An Appraisal of Stem and Leaf Rust Resistance in North American Hard Red Spring Wheats and the Probability of Multiple Mutations to Virulence in Populations of Cereal Rust Fungi

J. A. Kolmer, P. L. Dyck, and A. P. Roelfs

First and second authors: Agriculture Canada, Research Station, 195 Dafoe Rd., Winnipeg, MB, Canada R3T 2M9; third author: Cereal Rust Laboratory, USDA-ARS, Department of Plant Pathology, University of Minnesota, St. Paul 55108.

Contribution No. 1424, Agriculture Canada.
Paper 18,227, Scientific Journal Series Minnesota Agricultural Experiment Station.
Accepted for publication 5 September 1990 (submitted for electronic processing).

In a recent letter to the editor, Mundt (14) evaluated the hypothesis that combinations of resistance genes (pyramids) owe their durability of resistance to a low probability of mutation to the corresponding virulences in the pathogen population. Data of Green and Campbell (8) were used to support the argument that the number of stem rust (*Sr*) genes present in wheat cultivars grown in western Canada has no relation to the longevity of their resistance to stem rust. While we do not necessarily disagree with Mundt's final conclusion that the probabilities hypothesis cannot be properly tested given our current state of knowledge, we feel that the nature of stem rust resistance in North American hard red spring wheat cultivars was misrepresented. We wish to present a more complete analysis of stem and leaf rust resistance in these wheats and also to comment on the evidence concerning multiple mutations to virulence in field populations of cereal rusts.

Spring wheat cultivars bred for the rust area of the eastern Canadian prairies have been derivatives of either Thatcher (released in 1935) or Renown (1937). Stem rust resistance in Renown was derived from Yaroslav emmer through H-44a-24, and that in Thatcher was derived from Iumillo durum (17). Renown and Thatcher are usually moderately resistant to stem rust in the rust nursery at Winnipeg, with the notable exception of more severe and susceptible responses from 1950 to 1963, when race 15B-1 (Canadian race C10) was present at high levels in the nursery (8) (Table 1). These cultivars and their derivatives have shown resistance to stem rust in western Canada since the decline of race 15B-1 began after 1954. Mundt's Table 1 (14) and Green and Campbell's Table 2 (8) are not entirely correct in listing Thatcher and Renown as susceptible to stem rust in 1978. Renown was clearly resistant in the 1978 rust nursery (Table 1; also Table 4 in reference 8), and Thatcher, although given a susceptible rating, was moderately resistant for a number of years before and after 1978. Furthermore, in other areas of the 1978 nursery, stem rust readings of 30 MS (30% severity, moderately susceptible infection type) and 50 MS were recorded for Thatcher (P. L. Dyck, *unpublished data*). Also, Thatcher is more severely rusted at higher inoculum densities; at lower disease intensities the stem rust severity on Thatcher is much lower than on susceptible check cultivars. In one study (15) with the same race predominating, stem rust severities on Thatcher were 70 and 49% at two different locations, whereas on the susceptible check cultivar Baart, rust severities were 89 and 84%, respectively. At no time since the decline of race 15B-1 has Thatcher been considered completely susceptible to stem rust.

Mundt (14) concluded that the longevity of stem rust resistance has little relation to the number of *Sr* genes present in a cultivar. This conclusion was based on the number of *Sr* genes listed for selected cultivars in Table 2 of Green and Campbell (8). Upon further examination of the stem rust resistance of wheats grown in western Canada, it is evident that cultivars with combinations of effective resistance genes have been more resistant over a long period of time than closely related cultivars that have fewer of the same resistance genes.

The *Sr* genes listed in Renown (8,14) (and its derivatives Redman and Regent) are *Sr2*, *Sr7b*, *Sr9d*, and *Sr17*. *Sr7b* is ineffective against all of the predominant races of stem rust (Table 2); *Sr9d* confers resistance only to race C17 (race 56) and is ineffective against all other common races; *Sr17* is effective against races C18 and C33 and ineffective against races C10, C17, and C53. The adult plant resistance gene *Sr2* is most likely effective against all of these races with the exception of C10. Gene *Sr2* confers a high level of resistance; however, in heavily infected nurseries, plants with *Sr2* may have large uredinia at the nodes and in the spike (20), thus giving the appearance of susceptibility. Of the genes in Renown, only *Sr2* conditions resistance to race C53, which has been the most common race since 1977 (6). Mundt (14) stated that *Sr2* alone cannot explain the durability of stem rust resistance in Selkirk. However, the long-lived resistance of the Renown derivatives (including Selkirk) since the decline of race 15B-1 most likely results from the effectiveness of *Sr2* acting alone or in combination with *Sr9d* and *Sr17*. The cultivar Eagle, with only *Sr2*, maintained adequate resistance when grown in Kansas from 1974 to 1982 on over 500,000 ha annually (A. P. Roelfs, *unpublished data*). Vanderplank (21) claimed that Selkirk (*Sr2*, *6*, *7b*, *9d*, *17*, and *23*) was resistant to race C10 (15B-1) because of the combined presence of *Sr6* and *Sr9d*. However, the effect of *Sr9d* was overestimated, because of the presence of *SrH* (10) in the *Sr9d* differential lines. Race C10 is virulent to *Sr9d*, as are all of the common races with the exception of C17. Resistance to C10 in Selkirk is most likely derived from *Sr6*, since Selkirk is a backcross line of Redman.

None of the genes listed in Thatcher (*Sr5*, *9g*, *12*, and *16*; Table 2 of reference 8) confer resistance to any of the common races of stem rust (7,9) (Table 2). *Sr12* conditions resistance to race C17 only (A. P. Roelfs, *unpublished data*), which has not been important for over 20 years. Other genes are apparently responsible for the resistance of Thatcher to stem rust. Brennan (1) and Dyck (*unpublished data*) reasoned that the resistance of Thatcher is due to two recessive genes. Nazareno and Roelfs (15) indicated that this resistance is often associated with *Sr12* in lines derived from Thatcher. In any case, it is evident that independent action of the genes listed in Green and Campbell's Table 2 (8) is not responsible for the resistance of Thatcher to stem rust.

The Thatcher derivatives Manitou, Neepawa, Katepwa, and Columbus have been the predominant cultivars in western Canada since the mid-1960s (Table 1). These cultivars have all been resistant to stem rust since their release. Since detailed genetic studies of stem rust resistance in these cultivars have not been done, their genotypes are hypothetical. However, since Manitou is a Thatcher backcross derivative, it probably has the Thatcher resistance genes plus *Sr6* and *Sr7a*. Although Neepawa was developed by the pedigree method, it is closely related to Thatcher, originating from a cross between a Thatcher backcross line with *Sr7a* (and *Lr13*) and a Thatcher*3/Frontana selection. A limited genetic study of Neepawa (Dyck, *unpublished data*) indicated

TABLE 1. Adult plant reactions to stem rust (percent severity and response[a]) of selected wheat cultivars grown in the rust nursery at Winnipeg during selected years from 1952 to 1987

Cultivar	Year licensed	1952	1954	1957	1963	1966	1969	1975	1978	1981	1984	1987
Marquis	1923	60 S	90 S	80 S	80 S	80 S	80 S	80 S	70 S	60 S	70 S	50 M
Renown	1937	40 M	70 S	35 M	30 M	1 R	2 MR	20 MR	5 M	2 MR	20 MR	10 R
H-44a[b]	...	40 M	60 S	20 M	5 M	5 MR	5 R	5 MR	1 M	20 M	5 VR	5 R
Selkirk	1950		1 R	30 M	20 M	5 M	2 MR	TR	TR	TR	10 VR	5 R
Thatcher	1935	30 M	70 S	30 M	60 S	40 M	30 M	20 M	70 S	30 M	50 M	50 M
Manitou	1965					TR	1 R	TMR	20 M	5 M	20 R	20 R
Neepawa	1969						TR	5 MR	10 M	5 M	10 R	10 R
Columbus	1980										20 M	20 MR
Katepwa	1982										10 R	5 R

[a] Severities according to the modified Cobb scale (16): T = trace infection; VR = very resistant (hypersensitive flecks); R = resistant (flecks and small uredinia); MR = moderately resistant (flecks and small to moderate uredinia); M = mixed reaction (resistant flecks and large uredinia); MS = moderately susceptible (moderate to large uredinia); S = susceptible (large uredinia).

[b] H-44a was a breeding line used in the development of Renown.

TABLE 2. Seedling response[a] of stem rust resistance (*Sr*) genes to the predominant races[b] of wheat stem rust in Canada since 1935

	Race[c]				
Sr gene	15B-1 (C10)	56 (C17)	15B-1L (C18)	15B-1L (C33)	15B-1L (C53)
2[d]	...	...	...	...	...
5	S	S	S	S	S
6	R	R	R	R	R
7a	R	S	S	S	S
7b	S	S	S	S	S
9b	S	R	R	R	R
9d	S	R	S	S	S
9g	S	S	S	S	S
11	S	R	S	S	S
16	S	S	S	S	S
17	S	S	R	R	S

[a] R = resistant; S = susceptible.

[b] Predominant races according to Green (4-6).

[c] Standard race designations are given, with Canadian race designations in parentheses.

[d] *Sr2* is an adult plant resistance gene, not expressed in the seedling leaf.

that it has *Sr5*, *Sr9b*, and *Sr7a*. Since *Sr9b* is present, *Sr9g* is excluded. Therefore, the Neepawa genotype may be *Sr5*, *Sr7a*, *Sr9b*, *Sr12*, and *Sr16*. Columbus was developed by backcrossing sprouting resistance from RL 4137 into Neepawa. Columbus is not as resistant to stem rust as Neepawa and may have lost at least one of Neepawa's resistance genes. The cultivar Katepwa is from an intercross of two Neepawa backcross lines for stem rust resistance, one with *Sr11* and a second with an unidentified gene or genes from cereal introduction (CI) 8154/2*Frocor. Katepwa should have the Neepawa genotype for stem rust resistance plus at least two additional genes. Of the additional genes in Manitou (other than the Thatcher genes), *Sr6* is effective against all of the common stem rust races, whereas *Sr7a* conditions resistance to C10 only (Table 2). *Sr9b* is the only additional gene present in Neepawa that is effective against the current stem rust races. Gene *Sr11* in Katepwa conditions resistance to C17 only. Although CI 8154/2*Frocor is resistant to the common stem rust races, it is not known whether the resistance is due to one gene. Consequently it is not known whether all the resistance has been transferred. The higher levels of resistance of these wheats most likely results from the combined effects of the Thatcher resistance and *Sr6* (Manitou), *Sr9b* (Neepawa), and *Sr9b* and resistance from CI 8154/2*Frocor (Katepwa). Interactions between effective and normally ineffective genes may account for improved resistance. Singh and McIntosh (19) showed that in Chris, a cultivar also related to Thatcher, adult plant resistance may be due to an interaction between *Sr7a* and *Sr12*. Regardless of the possible explanations, the enhanced resistance of the Thatcher derivatives results from the combination of the Thatcher resistance with additional *Sr* genes. The Thatcher derivatives with additional *Sr* genes have clearly had higher levels of resistance over a long period of time than Thatcher (Table 1).

Mundt (14) and Vanderplank (21) associated *Sr6* with durable resistance in wheats grown in western Canada. This gene was certainly effective against race 15B-1 in Selkirk and is still effective against all of the common stem rust races (Table 2), but it is not solely responsible for the durable stem rust resistance in these wheats. For example, Manitou has *Sr6*, and the cultivars Neepawa and Katepwa, which have been predominant in western Canada since 1969, lack *Sr6*. All of these cultivars have had very similar if not identical stem rust readings in the rust nursery at Winnipeg. There has been no evidence of an increase of stem rust on Neepawa and Katepwa. Although virulence to *Sr6* has been reported in North America, it has never occurred in the common races (4,5). We conclude that the presence or absence of *Sr6* alone has not necessarily determined the durability of stem rust resistance in western Canadian wheats since the decline of race 15B-1.

Vanderplank (21) hypothesized that the rarity of stem rust races with virulence to Selkirk could be due to deleterious fitness effects that may be conditioned by virulence to *Sr6* in combinations with other virulences in stem rust isolates. It seems equally possible that stem rust isolates with virulence to Selkirk arose in poorly adapted genetic backgrounds. Races 15B-3 and 15B-5 are subraces of the original race 15B-1 and have additional virulence to *Sr6* and to *Sr6* and the cultivar Golden Ball, respectively (5). These races may also differ in other characteristics. Selective forces for characteristics other than virulence also affect race frequency. Green (5) noted that the first strains of race 15B had darker urediniospores and did not sporulate as abundantly as races 56 and 11. Furthermore, race 56 was found to compete better at high temperatures than race 15B (11).

The uncertainty as to which stem rust resistance genes are present in North American hard red spring wheats precludes attempts to relate the durability of resistance to specific genes, alone or in various combinations. The relationship between specific genes and durability will be obscure until the completion of detailed genetic studies that examine which genes are present and how they interact with each other and with unidentified resistance genes in these cultivars.

A more concise example relating durable resistance to combinations of specific resistance genes is found in the leaf rust resistance of some of these same wheat cultivars. Chris and Era, cultivars related to Thatcher, possess the leaf rust resistance genes *Lr13* and *Lr34* (Thatcher derivatives may also have *Lr22b*, an adult plant resistance gene that does not currently condition resistance to leaf rust at Winnipeg) (3,17) and have been highly resistant to this disease since their releases in 1966 and 1972, respectively (Table 3). The leaf rust resistance of Manitou, which has only resistance gene *Lr13*, has not been as effective over nearly the same period of time as the resistance in Chris or Era. The cultivar Columbus, a Neepawa backcross derivative that has *Lr13* + *Lr16* (18), has remained highly resistant since its introduction

TABLE 3. Adult plant reactions to leaf rust (percent severity and response[a]) of selected wheat cultivars in the rust nursery at Winnipeg during selected years from 1966 to 1989

Cultivar	Genotype	1966	1970	1974	1978	1982	1986	1989
Thatcher[b]	. . .	80 S	60 S	70 S	80 S	80 S	80 S	70 S
RL 6058	TC[c]*Lr34*			15 M	10 M	20 MR	10 M	15 M
Manitou	*Lr13*	10 R	20 M	60 S	40 M	40 MS	40 M	40 MR
Chris	*Lr13, 34*	TVR	10 MR	20 MR	3 R	VR	5 MR	5 VR
Era[d]	*Lr13, 34*		1 R	5 R	1 R	1 R	1 R	5 VR
Neepawa	*Lr13*		15 M	50 S	30 M	30 S	30 M	40 MR
RL 6005	TC*Lr16*		20 M	40 MR	20 M	30 MR	15 MR	20 MR
Columbus	*Lr13, 16*					VR	5 R	5 VR

[a] T = trace infection; VR = very resistant (hypersensitive flecks); R = resistant (flecks and small uredinia); MR = moderately resistant (flecks and small to moderate uredinia); M = mixed reaction (flecks and large uredinia); MS = moderately susceptible (moderate to large uredinia); S = susceptible (large uredinia).

[b] Thatcher has *Lr22b*, an adult plant resistance gene, which does not currently condition resistance to leaf rust at Winnipeg. The Thatcher-derived cultivars may also have *Lr22b*.

[c] Thatcher isogenic line *Lr34*.

[d] Era also has *Lr10* (3), which does not currently condition resistance to leaf rust in the Winnipeg rust nursery.

in 1980. Neepawa, with only resistance gene *Lr13*, has consistently had higher leaf rust severities than Columbus. Cultivars with combinations of adult plant resistance genes (Era and Chris) or combinations of seedling and adult plant genes (Columbus) have been widely grown over a large geographic area for a number of years and have maintained high levels of leaf rust resistance. There are numerous hypotheses to explain this durable resistance (interaction of resistance genes, deleterious fitness effects of the corresponding virulence phenotypes in the rust population, or epidemiological factors), but it is clear that cultivars with combinations of two or more effective adult plant or adult and seedling resistance genes have retained higher levels of resistance than closely related wheat cultivars with single resistance genes.

Mundt (14) used examples illustrating that multiple mutations to virulence or avirulence in cereal rust fungi have been obtained in mutagenesis experiments and, from this evidence, concluded that these mutation events are not always independent. In the cited experiments *N*-methyl-*N'*-nitro-*N*-nitrosoguanidine and X rays were used to obtain high levels of mutation rates for selected characters. These powerful mutagens would be expected to induce mortality and mutagenic rates much higher than expected under field conditions, and the survivors could also be expected to have multiple mutations. Moreover, there is little evidence that multiple changes in virulence commonly occur in field populations of cereal rust fungi. Green (5) showed that evolution within the race 15B-1L complex of wheat stem rust has occurred through a series of single-step changes to virulence or avirulence to single-gene differential lines. Kolmer (12) attributed the changes in wheat leaf rust races in the prairie region of Canada since 1974 to the introduction of single resistance genes in host cultivars that selected a series of single-step changes to virulence in the rust population. Martens and McKenzie (13) concluded that single-step changes in virulence have led to most of the virulence combinations of oat stem rust in Canada. While radical changes affecting virulence in rust populations could be a result of multiple mutations, simpler explanations are that these changes occur because of either migration of dissimilar rust populations (2) or selection of rust races due to factors unrelated to virulence (4,5).

Mundt stated that the empirical data (Green and Campbell's Table 2) are not supportive of the probabilities hypothesis. This is a narrow view, since it is apparent upon a more inclusive analysis of the literature on wheat stem and leaf rust that cultivars with combinations of effective resistance genes have displayed high levels of durable resistance, compared to related cultivars that have fewer of the same resistance genes. As such, the probabilities hypothesis should not be so readily dismissed. Although it is not wise to eliminate other reasons that may explain the longevity of this resistance, it is clear that durable resistance to both stem and leaf rust of wheat has been achieved by combinations of resistance genes.

LITERATURE CITED

1. Brennan, P. S. 1975. General resistance in wheat (*Triticum aestivum* L.) to wheat stem rust (*Puccinia graminis* Pers. f. sp. *tritici* Erik. and Henn.). Ph.D. thesis, University of Saskatchewan, Saskatoon. 142 pp.
2. Burdon, J. J., Marshall, D. R., Luig, N. H., and Gow, D. J. S. 1982. Isozyme studies on the origin and evolution of *Puccinia graminis* f. sp. *tritici* in Australia. Aust. J. Biol. Sci. 35:231-238.
3. Ezzahiri, B., and Roelfs, A. P. 1989. Inheritance and expression of adult plant resistance to leaf rust in Era wheat. Plant Dis. 73:549-551.
4. Green, G. J. 1971. Physiologic races of wheat stem rust in Canada from 1919 to 1969. Can. J. Bot. 49:1575-1588.
5. Green, G. J. 1975. Virulence changes in *Puccinia graminis* f. sp. *tritici* in Canada. Can. J. Bot. 53:1377-1386.
6. Green, G. J. 1979. Stem rust of wheat, barley, and rye in Canada in 1978. Can. Plant Dis. Surv. 59:43-47.
7. Green, G. J. 1981. Identification of physiologic races of *Puccinia graminis* f. sp. *tritici* in Canada. Can. J. Plant Pathol. 3:33-39.
8. Green, G. J., and Campbell, A. B. 1979. Wheat cultivars resistant to *Puccinia graminis tritici* in western Canada: Their development, performance, and economic value. Can. J. Plant Pathol. 1:3-11.
9. Green, G. J., and Dyck, P. L. 1975. The reaction of Thatcher wheat to Canadian races of stem rust. Can. Plant Dis. Surv. 55:85-86.
10. Green, G. J., and Dyck, P. L. 1979. A gene for resistance to *Puccinia graminis* f. sp. *tritici* that is present in wheat cultivar H-44 but not in cultivar Hope. Phytopathology 69:672-675.
11. Katsuya, K., and Green, G. J. 1967. Reproductive potentials of races 15B and 56 of wheat stem rust. Can. J. Bot. 45:1077-1091.
12. Kolmer, J. A. 1989. Virulence and race dynamics of *Puccinia recondita* f. sp. *tritici* in Canada during 1956–1987. Phytopathology 79:349-356.
13. Martens, J. W., and McKenzie, R. I. H. 1979. Virulence and race dynamics in *Puccinia graminis* f. sp. *avenae* in Canada. Can. J. Bot. 57:952-957.
14. Mundt, C. C. 1990. Probability of mutation to multiple virulence and durability of resistance gene pyramids. Phytopathology 80:221-223.
15. Nazareno, N. R. X., and Roelfs, A. P. 1981. Adult plant resistance of Thatcher wheat to stem rust. Phytopathology 71:181-185.
16. Peterson, R. F., Campbell, A. B., and Hannah, A. E. 1948. A diagrammatic scale of estimating rust intensity on leaves and stems of cereals. Can. J. Res. C26:496-500.
17. Roelfs, A. P. 1988. Resistance to leaf and stem rusts in wheat. Pages 10-22 in: Breeding Strategies for Resistance to the Rusts of Wheat. N. W. Simmonds and S. Rajaram, eds. CIMMYT, Mexico City.
18. Samborski, D. J., and Dyck, P. L. 1982. Enhancement of resistance to *Puccinia recondita* by interactions of resistance genes in wheat. Can. J. Plant Pathol. 4:152-156.
19. Singh, R. P., and McIntosh, R. A. 1987. Genetics of resistance to *Puccinia graminis tritici* in "Chris" and "W3746" wheats. Theor. Appl. Genet. 73:846-855.
20. Sunderwirth, S. D., and Roelfs, A. P. 1980. Greenhouse evaluation of the adult plant resistance of *Sr2* to wheat stem rust. Phytopathology 70:634-637.
21. Vanderplank, J. E. 1975. Principles of Plant Infection. New York, Academic Press. 216 pp.