I. Introduction

The essence of good model building is to find the right simplifications of reality: what are the most relevant aspects of a system for understanding the problem at hand? Naturally, models should be constructed in accordance with what is known about the biology of a given situation. However, paradigms of what nature is assumed to be, instead of what it is known to be, may receive undue emphasis in models.

Models can be classified as strategic or tactical (May 1974). Tactical models are built to understand a specific system, e.g., the interaction between barley and barley mildew. The aim of these models is often more applied, and the tailored nature of the model allows various empirically measured details to be included. These models may thus give exact predictions about, e.g., yield loss due to pathogenic infections. However, these models may include so many details that the structure of the system, and the effects of slight changes in the model are most difficult to understand. A full (global) analysis of the model is not possible, because only a limited number of parameter values and initial conditions can be studied in computer runs.

Strategic models are not meant to give precise quantitative predictions, but to give a qualitative insight into the system. May (1974) compares strategic model with idealized (like frictionless) systems in physics that are known to be unrealistic, but nevertheless form the backbone of classical physics. For a good strategic plant/pathogen model, we need to concentrate on the most general aspects of such systems.

The questions we want to address should determine the type of the model, although most actual models have both strategic and tactical aspects. Probably the best way to combine these alternatives is to formalize the problem as an analytical (strategic) model, and make use of computers when mathematical tools or skills no longer work. This section concentrates on the general aspects of strategic models.

Traditionally, plant pathology has aimed at understanding how yield of agricultural plants could be improved by introducing new one-locus resistance genes, but studies of nonmanaged plant/pathogen systems have been rare. Practices and theories for agricultural systems have developed quite independently from other biological disciplines.

In ecology/studies on interactions between species have always been in the mainstream. However, animal interactions have been the main target of modeling and data-collecting efforts, and until very recently, interactions between plants and pathogens have been largely ignored. Much
research has been devoted to the specific and unique genetic basis for plant/pathogen interactions, the gene-for-gene system (Flor 1942, 1956), which has seldom been found in interactions between other groups of organisms (Thompson and Burdon 1992). Instead of emphasizing the differences between systems, it is time to acknowledge the similarities in many antagonistic interactions where one species benefits and the other loses. Naturally, I will concentrate on plant/pathogen models, but I am convinced that more progress in this field could be made by integrating research on plant/pathogen systems more closely with related fields such as plant insect studies or animal/parasite studies.

Recent evolutionary biology has tended to merge ecological and genetic aspects of populations. It is now widely acknowledged that both quantity (density) and quality (resistance and virulence) of interacting species influence genetic and ecological characteristics of a given species (Antonovics 1994). The evolutionary process has been called coevolution (Janzen 1980), if the species show reciprocal adaptations. Plants should develop more defenses when attacked by virulent pathogens. In the same way, plants that are harder to attack should cause more aggressive pathogen genotypes to develop if this is evolutionarily possible.

A plant/pathogen interaction may lead to extinction of pathogen population, extinction of both species, or limited growth of both populations. If both species persist, genetic variation in one or both species may be maintained or lost. Most plant/pathogen models are equilibrium models, or have concentrated on predicting the outcome of these interactions in a homogeneous environment. In recent years, more realistic assumptions about the environment have produced nonequilibrium models, where the analysis of dynamics is more important than the final outcome.

Plant/pathogen models assume implicitly or explicitly many biological properties of the species, like the sexual system, speed of evolution in host and pathogen populations, or overlap of generations. These assumptions are typically made to allow clearer analysis of the studied problem. However, without further studies, it is extremely difficult to conclude whether the predictions of such models apply only to exceptional (or even impossible) cases.

II. Concepts and Assumptions

A. The Nature of Genetic Variation

Since the ingenious experiments of Flor (1942), the paradigm of plant/pathogen studies has been discrete major-locus variation. If pathogen isolates also differ in their ability to infect plant genotypes, the gene-for-gene hypothesis is routinely invoked. The evidence for gene-for-gene interaction in agricultural systems and some wild relatives of cultivated plants is well established (Alexander 1992), but opinions on the generality of the idea vary widely (Day 1974; Barrett 1985). The importance of these systems has increased with the possibility in the near future for molecular manipulation of major locus resistance genes (Gabriel and Rolfe 1990; de Wit 1992). The number of suggested gene-for-gene interactions is increasing (Thompson and Burdon 1992), but the actual number of proven cases is still somewhere around ten. Without doubt, plants have major locus resistance genes, and discrete variation in the ability of pathogens to attack plant genotypes exists. Most of these pathogens reproduce asexually, so their populations consist of separate lines. However, this does not prove that most plant/pathogen evolution is based on gene-for-gene interactions.

Quantitative resistance may be more common in types of pathogens that have not been intensively studied in agriculture (e.g., nonbiotrophs; Parlevliet 1989; Groth and Crist 1992), but there are good examples available on quantitative variation in agricultural plant/pathogen systems as well (Geiger and Heun 1989). Quantitative variation seems to occur in all studied plants, including the best examples of gene-for-gene system (Sidhu 1988; Parlevliet 1989). Further, genetic interactions between plant and pathogen genotypes are not limited to gene-for-gene systems; but genotypic interactions can be found in quantitative characters as well (De Nooij and Van Damme 1988a,b; Saloniemi 1993b).

Very little can be said about the importance or prevalence of different kinds of resistance. Nearly all models of plant/pathogen interaction are based on gene-for-gene assumptions, although explicit quantitative genetics has been employed in one predator-prey model (Saloniemi 1993a), and assumptions of continuous variation have been included in some models (Frank 1994).