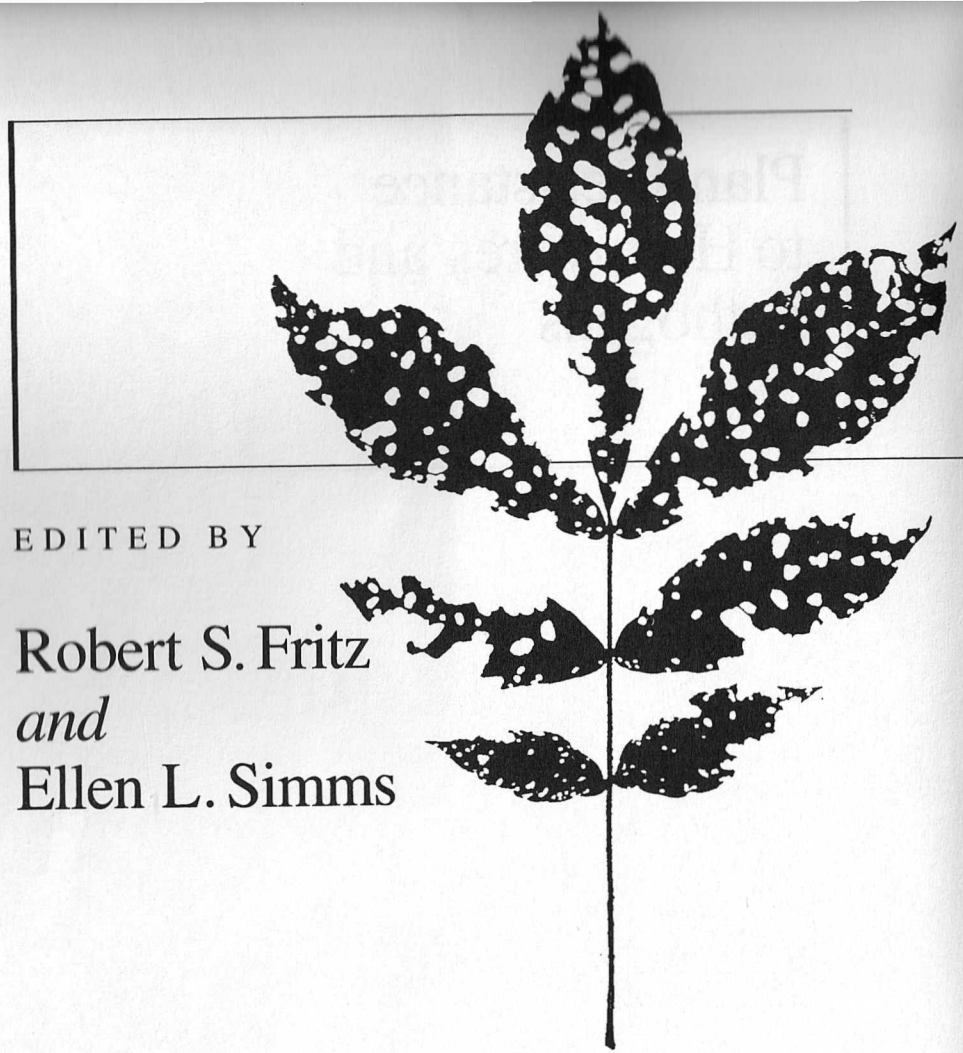

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18

Toward Community Genetics

Janis Antonovics

To anyone interested in the richness and diversity of the biological world, the concept of coevolution is as seductive as it is tantalizing. If interactions among organisms are indeed a driving force for reciprocal evolution, then given the obvious fact that such interactions are often complex, it is easy and tempting to cast coevolution as a major player in producing mutual adaptations that preserve and generate species diversity.

Such thoughts perforce flutter across the mind of any sensate natural historian, whether in a Costa Rican rain forest or in an English hedgerow, where like the dappled shade in early spring they cannot but stimulate, energize, and increase the sense of wonder. But to a professional biologist, often that selfsame natural historian back from a field trip, the study of coevolution is fraught with conceptual pitfalls and with practical difficulties that are often as prosaic as they are real.

The term "coevolution" has itself been applied to rather diverse phenomena, sometimes referring to processes that are macroevolutionary and recognizable through correlated phylogenies, or sometimes referring to processes that are defined as such only if there is a reciprocal interplay of selection pressures and gene frequencies among the ecological interactants (for discussion see Thompson 1989). For the ecologist interested in species interactions, it is essential to realize that a "species" is composed of a highly heterogeneous class of individuals differing phenotypically because of the varied influences of not only environment, age, or phenology, but also genotype. And genetic variation affecting the processes involved in species interactions will change those processes both in the immediate sense and in terms of future evolutionary change. Over the past 20 years the species as a unit recognized by taxonomists has come increasingly under attack as a valid unit of ecological analysis (Birch 1960; Antonovics 1976a; Harper 1982). At best the Latin binomial and what it represents is seen as an approximation, while at worst it is seen as dangerous and completely misleading. The major impasse, and one which to some extent leads

us to persist in a typological characterization of the species, is that at present we have no convenient (one is tempted to say simple-minded) substitute for the species concept. The papers in this volume cite numerous instances where genotypes within a species may behave in a quite contrasting manner in response to pathogens or herbivores. Conversely, different species may be treated as homogeneous by other pathogens or herbivores and should therefore perhaps be treated as "equivalent" genotypes (Janzen 1979). However, the technical difficulties in operationally defining genotypic or guild categories serve to make the prospect of abandoning the species as an ecological unit seem like a dangerous flirtation with prospective anarchy.

Probably the simplest (minimal) framework that encapsulates the essence of these complexities is shown in figure 18.1a. This diagram, originally developed by Levin and Udovic (1977) in the context of a mathematical model of coevolving populations, illustrates the different ways in which two noninterbreeding populations may interact with regard to their gene frequencies and their numerical abundances. For the theoretician, even this simple two-species model requires one to keep track of population size and gene frequency, and if one adds in the component ecological parameters (e.g., birth rates, death rates, disease transmission coefficients), the model can rapidly become overparameterized. Yet such an analysis becomes necessary if one wants to understand the dynamics and possible equilibria of relative species abundances, population sizes, and gene frequencies.

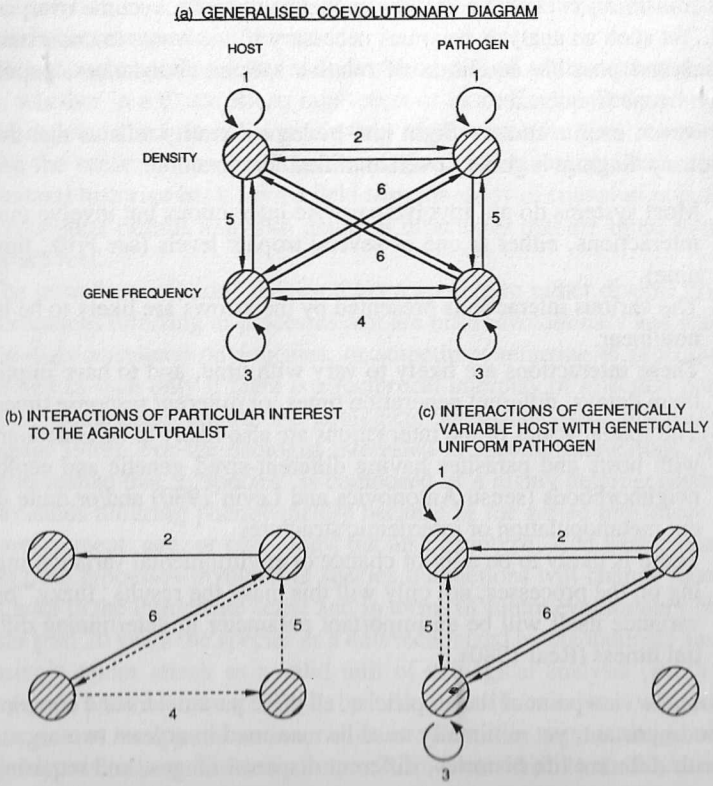
However, even a cursory flight into biological reality tells us that this coevolutionary diagram is greatly oversimplified. For example:

1. Most systems do not involve pairwise interactions but involve multiple interactions, either at one or several trophic levels (see Fritz, this volume).
2. The various interactions presented by the arrows are likely to be highly nonlinear.
3. These interactions are likely to vary with time, and to have implicit in them delays, different generation times, or different response times.
4. The spatial scales of the interactions are also likely to be noncongruent, with hosts and parasites having different-sized genetic and ecological neighborhoods (sensu Antonovics and Levin 1980) and/or quite different metapopulation or interdemic structures.
5. There is likely to be a lot of chance or environmental variation impinging on the processes; not only will this make the results "fuzzy," but the variance itself will be an important parameter in determining differential fitness (Real 1980).

From the viewpoint of the empiricist, all these parameters and complexities could be important, yet minimally must be measured in at least two organisms, often with different life histories, different dispersal ranges, and requiring dif-

ferent rearing techniques. Failure to coordinate just the latter can mean an ignominious end to an otherwise well-conceived project. Given this scenario, it may seem that any sensible biologist would abandon the study of species interactions, brush coevolution under the rug, and resort to simpler systems, to model evolutionary paradigms where, for example, toxic mine spoils or polluted cities provide an unresponding backdrop for the evolutionary theater. However, we often become professional biologists precisely because of our fascination with the intricacies of the biological world, and we would not be scientists if the study of complexity did not have its rewards, its modest inroads, or even its dreams of major insights.

The biological richness at the heart of species interactions, combined with the difficulties in analyzing them explicitly at either the empirical or the theoretical level, leads to the question of whether there is indeed not a much larger discipline which encompasses the discussions regarding the role of genetic variation in influencing species interactions and determining community structure. During a discussion of this issue a number of years ago with Dr. Jim Collins, of



Arizona State University, he suggested that we need a new level of analysis, a new area of biology called "community genetics." This discipline would emphasize the analysis of evolutionary genetic processes that occur among interacting populations in communities. There was also an earlier excursion into this idea by Wilson (1976), who discussed ways in which diffuse coevolution could occur at the level of communities. If molecular, cellular, developmental, and population genetics, why not community genetics? Conceptually, it may not be farfetched to partition the subject of ecological genetics into subdisciplines at the population, community, and perhaps even ecosystem level (Loehle and Pechmann 1988). Above all, the concept of community genetics frees us from the overly restrictive frame of reference, the reciprocality, that coevolutionists would choose for their own discipline. If our research is no longer dependent on returning with a genuine scalp of reciprocal coevolution, we can generalize community processes in terms of interactions that occur among genotypes as individuals, and by extension use our efforts as a vehicle for further understanding when and how taxonomic characterizations should be incorporated into ecological thinking, or when they are misleading.

Within the subject of community genetics, there are two major approaches, one reductionist and the other holistic, to understanding the underlying complexity of species interactions. The approach that links population biology to community interactions is clearly reductionist, and it is this approach that is emphasized in the present volume. It is also this approach that I wish to discuss

Opposite: Fig. 18.1 (*a*) Diagram of the possible types of interactions between densities and gene frequencies for two interacting species (after Levin and Udovic 1977). Arrows: (1) Intraspecific density dependence; (2) effect of density of one population on growth rate of the other; (3) differential fitness among genotypes unrelated to conspecific density, or composition of the other species; (4) density-independent differential fitness caused by the other species; (5) intraspecific density-dependent selection; (6) density of one species affecting relative fitnesses of genotypes in the other species, or gene frequency of one species affecting the density of the other.

(*b*) The subset of interactions that are of primary interest to the agriculturalist. Crop genotype changes pathogen abundance (solid arrow 6), which affects crop yield (arrow 2). Pathogen evolves virulent genotypes (arrow 4), which changes pathogen abundance (arrow 5), which in turn affects crop yield (arrow 2) and leads to further breeding for resistant varieties (dotted arrow 6).

(*c*) The interactions between densities and gene frequencies for a genetically variable host population and a genetically uniform pathogen population. Pathogen population size is influenced by and influences host numerical (arrows 2) and gene frequency dynamics (arrows 6). Density dependence occurs in the host (arrow 1) and there is genetic variation in costs of resistance that would result in genetic change in the absence of the pathogen (arrow 3). However, there may (if costs are in female fecundity) or may not (if costs are in terms of male fecundity) be direct effects of resistance on density regulation (arrows 5).

first, and to elaborate and extend upon using results from our own studies of a natural plant-pathogen system. Later, I will discuss more holistic approaches and suggest that they may be equally important for achieving a full understanding of community genetics.

The Reductionist Approach

By focusing on the plant component in plant-herbivore, plant-pathogen interactions, this book takes a reductionist approach to the study of community genetics. In doing so, it also immediately exposes the enormous commonality between the interests of the agricultural scientist and the population biologist (Harper 1967). The focus of both disciplines has been to document and understand the nature of variation in plant resistance, and to assess the degree to which such variation affects herbivores and pathogens and thus how resistance affects the fitness or yield of the plants themselves. This focus represents a small subset of the coevolutionary diagram (fig. 18.1b). It is immediately obvious from the papers in this volume that plant populations contain an abundance of genetic variation in resistance and susceptibility: direct studies of crop plants, of their wild relatives, and of other natural populations strongly support this contention. It cannot be overemphasized how dramatic these differences can be: within one population, individuals can be totally resistant or seemingly totally susceptible. What is equally clear, however, is that beyond the immediate documentation of such variation in resistance relatively little is known about its origin, stability, and distribution. It is tempting to assume that its origin is mutational or recombinational, that its greatest expression should be in areas of greatest herbivore/pathogen abundance and that it has been amplified and is being maintained by coevolutionary interactions. However, the chapters in this book seriously question this scenario. Agriculturalists often find substantial variation in resistance to a pathogen even in populations that seemingly have never been exposed to the pathogen, at least in the recent past. The lack of a cost to resistance, or even a positive correlation with fitness, if taken at face value, implies a nonequilibrium world with perhaps resistances spreading toward possible fixation. Absence of the occurrence of "losers" in the coevolutionary process is evident from the well-known cases of large-scale effects of introduced alien pests on the abundance of natural or seminatural populations (Gibbs 1978; Cullen et al. 1973), from the success of biological control programs, and from the use of pathogens directly as weed control agents (Wilson 1969; Templeton et al. 1979; Zettler and Freeman 1972). The studies described in the present volume make clear that assumptions about the coevolutionary process are at best hypotheses still in need of critical examination for the many different types of plant herbivore/pathogen interactions that occur in nature.

In spite of the common interests of the agriculturalist and the population biologist in the phenomenon of plant resistance, there are important differences

that need to be clarified so the two can continue to interact creatively. Earlier chapters (e.g., Alexander) have mentioned the conflict in any empirical science between achieving generality and achieving precision and realism. In any science, the pure and applied fields have quite different orders of priority in this regard. The pure scientist is primarily interested in generality, in theories that cast a broad sweep of understanding and unification over seemingly diverse aspects of a particular discipline. Excursions into particular systems are necessary to check the general theories and to develop new biological insights, but are to some extent secondary. Applied scientists, on the other hand, are interested in precision and realism. They have to operate within a specific real-world framework to achieve some direct predictability. Excursions into generality are secondary, necessary only to see the interconnectedness of ideas and approaches, so that these can be a stimulus to new answers for solving the particular problem at hand.

Pure and applied sciences also work on quite different time scales. The pure scientist (daily pressures of the job apart) would ideally like to be timeless, to generate truths that are eternal and irrefutable: underlying the scientific enterprise there is a belief that an objective reality exists that can eventually be understood. On the other hand, the applied scientist's goals are driven by urgencies and immediacies: achievement is more often seen and rewarded in terms of a rapid, transient success than by eternal solutions. For example, agricultural scientists want to predict immediately whether the herbivore/pathogen will reduce yield in that selfsame year so management practices can be put into force that minimize yield reduction. This is the reason for the focus on herbivore and pathogen effects expressed largely in terms of disease symptoms. Unfortunately, the concepts of resistance-susceptibility or virulence-avirulence are only tangentially related to demographic parameters of relevance in fitness estimation. Even in the agricultural literature, there needs to be frequent reminder of the fact that disease occurrence may relate only partially to yield decrement; overuse of pesticides and fungicides, in particular, may at times be motivated more by an aesthetic desire for clean crops than by economic considerations. This focus on symptoms rather than on fitness effects has led to discordant methodologies, to a noncorrespondence of concepts, and to differences in assumptions about the evolutionary process in the approaches of population biologists and applied plant pathologists (Antonovics and Alexander 1989).

Perhaps the greatest disjunction that has resulted from the focus on resistance per se has been a failure to appreciate the potential dynamical behaviors of plant-herbivore or plant-pathogen systems. Indeed, there has been a serious lack of explicit studies on how pathogens and herbivores influence the dynamics of plant populations, largely because of the focus on crop populations where numbers are preset at planting. For this reason the impact of plant pathogens and herbivores on plant population dynamics (defined as changes in numerical size of populations over successive generations) has not been a major concern

of the agriculturalist, except in cases of large-scale pandemics (Klinkowski 1970). Instead the concern has been with direct yield reduction, and with management of the less controlable component of the system, the pathogen or herbivore. As a result, there has been extensive study of pathogen spread in crop populations and this has provided information on spore dispersal, predictions of crop damage, and models for disease management (Leonard and Fry 1986, vol. 1). The emphasis however has been on epidemiology, on the short-term, within-season dynamics, rather than on long-term processes of the kind that characterize pathogen behavior in natural ecological systems. In order to understand and appreciate this dynamic behavior, it is important to express disease effects in terms of fitness impacts. Indeed, except for the purposes of recognizing pathogen presence or measuring transmission rates and modes, the nature of the disease symptoms is essentially irrelevant to the ecological and evolutionary dynamics.

Of particular concern to plant pathologists has been the genetic basis of disease resistance and susceptibility, or pathogen virulence and avirulence. But again, because these measures of resistance and virulence are difficult to translate into precise fitness effects (Nelson 1979; Antonovics and Alexander 1989), the agricultural literature can be used only tangentially for generalizations regarding natural populations. Moreover, it has been suggested that particular genetic interactions of plants and their pathogens (e.g., the gene-for-gene hypothesis) may be a product of selection in agricultural circumstances, rather than a reflection of patterns that actually exist in nature (Day 1974; Barrett 1985; but see Parleviet and Zadocks 1977). Indeed, many authors have appealed to natural systems as justification for a pathogen control strategy based on genetic diversity (Browning 1974; Harlan 1976), but others have cautioned that factors other than genetic diversity may also minimize disease incidence in nature (Schmidt 1978; Alexander 1988), and that more evidence based on ecological genetic studies is needed (Dinoor and Eshed 1984).

The above reasons make it perhaps understandable, but not less remarkable, that there have been, as far as I know, no explicit models of *numerical* dynamics of plant and pathogen populations, or of the interaction of such dynamics with gene frequency change. Models of gene frequency change in plant-pathogen systems (for review, see Leonard and Czochor 1980) have demonstrated that reasonable, empirically based assumptions can lead to the maintenance of genetic polymorphism in both the pathogen and the host, and have resulted in predictions for use of multiline mixtures for disease control (Leonard 1977; Barrett 1978; Marshall and Weir 1987). Generalized models of parasite-host systems developed by May, Anderson, and coworkers (Anderson and May 1979; May and Anderson 1979) provide the major theoretical paradigms that can be applied to natural plant-pathogen systems. They have been extended only in a limited way to analysis of genetic effects and evolution of sexual systems (May and Anderson 1983). While such models capture the essential dynamical properties of the systems they describe, their application to particular classes of disease requires empirical evaluation and extension to incorporate specific biological processes.

There obviously is also a dynamic in agricultural systems; the evolution of virulent pathogens may require the development of new varieties. But the dynamics will be slower and much less self-generating, being determined more by the pace of plant breeding programs and the rate of deployment of pest management strategies than by the biology itself. Rational deployment of multilines, where the opportunity exists for resetting plant genotype frequencies in each planting season, is probably the only situation in which joint dynamics of host and pathogen become critical in an agricultural context.

Finally it may be worth remembering that for the agriculturalist, the pursuit of intraspecific resistances may soon become a secondary process: given the possibility of interspecific gene transfer, resistances can be moved in from alien sources or engineered from the genes of the pathogen itself. Understanding the mechanics of interspecific variation in resistance (why do mosses not have fungal infections, even though they have no cuticle and live in cool moist habitats?) may become more crucial to the applied enterprise than understanding intraspecific variation.

In summary, the major goal of the agriculturalist is to understand and explore that small subset of the coevolutionary diagram that relates crop genotype, pathogen pressure, and yield. This is because crop genotypes and densities are normally reset at specific values each season and because it is essential to have immediate assessment of disease symptoms so as to invoke appropriate control measures. Longer-term dynamics are more a product of agricultural practices and possibilities than of the intrinsic biology of the host and pathogen. The population biologist, however, has a greater interest in a broader exploration of the coevolutionary diagram so as to include more of the direct feedback loops that are likely to be critical to the long-term numerical and gene frequency dynamics of natural populations. The generalities of the population biologist are in turn of interest to the agriculturalist at two levels: as a guide to strategies of crop deployment, particularly in low-input agricultural systems, and as a guide to gene conservation efforts. It is remarkable that a number of crop deployment and breeding strategies (e.g., multilines, hybrid varieties) are often justified beyond their intrinsic merits on the basis of a mythology of how the natural world should operate; empirical data on how it actually does operate (e.g., the role of genetic variation in ecological success, the extent of inbreeding depression) are often lacking or fragmentary at best.

The natural extension of many of the chapters in this book is therefore to ask how all the individual components fit together to produce an ecological and evolutionary dynamic that can be used to predict distributions, abundances, adaptations, and other evolutionary trajectories. In their discussion of the expected dynamics of the coevolutionary diagram, Levin and Udovic (1977) point

out that unless one makes unrealistic simplifying assumptions, the only generality to emerge is that it represents an "anything goes" situation. Populations that can coexist may not do so if there is genetic variation, and vice versa. Approach to gene frequency equilibrium may be accompanied by reduced population size. Or heterozygote deficiency may be associated with gene frequency equilibrium.

Fortunately, however, species interactions fall into various classes, each of which has distinct properties. The dynamical properties of plant-herbivore and plant-pathogen systems have not been explored sufficiently to say which types of interactions will have similar dynamics, but this book does suggest lines across which generalizations could be made. There is obviously the distinction between parasites, parasitoids, grazers, and predators: the ecological dynamics of these contrasting systems are well explored. Another distinction could be between situations where the plant is long-lived relative to the herbivore or pathogen and the reverse situation where the herbivore, for example, a mammalian grazer, is long-lived relative to the plants being grazed. In the former situation the host plants (whether at a phenotypic, genetic, or interspecific level) may be simply acting as a heterogeneous environment, and preexisting concepts and models of selection, habitat choice, and speciation in heterogeneous environments may be directly applicable to these cases (e.g., Maynard Smith 1966; Rausher 1984a). In the case of large grazers, the evolutionary forces acting on the plant community, as pointed out by Pollard (this volume), may have more direct parallels and be better described by models that are based on interspecific frequency–dependent behaviors characteristic of Mullerian (positive frequency–dependent) and Batesian (negative frequency–dependent) mimicry. Other possible distinctions might be systems with and without alternative hosts, whether pathogens are seed transmitted or not, and whether transmission is vector based or the result of wind dispersal.

To illustrate the nature of the gene frequency and numerical dynamics that can result from host-pathogen systems, I will digress into a description of some of the work we have been doing with the anther smut disease commonly found on members of the Caryophyllaceae. We in particular have been studying the anther smut (*Ustilago violacea*) of white campion (*Silene alba*).

Resistance and Susceptibility and the Dynamics of the *Ustilago-Silene* System.

This system was chosen for study because of the clear-cut effects of the pathogen on the host, its technical convenience, and its intriguing biology (Alexander and Antonovics 1988).

Silene alba or white campion (= *Melandrium album* or *Lychnis alba*, Caryophyllaceae) is a common weed of roadsides, old fields, and crops such as peas and alfalfa (McNeill 1977). It is a short-lived perennial that germinates in late summer and early spring. In growth chambers seed to flowering occurs in about six weeks; plants are dioecious and easily crossed. Under short days plants remain vegetative and can be cloned by cuttings.

Both male and female individuals of *Silene* infected by the anther smut fungus *Ustilago violacea* (Basidiomycetes, Ustilaginales) produce stamens with anthers that carry purple fungal spores. Infected females retain a rudimentary ovary but produce no seed. Except for initial stages of the infection process, usually all flowers on a plant are diseased and the individual is completely sterile (Alexander and Antonovics 1988). Otherwise infected plants appear normal, although they usually show increased flower production and a longer flowering period (Alexander 1987).

The spores (teliospores) produced in the anthers are diploid and transmitted by pollinators. Following transfer to another flower, they germinate and undergo meiosis to produce a short basidium of four haploid cells which in turn bud to produce yeastlike cells (sporidia). Fusion of sporidia of opposite mating types produces a binucleate infection hypha that penetrates the host tissue. Completely diseased plants have never been observed to recover and produce healthy flowers (Alexander and Antonovics 1988). There is no seed transmission, so newly established plants are disease-free.

The sporidial haploid stage can be maintained in liquid or solid agar culture using techniques similar to those for yeast (Cummins and Day 1977). Plants can be artificially infected at high frequencies by wounding rosettes with sporidial suspensions of mixed mating type or by soaking seedlings in such suspensions.

Our study populations are in the vicinity of Mountain Lake Biological Station, Pembroke, in southwestern Virginia. In this region, the pathogen is restricted to one host species, even though in Europe it is found on a wide array of hosts. In Virginia and other parts of the Southeast, we have found a similar anther smut infection in the fire pink, *Silene virginica*, but electrophoretic studies show it to be distinct from the anther smut on *S. alba* (Stratton 1990, personal communication).

In classical host-parasite models, the probability of an individual becoming diseased increases with the density of diseased individuals in the population. This assumes that spores are dispersed freely into the air, or that there is random encounter among mobile infected and uninfected individuals (as in animals). With pollinator-transmitted diseases, a different transmission process may be at play. It is known that pollinators increase flight distances when plants are more widely spaced (Levin 1972; Levin and Kerster 1974). As a result, given perfect "adjustment" of pollinator flight distances, disease transmission will be independent of density and dependent only on frequency of diseased individuals. A plant will receive spores if a pollinator visited a diseased plant on a prior visit, regardless of the absolute density of individuals within the population. Empirical evidence for this assumption comes from field studies (Alexander 1990b)

and experiments where we independently varied the density and frequency of diseased and healthy individuals in experimental arrays (Antonovics, unpublished data). Clearly, one would not expect this assumption to hold precisely, given spore carry-over or variations in pollinator behavior. However, the frequency-dependent nature of the disease transmission process is expected in any venereal disease where mate encounter rates are relatively independent of density, or in any vector-transmitted disease where vectors actively search out hosts and have a motility that exceeds that of the host. This anther smut disease is therefore a venereal disease not only by virtue of its being transmitted by pollinators, but also because of its transmission dynamics. Models of this disease therefore have broader relevance to venereal diseases in general, particularly those like gonorrhea, syphilis, and chlamydia that greatly reduce the fertility of their human hosts. In plants, anther smut diseases infect a large range of hosts in the Caryophyllaceae (Goldschmidt 1928) and related families such as the Portulacaceae (D. Ford, 1989, personal communication). Other pollinator-transmitted diseases are caused by fungi (Leach 1940; Broadbent 1960), by bacteria (Schroth et al. 1974), and by viruses (George and Davidson 1963; Cooper, Kelley et al. 1988).

Models of frequency-dependent transmission show that the conditions for host-pathogen coexistence are far more restrictive than in the case of "normal" diseases where transmission rates depend on density (Getz and Pickering 1983). Analysis of the *Silene-Ustilago* system is simplified because the possible state transitions are far fewer than in other host-parasite systems (fig. 18.2). We assume that individuals are either diseased or disease-free, and that diseased plants are completely sterile and never recover.

If we examine the purely numerical dynamics of this system (i.e., ignore

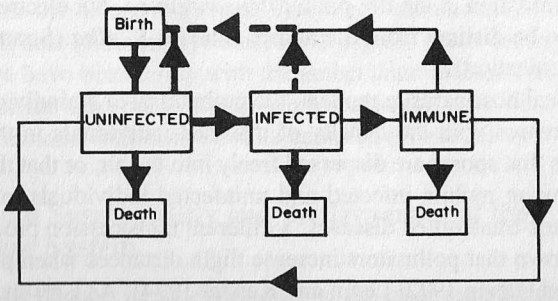


Fig. 18.2 Diagrammatic flow chart for state transitions in a directly transmitted disease (after Anderson and May 1979). The solid lines indicate state transitions considered in our theoretical models. Plants when first diseased may produce some healthy flowers, but this condition is transitory and rare: therefore this transition is indicated as a dotted line.

any genetic variation), then frequency-dependent transmission leads either to the population purging itself of the disease, or extinction of both disease and host (fig. 18.3). Moreover, unlike in the classical density-dependent transmission disease spread models (Kermack and McKendrik 1927), there is no threshold density for initial increase of the disease and therefore no critical density before epidemics occur. Initial disease spread will occur whenever the disease transmission coefficient, β, is greater than the death rate, d, of diseased individuals, regardless of density. However, if healthy individuals establish even more rapidly than the rate of disease spread, the disease becomes proportionately rarer in the population.

This simple model therefore predicts that the host and the pathogen should not stably coexist, and that the disease cannot regulate the plant population. One has either to invoke a nonequilibrium scenario, or to consider biologically realistic extensions of the model that may change these initial conclusions. The most obvious extension is to assume that the plant population is regulated by density-dependent factors that act independently of the disease itself (e.g., resource availability, safe sites for germination). Under these conditions (figs. 18.4 and 18.5), depending on the parameter values, equilibrium coexistence becomes possible (see also Alexander and Antonovics 1988 for an application of a similar model to a real population). In general, coexistence will occur if the density-dependent forces regulating the population act differentially on healthy and diseased individuals. This is very likely because density effects in plants, especially perennials, usually act most strongly at the seedling stage (Harper 1977; Shaw 1986). Yet the disease is transmitted largely at the adult flowering phase (but some seedling infection is known to occur if spores fall on young plants: Baker 1947; Antonovics and Alexander 1989; Alexander 1990b). Therefore the establishment of the healthy class is more likely to be influenced by density; diseased individuals can die only as adults and may be relatively insensitive to density. Again, this discordance in density-dependent effects on diseased and healthy plants may be generalizable to most venereal diseases, where almost by definition transmission is at an adult phase. We can therefore encapsulate the dynamics of such a system by assuming that density dependence acts solely on the birth rate of the healthy individuals, and not on the death rates of either the healthy or diseased. The result (table 18.1) is that as long as β is less than the birth rate at low density, b_0, and is greater than d, then coexistence of host and pathogen are possible.

While this may seem satisfying in that it provides an explanation of host-pathogen coexistence, it is important to calibrate this interpretation against reality. We currently have insufficient demographic data or data on transmission rates to assess whether real world populations fall in the "coexistence" region of parameter space or not. In one population that was studied intensively, coexistence was in fact not predicted (Alexander and Antonovics 1988). Moreover, the genetics of this host-pathogen system suggest that the model may be

DENSITY DEPENDENT
DISEASE TRANSMISSION

$$\frac{dX}{dt} = rX - \beta XY$$

$$\frac{dY}{dt} = \beta XY - dY$$

FREQUENCY DEPENDENT
DISEASE TRANSMISSION
(POLLINATOR)

$$\frac{dX}{dt} = rX - \beta \frac{XY}{N}$$

$$\frac{dY}{dt} = \beta \frac{XY}{N} - dY$$

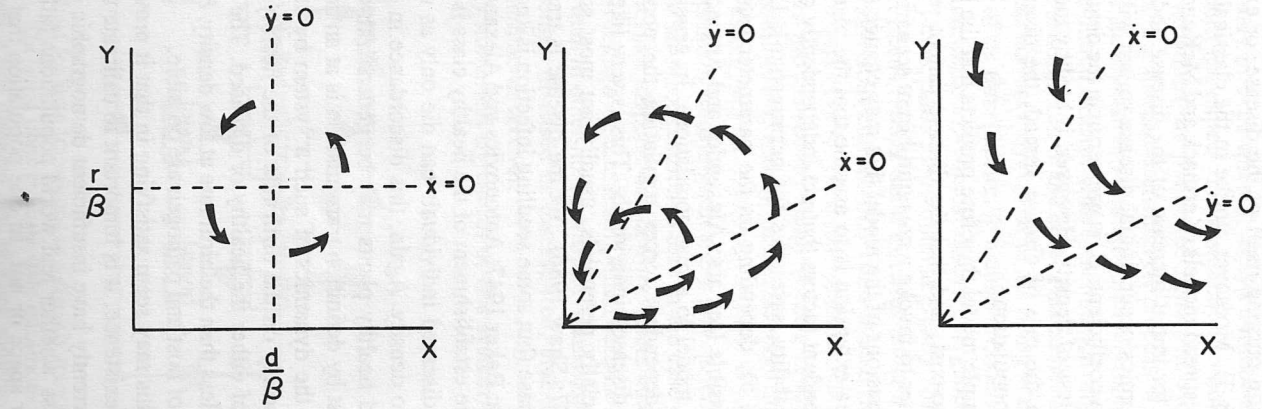


Fig. 18.3 Phase diagrams showing changes in numerical abundance of a host (X) and pathogen (Y) given density- or frequency-dependent disease transmission. Zero growth isoclines of host and pathogen are shown as dotted lines. r = birth-death rate of host, β = disease transmission rate, d = death rate of host, $N = X + Y$. Model is of *Silene-Ustilago* system illustrated in fig. 18.2.

DENSITY-DEPENDENT
DISEASE TRANSMISSION:
HOST SURVIVAL & REPRODUCTION
DENSITY DEPENDENT
$r = r - mN$, $d = d + sN$

FREQUENCY-DEPENDENT
DISEASE TRANSMISSION
(POLLINATOR):
HOST SURVIVAL & REPRODUCTION
DENSITY DEPENDENT
$r = r - mN$, $d = d + sN$

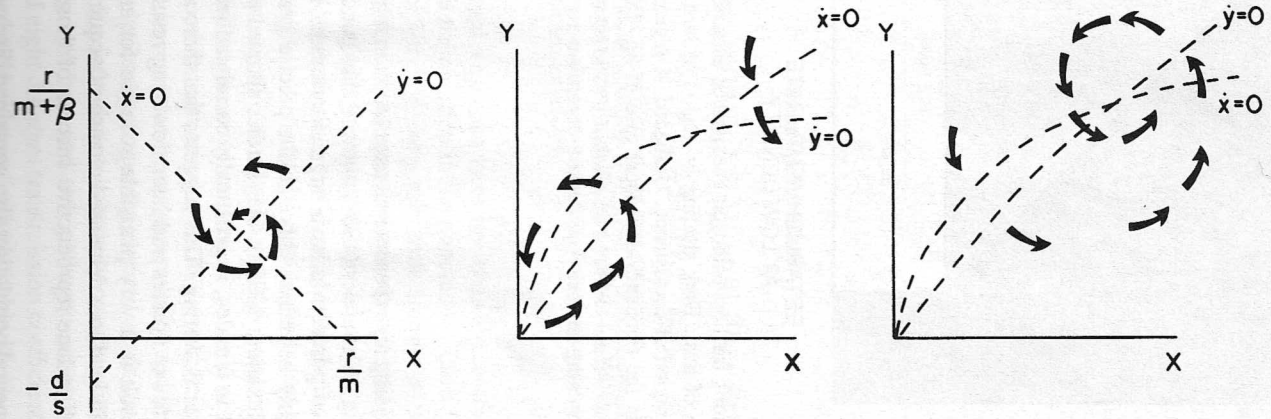


Fig. 18.4 Phase diagrams showing changes in numerical abundance of a host (X) and pathogen (Y) given density- or frequency-dependent disease transmission, but with linear density dependence of r and d on total plant density, N. Symbols as in fig. 18.3, with s and m = constants. Model is of *Silene-Ustilago* system illustrated in Fig. 18.2. Zero-growth isoclines for the frequency-dependent case are implicit functions in X and Y, and have been drawn only approximately.

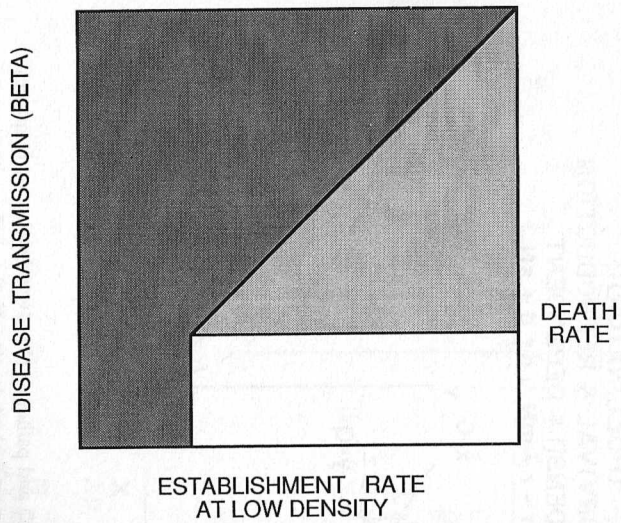


Fig. 18.5 Conditions for host-pathogen coexistence assuming density dependence of establishment rate of host. Dark shading = region of host and pathogen extinction. Light shading = region of coexistence. Unshaded = region of pathogen elimination. The model is as in the frequency-dependent case of fig. 18.4 except death rate d = constant, and $r = b/(kN+1)$ where b = establishment rate at low (zero) density, and k = constant determining intensity of density-dependent recruitment into the healthy class.

oversimplified. Using an experimental population in which genotypes were replicated by cloning, Alexander (1989) showed that genotypes sampled as seeds from one diseased population have large extremes of resistance. The level of resistance is highly heritable, although the precise genetic basis is still under investigation (Alexander 1990a). Her studies showed that resistance in males, but curiously not in females, is genetically correlated with lower flower production and with later flowering. This suggests that there are costs to resistance if we assume that fewer flowers and later flowering result in lower reproductive success. This result is a very plausible scenario for any venereal disease: reduced mating will lead to a decreased chance of acquiring the disease but correspondingly will reduce reproductive success of those individuals if they do remain healthy.

This experimental population also contained diseased individuals produced by artificial inoculation with six fungal isolates from the same population. Initial attempts to follow the fate of these isolates and to examine whether there is

TABLE 18.1
Components of a coevolutionary model of the *Silene-Ustilago* system that incorporates both plant and fungal genetic variation

Genotypes

X_1 Host genotype 1
X_2 Host genotype 2
Y_{11} Host genotype 1, fungal genotype 1
Y_{12} Host genotype 1, fungal genotype 2
Y_{21} Host genotype 2, fungal genotype 1
Y_{22} Host genotype 2, fungal genotype 2

Transmission coefficients

	Host 1		Host 2	
	Fungus 1	Fungus 2	Fungus 1	Fungus 2
Host 1	β1.11	β1.12	β1.21	β1.22
Host 2	β2.11	β2.12	β2.21	β2.22

Recursion Equations

$$X_1 = X_1\,[1 + r_1 - \frac{1}{N}(\beta 1.11 Y_{11} + \beta 1.12 Y_{12} + \beta 1.21 Y_{21} + \beta 1.22 Y_{22})]$$

$$Y_{11} = \frac{X_1}{N}(\beta 1.11\, Y_{11} + \beta 1.21\, Y_{21}) - d_{11}\, Y_{11}$$

Note: X_i represents numbers of healthy individuals of genotype i, Y_{ij} represents numbers of ith genotype of host diseased with jth fungal genotype. *Beta h.ij* represents the transmission rate of the fungus to the hth healthy genotype from the ith host genotype diseased by the jth pathogen. Recursions are shown only for X_1 and Y_{11}, but are analogous for other healthy and diseased genotypes.

genetic variation in fungal virulence have not been successful because of the low level of electrophoretic variation in the population. Essentially all the populations in this region of Virginia show very little among- or within-population variation for a large number of electrophoretic markers (Stratton 1990, personal communication). We therefore at present do not know the degree of genetic variation in virulence of the pathogen; it is plausible that during the introduction of the pathogen into the United States the populations went through a severe bottleneck effect and now show relatively little variation not only electrophoretically but also with regard to genes for virulence.

The models developed for numerical dynamics could be extended to incorporate genetic variation in resistance and susceptibility and therefore to reflect the entirety of the coevolutionary diagram presented at the outset of this paper. The problem is that such a formulation is heavily overparameterized: the minimal assumption of only two host and two pathogen genotypes results in six types of individuals and eight different transmission coefficients describing disease spread from diseased to healthy individuals (table 18.1).

In our analyses to date we have made the simplifying assumption that there is no genetic variation in the fungus and that diseased plant genotypes do not

show differential transmission (e.g., as a result of different pollinator visitation rates). In this way we are essentially examining the dynamics of the system assuming that the only genetic variation is in the resistance/susceptibility of the host. The model therefore still represents only half of the coevolutionary diagram (fig. 18.1C). We also assume that there are only two genotypes of the host (i.e., that the individuals are haploid); this is justified by the fact that the results of single-locus haploid models approximate those of diploid models if heterozygotes are intermediate.

To include some of the essential biological features, we have included plant genotypes with extremes of resistance and susceptibility, yet with costs to these resistances that reflect the results of the experimental studies described earlier (Alexander 1989). Thus in the simulations presented here, we have assumed widely divergent resistances, expressed by transmission coefficients of 1.0 (low resistance) and 0.1 (high resistance) for the two host genotypes, and costs that reflect the approximately twofold difference in flower production between the most resistant and the most sensitive genotypes in the experimental studies. In combination with the assumed birth and death rates, populations monomorphic for each of these genotypes would result in either population extinction for the low-resistance genotype, or failure of the disease to establish or persist for the high-resistance genotype. The results (fig. 18.6) show however that in polymorphic populations both genotypes will coexist, with an oscillatory approach to equilibrium. This shows that extremes of resistance and susceptibility can be maintained within one population and that given such variation the regions for host-pathogen coexistence are increased substantially. All our models so far have been deterministic: given the large fluctuations in population size, pathogen extinction in nature may occur owing to effects in small populations.

The costs of resistance however occur only in males, and we can modify the mating scheme in the model to reflect this. We assume that female success is unaltered (i.e., seed production of resistant and susceptible plants does not differ), but that susceptible genotypes are much more successful as males. This introduces frequency-dependent host "fitnesses" into the model: high-fecundity males are at a disproportionate advantage when they are rare, but at a disadvantage when they are common. But when they are common, disease spread will be more rapid. The resulting population dynamics show even more severe oscillations, an apparent stable limit cycle, and maintenance of polymorphism in resistance (fig. 18.7). It is important to note that if we were measuring costs solely in terms of seed production of females, then the costs would go unnoticed and we would be at a loss to explain the dynamics.

These results therefore provide insight into the ecological genetics, not only of this particular disease, but into venereal diseases in general. We can draw several important conclusions.

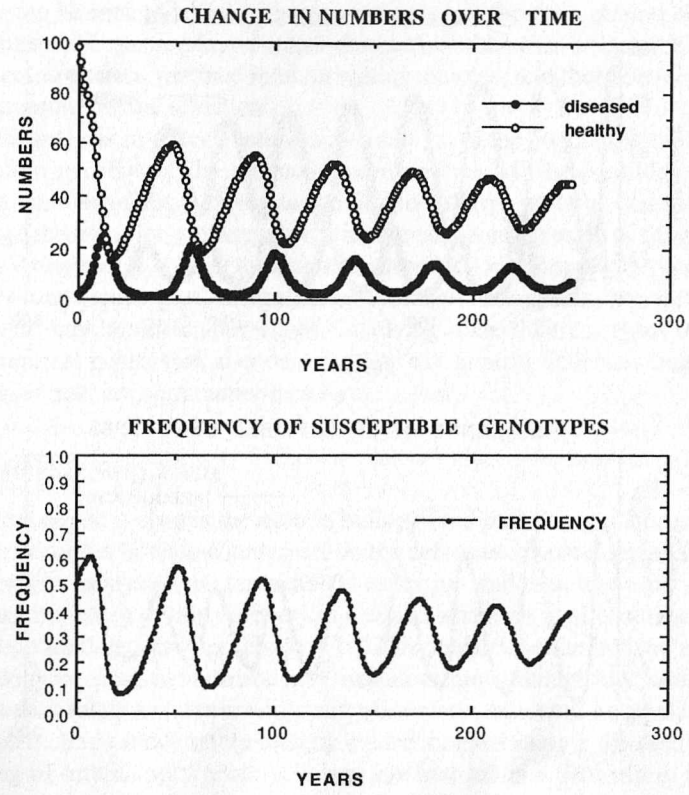


Fig. 18.6 Numerical and gene frequency dynamics of the *Silene-Ustilago* system assuming resistance costs are in terms of female fecundity. Model is as described in figure 18.5 and table 18.1, assuming density-dependent establishment and no genetic variation in the pathogen. Susceptible genotype: $beta = 1.0$, $b = 0.9$. Resistant genotype: $beta = 0.1$, $b = 0.6$; $k = 0.02$, $d = 0.3$.

1. Venereal disease dynamics can be stabilized by differential density effects on diseased and healthy plants.
2. Such effects are likely in the case of venereal diseases because their transmission is at the adult stage, so that the diseased class is likely to experience less density-dependent mortality than the healthy class which passes through a juvenile stage.
3. Resistance to venereal diseases is likely to have a direct cost whenever such greater resistance is achieved through reduced mating.
4. Variation in resistance is likely to be greater in the sex with the greatest variation in mating success; this will usually be males.

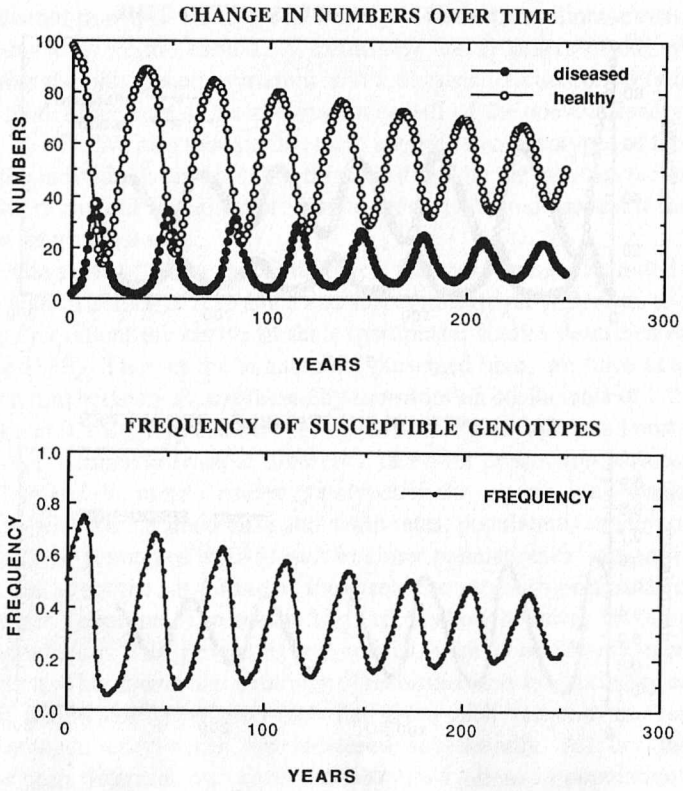


Fig. 18.7 Numerical and gene frequency dynamics of the *Silene-Ustilago* system assuming resistance costs are in terms of male fecundity. Model is as in fig. 18.6, except that relative male fitnesses are 1 and 0.667 for the susceptible and resistant genotypes. Recursions are modified to include differential male mating success.

5. These costs can permit the maintenance of large extremes of resistance and susceptibility in one population, and extend the regions for coexistence of host and pathogen.
6. Genetically variable hosts can result from the impact of genetically uniform pathogens. One does not necessarily need to invoke a coevolutionary "arms-race" to explain host-genetic variation (see also Parker, this volume).

I have focused on our study to illustrate how knowledge of plant resistance can be used to develop dynamic models that increase our understanding of the nuts and bolts of species interactions. Things that were extremely puzzling, such as the presence of highly susceptible and highly resistant individuals in the same population, no longer seem so. Conversely, such interactions immediately can be seen to have important repercussions for more general biological phenomena. For example, venereal diseases could be an important force in maintaining genetic variance in male mating success, and therefore in promoting continuing sexual selection.

The analysis of species interactions in terms of the population ecology and population genetics of the interacting components will be possible only in a limited number of cases. Even in the *Silene-Ustilago* system, it is difficult to envisage the behavior of the system with the added complexity of fungal variation in virulence. It is clear that while such reductionist approaches provide an important methodological bridge from population biology to community genetics, there may be other approaches, involving quite different types of theory and empirical goals, that also contribute to our general understanding of how genetics influences community structure.

The Holistic Approach

The reductionist is always the snob in biology (and probably in other sciences), putting his or her holistic counterpart on the defensive by proclaiming to have a more precise analysis, greater technical expertise, and therefore a surer handle on causality. Much of this snobbery is justified whenever the holist takes refuge in unsupported generalities. Harper (1982) has criticized the holistic approach to ecology because it is didactically unsound, methodologically weak (often being based solely on description), and motivated more often by emotional and intuitive feelings about nature than by evidence. For example, he describes the teaching of introductory ecology, where the first lab is a field trip to a woodland, as being equivalent to starting a chemistry class by showing them the structure of DNA. And he assails the blind belief that communities are "integrated and harmonious" because it leads to an answer that is always "safe and ignorant." However were it not for some virtues in holistic approaches, we would still all be paralyzed by physics envy, searching for individual quarks and electrons in a futile quest for biological understanding rather than discovering biologically based laws and generalities. What is critical is that higher levels of analysis have rigorously formulated questions, and that these be empirically tractable so that holistic approaches do not become a refuge for crypticism, mysticism, and evasiveness. It is also essential that holistic approaches do not violate ideas and generalizations attained by more reductionist levels. In technical terms (Rosenberg 1978) laws at higher levels should "supervene on" those at lower levels, but otherwise they can have a valid autonomy of their own.

Within the area of community genetics, there are numerous questions that do not require a case-by-case, species-by-species, year-by-year approach. Perhaps the most important of these is, What is the relationship between genetic diversity and species diversity? It is a question that can be asked within trophic

levels, among trophic levels, or within particular guilds or subsets of communities. For example, we simply do not know whether the abundant demonstration of ecotypic differentiation in temperate regions is a function of the greater abundance of scientists there (the Swedish tradition!) or whether intraspecific genetic variation in some way compensates for the low level of species diversity within these regions. The corresponding prediction might be a lower level of genetic diversity within species from the tropics. Alternatively, given that genetic and ecological diversity can be maintained by similar forces (Antonovics 1976a), one might argue that there should be a positive correlation between genetic diversity and species diversity of a community. I know of very few attempts to examine this question even descriptively (an exception is presented in figure 18.8).

Obviously, this larger question of the relationship between species diversity and genetic diversity is overgeneralized and should be broken down by considering subsets of genetic diversity and subsets of community components. For example, we can ask how the diversity of resistance genes relates to the presence or absence of particular pathogens. While numerous studies bearing on this issue have been done, the answers remain equivocal largely because information addressing his issue has often been obtained indirectly as part of studies with goals other than direct assessment of community genetic structure.

Perhaps it is feasible to address other, less grandiose questions. For example, there is currently an intense debate raging about the possible role of pests and pathogens in the evolutionary maintenance and (perhaps) origin of outbreeding and sexual reproduction (Hamilton 1980). And greater outbreeding and less parthenogenesis (hence a greater potential for generating genetic variation) have been correlated with greater pathogen and pest pressure (Levin 1975; Glesener and Tilman 1978). Such observations are certainly consistent with many micromodels of biotic interactions generating and preserving variation by negative-frequency dependent selection or genetic feedback.

It is important to emphasize that holistic approaches do not preclude experimental analysis, nor is it intrinsically easier or harder to carry out experiments at different levels of analysis. Certainly there has been the lack of an experimental tradition in much of plant community ecology, but this is rapidly changing. A vast array of experiments are possible. The role of genetic variance in species abundance can be addressed by manipulating genetic variance of species in experimental communities. For example, biological control measures have generated many natural experiments, and these have shown that more outcrossing, presumably more genetically variable, species are more recalcitrant to biological control measures (Burdon and Marshall 1981a). And we have artificially manipulated the genetic variance among arrays of individuals of *Anthoxanthum odoratum* in a seminatural grassland and shown that genetic variation is a critical component of demographic success (Antonovics and Ellstrand 1984; Ellstrand and Antonovics 1985; Antonovics and Ellstrand 1985; Schmitt and Antonovics 1986; Kelley et al. 1988). In these studies, circumstantial evidence strongly implicated the role of pests or pathogens.

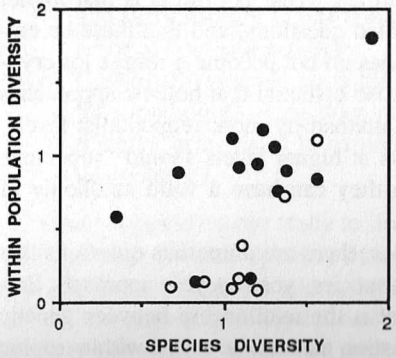


Fig. 18.8 Relationship between seed type diversity and weed species diversity among different populations of *Oryza sativa* (open circles) and *O. glaberrima* (closed circles) from rice fields in Africa. Seed types were identified on the basis of five polymorphic traits, and weed diversity was measured on the estimated biomass of 26 species. Diversity, *H*, is calculated using the Shannon-Weaver diversity index. From Morishima and Oka 1979.

A major impediment to the empirical understanding of coevolutionary systems is the recognition of genetic variants. This problem occurs at two levels. The first, and easiest to resolve in the future, is the ability to follow the fate of particular individuals and their progeny in natural and experimental populations. This requires the development of easy and reliable techniques for assessing genetic identity and statistical methods for inferring parentage (Meagher 1986). The need is to develop better and better DNAometers, whether through the use of electrophoretic variants, restriction-fragment-length polymorphisms, or eventually more direct analysis of sequence information. The lack of well-developed procedures in this regard is one of the major hurdles in the development of community genetics. The second, more difficult, problem is identifying the genetic basis of particular phenotypic variants. If the genotypes underlying these variants can be identified (e.g., identification of resistance genes by RFLP markers), then direct assessment of genotypic change is far easier. At present this hardly seems feasible for most systems, but may be an important source of interaction between the agricultural scientist and the field biologist: time and effort devoted to identifying specific loci may be worth the effort from an applied viewpoint and, given the availability of appropriate probes, these same loci could be studied in related wild populations. As it is, resolving the genetic versus environmental basis for intraspecific variation will continue to require classical experimental methods involving transplants and crosses, which are time-consuming and often remove populations from their natural context.

The other major problem confronting the discipline, and one that I hope became apparent in the previous section, is that given a set of interacting factors and processes, it is almost impossible to understand their joint dynamics by superficial intuition or by casual observation. It is necessary to use theoretical (computer or analytical) models to predict the consequences of multiple nonlinear interactions. It is necessary to do experiments to isolate the nature of the

particular interactions that might be involved. And finally it is necessary to calibrate the predicted outcomes against long-term studies of relative abundances of the interacting components. For no plant-based system (except perhaps in a few cases of biological control and major species introductions) do we have even the crudest long-term data on disease and host abundance.

All these problems are confounded by a too ready acceptance of ecology and evolution as sciences that can be done on the cheap. A thorough analysis of ecological and evolutionary dynamics is an extremely expensive enterprise, requiring laboratories to identify genetic markers and to rear organisms; requiring garden and growth chamber facilities for crosses and experiments; requiring computer facilities for modeling; and requiring time and personnel to handle the extensive, thorough analysis of field processes. Just as a molecular biology lab should perhaps not be without its high-speed centrifuge, its DNA synthesizer, and its attendant culture collections, so a community genetics lab should not be without is gene-detection equipment, its growth chambers, its computers, and its accessible field sites. Perhaps practicing ecologists realize these issues, but they need to be far more impatient and insistent in imparting the lesson to administrators and politicians who hold the purse strings, and who are only too willing to imagine that all an ecologist needs is a shovel, boots, and binoculars as if, by analogy, a modern molecular biologist could solve major issues in that science using a pestle and mortar, a bit of toluene, and some chromatography paper.

Conclusion

There is enormous richness and opportunity in the discipline of community genetics, the study of the genetics of species interactions and their ecological and evolutionary consequences. This opportunity presents itself at levels ranging from issues that form the focus of the present volume, namely understanding intraspecific variation in plant resistance, to well beyond. It extends to a detailed analysis of the numerical and genetic dynamics of particular systems, as outlined here for the *Silene-Ustilago* systems, and to answering quite general questions about the relationship between trophic structure and genetic structure. The reductionist aspects of the discipline have goals and methodologies almost identical to those of the agriculturalist concerned with pest-induced yield reduction. The holistic aspects also have applied repercussions: knowledge of the relationship between species diversity, abundance, and genetic diversity will provide a backdrop against which crop deployment, gene conservation, and species conservation strategies can be rationally developed. Both reductionist and holistic components will demand a combination of theoretical, experimental, and observational analysis to resolve the processes in such richly interacting systems, and to replace what is often now only a mythology by real, tempered understanding. Much of the development of the field will depend on

advances in genetic analysis and monitoring, on improved DNAometers. In general, it will be a far more expensive enterprise than either pure ecology or pure genetics. The obvious but impressive message of this volume is that genetic variation at the intraspecific level can be critically important to ecological processes. But equally this volume impresses upon us the fact that our knowledge is at best fragmentary. We need to know far more about the properties of intraspecific variation in resistance, the degree to which such resistance variation is related to the presence of herbivores and pathogens, the kinds of ecological and evolutionary dynamics that this generates (or is a product of), the degree to which this affects species distributions and abundances, and the degree to which this has consequences for major evolutionary processes such as the evolution of breeding systems and speciation. These issues will form the agenda for community genetics well into the next century.

Acknowledgments

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