Virulence and Race Dynamics of *Puccinia recondita* f. sp. *tritici* in Canada during 1956–1987

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ABSTRACT

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The changes in virulence and Unified Numeration (UN) race percentages of *Puccinia recondita* f. sp. *tritici* since 1956 in three regions of Canada were examined. Virulence frequencies to *Lr2c* and *Lr3* in eastern Canada and the Pacific region generally fluctuated between 20–100%, even though these genes are not known to be present in wheat grown in these regions. UN races in the eastern and Pacific regions fluctuated between low (0–20%) and intermediate (20–60%) levels, with no strong directional trends. In the prairie provinces, levels of virulence on lines having specific resistance genes increased in frequency after the corresponding resistance genes were released in the host population. In this region, changes in the race composition could be related to the changes in level of individual virulences and the use of resistance in the host population. Examples supporting and contradicting the concept of stabilizing selection are discussed with reference to wheat leaf rust populations in Canada.

Additional keywords: gene-for-gene relationships, physiologic specialization, *Triticum*.

Annual physiologic race surveys of *Puccinia recondita* f. sp. *tritici* Rob. ex. Desm have been conducted at the Agriculture Canada Research Station in Winnipeg since 1931. The surveys provide continuous information detailing the evolutionary trends of physiologic specialization by the wheat leaf rust fungus over a large geographic area. Before 1961, survey results were published in mimeographed reports issued by the Canada Department of Agriculture, Winnipeg. During 1961–1979, results were published in the *Canadian Plant Disease Survey*. Since 1980, the surveys have been published in the *Canadian Journal of Plant Pathology*. Johnson (9) summarized the results of the surveys during 1931–1955. Physiologic race surveys of *P. recondita* have also been conducted in the United States and are summarized by Johnston (12) and Long et al (16).

With the advent of modern plant breeding techniques, evolution in the cereal rust fungi became largely a human-guided process (10). Repeated documentation has shown that the use of rust-resistant cereals greatly influences the physiologic race structure of the corresponding rust populations (18,28,36). Use of leaf rust-resistant cultivars in the Canadian prairies began when the cultivar Renown (host-resistance gene *Lr14a*) was released in 1937 (26). Renown was followed by Lee (*Lr10*) in 1950 and Selkirk (*Lr10 + Lr16*) in 1954. The acreage planted to Selkirk declined when Manitou and Neepawa, both with adult plant-resistance gene *Lr13*, were introduced in 1966 and 1969, respectively. These cultivars were resistant to the prevalent leaf rust populations when initially released; within a few years, however, the corresponding virulences in the population of *P. recondita* increased and the resistance genes were less effective.

At present, Neepawa, Katepwa (*Lr13*), and Columbus (*Lr13 + Lr16*) are the most popular wheat cultivars grown on the prairies of western Canada. Neepawa and Katepwa are moderately resistant, whereas Columbus is still highly resistant to leaf rust. Susceptible cultivars have generally been grown in the soft white winter wheat areas of southern Ontario and eastern Canada. Most wheat cultivars in British Columbia and southern Alberta are also susceptible to leaf rust. Use of resistance genes in the wheat production areas of the United States also influences the leaf rust population in Canada. This was demonstrated with the release of the cultivar Agent (*Lr24*) in Oklahoma and Texas in 1971 and the

subsequent detection in Canada of isolates with virulence to this gene (26). Previously, isolates of *P. recondita* with virulence to differentials with *Lr24* had not been detected in Canada.

Gene-for-gene relations in the wheat leaf rust disease were demonstrated by Samborski and Dyck (27) and Dyck and Samborski (6,7,8). Avirulence in the rust fungus to resistance gene *Lr1* is conditioned by a single dominant allele (designated as *P1*). Avirulence to *Lr2a* and *Lr2c* is conditioned by an identical dominant allele (*P2*); however, an independent dominant allele (*I2c*) acts as an inhibitor of avirulence to the *Lr2c* allele. When dominant alleles are present at the *P2* and *I2c* loci in the fungus, avirulence to *Lr2a* and virulence to *Lr2c* will result. Virulence to the *Lr3* resistance gene is conditioned by a dominant allele (*P3*).

This paper is intended to serve as an initial examination of virulence and race frequencies of *P. r. tritici* in three geographic regions of Canada since 1956. Where it was possible, virulence frequencies to specific *Lr* genes were related to the known presence or absence of the corresponding resistance genes in the host populations.

MATERIALS AND METHODS

The methods employed in the annual physiologic race survey of *P. recondita* have remained essentially unchanged since the survey's inception in 1931. Collections of rust-infected leaves are obtained from commercially grown wheat and also from uniform nurseries throughout the country. The collections are increased on susceptible wheat seedlings (cv. Little Club), and urediniospores from a single pustule are isolated and increased from each collection. The single pustule isolates are then evaluated for virulence on a set of wheat leaf rust differential seedlings. Infection types are recorded 12 days later with the scale developed by Stakman and Levine (30). Infection types 0, 1, and 2 are considered as avirulent, and infection types 3 and 4 are classified as virulent. The average number of single pustule isolates in the annual survey was 300. The majority of the collections were from Manitoba and Saskatchewan, with the remainder split between eastern Canada, British Columbia, and Alberta. Techniques for the study of physiologic specialization of wheat leaf rust were summarized by Browder (4).

Since physiologic races of leaf rust were first described by Mains and Jackson in 1921 (17), four different nomenclature systems have been employed to designate physiologic specialization in

wheat leaf rust. The International Register of Standard Races used eight differential cultivars in classifying races of *P. recondita*. The differentials Malakof, Webster, Loros, Democrat, and Hussar contained resistance genes *Lr1*, *Lr2a*, *Lr2c*, *Lr3*, and *Lr11*, respectively. The Unified Numeration (UN) nomenclature proposed by Basile (2) and Johnston (11) and the modified UN system (16) used genes *Lr1*, *Lr2a*, *Lr2c*, and *Lr3* to differentiate isolates of leaf rust into physiologic races. International Standard races included within the UN groups produced identical or similar infection types on differentials with these resistance genes. A combination of International Standard race designation and UN system was used to describe physiologic races in Canada during 1956–1966.

Additional resistance genes were subsequently identified in other common wheats and in related genera such as *Aegilops* (*Lr9*) and *Agropyron* (*Lr19*). Browder (5) has summarized the information on *Lr* genes through *Lr29*. There are currently 34 designated *Lr* genes. After 1967, lines of wheat isogenic for *Lr* genes were used as differentials to identify physiologic races. During 1967–1986, avirulence/virulence formulae were used to designate virulence combinations. A new nomenclature proposed by the North American Leaf Rust Workers Committee in St. Paul, Minnesota in April 1986 is currently being used to describe virulence combinations of *P. recondita* in Canada (15).

The physiologic race designations of virulence combinations in the surveys were converted to the modified UN system. Percentages of isolates virulent on *Lr1*, *Lr2a*, *Lr2c*, and *Lr3* since 1956 were calculated because these genes were present in the original International Standard race differentials. Virulence percentages from avirulence/virulence formulae were determined for *Lr16*, *Lr17*, *Lr18* since 1969, and *Lr3ka*, *Lr9*, *Lr10*, *Lr11*, *Lr24*, *LrB* since 1976.

The survey results were analyzed separately for three geographic regions: the Pacific region of British Columbia and Alberta, the prairie region of Saskatchewan and Manitoba, and the eastern region comprising southern Ontario, Quebec, and the Maritime provinces. Samborski (26) and Johnson (9) had recognized previously that distinct leaf rust populations could be identified in each of these areas.

RESULTS

Virulence dynamics. Levels of virulence to differential lines with *Lr1*, *Lr2a*, and *Lr2c* followed similar trends in the eastern and Pacific populations (Figs. 1A, 2A). In both regions virulence to *Lr2c* fluctuated between 15–100%, without a clear directional trend. Virulence to *Lr2a* in both populations was consistently low, whereas virulence to *Lr1* was low after 1962 and increased steadily after 1974. Virulence to *Lr3* fluctuated between 15–100% in the eastern region and was consistently between 50–100% in the Pacific region. A directional trend of virulence dynamics occurred in the prairies. *Lr1* and *Lr2c* virulence frequencies varied between 10–50% during 1956–1962 and 0–10% during 1963–1976, after which both steadily increased (Fig. 3A). Virulence to *Lr2a* varied between 0–10% during 1956–1976, after which it also began to rise. Virulence to *Lr3* has been near 100% since 1962.

Virulence levels on resistance genes *LrB*, *Lr3ka*, *Lr9*, *Lr11*, *Lr16*, *Lr17*, *Lr18*, and *Lr24* did not change in a consistent direction in any of the three regions (Table 1). Virulence to *Lr3ka*, *Lr9*, and *Lr11* fluctuated between 5–95% in the eastern population; however, virulence to these genes were usually between 0–10% in the other two regions. Virulence to *LrB* was consistently low in the prairie region and fluctuated between 6–96% in the eastern and Pacific regions. Virulence to *Lr18* varied between 3–80% in the prairie and Pacific regions and between 30–100% in the eastern region. Levels of virulence to *Lr10* fluctuated between 39–93% in the eastern region and were consistently near 100% in the Pacific region. In the prairies, virulence on differentials with *Lr10* increased starting in 1972 and is currently at 100%. Virulence to *Lr24* has generally been low in all regions; in the prairies, however, it reached 30% in 1987.

Race dynamics. Five different UN races were found at intermediate levels in both the eastern and Pacific populations of

P. recondita (Figs. 1B, 2B). UN races 2 and 3 accounted for 50–100% of the population in the Pacific population during 1956–1979. UN race 6 reached levels between 30–70% during 1980–1985, after which UN races 5 and 13 also became prevalent.

In the eastern population, UN race 3 was dominant during 1956–1960, after which race 2 also became common. These races accounted for 50–100% of the population until 1974, when the frequency of race 6 increased, reaching 60% in 1980. UN races 6 and 3 accounted for the majority of the population in the east during 1977–1985, after which races 5 and 13 also became common.

A different succession of races occurred in the prairies (Fig. 3B). During 1956–1961, UN races 2 and 5 were between 30–80% of the population. UN race 2 was at nearly 100% during 1964–1977, after which it started to decline to an extremely low level. UN races 17 and 5 have increased since 1979 up to levels of 50%. UN race 13 was at very low levels until 1975, when it increased slightly, and in 1983, when it began to increase to its present intermediate level of 50%.

Distribution of resistance genes. Leaf rust-resistance genes *Lr9*, *Lr11*, *Lr12*, and *Lr13* have been used in the soft red winter wheat areas of the eastern United States (Table 2). In Michigan, New York, and southern Ontario, susceptible soft white winter wheats have generally been grown, although resistant cultivars have been introduced recently in New York. Resistance genes *Lr1*, *Lr2a*, *Lr2b*, *Lr3*, *Lr10*, *Lr13*, and *Lr34* have been used recently in hard red spring wheats grown in North Dakota (Table 2). Most cultivars grown in the Pacific Northwest of the United States, British Columbia, and southern Alberta are susceptible to leaf rust (19,25). Resistance gene *Lr1* has been used in cultivars grown in Washington (20). In the rust areas of the eastern Canadian prairies, genes *Lr1*, *Lr2a*, *Lr10*, *Lr12*, *Lr13*, and *Lr16* have been used in different cultivars since 1950 (Table 2).

DISCUSSION

The use of leaf rust-resistant cultivars in Manitoba, Saskatchewan, and the north central United States has resulted in large virulence shifts in the population of *P. recondita* of the Canadian prairies. Levels of virulence to *Lr1*, *Lr2a*, *Lr2c*, *Lr3*, and *Lr10* increased rapidly after cultivars with these resistance genes were released in North Dakota in the early 1970s. Resistance gene *Lr1* has also been used in hard red winter wheats grown in Nebraska (16), whereas *Lr3* has been used in wheats grown in the southern U.S. Great Plains starting in 1943, when Pawnee was released (26). Virulence to *Lr10* was also very high during 1958–1966, when Selkirk (*Lr10* + *Lr16*) was grown widely on the Canadian prairies and the adjacent northern states (26). Virulence to *Lr16* was also high during this period, reaching a maximum of 55% in 1966 (26). When Selkirk was replaced with Manitou (*Lr13*) in 1966, virulence to *Lr16* declined, and virulence to *Lr13* increased rapidly. In 1969, only two of 28 isolates tested were virulent on *Lr13* (24). By 1971, 84% of the isolates from Manitoba and Saskatchewan were virulent on *Lr13* (25). Manitou and Neepawa (*Lr13*) were highly resistant to leaf rust when first released; however, this resistance became less effective after these cultivars with *Lr13* were grown extensively on the prairies. Gene *Lr13* may also be exerting a selective effect on unrelated virulences. Alexander et al (1) demonstrated that nonhypersensitive resistance to rust in dry beans (*Phaseolus vulgaris*) exerts a selective effect on noncorresponding virulences in the bean rust fungus (*Uromyces appendiculatus*). Genes *LrB*, *Lr3ka*, *Lr9*, *Lr11*, *Lr17*, and *Lr18* have never been used in the Great Plains region of North America, and the corresponding virulences are currently at low frequencies in the Canadian prairies. The intermediate frequency of virulence to *Lr24* in 1987 can be attributed to its continued use in winter wheats grown in the southern plains of the United States (28).

In the Pacific population of *P. recondita*, only virulence to *Lr1* displayed a directional increase in frequency consistent with the use of that resistance gene in the host population. Gene *Lr1* has been used in cultivars derived from Washington 101 (CI 13438), which are grown in the U.S. Pacific Northwest (20). Virulences to *Lr2c* and *Lr3* fluctuated 20–100%, even though these genes have

never been used in wheat grown in this region.

A similar trend of increasing virulence frequency to *Lr1* and intermediate to high levels of virulence to *Lr2c* and *Lr3* were observed in the eastern population. *Lr1* has been used in the soft red winter cultivar Blueboy, grown in the eastern United States (5). Genes *Lr9* and *Lr11* have also been used in the eastern U.S., accounting for the intermediate levels of virulence to these genes that have been found in the eastern population. Resistance genes *Lr2c* and *Lr3* are not known to have been used in eastern North America.

Susceptible host populations in the eastern and Pacific regions have maintained intermediate levels of virulence in their respective populations of *P. recondita* to resistance genes that have no history of use in either region. This is in contrast to the prairies, where resistant cultivars have been grown continuously since 1937 and virulence trends can be generally explained by the use of the corresponding resistance genes. The continuous introduction of different resistance genes in this region may have resulted in displacement effects occurring in the population of *P. recondita*. Isolates virulent on only *Lr3* would have been displaced by isolates
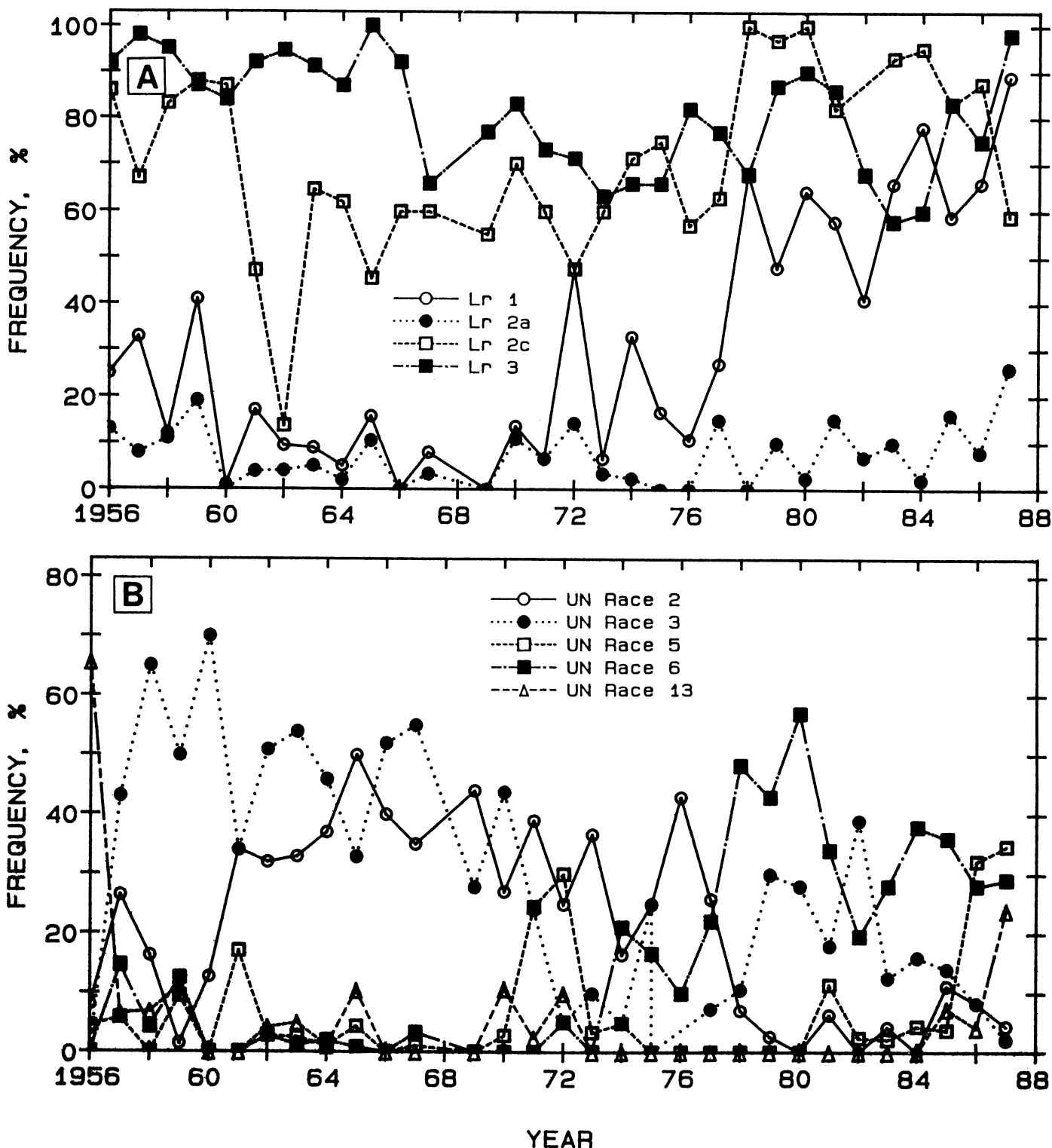

Fig. 1. Virulence and UN race frequencies of *P. recondita* f. sp. *tritici* in eastern Canada (Ontario, Quebec, Maritimes) during 1956–1987. **A**, Percent of isolates of *P. recondita* virulent on *Lr1*, *Lr2a*, *Lr2c*, and *Lr3*. **B**, Percent of UN races.

with additional virulence on *Lr1* and/or *Lr2a* after cultivars with these genes were introduced in North Dakota. This displacement effect may also have had an effect on virulence levels to other resistance genes that have never been used in this area.

Because the North American population of *P. recondita* reproduces asexually, associations between virulences that are directly selected by corresponding resistance genes in the host population and those virulences that are not under direct selection pressure may be very important in determining the final frequency of the unselected virulences. Populations of *P. recondita* in the eastern and Pacific regions would not have been subjected to the

same degree of displacement and may have different virulence associations (if any) than the population in the prairies. This may partly explain the unneeded virulence to genes *Lr2c* and *Lr3* in both of these regions. The different levels of virulence across all regions to genes *LrB*, *Lr3ka*, *Lr17*, and *Lr18* may be due to the combined effects of differing degrees of displacement and different associations between selected and unselected virulences between regions. These resistance genes have no history of use in any region of North America.

From the virulence dynamics as presented here, it is impossible to make any definitive judgment concerning the validity of
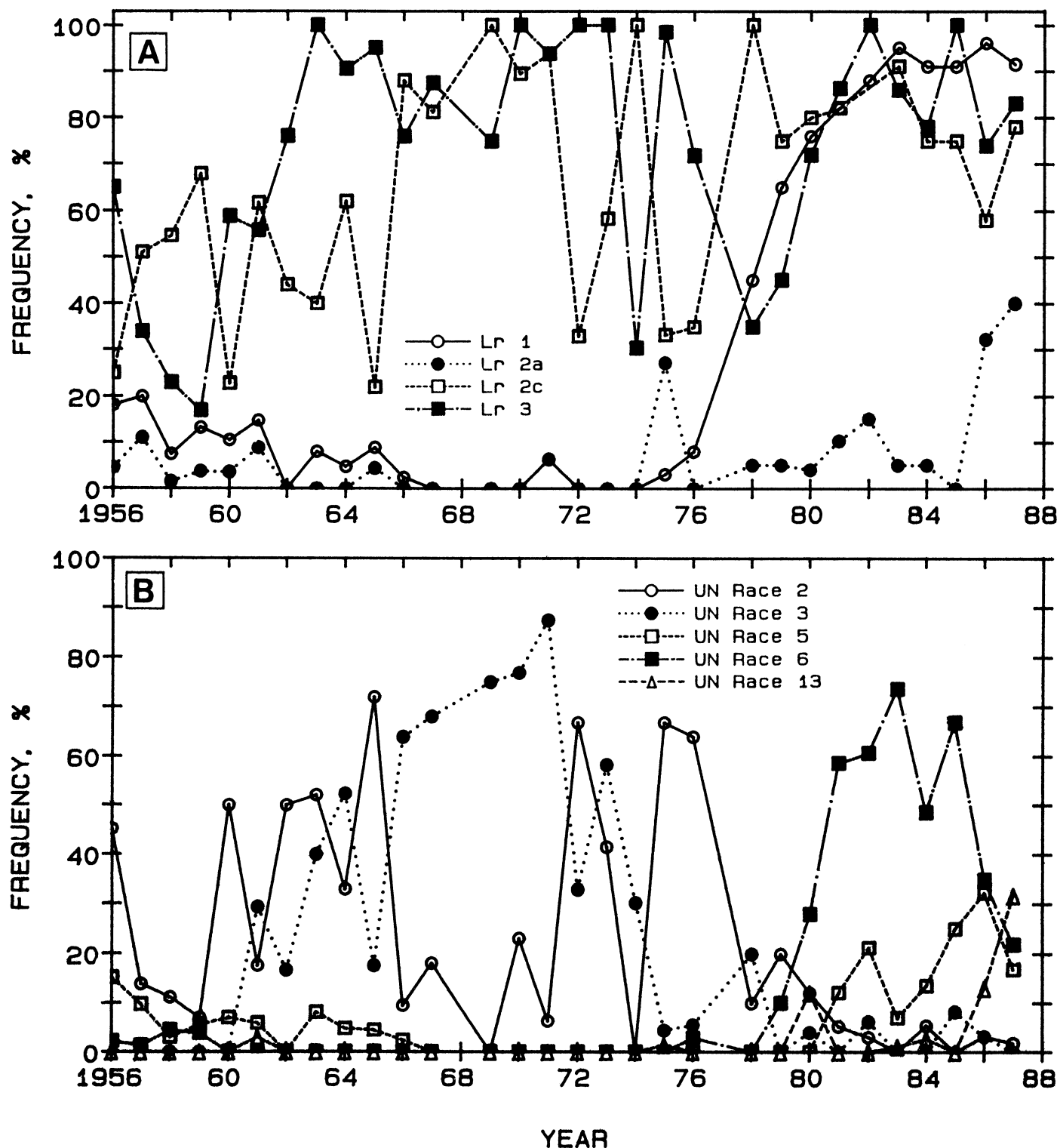

Fig. 2. Virulence and UN race frequencies of *P. recondita* f. sp. *tritici* in Pacific Canada (British Columbia, Alberta) during 1956–1987. **A,** Percent of isolates of *P. recondita* virulent on *Lr1*, *Lr2a*, *Lr2c*, and *Lr3*. **B,** Percent of UN races.

Vanderplank's (35) theory of stabilizing selection. Several examples can be illustrated that support and contradict this concept. Virulence frequencies to *Lr1*, *Lr2a*, and *Lr2c* in the prairies were at a low level and then increased when these resistance genes where introduced in adjacent North Dakota. Similarly, virulence to *Lr1* was at a low level in the Pacific region and then increased in frequency when this gene was used in wheat grown in the Pacific Northwest of the U.S.

In both of these examples, the virulences were maintained at low levels before the introduction of the resistance genes and increased in frequency only after their introduction. These examples support the idea that isolates with unneeded virulence genes are at a selective disadvantage relative to those with the minimum of required virulence genes. However, as previously noted in the eastern and Pacific populations, virulence frequencies to *Lr2c* and *Lr3* are maintained at intermediate levels, even though these genes are not known to have been present in either of these areas. The decline of virulence to *Lr10* and *Lr16* in the prairies after Selkirk was replaced could either be attributed to the deleterious effects of unneeded virulence or to a displacement effect, as virulence on Manitou occurred in that portion of the rust population that was avirulent to *Lr16* (26). Virulence levels to *Lr10* and *Lr16* may have
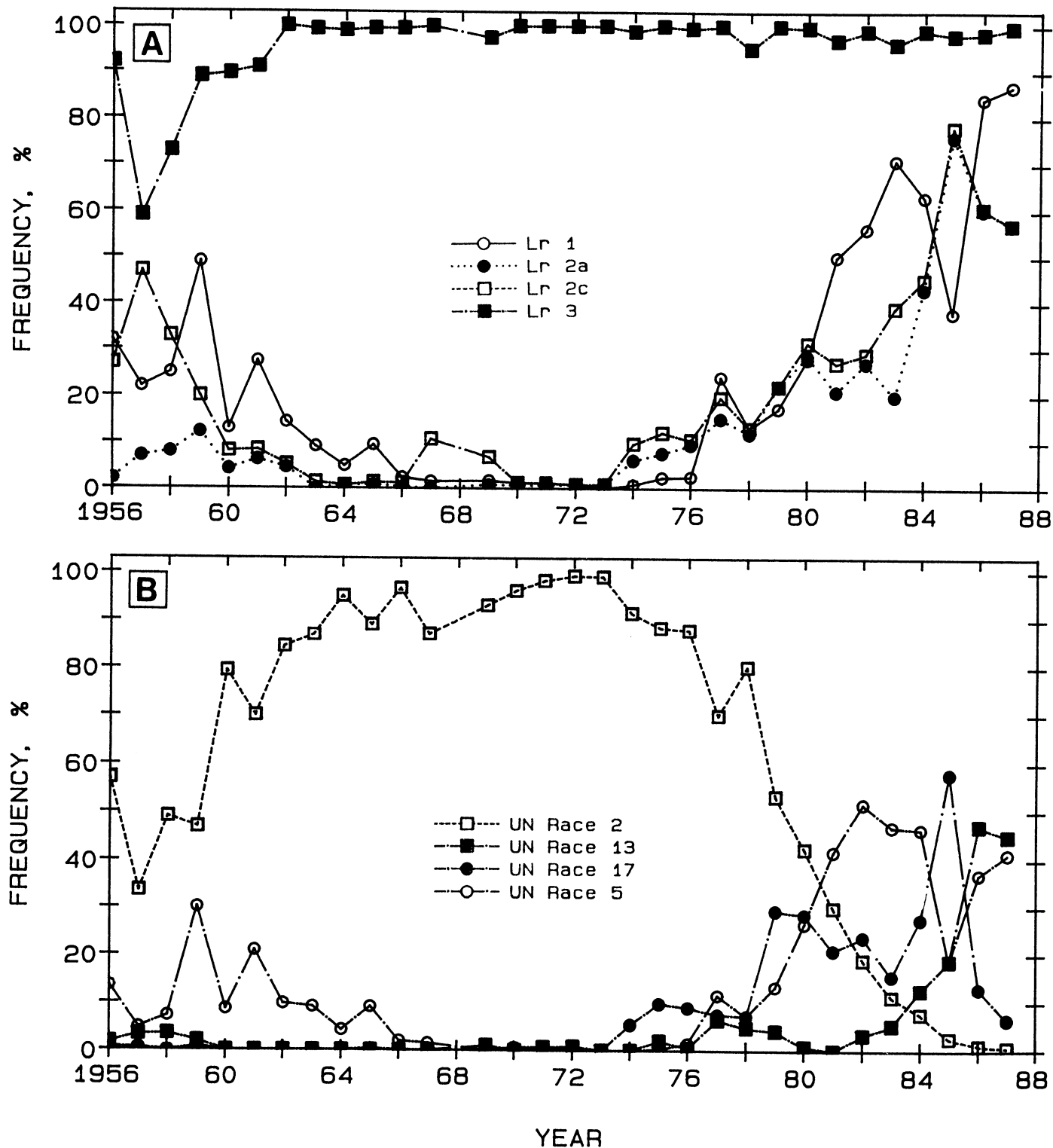

Fig. 3. Virulence and UN race frequencies of *P. recondita* f. sp. *tritici* in the Canadian prairies (Manitoba, Saskatchewan) during 1956–1987. **A**, Percent of isolates of *P. recondita* virulent on *Lr1*, *Lr2a*, *Lr2c*, and *Lr3*. **B**, Percent of UN races.

remained high if Selkirk had been replaced by a susceptible cultivar. Virulence to *Lr14a* has remained extremely high in the prairies, even though this gene is no longer used in commercial wheat cultivars (26).

Contradictory evidence in evaluating the validity of stabilizing selection has also been found in other host-parasite systems (23,36). In the oat stem rust pathogen (*P. graminis* f. sp. *avenae*), Martens et al (18) described the increase in frequency of virulence to resistance gene *Pg2* when cultivars with this gene were introduced in the U.S. and Canada in the 1940s. Virulence to *Pg1* increased, and virulence to *Pg2* decreased when cultivars with *Pg1* replaced those with *Pg2*. As found with *P. recondita*, virulence changes of this type are consistent with the idea of stabilizing selection as originally described by Vanderplank. However, in the same study virulence levels in *P. graminis* to *Pg3* were extremely high in both eastern and western Canada, even though this gene was not present in either host population. High levels of virulence without the presence of the corresponding resistance genes were also found for *Pg8* and *Pg9* in western and eastern Canada, respectively. Leonard (13) examined the effects of unneeded

TABLE 1. Percentage of isolates of *Puccinia recondita* f. sp. *tritici* in Canada virulent on backcross lines with single *Lr* genes from 1969 to 1987

Year	*LrB*	*Lr3Ka*	*Lr9*	*Lr10*	*Lr11*	*Lr16*	*Lr17*	*Lr18*	*Lr24*
Eastern Canada (Ontario, Quebec, Maritimes)									
1969	...	...	...	77	...	0	0	33	...
1970	...	...	...	70	...	0	0	70	...
1971	...	...	...	46	...	0	2	62	...
1972	...	...	...	66	...	0	10	33	...
1973	...	13	...	53	...	7	0	66	...
1974	...	33	...	40	...	0	5	66	...
1975	...	41	...	41	...	0	16	58	...
1976	57	40	10	39	0	0	0	57	21
1977	48	44	4	51	0	0	7	30	7
1978	96	56	4	87	0	0	0	100	0
1979	82	76	30	89	0	0	2	85	0
1980	95	86	33	88	5	0	2	90	0
1981	50	45	25	87	17	0	3	32	3
1982	82	48	29	58	41	0	15	68	0
1983	70	35	18	70	52	...	10	75	0
1984	65	41	10	80	58	2	9	58	0
1985	29	29	11	72	48	0	18	57	0
1986	46	33	0	70	20	0	0	50	0
1987	28	37	0	93	22	0	4	39	15
Canadian prairies (Manitoba and Saskatchewan)									
1969	...	...	...	38	...	4	4	20	...
1970	...	...	...	45	...	7	3	14	...
1971	...	...	...	18	...	5	0	15	...
1972	...	...	...	42	...	6	1	25	...
1973	...	0	...	54	...	8	0	27	...
1974	...	0	...	42	...	1	4	21	...
1975	...	0	...	72	...	0	0	9	...
1976	9	7	0	66	0	1	0	5	31
1977	1	3	0	71	0	0	19	9	9
1978	0	2	0	73	0	0	6	5	3
1979	0	0	0	85	0	0	3	5	8
1980	0	0	0	96	2	0	2	1	0
1981	1	0	1	95	0	0	11	5	2
1982	2	0	0	91	4	0	3	7	4
1983	4	1	0	92	0	0	13	2	6
1984	1	0	0	94	2	16	13	9	0
1985	1	0	0	99	1	4	14	15	1
1986	1	2	0	99	1	3	10	27	7
1987	0	0	0	100	3	4	8	30	30
Pacific Canada (British Columbia and Alberta)									
1969	...	...	...	100	...	0	25	0	...
1970	...	...	...	100	...	15	62	0	...
1971	...	...	...	100	...	0	94	0	...
1972	...	...	...	42	...	4	29	4	...
1973	...	0	...	83	...	16	58	17	...
1974	...	0	...	100	...	0	30	70	...
1975	...	0	...	72	...	0	5	3	...
1976	27	3	0	89	0	0	8	27	5
1977[a]	...	...	...	...	...	...	...	...	...
1978	45	0	0	100	0	0	20	80	0
1979	50	5	0	85	0	0	15	45	10
1980	28	16	0	100	0	0	44	28	0
1981	12	0	0	95	0	0	0	16	0
1982	0	0	0	91	3	0	57	0	0
1983	6	0	0	92	0	4	71	14	0
1984	19	14	0	94	5	5	48	22	0
1985	0	0	0	99	0	0	66	16	0
1986	12	10	0	99	3	0	16	10	0
1987	21	7	0	100	0	3	20	20	3

[a] Only three collections were obtained in 1977.

virulence genes in oat stem rust in greenhouse experiments with a sexual population of the fungus. He demonstrated that isolates of the fungus with excess virulence genes had a selective disadvantage of 14–39% compared with simple isolates when maintained for 10 uredial generations on susceptible hosts.

It is evident from this study that the concept of stabilizing selection, as used in Vanderplank's sense, cannot account for all virulence frequencies in *P. recondita*. However, this should not be entirely unexpected, as it is extremely difficult to account for all the factors that influence virulence frequency in cereal rust populations. Given the asexual reproduction of *P. recondita* in North America, it is impossible to distinguish between the fitness effects of the virulence genes and their genetic background. As previously explained, the best adapted, most fit genotypes may carry along virulences that are unrelated to the resistance genes in the host population. A further complicating factor is the difficulty in calculating virulence gene frequency in cereal rusts. Because of its asexual nature, the population of *P. recondita* in North America would not be expected to be in either Hardy-Weinberg or linkage equilibrium (14). It is then impossible to calculate virulence gene frequency, even if the frequency of the homozygous recessive, virulent genotypes is known.

A high degree of heterozygosity at virulence loci has been detected in *P. recondita* (7,27,31,32,37). These heterozygous isolates carry virulence genes that remain undetected in race surveys. Thus, the virulence frequency to a particular resistance gene would not necessarily be equal to the virulence gene frequency. If heterozygotes have a higher fitness than either homozygote, unneeded recessive virulence genes would then be maintained in the population. Recurrent mutation at heterozygous loci could then produce isolates that would be homozygous recessive for virulence alleles. Virulent isolates would then occur in the population at a rate dependent on the mutation rate and the fitness of the virulent genotypes.

Samborski and Dyck (27) selfed an isolate of UN race 2 (International Standard race 15) and determined it was heterozygous at loci conditioning virulence to *Lr1*, *Lr2a*, and *Lr2c* (*P1p1*, *P2p2*). The same isolate was homozygous virulent at the locus conditioning virulence to *Lr3* (*P3P3*) and homozygous recessive at the locus that acts as an inhibitor of avirulence to *Lr2c* (*i2ci2c*).

UN race 2 was the dominant race in the prairies during 1960–1978, making up at least 80% of the population. Of course, not all isolates of UN race 2 would have the same genotype; other isolates of race 2 could be homozygous at the *P1* and *P2* loci and heterozygous at the *P3* locus. However, it may be reasonable to assume, given the prevalence of heterozygosity in *P. recondita*, that many of the race 2 isolates are heterozygous at the *P1*, *P2*, and *P3* loci. If so, the succession of UN races in the prairies can easily be explained by a series of single-step mutations. Single mutations at the *P1* and *P2* loci in isolates of race 2 heterozygous at these loci would result in isolates of race 5 (*p1p1*, *P2p2*, *P3-*, *i2ci2c*) and race 17 (*P1p1*, *p2p2*, *P3-*, *i2ci2c*), respectively. A single mutation at the *P1* locus in an isolate of race 17 with this genotype would result in an isolate of race 13 (*p1p1*, *p2p2*, *P3-*, *i2ci2c*). Dominance of virulence at the *P3* locus could be a contributing factor to the extremely high levels of virulence to *Lr3* in the prairies and the fluctuating intermediate to high levels found in the eastern and Pacific populations. At this locus, isolates of the fungus would need only one dominant allele to be virulent.

Nearly all isolates from the prairies produced identical infection types on differentials with *Lr2a* and *Lr2c*. In the eastern and Pacific regions, many isolates were virulent to *Lr2c* and avirulent to *Lr2a*. This regional difference in relationships between *Lr2a* and *Lr2c* can be explained by different frequencies of the inhibitor allele (*I2c*) (7) in the populations. In the prairies, the race 2 isolates would have been homozygous recessive at the inhibitor loci. Races 5, 17, and 13, which most likely developed from race 2, would have remained homozygous recessive at this locus. UN race 3 (International Standard race 161) was a predominant race in the Pacific region during 1966–1973. An isolate of this race was selfed and determined to be heterozygous at the inhibitor locus (27). Dominant inhibitor alleles have remained in the Pacific and eastern regions, contributing to the greater diversity found in these populations of *P. recondita* (Kolmer, *unpublished data*).

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TABLE 2. Distribution and acreage of leaf rust-resistance genes, singly and in combinations, in wheat grown in the United States and Canada

Area and wheat type	Year	*Lr*[a] genes	Acreage (%)[b]
Southern U.S. and Ohio Valley Soft Red Winter	1959[c]	None	29.8
		13	5.10
		12	24
		?	23.8
	1969[d]	None	8.6
		1, 10	7.6
		12	12.2
		13	9.10
		1	37.7
		?	10.05
	1979[e]	None	2.95
		9	63.1
		11	4.21
		9, 11	3.24
	1984[e]	None	32.5
		9	13.14
		11	8.59
		?	16.66
Michigan Soft White Winter	1959[c]	None	83
	1979[e]	None	65.8
		?	3.50
New York Soft White Winter	1979[c]	None	83.11
	1984[e]	None	37.71
		10, 13	33.79
		?	5.96
North Dakota Hard Red Spring	1980[f]	1	3.50
		10	62.4
		2a, 10	20.9
		2b, 10	1.70
		10, 13, 34	3.70
		2b, 3, 10	.80
Canadian prairies Hard Red Spring	1966[g]	None	65
		10, 16	33
		13	2
	1975	None	15
		13	77
		10, 16	5
	1987	None	10
		13	69
		13, 16	18
		1, 2a, 12, 13	2

[a] *Lr* genes for resistance known to be present in cultivars grown in the area. Data in part from P. L. Dyck and A. P. Roelfs (personal communication), Briggle and Reitz (3), and Statler (33).
[b] Percentage of wheat acreage for each area.
[c] Distribution and wheat acreage from Reitz and Briggle (21).
[d] Distribution and wheat acreage from Reitz et al (22).
[e] Distribution and wheat acreage from Sieganthaler et al (29).
[f] Distribution and wheat acreage from Statler et al (34).
[g] E. M. Czarnecki (personal communication).

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