# Reduced Receptivity to Infection Associated with Wheat Gene *Lr2c* for Low Reaction to *Puccinia recondita*

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## ABSTRACT

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Tests were made to determine if *Lr* genes in wheat (*Triticum aestivum*) influenced the number of uredinia produced by virulent cultures of *Puccinia recondita*. Near-isogenic wheat lines, having genes *Lr1*, *Lr2c*, or *Lr3a* singly or in various combinations, and their recurrent parent were compared for receptivity to infection. We found that virulent cultures produced as many uredinia with lines having *Lr1* or *Lr3a* as with their recurrent parents. Wheat lines having *Lr2c* produced fewer uredinia than their recurrent parents when inoculated with some virulent cultures, but not when inoculated with other virulent cultures. Preliminary test results revealed differences between lines having *Lr2c* and the recurrent parent; at 15, 20, and 25 C, significantly fewer uredinia were produced with lines having *Lr2c*. These results are similar to those reported by others in that not all of the specific genes tested could be shown to have a residual effect on quantitative aspects of infection. Others have reported that some virulent cultures overcome the residual effect of specific genes, but have not emphasized this aspect of their results. These results indicate that genetic interactions other than those responsible for low or high infection type may affect the number of uredinia produced in these materials.

Flor (5) found that for every gene for resistance in flax, *Linum usititatissimum* L., there was a corresponding gene that conditioned avirulence in *Melampsora lini* (Pers.) Lev. In terms of host:parasite interaction, Flor's discovery indicated that both host genotype and parasite genotype function together to condition an interaction phenotype. Loegering (8,9) has suggested that the association of host and parasite results in a third entity, the aegricorpus, which manifests a phenotype, but that host and parasite only have genotypes that together condition the aegricorpus phenotype with respect to their association.

The discovery of the gene-for-gene relationship by Flor (5) brought about a situation in which previously used terminologies became contradictory and led to ambiguity in that genes for resistance interacting with genes for virulence were said to bring about susceptibility. Interactions without genes for resistance also could bring about susceptibility, however. Upon recognition of this ambiguity, Loegering (8) proposed a terminology and symbolization to describe the various elements of a host:parasite association. He used reaction to describe the host character relating to response to infection by a parasite and pathogenicity to describe the ability of the parasite to cause damage to the host. Infection type was used to denote a specialized kind of phenotype of the host:parasite interaction, the aegricorpus. Loegering chose "low" and "high" in each case to describe the possible allelic condition of a specific host and a specific corresponding parasite locus. He also used low and high to describe the two possible infection type conditions that result from the interaction of a single gene pair in one environment. Within the principle elucidated by Flor (5), the interaction of a host gene for low reaction and a corresponding parasite gene for low pathogenicity conditions a low infection type, while interaction of a gene for low reaction with a gene for high pathogenicity conditions a high infection type. Both interactions involving the gene for high reaction condition a high infection type.

A gene-for-gene relationship has been demonstrated in the association of *Puccinia recondita* Rob. ex Desm. and *Triticum aestivum* L. (17) in the same way that Flor (5) described the relationship in the flax rust system. Dyck and Samborski (3) have shown that reaction to *P. recondita* was simply inherited in several of the International Standard wheat leaf rust differential cultivars. They demonstrated that cultivars Malakof, Loros, and Democrat had genes *Lr1*, *Lr2c*, and *Lr3a*, respectively, and developed three near-isogenic lines, each having one of these *Lr* genes by using cultivar Thatcher as the recurrent parent. Soliman et al (19) studied the same differential cultivars and obtained the same results. Johnston and Heyne (7) developed a set of near-isogenic lines in the cultivar Wichita background. In a further study, Samborski and Dyck (18) found that pathogenicity in *P. recondita* to lines having *Lr1*, *Lr2c*, and *Lr3a* singly was inherited as single Mendelian factors.

The suggestion of Vanderplank (20) that there are two kinds of disease resistance in plants, vertical and horizontal, has created great interest in the possibility of developing cultivars with stable resistance. Many hypotheses have been put forth to explain observations of resistance different from that derived from interaction of genes for low reaction and low pathogenicity. We will address one such hypothesis in this paper. Martin and Ellingboe (11) found that cultures of *Erysiphe graminis* DC virulent to wheat lines having single *Pm* genes produced fewer elongated secondary hyphae with lines having some *Pm* genes than with the recurrent parent cultivar of the lines. They found that this phenomenon was detectable with lines having *Pm4* but not with lines having other *Pm* genes.

Nass et al (15) studied the same host:parasite system and found that fewer sporulating lesions formed with wheat lines having *Pm3c*, *Pm4*, and *PmMA* than with their recurrent parent when inoculated with a virulent culture. They proposed that this may be a widely occurring phenomenon in host:parasite associations and as such could explain "rate-reducing" resistance. If this is a general phenomenon, the resistance conditioned would be stable.

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The study reported here was made to test that hypothesis with *P. recondita* and *T. aestivum*. Our data do not support the hypothesis that a "residual effect" of "defeated" *Lr* genes is a widely occurring phenomenon.

## MATERIALS AND METHODS

Information about 35 genes for low reaction to *P. recondita* in *T. aestivum* was summarized by Browder (1). At least two reports (17,18) have documented the Mendelian inheritance of pathogenicity in *P. recondita* corresponding to many of these *Lr* genes.

Based upon the great trend of evidence from these studies and cited in these reports, we accept a gene-for-gene relationship as the most probable explanation of variation in infection type of *P. recondita*:*T. aestivum*. This premise is made in the same way as the premise of Martin and Ellingboe (11) and of Nass et al (15) for their studies.

**Host lines.** Series of near-isogenic lines having several different *Lr* genes singly in a Thatcher background were available from the work of Dyck and Samborski (3) and in a Wichita background from the work of Johnston and Heyne (7). In addition to these lines, a complete set of eight lines that collectively have all possible combinations of 0, 1, 2, and 3 of the genes *Lr1*, *Lr2c*, and *Lr3a* was available in a Wichita background (C. O. Johnston, E. G. Heyne, and L. E. Browder, *unpublished*).

We chose to work with this group of three *Lr* genes because of the availability of the complete set of lines having gene combinations and because there is adequate evidence of a gene-for-gene relationship with respect to them. This indicates there are indeed three loci in *P. recondita* that correspond directly to these three *Lr* genes and the alternate allelic condition at these parasite loci along with the allelic condition of the wheat locus determines the phenotype of the resulting aegricorpus. We also worked with some near-isogenic lines having other, single *Lr* genes.

The host lines with the Thatcher background that we used are available from the originators, Agriculture Canada, 192 Dafoe Rd., Winnipeg, Manitoba, Canada R3T 2M9. The host lines with the Wichita background are available from the second author.

**Parasite cultures.** With the addition of one *P. recondita* culture, X47B1, kindly supplied by G. D. Statler (Department of Plant Pathology, North Dakota State University, Fargo) we were able to assemble a complete set of eight cultures that collectively had all possible combinations of genes for low pathogenicity to 0, 1, 2, and 3 of the wheat lines having *Lr1*, *Lr2c*, or *Lr3a*. We inoculated this complete set of host lines with the complete set of cultures of *P. recondita*. The infection-type data obtained from these inoculations are presented in Table 1. In some cases, more than one culture was available having a specific pathogenicity. The data obtained were as expected if a gene-for-gene relationship were operating according to the models of Ellingboe (4) and McIntosh and Watson (12) but with three corresponding gene pairs. These data confirm the results of previous studies (3,7,17,18). Based on these results, we derived putative genotypes for pathogenicity of each of the cultures (Table 1). The symbols "*Lp*" and "*Hp*" are used to denote putative genes for low pathogenicity and high pathogenicity, respectively, at a parasite locus. These designations are derived from the presence of an infection type lower than 99P on a wheat line known to have a known *Lr* gene. The symbol "*Lr*" is used to denote that a host has a particular gene for low reaction to *P. recondita*. The symbol "*Hr*" is used to denote the absence of the *Lr* gene at a particular host locus. Virulence is used to describe the ability of a culture to produce an infection type 99P with a particular host line, regardless of the genotype of the host line.

Many of the cultures used are deposited in the American Type Culture Collection, 12301 Parklawn Drive, Rockville, MD 20852, and are identifiable by their PRTUS numbers (Table 1). The other cultures are available from the second author.

Cultures will be indicated in the Results section and in the Discussion section by culture numbers as abbreviations of the PRTUS culture names shown in Table 1; for example, culture 6 for PRTUS6.

**Methods.** All experiments were conducted using seedling plants grown in a greenhouse at about 20 C. In experiments planned for infection-type data, plants were inoculated with an oil:urediniospore suspension of the various cultures just before emergence of the second leaf, placed in a moist chamber overnight and transferred to a growth chamber at 20 C and a 12-hr photoperiod. Infection types were observed 10 days after inoculation. The infection-type coding system of Browder and Young (2) was used to record these observations. In that system, relative sporulation and relative lesion size are coded in a 0 to 9 scale, with 0 as none and 9 as the maximum amount or size observed for the host:parasite system. An alphabetic descriptor code was used in each infection-type code to describe the area around lesions, such as C for chlorotic or P for pale.

In experiments planned for counting relative numbers of uredinia formed with wheat lines having and not having specific *Lr* genes, plants were grown in 8-cm-diameter pots in the greenhouse with one or two seedlings of each line in each pot. Each pot contained all the lines to be compared in the experiment. Just before emergence of the second leaf, plants were inoculated at the same time in a quantitative inoculating machine similar to those described by Mortensen et al (14) and by Melching (13). Three milligrams of urediniospores were used to inoculate the plants in each group of 16 pots within each single inoculation. Inoculated plants were placed in a chamber and subjected to a fine water mist produced by a mechanical continuous-flow nebulizer for about 18

TABLE 1. Infection types observed on near-isogenic wheat lines in a cultivar Wichita background in interaction with different cultures of *Puccinia recondita* in the growth chamber at 20 C

| Host near-isogenic line | PRTUS culture number: / Host genotype | 1 | 3,8,19 | 20 | 4,17, 15 | X47B1 | 7,14 | 5,11 | 6,9,18, 21,22 23 |
|---|---|---|---|---|---|---|---|---|---|
| | | *Lp1*[y] *Lp2c* *Lp3a* | *Lp1* *Lp2c* *Hp3a* | *Lp1* *Hp2c* *Lp3a* | *Lp1* *Hp2c* *Hp3a* | *Hp1* *Lp2c* *Lp3a* | *Hp1* *Lp2c* *Hp3a* | *Hp1* *Hp2c* *Lp3a* | *Hp1* *Hp2c* *Hp3a* |
| LR1LR2CLR3A(WI) | *Lr1Lr2cLr3a* | 01C[z] | 01C | 01C | 01C | 03C | 03C | 03C | 99P |
| LR1LR2C(WI) | *Lr1Lr2cHr3a* | 01C | 01C | 01C | 01C | 03C | 03C | 99P | 99P |
| LR1LR3A(WI) | *Lr1Hr2cLr3a* | 01C | 01C | 01C | 01C | 03C | 99P | 03C | 99P |
| LR1(WI) | *Lr1Hr2cHr3a* | 01C | 01C | 01C | 01C | 99P | 99P | 99P | 99P |
| LR2CLR3A(WI) | *Hr1Lr2cLr3a* | 03C | 03C | 03C | 99P | 03C | 03C | 03C | 99P |
| LR2C(WI) | *Hr1Lr2cHr3a* | 03C | 03C | 99P | 99P | 03C | 03C | 99P | 99P |
| LR3A(WI) | *Hr1Hr2cLr3a* | 03C | 99P | 03C | 99P | 03C | 99P | 03C | 99P |
| Wichita | *Hr1Hr2cHr3a* | 99P | 99P | 99P | 99P | 99P | 99P | 99P | 99P |

[y] Culture names are abbreviated to the number of PRTUS (*Puccinia recondita tritici* United States) culture series in the American Type Culture Collection; these names are for purposes of retrieval only and do not relate systematically to pathogenicity. Putative genotypes for pathogenicity of these cultures at loci 1, 2c, and 3a are given in three rows below the culture name and above the infection types produced by these cultures with the host lines indicated in the rows.

[z] Infection types are coded at 0 to 9 estimates of relative sporulation, as 0 to 9 estimates of lesion size, and end with an alphabetic descriptor of tissue around the sporulating area of the pustule. In this table, C indicates chlorosis and P indicates a pale zone around the sporulating area.

hr at 20 C. Plants were then removed from the moist chamber and placed in growth chambers. Some experiments were conducted to compare relative numbers of uredinia produced by lines of different genotypes inoculated with cultures of different genotypes at latent period temperatures of 15, 20, and 25 C. Materials in other experiments were grown only at a 20 C latent period. Numbers of fully developed uredinia were counted on the primary leaf of 20–35 plants for each host-line/parasite-culture/temperature-treatment unit. Leaf areas on which uredinia were counted were calculated by multiplying the leaf lengths by leaf widths by 0.74. The factor 0.74 was obtained by plotting the product of leaf length times leaf width and the actual leaf area of a 20-leaf sample. The leaf margins were traced on graph paper; 0.74 was found to be the slope of the line. The total number of uredinia on each leaf was divided by the leaf area to obtain the number of uredinia per square centimeter. Some experiments were repeated twice; others were repeated only once because some inoculations failed for unknown reasons.

**Statistical analysis.** The SAS statistical package (6) was used to conduct one-way analysis of variance tests. No significant differences were found due to inoculations; thus, data from two inoculations were combined. Duncan's multiple range test was used to separate means of number of uredinia on each line in each experiment. Statistical comparisons were made only among lines inoculated with the same culture because we had no adequate method to account for possible differences in aggressiveness among the different cultures that were used. We did, however, make one comparison among cultures by expressing receptivity as percentage of uredinia that developed with a near-isogenic line relative to the number of uredinia that developed with the recurrent parent in the same inoculation.

## RESULTS

**Experiment 1.** Culture 6 produced infection type 99P with all eight near-isogenic lines in the Wichita background (Table 1). This set of host lines and this culture were selected for a preliminary experiment to evaluate the effect of *Lr1*, *Lr2c*, and *Lr3a* on receptivity to infection. The mean numbers of uredinia per square centimeter produced by culture 6 are presented in Table 2. Variation in the number of uredinia showed significant effect due to host genotype. There were two distinct groups within the eight lines. Lines LR1(WI), LR3A(WI), and LR1LR3A(WI) did not differ significantly from Wichita in receptivity to infection by this virulent culture. All lines having *Lr2c* had significantly fewer uredinia per square centimeter than did Wichita and the near-isogenic lines having *Lr1* or *Lr3a*. Reduction in numbers of uredinia ranged from 24 to 57% from that of Wichita.

Significant reduction in number of uredinia per square centimeter occurred at all three temperatures (Table 2).

*Experiment 2.* Another experiment was conducted with the same set of eight near-isogenic lines in the Wichita background, but with cultures 17, 9, and 4. Cultures 17 and 4 produced infection type 99P only with lines lacking *Lr1*; culture 9 produced infection type 99P with all of the eight lines (Table 1). Culture 17 produced significantly fewer uredinia per square centimeter with the lines having *Lr2c* than with the lines having *Lr3a* or with the Wichita recurrent parent (Table 3). Although cultures 4 and 17 produced similar infection types with all the lines (Table 1), analysis of data from inoculations with culture 4 revealed no significant differences in the numbers of uredinia per square centimeter with any of the four lines that developed infection type 99P, while differences with culture 17 were observed (Table 3). Culture 9, which produced infection type 99P with all of the eight lines, did produce significantly fewer uredinia per square centimeter with lines having *Lr2c* than with the other four lines that did not have *Lr2c* (Table 3). Relative to Wichita, the average reduction in number of uredinia per square centimeter was 18 and 20% with LR2C(WI) and LR2CLR3A(WI), respectively.

**Experiment 3.** Near-isogenic lines having *Lr2a* or *Lr2c* were available both in a Wichita and in a Thatcher background. A line having *Lr2b* was available only in a Thatcher background. We conducted a further experiment with these five lines along with Thatcher and Wichita. In this experiment, we used cultures 9, 6,

TABLE 2. Mean number of uredinia per square centimeter on primary leaves of a set of near-isogenic wheat lines and their recurrent parent infected with culture PRTUS6[y] of *Puccinia recondita* and grown at three latent period temperatures

| Wheat line: | Uredinia per square centimeter produced at: | | |
|---|---|---|---|
| | 15 C | 20 C | 25 C |
| Wichita | 21 a[z] | 35 a | 51 a |
| LR1(WI) | 20 a | 37 a | 48 a |
| LR3A(WI) | 20 a | 34 a | 50 a |
| LR1LR3(WI) | 19 a | 39 a | 53 a |
| LR1LR2CLR3A(WI) | 12 b | 16 c | 32 b |
| LR2CLR3A(WI) | 12 b | 23 b | 38 b |
| LR1LR2C(WI) | 12 b | 22 b | 36 b |
| LR2C(WI) | 9 b | 15 c | 35 b |

[y] PRTUS cultures are referred to only by their numbers in the text.
[z] Means within the same column followed by the same letter are not significantly different according to Duncan's multiple range test, $P = 0.05$.

TABLE 3. Mean number of uredinia per square centimeter on primary leaves of a set of near-isogenic lines of wheat inoculated with three cultures[x] of *Puccinia recondita* and grown in a growth chamber at about 20 C

| Wheat line: | Uredinia per square centimeter produced with: | | |
|---|---|---|---|
| | PRTUS17 | PRTUS9 | PRTUS4 |
| LR2CLR3A(WI) | –[y] | 50 a | – |
| LR1(WI) | – | 49 ab | – |
| Wichita | 34 a[z] | 49 ab | 38 a |
| LR3A(WI) | 37 a | 45 abcde | 39 a |
| LR2CLR3A(WI) | 26 b | 45 abcde | 35 a |
| LR1LR2C(WI) | – | 44 bcde | – |
| LR2C(WI) | 27 b | 40 cde | 38 a |
| LR1LR2CLR3A(WI) | – | 39 e | – |

[x] PRTUS cultures are referred to only by their numbers in the text.
[y] A minus (–) indicates low infection type; no pustules developed.
[z] Means within the same column followed by the same letter were not significantly different according to Duncan's multiple range test, $P = 0.05$.

TABLE 4. Mean number of uredinia per square centimeter on primary leaves of Wichita and Thatcher near-isogenic lines having different *Lr* alleles at the *Lr2* locus infected with three cultures[x] of *Puccinia recondita* and grown in a growth chamber at about 20 C

| Wheat line: | PRTUS9 | PRTUS6 | PRTUS4 |
|---|---|---|---|
| Wichita | 49 ab[z] | 50 a | 48 a |
| LR2A(TC) | 49 ab | –[y] | 44 a |
| Thatcher | 47 ab | 41 b | 49 a |
| LR2A(WI) | 46 abc | – | 44 a |
| LR2B(TC) | 45 abcde | – | 42 a |
| LR2C(WI) | 40 cde | 34 c | 47 a |
| LR2C(TC) | 39 e | 33 c | 49 a |

[x] PRTUS cultures are referred to only by their numbers in the text.
[y] A minus (–) indicates low infection type; no pustules developed.
[z] Means within the same column followed by the same letter were not significantly different according to Duncan's multiple range test, $P = 0.05$.

TABLE 5. Relative numbers[a] of uredinia of *Puccinia recondita* produced on seedling leaves of wheat line Lr2C(WI) and its recurrent parent cultivar, Wichita

| Wheat line or cultivar | Cultures of *P. recondita* | |
|---|---|---|
| | PRTUS4[a] | PRTUS6[a] |
| LR2C(WI) | 68 | 98 |
| Wichita | 100 | 100 |

[a] Values derived from data in Table 4.
[b] American Type Culture Collection number.

and 4. Because culture 6 produces low infection types with lines having *Lr2a* or *Lr2b*, comparisons of only four of the wheat lines with culture 6 were possible in this experiment. In this experiment, a one-way analysis of variance showed significant differences due to host genotype. With culture 6, lines LR2C(TC) and LR2C(WI) produced significantly fewer uredinia per square centimeter than their respective recurrent parents (Table 4). The genetic background in which *Lr2c* occurred did not affect receptivity to culture 6. Wichita also had significantly more uredinia per square centimeter than did Thatcher. Culture 4 produced infection type 99P with all lines in this experiment. No significant differences in numbers of uredinia per square centimeter were found in this experiment among any of the lines inoculated with culture 4. Culture 9 produced infection type 99P with all of the wheat lines in this experiment. As in the previous experiment, significantly fewer uredinia per square centimeter were produced by culture 9 with lines having *Lr2c* than with lines not having *Lr2c* (Table 4).

## DISCUSSION

The lower receptivity to infection by *P. recondita* in lines having *Lr2c* than in lines not having *Lr2c* when inoculated with cultures 6 and 17, but the absence of such a difference in lines having *Lr1* or *Lr3a* suggests that there is a residual effect of specific genes that relate to low infection type in some cases but not in others. It is possible, of course, that small differences that we were not able to detect occurred in all three cases.

In further studies with lines having *Lr2c*, culture 4 overcame the quantitative residual effect, culture 17 did not. Uredinia counts from inoculations with culture 9 did not separate lines having and not having *Lr2c*. This suggests to us that the low receptivity to infection is possibly controlled by specific corresponding gene pairs. We had no adequate method of completely eliminating the possible effects of differences in aggressiveness in the cultures that we used. On the other hand, if receptivity is a host:parasite interaction character, differences in parasite genotype at loci corresponding to genes for receptivity would be a basis for differences in aggressiveness.

In comparing relative results among lines inoculated with different cultures in the same experiment, we found that we could demonstrate differences in lines with some cultures but not with other cultures. This is similar to the results of Martin and Ellingboe (11) that showed differences in elongated secondary hyphae formed in different parasite/host interactions of *Erysiphe graminis* and *T. aestivum*. Our results imply that cultures are different with respect to infectivity to lines having *Lr2c*. One possible way of adjusting for differences in aggressiveness between two cultures is to calculate the percentage of uredinia per square centimeter produced with a line having *Lr2c* relative to the number produced with the recurrent parent with the same cultures in the same experiment. We used data from Table 4 to make such a comparison (Table 5).

This method provides a measure of difference in the ability of two cultures to cause infections with LR2C(WI) because their infectivity to Wichita is used as the base of 100% in both cases. No statistical test of the difference between 68 and 98% is available; however, the numerical difference is quite large. In such a comparison, it is clear that differences in LR2C(WI) and Wichita occurred with culture 6, but not with culture 4. Several other such comparisons showing similar differences are possible with our data.

A possible explanation of the data obtained is that there is a host gene closely linked to *Lr2c* that was transmitted with *Lr2c* in developing LR2C(TC) and LR2C(WI) and that the interaction of this gene with a corresponding gene in *P. recondita* results in reduced receptivity. Several reports indicate that the *Lr2* locus in wheat is a complex locus (3,10,18,19).

Although our data are generally in agreement with those of Martin and Ellingboe (11) and Nass et al (15), we do not interpret our data to support the hypothesis that specific genes related to production of low infection type have a residual effect on quantitative aspects of infection when inoculated with any virulent culture. Based on our results, the residual effect of the gene for low reaction is observed with some cultures but not with others. Royer et al (16) have recently published similar results from experiments with *E. graminis* and *T. aestivum*. Thus, we would not expect that resistance due to such a phenomenon with respect to any specific gene would necessarily be stable in time and space.

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