Interpreting Residual Effects of "Defeated" Resistance Genes

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Recently Nass et al (4) used near-isogenic winter wheat lines to attempt to determine the effect of individual genes for resistance to *Erysiphe graminis* (the wheat powdery mildew pathogen). Compared to the susceptible cultivar Chancellor, lines near-isogenic to Chancellor and carrying mildew resistance genes *Pm*3c, *Pm*4, or *MA* had lower numbers of sporulating colonies and lower spore production per lesion when inoculated with *E. graminis* isolate 144, even though this isolate is considered virulent against these genes on the basis of its infection type. They concluded that these effects on isolate 144 were residual effects of *Pm*3c, *Pm*4, and *MA*. The observed reduction in infection and sporulation would reduce the apparent infection rate, *r*. Nass et al (4) concluded that their data conflicted with prevailing resistance theory, and as representative of such theory they cited Vanderplank (7,8) as attributing reduction in *r* solely to genes for race-nonspecific, horizontal resistance.

While Vanderplank (page 23 in reference 8) did make a statement to this effect in an abstract, it referred to a simplified and generalized case and should not be taken out of context. Vanderplank (8) provided for exceptions to the generalizations that race-specific, vertical resistance reduces the effective initial inoculum, X_0, while horizontal resistance reduces *r*, by giving examples in which vertical resistance reduced *r* (page 192 in reference 8) and horizontal resistance reduced X_0 (page 120 in reference 7). More importantly, his concept that vertical resistance generally does not reduce *r* is restricted to resistance high enough to stop the pathogen from reproducing on the host (page 120 in reference 7). Clearly, partial or minor gene resistance, such as that observed by Nass et al (4), will reduce the *r* of some or all races whether the resistance is vertical or horizontal.

Examination of the data of Nass et al (4), to compare the number of sporulating colonies on Chancellor versus the near-isogenic lines, reveals that the differences in means ranged from 287 to 2,567% of the mean differences (Table 1 in reference 4). This indicates either that errors in calculation were made or that a powerful, but unstated, transformation was used in the statistical analysis.

Rigorous investigation of host or pathogen genes requires that the genes be present in a random or uniform genetic background so that effects of the genes under study are not confounded with those of other genes. If the genetic background is random, a sample population of genotypes must be used that is sufficiently large to average out any effects of the randomly segregating genes. Alternatively, near-isogenic lines can be used to approximate a uniform genetic background so that a large sample is not required. Unfortunately, the development, maintenance, and verification of near-isogenic lines is fraught with technical difficulties. Whether the "residual" effects on isolate 144 ascribed to genes *Pm*3c, *Pm*4, and *MA* (4) are actually due to these genes depends on whether the near-isogenic lines are in fact isogenic for any quantitative resistance genes against which isolate 144 is avirulent. The observation that secondary colonies formed around the primary colonies on the recurrent susceptible parent Chancellor but not on the near-isogenic lines produced by backcrosses to Chancellor (4) suggests that the lines were not as nearly isogenic as intended and that quantitative resistance genes may have been transferred from the resistant parents to these lines, along with the *Pm* resistance genes. Three mechanisms could account for the transfer of unidentified quantitative resistance genes to the near-isogenic lines during their breeding. The first mechanism is gene linkage. For example, nine loci for resistance to barley powdery mildew are located on barley chromosome 5 (3). Linkage can significantly slow the approach to the isogenic state and is difficult to detect when genes for quantitative characters such as infection efficiency and sporulation per lesion are involved. The second mechanism is genetic drift. The data presented by Nass et al (4) suggests that one near-isogenic line, *Pm*2+, had a greater number of sporulating colonies than Chancellor; two lines, *Pm*2 and *Pm*5, had about the same number; and three lines, *Pm*3c, *Pm*4, and *MA*, had a lesser number. This range of results in a quantitative trait is compatible with the hypothesis that genetic drift of quantitative resistance genes, unrelated to the *Pm* genes, occurred during the breeding of the near-isogenic lines. (Data on paired comparisons of the mean number of sporulating colonies on Chancellor and the near-isogenic line with *Pm*2+ were not presented (Table 1 in reference 4), although it was reported that differences between these lines were not statistically significant. However, results of subsequent relative infection efficiency experiments 1 and 2 appear reliable since they are well correlated with each other and with previous relative disease efficiency tests (Tables 1 and 2 in reference 4). The near-isogenic line with *Pm*2+ had 76% more sporulating colonies per leaf than Chancellor in experiment 1 and 58% more in experiment 2 (Table 2 in reference 4).) The third mechanism is disease selection pressure during the breeding process, but there was no evidence of this. If quantitative resistance genes were transferred to the near-isogenic lines by one of the above mechanisms, then the results observed (4) may be due to unidentified genes rather than residual effects of *Pm*3c, *Pm*4, and *MA*.

Support for the hypothesis of Nass et al (4) comes from the observation that radioactive sulphur transfer to virulent culture MS-76 was less from a near-isogenic line with *Pm*1 than from Chancellor (6). Martin and Ellingboe (2) used three isolates virulent for infection type on *Pm*4. A near-isogenic line with *Pm*4 had fewer infections than Chancellor with isolates MS-3 and Kh×Cc[7]. In contrast, the line with *Pm*4 had the same number of infections as Chancellor when inoculated with isolate MS-2, although infection development was 2 hr slower. Therefore, any residual effects of *Pm*4 were not effective against all isolates. Martin and Ellingboe pointed out that the differences among isolates could be due to multiple allelism or to effects of modifier loci in the pathogen. The fact that mutant pathogen strains of several levels of tolerance are often obtained in fungicide tolerance screening trials suggests that suboptimal multiple alleles and/or modifier genes are common in pathogens. If "defeated" major resistance genes have residual effects against certain isolates, and if this is only because these isolates have suboptimal virulence alleles, then such residual effects would be likely to disappear under field conditions as more virulent strains, similar to MS-2, become predominant through natural selection. Such selection may occur rapidly since the mutation rate towards virulence in *E. graminis* may be as high as 2,000/locus/ha/day (9). Furthermore, residual effects might mask, and therefore hinder selection of, other genes for slow mildewing in breeding plots.

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It is now clear that there are several distinct hypotheses about effects of host/pathogen genotypes that condition a susceptible infection type. For practical purposes the apparent infection rate can be chosen as the measure of any such genotype effects. Let *px* and *Px* represent pathogen genotypes at locus *x* for virulence and avirulence, respectively; let *pmx* and *Pmx* represent the corresponding host genotypes for susceptibility and resistance; and let *r* represent the apparent infection rate as measured using pathogen strains isogenic except at locus *x* and host lines isogenic except at locus *x*; or, alternatively, let *r* represent the average of a large sample of apparent infection rates where the host or pathogen alleles being compared are present in a random genetic background. (If modifier alleles or multiple alleles for virulence or resistance exist, each must be treated as an individual case.) In all hypotheses $r(Px/Pmx)$ is very much less than r of all other genotypes and can be disregarded. The first hypothesis is that $r(px/Pmx) = r(px/pmx) = r(Px/pmx)$. The second hypothesis (7) is that $r(px/Pmx) = r(px/pmx) < r(Px/pmx)$. The third hypothesis (5) is that $r(Px/pmx) < r(px/Pmx) = r(px/pmx)$. The fourth hypothesis (4) is that $r(px/Pmx) < r(px/pmx) = r(Px/pmx)$. The theories behind the second and fourth hypotheses could both hold, in which case $r(px/Pmx) < r(px/pmx) < r(Px/pmx)$. The theories behind the third and fourth hypotheses could both hold, in which case $r(px/Pmx)$ could be lower, equal to, or higher than $r(Px/pmx)$ but both would be lower than $r(px/pmx)$. Thus, all these alternatives are clearly distinguishable from each other. There are 16 possible sequences of the three susceptible genotypes, but the eight that are not listed above do not correspond to any known theory and are therefore improbable. A thorough evaluation of the hypotheses will require progeny analysis of suitably large samples from crosses of host cultivars and crosses of pathogen strains. Analogous hypotheses can be proposed about the effects of resistance genes on host yield in the absence of disease, and accurate yield tests of near-isogenic lines could provide some information on such effects.

Progeny analysis of crosses between Chancellor and the near-isogenic lines with *Pm*3c, *Pm*4, and *MA* could be used to eliminate the possibility that unidentified unlinked genes are responsible for resistance of the near-isogenic lines and to reduce the possibility that unidentified linked genes are responsible. Hopefully, future tests of isolates will be on adult plants rather than on 14-day-old seedlings as used by Nass et al (4), since results with seedlings and adult plants are poorly correlated (1) and seedling response is of lesser epidemiological interest. Genetic studies on host-pathogen relationships require tests of inoculum purity, and papers should report that these have been performed.

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