Chapter 7

The Concept of Fitness in Plant-Fungal Pathogen Systems

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INTRODUCTION

In plant pathology, the concept of "fitness" has played a central role in debate about such issues as the effect of host resistance on the genetic composition of pathogen populations (Leonard and Czochor, 1980; Browning, 1980, 1981; Parlevliet, 1981), the potential loss of virulence genes in the absence of resistant varieties ("stabilising selection" sensu Van der Plank, 1963; Nelson, 1979; Leonard and Czochor, 1980; Mundt and Browning, 1985), and the loss of generalized resistance in plants bred for resistance to specific pathogen types ("the Vertifolia effect"; Harlan, 1976). Nevertheless, close examination of the use of the term "fitness" by different plant pathologists reveals that the concept is used not only in a wide variety of ways, but often inconsistently with its usage in population biology, from which it was borrowed. This confusion is more than just a semantic problem. Different interpretations of the concept of fitness affects both the way we predict interactions between plants and pathogens as well as the strategies we adopt for disease control.

It is the purpose of this chapter to explore and clarify differences in the usage

of this concept between the disciplines of plant pathology and population biology. We also illustrate the application of the concept of fitness by describing approaches that have been used to study the ecology and evolution of natural or seminatural plant-pathogen systems.

THE INDIVIDUAL VS. THE POPULATION

Population biology, defined in the broad sense of including both ecological and genetic approaches to populations, has developed largely independently of plant pathology. This independence of the two fields has resulted not from the lack of a sphere of common interest, but from a difference in their respective goals. Plant pathology has encompassed largely applied goals, being concerned with crop losses due to pathogens in agricultural populations. Population biology has been concerned with understanding the causes of changes in numerical abundance and gene frequency in natural populations. These differing goals have resulted in divergent methodologies and divergent terminology. The agricultural scientist is mostly concerned with the overall performance of a *group* of individuals, the crop of plants grown in a field, and thus its total yield (be it biological or economic yield). The population biologist, by contrast, is concerned with the performance of the *individual*; particularly its performance relative to other individuals in the population.

The difference between a focus on the individual and on the group (or population) is not a trivial one, especially when one is discussing evolutionary processes. Theoretically, evolutionary change can result from differential performance of any kind of distinct "units," whether such "units" are groups of organisms, individual organisms, organelles, or even genes themselves (Brandon and Burian, 1984). Natural selection is a process that results from these "units" differing in their properties (in their "fitness") such that some leave more descendants than others. Normally when we speak of natural selection in an unqualified way we refer to the process that involves differential performance of individuals (i.e., "individual fitness"). We do this for reasons that are historical (this was the context in which the idea of natural selection was first formulated by Darwin), for reasons of convenience and practicality (individuals are clearly definable entities whose survival and reproduction can be measured), and because in the absence of counterevidence, individual selection is seen as being the most likely, although not necessarily the only, process responsible for genetic change in natural populations. Group selection, in contrast, implies that genetic change results from certain groups of individuals (populations, demes) having greater persistence and leaving more descendants than other groups (Wilson, 1983). Group selection is seen as more complex, perhaps less likely, and is certainly more difficult to study. When the terms "selection" and "differential fitness" are used without qualification by evolutionary biologists, emphasis on the individual level is assumed.

This distinction between individual and group selection is important because the two processes may have different outcomes and different time scales and

dynamics. For example, individual selection may result in traits that not only increase the individual's performance but that also decrease the performance of its neighbors. Thus, taller plants not only receive more sunlight in a dense canopy but also shade neighboring plants. Short stature on the other hand, while detrimental to a specific individual, may permit a group of such individuals to devote more resources to reproduction and so increase group performance (= yield). With regard to time scales and dynamics, group selection occurs by differential extinction and proliferation of groups of individuals; the extinction and multiplication rates of such groups are likely to occur on quite a different temporal and spatial scale from mortality and reproduction of individuals.

The distinction between selection acting on the individual and group levels is often lost in discussions of pathogen fitness. One evolutionary focus in plant pathology has been on the idea that obligate pathogens should not become too virulent lest they eliminate their host plants and thus lead to their own demise (e.g., Knott, 1972; Nelson, 1979; Browning, 1980, 1981; Parlevliet, 1981). The likelihood of such a scenario taking place depends on whether the process of selection is operating on an "individual" or "group" level. Within a pathogen population, individuals that are most fit, in the sense of leaving the greatest contribution to the next generation, are likely to produce the greatest number of propagules, and will therefore have relatively high levels of virulence (in the sense of causing severe disease). Natural selection acting on the individual level will thus favor virulence, but only to the point where virulence does not take on such an extreme form that the pathogen kills the individual host plant or reduces its vigor so that the pathogen's own longevity and hence fitness is reduced. In contrast, the scenario where selection against virulence occurs because virulent pathogens reduce host population size (and therefore themselves) depends on selection operating on the "group" level. In this case, variation in virulence exists *among* different populations of pathogens (due perhaps to differing frequencies of virulent individuals *within* those populations). If certain pathogen populations contain so many virulent individuals that they reduce the size of their host populations, selection could then operate *among* populations at the "group selection" level to favor populations of pathogens that have a less severe impact on their hosts. Both modes of selection could, therefore, lead to the same result (pathogens having intermediate virulence).

Data are lacking on the frequency with which pathogens eradicate their host populations. Large-scale pandemics, such as chestnut blight or Dutch elm disease, strongly suggest this possibility. Moreover, such effects are often caused by introduced pathogens, rather than endemic ones. Therefore, the inference is often made that coevolved host-pathogen systems show reduced virulence as a result of (group) selection. In spite of such circumstantial reasoning, population biologists generally believe that the mechanism of individual selection is both more probable and more important than group selection, even though mechanisms of group selection have been shown to be feasible (Gilpin, 1975; Levin and Kilmer, 1975; Wilson, 1983). The reasons for this are several.

First, there has to be some mechanism for the generation of group differences.

Genetic drift is often invoked as a potential source of among-group differences; hence, the process becomes restricted to small populations or populations with only a few founders. Second, populations have to be distinct such that gene flow between them does not eradicate any difference that becomes established. Third, group extinction and reestablishment have to be frequent; if the "generation time" of a group is too long, processes occurring within the group (determined by the generation time of individuals) will predominate over those occurring among groups. Most population biologists consider that these conditions are unlikely to be generally present in natural populations. These conditions seem particularly improbable for many pathogens such as the rusts with their large populations and presumably widely dispersed spores.

Thus, although pathogens may have evolved reduced virulence by elimination of host populations due to group selection (this possibility has been explicitly argued in theoretical models of predator–prey systems by Gilpin, 1975), there is a whole suite of alternative explanations that are consistent with individual selection. For example, increased virulence of an individual may result in reduced residence time on its host and therefore decreased spore production. Increased virulence may also reduce the probability of disease transmission if disease vectors have a reduced probability of finding the host (e.g., if the host is very short lived). Further, virulence is rarely universal, but instead is specific to genotype, developmental stage, or physiological condition; thus, increased virulence on one host phenotype may result in decreased performance on alternative phenotypes or alternative hosts (see Pathogen Fitness below). Last, one must not forget that host populations in nature are themselves evolving to reduce the detrimental effect of the pathogen.

The population biologist is therefore confused and uncomfortable when plant pathologists write as in the following examples:

> The most spectacular *coup de grace* ever accomplished by parasitic fungi throughout their long evolution was their ability to coexist with their hosts in genetic equilibrium. It was fitness personified. In an atmosphere of relaxed selection pressure both parasite and host had learned that coexistence was preferable to the alternating thrill of victory and the agonies of defeat. No longer would the improved fitness of one result in the diminished fitness of the other. (Nelson, 1979)

> Obviously, the most *fit* pathogen cannot necessarily be the one that sporulates best as, in the long term, that would tend to destroy the host and minimize the probability of either pathogen or host leaving descendants. (Browning, 1980)

> Because the host plant competes with other plants, a slight reduction in its fitness, and thus its competitive ability, could result in a serious decline of the host population. A biotrophic pathogen does influence the fitness of its host population considerably, and maximizing its pathogenicity could endanger its host and thus itself. (Parlevliet, 1981)

The above statements strongly imply that selection acts by differential performance of populations (i.e., group selection) and that fitness is a population attribute. If such statements were applied to particular pathogen systems in which group selection processes had been characterized and documented, they would not in themselves be incorrect. But when presented as generalized remarks of how selection occurs, they clearly reflect acceptance of group selection as a likely process. A population biologist intuitively thinks of individual selection and individual performance; a crop scientist is intuitively concerned with group properties and group performance.

The definition of an "individual" is critical in measures of pathogen fitness because of the prevalence of asexual reproduction in many pathogen populations. These same problems exist in plant populations. Vegetatively reproducing plant populations will consist of physiologically separate individuals or "ramets," which can be grouped into "genets" each consisting of the ramets from a common zygotic origin. (The term "genet" is used instead of "genotype" because independently originating zygotes could have identical genotypes.)

Individual phenotypes and genotypes in microbial populations can be identified by using such techniques as single spore isolations, screening tests to identify virulence traits, or electrophoretic markers. The fact that an individual is not an easily visualized entity for many pathogens is probably another reason for the confusion between fitness on the individual and group level, discussed earlier. A practical solution, at least for some fungal pathogens, is to first use single spore isolations to define an "individual." Subsequent measurements of fitness for that individual, for example, measures of infection efficiency, will involve inoculation procedures using millions of spores asexually derived from the originally isolated spore. Such a test can be considered a measure of *individual* fitness, however, since the "population" of spores used are replicate samples of the originally isolated spore. This measure of individual fitness can be contrasted with a measure of, say, infection efficiency using a bulked collection of spores of mixed genetic origin. In higher plant populations too, it is commonplace to measure "individual" fitness of a phenotype by measuring the average fitness of a set of individuals sharing the same phenotype. This is done because neither accurate measures nor statistical estimation is possible if only one individual is used.

Clearly, such "surrogate" measures of individual fitness have their drawbacks. One must be careful that the individuals being used for fitness estimation do not interact in some unforeseen way; for example, genetic similarity among plants grown in proximity may affect disease incidence (see Schmitt and Antonovics, 1986). And there are theoretical and statistical problems involved in estimating fitness from sums of individual birth and death schedules (Lenski and Service, 1982).

Fungi, like higher plants, alternate haploid and diploid generations. In plants, separate accounting of the fate of the haploid phase (usually pollen) prior to fertilization is sometimes useful (e.g., for incompatibility systems) or is itself an object of the study of differential fitness (gametophytic selection; see Mulcahy, 1975; Mulcahy et al., 1986). The complex nature of the life cycle of many plant pathogens requires that either the complete life cycle be included in any research on fitness or that the predictive aspect of any study is confined to one stage. Indeed,

many epidemiological models focus only on within-season spread of pathogens propagating by asexual means over successive "generations." In this context fitness estimates would be of each asexual generation. For longer term population and evolutionary models the inclusion of all life cycle phases would be essential.

THE CONCEPT OF FITNESS IN POPULATION BIOLOGY

Definitions of Fitness

What then is the concept of "fitness" to a population biologist, and how can it be operationalized? In the context of individual selection, fitness is most simply defined as the expected contribution of a phenotype to the subsequent generation (Roughgarden, 1979; Endler, 1986; Brandon, 1978). We refer to the *expected* contribution of an individual, because we want to exclude cases where differential contribution to the next generation is by chance alone (e.g., genetic drift) and not an actual consequence of the phenotype of the individual. Otherwise, what is fittest is always that which contributes the most and the concept of selection becomes a tautology.

We also refer to fitness of a phenotype and not of a genotype. It has perhaps been more common to define fitness in terms of the contribution of genotypes rather than phenotypes (Dobzhansky, 1970; Crow and Kimura, 1970), and this is usually done in single-locus models of selection. Here it is clear that certain genotypes cause particular phenotypes. When we are dealing with quantitative traits (i.e., traits determined by many loci), it is more useful to define fitness in terms of phenotypes because in such situations genotypes are not easily characterized, and moreover they are "ephemeral," being broken down and reformed by recombination. If very many loci are involved, a particular genotype may have a very low probability of ever recurring again in a finite population! When single loci are involved, genotypes are equatable with phenotypes and their "reconstruction" each generation is readily predictable from Mendelian principles. Moreover, a major goal of evolutionary biology is to explain the frequency of phenotypes in a population (e.g., disease-resistant and susceptible plants).

Perhaps the most compelling reason for the measurement of fitness of phenotypes is that it facilitates the "accounting process" involved in predicting evolutionary change of a particular trait. This is because such change is the result of two rather distinct processes: differential fitness of phenotypes and the genetic transmission of the trait from one generation to the next. This probability of transmission is deduced from Mendelian behavior for single gene traits or from general measures such as heritability for polygenic traits. By examining fitness at the level of the phenotype, we distinguish it from the genetic process of transmission, and each can thereby be measured and understood separately. This is *not* a logical necessity but a matter of usage and convenience. Thus, we could speak of the fitness of an allele, a quantity that would measure the change in the number of copies of that allele from one generation to the next. However, such a fitness measure will then be a compound of the transmission properties of the gene plus the effects it has on the phenotype.

This approach is at times very useful, as for example, when dealing with genes that produce phenotypes that affect the transmission process itself. It has also been conceptually "catchy" in the sense of "selfish genes," the idea that organisms are simply vehicles whereby genes increase their own transmission (Dawkins, 1976). However, ascribing fitness values to particular alleles that incorporate their joint impact on survival and reproduction as well as on transmission, while of some theoretical interest, has been of less value for standard analyses of evolutionary change than formulations that consider fitness and transmission of genotypes or phenotypes as separate processes.

The most simple operational definition of the fitness of a given phenotype of a diploid organism is the average (or expected) number of zygotes that a single zygote with that phenotype contributes to the next generation. (The probability that the zygotes in the next generation actually display that phenotype is then dependent on the transmission probability of the trait.) An equivalent definition for phenotypes measured in the haploid phase would be in terms of haploid (spore, gamete) rather than zygotic contribution. The above definition describes measurement of fitness on an "absolute" scale. In the case of "relative fitness," this is the number contributed relative to other individuals in the population, with the greatest contribution often being set to 1.

It should be recognized that "fitness" is used in population biology in a short-term sense and that it is measured on time scales of one to a few generations. Various authors have proposed explicit conceptualizations of "long-term fitness," i.e., fitness measured in terms of genetic contribution after hundreds, even millions of years (Thoday, 1953; Cooper, 1984). However, there is little agreement on what definition is most appropriate, and little likelihood that any of the proposed measures could actually be made in real-world situations (Endler, 1986). Therefore, when long-term fitness is invoked as some ultimate measure, population biologists are left somewhat in despair. The only legitimate sense in which the term could be operationalized is perhaps if "long-term fitness" is equated with fitness of a particular recognizable group, and processes are analyzed both at the level of the individual and of the group. However, except in family-structured populations and some highly compartmentalized host-parasite systems (e.g., fig wasps, flower mites), measurement of group fitness has proved extremely difficult. Such processes are deserving of study in plant-pathogen systems, but to date no one has done so sufficiently to draw any conclusions about "long-term" fitness of particular groups.

Nelson (1979), Browning (1981), and others have considered spore production to be primarily a measure of "short-term" fitness and therefore in some sense an incomplete measure of fitness because of the argument that pathogens with high reproductive rates will destroy their host. This interpretation is not helpful. First, there is no practical way in which "long-term fitness" can be defined operationally. Second, such an interpretation can readily confuse individual selection (with one

pathogen genotype eliminating its host and therefore reducing its own longevity) with group selection (a group of genotypes causing such heavy infection as to lead to elimination of all hosts and thus the pathogen population).

Measurements of Fitness and Its Interpretation

Biologists have used two general methods for measuring fitness. One method is based on single-generation measures of expected contribution of an individual. In this "predicted fitness" method, the contribution of an individual (or of a phenotypic or genotypic class) is estimated by measuring traits ("fitness components") that are likely to directly influence such a contribution. The other method is based on multigeneration measures of changes in phenotype frequency, gene frequency, or (in the case of pathogen lines) changes in strain frequency. In this "realized fitness" method, the observed change is translated into a single generation fitness difference by assuming the outcome is the result of a particular (usually constant) process of selection and of transmission in each generation. The use of "realized fitness" estimates is clearly more feasible in situations where the organism has a short life cycle, as in the case of many fungi, and conversely is less applicable to longer-lived plants.

The distinction between these two methods breaks down if phenotypes affect each other's fitness within and across generations. An obvious case is when parental infection results directly or indirectly in disease transmission to seed or seedling. Less obvious but more universal are the cases where the level of maternal provisioning of the seed affects its fitness or where progeny interact with each other. No longer is a simple single-generation zygote-to-zygote measurement sufficient. Instead, we need measurements that span two (or more) generations and that therefore have to incorporate transmission properties in the form of covariances among relatives. This is the complex subject of kin selection, which we do not have space to address (for recent discussions of methodology see Michod and Anderson, 1979; Uyenoyama and Feldman, 1981). Kin selection can be viewed as a special subset of group selection, with the groups now being composed of interacting related individuals (Wilson, 1983).

"Predicted Fitness" Methodology The factors that determine an organism's contribution to a subsequent generation are complex and involve many aspects of the life cycle such as survival, mating, gamete transmission, gamete fusion, embryo maturation, birth, and dispersal. For these reasons population biologists often invoke the idea of "fitness components" as a way of acknowledging explicitly that measurement of all the component processes is often not possible. The use of data on only a subset of the life cycle is valid if the individuals of interest do not differ in other unmeasured components. This is often reasonable biologically or can be confirmed by subexperiments. For example, a component that is usually not measured in higher plants is pollen tube growth rate; the measurement of this component is considerably more difficult than measuring, say, total seed

output. This omission may be reasonable if we are dealing with traits such as disease resistance or susceptibility because it is not expected that resistance genes would in and of themselves affect pollen tube growth rates. However, it would clearly be folly to ignore these fitness components when dealing with incompatability loci.

When considering plants, there is a direct analogy between "fitness components" and "yield components" (Primack and Antonovics, 1982). A "major" fitness component is a trait that is expected to be directly responsible for determining fitness. Thus, a crop scientist will speak of corn yield as being determined by weight per kernel, number of kernels per ear, and the number of ears per plant. Similarly, a plant population biologist would view seed size, number of seeds per capsule, and number of capsules per plant as important fitness components. But there are limits to the yield-fitness analogy. Thus, while the crop scientist might include number of plants per unit area as a yield component, such a measure is a group attribute and usually not a valid fitness component to the population biologist.

The simplest measure of fitness is the net reproductive rate (or lifetime fecundity):

$$R = \sum l_x m_x \tag{1}$$

where l_x = probability of survival to age x, and m_x = number of offspring produced by an individual of age x. For this to be a reasonably valid measure of fitness we have to make two major assumptions: the different phenotypes contribute equally as males and females to zygote formation, and generations do not overlap with phenotypes differing in their age-specific survival and reproduction.

Particular males or females may contribute differentially to zygote formation (this has been termed "sexual selection"). A simple female-based accounting of zygote fates has the danger of ignoring the likelihood that there may have been differential contribution of particular phenotypes to the zygotes via male function. Recently, a number of authors have emphasized the importance of considering both male and female functions in studies of plant fitness (Willson, 1983; Lloyd, 1984). The male pollen contribution is difficult to measure but this can be done by using electrophoretic markers (Meagher, 1982). For rust fungi, an analogous problem would be to determine whether strains differ in the rates at which spermatia fertilize pycnia on other strains rather than the simpler female-based measure of subsequent urediospore production. Clearly, these difficulties don't arise in fitness estimates of genotypes in purely asexual populations.

In most natural populations, generations overlap and are not discrete as they are in annual crops. This introduces complexity into the accounting of zygote fates; populations consist of individuals born at different times and whose survival and reproduction may vary with age (see Charlesworth, 1980). In such situations, it is necessary to take into account not only the absolute number of zygotes produced, but also the timing of their production; all else being equal, earlier reproduction will be favored because zygotes produced earlier will themselves produce zygotes

sooner. In these situations, fitness of the *ij*th genotype (or phenotype) can be estimated by (Charlesworth, 1980)

$$W_{ij} = \sum l_{ij}(x)\, m_{ij}(x)\, e^{-\hat{r}x} \qquad (2)$$

where x = age, $\hat{r}$ = growth rate of the population as a whole, and $l(x)$, $m(x)$ = age-specific survival and fecundity, respectively. If the population is increasing ($r > 0$), later age classes are discounted more than earlier age classes, thus leading to a higher fitness for those genotypes reproducing earlier, or to a greater extent in the early age classes.

This formulation is in itself an oversimplification, making the assumption that populations are in stable age-class distribution, and that the genotypes (if they interbreed) mate at random, show no sex differences or sex-ratio bias, and that fecundity is determined solely by the age and genotype of the female. For cases where these assumptions do not hold, more complex formulations are needed (Charlesworth, 1980).

These principles, while they have been developed in relation to higher plants and animals, also apply to fungal pathogens. For a fungal pathogen, the probability of inoculum or spores reaching a host plant is an important initial fitness component. Its value will depend on the spore dispersal distance, something that is influenced by the pathogen's effect on host morphology, position of lesions, and time of spore release. It will also depend on the density and relative distribution pattern of diseased and healthy plants. Following spore dispersal, the "infection efficiency" (the number of successful infections resulting from a known amount of inoculm) is the most important fitness component. As pointed out by Nelson (1979), this in itself is a composite trait involving survival of the pathogen at successive stages from spore germination through production of a functional lesion. Once the pathogen is established, inoculum production as a measure of pathogen reproduction is a subsequent fitness component.

Tooley et al. (1986) measured the fitness of *Phytophthora infestans* isolates from sexual and asexual populations by using the fitness components infection frequency (proportion of leaflets on which lesions developed), lesion area (per leaflet), and sporulation capacity (number of sporangia per unit area of lesion). The product of these three quantities gave a "composite fitness index" in terms of total sporangia produced per inoculum. Sexual and asexual populations differed in the contribution of the different fitness components, with sexuals having a higher infection frequency but a smaller lesion area, even though they did not differ in overall fitness. In the sexual population, fitness components tended to be negatively correlated with the number of virulence factors, although this trend was not significant. Other analyses of fitness components in pathogen populations have been carried out by Oard and Simons (1983) and Prakash and Heather (1986). Similar studies have been done in populations of *Drosophila* (Prout, 1971; Haymer and Hartl, 1983), plants (McGraw and Antonovics, 1983; Clegg et al., 1978), and vertebrates (Arnold and Wade, 1984b).

The analysis of fitness in terms of components is often an intermediate step in

estimating overall fitness. However, it is also of value in a number of other ways. It permits an evaluation of how each component contributes to total fitness, especially if the latter can be estimated or approximated by some measure that is independent of the component measures themselves. This can show whether the measurement of some components is in fact superfluous. An examination of the phenotypic and genetic correlation structure among the components will show whether there are tradeoffs among them, such that increasing or decreasing one component is compensated for by a change in another component (see, for example, Primack and Antonovics, 1982; Roach, 1986). And if the components can be placed in a developmental or temporal sequence, then their causative relationships can be investigated by using path analysis (Maddox and Antonovics, 1983). From the standpoint of disease control, it is important to understand how plant characteristics can be modified to impact on fungal fitness components and therefore on the spread of disease (Parlevliet, 1979). Fitness components therefore permit the critical analysis of processes leading to successful or unsuccessful pathogenicity.

When generations overlap, as they do in many cases where spore production and infection occur as a continuous process, the latent period, the longevity of the lesion, and the pattern of spore production over the course of the lesion become as important, if not more important, than the total spore production. Leonard and Mundt (1984) used an overlapping generation model based on a triangular approximation to an age-specific fecundity curve (Lewontin, 1965) to estimate growth rates of pathogens. Three time components were considered: age to first reproduction (latent period), age at peak reproduction, and age of last reproduction. They showed that changes in the latent period can have a much larger proportionate effect than changes in the total spore production especially at higher rates of population growth. Similar results were obtained for *Drosophila* by Lewontin (1965).

Such analyses are an approximation of more precise life-table analyses used frequently in population biology (see Charlesworth, 1980). There is a continuing need to place assessments of fitness components in rigorous life-table terms, so they can be used predictively. At times, whole suites of fitness components are presented rather uncritically; these components may simply be algebraic functions of each other (e.g., spore weight/uredium, spore weight/uredial area, spore weight/uredial size; Oard and Simons, 1983) or are of very limited value in population projection (e.g., latent period to 50% lesions; Prakash and Heather, 1986). Fitness components have value only if they each add information that can be rationally combined to produce valid predictions of future contribution.

"Realized Fitness" Methodology Fitness estimates based on single-generation measures of fitness components are always open to the criticism that some potentially important component has been unmeasured. An alternative approach that avoids these difficulties is to estimate fitness based on actual observed changes in phenotype or genotype frequency across one or more generations. However,

these "realized fitness" estimates have their own potential pitfalls because their calculation requires assumptions about the type of selection acting and the mode of inheritance of the trait in question, both of which may change over successive generations. For example, if one observes a change in phenotype frequency over several generations, then the estimate of fitness will require knowledge of the heritability of the trait (and perhaps the assumption that it remains constant) and the type of directional selection acting (for example, whether it is truncation selection or a linear relation between phenotype and fitness). Even in the simpler case of a single diallelic locus, observed changes in genotype frequency over time can be translated into precise fitness estimates only if the dominance relations of the trait are known and if fitness does not change with genotype frequency (see Manly, 1985, Chap. 8). Even then care has to be taken regarding the precise life-history stage at which the census is made (see Prout, 1969, 1971).

The simplest case would seem to be the situation where one is dealing with two identifiable lines, such as two asexual pathogen strains. However, even here fitness estimates depend critically on assumptions about whether the growth rates of the two strains are or are not resource limited and on whether the strains are competing or not for a limited amount of host tissue (MacKenzie, 1978; Skylakakis, 1980; Groth and Barrett, 1980; Fleming, 1981). Furthermore, all growth models require that several assumptions be made (e.g., constant effect of environment, constant density dependence, or constant age-structure in the population), which are likely to be invalidated with most host–pathogen situations (Barrett, 1983). It has been argued that in such situations (MacKenzie, 1980) the concept of pathogen fitness is different from the concept of biological fitness. While there has certainly been confusion between similar symbols being used to indicate different quantities (as pointed out by Barrett, 1983), there seems no justification for trying to carve out a meaning for the term "pathogen fitness" that is in any way different from that used by population biologists working with higher plants or animals: the principles involved are identical and powerfully generalizable to all systems.

Successful estimates of rates of growth of strain isolates from disease progress curves have been made by a number of workers (e.g., Burleigh et al., 1969). Where such curves are available for two different strains simultaneously, they can be used to estimate relative fitness. MacKenzie (1980) used logistic functions to estimate relative fitness of benomyl sensitive and resistant strains and showed that the sensitive strain had a lower fitness not only in the presence of benomyl, as expected, but also when benomyl was absent. Tooley and Fry (1985) estimated the fitness of several isolates of *Phytophthora infestans* by using both logistic and exponential functions. They found general qualitative but not quantitative agreement between estimates of fitness based on the two models, but they were making estimates based on early disease progress when both functional descriptions are likely to be similar.

As mentioned earlier, choice of model is critical in realized fitness estimation, and criteria should be based on both biological knowledge as well as model structure. Thus, where proportional disease estimates are made, the logistic model

is logically the most appropriate, whereas when absolute numbers are measured in the early stages of an epidemic, exponential models may be more appropriate.

Knowledge of the detailed epidemiology of the pathogen may permit the use of more elaborate models. Realized fitness estimates have been also carried out in *Drosophila* (e.g., Prout, 1971) as well as microorganisms (e.g., Cox and Gibson, 1974; Helling et al., 1981; and Paquin and Adams, 1983). In these cases, the models used have made appropriate assumptions about modes of selection or about competition for resources in chemostat systems.

The choice of model should be based on a priori knowledge of the system, rather than arbitrary curve fitting; ultimately the test of a good model should come from independent confirmation of the model assumptions and from the agreement among predicted and realized fitness components. However, we know of no such integrated study either in pathogens or higher plants (but see Tooley, et al., 1986, where some qualitative agreement between estimates made in the two ways is suggested). Both approaches have sometimes been used in experimental *Drosophila* populations (e.g., Kojima and Yarborough, 1967; Spiess, 1977).

Environmental Effects on Fitness The fitness of an individual or class of phenotypes will be influenced not only by the genetically determined attributes of those individuals, but also by the environments in which they find themselves. Thus, it is possible to speak of the effect of an environment (e.g., infection by a pathogen) on the fitness of a (host) plant. It is important to realize that when two phenotypes have different fitnesses in the same environment (or phenotypes are deconfounded from or randomized over environments using experimental designs), we can with knowledge of the mode of inheritance make an *evolutionary* prediction (i.e., effect on gene or genotype frequency). When, however, similar phenotypes have differential fitness in different environments, this observation can only be used to make an *ecological* prediction (i.e., effect on population growth). Such ecological "fitness" measures are often based on female contribution, since it is this that determines population growth.

More problematical is the situation where we show by a descriptive study that in a plant population plants with disease have a greater fitness than those without disease. In such situations, before any evolutionary statement can be made, it must be assumed that the different observed phenotypes (diseased, vs. nondiseased) are the product of inherent properties of those individuals and not solely the product of some other (nonpathogen) environment. This cannot be determined by a purely descriptive study, since it is quite conceivable that some nonpathogen environmental factor (e.g., low nutrients) may independently affect probability of infection and plant size (and hence fitness). If environmental effects are confounded with genetic effects, it is not possible to make predictions about evolutionary outcomes.

Modes of Selection Population geneticists recognize several different modes of selection. In single-locus diploid models, where fitnesses can be ascribed to specific genotypes, the outcome of selection will depend on the degree of

dominance (with regard to fitness); overdominance in fitness (heterozygote advantage) will maintain genetic polymorphism. Single-locus haploid models are formally equivalent to models of noninterbreeding lines or species (assuming haploids do not differ in their mating success).

The fitness of genotypes may also depend on their density and frequency: such density- and frequency-dependent selection may have many biological causes. For example, some genotypes may better exploit an abundance of resources at low density, whereas other genotypes may more efficiently use resources and perform better at high density. In pathogen populations, it is easy to envisage circumstances that may lead to frequency-dependent selection. If the host population is genetically heterogeneous for different resistance alleles, then a virulence allele will have reduced relative fitness as it becomes common because there will be more disease-free tissue available to an alternative virulence allele capable of attacking a different host genotype.

If the phenotype can be measured on a linear scale (e.g., degree of virulence, degree of susceptibility), we can recognize several qualitatively different types of selection depending on the relationship of fitness to phenotype. If individuals toward one extreme of the phenotypic distribution have a greater fitness, this is termed *directional selection*. If intermediate individuals have the greatest fitness, this is termed *stabilizing selection*. Conversely, lower fitness of intermediate individuals is termed *disruptive selection*. These relationships can be quantified by describing the statistical relationship between phenotype and fitness (Arnold and Wade, 1984a, 1984b). The regression of fitness on the phenotype may have linear and quadratic components. The linear component reflects the forces of directional selection, whereas the quadratic component if negative, represents stabilizing selection, or if positive, represents disruptive selection. We can also apply the concept of frequency- and density-dependent selection to quantitative traits, where now the terms apply to the frequency or density of the different phenotypic classes.

It is important to note that the terms *directional, disruptive,* and *stabilizing* do not refer to expectations of the future composition of the population. The outcome will be dependent on the genetic transmission of the phenotypes, as well as on other properties of the system (e.g., relative abundance of host and pathogen). Thus, plant pathologists use the term *stabilizing selection* (after Van der Plank, 1963) in a way totally inappropriate for a population biologist (see also below, Measurement of Pathogen Fitness). With Van der Plank's usage, *stabilizing selection* refers to a dynamic outcome and only obliquely to a fitness concept (i.e., that *if* there is a cost to virulence, then virulent types will not increase in number, but be "stabilized" in the absence of resistant hosts). General methodologies of fitness estimation can be found in recent books by Manly (1985) and Endler (1986) and many of the references therein.

The following section will focus on measuring fitness in natural or semi-natural plant–pathogen systems; such information is crucial to understanding the ecological and evolutionary interactions involved in disease processes. The effect of disease on plant fitness, and generally the importance of disease in plant ecology

and evolution, have been largely ignored by population biologists. Several recent studies, however, will be used to show the types of approaches. There are also essentially no studies of differential fitness in populations of plant pathogens interacting with nonagricultural plants. Thus, for the section on pathogen fitness, we will illustrate our discussion with examples from the applied plant pathology literature.

MEASUREMENT OF PATHOGEN FITNESS

Fitness and Disease

The traditional terms used in plant pathology to describe the disease process have only an indirect relationship to the term *fitness* (of host or parasite) as used by the population biologist. The plant pathology terminology often contains *implicit* assumptions about fitness effects, but they are not *explicit* or precise fitness measures. Thus, a plant that is diseased will usually leave fewer offspring than one that is not, but "disease" refers to the overt manifestations of an infection and not to the fitness effect on the host.

The relationship between disease and fitness can further be complicated by phenomena such as tolerance (Schafer, 1971). Resistance and susceptibility of host plants and virulence and avirulence of pathogens also refer to the appearance of disease symptoms given a particular combination of types of host and pathogen, and are not measures of survival or reproduction of either host or pathogen. Only in extreme cases, where a host is so susceptible to disease that it is killed prior to reproduction or a pathogen is so completely avirulent that it cannot infect, is it possible to equate these terms to (zero) fitness.

The plant pathologist is of course directly concerned with the fact that the virulence of the pathogen will depend on the particular genotype of the host. This has a direct analogy with the concern of the population biologist with the equally obvious fact that the relative fitness of a genotype will depend on its environment. The pathologist will speak of the "vertical" and "horizontal resistance" (of the host) or of "virulence" and "aggressiveness" of the pathogen (sensu Van der Plank, 1982) to indicate that there is or is not a strong dependence of the incidence of disease on the "biotic environment." The population biologist will similarly speak of "specialist" and "generalist" genotypes. All these types of terminology are dangerous because they impose qualitative distinctions on processes that are essentially quantitative in nature. The terminology becomes even more misleading if it is coupled with expectations about the modes of genetic determination of the specialization (e.g., single locus vs. polygenic).

A much more quantitative approach is to consider that some ensuing outcome (disease incidence, fitness of host, fitness of pathogen) is a function of the host genotype, the pathogen genotypes, or their interaction (Van der Plank, 1982). The term *vertical resistance* is a statement that the pathogen–host interaction is high and that there is a strong negative genetic correlation in performance of pathogen

strains over host genotypes, whereas the term *horizontal resistance* is a statement that there is only a main effect of host genotype, no interaction, and genetic correlations are positive. The qualitative nature of the terms *horizontal* and *vertical resistance* has hindered the development of rigorous measures describing the plant–pathogen interaction process. These terms, like *stabilizing selection*, have also erected artificial semantic barriers between the disciplines of plant pathology and plant population biology.

Pathogen Fitness in Natural Populations

In contrast to studies of economically important plant pathogens, very little is known about fitness for pathogens interacting primarily with natural plant populations. The few studies of such pathogen populations are usually of a descriptive nature, focusing on the phenotypic composition of the population in terms of virulence on various hosts (Eshed and Wahl, 1975; Eshed and Dinoor, 1981; Oates et al., 1983). It has been suggested by Leonard and Czochor (1980) and others that pathogen populations are often diverse for virulence traits, particularly in regions where the host and pathogen apparently evolved or where environmental conditions are particularly advantageous for the pathogen. High diversity of resistance genes in host-plant populations have also been found in similar situations (Wahl, 1970; Wahl et al., 1978; Burdon et al., 1983).

Such relationships led Leonard and Czochor (1980) to hypothesize that the polymorphism for virulence in natural populations of pathogens and resistance in plant populations are likely to be "balanced," such that individuals with either extremely high or extremely low levels of virulence are less fit than those with more intermediate phenotypes. This conclusion is based on the rationale that selective pressures on both host and pathogen will be strongest when conditions are conducive to disease. Thus, if transient polymorphisms for virulence were the rule, i.e., that selection occurred in a single direction for virulence or avirulence until fixation occurred, one would expect that more virulence genes would be maintained (= greatest diversity) in regions with low selective pressures where environmental conditions were marginal for disease development. Since this pattern is not borne out by virulence surveys, Leonard and Czochor (1980) conclude that "costs" for virulence must exist that, along with obvious fitness costs of "avirulence," lead to maintenance of intermediate virulence levels.

Apart from these general considerations, we know almost nothing precise about the differential fitness of pathogen genotypes sampled from natural populations. The study of pathogen fitness in nature is likely to be a difficult endeavor, requiring extensive measurement of components of fitness of individual isolates on particular host genotypes or the use of electrophoretic or molecular markers to "follow" particular genotypes and so estimate realized fitness on different host genotypes under field conditions. However, many questions of interest to the plant pathologist (as well as population biologist) could be addressed. For example, are gene-for-gene systems common in nature or are they a product of agricultural selection? Is there a positive correlation between pathogen abundance and number

of virulence genes? By what mechanism is genetic diversity in virulence maintained in nature? Is there a cost to virulence in natural populations? What regulates the size of pathogen populations in nature? All these questions are of direct applied interest in providing a context within which to interpret results obtained from crop populations.

"Stabilizing Selection" in Plant Pathology and Fitness Concepts

The major focus of fitness studies in plant pathology has been on the relationship between virulence and the overall fitness of the pathogen. This has developed into a controversial topic in plant pathology. The basic question is whether or not pathogens that are virulent on a wide range of host genotypes are less "fit" than pathogens with narrower ranges. Van der Plank (1963, 1968) addressed this issue by suggesting that on susceptible hosts, pathogens with "unnecessary" virulence genes had a lower fitness than pathogens lacking such virulence. Such a phenomenon would tend to stabilize the genetic composition of the population in the sense of maintaining genes for conferring both virulence and avirulence. In plant pathology this idea has come to be known as the concept of *stabilizing selection* (sensu Van der Plank, 1963, 1968). As mentioned earlier, this use of the term *stabilizing selection* is quite alien to its use in population genetics (Leonard and Czochor, 1980). Population geneticists use the term to refer to situations where any extremes of a phenotype (i.e., in this case, very high virulence or very low virulence) have low fitness compared with phenotypes with intermediate measures of the trait.

In discussions of virulence and pathogen fitness, there are three issues that are often confounded. The first issue is whether a particular virulent strain has a lower fitness than a particular avirulent strain on susceptible plants. The second issue is whether particular genes for virulence reduce the fitness of the pathogen. And the third issue is whether virulence genes in the population as a whole result in average lower fitness of the individuals carrying them.

The first issue can be addressed readily by direct fitness comparison of the two strains in question. The second issue is more difficult to resolve because it requires that the particular virulence genes be compared on a uniform genetic background, so that other loci in which the strains differ do not confound the result. This is particularly true if there is asexual reproduction, an important part of many pathogen life cycles, since this can effectively link together traits with varying effects on fitness so that one cannot distinguish adverse effects of any one trait such as virulence (Leonard, 1977a). The problem is the same as is encountered by population biologists who wish to ascribe fitness effects to any particular locus: it is often not clear whether the effects are due to the locus itself or other alleles that may be in linkage disequilibrium with that locus (see Lewontin, 1970, for extensive discussion of this problem). There is unfortunately no foolproof solution beyond reducing the likelihood of such associations by backcrossing, induced mutation in isogenic lines, by randomizing the genetic background as much as possible, or in the future, by gene transfer.