Letter to the Editor

Races, Pathogenicity Phenotypes, and Type Cultures of Plant Pathogens

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Contribution 80-194-A, cooperative investigations of USDA-SEA-AR and the Kansas Agricultural Experiment Station, Department of Plant Pathology, Kansas State University, Manhattan 66506.
Accepted for publication 16 April 1980.

Pathogenic specialization in plant parasites and specific host plant reactions are important phenomena in plant disease control. Biologically, neither pathogenic specialization nor specific host reaction occur alone. Parasite and host interact to produce a third entity, the host:parasite association. The association phenotype is dependent on host genotype, parasite genotype, and environment. Historically, host and parasite characters have been, and must continue to be, considered separately because the host is manipulated in response to parasite population changes. Thus, a method is needed to directly relate data about the host:parasite association to both host and parasite.

Races. The term race has been widely used to describe pathogenic specialization in plant parasites. Race has become ambiguous because it is used to convey at least three different concepts:

1. A taxon of rank below species (18). A taxon is a group of individuals having characters in common, with a formal name attached.
2. An abstract group of real or hypothetical individuals having specified characters in common, with no formal name attached.
3. The biological material of an organism that is under experimental control. The use of race to convey this concept is clearly incorrect. Culture and isolate are proper terms to describe this meaning.

Terms such as strain, form, variant, pathotype, and pathovar have been used as synonyms of race to convey one or more of these concepts. Usually it is not clear which of the conceptual meanings is intended when either race or one of the other terms is used.

This ambiguity of terms is not necessary. We suggest that the term race, as used in studies of host:parasite specificity, should have only one meaning, that of an abstract group of individuals having specified characters in common, with no formal name attached. Using the term to convey the concept of biological material (concept 3) is incorrect; the formal naming of parasite population groups (concept 1) presents problems that seem to be unresolvable.

Taxonomy and race. Historically, race description and naming has been a taxonomic procedure (18). Portions of a species that had pathogenicity characters in common were described and named. The application of this procedure to studies of pathogenic specialization violated two principles of plant taxonomy (17). Races were described and named on the basis of their pathogenicity to "closed sets" (19) of host differential cultivars. This violated the taxonomic principle that the basis for classification of organisms is "open-ended." In an open-ended system, characters used in classification can be added, or no longer used, according to the investigator's needs and judgment. Classic race identification has no means of adding a differential or not using any differential of a closed set. Secondly, race classification and naming systems have not provided for type cultures to serve as a basis of nomenclature when numbers of characters used in classification are increased or reduced. This violated the principle that there is to be a "type" for every named taxon below the rank of family (17). Types obviously were not established in the early work with races because the necessary types could not be adequately preserved as living cultures.

Failure to observe these two principles apparently precluded the acceptance of pathogenic race as a valid taxon by the International code of Botanical Nomenclature (17). Attaching race names (numbers) to parasite groups without observing these two principles will always result in ambiguous names. A valid system for classification and naming of races could be developed, but we believe this is not necessary. It is not necessary because the functional unit of pathogenicity is not race as a total or arbitrary combination of pathogenicity, but genes for pathogenicity to the host genes for low reaction that occur in crops being grown at a particular time and place.

Implications of the gene-for-gene relationship. We believe that to resolve the problem of ambiguity of race names, plant pathologists should cease naming races on the basis of pathogenicity to *specified* sets of host cultivars. Instead of naming races, we suggest using the implications of the gene-for-gene relationship and a pathogenicity-formula description system to portray pathogenicity to *any* set of cultivars that are useful in a particular situation.

The pioneering work of Flor (4) led to development of an important body of theory concerning the gene-for-gene relationship (3,8,9,14), as well as a large amount of information about details of some important host:parasite associations (3,13,15). We believe this available theory could be better used in studies of pathogenic specialization. The number of possible genotypes for host reaction or parasite pathogenicity can be calculated as 2^n in which n is the number of functional corresponding gene pairs. The possible number of genotypes becomes very large; for example, 2^{35} in the *Triticum:Puccinia recondita* system.

If every host gene for reaction were represented singly in a set of differential hosts, the number of possible races would be equal to the number of possible genotypes for pathogenicity. Thus, the number of possible races becomes so large in most systems as to eliminate any hope of naming the total variation as races, or even visualizing it theoretically. Even if all races could be described and named, reporting of data as frequency of race names would obscure the important information from a survey—the frequency of genes for low or high pathogenicity in a parasite population.

The major problem in all race nomenclatural schemes so far proposed is that there is no direct way to relate results from one differential set to another, although the sets may have host cultivars in common. We cannot conceive of a race nomenclatural scheme by which results from two or more differential sets could be related, because the race name would always obscure the number and kind of differentials used in each of the sets. The pathogenicity formula method is an excellent method of presenting results if a name is not attached to each formula.

Pathogenicity formulae. Green (5) proposed a method by which races were described by a "virulence formula" of the form "effective/ineffective host genes." In his application of the method,

a code number is assigned to each formula; these code numbers serve the function of a race name. For example, the formula 5,6,7,9a,9b,10,11/8 for *Puccinia graminis tritici* was assigned C1 and is synonymous to International Standard race 17 (5). This method has since been adapted to describe pathogenicity differences in *Puccinia striiformis* (7), *P. graminis avenae* (12), and *P. recondita* (10). Attaching a name to each formula has most of the same shortcomings of the more classic method of naming races and describing them with a key.

We believe that the pathogenicity formula method, used conceptually in a different way, will resolve the confusion about race names now extant in host:parasite specificity studies. The mechanics of studying pathogenic specialization and host reaction genetics in order to manipulate plant breeding materials toward resistance involves two major aspects of describing the parasite; a) the monitoring and description of pathogenicity in parasite populations, and b) the description of specific pathogenicity in particular parasite cultures that are used in genetics and plant breeding. The pathogenicity formula method can serve both these purposes well.

We will illustrate each of these aspects, using data from the *Triticum:P. recondita* relationship. The illustration portrays pathogenicity to fewer host lines than are currently being studied; we do this for clarity of presentation. Assume that a sample from a parasite population was taken and assayed for pathogenicity to four host lines, each having a gene for low reaction, *Lr1, Lr2a, Lr2c,* or *Lr3*, respectively. The data can be efficiently presented as:

Pathogenicity formula	Percent frequency
1, 2a, 2c, 3/	33
1, 2a, 2c/3	40
2a, 2c/1, 3	13
1/2a, 2c, 3	14

where avirulence is listed on the left of the virgule (/) and virulence is listed on the right of the virgule.

With this presentation, virulence frequencies to host lines with each of the genes can be readily calculated: 13%, 14%, 14%, and 67%, respectively, to the lines with *Lr1, Lr2a, Lr2c,* or *Lr3*. Pathogenicity associations can readily be calculated. For example, avirulence to lines with *Lr1* or *Lr2a* was associated in 73% of the population sample and no cultures in the sample were virulent to lines with *Lr1* and *Lr2a*. Pathogenicity to lines *Lr2a* or *Lr2c* was associated in all cases. In North American *P. recondita* populations, the pathogenicity formulae presented above are roughly equivalent to UN races, 1, 2, 5, and 17 (1). Presentation of the above data as 33% UN race 1, 40% UN race 2, 13% UN race 5, and 14% UN race 17 would be accurate, but would obscure both the frequencies of virulence to single host lines and the associations of pathogenicity to two or more host lines. When different host line sets with some lines in common are used in separate studies, data can readily be compared if the pathogenicity formula method is used to portray the results of both studies. Samborski (16) used this method to present his results from a *P. recondita* pathogenicity survey. Frequencies of virulence and associations of pathogenicity are the important results from such studies, not named race frequencies.

When cultures are used in genetic studies, they can readily be described by pathogenicity formulae. It would be much more meaningful in interpreting genetic data to indicate that *P. recondita* culture 1 with a pathogenicity formula of 1, 2a, 2c, 3/ and culture 2 with a pathogencity formula of 2a, 2c/1, 3 were used rather than cultures of UN races 1 and 5. The pathogenicity formula-descriptive method is direct; the race-naming method is indirect and obscures genetic relationships. Kuhn et al (6) used the pathogenicity formula method to describe *P. recondita* cultures. Their culture 14 and our laboratory culture UN09-66A have the same formula: 3,9,11/1,2a,2b,2c,2d and relate directly to one another.

The pathogenicity formula method can be used when the reaction genotype of one or more of the cultivars within a differential set is unknown. Green (5) used such a cultivar, Golden Ball, in his differential set and abbreviated it as GB in the formulae.

Type cultures. The genetics of host reaction has been studied by observing the low- or high-infection types produced by one or a relatively few cultures on plants of segregating populations from controlled host crosses. Usually, only the host materials have been preserved; but sometimes even the host lines having named genes have not been preserved. The development of knowledge concerning gene-for-gene relationships and the possibility to test genetic hypotheses without making host crosses (8,9), make the preservation and documentation of parasite cultures used in genetic studies very important. Obviously, there is no need for type cultures to document named races, but there is a great need for cultures to document corresponding gene pairs in the various host:parasite systems. Technology is now available to preserve this valuable biological material (2). We believe documentation of a parasite culture to detect a host gene for low reaction should be a requisite for naming the gene, actually the gene pair.

Conclusion. We conclude that race, as applied to studies of host:parasite specificity, is a group of individuals in a parasite population with pathogenicity characters in common. This is in keeping with the general use of race in biology. The confusion associated with different usages of race can best be resolved by using race only for that meaning. Further, no term is needed to convey the concept of a formal taxon with specified pathogenicity phenotype because the formal taxon is not needed. We believe that studies of pathogenic specialization in systems in which genetics are not known would be more useful if their objective were to elucidate genetic relationships rather than merely to describe and name parasite variation for pathogenicity to host cultivars.

Luttrell (11) suggested that the value of taxonomy in mycology is to facilitate storage and retrieval of information. We suggest that taxonomy is extremely valuable for this purpose in all of biology, and that information about host:parasite specificity can best be stored and retrieved on the basis of genes for host reaction interacting with parasite genes for pathogenicity—on the basis of corresponding gene pairs.

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