Disease Tolerance—an Indicator of Thresholds?

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Subsequent to discussions at the Third International Congress of Plant Pathology (Munich, 1978) and the recent Stakman Commemorative Symposium on Crop Loss Assessment (Minneapolis, 1980), I am prompted to present my views on the concept of plant tolerance to disease. Perhaps the most severe problems associated with the term "tolerance" arise because it is used by workers in diverse disciplines to describe a wide range of concepts. In recent reviews, Mussell and Malone (10) and Mussell (9) included in the concept of "disease tolerance" the phenomenon of reduced symptoms in the presence of equal populations of pathogen propagules; the inactivation of toxins and enzymes produced by the pathogen; "slow rusting" and epidemic development delayed by agronomic practices and the use of multilines. Each of these may be considered to be something other than tolerance to disease, and explainable either in terms of tolerance to pathogens, to forms of resistance, or to epidemiological manipulation via resistance or cultural practices. All have in common the fact that less disease develops in the given situation. Schafer (15) suggested that tolerance to disease may be defined as "that capacity of a cultivar resulting in less yield or quality loss relative to disease severity or pathogen development when compared with other cultivars or crops." As he pointed out, however, tolerance so defined is a difficult concept to identify and quantify, and this has been reflected in difficulties that have arisen in attempts to measure and breed for tolerance to disease; eg, Simons (16); Clark and Johnston (1); Ziv and Eyal (17). In view of these difficulties and our increasing knowledge of the causal relationships between disease and yield loss (4,5,8), it is timely to reassess the situation and to ask whether tolerance to disease is a useful concept or whether the term should be restricted to usage in tolerance to pathogens.

When the term is applied to plants that "appear susceptible to a disease without sustaining severe losses in yield or quality" (17) certain assumptions are implicit in the usage of the term. First, it is assumed that the parameter of yield measured fully described the harvestable portion of the plant. In most studies yield has been measured as weight per unit area, and often this has been subdivided into various yield components to more closely define the cause of loss. Simons (16) suggested that kernel density alone may be used as an index of tolerance, but the correlation between this and yield response to rust infection was not high ($r = 0.658$). Single components of yield are less likely to describe disease effects on yield than yield measurement itself. In most yield studies in infected crops, quality, such as protein content, usually is not assessed; this represents a deficiency in the analysis that is not confined to studies of tolerance. Second, it is assumed that the measurement of disease is a true representation of the influence of disease on the plant. This is a more difficult area, and may well be the main source of error in the proposition of tolerance to disease in many cases. As pointed out by Schafer (15), it is necessary to measure disease throughout the epidemic and not at a single point; otherwise differences in epidemics that achieved similar final levels may be overlooked. It is also important that the chosen parameter truly reflects the total effects of the disease. For example, disease assessment based on sporulation rate per unit leaf area describes only one facet of the disease, and may bear little relevance to the mechanism of yield reductions. It is illogical for these purposes to describe disease on the basis of the amount of pathogen. Percent disease severity based on lesion size and associated chlorosis represents a more satisfactory parameter, but it still ignores some of the effects of disease on the ability of the plant to grow and develop its yield potential. This would apply especially for disease syndromes of long duration when the influence on leaf size has not been included in the assessment (8). This aspect recently was discussed by Kramer et al (7); they suggested that a more meaningful method of assessment was desirable, but perhaps difficult or impossible to achieve.

The lack of cultivar response to disease is often assumed to be specific to the disease situation. I believe that this may not be so, and that it may more accurately be considered to be a lack of response to any constraint situation such as drought or a reduction in incident radiation, and not to disease specifically. For example, Fischer (3) showed that shading treatments for short durations at different growth stages in field crops of wheat caused different responses by the crop in terms of final yield. At some growth stages, final yield was unaffected either because the plant was able to compensate at later growth stages, or because yield production was not limited at that time by incident radiation. In the plant pathological literature little attention has been paid to such effects, although Romig and Calpouzos (13) noted the existence of threshold levels of disease in their crop loss studies, and King (6) also demonstrated threshold levels in his studies on yellow rust on wheat. It may be argued that the tolerance of plants to disease is a nonspecific character that is inherent to a particular cropping situation and may have no direct relationship to disease. In given situations, certain cultivars may, at some or all stages of growth, have spare production or compensation capacity, and if a potential pathogen influences the crop at these stages, there will be no reaction to the presence of the pathogen in terms of yield reduction. In a different environment, these same cultivars may not show such a lack of response to the presence of disease. This suggestion is supported by the fact that many cultivars described as tolerant have low yield potential in the absence of disease as shown by Kramer et al (7) in their work with leaf rust of barley. Also, Roberts (12), working with *Puccinia recondita* on wheat, suggested that tolerance may not be stable since he was able to change the degree of yield response of different cultivars by varying the time of inoculation or the environment at the time of attack. Ellis (2) showed that low-yielding local cultivars of maize were unaffected by moderate infection of *Puccinia polysora*, and postulated that these cultivars may not express full capabilities of their metabolism and the development of grain yield. "This," he suggested, "would allow for a reservoir which absorbs the inroads of disease, and until it was exhausted no loss in yield would be encountered."

In view of the above discussion, I believe that the concept of tolerance to disease requires reappraisal, and that true disease tolerance (15) in fact may not exist. Schafer himself suggested, in view of our imprecise understanding of the concept at that time, that tolerance to disease eventually may be explained as a type of resistance and that the term be restricted to the lack of or reduced reaction to a pathogen (ie, not to disease). Slow-rusting is a possible example of this, and I would add to Schafer's comment that tolerance may be explained as a more general characteristic of crops. The following criteria and guidelines are suggested for identification of the possible existence of tolerance to disease:

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1. Potential yield should be equivalent to that of nontolerant cultivars.
2. Studies should be conducted in cropping situations and not in single-spaced plants, single rows, or hill plots. Competition between plants is an important factor in the analysis of any crop loss vs disease interactions.
3. Disease should be measured by several different parameters, including host-based parameters such as total leaf area, senescence, etc. As techniques are refined, measurements in crops of host factors such as transpiration, photosynthesis, partitioning of assimilates may become useful in this context.
4. Disease should be measured frequently during the whole epidemic.
5. Yield should be analyzed as final harvested weight per unit area, and as primary yield components to identify more closely the development of yield potential. Further analysis (see Gaunt [5]) may be required if it is necessary to define more closely the stage of growth at which losses were induced.
6. If possible, yield quality should also be determined.

The existence of tolerance to disease requires further investigation before the concept may be accepted or rejected as a breeding objective (11,14). By the type of analysis described above, the plant characteristics which confer spare production capacity may be defined as breeding objectives to ensure the existence of disease threshold levels below which yield is not affected. Tolerance to disease as a term is then redundant, and tolerance to pathogens may be restricted to descriptions of the situation in which there is susceptibility to infection and full expression of "signs," but either lesser or no symptoms of disease are evident.

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