Editorial

Disease Resistance in Crop Plants

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Twenty-five years ago graduate students in plant pathology were taught that the approach to developing disease resistance in crop plants was first to find a source of resistance. The next task was to determine the mode of inheritance. If it turned out to be polygenic, then the material was frequently abandoned and the search was continued until a monogenic source could be found. Once located, and assuming disease screening techniques were adequate, we had only to backcross into a desirable genotype.

No instructions were made available to farmers and extension personnel relative to gene management; in fact, we were not particularly concerned with such things. Our objectives were merely to find the genes, work them into acceptable genotypes, and release them to a world in which they were certain to be abused. We sat back and waited for races to develop. We knew they would occur and the job would have to be repeated. But that was the way it was and apparently had to be. With a few exceptions, developing disease resistance in crop plants amounted to biological manipulations offering only temporary effectiveness. We were playing games with nature and expecting always to lose.

Paradoxically, development of disease-resistant crop plants was considered a great technological triumph, amounting to one of the most important human-directed endeavors in all of biology. The activity was far too important economically to be stopped, but the discipline was deeply troubled.

That was the situation 25 years ago. Today the prospects of "boom or bust" cycles and the frustrations of never-ending gene-for-gene struggles no longer loom as inevitable. It is time to announce to our fellow plant pathologists, who might have lost touch with disease resistance research, that the dismal dilemma no longer exists. Research in this vital area has progressed splendidly during the past two decades. Our concepts now embrace epidemiology, resistance gene quality, and qualitative and quantitative aspects of pathogen population dynamics as integral and inseparable components of disease resistance programs. Objectives of many disease resistance breeding projects logically include devising gene management schemes designed to promote stable deployment of genes. An appreciation of these concepts has enabled us to replace instability with stability. I do not mean to imply that we no longer must contend with races and instability. These advances reflect the present state of the science. Technology has by no means kept up, and I think several reasons are obvious.

In the first place, many plant breeders, for various reasons, hold on to the concepts and techniques of the early 1900s. Also, I would imagine that most farmers would not even know the meaning of "gene management," indicating insufficient effort expended by extension plant pathologists. Another reason for the continuing problem relates to activities of farmers who have been informed but continue to operate with naive disregard for biology. These "agricultural miners" show no interest in practicing gene management.

As an example, consider the I gene that has been used throughout the world to condition for Fusarium wilt resistance in tomato. Race 2 of *Fusarium oxysporum* f. *lycopersici*, which compromises the I gene, has been reported widely but sporadically. Stable, nonspecialized populations of the fungus (to which the I gene confers resistance) have demonstrated almost indefinite field survivability in the absence of tomato, but populations of race 2 were shown to be reduced to below detectable levels after only 2 years' absence of tomato. Therefore, by giving minimum attention to gene management, the invaluable I gene would continue to be effective indefinitely. In several of the most important tomato-producing areas in the United States, however, the decision has been not to rotate. As a result, plant pathologists have resorted widely to use of gene I_2, which conditions for resistance to race 2. I understand the pressure my colleagues are under, but this doesn't change the fact that our heritage of resistance genes is being squandered.

We are not likely to overstate the importance associated with developing the potential for stably deploying monogenic or oligogenic disease resistance, but I feel that the most important and exciting advances have occurred with polygenic resistance. About the only positive idea we had 25 years ago relative to polygenic resistance was that it appeared to be highly stable. We purposely tried to steer clear of it because we considered it more or less unusable. We thought it was difficult to detect and that complicated techniques requiring excessively large populations and unacceptably long periods of time were required for transferring the resistance into desirable genotypes. And no more than mediocre-level resistance was expected.

Fortunately, we were wrong on every negative idea. Usable levels of polygenic resistance can easily be detected, even in plants with monogenic or oligogenic resistance. Small-population techniques have been developed that effectively accumulate the factors for polygenic resistance into desirable genotypes in relatively short periods of time. Although extremely high levels of polygenic resistance would be unnecessary or even undesirable in many cases, we certainly are not limited to low or mediocre levels. In experimental models, polygenic resistance to specific diseases has been extended to levels approaching immunity.

With polygenic resistance, the techniques accommodate development of resistance to several diseases simultaneously. Resistance from a wild relative can certainly be utilized, but we are less dependent on exotic imported germ plasm than with monogenic resistance. A single source of resistance is not necessary, nor are we limited to sources of high resistance. In fact, with polygenic resistance the final product may be far more resistant than any source used in its development. Conceivably, susceptible crop varieties could be the only sources for development of a resistant variety. Thus, vast untapped sources of stable disease resistance are readily available for crop improvement.

These ideas have not been universally accepted and the techniques have been tested on only a few crops, but this will change with time. I am convinced that we should continue in the future to use monogenic resistance in many instances. However, the emphasis will shift during the 1980s to polygenic resistance.