Nomenclature for Pathogenicity and Virulence: The Need for Precision

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In a recent review, Shaner et al (9) considered the different meanings of terms related to pathogenicity as used in plant pathology literature and stressed the need for common designations of basic concepts. They proposed that avirulence and aggressiveness should be abandoned and replaced by nonpathogenicity and parasitic fitness, respectively. They also suggested a dichotomous hierarchy of terms describing pathogenicity, composed of virulence on one side and parasitic fitness on the other. The latter component was split further into specific pathogenicity and reproductive fitness. Finally, a quantitative meaning for virulence was proposed. Unfortunately, and as recognized by the authors themselves, no explicit definitions of these suggested designations are given, although the introductory sentence of the review rightly states that "precision of names given to concepts, structures and phenomena is indispensable to communication in science" (9).

In my opinion, the proposals suggested by Shaner et al (9) increase the confusion instead of clarifying the current nomenclature. The aim of this letter is to show that the concepts covered by the terms nonpathogenicity, avirulence, virulence, aggressiveness, and parasitic fitness all accept distinct definitions, and that they all are of value to plant pathologists for describing different realities.

Avirulent pathogens versus nonpathogens. Part of the problem in accurately defining pathogenicity-related terms arises from the lack of a clear definition of what an infectious disease is. It is interesting to note that such a definition is absent from many major textbooks on plant pathology (e.g., 7,15,16), although it is the basis for all further concepts. The following discussion is restricted to infectious diseases, because physiological disorders do not imply the interaction of two organisms and, therefore, are outside the scope of the definitions of virulence, avirulence, aggressiveness, and parasitic fitness.

An infectious disease can be defined as a harmful alteration of the normal physiological state of an organism, called a host, due to challenge by another, called a pathogen. It results in "visible or otherwise detectable abnormalities," called symptoms (10). A pathogen succeeding in entering host tissue and deriving part or all of its nutritive substrates from it is called a parasite and is said to infect its host (10). The definition of a pathogen implies that a nonpathogen is an organism not inducing disease when challenging another (10); the challenged organism is then called a nonhost.

A major feature of nonhost resistance is that all members of the nonhost species are resistant to the nonpathogen (4,6). The opposite extreme is when all members of a host species are susceptible to a pathogen. More commonly, only part of the genotypes constituting the host species are resistant to the pathogen; their resistance is called host resistance, and the pathogen is said to be avirulent to them. The challenge of a resistant host by an avirulent pathogen results in an incompatible reaction (6). On the other hand, the pathogen is said to be virulent to a host when it is able to infect it and, usually, to reproduce on it; the host is then said to be susceptible and the interaction compatible. These definitions make virulence and avirulence clearly qualitative traits, as used by Vanderplank (14,15) and many subsequent authors (e.g., 1,2,5,7,11,12).

It is clear that definitions of pathogenicity and virulence apply to pairs of organisms; therefore, one organism may be a pathogen for some species and not for others. A classic example is *Phytophthora infestans*, a pathogen of potato and tomato but not of wheat, citrus, or pines.

The definitions are simple to formulate, but recognition of the nonhost status of a resistant species is sometimes difficult. Niks (6) suggested that nonhost resistance is generally characterized by either avoidance mechanisms or by immunity, i.e., a high proportion of early abortion of the nonpathogen and infrequent necrosis of host cells. On the other hand, host resistance is most commonly expressed as either a hypersensitive reaction or necrosis associated with limited growth and reproduction of the pathogen (6). This led Tosa (13) to consider wheatgrass (*Agropyron repens*) as a host of *Erysiphe graminis* f. sp. *tritici*, because their interaction results in hypersensitive necroses of the plant cells. However, nonhost resistance is sometimes expressed mainly as necrotic reactions (6), showing that immunity cannot be equated with nonhost resistance. In spite of this difficulty in differentiating host from nonhost resistance, their separation into two different concepts is supported by mechanistic and genetic data. Day (3) suggested that a basic mechanistic difference existed between avirulence reactions, which are mechanisms of resistance "superimposed on an interaction that already has most if not all the elements needed for compatibility," and nonhost resistance, which is "generally due to failure to induce susceptibility." There also is accumulating evidence that the two types of plant resistance are mediated by different genetic systems (9).

Aggressiveness versus parasitic fitness. Although several meanings have been associated with the term aggressiveness in plant pathology literature (8,9), most plant pathologists currently use this term, as originally defined by Vanderplank (14,15), to designate the quantity of disease induced by a pathogenic strain on a susceptible host (e.g., 1,7). From this definition, it appears that aggressiveness depends primarily on the pathogen but also on the host and the environmental conditions; it is, therefore, a characteristic of a host-pathogen interaction rather than of a pathogen alone. This appears clearly in a number of reports, showing that the same pathogenic isolate can induce different amounts of disease on a series of susceptible hosts (e.g., 7,14,15). There is, therefore, no such thing as "the aggressiveness of one strain" per se, measurable as the area of diseased tissue or the number of offspring produced by this strain on any susceptible host. Aggressiveness always depends on the partial resistance features of the host on which it is measured (15). Another attribute of aggressiveness is that it can be measured repeatedly in standard environmental conditions and is then a stable trait for a given host-pathogen pair.

On the other hand, fitness classically designates the contribution of a given genotype to the gene pool of the next generation of the organism considered (9). Thus, parasitic fitness is basically an attribute of a pathogenic strain within a population, rather

than of a single host-pathogen interaction. It obviously depends on the aggressiveness of the strain on the different hosts available for infection, as correctly outlined by Shaner et al (9), but also on the aggressiveness of the other genotypes composing the pathogen population on the same set of hosts. Because it is a result of the relative parasitic and reproductive abilities of a parasitic population on a host population, parasitic fitness of one pathogen strain is in practice not a stable trait, because populations and environmental conditions are never the same in two experiments. Consequently, the suggestion of Shaner et al (9) of equating aggressiveness and parasitic fitness is irrelevant, because these terms designate clearly distinct concepts.

The dichotomous separation of pathogenicity between virulence and parasitic fitness proposed by Shaner et al (9) also is a source of confusion, because the authors meant a quantitative definition of virulence. I cannot see where the separation is between a quantitative virulence (including, at least to some extent, the amount of disease produced by the pathogen) and parasitic fitness as equated to aggressiveness, because the latter term seems to be part of, but not separate from, the former. Although Shaner et al (9) stated that "we should not enforce dichotomies where none exist," they obviously did so by separating two overlapping concepts.

Epidemiological implications. Vanderplank (15) showed that race-specific resistance and race-nonspecific resistance have largely different epidemiological consequences, for the former delays the onset of the epidemic, whereas the latter reduces the rate of epidemic progression. Heath (4) stated that one of the most prominent features of nonhost resistance is the provision of a highly effective, durable protection of the nonhost plant. Therefore, the separation of pathogenicity both from the genetic and the epidemiological standpoints into three qualitative categories (nonpathogens, avirulent pathogens, and virulent pathogens) seems to be fully justified. Aggressiveness relates only to the latter qualitative category and depicts the amount of disease produced in a particular susceptible host-parasite interaction. Parasitic fitness is a measure of the success of one pathogenic genotype in a given population challenging a range of hosts. Thus, these five concepts are markedly distinct, and the terms used to designate them, therefore, cannot be used interchangeably.

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