The genomics of stem rust resistance in wheat

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CATEGORIZING GENES FOR RESISTANCE

Genes for stem rust (*Puccinia graminis Pers*. f. sp. *tritici*) resistance having a variety of effects have been described. Often genes controlling the same type of resistance have been given several different names. Many genes have been postulated but were not sufficiently distinct to be named. The following are some of the categories that have been used.

1. Morphological resistance. In some of the earliest work on rust resistance, the effect on resistance of characters such as stomatal size and frequency, and stem composition were measured (e.g., Hart 1931). The effects were often not large and genetic studies were difficult or impossible.

2. *Physiological, hypersensitive, major, monogenic, race-specific, vertical and gene-for-gene resistance*. Race-specific and gene-for-gene provide the clearest description of this type of resistance. The resistance is effective only against specific rust races and there is a specific interaction between the products of the genes for resistance and the products of the corresponding genes for avirulence. The term hypersensitive is misleading. It suggests that the interaction is rapid and results in a high degree of resistance. In fact, gene-for-gene interactions can result in a range of infection types from immunity to very moderate resistance depending on how rapidly resistance develops. Genes for resistance were called major if their effects were large enough to make them readily identifiable as opposed to genes that had only small effects. The distinction probably serves little purpose. Suppressors of race-specific genes appear to be gene-specific and probably belong in this category.

3. *Polygenic, non-hypersensitive, race-non-specific, horizontal, general, minor, partial, slow rusting and residual.* These terms have all been used to describe resistance that often behaves like a quantitative character, is generally complex in inheritance and controlled by several genes. Usually the individual genes have small effects that may be additive or even multiplicative. Residual or ghost resistance results from the postulated residual effect of race-specific genes whose effect has been overcome but some residual effect remains. It is not clear whether these types of resistance have similar methods of genetic control or not. The resistance is often thought to be race-non-specific or general (i.e., effective against all races of rust). However, it is not clear whether this is really the case or the genes have such small effects that it is impossible to detect gene-for-gene effects. Perhaps, this category includes several distinct types of resistance.

Interactions among Genes for Resistance

The stem rust resistance of a wheat plant results from the combined effects of all of its genes that in any way affect its rust resistance. Many genes are involved and have effects of various sizes on a variety of characters. The situation is complicated by the fact that the genes interact with environmental factors such as temperature and light, as well as with one another.

Genes for Specific Resistance

The products of genes for specific resistance interact directly with the products of genes for avirulence in the fungus. An interesting question is whether the products of different genes for specific resistance also interact with one another in any way. It appears unlikely that they do. The evidence indicates that a plant carrying two genes that provide specific resistance to different races is resistant to all races to which either gene provides resistance but not to any additional races. In other words, the two genes do not interact to provide resistance to additional races.

However, there is evidence that genes that condition resistance to the same race may combine to increase the level of resistance to that race. For example, the cultivar Thatcher gives a fleck reaction to race 56 ((MCCD). It has several genes for resistance but none conditions an infection type more resistant than a type 2. Two or more of its genes must combine to produce the fleck type (Green and Dyck, 1975 and Nazareno and Roelfs, 1981. It is not clear how frequently this occurs.

Knott and Weller (1988) did extensive tests on lines carrying combinations of two and three genes for stem rust resistance in the genetic background of the cultivar Marquis. Four near-isogenic lines of Marquis, each carrying one of *Sr7a, Sr8a,* Sr9b *or Sr11,* were inter-crossed to produce the six possible two-gene genotypes and three of the four possible three-gene genotypes. The nine genotypes and the parents were tested separately in growth chambers with each of five rust races, giving 65 combinations in all. In seven of the 45 combinations involving genotypes with two or three genes for resistance, the genotypes gave a detectably lower infection type than their single gene parents. However, none of the differences was large. All seven involved the gene *Sr7a* which conditions an unusual infection type. The presence of *Sr7a* results in fewer pustules, often somewhat variable in size, with extensive yellow chlorosisaround the pustules and along the leaf margins, particularly at the tip. This makes it more difficult to compare its infection type with those of other genotypes. In two of the seven cases, *Sr7a* did not condition resistance to the

tester race but still appeared to interact with another resistant gene or genes to increase resistance.

The same lines were tested in two field nurseries, each inoculated with a different race. At maturity, the rust severity on the lines was recorded in percent. Ten of the 18 genotypes showed a significant reduction in rust severity compared to the most resistant of the parents of the genotype. In the case of the genotype, *Sr9bSr9bSr11Sr11,* in which neither gene conditions resistance to race TMRT, the combination was significantly more susceptible than the most resistant parent, *Sr9bSr9b.*

For a number of cereal rusts including stem rust of wheat, it has been suggested that resistance genes that are no longer effective against a race can have residual or ghost effects. Brodery et al. (1986) studied the effect of genes *Sr6, Sr8* and *Sr9a* against a race virulent on all three. Each gene alone had an effect on pustule size and sporulation. The two gene combinations were more effective and the three gene was the most effective. Pederson et al. (1988) suggested the pyramiding of defeated genes to maintain their effects. Residual gene effects add to the complexity of the inheritance of stem rust resistance.

Genes for General Resistance

By definition, genes for general resistance to stem rust do not interact with specific genes for avirulence in the rust. As noted above, resistance that is probably general can go under a variety of names but it is not always clear that their inheritance is similar. It is generally thought that general resistance is controlled by a few genes, each having a relatively small effect. Some authors have concluded that it is not possible to obtain a high degree of resistance and, therefore, use the name partial resistance. However, there is good evidence in at least some cases that genes with small effects can be combined to produce near immunity (e.g., Knott and Padidam 1988). Their data for six wheat lines lacking genes for specific resistance to race 15B-1 (TMRT) indicated that three or four genes were involved in field resistance and that their effects were probably multiplicative.

Genes for general resistance must have effects in wheat plants that result in resistance to all races of stem rust. This may be from the production of antifungal compounds such as phytoalexins, or compounds that provide a barrier to the fungus such as lignins and waxes, etc. Different types of resistance mechanism s will almost certainly be genetically independent. Consequently, their effects are likely to be independent and may be simply additive or possibly multiplicative.

Niks and Rubiales (2002) analysed the infection process in airborne pathogens like rusts. They divided the process into stages – spore deposition, spore germination and germ tube development, finding a stoma, stoma recognition and appresorium formation, stoma penetration and cell wall penetration, and haustorium formation – and studied them separately. Resistance can occur at any stage. Similarly, different cultivars can affect characters such as the length of the latent period and volume of spore production. These characters are presumed to be controlled by genes having small effects which can be selected for.

Suppressors and Modifiers

The effects of genes for resistance can be modified by genes that do not affect resistance directly but modify the effects of other genes. In the must extreme cases, a gene may completely suppress the effect of a gene for resistance. Although suppressors have been reported occasionally, particularly in interspecific crosses, they are probably more frequent than is realized. For example, Knott (unpublished) selected seven durum cultivars (*Triticum turgidum* L.) that are susceptible to stem rust race TMRT and made 20 of the possible 21 crosses among them. The F_1 plants from all 20 crosses were susceptible to race TMRT. However, the F_2 families from 11 of the 20 crosses segregated for resistance to TMRT. The fact that the F_1 plants were susceptible indicated that pairs of dominant complementary genes were not involved. This was confirmed by the fact that none of the F_2 segregations fit a ratio of 9 resistant to 7 susceptible seedlings. Six of the seven durum wheats were also crossed with LMPG-6S, a susceptible common wheat (*Triticum aestivum* L.). All six crosses segregated for resistance to TMRT. LMPG-6S may carry a resistance gene plus a suppressor and none of the durum cultivars carry the same suppressor as LMPG-6S. However, the variation in the infection types and in the segregations in F_2 families suggest that it is more likely that the durum cultivars carry different resistance genes and suppressors and that LMPG-6S does not carry any of the same suppressors.

The data suggest that suppressors of resistance are much more frequent than has been realized. They almost certainly act against specific genes, not in general. The mechanism by which they suppress a resistance gene is not clear. Their frequency suggests that they must have a selective advantage which maintains them in a population

Other genes that modify the effects of resistance genes but less drastically than suppressors have been postulated. If their effects are small, their presence will be difficult to demonstrate.

Defensins

In recent years, a group of antimicrobial compounds called defensins has been identified in a number of plant species and are probably present in all plant species (Graham et al., 2008). Large numbers of defensins are present in plants. Their mode of action is unknown but they are non-specific. Although it is not known whether defensins are effective against stem rust of wheat, they probably are. Thus, defensins add another layer of complexity to the genomics of stem rust resistance.

CONCLUSIONS

The genomics of stem rust resistance in wheat are extremely complicated. A large number of the genes in wheat are probably involved directly or indirectly.

REFERENCES

N.B. This is not a comprehensive reference list but rather a list of a few key references.

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