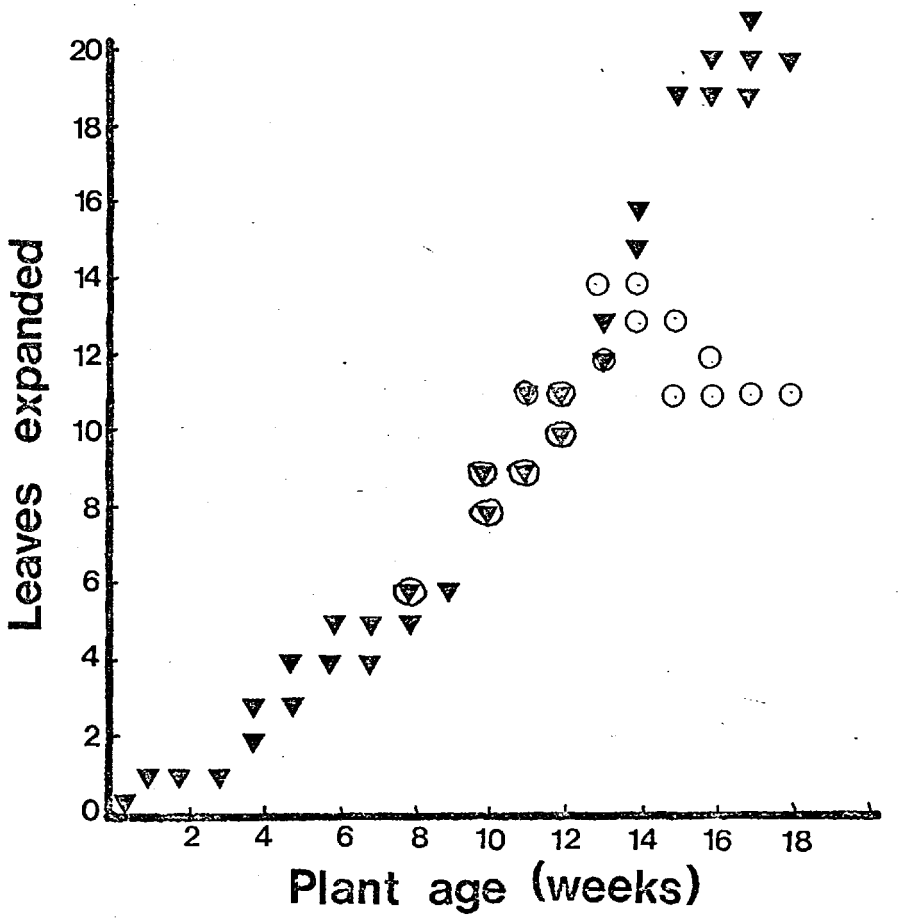


Figure 20. The number of leaves expanded in control (▼) and treatment (O) plants at different plant ages. Aphid infestation began at the start of the 8th week. The infested plants did not actually decrease in height; the points in this figure are for individual plants.

c) Growth of Individual Leaves

The leaves of a growing plant can be considered in two broad categories as experiencing either a net import, or a net export of photosynthesized reserves. These are referred to as 'sink' and 'source' leaves respectively. An aphid aggregate established on a source leaf will reduce the amount of reserves available for growth in those parts of the plant to which reserves are transported from the node in question. It should not have any significant effect on the rate of dry matter increase of the source leaf itself, if only for the reason that the leaf will be at, or near, its maximum weight already (Fig. 21a). Aphid feeding on sink leaves, however, can drastically affect their rate of growth. In effect, the aphids compete with the growing cells for the available reserves, but have an advantage in that they remove the photosynthates from the pipeline before the material reaches the cells. The result of this competition is that the reserves are incorporated into aphid body tissues or excreted as honeydew, and the demand of the growing leaf for reserves (its sink strength) is unabated. The aphids benefit in consequence, because the rate of flow of photosynthates is maintained. If the reserves had been incorporated into leaf tissue, the sink strength would have been reduced, and the leaf would eventually become a source (i.e. an exporter of material). In Fig. 21b the growth of a sink leaf (the eleventh) is compared for treatment and infested plants.

Fig. 2i backs up the assumption that aphid feeding affects source (a) and sink (b) leaves differently; there is no significant difference between the weight of the seventh (source) leaf, but the eleventh leaf is significantly smaller in

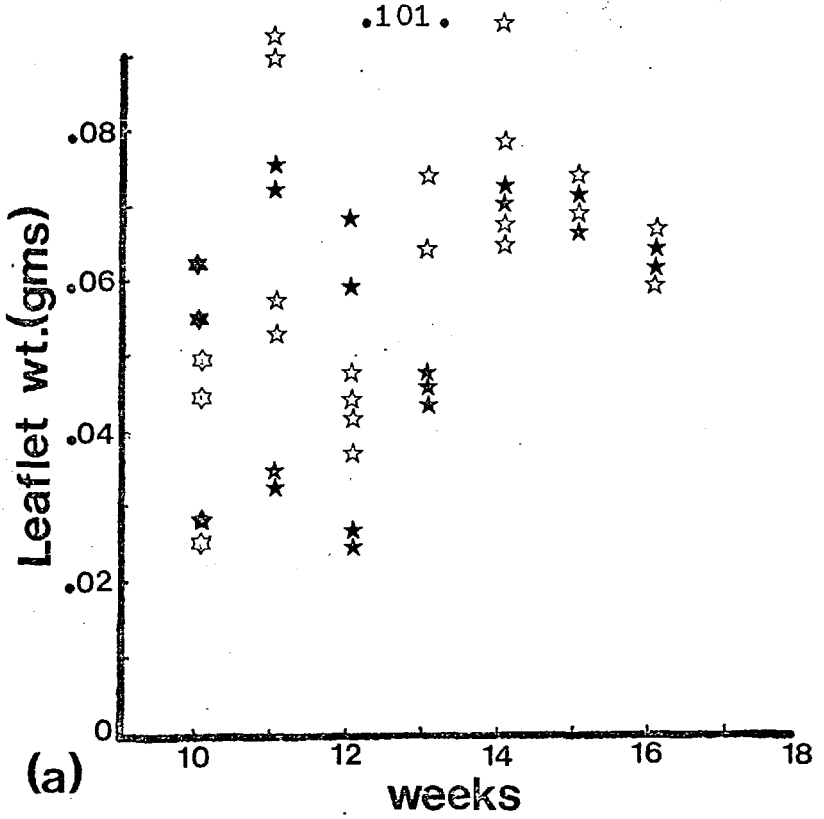


Figure 21a. The growth of control (★) and infested (☆) source leaves (7th node).

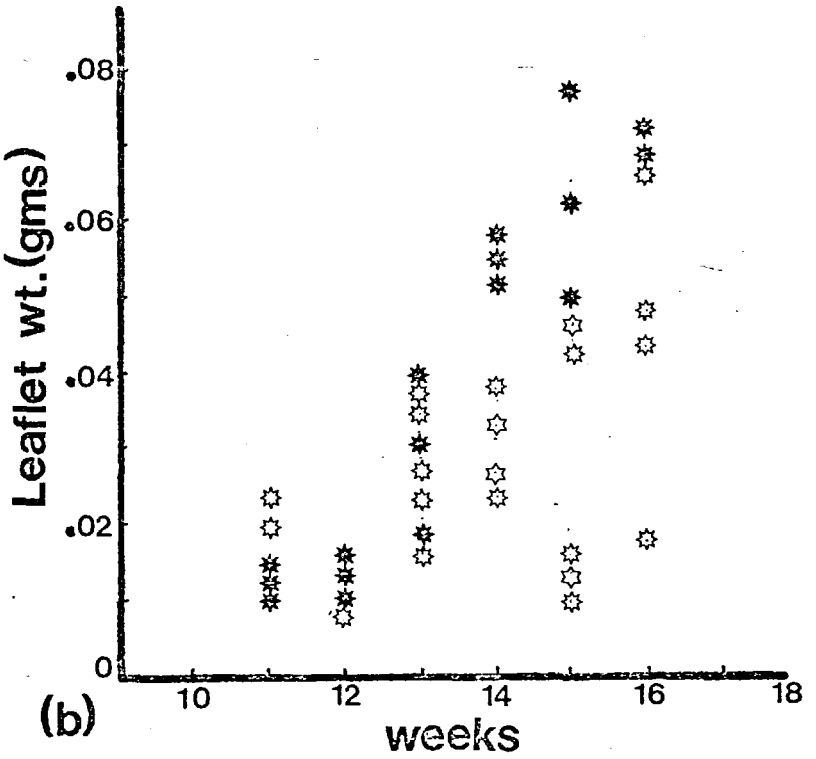


Figure 21b. The growth of control (✲) and infested (✡) sink leaves (11th node).

treatment plants.

In addition to the removal of carbohydrates and amino acids, aphid feeding can account for a substantial water loss (Wearing, 1972). Depending upon the extent to which the plant can compensate for this loss by increased transpiration and osmosis from adjacent cells, the rate of photosynthesis within the leaf will be detrimentally affected (Jones, 1973). It was not possible to guage this effect directly in the experiment, but it was clear that leaves bearing large aphid aggregates contained less water per gram dry weight than did leaves of the same age from control plants (Fig. 22).

The effects of cuticle damage by stylet insertion, and the blockage of stomata by honeydew could alter both the water balance of the leaf and the rate of gas exchange. Again, the quantification of this effect is impossible from this experiment, but the amount of honeydew and aphid debris on leaves were clearly inversely correlated with leaf 'health', at least on a visual assessment. The other unknown quantity in understanding the effects of aphid infestation on leaf growth is the extent to which the growth of fungal and virus species affects the rate of photosynthesis, and the incorporation of imported reserves into leaf tissues.

d) Stem and Petiole Growth

Although the stems and petioles of Vicia faba do contain chloroplasts, it is thought that these organs contribute only slightly to overall plant growth (Ishag, 1969); they can therefore be considered as sink tissues. The analysis is complicated by the fact that reserves flow through stems and petioles on their way to and from other organs, and so aphid feeding might act as on source leaves to reduce the amount of material available in other parts of the plant. Unless the

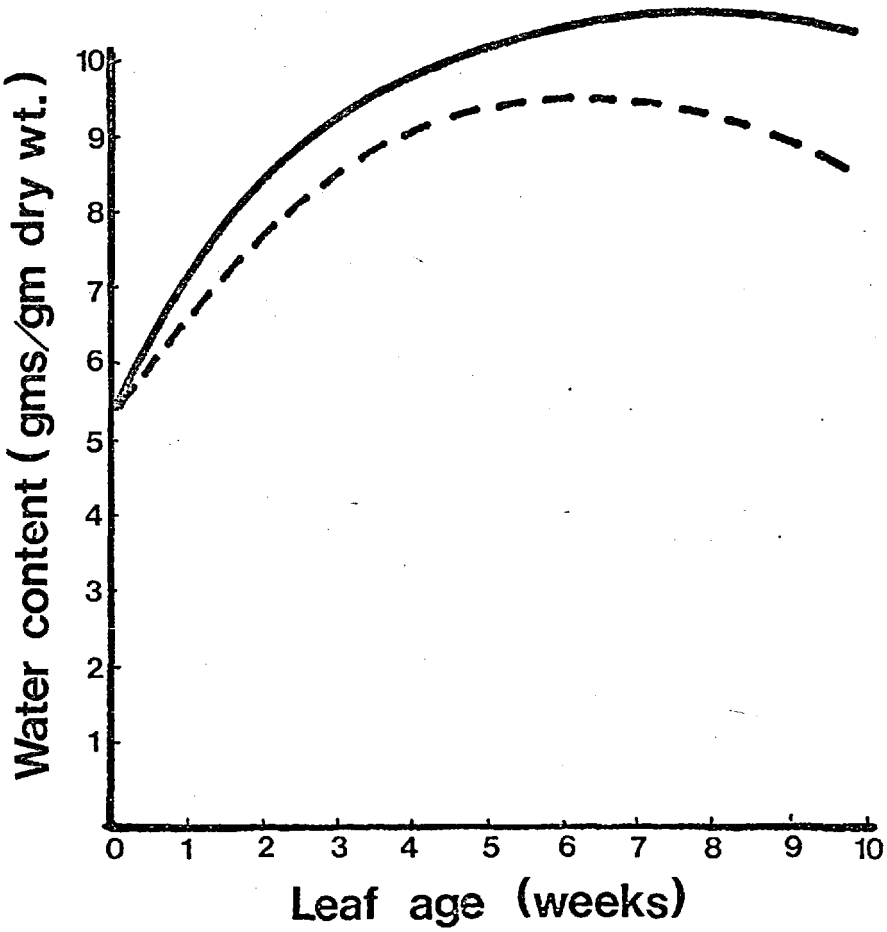


Figure 22. The water content of leaves (grams of water per gram leaf dry weight) of different ages from control (solid curve) and treatment (dashed curve) plants.

aphids feeding on a particular internode or petiole were tapping those photosynthates specifically destined for the dry matter increase of these organs, we should not expect the presence of an aphid aggregate to affect the growth of the stem or petiole relative to other parts of the plant. The picture is again complicated by the possible effects of tissue damage and saliva injection. It could be that the relative growth of an internode is unaffected by the amount of reserve passing through its phloem vessels, and that any reduction in growth rate relative to other less heavily infested internodes is due entirely to surface damage effects. To test this hypothesis we can plot the growth of one stem section relative to another for infested and control plants (Fig. 23). The null hypothesis is that the number of feeding aphids has no effect on relative stem growth, but simply reduces overall growth; the gradient of the curve for the growth of the 10th (more heavily infested) internode relative to the 7th should be the same for control and treatment plants. There is an indication in Fig. 23 that the relative growth of the more heavily infested stem section is reduced, and therefore, that the null hypothesis of overall reserve reduction is not applicable. It seems, in other words, that the relative growth of a stem section is determined (at least in part) by the number of feeding aphids upon it.

e) Root Growth

Aphis fabae does not feed on the roots of Vicia faba, nor were any other insects seen to do so in this experiment. Any reduction in root growth in the treatment plants can therefore be ascribed to either the reduction in the amount of

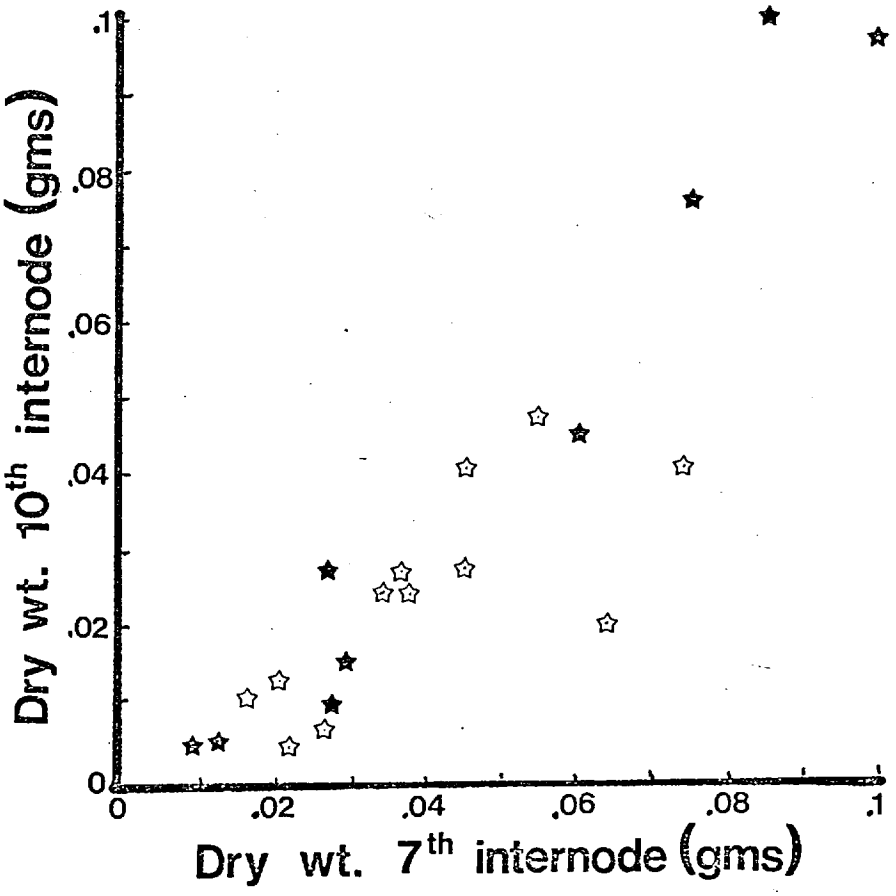


Figure 23. The relative weights of the 10th (heavily infested) and 7th (lightly infested) internodes for control (★) and treatment (☆) plants. If the number of feeding aphids had no effect on relative stem growth the two curves would be of equal gradient. It appears that the number of feeding aphids does affect relative stem growth.

reserve available, or to the injection of chemicals affecting the pattern of dry matter distribution between the root and shoot system. As material is removed from the above ground parts of plants it is commonly found that reserves are mobilized from the root in compensation (Cavers, 1971). Other plants may reduce the fraction of current production flowing to roots (Crider, 1955; Brouwer, 1963; Schuster, 1964; Das Gupta, 1968).

The weights of the root systems of treatment and control plants are compared in Fig. 24. Because the amount of reserves removed by the aphids in feeding, and the amount of material they might inject into the plant via their saliva are so closely correlated, it is not possible to state whether the observed reduction in root growth is due to either one of these causes, or to a combination of them both. In any event, it is unlikely that the size of the root system will limit plant growth in this experiment, because all the effects of aphid feeding are experienced first in the shoot, and the plant itself responds by reducing the rate of root growth. It would presumably be possible for the plant to make good any excessive reductions in the relative amount of root, should the shoot recover sufficiently to merit it.

f) Pattern of Dry Matter Distribution

Not only does aphid feeding reduce the rate of growth of the plant, but it also affects the pattern of distribution of such dry matter production as occurs. I have already discussed the possible effects of aphid feeding in competing with sink organs for the reserves available, and the potential effects of aphid damage on the relative growth of one organ over another.

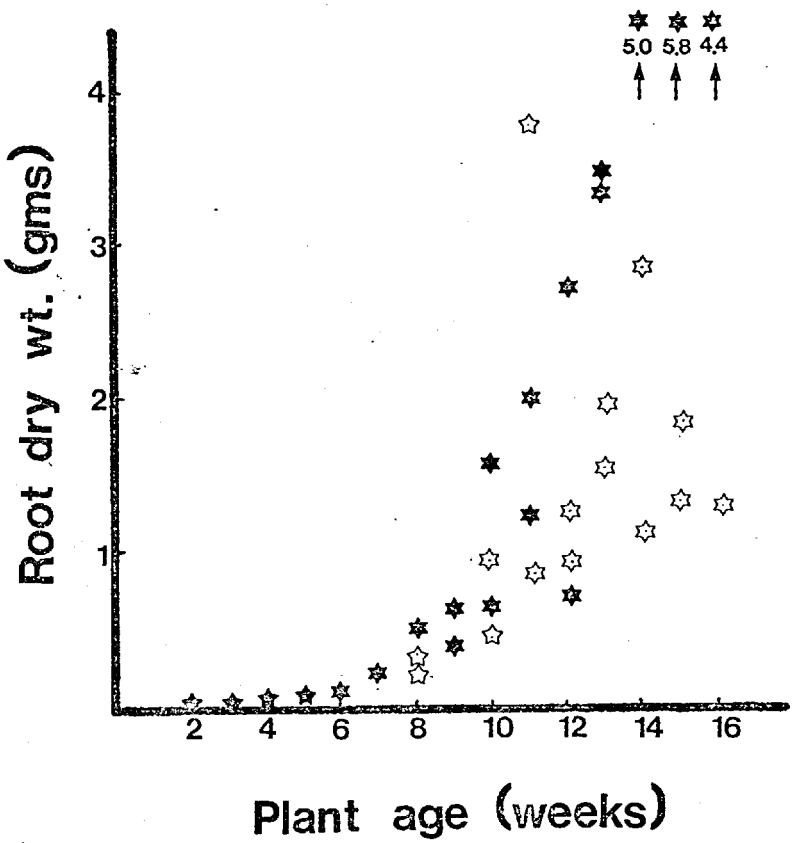


Figure 24. The pattern of root growth in control (✡) and treatment (✡) plants.

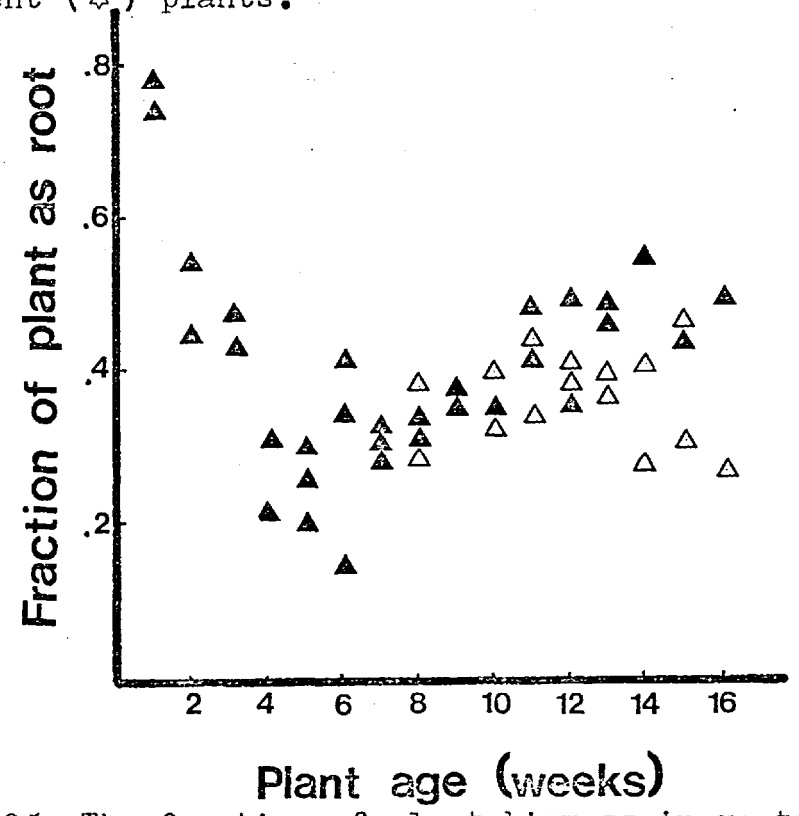


Figure 25. The fraction of plant biomass in roots for control (▲) and treatment (△) plants.

Figure 24 shows that less growth occurs in the roots of infested plants, but it can be redrawn to establish whether the proportion of net production channelled into root growth has been altered. We assess this effect by comparing changes in the fraction of plant dry weight in roots at different plant ages between treatment and control plants (Fig. 25). The observed reduction in the relative growth of roots suggests that the plant compensates for dry matter losses occurring in the shoot by increasing the fraction of dry matter production channelled into shoot growth.

The main interest in the effect of aphid infestation on the pattern of dry matter distribution centres on the shoot, and on the fraction of production going into fruit growth in particular. Unfortunately, all the treatment plants died prior to seed set (the infestation was made too early), and so we can gain no insight into this aspect of the process. It is interesting, however, to observe the effects of aphid feeding on the relative growth of leaves, stems and petioles.

Fig. 26 shows the distribution of leaf weights between different nodes of the plant at three ages (11, 13 and 15 weeks) in infested and control plants. The control plants have more leaves expanded, and more leaf dry weight per node than their infested counterparts. The gross pattern of leaf distribution with height is more or less similar, however, with the maximum leaf weight occurring mid-way between the highest and lowest leaf-bearing nodes.

We can now compare the distribution of dry matter within a node between leaf, stem and petiole tissues. Consider the fate of the first leaf produced after the aphid infestation

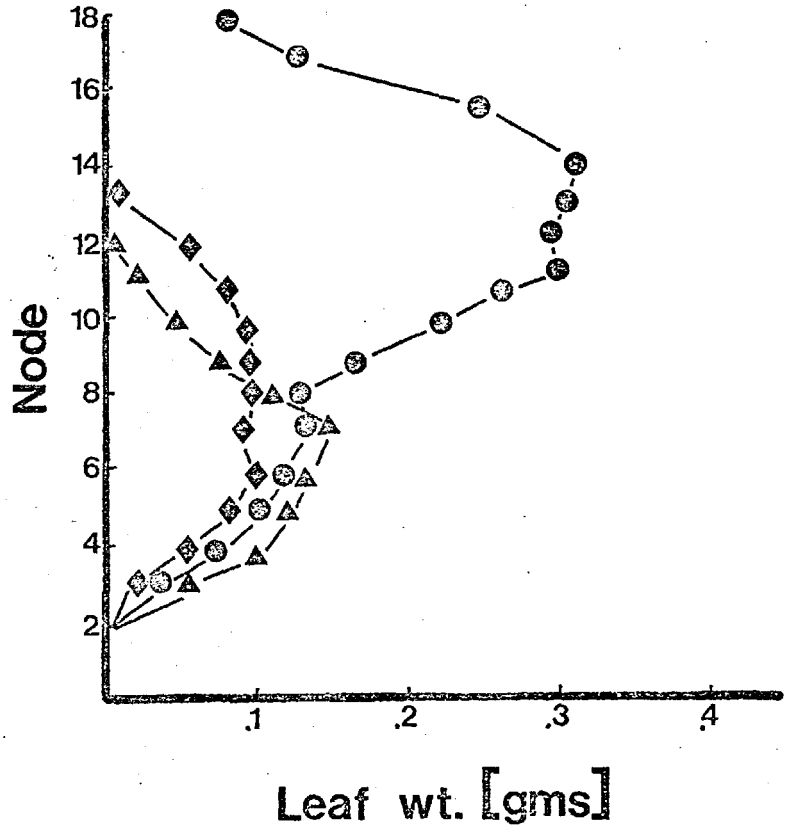


Figure 26. Distribution of leaf weight with height in control plants. Plant age 11 (▲), 13 (◆), and 15 (●)

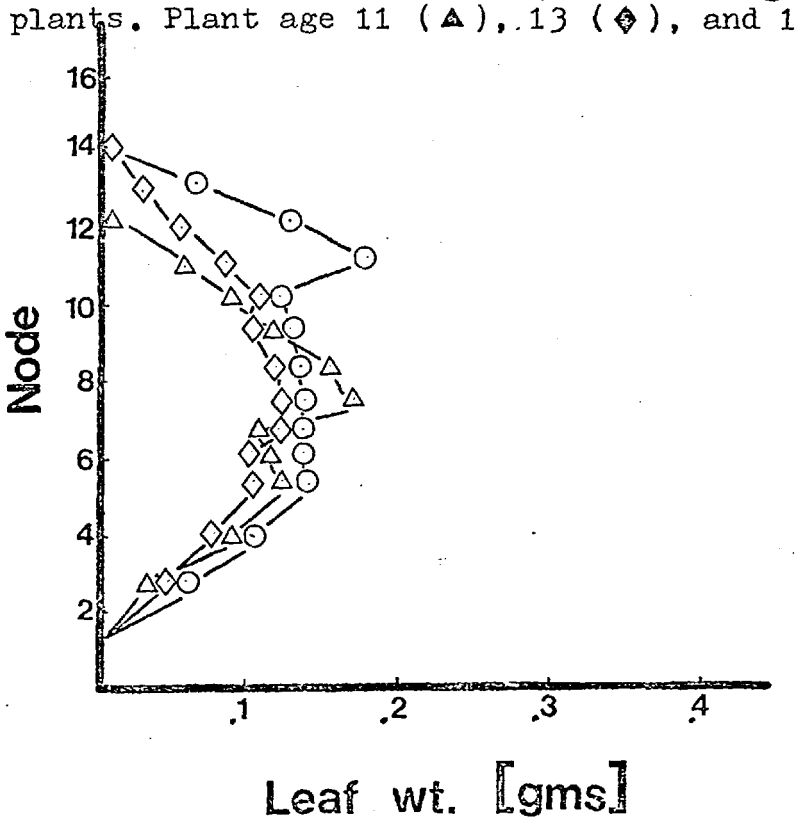


Figure 26. Distribution of leaf weights with height in infested plants. Plant age 11 (△), 13 (◇), and 15 (○).

(the seventh). This leaf will have been affected by the aphids from its appearance, because the aphids were introduced into the terminal cluster of leaves. In the controls, the proportion of nodal biomass in stem increases as the node ages; the seventh is an essentially structural node (it does not bear flowers), and the stem elongates and thickens considerably. In the treatment plants, on the other hand, stem elongation is negligible, due not only to a reduction in the growth of the whole node, but also to a reduction in the proportion of dry weight allocated to the stem (Fig. 27).

The relative growth of the organs in a node formed after aphid infestation is different. In the control plants, stem fraction remains rather constant, and the decrease in the fraction of leaf is matched by an increase in the fraction of fruit (Fig. 28). Infested plants, in contrast, show the same pattern as before, with an increase in the fraction of leaf relative to stem.

Finally, we can compare the relative growth within a node formed before aphid infestation (the fifth). Here, the fractions of leaf and stem are constant in the infested plants, but stem comprises a much higher fraction of nodal weight in the control plants (Fig. 29).

The causes of these differences seem to be threefold. First, by reducing the size of the infested plants, the aphids reduce the 'need' of the plant to allocate reserves to structural thickening of the lower stem sections; there is less shoot for them to support. Second, by reducing the amount of reserve available for growth at each node, the aphids are capable of suppressing flower production (Fig. 28). Third, aphid feeding on leaves may reduce leaf growth less than

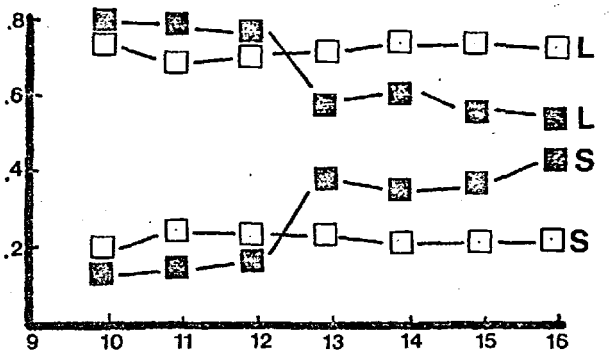


Figure 27. Fractions of biomass in leaves (L) and stems (S) for treatment (□) and control (■) plants of different ages. Seventh node.

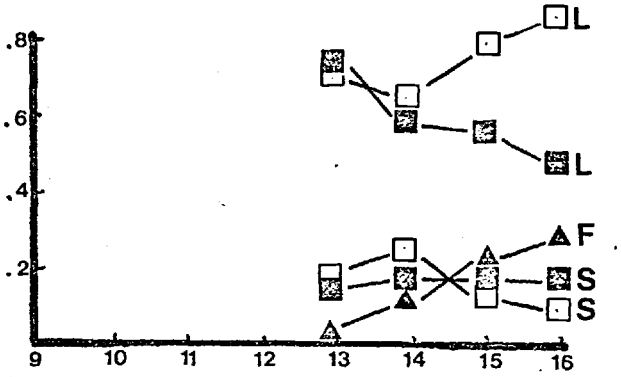


Figure 28. Fractions of biomass in leaves (L), stems (S) and fruit (F) at the 11th node. Treatment plants (□) and controls (■).

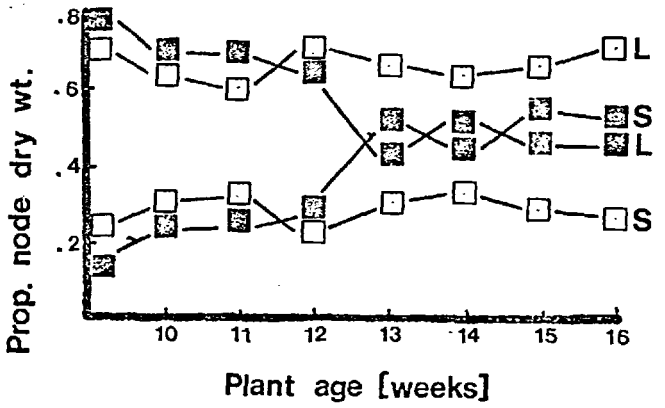


Figure 29. Fractions of biomass in leaves (L) and stems (S) for the fifth node in treatment (□) and control (■) plants.

aphid feeding on stems reduces stem growth. The possible mechanisms for this effect remain to be elucidated.

g) Aphid Population

At each node of the plant the course of aphid population change was of a similar pattern; a spell of immigration from other nodes was followed by a rapid build-up in numbers, then a steep decline occurred, associated with both apterous and alate emigration. The nodal populations, however, were out of phase with one another, so that aggregates were declining in size at some nodes, while increasing at others. The population build-up on the entire plant is shown in Fig. 30, and the pattern at different nodes in Fig. 31.

Few predators and parasites entered the experimental greenhouse, and so the majority of aphid deaths must be ascribed to occidents, old age, starvation, and losses during dispersal. The reproductive rate of individual virginoparae was not measured, but several estimates exist in the literature (Banks and Macaulay, 1964; Milne, 1971; Sharma, 1971). From these we can calculate the potential increase rates of the population at different mortality patterns. Using the simple model of aphid population growth described in Chapter IV (used in determining the reproductive values of aphids under different predation regimes) it is possible to plot the numerical increase which would be observed with different birth and death rates. These are compared with the experimental outcome in Fig. 32. Clearly, the greenhouse population increased at a lower rate than it might have done under optimal conditions. Since mortality and emigration appeared to be negligible in the early stages, it is likely that this

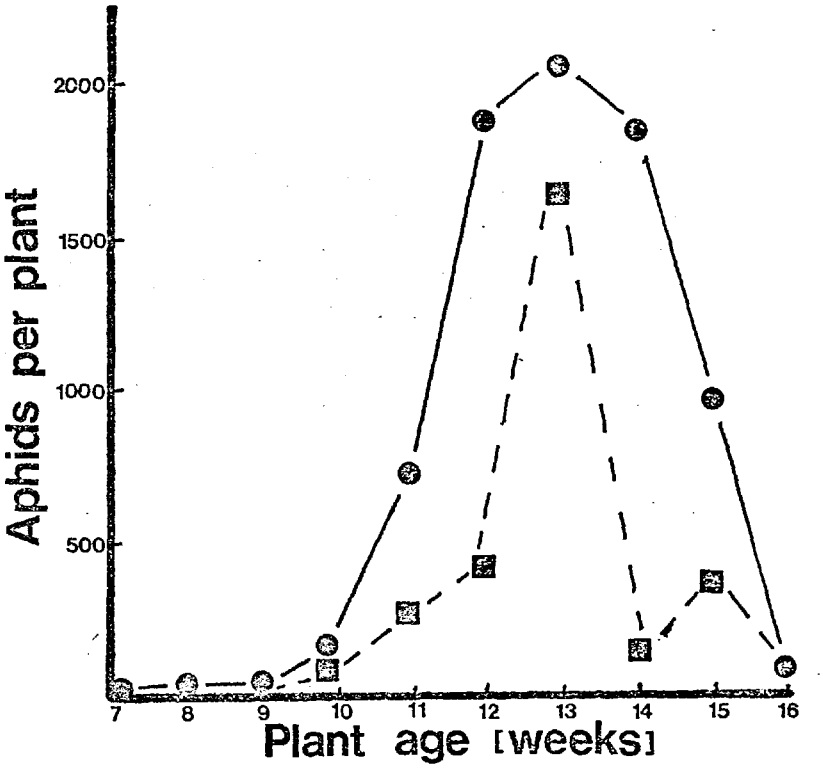


Figure 30. The number of aphids per plant on large (●) and small (■) plants at different plant ages.

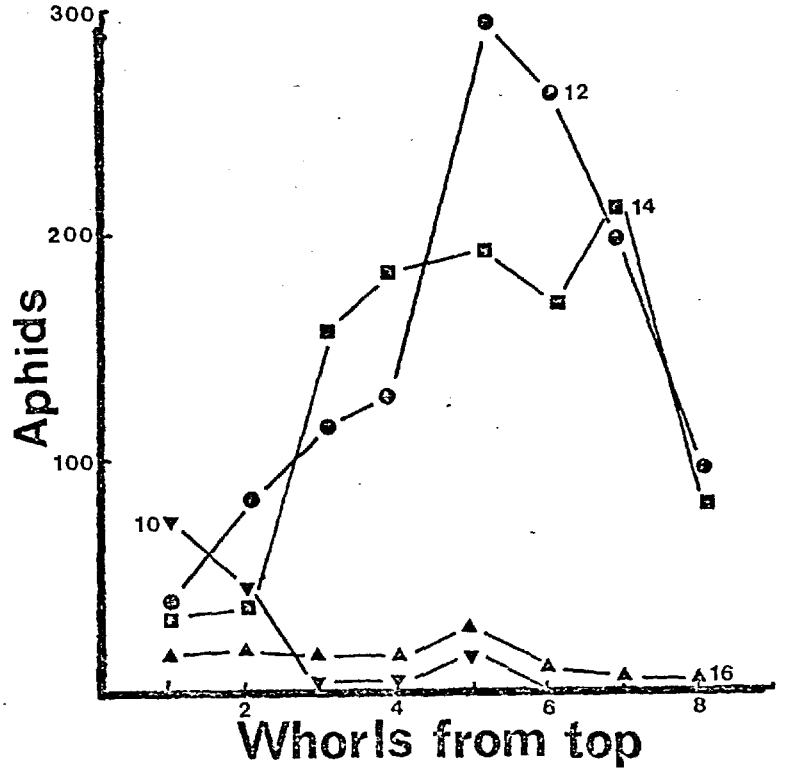


Figure 31. The pattern of aphid abundance at different heights below the top of the plant at four stages in population development (weeks from planting)

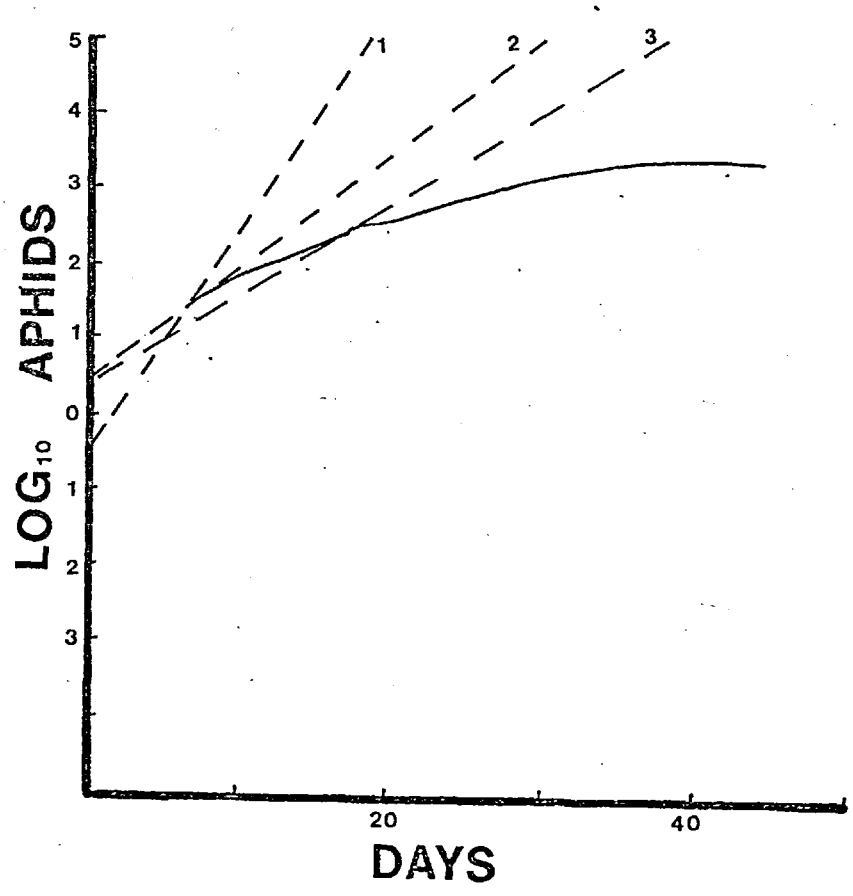


Figure 32. The actual population build-up observed (——) compared with three calculated curves based on the fecundity data of Banks and Macaulay (1964) and assuming 95% (1), 80% (2) and 70% (3) daily survival.

reduction is due to a depression of individual reproductive rates. This could be brought about by the effects of high or low density (Way, 1968), or by the sub-optimal nutritional condition of the plant (Auclair, 1963). Because the population grew less quickly than predicted by the accepted fecundity and survival values it is clearly unwise to use these directly in pest control decisions; they would lead to a consistent over-estimation of aphid damage (see Chapter IV).

The distribution of aphid numbers over the whole plant changed as the infestation progressed. In the early stages, the uppermost leaves and stems bore the densest aphid aggregates, as shown for the curve for 10 weeks in Fig. 33. At 12 weeks the aphids were more numerous on all nodes, but the peak numbers occurred lower down the plant. Between the 12th and 14th weeks the aphid populations on the lower leaves decreased, while numbers on the higher nodes increased, and later still, as the population crashed, the distribution of aphids with height became much more even, and all aggregates were reduced to very low numbers (curve 16 in Fig 33).

h) Damage

The simplest representation of the damage caused by the aphid infestation is a plot of the difference between the dry weights of control and treatment plants against time (Fig. 34). From this figure, it is clear that there is a rather abrupt threshold between 13 and 14 weeks, after which plant damage increases very rapidly with time. This is due to the cessation of growth in most of the treatment plants after 14 weeks. Up to this point, however, the plant seems to be affected rather little by the aphid infestation, and it is interesting to investigate the damage (in terms of dry weight

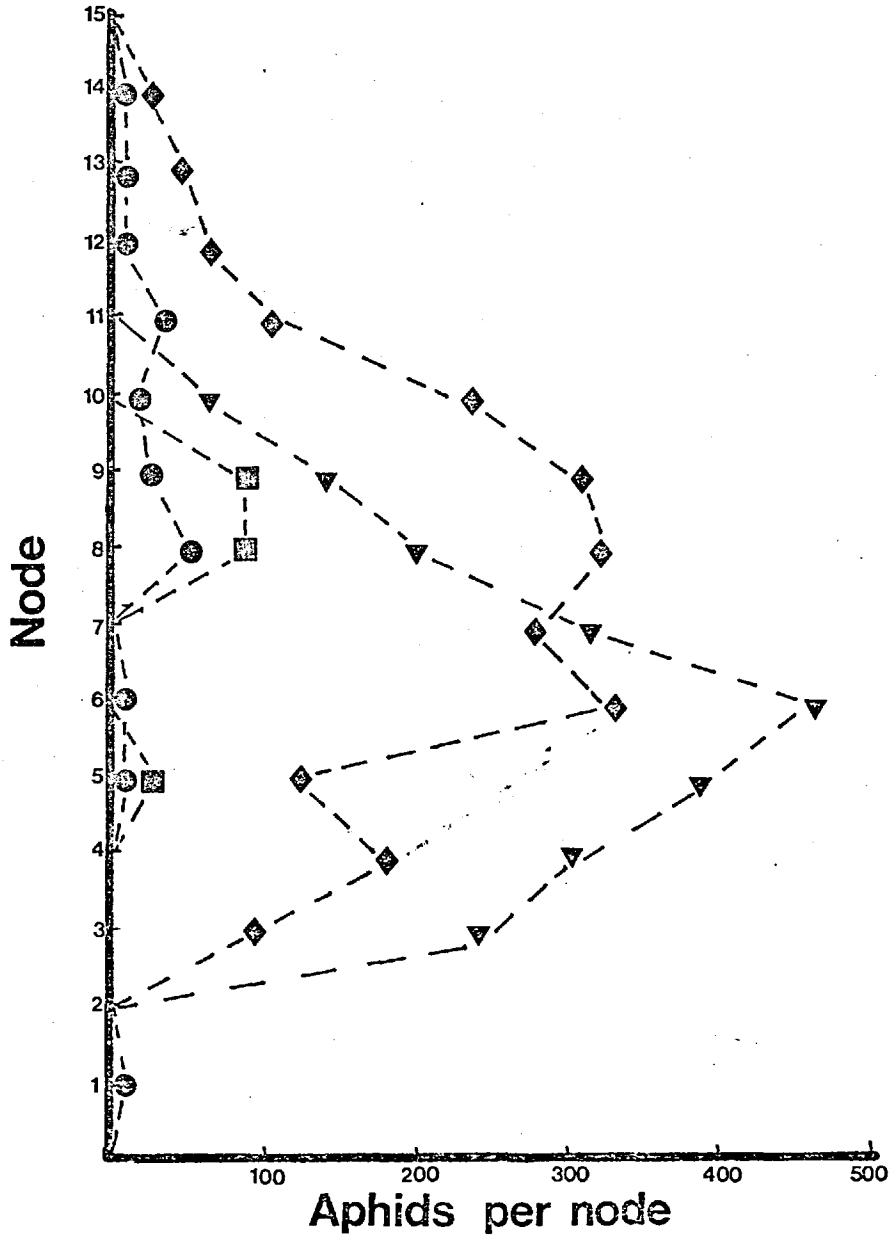


Figure 33. The number of aphids at each node of the plant at 10 (▣), 12 (▼), 14 (◆) and 16 (⊙) weeks.

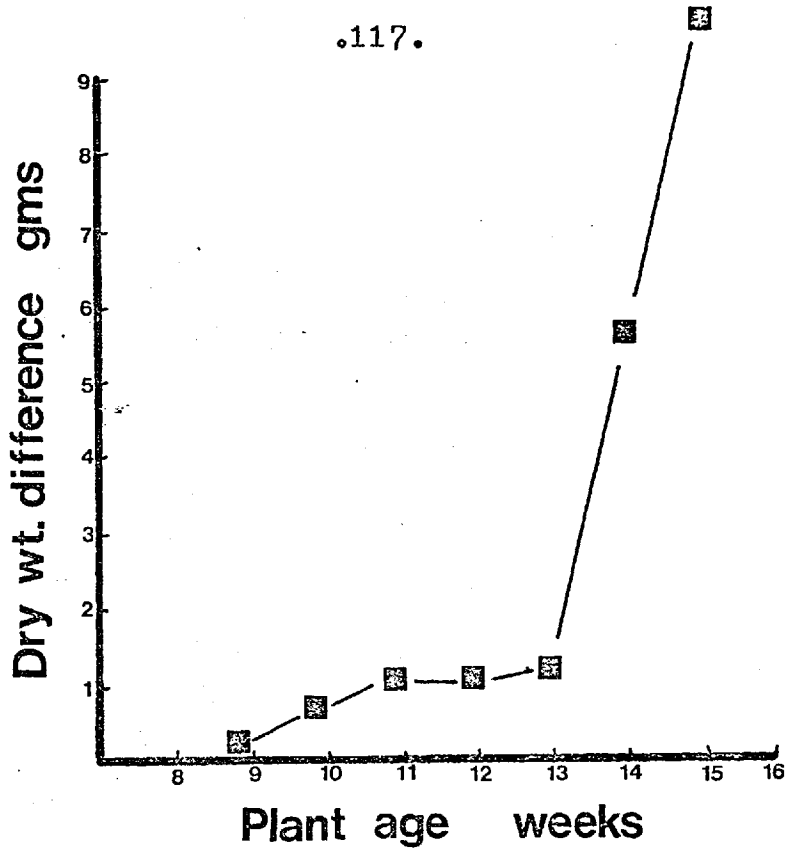


Figure 34. The difference in dry weight between control and treatment plants at different times.

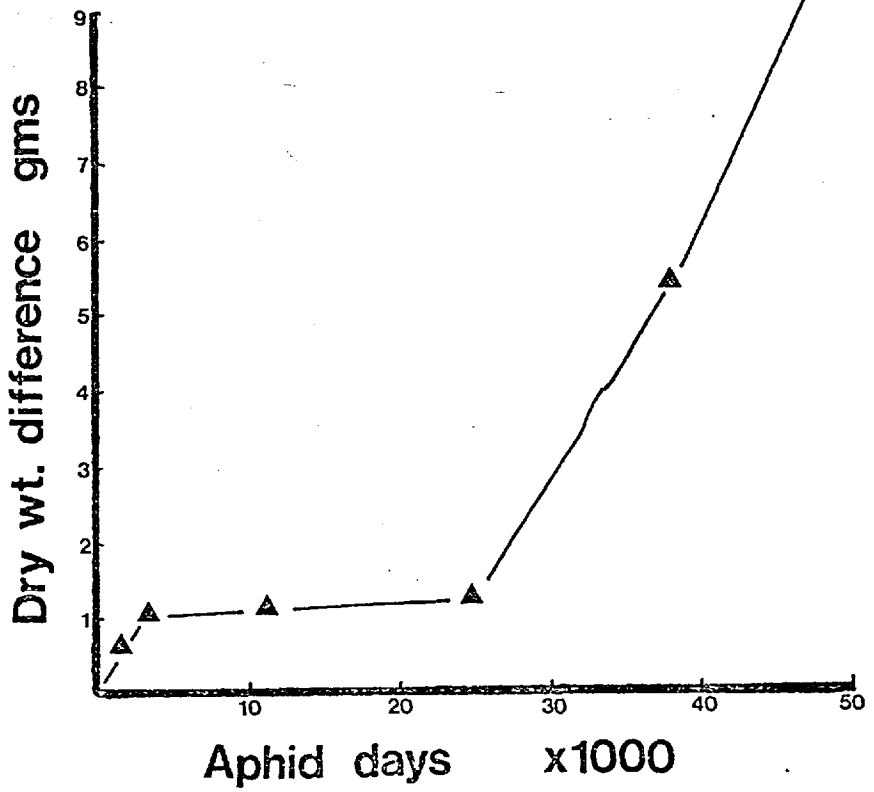


Figure 35. The difference in dry weight between control and treatment plants plotted against the total number of aphid-days experienced by the plant.

difference) caused by unit aphid feeding. Let us assume that the amount of food removed from the plant per day by the 'average aphid' can be considered as a constant, and also that the effects of aphid feeding are additive through time. If these assumptions are robust, then we should expect a linear relationship between yield reduction and the total number of feeding-days experienced by the plant (hereafter called aphid-days). We therefore replot Fig. 34 using the number of aphid-days on the x-axis; the figure for aphid-days is the approximate integral of the aphid abundance curve (Fig. 30) up to the mid-point of the week in question. Fig. 35 shows that the null hypothesis of constant damage per aphid-day does not hold, rather the curve seems to consist of three sections. There is an initial phase of quite high damage per aphid-day ($0.34\ 10^{-3}$ gms aphid-day^{-1}) followed by an extended period when the damage is far lower ($0.0061\ 10^{-3}$ gms aphid-day^{-1}). Finally, when the accumulated aphid population reaches 25,000 aphid-days the damage function again becomes quite steep ($0.33\ 10^{-3}$ gms aphid-day^{-1}). The discrepancy between the experimental results and the null hypothesis could be due to the inapplicability of the assumptions, but it could equally well be brought about by compensation for food removal in the mature plant.

To investigate the possibility of plant compensation we can compute the mean weekly rate of dry matter production per sq. cm. of leaf in control and treatment plants (Fig. 36). We should expect that the unmodified results show a lower unit leaf rate in the treatment plants, because of the dry matter removed by the aphids. This is what we observe. If, however, we can estimate the actual amount of food removed

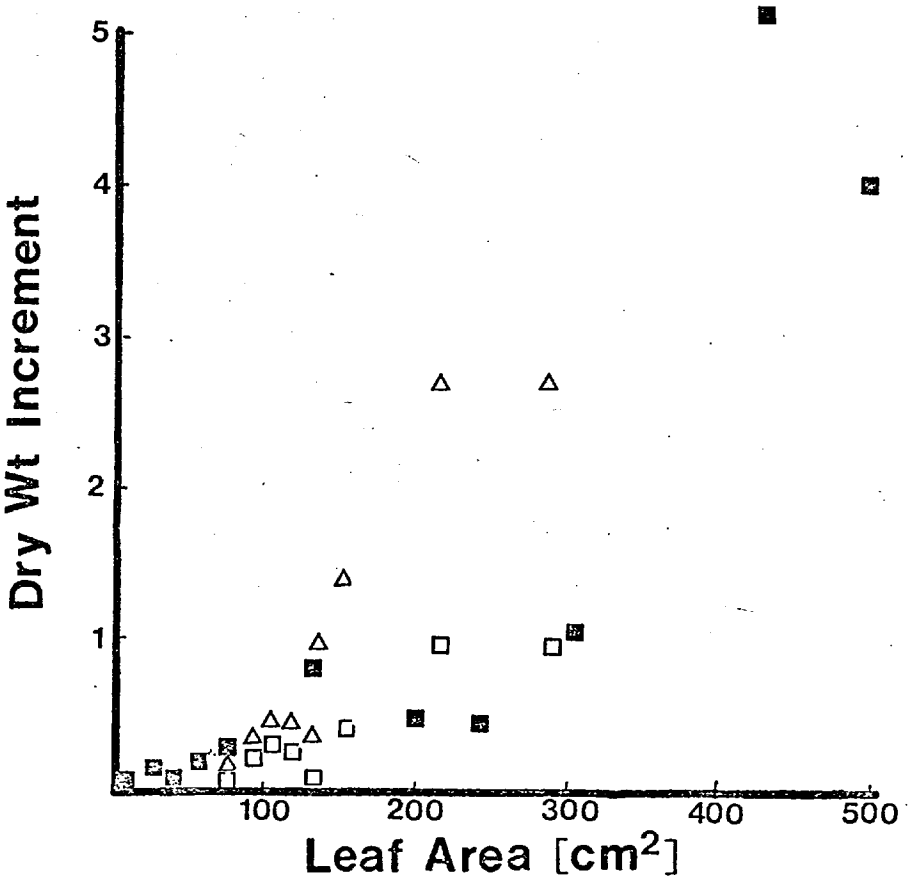


Figure 36. The relationship between leaf area and dry weight increment of the plant (the unit leaf rate, ULR). The ULR of treatment plants is rather lower than that of the controls (compare □ and ■). When estimated food removal is added to the treatment dry weight increments (△) the points are generally higher than the controls (■). This suggests that the plant has some ability to compensate by increasing its ULR

per aphid-day from an independent set of data, then it should be possible to suggest whether the plant can compensate for aphid feeding by increasing the rate of dry matter production. Banks and Macaulay (1964) calculate that the mean weight of sap removed by Aphis fabae in its lifetime is in the order of 30.0 mg. Now if the aphid lives 25 days (same data) we have a mean daily intake of 30/25 = 1.12 mg of sap, and taking the dry matter content of phloem sap to be 15% by weight (Auclair, 1963), we have a dry weight removal rate of 0.18 mg per aphid-day, and hence of 1.126 mg per aphid-week.

If we can approximate the number of aphids feeding for a whole week from the mean; i.e.

$$A = (APHID_{t} + APHID_{t+1}) / 2$$

then the total dry weight removed by aphid feeding during the week will be 1.126 * A mg. By adding this to the weekly production of the treatment plants we can recompute thier unit leaf rate from

$$ULR = (P + 1.126 * A) / LA$$

where P is the actual dry matter production and LA is the leaf area. If the plant is compensating for aphid feeding, then the plot of net production with aphid feeding added against leaf area should have a gradient greater than the curve for the control plants (Fig. 36). If the curves are of equal gradient, then the plant is producing dry matter at exactly the same rate with and without aphid feeding, while if the adjusted unit leaf rate of the treatment plants is lower than that of the controls we could suggest that the aphids were inhibiting the productive process. The data in

Fig. 36 are inconclusive for the most part, but there is a suggestion that some compensation occurs, especially at intermediate leaf areas (which match the period of low damage per aphid-day in Fig. 35). In any event, the compensation was quite insufficient under such intense aphid infestation as the plants experienced in the greenhouse, and all the treatment plants died. In the field, however, where the aphid populations might be considerably lower (predation, parasitism, rain, frost and so on), and peak density could occur much later in plant development, plant compensation could act as an important mechanism in defraying bean yield losses.

All the conclusions emerging from this experiment must be considered in relation to its several shortcomings. Principal amongst these is the lack of statistical rigour arising from the very low replication of each observation. It would be advisable to have at least four replicates of control and treatment plants at each harvest instead of the two used here; although the labour and greenhouse space would be doubled, the precision would be greatly enhanced.

Again, only one time of infestation was studied, and this turned out to be rather early, since almost all the treatment plants died before flowering. It would be interesting to know how the date of infestation affected both the rate of dry matter production and its pattern of distribution between the organs of the plant, and particularly the effects of aphid feeding on seed-fill. On altering the date of infestation in a model of the growth of Vicia faba (Chapter III) it has been observed that yield increases asymptotically with the time lag between sowing and infestation (Fig. 37), and it would be useful to validate this result with real bean plants.

A number of factors in the greenhouse environment will

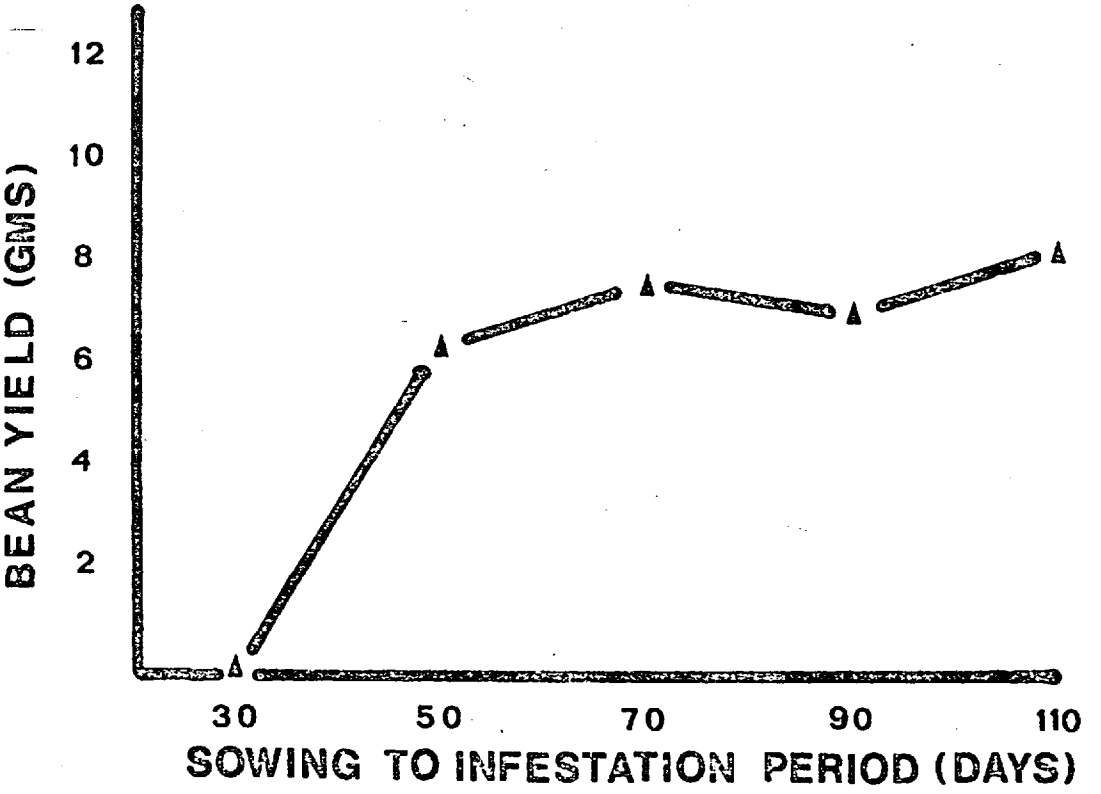


Figure 37. The output gained from a model of Vicia growth (Chapter III) which shows the importance of the date of aphid infestation relative to crop development in affecting final yield. In all cases a total of 14 gms of material were removed from the plant.

have affected the course of dry matter production and distribution differently than would the same factors in the field. Fluctuating light and temperature conditions, for example, are known to affect photosynthetic rate and aphid reproduction in the field (McCree and Loomis, 1969; Strain and Chase, 1966).

Discussion

Plants infested with aphids showed a lower rate of dry matter increase than the controls. First, the aphids removed a certain amount of photosynthate directly (about 0.18 mg per aphid-day), and second, by reducing the rate of appearance of new leaves, they lowered the potential for future dry matter production. The aphids may also have acted to affect the rate of photosynthesis by injecting chemicals with their saliva (Miles, 1968), or by damaging the surface of the leaves, but this can not be shown from the present experiment.

The pattern of dry matter distribution was also affected by aphid feeding. The roots of infested plants grew relatively less (Fig. 25), and, while leaf weight showed the same overall distribution with plant height (Fig. 26), it formed a larger fraction of nodal biomass than in the controls (Figs. 27, 28, and 29). This could be because a) infested plants are smaller, and their stems, having less shoot weight to support, need not be so thickened, or b) aphid feeding on leaves reduces leaf growth less than aphid feeding on stems reduces stem growth. The growth of stems is affected by the number of aphids feeding directly upon them (Fig. 23), which suggests that they are affected as if they were sink organs (and not merely pipelines through which reserves travel to other parts of the plant).

The leaf water content of aphid infested leaves is lower than that of leaves of the same age from control plants (Fig. 22); this suggests that the plant is unable to compensate for high rates of water removal, and this, in turn, might have a detrimental effect on the rate of dry matter production (Brix, 1962; Troughton and Slatyer, 1969).

Flower production was inhibited in infested plants (Fig. 28); this could be due to their reduced stature (Fig. 19), or to the indirect effects of aphid feeding (leaf damage, injections, and so on).

The ability of Vicia faba to compensate for aphid feeding appears to be rather limited when the aphid infestation begins as early as it did in this experiment (Fig. 36). There is a period, however, between about 5,000 and 25,000 aphid-days, when the rate of damage to the plant is less than the rate of feeding ($0.0061\ 10^{-3}$ compared with $0.180\ 10^{-3}$ gms per aphid-day; see Fig. 35). Under field conditions, when aphid populations would be lower, and infestation occur later, this compensation ability may buffer losses in yield to a significant extent. In general, it should be possible to tell whether a plant is compensating or not from quite straightforward data. If we plot estimated aphid dry matter removal as one block of a histogram and compare this with the corrected net production of the plant (net growth plus aphid feeding) we would obtain one of the cases shown in Fig. 38. The type of response might change through time, associated with changes in the sensitivity of different phenological conditions of the plant, and different intensities of aphid feeding.

In terms of pest control, it will be more important to slow down the rate of aphid feeding when the plant is under-

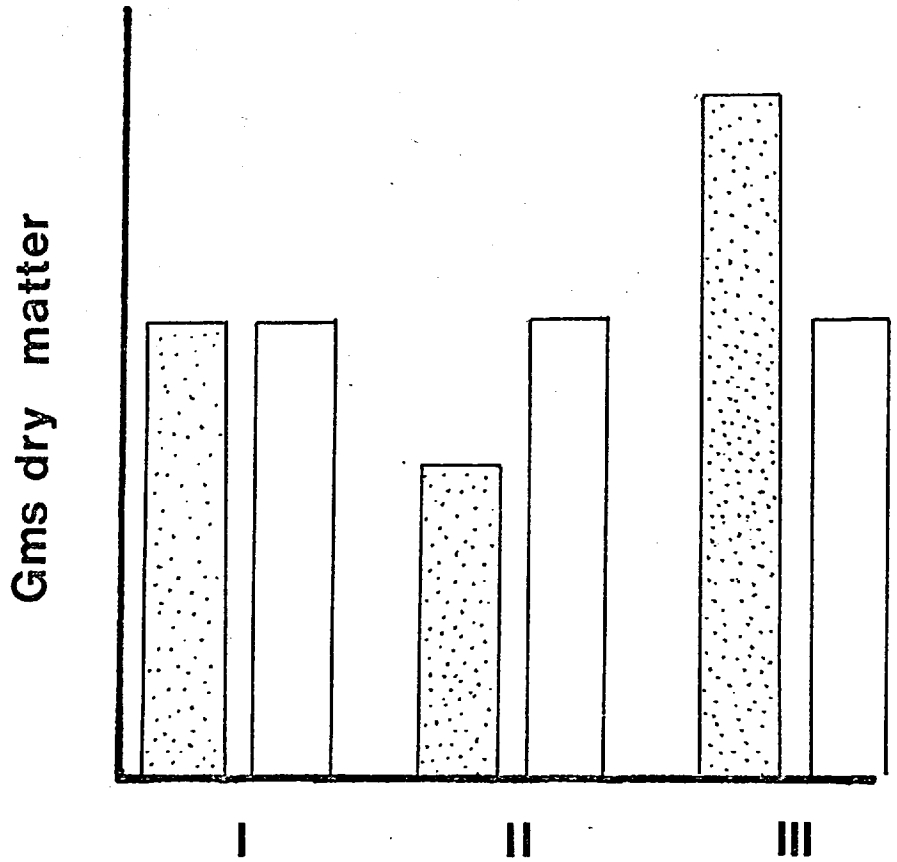


Figure 38. A simple means of demonstrating plant compensation. The stippled columns represent plant yield reduction; the open, aphid food removal.
I. Damage equal to feeding, the null case
II. Damage less than feeding, compensation occurring
III. Damage greater than feeding, a run down in the productive processes themselves.

compensating (i.e. when it is suffering damage in excess of aphid food removal due to a run-down of the photosynthetic machinery), than when the unit leaf rate has been increased, and the plant is suffering less dry matter loss than occurs through aphid feeding. The mechanisms of this compensation response are discussed in the following chapter.

Similarly, because the plants appear to be very sensitive to the time of infestation, the necessity for pest control measures increases with the earliness of aphid arrival in the crop. Control measures must be related to the intensity of the infestation as well as the time of its occurrence, because it would be unrewarding (not to say wasteful) to spray an entire field, or release a costly batch of natural enemies, when only one or two aphids are observed in the crop at an early date. Ideally, there should be some experimentally determined threshold density of aphids related both to the time of year and to the developmental stage of the plant, below which pest control can be said to be unnecessary, and above which an economic return is likely (through enhanced yield defraying the costs of the control measures).

This experiment shows us how Vicia faba responds to Aphis fabae infestation under one set of greenhouse conditions. Our main objective, however, is to discover the pattern of aphid feeding which can be tolerated before a significant economic loss of beans occurs under a range of field conditions. To increase the generality of the findings, I shall now discuss a simulation model of Vicia growth, which uses information from uninfested plants (collected by Ishag, 1969) to predict the potential bean yield under different conditions, and upon which we can superimpose a number of different models for the

effects of aphid feeding on plant growth. I can then use the results of the present experiment to suggest which of the models of aphid feeding produces the most realistic responses in the model plant.

CHAPTER III A SIMULATION MODEL OF THE GROWTH AND FRUIT DEVELOPMENT OF AN ANNUAL PLANT UNDER APHID ATTACK

Introduction

The effects of phytophagous pest insects are generally assessed in terms of the yield reduction they cause. In any given situation, however, it is difficult to predict the degree of damage with any precision because the regression of damage against insect numbers often shows only a poor fit (see Stern, 1973; Hussey and Parr, 1963). In designing strategies of integrated and biological pest control it is important to know not only how to reduce pest numbers, but also the response of the crop to various levels of attack at different stages in its development. It may be that the most effective control (the economic optimum) does not involve a minimization of total pest numbers, but rather a course of control related to the sensitivity of the plant to damage at different times.

The present model has been developed to investigate

the effects of different levels of feeding by Aphis fabae on the growth and seed yield of the broad bean, Vicia faba. The model deals with the effects of the timing of aphid infestation relative to plant development, and with the effects of different spatial distributions of aphid feeding within the plant canopy and between the plant organs. By determining the periods during which the plant is most sensitive to damage by aphid feeding, and the quantitative responses of bean yield to aphid numbers, it should be possible to suggest the basis of a rational and biologically feasible strategy for aphid control on this crop.

It seems to be a sound generality that the value of an ecological model increases in relation to the precision with which its objectives are explicitly stated. There are two principal reasons for this. First, a rigid definition of the problem to be tackled allows us to make a clear and concise delimination of the structural aspects of the sub-system to be modelled. Second, a clear picture of the objectives is invaluable once the model has been built, in deciding on the set of runs to perform (Chapter I).

Plant Growth Models

Plant growth models generally consist of two sections; one concerned with predicting the rate of net biomass increase, and another with determining the pattern of distribution of this net production between the different plant organs. There has been a tendency in the past to build models which concentrate on one aspect of the problem, and to treat either the processes of photosynthesis and respiration (Anderson, 1968, 1970; Connor and Cartledge, 1970; Gaastra, 1959, 1963; Lake,

1967; Monteith, 1963, 1966; Neals and Incoll, 1968; Pearce, 1967; Ross, 1964, 1966, 1970; Tooming, 1970), or the pattern of dry matter distribution (Monsi and Murata, 1970; Brouwer, 1962; Davidson and Milthorpe, 1965; Alberda and de Wit, 1961) in relatively greater detail. A number of rather complex simulation models do exist in which both net production and dry matter distribution are described in detail, notably ELCROS (the Elementary Crop Growth Simulator) from the Dutch school of de Wit and his colleagues (de Wit and Brouwer, 1968; Brouwer and de Wit, 1968; de Wit et al., 1970).

1 Net Production

The processes which are normally considered as acting to affect the rate of net production are diagrammed in Fig. 39. The interception of light has commanded considerable attention in the literature (Alberda and de Wit, 1961; Anderson, 1964, 1968; Black, 1963; Cowan, 1968; Donald, 1961; Kriedman et al., 1964; Loomis et al., 1968; McCree and Troughton, 1966; Monteith, 1965; Saeki, 1963). The variables of importance are the intensity of the incident radiation in the photosynthetic wavelength spectrum, its duration (photoperiod), and the amount and spatial disposition of leaf tissue available to intercept it. The relationship between the actual amount of leaf present, and the effective leaf area is complex, and depends upon the angles of the leaves relative to the sun (Loomis et al., 1968; Monteith, 1965), the depth of the canopy (Anderson, 1968; Ross, 1970), and the degree to which the leaves shade oneanother (Donald, 1961). The concept of leaf area index (LAI) (Watson, 1947; 1958), which expresses the area of leaf surface relative to the area of ground covered

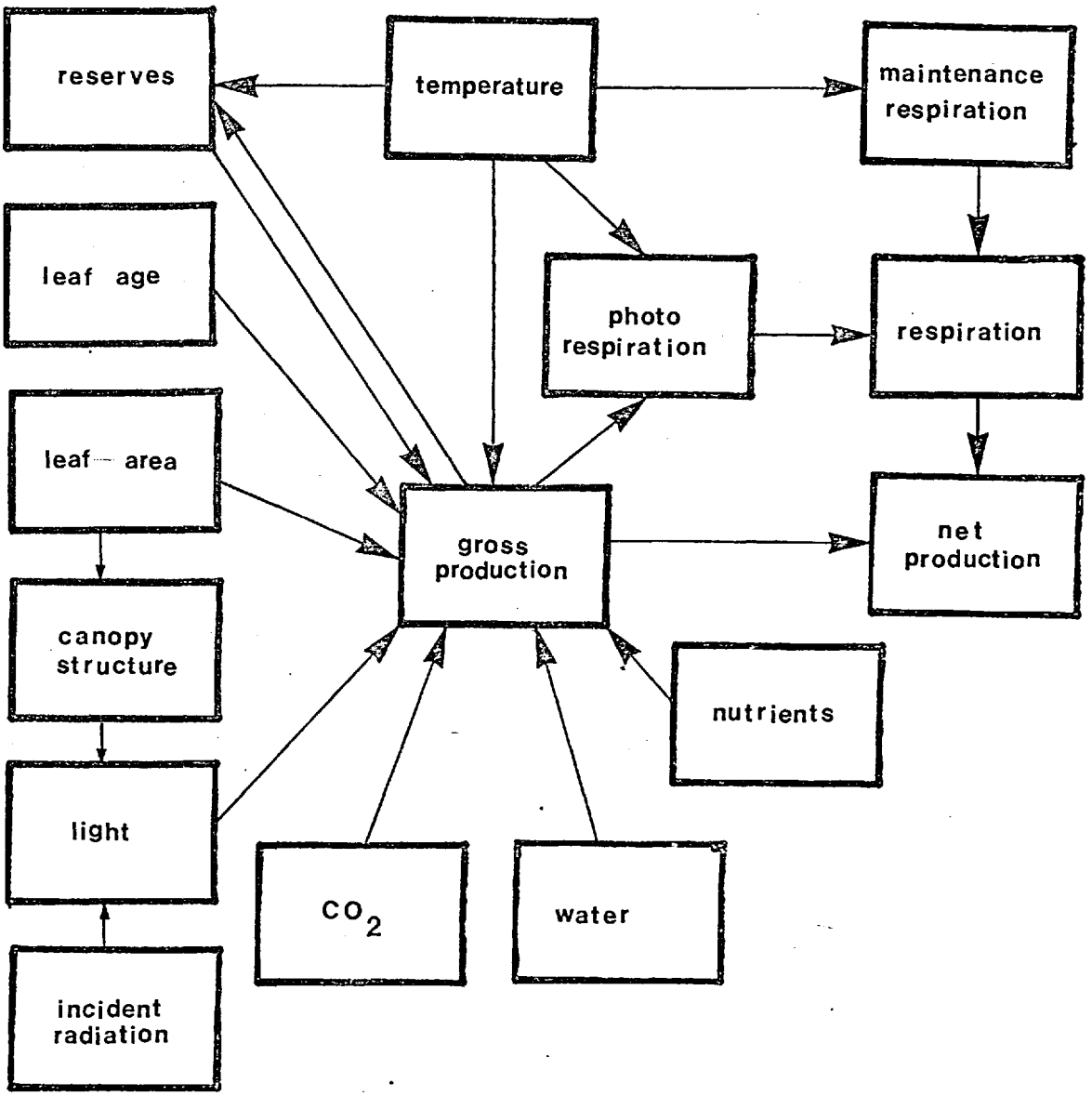


Figure 39. A flow diagram of the factors envisioned as affecting the rate of net production in many plant growth models

by the canopy, has been usefully employed in applied studies of agricultural producion to subsume the effects of individual leaf angles and variations in leaf distribution through the canopy (e.g. Black, 1964).

The conversion of a given flux of radiation into a store of potential energy in carbohydrates occurs when water and carbon dioxide are combined in photosynthesis. A model of this process can operate by considering the proportional efficiency of the leaf in accumulating energy, or by calculating the rate at which CO_2 is fixed, in a given light intensity. Both models ignore the fact that the products of photosynthesis are converted into compounds of different calorific values and carbon contents, and that the proportion of any one type of compound in the plant might well change with time. A regression of the dry weight of organic material incorporated into the plant against either the energy fixed, or the weight of CO_2 assimilated, will therefore show an imperfect fit to empirical data.

Much of the detail in models of net production centres about those factors which affect the rate of photosynthesis, and, in particular, the effects of light intensity, air temperature, and CO_2 concentration (Gaastra, 1963). Fig. 40 shows the interaction between these three primary factors. In addition, experimental work has clearly shown the dependence of photosynthetic rate upon leaf age (Treharne et al., 1968), soil water availability (Schroder, 1966), stomatal aperture (Gaastra, 1963), and the level of accumulated reserves in the leaf (Neals and Incoll, 1968).

It would be difficult, not to say impractical, to incorporate these factors in the algebraic models of canopy

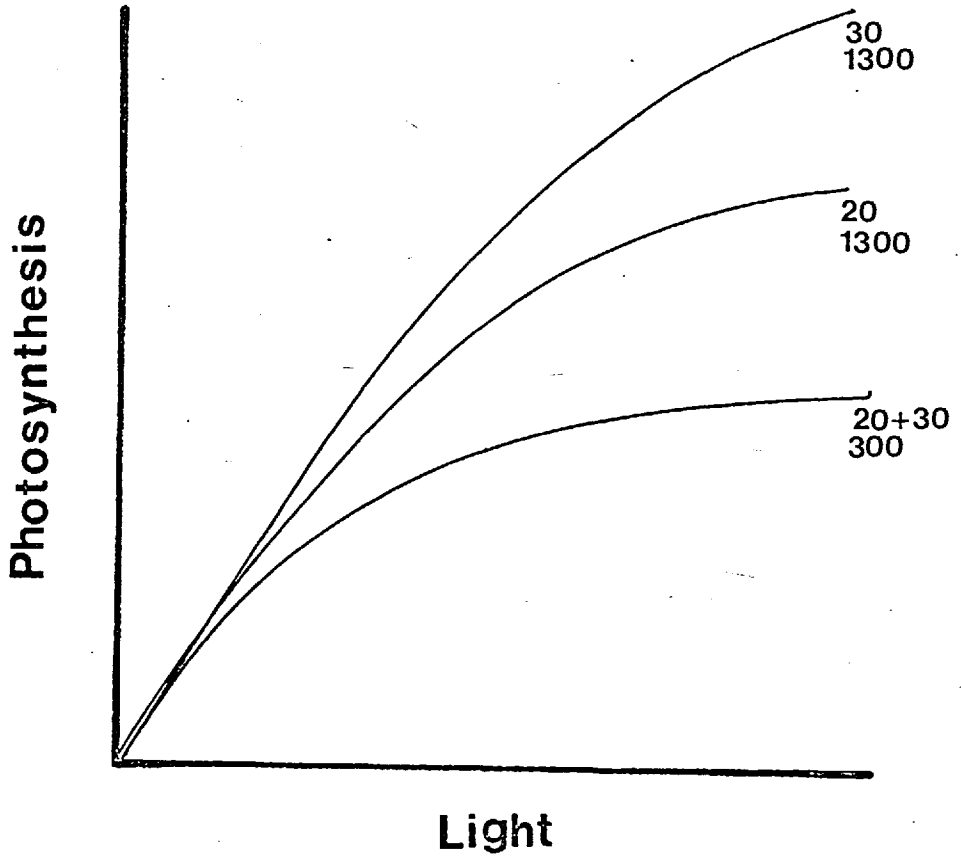


Figure 40. The interaction between temperature and carbon dioxide concentration in affecting the relationship between light and photosynthetic rate (after Gaastra, 1963). The upper figures are temeratures in centigrade, and the lower are CO2 concentrations in ppm

structure and photosynthesis (e.g. Tooming, 1970; Acock et al., 1970), but simulation models do hold the potential to deal with such complexity (see de Wit et al., 1970). The level of complexity at which a problem is tackled depends, however, not so much on the types of model available, as on the purpose to which the model will be put. It is quite clear that of a family of models which fulfil a given purpose the most useful is that which is conceptually most straightforward. Because our aim is to understand the dynamics of plant growth under aphid attack, we shall have to consider more variables than in prediction of yield reduction were our sole objective.

Gross production is computed as a function of those variables of crop architecture, plant physiology and environment which are relevant to a particular problem. In order to predict the amount of photosynthate available for growth (net production), it is necessary to calculate the loss of reserves through respiration. The classical picture is that presented by Davidson and Philip (1958), and shown in Fig. 41. This model assumes that respiration is proportional to the weight of leaves present, and that there is an optimal leaf area at which the difference between photosynthesis and respiration is maximal. The photosynthesis curve is asymptotic because of mutual shading of the leaves as the leaf area increases above a certail threshold (the optimum leaf area).

As de Wit et al. (1970) point out as a result of their simulations, and McCree and Troughton (1966) and Treguna et al. (1964) have shown experimentally, the respiration rate of crops is not proportional simply to some measure of above ground biomass, but is much more closely related to the

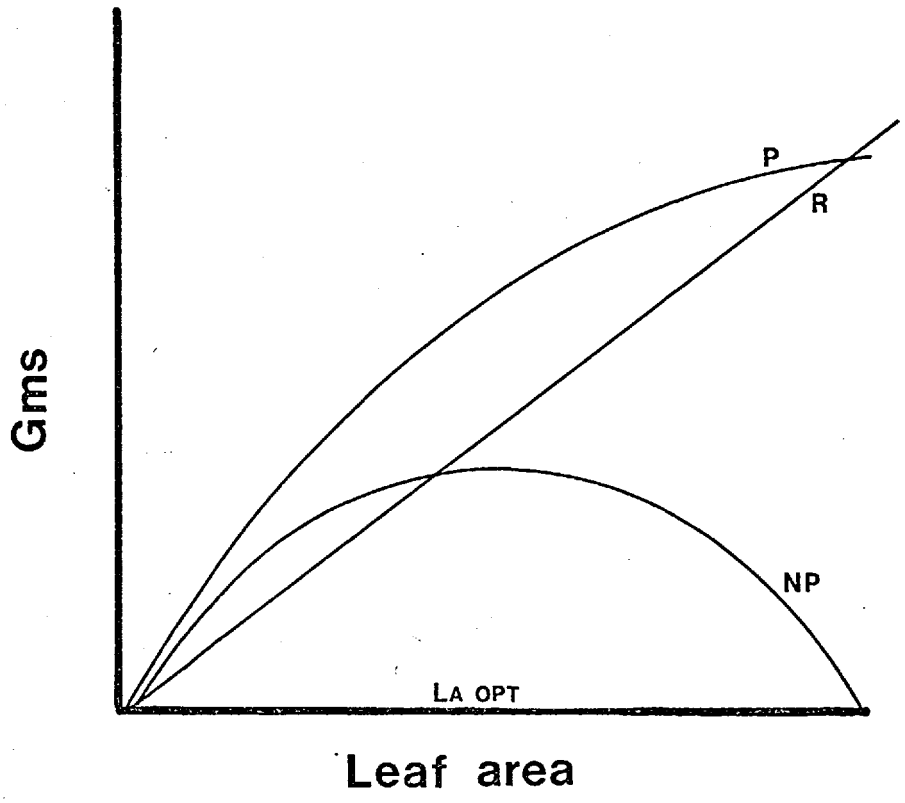


Figure 41. The classical picture of the relationship between leaf area and net production (see text)

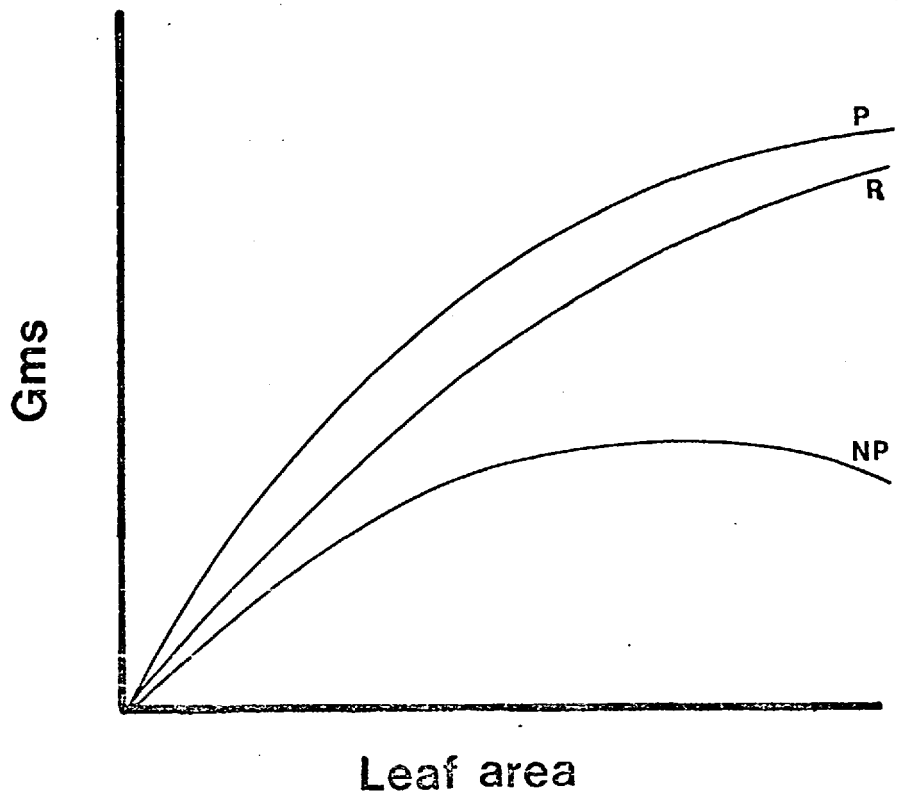


Figure 42. The modern picture (after McCree and Truoghton, 1966) including photorespiration (see text)

photosynthetic rate of the crop surface (see Fig. 42, after McCree and Troughton, 1966). The practice in models such as ELCROS (de Wit et al., 1970) has been to define a respiration coefficient which is expressed as the weight of dry matter which is lost during the synthesis of one weight unit of structural material. To this synthetic respiration must be added the loss of material necessary to sustain the productive machinery of the plant; the maintenance respiration. Using such a model de Wit has been able to produce simulated experiments whose results are in close agreement with the available data. This agreement is not possible when the assumption of simple weight dependence in respiration rate is employed in the model (loc. cit.).

The general practice in simulating photosynthesis is to assume that in optimal light conditions, the amount of dry matter produced is a function of the area of leaf available to intercept radiation (references above). If L is the weight of leaves, and d is the maximum rate of biomass accrual per unit leaf weight, we can assume that leaf area bears some linear relation to leaf weight and write

$$NP = d.L \qquad \ldots\ldots (1)$$

where NP is net production. As the models become more sophisticated, the constant term d is expanded to account for the effects of temperature, leaf age, reserve levels, and so on, while the area of leaf (or its weight equivalent L) is subdivided into strata to allow for light variations within the canopy. Considering photosynthesis within one stratum, we can put

$$d_i = f\,(\text{TEMP, LEAF AGE, RESERVES}) \qquad \ldots\ldots (2)$$

The light at this level in the canopy will be some reduced fraction of the radiation incident at the crop surface. If $\sum L$ is the accumulated leaf biomass above the ith layer, we can state that the light intensity I_i will be

$$I_i = I_o f\left(\sum_i^n L_j\right) \quad \ldots\ldots (3)$$

I_o being the incident radiation. It is often assumed that $f(\sum L)$ decreases exponentially with the depth in the canopy (e.g. Monsi and Saeki, 1953).

Photosynthesis can then be given by

$$p_i = d_i . L_i . I_i \quad \ldots\ldots (4)$$

for the ith layer, and for the plant as a whole by

$$P = \sum_1^n p_i \quad \ldots\ldots (5)$$

where n is the number of layers in the canopy.

Taking respiration to consist of two aspects, maintenance and photosynthetically coupled, we can follow McCree and Troughton (1966) and write

$$R = aP + bW \quad \ldots\ldots (6)$$

which states that the total dry weight loss through respiration (R) is simply a photosynthetic component (of value aP) added to a maintenance component (bW) determined by plant weight (W). This very simple model assumes the two aspects to be independent, but, more importantly, assumes a linear relation between photosynthetic rate and photorespiration, and between plant weight and maintenance respiration. Also implicit is

the assumption that maintenance respiration is independent of temperature, and photorespiration only temperature dependent to the extent that the rate of photosynthesis itself is affected by temperature. It is probably better to use a more general model form, and write

$$R = f(P).\, f'(T) + g(W).\, g'(T) \qquad \ldots\ldots (7)$$

where T is air temperature, and f'(T) does not necessarily equal g'(T). Here, no assumption is made about the shape of f(P) or g(W); these can be stated independently for a given plant species using real data.

Net production is now, quite simply,

$$NP = P - R \qquad \ldots\ldots (8)$$

and if the plant has lost no biomass through death or abscission, the new plant weight can be incremented by NP

2 Distribution of Dry Matter

For some purposes it may be quite sufficient to specify that a constant fraction of net production is channelled into leaf growth, and to simulate dry matter distribution simply by incrementing leaf weight by that fraction of current production; i.e.

$$WTL_{t+1} = WTL_{t} + f_{l} . NP$$

where WTL is the dry weight of leaf, f_l is the distribution fraction to leaf, and NP is net production.

Alternatively, if the pattern of dry matter distribution varies with time in a consistent fashion, it will be possible to set up a vector of distribution fractions describing the

proportion of net production added to leaves at each age of the plant; i.e. f_{lt} where t is plant age.

In the context of relating aphid feeding to bean yield we must consider rather more detail. We need to know the fraction channelled into fruit growth as well as into leaf, and also the effects of aphid feeding on this pattern. Aphid infestation may well alter the growth of some organs relative to others (Chapter II), in much the same way as grazing alters the distribution of dry matter in grasses (Troughton, 1960; Alberdā, 1966).

Monsi and Murata (1970) review the environmental factors affecting the pattern of dry matter distribution (with emphasis on light and soil-water conditions). They point out that the distribution fractions to different organs are quite responsive to external conditions; for example, transferring young soybean plants from a moist to a dry environment caused a doubling in the fraction of photosynthates incorporated into roots (20 to 40%). In ELCROS de Wit and his colleagues simulate dry matter distribution in relation to the relative growth rates of the organs. They assume a constant rate of leaf appearance, however, and this is unsuitable in the present model because aphid infestation is known to slow the rate of new leaf formation appreciably (Chapter II).

I have assumed that each organ of the plant at each node competes for the reserves available, and that dry matter is distributed in relation to the relative competitiveness of each organ. I have considered dry matter distribution as a net effect, so that a leaf which is importing more reserves than it exports has been designated a 'sink' leaf, while an organ exporting more reserves than it imports has been defined as a 'source'. In order to simulate the distribution

of net production it is necessary to quantify the competitiveness of each organ; that is, to determine its relative sink strength. This has been achieved by assuming first, that each plant organ at each node has some maximum weight which would be reached under optimal conditions for the growth of the species, and second, that in an undisturbed plant in a constant environment, this maximum weight would be approached in a logistic fashion.

If we let the maximum dry weight of the organ be K gms, the potential rate of dry matter accumularion be r, and the initial weight be W_o, then the weight of the organ at time t will be (using the integral equation for the logistic curve)

$$W_t = \frac{K}{1 + e^{-r.t\left[\frac{K - W_o}{W_o}\right]}} \qquad \ldots\ldots (9)$$

If we now express the logistic as a difference equation, we can compute the increase in dry weight over a given time interval (one day perhaps) from

$$W_t = W_{t-1} \cdot e^{r.\left(\frac{K - W_{t-1}}{K}\right)} \qquad \ldots\ldots (10)$$

and the weight increase is clearly $W_t - W_{t-1}$. Now if this is the potential weight increase of the organ under optimal conditions, it is also an indication of the sink strength of the organ, and so, even if the dry matter production of the whole plant (DM) is less than the total potential growth which all the organs W_{it} (i=1,2,... n; where n is the number of organs) could make; i.e.

$$DM < \sum_{i}^{n} (W_{it} - W_{it-1}) \qquad \ldots\ldots (11)$$

we might still expect the dry matter to be distributed in proportion to relative sink strength. That is,

$$F_j = \frac{(W_{jt} - W_{jt-1})}{\sum_{1}^{n} (W_{it} - W_{it-1})} \quad \ldots\ldots (12)$$

where F_j is the fraction of current production channelled into the jth organ. Clearly, then, the actual weight increase of the jth organ will be $F_j . DM$, and so its new weight becomes

$$W_{jt} = W_{jt-1} + F_j . DM \quad \ldots\ldots (13)$$

In many cases the most important aspect of dry matter distribution is the extent to which it is affected by variations in the environment, both natural and man-induced. In a grazing model, for example, it will be necessary to relate the fraction of net production channelled into leaf growth to the amount of leaf removed through grazing, and to the general run-down of the plant through trampling, fouling and the like. To model such effects we can define a variable E which lies in the range 0 to 1, and expresses the degree to which the growth (and hence the sink strength) of an organ is reduced at a particular time. When E = 0 the organ does not grow, and growth is unaffected when E = 1. We then calculate a value for E in relation to the relative sub-optimality of all those environmental factors tending to alter the pattern of plant growth. Rewriting equation (10) to give the potential dry matter increment, A, we have

$$A = W_{t-1}\left[e^{r.\left(\frac{K - W}{K}\right)_{t-1}} - 1\right] \quad \ldots\ldots (14)$$

to which we can add the E term by assuming that it effects a reduction in the exponent term in the same overall way as the proportional departure from maximal weight (K-W)/K. Now we can calculate the potential dry matter increment in a sub-optimal environment as

$$A = W_{t-1}\left[e^{r.E.\left(\frac{K - W}{K}_{t-1}\right)} - 1\right] \quad \ldots\ (15)$$

and the fraction of dry matter partitioned into the growth of the jth organ will be

$$F_j = \frac{A_j}{\sum_{1}^{n} A_i} \quad \ldots\ (16)$$

The new weight of the organ can then be calculated from equation (13) as before. A family of curves showing the growth of an organ with different values of E is shown in Fig. 43.

THE MODEL

The precise problem to be tackled here is to determine the effects of the timing and intensity of an aphid infestation on the yield of beans from Vicia faba. As in most plant growth models this involves a consideration of two sets of processes; the production of dry matter and the distribution of a fraction of this production into seed. Both will be affected to a greater or lesser extent by aphid infestation. Fig. 44 plots the variables which have a significant effect upon bean yield under aphid infestation in flow-diagram form. This constitutes the structural aspect of the model, and embodies those biological properties of the plant which are

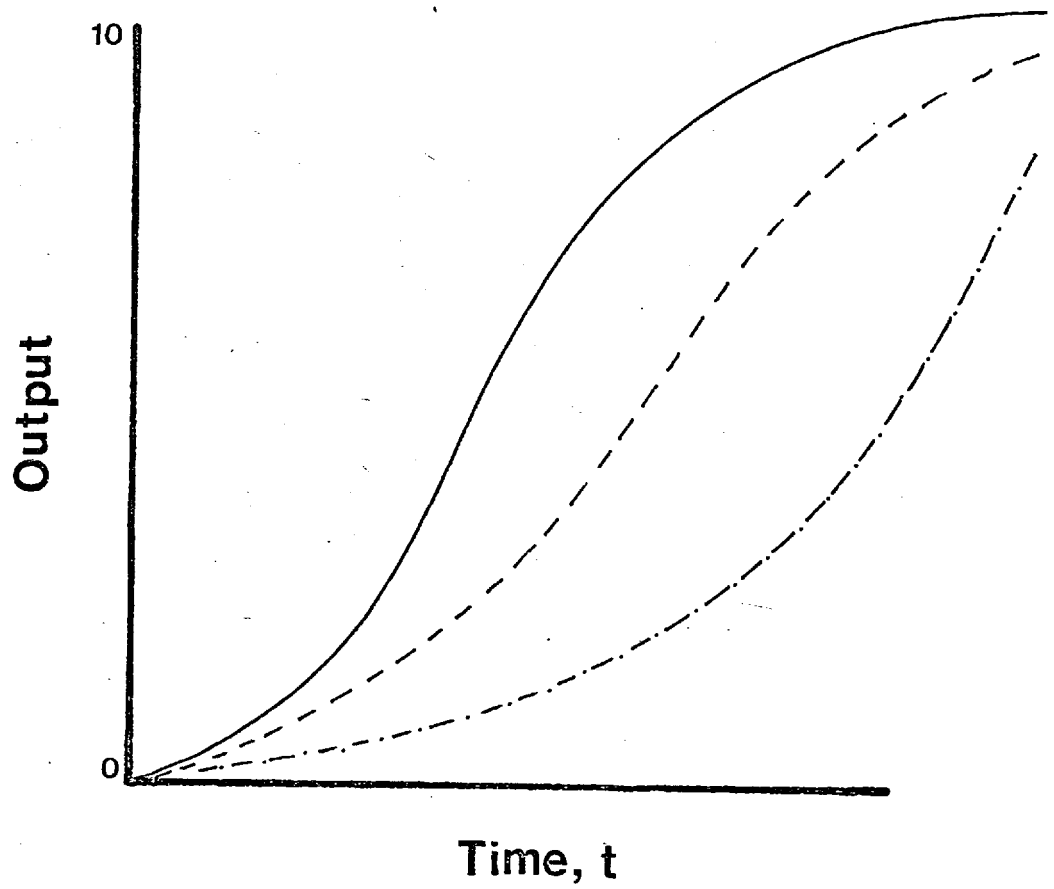


Figure 43. Logistic, or sigmoid growth, assuming that E in Equ. 15 has constant value 1 (—), decreases (- -), or increases linearly with time, t (-·-)

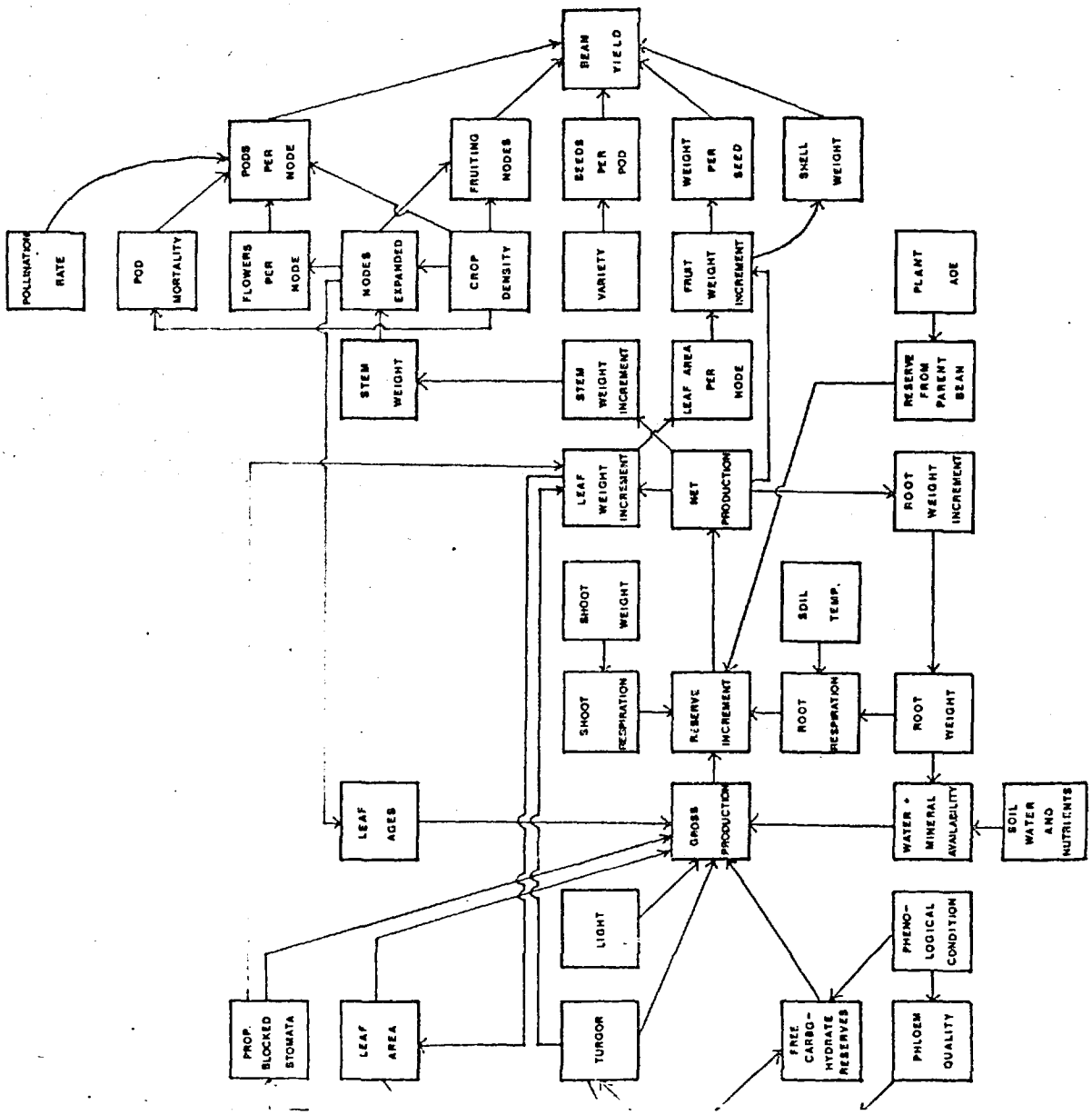


Figure 44. The flow diagram of the variables and processes involved in the plant growth model

considered to be relevant to the question in hand.

For the purposes of this model the plant is envisioned as consisting of a root system, and of a shoot which is divided into a number of nodes. In Vicia faba one leaf is produced at each node in an opposite pattern, and the stem is usually unbranched. Older plants occasionally produce branches at the first and second nodes where leaves are absent. Each node consists of a length of stem (the internode), a stipule, a petiole arising at the junction of the stipule and the stem, a number of leaflets attached to the petiole, and at the higher nodes of older plants a cluster of flowers or pods. The plant parts, and their relative dispositions are shown in Fig. 45.

New nodes arise in the apical meristem by the production of leaf primordia, but are not considered as contributing to the physiological behaviour of the plant until they reach a certain threshold size. Up to this time all the leaf primordia, young leaves and the meristem itself are considered as a single functional unit, the terminal cluster.

Each of the tissues within a node consists of three types of carbohydrate. There are structural carbohydrates like cellulose and hemicellulose which we can consider as fixed pools of reserve. In addition, there are water insoluble carbohydrates such as starch which form reserve pools from which material can be mobilized under certain conditions. Finally, there are water soluble carbohydrates represented by the sugars, and by sucrose in particular, forming a mobile reserve pool within each tissue. Clearly, each type of tissue will contain these carbohydrates in different proportions; a stem will contain proportionately more cellulose than a leaf, and a fruit will often contain more starch than a stem. The

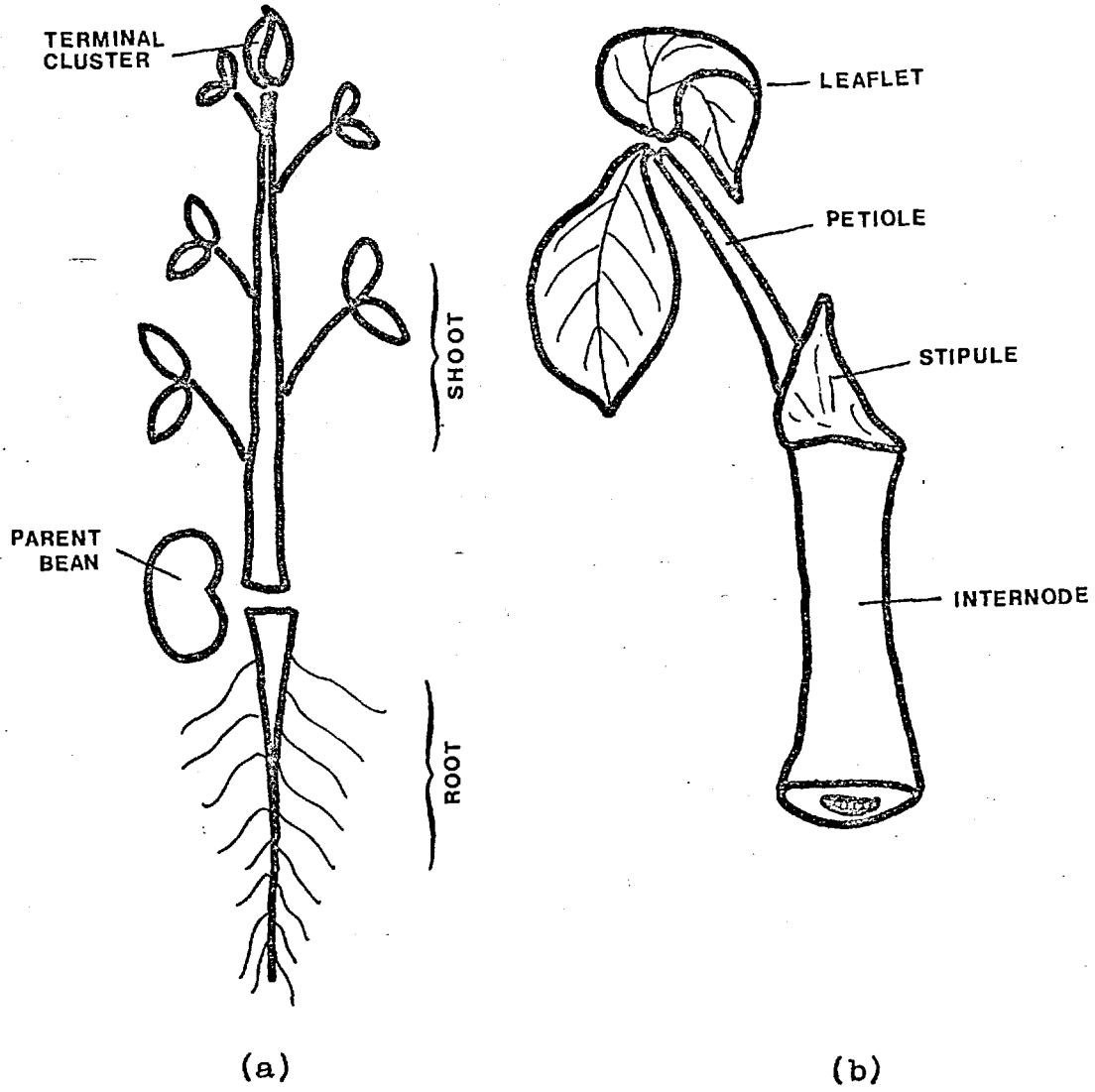


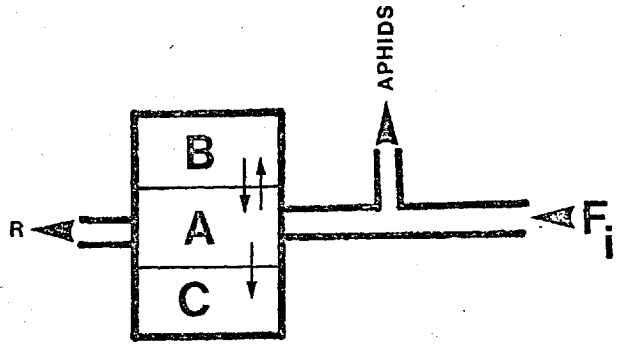
Figure 45. (a) Diagrammatic representation of a plant of Vicia faba as envisioned by the model. This plant has five nodes expanded which, including the two leafless nodes, means that NODES = 7

(b) A single node expanded to show its component parts. If and when flowers are produced, they appear in the axil of the leaves, at the junction of the petiole and internode

question to be asked is whether it is necessary in the context of simulating the effects of aphid feeding to consider the dynamics of the three carbohydrate types within each organ.

Aphid feeding acts to reduce the amount of soluble carbohydrate in the phloem vessels (Kennedy and Mittler, 1953). The simplest assumption is that the aphids do not affect the partitioning of reserves between the three carbohydrate types, and that the removal of a certain amount of sucrose from the phloem will reduce the rate of increase of each type equally. A model built on this assumption would only have to consider a single measure for the total amount of carbohydrate in each organ. Typically, this would be its dry weight.

The alternative treatment would consider aphid feeding as affecting only the amount of soluble carbohydrate in each tissue (Fig. 46). A source leaf would therefore export less material, but sink organs such as young leaves and all other plant parts requiring more assimilate than they produce (stems, roots, petioles and fruits), would have to be considered differently. In these cases, the pool of mobile reserves is reduced, but the model would have to consider the possibility of mobilization of stored carbohydrates in compensation. These might come from the organ itself, or be translocated from other parts of the plant. Similarly, the rate of incorporation of mobile reserves into new structural material would have to be considered in relation to the relative sizes of the three pools within the tissue. While it would be possible to model this process by calculating net flow rates between the three compartments, there are no data available, and it seems reasonable to build a model assuming a constant partitioning in the first instance, and only move to the more detailed



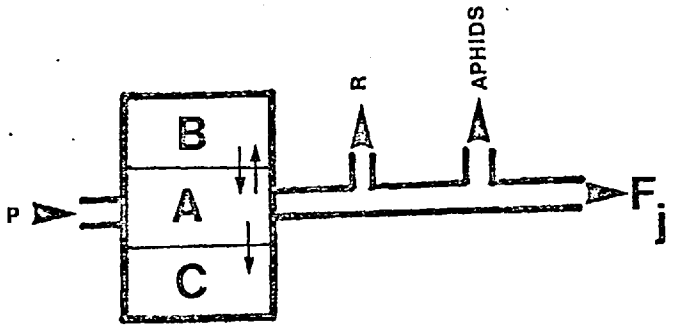
SINK

A SOLUBLE CARBOHYDRATES
B INSOLUBLE STORAGE CARBOHYDRATES
C STRUCTURAL CARBOHYDRATES

F_i RESERVE INFLOW

F_j RESERVE EXPORT

R RESPIRATION
P PHOTOSYNTHESIS



SOURCE

Figure 46. The pattern of reserve movement in source and sink tissues. Aphid feeding probably affects the internal carbohydrate balance of source leaves relatively little

consideration if the model behaves unrealistically (Chapter VI). Consequently no allowance has been made in the present model for the mobilization of reserves in compensation for high rates of carbohydrate removal by the aphids.

Consider the plant as being divided into above and below ground parts, and let these be called SHOOT and ROOT respecively. The ROOT can be considered as a single unit of biomass, while the shoot consists of a number of NODES at any time. Each node of the plant includes a length of internode, a petiole, a leaf, and a number of flowers or pods. Let WTS(N), WTP(N), WTL(N) and WTF(N) be the dry weights of these organs in the Nth node. Finally, let the weight of the terminal cluster be TCWT grams. Therefore, at any given time, the shoot weight is given by

$$SHOOT = \sum_{1}^{NODES} (WTL(N) + WTS(N) + WTP(N) + WTF(N)) + TCWT$$

and the whole plant weight by

$$PLANT = SHOOT + ROOT$$

1 Germination and Establishment

Let the weight of the planted seed be PBWT, and the dry weight of reserves mobilized from it on any one day be PBRES. Now let the rate at which these reserves become available be a function of soil temperature (Smoliak and Johnston, 1968), which is to say

$$PBRES = f(SOILTMP)$$

then, at the end of the day, the seed weight will be

PBWT = PBWT - PBRES

Plant weight is therefore incremented by PBRES daily (ignoring respiration at this stage), and if OPTRPR(1) is the fraction of dry matter channelled into root growth in a plant of 1 node in size, we can increment root weight by

ROOT = ROOT + OPTRPR(1) * PBRES .... (17)

and the shoot weight by

SHOOT = SHOOT + (1. - OPTRPR(1)) * PBRES . (18)

When the shoot has increased in size to a specified threshold weight, an initial distribution of stem tissues is set up; the stem is divided into two nodes (leafless), and a terminal cluster containing the shoot apex and the leaf primordia. At this stage, the weight of the whole plant is

PLANT = ROOT + WTS(1) + WTS(2) + TCWT .... (19)

The terminal cluster then grows (as described later) until the first leaf is sufficiently large as to be considered separately. There are three nodes at this stage, and, in addition to the reserves from the seed, photosynthate becomes available for growth and maintenance. When the seed has reached a specified low weight (representing the weight of the seed-coat plus any non-mobilizable material in the cotyledons), or such time elapses as would account for the rotting of the seed, then these reserves are permanently cut off, and the plant continues to function solely from its photosynthates.

2 Photosynthesis and Respiration

Assume that photosynthesis only occurs to a significant

extent in leaves, and that the amount of photosynthesis is an increasing function of leaf area. It is quite straightforward to include stem photosynthesis in the model should it be required. The basic model is therefore

$$PHOTO = f(LA) \quad \ldots\ldots (20)$$

where PHOTO is the gross production in grams of carbohydrate, and LA is the area of green leaf. We can consider two sections to this equation; a section dealing with the rate of photosynthesis per unit leaf area, and another concerned with the effects of canopy structure on the environmental conditions experienced by different leaves in the crop.

To achieve this we divide the canopy into strata, in this case defining each node of the plant as a discrete layer. Now assume that a leaf of Vicia faba has a maximum potential rate of photosynthesis which we can call PEFFIC (photosynthetic efficiency), expressing the rate of accumulation of carbohydrate by one sq. cm. of leaf in completely optimal conditions. The actual rate of photosynthesis will therefore be some fraction, usually less than 1.0, of this maximum rate. The first section of the photosynthesis model must determine the value of this fraction in terms of the current levels of the factors which affect it.

The primary factors affecting the rate of photosynthesis are light intensity, air temperature, and CO_2 concentration (see Gaastra, 1963). For the purposes of this model it is possible to assume that CO_2 concentration is constant from day to day, although it may well vary within a day (Monteith et al., 1964). We then provide data sets describing the effects of air temperature and light intensity on the fraction

of PEFFIC which is achieved under controlled conditions; these might be of the form shown in Figs. 47 and 48. The actual contribution of each factor to the actual photosynthetic rate is then determined by interpolation (Chapter I), writing

$$RT = F(TEMP)$$

and

$$RL = F(LIGHT)$$

where F is the routine of interpolation, and RT and RL are the fractions of PEFFIC which would be achieved if all other factors were optimal. From Fig. 40, above, it is clear that light and temperature interact with one another, so we can write RTL, the fraction at a given light and temperature, as

$$RTL = RT * RL$$

This means that if LIGHT was such that RL was 95% maximal, and TEMP was too low, so that only 75% of the maximal rate of photosynthesis was possible, we should observe

$$RTL = .75 * .95 = 0.7125$$

or actual photosynthesis of 71.25% PEFFIC.

Other factors affect the realized fraction of PEFFIC under most circumstances. We know that the age of the leaf tissue affects its rate of photosynthesis (e.g. Treharne et al., 1968), so we can draw a graph of the relative rate of photosynthesis against leaf age (Fig. 49). The maximum rate at a given leaf age, LFAGE, will then be

$$RA = F(LFAGE)$$

The effects of accumulated leaf reserves (Nealls and Incoll, 1968), and the effects of leaf water content (Catsky, 1965; Troughton and Slatyer, 1969) can be calculated in a similar

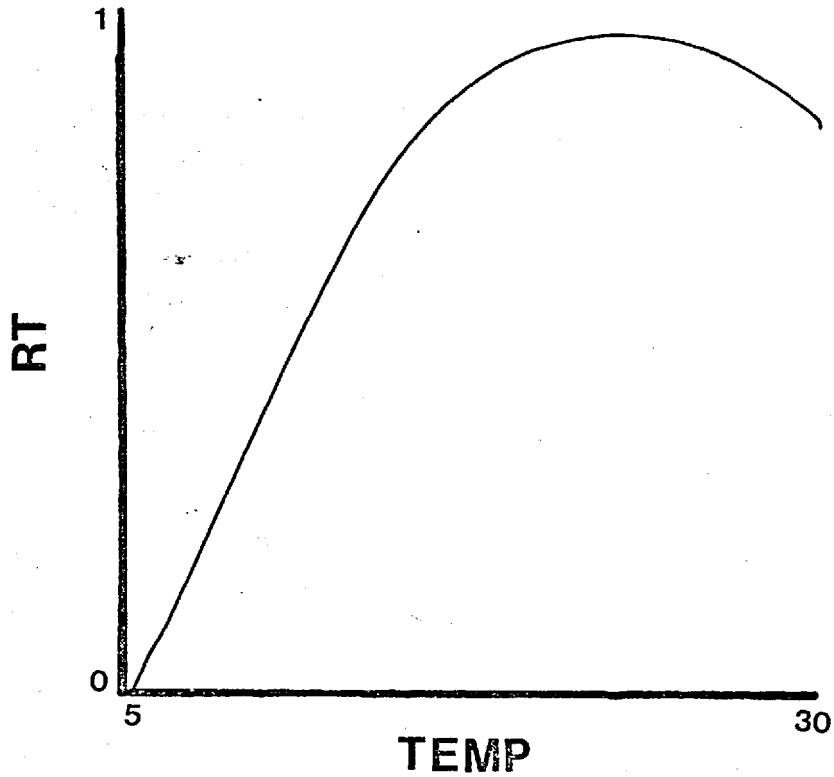


Figure 47. The effects of air temperature (TEMP) on relative photosynthetic rate, RT

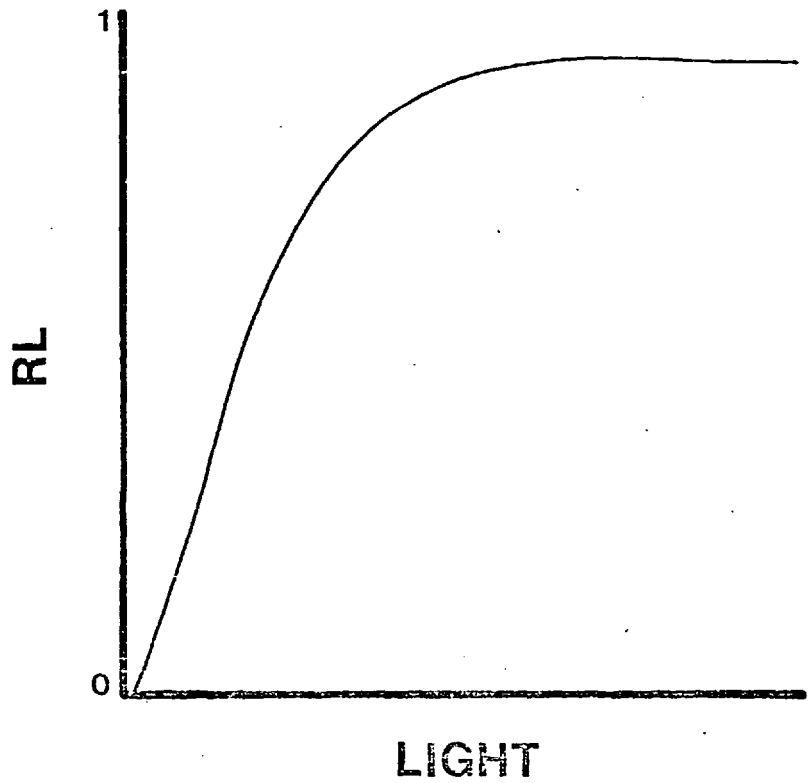


Figure 48. The effects of light intensity on the relative photosynthetic rate, RL

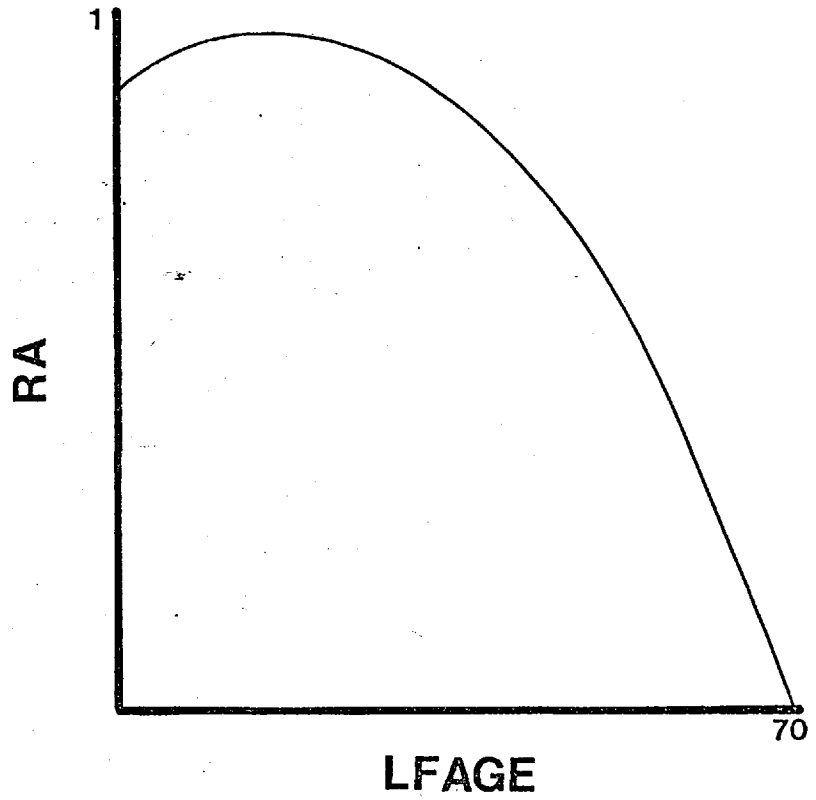


Figure 49. The effects of leaf age (LFAGE) on relative photosynthetic rate, RA

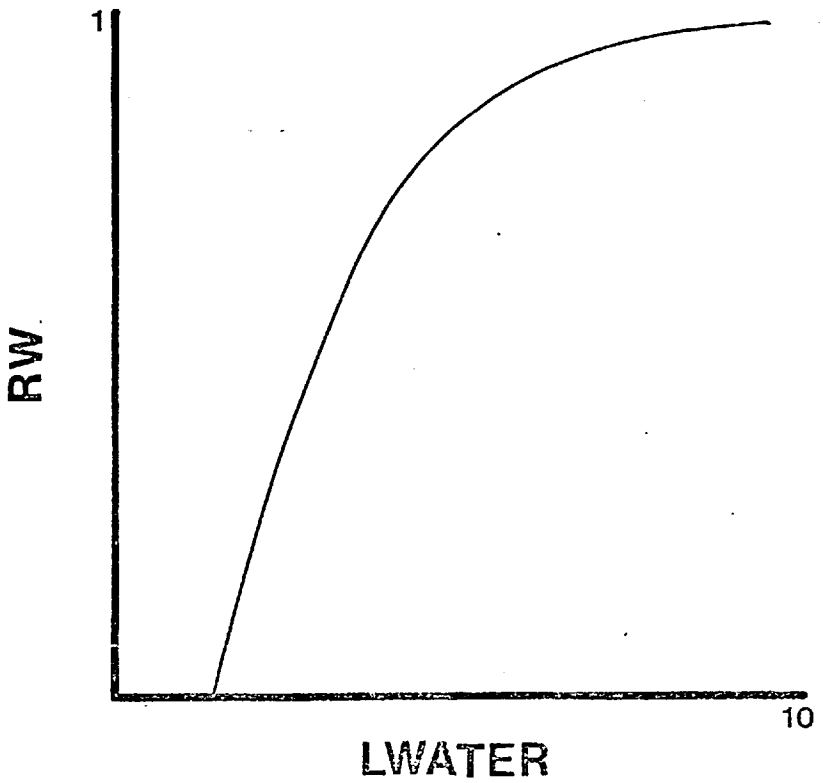


Figure 50. The effects of leaf water content (LWATER) on relative photosynthetic rate, RW

fashion to give

$$RR = F(CHO)$$

$$RW = F(LWATER)$$

where CHO is the carbohydrate level in the leaf and LWATER is the leaf water content (Fig. 50). The problem is then to calculate the actual fraction of PEFFIC which is realized under a particular set of values for each factor. We could (see Chapter I) either model the process by assuming that the lowest of the fractions limited the rate of photosynthesis (Liebig's law); i.e.

$$RATE = \min(RA, RR, RW, \text{and so on}) \quad .... (22)$$

or we could suggest that like light and temperature they interact with one another; i.e.

$$RATE = RA * RR * RW \quad .... (23)$$

In the absence of data from factorial experiments, it is not possible to determine which approach is the most realistic. For this model I have assumed that the effects are multiplicative, and so, for all factors we can write the actual rate as

$$AEFFIC = PEFFIC * RT * RL * RA * RR * RW \quad .. (24)$$

The factors have no depressive effects on photosynthesis when their levels are optimal since, by definition,

$$1 = F(\text{optimal value})$$

The second aspect of the photosynthesis model deal with the conditions prevanlent at each node of the plant. We know that light intensity decreases through the canopy because the upper leaves shade the lower. To model this we write I_i,

the light intensity in the ith layer of the canopy, as

$$I_i = f\left(\sum LA\right) . I_o \qquad \ldots\ldots (25)$$

where $\sum LA$ is the accumulated leaf area above the ith node, and I_o is the incident radiation. It is commonly assumed that this decay in light intensity is exponential (e.g. Monsi and Saeki, 1953), but we can supply the model with graphical data from field measurements without specifying the precise mathematical form (Fig.51). The actual light is then given by interpolation;

$$\text{LIGHT} = F\ (\text{ALA}) * \text{IO} \qquad \ldots\ldots (26)$$

ALA being the accumulated leaf area, and IO the incident light.

If we know the weight of leaves at each node of the plant to be WTL(N), and the age of these leaves to be LFAGE(N), we can determine the leaf area at each node by dividing the leaf weight by the leaf density (which is a function of leaf age). That is, setting K = LFAGE(N),

$$\text{AREAL(N)} = \text{WTL(N)} \ / \ \text{WTSQCM(K)} \qquad \ldots\ldots (27)$$

where WTSQCM(K) is the density in gms cm^{-2} of a k-day-old leaf. Summing leaf area from the ith node to the top of the plant gives ALA;

$$\text{ALA} = \sum_{N=i}^{\text{NODES}} \text{AREAL(N)} \qquad \ldots\ldots (28)$$

and the actual light conditions are calculated from equation (26). Actual gross production at each node is therefore

$$\text{GPROD(N)} = \text{AREAL(N)} * \text{LIGHT} * \text{AEFFIC} \qquad \ldots\ldots (29)$$

where AREAL(N) is known, and AEFFIC is obtained from equation (24). Summing over all nodes gives the total gross photosynthesis, PHOTO, as

$$PHOTO = \sum_{N=3}^{NODES} GPROD(N) \quad \ldots\ (30)$$

Finally, we can allow that the rate of gross production is affected by the availability of soil water and nutrients. There are two ways in which such a shortage might occur;
a) the situation in which there is too little root biomass to take up enough water or nutrients to make good the demands of the shoot system, even at maximal uptake rates:
b) those cases where the density of water or nutrient ions in the effective vicinity of the root system is so low that the demand can not be fulfilled at the necessary rate.

Let the fraction of the shoot's demand which a given root biomass can fulfil be UPFRAC, and the optimal proportion of roots for a plant of size NODES be OPTRPR(NODES). If there are sufficient roots, that is if

$$OPTRPR(NODES) \leq ROOT / PLANT$$

then UPFRAC = 1. Otherwise, let us define a new term SRAT, being the optimal ratio of shoot to root; this is given by

$$SRAT = [1 / OPTRPR(NODES)] - 1 \quad \ldots\ (31)$$

since OPTRPR(NODES) = ROOT/(ROOT+SHOOT) in optimal conditions. Then if the actual shoot/root ratio is more than SRAT

$$SHOOT / ROOT > SRAT$$

there is too little root to supply the shoot. We then make UPFRAC some decreasing function of the difference between

SHOOT/ROOT and SRAT. A simple linear model of this is given by

$$UPFRAC = 1. - ((SHOOT/SRAT)-ROOT)/(SHOOT/SRAT) \quad (32)$$

and the relative root shortage can be computed as a function of this value of UPFRAC

$$ROOTF = F(UPFRAC)$$

Now if the relative availability of water and nutrients in the vicinity of the root system is SOILF, and the ammount available is SUPPLY, we can determine whether gross production will be limited by these factors. The amount of water and nutrients removed from the soil on a given day will be a function of the root biomass and ROOTF; for example

$$UP = ROOT * ROOTF \quad .... (33)$$

then if SUPPLY < UP there is too little water and nutrient to fulfil demand. SOILF will be a decreasing function of the difference between UP and SUPPLY which can be modelled by

$$SOILF = 1. - ((UP-SUPPLY) / UP) \quad .... (34)$$

The revised level of gross production is computed by multiplying the shoot potential PHOTO by these two fractions;

$$PHOTO = PHOTO * ROOTF * SOILF \quad .... (35)$$

Respiration, as indicated earlier, can be considered as comprising a maintenance and a photorespiratory component. The general model can be written as equation (7) which, in our model symbolism, is

$$TRESP = F(PHOTO)*F(TEMP) + F(PLANT)*F(TEMP) \quad . (36)$$

We must supply four data sets, then, to allow interpolation of the actual dry weight loss through respiration. The shapes of the functions for PHOTO and PLANT are shown in Figs. 52 and 53; these are intuitive, based on the results of McCree and Troughton (1966). As with photosynthesis, it is simplest to specify the actual respiration rate at a given temperature as some fraction of its maximum rate. To do this the temperature functions are plotted on a relative scale, assuming a doubling in respiration for each 10°C rise in temperature. The weight loss data in Figs 52 and 53 are expressed at 20°C and multiplied by the temperature fraction (which in this case can exceed unity) to give the actual respiratory loss. Net production is the calculated by subtracting the total respiration TRESP from gross production; i.e.

$$\text{PRODNET} = \text{PHOTO} - \text{TRESP} \qquad \ldots\ (37)$$

3 Dry Matter Distribution

a) Root Growth

Assume that at a given height, NODES, and phenological condition, COND, the plant has an optimal root proportion OPTRPR. Dry matter is distributed between root and shoot such that OPTRPR is approached; if the root is relatively light, then proportionately more of the net production is channelled into root growth, and if the root is too heavy, more reserves are allocated to the shoot.

After the net production has been distributed, the new plant weight will be

$$\text{PLANT} = \text{PLANT} + \text{PRODNET}$$

and from this, the optimal root weight will be

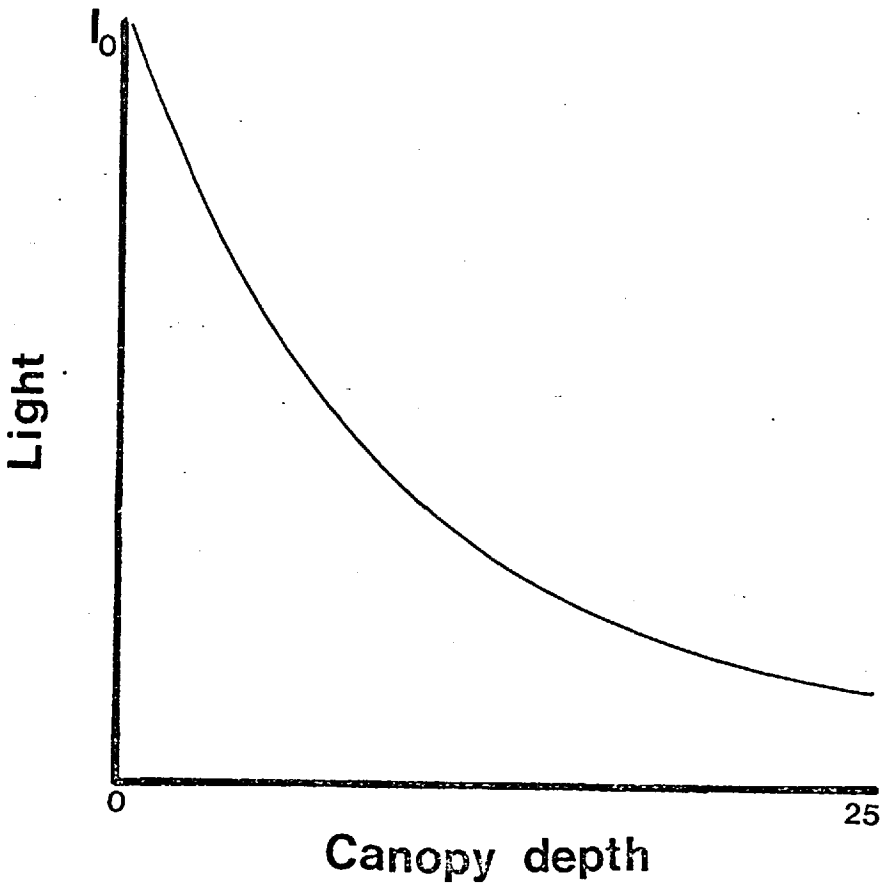


Figure 51. The relation between canopy depth (in nodes) and light intensity. In the model light intensity is plotted against accumulated leaf area above a point, to allow for variation in the leaf area for a given height of the whole plant

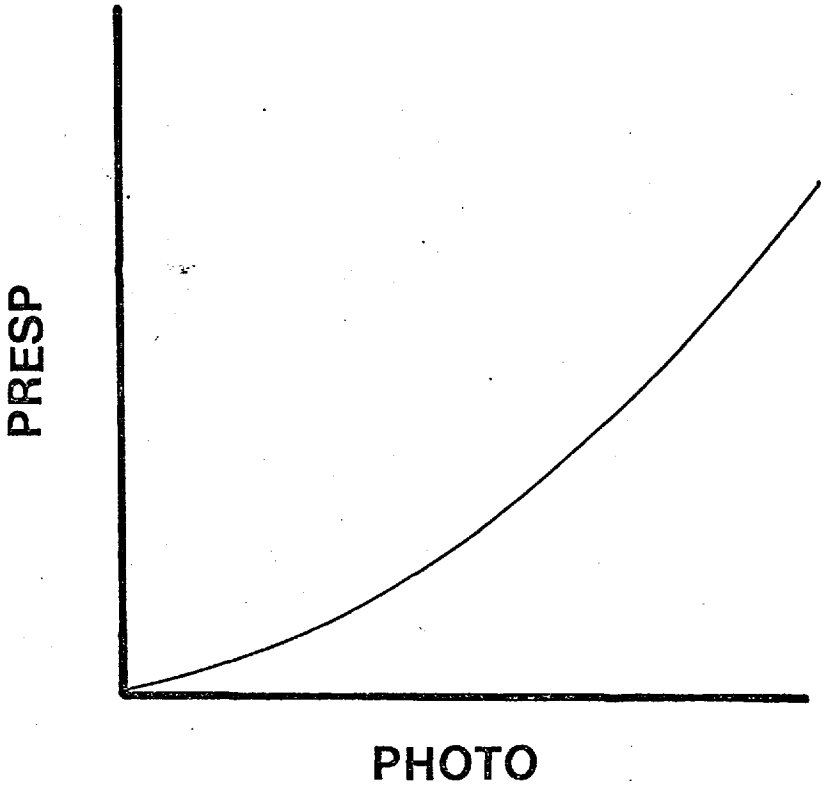


Figure 52. The relationship between photosynthetic rate (PHOTO) and photorespiration (PRESP)

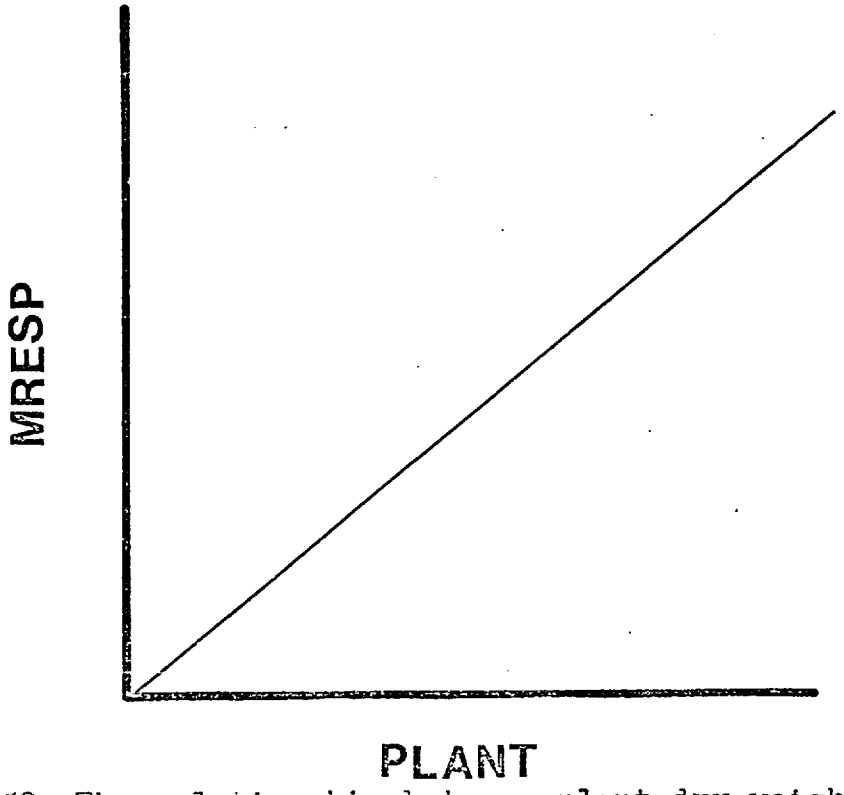


Figure 53. The relationship between plant dry weight (PLANT) and maintenance respiration.

$$OPTRUT = PLANT * OPTRPR(NODES) \quad .... (38)$$

If the current root weight is less than OPTRUT, root growth occurs as follows;

$$PROD11 = PRODNET - (OPTRUT - ROOT) \quad .... (39)$$

so PROD11 is the dry matter incrmement remaining after root growth has occurred. Now if PROD11 > 0. then we set

$$ROOT = OPTRUT$$

and $$SHOOTIC = PROD11$$

where SHOOTIC is the shoot dry weight increment.

If, on the other hand, the root is so light that all today's production can not bring it back to OPTRUT, then

$$PROD11 \leq 0.$$

and $$ROOT = ROOT + PRODNET$$

so $$SHOOTIC = 0.$$

Again, if the root is already large enough and

$$ROOT \geq OPTRUT$$

then $$SHOOTIC = PRODNET$$

and all net production is channelled into shoot growth.

b) Shoot Growth

The shoot is made up of a number of tissue types distributed vertically through a number of nodes. Root gowth was simulated simply as an increment to a single below-ground biomass; for the shoot, on the other hand, the distribution of the dry weight increment SHOOTIC is more complex. It must be partitioned between nodes, and between the tissues within

a node.

To compute these distribution fractions, assume that each tissue (stem, lamina, petiole, flowers anf fruit) has a maximum obtainable dry weight at any given node, and that it grows towards this weight according to an equation of the form (explained in Distribution of Dry Matter, page 138)

$$W_{t+1} = W_t \cdot e^{(GR.\ E.\ (K-W)/K)} \quad \ldots\ (40)$$

where K is the maximum attainable weight, GR is the growth rate describing the rate of approach of W to K, and E is a variable whose value lies in the range 0 to 1 constituting an integration of the various deleterious environmental effects tending to reduce the growth rate. If aphid feeding affects not only the amount of dry matter produced, but also the pattern in which the remainder is distributed, then E would contain an estimate of the relative feeding intensity at each node.

Next, let the potential dry matter increment represent the strength of the sink created by the growth of a tissue at this node; that is, for leaves,

```
SINKL(NODE) = WTL(NODE) *
    [EXP((K - WTL(NODE))/K * GR * E) - 1.]      (41)
```

By summing the leaf sinks for all parts of the plant we obtain the total leaf sink, TSINKL

$$TSINKL = \sum_{1}^{NODES} SINKL(NODE)$$

This procedure, when repeated for each tissue type, gives a value for the total shoot sink strength of

SIGSINK = TSINKL+TSINKP+TSINKS+TSINKF+TCSINK

TCSINK, the sink created by the growth of the terminal cluster, is calculated in the next section.

The dry weight increment of any tissue type in the shoot is therefore (again using leaves as an example)

LEAFINC = SHOOTIC * TSINKL / SIGSINK .. (42)

and, to determine the growth of lamina at any given node we have

WTINCL(NODE) = LEAFINC * SINKL(NODE)/TSINKL

The weight of the organ is then updated

WTL(NODE) = WTL(NODE) + WTINCL(NODE) .... (43)

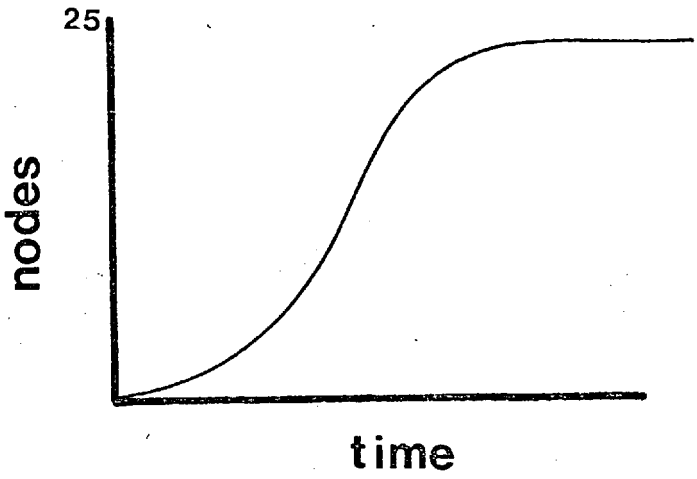
and the new leaf area will be

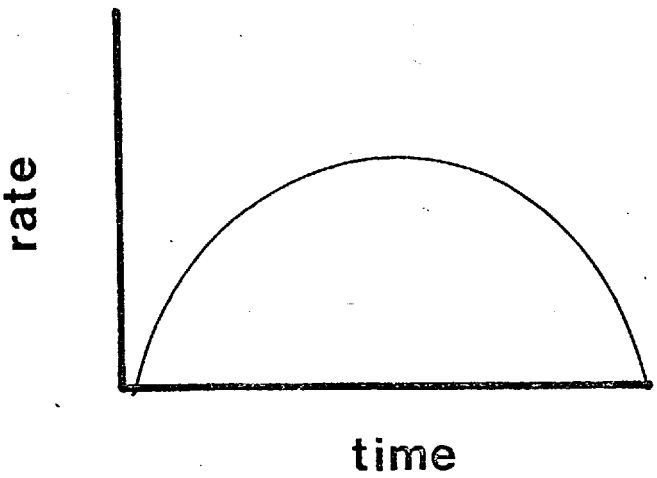
AREAL(NODE) = WTL(NODE) / WTSQCM(K) .... (44)

wnere K = LFAGE(NODE), the age of the leaf in days.

c) Height Growth and Leaf Production

The pattern of appearance of new leaves in a single stemmed plant like Vicia faba is generally a sigmoidal function with time, as shown in Fig. 54 from Chapter II and Ishag (1969). The rate of leaf production is therefore an N-shaped curve approaching zero as the plant approaches its maximum node number (Fig. 54). To model this, assume that the terminal cluster has a maximum weight at which a leaf is on the threshold of becoming photosynthetically independent, and a minimum weight representing the dry weight of the stem apex and the smaller leaf primordia. Reference to Fig. 55 (from





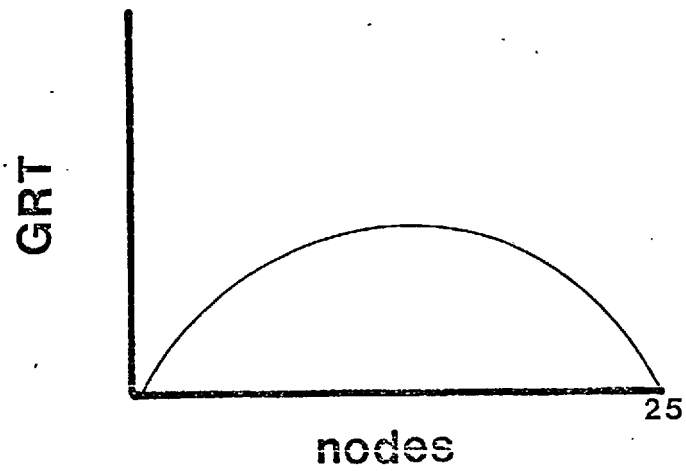


Figure 54. The observed patterns in the number of nodes (top) and rate of node production (centre). The lower relationship is used in the model to mimic these effects without relying on a time-based curve

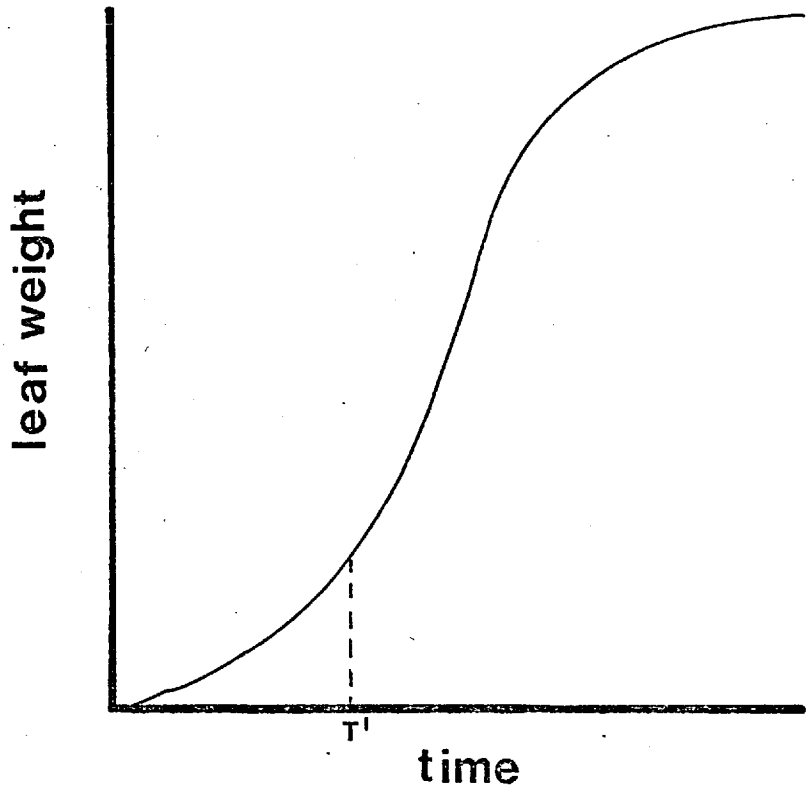


Figure 55. Generalized growth curve for a leaf. Up to the point T' growth can be assumed to follow an exponential curve

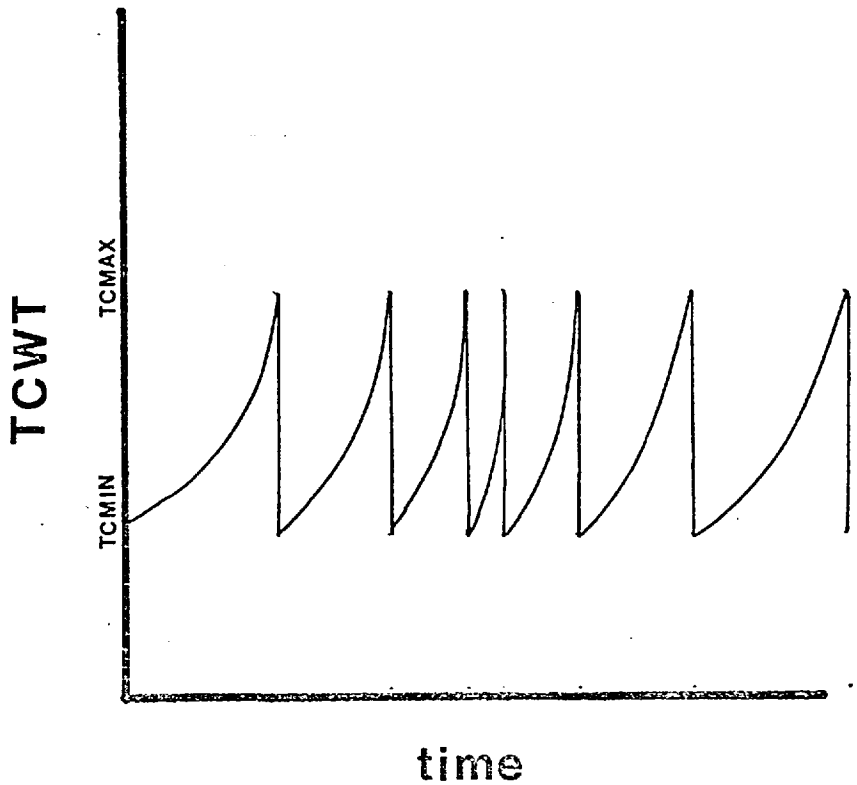


Figure 56. The pattern of dry weight change in the terminal cluster as new nodes are produced (see text)

Ishag, 1969) shows that the weight increase of a single leaf can be approximated by a sigmoid curve, and that up to a threshold size T, the pattern of growth is more or less exponential. So let the strength of the terminal cluster sink increase exponentially with time

$$TCNEW = TCWT * EXP(GRT(NODES) * E) \quad .... (45)$$
$$TCSINK = TCNEW - TCWT$$

where GRT(NODES) is the potential rate of growth, and E is the integral of the environmental factors tending to reduce this growth. Unlike the growth rates of the other stem organs, that of the terminal cluster varies in an N-shaped fashion with the size of the plant (Fig. 54). From this assumption, the time taken for the terminal cluster weight to increase from minimum to maximum changes as the inverse to the curve; i.e. in a U-shaped fashion. A graph of TCWT against time is therefore of the form shown in Fig. 56.

Given the relative sink strength of the growing apex (TCSINK) we calculate the biomass increase as

$$TCINC = SHOOTIC * TCSINK / SIGSINK \quad .... (46)$$

and the new weight is therefore

$$TCWT = TCWT + TCINC$$

Now if TCWT $\geq$ TCMAX a new leaf will be produced, and NODES = NODES + 1. The weight of the new node is

$$WTNEW = TCWT - TCMIN$$

and the terminal cluster returns to its basal weight,

$$TCWT = TCMIN$$

Depending upon the current height (NODES) of the plant, and its phenological condition (COND), the biomass of the new node is distributed between the four tissue types;

```
WTL(NODES) = WTNEW * FRACTL(COND, NODES)
WTP(NODES) = WTNEW * FRACTP(COND, NODES)
```

and so on, for the other tissues.

The age of the new leaf is assigned a starting value of one day which will be incremented every day of its life;

```
LFAGE(NODES) = 1
```

Leaf age is included in the model as affecting photosynthesis (equation 23), leaf density (equ. 27), and leaf water content (next section). On reaching the maximum age for which a leaf at a given node can actively photosynthesize, the tissue is assumed to die and no longer to contribute any photosynthate to the pool of gross production. We write

```
IF (LFAGE(NODE).GT.MAXAGE(NODE)) LIVE(NODE) = 0
```

where LIVE(NODE) has a value of 1 for living tissues. Then a leaf only respires or photosynthesizes if LIVE(NODE) = 1; similarly, leaves which are senescent do not give rise to active sinks, so that

```
IF (LIVE(NODE).EQ.0)  SINKL(NODE) = 0
```

The weight of dead leaves held on the plant is assumed to decrease exponentially according to the equation (computed daily)

```
IF (LIVE(NODE).EQ.0) WTL(NODE)=WTL(NODE)*DECAY
```

where DECAY is a constant describing the rate at which material

is leached or withdrawn actively from the leaf.

This treatment of height growth allows that the observed pattern of leaf production will be observed under control conditions, and also that the rate of appearance can be affected by aphid feeding (as observed in Chapter II). The aphids act to slow the production of new nodes by reducing the amount of reserve available (PRODNET), and also by damaging the tissues of the terminal cluster, thereby reducing E and hence the relative competitiveness of the terminal cluster sink. So both the amount of carbohydrate available, and the fraction of the pool incorporated is dependent upon aphid feeding.

4 Leaf Water Content

Leaf water content plays two crucial roles in the model. First, it affects the rate of photosynthesis in the leaf; the mechanism is not specified, but reductions in water content are taken as being indicative of a general lowering in photosynthetic potential (Troughton and Slatyer, 1969; Jones, 1973). Second, leaf water content affects the rate at which the aphids feed, and the desirability of a given leaf for aphid colonization.(Wearing, 1966, 1972).

In turn, leaf water content is decreased by aphid feeding when the rate of water removal exceeds the rate at which the leaf can compensate by increased transpiration, and by low soil water availability.

Let the weight of water held per gram of leaf dry weight at full turgor, vary with the age of the leaf (Chapter II, Fig. 22). This curve represents the optimum leaf water content for leaves of any age; departures from this optimal will affect the rate of photosynthesis and the rate of aphid feeding. For any leaf, then, we can calculate the optimum weight of

water per gram dry weight by interpolation from Fig. 22;

OPTWAT = F (LFAGE(NODE))

and we can define a proportional water deficit DIFWAT as being

DIFWAT = LWATER(NODE) / OPTWAT

where LWATER(NODE) is the current water content. To account for daily variations in this parameter, water losses must be balanced against the current ability of the plant to compensate by enhanced uptake. Assume that the value SOILF (above) represents the extent to which water can be taken up by the root system under a given set of atmospheric conditions. Now, allowing that the amount of water removed from a leaf during feeding is a function of the amount of food taken by the aphids at a node, we can put

APHWAT = F (APHFEED(NODE))

This feeding accounts for a proportion of the water content of the leaf at any instant, and although compensation by the plant to water loss is a continuous process, we can approximate by stating that the overall reduction in the course of a day will be related to the ratio of the total water removed with the food per gram of water in the tissue at equilibrium. The maximum weight of water available in the leaf is given by

WATER = LWATER(NODE) * WTL(NODE)

The actual water holding potential, however, is a function of the balance between soil and atmospheric conditions, and the stature and uptake potential of the plant. We can write, therefore,

ACTWAT = F (SOILF) * WATER

where the fraction SOILF lies in the range 0 to 1, and the function is of the general form shown in Fig. 57. Here, a threshold of dehydrating factors has been assumed, above which ACTWAT = WATER. The relative intensity of water removal is then

WATRAT = APHWAT / ACTWAT

and the effects of this intensity on the new value of LWATER(NODE) will be

APHWF = F (WATRAT) $\qquad 0 \leq APHWF \leq 1$

If SOILF is above the threshold shown in Fig. 57 we can allow the tissue to increase in water content as long as LWATER is less than or equal to OPTWAT (i.e. DIFWAT $<$ 1). The extent of this compensation is related to DIFWAT by

COMP = F (DIFWAT)

and the function is graphed in Fig. 58. The new values of leaf water content is then calculated;

LWATER(NODE) = LWATER(NODE) * APHWF * COMP

5 Phenological Condition

Four phenological conditions of the plant are recognised in the current model, though any number could be incorporated quite readily. These conditions are germinating, vegetative, early reproductive and late reproductive. In each, the pattern of dry matter distribution is different. The germinating plant develops an anchoring and absorbing root with which to

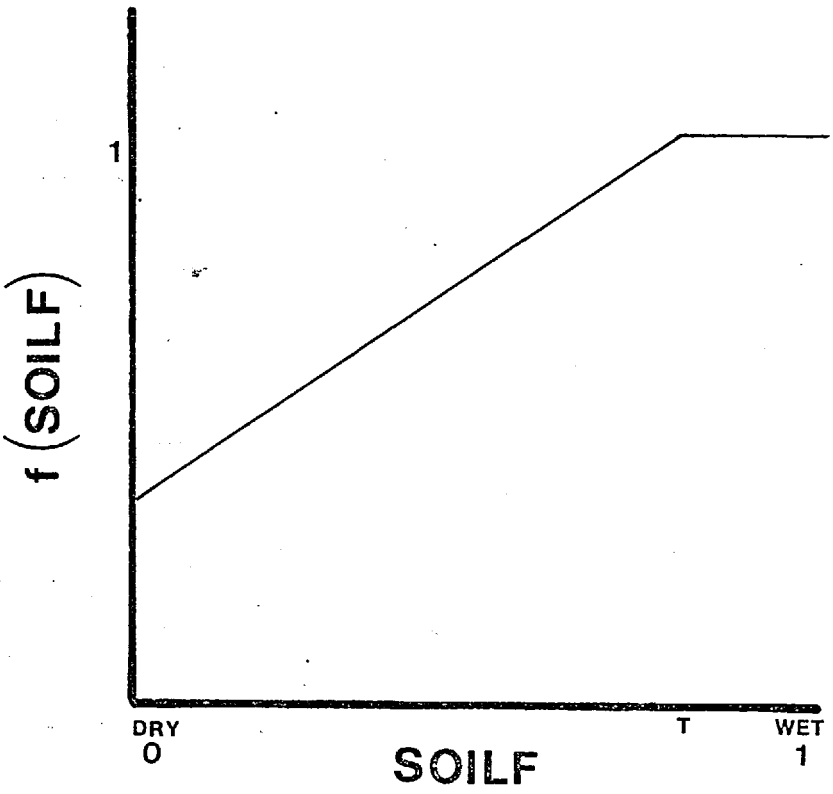


Figure 57. The relationship between soil water availability (SOILF) and the relative ability of the plant to make good water losses

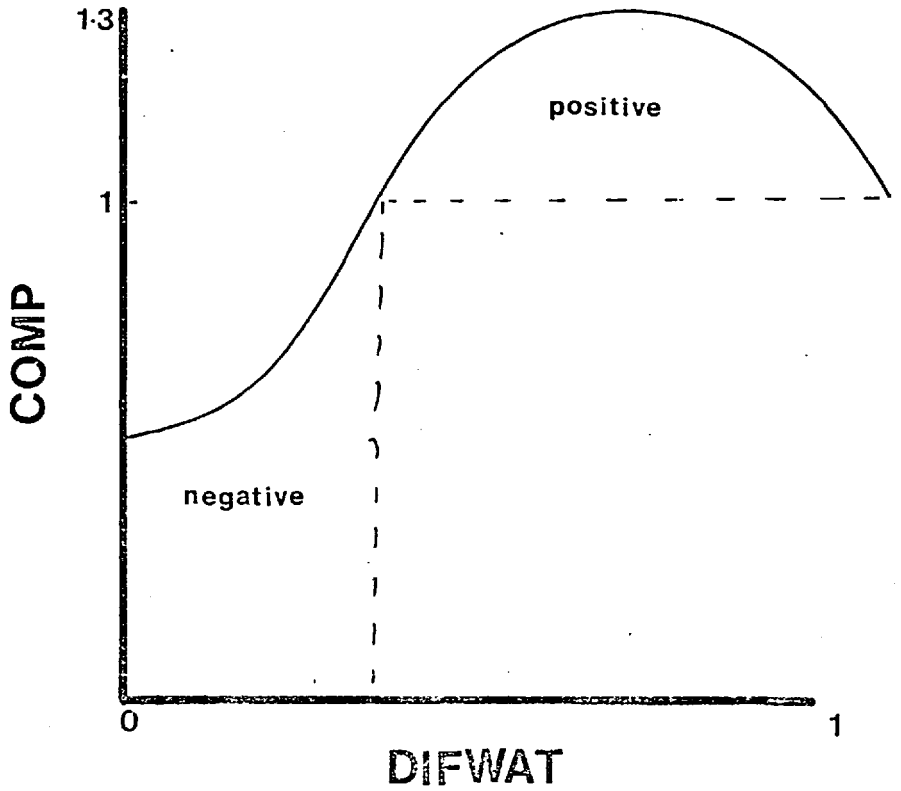


Figure 58. The relationship between the sub-optimality of leaf water content (DIFWAT) and compensation ability (COMP)

supply the necessary materials to allow elongation of the shoot and the production of the first leaves. The energy source for this activity is derived principally from the parent seed. The vegetative phase of growth consists of an overall increase in stature and photosynthetic productive power to supply the energy and nutrient needs of the early reproductive stage, when flower buds are produced and developed. The materials for this growth are, in the main, photosynthetic products. Towards the end of the life of an annual plant, reserves are mobilized from the older leaves whose photosynthetic function is now obsolete, to augment the flow of energy-rich materials into seed production; this late reproductive phase is the fourth condition dealt with in the model.

Changes from one condition to another occur at three thresholds which are modelled as follows. I assume that the germinating plant becomes vegetative when it has produced three nodes; it has, in other words, one leaf fully opened. We write

$$\text{if} \quad \text{NODES} \geq 3 \quad \text{then} \quad \text{COND} = 2$$

Similarly, if shoot weight exceeds the minimum stature necessary for flowering, and the daylength exceeds the flowering photoperiod, the plant becomes reproductive;

$$\text{if} \quad \text{DAY} \geq \text{PHOTHR} \quad \text{and} \quad \text{SHOOT} \geq \text{SHTMIN}$$
$$\text{then} \quad \text{COND} = 3$$

Finally, when the shoot weight exceeds another threshold, THR4, the plant begins to channel all its net production plus whatever reserves can be mobilized from dyning tissues into seed-fill;

if SHOOT ≥ THR4 then COND = 4

Clearly, the values of these three thresholds will be related to a number of environmental parameters (including the intensity of aphid feeding), and they can readily be made responsive to such factors in the model. In the present version, however, they are included as constants, because they are no data available.

6 Flowering and Fruiting

The precise time of flower initiation in most annual plants is under a complex of controls including temperature, photoperiod, plant stature and general physiological condition. Evans (1959) describes the environmental factors important to Vicia faba, and emphasises the variability in regard to flowering time between phenotypes of this species. All these controls affect the time at which COND changes from vegetative to reproductive (from 2 to 3) and the flow of dry matter into flowers begins (previous section).

Suppose that the plant has just entered the reproductive phase. An initial distribution of floral primordia must be set up, so that their relative sink strengths approximate the relative growth of flower buds in real plants. In V. faba the lowest flowering node is generally the ninth, but this varies with the variety of the plants and with crop density (Ishag, 1969). We therefore write NOFLO = 9, and, because the lowest nodes flower first in this species, we assign a relatively large flower bud to this node;

WTF(NOFLO) = WTINIT(1)

where WTF(NOFLO) is the weight of the flower bud at the NOFLOth

node, and WTINIT is a vector of initial bud-weights reflecting both the decreasing weights of the higher nodes, and the more advanced development of the floral primordia on the lower nodes. The increments, WTINIT, are made up from today's photosynthesis. So incrementing all the nodes higher than NOFLO which are expanded when flowering begins, we have

```
      Do 10 NODE = NOFLO, NODES
      N  =  NODE - NOFLO + 1
10    WTF(NODE)  =  WTINIT(N)
```

After this time, all the nodes developing from the terminal cluster will include a flower bud (page 168).

Let each flower have a maximum weight, WTFLOWR, and let the initial number of flowers at each node be FLOWRS(NODE). The maximum weight of flowers will therefore be

```
WTFLMAX  =  FLOWRS(NODE) * WTFLOWR
```

The flowers are assumed to increase exponentially in weight up to pollination, with the rate of growth determined by aphid feeding, temperature, and any other deleterious factors.

```
SINK(NODE) = WTF(NODE) * (EXP(GRF * E) - 1.)
```

where GRF is the growth rate of flowers, and E describes the intensity of the adverse factors at the node. The total flower sink of the plant is

$$TSINKF = \sum_{NOFLO}^{NODES} SINK(NODE)$$

so the fraction of the shoot weight increment incorporated in flower growth will be TSINKF/SIGSINK, and the flower weight increment at a given node will be

```
WTF(NODE) = WTF(NODE) + SHOOTIC * SINK(NODE)/SIGSINK
```

In Vicia faba there is a high flower mortality (Evans, 1959; Ishag, 1969), which is simulated by assuming that when the flowers have reached their maximum weight, and are pollinated (or not), then a fraction of the FLOWRS(NODE) are lost. The fraction of flowers surviving to become pods at a given node is FLOSURV(NODE) which is supplied as data.

The number of pods is then given by multiplying the initial number of flowers by the survival rate (to obtain the number of mature flowers), and then by the plooination rate;

```
PODS(NODE) = FLOWRS(NODE) * FLOSURV(NODE)
                          * POLLEN(IWEEK)
```

The rate of pollination is assumed to vary with time (related to bee activity) and to be constant between nodes. Next, the weight of pods is computed by multiplying the weight of mature flowers by a similar fraction;

```
WTF(NODE) = WTF(NODE) * FLOSURV(NODE)
                      * POLLEN(IWEEK)
```

and the biomass of dead flowers is lost to the plant.

The pods are assumed to grow sigmoidally according to the equation

```
SINK(NODE) = WTF(NODE) * (EXP(G * A * E) - 1.)
```

where G is pod growth rate, E summarizes the deleterious effects of environmental factors on pod growth, and A is the fraction of unfulfilled growth as follows;

```
PODMAX = PODS(NODE) * WTPOD
```

so PODMAX is the weight limit of logistic growth (the product of the number of pods and their individual weights), and

$$A = (PODMAX - WTF(NODE)) / PODMAX$$

Summing the pod sinks over all nodes bearing pods we obtain TSINKF, and hence the total pod weight increment

$$FRUTINC = (TSINKF / SIGSINK) * SHOOTIC$$

The pod weight increment at a given node is therefore

$$FRUTINC * SINKF(NODE) / TSINKF$$

When the weight of pods at a given node reaches a threshold, THRMORT, there is a further loss to the plant through pod mortality. That is,

if WTF(NODE) $\geq$ THRMORT
then PODS(NODE) = PODS(NODE) * FRUSURV
and WTF(NODE) = WTF(NODE) * FRUSURV

where FRUSURV is the fraction of pods surviving to maturity. After a prescribed period, or when the plant is fully grown, the crop is harvested. The yield of beans is given by

$$YIELD = BPOD * \sum_{N=NOFLO}^{NODES} WTF(N)$$

where BPOD is the fraction of the reproductive tissue which is beans (rather than shells or stalks).

7 Changes in the Phloem Sap

To the aphid feeding on the leaf or stem, the most profound changes in diet quality occur as a result of switches

in the physiological state of the plant, which manifest themselves in rearrangements of the proportions of the various chemicals making up the nutritious fraction of the phloem sap. The effects of such changes in diet quality can have significant effects on the rate at which the aphids feed (Auclair, 1963), their rate of development (Banks and Macaulay, 1965; Mittler and Dadd, 1966), and their reproductive rate (Lees, 1966; van Emden, 1969). Nutritional changes in the plant have also been invoked as one of the factors affecting the production of alate morphs (Lees, 1966; Hille Ris Lambers, 1966).

Let us assume that phloem quality (with respect to the aphid) is a function of plant phenological condition, and reflects changes in the amount of soluble nitrogen available. This will give us four basic values showing the differences in nutritional quality of germinating, vegetative, early reproductive and later reproductive plants (Kennedy, 1958). At any given node, however, food quality will also be a function of leaf age (Kennedy, 1958; Wearing, 1966; van Emden, 1969), so that young and senescent leaves give a higher diet quality than mature tissues. Now if we allow that FOODQU(COND) is the maximum phloem quality attainable in a given phenological condition, then the actual quality is given by

$$QUAL = FOODQU(COND) * F(LFAGE(NODE))$$

This measure of food quality can then be used in those processes involving aphid physiology (feeding rate, population increase, and so on). Lowering the quality of the phloem sap to the aphid pest can act as a powerful tool in reducing crop loss, especially in the breeding of 'resistant' varieties

(van Emden et al., 1969).

Running the Model

There are three principal sets of tests which we must make with the model in order to observe the effects of each component of the aphid feeding process on bean yield.

a) We must observe the response of the modelled system to the removal of photosynthate, and to the temporal pattern of this removal:

b) we must assess the effects of leaf water reduction on yield, accounting for the temporal pattern of feeding and the spatial distribution of attack within the plant (what is the difference between lowering the water content of young and mature leaves ?):

c) finally, since it is known that aphid infestation affects the pattern as well as the amount of growth (Chapter II), we must determine the effects of different distributions of aphid feeding on the relative growth rates of different organs.

For comparison with the output of these tests, a 'control' run is described; this traces the development of an uninfested plant through the same environmental conditions as will later be used with infested plants.

1 Control

Assume that the plant lives for two hundred days from planting to harvest, and that, for this period, we have weekly means of air temperature, radiation, soil moisture, and soil nutrient availability. The weight of the planted seed is supplied as input, and the total weight of beans, and their distribution with height appear as output (Fig. 59).

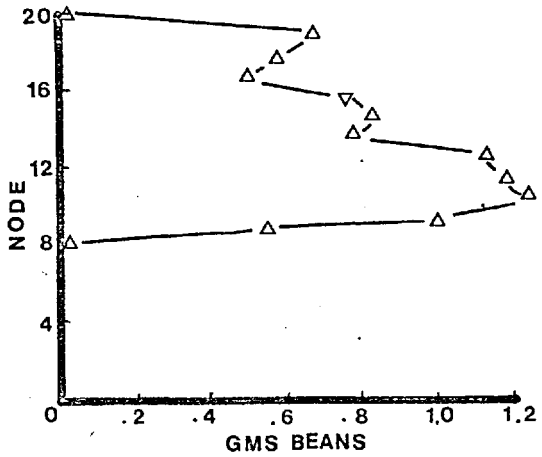


Figure 59. The final distribution of bean weights with height in a control run of the model

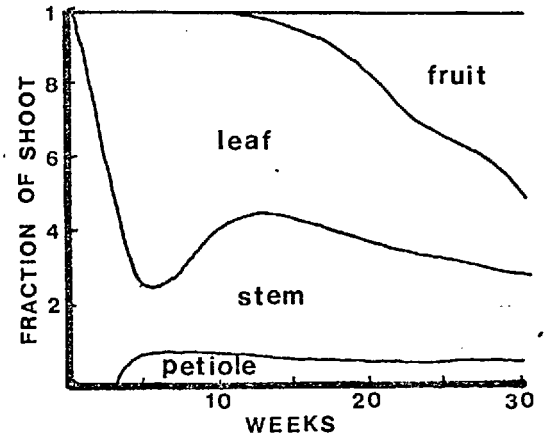


Figure 60. The proportional make-up of shoot biomass over the course of the same control run

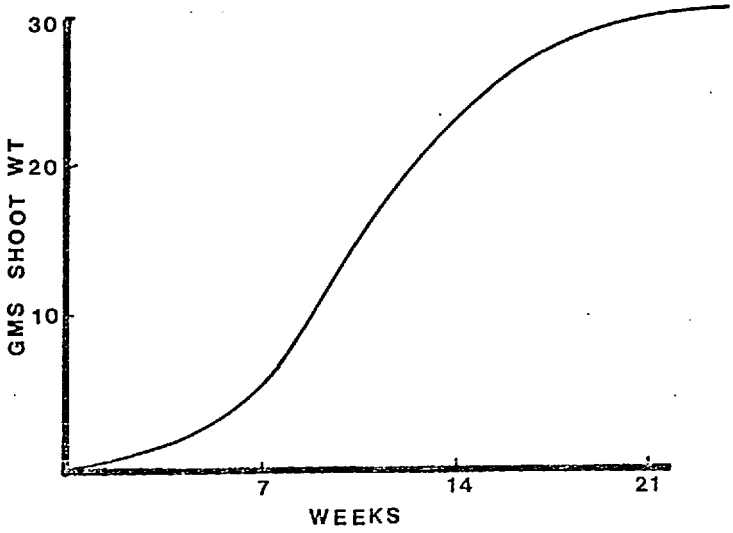


Figure 61. The pattern of shoot dry weight accumulation over the course of a control run

In addition to this, we can plot changes through time in the proportional make-up of plant biomass in the shoot between leaf, stem, petiole and fruit (Fig. 60), and the pattern of dry matter accumulation over the growing period (Fig. 61).

These results agree quite well with the data given by Ishag (1969) for the growth of Vicia faba in the field. Since several of his data sets were used in the construction of the simulation, this agreement does not constitute a validation of the model, but it does show that the several intuitive data sets, included to make up for the more important data shortages, have not grossly distorted the behaviour of the model. From this starting point it seems reasonable to proceed with the manipulatory runs, bearing in mind that the similarity in behaviour between the model and real systems under control conditions does not necessarily mean that the results of the model experiments will be realistic (Chapter I, VI).

2 The Removal of Photosynthate

The first set of manipulatory runs consideres the effects on plant growth of the removal of photosynthate alone; the pattern of aphid feeding between the nodes, and the effects of leaf damage are ignored. Clearly these runs can not be validated directly, as the experiments involved are impossible (that is, with real aphids we can not have a reduction in photosynthate without the other effects). To model this process we specify a particular time-pattern of feeding (Fig. 62) which is equivalent to an increase in aphid numbers followed by a population crash (Chapter II, Fig. 30). The intensity of the infestation is then altered by changing the height of the curve and keeping the overall shape and position the same. The integral of this curve is then an index of the damage done to the plant; it is the total dry weight of

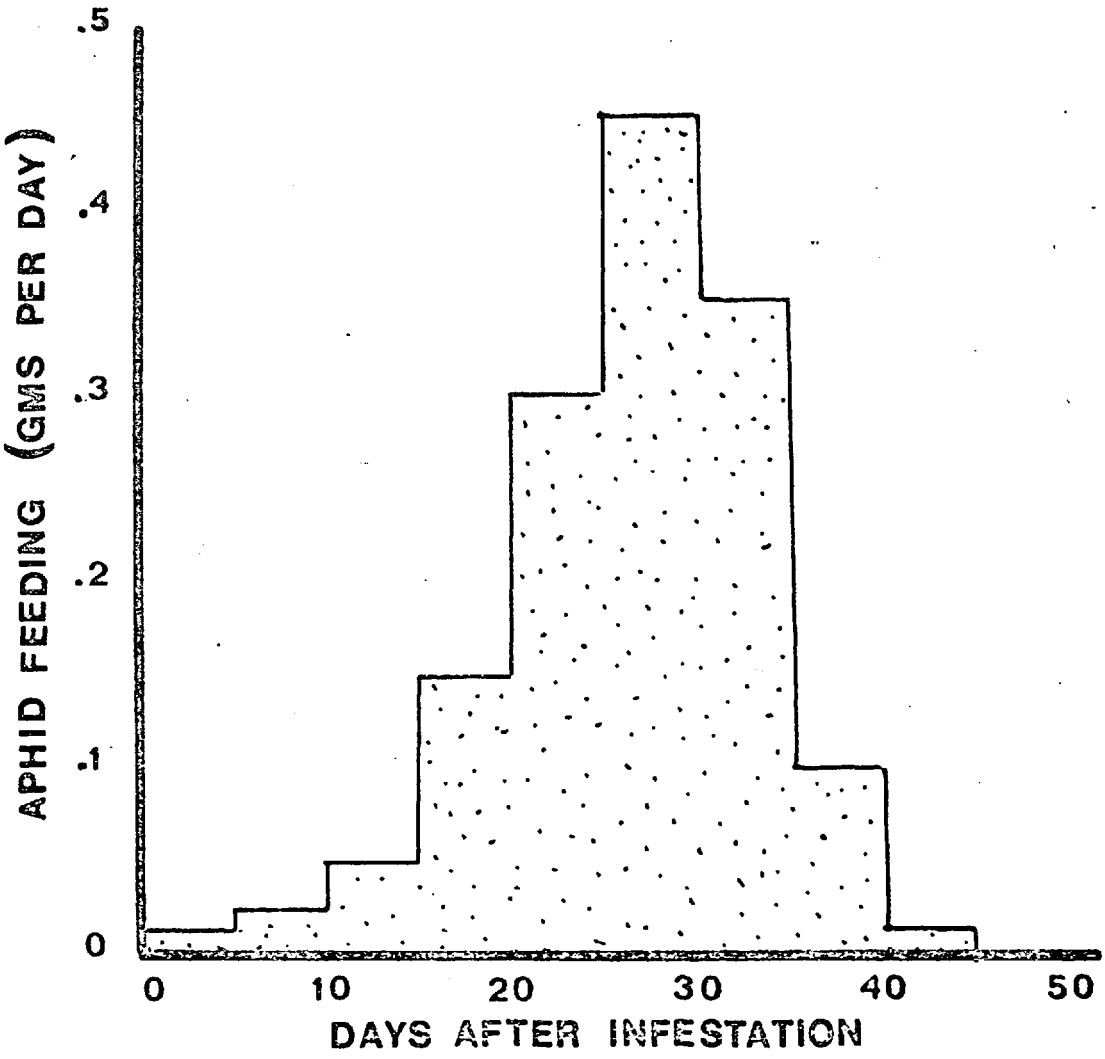


Figure 62. The time pattern of aphid food removal used in the model to mimic the effects of aphid population growth and decline. It is necessary to specify the integral of the curve (the total food removed), and the date upon which the infestation begins

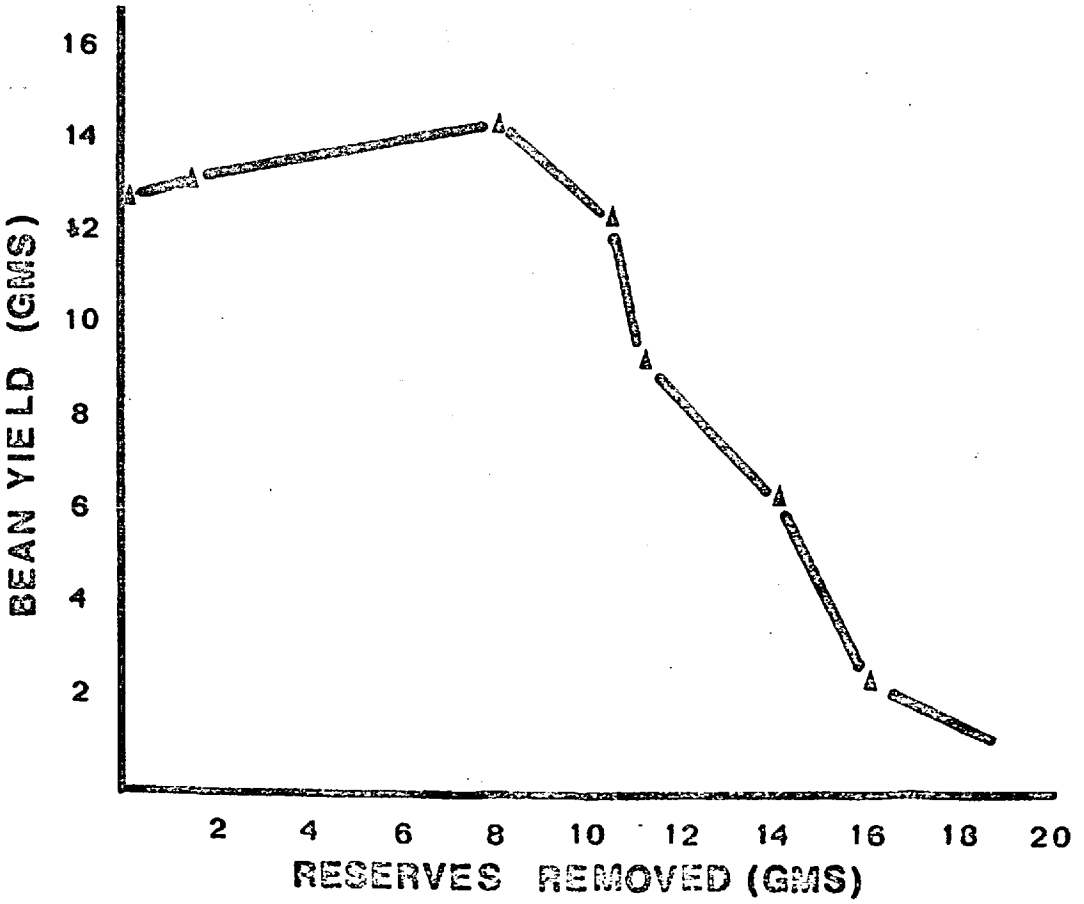


Figure 63. The relationship between the total weight of reserves removed by the aphids during the growing season and the final yield of beans

photosynthate removed over the feeding period. In Fig. 63 we plot the predicted yield of beans against the intensity of the aphid infestation, and note that up to a point (about 10 gms) there is no net loss of beans (indeed, there is a slight increase), but that after this point, yield falls off rapidly with increases in the weight of food removed.

Now, selecting a sequence of starting dates for the infestation, we can test the sensitivity of bean yield to removal of the same weight of photosynthate (say 14 gms) at different stages in plant development. This effect is graphed in Fig. 64, which shows bean yield rising asymptotically as the time lag between germination and infestation increases.

3 Leaf Water Reductions

The water content of individual leaves is maintained as a balance between losses occurring through evapotranspiration and aphid feeding, and gains occurring through osmosis and transpiration. The rate of aphid feeding and the drying ability of the air (wind speed, temperature and humidity) affect the rate of water loss, while the availability of soil water and the extent of the root system influence the rate of water replacement (Kozlowski, 1968).

The model has been built on the assumption that leaf water content can affect both the rate of photosynthesis and the sink strength of a growing leaf (above). Fig. 65 shows the response of leaf water content through time to two constant rates of sap removal. Because the curves are qualitatively different, the effects of leaf water reduction on the rate of photosynthesis will depend upon the absolute distribution of aphid feeding between different leaves; in other words,

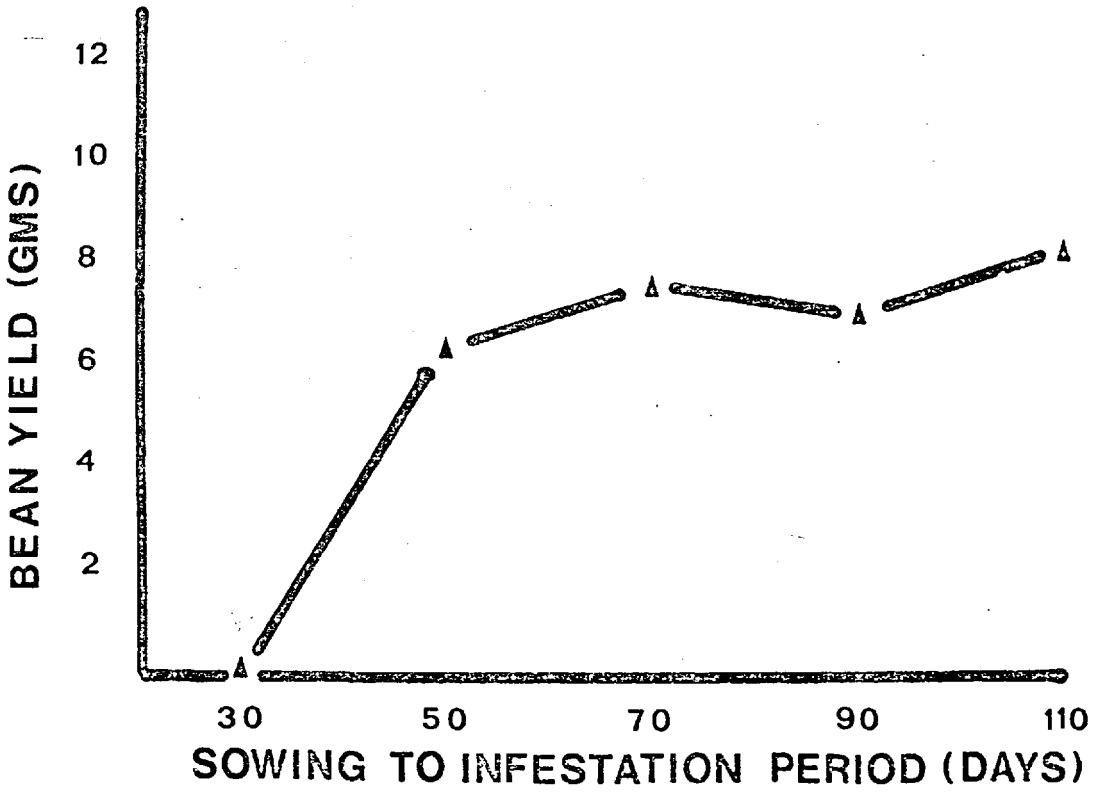


Figure 64. The relationship between the length of the time lag between sowing and aphid infestation and the final bean yield. The total food removal by th aphids was 14 gms.

there is no mean effect, and 100 aphids on one leaf will affect the plant differently than 10 aphids on 10 leaves. Since the leaves differ in their ages, and in the light they receive, the pattern of aphid feeding will be important in assessing the effects of unit leaf water reduction on net production.

When the available data show that the sink strength of the growing leaf is reduced by lowering its water content, then the process can be modelled by making the environmental parameter E in equation 41 a function of leaf water. Fig. 66 plots an hypothetical graph of E against LWATER in which it is assumed that leaf growth ceases (i.e. E = 0) before the leaf is entirely dessicated (i.e. LWATER > 0). The results of running the model with different rates of water loss are shown in Fig. 67 where bean yield is plotted against removal rate, and Fig. 68 where the growth curves of control and water-reduced plants are compared.

4 Pattern of Dry Matter Distribution

There are two ways in which we can alter the pattern of dry matter distribution in the model. The first is to consider that the reserves taken during aphid feeding are removed from the sink of the organ itself, rather than from the total pool available to the plant (as in section 2, above). In this case, sink tissues will grow in proportion to the number of aphids feeding upon them, and source leaves will export less material. In particular, if we have a sink tissue of weight W and maximum weight K then, by our previous definition, the fraction of the shoot weight increment which will pass into its sink is given by

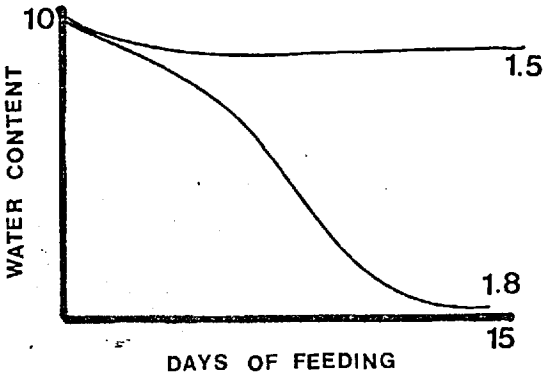


Figure 65. The effects of aphid feeding on leaf water content at two rates of removal

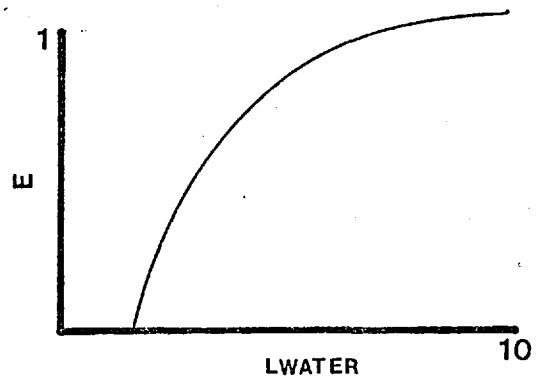


Figure 66. Hypothetical relationship between leaf water content and relative leaf growth rate (E)

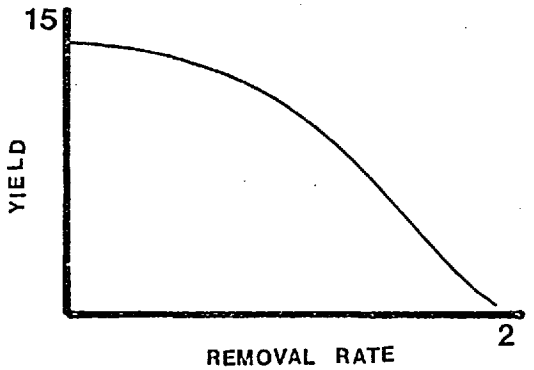


Figure 67. The relationship between leaf water removal rate and the final yield of beans from the model plant

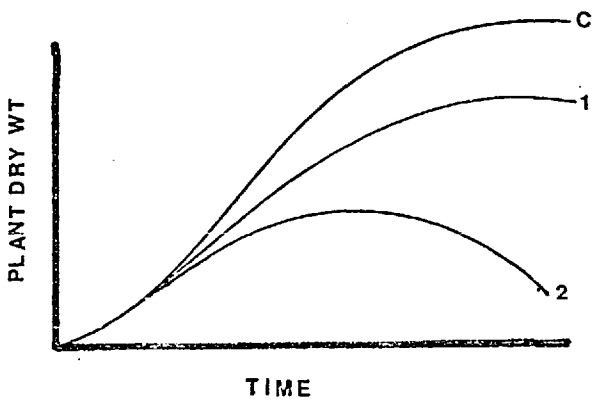


Figure 68. The time pattern of dry matter increase in control (C), 1.5 (1), and 1.8 (2) water removal rates.

$$f = W\left(e^{r\left[\frac{K - W}{K}\right]} - 1\right) \Big/ \sum_j W_j\left(e^{r\left[\frac{K_j - W_j}{K_j}\right]} - 1\right)$$

and the actual weight by

WSINK = SHOOTIC * f

Then, by this first system of considering aphid feeding, the actual weight increase will be this input less the dry weight removed by the aphids; i.e.

WTINC = WSINK - APHFEED(NODE) * FAF(NODE,J)

where APHFEED(NODE) is the weight of food removed by the aphid population at this node, and FAF(NODE,J) is the fraction of the aphids feeding at this node on the Jth tissue. The final growth of the tissue is therefore

W = W + WTINC

Source organs are defined as being leaves over 20 days in age, and these are treated differently. Since they are net exporters, their production is simply reduced in relation to the number of feeding aphids, so if the leaf at the Nth node produces PRODN grams of carbohydrate there will be

PRODNEX = PRODN - APHFEED(N) * FAF(N,J)

grams to export. Of this amount, however, the leaf removes some material for its own growth

PRODNEX = PRODNEX - WTINCL

and so the total amount of reserve available to the plant is

$$GPROD = \sum PRODNEX$$

the exportable material summed over all source leaves.

The second option open to us in modelling changes in dry matter distribution can be used with either pool- or tissue-specific reserve removal. It consists of making the sink strength of the tissue dependent upon the number of feeding aphids, so that the tissue is 'damaged' and its growth impaired. The method is described in equations 10 to 16 in the introduction, and the model is run by specifying the relationship between the number of aphids feeding on the tissue (APHFEED(N) * FAF(N,J) as above) and the value of E, the relative reduction in sink strength. Fig. 69 shows two typical curves of E against feeding; the upper curve assumes that only at very high levels of reserve removal is the relative growth of the organ affected, and at no time do the aphids stop growth completely (i.e. $E \neq 0$), while the lower curve describes the situation in which aphid feeding accounts for quite dramatic reductions in sink strength, and the tissue stops growth completely above certain levels of feeding ($E = 0$).

This treatment of dry matter distribution has the most profound effects upon bean yield, depending upon the shape of the function employed (Fig 69). This aspect of model structure was not included to deal with the direct effects of carbohydrate removal, but rather to consider the effects of tissue damage on reserve distribution. We should therefore plot the value of E not against current feeding, but against the total number of aphid-days experienced by the tissue. This allows that the damage effects will be cumulative (as they would be in real plants; see Chapter II). A better picture

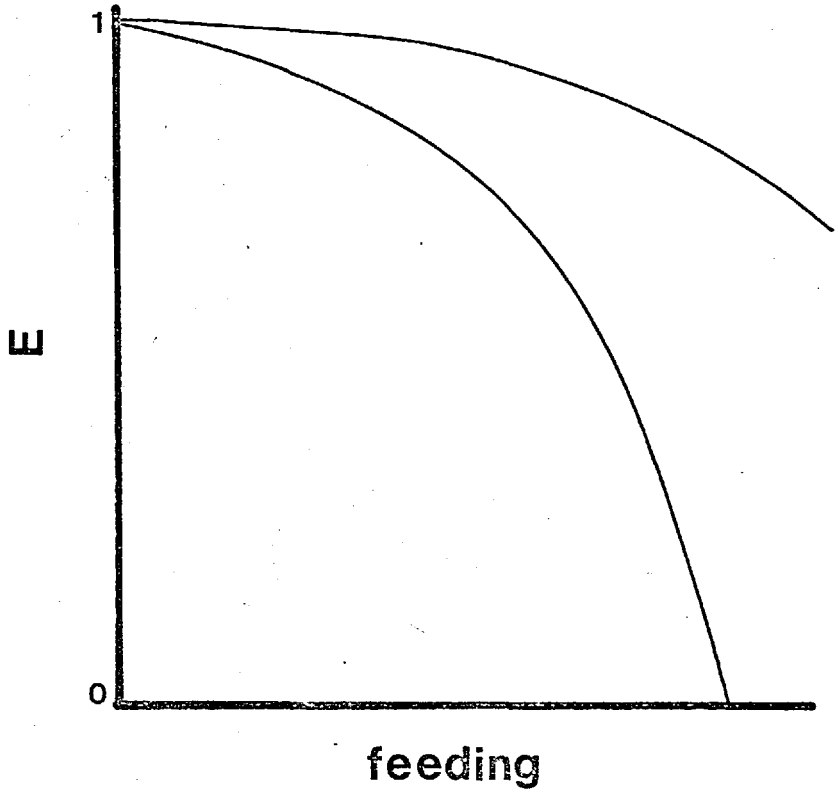


Figure 69. Two possible relationships between the amount of aphid feeding an the relative growth of the host organ (E)

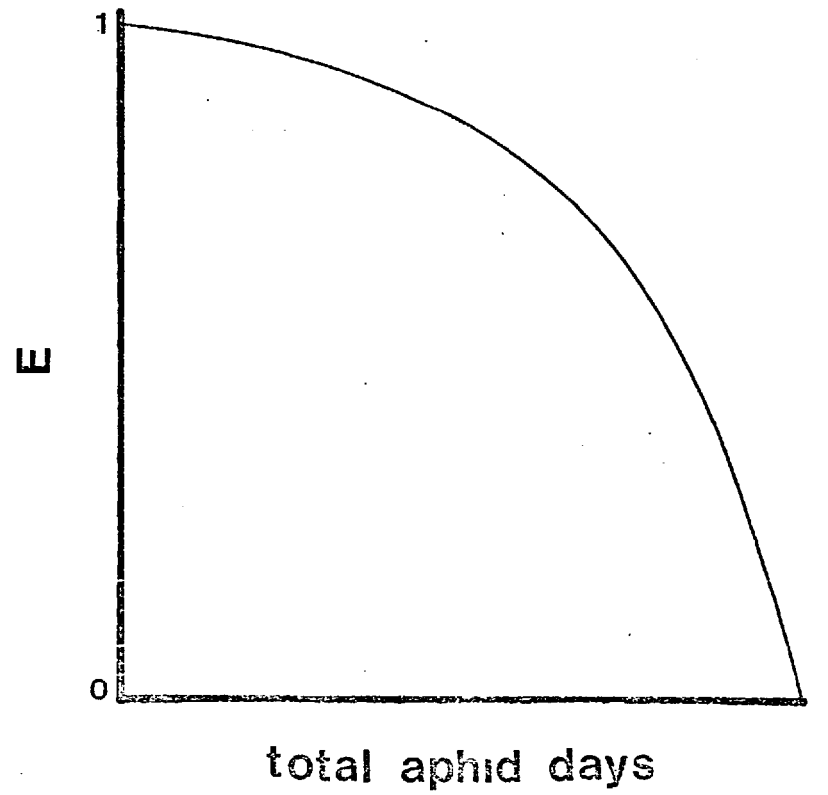


Figure 70. Relative tissue growth as a function of total damage (measured as aphid days)

of the overall effect of aphid infestation on dry matter distribution is gained by employing the first system of aphid feeding (direct subtraction from the sink of each tissue), and allowing that the strength of each sink is determined by the damage it has experienced (Fig. 70). Under this regime, the growth of a tissue would only be reduced by the amount of reserve removed when the infestation was young (and E = 1 in Fig 70), but would suffer a reduction in sink strength as well as a direct removal of carbohydrate as the infestation aged, and the effects of damage became more prevalent (and E becomes significantly lower than 1).

Discussion

The purpose of this model is to investigate the relationship between aphid feeding and bean yield. Three critical aspects of the problem have emerged during the study; the timing of the aphid infestation relative to crop establishment and to flowering: the numerical pattern of aphid feeding over the period of infestation: and the distribution of aphid feeding and tissue damage between the different nodes and organs of the plant.

a) Timing

Aphids arrive in a bean crop as alatae. These animals are produced during the early spring build-up of numbers on the primary, over-wintering host plant, Euonymus europaeus, and they fly off to find new host plants either when their density on spindle becomes intolerably high, or a specific photoperiod is reached. Way and Banks (1964, 1968) describe the biology of this spring phase of the life cycle of Aphis

fabae. Because of the duality in the mechanisms causing alatae to emigrate from the winter hosts, there is a wide range of time over which this dispersal can occur. If spring conditions are such that the eggs laid on spindle hatch rather early, then the available area of leaf on which feeding can occur is minimal, and crowding will rapidly bring about alate production. Under these conditions, however, aphid survival is likely to be rather poor, and so a small early infestation is likely on the bean crop.

If spring conditions delay egg hatching, the spindle bushes will have developed a considerable leaf area by the time that larval feeding reaches its peak. In this case, the population can build up to high levels before crowding becomes important, and the emigrants will be more numerous. It is possible, however, that emigration is delayed so long that the bean plants in the crop are no longer attractive to the aphids, and infestation might be slight.

The density of eggs laid in the autumn will also affect the timing of the spring emigration. At low egg densities crowding will occur later in the season (if at all), while high egg densities will tend to give rise to crowded conditions and alate production relatively early. Here again, the precise effect will be modified by the time of hatching in relation to leaf expansion in Euonymus.

The model suggests (Fig. 64) that aphid infestations of moderate intensity can not be tolerated by the plant if infestation occurs much before the 40th day after germination. It is not the purpose of the model to attach any particular significance to this number, but rather to show that there is a critical threshold in the time of infestation,

below which yield reduction will be highly significant. The model behaves in this way for two main reasons. First, in small plants the aphids are relatively more clustered in the terminal leaves around the meristem, and by their feeding and damaging effects, slow down the rate of leaf production significantly. Second, smaller plants have proportionately less potential to compensate for carbohydrate losses; their leaf area is lower, and a greater fraction of it is directly affected by the pest. Additionally, in very young plants the bulk of the reserves used in growth originate from the seed, and if this material is channelled into aphids rather than new leaves, the plant will be doomed as soon as the seed reserves are exhausted.

A second aspect of the timing of the aphid-plant interaction concerns the duration of the infestation. As we have seen, the initiation of the aphid population is under a comples control; so it is with the termination. It is possible to enumerate a set of factors which bring about the downfall of the pest population, some of which are more desirable than others. In the undesirable class we can place plant death and, in most circumstances, the mass emigration of aphids due to crowding (because this crowding will usually have been associated with significant plant damage). If the aphids arrived on the plant relatively late in the season, or the crop was planted late, it is possible that the aphids emigrate because of decreasing photoperiod (Lees, 1966) before causing significant damage to the crop. Finally, we can terminate the aphid infestation by management intervention, but in this case the costs and the benefits must be carefully weighed.

b) Number of Aphids

The model was run by specifying a pattern and intensity of aphid feeding for each simulation (Fig. 62). In the field, however, there is a feed-back between the plant and the number of aphids feeding upon it. Various aspects of plant quality (nitrogen content, water content, genetic resistance, and so on) affect the developmental and reproductive rates of the animals (thereby affecting the number of feeders), and their growth and feeding rates (thereby altering the extent to which each aphid damages the plant). This model does not attempt to simulate these interactions, but incorporates their effects directly in the shapes of the exploitation curves (Fig. 62). The mechanisms by which these processes act, and their relative effects on total aphid feeding, are considered in Chapter IV.

From Fig. 63 it seems that the plant can tolerate total dry weight removals of up to about 10 gms without suffering any significant loss in bean yield (as long as infestation occurs late enough; see above). The mere presence of aphids on the plant is therefore not necessarily a justification for employing control measures; it also means that biological control is a possibility because the plant can sustain a number of aphids, and hence a number of predators, without an economic loss.

The variable of principal concern is not the number of aphids, but rather the rate of dry matter removal (the product of the number of aphids and their individual feeding rates); similarly, the need for pest control measures is not only the rate of food removal, but the rate of food removal relative to the stature and developmental stage of the plant.

c) Pattern of Feeding Within the Plant

As shown in Chapter II, the aphid population is distributed non-randomly over the plant surface; some nodes are preferred to others, and the tissues within each node bear aphid aggregates of different densities. This being the case we should expect that the pattern of growth of aphid infested plants differs from the controls, and that the relative growth of a tissue is a function of the number of aphids feeding on it.

Runs of the model have shown that it is possible to mimic the type of response observed in the experiment (Chapter II) only by removing reserves directly from the sink tissues, and by considering the effects of tissue damage on sink strength. There are no data available to suggest that the rate of feeding by Aphis fabae differs with the tissue type, and so we must assume that the amount of food removed bears a consistent relation to the number of feeding aphids for all tissue types. This rules out the possibility of simulating the observed relative decrease in stem growth simply by direct removal of reserves, since fewer aphids fed on the stems than on the leaves. It is essential to consider the effects of aphid presence on the sink strength of the tissue to obtain model output which agrees with the experimental results. We should take care, however, to ensure that the model does not simply become a crude means of curve fitting; with so many effects it would be quite simple to juggle the parameters in the damage sub-model to fit any set of validation data.

The important point is that the model shows great sensitivity to the gradient of the relationship between the damage variable E and aphid numbers, and also to the system by which reserves are removed (either from a central pool, or from the

sinks of the tissues directly). Data on these effects have never been collected, and the model does suggest some potentially rewarding experiments; the difficulty, of course, would arise in attempting to damage tissues in an aphid-like way without removing carbohydrate.

d) Plant Compensation

While allowance has been made in the model for recovery of leaf water content after aphid feeding (Fig. 54), no specific mechanism for compensation in net production has been included. The results of the experiment in Chapter II do suggest, however, that the plant has some ability to compensate for carbohydrate removals, particularly in the late vegetative stage. We defined this compensation by graphical means in Fig. 38; if yield reduction and aphid feeding are equal then there is neither positive nor negative compensation, while yield reductions greater than or less than food removal demonstrate negative and positive compensation respectively. The mechanisms by which positive compensation might occur could be through the removal of photosynthesis-limiting build-ups of carbohydrate (Maggs, 1964; Neals and Incoll, 1968), or through a redirection of reserve translocation towards the shoot and away from the root (Davidson and Milthorpe, 1965; Marshall and Sagar, 1965; Thrower, 1962). Similarly, reserves may be mobilized from a storage tissue to redress the balance (Cavers, 1971).

Negative compensation would occur if the feeding aphids inhibited the process of photosynthesis directly, as they might by injecting saliva (Dixon, 1970; Miles, 1969; Mittler and Dadd, 1964), or by damaging the leaf surface (Chapter II).

The model does include an aspect of this effect, in making the rate of photosynthesis a function of the proportion of stomata blocked by honey-dew, and of leaf water content (equ. 24). There is an element of passive compensation in the model in so far as the rate of root growth is coupled to the rate of shoot growth by means of the optimal root proportion parameter (OPTRPR), and so reductions in shoot weight will lead to a relative increase in the amount of reserve channelled into the shoot. The potential to make the rate of photosynthesis an increasing function of the rate of carbohydrate use, and hence allow for real positive compensation, is included in the model as a relationship between photosynthetic rate and the amount of free reserves in the leaf (CHO on page 155), but since the decision was made not to consider the separate types of carbohydrate, this process is redundant. In order to model compensation realistically, it would be essential to model the dynamics of the soluble carbohydrate pool.

e) Control Recommendations

The collected attributes of an aphid infestation which would cause no significant loss of beans can be determined from the simulation runs. The aphids should arrive late in the crop and stay for a brief period. The rate of damage per aphid should be as low as possible, which will occur with minimal individual feeding rates and maximum plant tolerance of feeding. There will be a threshold between these effects related, but not identical to the economic threshold (Stern, 1973): an example is given in Fig. 71 for a plant with maximal resistance just prior to flowering.

Low aphid feeding rates will be associated with low individual growth rates and low temperatures (Chapter IV),

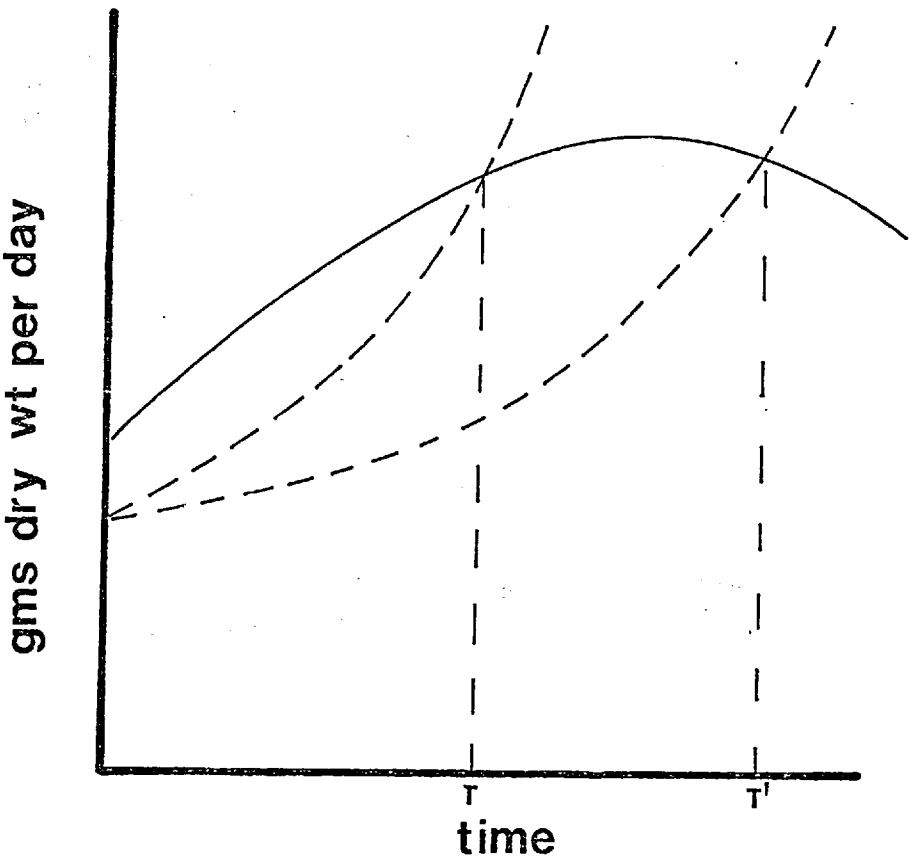


Figure 71. The relationship between plant tolerance (solid curve) and aphid food removal rates (hatched lines) showing how the rate of aphid population growth can affect the time at which damage is felt (T' versus T). Clearly increasing the steepness of the plant tolerance curve would have the same desired effect

but the net effect will depend upon whether photosynthetic production and aphid feeding are affected to the same extent by changes in temperature. In addition, the rate of aphid population increase should be slow; this would be affected by the nutritional quality of the plant, by weather conditions, and by the abundance and synchrony of natural enemies. These effects are considered in detail later (Chapters IV and V). Finally, the damage to the plant will be minimized for any given time and intensity according to the pattern of feeding within the plant. Feeding should be minimal on very young leaves and on the terminal cluster in particular, since the rate of new leaf production is critical to final bean yield. Feeding on mature leaves tends not to affect the pattern of growth of the plant, but only its rate, while senescent leaves contribute rather little to plant growth and consequently aphid feeding at the older nodes is less damaging. In the field, however, the inverse of these ideal feeding arrangements is observed, with aphids concentrating (at least initially) on the youngest leaves. It seems that what is worst for the plant is best for the aphid.

We can summarize the relative importance of pest control measures in relation to the time of infestation and the rate of increase of the aphid population.

RATE OF INCREASE	TIMING		
	EARLY	MID-SEASON	LATE
LOW	0	0	0
MED 1	1	0	0
MED 2	2	1	0
HIGH	3	2	1

Here 0 indicates no control necessary and 3 indicates that pest control would be economically advisable.

f) Assessment of the Model

The simplest model which could be employed to predict the effects of an aphid infestation of given size on the ultimate yield of beans from the crop might consider the plant as a single unit of biomass, which increases in weight under the control of a simple growth equation like the logistic. Our only biological parameter would be W, the dry weight of the plant, and we should need two constants to describe the maximum weight of the plant (K) and the rate of approach of W to K (r). The weight at any time is then given by

$$W_t = K / \left(1 + e^{-r.t\left[\frac{K - W_o}{W_o}\right]}\right)$$

The growth occurring in a finite time interval is therefore the difference equation

$$W_{t+1} = W_t \cdot e^{r\left[\frac{K - W_t}{K}\right]}$$

If data were available describing the pattern of dry matter accumulation of aphid infested plants it would then be possible to fit another parameter to the equation to describe the deleterious effects of aphid feeding. If we call this parameter A, and rewrite the difference equation as

$$W_{t+1} = W_t \cdot e^{r \cdot A_t\left[\frac{K - W_t}{K}\right]}$$

it will be possible to compute a set of values for A_t at each time t so that the pattern of growth fits any observed data. Fig. 72 shows the growth of an hypothetical plant under control and infested conditions, and shows the pattern of values for A_t necessary to fit the model to the lower curve.

The problem involved in this approach (which is no more than a curve-fitting procedure) is the extent to which changes in the aphid population are related to changes in the value of A_t. For the model to have biological as well as applied value it must be possible to calculate A_t given the number of aphids N. Fig. 73 shows the model which might be fitted to the data in the first instance, namely a linear decreasing curve of A on N. Depending upon the fit of the data points to the curve employed, we can guage whether this model accounts for sufficient variation to meet the needs of the problem in hand. If the main objective is to obtain quantitative predictions of yield reduction under different patterns of aphid feeding this approach could be very powerful, but if, as in the present context, the principal aim is to assess the qualitative effects of the biological interactions involved, the method falls short, because it considers so few of the processes occurring. In particular, a linear regression of A_t on N assumes that

a) the aphid population feeds at a rate which is directly proportional to its size (i.e. that it has a stable age distribution, and constant rates of feeding at each age):

b) the plant is affected to the same degree by unit aphid feeding at all stages in its development:

c) the position of aphid feeding within the plant does not affect the final yield of beans:

d) the plant does not compensate for aphid feeding:

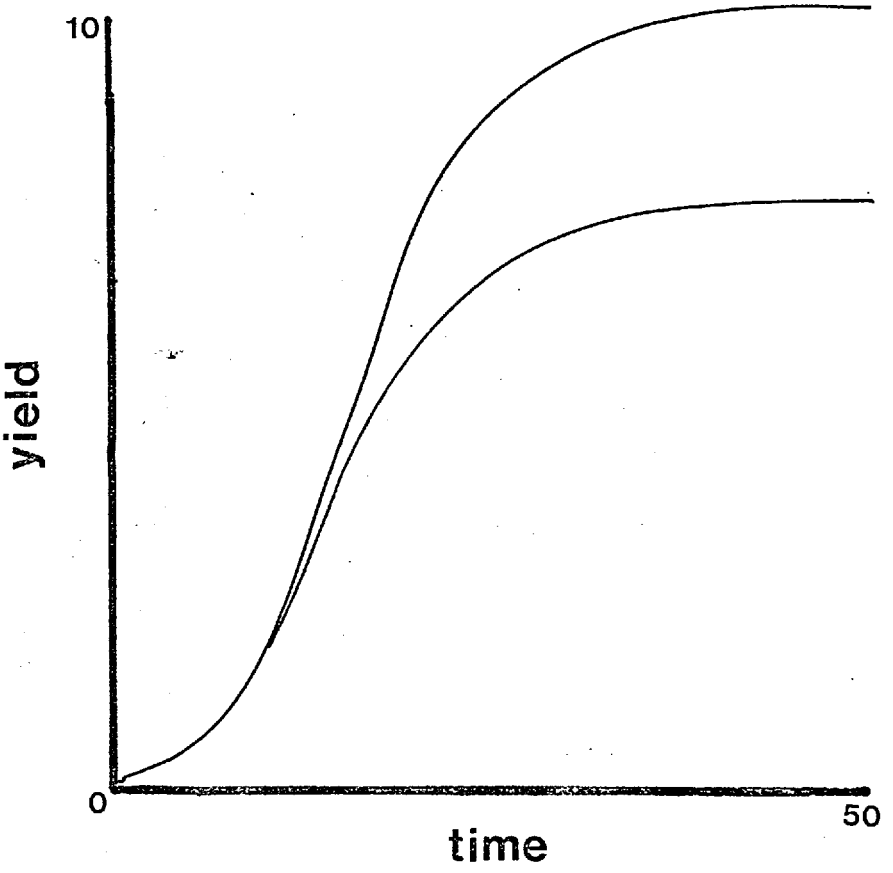


Figure 72. The growth of control and infested plants. The lower curve is developed by assuming values of A of 1,.7,.5,.3,.08, .03, and .0008 at times 15, 20, 25, 30, 35, 40 and 45.

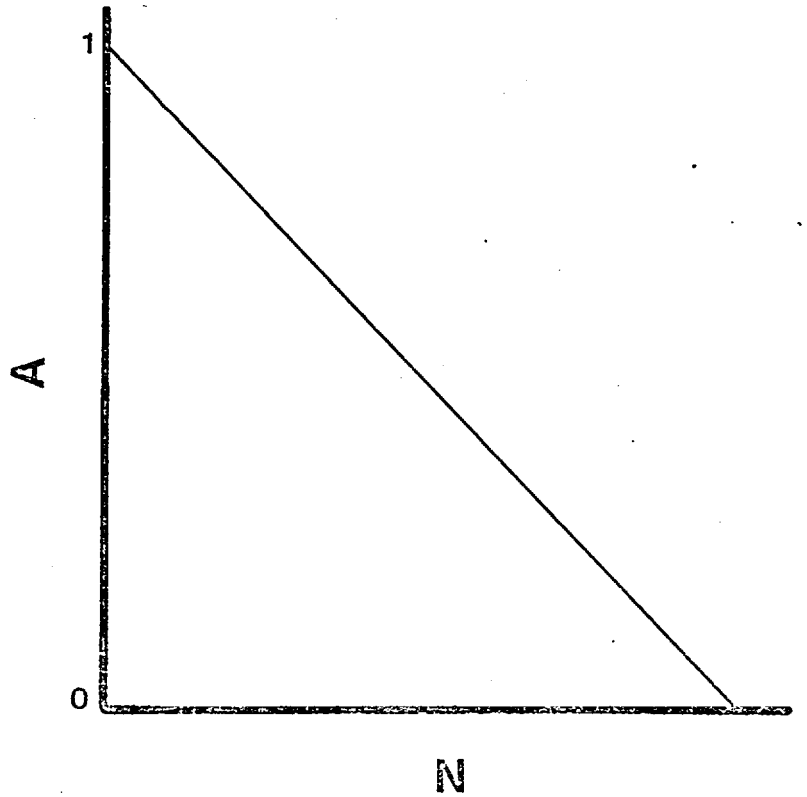


Figure 73. An idealized model of the relationship between prey numbers and the relative gorwth rate reduction (A).

e) the effects of aphid feeding are not compounded through damage to the plant's productive processes:

f) the aphid population does not affect the pattern of dry matter distribution, or that the pattern of dry matter distribution does not affect bean yield.

All these effects would tend to increase the scatter of the data points about the curve fitted between A and N.

The present model has been built to consider these effects, and to gain qualitative insight into their interaction. The model does not represent the most efficient means of predicting the actual weight of beans lost under a given aphid infestation (this could best be achieved empirically), but it is a step towards understanding the effects of sap-feeding insects on plant growth and crop yield.

The only strategy which can emerge from the simple model is that pest control consists of a reduction in the number of feeding insects; the present model investigates the pattern and timing of infestation which has least depressive effect on bean yield, and hence allows the formulation of control strategies which relate the benefits of increased yield to the costs of killing the insects at different stages of plant growth. This fundamental distinction between models designed to produce accurate numerical answers, and models designed to investigate the behaviour of complex processes is considered in Chapter VI.

The difference between the two approaches lies in the number of variables considered. Instead of treating the plant as a single unit of biomass, I have divided the plant into tissues and nodes, to allow for changes in the distribution of dry matter between the organs, and variation in the intensity of aphid feeding between nodes. The model is not

phrased in terms of internal equilibria; if the aphids were removed from the simple model (i.e. A_t were set to 1) the plant would eventually reach its maximum weight, and the aphids, in effect, only slow down the rate of growth. In the present model, on the other hand, the effects of aphid infestation are lasting, in that the plant would not reach its potential maximum weight if the aphids were removed; tangible damage is done to each of the plant organs infested, and so long as the rate of new leaf production is slowed down sufficiently that the existing leaves age (and hence loose photosynthetic capacity) more rapidly than leaves are produced, the plant will never recover.

Because the number of variables included is limited by the demands of the problem under scrutiny, the present model is not a particularly sophisticated representation of the current state of plant physiology (see, for example, Evans, 1972). Each plant physiological process modelled could have been stated in more detail, but every process must be considered in relation to the likelihood that aphid infestation would affect its operation, and to the availability of data to describe these effects. The fact that data are available does not argue for the inclusion of a process which can not be shown to be important in model structure however.

There remain several obstacles to the construction of realistic plant growth models, not the least of which is the relationship between the three types of carbohydrate, and the translocation and incorporation of assimilates in different parts of the plant. Because of this, and the problems associated with obtaining data on individual processes, there have been few attempts to model the effects of grazing on plant growth. As I have suggested, it is very difficult

to separate the interacting effects of carbohydrate removal, leaf water reductions, surface damage and saliva injections on the rate of photosynthesis and the sink strength of growing tissues. These problems will only be solved by sophisticated and carefully designed experiments.

The aim of this model was to suggest the pattern and intensity of aphid feeding which we should aim to achieve by biological control. The properties of the aphid feeding pattern which emerged can now be used to determine the strategy of predation necessary to maintain bean yields in a model population of Aphis fabae.

CHAPTER IV A SIMULATION MODEL OF THE GROWTH AND FEEDING OF AN APHID POPULATION

Introduction

The simplest mathematical description of the growth of an animal population is the exponential, or Malthusian equation. This expresses the number of animals after time t (N_t) in terms solely of the initial population size (N_o), and the potential rate of increase, r. We have, therefore,

$$N_t = N_o \cdot e^{r.t}$$

and, for any particular time interval, we can write the difference equation

$$N_{t+1} = N_t \cdot e^{r}$$

If the animal population is restricted in its growth

so that the environment can support a maximum of K individuals (because of space shotage, or lack of resources), then the equation can be modified to the form suggested by Verhulst; the well-known logistic function. Here, the number of animals at time t will be

$$N_t = K / \left(1 + e^{-r.t} \cdot \left(\frac{K - N_o}{N_o}\right)\right)$$

after Pielou (1969), setting her constants a/b = K, a = r. The logistic can equally be presented as a difference equation, so that

$$N_{t+1} = N_t \cdot e^{r\left[\frac{K - N_t}{K}\right]}$$

The abundance of many animal populations is related to the abundance of their competitors and natural enemies, and to model this phenomenon Lotka (1923) and Volterra (1926, 1931) independently developed equations expressing the rate of increase in numbers as a function of the density of competing or predating species (see Chapter V).

These elementary models of population growth are ideally suited to demonstrating the importance of the number of animals relative to the environmental capacity, and the number of animals relative to the abundance of their natural enemies, in affecting the pattern of numerical change. They have, in consequence, been usefully employed in tackling problems in which such considerations take precedence (species diversity (MacArthur, 1972), biogeography (MacArthur and Wilson, 1967; Simberloff, 1969), and studies on the population-structure

of ecosystems (Gallopin, 1971; May, 1971) for example).

Many ecological problems, however, involve variables and processes which it would be difficult to express in a form as analytically attractive as these examples. This is particularly true when applied ecological problems are considered, in which it is necessary to investigate the effects of man-induced changes to the system, and to assess their effects not only on the numbers of animals present, but also on the permanence or stability of the system after intervention. In such cases it is often useful to resort to simulation modelling, since this approach facilitates the construction of a model which is oriented towards the specific problem in hand, and which can be structured to consider as many variables as are known to affect the behaviour of the system to an important extent.

In this Chapter I shall discuss a simulation model of an aphid population (Aphis fabae Scop.) designed to investigate the effects of different strategies of predation on the damage done to the host plant (Vicia faba L) by aphid feeding. The model differs from the examples given above because it must consider the effects of prey selection by age and size, and the aphid population must be divided into age classes. The age structure of the population (the fraction of the total number of animals in each age class) may not be stable, and so we must consider birth and death processes as time-related. Again, the rate of food removal from the plant will not be a linear function of total aphid numbers when the age structure is non-stable, and so this too must be determined in more detail. The dynamics of the predator population are not considered in detail in this particular model, since the

strategies of predation can be defined simply as a set of rates of aphid-kill per age (or size) class, independent of the precise patterns of numerical change in the predator species (these are discussed in Chapter V).

The Model

In Chapter III the attributes of a population of Aphis fabae which would cause no significant economic damage to a crop of broad beans were defined. The problem to be tackled here is to decide on the strategy of predation which most effectively brings a model population of aphids to this tolerable level of abundance.

A strategy of predation can be defined in terms of three attributes; the number of aphids eaten, the time-pattern of attack, and the proportion of aphids of different ages (or sizes) killed. The input to the model will therefore need to state these three parameters.

As output from the model we have the parameters of aphid infestation defined in Chapter III; late development, short duration, low peak numbers, low individual feeding rates, and so on. With the inputs and outputs defined in this way, we can draw a flow-diagram of cause and effect between the strategy of predation, and the amount of feeding by the aphid population (Fig. 74). The variables included in this flow-diagram will then form the structure of the model.

A search of the literature, supplemented by information collected during the experiment (Chapter II) will then supply the data necessary to quantify the relationships represented by the arrows in the flow-diagram. Where no data exist, but the relationship is thought to be importantly affected by

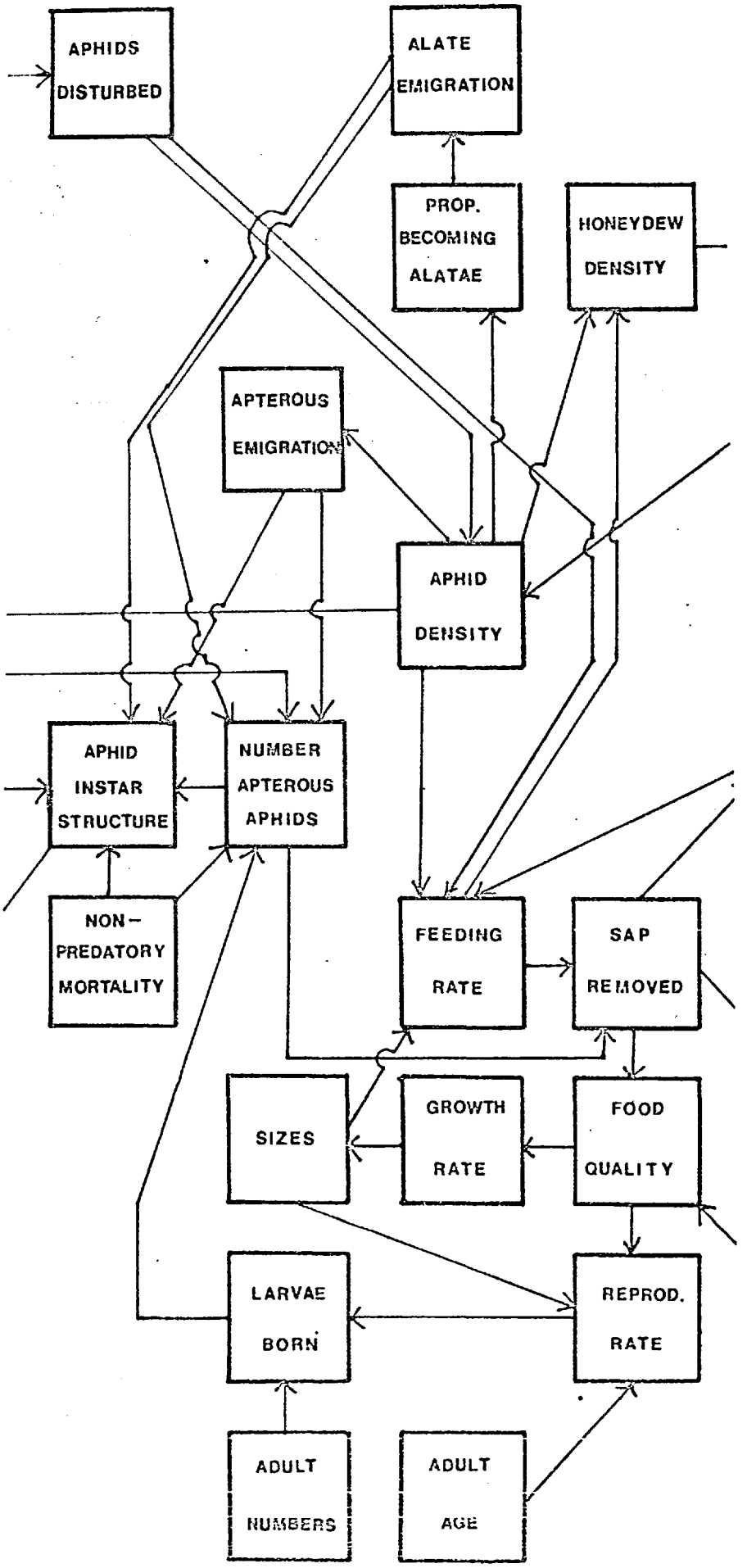


Figure 74. Flow diagram of the aphid population model

the strategy of predation, then intuitive data sets have been included (Chapter I).

Consider the aphids living within a meter-square of crop as representing a population unit, and let the total number of aphids present by TOTAPH. Now let the population be divided into a number of age classes, and let the number of apterous (wingless) aphids of any particular age in days be APTERAE(I), where I takes any value between 1 and the maximum age to which an individual of Aphis fabae can live. This population vector will then form the backbone of the model, and we shall consider in detail the processes which act to increase aphid numbers (birth and immigration), and those acting to decrease abundance (death and emigration, with special emphasis on death by predation).

1 Rate of Development

The single most important factor in affecting the rate of development of an insect from birth to maturity is temperature. In order to include this effect in their model of aphid population growth, Hughes and Gilbert (1968; Gilbert and Hughes, 1971) assumed that the most crucial aspect of the process was the number of day-degrees experienced by the insect above its emergence threshold (the lowest temperature at which development is possible). Further, by assuming that each larval instar of the aphid was of the same duration at constant temperature, they could run the model using a 'physiological time-scale', namely instar-periods (see Hughes, 1963).

There are three main draw-backs to this approach. First, if the model is to consider more than one species (a predator

as well, for example) then both species must have the same temperature thresholds of emergence, and a constant relationship between the duration of their respective instars and temperature. Second, all the instar periods are assumed to be of the same duration, and, while this may hold for Brevicoryne brassicae in Australia, it does not apply to Aphis fabae under British field conditions (Milne, 1971). Third, all the time units of the model (instar periods, or, in Hughes and Gilbert (1968), quarter-instar-periods; quips) are assumed to be similar. If we consider a particular aphid instar which lasts two days at a given temperature (say 15°C), then the quarter-instar-periods (the quips) can not possibly be similar, since each quip is roughly equal to half a day in real time units. Hence, if the quips are related to noon and midnight, the morning quip will be longer than the afternoon quip (because the mean morning temperature is lower), and if they are related to sunrise and sunset, then one will be in darkness and the other in light.

To avoid these shortcomings, the current model runs with real time units (days), and simulates the effects of temperature on development rate by assuming that each instar lasts for a certain number of day-degrees above the temperature threshold. This threshold can differ from species to species. Now, we define DAYDEG(I) as the total number of day-degrees above the threshold experienced by an aphid of age I days since its birth. This is updated each day so that

$$\text{DAYDEG(I)} = \text{DAYDEG(I)} + \text{TEMP} \qquad \ldots\ldots (1)$$

where TEMP is the mean temperature for the day less the development threshold (net day-degrees).

Let INSTAR(I) be the instar in which the aphids of age I are currently disposed, and let DEGTHR(K) be the number of day-degrees which must pass before an aphid in the Kth instar passes into the K+1th. Setting K = INSTAR(I), we can write

if DAYDEG(I) DEGTHR(K)
then INSTAR(I) = INSTAR(I) + 1 ..... (2)

In this way, the duration of a given instar (at a mean temperature T) will be given by

IDUR(K) = (DEGTHR(K+1) - DEGTHR(K)) / T .. (3)

where IDUR(K) is the duration of the Kth instar in days.

The fundamental assumption of this approach is that the insects take a constant number of day-degrees for development irrespective of mean temperature. To test its validity we can compare the model output with real data presented by Sharaf Eldin (1970) for Myzus persicae (Sulz.). He measured the time taken in days for the animals to develop from birth to adulthood at five temperatures. Multiplying the time in days by the temperature in Centigrade gives us the total number of day-degrees in development. This number varies from 198 day-degrees at 10°C to 145 at 29°C; taking the mean over all temperatures (160) and using this in the model, so that development time is 160/T, we obtain good agreement between model and observed values (Fig. 75).

The course of development in Aphis fabae is as follows. Animals are born (i.e. larviposited) as active first instar larvae, produced within the mother by parthenogenesis. All births occurring on the bean plant are of females, and the larvae pass through four instars before becoming adult. The

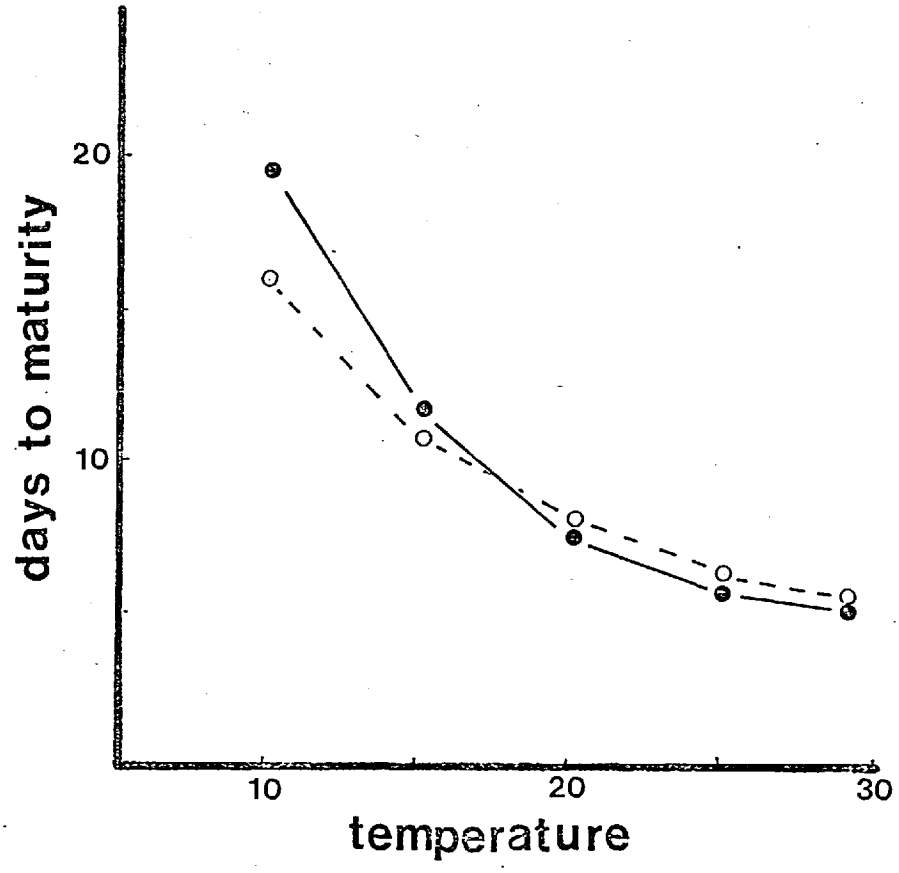


Figure 75. A comparison of the output of the aphid development sub-model (o) and data on a real population of Myzus persicae (⊙) given by Sharaf Eldin (1970)

omission of a dormant egg or pupal stage from the life cycle makes for very rapid potential population increase, and it can take as little as eight days to develop from birth to sexual maturity for this aphid at 20°C (Milne, 1971). Adult life extends over a period determined by air temperature, and each adult deposits larvae for most of her life (Kennedy and Stroyan, 1959; Dixon and Wratten, 1971; Way, 1968; Banks and Macaulay, 1964).

2 Growth and Feeding

Let us assume that individuals of Aphis fabae have a maximum dry weight, which they attain after the final moult under completely optimal conditions. This value might be of the order of 0.93 mg (Banks and Macaulay, 1964), and we can write it as W_{max}. Further, let us allow that this maximum weight is achieved during larval growth in a logistic form under ideal conditions; this pattern has been observed in Acyrthosiphon pisum (Murdie, 1965; his Fig. 30), and Aphis fabae (Banks and Macaulay, 1964; their Fig. 2). At any time before maximal weight is achieved we can threfore write the current weight, W_t by the difference equation

$$W_{t+1} = W_t \cdot e^{r\left[\frac{W_{max} - W_t}{W_{max}}\right]}$$

where r describes the maximum rate of weight increase.

When the environment is sub-optimal in some respect, we can allow that the rate of weight increase is reduced by defining a variable E to describe environmental clemency. This has the value 1 in an optimal environment, and 0 in an environment allowing no growth; if the environment is so poor

that the aphid actually loses weight, then E will become negative. We now put

$$W_{t+1} = W_t \cdot e^{r \cdot E\left[\frac{W_{max} - W_t}{W_{max}}\right]} \qquad \ldots\ldots (4)$$

and define E as a function of those factors tending to reduce growth (temperature, crowding, nutritional condition of the phloem sap, and so on).

$$E = f\ (TEMP,\ CROWD,\ FQUAL)$$

We can obtain graphs for the relative effects of the different factors from a number of sources. Murdie (1965) found that the adults of Acyrthosiphon pisum were smaller at high temperatures than at low, and we can graph this effect in Fig. 76. Similarly, Auclair (1963) and Banks and Macaulay (1964) have shown that aphid growth increases with plant nutritional quality, so using FQUAL (Chapter III) we can draw Fig. 77. The effects of aphid density (the number of animals per cm^2 within an aggregate or cluster of individuals) have been shown to affect the growth of Aphis fabae by Way and Banks (1967), from whose description Fig. 78 is drawn.

These three graphs define the effects of the different factors on aphid growth rate. We can synthesize the information they contain to give an estimate of the growth rate at any particular temperature, food quality and aphid density by interpolation from the three curves

$$\begin{aligned} TGF &= F\ (TEMP) \\ QGF &= F\ (FQUAL) \\ CGF &= F\ (DENSITY) \end{aligned} \qquad \ldots\ldots (5)$$

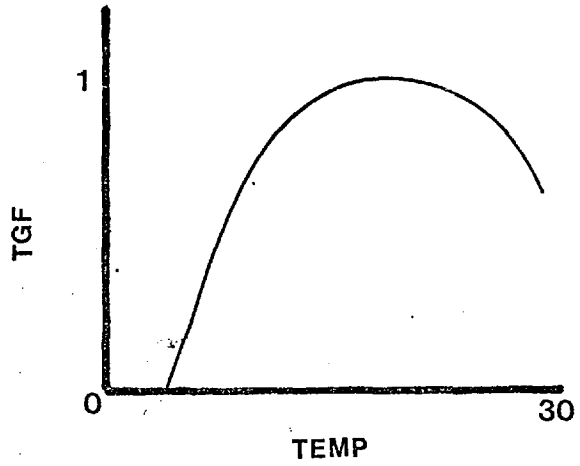


Figure 76. The relationship between air temperature and relative aphid growth rate

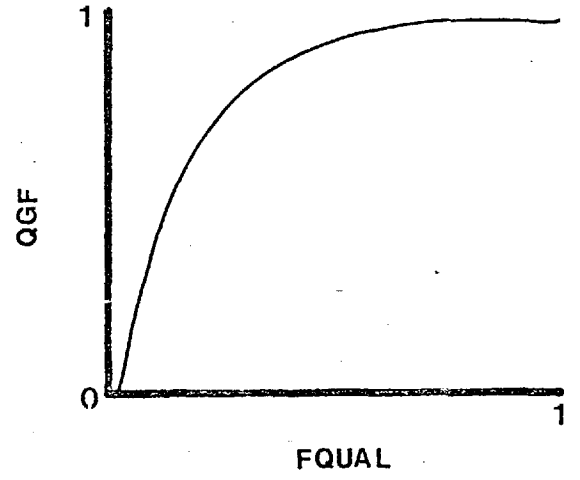


Figure 77. The relationship between plant food quality (e.g. nitrogen content) and relative growth rate

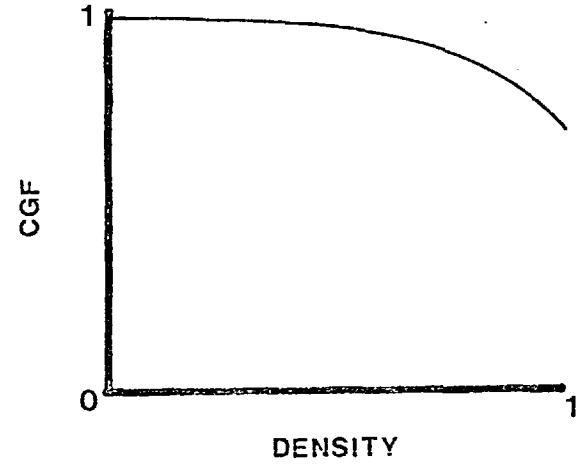


Figure 78. The relationship between aphid density and relative aphid growth rate

where TGF, QGF and CGF lie in the range 0 to 1. The actual growth rate depression can then be determined by assuming only the minimum factor to be important

$$E = \min (TGF, QGF, CGF) \quad \ldots\ldots (6)$$

or that the factors interact with one another (Chapter I)

$$E = TGF * QGF * CGF \quad \ldots\ldots (7)$$

Now let the dry weight of the APTERAE(I) be SIZE(I), and their maximum size be SIZEMAX(I); this will be determined by the weight of the aphid at birth (Murdie, 1965; see below). The proportion of unrealised growth, A, is therefore

$$A = \frac{SIZEMAX(I) - SIZE(I)}{SIZEMAX(I)} \quad \ldots\ldots (8)$$

We can now determine the maximum growth rate of the animals by fitting equation 4 to data for weight increase in optimal conditions (when E = 1); the expression for r has only one real positive root. This value is then supplied as GR, and with the calculated value of E we can express growth as

$$SIZE(I) = SIZE(I) * EXP(E * GR * A) \quad \ldots\ldots (9)$$

To compute the amount of sap removed from the phloem as food, let us assume that the aphid feeds at a rate which is determined by its size (Auclair, 1963; Banks and Macaulay, 1964), the water status of the plant tissue (Wearing, 1966, 1972), the air temperature (Mittler, 1962), the density of aphids in the aggregate (Way and Cammell, 1972), and the nutritional quality of the phloem sap (Auclair, 1963). As before, we can plot graphs of the relative effects of these factors as in Fig. 79. Then, by interpolation, the actual

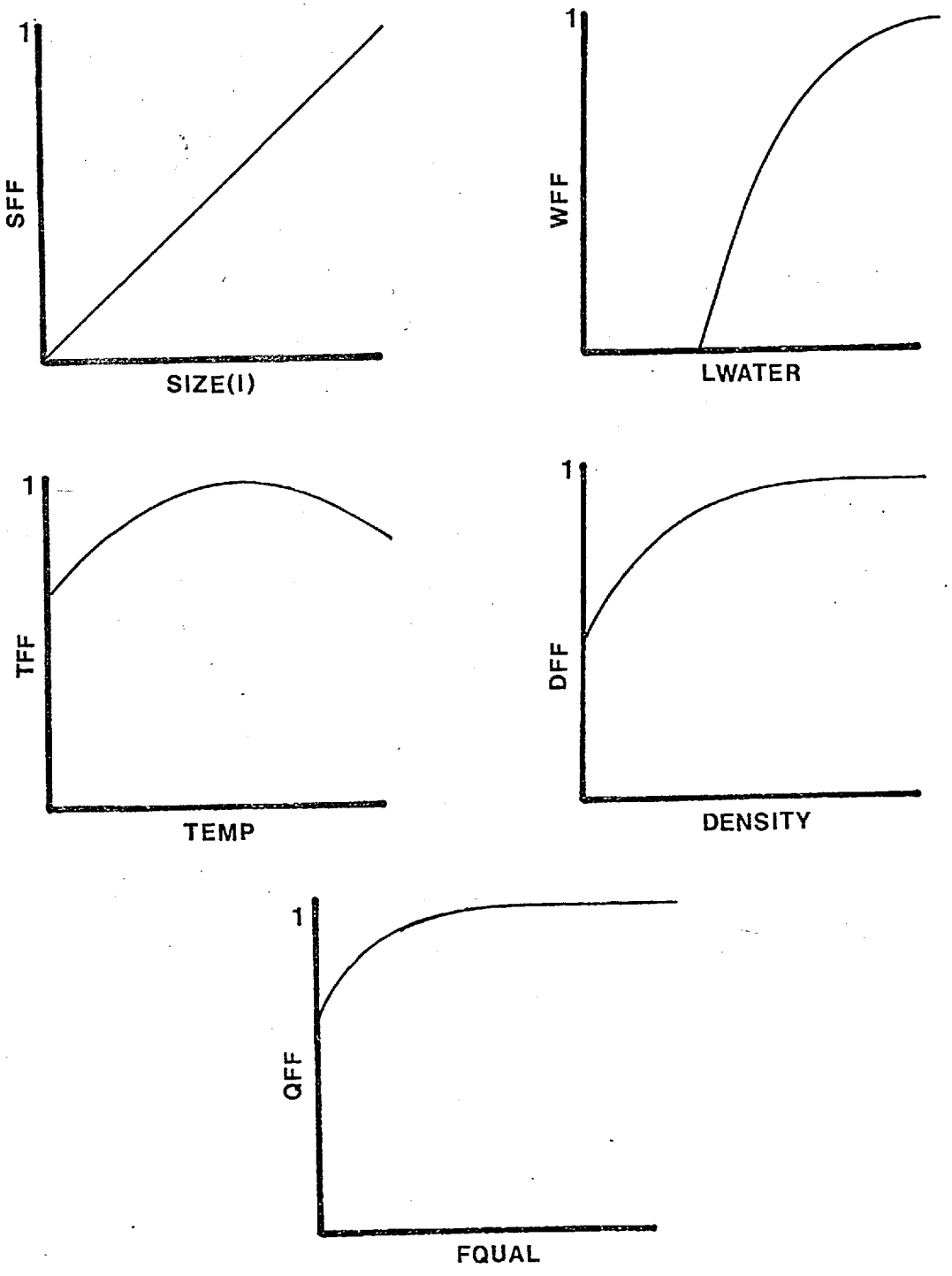


Figure 79. The factors which are assumed to affect aphid feeding rate in the model and their relative effects. Aphid dry weight (SIZE(I)), leaf water content (LWATER), temperature (TEMP), aphid density (DENSITY), and plant food quality, e.g. nitrogen content (FQUAL)

fraction of the maximum potential feeding rate can be determined for each age class of aphids in turn. If the suffix 'FF' represents 'feeding factor' we can write

$$SFF = F(SIZE(I))$$
$$WFF = F(LWATER)$$
$$TFF = F(TEMP)$$
$$DFF = F(DENSITY)$$
$$QFF = F(FQUAL)$$

for the relative effects of size, water status, temperature, density and food quality respectively. If FEEDMAX is the maximum possible feeding rate measured in grams dry weight of carbohydrate per aphid per day, we can set

$$E1 = SFF * WFF * TFF * DFF * QFF$$

or

$$E1 = \min(SFF, WFF, TFF, DFF, QFF) \quad .... (10)$$

and calculate the feeding by any age class of animals from

$$FEED(I) = APTERAE(I) * FEEDMAX * E1 \quad .... (11)$$

and therefore the total feeding by the population will be

$$APHFEED = \sum_{1}^{N} FEED(I) \quad .... (12)$$

APHFEED is then the measure of daily crop damage, and, summed over the period of infestation, will represent the total reserves lost to the plant. The effects of this removal were covered in Chapters II and III.

3 Birth and Immigration

The infestation is started by assuming an influx of alate adults into the crop at a specified time after germination.

The age of these adults, and the date upon which they arrive, can be varied at will. After the initial immigration, subsequent arrivals can be dealt with simply by adding the number of new aphids to the relevant age class of the established population.

Virginoparae of Aphis fabae reproduce parthenogenetically on the broad bean, giving birth to active first instar larvae (Kennedy and Stroyan, 1959). The number of larvae deposited per day by a single female is a function of her age (Dixon and Wratten, 1971), the air temperature (Kenten, 1955), the density of the population (Way, 1968), and her nutritional history (Banks, 1965). To simulate this process, let the vector FECMAX(I) be the maximum rate of larval deposition for a female of age I days. Then assume that the other factors act in such a way as to reduce the actual birth rate below this level; let PRTEMP be the proportion of the maximum which is possible at today's temperature (TEMP). Then

$$PRTEMP = F(TEMP) \qquad \text{Fig. 80}$$

Similarly, let there be a density-dependent effect, DDFEC, whose value is given by interpolation from Fig. 81;

$$DDFEC = F(DENSITY)$$

where DENSITY is the mean density of aphids in an aggregate.

Then the total number of larvae born on any given day will be

$$BORN = \sum_{MINAD}^{MAXAD} (APTERAE(I) * FECMAX(I) * PRTEMP * DDFEC) \quad (13)$$

where MINAD and MAXAD are the minimum and maximum ages of

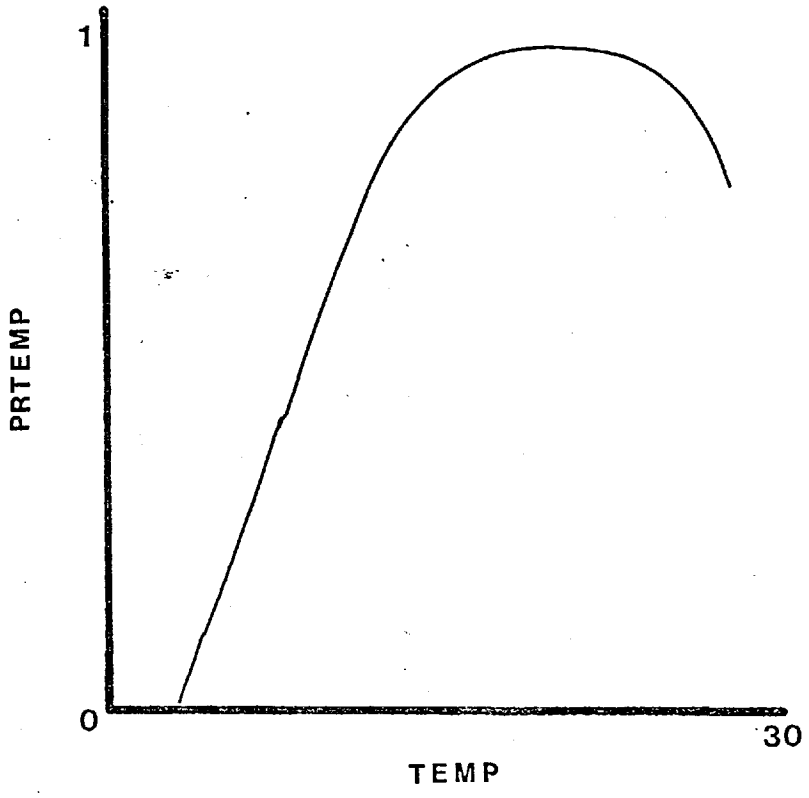


Figure 80. The relationship between air temperature and relative fecundity

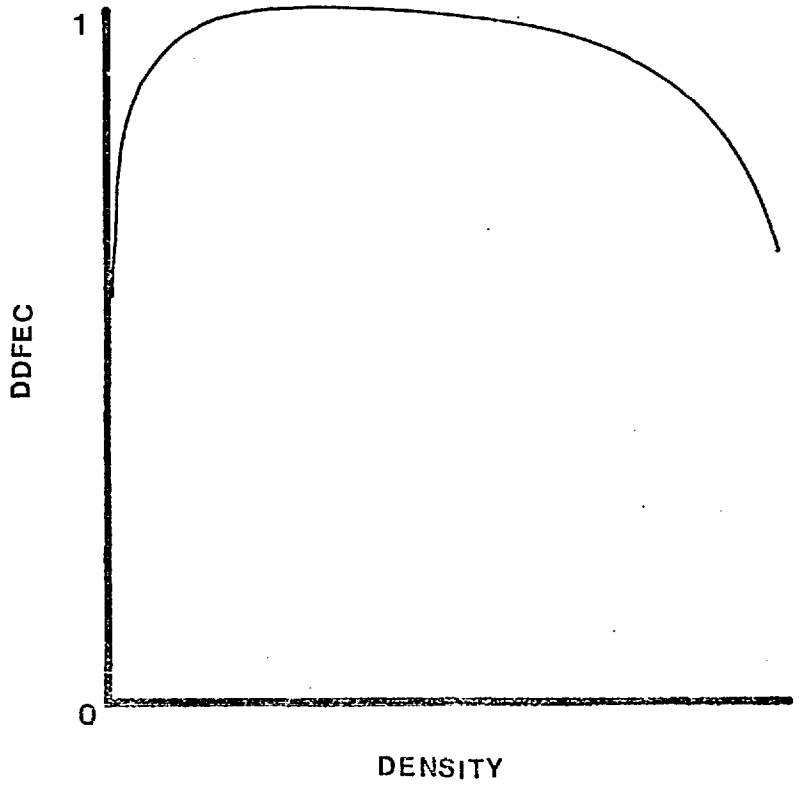


Figure 81. The relationship between aphid density and relative fecundity

adults in the population (since adults alone reproduce).

In Section 1 it was shown that development rate is a function of temperature and, this being the case, aphids will become adult (reach the fifth instar in the case of Aphis fabae) at different times after their birth, as determined by weather conditions. In order to take this into account in the model, it is necessary to store the age at which each age-class of aphids became adult in a vector IAGEAD(I). This is computed as follows;

> let K = INSTAR(I), the current instar of I-aged aphids
> then if DAYDEG(I) $\geq$ DEGTHR(K) let INSTAR(I) be incremented;
> and if INSTAR(I) now equals 5 then IAGEAD(I) = I

To determine the number of day classes which are adult, and therefore capable of reproduction, the model simply loops through I until INSTAR(I) = 5 (at which point, MINAD = I); the vector IAGEAD(I) is used within this loop to give a value for the maximum fecundity, since this is a function not of age since birth, I, but of age since becoming adult, I - IAGEAD(I). Now equation 13 becomes

$$\text{BORN} = \sum_{\text{MINAD}}^{\text{MAXAD}} (\text{APTERAE(I)} * \text{FECMAX(J)} * \text{PRTEMP} * \text{DDFEC}) \quad (14)$$

where J = I - IAGEAD(I). By this means we determine the births on day t; these animals will be APTERAE(1) on day t+1.

The problem now arises of determining the mean weight of these recruits. Their parents will have been of a broad range of sizes and ages, and the weight of the individual progeny varies both within and between families. The simplest approach, and that employed in the current model, is to assume that for

any given female, the weight of her progeny is an experimentally determined function of her bogy weight, and the air temperature. These effects are discussed by Murdie (1965) and by Banks and Macaulay (1964).

So let SIZEPRG(I) be the mean progeny size from aphids of SIZE(I) (see Fig. 82), giving

$$SIZEPRG(I) = F(SIZE(I)) * F(TEMP)$$

where the temperature function lies in the range 0 to 1, decreasing as temperature rises (Murdie, 1965). The proportion of recruits of this mean weight will be the proportion of births from adults of this age; i.e.

$$P = BORN(I) \Big/ \sum_{MINAD}^{MAXAD} BORN(J)$$

so the overall mean weight of the progeny from all adults will be

$$PRSIZE = \sum_{MINAD}^{MAXAD} (BORN(I)*SIZEPRG(I)) \Big/ \sum_{MINAD}^{MAXAD} BORN(I)$$

As stated earlier, the weight at birth determines the maximum potential weight at adulthood; we therefore write

$$SIZEMAX(1) = F(PRSIZE)$$

and determine the value by interpolation from Fig. 84.

4 Mortality and Emigration

Aphids die from a host of causes. Biological factors including predation by coleopteran, syrphid, neuropteran, and cecidomyid larvae, parasitism by numerous hymenoptera,

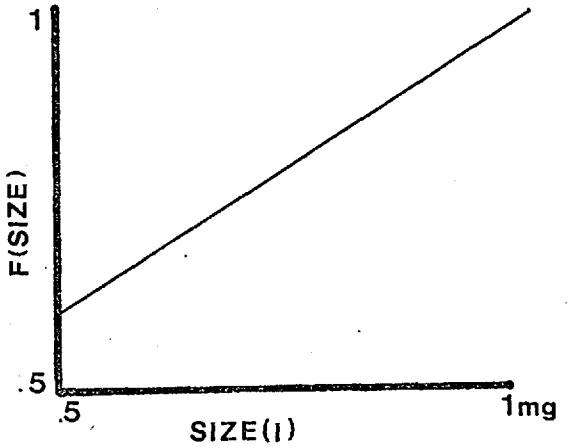


Figure 82. The relationship between the size of an adult aphid and the relative size of her progeny

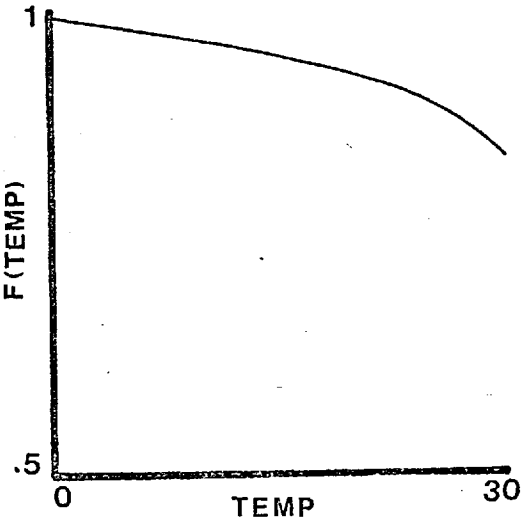


Figure 83. The relationship between air temperature and relative progeny size (after Murdie, 1965)

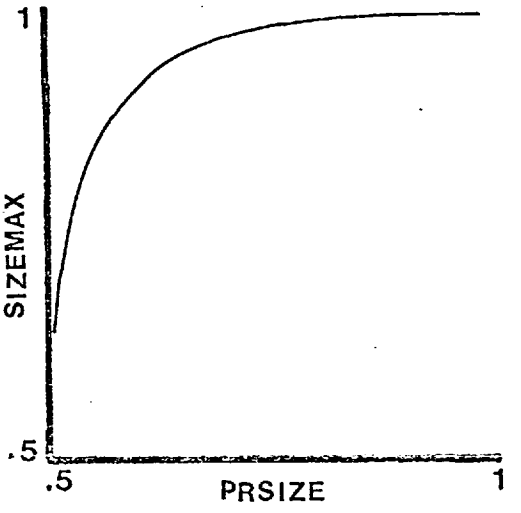


Figure 84. The relationship between relative progeny size and maximum potential adult size (SIZEMAX).

bacterial and virus diseases, and starvation can act in a density dependent fashion under certain circumstances (see Solomon, 1949), while abiotic (and typically density independent) mortality factors can eliminate a considerable fraction of the population through frost, high winds, heavy rain, and so on.

The practice in many models (e.g. Gilbert and Hughes, 1971) is to assume that these factors can be taken as contributing to a constant age-dependent survival rate ('background mortality'), while one particular agency of mortality is considered in more detail (parasitism in Gilbert and Hughes' model). This treatment overlooks the possibility that several of the biotic mortality factors interact with one another, so that, for example, as the rate of predation increases, the rate of parasitism might decrease (Flanders and Badgely, 1960). In the current model the abiotic factors are treated as a deterministic survival rate, while the modeller himself acts as the agency of all biotic factors by 'predating' from the population under different strategies.

If the age-specific survival rate is SURV(I), then

APTERAE(I) = APTERAE(I) * SURV(I) - 'predation' (16)

In other words, the survival rate is applied to the population before predatory loss is simulated. This is arbitrary, but it makes little difference if the treatment is consistent throughout. As an example, if the population is 100, and the survival and predation rates are .9 and .1 respectively, then predating first we have 100 * 0.1 = 10 individuals eaten, whereas applying the survival rate first gives us 100 * 0.9

= 90; 90 * 0.1 = 9 individuals predated. In both cases, of course, there are 81 survivors.

The factor which complicates the modelling of aphid populations is their polymorphism. The infestation is started by immigrant alatae whose progeny are generally apterous virginoparae. As the infestation continues, an increasing proportion of the aphids become alatae and fly off to find new host plants on becoming adult. Coupled with this, there is an increasing rate of apterous emigration in which the aphids walk down the stem and over the soil surface in search of fresh plants.

The great variety of factors which can induce an apterous mother to produce predominately alate progeny, and which can act to cause an aphid destined to become alate to revert to an apterous form (before the third instar), are reviewed by Lees (1966) and Hille Ris Lambers (1966).

Despite the large number of factors which have been suggested as affecting the rate of alate production, the overriding concensus is that 'crowding' of the aphids is of paramount importance (Awram, 1968; Kawada, 1964, 1965; Lees, 1961, 1966, 1967; Johnson, 1965, 1966a, b; Shaw, 1968; Toba et al., 1967; Way, 1968). It is frequently observed that the proportion of fourth instar larvae with wing pads increases with time as in Fig. 85. On becoming adult, a proportion of these alatae will leave the plant (Shaw, 1968), and the population is reduced in consequence. Also, the density of aphids in an aggregate (the number per cm^2) tends to decrease as the total number of animals in the aggregate increases (Way, 1968; and Fig. 86), so that the rate of increase in aggregate area exceeds the rate of increase in

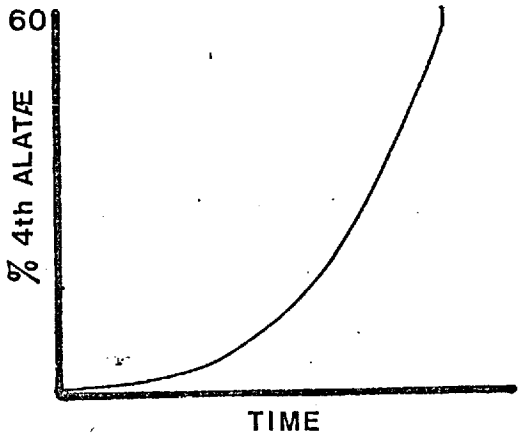


Figure 85. The observed pattern in the frequency of alate fourth instar aphids. This pattern is often attributed to the effects of aphid density.

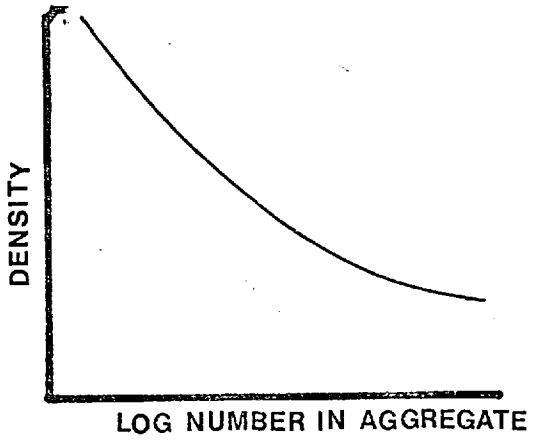


Figure 86. The density of aphids has been shown (Way, pers. comm.) to decrease in certain cases as the number of aphids increases. The aggregate becomes thinner on the ground as it ages, in other words

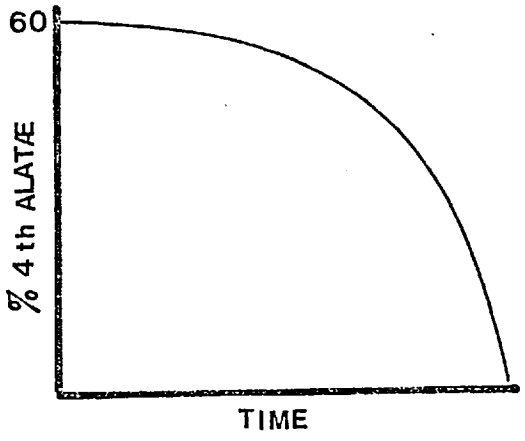


Figure 87. The consequence of assuming that % alate is a direct function of density. Density decreases through alate emigration and by the effect in Fig. 86, so each day there will be a lower rate of alate production. This is not observed in field or laboratory experiments

numbers. From these observations it would be incorrect to model the increase in the proportion of alatae as caused by increasing aphid density. Indeed, a model based on this assumption would show a trend as in Fig. 87; the opposite of the real situation. The alternative simple suggestion would be that more alatae were produced as density decreased, but this, of course, is not borne out by experimental results (Lees, 1966).

The process is clearly time-based, to a certain extent at least, since the proportion of alatae continues to increase even as the number of aphids per plant decreases (Figs. 85 and 86). It is likely, therefore, that some historical or cumulative effect is operating. This could be the result of a general run-down in the suitability of a particular area of the plant related to cumulative aphid presence; decrease in leaf turgor, increase in honey-dew, exuviae, inserted stylets and the like. This in turn might lead to an increase in the restlessness of each individual, causing the rate of encounter between aphids (and hence mutual stimulation) to increase.

The current model is based on this premise, and uses the total number of aphid-days experienced by the plant as the measure of the historical factor; this increases monotonically with time

$$ADAYS = \sum_{1}^{t} TOTAPH_i$$

The actual cause of alate production is then assumed to be the summation of all those cumulative and density-related factors subsumed into a single variable called FILTH. This is an

expression of the degree of exploitation per unit area of plant surface; in other words

$$FILTH = F\ (ADAYS\ /\ TOTAREA)$$

The variable FILTH tends to increase as the number of aphid-days experienced by the plant increases, and to decrease as the surface area of the plant increases by growth (TOTAREA). In order to mimic the effects observed in the field, and in many laboratory experiments (e.g. Way, 1968; Shaw, 1968) the variable FILTH must be related to the proportion of the population becoming alatae so that the observed trend (Fig. 85) is realized. Clearly there is a threshold value of FILTH below which very few alatae are produced (Chapter II), and, since the proportion of fourth instar alatae does not decrease once the population decline has begun, the function must be monotonically increasing. This is shown in Fig. 88.

In addition to the functionally important increases in the proportion of alatae in the population, there is a purely arithmetic reason for part of the effect. Since the wings only open when the aphid becomes adult, all losses to the population through flight emigration are of adults, and hence the rate of production of young by the whole population falls. This means that the younger age classes form a smaller proportion of the whole population and, therefore, that the fourth instar forms a larger part (see Fig. 89). This effect is further accentuated by the reduction in the size of the adult age group through emigration.

It is not completely clear from experimental work at which stage in the life history the alate morph is determined (Lees, 1966). There is evidence that crowded mothers produce

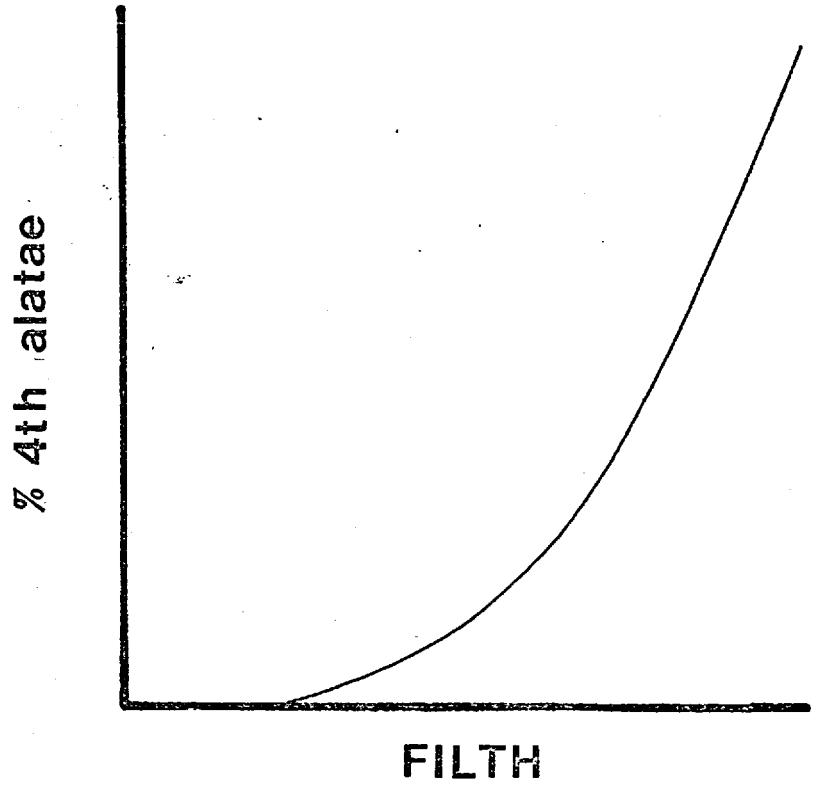


Figure 88. The relationship included in the model to predict the proportion of the population which will become alate. The derivation of the variable FILTH is explained in the text

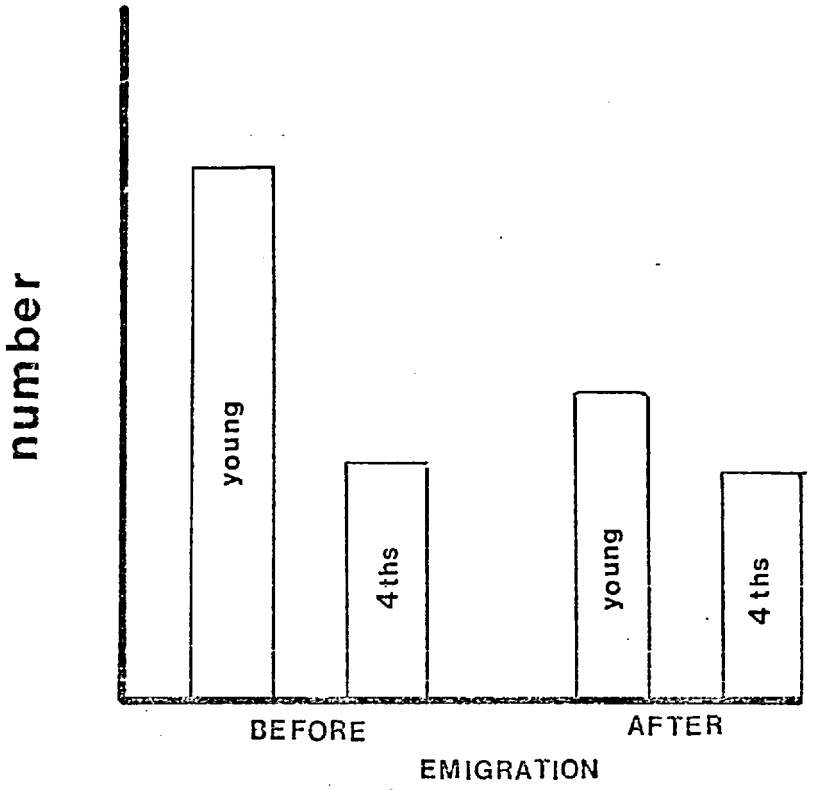


Figure 89. The reason why the fraction of the population as fourth instars increases after adult emigration; with fewer adults, fewer young are produced

alatae progeny (i.e. that the determination occurs before birth), but other evidence points to the ability of young, pre-third instar larvae to revert to the apterous form if the conditions in which they are reared are suitable (i.e. that determination is not absolute until the third instar). The sudden beneficial changes in the local environment which are necessary for this reversion are not likely to occur in the field, however, and so it will not be particularly important to the behaviour of the model whether one assumes wing morph determination in relation to the conditions experienced by the mother, or by her progeny at the second instar stage. There will be a time-lag introduced whose duration will depend upon temperature, reflecting the length of the first and second instars, but because FILTH is a continuously increasing function with time the effects of this lag on model behaviour will be negligible.

We let PROP2 represent the proportion of today's second instar aphids which tomorrow will be 'alate' third instars and write

$$PROP2 = F(FILTH)$$

and interpolate the value from Fig. 88. In order to accomodate the two morphs in the model we require an additional population vector to describe the number of alate-determined aphids of age I days, ALATAE(I). All the vectors associated with APTERAE(I) must be duplicated if the morphs differ significantly in any respect of their behaviour; they are known, for example, to develop at different rates under the same temperature conditions, and to give birth to different numbers and sizes of young (Banks and Macaulay, 1964).

The number of alatae determined on a given day is

$$ALES = AL(2) * PROP2$$

where AL(2) is the number of apterous second instar aphids. This number is then subtracted from the APTERAE(I) and added to the ALATAE(I). On becoming adult, the ALATAE(I) are capable of emigration by flight. Only a fraction of this group actually leave the plant completely, others staying in the vicinity and some remaining on the plant to feed and produce young (Shaw, 1968). The number of emigrants each day is taken to be those aphids who no longer affect the plant or the aphid aggregate; i.e. categories one and two above. This number, EMIGNO is calculated from

$$EMIGNO = \sum_{MINAD}^{MAXAD} (ALATAE(I) * FALEM(I))$$

where FALEM(I) would be an experimentally determined fraction of the adult population of age I leaving the plant in one day. Those ALATAE(I) which stay on the plant reproduce in the same fashion as the APTERAE(I) (equation 14), but a different fecundity vector is employed.

5 Parameter Updating

All the foregoing processes are simulated once every day. Before beginning the next day's calculations, however, the aphids, and the parameters associated with them must be updated. To 'age' the vectors we loop through their subscripts from the maximum age to the minimum, and make

$$parameter(I) = parameter(I-1)$$

much like the classes in a school are advanced at the end of each year. This procedure loses the maximum values (associated

with the aphids of maximum age), and these animals are assumed to die (or emigrate, or in some other way become unimportant to the future functioning of the system). Thus,

```
      DO 11 I = MAXAGE to 2 in steps of -1
      ALATAE(I) = ALATAE(I-1)
      APTERAE(I) = APTERAE(I-1)
      DAYDEG(I) = DAYDEG(I-1)
      IAGEAD(I) = IAGEAD(I-1)
      INSTAR(I) = INSTAR(I-1)
      SIZE(I) = SIZE(I-1)
   11 SIZEMAX(I) = SIZEMAX(I-1)
```

In this way all the first elements of the vectors are left vacant; these can now be initialized with constants; e.g.

```
INSTAR(1)  =  1
DAYDEG(1)  =  0
ALATAE(1)  =  0
```

or with values computed earlier in the model

```
APTERAE(1)  =  BORN
SIZE(1)  =  PRSIZE
SIZEMAX(1)  =  F (SIZE(1))
```

It only remains now to compute a number of totals;

```
TOTAPH  =    (ALATAE(I) + APTERAE(I))    population
K  =  INSTAR(I)
AL(K)  =    APTERAE(I)                   instar (apterae)
ALA(K)  =    ALATAE(I)                   instar (alatae)
TOTAREA  =  ALEAF + ASTEM                plant area
DENSITY  =  TOTAPH / TOTAREA             aphids cm^-2
```

Any output figures required at the end of each day are printed at this stage; food consumed, aphid days experienced by the plant, population age structure and so on.

Output from the Model

1 Temperature Effects

Temperature has been included in the model structure as affecting the rate of development, the birth rate, individual growth rate and feeding rate of the aphids. Its effects on the output of numbers and total food removed are therefore likely to be complex. Using a standard set of survival rates (95% per day), and density independent fecundity values (from Banks and Macaulay, 1964) the model was run for 100 days at 5, 10, 15, 20 and 25°C. The number of aphid-days and the total food removed from the plant are plotted in Fig. 90.

At some temperature higher than 25°C the numbers and feeding would decrease, but the model has used data only up to this temperature, because it is unlikely that higher field temperatures would be encountered in Great Britain. The effect of temperature on fecundity is shown in Fig. 91, and the development rates are as follows:

Instar	I	II	III	IV
Day degrees from birth to completion of instar.	40	80	120	160

In other words, at a temperature of 20°C, each instar would last two days.

By plotting the average food per aphid-day against temperature an interesting hypothesis is suggested (Fig. 92), namely that at low temperatures the cost-effectiveness of pest control measures is higher than at high temperatures.

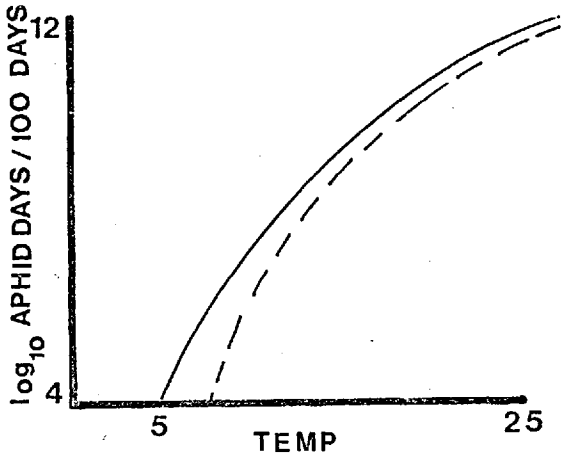


Figure 90. The number of aphid-days ( ) and the weight of plant tissue eaten ( ) at different temperatures

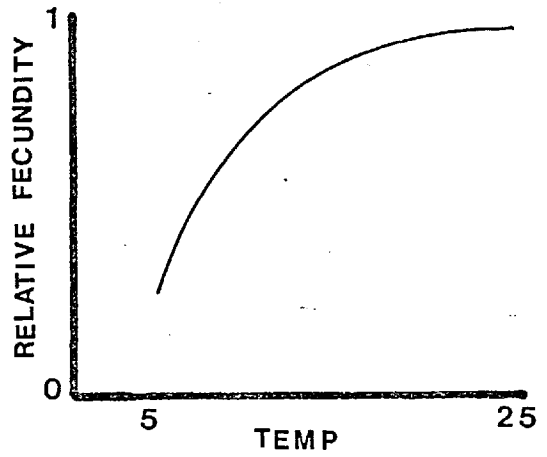


Figure 91. The effect of temperature on relative fecundity included in the model

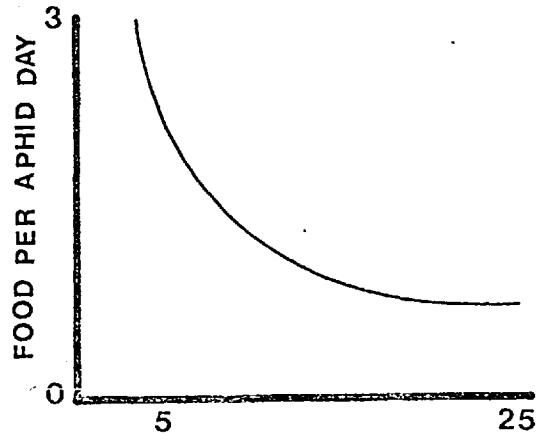


Figure 92. The mean weight of plant tissue eaten per aphid-day at a range of temperature. For explanation see text

This effect will tend to be of limited importance in the field, however, where temperatures will generally exceed 12°C.

The model produces this result because of the relationships between temperature and body weight, and body weight and feeding rate. Murdie (1965) suggests that body size of adult Acyrthosiphon pisum decreases with rising temperature, and that feeding rate is an increasing function of body size. At low temperatures, therefore, body weight and feeding rate are both relatively high, so the death of one aphid is more effective in reducing the rate of damage to the plant. In fact, it may be that as temperature increases, the feeding rate per unit body weight increases, and that this compensates for the effect observed in the model. The observed increase in damage with temperature is due to the rise in the number of feeding animals, brought about by increased birth and development rates.

For the purposes of comparison with later tests, the model was run under standard conditions (i.e. at 20°C with our manipulation (predation) absent). The pattern of numerical change is shown in Fig. 93, with aphid numbers increasing to a peak and then declining quite rapidly as alatae are produced and emigrate from the system. Fig. 94 plots the age structure of the population up to the peak in numbers; just as the age structure is about to stabilize, large numbers of adults fly off, and the number of births is reduced, so the age structure begins to destabilize again.

2 Density Dependent Effects

Way (1968) and Way and Cammell (1970) have demonstrated the importance of a number of density-related processes

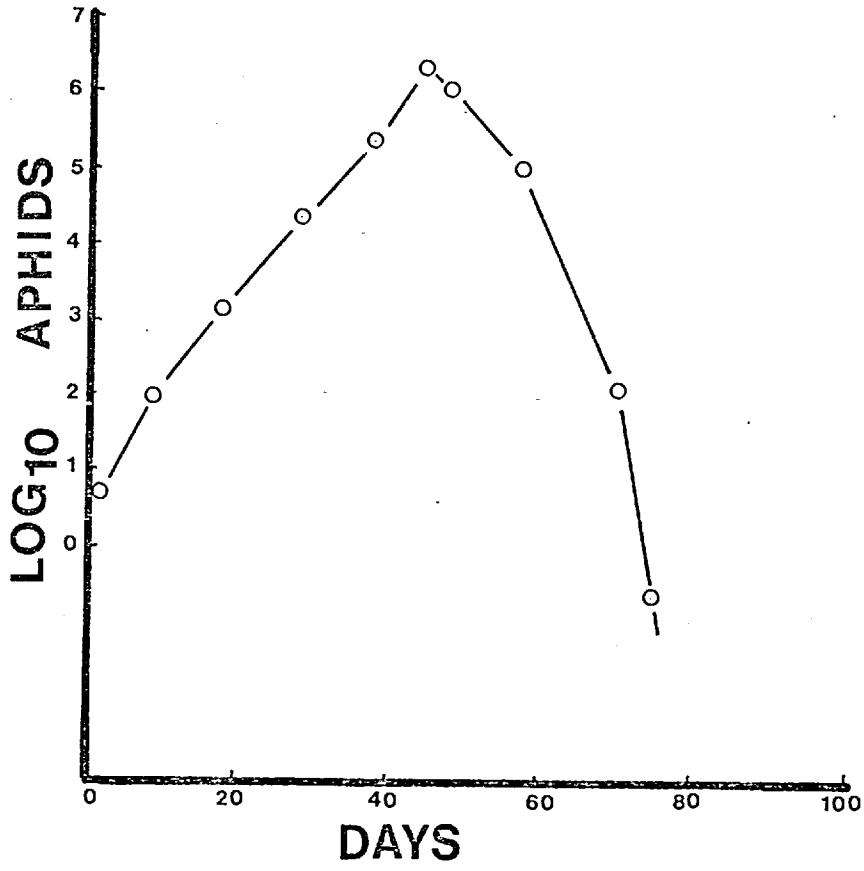


Figure 93. The time pattern of aphid abundance in a control run.

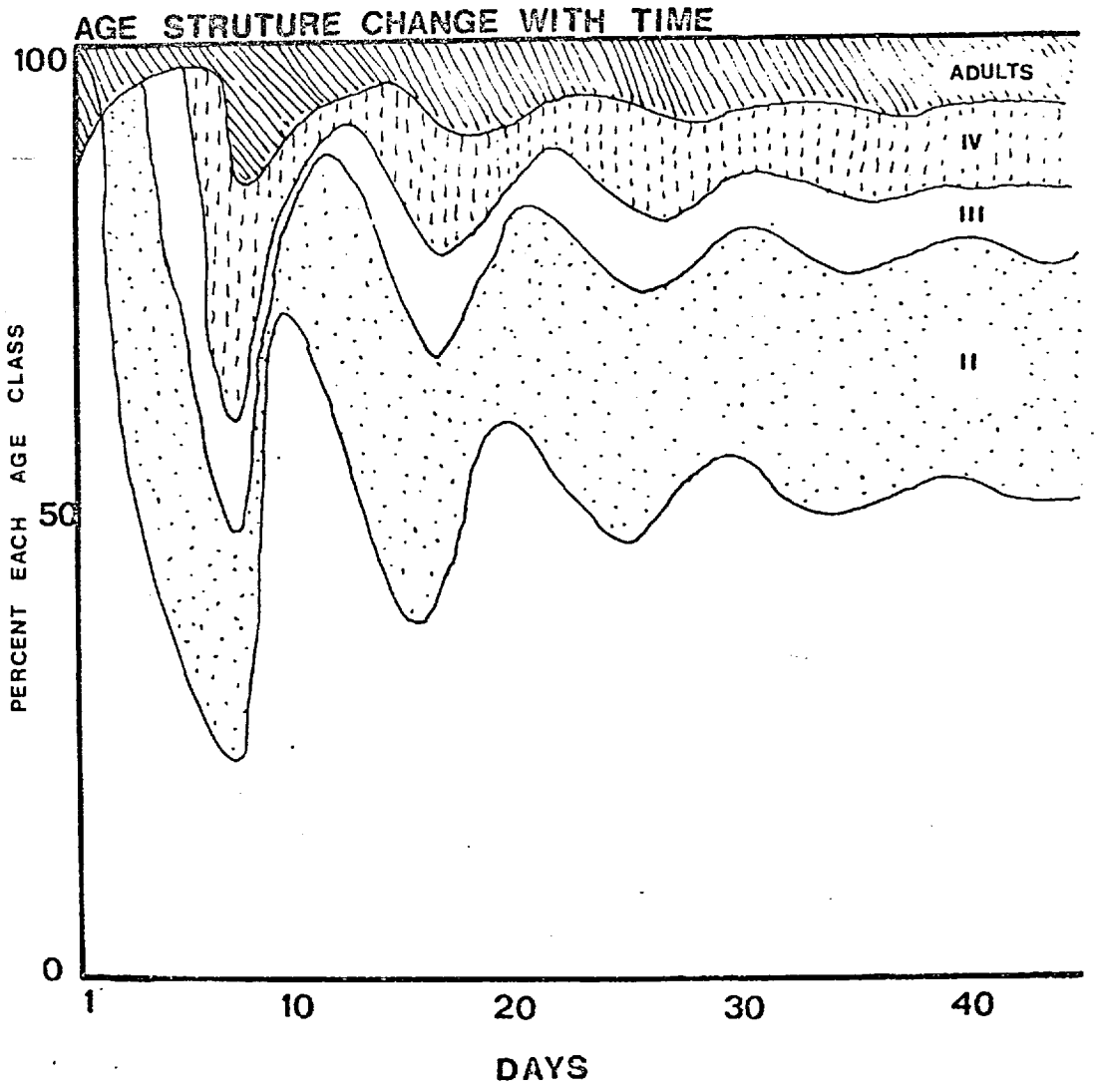


Figure 94. Changes in the age-structure of the aphid population up to peak numbers. The infestation began on day 0 with 100% adults, and fourth instars do not appear at 20°C until day 8. By day 40 the percentage of the population in each instar is almost constant.

in affecting the dynamics of aphid population change. Principal amongst these in the host-plant-alternating aphids is the density affected emigration rate of apterae and alatae. As shown above (Mortality and Emigration) the interpretation of this as being a density effect is not strictly correct, because as the proportion of emigrants increases, the density decreases.

Density dependence in its strict usage (Solomon, 1949) can be applied in the model to both the survival and fecundity vectors, and we can observe its effects without necessarily specifying the mechanisms by which it operates. For example, let the maximum number of aphids which it is possible to confine to the habitat unit be 5000. This number is arbitrary, and simply sets the level of population density at which the density dependent effects are at their maximal intensity. Now consider a parameter DD which behaves in such a way that at any DENSITY we have

$$DD = F(DENSITY)$$

The shape of the function is defined so that F(0) = 1 and F(5000) = the maximum degree of density dependence we wish to test. If we want to look at the effects of density on fecundity, and the minimum fecundity we expect is 40% of the maximum, then we set F(5000) = 0.4. Fig. 95 shows this effect; we can now say that the "percentage density dependence" is

$$DD\% = (1 - 0.4)\ 100 = 60\%$$

and calculate the actual reduction at any DENSITY from

$$DD = 1 - DENSITY * 0.4 / 5000$$

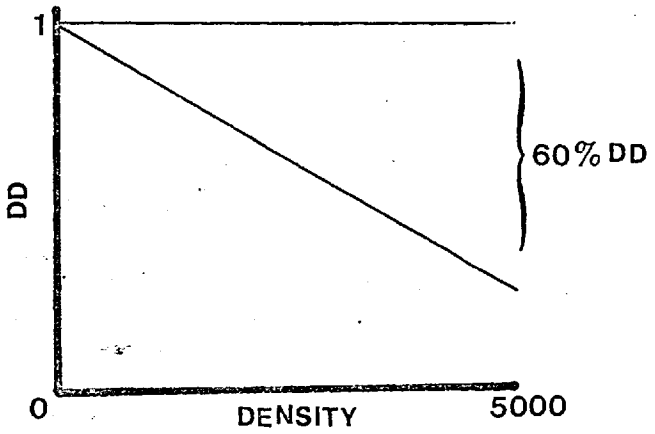


Figure 95. A simple means of representing the intensity of a density dependent factor. If DD is reduced to 0.4 at some maximum aphid density we say that the intensity of density dependence is 60%

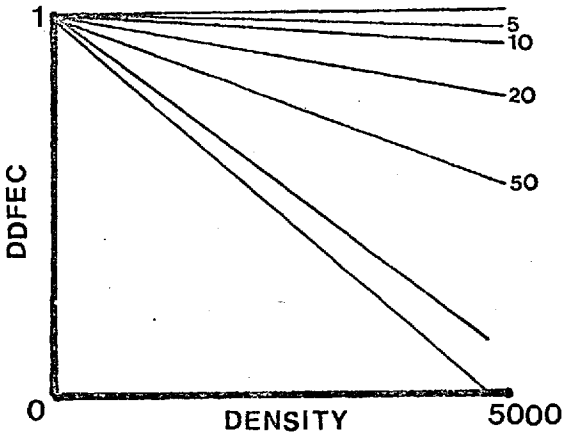


Figure 96. The effects of aphid density on relative fecundity (DDFEC) at different intensities of density dependence

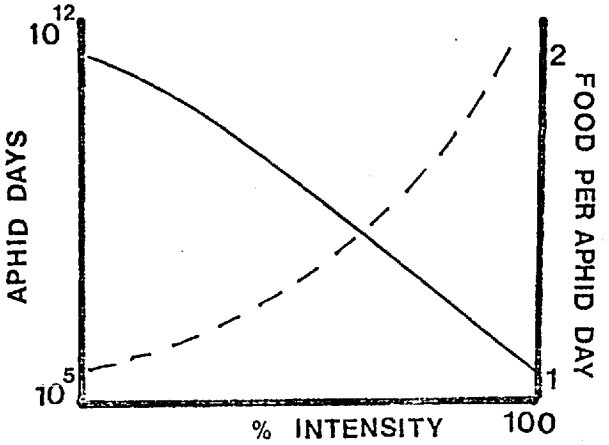


Figure 97. The number of aphid days experienced by the plant, and the mean weight of food removed per aphid-day plotted against the intensity of density dependence in fecundity

a) Density Dependent Birth Rate

The model has been run with maximal density dependence in birth rate set at 0, 5, 20, 50, 80 and 100%; the relationships between DDFEC and DENSITY are therefore as shown in Fig. 96. On any given day, DDFEC is the proportion of potential births realized, so that

BORN = BORN * DDFEC

Now, graphing aphid-days after 100 days and total food removed after 100 days against the intensity of density dependence we obtain Fig. 97; the reduction in aphid days and feeding can be seen to be more or less exponential with increasing intensity of density dependence, though mean food per aphid day increases with intensity, because mean aphid age is higher when the birth rate is reduced.

Way (1968) has shown that in Aphis fabae there is a curvilinear relationship between density and reproductive rate, so that at very low densities there is a depression in the birth rate as well as at very high densities. The importance of this effect in the model (Fig. 98) depends upon the proportion of the infestation during which the birth rate is inhibited by DENSITY being too low; after long periods, the population largely compensates for this effect, and the model behaves as if the monotonically decreasing curve had been employed.

b) Density Dependent Survival

Density dependent survival is assumed to act in addition to density independent survival. Each element in the basal age specific survival vectors APTSURV(I) and ALASURV(I) is multiplied by DDSURV which is computed from the current

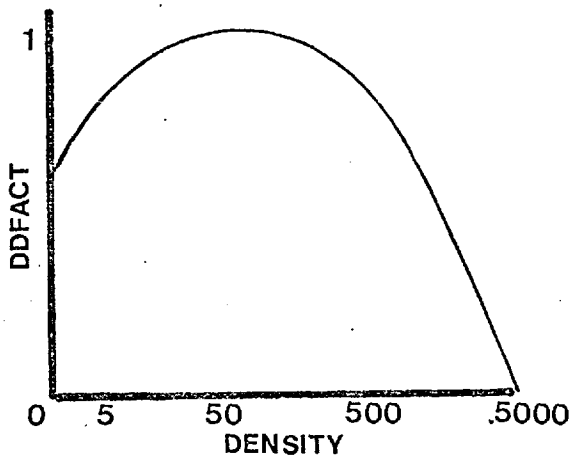


Figure 98. The relationship between relative fecundity (DDFACT) and aphid density (after Way, 1968)

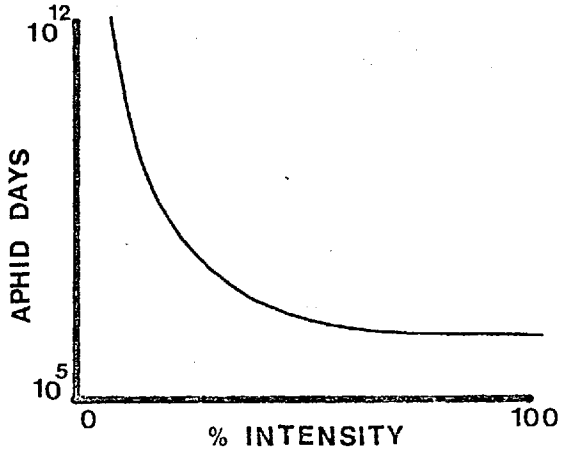


Figure 99. The effects of the intensity of density dependent survival on the damage experienced by the plant (aphid days)

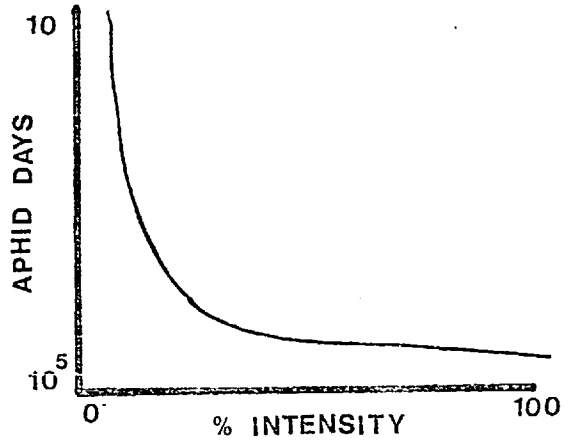


Figure 100. The effects of the intensity of density dependent survival and fecundity on plant damage. Each point represents the action of both factors at the same intensity (e.g. 20% survival reduction and 20% fecundity reduction)

density level by interpolation. A family of intensity curves similar to those used in the birth rate analysis (Fig. 96) were employed to investigate the effects of changes in the survival rate on numbers and feeding (Fig. 99).

It can be seen that as the intensity of density dependence increases, the population history approaches a limit asymptotically. The level at which the number of aphid-days per hundred days becomes constant (i.e. independent of changes in the intensity of density dependence) is set by the ceiling which is placed on density. If, therefore, the population were constant and maximal for the full period, we would have

$$5000 * 100 = 5.0\ 10^{5} \text{ aphid days}$$

That the observed asymptote is lower than this level reflects the fact that the aphid population was started by only two females on day 1.

This apparent population regulation, however, is only approached when very intense levels of density dependence are assumed; levels which would be unlikely in most field situations. For example, the third point on the graph (the first to show a really significant reduction) requires that 20% of the population is killed each day when aphid abundance is near the limit; in other words, the regulation would be very violent at density dependent survival intensities of this order. In addition, it becomes increasingly difficult to postulate mechanisms which might work with the intensities modelled (see Chapter V).

This is not to say that the stability (in the sense of relative numerical constancy) of the population is unaffected by low intensities of density dependence. On the contrary, any immediate response to density will slow down the rate

of increase or decrease in numbers; the stability achieved, however, will increase as the intensity of density dependence increases (unless, of course, survival rate increases with density in which case the opposite is true). In terms of pest control we are concerned to minimise the integral of the population curve, and not necessarily to increase the stability of pest numbers. In practice, of course, the two aims are generally quite closely correlated because the probability of pest outbreak increases with the amplitude of population fluctuations.

From a comparison of Figs. 97 and 99 it is clear that the conditions built into the model are sufficient to back up the intuitive suggestion that density dependence of survival rate is more effective in bringing about a reduction in numbers and feeding than a similar effect on fecundity. This is simply due to the fact that fecundity is only a property of adult animals, whereas survival rates apply to all ages. At any population level, a given change in the intensity of density dependent mortality will bring about a greater proportional change in abundance than will the same change in birth rate.

In Fig. 100 we plot the output of a set of runs in which both birth rate and survival are affected by population density. The intensities are the same as before, but each point represents the action of two density dependent effects of the same intensity (e.g. 20% survival and 20% birth rate). There is rather little difference between this combination and the effects of survival alone (Fig. 99). This is because reduced survival enhances birth rate to a greater extent than reduced birth rate increases survival.

Both factors are powerful agencies in reducing the

enormous potential rate of increase of the model population, and there is some evidence that they operate in real aphid populations (Way, 1968; Way and Cammell, 1970). It is possible, however, to cause a model of this type to behave in almost any way desired, by a judicious manipulation of these two density dependent processes. Until reliable data become available it is probably better to investigate the behaviour of the model under, at most, rather weak density dependent influences, since this allows us to interpret the output in terms of the tests applied, rather than having it over-ridden by the effects deriving from the shape of the density dependent relationships we happen to have included. In subsequent runs, both density dependent effects are assumed to act at a maximum 5% intensity (i.e. weakly), taking survival as linear, and birth rate as curvilinear functions of density (after Way, 1968).

3 The Effects of Predation

The purpose of this set of runs is to determine the pattern of predation to which the model population is most responsive. Three parameters are of particular importance in this context; the predation rate (the proportion of the prey population eaten on a given day), the pattern of change in the predation rate (reflecting numerical changes in the predator population), and the preference of predators of different sizes for aphids of different sizes. If there is an optimal combination of these parameters in terms of the reduction in food removal from the plant, then it will be interesting to see the extent to which real predator populations approach such a strategy (Chapter V).

a) Predation Rate

Consider the situation in which predation rate is constant with time, equivalent to the aphid being prey to a very common, widely polyphagous predator, whose alternate prey species have a constant relative abundance. At low rates of predation (in the order of 10% per day) the predator has little influence on the damage experienced by the plant (Fig. 101) even though, after a time, large numbers of aphids are eaten (Fig. 102). In contrast, high predation rates (90% per day) will reduce damage considerably and relatively few aphids are eaten over an extended period (because the reproductive stock is so diminished, the high predation rate only amounts to the removal of a few individuals).

In terms of biological control, this simply tells us what we knew already, namely that the higher the predation rate, the better our damage control. The model also shows (though rather incidentally) how we can harvest the maximum biomass of aphids from the system, by predating at a rate of between 1 and 10% (Fig. 102).

One of the principal criticisms which can be levelled against this model of predation is that it assumes homogeneity in the treatment of prey by the predator; prey selection has been ignored.

b) Preference

In these runs I assume a constant rate of predation, but, to highlight the effects of age and size preference, apply this predation to only one of the five instars of the aphid life cycle. In this extreme situation, where four of the five instars escape predation, the effects of prey selection should be exagerated, but not qualitatively distorted, pictures of the

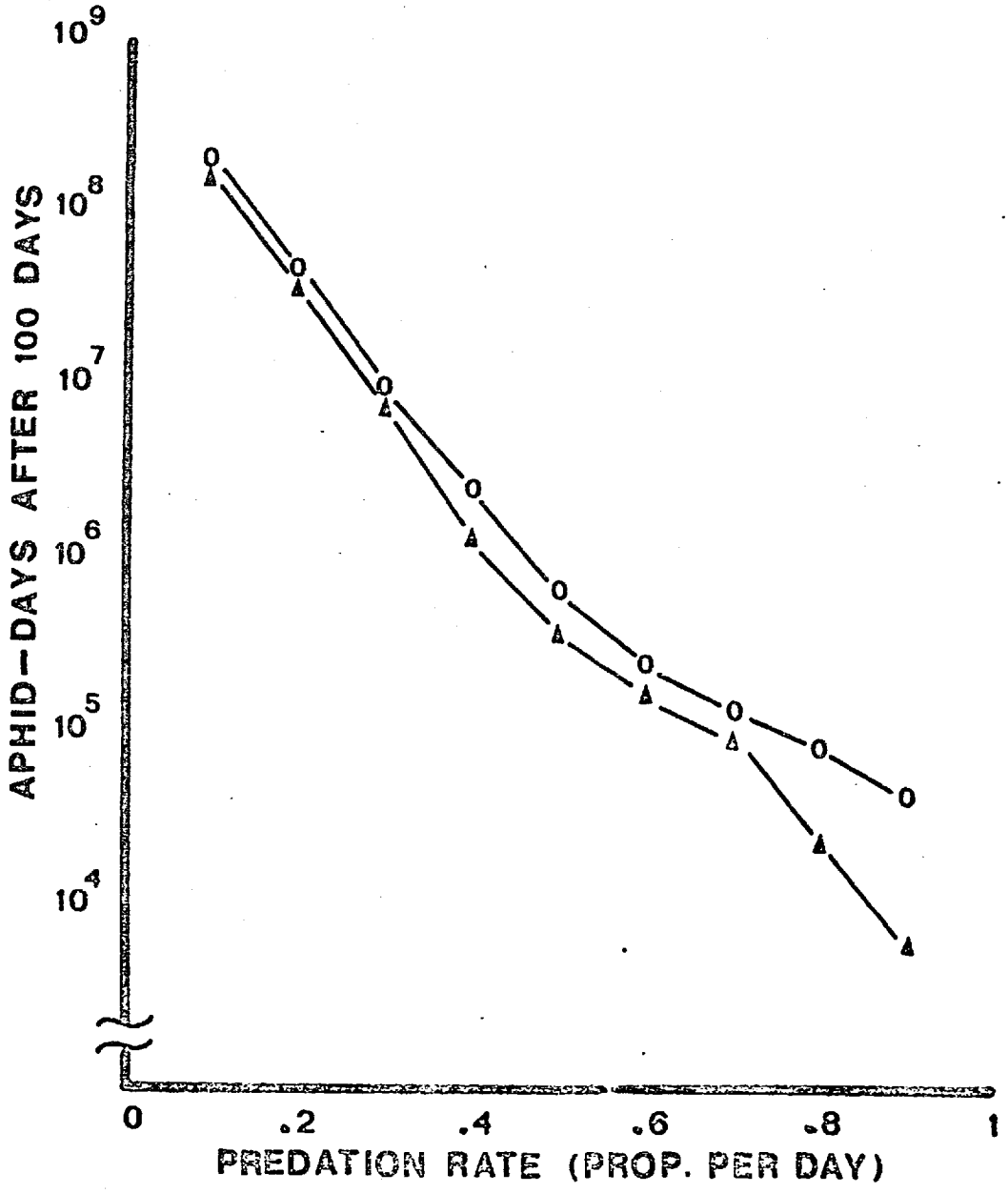


Figure 101. The relationship between the predation rate (percent of the prey population killed per day), and the damage experienced by the host plant (total aphid-days). The graph distinguishes the effects of selecting first instar animals (▲) and adults (O) on the outcome.

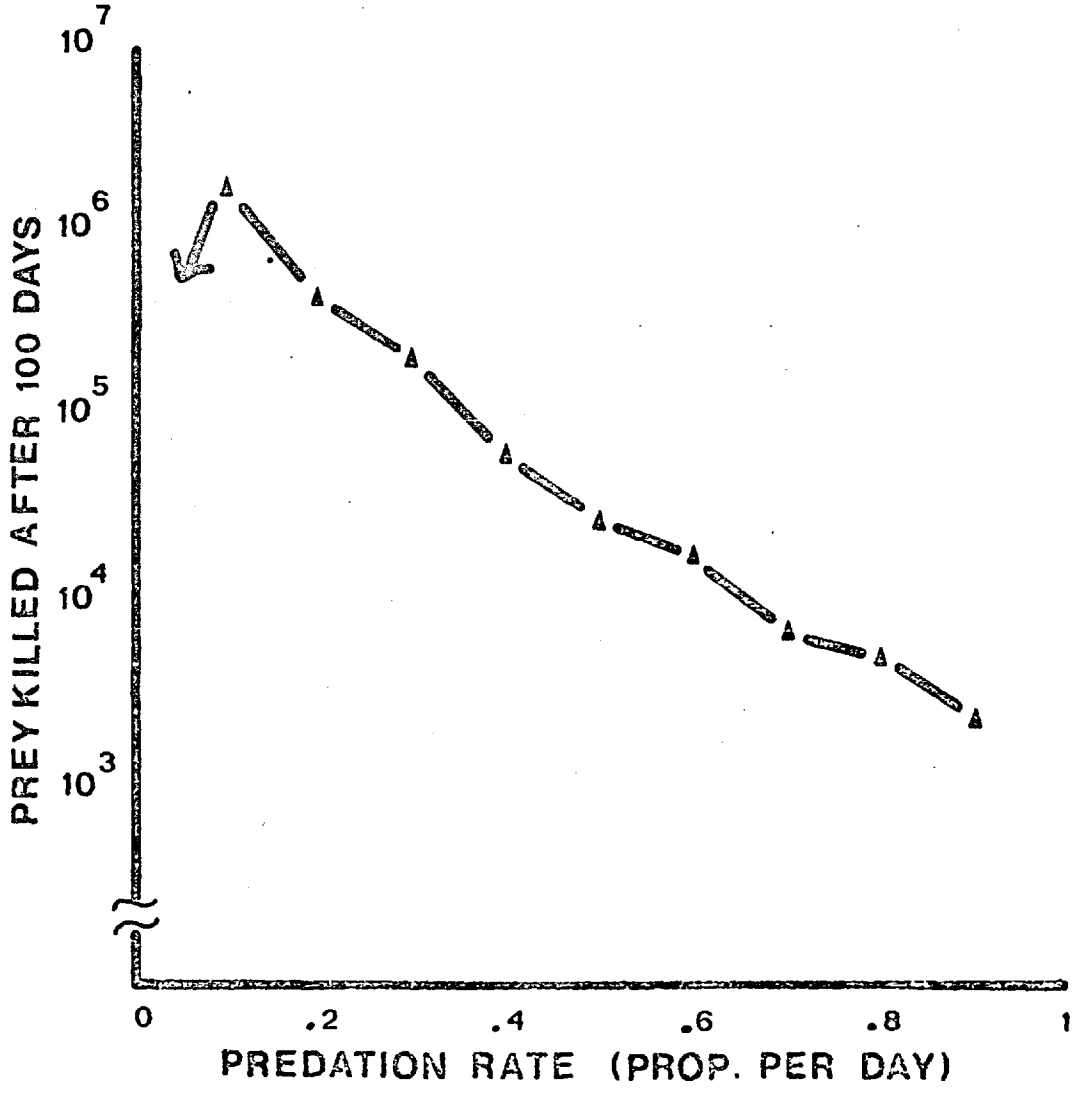


Figure 102. The relationship between predation rate and the number of aphids killed after 100 days. The curve clearly must pass through the origin (no predation; no kill). The total kill decreases as the predation rate increases because the reproductive stock is so diminished that there are fewer aphids from which to select.

more realistic case in which preference is a continuous, less rigid function of size (Fig. 103). All predators are assumed to have similar preferences, irrespective of their size or age.

Figure 101 shows the difference between selecting only adults, or only first instar larvae on the number of aphid days experienced by the plant. It is clear that selecting first instars is advantageous to the plant at all predation rates, and that the difference between the two treatments increases with the predation rate. This is not intuitively obvious, and one might expect that in selecting younger aphids the predator is wasting a certain amount of effort on animals which would have died before reaching maturity through other factors. This wastage of effort applies, of course, to our biological control, and not to the predators own survival strategy.

The reason for this counter-intuitive result stems from our predating by <u>proportion</u> and not by actual number killed; in these runs the food demand of the predator is not fixed in any way, and the number of aphids eaten is determined comletely by aphid population size. In retrospect, it is clear that under such a strategy the most abundant age class (in this population, the first instar) will bring about the greatest population reduction when predated. This aspect is detailed in the next section.

It is of interest to examine the changes in the efficiency of predation which might occur as a result of selective feeding. We can define the efficiency of a biological control predator in terms of the reduction in plant damage per gram of aphid consumed. Fig. 104 shows that this efficiency decreases as larger aphids are selected, and as the predation

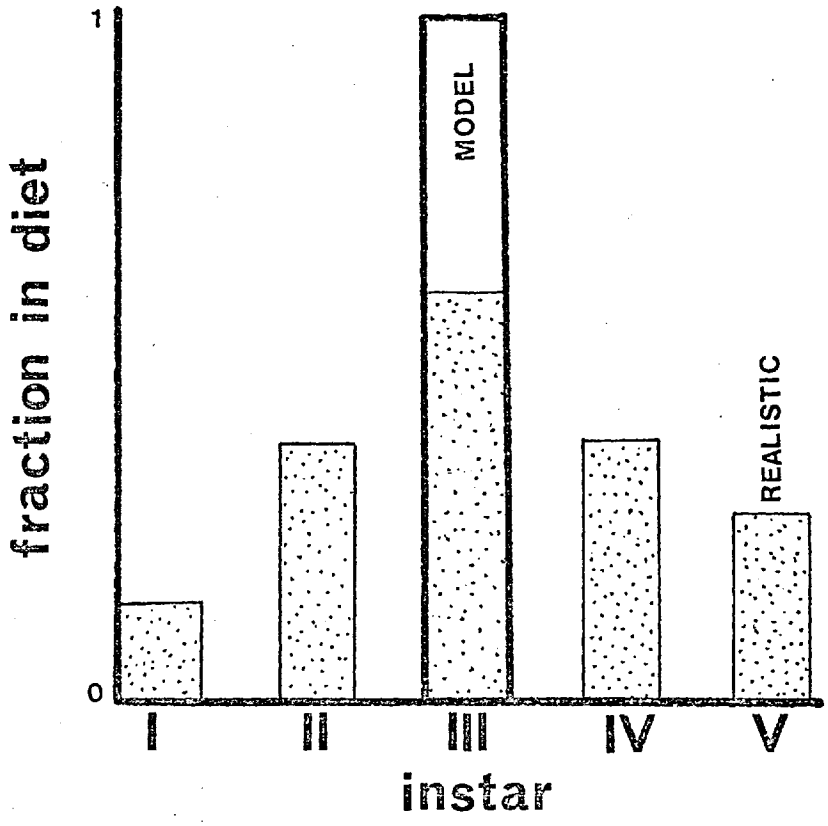


Figure 103. The preference of a predator for different aphid instars in the model (open bar) and realistic situations (stippled bar)

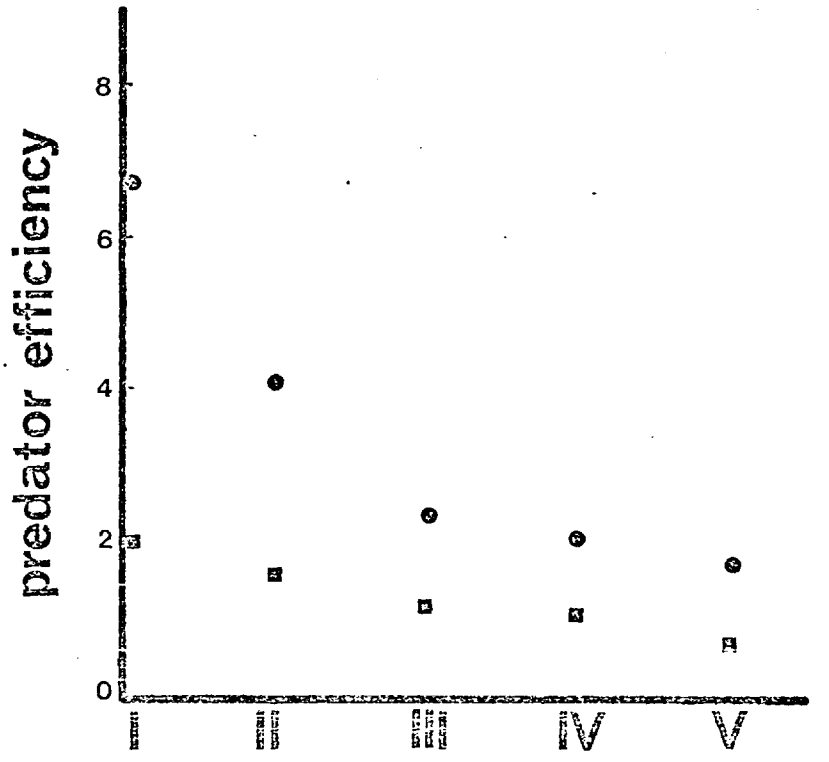


Figure 104. Predator efficiency (see text) when selecting different instars. Predation rate 20% (⊖) and 90% (⊟)

rate decreases. This finding also holds only for proportional predation as we shall see.

4 Numerical Predation

To simulate predation as the removal of a set number of individuals from the model population involves a number of problems. First, we must specify a temporal pattern of predation, which reflects changes in predator abundance and voracity. This may or may not be related to aphid abundance (below), depending upon whether the predator is relatively narrow or relatively catholic in its selection of food species, and whether it is highly mobile and has the ability to seek out prey in new areas when its current food source is depleted.

Second, having decided upon the number (or weight) of aphids to be taken from the population on a given day, one must apportion this predation between age (or size) classes. This can be done either by assuming that the predator exhibits no selective ability, and eats aphid age classes in the proportion in which they occur in the population, or by assuming some degree of selective behaviour, and weighting the proportion taken from any class of the population accordingly.

a) Pattern of Predation

For patterns of predation intensity are examined here; these are constant, exponential, logistic, and density-dependent (Fig. 105).

The conditions under which a constant number of individuals are eaten each day are rather difficult to conceive in a natural setting, but a monophagous predator with a long generation time, and long-lived individuals could approximate

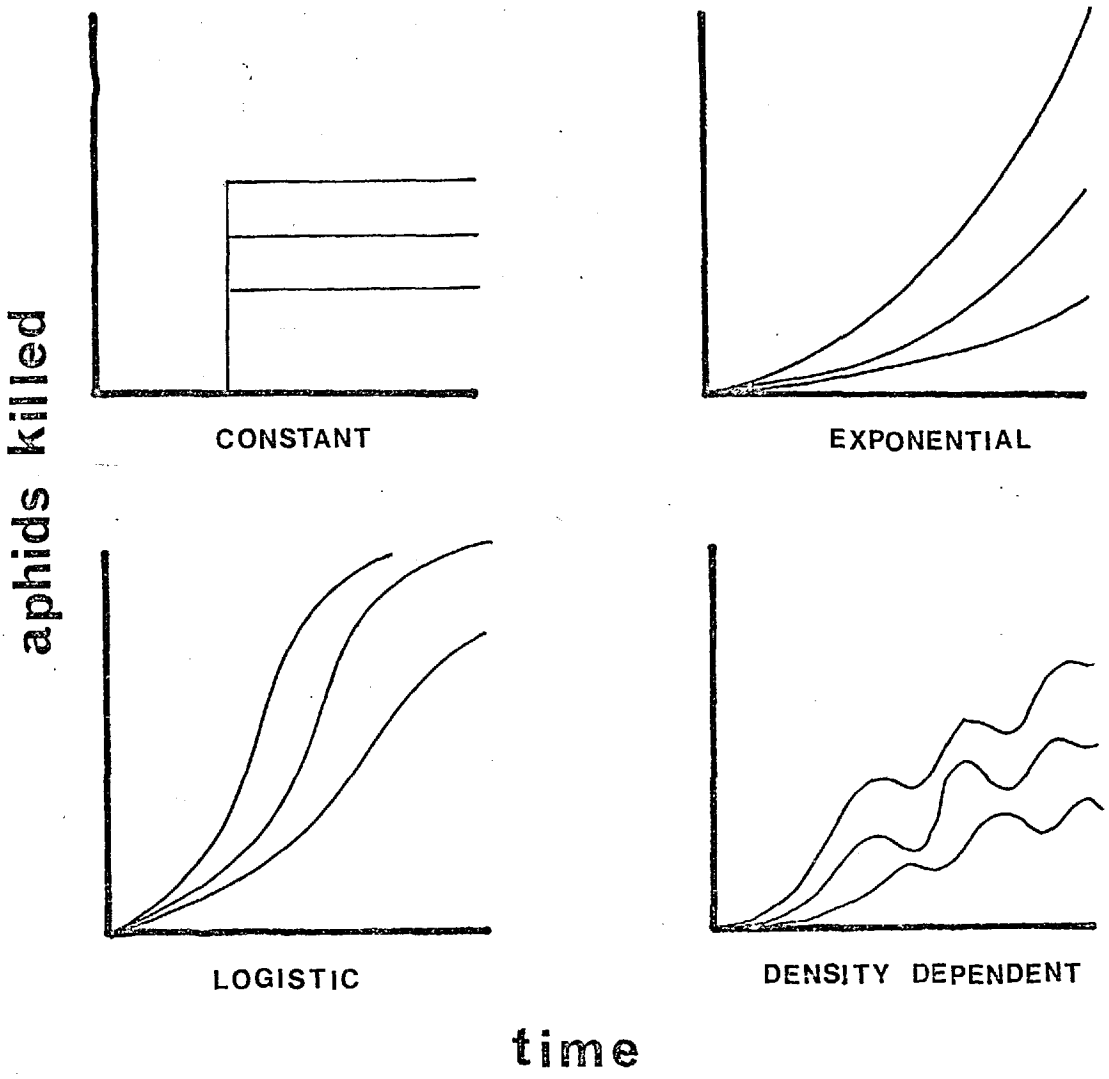


Figure 105. Four patterns of numerical predation tested in the model. Each pattern is tested at three intensities

such a pattern. As one might expect, the degree of control obtained with such a predator at a given level of abundance depends entirely upon the stage of the aphid infestation at its arrival. If the population is low relative to the predator's daily food requirements, then the aphid will be exterminated in a short space of time, while if the aphids are significantly more abundant than the daily food requirement of the predator, then little control will be exerted.

Exponentially increasing predation can be simulated by allowing that the number of aphids eaten per day (PRED) varies as follows;

$$PRED_{t+1} = PRED_{t} * EXP(r)$$

and by specifying an initial predator food demand $PRED_0$, and the date after the aphid infestation began on which the predators arrived in the system we can calculate the number of aphids killed on any day. Such a pattern of predation might occur given a predator which attacked only the aphid in question, and whose searching efficiency was high enough to make good reductions in prey abundance which its own exploitation brought about (see Chapter V).

We can also allow that predation is density dependent, so that the rate of increase in aphid consumption (r) is a function of aphid abundance, and therefore

$$r = F(TOTAPH)$$

shown in Fig. 106. Similarly, we can make the density dependence delayed by writing

$$r = F(OLD)$$

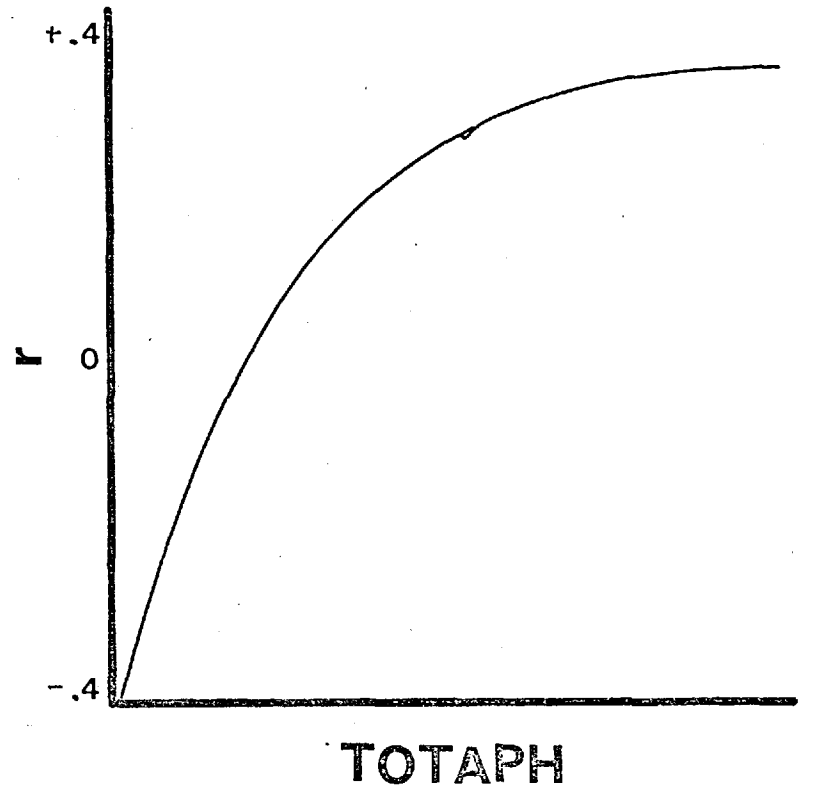


Figure 106. The relationship between the rate of increase in aphid consumption (r) and total aphid density (TOTAPH) in density dependent predation

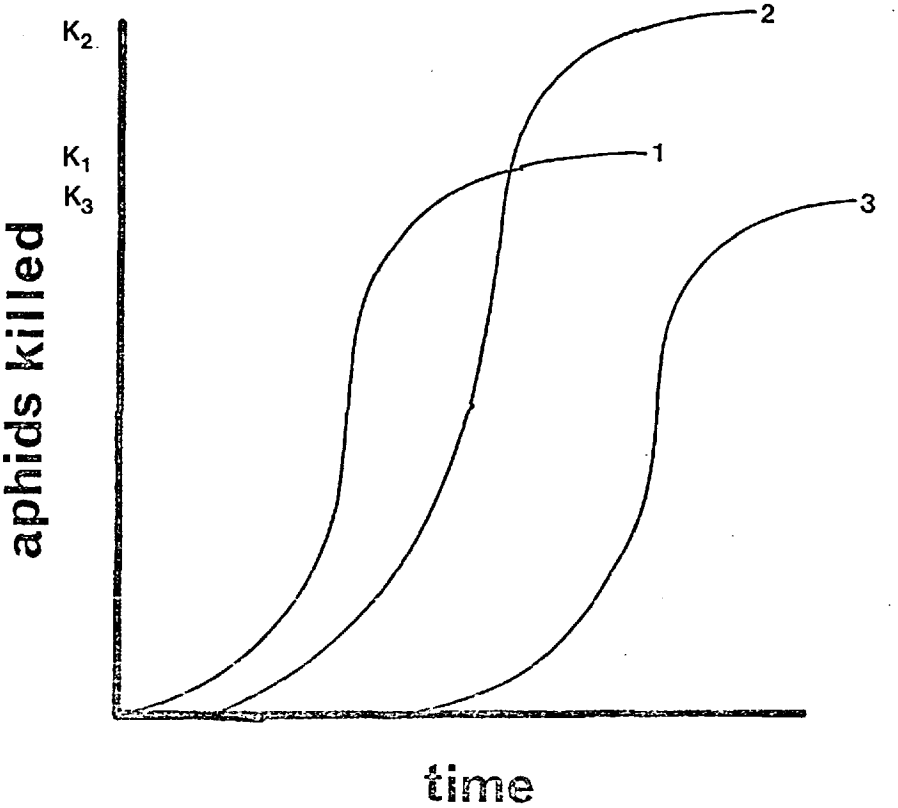


Figure 107. A graphical representation of a succession of 'logistic' predators which might act in biological control

where OLD is the aphid abundance at some time (LAG) earlier. We can observe the effects of changing LAG on the degree of control achieved.

A logistic change in the rate of prey consumption with time could occur when the aphid population is subject to attack by a rather specific predator whose own abundance is determined by factors other than the abundance of prey (by parasites, or the availability of oviposition sites, for example). A curve of predation of this type might be expected to be effective in pest control uncer two conditions. First, that the timing of the exponential phase of the curve matched the period of exponential increase in the prey population (the two species are synchronous in their development), and second, that the level K at which the predator population is limited, is sufficiently high relative to prey density at the time that the prey population does not escape control. An aphid could clearly be controlled by a temporal sequence of predators with logistic population growth curves, so long as each predator species overlapped with its neighbour in time, such that one species entered its period of rapid growth as the other reached its maximum population level (Fig. 107).

In such a case, it is possible to imagine a large increase in the number of predator species which could be supported by an aphid resource system, as long as each predator species is limited in abundance by factors other than prey density (see the discussions by Levins, 1968; MacArthur, 1972; Pianka, 1966). Here, the abundance of the aphid would be greater in the absence of any of the predators, but it is not strictly correct to say that these predators regulate aphid numbers, because there is no feed-back between prey

and predator density. The number of aphids would vary from year to year depending upon those factors which determined the K values of the predator species. If all the Ks were low in a particular year, then the aphid could reach outbreak abundance; there would be no way for the predators to increase in numbers to lessen the damaging effect.

The results of employing these different patterns of predation in the model are shown in Figs. 108, 109, and 110, where aphid numbers are plotted against time. The removal of a constant number of aphids per day can be highly effective or highly ineffective in reducing the aphid population, and the same observation applies to an exponential increase in predation (Figs. 108, 109). The effectiveness of control achieved is very sensitive to the rate of aphid removal; in Fig. 108 removing 175 aphids per day has little effect on the total numbers over the season, while a kill of 185 per day exterminates the population before the 30th day. The reason for this behaviour lies in the relationship between the reproductive rate of the aphid and the rate of removal of aphids by the predator. Imagine a number of aphids as representing the 'reproductive capital' or stock of the system; this stock will produce a certain number of aphid progeny per day. As long as the predation rate is less than the progeny production rate the aphid population will continue to increase, but as soon as the predation rate exceeds the progeny production rate, the predators will begin to deplete the reproductive capital, and the aphid population will inevitably become extinct. The only condition of stability in this system occurs when the predation rate exactly equals the birth rate. But, because the age structure of the aphid population is time-variable, the birth rate changes from day

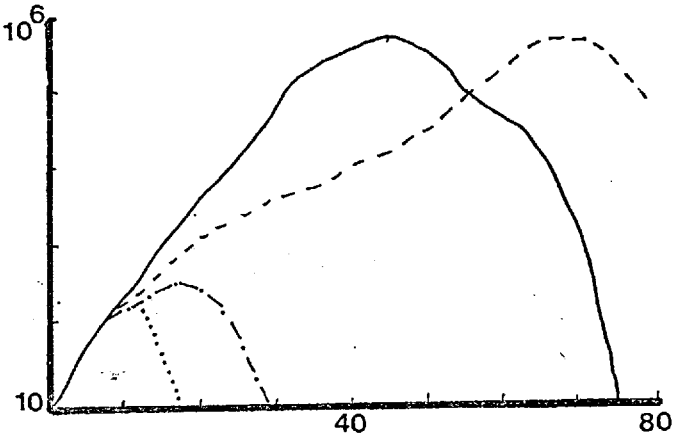


Figure 108. Aphid abundance under numerical predation. Constant removal rates of 0 (—), 175 (--), 185 (-·-) and 300 (···) aphids per day

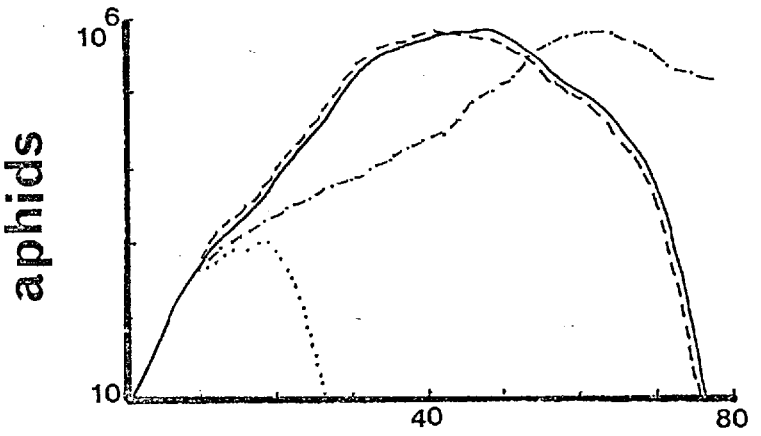


Figure 109. Aphid abundance under numerical predation. Exponential increase in removal rate beginning early (···), mid (-·-) and late in the outbreak.

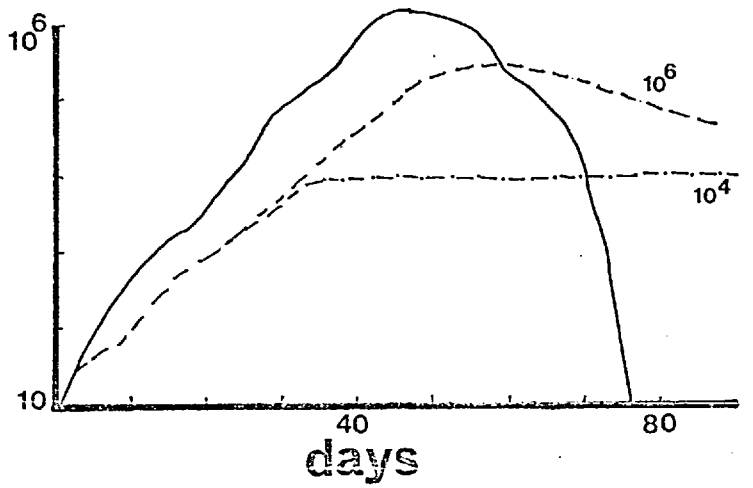


Figure 110. Aphid abundance under density dependent predation. The control curve (—), and the 80% intensity curves at two density maxima are shown (see Fig. 111)

to day, and no constant rate of predation will maintain the aphid population at equilibrium. It does not matter whether the predation rate increases exponentially or logistically with time; the same result will hold as long as the rate of aphid kill is independent of aphid numbers.

Fig. 110 demonstrates the effects of density dependence in the rate of predation. For these runs, the proportion of the aphid population attacked was taken to increase with aphid density (Fig. 111), and the different curves represent different maxima and different gradients in the density dependence of predation. The model now behaves in quite a different fashion. First, it is not possible for predation to exterminate the aphids, since however few there are left, only 10% of them can be killed (Fig. 111). Second, the maximum density which can be achieved depends on our choice of the density maximum (D1 or D2 in Fig. 111), and no longer on the crowding conditions which can be tolerated by the aphids. Because of this, the aphid population tends to last for a longer period than it does under constant predation, as the onset of alate production is delayed. From Fig, 110 it is clear that such a predator increases in effectiveness (in terms of reducing the integral of the aphid abundance curve) by having intense density dependence (eight times the predation rate at high than at low aphid densities as compared to five times), and a low density maximum (10^4 rather than 10^6 aphids at maximum predation rate).

b) Reproductive Value and Predator Preference

Animals of different ages are likely to contribute in different degrees to the future growth of a population. Young animals may die before producing any offspring, while older

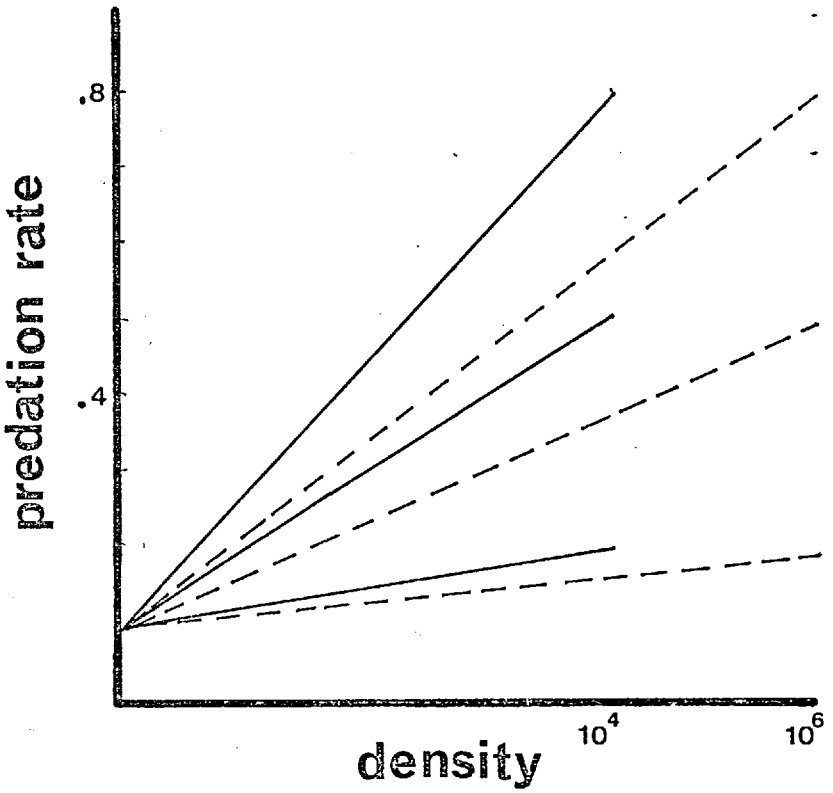


Figure 111. Six strategies of density dependent predation tested. Two 'density maxima' and three 'intensities' were employed.

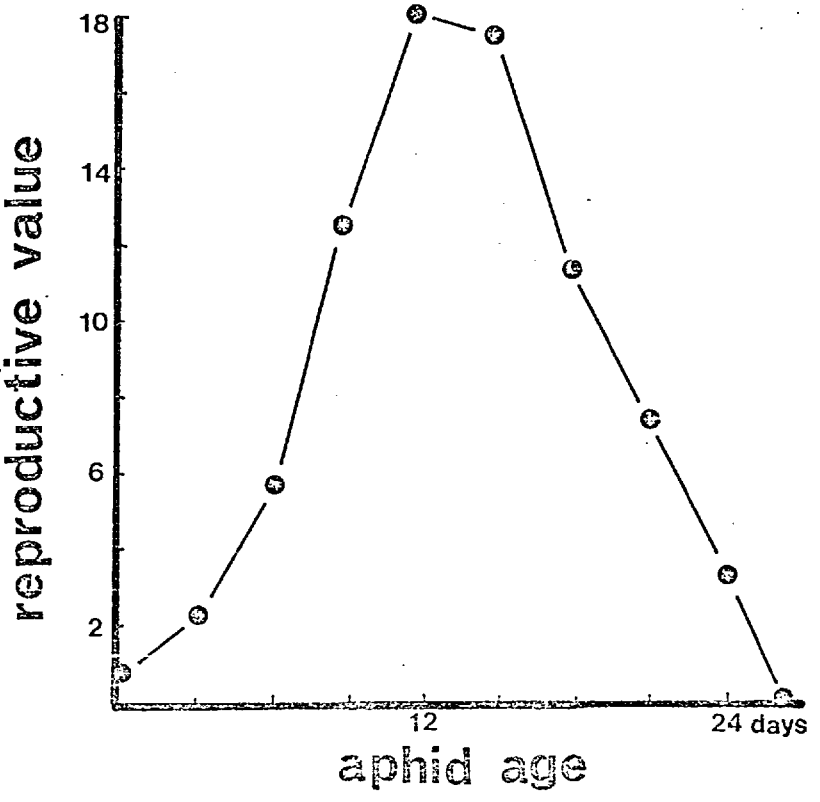


Figure 112. Fisher's reproductive value curve for Aphis fabae. Birth data from Banks and Macaulay (1964) with 95% daily survival assumed

animals may be beyond reproductive age on a given data. In both cases, their future contribution is zero. Between these two extremes there must be an age at which individuals contribute most to the growth of the population, and to determine the value of this age, Fisher (1929) proposed the equation

$$\frac{v_x}{v_o} = \frac{e^{r.x}}{l_x} \int_x^\infty e^{-r.t} \cdot l_t \cdot b_t \cdot dt$$

which he called the reproductive value at age x. In this equation l_x is the survivorship curve of the population (monotonically decreasing), b_x is the birth rate per head in the time interval x to x+1 (which is zero for pre and post-reproductive animals), and r is the intrinsic rate of increase of the population.

The limitations applying to this equation are that it assumes a stable age distribution, and time-invariant values of l_x and b_x. It is still a potentially useful measure in real populations, since it weights a simple count of heads in relation to their potential contribution to future generations. Its use has been suggested on these grounds in studies of species diversity (Lloyd, 1964) and predation (MacArthur, 1960).

It is intuitively obvious that the optimal strategy in reducing a population by predation would be to remove those animals with the highest reproductive values. We have seen, however, that a proportional treatment of predation suggests that taking the youngest (or, in general, the most abundant) age class most effectively reduces the population-integral (but it involves the removal of more animals). If we predate numerically, on the other hand, the proportion

of the population killed in each time-interval will vary, and so the age structure will be disturbed. To determine the error involved in predicting the age of optimal prey selection by Fisher's reproductive value in non-stable age distributions, the following simulations were carried out.

We must first specify the vectors for survivorship and birth with age, l_x and b_x, and we shall assume that these apply to discrete time units (in this case, days). We shall therefore have l_5 as the fraction of the animals born on day 0 alive at the beginning of the 5th day, and b_{10} as the number of aphids born to a 10-day-old adult. From these two vectors we can compute the natural rate of increase, r, by numerical approximation. We know that

$$\int_0^\infty e^{-r.t} \; l_t \, . \, b_t \, , \, dt \; = \; 1$$

(Fisher, 1929), and for discrete time intervals we can put

$$\sum_0^m e^{-r.t} \; l_t \, . \, b_t \; = \; 1$$

where m is the maximum age at which reproduction occurs. This polynomial will have only one real positive root, which can be determined quite simply by successive approximation (see Fig. 113). The simulations which follow take r to four decimal places. Once r is known for a given l_x and b_x, the reproductive value can be calculated. Again, we can approximate the integral with a summation over discrete time-units, so

$$\frac{v_x}{v_o} \; = \; \frac{e^{r.x}}{l_x} \sum_x^m e^{-r.t} \; l_t \, . \, b_t$$

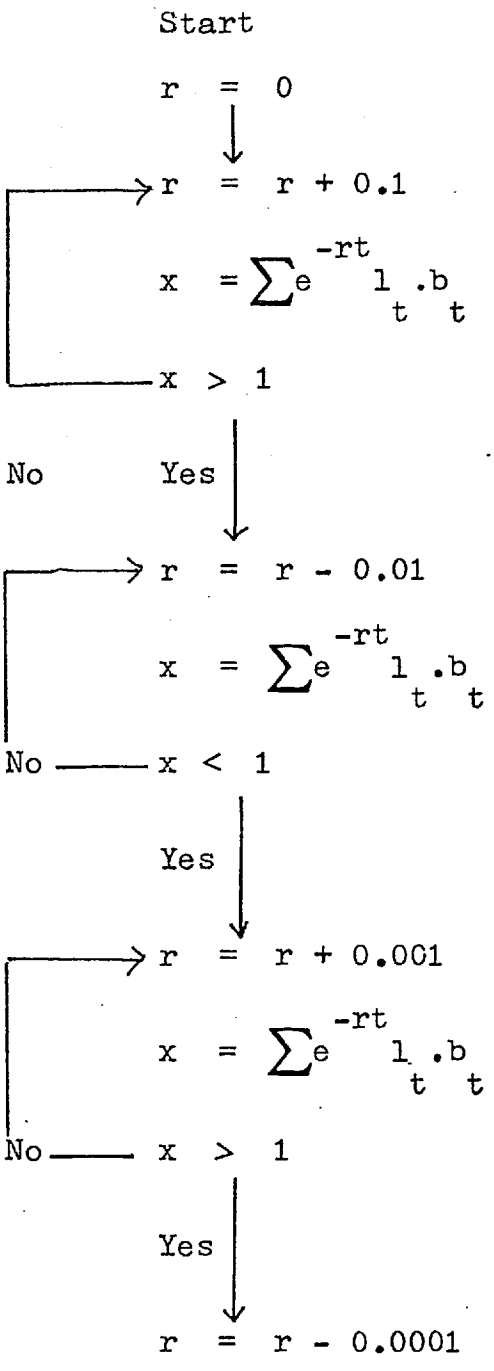


Figure 113. A very simple method for calculating r, the natural rate of increase, by successive approximation.

We now evaluate this expression for each age in the aphid's life. We shall use the b_x data as before (Banks and Macaulay, 1964), and calculate an l_x curve assuming a constant daily survival of 95%; graphing reproductive value against age gives us the curve in Fig. 112.

In order to observe the effects of age-specific numerical predation, we require a simple model of aphid population growth. Let A(I) be the number of aphids of age I and let B(I) be the age specific birth rate. If S is the daily survival rate in the absence of predation, so that $l_x = S^x$, we can trace the dynamics of the aphid population. Let us begin with a population near the stable age distribution; put 1000 aphids at age 1 and divide aphids between the other age classes so

```
A(I)  =  A(I-1) * S
```

Now we can test the effects of removing a number of aphids from the Kth instar (PREDS), writing

```
      D=0.
      DO 2 IDAY = 1,30
      BORN = 0.
      DO 3 I = MINAD, MAXAD
    3 BORN = BORN + A(I) * B(I)
```

to give us the number of births on any day, and

```
      DO 4 I = 1, MAXAD
      D = D + A(I)
    4 A(I) = A(I) * S
```

to compute the number of aphid-days (D) and survival respectively. The population reduction due to our experimental predation is then

```
A(K) = A(K) - PREDS
```

and it only remains to update the age of the animals before

simulating another day;

```
      DO 5 I = 1, (MAXAD-1)
      M = MAXAD - I + 1
    5 A(M) = A(M-1)
```

and putting the births into the now vacant first age class

```
      A(1) = BORN
```

the cycle is complete. After 30 days we can write out the total number of aphid-days experienced by the crop

```
    2 CONTINUE
      PRINT, D
      STOP
      END
```

If Fisher's reproductive value is a robust model, in the sense that it gives reasonable predictions even when its assumptions are contravened, we should observe a decreasing, linear relationship between the number of aphid days in the pest outbreak (D), and the reproductive value of the individuals of age K which we predated. If the fit of the data to this regression is good, then the model is robust, and variations in the age structure brought about by removing a given number (and hence a variable proportion) of animals from a single age class does not importantly affect its behaviour. Since, by changing the l_x each day we are also changing the value of r (see above), this conclusion would be difficult to prove analytically.

Figure 114 demonstrates such a graph. The two curves shown are non-linear because the fecundity curves are non-linear with age (the b_x values). The interesting point to emerge from this graph is that although the reproductive value might be the same at two ages (Fig. 112), it is better

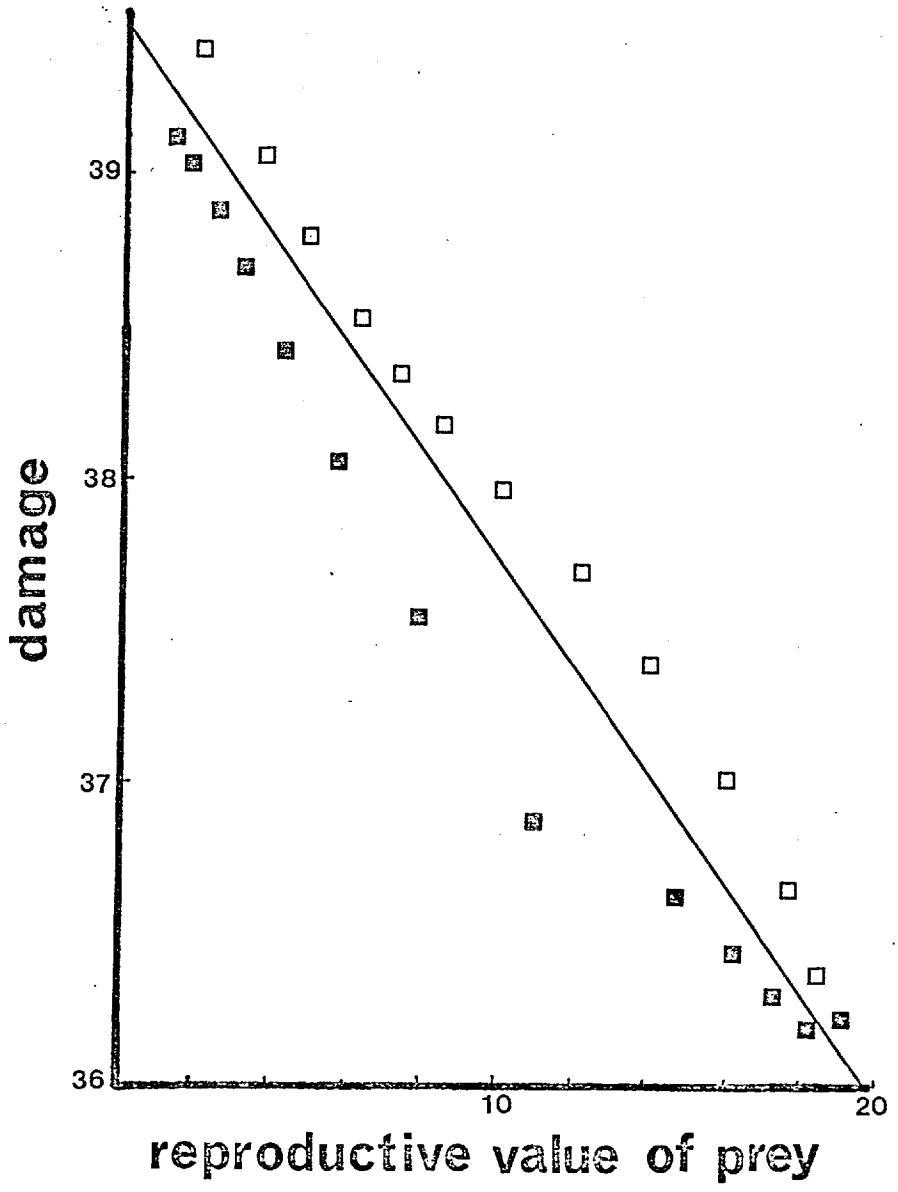


Figure 114. The relationship between the reproductive value of the aphid age class predated and the damage done to the crop (expressed as thousands of aphid-days after 30 days)

from the point of view of minimizing plant damage to remove the younger animals; compare the lower (younger) and upper (older) curves. The cause of this difference is, quite simply, that the animals spend a certain number of aphid-days on the plant between reaching a particular reproductive value on the up-slope of the curve (Fig. 112) and reaching it again on the down slope.

It is also clear from Fig. 114 that the removal of individual aphids of maximum reproductive value (i.e. aged 13 days; Fig. 112) brings about the greatest reduction in plant damage. The error introduced by applying Fisher's method to a non-stable age distribution were negligible in this context.

Discussion

The aim of this model is to determine the strategy of predation which constrains the growth of an Aphis fabae population in those ways suggested in Chapter III; namely late development, low peak numbers, and low overall feeding rate. We can consider the predation strategy in relation to its timing, its type (proportional or numerical), and its age-selectivity.

1 Timing

Just as the time of aphid infestation was crucial to the degree of plant damage, so it is that the synchrony between aphid infestation and the onset of predation determines the peak abundance reached by the aphids.

Most of the predators of Aphis fabae overwinter outside the arable areas of farmland, in hedgerows, gardens and woodlands. In spring they emerge from their dormant phase and typically pass through a number of developmental stages on

weeds and wild plants before immigrating into the bean crop (van Emden, 1965a, 1965b). If the predators immigrate early, then the abundance of aphids may be too low to allow population build-up, and the predators may leave the crop rather early. This would allow late infesations of aphids to escape control, and, depending on the developmental state of the crop plants, cause an economic loss in bean yield. Again, if the immigration of predators is delayed until aphid numbers in the crop are high, the predator may be unable to increase at a sufficient rate to subdue the pest population. The model, while not simulating the dispersive behaviour of the predators, does demonstrate these effects by predicting that early immigrations (equivalent to high early predation rates) will lead to aphid extinction, and hence to predator starvation or emigration, allowing the possibility of reinfestation by alatae. Similarly, late immigration (equivalent to low early predation rates) tends to be relatively ineffective in reducing aphid numbers, unless the predator is highly density dependent in its feeding and reproductive behaviour. This is shown in Figs. 108, 109 and 110.

In addition, the duration over which the predators feed in the crop will clearly affect the level of damage experienced by the plants. If the predators emigrate before pod-fill, or before the plants become undesirable to the aphids, then late season population build-up could give rise to considerable yield reductions. The ideal predator will therefore be rather tolerant of low aphid densities, so that emigration does not occur while a potentially damaging reproductive stock of aphids remains on the plant, and rather flexible in the mechanisms which cause it to leave the crop in search of overwintering sites or new sources of food organisms. A predator

which always left the crop on August 1st because of a photoperiodic response would clearly be less effective than a species which stayed on the plant until all the aphids were dispersive alatae.

The desirable attributes of predator immigration are rather less easy to define. As I have said, the immigration should occur neither too early nor too late, but the difficulty is that the precise timing seems to be more a function of the conditions of prey availability on the wild host plants than of the number of Aphis fabae on the young bean crop (R. M. Perrin, pers. comm.). If the aphids are abundant on the wild hosts, the predators may not fly into the bean crop until Aphis fabae is very abundant and essentially beyond their control, whereas low aphid availabilities on the wild plants may cause the predators to leave so early that they suffer very high mortality in seeking out areas of high prey availability. It would seem that unless the predators are reared and released into the crop by the grower himself, then a certain amount of asynchrony between the predator and the pest is inevitable. Depending upon the extent of the asynchrony, additional control measures such as the application of a specific aphicide may be necessary.

2 Numerical versus Proportional Predation

Whether a predator predates numerically or proportionately will depend upon the biological attributes of the predator species (its size, dispersive ability, and so on), and on the availability of the aphid prey. As we have seen, the two types of predation can lead to widely divergent consequences in terms of aphid abundance and crop damage. Basically, numerical predation will be observed in those predator species whose

abundance is determined by factors other than the availability of Aphis fabae; by the availability of other prey species, by parasites or diseases, or by some environmental resource like oviposition sites for example. When the predator is sufficiently common relative to Aphis fabae then it can exert a controlling influence on pest abundance, but, since numerical changes in the predator population occur largely independently of changes in the number of Aphis fabae, such a predator is unlikely to exert a long-term controlling effect. As the number of predators of this type in the crop increases, however, the mean abundance of the target aphid should be reduced. It seems that unless the abundance of the predator species is responsive to changes in prey availability then the pest will always be likely to escape control when environmental conditions (and particularly weather patterns) are somewhat atypical. This numerical response is considered in Chapter V, and all we require to note at this stage is that it can operate either by increased fecundity, reduced mortality, increased immigration, or decreased emigration. If one or all of these aspects of predator biology is affected by the abundance of Aphis fabae then its potential in control is increased.

Density dependent predation requires that the proportion of the aphid population eaten per day increases with aphid density. In the absence of numerical change in the predator population, this could only occur if the daily intake per predator was sufficiently flexible to absorb any net increases in aphid abundance occurring within the day. The more likely situation is that the predator population responds to changes in prey density by the means outlined above. These can act quite rapidly (immigration and emigration), but often only

after an appreciable time lag (enhanced survival or fecundity). Irrespective of whether the proportion of the aphid population killed increases with aphid abundance, a predator which predates proportionally will be more effective in control than a predator which eats a given number of prey each day. We can see this best by an example. Let us define three predators; one predates numerically at a constant rate of 40 aphids per day, the second predates proportionately at 40%, and the third predates proportionally but in a density-dependent fashion (Fig. 115). Assume that the aphid population doubles each day, then, starting with 100 aphids we should observe the following;

PREDATOR	DAY	1		2		3		4	
	Aphids	100	60	120	80	160	120	240	200
1	# Kill	40		40		40		40	
	% Kill	40		33		25		17	
	Aphids	100	60	120	72	144	86	172	103
2	# Kill	40		48		58		69	
	% Kill	40		40		40		40	
	Aphids	100	60	120	67	134	71	142	74
3	# Kill	40		53		63		68	
	% Kill	40		44		47		48	

To demonstrate the dependence of the number of aphids killed on aphid density we need only plot these data (Fig. 116); clearly predators 2 and 3 take prey in relation to its availability. If we now plot the proportion of the population eaten against density (Fig. 117) we see species 2 constant (by definition), species 1 a decreasing curve (since births exceed 40 per day in this example), and species 3 increasing (again, by the definition of density dependence).

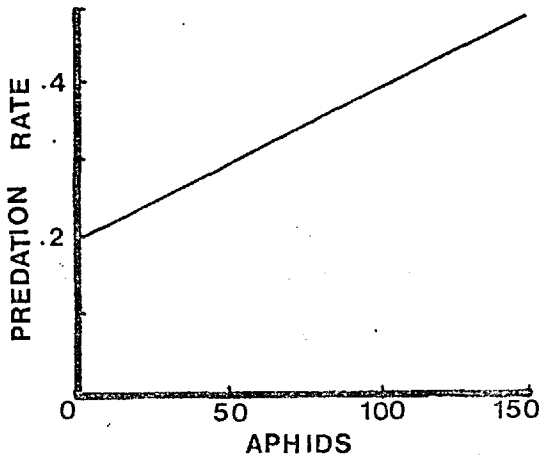


Figure 115. Density dependent predation; the fraction of the aphids killed increases with aphid density

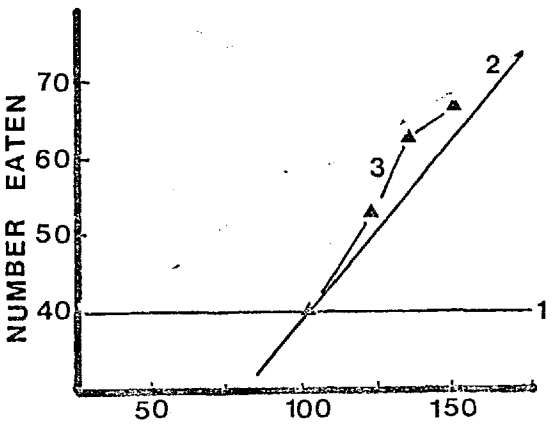


Figure 116. The number of aphids eaten at different aphid densities with constant predation (1), proportional predation (2), and density dependent predation (3)

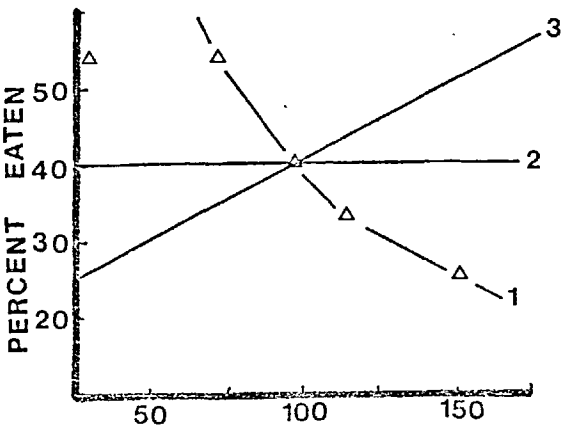


Figure 117. The proportion of the aphid population eaten at different aphid densities. Symbols as above

In short, the effectiveness of our predator species increases with its ability to adjust its rate of consumption of Aphis fabae to the abundance of the pest, and with the degree to which it can increase the proportion of the population killed as the aphid increases in numbers (i.e. the intensity of density dependence). The first criterion is quite simple to fulfil; the predator would only have to consume Aphis fabae in relation to the proportion of the aphids in the environment of this species. The difficulty is simply that the food intake capacity of the predator population is limited, and so functional responses can only be effective up to a certain ratio of aphids per predator (see Chapter V). The second criterion requires the predator to be rather well adapted to Aphis fabae, and either to aggregate in areas of high aphid availability (Hassell and May, 1973), or show highly effective numerical responses to prey density (Chapter V).

3 Age Selectivity in Predation

The optimal strategy of prey selection in terms of biological control depends upon the way in which a particular predator species treats the aphid population. If the predator is of such a size, or has such a behaviour pattern that all individuals in the aphid population are not equally available to it, then we must consider the aphid population as consisting of a number of food resources, only a few of which are actual target prey. It is possible to imagine an insect predator too small to consume aphids larger than the first instar, and it is equally possible to think of another which would feed exclusively on larger prey. With such a predator the same arguments as outlined in the previous section would

apply, but feeding would be concentrated in one particular size class. In this case we might ask which age class when predated proportionally reduces the overall population to the greatest extent ? The results of the simulation runs showed that this type of predation was most effective when applied to first instar aphids. In retrospect it is quite straightforward to show that this 'multiple-resource' type of preference will be most effective when applied to the most abundant age class, since the number killed depends only on the product of predation rate and availability. If there was no predator species available to attack the entire range of aphid sizes, then we should clearly choose that species which concentrates on the most abundant size class. In Aphis fabae this tends, at most stages of the infestation, to be the first instar (see Fig. 94), but, if adult life is rather prolonged, and all adults are more or less similar in size, a predator concentrating on adults may be almost as effective.

It would seem intuitively obvious that an ideal predator would not be limited to the consumption of a particular size class, but could eat aphids of all sizes. If such a predator showed no preference for prey of different sizes, then it would kill aphids in the proportion it encountered them, and, if the aphids were distributed randomly in space, this would be the proportion in which the aphids of a given size occurred in the population. We know, however, that Aphis fabae do not distribute themselves randomly, but live in very tight-knit aggregates (Chapter II; Dixon and Wratten, 1971; Ibbotson and Kennedy, 1951). Within an aggregate, on the other hand, most age classes are represented, and if the aggregates do not differ significantly from one another in terms of their age-structure, it will be reasonable to suggest that the predator

could encounter age-classes in the proportion they occur in the whole population. So if A_i is the fraction of the population of aphids in the ith age-class, and D_i is the fraction of this food in the predator's diet, with no preference we should observe $A_i = D_i$ for all values of i.

Now, since the predator can attack aphids of all sizes, and the total intake rate is limited by the number of predators and their individual food requirements, the proportional strategy of prey selection will not apply. Instead, we should ask which age of aphids are most important in pest population growth, and which, therefore, will bring about the greatest reduction in population increase per individual removed? This was shown to occur in the model by removing aphids of maximum reproductive value (i.e. of age 13 days). We can state that the ideal biological control predator which can feed from the whole aphid population would eat more individuals of this age than of any other. But, since individuals of age 13 days are less abundant than all younger age classes, the predator must adjust its feeding behaviour to achieve this end; it must show positive preference for 13-day-old aphids.

We can define preference empirically by plotting the proportion of a food item in the diet against the proportion of the item in the environment. No preference will shown when $D_i = A_i$ for all i, and positive preference when $D_i \geq A_i$ for all i (Fig. 118). The numerical value of 'preference' can be read off from such a curve as follows. If there are N types of food item in the environment then

$$1 = \sum_{1}^{N} D_i$$

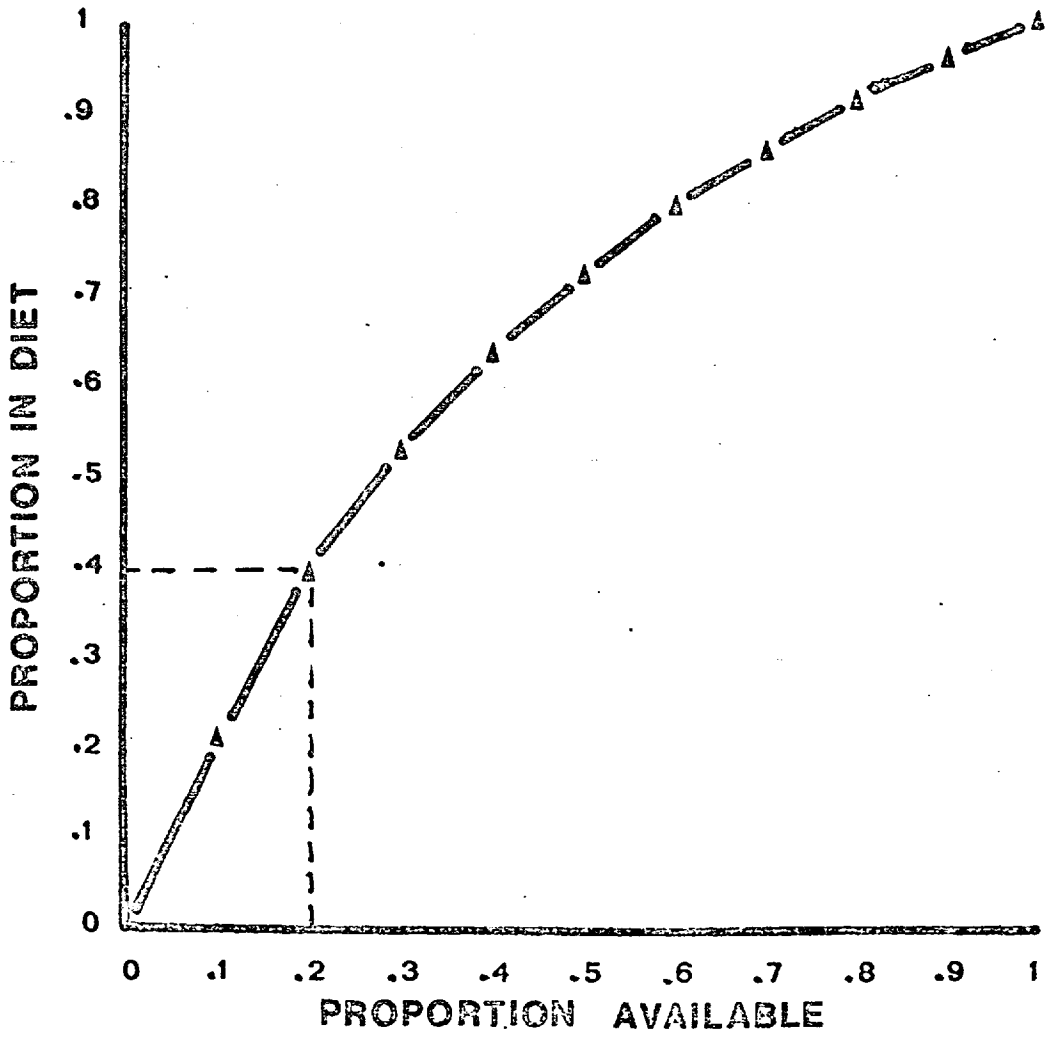


Figure 118. An empirical preference curve. The value of preference for the ith food item is read off at A = 1/N; in this case at A = .2 (i.e. five items to choose from). The relative preference for the ith item is therefore 0.4

and when they are all equally abundant in the environment we would have

$$A_i = 1 / N$$

for all i. In this instance, when availability does not affect the fraction taken, the proportion of the ith item in the diet will be a measure of the preference factor of the predator for this food item. We determine its value by reading off D_i at $A_i = 1/N$ (see Fig. 118). Plotting the curves for all N food types will give us N estimates of preference which, by definition, must sum to unity.

The actual number of aphids eaten from each size class will depend not only on the preference factor, but also on the availability of each size of prey. We can model this by assuming that the predator orients its behaviour so as to attack prey in relation to the product of preference and availability; i.e.

$$ENC_i = PREF_i * AVAIL_i$$

This assumes that the predator encounters the aphid size classes in the proportion they occur in the environment, but that it ignores the less preferred types in relation to a weighting factor (its preference). It is a statistical estimate not designed to predict the type of prey to be encountered at any given time, but rather to describe the average fraction consumed over a long period. To compute the fraction of the ith food type in the proedator diet we simply divide the total number of encounters with the ith type by the number of encounters with all types, writing

$$D_i = \frac{PREF_i * AVAIL_i}{\sum_{1}^{N} PREF_j * AVAIL_j}$$

We can now state the selective strategy of our optimal predator in temrs of its preference and the reproductive value of the types preferred (Fig. 119). In general, preference should be a monotonically increasing function of the reproductive value of the prey; the more right-biased the curve, the better the selection strategy (in other words, the more the predator prefers high reproductive value prey the better).

4 Predation and Feeding Rate

Since the model includes aphid size as affecting the rate of sap removal from the plant, it is clear that age-selective predation will alter the mean feeding rate per aphid of the population. Each adult aphid killed will reduce the immediate rate of removal more than each young aphid eaten, but we recognise that to reach a higher age an aphid will have consumed more carbohydrate in total.

Also, by reducing aphid numbers, predation will act indirectly on all those density-related processes affecting feeding rate (density itself, leaf water content, food quality; see Fig. 79). Because some of these processes act to decrease the feeding rate as aphid density increases, it could be that predation acts to moderate their effect and so, overall, to increase the mean aphid feeding rate.

The precise recommendations as to the type of predation which minimises feeding rate as well as population growth depend upon the relationship between aphid size and individual

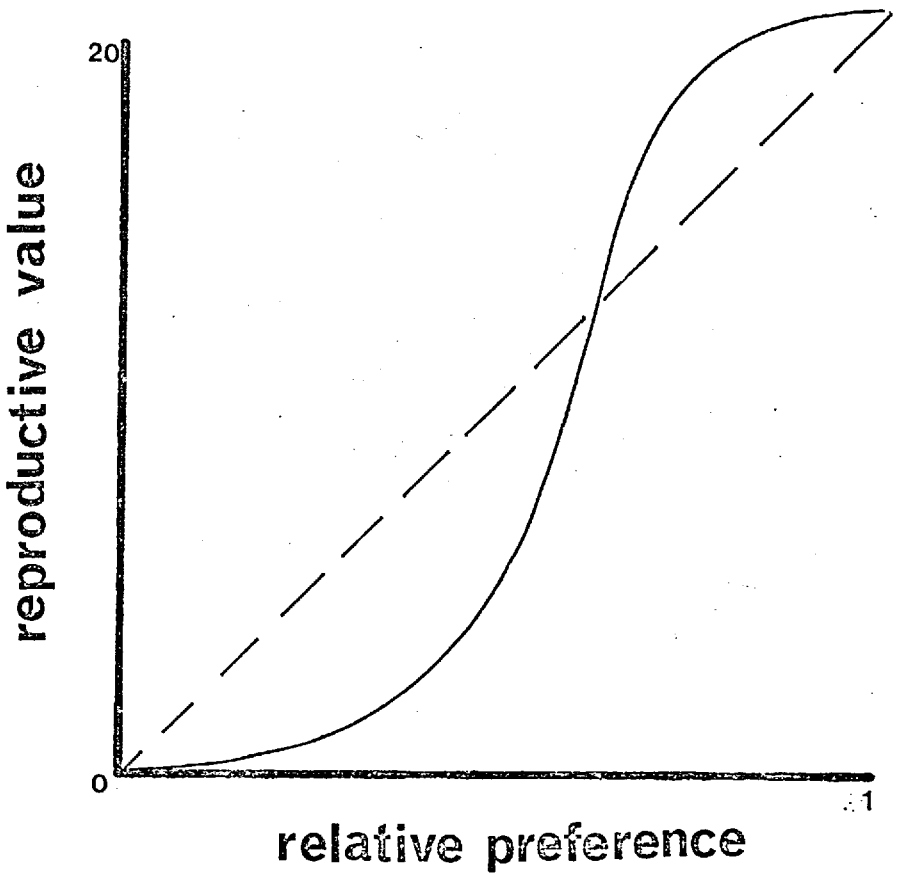


Figure 119. The general (hatched line) and the optimal (solid) strategy of prey selection by the predator. Aphids of high reproductive value should always be preferred, but, since they are typically less abundant than younger aphids, the predator should show a proportionately stronger preference for them

feeding rate. If small aphids feed almost as rapidly as older aphids, then the age of optimal predation will be shifted below the age of maximum reproductive value, while if old aphids feed at a vastly greater rate than the young, then the optimal age for predation may be old-shifted.

5 Control Strategy

We can now define the type of predation pattern necessary to keep an Aphis fabae population below the level at which it would cause significant economic loss of bean crop.

a) The predator should be introduced into the crop after the aphids have arrived, but before pest abundance is too high;

b) ideally, predator immigration should be spread over an extended period, since this will minimise the probability of aphid reinfestations leading to populations of outbreak level;

c) the predator should respond to increases in prey abundance by increasing its food intake and/or increasing the number of predators;

d) if the available predator species can only handle a limited number of aphid size classes, we should choose that predator species which concentrates upon the most abundant class (in Aphis fabae, the first instar);

e) if the predator can attack all stages of the pest, its preference should be maximal for aphids of maximum reproductive value;

f) the rate of increase in predator numbers should be at least as responsive to increases in temperature as the rate of increase in aphid reproduction, otherwise high temperatures will produce aphid outbreaks (the converse should also hold);

g) the predator should respond to low prey densities by reducing the rate of exploitation: this will increase the

stability of the biological control system;

h) the predator should maximise the plant damage reduction per aphid killed, by selecting aphids from the young-side of the reproductive value curve (Fig. 114).

6 Model Evaluation

The type of answer produced by a model depends on the terms in which the question is phrased, and on the way in which these terms are incorporated into the model structure. For example, van Emden (1966) investigated the effectiveness of predators in reducing aphid populations by using a very simple, two parameter model attributed to Bombosch (1963);

$$a_n = a_o \cdot q^n - k\left[\frac{q^n - 1}{q - 1}\right]q$$

where a_o and a_n are the starting and final populations, q is the daily multiplication rate, and k the rate of predation. When $q = 1$ then $a_n = a_o - k.n$. From this model it is clear that the voracity of the predators (k) and the reproductive rate of the aphids (q) are important; indeed, they could not be otherwise, since they are the only parameters in the model. The effects of predator synchronization can be demonstrated by splitting the right-hand side of the equation into two sections, one for pre-predation growth, and another for post-predation. This gives

$$a_n = a_o \cdot q^s + a_s \cdot q^{n-s} - k.q\left[\frac{q^{n-s} - 1}{q - 1}\right]$$

and by varying the time lag between aphid infestation and predator arrival (s) the effects of asynchrony can be observed.

The purpose of the present model has been to investigate the necessary predation pattern for aphid control in more detail; by considering more variables, and by considering the processes by which the parameters above (and the reproductive rate, q, in particular) are affected. Van Emden's model is essentially a balance sheet for aphid numbers; so many births, so many deaths, and hence net population change. My objective has simply been to extend this analysis so that the birth rate q is non-constant, and to interpret the effects of this variability on predator strategy.

In terms of form, the present model resembles the simulation program developed by Gilbert and Hughes (1972) in several respects. In both models the aphid population is divided into age classes, and many of the population processes (birth and death rates, for example) are age-dependent. The main difference, however, is that the present model was built specifically to consider the problem of optimal predation strategy, while Gilbert and Hughes apparently built their model to describe the life-system of an aphid (Brevicoryne brassicae) in a particular part of Australia, and from the completed model went on to make observations on the evolutionary adaptation of the principal parasite.

This distinction may appear rather trivial, but unless a model is built to tackle a specific problem, and the problem is explicitly stated at the outset, it is very difficult to judge the realism of model behaviour (Chapter I). To be formally comparable with the present model, Gilbert and Hughes' simulation would have to investigate the relationship between the number of eggs laid per parasite (its evolutionary adaptedness) and the number of parasites produced per season

(its fitness) explicitly. This would involve the inclusion of more information on parasite life-history (especially on survival), and perhaps rather less emphasis on the dynamics of the aphid host.

The features of the present model which are new include the time-based treatment of temperature-affected development, the relationship between population growth and size growth, the effects of predation on feeding rate, and the application of reproductive value to non-stable age distributions. As with the plant growth model of Chapter III a number of processes have been included for which no real data exist, especially in the section dealing with the computation of individual feeding rates. There is a clear need for experiments to be carried out to determine first, whether feeding rates under field conditions do vary appreciably, and second, to which factors this variability can be attributed. The whole field of pest control hinges upon reducing damage to the crop, which, when feeding rate and plant sensitivity are variable, may bear only a very tenuous relationship to a straightforward reduction in aphid numbers. Clearly the predation strategy which minimises aphid feeding is more appropriate to pest control than the strategy which minimises aphid numbers; without data on the factors influencing aphid feeding rates in the field, it is not possible to separate one strategy from the other.

CHAPTER V A SIMULATION MODEL FOR INVESTIGATING THE POTENTIAL OF INSECT PREDATOR SPECIES IN BIOLOGICAL CONTROL

Introduction

Parasitism and predation are probably the most modelled phenomena in ecology (Royama, 1971; Hassell and May, 1973). They possess a set of properties which make them attractive subjects for mathematical analysis; first of all, they are fundamental ecological processes, considering as they do the relationship of one species to another over a period of time. Second, it is possible to consider both populations in terms of their abundances alone, since the interaction operates (at least in the simple case) between an individual parasite and an integer number of hosts which it parasitizes. Third, and of considerable importance, is the fact that even with a very simple set of assumptions, a complex pattern of output can be obtained from the model. This means that the model-building exercise tends to be rewarding in terms of the number of testable hypotheses brought to light, and in the diversity of

behaviour which can be mimicked.

Most models of predation and parasitism start with the basic assumption that the rate of increase in host abundance is a decreasing function of the abundance of parasites (e.g. Lotka, 1925; Volterra, 1926; Nicholson and Bailey, 1935). So, if we have r as the natural rate of increase of the host population (of which there are H individuals), and P active parasites, we should observe that

$$\frac{dH}{dt} = H (r - f(P)) \qquad \ldots\ldots (1)$$

To simplify the model, f(P) is usually assumed to increase linearly with P (e.g. Lotka, 1925; Volterra, 1926). That is, replacing f(P) by c.P in (1),

$$\frac{dH}{dt} = H (r - c.P) \qquad \ldots\ldots (2)$$

where c is a positive constant.

The parasite population is then assumed to increase in relation to the number of hosts available, and to decrease exponentially when no hosts are present. The constant r' describes this rate of population decline in the absence of hosts, and we can write

$$\frac{dP}{dt} = P (- r' + g(H)) \qquad \ldots\ldots (3)$$

Again, simplifying g(H) to a linear relationship we gain

$$\frac{dP}{dt} = P (- r' + a.H) \qquad \ldots\ldots (4)$$

where a is a positive constant.

Pielou (1969) shows that this pair of typical Lotka-Volterra equations can be solved by putting

$$\frac{dH}{dP} = \frac{(r - c.P)\,H}{(-r' + a.H)\,P}$$

and rewriting as

$$r'.\frac{dH}{H} - a.dH + r.\frac{dP}{P} - c.dP = 0 \qquad \ldots\ldots (5)$$

which integrates to give

$$r'.\ln H - a.H + r.\ln P - c.P = \text{constant} \qquad (6)$$

The solution of this expression yields a family of closed curves, with one curve per value of the constant of integration. This value in turn depends upon the initial values of H and P. There is no damping in this system, and the relative abundances of parasites and hosts continue in closed cycles indefinitely, unless the starting values were

$$H = r'/a \qquad \text{and} \qquad P = r/c$$

in which event the densities of host and parasite are constant indefinitely.

It is rather difficult to increase the biological realism of these equations while maintaining their analytical tractability. Consequently, most of the later attempts at modelling the predator-prey interaction have abandoned this format, and moved instead to a consideration of the number of prey eaten (or hosts attacked) at a given abundance of predators and prey. The principal objective has been to relieve the constraints of linearity and time-constancy in the effect of predation on the rate of prey increase, and in the effect of prey density on

predator abundance (see equs. 2 and 4).

In general, we shall be looking for an expression of N_{ha}, the number of hosts attacked, in terms of the parasite density (P), host density (N), and those biological processes which affect the interaction.

Thompson (1924) suggested a model which accounted for the fact that the number of eggs laid per female parasite was limited (by her age and reproductive physiology). If each parasite laid a constant C eggs, then the population could lay C.P eggs. The proportion of the host population affected is then C.P/N, and he suggested that the number of hosts attacked would be given by

$$N_{ha} = N (1 - e^{- C.P/N}) \qquad \ldots\ldots (7)$$

assuming that the fraction of hosts escaping attack is given by the zero term of a Poisson distribution ($e^{-CP/N}$). Some hosts, in other words, receive more than one parasite egg, but only one adult parasite emerges.

Nicholson and Bailey (1935) modelled the process in a different way; they assumed that the fraction of the host population encountered would increase linearly with the number of searching parasites, and that the egg-laying potential of the parasites did not affect the number of hosts attacked. The constant of proportionality describing the rate of encounter between parasites and hosts they called the 'area of discovery' a, and again assuming random search, they wrote

$$N_{ha} = N (1 - e^{- a.P}) \qquad \ldots\ldots (8)$$

These two models behave differently, in that the number of hosts encountered per parasite increases linearly with host

density in Nicholson's model, but is constant in Thompson's (Fig. 120). Both models express the number of hosts attacked as a fraction (the bracketted term) of the number of host present.

In 1959 Watt proposed a new equation for the number of hosts attacked, which included a maximum attack rate (like Thompson's model), and a new concept of interference between searching parasites (see also Hassell, 1970). Watt's model differs from the two previous equations by stating that the number of hosts attacked is a fraction of the number of parasite eggs available (rather than of the number of hosts). We have, therefore, a total of P.C parasite eggs of which less than 100% will find their way into unparasitized hosts. Watt writes

$$N_{ha} = P.C\ (\ 1 - e^{-a'.N.\left[P^{1-b}\right]}\ )$$

and the bracketted term expresses the proportion of parasite eggs which are not laid in hosts as a function of an attack coefficient, a', (which is related though not identical to Nicholson's area of discovery), and an interference term, P^{1-b}, where b is a constant of interference. Watt's model has been criticised by Hassell and Rogers (1972) and by Royama (1971). Its general form, however, does seem to be rather more meaningful in biological terms, because it puts the emphasis on the parasite population, calculating N_{ha} as a fraction of the number of parasite eggs available, rather than as a fraction of the number of hosts as before. This will tend to minimise the risk of looking at parasitism as a means of 'self-regulation' by the host population, and also allows that under many circumstances the rate of parasitism (the fraction of available hosts which are parasitized) will be independent of host density. There is a danger in the models expressing

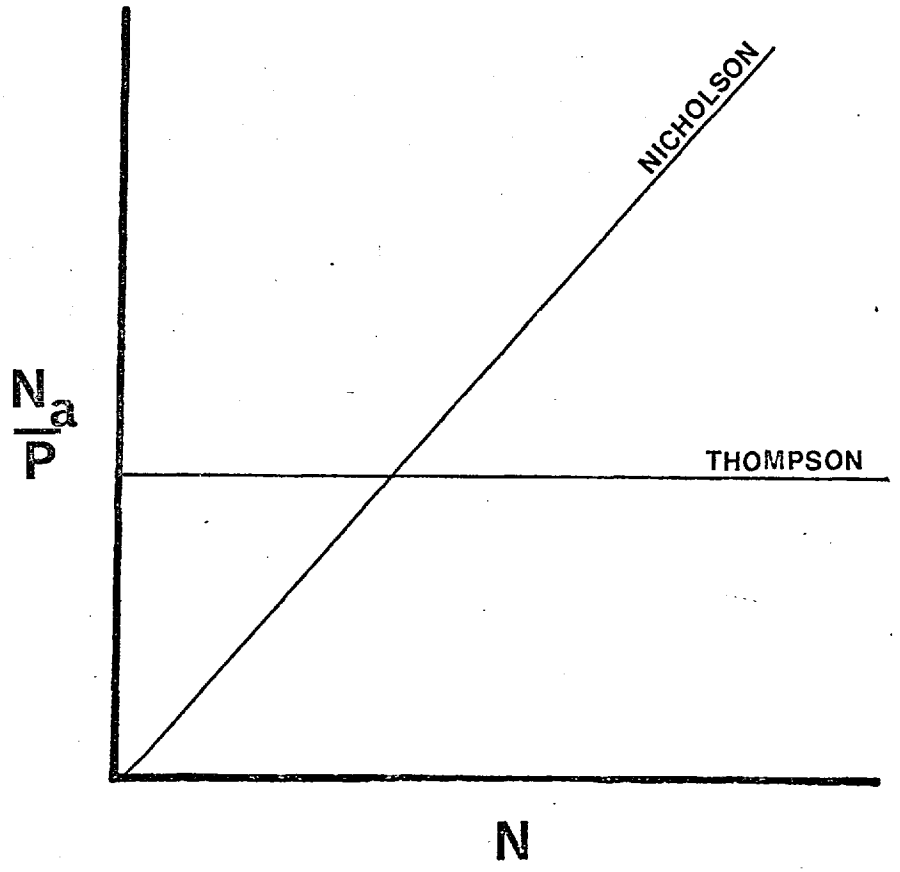


Figure 120. The number of encounters per parasite which are assumed under Thompson's and Nicholson's models of parasitism at different levels of host (prey) abundance

the number of hosts attacked as a fraction of the number available that density-dependence is implicitly assumed (e.g. Nicholson's model in Fig. 120).

Holling (1959) considered the effects of predator time-allocation between various aspects of searching behaviour in arriving at his equation. This model, as Rogers (1972) stresses, generates the number of prey encountered, but does not predict the number attacked, since host exploitation is not considered. If T is the total time of exposure between the prey and predator populations, and T' is the time taken to pursue and eat an individual prey (the handling time), Holling puts

$$\frac{N_a}{P} = \frac{a''.\,T.\,N}{1 + a''.\,T'.\,N} \qquad \ldots\ldots (10)$$

where a" is yet another attack coefficient. Each predator's hunting time is taken as $T - T'N_a$ and in unit time each predator will encounter $a''.N_a$ prey. The number of encounters is then $a''.N(T - T'N_a)$ which can be rearranged to give (10). Later (Holling 1965, 1966a) these temporal aspects were further sub-divided to consider the effects of non-feeding activity, and the time spent in pursuit, ingestion, and digestion prior to initiating another search pattern. Clearly, these parameters apply to a predator, but they do have analogues in parasite behaviour (duration of oviposition, post-oviposition rest, time spent avoiding super-parasitism, and so on). Similarly, the attack coefficient was analysed into sub-components dealing with the perceptive range of the predator, its speed of movement, the prey's speed of movement, and the capture success (measured as the fraction of prey coming within the 'range' which are successfully captured).

Hassell and Varley (1969) modified the basic Nicholson-Bailey model to account for the observation (e.g. Hassell, 1971) that parasite searching efficiency is dependent upon the density of searching parasites. The attack coefficient a (the area of discovery) was made exponentially dependent upon parasite density to account for mutual interference. They set

$$a = Q . P^{-m}$$

Q being the area of discovery at P = 1, and the mutual interference constant, m, being the slope of the linear regression of log a on log P. This model is equivalent to Nicholson's only when m = 0; otherwise the behaviour is markedly different, especially with respect to the stability of the interaction (see Hassell and May, 1973).

Following on this, Hassell and Rogers (1972) incorporated Holling's ideas on the effects of handling time. The number of encounters could then be expressed as

$$N_a = \left[\frac{a'' \alpha P^{-\beta} T N}{1 + a'' . T' . N} \right] P \qquad \ldots\ldots (11)$$

and the number of attacks (following Rogers (1972) and Royama (1971)) can be calculated by substituting N_a in

$$N_{ha} = N \left(1 - e^{-\frac{a}{N}} \right) \qquad \ldots\ldots (12)$$

The term $\alpha P^{-\beta}$ models the interference component, making the time that the hosts are exposed to parasitism dependent upon parasite density.

Functional Responses

1) Functional Response to Prey Density

A predator species can be said to act in a density-dependent fashion if the proportion of prey attacked increases as prey density increases. The response is said to be 'functional' if, in Solomon's words (1949), "as host density rises, each enemy will attack more host individuals, or it will attack a fixed number more rapidly."

The processes by which functional responses operate tend to fall into two categories; behavioural responses relating the way in which the predators search for and capture their prey, and physiological responses governing the number of prey which each individual predator can consume in a given time period.

It is not clear from Solomon's statement that the functional response can not be a continuously increasing function of prey density, since the number of animals attacked per predator has an upper limit in the gut capacity of the individual (although it is possible that more prey are killed than are eaten), and the rate at which animals are attacked has an upper limit in the handling time of each prey item (assuming also a limited feeding period). If the handling time per capture is HAND, and the time available is TIME, then the maximum kill will be

$$KILL = TIME / HAND \quad \ldots\ldots (13)$$

this being the case when prey are so common that searching time approaches zero.

This limitation to the effectiveness of the functional response has been incorporated in several of the predation/parasitism models, and operates to produce an analogue to the

economic law of diminishing returns (Nicholson and Bailey, 1935; Holling, 1959; Ivlev, 1955). The theoretical aspects of the assumptions included in these models, and their analytical accuracy are reviewed by Royama (1971).

The fundamental difference between models of predation and models of parasitism is that to determine the total number of attacks in a given period of time, rather than simply the attack rate, models of predation must account for the removal from the pool of available prey of those animals consumed prior to any instant of feeding. In the case of parasitism, of course, the prey (hosts) do not disappear from the pool available once they are parasitized, and remain to be super-parasitized (unless the species in question avoids super-parasitism in its oviposition behaviour, in which case the model will be of the predation form with an allowance made for the time spent in avoidance). In other words, models of predation must account for the reduction in prey density DURING the time-period modelled.

The number of prey in a unit area of habitat affects the attack success of a given number of predators in two ways. First, when prey are very common, the time it takes for a predator to search for a prey item becomes relatively low. This means that the number of attacks per day can reach the maximum possible, which is determined by the handling time per capture, and by the gut capacity of the individual. These parameters will undoubtedly be inter-related over the course of evolution such that at high prey densities the handling time necessary to capture sufficient prey for satiation will be considerably less than the total hunting time available (otherwise the predator would invariably starve !). This means that at high prey density we should observe an attack

rate of

$$KILL = PREDS * INTAKE \quad \ldots\ldots (14)$$

if the predators only kill as many prey as they consume. In the situation defined, we have INTAKE (the number of prey required per predator per day) independent of PREY, and affected only by the age, sex, size and physiological condition of the predator; i.e.

$$INTAKE = f\ (AGE, SEX, SIZE, etc.) \quad \ldots\ldots (15)$$

As prey density decreases, however, it becomes progressively more difficult for each individual predator to find prey items. The searching time will therefore increase as prey density decreases until a point is reached at which there is insufficient time to search for and capture enough prey to fill the gut. We can now state that under these conditions

$$INTAKE = f\ (\ AGE, SEX, etc\ ),\ g\ (SEARCH) \quad (16)$$

where SEARCH is a measure of the searching time per encounter (specifically, the fraction of the hunting time spent in search). To observe this effect, we can plot SEARCH as a function of prey density (PREY) as in Fig. 121, bearing in mind that when SEARCH is less than a threshold (TS) the prey intake rate will be unaffected (above). The curve will tend to be of an hyperbolic form with

$$SEARCH \propto \frac{1}{PREY}$$

If this is the case, then the relationship will be asymptotic to both axes, and it will be necessary to specify a threshold of prey abundance below which all hunting time is expended

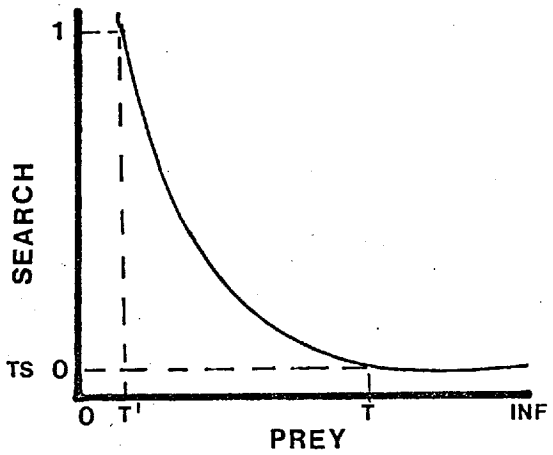


Figure 121. The relationship between prey density and the fraction of hunting time spent in search

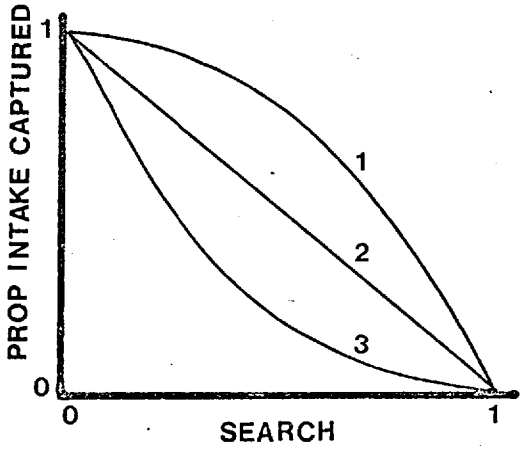


Figure 122. The relationship between the fraction of hunting time spent in search and the proportion of the food requirement which is fulfilled for three types of predator (see text)

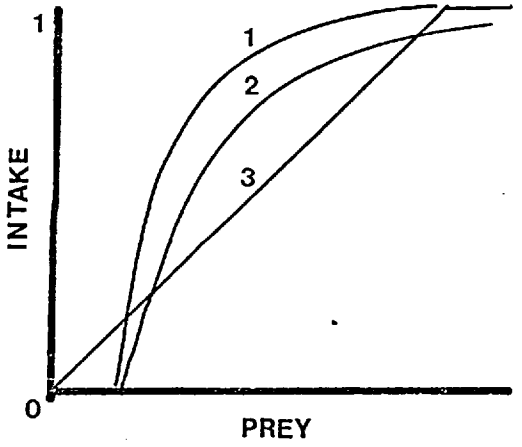


Figure 123. The functional responses of the three predators to prey density obtained by combining the two graphs above

in searching (T', at which point SEARCH = 1); the threshold reflects the relationship between the hunting efficiency of the predator and the area over which it must search. Similarly, the asymptote as prey become very abundant reflects the limit at which each predator spends no time at all in search, and the number of prey killed is limited by the capacity of the gut. If we now suggest that below prey densities of T' search is completely unrewarding, we can write

$$\text{SEARCH} = T' / \text{PREY} \qquad \ldots\ldots (17)$$

and allow that when SEARCH < TS feeding is unaffected by prey density (so that in Equ. 16, g(SEARCH) = 1).

The function g(SEARCH) in Equ. 16 will clearly decrease with SEARCH, since it is not possible to increase the rate of attack as searching time increases relative to total hunting time (it is possible to model this process, but it implies having the searching efficiency determined by prey density). To determine which particular shape of function best describes this relationship, we can examine three possible curves (Fig. 122).

The usual representation of the functional response to absolute prey density is given as the number of prey killed against the number of prey. This is equivalent to plotting g(SEARCH) against PREY, since g(SEARCH) is simply the fraction of the maximum possible prey intake which is killed at any given prey density. In Fig. 123 we plot the functional response curves derived by adopting each of the three forms for g(SEARCH) (from Fig. 122) in turn, and assuming an hyperbolic relationship between SEARCH and PREY (Fig. 121).

The shape of the response chosen in Fig. 122 determines how the predator species reacts to changes in prey density.

Curve 1 represents an animal which can respond to increases in the time it takes to find a prey individual either by increasing its rate of search, or by searching more efficiently. In consequence, the food intake rate drops rather slowly with decreases in prey numbers until very low prey densities are reached at which time the functional response curve falls steeply. This situation approximates the model adopted by Thompson (1924) which can be seen by comparing curve 1 with Fig. 120 above; Thompsons 'predators' fed at a rate which was independent of prey density.

Curve 2 describes the feeding strategy of a predator whose hunting behaviour is completely inflexible; it is unable to change its attack rate or searching efficiency as prey density decreases, and so the relationship between the fraction of the diet realized, and the proportion of time spent in search decreases linearly. In other words, at prey densities such that the searching time per encounter is doubled, the prey intake will be halved. This type of behaviour produces a functional response curve in Fig. 123 very like Holling's disk equation (1959); this is what we would expect, because the experimentors in the disc-collecting exercise did not vary their rate of search as prey density changed (in fact, since they were blindfold, they could not tell whether prey density had been reduced).

The last response we can consider is that in which each unit increase in search-time produces a lower reduction in food intake, so that the curve falls steeply at first. Such a response would be observed in a predator whose presence in an area depended upon there being rather high prey densities, or by a predator species whose individuals were debilitated by starvation. In this latter case, each day spent in sub-optimal

prey densities would decrease the animal's performance on the following day. The functional response curve for this behaviour pattern increases linearly with prey density when curve 3 is an hyperbola, because Figs. 121 ans 122 cancel to give a straight line (SEARCH = T'/PREY; fraction of diet = G/SEARCH; therefore fraction of diet = G/T'/PREY which is to say PREY * K where K = G/T'). In general, we could express the relationship shown in Fig. 122 in terms of two parameters, and write

$$F(SEARCH) = 1 - SEARCH^{v}$$

where v is the coefficient of hunting strategy. Then if $v > 1$ we have a response like curve 1; $v = 1$ a response like curve 2; and $v < 1$ a response such as curve 3. Two parameter equations of this type are widely used in predation models (e.g. Hassell and May, 1973; Rogers, 1972), and for visualization of the equations which follow, a family of curves of the form $y = x^{b}$ are shown in Fig. 124, and $y = 1 - x^{b}$ in Fig. 125. The values of the power coefficients can be obtained from data by a regression of $\log_{10} y$ on $\log_{10} x$; the power b is then the slope of the graph.

We can now restate this model of the functional response to prey density in the format employed by other ecologists. The food requirement is calculated as the product of the number of predators (P) and their individual food requirements (C); therefore P.C is the total number of prey which would be taken in optimal conditions. When prey density is low, only a fraction of this number will be taken, determined by the mean searching time per encounter, f(N), and the response of the food intake to this searching time g(f(N)); so

$$N_a = P.C\ (\ g(f(N))\ )$$

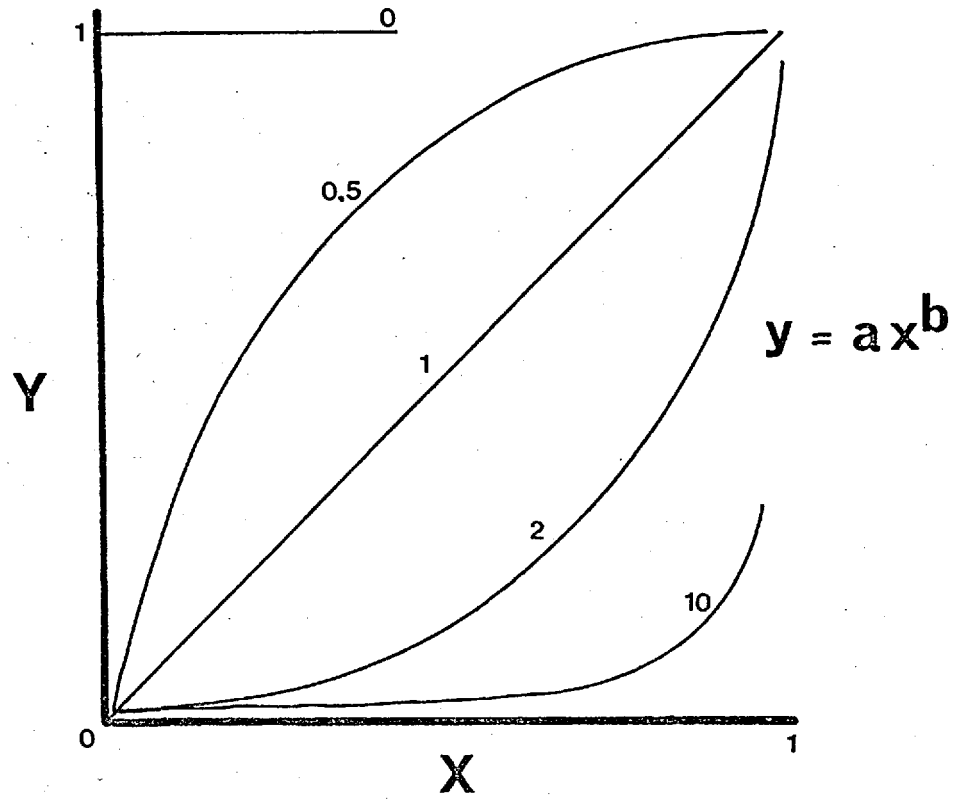


Figure 124. Some examples of curves in the two-parameter family with y an increasing function of x. The b values giving rise to the particular curves are shown.

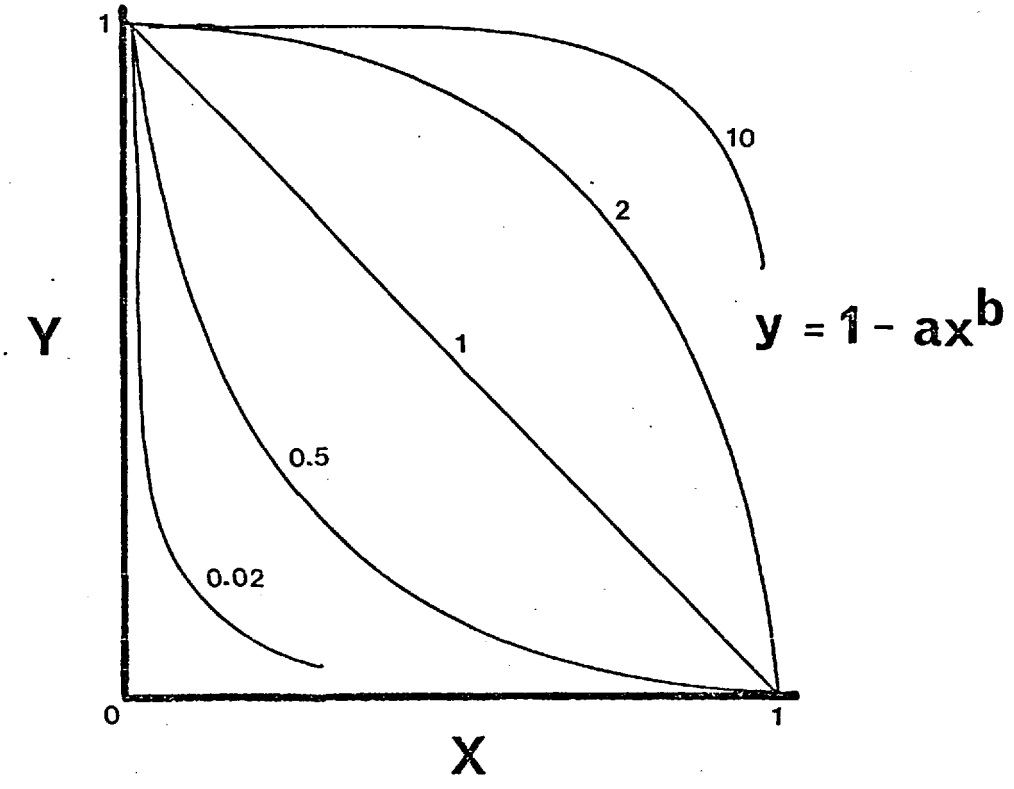


Figure 125. As above, but with y a decreasing function of x

and we can specify f(N) as an hyperbola (above), with a constant T' being the prey density at which all time is spent in search and no kills are made. This, of course, will only be an expectation in a stochastic process, because as long as $N > 0$ there will be a finite probability that an encounter will occur. Now setting f(N) to T'/N we have

$$N_a = P.C\ (\ g(T'/N)\ ) \qquad \ldots\ldots (19)$$

We saw that the fraction of the diet taken could be expressed as a two parameter formulation of T'/N, so the equation can be completed by writing

$$N_a = P.C\ (\ 1 - (T'/N)^{v}\ )$$

Altering T' will change the prey density at which the functional response curve cuts the prey axis (i.e. there is no feeding), while changing v will alter the shape of the functional response curve as it increases with prey density.

2 Functional Responses to Relative Prey Density

Relative prey density, the number of prey per predator (N/P) also has a functional response associated with it. Consider the case in which prey are abundant, and therefore searching time is non-limiting in determining the number of prey attacked. In this case we have seen that the intake of prey should be PREDS * INTAKE. But if we now allow that predators are also very abundant we might arrive at the absurd situation that PREDS * INTAKE > PREY. In this case, it will not be the difficulty in finding a prey organism which limits the number of prey killed, but rather the difficulty of finding a prey which is not already being pursued by a predator.

Clearly, in this situation, the predators will have to compete with one another for the prey which are available. This competition, in turn, will lead to a reduction in the mean INTAKE per animal, and to a reduction in the total number of prey attacked.

Competition between predators at low relative prey densities will operate in an homogeneous population (where all individual predators are alike with regard to their food requirements and competitive ability) simply to reduce the mean intake per predator. Exploitation of the prey population will make the situation increasingly severe so that, in the next time interval, the relative prey density is even lower. Two factors could act to ameliorate the situation, however. First, if prey reproduction is high relative to the food requirements of the predator population, and more than matches any increase in predator numbers over the same period, then competition will be reduced. Second, the predators may respond to competition by decreasing their abundance either through dispersal of the active stages, or increased mortality amongst the more sedentary immatures. This latter response is dealt with in the next section, under Numerical Responses.

If the predator population is heterogeneous, and the animals differ from one another in size and voracity, the situation will be modified. In this case, certain classes of the predator population will be relatively more competitive than others, and any food deprivation resulting from competition will be unevenly distributed over the size classes. We might suggest that success in intraspecific competition for prey increases with size and mobility, so that the older animals can fend off the advances of smaller predators for the prey item being pursued. In this case, the model would have to

compute a weighting factor describing the distribution of unit food deprivation between the instars. A simple model of this phenomenon can be given by writing D as the total prey deprivation (the number of prey which would have been captured had prey density been optimal, over and above those actually captured), and dividing it between the predator size classes in relation to their abundance, P_i, and the weighting factor d_i. Now, assuming that the abundance of the size class and the weighting factor interact (so that a small group of large predators suffer proportionately less than a large group of small predators) we can write the actual food deprivation of the ith size class as

$$FS_i = D \frac{P_i \cdot d_i}{\sum_{1}^{n} P_j \cdot d_j}$$

The growth of the individuals in the ith size class can then be related to food shortage (FS_i).

We require an equation relating the number of prey attacked to the intensity of intraspecific competition. We have already defined this competition as a decreasing function of relative prey density. Now, expressing competition as the fraction of the total food demand which is realized, and representing this by E we have

$$E = f(N/P)$$

and, if we can define a limit in the ratio of prey per predator at which feeding approaches zero ($E \rightarrow 0$) we can specify that f(limit) = 0, and defining an upper limit at which feeding is unaffected by competition we can put f(upper limit) = 1. To do this, it is more meaningful to express the ratio as the

number of prey relative to the number of prey required as food.

$$\text{Ratio} = N / (P.I)$$

where I is the food demand per predator. Clearly, when this ratio is less than or equal to unity, all the prey would be consumed if no competition occurred. We require a graph of E in terms of Ratio (Fig. 126); we have defined that at an upper limit in Ratio (R') E approaches unity, and that at a lower limit feeding approaches zero. This lower limit must be carefully considered, since, as stated at the outset, prey may be abundant in absolute terms, and it would be unrealistic to suggest that none are consumed simply because the predators are competing with one another. To counter this, let the graph pass through the origin, so that no feeding occurs when and only when there are no prey. In this case, of course, the first functional response would have modelled the phenomenon.

The simplest model is a linear relationship implying that intraspecific food competition occurs at a constant rate (curve 1 in Fig. 126). It may be, however, that competition is at first relatively light, but increases in intensity with decreases in the ratio of prey to prey required (curve 2). For these two graphs we arrive at terms for E as

$$\begin{aligned} E &= \text{Ratio}\ (1/R') && \text{for Ratio} \quad R' \\ E &= 1 && \text{for Ratio} \quad R' \end{aligned} \qquad \ldots\ldots (21)$$

or, for the second case we might write

$$E = 1 - e^{-(b/\text{Ratio})}$$

where b is a positive constant.

The general equation is then

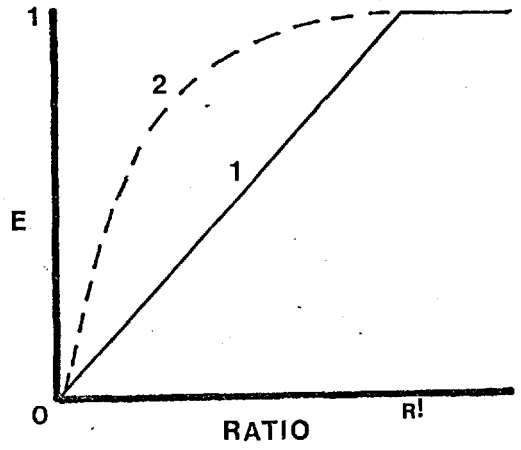


Figure 126. Two functional response curves for the effect of relative prey availability (RATIO) on the fraction of the diet fulfilled (E); see text

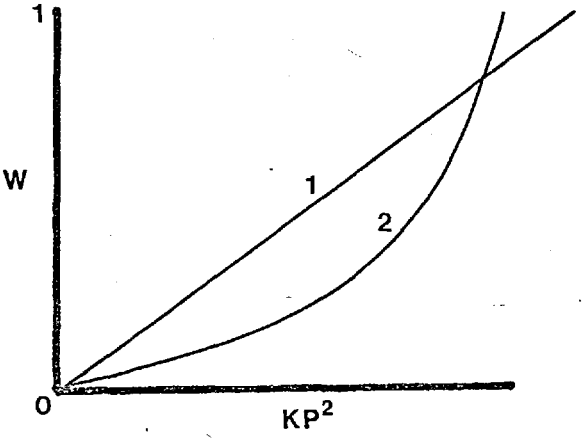


Figure 127. Two functional response curves for the effect of intraspecific predator contacts (KP) on the fraction of hunting time wasted, w

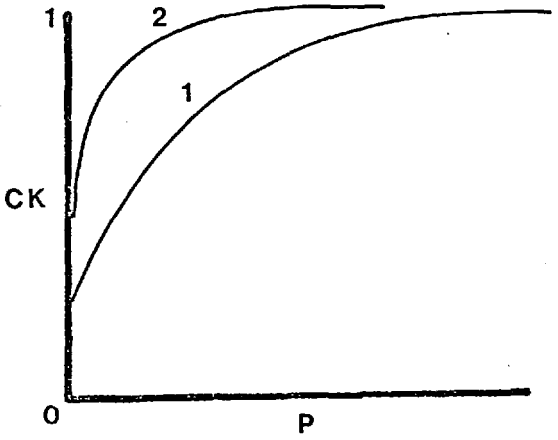


Figure 128. Two functional response curves for a predator species which fed more efficiently in the company of others. In this case intake rises as prey density increases

$$N_a = P.C.E$$

where the equation for E is of the form best fitting the data.

3 Functional Response to Predator Density

The final case which must be considered as a component of the functional response is the effect of high absolute predator densities. One of the possible mechanisms of this response has already been suggested in the introduction, in discussing the mutual interference between adult parasites (Watt, 1959; Hassell and Varley, 1969; Hassell and Rogers, 1972). In this instance, the time during which the parasite and host populations are exposed to one another depends upon parasite density, since time is wasted by adult parasites following intraspecific contacts. This effect operates independently of host density. The exception to this independence occurs in models which account for the distribution of parasites in relation to host density, so that differences in host density between one area and another can lead to increases in the number of intraspecific contacts in those species which tend to aggregate over higher relative prey densities (below). This time-wasting effect could equally well apply to larval forms if they exhibited any form of avoidance or combative behaviour with others of their own kind.

Another mechanism which could act to much the same end has been described for Bupalus piniarius by Gruys (1971), in which nocturnal contacts between larvae lead to reduced growth and subsequent fecundity. This process operates at looper densities well below the level at which food competition might occur, and could act, therefore, as a preventative mechanism reducing the probability of over-exploitation.

In this type of functional response we allow that

$$N_a = P.C.\ f(P) \qquad \ldots\ldots (23)$$

so that, in general, f(P) decreases with increasing P. Hassell and Varley's (1969) approximation for this function was $Q.P^{-m}$ (described in the introduction). If the predators contact one another due to random motion, then the number of contacts should be proportional to P^2. That is, putting s as the number of non-reproductive intraspecific predator contacts,

$$s \propto P^2 \qquad \ldots\ldots (24)$$

Following on this, it is necessary to specify the shape of the function relating the number of contacts to their effect on hunting activity. The simplest model would again be linear; i.e. each contact contributing equally to the reduction in feeding. In this case we should have a constant interference component representing the fraction of hunting time lost per encounter; we can call this J. Therefore

$$W = J.s$$
$$= J.K.P^2 \qquad \text{(from 24)}$$

where W is the fraction of hunting time lost.

If the response is non-linear, then we must graph W against $K.P^2$ (Fig. 127). If behaviour was such that the first encounter was the most traumatic, after which the animal became used to meeting its fellow predators, W would decrease with KP^2, perhaps in an exponential manner. More likely, however, would be the case in which each encounter reinforced the detrimental effects of the last (curve 2 Fig. 127). In any event, we shall have

$$W = g(K.P^2)$$

and hence

$$N_a = P.C\ (1 - g(K.P^2))$$

or

$$N_a = P.C\ (1 - (K.P^2)^w) \qquad \ldots\ldots (25)$$

where w is the coefficient of interference time-loss.

The functional response to predator density may not give rise to a reduction in food intake with increasing predator numbers in all species. Over the lower ranges of density at least, many predator species are aggregatory, or hunt in packs. Here, the functional response curve would be as in Fig. 128; curve 1 would be the curve for a species which needed many neighbours to allow feeding to occur at its maximum rate, while curve 2 describes a species whose feeding only decreases at very low predator densities.

In summary, there are three aspects to the functional response; the effects of low prey density upon the searching time of individual predators, the effects of low relative prey density on intraspecific food competition between predators, and the effects of high predator density on mutual avoidance behaviour. The conditions under which these three types might operate are listed in Fig. 129.

In the simulation model which follows, allowance has been made for the operation of these different aspects, and for a consideration of the effects which might result from the consequent under-nutrition. The situation differs here again between predators and parasites; a predator which does not catch its quota of prey will be hungry and, over a period of time, will become debilitated. A parasite, on the other hand, is less likely to be affected in this way by being unable to

PREY DENSITY	PREDATOR DENSITY	TYPE OF FUNCTIONAL RESPONSE
LOW	LOW	1, 2
MEDIUM	MEDIUM	2
HIGH	HIGH	2, 3
LOW	HIGH	1, 2, 3
MEDIUM	HIGH	2, 3
HIGH	LOW	-
LOW	MEDIUM	1, 2
MEDIUM	LOW	-
HIGH	MEDIUM	2

1 = Response to low prey density (N)

2 = " " low relative prey density (N/P.C)

3 = " " high predator density (P)

Figure 129. The conditions of prey and predator density under which the three types of functional response might be expected to operate.

lay all its eggs.

The three responses can be expressed as a single equation for comparison with previous models. In the absence of exploitation, the number of predator prey encounters would be

$$N_a = P.C.\ (1 - (T'/N)^{v})(1-e^{-b.P/N})(1 - (K.P^{2})^{w}) \qquad (26)$$

and, assuming random search we can follow Rogers (1972) and Royama (1971) and calculate the number of prey killed from

$$N_{ha} = N\ (\ 1 - e^{-(N_a/N)}\ )$$

Numerical Responses

Despite the extent of the literature on functional responses, this aspect of predator behaviour can only exert a minor influence on the biological control potential of a species. When prey density is high, as we have seen, each predator will feed at a maximum rate determined by the capacity of its gut and the physiological factors which affect its rate of intake (temperature, food digestibility, body size, and the like). While the functional responses may act to lend stability or permanence to the interaction at low prey densities, it is the ability of the predator species to respond to changes in prey density by altering the size of its own population which will ultimately determine its potential in biological control.

We can isolate three mechanisms by which numerical responses might occur, and deal with each in turn. These are the effect of increased prey density upon enhanced survival (particularly of the immature stages) and increased fecundity (with more food available for egg production), and the effect of increased prey density upon the dispersal behaviour of the predator (less tendency to emigrate, or an increased rate

of immigration into areas of high prey density). It will also be necessary to determine the extent to which the functional and numerical responses interact, since, in the case of survival and reproductive rates, it is the degree of starvation (the intensity of the functional response) which depresses the rate of increase in the predator population.

1 Survival

In general, numerical responses due to changes in survival rate can be visualized as acting to increase the probability of death as the degree of starvation increases. If we define relative starvation as being a function of the fraction of the food intake realized (see Functional Responses, above) as in Fig. 130, we can use the value of STARVE to determine the magnitude of the numerical response. This approach is based on the assumption that numerical responses can not occur unless predator and prey densities are such that the actual food intake per predator has been reduced because of functional responses of one type or another. Survival rate changes under the assumption that predators weakened by starvation are more likely to fall to diseases, accidents and natural enemies.

The relationship between relative starvation (STARVE) and survival rate can now be supplied graphically, plotting the proportional mortality due to starvation-related factors on the y-axis (Fig. 131). Given any value of STARVE from Fig. 130 the new survival fraction can be computed by interpolation (Chapter I), and the actual daily survival computed from

$$SUVACT = SUV * F(STARVE) \quad \ldots\ldots (27)$$

where SUV is the survival rate with full satiation. The shape

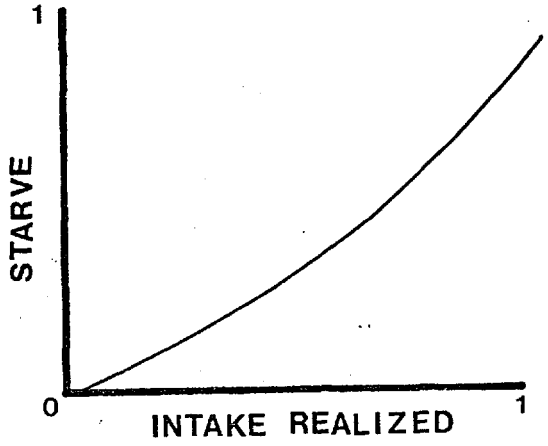


Figure 130. The relationship between the relative intensity of the functional responses (the fraction of intake realized) and the relative starvation resulting

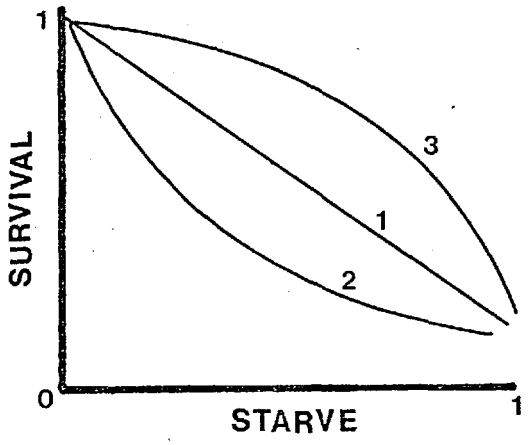


Figure 131. The relationship between relative starvation and survival rate for three types of predator (see text)

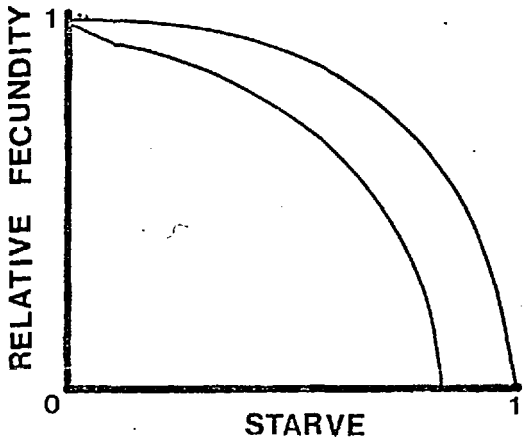


Figure 132. The relationship between relative starvation and relative fecundity for two types of predator (see text)

of curve employed in Fig. 131 will reflect the sensitivity of the predator species to food shortages; curve 2 would apply to a 'starvation-sensitive' predator, while the type shown as curve 3 would be capable of withstanding high starvation before becoming prone to increased mortality.

2 Reproduction

Starvation can also affect the rate of egg production by adult predators (Holling, 1965; Huffaker and Kennett, 1969). As in the preceeding section, we can plot relative egg production against the degree of food shortage (Fig. 132), and obtain the actual value by interpolation.

If the egg-laying rate of adult predators varies with their age, and if the maximum oviposition rate on the Kth day of adult life is FEC(K), then the actual rate will be

$$EGGS = FEC(K) * F(STARVE) \quad \ldots\ldots (28)$$

where F(STARVE) is less than or equal to one. This process means that as prey density decreases the number of predator eggs laid is reduced, and if the predators take a finite time from egg laying to the initiation of feeding, then there will be a time-lag between the numerical response of the egg layers to reduced prey density, and the effects of this reduction in terms of lower attack rate. Depending on the duration of the non-feeding egg stage, and the time taken to develop to the size of maximal prey intake, this time-lag will have more or less profound implications. If the period is relatively brief, the abundance of the predator population should follow the pattern of prey abundance rather closely, while if the period is extended, the predator population may become seriously out of phase with its prey. In the latter case, there will be too

many predators when prey are scarce, and too few when prey are abundant, so prey density will tend to vary between rather extreme limits.

3 Dispersal

The third way in which the predator population can change in abundance as a function of prey availability is through dispersal. Predators which have been unable to find sufficient prey to satiate themselves may, instead of staying in the area and starving, depart in search of other areas where prey density is higher. This dispersive response can act immediately to changes in prey availability (when the predator leaves as soon as it can not consume its fill), or after a time-lag (in those cases where dispersal is dependent on the achievement of a certain threshold in starvation). In any event, the effect of this aspect of the numerical response on the future interaction between predators and prey will depend on the particular life-stages of the predator given to dispersive behaviour. For example, only the adults of insect predator species have wings, and if they are prone to migrate under conditions of low prey availability, they can travel considerable distances (Johnson, 1969). Dispersal in the egg and pupal stages can only be passive, and the movement of larvae over long distances may be fraught with difficulties (low mobility, exposure to predators, dessication and so on). In most cases it will be possible to rank each developmental stage of the predator in terms of its tendency to disperse under conditions of low prey availability; generally the adults will be most responsive, followed by the larval stages in decreasing relation to their size. The fraction of each instar leaving the area during a specified time-interval

can then be set as a function of relative starvation, and so the number of predators in the Jth instar remaining will be

$$PREDS(J) = PREDS(J) * F_j(STARVE) \quad \ldots\ldots (29)$$

where the shape of the F_j reflects the different tendencies of each instar to disperse (Fig. 133).

The Behaviour of Predator-Prey Models

Models of predator-prey interactions tend to behave in one of three ways, depending upon the structure of the model and the initial conditions which are supplied;

1: the prey population becomes extinct through over-feeding by the predator population, which, in the absence of food, becomes extinct itself;

2: the prey population, because of under-exploitation and lack of density-dependent controls on its own rate of increase, becomes infinitely abundant;

3: both populations persist indefinitely.

1) Prey Population Becomes Extinct

This type of result is commonly observed in laboratory experiments on competition and predation (Crombie, 1947, for a review), essentially because homogeneous environments are used. It is very unlikely that one homogeneous environment will be optimal for two species in all respects of their biology, by merit of the very fact that the species are different. Crombie (1945, 1946) has shown that simply through making the environment spatially heterogeneous, by placing short lengths of fine glass tubing in a flour-wheat medium, two species can coexist (Tribolium confusum and Oryzaephilus surinamensis) where only one persisted in the homogeneous

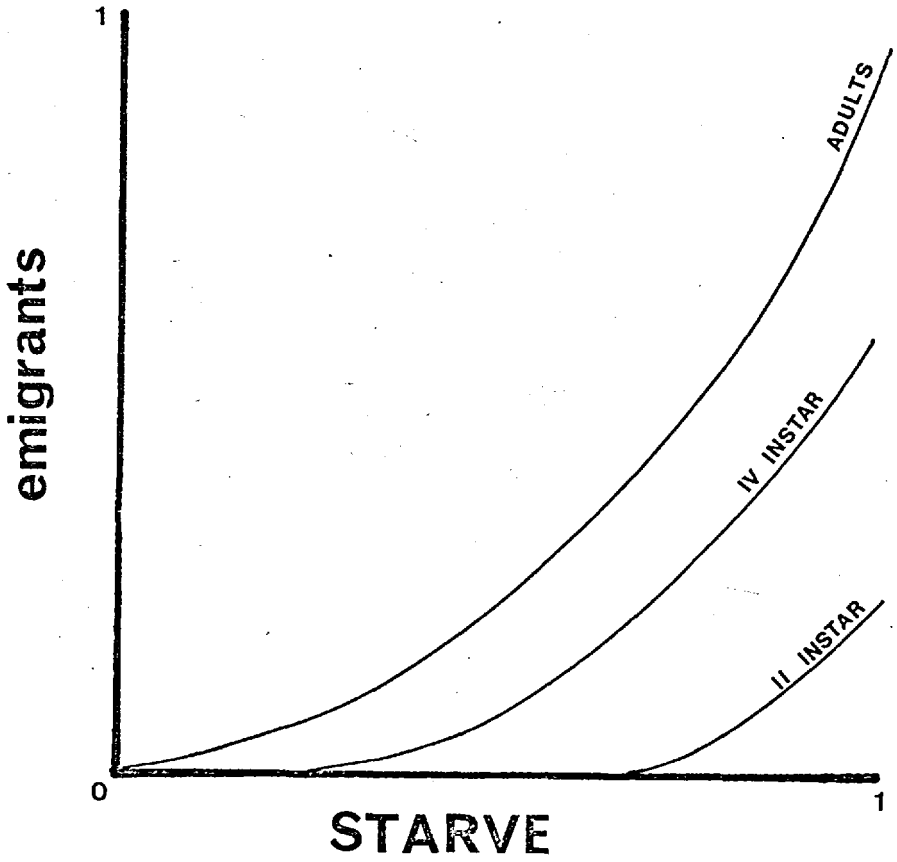


Figure 133. The relationship between relative starvation (STARVE) and the proportion of the predators emigrating per day for different predator instars

substrate (T. confusum). The explanation of this result lies in the provision of a 'refuge' for the larvae and pupae of O. surinamensis from predation by T. confusum. When larger bore tubing was used in the medium the predators were able to encounter and consume the entire competing population; the refuge was no longer effective.

Models of predator-prey population dynamics tend to behave in a similar fashion when their structure allows that the entire prey population is available to the predator. A necessary condition for the persistence of the prey (and hence of the predator) is that the last prey individual capable of reproduction must never be consumed. This can be achieved in two ways.

First, the functional responses to prey density and to relative prey density can act to minimise the probability of prey extinction. The former response can be especially effective if the rate of prey consumption falls to zero at prey densities greater than zero (see Fig. 123). The problem with including this assumption in the model is that the mechanism by which the prey escape predation is not elucidated, and we are asked to believe that difficulty in making encounter with prey organisms completely curtails feeding. I shall return to this point when discussing the running of the model. In essence, however, this approach implicitly assumes that a certain number of prey are unavailable to the predators; they are in a refuge from attack.

The second possibility open to us in attempting to prevent extinction in the prey population is the explicit provision of a refuge from predation (Crombie, 1947; Hassell and May, 1973). The refuge can be visualized in one of two ways.

Hassell and May (1973) have allowed that a particular proportion of the prey population is unavailable because of some spatial attribute of the environment which affects the behaviour of the predators but not the prey. For example, an avian predator might forage only on the outer leaves of an oak tree, whilst the caterpillars it feeds upon are distributed throughout the canopy; similarly, a parasite with a given length of ovipositor can only parasitise that fraction of a grain-dwelling host species which is in the upper layer of the substrate, and within range of the ovipositer. The disadvantage of this approach is that one must assume continual redispersal by the prey individuals so that the proportion of the prey population in the refuge is always equal to the proportion of the environment which is defined as being refuge.

An alternative method of modelling a prey refuge is to allow that the predator species has access to all the habitats occupied by the prey, but that within any habitat type there are a finite number of 'hiding places' for the prey. This number of refuges need not be constant, particularly if, for example, the refuge consists of some part of an annual plant. The essential difference between the two treatments is that the proportional refuge operates independently of prey density, while the finite refuge has a very small effect on predator-prey interaction at high prey densities and an important effect when prey are scarce. If it is assumed that the refuges are always filled, then the finite treatment can bring about a complete cessation in predator feeding when the number of prey is lower than or equal to the number of refuges. With a proportional refuge some prey are attacked however rare they become. It is probable that both types of refuge occur in nature, but for any particular interaction between two species,

one is likely to be of over-riding importance.

2 Prey Population Becomes Infinitely Abundant

Under certain sets of input conditions and constraints, mathematical models are apt to suggest results which are clearly absurd. So it is that some models of predation (e.g. Thompson, 1924) predict that the prey population can become infinitely abundant.

There are a number of cases in which natural populations of animals have increased very rapidly after a reduction in the intensity of predation, notably the Mule Deer on the Kaibab plateau (Rasmussen, 1941; Leopold, 1943) and in northern Utah (Doman and Rasmussen, 1944), and many species of pest insect after insecticide applications have eradicated their natural enemies (Stern et al., 1959), or they have been introduced into areas where no natural enemies existed (Elton, 1958; Lack, 1954). In all these cases, however, the increase in numbers has in no sense been infinite (the Mule Deer on Kaibab multiplied 25 fold in eighteen years), and it has generally been matched by an equally swift decline.

3 Both Populations Persist Indefinitely

The ideal situation for biological control is that the predator and prey (pest) persist indefinitely; this will reduce the costs associated with continual rearing and release of natural enemies, and also reduce the necessity of expensive predator monitoring schemes and the possibility of pest outbreak following an un-noticed decline in predator abundance. The critical question is what level of pest abundance is necessary to maintain a permanent predator population ? If the level is too high biological control may be economically

infeasible, and if it is very low there is a possibility that chance extinctions of either the predator or the pest may lead to subsequent pest outbreak.

The picture is further complicated when annual crop situations are considered, since permanent interaction between the predator and prey is not possible (the crop does not exist for extensive periods of time). This is especially important when the crop is grown in rotation; in this case a predator species which overwintered in the soil below the crop would emerge in the following year to discover a completely different set of prey insects. Clearly, the only viable long-term permanence in predator-prey interaction on annual crops will occur when large, reliable pools of overwintering predators exist within reach of the crop area. Coupled with this, there must be a well-tuned synchrony between predator immigration and prey abundance within the crop (Chapters III and IV).

The problem in applying most predator-prey models to the biological control situation is that they tend to consider permanent predation in a single area, while the applied situation demands a treatment of discrete predation periods in a succession of different areas. It is interesting to note that most of the successful applications of biological control have occurred with pests of perennial crops (see de Bach, 1964); situations to which the established predation models are more applicable. Whether the apparent failure of biological control in annual crops is a real indication of the improbability of the necessary conditions of pool-availability and synchrony, or simply reflects a lack of research, must remain to be seen.

The Model

The purpose of the following simulation model is to incorporate the ideas presented on the general behaviour of predator systems in tackling the question "What collection of biological properties in a predator species make it an ideal control agent for Aphis fabae on beans ?" We know from the model of aphid population growth (Chapter IV) the pattern of predation which will be most effective, and we also know the rate at which different aphid instars should be attacked. It is now necessary to investigate the relationship between the physiological parameters of the predator and its behavioural responses to prey density, and to determine that combination of attributes which defines the optimal predator for this system. The processes which operate can be demonstrated by a flow diagram (Fig. 134), linking our inputs (the biological attributes of the predator species) to the outputs (the pattern of predation defined in Chapter IV).

Unlike the models of plant growth and aphid population dynamics in the preceeding chapters, the predator model is not concerned with any particular species, but rather with defining the attributes of an hypothetical optimal predator. From these attributes it should be possible to rank actual biological control species in relation to their expected efficiency when designing field trials to assess their actual performance. Because all the biological attributes will be manipulated in assessing their effects upon the number of aphids attacked, there are no real data included in the model. All the graphs are intuitive, but they are constrained by the bounds of possibility; for example I shall not consider the potential of a species whose individuals can lay 500 eggs per day over extended periods, nor of one whose developmental

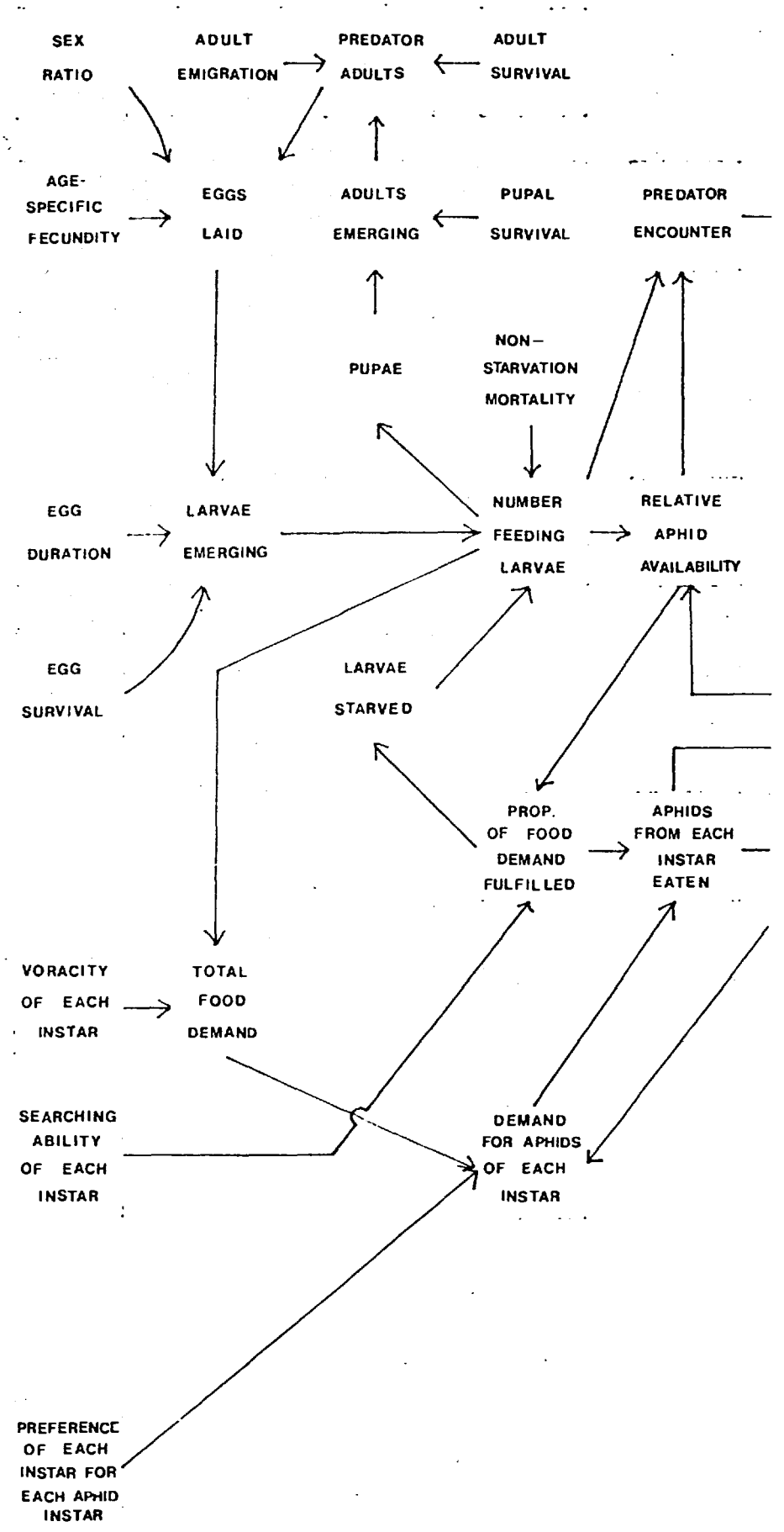


Figure 134. Flow diagram of the variables and processes included in the predation model

period is less than one week from egg to senescence. The construction of the model, however, and the means of interpretation, are precisely the same as before (see Chapter I).

The model is built in two parts. The first considers the effects of physiological and behavioural attributes of the species in determining the number of prey killed and the number of predators surviving, while the second is concerned with the effects of adult predator behaviour in determining the pattern of egg-laying between areas of different prey availability.

1 Population Growth and the Number of Prey Attacked

Consider the events occurring within a predator population during the course of one day. Let the number of predators of age I days be PREDS(I), and consider those processes acting to alter PREDS(I) within a period of one day (birth and immigration, and death and emigration).

a) Rate of Development

As with the aphid population in Chapter IV it is necessary to deal with the effects of air temperature on the rate of development of the insects from oviposition to adulthood. To do this, let NINST be the number of instars through which the predator must develop before becoming adult. Now if each instar lasts for a given number of day-degrees (Hughes, 1963; Chapter IV), we can calculate the number of day-degrees experienced by each age class of predator from birth by writing

$$DAYDEG(I) = DAYDEG(I) + TEMP \quad \ldots\ldots (30)$$

where TEMP is the mean temperature over the day in excess of the development threshold. So, for example, if a given predator could not develop below 6°C, and the mean temperature was 15°C

we should have TEMP = 15 - 6 = 9 day-degrees of development. When DAYDEG(I) exceeds a threshold we can say that the animal emerges from the egg to the first larval instar, and write

```
IF (DAYDEG(I).GE.DEGTHR(K))
then INSTAR(I) = INSTAR(I) + 1          ..... (31)
```

where INSTAR(I) is the instar in which PREDS(I) are disposed (taking a value of 1 for eggs and NINST+1 for adults). K is calculated before the conditional statement is evaluated as K = INSTAR(I). DEGTHR is a vector of accumulated day-degree thresholds marking the transition from one instar to the next. This procedure is repeated for each age class of predator, and we can compute the mean time for development from egg to adult by dividing the NINSTth value of DEGTHR by the mean temperature over the period; i.e.

```
DAYS  =  DEGTHR(NINST) / MEANT          ..... (32)
```

The duration of adult life can be specified as a constant (the approach adopted here), or allowed to be a function of temperature. In the latter case all PREDS(I) for which

```
DAYDEG(I).GT.DEATH
```

would be assumed to die of old age; DEATH is therefore the number of day-degrees from birth to death.

b) Birth and Immigration

The population of predators is initiated by allowing immigration to occur over a period of IMPER days. Before this

```
PREDS(I) = 0      for all I
```

and to simulate immigration we simply write

```
IF (IDAY.LE.IMPER)
then PREDS(I) = PREDS(I) + PIMIG(I)     ..... (33)
```

for all ages I. PIMIG(I) is the number of I-day-old immigrants on the IDAYth day, and this can be constant or variable (its value is read into the model daily). PIMIG will usually have the value 0 for eggs and pupae and other immobile stages.

Population increase also occurs by reproduction; each adult female is assumed to lay a certain number of eggs each day. If FEC(K) is the maximum potential egg-laying rate of an adult K days after its pupal moult, then the total number of eggs laid by the population will be given by multiplying the total number of fertilized females by their individual fecundity rates, and summing over all the reproductive age classes. If we let SEXR(I) be the fraction of PREDS(I) which are females, and let FERT(I) be the fraction of these females which have been successfully mated, then the maximum number of eggs which can be laid will be

$$EGGS = \sum PREDS(I)*SEXR(I)*FERT(I)*FEC(K) \qquad (34)$$

summing I over all the adult ages. In order to determine K, the age since becoming adult, we must note the day on which the PREDS(I) changed from being pupae to first-day-old adults. Recall we wrote

```
IF (DAYDEG(I).GE.DEGTHR(K)) INSTAR(I)=INSTAR(I)+1
```

so now we add

```
IF (INSTAR(I).EQ.NINST+1) IAGEAD(I) = I
```

In other words, if the animal has just become adult (INSTAR =

NINST + 1) then the age it is today (I) is the age at which it became adult (IAGEAD(I)). With this information we can calculate the number of adult days already passed by writing

$$K = I - IAGEAD(I)$$

and determine the fecundity of an adult of this age from FEC(K).

Several factors act to reduce the actual number of eggs laid to a level below the maximum potential. Principal amonst these in the present context are the effects of starvation brought about by functional responses to low prey availability (see above). This effect is incorporated by setting actual reproduction as a function of food shortage experienced by an I-day-old adult, STARVE(I). We now write

$$EGGS = EGGS * F(STARVE(I)) \quad \ldots\ldots (35)$$

where EGGS is given by Equ. 34, and the function F(STARVE(I)) takes a form as shown in Fig. 132. In this way we can make a numerical response to prey availability by reducing birth rate in relation to starvation.

c) Death and Emigration

Only a fraction of the predators alive on one day will survive to the next, and the value of this fraction may well be a function of the age of the animal. Let SUV(I) be the fraction of the animals of age I days surviving to I+1, and let the mortality factors bringing about this reduction remain unspecified. In addition, however, let survival be a function of the degree of starvation, so that numerical responses occurring through the reduction of food intake can be simulated. To calculate the number of aphids in each age class we therefore write

$$PREDS(I) = PREDS(I)*SUV(I)*F(STARVE(I))$$

assuming that starvation-related mortality acts over and above basal survival rate. This assumption will only be unrealistic at very low predator densities, when it would be more likely that the basal survival rate would be increased, with most of the individuals which would have been parasitized, predated or accidentally killed being taken from those animals destined to die of starvation or related causes.

Emigration can also act as a factor of numerical response. There is no need to consider causal mechanisms for emigration in as much detail as in Chapter IV since the predators we are considering do not undergo wing polymorphism as did the aphids. We shall simply state that the tendency of a particular instar of the predator to emigrate is related to the degree of starvation it experiences, and to its mobility (Fig. 133). For each age class, then, we write

$$PREDS(I) = PREDS(I) - PEMIG(I) \qquad (36)$$

where $$PEMIG(I) = PREDS(I) * F\ (INSTAR(I), STARVE(I))$$

d) Growth

Let the dry weight of an individual in the age class PREDS(I) be SIZE(I) gms, and assume, as in Chapter IV, that growth occurs in a logistic fashion under optimal conditions until a maximum dry weight (SIZEMAX) is achieved after the final moult.

The growth occurring during one day will then be given by the difference equation

$$SIZE(I) = SIZE(I) * EXP(GR * A) \qquad ..... \quad (37)$$

where A = (SIZEMAX - SIZE(I)) / SIZEMAX

and GR is the growth rate (a constant representing the rate at which SIZE(I) approaches SIZEMAX).

When the environment is sub-optimal (because of starvation or temperature extremes for example) growth will be reduced, and we model this by defining a parameter E to express relative environmental clemency. Plotting E against STARVE(I) and against air temperature (Figs. 135, 136) gives us an actual value for E by interpolation;

E = E1 * E2

and a new growth equation of

SIZE(I) = SIZE(I) * EXP(GR * A * E) ..... (38)

Certain of the life stages, of course, undergo no feeding or weight increase (eggs and pupae), while the weight of adults will fluctuate as they feed and lay eggs. Non-feeding instars are treated as follows;

IF (INSTAR(I).EQ.1.OR.INSTAR(I).EQ.IPUPL)
then E = 0

where IPUPL is the number of the pupal instar (from egg = 1). With E = 0 these animals will not grow (Equ. 38).

e) Feeding

The model assumes that the active stages of the predator feed at a rate determined by their body size (Fig. 137), and that their food requirement can be expressed as a number of calories, FOOD(I). We write, therefore,

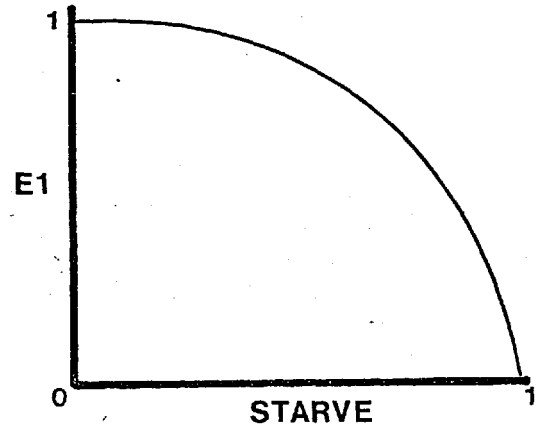


Figure 135. The effect of starvation on the relative growth rate of juvenile predators

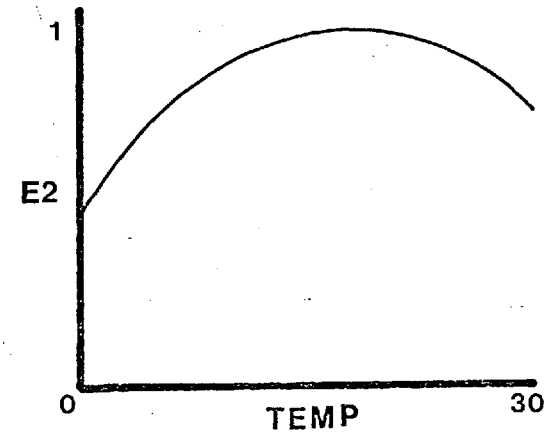


Figure 136. The effect of air temperature on the relative growth rate of juvenile predators

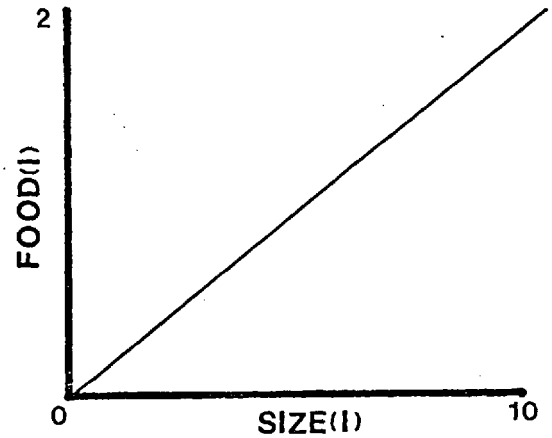


Figure 137. The relationship between the body weight of a predator and the magnitude of its food requirement (in calories) required for satiation and maximum growth

```
IF (INSTAR(I).EQ.1.OR.INSTAR(I).EQ.IPUPL) GO TO 71
FOOD(I)  =  F (SIZE(I))                          ..... (39)
```

and to determine the number of aphids required, we divide FOOD(I) by the mean number of calories per aphid eaten by predators of SIZE(I). The number of aphids required for satiation, NKILL(I), is

```
NKILL(I)  =  FOOD(I) / PREYSZ(I)                 ..... (40)
```

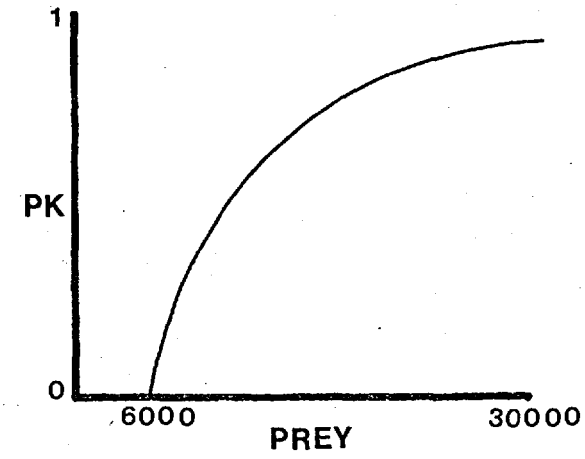
and for the inactive stages we put FOOD(I) = NKILL(I) = 0.

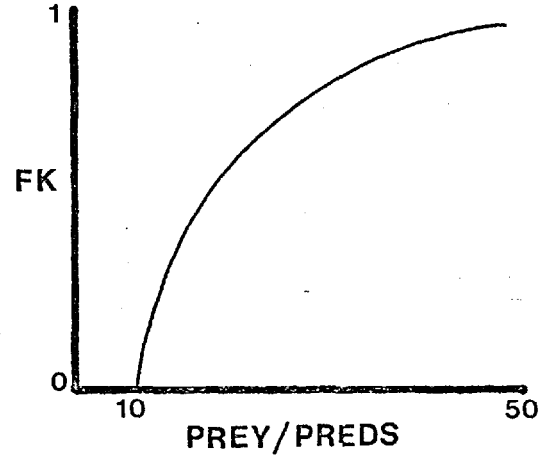
f Functional Responses

As we saw earlier, the actual number of prey eaten is often less than the number required for satiation. The functional responses which bring about this reduction can be due to low prey density, low relative prey density, or high predator density, and are caused by difficulty in finding prey, intraspecific food competition, and intraspecific interference respectively. To model the phenomenon we require three functions expressing the fraction of the food requirement which will be fulfilled under the prevailing conditions of predator and prey abundance. Let us call the potential realizable fractions of the food requirement PK, FK, and CK for the three functional responses, and plot their values as in Fig. 138. We calculate their levels by interpolation as

```
PK  =  F (PREY)
FK  =  F (PREY/(PREDS(I)*NKILL(I)))
CK  =  F (PREDS(I))
```

and we can suggest that the actual fraction of the food requirement which is realized (ACTK) will be





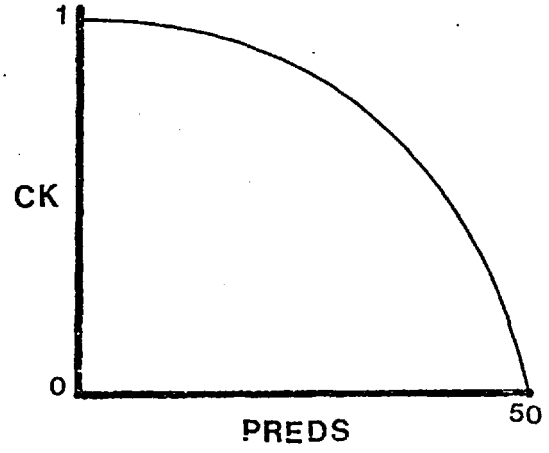


Figure 138. The three functional responses included in the model. The y-axes show the fraction of the food requirement which can be realized under given conditions

$$ACTK = \min (PK, CK, FK) \quad \ldots\ldots (41)$$

We assume, in other words, that the three responses do not interact with one another which seems reasonable, since they are simply three sides of the same problem: how many of the food items required can be caught in the time available ?

The actual number of prey which would be encountered in the absence of exploitation will now be given by

$$ENC = ACTK * NKILL(I) \quad \ldots\ldots (42)$$

and the total aphid-consuming potential of the Ith age class is

$$KILL(I) = PREDS(I) * ENC \quad \ldots\ldots (43)$$

The next problem which must be considered is the way in which the predators exploit the prey; we must decide how the different age classes of predator compete with one another, and how the effects of prey depletion affect the number of aphids killed per predator age class. Competition is dealt with in a straightforward fashion in the model by assuming that the largest predators get 'first pick' of the aphids, and the smallest (the young first instars) the last. We assume, in other words, that the predator population exhibits ranked exclusive competition, with one age class eating its fill before the next age class feeds. This is a gross simplification of reality, of course, because all instars feed more or less continuously with one another, but its purpose is to mimic the relative responses of different age classes to food shortages. If we wish to consider a different competitive ranking (with, say, the youngest adults as the most competitive stage), we simply rearrange the order of computation.

Prey exploitation is modelled by assuming random search

by the predators, and allowing that the actual number of aphids captured is

$$N_{ha} = N \left(1 - e^{-Na/N} \right) \qquad \ldots\ldots (44)$$

as explained in the Introduction. Transferring this to the model notation we have

```
P  =  KILL(I)
P1  =  PREY * ( 1. - EXP( -P / PREY ))      ..... (45)
```

and the corrected number killed will be P1 rounded to the nearest integer; i.e. AKILL(I) = P1 + 0.5. The realized calorific value of the diet is then

```
EATEN  =  AKILL(I) * PREYSZ(I)              ..... (46)
```

and the degree of starvation (used in all the numerical responses) can be calculated as

$$\text{STARVE}(I) = 1 - \frac{\text{number eaten}}{\text{number required for satiation}} = 1 - \frac{\text{AKILL}(I)}{\text{NKILL}(I) * \text{PREDS}(I)}$$

Determining the feeding for each of the day classes in decreasing order of size we write

```
      DO 7 J = 1, MAXAD
      I = MAXAD - J + 1
      ......
      compute values for AKILL(I) and STARVE(I)
      ......
      PREY  =  PREY - AKILL(I)
      ......
    7 CONTINUE
```

which means that each succeeding age class experiences a lower

prey density. This implies that in cases where prey density borders on the limiting, the young may experience food shortage while the adults are satiated, and that the functional responses experienced by the young are always stronger than those experienced by the old.

When all feeding life-stages have been considered we can compute the total number of prey killed by summing AKILL(I) over I to give us the principal output for the model. The prey population can then be considered; its dynamics are simulated very simply in this model, since a detailed appraisal has already been made (Chapter IV). Only two variables are included; the net reproductive rate, PREP, and the size of the prey refuge, REFUGE (see above). Only aphids in excess of the number in the refuge are available for predation, so the total prey population size PREY1 is given by adding the refuge to the prey left after predation; i.e.

PREY1 = PREY + REFUGE

PREY1 is then the reproductive prey population, which will increase in size to

PREY1 = PREY1 * PREP

in the time interval. At the beginning of the next day there will therefore be

PREY = PREY1 - REFUGE

aphids available to the predator population. If we wish to make the refuge proportional (Hassell and May, 1973; and above), we simply write

REFUGE = PREY1 * HID

where HID would be the fraction of the prey population out of reach of the predators.

Updating

It now only remains to update the predator population attributes before simulating the events occurring during another day. This is done in precisely the same way as with the aphids in the previous chapter (see page 233).

2 Spatial Effects

We can use the preceeding model to determine the effects of the functional responses, and to investigate the relationship between the reproductive biology of the predator and the numerical pattern of prey attacked. It is also of interest, however, to suggest the ways in which out optimal predator might respond to patchy prey distributions, since it is known (Hassell, 1968, 1969) that several parasites aggregate in areas of high host density, and some coccinellid predators behave in a similar fashion (Hagen, 1966).

We can model this process by assuming that the predators are mobile and can distinguish the desirability of different areas in terms of the availability of aphid prey within them. Another section to the model is therefore required; we need to consider the population dynamics of the prey in a number of areas, and the dispersive behaviour of the predators between these areas as relative prey density changes.

Consider an area of crop, A, whose extent is defined by the behaviour of the adult of the predator species in question. Assume that any adult can effectively search the area A during the course of one day, and relate its pattern of oviposition

and feeding (if the adults do feed) to the prey abundances which it discovers. Now allow that the predator larvae are less mobile than the adults, and define an area B which can be searched by a feeding larva in one day. The area A can now be divided into N sub-areas where

$$N = A / B$$

and

$$A = \sum B_i$$

It is known that the larvae of many predator species are more mobile when prey availability is low (Wellington, 1957; Smith, 1966), so we can suggest that

$$B_i = f(PREY_i)$$

where the function might take the form shown in Fig. 139. For the moment, however, let us consider the case in which all the sub-areas B_i are the same size, an assumption equivalent to defining the extent of the B_i by the maximum searching rate of the larvae.

Now for each area B_i let there be a distinct prey population, and a distinct population of immature predators, including the egg and pupal stages (if they occur), represented by a unique model like that outlined in Section 1, above. In addition, let there be a population of adult predators which is confined to the area A but which can move freely amongst the areas B_i. Assume that within any B_i the aphids are distributed at random, and that differences in prey availability are appreciable only between the sub-areas.

Let the total number of adult predators of age K days be PN(K). Their individual dry weight can then be represented by

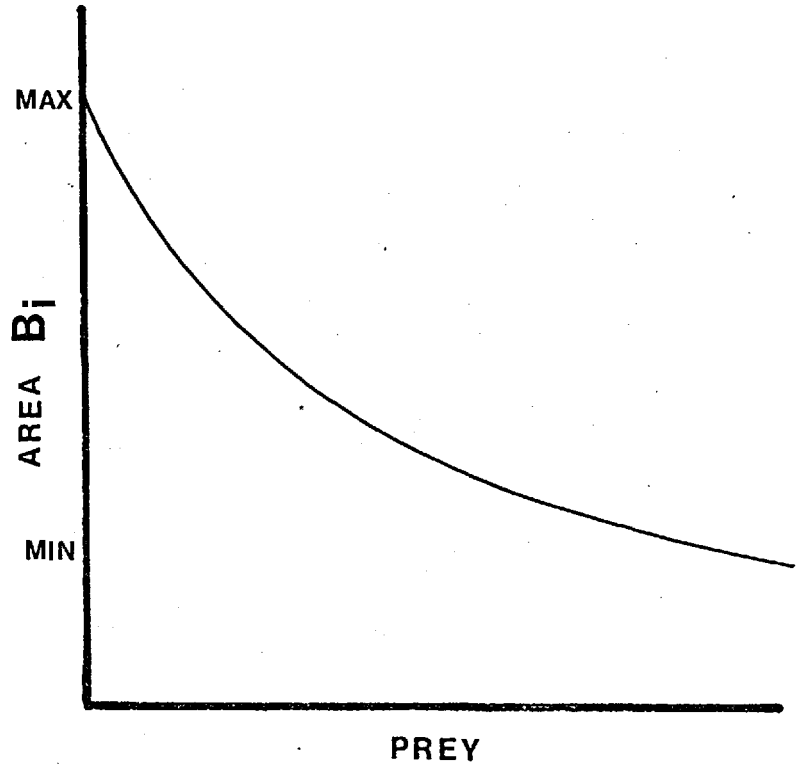


Figure 139. The relationship between the area searched by larval predators (Bi) and mean prey availability

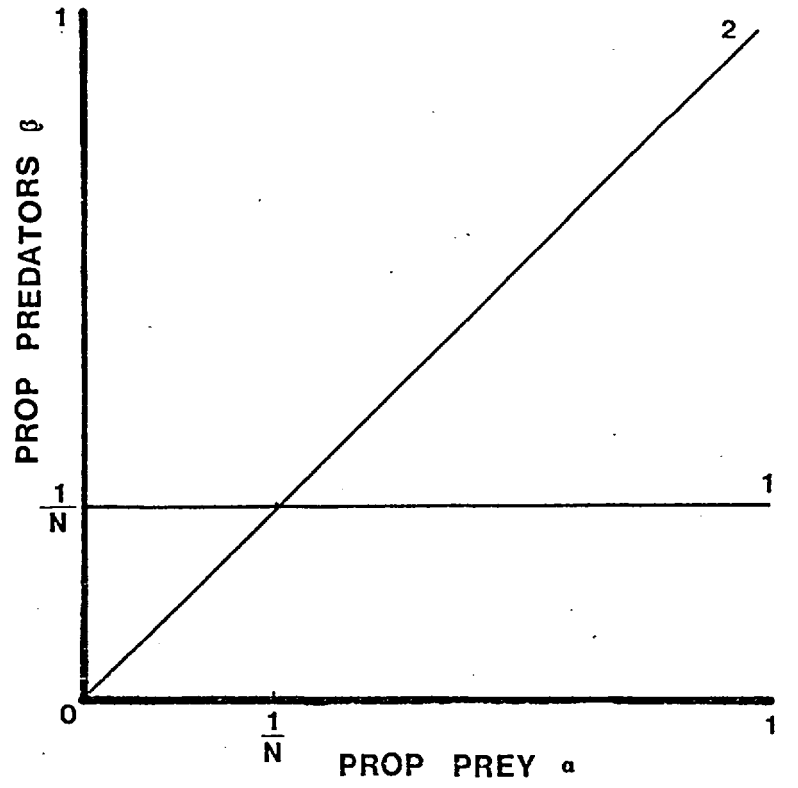


Figure 140. Two relationships between the proportion of the prey population in a sub-area and the proportion of predators attracted

SIZEAD(K), which changes as some function of the number of eggs laid, and the number of prey eaten (if any). Animals enter the adult section of the model in two distinct ways. Initially, all recruitment is by immigration from adjacent areas - from overwintering sites for example - but later recruits enter the adult stage from the larval sub-populations which originated from the eggs of the immigrants.

The object of the dispersal section of the model is to distribute oviposition and feeding over the areas B_i in relation to the relative availability of prey in each sub-area. So let the distribution of aphid abundances be PREY(I), where I is the sub-area (I = 1,2,...,N). The total prey available in the area A is therefore

$$\text{TOTPREY} = \sum_{1}^{N} \text{PREY(I)} \qquad \ldots\ldots (48)$$

and the mean availability per sub-area

$$\text{AM} = \text{TOTPREY} / \text{N}$$

Now, as discussed earlier, many predators lay their eggs in relation to prey availability such that at low prey densities the eggs are relatively aggregated in areas of higher prey density, while at high overall densities the eggs are more evenly distributed (Hassell, 1968, his Fig. 2; Hassell and May, 1973).

This dispersive behaviour can be most clearly understood by plotting the proportion of the adult predator population in a particular sub-area against the proportion of the total prey in the sub-area. Fig. 140 shows two simple cases; the first, in which predators are distributed independently of relative prey density, so that given N sub-areas the fraction

of predators β_i is 1/N for all i, and the second case which assumes that the predators respond directly to relative prey density so $\beta_i = a_i$ for all i. We might expect that the first case would be approximated at very high prey densities, and the second when prey were rather scarce. The model is not particularly general in this form, and we really require a continuous function describing the degree to which the predators aggregate in terms of the mean density over all the sub-areas (AM). To do this, we can make β_i a curvilinear function of a_i by creating another parameter μ to describe the degree of aggregation; we then write

$$\beta_i = \frac{1}{\sum a_i^{\mu}} \cdot a_i^{\mu} \qquad \ldots\ldots (49)$$

in which the term $1/\sum a_i^{\mu}$ normalizes the values of β_i so that $\sum \beta_i = 1$ ($\sum a_i = 1$ by definition). This equation shows that when $\mu = 1$ then $\beta_i = a_i$ as before, and when $\mu = 0$, $a_i^{\mu} = 1$ so $\beta_i = 1/N$ again, as shown in Fig. 140. The curve of β_i on a_i for values of μ greater than 1 ($\mu = 2$) and less than 1 ($\mu = 0.5$) are shown in Fig. 141, and correspond to aggregation greater than and less than curve 2 in Fig. 140.

From Fig. 141 it is clear that as μ increases, so the proportion of the predator population in relatively high prey density areas increases. The limit, when μ is infinitely large means that all predators aggregate in the one sub-area of highest prey density. In order to mimic the effects reported by Hassell (1968) we need only plot the value of the aggregation index μ against mean overall prey density (AM); this is done in Fig. 142.

On any given day, we calculate a_i for all the sub-areas;

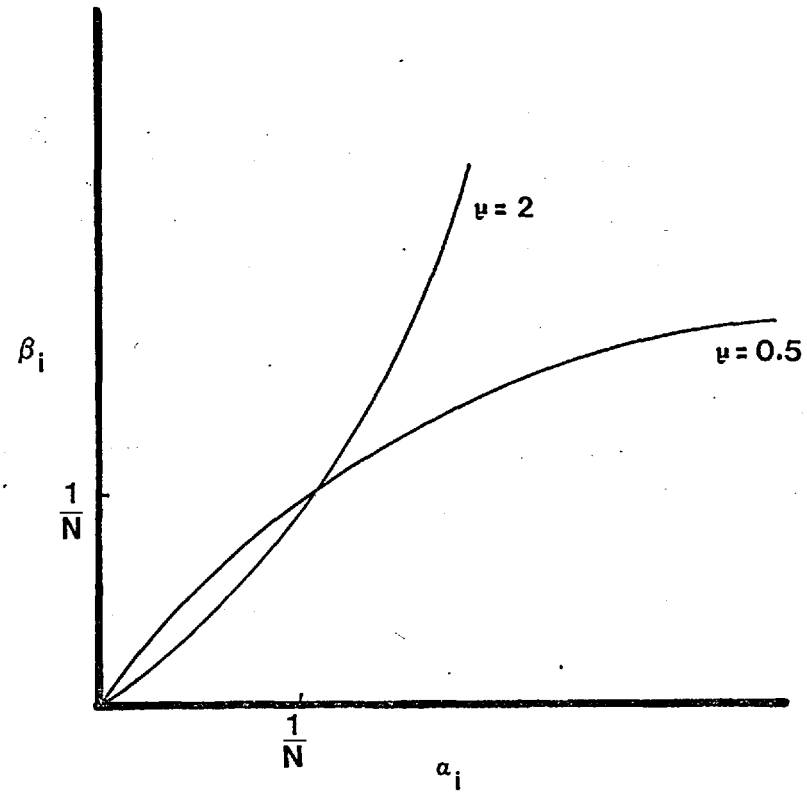


Figure 141. The relationship between the proportion of prey in an area ( ) and the proportion of predators entering the area ( ) when the aggregation coefficient is greater and less than 1. When the proportion of prey is 1/N the proportion of predators will also be 1/N.

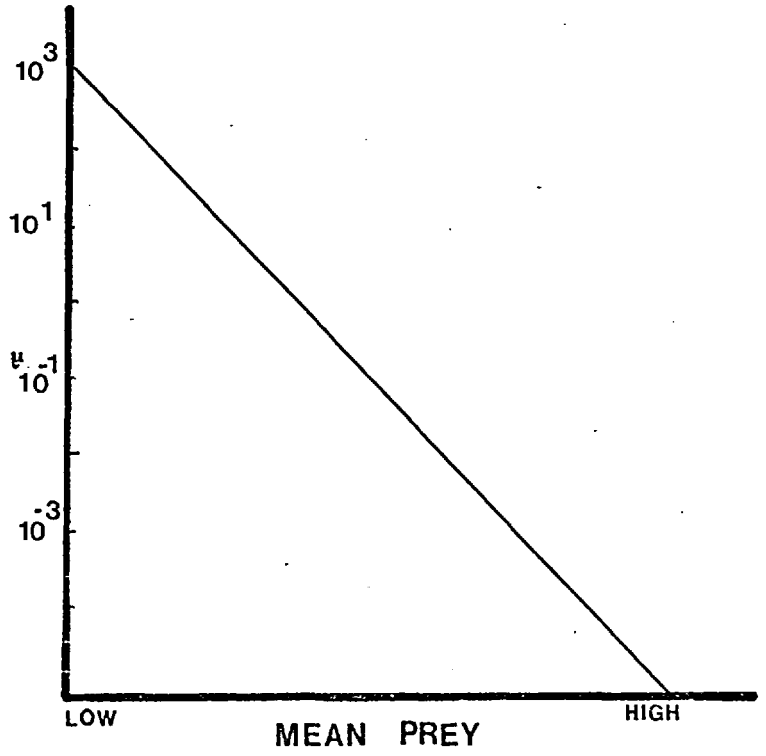


Figure 142. The relationship between predator aggregation and mean prey density employed to explain Hassell's observations (see text). Predators are assumed here to aggregate strongly at low prey densities

$$ALPHA(I) = PREY(I) / TOTPREY \quad \ldots\ldots (50)$$

and compute mean prey density

$$AM = TOTPREY / N \quad \ldots\ldots (51)$$

Then we obtain a value of $\log_{10} \mu$ by interpolation from Fig. 142;

$$ULOG = F(AM) \quad \ldots\ldots (52)$$

and derive μ as

$$U = 10 ** ULOG \quad \text{(10 to the power ULOG)} \quad (53)$$

For all sub-areas we can now compute β_i using Equ. 49,

$$BETA(I) = SIGALPH * ALPHA(I) ** U \quad \ldots\ldots (54)$$

where $$SIGALPH = \frac{1}{\sum ALPHA(I) ** U} \quad \ldots\ldots (55)$$

The adult predators are then distributed over the sub-areas in relation to the value of BETA(I). When predators are scarce, it is unrealistic to distribute the animals themselves over the sub-areas, because there may be more areas than predators. To avoid this error the total egg-laying potential of the adults is calculated (and their total food requirement) and the eggs, rather than the laying adults, are distributed by BETA(I).

Each day a certain number of adults emerge from the pupal stage within each area, and these numbers are recruited to the mobile adult population after a time-lag IDISP (the threshold time in days between emerging from the pupal moult and initiating oviposition). Within the predator sub-models we therefore write the number of emigrating adults as

$$EMIGAD = PREDS(K)$$

where $$K = IAGEAD(I) + IDISP$$

and we sum this number over all sub-areas to obtain the total number of adult recruits;

$$ANEW = \sum_{1}^{N} EMIGAD(I) \qquad \ldots\ldots (56)$$

The IDISPth age class of the adult population is then set to ANEW, and all the other age classes are shifted up one day.

Since the adult recruits have emerged from heterogeneous sub-areas, they will differ in size even though they are the same age-since-becoming-adult. It will be necessary, therefore, to calculate the mean size of the recruits by writing

$$ADSIZE = \frac{\sum PREDS(I) * SIZE(I)}{ANEW} \qquad \ldots\ldots (57)$$

so we can set the IDISPth age class of adults to this size

$$SIZEAD(IDISP) = ADSIZE$$

$$PN(IDISP) = ANEW$$

The total number of eggs produced by the mobile adult population is computed just as before

$$EGGS = \sum_{K=IDISP} PN(K)*SEXR(K)*FERT(K)*FEC(J)*F(STARVE(K)) \qquad \ldots\ldots (58)$$

where J is now given by K - IDISP. The fraction of these eggs laid in the Ith sub-area is BETA(I), and the actual number

$$ELAY(I) = EGGS * BETA(I) \qquad \ldots\ldots (59)$$

so that on the next day, the first age class of eggs in the Ith area is set to ELAY(I)

$$PREDS(1) = ELAY(I)$$

In this way we can simulate the third aspect of the numerical response through dispersal; eggs are laid in relation to mean prey density (as it determines the value of the aggregation index μ) and relative prey density (α_i).

In those cases when we wish to observe the effect of adult feeding as well as oviposition behaviour the model must be a little more complex. In the population growth model we set food requirement as a function of body size, FOOD(K) being the number of calories required for satiation by each individual of age K days. We can do precisely the same for adult feeding except that in this case we are calculating the number of prey required by the adult population from the entire prey population, rather than from one of the sub-populations as was the case before. It is reasonable to assume that adult feeding will bear a similar relation to prey availability as did oviposition behaviour, so having summed the food demand over all adult ages

$$KILL(K) = FOOD(K) \;/\; PREYSZ(K)$$
$$TKILL = \sum KILL(K)$$

we can distribute this potential attack over the sub-areas by writing

$$ATTACK(I) = TKILL * BETA(I) \qquad \ldots\ldots (60)$$

This potential attack is then used in the individual models to calculate functional responses and exploitation effects (Section f), and we arrive eventually at an actual number killed from which it is possible to calculate the degree of starvation experienced by those adult predators which chose to feed in each sub-area. Since we have assumed that the adults distribute themselves over prey areas in the

same way at all ages, we can only arrive at a mean figure for the starvation of the adults from a particular sub-area. At the end of the day, the mean starvation of the whole adult population can be calculated by weighting the actual starvation experienced by each animal from a sub-area by the number of animals which fed in the sub-area. If STARVE(I) is the actual starvation for an adult in the Ith area, to which BETA(I) of the predators moved, the mean adult starvation will be

$$\text{STVMEN} = \text{STARVE(I)} * \text{BETA(I)} \quad \ldots\ldots (61)$$

and this value can be used in calculating their fecundity and survival on the next day.

In short, the two models are run together so that the population growth section operates for each sub-area separately and deals with the animals only up to adult age, IDISP. From the population models a number of adults emerge which then form a single population unit capable of ranging over all the sub-areas. A prey population is simulated within each sub-area; this is decreased by the feeding of those larvae confined to its sub-area, and by the feeding of those adults which spend the day in the sub-area. The sub-areas can be made heterogeneous in three ways. First, the initial distribution of prey can be non-regular so that some areas contain more aphids than others; second, the rate of aphid increase (PREP) can be set to different levels for each sub-area, so that we can assess the effects of patchy environmental quality for the prey species; third, we can start the simulation with non-regular distributions of predator eggs, and examine the ability of the model to simulate the recovery of prey equitability between the areas.

Running The Model

A) The Population Model

1 Functional Responses

The functional responses to prey density, relative prey density, and predator density are shown in Fig. 138, and they reflect the fraction of the dietary requirement which can be satisfied under a given set of conditions. We can now run the model using different shapes and combinations of the three responses to investigate how they affect the number of prey attacked. At first we shall consider their operation in the absence of any numerical responses, so in the early runs predator density is not affected by the number of aphids.

a) Response to Prey Density

In all these tests we shall make the response rather extreme so that its effects on the output (number of prey, and number of prey killed) are accentuated. For this run we set the other two functional responses to unity (so they have no effect), and set PK = F(PREY) as in Fig. 138. Now, running the model for 50 days we can plot the number of prey and the number of prey consumed. A refuge of 5000 aphids has been assumed throughout, and since no aphids are eaten below densities of 6000 in excess of the refuge (Fig. 138), we should expect the system to come to equilibrium at about 11000 prey individuals, with a daily consumption equal to the reproduction from a stock of this size (i.e. (PREP - 1) * 11000). The output is plotted in Fig. 143.

The curve of prey consumed shows a characteristic lag behind the curve of prey numbers (about three days) because the response to prey density is not completely immediate. There

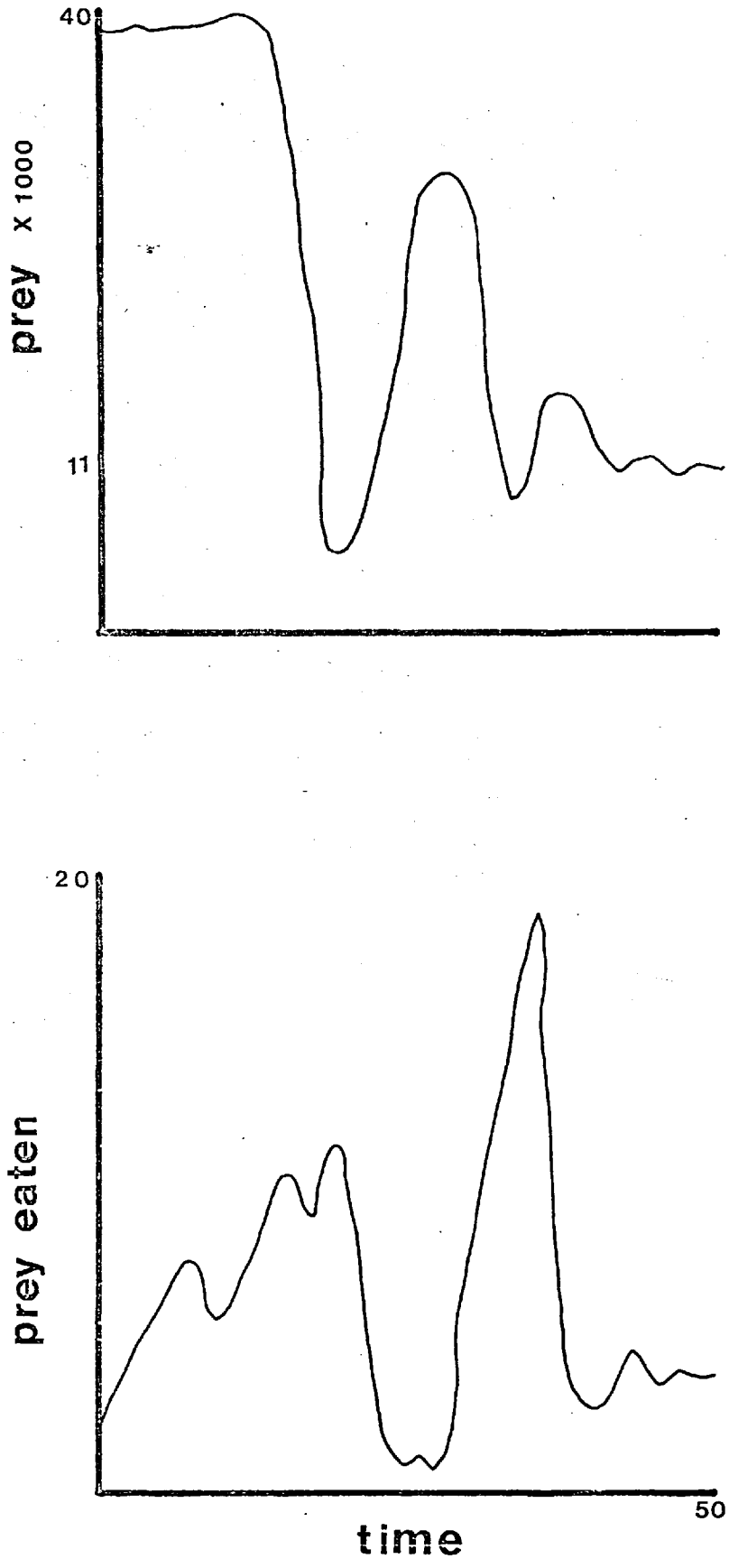


Figure 143. The patterns of aphid abundance and aphid consumption observed when the functional response to prey density is included in the model

are a number of reasons for this; first, the age structure of the predator population changes with time and so, even though the predators continue to rise in numbers, the rate at which feeding increases is variable. Second, the shape of the functional response curve has not been drawn so as to compensate perfectly for changes in prey availability; this would not be possible in any case, so long as the age structure of the predator population varies.

The prey population is stabilized, however, and at the level we predicted (about 11,000 individuals). The functional response to prey density is seen to be highly stabilizing, but this effect is dependent upon the curve (Fig. 138) bisecting the PREY axis with PREY greater than 0. If feeding is not curtailed before prey density reaches zero then the interaction will be unstable, and the prey population will become extinct. We must also remember that no numerical responses are considered at the moment, and it would take a very starvation-tolerant predator species to follow the feeding curve shown in Fig. 143 without fluctuating in abundance.

b) Response to Relative Prey Density

In building the model we made the assumption that below a certain threshold in the number of prey per predator, intraspecific food competition would begin to limit the rate of food intake. In Fig. 138 we graphed the fraction of the diet realized against the ratio of prey to feeding predators so that above 100 prey per predator feeding is unaffected, but that with only 20 prey per predator all the animal's time is spent in chasing other predators, and no aphids at all are caught. Again, an extreme example has been chosen to highlight

the effects of the response; we would not expect a real predator species to completely curtail feeding because of intraspecific competition.

As before, the model is run for 50 days and the number of prey, and the number of prey consumed per day are plotted as output (fig. 144). The first and most obvious difference with this functional response is that the peak prey density reached is five times higher than that reached with the prey density response (200 as compared to 40 thousand). This is because absolute prey density does not affect the number of aphids killed; in fact, the number of prey killed falls for the first 15 days, while prey numbers increase continually over this period. The response depends upon the rate at which the predator population increases relative to the aphids, and, without any numerical responses in the model, this rate will vary, and the response will be poorly coupled to prey density.

The second point to emerge is that this response does not stabilize prey density to the same extent as did the first response. If the time axes of the graphs were extended further large peaks in prey numbers would be observed. This behaviour is brought about by the fact that with less than 20 prey per predator no feeding can occur, and so the predator population has to wait until prey numbers have built up to a sufficient level so that the ratio of prey per predator again exceeds 20. Since, however, the predator population is increasing continuously (there being no numerical response) this time period in which no feeding occurs can be quite prolonged (indeed, if the predator population increased rapidly enough there would never be any more feeding, as the ratio of prey per predator would decrease continuously). The key point in considering this functional response is the level to which

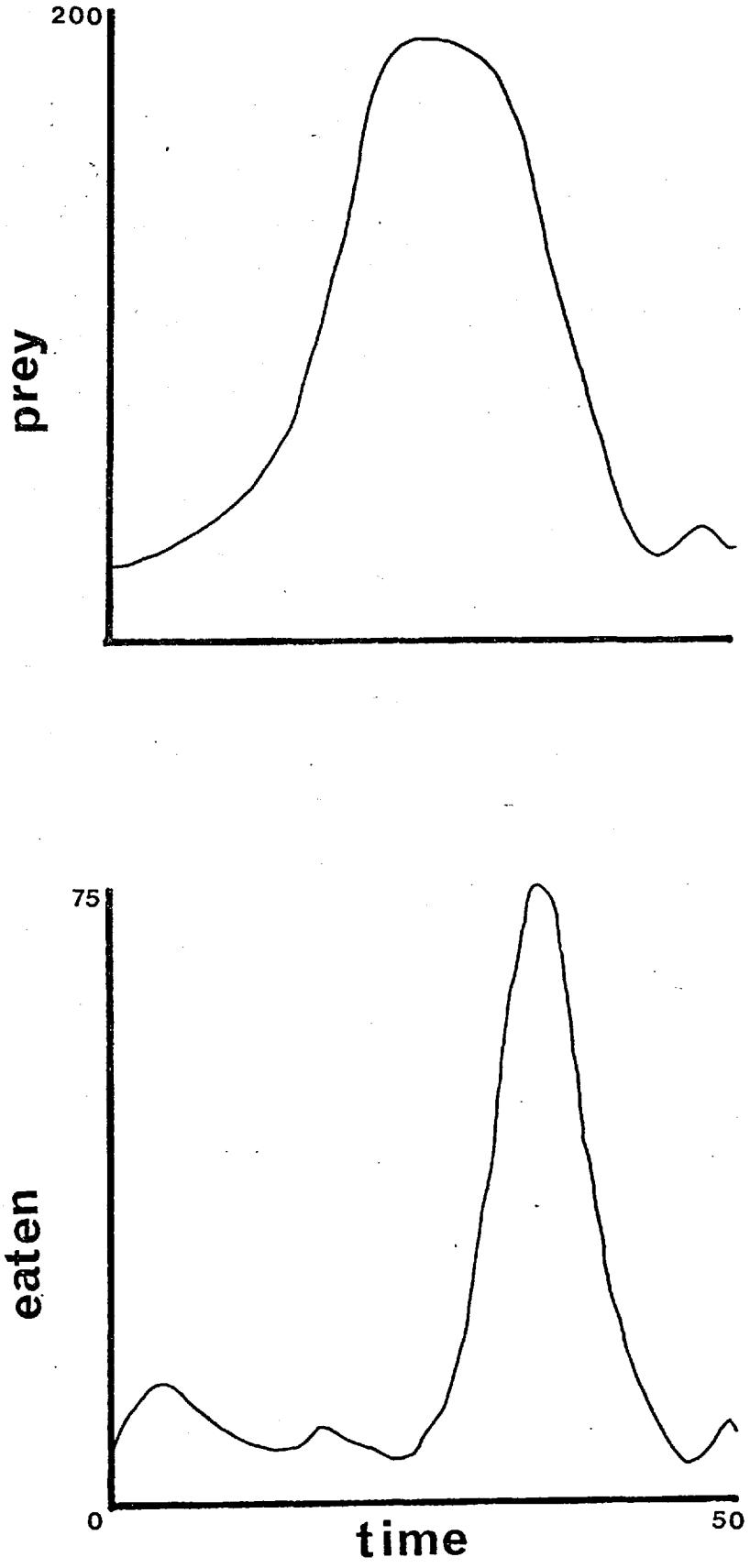


Figure 144. The patterns of aphid abundance and aphid consumption observed when the functional response to relative prey density is included in the model

it could actually reduce food intake at extreme relative prey shortages.

c) Response to Predator Density

The final functional response concerns intraspecific interference (Hassell, 1971), during which feeding time is wasted in avoiding (or following) intraspecific contacts. To assess the effects of this response on the behaviour of the model populations we can plot relative food intake against the number of predators of a given age, PREDS(I) as in Fig. 138. At most times in the predator population cycle the number in any age class will be proportional to the total number in the population, and the specific assumption that predators only interfere with their contemporaries is not limiting to the generality of the model.

As we might expect, this response is highly destabilizing when there are no numerical responses associated with the starvation brought about by mutual interference. As predator density increases, the number of prey killed per predator falls off until no prey are consumed. But the density of predators continues to incease, and so the prey multiply unimpeded (Figs. 145, 146).

When these strong functional responses are run in combination we observe the following. The response to predator numbers through interference is of over-riding importance in combination with any other, because predator numbers increase indefinitely without numerical responses. The combination between the prey density and relative prey density responses is more interesting. For the early part of the interaction the relative prey density response is over-riding, but after the population has been reduced to low levels the

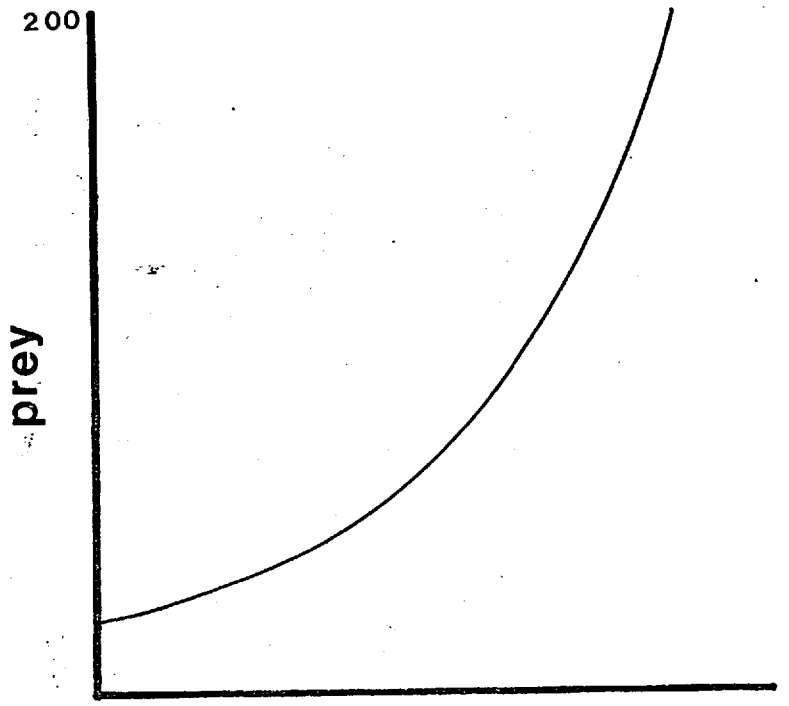


Figure 145. The pattern of aphid abundance when the functional response to predator density is included in the model

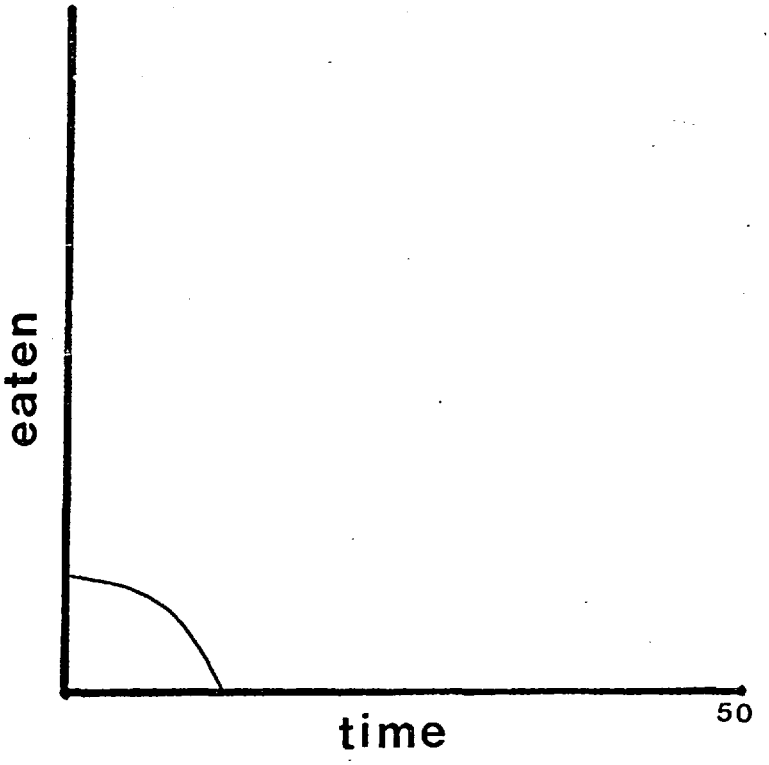


Figure 146. The pattern of aphid consumption observed when the functional response to predator density is included

absolute prey density response takes over. The relative prey density response stops the absolute response from acting to its full stabilizing potential, and prey numbers rise to much higher levels in consequence (like Fig. 144). In order to observe these effects more realistically, we must move on to consider the numerical responses of the predator population to starvation.

2 Numerical Responses

The model assumes that the degree of starvation experienced by an individual predator affects its survival rate, its reproductive rate (if it is an adult), and its emigration rate. I shall consider the first two effects here, and deal with dispersal when running the adult dispersal model (below). Since the numerical responses all operate as functions of starvation, and there can be no starvation without functional responses, it is not possible to observe the effects of the numerical responses in isolation.

a) Effects on Survival Rate

If we make the probability that an individual will survive from one day to the next dependent upon the amount of food the animal took in over the previous day (or some more extended period) we can employ a graph of survival under starvation like Fig. 131. Because adults feed first in the intraspecific competition for aphids (see above), they will starve relatively less than the juveniles; similarly, because there tend to be more juveniles than adults at most stages in population development, the reduced survival of young predators will amount to a greater net reduction in predator numbers than would the application of the same survival rate to the older instars.

When both the functional responses to prey and to relative prey densities contribute to the starvation mortality we obtain output as in Fig. 147. The integral of the aphid abundance curve is much lower than was the case when these functional responses were run without their effect on survival (see Fig. 144), but there are still more prey than occurred with the density-dependent functional response alone (Fig. 143). Clearly the numerical response in survival rate acts to reduce aphid numbers, but it also tends to reduce the period of prey oscillation. In Fig. 144 there is only one prey cycle per 50 days, while in Fig. 147 there are two. It is also apparent from the latter figure that the variations in aphid numbers are being damped; they are increased in frequency and decreased in apmlitude compared to the curves with no numerical response. The curve of prey consumed in Fig. 147 resembles that in Fig. 143 (the prey density response) far more than it resembles Fig. 144 (the relative prey density response).

b) Effects on Birth Rate

We have assumed that the number of eggs produced by an adult predator in a day depends upon the number of aphids she has eaten. It is therefore possible to plot relative egg-laying rate as a function of STARVE(I), and to obtain particular values by interpolation from Fig. 132. The actual number of eggs laid (Equ. 34) is then reduced by this fraction.

Because the response has been made very strong, and we have allowed that no eggs at all are laid by unfed females, we would expect prey numbers to be rather well regulated, and this is what we observe (Fig. 148). After about 30 days numbers remain very stable about their basal level (determined by the size of the refuge, and the cut-off point of the prey-

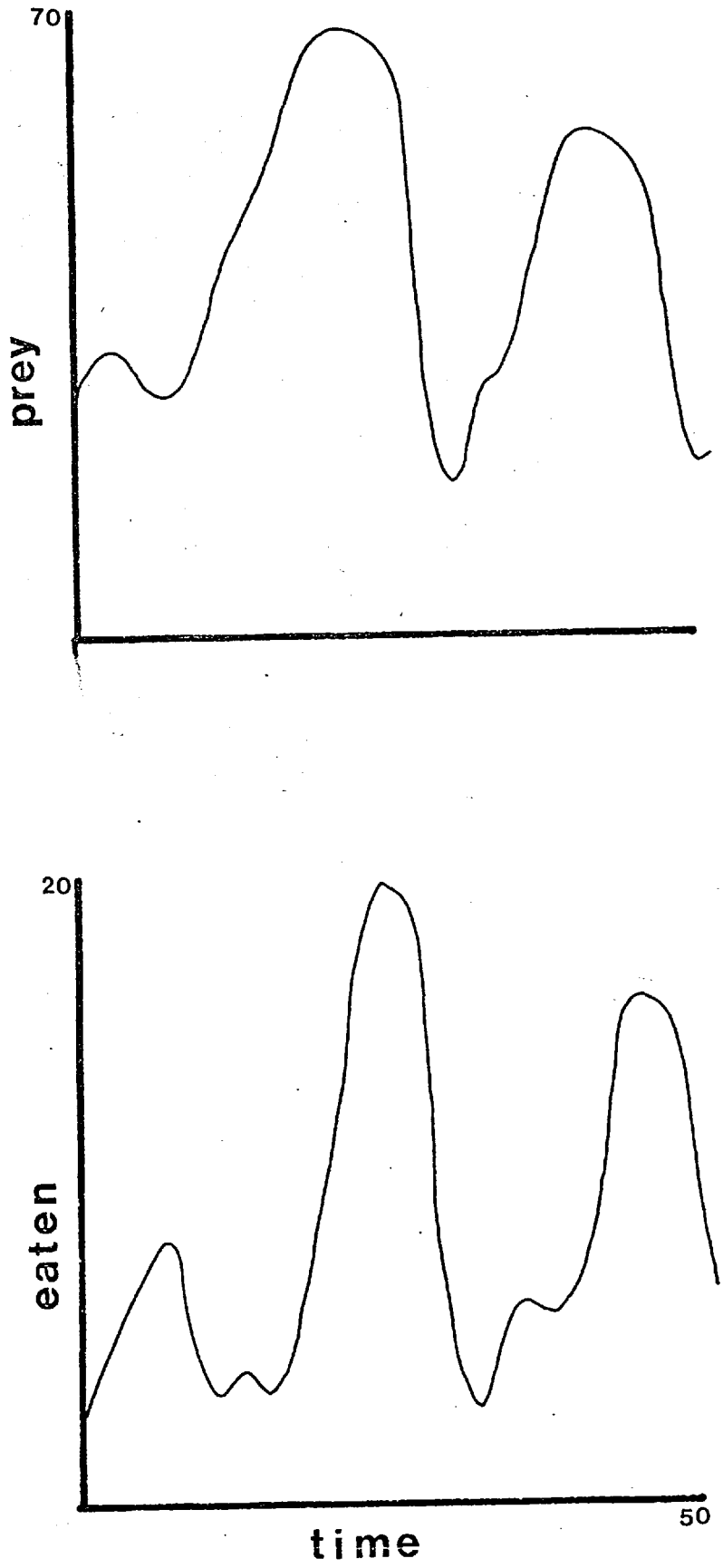


Figure 147. The number of aphids and the number of aphids eaten plotted against time since predator immigration. The numerical response in survival rate alone is included

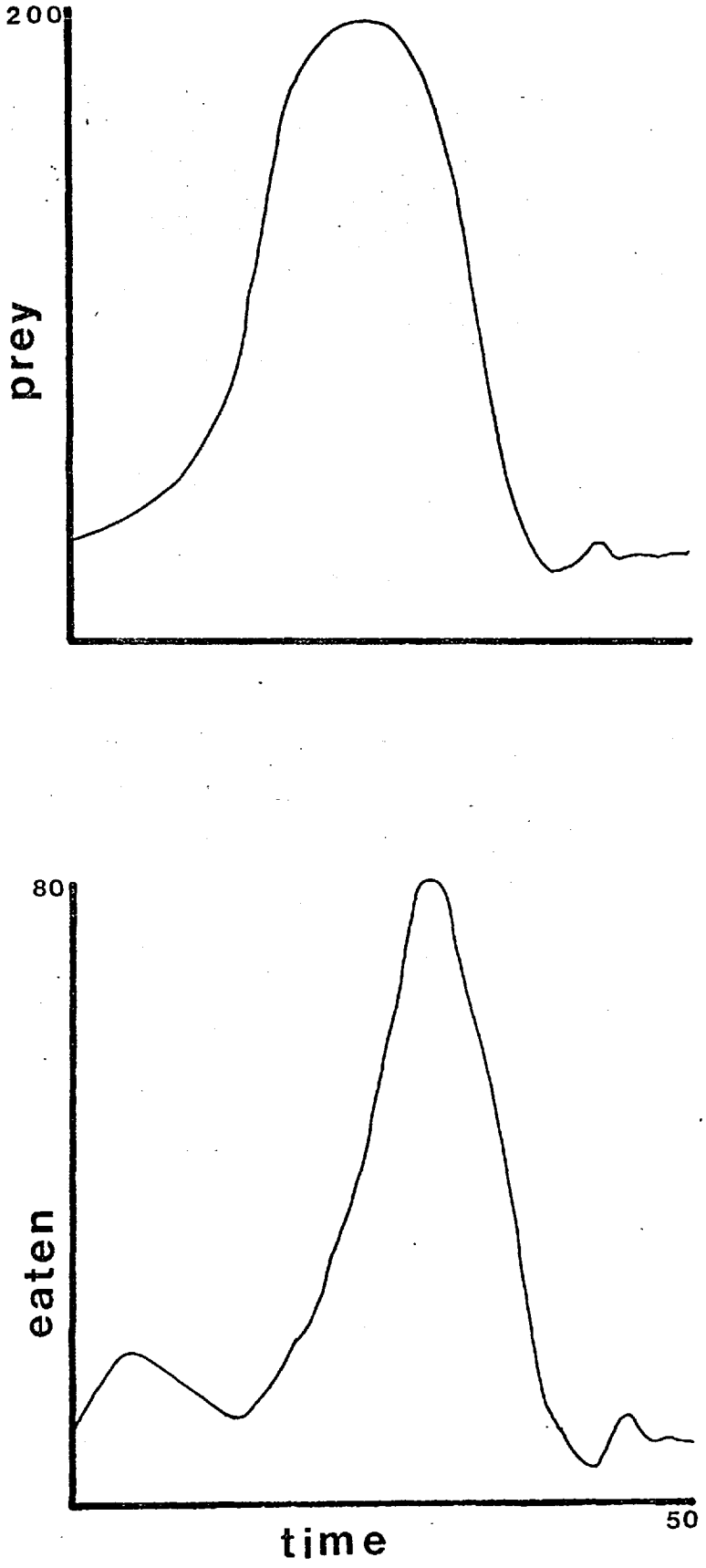


Figure 148. The number of aphids and the number of aphids eaten plotted against time since predator immigration. The numerical response in birth rate alone is included

response curve), and no more peaks in numbers occur. The shape of the feeding curve in Fig. 148 resembles the shape of that observed when the relative prey density response acted alone (Fig. 144), and so we might suggest that under these conditions the relative response was more important than the response to absolute prey density. Numerical response in birth rate would therefore appear to be a potent agency in both reducing prey numbers and in maintaining them at a constant low level. We can not compare the outcome directly with the survival response, because the two effects act with different intensities; it would be rather unrealistic to assume that all the predators in a particular age class died after missing only one day's feeding. The interesting question to emerge is whether the response to relative prey density is most important when births are limited, or when a complete (i.e. 100%) numerical response is allowed ? To test this we can run the model with both birth and survival rates dependent on feeding success.

c) Effects on Both Survival and Reproduction

Figure 149 plots the pattern of prey numbers and the pattern of prey consumption when both survival and birth rate are made dependent upon starvation. It is quite clear that the two curves follow almost exactly the trends shown in Fig. 147, and that, therefore, the responses in survival rate tend to be of over-riding influence. The peak numbers reached with both responses are only fractionally higher than with the survival response alone (67,112 and 66,726). In retrospect, the reasons for this are clear; first, increases in mortality act immediately and so the responses in feeding rate follow more closely on changes in food availability, and second,

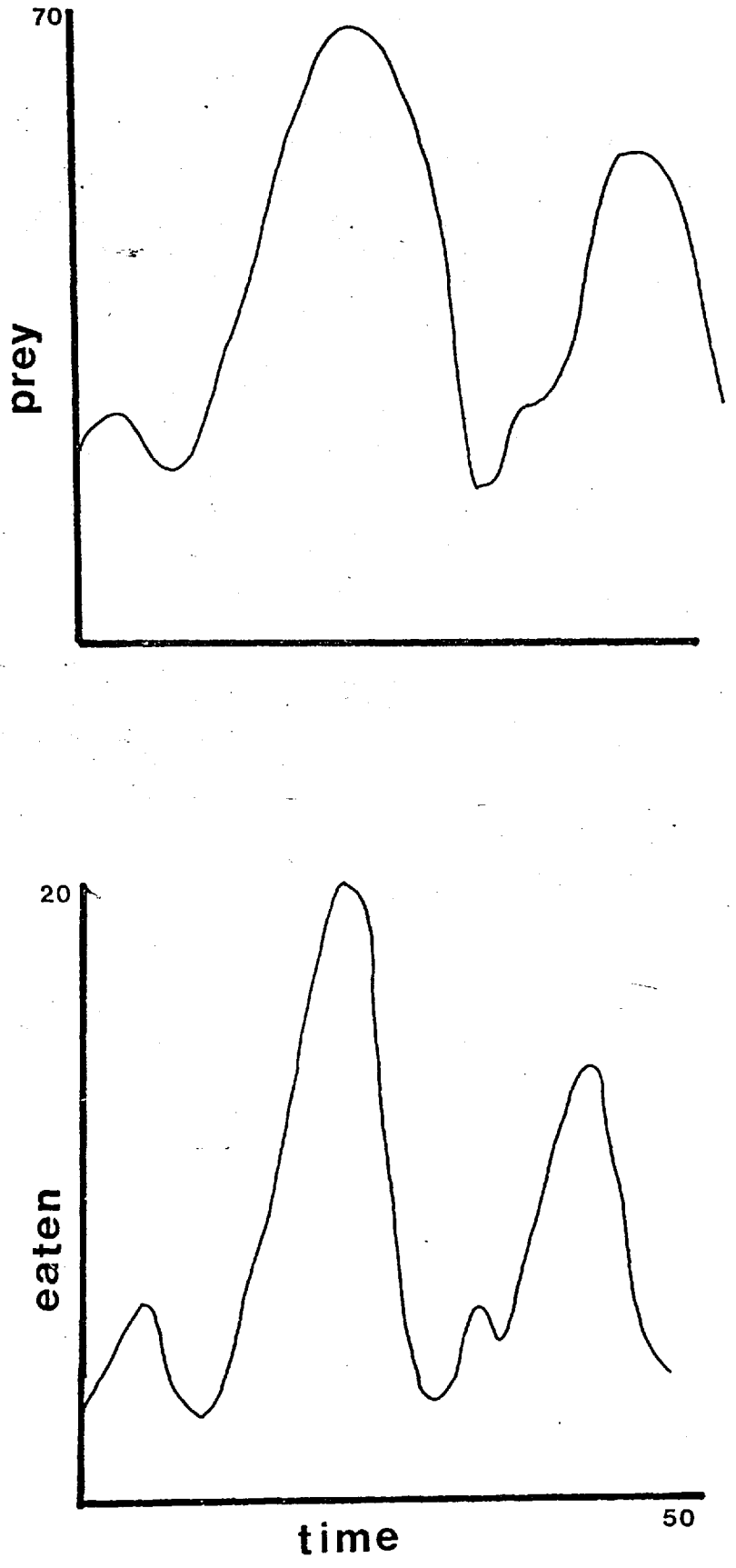


Figure 149. The number of aphids and the number of aphids eaten plotted against time since predator immigration. Numerical responses in both survival and reproductive rates are included

all animals can die of starvation (except eggs and pupae, of course), while only adults can reproduce. This means that the birth response acts on a smaller fraction of the feeding population than the survival response and is consequently less effective at stabilizing numbers of prey.

As with the survival response alone, the prey population is reduced by a series of asymmetrical damped oscillations, and predator numbers follow a similar pattern but with a slight time-lag.

3 Duration of Non-feeding Periods

A predator species which spends the greater part of its life cycle as an egg or a pupa will tend to be less effective in pest control than a predator with very brief non-feeding stages. There are two reasons for this; first, the shorter the dormant periods, the greater the proportion of the predator population which is actively feeding, and second, with brief dormant phases development to adulthood can be more rapid, which allows for quicker build-up in predator numbers. To observe the effects of these two processes I have made three runs using different durations of the pupal stage, running the simulation for 50 days at 20°C so that the pupal stages lasted 3, 7, and 14 days respectively. The pattern of prey abundance with these three treatments is plotted in Fig. 150.

For the first 20 days the functional and numerical responses were of over-riding importance as the two populations built up towards a more stable age distribution. Once the initial population adjustment had occurred it became clear that the stability of aphid numbers was very largely dependent upon the duration of the predator's pupal stage. As the duration decreased, so the frequency of prey oscillations, and

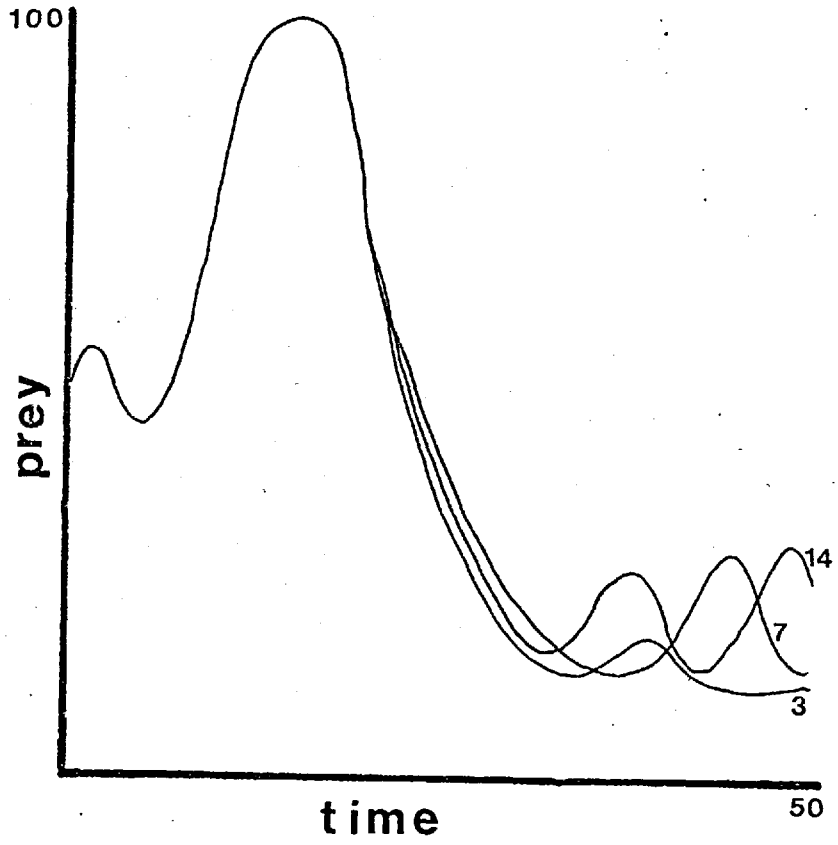


Figure 150. The pattern of aphid abundance when the duration of the pupal stage was set to 3, 7, or 14 days. Stability appears to increase as the duration of the stage decreases.

the amplitude of the fluctuations decreased in turn. The same effects would be observed if the egg duration were reduced but the magnitude would be diminished somewhat by the fact that a lower proportion of eggs survive to become reproductive.

4 Age-specific Fecundity

The model runs on the assumption that the number of eggs laid by a female predator on a given day is a function of her age since becoming adult. There are numerous patterns of egg-laying which we can investigate, but, since the runs performed to date have shown the importance of a relatively stable age distribution (with no periods when no prey are consumed), I shall limit consideration to those patterns of oviposition which occur over an extended period. It is quite clear that if every female lays all her eggs on one day, and these eggs develop in synchrony, the age-structure of the population will be completely discrete, with only one instar present at any given time; when the population pupates all pest control activity will cease and the aphid can multiply unimpeded.

In Fig. 151 are shown three patterns of age-specific fecundity; constant, decreasing, and increasing with age. They all have the same integral of 300 eggs per female over her reproductive life-span. In the model, however, the curve which allows increasing fecundity with age will tend to give a lower total number of eggs per generation, because each female has a lower probability of reaching the age of maximum fecundity. The pattern of prey abundance resulting from the inclusion of each of these three fecundity vectors in turn is shown in Fig. 152.

The pattern of age-specific fecundity did affect the peak number of aphids in the initial phase of increase, with the

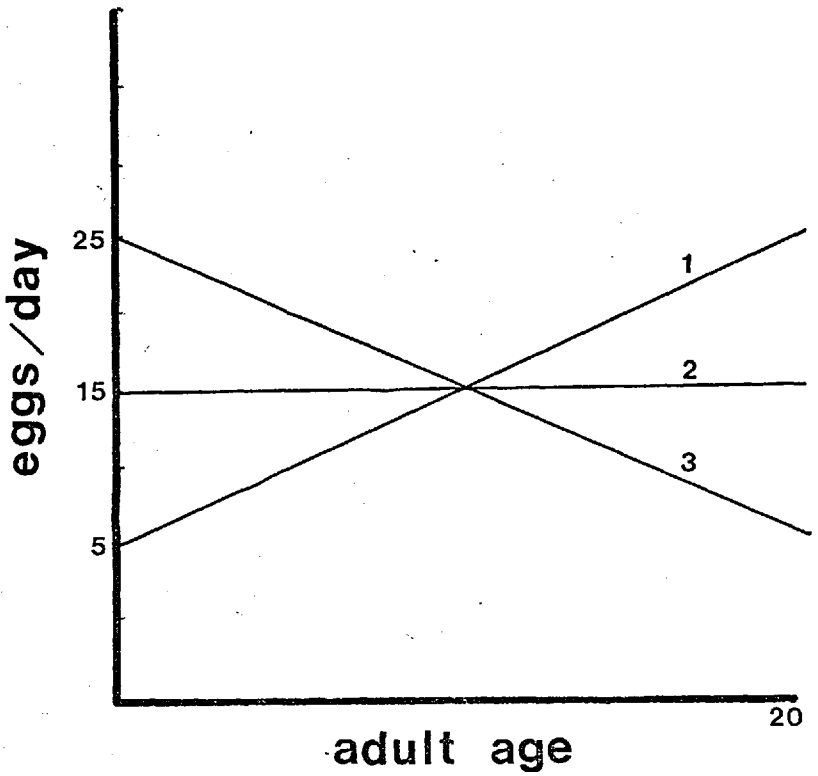


Figure 151. Three patterns of age-specific fecundity tested in the model. All have the same integral of 300 eggs/life

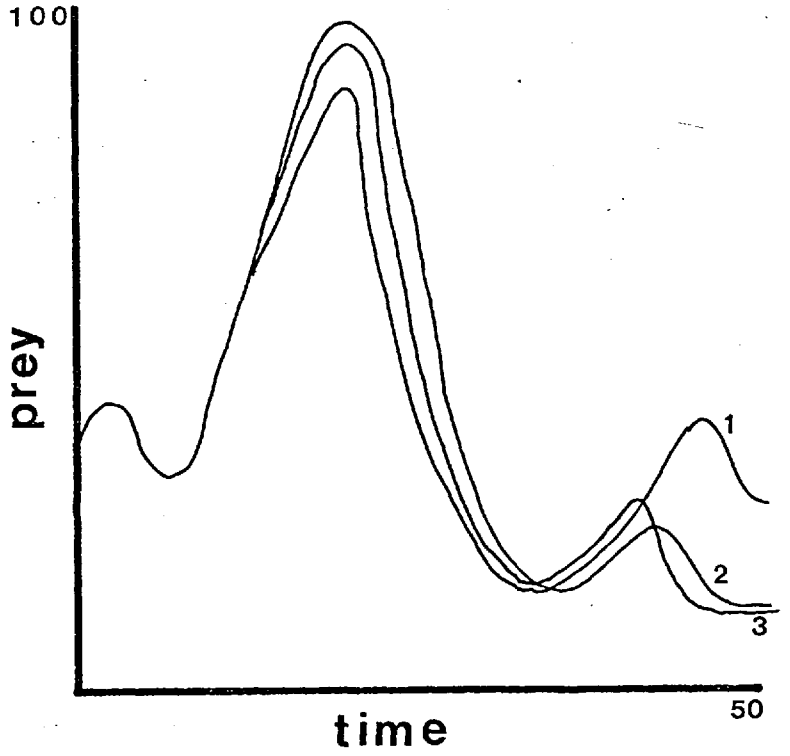


Figure 152. The pattern of aphid abundance resulting from the inclusion of each of the three fecundity vectors

increasing vector bringing about the greatest population reduction. This is due to a reduction in predator numbers in the early stages, and hence to a lessening of intraspecific competition and a consequent rise in the number of prey consumed. After the prey had been reduced to a low level, however, the situation was changed, and the more young produced by younger predators, the lower the fluctuations in aphid abundance.

From the last two sections it has become plain that the degree of control exerted by a predator species can be affected by almost every aspect of the animal's reproductive and developmental biology, as well as by the more frequently discussed attributes of its functional and numerical responses; its 'density-dependent properties'. Even with the very strong responses incorporated in these runs of the model, there is sufficient variation in prey abundance brought about by the duration of the non-feeding stages, and by the shape of the fecundity curve, to mean the difference between successful control and pest outbreak (Figs. 150 and 152).

B) The Adult Dispersal Model

If we now take the population model and apply it to five areas, each with its own independent prey population, we can observe the effects of adult dispersive behaviour on the pattern of pest abundance in the different areas. We can investigate how heterogeneity in initial prey densities affects the pattern of predator attack, and how non-regular predator distributions affect the outcome. It will also be possible to test the stabilizing effects of predator aggregation reported by Hassell and May (1973) when the aggregation index is a variable determined by mean prey density (see above). The functional responses are included as before, but the inter-

ference response is set a very low level because of its strong destabilizing effects (Section A, 1c). Adults are assumed to leave the sub-areas in which they developed after four days of adult life (IDISP = 4), and to spend the remainder of their days in the mobile adult population which is confined to the total area, A. Only adult emigration from the system is permitted, this occurring when prey density becomes uniformly low.

In order to check that the model has not been inadvertently altered by the inclusion of adult dispersal a control run was made using the same initial aphid density as had been employed previously (35,000). All five prey sub-areas were similar, therefore, in all respects, and so there should be no predator aggregation (since there are no grounds for discrimination). In Fig. 153 the pattern of aphid abundance is plotted on a $\log_{10}$ scale against time since predator immigration; the output matches that shown in Fig. 152, and the prey populations in each sub-area are equally abundant. We can therefore conclude that the model behaves as expected.

The initial intention of this part of the model was to investigate the relationship between the degree of predator aggregation and the level and constancy in prey numbers resulting. It was also hoped that the effect reported by Hassell (1968), in which aggregation decreased as mean prey density increased, could be mimicked. Since the value of μ is an indication of the extent to which adult predators prefer to feed and oviposit in areas of high relative prey availability, Hassell's observations were equivalent to the assumption that μ decreased as mean prey density increased. As I had no idea which values of μ should match with a particular level of aphid abundance an arbitrary relationship was drawn as a

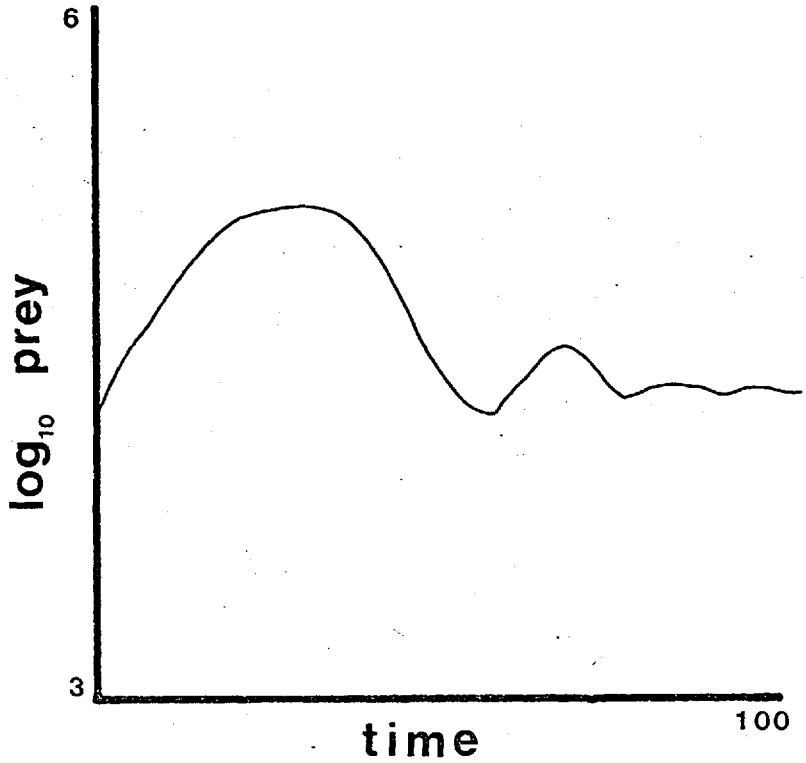


Figure 153. Aphid abundance in five areas against time since predator immigration. All areas have the same number of aphids in this control run.

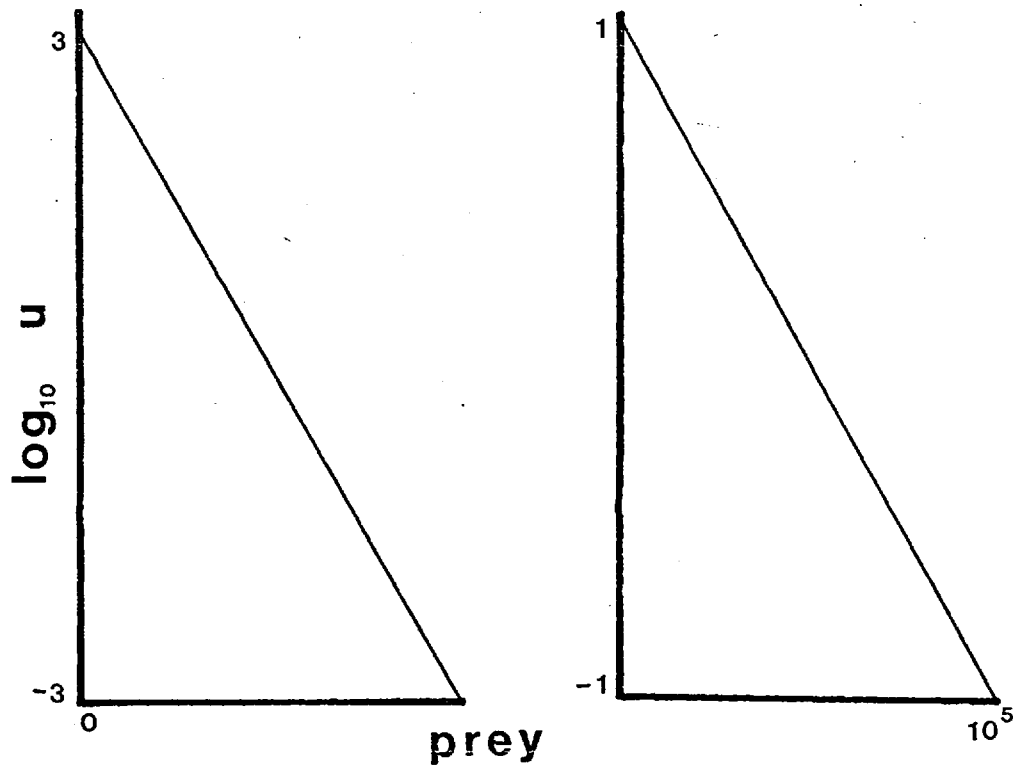


Figure 154. Two test patterns of the relationship between predator aggregation (u) and mean prey density. Both assume that predators are more aggregated at low aphid abundances.

starting point (Fig. 154 A).

The first tests which can be made assess the effects of initial prey densities; three sets of conditions were tested with (35, 25, 15, 5, 0.5), (20, 5, 1, 0.5, 0.1) and (10, 9, 8, 7, 6) thousand aphids in sub-areas one to five respectively. The pattern of prey numbers with these initial conditions is shown in Fig. 155. They are, without exception, both more unstable and show higher levels of pest abundance than the control in which aggregation is not operating; see Fig. 153. There is rather little to choose between the three runs in deciding on the relationship between stability and initial prey distribution.

The aggregation response employed is clearly destabilizing; we must determine now whether it was too intense (i.e. gave too extreme a range of values for μ) or too weak. Since the initial range was from 1000 to 0.001 I assumed that the response was too intense, and replaced the upper and lower limits on μ by 10 and 0.1 respectively (Fig. 154 B). With the same three initial prey distributions as before, we now obtain the output graphed in Fig. 156 . Stability is greater than before, and can be seen to decrease as the difference between the initial prey densities increases.

The general outcome therefore seems to be that the aggregation response is destabilizing when made a decreasing function of mean prey density (Fig 154); additionally, stability of aphid numbers increases with the homogeneity of initial aphid distribution. Perhaps predator aggregation would be more effective in terms of pest control if it were a constant ? Setting μ equal to 6 and running the three initial prey conditions gives us Fig. 157, in which stability has again improved, and the most unstable case still occurs with the

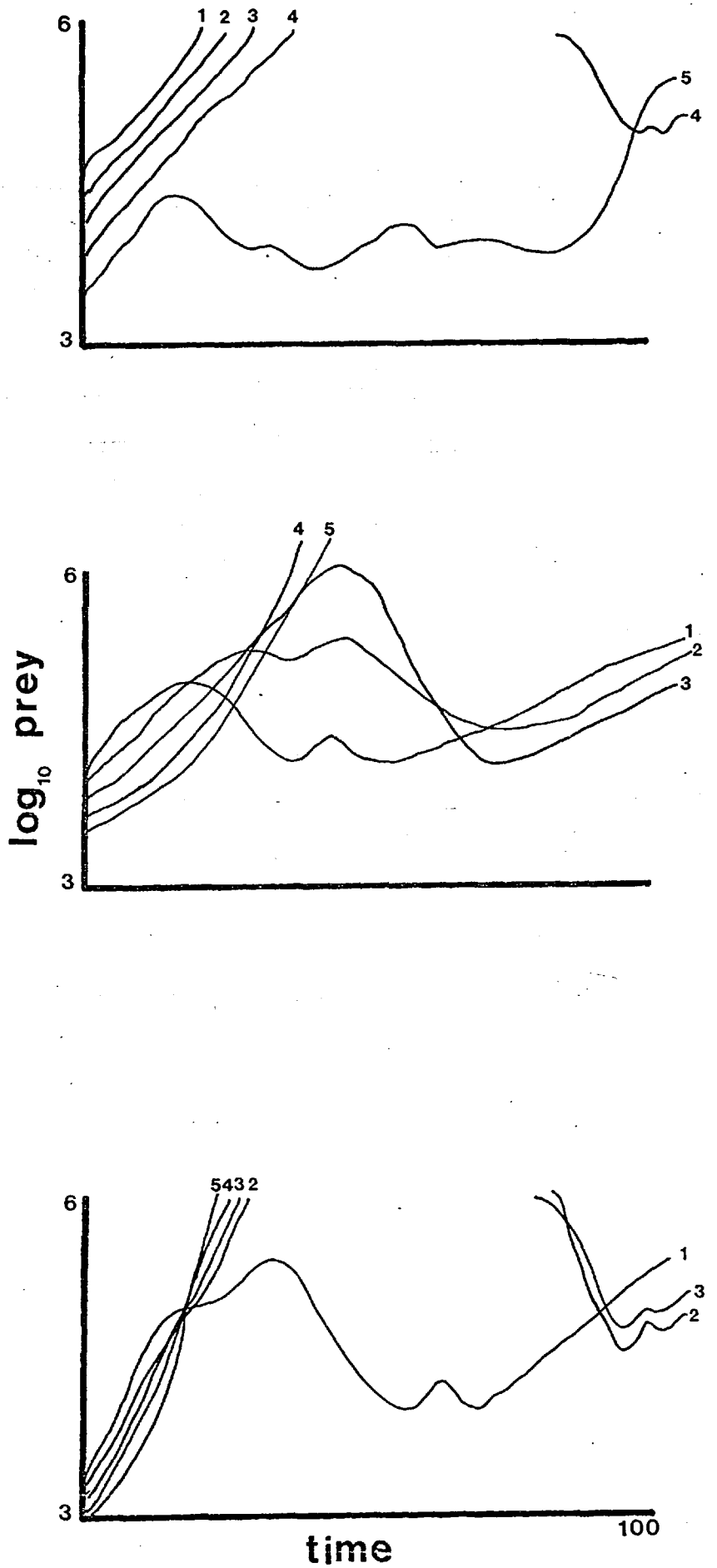


Figure 155. The pattern of aphid abundance in five areas with three initial prey distributions (see text). Aggregation a strongly decreasing function of prey density

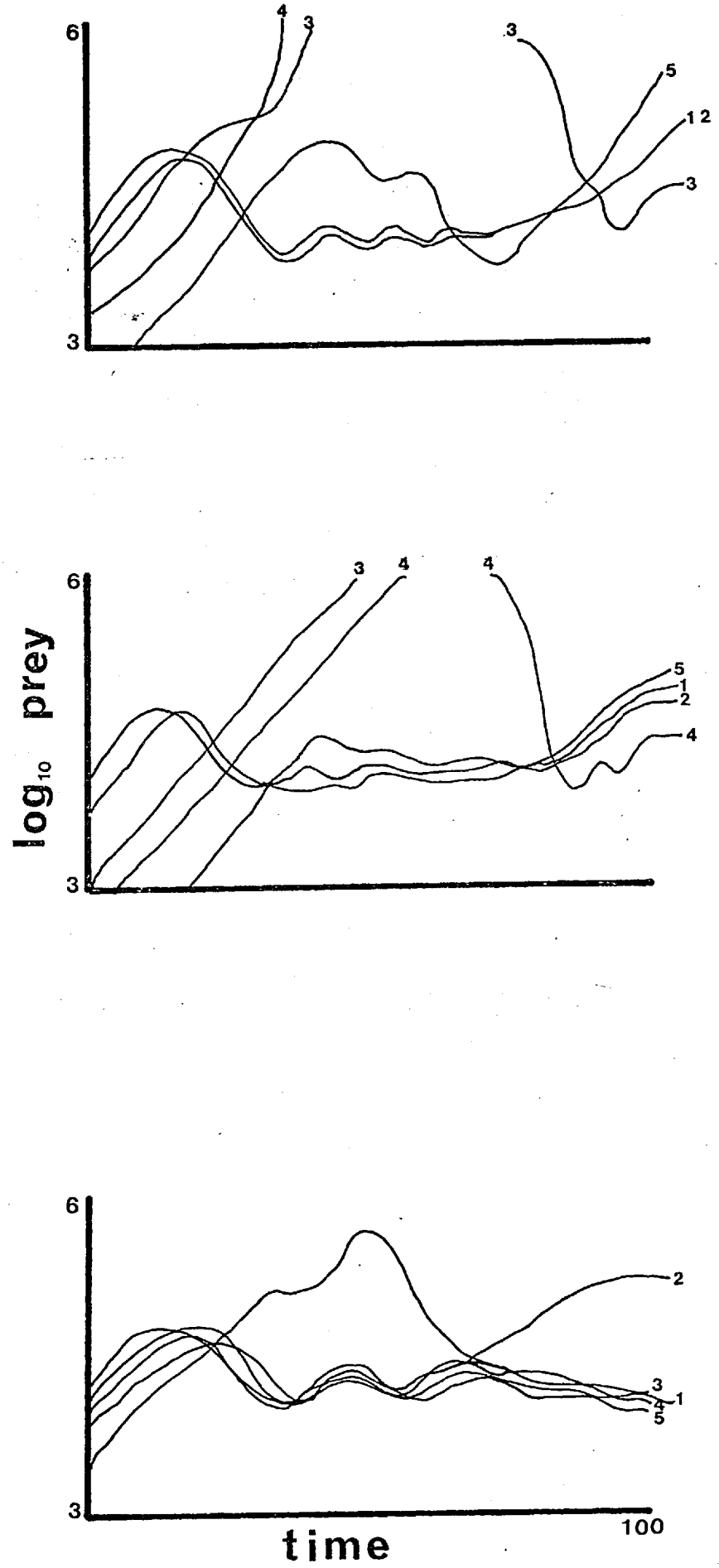


Figure 156. The pattern of aphid abundance in five areas with three initial prey distributions. Aggregation a weakly decreasing function of mean prey density

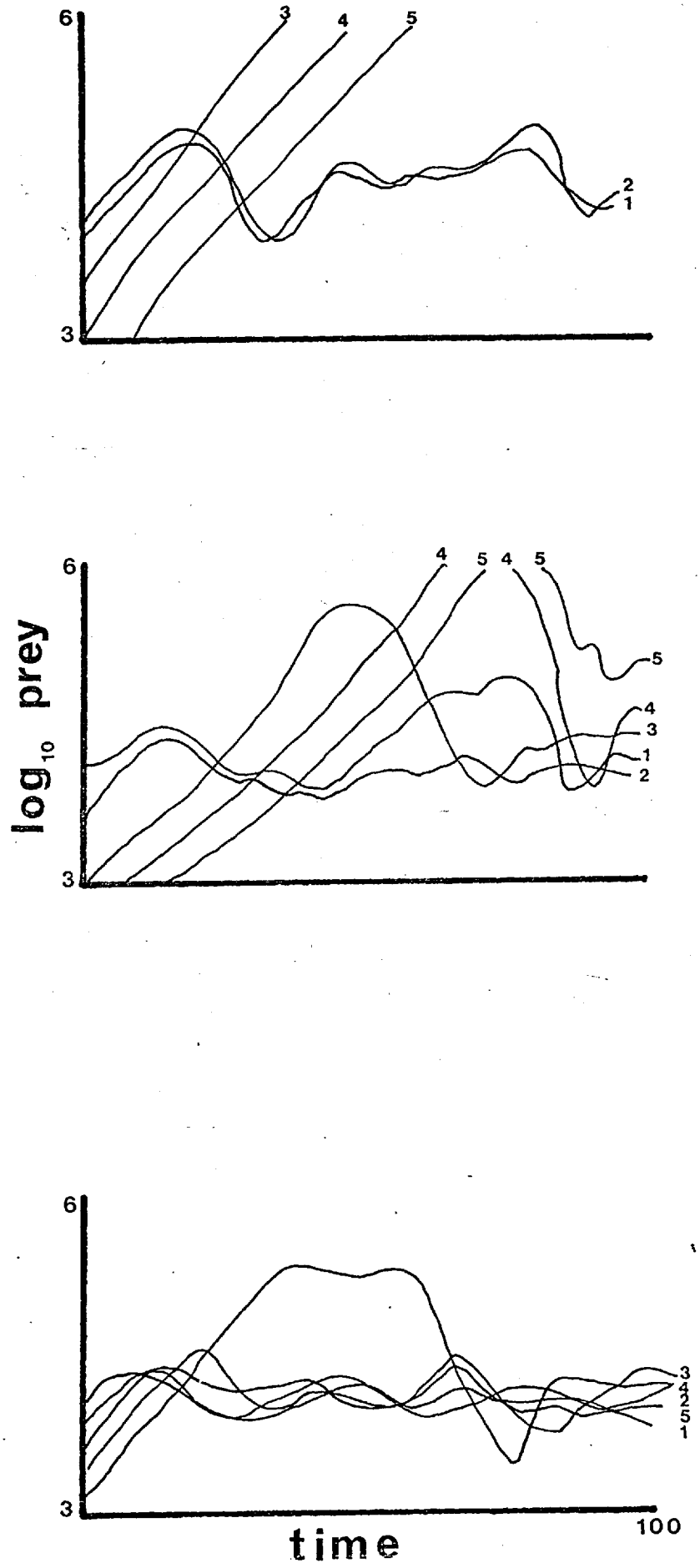


Figure 157. The pattern of aphid abundance in five areas with three initial prey distributions. Aggregation constant and high at 6.0

widest difference in initial prey density.

Assuming still that μ is time invariant, we can set it to a lower value and re-run the model (μ = 0.1); the results are shown in Fig. 158. In this case all the runs are strongly stabilized, and after 100 days there is little difference in prey abundance between sub-areas. The final abundance is related to the initial prey distribution, however.

Apparently stability can be increased by removing the prey-density effects on μ, by lowering the mean level of μ (from 6 to 0.1), and by starting with an homogeneous prey distribution. These conclusions differ from those of Hassell and May (1973) in many respects (see Discussion). Finally, we can test the response of the model to the inverse of our original assumption; namely that predator aggregation μ increases as mean prey density increases. We can use a reversed image of Fig. 154 B to calculate μ at each prey density. From Fig. 159 it appears that the assumption of increasing predator aggregation with increasing mean prey density is highly stabilizing. The mean values of μ during these runs were about 0.3, 0.25 and 0.2 respectively; this is what we might expect, since the more heterogeneous case again gave rise to the highest mean prey abundances.

Discussion

My purpose in this Chapter has been to present a model of predation which considers the functional and numerical responses of a predator population with overlapping generations, and with variable synchrony to its prey. This contrasts with the existing models, which tend to treat parasitism rather than predation, and to deal with discrete generations of host and parasite. The model is designed to investigate how each of

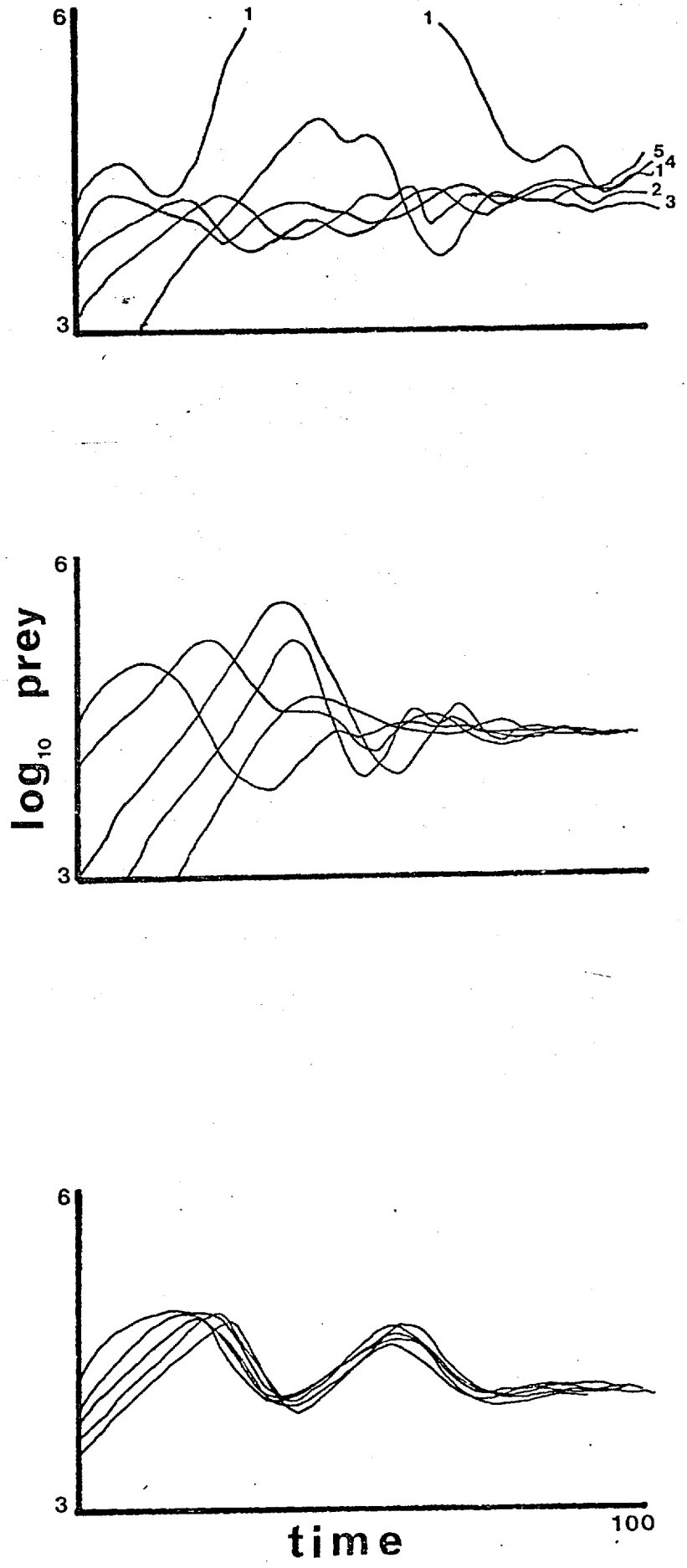


Figure 158. The pattern of aphid abundance in five areas with three initial prey distributions. Aggregation constant and low at 0.1

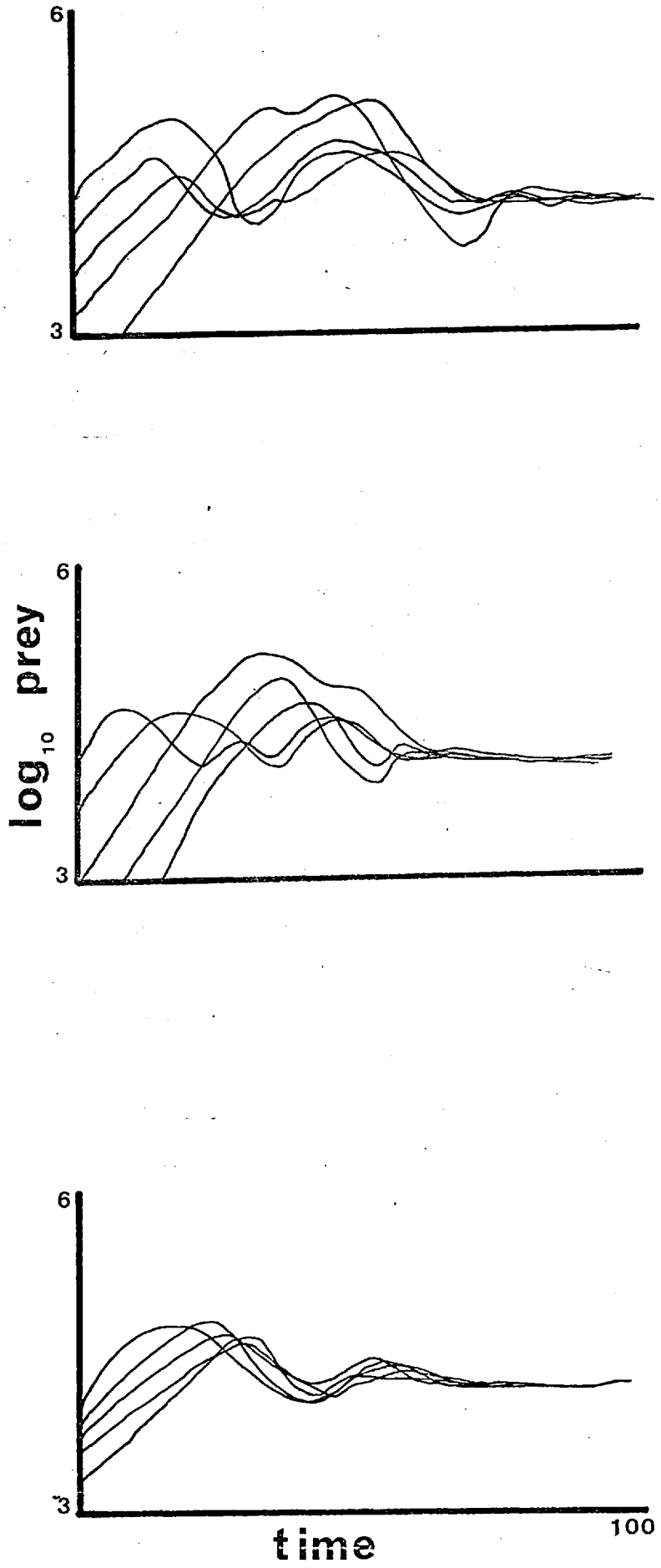


Figure 159. The pattern of aphid abundance in five areas with three initial prey distributions. Aggregation an increasing function of mean prey density

the biological attributes of potentially useful pest control predators affects both the number and the time-pattern of prey killed. The optimal pattern, and the ideal age-selectivity have been specified by running the aphid population model described in Chapter IV.

Predation in biological control, especially of pests of annual crops grown in rotation, is somewhat removed from the idealized situation of a simulation model. By integrating all the available information on potential predator species, however, the model can form a basis for choosing between candidates, even if it can not predict with great precision the actual level to which a given species will reduce aphid numbers in a particular situation. The latter type of information is best gained by direct experimentation, but the model can help in this too, by showing which types of experiment are most likely to be rewarding.

1 Functional Responses

I have used three functional responses which between them tend, under most circumstances, to reduce the intake of prey per predator below the optimum required for growth and reproduction. Two of the responses (to absolute and to relative prey density) tend to stabilize the interaction between the predator and aphid numbers, and, depending on the density at which prey consumption is minimal, can reduce pest numbers to a level at which no economic loss of crop would occur.

The third functional response concerns the time wasted by individual predators in what Hassell (1971) has called mutual interference. This response is destabilizing because prey intake tends to decrease as predator numbers increase. If, then, the high predator numbers were brought about by

increasing prey density, the prey will continue to multiply, but at an increasing rate (since fewer and fewer are being eaten). The only time at which this response could act to stabilize the interaction would be when prey were scarce and predators were very abundant. In this case, however, the functional response to relative prey density would act to decrease the feeding rate, and prey numbers could recover. This observation is in direct contrast to the observations of Hassell and Rogers (1972) and Hassell and May (1973) where it was shown that mutual interference was stabilizing under most conditions. This discrepancy only highlights the differences between models of discrete generations with very simple numerical responses, and simulations of overlapping generations with complex numerical responses. It is clear that the indiscriminate incorporation of apparently robust theories at one level into models at another level should be carefully avoided. This serves to highlight the point (Chapter I) that models should not be used to tackle questions they were not specifically designed to answer.

2 Numerical Responses

The functional responses, by reducing the aphid intake of individual predators, act to produce a state of starvation which varies in intensity between the different age classes of predators (because they are of different sizes, and compete for prey with different success). This starvation in turn has been assumed to affect the mortality, birth and emigration rates; it is responsible for determining the magnitude of the numerical response. The three effects act in different ways upon subsequent prey population change. The survival response can be very important, and strongly stabilizing, depending

on the degree to which starvation affects mortality. It also acts immediately, since an animal dying today can not feed tomorrow, but the synchrony of the response does depend upon the number of days of starvation necessary to increase the death rate.

The dispersal response can also act immediately, so that adult predators will leave the area if they can not find sufficient prey. Redispersal is also associated, however, with changes in the pattern of oviposition (see below), and the response to decreases in fecundity can only act after a time lag. If a starved female lays fewer eggs today, the predator population will not exert a reduced feeding pressure on the aphids until these eggs hatch into feeding larvae. The effectiveness of a numerical response in birth rate will therefore depend on the length of the egg stage, as well as on the extent to which fecundity is affected by starvation. The response can clearly stabilize prey numbers (Fig. 148) but it tends to be less effective than the survival response, simply because the latter acts on the whole population, while only the adults lay eggs. Additionally, because adults are assumed to be superior in competition for prey, they tend to starve less than the immature stages, and hence the birth response tends to be under-compensatory.

3 Adult Dispersive Behaviour

The ability of adult predators to discriminate between areas of different prey availability has been shown by several authors (Hagen, 1966; Hassell, 1968). Hassell and May (1973) built a discrete-generation model of the interaction between a parasite and its host population, and investigated the effects of changing the value of the aggregation coefficient μ on the

stability of the system. They came to four conclusions; a) that increasing μ increased stability: b) that stability increases as the heterogeneity of initial prey distribution increases (they wrote "when there are more low host density regions"): c) that stability exists in a wider range of conditions for μ about 0.5 than at other levels: and d) that stability breaks down as the reproductive rate of the prey increases.

It is interesting that in the first two conclusions the present model produces the opposite results (i.e. decreasing μ increases stability; compare Figs. 157 and 158; and increasing heterogeneity in initial prey distributions can decrease stability; see Fig. 157), while both models agree on the last two points. This further emphasizes the difference between discrete and overlapping generation models, and, perhaps to a lesser extent, between models of predator and parasitoid attack.

The results obtained in the present model can be explained in terms of the time lag between oviposition and the onset of feeding. When the aggregation response is very strong, large numbers of predators are attracted to the regions of high relative prey density, where they feed and lay many eggs. On the next day, however, the same areas will still be attractive because the eggs laid by the adults can not affect prey numbers. In this way the same area can remain highly attractive until the larvae hatch and begin to feed. Depending on the duration of the egg stage, there will be a massive over-compensation to prey density with the result that, when all the eggs have hatched, there will be a rapid depletion of prey followed by predator starvation. High aggregation within a generation therefore tends to be destabilizing because it

is over-compensatory; the adults do not appreciate the starvation they are about to inflict on their progeny.

This point serves to suggest new ways in which we might look at predator aggregation. The behaviour outlined as accounting for the results of the simulation would probably not be followed by a real predator. In the first place, it could sense the number of eggs laid by members of its own species in a sub-area, and could well show a tendency to avoid heavily attacked prey regions (this would parallel Hassell's mutual interference - 1971). Again, because of the time lag in feeding response, areas which are initially low in prey will be given an extra period after they have become attractive, but before feeding greatly increases, in which to increase in abundance. This period may be sufficient to allow the prey population to escape control (see Figs. 156, 157).

To return to the original example, the decreasing aggregation of Cyzenis albicans with increases in the density of its hosts must be explained not as a stabilizing mechanism (as I originally suspected), but rather as a straightforward reduction in the necessity to aggregate as mean host density increases. In other words, when prey are abundant, the parasites can afford to be less discerning in choosing areas for oviposition. Here again, we highlight the difference between what a real species does under natural conditions, and what an ideal biological control agent should do in a crop monoculture.

An improved model of predator aggregation should therefore consider the relative density of prey, but also the number of predator eggs already laid. This would involve calculating μ as a function of both mean prey density and either the number of predator eggs, or the number of predators attempting to oviposit in the area. In this sense, the

aggregation model should include an element of intraspecific discrimination on the part of the predators, perhaps after the fashion of Hassell's mutual interference equation (see Functional Responses, above).

4 Reproductive and Developmental Biology

A predator species will only be as effective in biological control as its ability to increase in numbers parallel to pest abundance. While the numerical reponses act to tune the rate of predator increase to prey density, they can only exert a depressive effect; that is, starvation can only lower the number of eggs produced per female. Unless the maximum rate of oviposition (in the absence of prey-limitation) is sufficient to keep the predator's population within 'reach' of the prey the species will not be effective in pest control. The rate of increase in predator numbers is not determined by egg-laying alone, of course, but also by the time it takes for eggs to develop into breeding adults. As shown in Section B-3 of this Chapter, both egg and pupal durations have a significant effect on the pattern of aphid abundance.

On becoming adult, it is also apparent from the simulations that the pattern of egg-laying with age is important to the degree of control exerted (Fig. 152). This is unlikely to present any problems in reality, since few predators lay more eggs as they age (the least effective strategy).

5 Growth and Voracity

In general, large animals have large food requirements even though, by merit of their relatively low surface area to body weight ratio, they may be rather efficient at using the food they obtain. The most obvious suggestion for an ideal

predator would then be that it should be large (with, by implication, a high growth rate), and voracious (e.g. van Emden, 1966). The problem with this assertion is that animals with high individual food requirements will require higher prey densities for satiation; the equilibrium level of pest abundance will therefore tend to be higher, and hence more damaging to the crop. In addition, because of their high demands, large predators will tend to immigrate into the crop later in the season, and emigrate from it earlier; both conditions which seem to be undesirable (Chapter III and IV).

It became clear when running the model of aphid population growth that the ideal predator should be able to attack all aphid instars, and so this constraint sets a lower limit to predator size. From this Chapter, however, we have seen the importance of being small enough to tolerate rather low prey densities. It is clear that our optimal predator must be of intermediate size.

By allowing that the growth rate of the larval predator is determined by the number of prey it consumes (as in the present model) this size constraint can be somewhat relaxed, as it leads to a situation of having large predators when aphids are abundant and small predators when they are scarce. The ideal species would therefore be able to show a wide range of sizes within any instar, depending upon the conditions of aphid availability.

6 Age Structure of the Predator Population

Population models are often built on the assumption that the age structure of the population is constant, and that the proportion of the animals in any particular developmental stage is invariant. In most field situations where the envir-

onment is seasonal this is very rarely the case. Animals emerge in the spring from their dormant phase, and at this time they are all in the same instar (if they overwinter as eggs, they will be first instar larvae, or if as pupae, they will be adults). The age structure is therefore discrete; some age classes have no individuals in them at all.

The importance of the age-structure is twofold; first, with a discrete structure there are periods of complete inactivity during which no prey are killed, and second, there are times during which there are no adult insects, and hence no reproduction.

The age-structure will tend to stabilize with time, however, under three processes. First, spring emergence is not usually completely synchronous, and so some predators are just emerging from dormant eggs while others have reached maturity. Second, if the adults lay their eggs over an extended period rather than in one batch, then more age classes will be represented which, on becoming adult in their turn, will continue the process of age-stabilization. Finally, in the context of an annual crop, the longer the period of adult immigration, the more rapidly will the age-structure of the predator population stabilize. In terms of field practice this suggests that a predator spending its early spring stages on a variety of wild plant species will be ideal, as adults will emigrate form the different plants at different times. The importance of the timing of predator immigration relative to aphid population growth has been discussed in Chapter IV.

7 Polyphagy or Monophagy ?

One question we must ask is whether it is better to have

a completely specific predator, or a species which eats many types of aphid but prefers Aphis fabae ? Rather few real aphidophagous predators are, in fact, monophagous (Iperti, 1966; Hodek, 1966), but a number of parasites may be (Evenhuis, 1966). The model shows that monophagy is desirable under all circumstances except those which would otherwise cause the predators to emigrate. In other words, our ideal animal would feed exclusively on the target species until its availability was so low that the predator would normally emigrate (thereby allowing the possibility of reinfestation and crop loss), at which time it would switch to the consumption of other prey living in the same area. This feeding strategy is simply the extreme of a straightforward 'attack in relation to availability' predator; presumably the target pest species is the most abundant animal feeding in the crop (if it were not, then the more abundant species would presumably be the main pest), and so it will be attacked to a greater extent even by polyphagous predators. The main point of concern is that the alternative prey consumed are essentially wasted in terms of pest control because their populations are below the economic threshold. We must bear in mind, of course, that the other insect species dwelling in the crop are non-pests simply because they are prey to polyphagous predators, and that because of this, there may not be sufficient of them to support a population of monophagous predators.

In short, we require a predator which can be monophagous at high pest densities and polyphagous when the target aphids are rare. Mechanisms for such a switching in predation have been discussed by Murdoch (1969).

8 Pattern of Mortality

I have already discussed the effects of starvation on mortality, and the contribution which this process makes to the numerical response of the predator to prey density. Equally important, however, is the magnitude of the non-starvation mortality, since this will determine the rate at which the predator population grows in conditions of optimal prey availability. The agencies of mortality of concern here are predation by birds and small mammals, attack by insect parasitoids, and exposure to extremes of climatic environment. They may, in the case of parasitism and predation, act in a density-dependent fashion so that as the abundance of our predator increased, so would its death rate. This is obviously undesirable, since it would act in the same destabilizing way as did the mutual interference response in Section A-1c; the rate of aphid consumption would decrease as the number of aphids increased, and an outbreak would occur.

In consequence, our predator must suffer low mortality rates in general (to allow rapid build-up), and very little density-dependence in mortality in particular (to avoid under-exploitation of the pest aphid). This would be possible if our predator remained rather scarce, or developed protective devices either by being distasteful, or by mimicking a distasteful species. We have already seen, however, that abundance is preferable to large size (when a given number of prey must be consumed), so the avoidance of density-dependent mortality through remaining scarce is not an attractive alternative. Our objective, then, is to find a predator which is food-limited with regard to its mortality rate, and following on the same line of argument used in Chapter IV, mortality

should be minimal at the age of maximum reproductive value, and maximum at low reproductive values on the 'old side' of the optimum (since we would like the predator to have eaten many aphids before it dies). This is the converse of the mortality pattern we demand in the pest population; there we want maximum mortality at maximum reproductive value, and high mortality in the young stages so that less damage is done to the plant.

9 Searching Behaviour

The most efficient pattern of searching behaviour in seeking out a prey species which lives in tight-knit aggregates is clearly to find an aggregate as quickly as possible (by sight or scent), and then to stay in the vicinity until as many prey have been consumed as possible. This has been demonstrated in model populations (Murdie and Hassell, 1973) and with aphid predators in the field (coccinellids by Banks, 1964; syrphids by Chandler, 1969). Searching efficiency appears in the model as the response of the predator to prey density; in effect, the steeper the functional response curve in Fig. 138, the better the predator at discovering, or staying close to, having once discovered, aphid aggregates. The processes of search themselves, whether they be random walks or klinokinesis, are not considered in detail. I do consider, however, the response of the more mobile, adult stages to differences in prey availability over larger areas. This is not so much a pattern of searching behaviour as it is a dispersive response which allows that searching will subsequently be more rewarding.

10 The Properties of the Optimal Predator of Aphis fabae

Having discussed the biological attributes of the ideal predator for the biological control of Aphis fabae on broad

beans in some detail, we are now in a position to catalogue them. The order in which the attributes are listed does not signify their relative importance.

Body Size

Large enough to attack all sizes of aphid, but small enough to tolerate low prey densities without emigrating.

Growth Rate

Commensurate with optimal body size; ideally a function of food intake so that body size and hence food demand varies with prey availability. High prey, high growth rate; low prey low growth rate.

Sex Ratio

As high as possible, constrained by the probability of achieving 100% mating success. Sex ratio can be lower if the males feed voraciously.

Fecundity

As high as possible, and spread over adult life (not a single egg batch) to rapidly stabilize age structure. Fecundity should decrease with age if only a certain number of eggs can be produced, otherwise it should be constant.

Number of Instars

Not itself important, but the fewer the better (below). Some feeding time is wasted during and immediately following each moult.

Duration of Instars

All instars except the adult should be as brief as possible, If the only predator available does not feed on aphids as an adult, then rapid progression through the instars is less important, as only the larvae act as biological control agents (e.g. Syrphidae and Cecidomyidae).

Dormant Stages

The duration of non-overwintering egg and pupal stages should be as brief as possible; they slow population increase and do not feed.

Voltinism

Multivoltine; a univoltine predator can not undergo numerical responses within the season.

Feeding Rate

In the sense of weight of prey required for maximum growth, survival and fecundity. Should be high but flexible (see Growth Rate above) and determined by body size.

Survival Rate

High; all mortality other than that caused by starvation (below) should be independent of predator density. Abiotic factors ideal.

Natural Enemies

Few; those which do attack should not do so in a density-dependent fashion. Since the predator will sometimes be abundant it should be distasteful, or a colourful mimic, to reduce density-dependence in mortality.

Functional Response to Prey Density

Strong; there should be an aphid density greater than zero at which no prey are consumed. The shape of the response (Fig. 138) affects the frequency and amplitude of prey numbers, and the optimal value of v (Equ. 26) depends upon the relationship between the economic threshold and the zero-feeding density.

Functional Response to Relative Prey Density

Strong; the predators should respond to food competition. When this response occurs it means that the predator is doing, or about to do, too well in reducing aphid numbers, and runs the risk of eliminating its food source.

Functional Response to Predator Density

Very weak or non-existant. This response is highly destabilizing, and the only time it might effect a useful reduction in predator feeding would be at very low relative prey densities. The Relative Response, above, could do this equally well. There should, however, be a response in oviposition behaviour such that areas of high egg density are avoided.

Numerical Response in Survival Rate

Depends upon birth rate; if the fecundity is high then this allows a strong, immediate response (the most effective). Otherwise a compromise is necessary; either a less strong response , or a strong response after a time lag. The situation to be avoided is over-compensation so that starvation-induced mortality lowers the predator population to a level from which it can not control the subsequent prey resurgence.

Numerical Response in Birth Rate

Less important than the above, but still strongly stabilizing. If maximum birth rate is high enough to allow good recovery, then the response should be strong (e.g. no births when STARVE(I) = 1).

Numerical Response in Emigration Rate

Depends on the likelihood of re-immigration if prey numbers increase; this in turn depends on the minimum number of aphids which forms an attractive population. If the predators are unlikely to return, the response should be slight, especially if the threshold aphid density at which they emigrate is high (as it might be if the predators were large). Otherwise, if predator return is likely, and the emigration threshold density is very low, then the response can be strong, and will be immediate and highly stabilizing.

Polyphagy

Monophagous at high prey density; polyphagous at low.

Timing of Immigration

After the aphids have arrived in the crop and before they have built up to high levels. Of crucial importance to the effectiveness of control.

Probability of Immigration

Dependent on the release of predators from other host plants; this aspect is therefore highly probablistic. The alternative is costly release of predators by the grower. Once released from wild plants the probability of immigration depends on prevailing winds and so on, but also on the

threshold density of aphids in the crop necessary for attraction. Predators should come from a range of wild plants to increase the period of immigration.

Temperature Threshold of Emergence

Eqaul to or less than that of the aphid pest. If no inremediate plants are colonized then the threshold can be higher, but it can not be so high that it is only reached when the aphids have arrived in the crop.

Temperature and Physiology

The rate of population growth in the predator must equal or exceed that of the prey at all temperatures likely in the field, otherwise increases or decreases in mean temperature would bring about a pest outbreak.

Weather Factors

The climatic tolerances of the predator should be optimal for all areas where the pest is found; in general this will mean that the area over which a species is distributed naturally will be an indication of its likelihood of acting as a good biological control agent in a new area.

Food Preference

Depends upon predator size; if the predator is large enough to consume all sizes of prey, then it should concentrate on those of maximum reproductive value, taking relatively younger aphids in preference to relatively older (see Fig. 114 in Chapter IV). If no predator can be found to consume all age classes, then more prey will be killed by selecting a predator to attack the smallest aphids (or, in general, the most abundant size class).

Dispersive Ability

Predators should be able to differentiate between areas of different prey density, and to lay their eggs in relation to the number of eggs already present as well as to the relative prey density. The intensity of the aggregation response should increase with mean prey density.

Prey Refuge

The structure of the environment, or the behaviour of the predators, should be such as to allow for the survival of a number of pest insects. This number must be sufficiently large to give rise to an attractive population which will maintain predator numbers, and sufficiently low so as to cause no significant crop loss.

Spatial Pattern of Feeding

Near the surface of the bean canopy, where aphid feeding causes most damage (see Chapters II and III).

Age Structure

As stable as possible; this will be increased by prolonged immigration and by rapid development and extended oviposition, and decreased by a tendency to lay all eggs in one batch (if development is synchronous).

11 Candidate Species

Aphids fall prey to many types of predators including birds, spiders, and many insects (Imms, 1947). Of the latter, beetles (Coleoptera, and Coccinellidae in particular), lacewings (Neuroptera), hover-flies (Syrphidae) and some midges (Cecidomyidae), form the most important groups. No single

species from any category is optimal in all respects, but the groups can definitely be ranked, and candidate species within any group classified in terms of their control potential.

Neither adult syrphids nor adult cecids feed on aphids, and their control potential is thereby diminished. They may, under certain circumstances, be useful in biological control by preferring an aphid which is ignored by other groups (J. J. Brightman, pers. comm.).

The model could best be used in discriminating between various potential coccinellids, syrphids or lace-wings whose biology was rather well known; the principal shortcoming of the model is that it requires a rather detailed understanding of the biology of the candidate species. On the other hand, the completely empirical approach advocated by some ecologists (e.g. Gifford, 1971) would demand numerous expensive field trials with animals whose control potential could, had a model been employed, have been eliminated a priori. The model only proposes an order in which to conduct the field trials, and does not presume to predict exactly which predator species will be optimal in a particular situation.

One important aspect of the list of attributes stands out. This is that while no one species is optimal in all respects, there is no respect in which a species of some sort might not be optimal. This fact would argue for the implementation of multiple-species control, especially when the optimal attributes appear to change as the season progresses. Thus we find that our ideal predator must be highly synchronized with the pest, so that it immigrates before economic losses are caused, and have the ability to increase rapidly in numbers in the early stages of the infestation. On the other hand, it must be able to withstand rather low pest densities if later

outbreaks are to be avoided. Again, the animal should be monophagous at high aphid densities but polyphagous when prey are scarce. These requirements are unlikely to be combined in a single species, because selection would have operated to produce one set of attributes or the other. A sequence of predator species, however, might combine all the desirable attributes; a fast-breeding ladybird could be optimal in the early period of growth, followed by a syrphid species which could withstand lower pest densities and maintain the aphids below the economic threshold. Finally, perhaps, a predator which tended to stay in the crop up to harvest, feeding only rather slowly but ensuring continued control, might be released (a lace-wing species for example). It is unlikely that a single predator species of Aphis fabae could be found which would work alone in the conrol of the pest under British conditions.

12 Model Assessment

Of the models of parasitism and predation reviewed in the introduction, the population model presented here most closely resembles that of Hassell and Rogers (1972). It differs from their model in several respects because it was built specifically to investigate the properties of an ideal predator, rather than to describe extant bodies of data. First of all, it considers predation rather than parasitism; this distinction is discussed in depth by Royama (1971). Second, I have laid emphasis on the number of predators rather than on the number of prey in calculating the number of prey killed (Watt, 1959, made the same distinction), and this treatment removes any implicit assumptions of density dependence in predation which may occur when writing $N_{ha} = f(N).N$ instead of $N_{ha} = f(N).P.C.$

Third, the predator population is considered by age classes, and no assumptions of stable age-distribution are made; the incorporation of non-feeding stages in the life-cycle, and the dependence of development time upon temperature further complicate an analytical comparison of the two models, but as shown earlier, it is possible to write an algebraic approximation to the attack equation for one predator age class. The calculation of the number of encounters is given by Hassell and Rogers (1972) as

$$N_a = \left[\frac{a''.a P^{-\beta}.T.N}{1 + a''.T'.N}\right] P$$

while the functional responses in the present model can be approximated by writing

$$N_a = P.C\,(1 - (T'/N)^{v})(1 - e^{-(b.P/N)})(1 - (K.P)^{2w})$$

where the notation is as described on page 309. The principal difference in content is that I have considered an additional functional response, namely that to relative prey density, appearing as the second bracketted term in the equation. Both these models must assume some pattern of searching behaviour to allow for prey exploitation; typically we assume random search and allow that the fraction of the prey population escaping attack is given by the zero term of the Poisson Distribution, $e^{-Na/N}$ (see Pielou, 1969). The actual proportion encountered is therefore $1 - e^{-Na/N}$, and so the number killed is

$$N_{ha} = N\,(1 - e^{-Na\,/\,N})$$

I have assumed that there is exploitation within the

predator population, so that the larger predators are allowed first pick of the aphid crop. The prey population is then depleted and the next size class is allowed to feed. As discussed earlier, this system is completely arbitrary, its sole purpose being to distribute the relative effects of starvation differentially between the predator instars. If we thought that the young suffered starvation less frequently than the adults, it would be quite straightforward to reverse the feeding order and model the observed effect.

Most of the conventional models of predation and parasitism assume that within a generation feeding or oviposition occurs instantaneously, and that the number of parasites in the following generation is simply the number of eggs successfully laid (perhaps multiplied by a survival rate). In biological control situations, this type of approach might apply to pests of perennial crops with highly specific parasites, but in an annual crop with poyphagous predators it almost certainly does not. On the contrary, the details of the numerical responses occurring within a set of overlapping generations will be the factor of over-riding importance in determining whether pest control is achieved or not. For this reason the current model deals with numerical responses in some detail, and fecundity, mortality and emigration rates are all computed on a daily basis in relation to the current conditions of prey availability. The model is simple in its present treatment of these responses in so far as it only considers the starvation experienced on the preceeding day; no account is taken of any accumulative effects of starvation, for example.

The novel feature of the model is the combination of a relatively complex treatment of functional responses with a

consideration of the effects of predator aggregation behaviour. This has brought to light two main points. First, that while a functional response to predator density (mutual interference) is stabilizing in discrete-generation models (e.g. Hassell and Rogers, 1972) it has the opposite effect when applied to models of overlapping generations. Second, that the factors affecting the stabilizing properties of predator aggregation differ in discrete models of parasitism and overlapping models of predation. In the former, stability is enhanced by increases in the aggregation index, and by increased heterogeneity in initial prey distribution; in the latter, the converse is true. It has also become clear that a treatment of predator aggregation should not ignore the effects of predator density (especially the density of non-feeding egg and pupal stages) in determining the pattern of predator egg-laying over the areas.

All the data sets in the model are intuitive. This is deliberate, and allows that the attributes of the ideal predator will not be coloured by any preconceptions about particular species.

CHAPTER VI ON THE RELATIONSHIP BETWEEN THE COMPLEXITY AND THE UTILITY OF SIMULATION MODELS

Introduction

The models described in the preceeding chapters serve to highlight a number of points about management models in general. For the purposes of discussion we can define two poles in the spectrum of resource management problems; those concerned with accurate numerical predictions (usually in relation to economic analyses), and those concerned with obtaining a broad, qualitative understanding of the response of the system to a given manipulation (usually by considering the detailed biology of the organisms involved). As I shall attempt to show, these two types of problem require quite different approaches in model building, and have divergent data requirements. It is of crucial importance to the success of an ecological investigation to determine explicitly whether

a quantitative or a qualitative answer is required, and to go about the analysis and modelling in the appropriate fashion.

1 Quantitative Problems

A large class of management problems poses questions of the type 'How much ?', 'When ?', 'Where ?', 'How many ?', and so on; these we shall call quantitative problems. The answers they demand are accurate numerical predictions which can be used directly and immediately in management.

The only efficient means of answering this class of problems is empirically; that is, by going out and doing the experiments. In most cases modelling will only be a time-consuming and irrelevant diversion, and is most unlikely to benefit the management effort. In some instances, however, the management problem suggests experiments which are either very difficult or very expensive. Here, it may be possible to solve the problem theoretically by resort to some form of economic analysis (e.g. linear programming; Noble, 1964). The important point is that biological simulation models as described in Chapter I hold very little potential for answering this class of problem.

2 Qualitative Problems

The second class of management problems demands solutions which are essentially qualitative. The aim is not to predict exactly what will happen, but rather why it should have happened. The questions are directed at increasing our understanding of the resource system, so that the quantitative questions we ask in the future can be better geared to the system's biological behaviour.

Qualitative management problems will usually be complex

(in the sense that they can not be tackled by mental deduction), and it is important to decide on the degree of biological complexity which must be considered in the model in order to fulfil its objectives. As the complexity of a model increases, however, so do the costs of its construction and the difficulty of its final interpretation. We should therefore aim for the minimum complexity necessary to the solution of the problem, but to do this, we must understand the relationship between complexity and utility.

a) Complexity

i/ Number of Variables

In broad terms, there is an increasing relationship between the number of variables considered and the complexity of the resulting model. The number of variables in the model will increase as the manipulation increases in 'causal-distance' from the output (the 'width' of the flow-diagram; see Fig. 6), and with increases in the degree of biological detail into which each component process is analysed. We can not reduce the 'causal-distance', but the degree of analytical detail must be decided. Throughout the preceeding chapters I have used as a criterion for assessing the merits of further analysis whether or not the process in hand is likely to vary in its behaviour in such a way as to affect the output. If the process behaves in a consistent and predictable pattern, there is no need to analyse it further. If, on the other hand, the process is known to vary under the manipulation (e.g. photosynthetic rate under aphid feeding), and this variation is likely to affect the output (bean yield), then further analysis is warranted. Instead of including photosynthetic rate as a constant, therefore, the factors which affect its

rate are elucidated, and inspected in the same way. So it is that some processes are analysed in considerable biological detail (e.g. photosynthesis), while others in the same model which are either less strongly affected by the manipulation or are inherently less variable (e.g. water and nutrient uptake) are treated very simply.

ii/ The Inter-relatedness of Variables

The complexity of a model also increases with the extent of the inter-relatedness of its variables. If we have a number of variables, the simplest model is that in which each affects, and is affected by, only one other; the model, in other words, is a causal chain. Clearly, then, the most complex model of a given system will be that in which each variable affects, and is affected by, all the others; this model is a complete causal network.

Just as before, when deciding upon the number of variables to consider, the criteria for including a particular interaction must be a) whether the interaction importantly affects the variables concerned, and b) whether it is likely to occur at an intensity which will be important. Both of these criteria will be implicit in the question under scrutiny.

It is essential that each interaction be carefully considered, because it is from interactive processes that much of the interesting behaviour of the model springs (the interaction of different organs in the distribution of photosynthate in Chapter III, and the effects of density-related processes like birth and dispersal rates in Chapters IV and V).

As the number of interactions increases then so will the number of data sets required by the model. In a causal-chain model with N variables, there will be (N-1) data sets needed,

but if the model is a complete network, the data requirements will rise to (N^2-N). There is an obvious incentive to keep the number of interactions as low as possible.

Not only does the number of data sets increase with the number of interactions, but also the difficulty of obtaining the data. When interactions occur one must either make potentially misleading assumptions as to the form of the interaction (see Chapters I, III), or collect the data by factorial experiments. Factorial data are less abundant in the literature (and therefore the relationships are less likely to be already quantified), and also more expensive to gather, than are data from controlled-environment, single independent-variable experiments.

iii/ Levels of Organization

Odum (1959) lists a number of 'levels' at which biological processes can act, namely biochemical, organelle, cell, tissue, organ, individual, population, community, ecosystem and biosphere. The third aspect of model complexity relates to the number of levels which must be considered in solving a given problem. We can suggest that the more levels involved, the more complex the model will be. This is because processes which operate on one level are affected by the summation of those processes operating at lower levels; the growth of an animal can be expressed at the individual level as a single dry weight increment, but at the lower level this must be replaced by the sum of the dry weight increments of its component organs. Since increasing the number of levels will inevitably increase the number of variables (and probably the number of interactions), the data-requirements will be higher for many-level than for single-level models. Also, because

data tend to be collected at one level without a view to synthesis to a higher level, it is not always clear how low-level processes act together to produce an effect at a higher level (for example how plant 'sinks' compete for the reserves available, and how this process manifests itself in the dry weight increase of a single sink organ).

The models considered in the foregoing chapters ranged in level from organs (the different plant parts in Chapter III), through individuals (the aphids in Chapter IV; their growth and feeding rates), to populations (of aphids and predators in Chapters IV and V). The question tackled had the output expressed at the level of the organ (the weight of beans), and the manipulation stated jointly in terms of individual and population attributes of the ideal predator species.

b) Utility

The utility of a model can be expressed most simply as the extent to which it answers the questions posed. The difficulty lies in discovering the accuracy of the answers, since, in most cases, it is not known in advance what the precise repercussions of the manipulation will be in a real ecosystem. This raises again the important issue of validation; the determination of how realistically a model represents the real system, and the degree of confidence with which its suggestions can be implemented as management practices.

A model can be said to be validated if, under a range of conditions, the output from the model agrees with observations of the real system. The definition of this agreement should be made in advance, so that, for instance, we might allow that the model be validated when there is less than 10% disagreement between observed and predicted levels in the

output variable.

It is crucial, as Goodall (1968, 1971a, b) points out, that the data used to validate the quantitative behaviour of the model be collected independently of the data used to describe relationships within the model. If this is not observed then the validation simply measures the extent to which the model distorts its internal data sets in producing an output value ! Validation data typically consist of a time-series of measurements of the output variables, and of the conditions under which these outputs developed (often a series of meteorological observations). The model is then assessed by running it over a similar time period, using the field-measured conditions as driving variables; the sequence of real and predicted outputs can then be compared to assess the quantitative and behavioural validity of the model.

Once the validation data have been collected, and the outputs compared, the discrepancies which appear must be explained. There is a school of thought (see, e.g., Plinston, 1972) that the best strategy at this point is to feed the validation data into the computer and run the model under an optimization program to determine the set of parameters which gives the best fit between the observed and predicted outputs. This method has the serious draw-back that it distorts the data sets which were incorporated in the initial model. This will be irrelevant if the data sets were all intuitive, but it will be seriously misleading if they were the result of careful, well-replicated experiments. In fact, this method of parameter-fitting can be more than a little dangerous, and, to use de Wit's words (1970), "the model degenerates from an explanatory model into a demonstrative model which cannot be used anymore for extrapolation, and the

technique reduces into the most cumbersome and subjective technique of curve fitting that can be imagined." Additionally, the more complex the model becomes, in terms of the number of variables included and the degree of their inter-relatedness, the easier it is to obtain a perfect fit between predicted and observed values by 'parameter-fiddling'.

The difficulty in assessing the causes of departure between the behaviour of the model and real systems lies in the fact that there is no a priori method of determining whether the discrepancies are due to qualitative or quantitative errors in the model; whether our structure is incorrect through the omission of important variables or inter-relations, or whether some of the data sets are inaccurate or inappropriate.

If the model includes only data sets of high quality (as defined in Chapter I), then the structure of the model must be inadequate in some sense, since we know that if only X affects Y then the value of Y will be accurate for any X (by the definition of a high quality data set). In this case the model should be re-examined to see which potentially important variables might have been omitted, and experiments carried out where necessary to obtain the data necessary for their inclusion.

The more common situation, however, is that in which the data sets included are 'patchy' with respect to their quality, and some processes are better understood than others. Here, the problem of rectifying the discrepancies in model behaviour is more difficult. The only objective and rigorous procedure is to set up a feed-back between experimentation and modelling in each sub-section of the system, starting with those sections in which confidence is minimal, and progressing in turn to the best understood sections. Clearly, this procedure will be both expensive and time-consuming. It is necessary, therefore, to

relate this phase of the modelling process directly to the objectives of the research; if the exercise is principally theoretical, then the feed-back can continue indefinitely, whereas an applied problem will demand a strict definition of the accuracy required of the model, and of the resources available for the attainment of the desired level of precision.

Model utility therefore depends not only upon the answers obtained, but also on the possibility of showing them to be correct. In general, we can conclude that the utility of a biological simulation model increases with complexity so long as

1. the model remains compact enough to run in the computer at reasonable cost;
2. the data requirements are manageable;
3. the interpretation of model behaviour is unimpaired;
4. validation is possible.

Other Uses

Over and above their use in answering specific questions, biological simulation models have great additional utility. First of all, they act to impose a strict and rigorous logic on the analysis of a scientific problem by demanding a knowledge of precisely what affects what in the system, and in what way. By this means, apparently established ideas are brought into contexts in which their applicability can be questioned (e.g. mutual interference in predator feeding, Chapter V; the relationship between aphid density and alate production, Chapter IV).

Second, they form a useful precursor to experimentation, by showing which variables and processes are likely to be important in a given context. A great deal of time and expense

can be saved by collecting only those data which will actually be used in disproving an hypothesis (and see Watt, 1968).

Third, in building, running, and interpreting a biological simulation model, many novel hypotheses are suggested about the behaviour of the system. These can form the basis of later experimentation and research (e.g. does plant compensation significantly defray the yield reductions caused by aphid feeding ? will useful biological control predators aggregate more strongly at high rather than low prey densities ? and so on).

Finally, simulation modelling has great heuristic and educational value in uncovering the consequences of the synthesis of even the most simple ideas (Innis, 1971; Patten, 1966). For example it was impossible to tell before building the model whether the curve of predator aggregation reported by Hassell (1968) was a stabilizing or a destabilizing process. Again, it became clear that the functional response to predator density has quite divergent consequences when applied to aphid consumption and to predator oviposition patterns (Chapter V).

In short, while simulation modelling has a considerable number of drawbacks (Chapter I), and is ill-suited to answering quantitative problems (above), the benefits which it brings to the analysis of complex qualitative problems make it a very potent tool of ecological research. Its usefulness is not to be judged in terms of the number of spectacular predictions it fosters, but rather in the insight it allows into the functioning of the biological resource systems upon which we rely.

SUMMARY

1. A method is explained for the construction of problem-oriented computer simulation models, and a brief review presented of other types of model. A system is devised for tackling the complex problem of determining the biological attributes of a predator for Aphis fabae which would maintain aphid abundance below the level at which significant crop loss occurred.

2. An experiment is described to determine the effects of infestation by Aphis fabae on the growth and pattern of dry matter distribution in broad bean plants (Vicia faba). Aphid infestation reduced the rate of new leaf production (and hence subsequent net production), and reduced the fraction of dry matter incorporated into stem and fruit tissues.

3. The growth and fruit development of a model plant of V. faba is evaluated under different patterns of reserve removal

(aphid feeding). Factors of importance are the time of first feeding relative to germination, the amount of reserve removed, the pattern of removal from different nodes, and the surface damage caused by aphid presence (honeydew deposition, stylet insertion, and so on).

4. A model of the growth and feeding of an aphid population under different predation strategies shows that the ideal predator must select aphids of maximal reproductive value (or younger), be highly synchronized with aphid population increase, and show strong functional and numerical responses to prey density (so that it neither over- nor under-exploits the aphid population).

5. A model of the relationship between the biological attributes of a predator species and the number of aphids eaten is developed. Additionally, a scheme is laid out for investigating the effects of predator dispersal on the stability and abundance of aphids in different areas. The attributes of the optimal predator species are listed (page 381), and compared with known aphid predators. The applicability of multiple-species control is assessed.

6. The relationship between the complexity of a model and its utility is explained. Two discrete types of answer to resource management problems (accurate prediction and qualitative understanding) emerge which require quite different model-structures for their solution.

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APPENDIX

There follows a computer listing of the three models discussed. Program VICIA is the code of the plant model, APHIS of the aphid population, and PRED of the predator population including adult dispersal (Chapters III, IV and V respectively). All the subroutines these models call are included with the listings, and all the programs should run as they stand on CDC FORTRAN machines.

```
      PROGRAM VICIA (INPUT,OUTPUT,TAPE5=INPUT,TAPE6=OUTPUT)
      COMMON /BD01/BLANK,DOT,CROSS,NUMB(6)

C     ************************************************************************
C     *                                                                      *
C     *     A MODEL TO SIMULATE THE GROWTH AND FRUIT DEVELOPMENT OF          *
C     *     VICIA FABA L. UNDER AN INFESTATION OF APHIS FABAE SCOP.          *
C     *                                                                      *
C     ************************************************************************


C     ************************************************************************
C     *                                                                      *
C     *     DECLARE THE DIMENSIONS OF THE VECTORS AND ARRAYS USED            *
C     *                                                                      *
C     ************************************************************************

      DIMENSION TEST(200)
      DIMENSION IHEAD(20),LABELS(6,3),LABOUT(4,2)
      DIMENSION DIV(6),BNDS(12)
      DIMENSION
     +     X69(4),Y69(4),
     +     X70(4),Y70(4),
     +     X71(4),Y71(4),
     +     X72(3),Y72(3),
     +     X30(4),Y30(4),
     +     RADIAT(52),
     +     AREAL(25)
      DIMENSION
     +     WTL(25),
     +     THRMORT(25),
     +     PODWT(25),
     +     WTS(25),
     +     WTP(25),
     +     WTF(25),
     +     LIVE(25)
      DIMENSION
     +     LFAGE(25),
     +     TCGR(25),
     +     TEMP(25),
     +     TEMPOPT(4),
     +     FRACTL(4),
     +     FRACTS(4),
     +     FRACTF(4),
     +     FRACTP(4)
      DIMENSION
     +     OPTRPR(25),
     +     SINK(25),
     +     WTLMAX(25),
     +     WTSMAX(25),
     +     WTPMAX(25),
     +     DISTL(25),
     +     DISTS(25),
     +     DISTP(25),
     +     DISTF(25)
      DIMENSION
```

```
     +      NFLOWR(25),
     +      FLOSURV(25),
     +      FRUSURV(25),
     +      WTINIT(25),
     +      LPODAGE(25),
     +      FLOWRS(25),
     +      PODS(25),
     +      POLLEN(52)
      DIMENSION
     +      DENSLF(250),
     +      X20(4),Y20(4),
     +      X21(5),Y21(5),
     +      X23(4),Y23(4),
     +      X33(4),Y33(4),
     +      X73(5),Y73(5),
     +      X76(3),Y76(3),
     +      X77(3),Y77(3),
     +      X78(3),Y78(3),
     +      X80(4),Y80(4)
      DIMENSION
     +      TURGOR(25),
     +      APHFEED(25),
     +      QUALITY(25)
      INTEGER
     +      PBDEATH,
     +      COND
      REAL
     +      LEAFINC

C     ************************************************************************
C     *                                                                      *
C     *     DATA STATEMENTS ASSIGN INITIAL VALUES TO ARRAYS AND              *
C     *     PARAMETERS WHEN THE PROGRAM IS COMPILED                          *
C     *                                                                      *
C     ************************************************************************

      DATA TEST/40*0.,20*.09,20*.17,20*.27,20*.17,80*0./
      DATA
     +      APHFEED/25*0./,
     +      LFAGE/25*0/,
     +      DISTL,DISTS,DISTP,DISTF/100*0./,
     +      WTSMAX/.25,.08,23*.15/,
     +      WTPMAX/2*0.,.005,.007,.009,.01,.011,.012,.015,.022,.029,
     +      .035,.036,.038,.042,10*.045/,
     +      X69/0.,.2,.3,1./,Y69/0.,.5,.9,1./,
     +      X70/0.,.3,.6,1./,Y70/0.,.5,.95,1./,
     +      X71/0.,.5,.8,1./,Y71/1.,.95,.6,0./,
     +      X72/0.,.7,1./,Y72/.5,.9,1./
      DATA
     +      X30/0.,300.,600.,1000./,Y30/1.,.5,.3,.2/,
     +      X73/0.,5.,7.,9.,10./,
     +      Y73/1.,.9,.75,.4,0./,
     +      X76/0.,10.,20./,
     +      Y76/.3,.7,1./,
     +      X77/0.,8.,15./,
     +      Y77/.3,.7,1./,
```

```
     +      TEMPOPT/10.,15.,16.,14./,
     +      AREAL/25*0./,
     +      LIVE/25*1/,
     +      WTINIT/8*0.,7*.0008,5*.0005,5*.0003/,
     +      POLLEN/52*1./,
     +      TCGR/2*0.,.16,.2,.245,.285,.325,.355,.415,.44,.465,.44,
     +      .415,.38,.35,.32,.28,.22,.15,.1,.08,.05,2*0./,
     +      X33/0.,50.,80.,110./,Y33/1.,.95,.5,0./
      DATA
     +      WTL,WTS,WTF,WTP/100*0./,
     +      FRACTL/.786,.807,.765,.6/,
     +      LPODAGE/25*0/,
     +      THRMORT/25*0.4/,
     +      PODWT/25*1.8/,
     +      FRACTS/.137,.129,.092,.16/,
     +      FRACTF/0.,0.,.073,.23/
      DATA
     +      OPTRPR/25*.5/,
     +      WTLMAX/2*0.,.04,.058,.078,.103,.142,.174,.198,16*.2/,
     +      RADIAT/20*.08,32*1.5/,
     +      FLOSURV/8*0.,.5,1.,.8,.715,.625,.5,.429,.5,.4,.5,.6666,
     +            1.,5*0./,
     +      FRUSURV/8*0.,1.,.6666,.75,2*.6,.5,2*.6666,3*.5,6*0./,
     +      FLOWRS/8*0.,2.,3.,5.,7.,2*8.,7.,6.,5.,4.,3.,2.,5*0./
      DATA
     +      X23/.5,.7,.8,1./,Y23/.5,1.,1.2,1./,
     +      X20/0.,.3,.6,1./,Y20/1.,.98,.86,.7/,
     +      X21/0.,4.,6.,8.,14./,Y21/5.,7.,9.,9.5,10./,
     +      DENSLF/250*.0035/,
     +      TURGOR/25*5./,
     +      X80/4.,6.,8.,10./,Y80/1.,.5,.5,1./
      DATA BNDS/25.,0.,25.,0.,10.,0.,10.,0.,10.,0.,.05,0./

C     ***************************************************************************
C     *                                                                         *
C     *     SET INITIAL VALUES OF THE CONSTANTS USED                            *
C     *                                                                         *
C     ***************************************************************************

      READ(5,201) (IHEAD(J),J=1,20)
      READ(5,202) ((LABELS(I,J),J=1,3),I=1,6)
      READ(5,203) ((LABOUT(I,J),J=1,2),I=1,4)
201   FORMAT (20A4)
202   FORMAT (6(3A4))
203   FORMAT (4(2A4))
      CALL SCALE (BNDS,DIV,IHEAD,LABELS,LABOUT)
      PBLOCK=0.
      PBDEATH=6
      THRLF=0.0085
      SUPPLY=50.
      DECAY=.9
      SHOOT=ROOT=0.
      WTPLANT=0.
      SIGAPHF=0.
      PRODNET=SIGSINK=0.
      NODES=1
```

```
      MI=0
      EFFIC=0.02
      SHOOTIC=0.
      GPROD=TOTLA=0.
      MAXAGE=70
      X33(4)=FLOAT(MAXAGE)
      PBTHR=0.158
      PBRES=0.0077
      PBRES1=0.0025
      PBWT=0.435
      RESPR=0.01
      RESPS=0.01
      CHOEAT=0.
      WTTHR1=0.03
      WTTHR2=0.0336
      BASEWT=0.0076
      NNFL=10
      IPHOTP=75
      NDAYFL=0
      N4THR=120
      TCWT=0.
      GRL=0.07
      GRS=0.065
      GRP=0.06
      GRFL=0.3
      GRFR=0.08
      PODMAX=1.75
      NFTHR=13
      INITNO=9
      NOFRU=NOFLO=0
      WTFLOWR=0.025
      XYZ=.1
      MORTPT=14
      CWATER=1.
      THRLA=15.
      TLATHR=480.
      THR4=30.
      COND=1
      TOTL=TOTS=TOTF=TOTP=TOTLA=0.
      NW=2

C     ************************************************************************
C     *                                                                      *
C     *     BEGIN THE CYCLE OF DAYS                                          *
C     *                                                                      *
C     *     ALL THE EVENTS BETWEEN HERE AND STATEMENT NUMBER 1 ARE           *
C     *     SIMULATED ONCE EVERY DAY. DAY 1 IS ASSUMED TO BE THE DAY         *
C     *     UPON WHICH THE BEAN CROP WAS SOWN.                               *
C     *                                                                      *
C     ************************************************************************

      WTTHR=WTTHR1
      DO 1 IDAY=1,200
      IWEEK=IDAY/7+1
      DO 3653 NODE=1,NODES
3653  APHFEED(NODE)=0.
```

```
      IF (COND.EQ.3) NDAYFL=NDAYFL+1
      AIRTEMP=TEMPOPT(COND)
      SOILT=AIRTEMP-5.
      DO 42 NODE=1,NODES
42    TEMP(NODE)=AIRTEMP
C     ***************************************************************************
C     *                                                                         *
C     *     UPDATE LEAF WATER CONTENT IN RELATION TO APHID FEEDING              *
C     *     AND NATURAL WILTING.                                                *
C     *                                                                         *
C     ***************************************************************************

      IF (NODES.LT.3) GO TO 21
      DO 20 NODE=3,NODES
      AGE=FLOAT(LFAGE(NODE))
      WATER=TURGOR(NODE)*WTL(NODE)
      APHWAT=APHFEED(NODE)*CWATER
      WATRAT=APHWAT/WATER
      APHWF=F(WATRAT,X20,Y20,4)
      OPTWAT=F(AGE,X21,Y21,5)
      DIFWAT=TURGOR(NODE)/OPTWAT
      IF (DIFWAT.GT.1.) DIFWAT=1.
      DIFWAT=F(DIFWAT,X23,Y23,4)
20    TURGOR(NODE)=TURGOR(NODE)*APHWF*DIFWAT
21    CONTINUE
      TOTLA=GPROD=0.
      RESPB=0.
      APHF=1.
      TSINKL=TSINKS=TSINKP=TSINKF=0.

C     ***************************************************************************
C     *                                                                         *
C     *     CALCULATE RESPIRATORY LOSS                                          *
C     *                                                                         *
C     ***************************************************************************

      TOTLR=0.
      DO 649 NODE=3,NODES
      IF (LIVE(NODE).EQ.0) GO TO 649
      TOTLR=TOTLR+WTL(NODE)
649   CONTINUE
      SRESP=(SHOOT-TOTL+TOTLR)*RESPS
      IF (NODES.LE.3) SRESP=SHOOT*RESPS
      SRESP=SRESP*F(AIRTEMP,X76,Y76,3)
      RRESP=ROOT*RESPR
      RRESP=RRESP*F(SOILT,X77,Y77,3)
      TRESP=SRESP+RRESP

C     ***************************************************************************
C     *                                                                         *
C     *     CALCULATE RESERVES MOBILIZED FROM PARENT BEAN                       *
C     *                                                                         *
C     ***************************************************************************

      IF (PBWT.LT.PBTHR.OR.IWEEK.GT.PBDEATH) GO TO 40
      RESPB=PBRES
```

```
      IF (IWEEK.LE.2) RESPB=PBRES1
      PBWT=PBWT-RESPB
40    RESERVE=RESPB
      IF (COND.EQ.1) GO TO 2

C     ************************************************************************
C     *                                                                      *
C     *     CALCULATE GROSS PHOTOSYNTHETIC PRODUCTION                        *
C     *                                                                      *
C     ************************************************************************

      GPROD=0.
      TOTLA=0.
      ND=NODES-2
      DO 30 NODE=1,ND
      NN=NODES-NODE+1
C     ************************************************************************
C     *                                                                      *
C     *     FROM THE TOP OF THE PLANT DOWNWARDS, COMPUTE THE FOLLOWING       *
C     *                                                                      *
C     *     1. EFFECTS OF SUB-OPTIMAL TURGOR                                 *
C     *                                                                      *
C     ************************************************************************

      AGE=FLOAT(LFAGE(NN))
      OPTWAT=F(AGE,X21,Y21,5)
      TURGF=F(TURGOR(NN)/OPTWAT,X70,Y70,4)

C     ************************************************************************
C     *                                                                      *
C     *     2. EFFECT OF STOMATAL BLOCKAGE BY HONEYDEW                       *
C     *                                                                      *
C     ************************************************************************

      BLOKF=F(PBLOCK,X71,Y71,4)

C     ************************************************************************
C     *                                                                      *
C     *     3. EFFECTS OF RESERVE ACCUMULATION IN THE LEAF                   *
C     *                                                                      *
C     ************************************************************************

      CHOF=1.
      IF (PRODNET.GT.SIGSINK) CHOF=F(SIGSINK/PRODNET,X72,Y72,3)
C     ************************************************************************
C     *                                                                      *
C     *     4. EFFECTS OF TEMPERATURE                                        *
C     *                                                                      *
C     ************************************************************************

      TD=ABS(TEMPOPT(COND)-TEMP(NN))
      TEMPF=F(TD,X73,Y73,5)

C     ************************************************************************
C     *                                                                      *
C     *     COMPUTE THE TOTAL RATE REDUCTION, FACTORS                        *
```

```
C     *                                                                     *
C     ***********************************************************************

      FACTORS=TEMPF*CHOF*BLOKF*TURGF

C     ***********************************************************************
C     *                                                                     *
C     *     COMPUTE THE LIGHT AT THIS LEVEL IN THE CANOPY                   *
C     *     CURRENTLY I ASSUME AN EXPONENTIAL DECREASE IN INTENSITY         *
C     *     WITH ACCUMULATED LEAF AREA                                      *
C     *                                                                     *
C     ***********************************************************************

      IF (NN.EQ.NODES) GO TO 31
      TOTLA=TOTLA+AREAL(NN+1)
      ALIGHT=F(TOTLA,X30,Y30,4)*RADIAT(IWEEK)
      GO TO 32
31    ALIGHT=RADIAT(IWEEK)
32    PRODN=ALIGHT*AREAL(NN)*EFFIC*FACTORS

C     ***********************************************************************
C     *                                                                     *
C     *     COMPUTE THE EFFECT OF LEAF AGE ON PHOTOSYNTHETIC RATE           *
C     *                                                                     *
C     ***********************************************************************

      IF (PRODN.LE.0.) PRODN=0.
      LFAGE(NN)=LFAGE(NN)+1
33    AGEF=F(AGE,X33,Y33,4)
      PRODN=PRODN*AGEF
      IF (LFAGE(NN).LE.MAXAGE) GO TO 30
      LIVE(NN)=0
      WTL(NN)=WTL(NN)*DECAY
30    GPROD=GPROD+PRODN

C     ***********************************************************************
C     *                                                                     *
C     *     COMPUTE THE OTHER LIMITS TO GROSS PRODUCTION                    *
C     *                                                                     *
C     *     1. TOO LITTLE ROOT BIOMASS TO SUPPLY THE NEEDS OF THE SHOOT     *
C     *                                                                     *
C     ***********************************************************************

      UPFRAC=1.
      SRAT=1./OPTRPR(NODES)-1.
      IF (SHOOT/ROOT.LE.SRAT) GO TO 60
      UPFRAC=1.-((SHOOT/SRAT)-ROOT)/(SHOOT/SRAT)

C     ***********************************************************************
C     *                                                                     *
C     *     2. TOO LITTLE WATER OR NUTRIENTS IN THE ROOT REGION             *
C     *                                                                     *
C     ***********************************************************************

60    UP=ROOT*UPFRAC
      SOILF=1.
```

```
      IF (SUPPLY.GE.UP) GO TO 61
      SOILF=1.-((UP-SUPPLY)/UP)
61    ROOTF=F(UPFRAC,X69,Y69,4)

C     ************************************************************************
C     *                                                                      *
C     *     COMPUTE TOTAL GROSS PRODUCTION                                   *
C     *                                                                      *
C     ************************************************************************

      GPROD=GPROD*SOILF*ROOTF
      IF (SIGAPH.GT.GPROD+RESERVE) SIGAPH=GPROD+RESERVE
      GPROD=GPROD+RESERVE-SIGAPH
      GO TO 3
2     GPROD=RESERVE

C     ************************************************************************
C     *                                                                      *
C     *     CALCULATE NET PRODUCTION. GPROD LESS RESPIRATION LESS FEED       *
C     *                                                                      *
C     ************************************************************************

3     PRODNET=GPROD-SRESP-RRESP

C     ************************************************************************
C     *                                                                      *
C     *     IF RESPIRATION EXCEEDS GROSS PRODUCTION, THEN REDUCE             *
C     *     TISSUE WEIGHTS ACCORDINGLY                                       *
C     *                                                                      *
C     ************************************************************************

      IF (PRODNET) 17,750,16
17    RLOSS=SRESP+RRESP-GPROD
      ROOT=ROOT-(RLOSS*ROOT/WTPLANT)
      TCWT=TCWT-(RLOSS*TCWT/WTPLANT)
      DO 18 NODE=1,NODES
      WTL(NODE)=WTL(NODE)-(RLOSS*WTL(NODE)/WTPLANT)
      WTS(NODE)=WTS(NODE)-(RLOSS*WTS(NODE)/WTPLANT)
      WTF(NODE)=WTF(NODE)-(RLOSS*WTF(NODE)/WTPLANT)
18    WTP(NODE)=WTP(NODE)-(RLOSS*WTP(NODE)/WTPLANT)
      WTPLANT=WTPLANT-RLOSS
      SHOOT=WTPLANT-ROOT
      GO TO 750

C     ************************************************************************
C     *                                                                      *
C     *     DISTRIBUTE THE NET PRODUCTION                                    *
C     *                                                                      *
C     *     PRIOR TO THE FIRST LEAVES APPEARING AT WHORL 3 DO                *
C     *     THE FOLLOWING.                                                   *
C     *                                                                      *
C     ************************************************************************

16    IF (NODES.GE.2) GO TO 15
      ROOT=ROOT+PRODNET*OPTRPR(NODES)
      SHOOT=SHOOT+PRODNET*(1.-OPTRPR(NODES))
```

```
      IF (SHOOT.LT.THRLF) GO TO 760
      SHOOT1=SHOOT-BASEWT
      WTS(1)=SHOOT1*0.6666
      WTS(2)=SHOOT1-WTS(1)
      TCWT=BASEWT
      NODES=2
      GO TO 760
15    APHF=1.
      NODNEW=NODES+1
      TCNEW=EXP(TCGR(NODNEW)*APHF)*TCWT
      TCSINK=TCNEW-TCWT

C     ************************************************************************
C     *                                                                      *
C     *     NEXT, THE ROOT INCREMENT IS CALCULATED                           *
C     *                                                                      *
C     ************************************************************************

      WTPLANT=WTPLANT+PRODNET
      OPTRUT=WTPLANT*OPTRPR(NODES)
      PROD1=PRODNET
      IF (ROOT.GE.OPTRUT) GO TO 552
      PROD11=PROD1-OPTRUT+ROOT

C     ************************************************************************
C     *                                                                      *
C     *     IF NET PRODUCTION IS INSUFFICIENT TO SATISFY ROOT GROWTH         *
C     *     THEN SHOOT GROWTH IS ZERO.                                       *
C     *                                                                      *
C     ************************************************************************

      IF (PROD11.GT.0.) GO TO 551
      ROOT=ROOT+PROD1
      SHOOTIC=0.
      GO TO 750
551   ROOT=OPTRUT
      SHOOTIC=PROD11
      GO TO 553
552   SHOOTIC=PROD1
553   CONTINUE

C     ************************************************************************
C     *                                                                      *
C     *     COMPUTE THE RELATIVE SINK STRENGTHS OF LEAF, STEM,               *
C     *     FRUIT, AND PETIOLE SHOOT TISSUE.                                 *
C     *                                                                      *
C     ************************************************************************

      IF (COND.GE.4) GO TO 105

C     ************************************************************************
C     *                                                                      *
C     *     1. LEAF SINK                                                     *
C     *                                                                      *
C     ************************************************************************
```

```
      TSINKL=0.
      IF (NODES.LT.3) GO TO 571
      DO 56 NODE=3,NODES
      IF (LIVE(NODE).EQ.0) GO TO 6587
      FTURG=1.
      FHONEY=1.
      FWT=(WTLMAX(NODE)-WTL(NODE))/WTLMAX(NODE)
      SINK(NODE)=WTL(NODE)*EXP(GRL*FWT*FTURG*FHONEY)
      SINK(NODE)=SINK(NODE)-WTL(NODE)
      GO TO 56
6587  SINK(NODE)=0.
56    TSINKL=TSINKL+SINK(NODE)
      DO 57 NODE=3,NODES
57    DISTL(NODE)=SINK(NODE)/TSINKL

C     ************************************************************************
C     *                                                                      *
C     *     2. STEM SINK                                                     *
C     *                                                                      *
C     ************************************************************************

571   TSINKS=0.
      DO 58 NODE=1,NODES
      FADENS=1.
      FWT=(WTSMAX(NODE)-WTS(NODE))/WTSMAX(NODE)
      SINK(NODE)=WTS(NODE)*EXP(GRS*FWT*FADENS)
      SINK(NODE)=SINK(NODE)-WTS(NODE)
58    TSINKS=TSINKS+SINK(NODE)
      DO 59 NODE=1,NODES
59    DISTS(NODE)=SINK(NODE)/TSINKS

C     ************************************************************************
C     *                                                                      *
C     *     3. PETIOLE SINK                                                  *
C     *                                                                      *
C     ************************************************************************

      TSINKP=0.
      IF (NODES.LT.3) GO TO 711
      DO 70 NODE=3,NODES
      FADENS=1.
      FWT=(WTPMAX(NODE)-WTP(NODE))/WTPMAX(NODE)
      SINK(NODE)=WTP(NODE)*EXP(GRP*FWT*FADENS)
      SINK(NODE)=SINK(NODE)-WTP(NODE)
70    TSINKP=TSINKP+SINK(NODE)
      DO 71 NODE=3,NODES
71    DISTP(NODE)=SINK(NODE)/TSINKP

C     ************************************************************************
C     *                                                                      *
C     *     4. FRUIT SINK                                                    *
C     *                                                                      *
C     *          4A. FLOWER SINKS                                            *
C     *                                                                      *
C     ************************************************************************
```

```
711   IF (COND.LT.3) GO TO 700
      IF (NOFLO.GT.0) GO TO 102

C     ************************************************************************
C     *                                                                      *
C     *     SET UP INITIAL DISTRIBUTION OF FLOWER WEIGHTS                    *
C     *                                                                      *
C     ************************************************************************

      NOFLO=NOFRU=INITNO
      DO 104 I=NOFLO,NODES
104   WTF(I)=WTINIT(I)
      GO TO 105
102   NFLO1=NOFLO
      DO 103 NODE=NFLO1,NODES
      WTFLMAX=FLOWRS(NODE)*WTFLOWR
      IF (WTFLMAX.GT.0.) GO TO 1021
      WTF(NODE)=0.
      GO TO 103
1021  CONTINUE
      IF (WTF(NODE).LT.WTFLMAX) GO TO 105
      PODS(NODE)=AINT(FLOWRS(NODE)*FLOSURV(NODE)*POLLEN(IWEEK)+.5)
      WTF(NODE)=WTF(NODE)*FLOSURV(NODE)*POLLEN(IWEEK)
      NOFLO=NOFLO+1
103   CONTINUE
105   TSINKF=0.
      DO 106 NODE=NOFLO,NODES
      APHFLO=1.
      SINK(NODE)=WTF(NODE)*EXP(GRFL*APHFLO)
      SINK(NODE)=SINK(NODE)-WTF(NODE)
106   TSINKF=TSINKF+SINK(NODE)

C     ************************************************************************
C     *                                                                      *
C     *          4B. POD SINKS                                               *
C     *                                                                      *
C     ************************************************************************

      IF (NOFRU.EQ.NOFLO) GO TO 101
      NF=NOFLO-1
      DO 107 NODE=NOFRU,NF
      IF (WTF(NODE).LT.THRMORT(NODE)) GO TO 1065
      PODS(NODE)=AINT(PODS(NODE)*FRUSURV(NODE)+.5)
      WTF(NODE)=WTF(NODE)*FRUSURV(NODE)
      THRMORT(NODE)=5000.
1065  PODMAX=PODS(NODE)*PODWT(NODE)
      IF (PODS(NODE).GT.0.) GO TO 1064
      SINK(NODE)=0.
      GO TO 107
1064  CONTINUE
      FWT=(PODMAX-WTF(NODE))/PODMAX
      APHFRU=1.
      SINK(NODE)=WTF(NODE)*EXP(GRFR*APHFRU*FWT)
      SINK(NODE)=SINK(NODE)-WTF(NODE)
107   TSINKF=TSINKF+SINK(NODE)
101   CONTINUE
```

```
      DO 109 NODE=NOFRU,NODES
109   DISTF(NODE)=SINK(NODE)/TSINKF
108   CONTINUE

C     ************************************************************************
C     *                                                                      *
C     *     DISTRIBUTE STEM INCREMENT BETWEEN THE SHOOT ORGANS               *
C     *                                                                      *
C     ************************************************************************

      IF (COND.GE.4) GO TO 600
      GO TO 701
700   TSINKF=0.
701   SIGSINK=TSINKL+TSINKS+TSINKP+TSINKF+TCSINK

C     ************************************************************************
C     *                                                                      *
C     *     THE TERMINAL MERISTEM IS INCREMENTED FIRST                       *
C     *                                                                      *
C     ************************************************************************

      TCINC=(TCSINK/SIGSINK)*SHOOTIC
      TCWT=TCWT+TCINC
      IF (TCWT.LE.WTTHR) GO TO 51
      NODES=NODES+1
      WTNEW=TCWT-BASEWT
      TCWT=BASEWT
      WTTHR=WTTHR2

C     ************************************************************************
C     *                                                                      *
C     *     SET UP THE BIOMASS DISTRIBUTION IN THE NEWLY FORMED NODE         *
C     *                                                                      *
C     ************************************************************************

      NODOLD=NODES
      IF (NODOLD.LT.3) GO TO 54
      WTL(NODOLD)=WTNEW*FRACTL(COND)
      WTS(NODOLD)=WTNEW*FRACTS(COND)
      WTF(NODOLD)=WTNEW*FRACTF(COND)
      WTP(NODOLD)=WTNEW-WTL(NODOLD)-WTS(NODOLD)-WTF(NODOLD)
      LFAGE(NODOLD)=1
```

```
      AREAL(NODOLD)=WTL(NODOLD)/DENSLF(1)
      TURGOR(NODOLD)=Y21(1)
      GO TO 51
54    WTS(NODOLD)=WTNEW
51    CONTINUE
      LEAFINC=(TSINKL/SIGSINK)*SHOOTIC
      STEMINC=(TSINKS/SIGSINK)*SHOOTIC
      FRUTINC=(TSINKF/SIGSINK)*SHOOTIC
      PETOINC=(TSINKP/SIGSINK)*SHOOTIC
      GO TO 601
600   FRUTINC=SHOOTIC
      IF (FRUTINC.GT.TSINKF) FRUTINC=TSINKF
      LEAFINC=STEMINC=PETOINC=0.
601   TOTLA=TOTL=TOTS=TOTP=TOTF=0.
      DO 200 NODE=1,NODES

C     ************************************************************************
C     *                                                                      *
C     *    DISTRIBUTE LEAF INC. VERTICALLY THROUGH THE CANOPY IN             *
C     *    RELATION TO THE RELATIVE SINK STRENGTH OF EACH NODE.              *
C     *                                                                      *
C     ************************************************************************

      IF (LEAFINC.LE.0.) GO TO 710
      WTL(NODE)=WTL(NODE)+DISTL(NODE)*LEAFINC
      LAGE=LFAGE(NODE)
      AREAL(NODE)=WTL(NODE)/DENSLF(LAGE)

C     ************************************************************************
C     *                                                                      *
C     *    DISTRIBUTE THE STEM INCREMENT                                     *
C     *                                                                      *
C     ************************************************************************

710   IF (STEMINC.LE.0.) GO TO 720
      WTS(NODE)=WTS(NODE)+DISTS(NODE)*STEMINC

C     ************************************************************************
C     *                                                                      *
C     *    DISTRIBUTE THE PETIOLE INCREMENT                                  *
C     *                                                                      *
C     ************************************************************************

720   IF (PETOINC.LE.0.) GO TO 730
      WTP(NODE)=WTP(NODE)+DISTP(NODE)*PETOINC

C     ************************************************************************
C     *                                                                      *
C     *    DISTRIBUTE THE FRUIT INCREMENT                                    *
C     *                                                                      *
C     ************************************************************************

730   IF (FRUTINC.LE.0.) GO TO 740
      WTF(NODE)=WTF(NODE)+DISTF(NODE)*FRUTINC

C     ************************************************************************
```

```
C     *                                                                *
C     *    COMPUTE TOTALS OF EACH BIOMASS TYPE                         *
C     *                                                                *
C     ******************************************************************

740   TOTLA=TOTLA+AREAL(NODE)
      TOTL=TOTL+WTL(NODE)
      TOTS=TOTS+WTS(NODE)
      TOTP=TOTP+WTP(NODE)
      TOTF=TOTF+WTF(NODE)
200   CONTINUE
      GO TO 780
750   TOTLA=TOTL=TOTS=TOTP=TOTF=0.
      DO 770 NODE=1,NODES
      TOTLA=TOTLA+AREAL(NODE)
      TOTL=TOTL+WTL(NODE)
      TOTS=TOTS+WTS(NODE)
      TOTP=TOTP+WTP(NODE)
770   TOTF=TOTF+WTF(NODE)
780   SHOOT=TOTL+TOTS+TOTP+TOTF+TCWT
760   WTPLANT=SHOOT+ROOT

C     ******************************************************************
C     *                                                                *
C     *    DETERMINE WHETHER THE PLANT SHOULD CHANGE PHENOLOGICAL      *
C     *    CONDITION, BY REFERENCE TO VARIOUS THRESHOLDS.              *
C     *                                                                *
C     ******************************************************************

      IF (COND.GT.1) GO TO 5
      IF (NODES.GE.3) COND=2
      GO TO 7
5     IF (COND.GT.2) GO TO 6
      IF (NODES.GE.NNFL.AND.IDAY.GE.IPHOTP) COND=3
      GO TO 7
6     IF (COND.GT.3) GO TO 7
      IF (NDAYFL.GE.N4THR) COND=4
7     CONTINUE
      A1=SHOOT
      A2=ROOT
      A3=TOTL
      A4=TOTS
      A5=TOTF
      A6=TCWT
      A7=WTPLANT
      A8=TOTF
      IF (MI.EQ.1) GO TO 7474
      MI=1
      CALL PRTPLT(BNDS,DIV,IDAY,A1,A2,A3,A4,A5,A6,A7,A8)
      GO TO 7475
7474  MI=0
7475  CONTINUE
1     CONTINUE
      WRITE (6,5002) TOTF
5002  FORMAT (≠ ≠,≠ TOTAL BEAN YIELD =≠,F8.4)
      STOP
```

```
      END
      REAL FUNCTION F(X,XVAL,YVAL,NDIM)
      DIMENSION XVAL(NDIM),YVAL(NDIM)
      IF (X.LE.XVAL(1)) GO TO 1
      IF (X.GE.XVAL(NDIM)) GO TO 2
      DO 3 I=1,NDIM
      IF (XVAL(I).LE.X) GO TO 3
      AM=(YVAL(I)-YVAL(I-1))/(XVAL(I)-XVAL(I-1))
      C=YVAL(I)-AM*XVAL(I)
      F=AM*X+C
      RETURN
3     CONTINUE
1     F=YVAL(1)
      RETURN
2     F=YVAL(NDIM)
      RETURN
      END
      SUBROUTINE SCALE(BOUNDS,DIV,IHEAD,LABELS,LABOUT)
      DIMENSION IHEAD(20),LABELS(6,3),LABOUT(4,2)
      DIMENSION
     +     BOUNDS(12),
     +     DIV(6)
      NOG=6
      WRITE (6,800) (IHEAD(I),I=1,20)
      WRITE (6,801)
      N=0
      DO 9 K=2,12,2
      N=N+1
      WRITE(6,802)BOUNDS(K),(LABELS(N,J),J=1,3),BOUNDS(K-1)
9     CONTINUE
      WRITE (6,808) ((LABOUT(I,J),J=1,2),I=1,4)
      WRITE (6,910)
      DO 1 ISCALE = 1, NOG
      IKX = 2 * ISCALE
1     DIV(ISCALE) = (BOUNDS(IKX-1)-BOUNDS(IKX))/70.
      RETURN
910   FORMAT (≠0≠)
800   FORMAT (≠1≠,T33,20A4)
801   FORMAT (≠0≠,/,T33,≠MINIMUM≠,T59,≠GRAPHING≠,T92,≠MAXIMUM≠)
802   FORMAT (≠ ≠,T33,E11.4,T51,3A4,T64,≠.VV.≠,T71,≠TIME≠,T92,E11.4)
808   FORMAT (≠ ≠,T4,2A4,T17,2A4,T105,2A4,T119,2A4)
      END
      BLOCK DATA
      COMMON /BD01/BLANK,DOT,CROSS,NUMB(6)
      DATA NUMB/1H1,1H2,1H3,1H4,1H5,1H6/
      DATA BLANK/1H /
      DATA DOT/1H./
      DATA CROSS/1H+/
      END
      SUBROUTINE PRTPLT(BND,DIV,I9,W1,W2,W3,W4,W5,W6,W7,W8)
      COMMON/BD01/BLANK,DOT,CROSS,NUMB(6)
      DIMENSION
     +     XV(8),
     +     ALINE(70),
     +     DIV(6),
     +     BND(12)
```

```
      INTEGER BLANK,DOT,CROSS,NUMB
      INTEGER ALINE
      NOG=6
      XV(1) = W1
      XV(2) = W2
      XV(3) = W3
      XV(4) = W4
      XV(5) = W5
      XV(6) = W6
      XV(7) = W7
      XV(8) = W8
      DO 1 I=1,70
1     ALINE(I)=BLANK
      ALINE(1 ) = DOT
      ALINE(70) = DOT
      DO 2 I = 1, NOG
      J=2*I
      NDIV = (XV(I)-BND(J))/DIV(I)+0.5
      IF (NDIV.LE.1 ) NDIV = 1
      IF (NDIV.GE.70) NDIV = 70
2     ALINE(NDIV)=NUMB(I)
      WRITE(6,3) I9,XV(1),(ALINE(II),II=1,70),XV(7),XV(8)
3     FORMAT (≠ ≠,T4,I4,T17,E11.4,T31,70(A1),T105,E11.4,T119,E11.4)
      RETURN
      END
```

•

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000112

DATE 03/07/73 NODUMP

```
      PROGRAM APHIS (INPUT,OUTPUT,TAPE5=INPUT,TAPE6=OUTPUT)
      COMMON /BD01/BLANK,DOT,CROSS,NUMB(6)
      DIMENSION PRATE(5)
      DIMENSION IHEAD(20),LABELS(6,3),LABOUT(4,2)
      DIMENSION DIV(6),BNDS(12)
      DIMENSION
     +     ALATAE(25),
     +     APTERAE(25),
     +     SIZE(25),
     +     IAGEAD(25),
     +     SIZEMAX(25),
     +     AL(5),
     +     TURGOR(25),
     +     FOODQU(25),
     +     ALA(5)
      DIMENSION
     +     INSTAR(25),
     +     DAYDEG(25),
     +     FECMAX1(17),
     +     FECMAX2(17),
     +     APTSURV(25),
     +     ALASURV(25),
     +     FALEM(25),
     +     APHFEED(25),
     +     DENSITY(25),
     +     DEGTHR(4)
      DIMENSION
     +     X101(4),Y101(4),
     +     X102(4),Y102(4),
     +     X103(4),Y103(4),
     +     X104(4),Y104(4),
     +     X100(4),Y100(4),
     +     X120(4),Y120(4),
     +     X121(4),Y121(4),
     +     X140(4),Y140(4),
     +     X150(4),Y150(4),
     +     X400(4),Y400(4),
     +     X1(4),Y1(4),
     +     X3(4),Y3(4),
     +     X2(4),Y2(4)
      DIMENSION
     +     P(5),
     +     AREAL(25),
     +     WTS(25)
      DIMENSION X99(3),Y99(3)
      DIMENSION X77(4),Y77(4)
      DIMENSION OLD(10)
      DIMENSION X55(3),Y55(3)
      DATA X55/1.,1.5,2./,Y55/1.,.5,0./
      DATA OLD/10*0./
      DATA X77/0.,2000.,3000.,5000./,Y77/-.3,0.,.3,.5/
      DATA X99/2.,3.,5./,Y99/0.,.7,1./
      DATA
     +     X101/0.,2.,5.,10./,Y101/0.,2.,5.,10./,
     +     X102/3.,5.,7.,10./,Y102/0.,.6,.9,1./,
     +     X103/0.,4.,8.,10./,Y103/.6,.65,.95,1./,
```

```
     +      X104/0.,3.,7.,10./,Y104/.8,.9,.95,1./,
     +      X100/5.,10.,15.,20./,Y100/.1,.6,.9,1./,
     +      X120/0.,.25,.6,1./,Y120/6.,7.5,9.,10./,
     +      X121/5.,7.,9.,10./,Y121/.6,.7,.9,1./,
     +      X140/0.,.5,.8,1./,Y140/0.,.3,.5,1./,
     +      X150/.3E4,.6E4,.4E5,.1E7/,Y150/0.,.4,1.,3./,
     +      X400/5.,10.,15.,25./,Y400/.265,.255,.24,.22/,
     +      X1/0.,10.,30.,40./,Y1/3.,5.,16.,20./,
     +      X2/0.,0.,5000.,6000./,
     +      Y2/1.,1.,.95,.95/,
     +      X3/0.,0.,5000.,6000./,
     +      Y3/1.,1.,.95,.95/
      DATA
     +      AL,ALA/10*0./,
     +      ALATAE,APTERAE/50*0./,
     +      DEGTHR/40.,80.,120.,160./,
     +      SIZE,SIZEMAX/50*0./,
     +      FECMAX1/7.,2*9.,3*10.,2*7.,4*4.,2*3.,3*1./,
     +      FECMAX2/5.,2*7.,3*8.,2*5.,4*2.,2*1.,3*0./,
     +      INSTAR/25*0/,
     +      DAYDEG/25*0./,
     +      FALEM/10*0.,.01,.03,.05,.1,.15,.2,.25,.3,.4,.5,.6,.7,.8,.9,
     +1./
      DATA
     +      TURGOR/25*10./,
     +      DENSITY/25*3./,
     +      FOODQU/25*1./,
     +      APTSURV/25*.95/,
     +      ALASURV/25*.9/,
     +      WTS/25*.1/,CSA/1./
      DATA BNDS/8.,0.,8.,0.,8.,0.,8.,0.,8.,0.,8.,0./
      READ (5,201) (IHEAD(J),J=1,20)
      READ(5,202)((LABELS(I,J),J=1,3),I=1,6)
      READ(5,203)((LABOUT(I,J),J=1,2),I=1,4)
  201 FORMAT(20A4)
  202 FORMAT(6(3A4))
  203 FORMAT(4(2A4))
      READ (5,713) (P(M),M=1,5)
  713 FORMAT (5F5.0)
      CALL SCALE(BNDS,DIV,IHEAD,LABELS,LABOUT)
      NODE=6
      TOTAPH=EM=DIF=0.
      ALTHR=.1
      TPC=TP=0.
      IPTHR=10
      FOODQUL=1.
      REF=1000.
      TOP=.8
      FILBTH=.5E7
      PR=0.2

C     ************************************************************************
C     *                                                                      *
C     *     INITIAL IMMIGRATION OF ALATE APHIDS                              *
C     *                                                                      *
C     ************************************************************************
```

```
      ALATAE(10)=2.
      INSTAR(10)=5
      SIZE(10)=9.
      SIZEMAX(10)=10.
      IAGEAD(10)=9
      NDAYS=100
      TFOOD=ADAYS=0.
      ALP=0.
      ADAYS=0.
      TEMP=20.
      DO 1 IDAY=1,NDAYS
      IWEEK=IDAY/7
      PPR=(TOP-.1)*.1E-3*TOTAPH+.1
      TP=PPR*TOTAPH
      TOTAPH1=OLD(10)
      PR=F(TOTAPH1,X77,Y77,4)
      PRTEMP=F(TEMP,X100,Y100,4)
C     ************************************************************************
C     *                                                                      *
C     *    GROWTH OF THE APHIDS OF EACH AGE IS ASSUMED TO BE SIGMOID         *
C     *    IN RELATION TO THEIR WEIGHT AT BIRTH, AND THEIR MAXIMUM           *
C     *    WEIGHT WHICH IS DETERMINED BY THIS. TEMPERATURE AND FOOD          *
C     *    QUALITY AFFECT THEIR GROWTH RATE.                                 *
C     *                                                                      *
C     ************************************************************************
      GRAPH=F(TEMP,X400,Y400,4)
      DO 400 I=1,25
      IF (SIZE(I).LE.0.) GO TO 400
      SIZE(I)=SIZE(I)*EXP((SIZEMAX(I)-SIZE(I))/SIZEMAX(I))*GRAPH*
     +FOODQU(NODE))
400   CONTINUE
      DAY=FLOAT(IDAY)
      AREAL(NODE)=F(DAY,X1,Y1,4)
C     ************************************************************************
C     *                                                                      *
C     *    APHIDS FEED AT A RATE WHICH IS A FUNCTION OF THE TURGOR           *
C     *    OF THE LEAF,THE DENSITY OF APHIDS IN THE AGGREGATE, AND           *
C     *    THE NUTRITIONAL QUALITY OF THE PHLOEM SAP                         *
C     *                                                                      *
C     ************************************************************************
      APHFEED(NODE)=0.
      DO 401 I=1,25
      FACTS=F(TURGOR(NODE),X102,Y102,4)*F(DENSITY(NODE),X103,Y103,4)
      FACTS=FACTS*F(FOODQU(NODE),X104,Y104,4)
      APHFEED(NODE)=APHFEED(NODE)+(APTERAE(I)*F(SIZE(I),X101,Y101,4)
     +*FACTS)
      APHFEED(NODE)=APHFEED(NODE)+(ALATAE(I)*F(SIZE(I),X101,Y101,4)
     +*FACTS)
401   CONTINUE
      TFOOD=TFOOD+APHFEED(NODE)
```

```
C     ************************************************************************
C     *                                                                      *
C     *    DENSITY DEPENDENT FECUNDITY AND SURVIVAL CAN BE INCLUDED          *
C     *    HERE IF THEY ARE REQUIRED                                         *
C     *                                                                      *
C     ************************************************************************

      DDFEC=F(TOTAPH,X2,Y2,4)
      DDSURV=F(TOTAPH,X3,Y3,4)

C     ************************************************************************
C     *                                                                      *
C     *    DETERMINE THE NUMBER OF DAY-DEGREES EXPERIENCED BY EACH           *
C     *    AGE CLASS OF APHIDS TO DATE                                       *
C     *                                                                      *
C     ************************************************************************

      DO 301 I=1,25
301   DAYDEG(I)=DAYDEG(I)+TEMP
      INSTAR(1)=1
      BORN=0.
      TSIZE=0.

C     ************************************************************************
C     *                                                                      *
C     *    COMPUTE THE NUMBER BORN TODAY. THIS IS A FUNCTION OF THE AGE      *
C     *    OF THE ADULT FEMALES AND THE TEMPERATURE                          *
C     *                                                                      *
C     ************************************************************************

      DO 200 I=1,25
      IF (INSTAR(I).NE.5) GO TO 200
      K=I-IAGEAD(I)
      A=BORN
      BORN=BORN+AINT(APTERAE(I)*FECMAX1(K)*PRTEMP*DDFEC)
      B=BORN-A
      TSIZE=TSIZE+B*SIZE(I)
      A=BORN
      BORN=BORN+AINT(ALATAE(I)*FECMAX2(K)*PRTEMP*DDFEC)
      B=BORN-A
      TSIZE=TSIZE+B*SIZE(I)
200   CONTINUE

C     ************************************************************************
C     *                                                                      *
C     *    DETERMINE THE PROPORTION OF SECOND INSTAR APHIDS WHICH ARE        *
C     *    DESTINED TO BECOME ALATE. THIS IS RELATED TO THE ACCUMULATED      *
C     *    POPULATION LIFE AT THIS NODE OF THE PLANT                         *
C     *                                                                      *
C     ************************************************************************

      ADAYS=ADAYS+TOTAPH
      TOTAREA=AREAL(NODE)+(WTS(NODE)*CSA)
      FILTH=F(ADAYS/TOTAREA,X150,Y150,4)
      IF (FILTH.LT.ALTHR) GO TO 500
      PROP2=F(FILTH,X140,Y140,4)
```

```
      BORN=BORN*F(FILTH,X55,Y55,3)
      DO 501 I=1,15
      IF (AL(2).LE.0.) GO TO 501
      IF (INSTAR(I).NE.2) GO TO 501
      DIF=AINT(APTERAE(I)*PROP2)
      APTERAE(I)=APTERAE(I)-DIF
      ALATAE(I)=ALATAE(I)+DIF
501   CONTINUE
500   CONTINUE
      IF (IDAY.LT.8) GO TO 527
      DO 526 I=1,25
      IF (INSTAR(I).NE.5) GO TO 526
      EMIGNO=AINT(ALATAE(I)*FALEM(I))
      EM=EM+EMIGNO
      ALATAE(I)=ALATAE(I)-EMIGNO
526   CONTINUE
527   CONTINUE

C     ************************************************************************
C     *                                                                      *
C     *    COMPUTE DAILY SURVIVORS                                           *
C     *                                                                      *
C     ************************************************************************

      DO 528 I=1,25
      APTERAE(I)=APTERAE(I)*APTSURV(I)*DDSURV
528   ALATAE(I)=ALATAE(I)*ALASURV(I)*DDSURV

C     ************************************************************************
C     *                                                                      *
C     *    UPDATE THE INSTARS OF THOSE AGES OF APHIDS WHOSE TEMPERATURE      *
C     *    THRESHOLDS HAVE BEEN PASSED                                       *
C     *                                                                      *
C     ************************************************************************

      DO 302 I=1,25
      K=INSTAR(I)
      IF (K.EQ.5) GO TO 302
      IF (DAYDEG(I).LT.DEGTHR(K)) GO TO 302
      INSTAR(I)=INSTAR(I)+1
      IF (INSTAR(I).LT.5) GO TO 302
      IAGEAD(I)=I
302   CONTINUE

C     ************************************************************************
C     *                                                                      *
C     *    AGE ALL THE APHIDS, AND THE PARAMETERS ASSOCIATED WITH THEM       *
C     *    BY ONE DAY                                                        *
C     *                                                                      *
C     ************************************************************************

      DO 303 J=1,24
      I=25-J+1
      ALATAE(I)=ALATAE(I-1)
      APTERAE(I)=APTERAE(I-1)
      DAYDEG(I)=DAYDEG(I-1)
```

```
      IAGEAD(I)=IAGEAD(I-1)
      INSTAR(I)=INSTAR(I-1)
      SIZE(I)=SIZE(I-1)
      SIZEMAX(I)=SIZEMAX(I-1)
303   CONTINUE
      APTERAE(1)=BORN
      ALATAE(1)=0.
      DAYDEG(1)=0.
      INSTAR(1)=1
      SIZE(1)=SIZEMAX(1)=0.
      IF (BORN.EQ.0.) GO TO 277
      AVADS=TSIZE/BORN
      SIZE(1)=F(AVADS,X121,Y121,4)
      SIZEMAX(1)=F(SIZE(1),X120,Y120,4)
      SA=0.
      DO 7117 I=1,25
      K=INSTAR(I)
7117  SA=SA+(APTERAE(I)+ALATAE(I))*P(K)
      DO 7118 I=1,25
      K=INSTAR(I)
      A=APTERAE(I)+ALATAE(I)
      IF (A.LE.0.) GO TO 7118
      FA=APTERAE(I)/A
      PKILL=TP*(P(K)*A)/SA
      PAPT=PKILL*FA
      PALA=PKILL-PAPT
      APTERAE(I)=AMAX1(0.,APTERAE(I)-PAPT)
      ALATAE(I)=AMAX1(0.,ALATAE(I)-PALA)
7118  CONTINUE
711   CONTINUE

C     ********************************************************************
C     *                                                                  *
C     *     COMPUTE TOTALS                                               *
C     *                                                                  *
C     ********************************************************************

277   DO 309 I=1,5
309   AL(I)=ALA(I)=0.
      TOTAPH=0.
      DO 310 I=1,25
      K=INSTAR(I)
      ALA(K)=ALA(K)+ALATAE(I)
      AL(K)=AL(K)+APTERAE(I)
310   TOTAPH=TOTAPH+ALATAE(I)+APTERAE(I)
      DO 444 I=1,9
      K=10-I+1
444   OLD(K)=OLD(K-1)
      OLD(1)=TOTAPH
      DENSITY(NODE)=TOTAPH/TOTAREA
      IF (TOTAPH.LE.0.) GO TO 11111
      A1=0.
      A2=0.
      A3=0.
      A4=0.
      A5=0.
```

```
      E=0.
      IF (AL(1).GT.0.) A1=ALOG10(AL(1))
      IF (AL(2).GT.0.) A2=ALOG10(AL(2))
      IF (AL(3).GT.0.) A3=ALOG10(AL(3))
      IF (AL(4).GT.0.) A4=ALOG10(AL(4))
      IF (AL(5).GT.0.) A5=ALOG10(AL(5))
      IF (EM.GT.0.) E=ALOG10(EM)
      CALL PRTPLT(BNDS,DIV,IDAY,A1,A2,A3,A4,A5,E,
     +TOTAPH,PRED)
    1 CONTINUE
11111 CONTINUE
      WRITE (6,966) Y3(3)
966   FORMAT (≠0≠,F6.2)
      WRITE (6,666) ADAYS,TFOOD
666   FORMAT (≠ ≠,2(E12.6,2X))
      XFFF=TFOOD/ADAYS
      WRITE (6,966) XFFF
      WRITE (6,666) TP,TPC
      STOP
      END
      REAL FUNCTION F(X,XVAL,YVAL,NDIM)
      DIMENSION XVAL(NDIM),YVAL(NDIM)
      IF (X.LE.XVAL(1)) GO TO 1
      IF (X.GE.XVAL(NDIM)) GO TO 2
      DO 3 I=1,NDIM
      IF (XVAL(I).LE.X) GO TO 3
      AM=(YVAL(I)-YVAL(I-1))/(XVAL(I)-XVAL(I-1))
      C=YVAL(I)-AM*XVAL(I)
      F=AM*X+C
      RETURN
3     CONTINUE
1     F=YVAL(1)
      RETURN
2     F=YVAL(NDIM)
      RETURN
      END
      SUBROUTINE SCALE(BOUNDS,DIV,IHEAD,LABELS,LABOUT)
      DIMENSION IHEAD(20),LABELS(6,3),LABOUT(4,2)
      DIMENSION
     +     BOUNDS(12),
     +     DIV(6)
      NOG=6
      WRITE (6,800) (IHEAD(I),I=1,20)
      WRITE (6,801)
      N=0
      DO 9 K=2,12,2
      N=N+1
      WRITE(6,802)BOUNDS(K),(LABELS(N,J),J=1,3),BOUNDS(K-1)
9     CONTINUE
      WRITE (6,808) ((LABOUT(I,J),J=1,2),I=1,4)
      WRITE (6,910)
      DO 1 ISCALE = 1, NOG
      IKX = 2 * ISCALE
1     DIV(ISCALE) = (BOUNDS(IKX-1)-BOUNDS(IKX))/70.
      RETURN
910   FORMAT (≠0≠)
```

```
  800   FORMAT (≠1≠,T33,20A4)
  801   FORMAT (≠0≠,/,T33,≠MINIMUM≠,T59,≠GRAPHING≠,T92,≠MAXIMUM≠)
  802   FORMAT (≠ ≠,T33,E11.4,T51,3A4,T64,≠.VV.≠,T71,≠TIME≠,T92,E11.4)
  808   FORMAT (≠ ≠,T4,2A4,T17,2A4,T105,2A4,T119,2A4)
        END
        BLOCK DATA
        COMMON/BD01/BLANK,DOT,CROSS,NUMB(6)
        DATA NUMB/1H1,1H2,1H3,1H4,1H5,1H6/
        DATA BLANK/1H /
        DATA DOT/1H./
        DATA CROSS/1H+/
        END
        SUBROUTINE PRTPLT(BND,DIV,I9,W1,W2,W3,W4,W5,W6,W7,W8)
        COMMON/BD01/BLANK,DOT,CROSS,NUMB(6)
        DIMENSION
       +     XV(8),
       +     ALINE(70),
       +     DIV(6),
       +     BND(12)
        INTEGER BLANK,DOT,CROSS,NUMB
        INTEGER ALINE
        NOG=6
        XV(1) = W1
        XV(2) = W2
        XV(3) = W3
        XV(4) = W4
        XV(5) = W5
        XV(6) = W6
        XV(7) = W7
        XV(8) = W8
        DO 1 I=1,70
  1     ALINE(I)=BLANK
        ALINE(1 ) = DOT
        ALINE(70) = DOT
        DO 2 I = 1, NOG
        J=2*I
        NDIV = (XV(I)-BND(J))/DIV(I)+0.5
        IF (NDIV.LE.1 ) NDIV = 1
        IF (NDIV.GE.70) NDIV = 70
  2     ALINE(NDIV)=NUMB(I)
        WRITE(6,3) I9,XV(1),(ALINE(II),II=1,70),XV(7),XV(8)
  3     FORMAT (≠ ≠,T4,I4,T17,E11.4,T31,70(A1),T105,E11.4,T119,E11.4)
        RETURN
        END


UT
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 000112



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```
      PROGRAM PRED (INPUT,OUTPUT,TAPE5=INPUT,TAPE6=OUTPUT)
      COMMON /BD01/BLANK,DOT,CROSS,NUMB(6)
      COMMON/BD02/X1(4),Y1(4),FEC(50),SUV(50)
      COMMON/BD03/BETA(5)
      COMMON/BD04/IPRINT,REFUGE
      DIMENSION DIV(6),BNDS(12)
      DIMENSION IHEAD(20),LABELS(6,3),LABOUT(4,2)
      DIMENSION ALPHA(5),
     +     X111(2),Y111(2),
     +     EGG(5),
     +     ADS(20),
     +     AKILL(5),
     +     PREY1(5),
     +     SEXR(20),
     +     P(5,50),
     +     IA(5,50),
     +     DD(5,50),
     +     S(5,50),
     +     IN(5,50),
     +     SV(5,50)
      DATA P/250*0./,S/250*0./,DD/250*0./
      DATA IN/250*1/,SV/250*1./,IA/250*20/
      DATA ADS/20*0./
      DATA X111/0.,100000./
      DATA Y111/-1.,1./
      DATA SEXR/20*.5/
      DATA BNDS/6.,3.,6.,3.,6.,3.,6.,3.,6.,3.,1000.,0./
      READ (5,99) REFUGE
      READ (5,99) (PREY1(I),I=1,5)
      WRITE (6,97) (PREY1(I),I=1,5)
      READ (5,98) IMP
97    FORMAT (≠0≠,*   INITIAL PREY DISTRIBUTIONS  *,5(F10.1,3X))
98    FORMAT (10I3)
99    FORMAT (10F6.0)
      READ (5,201) (IHEAD(J),J=1,20)
      READ(5,202)((LABELS(I,J),J=1,3),I=1,6)
      READ(5,203)((LABOUT(I,J),J=1,2),I=1,4)
 201  FORMAT(20A4)
 202  FORMAT(6(3A4))
 203  FORMAT(4(2A4))
      CALL SCALE(BNDS,DIV,IHEAD,LABELS,LABOUT)
      AD=0.
      ADFOOD=20.
      STT=0.
      READ (5,99) ADIM
      DO 1 IDAY=1,100
      IF (IDAY.GT.IMP) GO TO 111
      DO 11 I=1,20
11    ADS(I)=ADS(I)+ADIM
111   CONTINUE
      TP=0.
      DO 2 I=1,5
2     TP=TP+AMAX1(0.,PREY1(I)-REFUGE)
      DO 5 I=1,5
5     ALPHA(I)=AMAX1(0.,PREY1(I)-REFU7E)/TP
      AM=TP*.2
```

```
      ULOG=F(AM,X111,Y111,2)
      U=10.**ULOG
      SIGA=0.
      DO 3 I=1,5
3     SIGA=SIGA+ALPHA(I)**U
      DO 4 I=1,5
4     BETA(I)=(ALPHA(I)**U)/SIGA
      TAD=TEGGS=0.
      DO 6 J=1,20
      ADS(J)=ADS(J)*SUV(J)
      TAD=TAD+ADS(J)
6     TEGGS=TEGGS+ADS(J)*FEC(J)*SEXR(J)*F(STT, X1,Y1,4)
      TFOOD=0.
      DO 7 J=1,20
7     TFOOD=TFOOD+ADS(J)*ADFOOD
      DO 8 I=1,5
      EGG(I)=TEGGS*BETA(I)
8     AKILL(I)=TFOOD*BETA(I)
      DO 9 J=1,19
      I=20-J+1
9     ADS(I)=ADS(I-1)
      ADS(1)=AD
      IPRINT=0
      IF (IDAY.EQ.100) IPRINT=1
      CALL POPGRO (EGG,AKILL,PREY1,P,IA,DD,S,SV,IN,STT,AD)
      O1=ALOG10(PREY1(1))
      O2=ALOG10(PREY1(2))
      O3=ALOG10(PREY1(3))
      O4=ALOG10(PREY1(4))
      O5=ALOG10(PREY1(5))
      CALL PRTPLT (BNDS,DIV,IDAY,O1,O2,O3,O4,O5,U,TAD,U)
 1    CONTINUE
      STOP
      END
      SUBROUTINE POPGRO (E,A,PREY1,P,IA,DD,S,SV,IN,STT,AD)
      COMMON/BD02/X1(4),Y1(4),FEC(50),SUV(50)
      COMMON/BD03/BETA(5)
      COMMON/BD04/IPRINT,REFUGE
      DIMENSION E(5),A(5),
     +     PREY1(5),
     +     ST(5),
     +     P(5,50),
     +     IA(5,50),
     +     IN(5,50),
     +     DD(5,50),
     +     S(5,50),
     +     SV(5,50)
      DIMENSION PREDS(50),
     +     IAGEAD(50),
     +     DAYDEG(50),
     +     INSTAR(50),
     +     SIZE(50),
     +     FOOD(50),
     +     DEGTHR(10),
     +     PIMIG(50),
     +     PEMIG(50),
```

```
     +      KILL(50),
     +      NKILL(50),
     +      EATEN(50),
     +      STARVE(50),
     +      PREYSZ(50),
     +      SEXR(50)
      DIMENSION
     +      X2(4),Y2(4),
     +      X3(4),Y3(4),
     +      X4(4),Y4(4),
     +      X5(4),Y5(4),
     +      X6(4),Y6(4),
     +      X7(4),Y7(4),
     +      X8(4),Y8(4),
     +      X9(4),Y9(4),
     +      X10(4),Y10(4)
      DATA IX/0/
      DATA PREYSZ/50*.1/
10    FORMAT (26I3)
11    FORMAT (16F5.0)
      IDISP=4
      IF (IX.EQ.1) GO TO 1106
      READ (5,10) IRUN
      READ (5,10) NINST
      READ (5,10) IPUPL
      READ (5,11) (DEGTHR(I),I=1,NINST)
      READ (5,11) PREP
      READ (5,11) TEMP
      MINAD=DEGTHR(NINST)/TEMP+0.5
      MAXAD=MINAD+19
      READ (5,11) GR,SIZEMAX
      KK=MAXAD-MINAD
      READ (5,11) (FEC(I),I=1,20)
      READ (5,10) IMPER
      READ (5,11) (X1(I),I=1,4)
      READ (5,11) (Y1(I),I=1,4)
      READ (5,11) (X2(I),I=1,4)
      READ (5,11) (Y2(I),I=1,4)
      READ (5,11) (X3(I),I=1,4)
      READ (5,11) (Y3(I),I=1,4)
      READ (5,11) (X4(I),I=1,4)
      READ (5,11) (Y4(I),I=1,4)
      READ (5,11) (X5(I),I=1,4)
      READ (5,11) (Y5(I),I=1,4)
      READ (5,11) (X6(I),I=1,4)
      READ (5,11) (Y6(I),I=1,4)
      READ (5,11) (X7(I),I=1,4)
      READ (5,11) (Y7(I),I=1,4)
      READ (5,11) (X8(I),I=1,4)
      READ (5,11) (Y8(I),I=1,4)
      READ (5,11) (X9(I),I=1,4)
      READ (5,11) (Y9(I),I=1,4)
      READ (5,11) (X10(I),I=1,4)
      READ (5,11) (Y10(I),I=1,4)
1000  DO 1001 I=1,MAXAD
      INSTAR(I)=IAGEAD(I)=1
```

```
      PREDS(I)=DAYDEG(I)=SIZE(I)=STARVE(I)=0.
1001  SEXR(I)=0.5
1106  IX=1
      IF (IPRINT.EQ.0) GO TO 1108
      WRITE (6,202)
202   FORMAT (≠1≠,*INPUT DATA SUMMARY*,//)
      WRITE (6,203) IRUN,NINST,IPUPL
203   FORMAT (≠ ≠,*NUMBER OF DAYS IN RUN*,I3,*. PUPAL STAGE*,I3,2X,I3)
      WRITE (6,204) (DEGTHR(I),I=1,NINST)
204   FORMAT (≠ ≠,*TEMPERATURE THRESHOLDS*,10F7.1)
      WRITE (6,205) TEMP,PREP,GR,SIZEMAX
205   FORMAT (≠ ≠,10F10.4)
      WRITE (6,205) (X1(I),I=1,4)
      WRITE (6,205) (Y1(I),I=1,4)
      WRITE (6,205) (X2(I),I=1,4)
      WRITE (6,205) (Y2(I),I=1,4)
      WRITE (6,205) (X3(I),I=1,4)
      WRITE (6,205) (Y3(I),I=1,4)
      WRITE (6,205) (X4(I),I=1,4)
      WRITE (6,205) (Y4(I),I=1,4)
      WRITE (6,205) (X5(I),I=1,4)
      WRITE (6,205) (Y5(I),I=1,4)
      WRITE (6,205) (X6(I),I=1,4)
      WRITE (6,205) (Y6(I),I=1,4)
      WRITE (6,205) (X7(I),I=1,4)
      WRITE (6,205) (Y7(I),I=1,4)
      WRITE (6,205) (X8(I),I=1,4)
      WRITE (6,205) (Y8(I),I=1,4)
      WRITE (6,205) (X9(I),I=1,4)
      WRITE (6,205) (Y9(I),I=1,4)
      WRITE (6,205) (X10(I),I=1,4)
      WRITE (6,205) (Y10(I),I=1,4)
 1108 CONTINUE
      AD=0.
      DO 101 K=1,5
      DO 201 I=1,50
      PREDS(I)=P(K,I)
      IAGEAD(I)=IA(K,I)
      DAYDEG(I)=DD(K,I)
      SIZE(I)=S(K,I)
      INSTAR(I)=IN(K,I)
201   STARVE(I)=SV(K,I)
      PREY1(K)=PREY1(K)*PREP
      PREY=AMAX1(0.,PREY1(K)-REFUGE)
      IP0=1
      IF (PREY.LE.0.) IP0=0
C     *     1.  BIRTHS                                              *
      BORN=0.
      DO 1 I=MINAD,MAXAD
      IF (PREDS(I).LE.0.) GO TO 1
      IF (INSTAR(I).NE.NINST+1) GO TO 1
      J=I-IAGEAD(I)+1
      BORN=BORN+AINT(PREDS(I)*FEC(J)*SEXR(I)*F(STARVE(I),X1,Y1,4))
1     CONTINUE
      SEXBOR=0.5
C     *     2.  DEATHS                                              *
```

```
      DO 2 I=1,MAXAD
      IF (INSTAR(I).EQ.7.AND.I-IAGEAD(I).GE.IDISP) GO TO 7177
      GO TO 7178
7177  AD=AD+PREDS(I)
      PREDS(I)=0.
7178  IDISP=4
2     PREDS(I)=AINT(PREDS(I)*SUV(I)*F(STARVE(I),X2,Y2,4))
C     *     4.  DEVELOPMENT                                          *
      DO 4 I=1,MAXAD
      DAYDEG(I)=DAYDEG(I)+TEMP
      KIN=INSTAR(I)
      IF (KIN.GE.NINST+1) GO TO 4
      IF (DAYDEG(I).LT.DEGTHR(KIN)) GO TO 4
      INSTAR(I)=INSTAR(I)+1
      IF (INSTAR(I).EQ.NINST+1) IAGEAD(I)=I
4     CONTINUE
C     *     5.  GROWTH                                               *
      DO 5 I=1,MAXAD
      EE=F(STARVE(I),X4,Y4,4)*F(TEMP,X5,Y5,4)
      AA=(SIZEMAX-SIZE(I))/SIZEMAX
      IF (INSTAR(I).EQ.1.OR.INSTAR(I).EQ.IPUPL) EE=0.
5     SIZE(I)=SIZE(I)*EXP(GR*AA*EE)
C     *     6.  FEEDING                                              *
      DO 6 I=1,MAXAD
      STARVE(I)=0.
      IF (INSTAR(I).EQ.1.OR.INSTAR(I).EQ.IPUPL) GO TO 61
      FOOD(I)=F(SIZE(I),X6,Y6,4)
      NKILL(I)=FOOD(I)/PREYSZ(I)
      GO TO 6
61    FOOD(I)=0.
      NKILL(I)=0
6     CONTINUE
      AD1=A(K)/20.
      IF (AD1.LE.0.) GO TO 616
      IF (PREY.LE.0.) GO TO 616
      PK=F(PREY,X7,Y7,4)
      FK=F(PREY/AD1,X8,Y8,4)
      CK=F(AD1,X9,Y9,4)
      ACTK=AMIN1(PK,FK,CK)
      ADKIL=ACTK*A(K)
      PP1=PREY*(1.-EXP(-ADKIL/PREY))
      PREY=PREY-PP1
      ST(K)=AMAX1(0.,1.-PP1/A(K))
      GO TO 617
616   ST(K)=0.
617   CONTINUE
C     *     7.  FUNCTIONAL RESPONSES                                 *
      DO 7 J=1,MAXAD
      I=MAXAD-J+1
      IF (PREY.LE.0.) GO TO 71
      IF (NKILL(I).EQ.0) GO TO 71
      IF (PREDS(I).LE.0.) GO TO 71
      PK=F(PREY,X7,Y7,4)
      FK=F(PREY/PREDS(I),X8,Y8,4)
      CK=F(PREDS(I),X9,Y9,4)
      ACTK=AMIN1(PK,FK,CK)
```

```
      KILL(I)=AINT(FLOAT(NKILL(I))*ACTK)*PREDS(I)
      P2=FLOAT(KILL(I))
      P1=PREY*(1.-EXP(-P2/PREY))
      KILL(I)=P1+0.5
      STARVE(I)=AMAX1(1.-P1/(FLOAT(NKILL(I))*PREDS(I)),0.)
      EATEN(I)=FLOAT(KILL(I))*PREYSZ(I)
      GO TO 7
71    KILL(I)=0
      EATEN(I)=0.
      STARVE(I)=1.
      IF (INSTAR(I).EQ.1.OR.INSTAR(I).EQ.IPUPL) STARVE(I)=0.
7     PREY=PREY-FLOAT(KILL(I))
      TKILL=0.
      NTKILL=0
      DO 72 I=1,MAXAD
      TKILL=TKILL+EATEN(I)
72    NTKILL=NTKILL+KILL(I)
      IF (IP0.EQ.0) GO TO 7964
      PREY1(K)=PREY+REFUGE
7964  CONTINUE
C     *     8.  UPDATE                                              *
      M=MAXAD-1
      DO 8 J=1,M
      I=MAXAD-J+1
      PREDS(I)=PREDS(I-1)
      DAYDEG(I)=DAYDEG(I-1)
      SIZE(I)=SIZE(I-1)
      IAGEAD(I)=IAGEAD(I-1)
      STARVE(I)=STARVE(I-1)
      INSTAR(I)=INSTAR(I-1)
8     SEXR(I)=SEXR(I-1)
      PREDS(1)=BORN+E(K)
      DAYDEG(1)=0.
      IAGEAD(1)=INSTAR(1)=1
      SEXR(1)=SEXBOR
      SIZE(1)=.5
C     *     9.  TOTALS                                              *
      TEG=T1=T2=T3=T4=TPUP=TAD=0.
      DO 9 I=1,MAXAD
      IF (INSTAR(I).EQ.1) TEG=TEG+PREDS(I)
      IF (INSTAR(I).EQ.2) T1=T1+PREDS(I)
      IF (INSTAR(I).EQ.3) T2=T2+PREDS(I)
      IF (INSTAR(I).EQ.4) T3=T3+PREDS(I)
      IF (INSTAR(I).EQ.5) T4=T4+PREDS(I)
      IF (INSTAR(I).EQ.6) TPUP=TPUP+PREDS(I)
      IF (INSTAR(I).EQ.7) TAD=TAD+PREDS(I)
9     CONTINUE
      DO 301 I=1,50
      P(K,I)=PREDS(I)
      IA(K,I)=IAGEAD(I)
      DD(K,I)=DAYDEG(I)
      S(K,I)=SIZE(I)
      IN(K,I)=INSTAR(I)
301   SV(K,I)=STARVE(I)
101   CONTINUE
      STT=0.
```

```
      DO 103 I=1,5
103   STT=STT+ST(I)*BETA(I)
      RETURN
      END
      REAL FUNCTION F (X,XVAL,YVAL,NDIM)
      DIMENSION XVAL(NDIM),YVAL(NDIM)
            IF (X.LE.XVAL(1)) GO TO 1
            IF (X.GE.XVAL(NDIM)) GO TO 2
            DO 3 I = 1, NDIM
                  IF (XVAL(I).LE.X) GO TO 3
                        AM=(YVAL(I)-YVAL(I-1))/(XVAL(I)-XVAL(I-1))
                        C=YVAL(I)-AM*XVAL(I)
                        F=AM*X+C
                  RETURN
3           CONTINUE
1           F=YVAL(1)
            RETURN
2           F=YVAL(NDIM)
            RETURN
      END
      SUBROUTINE SCALE(BOUNDS,DIV,IHEAD,LABELS,LABOUT)
      DIMENSION IHEAD(20),LABELS(6,3),LABOUT(4,2)
      DIMENSION
     +      BOUNDS(12),
     +      DIV(6)
      NOG=6
      WRITE (6,800) (IHEAD(I),I=1,20)
      WRITE (6,801)
      N=0
      DO 9 K=2,12,2
      N=N+1
      WRITE(6,802)BOUNDS(K),(LABELS(N,J),J=1,3),BOUNDS(K-1)
9     CONTINUE
      WRITE (6,808) ((LABOUT(I,J),J=1,2),I=1,4)
      WRITE (6,910)
      DO 1 ISCALE = 1, NOG
      IKX = 2 * ISCALE
1     DIV(ISCALE) = (BOUNDS(IKX-1)-BOUNDS(IKX))/70.
      RETURN
910   FORMAT (≠0≠)
800   FORMAT (≠1≠,T33,20A4)
801   FORMAT (≠0≠,/,T33,≠MINIMUM≠,T59,≠GRAPHING≠,T92,≠MAXIMUM≠)
802   FORMAT (≠ ≠,T33,E11.4,T51,3A4,T64,≠.VV.≠,T71,≠TIME≠,T92,E11.4)
808   FORMAT (≠ ≠,T4,2A4,T17,2A4,T105,2A4,T119,2A4)
      END
      BLOCK DATA
      COMMON/BD01/BLANK,DOT,CROSS,NUMB(6)
      COMMON/BD02/X1(4),Y1(4),FEC(50),SUV(50)
      DATA NUMB/1H1,1H2,1H3,1H4,1H5,1H6/
      DATA BLANK/1H /
      DATA DOT/1H./
      DATA CROSS/1H+/
      DATA X1/0.,1.,2.,3./,Y1/1.,1.,1.,1./
      DATA SUV/50*.95/
      DATA FEC/50*0./
      END
```

```
      SUBROUTINE PRTPLT(BND,DIV,I9,W1,W2,W3,W4,W5,W6,W7,W8)
      COMMON/BD01/BLANK,DOT,CROSS,NUMB(6)
      DIMENSION
     +     XV(8),
     +     ALINE(70),
     +     DIV(6),
     +     BND(12)
      INTEGER BLANK,DOT,CROSS,NUMB
      INTEGER ALINE
      XV(1) = W1
      XV(2) = W2
      XV(3) = W3
      XV(4) = W4
      XV(5) = W5
      XV(6) = W6
      XV(7) = W7
      XV(8) = W8
      DO 1 I=1,70
    1 ALINE(I)=BLANK
      ALINE(1 ) = DOT
      ALINE(70) = DOT
      DO 2 I=1,6
      J=2*I
      NDIV = (XV(I)-BND(J))/DIV(I)+0.5
      IF (NDIV.LE.1 ) NDIV = 1
      IF (NDIV.GE.70) NDIV = 70
    2 ALINE(NDIV)=NUMB(I)
      WRITE(6,3) I9,XV(1),(ALINE(II),II=1,70),XV(7),XV(8)
    3 FORMAT (≠ ≠,T4,I4,T17,E11.4,T31,70(A1),T105,E11.4,T119,E11.4)
      RETURN
      END


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