INTERNATIONAL ATOMIC ENERGY AGENCY
UNITED NATIONS EDUCATIONAL, SCIENTIFIC AND CULTURAL ORGANIZATION
INTERNATIONAL CENTRE FOR THEORETICAL PHYSICS
I.C.T.P., P.O. BOX 586, 34100 TRIESTE, ITALY, CABLE: CENTRATOM TRIESTE

SMR.478 - 3

THIRD AUTUMN COURSE ON MATHEMATICAL ECOLOGY

(29 October - 16 November 1990)

"Mathematical Modelling in Plant Biology: Implications of Physiological Approaches for Resource Management"

Louis J. GROSS
University of Tennessee
Department of Mathematics and
Graduate Program in Ecology
Knoxville, TN 37996-1300
U.S.A.

These are preliminary lecture notes, intended only for distribution to participants.

MATHEMATICAL MODELING IN PLANT BIOLOGY: IMPLICATIONS OF PHYSIOLOGICAL APPROACHES FOR RESOURCE MANAGEMENT

Louis J. Gross
Department of Mathematics and
Graduate Programs in Ecology and
Plant Physiology and Genetics
University of Tennessee
Knoxville, TN 37996-1300

Abstract

I provide a brief overview of mathematical models that have been developed for particular plant physiological processes, with emphasis on the difficulties involved in taking these upscale to deal with whole plant, crop and forest growth analyses. As the issues addressed by physiologists are often highly reductionist in nature, I point out the gap which has developed between our detailed knowledge of certain physiological processes and our general ignorance of appropriate ways to integrate these processes over whole plant or canopy scales. The importance of accurate integration for crop and forest management is discussed. Finally, I review models for the spread of plant pathogens, and indicate how these may be modified to take account of the spatial nature of plant infection and the continuum of resistance types within a natural population.

1. Introduction

Mathematical models have been applied to a wide variety of topics in plant physiology (Thornley, 1976). The majority of these focus on processes that are modeled independently such as photosynthesis, fluid transport, respiration, transpiration and stomatal response. The general goal of these models is to predict the effect of a variety of environmental factors, including radiation input, humidity, wind, CO_2 concentration and temperature, on the process rates. The models tend to be more general and realistic than precise, though all are empirical at some level. Modelers have an advantage in the wealth of physiological data which has been collected on these processes, but at the same time this is often too much of a good thing due to a general

lack of accepted theory in the subject. Thus it is typically not clear what in the plethora of detailed biochemical knowledge about specific processes should be included and what can be safely ignored or lumped in with other components of the process under consideration.

The uncertainty of appropriate components to include in these models becomes even more critical when dealing with questions on longer time or larger spatial scales than those over which typical physiological approaches operate. Much of the detailed physiological models deal with processes on a cellular or organ (such as leaf) spatial scale and the natural time scales associated with these, normally from seconds to daily. I have argued elsewhere (Gross, 1986) that in dealing with plant processes, a natural breakdown of time scales would be physiological (within a day or so), acclimation (within genet lifespan), and evolutionary (over many generations). The appropriate time scale for many management applications is the acclimation one, and there has been much less work at this scale than at the physiological one. My purpose here is in part to indicate the variety of modeling approaches which have been undertaken at the physiological scale, and try to indicate a number of open areas regarding how to appropriately go to acclimation scales from these. A similar problem regarding scaling is discussed in the last section, namely how to make more realistic models for the spread of plant diseases which take into account the spatial interactions which are necessary for the disease to spread.

2. Physiological Models

The aim of most physiological models is to express the equilibrium rate of certain physiological processes as a function of environmental inputs and the current physiological state of the plant. A general form for this would be

$$\begin{aligned} R_1 &= f_1(E_1,\ldots,E_k,P_1,\ldots,P_m,R_2,\ldots,R_n) \\ &\vdots \\ R_n &= f_n(E_1,\ldots,E_k,P_1,\ldots,P_m,R_1,\ldots,R_{n-1}) \end{aligned} \qquad (1)$$

where $R_1,\ldots,R_n$ represent equilibrium rates of various physiological processes, $E_1,\ldots,E_k$ represent environmental factors, and $P_1,\ldots,P_m$ represent various physiological states. The physiological states could be viewed as independent of the R_i's but more realistically are controlled by the R_i's on a longer time scale than this equilibrium

approach is meant to handle. For example, photosynthetic rate is affected by the chlorophyll concentration within a leaf, and this may change over time scales of days due to changes in a variety of reaction rates over the recent past. Thus the R_i's operate on the above mentioned physiological time scale, while the P_i's change on an acclimation time scale.

The form of (1) really is derived as the equilibrium solution of a system of differential equations which would track the concentrations of biochemical components of the associated reactions. It is somewhat rare to see a model actually applied in this form however, due to a general lack of knowledge about the dynamics of the component reactions. In fact, the vast majority of models of the form (1) are really derived as empirical fits to experimental data. In reality of course, both the physiological state and the environmental inputs are time-varying. A typical assumption is that these change on a slow time scale relative to that of the reaction rates, so that the equilibrium solution (1) just continuously tracks these changes. For a variety of processes, including stomatal conductance and photosynthetic rates, this assumption is not justified (Gross, 1986; Kirschbaum et al., 1988).

In addition to the dynamic assumptions inherent in (1), the approach is also inherently a local one, for it is assumed that the reactions guiding the process have no spatial component. That is, all the variables of the model should be viewed as spatial averages over whatever scale the process is assumed to be operating. Thus photosynthesis models at the leaf level based on biochemical reactions typically assume uniform concentrations of the various biochemical components of the process over the entire leaf. The nature of the averaging which is implied by this is non-trivial due to the nonlinearities in the models governing the process as a function of the reactants. Another assumption of these models concerns the simplifications of the biochemistry which are used in order to reduce the number of model parameters to tractable levels and to allow them to be estimated from available data. Many of the sub-reactions which determine the R_i's are extremely complicated, and though some aspects of them may be known in detail, typically one rapidly reaches the current level of ignorance when constructing a model. Thus it becomes necessary to lump component reactions.

Before leaving the general topic of physiological modeling, it should be emphasized that biophysical approaches are the basis for investigating the effects of most environmental factors in these models (Nobel, 1974). This is true of water relations, in which the

volume changes of cells is related to external osmotic pressure through the Boyle-Van't Hoff relation and flux of water between various plant parts is governed by a transport equation based on the chemical potential of water in the parts. Similarly, mass and heat transfer between the plant and surrounding environment is derived from micrometeorological approaches (Montieth, 1975).

3. Whole Plant Processes

How are physiological processes scaled up to the whole plant? Though there are a variety of approaches, none are entirely satisfactory. One method is to use a macrodescriptor which is essentially empirical. For example, a frequently used estimate of whole plant respiration over a day is

$$R = kP + cW$$

where R is respiration, P is gross photosynthesis during the light period, W is plant dry weight, and c and k are constants. This was originally obtained from data on clover (McCree, 1970), but has since been applied in many simple growth models. Similar whole plant descriptors have been used for photosynthesis as a function of incident radiation and temperature, for transpiration as a function of these along with wind speed and humidity, and for a variety of other plant processes. These will sometimes have a mechanistic basis, but more typically use a regression approach to determine the interactions of the variety of factors affecting any of the processes of concern.

There are hosts of plant growth curves derived along these empirical lines, viewed as describing the time course of whole plant or community growth (Hunt, 1982). The difficulty is that, without the data to decide which curve is appropriate, one must proceed in an ad hoc manner in choosing a curve. If the data are available, then one really doesn't learn much new from the curve anyway, the typical use being interpolation. It also isn't clear how the growth curve should be modified by considering a plant in somewhat different conditions than those for the data from which the curve came. When large amounts of data are available, for example on crop varieties that have been planted in many different conditions for many years, these regression approaches work extremely well in predicting harvests. In fact, they are much better predictors than very complex mechanistic models such as those mentioned below. This is just a special case of the rule that, conditional on the availability of adequate data and ignorance

of the exact mechanism of a process, statistical techniques provide far more accurate process prediction within the range of conditions included in the data base than mechanistic approaches with poorly understood functional forms or parameter values.

Alternative to descriptive approaches, one can build up to the whole plant level using brute force, meaning that one simply integrates the process of concern over the entire plant surface. For example, to estimate whole plant photosynthetic rate from a model for the rate of individual leaves

$$P = F(E_1,\ldots,E_k)$$

where P is photosynthetic rate per unit leaf area and the E_i's are environmental factors, one merely sums

$$P_{tot} = \int F(E_1(x),\ldots,E_k(x))\ A(x)\ dx \qquad (2)$$

where P_{tot} is the photosynthetic rate for the whole plant, $E_i(x)$ is the value of environmental factor i at position x, and A(x) gives the amount of leaf area at position x. Here x will in general be in three space, and it may be very difficult to predict the spatial variation of environmental factors throughout the plant. In fact there are very sophisticated models to describe the spatial patterns of radiation throughout plant canopies, based on the architecture of the canopy, transmittance and reflectance of the leaves, quantity of branches, etc.(Ross, 1981).

Typically (2) is solved in a discrete manner, by breaking a canopy into layers and simply considering the fraction of leaf area in each layer subject to direct beam versus diffuse radiation. Although tests have been done of the radiation penetration portions of these (Baldocchi et al., 1985), the photosynthetic rate predicted from (2) hasn't been adequately tested yet. This is due to the difficulty of measuring whole canopy photosynthesis in field conditions. Note that (2) is really a simplification because the $E_i(x)$'s are time-dependent and the functional form of F will change with position in canopy.

Although approaches similar to (2) can be carried out for many physiological components of plant growth, the vast amount of variation of both environmental factors and physiological state throughout a plant canopy (I'm speaking here of commercially important crop and forest plants) limits the technique. One alternative is to use a highly simplified form for the variation in these factors, and derive

from (2) a general relationship which might indicate how changes in basic parameters affect the process. For example in the photosynthetic case, the Monsi-Saeki theory says that light extinction in a canopy may be approximated by an exponential decay with depth (measured in units of leaf area per unit ground area, the leaf area index L) from the top of the canopy. If F(I) gives photosynthetic rate at irradiance I, then (2) becomes

$$P_{tot} = \int_0^{L_T} F(K \exp(-cL))\ dL \qquad (3)$$

where c is an extinction coefficient, L_T is the total leaf area index of the canopy and K is a species-specific constant which depends on the transmission of a leaf as well as the irradiance at the top of the canopy (see France and Thornley, 1984, chap. 7, for more details). One advantage of this method is that it allows one to derive at least qualitative conclusions as to how changes in basic plant characteristics will affect the process. It provides a more mechanistically-based descriptor of whole plant processes than a purely empirical approach.

4. Growth and Yield Models

Just as there are several methods to move upscale from cellular level phenomena to the whole plant, there are a variety of techniques to model growth at the plant, canopy and community level. Descriptive approaches were already mentioned above, but it should be pointed out that some of these seem to be fairly general. For example there is strong evidence from both crop and forest data that annual biomass production for a crop or stand is given by

$$B = \alpha Q \qquad (4)$$

where Q is the annual interception of photosynthetically active radiation by the crop (McMurtrie, 1985). The constant of proportionality α will of course vary with species, nutrient and water conditions at the site, and probably stand history as well. However, estimates of the constants c and K in (3) are available for a wide variety of forests (Jarvis and Leverenz, 1983) from which it is possible to estimate Q. If a time series of data are available to estimate how α varies with time, it is possible to iterate (4) on a time scale shorter than that over which α changes significantly to

estimate the time course of B, and this has been done for a number of crops (Charles-Edwards, 1982). This is an illustration of a top-down approach to growth modeling, in which the details of the physiology are lumped into a single parameter (here α). One could proceed from here to derive a physiologically based model for how α depends on environmental factors, a method which is utilized in some way in many growth models (Landsberg and McMurtrie, 1985).

Alternative to approaches based on (4), compartment models for whole plant growth are quite common (Thornley, 1976; France and Thornley, 1984). These models break up a plant or crop into compartments such as shoot and root, tracking perhaps several components of each, such as structural and storage dry weight and carbon and nitrogen concentration. They may be coupled to models for photosynthesis, soil nutrient uptake, and other possible inputs. Then differential equations are written to describe changes in each compartment, with respiratory losses typically being taken as proportional to the dry weight in a compartment.

An area of controversy in this approach concerns the nature of partitioning of new substrates (usually just taken to be carbon and nitrogen) among the various compartments. One approach is to simply assume that transport of these substrates follows a Fick's law of diffusion, so for example

$$J_c = \frac{\beta(C_s - C_r)}{r_c} \qquad (5)$$

where J_c is the flux of carbon from shoot to root, C_s and C_r are the carbon concentrations in the shoot and root respectively, β is a scaling factor, and r_c is a resistance to movement of carbon (France and Thornley, 1984). An alternative to this is to prescribe the partitioning of nutrients in a "goal-seeking" manner such that either a fixed carbon-to-nitrogen ratio is set and the dynamic behavior of the model is forced to seek this ratio (Reynolds and Thornley, 1982) or else this ratio is set in a way that depends on the root-to-shoot ratio (Johnson, 1985). Still another approach to partitioning is to assume that there are organizing principles of evolutionary origin which specify the partitioning of nutrients so as to maximize some measure of fitness. This is an outgrowth of life history theory, utilizes optimal control techniques, and has been applied mainly to plants broken into roots, shoots and reproductive compartments (see Roughgarden, 1986, for a review).

Yet another approach to growth modeling is a systems one, in which a large collection of physiologically detailed process models are coupled. These typically have submodels for light interception and photosynthesis, root activity and nutrient uptake, partitioning of substrates, transpiration, growth and respiration, leaf area expansion, initiation and development of plant organs, and senescence, though not all these may be included in each model. The models then iterate, typically on a daily or hourly time step, keeping track of levels of nutrients and dry weights of various structural compartments. This amounts to solving non-autonomous difference equations and thus is essentially limited to being simulated on a computer. These models have been constructed for a wide variety of crops (Barrett and Peart, 1981; Loomis et al.,1979; Reynolds and Acock, 1985), involve large numbers of parameters that are sometimes difficult to estimate, and are mainly used as research tools to point out which subprocesses are not well understood. As with many large systems models, they are extremely difficult to validate, due to the ability to tune the large number of parameters to the available data. It is only in rare circumstances that data sets independent from those used to estimate the parameters are available for model validation. The models are rarely spatial, merely assuming the variables are uniform over the scale of the plot under consideration. In this sense, the models are limited to monocultures of fairly even age for which spatial heterogeneity in stand structure is not a significant factor for stand growth.

One method to take account of spatial factors is to use individual-based models. These track all individuals in a stand, using some type of growth model for each species in the stand, and take into account the competitive interactions between neighbors through shading and root competition. They have been applied extensively to investigate patterns of succession in a wide variety of forests (Shugart, 1984) by considering species composition in small plots in a stand. Each plot is typically only slightly larger in area than the crown area of a single dominant adult tree. Gaps are created when the dominant dies, and the models track the transients of composition in the plot over a time scale of centuries. From Monte-Carlo runs it is possible to make statistical predictions about the effects of alternative disturbance regimes on forest composition. There is no inherent reason aside from computer limitations why this cannot be applied on larger spatial plots.

These models typically use very simple individual growth models, though more complicated ones have been applied in the case of species

for which a good physiological data base is available (Makela and Hari, 1986). Despite their general lack of physiological detail however, these models produce quite realistic predictions for forest dynamics that have been validated in a few cases. Perhaps their weakest component is the handling of competitive effects, for which there is not much general agreement. Recent models of plant populations which take into account explicit neighborhood effects on survivorship, mortality and fecundity may provide some basic theory appropriate in this regard (see Pacala, 1988, for a review). Despite their lack of physiological detail and the fact that these models often include so many parameters that model tuning is a real problem, the approach offers great hope for investigating how altering physiological characteristics of the component species will affect community-level processes (Huston and Smith, 1987).

With regard to resource management, it should be clear from the above that we are still very ignorant about how to scale up from the detailed knowledge available on cellular and organ levels to even a whole plant, let alone to stand and regional scales. But when is it really necessary to do this? Many of the models that are currently used by agronomists and foresters to predict harvests, and schedule fertilization, irrigation and pesticide application are empirical in form. These models work well as long as the data base upon which they are based is adequate. For predictions outside the range of available data however, mechanistically-based models are necessary. One example, discussed below, concerns the long term effects of atmospheric CO_2 increases. For management at the level of individual farmers, an approach which couples a mechanistic model with expert systems methods may well be the best combination of empiricism (from the intuition and experience of the expert opinions solicited) with mechanism (Lemmon, 1986). A major limitation in all these approaches is the unpredictability of the environmental inputs. Stochastic simulators of variables such as rainfall can be included in most approaches, leading to estimates of the variance or even the full probability distribution of yield. Management decisions will then depend upon the manager's assessment of how much risk is acceptable.

It has been argued that in order to make reasonably accurate predictions of the long-term effects of atmospheric CO_2 increases on world productivity, it is necessary to construct systems models which are capable of extrapolative prediction on an ecosystem level, based on mechanistic models for physiological responses to CO_2 (Reynolds and Acock, 1985). While I am sympathetic with the reductionist sentiment which underlies this, I am also quite pessimistic that detailed

physiological models are either possible to apply or to validate at regional or world scales. I believe that the best that one could hope for from such models is that they would suggest relatively simple empirical models, that though they lack the details of the mechanistic approach, would still be fairly accurate predictors. This uses a complicated model to determine what parameters really matter, and suggest macrodescriptors that would be fairly robust. On these scales, robust might be defined as within an order of magnitude of the actual.

On world scales, I believe it is much more reasonable to pursue top-down models. These may still include physiologically reasonable formulations, as Landsberg and McMurtrie (1985) argue for in the case of forest management. As an example of how this might be formulated, I'll describe an (admittedly fairly obvious) approach to investigating world productivity changes due to CO_2. My objective is to point out how one might integrate models on different scales, in essence by decoupling them.

Suppose that the world is broken up into several different vegetation types, $i = 1,..,n$, such as deciduous forest, grasslands of different types, etc. Let

$A_i(u)$ = land area of vegetation type i under world conditions u

$P_i(u)$ = productivity (e.g. biomass production per unit time) per unit area of vegetation type i under world conditions u

$u(t)$ = a time-dependent vector of world conditions (i.e. the distribution of temperature, precipitation, CO_2, etc. over the earth's surface) at time t.

Then u(t) would be generated by a climate model, and if there were several such models then their outputs would each be used to give some estimate of the potential variance in productivity. Then total productivity in year t is

$$P(t) = \sum_i A_i(u(t))\, P_i(u(t)) \qquad (6)$$

This allows a decoupling of abiotic effects from biotic ones since the $A_i(u)$ might be estimated from Holdridge-type diagrams obtained from a climate model. In this simple case, direct effects of CO_2 on plants are viewed as not being important in determining future world distributions of vegetation types, compared to the effects of climate change.

To estimate $P_i(u)$, consider there to be many species types within the vegetation type i. Then

$$P_i(u(t)) = P_i(u_0) + \delta P_i(u(t)) \qquad (7)$$

where $P_i(u_0)$ is the value of productivity in vegetation type i at present and $\delta P_i(u(t))$ is its change from the present to time t. The models may well be much more accurate predictors of changes than of absolute values of productivity and, furthermore, whatever data is collected to validate the models will only be at one or a few CO_2 levels. Then letting

$f_{ij}(u)$ = the fraction of ground area in vegetation type i occupied by species j under world conditions u

$B_{ij}(u)$ = productivity per unit area of species type j in vegetation type i under world conditions u

with $\delta B_{ij}(u)$ and $\delta f_{ij}(u)$ representing changes in these when conditions change from present (u_0) to u, we have

$$\delta P_i(u) = \sum_j \{ \delta B_{ij}(u)\, f_{ij}(u) + \delta f_{ij}(u)\, B_{ij}(u_0) + \delta f_{ij}(u)\, \delta B_{ij}(u) \}; \qquad (8)$$

Here the sum is over all species types in the vegetation type (i.e weeds, pines, grasses, etc.)

In the above, the f_{ij} would presumably come from community-level models taking into account relative competitive ability changes under elevated CO_2, differential abilities for species to adapt to elevated CO_2, etc. The B_{ij} could come from a relatively simple empirical model, possibly derived from complicated physiologically-based models. The above procedure allows one to set up a variety of "null models" since it decouples the community level effects from the direct effects on physiology. For example, one could investigate the assumption that relative species compositions within vegetation types will not change under elevated CO_2 by setting $\delta f_{ij} = 0$. This approach also allows one to estimate how sensitive the larger scale results are to changes in models on smaller scales. In this way it may be useful in providing some confidence interval, or range, for possible effects on world scales from confidence intervals on parameters in the lower-level models. From a public policy perspective, even fairly rough confidence intervals from procedures such as the above would provide a rational basis for analyzing the long-term effects of alternative governmental responses.

5. Spatial Aspects of Plant Epidemiology

The purpose of this section is to point out some relatively unexplored problems in epidemiology that arise from considerations of spatial scale. The vast majority of mathematical work in epidemiology concerns the spread of disease in homogeneously mixing populations. This assumption is reasonable for many animal populations in which the spread of the disease is caused by contact between animals that move about. Even so, it is not realistic in cases for which there is either spatial or some other structure in the population which causes there to be higher contact rates within certain groups than between these groups. There have been a variety of models developed to analyze the effects of such structure in the host population on disease spread (Hethcote, 1978; Post et al., 1981; May and Anderson, 1984). In contrast to the situation in animal epidemiology, there has been relatively little theoretical development in the spatial aspects of plant epidemiology.

Plant epidemiology offers two important differences from the epidemiology of animal diseases, at least as far as modeling is concerned. First, since plants are fixed in space for much of their lifespans (and in essence all of it for crops), disease spread does not occur due to contacts between individuals but rather through the dispersal of the pathogen itself. There is thus an explicit spatial aspect in plant epidemics that has long been noted, but generally ignored from a modeling perspective (Gilligan, 1985). Secondly, plants generally have a continuum of resistance levels to any particular pathogen. The effect of infection by a pathogen on a given plant may range from severe damage or death to no damage at all. In many crop plants, infection leads to reduced growth and yield, but rarely to premature death of the host. This implies that the usual mathematical structure of epidemiological models - classes of infected, susceptible, immune and removed individuals - is inappropriate for most plant situations. The situation is similar to the case of macroparasitic infections in humans (Anderson and May, 1982).

Due to the above, even in monocultures of genetically uniform crop plants, it is inappropriate to assume that disease is spread either uniformly or according to a Poisson distribution over the host population. Despite this, much of the work on the temporal spread of a disease makes this assumption (Rouse, 1985; Hau et al., 1985). What

work has been done on the spatial aspects of disease spread tends to be either highly empirical (i.e. statistical models for spore dispersal) or based on physical transport models with very little emphasis on biological effects (McCartney and Fitt, 1985). With few exceptions (i.e. Kampmeijer and Zadoks, 1977) there has been little work which attempts to simulate the dispersal of a crop disease and couple it with the growth of the crop. At the same time, essentially no attempts have been made to analyze the stochastic nature of the spatial spread of crop disease (Gilligan, 1985).

One approach to this is to utilize the individual-based growth models mentioned above. A somewhat preliminary investigation of this method was undertaken by a former student of mine (Bullock, 1986). The objective here was to investigate the effects of alternative spatial patterns of mixtures of resistant and non-resistant plants on the spread of a fungal pathogen. The method used a very simple individual growth model, logistic in form, with no neighborhood competition or physiological effects of environment. Pathogen was introduced according to a random process, with local growth dependent upon leaf area available, and dispersal occuring once pathogen density on a host reached a certain fraction of the host's carrying capacity. Dispersal was determined by an exponential random variable, with the capability to bias the spread due to prevailing winds. Host growth rate was either reduced as a function of pathogen load (for non-resistant plants) or independent of pathogen density (resistant plants). Several alternate spatial patterns were considered including uniform, bordered, striped and checkerboard. Criteria investigated were mean total biomass at end of season as well as probability that biomass at season end was below some threshold (presumably that at which profit is zero). Results indicated that generally the more divided the field, the smaller the amount of damage there was. Also, it was determined that bordering a field with resistant plants, as is sometimes suggested to farmers, had little effect on slowing an epidemic unless pathogen dispersal distances were very small.

It is possible to formulate an analytic approximation to the above situation, if one is willing to make certain assumptions. Consider the situation in one dimension only, and suppose the pathogen spreads according to a diffusion process with local growth dependent upon the local densities of the two host types. Also suppose there is no neighborhood competition in the host, so that plant growth is given by an ordinary differential equation. Then a general form of the problem is

$$\frac{\partial r}{\partial t} = D r_{xx} + f(r, g_1, g_2)$$

$$\frac{dg_1}{dt} = h_1(r, g_1, g_2) \qquad (9)$$

$$\frac{dg_2}{dt} = h_2(r, g_1, g_2)$$

with $g_i(x,0) = k_i(x)$, $i=1,2$, $r(x,0) = r_0(x)$, $0 \le k_i \le C$, and

$$\int (k_1 + k_2)\, dx = M.$$

In the above, the $g_i(x,t)$'s are the densities of the two plant types at location x at time t, $r(x,t)$ is the pathogen density there, f is the local growth rate of the pathogen, the h_i's are the growth rates of the plants, the k_i's are the initial planting densities of the two plant types which are bounded by C and total initial planting is M, and $r_0(x)$ is the initial pathogen distribution. In addition one could attach zero boundary conditions for the pathogen density on the plot of length L say. The above becomes a control problem if the object is to choose the initial densities k_i so as to maximize the total biomass at end of season T

$$Y = \int_0^L (g_1(x,T) + g_2(x,T))\, dx. \qquad (10)$$

In even the non-control case, this problem is extremely difficult to analyze. In part motivated by this model, R. S. Cantrell and C. Cosner of the University of Miami have investigated a steady-state version of (10) in the simplified case of fixed "good" and "bad" regions for the growth and dispersal of a pathogen. In the control problem, even proving that bang-bang is optimal is very hard. One can pretty much intuit what the answers should be in some special cases depending upon the dispersal rate of the pathogen, the size of the plot, and relative growth rates of the two plant types.

The above is partly meant to show how rapidly mathematical models can become intractable, but also that simplifications of the model can lead to intriguing mathematical problems. There have been a number of other models for disease spread that essentially produce travelling waves. One is a simulation approach similar to the individual model described above (Minogue and Fry, 1983) and another considers the diffusion of pathogen from a focus (van den Bosch et al., 1988).

van den Bosch, F. J. C. Zadoks and J. A. J. Metz. 1988. Focus expansion in plant disease I: the constant rate of focus expansion. Phytopathology 78: 54-58 .

Bullock, M. T. 1986. A Spatial Simulation Model for Disease Spread in a Crop. Master's thesis, Univ. of Tenn., Knoxville.

Charles-Edwards, D. A. 1982. Physiological Determinants of Crop Growth. Academic Press, Sydney.

France, J. and J. H. M. Thornley. 1984. Mathematical Models in Agriculture. Butterworths, London.

Gilligan, C. A. 1985. Introduction to: Mathematical Modelling of Crop Disease, C. A. Gilligan (ed.). Advances in Plant Pathology, Vol. 3, Academic Press, London.

Gross, L. J. 1986. Photosynthetic dynamics and plant adaptation to environmental variability. P. 135-170 in L. J. Gross and R. M. Miura (eds.), Some Mathematical Questions in Biology - Plant Biology. American Math. Soc., Providence.

Hau, B. S. P. Eisensmith and J. Kranz. 1985. Construction of temporal models II: Simulation of aerial epidemics. Chap. 3 in C. A. Gilligan (ed.). Advances in Plant Pathology, Vol. 3, Academic Press, London.

Hethcote, H. W. 1978. An immunization model for a heterogeneous population. Theor. Pop. Biol. 14: 338-349.

Hunt, R. 1982. Plant Growth Curves. Arnold, London.

Huston, M. and T. Smith. 1987. Plant succession: Life history and competition. Amer. Natur. 130:168-198.

Jarvis, P. G. and J. W. Leverenz. 1983. Productivity of temperate, deciduous and evergreen forests. Chap. 8 in Encyclopedia of Plant Physiology, Vol. 12D. Springer-Verlag, Berlin.

Johnson, I. R. 1985. A model of the partitioning of growth between the shoots and roots of vegetative plants. Ann. Bot. 55: 421-431.

Kampmeijer, P. and J. C. Zadoks. 1977. EPIMUL, a Simulator of Foci and Epidemics in Mixtures of Resistant and Susceptible Plants, Mosaics and Multilines. Pudoc, Wageningen.

Kirschbaum, M. U. F., L. J. Gross and R. M. Pearcy. 1988. Observed and modelled stomatal responses to dynamic light environments in the shade plant Alocasia macrorrhiza. Plant, Cell and Envir. 11: 111-121.

Landsberg, J. J. and R. McMurtrie. 1985. Models based on physiology as tools for research and forest management. In J.J. Landsberg and W. Parsons (eds.), Research for Forest Management. CSIRO, Melbourne.

Lemmon, H. 1986. Comax: an expert system for cotton crop management. Science 233: 29-33.

Loomis, R. S., R. Rabbinge and E. Ng. 1979. Explanatory models in crop physiology. Ann. Rev. Plant. Physiol. 30: 339-367.

Makela, A. and P. Hari. 1986. Stand growth model based on carbon uptake and allocation in individual trees. Ecol. Model. 33: 205-229.

Neither of these consider the effects of the pathogen on the plant however.

6. Conclusions

In sum, I have argued that a physiological perspective is often useful, even when the scales of the problem of concern are considerably longer temporally and larger spatially than would normally be addressed by consideration of physiology. My key point might be succinctly stated as "A little reductionism is good for the soul, too much reductionism is bad for the heart". Thus, we gain a mechanistic understanding of the functioning of complex natural systems by taking a physiological perspective. At the same time, there are clear limits to the utility of a reductionist approach, evident from the large number of poorly understood parameters and functional forms which appear in large systems models. I argue for an intermediate approach which uses physiologically-based models to indicate appropriate macrodescriptors for large-scale phenomena. When this is coupled with an analysis of the system's structure according to the rates of the processes appropriate to the questions being addressed (O'Neill et al., 1986), we will have available a truely hierarchical approach to natural systems.

7. Acknowledgements

I thank an anonymous referee for helpful comments. My remarks on the effects of atmospheric CO_2 increases were initiated due to an invitation to attend a U. S. Department of Energy workshop on the topic, and I thank James Reynolds for the invitation to attend. I also thank Carole Levin for urging me to come up with the one sentence summary of my views mentioned in the conclusions above (as well as for a luscious meal during the Conference).

References

Anderson, R. M. and R. M. May. 1982. The population dynamics and control of human helminth infections. Nature 297: 557-563.

Baldocchi, D. D., B. A. Hutchison, D. R. Matt and R. T. McMillan. 1985. Canopy radiative transfer models for spherical and known leaf inclination angle distributions: a test in an oak-hickory forest. J. Appl. Ecol. 22: 539-555.

Barrett, J. R. and R. M. Peart. 1981. Systems simulation in U. S. agriculture. P. 39-59 in Progress in Modeling and Simulation, Academic Press, NY.

May, R. M. and R. M. Anderson. 1984. Spatial heterogeneity and the design of immunization programs. Math. Biosci. 72: 83-111.

McCartney, H. A. and B. D. L. Fitt. 1985. Construction of dispersal models. Chap. 5 in C. A. Gilligan (ed.). Advances in Plant Pathology, Vol. 3, Academic Press, London.

McCree, K. J. 1970. An equation for the rate of respiration of white clover plants grown under controlled conditions. In: I. Setlik (ed.), Prediction and Measurement of Photosynthetic Productivity, Pudoc, Wageningen.

McMurtrie, R. E. 1985. Upper limits to forest productivity in relation to carbon partitioning. In: The Application of Computer Models in Farm Management, Extension and Research. Austr. Inst. Agric. Sci., Queensland.

Minogue, K. P. and W. E. Fry. 1983. Models for the spread of plant disease: model description. Phytopath. 73: 1168-1173.

Montieth, J. L. (ed.). 1975. Vegetation and the Atmosphere. Vol. I. Academic Press, London.

Nobel, P. S. 1974. Introduction to Biophysical Plant Physiology. Freeman, San Francisco.

O'Neill, R.V., D. L. DeAngelis, J. B. Waide and T. F. H. Allen. 1986. A Hierarchical Concept of Ecosystems. Princeton University Press, Princeton, NJ.

Pacala, S. W. 1989. Plant population dynamic theory. In J. Roughgarden, S. A. Levin and R. M. May (eds.), Perspectives in Ecological Theory, Princeton Univ. Press, Princeton, NJ.

Post, W. M., D. L. DeAngelis and C. C. Travis. 1983. Endemic diseases in environments with spatially heterogeneous host populations. Math. Biosci. 63: 289-302.

Reynolds, J. F. and B. Acock. 1985. Predicting the response of plants to increasing carbon dioxide: a critique of plant growth models. Ecol. Model. 29: 107-129.

Reynolds, J. F. and J. H. M. Thornley. 1982. A shoot:root partitioning model. Ann. Bot. 49: 585-597.

Ross, J. 1981. The Radiation Regime and Architecture of Plant Stands. Junk, the Hague.

Roughgarden, J. 1986. The theoretical ecology of plants. P. 235-267 in L. J. Gross and R. M. Miura (eds.), Some Mathematical Questions in Biology - Plant Biology. American Math. Soc., Providence.

Rouse, D. I. 1985. Construction of temporal models I: Disease progress of air-borne pathogens. Chap. 2 in C. A. Gilligan (ed.). Advances in Plant Pathology, Vol. 3, Academic Press, London.

Shugart, H. H. 1984. A Theory of Forest Dynamics. Springer-Verlag, NY.

Thornley, J. H. M. 1976. Mathematical Models in Plant Physiology. Academic Press, NY.

ECONOMICS, MATHEMATICAL MODELS AND ENVIRONMENTAL POLICY

Jon M. Conrad
Department of Agricultural Economics
Cornell University
Ithaca, New York 14853

Abstract

This paper briefly reviews several models of externality which provide the theoretical basis of environmental economics. An externality may be defined as a *situation* where the output or action of a firm or individual affects the production possibilities or welfare of another firm or individual who has no direct control over the initial level of the output or activity. Pollution, resulting from the disposal of residual wastes, is a classic example of externality.

Three static models examine the optimality conditions for (1) a two-person externality, (2) a many-person externality (where the externality takes the form of a "pure public bad"), and (3) a two-plant polluter. In the case of a two-person externality negotiation between the affected parties may lead to the optimal level for the externality *regardless* of the initial assignment of property rights. In the many- person case, environmental policies, such as direct controls or economic incentives, may be required to achieve an optimal allocation of resources. Economic incentives may take the form of per unit taxes on emissions or transferable discharge rights. In the third model it is shown how a tax can induce optimal (least cost) treatment from a two-plant polluter.

Two dynamic models examine the cases where (1) a pollution stock may accumulate or degrade according to rates of discharge and biodegradation and (2) a toxic residual must be transported from sites where it is generated to sites where it may be safely stored. The latter problem poses environmental risks from spills in transit or leakage at storage sites.

While radioactive and toxic wastes are likely to continue to be regulated by direct controls some of the more "benign" residuals are suitable for regulation by economics incentives. Effluent taxes in France and the Netherlands, transferable discharge permits on the Fox River in Wisconsin, transferable stove permits in Telluride, Colorado and the EPA's emission-offset policy are indications that economic incentives will play a greater role in the future management of environmental quality.