The Use of Terms for Responses of Plants to Viruses: A Reply to Recent Proposals

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Scientific information in plant pathology has been attained at an increasingly fast rate in recent years, mainly because of the dramatic growth of related life sciences. However, our knowledge of certain biological phenomena involved in plant-pathogen interactions at the molecular, cellular, organismal, or population level is still elementary. Therefore, terms we use to describe these phenomena are often not precise and, in some cases, cause confusion. Recently, Cooper and Jones, in a letter to the editor (6), offered to dispel some of "the confusion that exists" with regard to the "inconsistent use" of certain phytopathological terms by plant virologists and plant breeders. Since they welcome comments, I would like to respond to their proposals.

Cooper and Jones (6) proposed use of the terms

> *resistant* and *susceptible* to denote the oppposite ends of a scale covering the effects of an infectible individual on virus infection, multiplication, and invasion, and the terms *tolerant* and *sensitive* to denote the opposite ends of a scale covering the *disease reaction* of the plant to virus infection and establishment.

A careful review of the literature on plant virus diseases, however, indicates that there are relatively few virus-host systems for which there is any definite information about yield losses (tolerant versus sensitive). In many instances, data on viral replication rates in given hosts are lacking (susceptible versus resistant). Plant breeders usually deal with large numbers of plants, so it is not feasible for them to assess the effect of the different plant genotypes on virus replication or translocation. As Cooper and Jones pointed out, virus concentration is often not directly related to disease reaction (which includes yield). Replication and accumulation of potato spindle tuber viroid (PSTV) is about the same in three tomato cultivars that may respond with severe (cv. Rutgers), mild (cv. Rentita), or no (cv. Hilda 72) symptoms following infection by the same strain of PSTV (13). Severity of symptoms induced on Essex soybean by four strains of soybean mosaic virus (SMV) was not related to virus concentration, which was comparable in all cases (9). Furthermore, virus replication is often controlled by both host and virus genes (10). I believe that Cooper and Jones's proposal on "resistant"/"susceptible" is impractical and confusing.

Cooper and Jones (6) suggested that one component of resistance to virus infection may be the "resistance to adsorption or attachment," which is "the incompatibility of surface properties possessed by virus nucleoprotein for surfaces of potentially infectible cells [that] may hinder infection." This hypothesis is not supported by scientific evidence. There is no evidence showing that plant viral coat proteins play a role in cell recognition (11). The requirement that plant viruses enter cells through wounds on the cell surface supports the hypothesis that plant viruses may have evolved a recognition system that bypasses any virus-cell surface interaction. Intact virus particles move from cell to cell through the plasmodesmata and cause infection while remaining within the plasma membrane (11). There is evidence, however, of recognition by a virus of a particular organelle or site within the cell (8,16), but this is not the type of recognition to which "resistance to adsorption or attachment" referred.

According to Cooper and Jones (6), another "component of passive resistance might be *resistance to a vector.*" Resistance to a vector, however, is a mechanism of disease escape (not resistance), such as in the situation in which an inherently susceptible plant (raspberry) does not become infected with a virus (raspberry mosaic) because it is a nonpreferred host of the insect vector. The term "klendusity" is used to characterize this particular type of disease escape (7).

In the phytopathological literature, tolerance has usually been used to designate the capacity of a cultivar to endure disease with less yield or quality loss relative to disease severity or pathogen development, compared with other cultivars (1,7,18). Cooper and Jones (6) correctly observed that tolerance has often been mistakenly considered by plant virologists as a type of resistance. The confusion that has led to the interchangeable use of the terms resistance and tolerance is due to the lack of information concerning virus concentration in the plants under study. The term tolerance (sensu Schafer [18]), however, has often been used correctly in the early and most recent plant virus literature (15,20). According to the Cooper-Jones proposal

> sensitivity is a subjective description of disease severity that will often be associated with conspicuous symptoms in an infectible organism and may indicate that infection with a specific virus diminishes the rate or amount of plant growth or marketable yield.

In certain virus-host systems, however, the reaction of the host (cowpea cv. California Blackeye) to virus infection (cowpea chlorotic mottle virus [CCMV]) is severe with regard to symptom expression, but yield is not affected (10). In contrast, cowpea cv. Iron shows mild symptoms upon infection with the same strain of CCMV, but its yield is diminished significantly. Concentration (milligrams of virus per gram of tissue) of CCMV is 0.533 in California Blackeye, 0.842 in Iron, and 0.036 in the resistant cv. PI 186465. Since Cooper and Jones (6) proposed (Fig. 1 of their letter to the editor) that a plant is tolerant when "little or no effect on the plant is apparent" following virus infection, both cultivars, California Blackeye and Iron, would be characterized as sensitive (sensu Cooper and Jones). This characterization is misleading, however, especially for plant breeders, in view of the fact that yield of California Blackeye is not affected by CCMV infection. On the contrary, California Blackeye would be classified as tolerant (based on virus titer, symptoms, and yield) to CCMV, whereas Iron would be classified as susceptible (based on the same criteria) according to the definition of resistance, susceptibility, and tolerance accepted to date (7,18). Consequently, PI 186465 would be classified as resistant on the basis of absence of systemic symptoms, low virus titer, and yield unaffected by CCMV infection. The current terminology on plant response to virus infection, if used properly, is clearly functional from the standpoint of communication among plant virologists and plant breeders as well as plant pathologists dealing with other categories of pathogens.

Cooper and Jones's (6) suggestion that immunity is "an absolute state of exemption from infection with a specified agent" is in agreement with the recommended use of the term (1,5,7,17). Their approach, however, of using "the term *immune* to denote plants in which virus cannot be detected after repeated challenge inoculations" is not characterized by the appropriate stringency. In true immunity, no virus replication could occur in the cells of a

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plant under any circumstances (11). The following example should demonstrate the limitations of Cooper and Jones's suggestion. One-thousand thirty-one lines of cowpea were surveyed for resistance to cowpea mosaic virus (CPMV) (4). Sixty-five lines were classified as operationally immune because no symptoms were observed and no virus was recovered 7–12 days after inoculation with purified CPMV at a concentration that was 100 times that which would uniformly infect susceptible lines (4). Some of the apparently immune cowpea lines, however, supported CPMV replication after graft inoculation (3). Moreover, CPMV replicated in protoplasts from 54 of the 55 tested immune lines. These apparently immune lines may have been giving an extreme hypersensitive reaction expressed in the form of microscopic local lesions in the leaves. In further experiments, protoplasts from the one apparently immune line (Arlington) could support replication of very small amounts of CPMV (1% of what was associated with similarly inoculated protoplasts from a susceptible line). In addition, CPMV replicated (0.3% specific radioactivity, recipient/donor) in one of the eight Arlington seedlings tested after graft inoculation (3).

One might argue that it is not possible to test for virus replication under every conceivable circumstance. So, for practical purposes it might be worthwhile to set some reasonable standards for acceptance of a demonstration of immunity. This approach, however, can lead to the use of "immune" cultivars that actually contain low amounts of virus. For example, *Solanum tuberosum* L. 'Atlantic' was reported to be immune to potato virus X (PVX) when it was released in 1976, only to be found infected with PVX a few years later (19). The consequences of misuse of the term immune can be devastating in situations where a susceptible cultivar is located near the "immune" cultivar, vector populations are high, cultural practices are poor, or the small virus population present in the "immune" cultivar gives rise to a mutant with increased virulence toward its host. I believe that the terms "extremely resistant" or "highly resistant" are more appropriate when careful serology and infectivity tests under greenhouse and field conditions fail to detect the presence of a given virus in a line of a plant species that is known to be a host of this virus. A plant cultivar might be designated as immune only when utilization of more stringent techniques, similar to those described previously (3,4), produces no evidence suggesting presence of the particular virus. I think that the great value in conjunction with the small number of potentially immune lines justifies the additional time and effort required by this rigorous approach, which is merely a safer compromise.

Finally, I would like to make a few "minor" comments on a statement that was supposed to be an example of plant tolerance to virus pathogens. Cooper and Jones (6) stated that "viruses that invade organisms without causing disease are described as *latent*." First, viruses that are often latent (ie, potato virus X) cause considerable (up to above 15%) yield losses (14). Because yield is part of the plant response to virus infection, the above statement is not a good example of tolerance sensu Cooper and Jones or sensu Schafer. Second, latent viruses may not cause easily recognizable symptoms, but they do cause disease according to the most refined definitions of disease (2,12). Third, Cooper and Jones suggested that "operationally the processes of *infection* and *invasion* [of other cells] are different." Yet, a few paragraphs later (see statement under question) they used the word "invade" to denote infection.

It is difficult to coin precise, universally accepted terms or to formulate definitions for these terms that accurately and completely describe all aspects of complex phytopathological phenomena. The events associated with pathogenesis of plant virus diseases are similar to those of diseases caused by bacteria, fungi, or nematodes. Introduction of "new" terms to describe certain aspects of plant-virus interactions is not only unjustifiable but also confusing, especially when a modified meaning is given to terms already used to describe different but related concepts of plant disease. I believe that use of terms defined in a broad but accurate and complete way facilitates scientific communication at the present time. As new knowledge is gained in plant pathology, existing terms and definitions could be reexamined and refined to reflect the latest advances of our exciting science.

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