Evolution of Distinct Populations of *Puccinia recondita* f. sp. *tritici* in Canada

J. A. Kolmer

Research scientist, Agriculture Canada, Research Station, 195 Dafoe Road, Winnipeg, MB, Canada R3T 2M9.
Contribution 1413.
I thank the past and present rust researchers of the Winnipeg Research Station.
Accepted for publication 17 September 1990 (submitted for electronic processing).

ABSTRACT

Kolmer, J. A. 1991. Evolution of distinct populations of *Puccinia recondita* f. sp. *tritici* in Canada. Phytopathology 81:316-322.

The eastern and prairie populations of *Puccinia recondita* f. sp. *tritici* in Canada had similar identities and frequencies of Unified Numeration (UN) races during the initial years of the annual wheat leaf rust survey (1931–1937) when susceptible cultivars were grown in both regions. Pathogenicity associations within each population also were very similar during this period. Differences between the eastern and prairie populations became evident after the introduction of leaf rust-resistant cultivars in the prairies in 1937. A series of resistant cultivars was released in this region, placing a continuous selection pressure on the corresponding virulences in the prairie leaf rust population. This resulted in cycles of selection and displacement of virulent and avirulent races, respectively, in this region. Susceptible cultivars continued to be grown in the eastern region, and the leaf rust population in this region has since had a different succession of UN races than the prairie population. The eastern and prairie populations of *P. r. tritici* are currently very different in terms of distinct phenotypes. These regional populations most likely originated from a common, introduced population of *P. r. tritici*, and the regional differences have resulted from the use of leaf rust-resistant and susceptible wheat cultivars.

Additional keywords: physiologic specialization, *Triticum*.

Distinct populations of *Puccinia recondita* f. sp. *tritici* Rob. ex. Desm. (causal organism of wheat leaf rust disease) in Canada have been characterized in the eastern region of Ontario and Quebec, the prairie region of Manitoba, Saskatchewan, and northern Alberta, and the Pacific region of southern Alberta and British Columbia (14). Very little overlap currently exists among the three populations. In the 1988 survey of physiologic specialization, Kolmer (11) did not find any single phenotype of *P. r. tritici* in all three regions. Of 44 distinct phenotypes identified in 1988, only two were found in common between the eastern and prairie populations, two between the eastern and Pacific populations, and two between the prairie and Pacific populations.

Kolmer (13) examined the history of virulence and Unified Numeration (UN) race dynamics in the three populations of *P. r. tritici* in Canada during 1956–1987. In the eastern and Pacific regions where susceptible wheat cultivars generally have been grown, UN race and virulence dynamics generally followed a fluctuating nondirectional trend. In the prairie region where a series of resistant cultivars has been released since 1937, UN race and virulence dynamics followed strong directional trends that were consistent with selection of *P. r. tritici* isolates virulent on the corresponding resistance genes in the host population.

The eastern and prairie populations were characterized further by their distinct associations of virulences to specific resistance genes and their differing levels of phenotypic diversity (8,12). Using the UN differentials, the eastern population generally has had higher levels of phenotypic diversity than the prairie region since 1959 (14). Using an expanded set of 12 differentials, phenotypic diversity was higher in the eastern and Pacific populations than in the prairie population (14) in the 1988 survey.

Johnson (10) summarized the results of the wheat leaf rust virulence surveys from 1931 to 1955. International Standard races 1 and 11 were prevalent in British Columbia; races 76 and 58 predominated in eastern Canada; and races 5, 9, 15, 126, and 128 were the most common phenotypes in the prairie region. The leaf rust resistance genes present in the original International Standard differentials have since been identified (6), making it possible to convert International Standard races to modified UN races and also to determine virulence frequencies to the resistance genes *Lr1*, *Lr2a*, *Lr2c*, and *Lr3* (13). This article examines virulence and UN race dynamics and pathogenicity associations in the eastern and prairie populations of *P. r. tritici* from 1931 to 1960 to determine the degree of similarity between the two populations before and during the initial release of leaf rust-resistant cultivars in the prairie region. Also examined were the changes in distinct phenotype frequencies and frequencies of individual virulences to specific resistance genes from 1986 to 1989 in the prairie region to determine the combined effects of directional selection and pathogenicity associations in this population.

MATERIALS AND METHODS

The survey data during 1931–1960 from the eastern and prairie regions were converted from International Standard race designations to the modified UN nomenclature as previously described (13) (Table 1). Percentages of isolates virulent on lines with resistance genes *Lr1*, *Lr2a*, *Lr2c*, and *Lr3* also were determined. Percentages of isolates virulent on the wheat cultivar Renown, which has resistance gene *Lr14a*, were determined from 1946 to 1954. Percentages of isolates virulent on the wheat cultivar Lee (*Lr10*) were determined from 1955 to 1963 for both regions. Frequencies of UN races 2, 3, 5, and 6 in both regions were examined through 1963. Pathogenicity associations in both populations from 1939 to 1946 and in the prairie population from 1986 to 1989 were examined using contingency tables and the *G* log-likelihood test for independence as previously described (1,11). The frequencies of the predominant *P. r. tritici* phenotypes during 1986–1989 in the prairie region were determined using isogenic lines with resistance genes *Lr1*, *Lr2a*, *Lr2c*, *Lr3*, *Lr9*, *Lr16*, *Lr24*, *Lr26*, *Lr3ka*, *Lr11*, *Lr17*, and *Lr30* (16).

RESULTS

Virulence and race dynamics from 1931 to 1959. The eastern and prairie populations had similar identities and frequencies of UN races from 1931 to 1944 (Figs. 1A and 2A). UN races 2, 3, 5, and 9 were found at varying frequencies between 0 and 60% in both populations during this period. Race 9 (10–60%) and race 2 (5–35%) were the most prevalent races in the prairie region and comprised 0–35% and 20–60%, respectively, of the

TABLE 1. Avirulence/virulence formulas of Unified Numeration (UN) races of *Puccinia recondita* f. sp. *tritici*

UN race	Avirulence (A) and virulence (V) on UN differential host			
	Lr1	*Lr2a*	*Lr2c*	*Lr3*
1	A	A	A	A
2	A	A	A	V
3	A	A	V	V
5	V	A	A	V
6	V	A	V	V
9	V	V	V	A
13	V	V	V	V
14	V	A	V	A
17	A	V	V	V

isolates in the eastern region. Race 3 comprised 0–35% of the isolates in the prairie region and 0–20% of the isolates in the eastern population. Race 5 varied between 0 and 30% in the prairie population and 0 and 15% in the eastern population.

Frequencies of virulence to the UN differentials during 1931–1944 were generally intermediate to high in both regions (Figs. 1B and 2B). Virulence to *Lr1* was 30–88% in the prairies and 20–90% in the eastern region. Virulence to *Lr2a* was 40–55% in both regions. Virulence to *Lr2c* was 50–90% in the prairies and 50–75% in the eastern region. Virulence to *Lr3* was 50–85% in both regions.

Differences in race and virulence frequencies between the two populations increased after the widespread introduction of leaf rust-resistant cultivars in Canada and the United States. In 1937

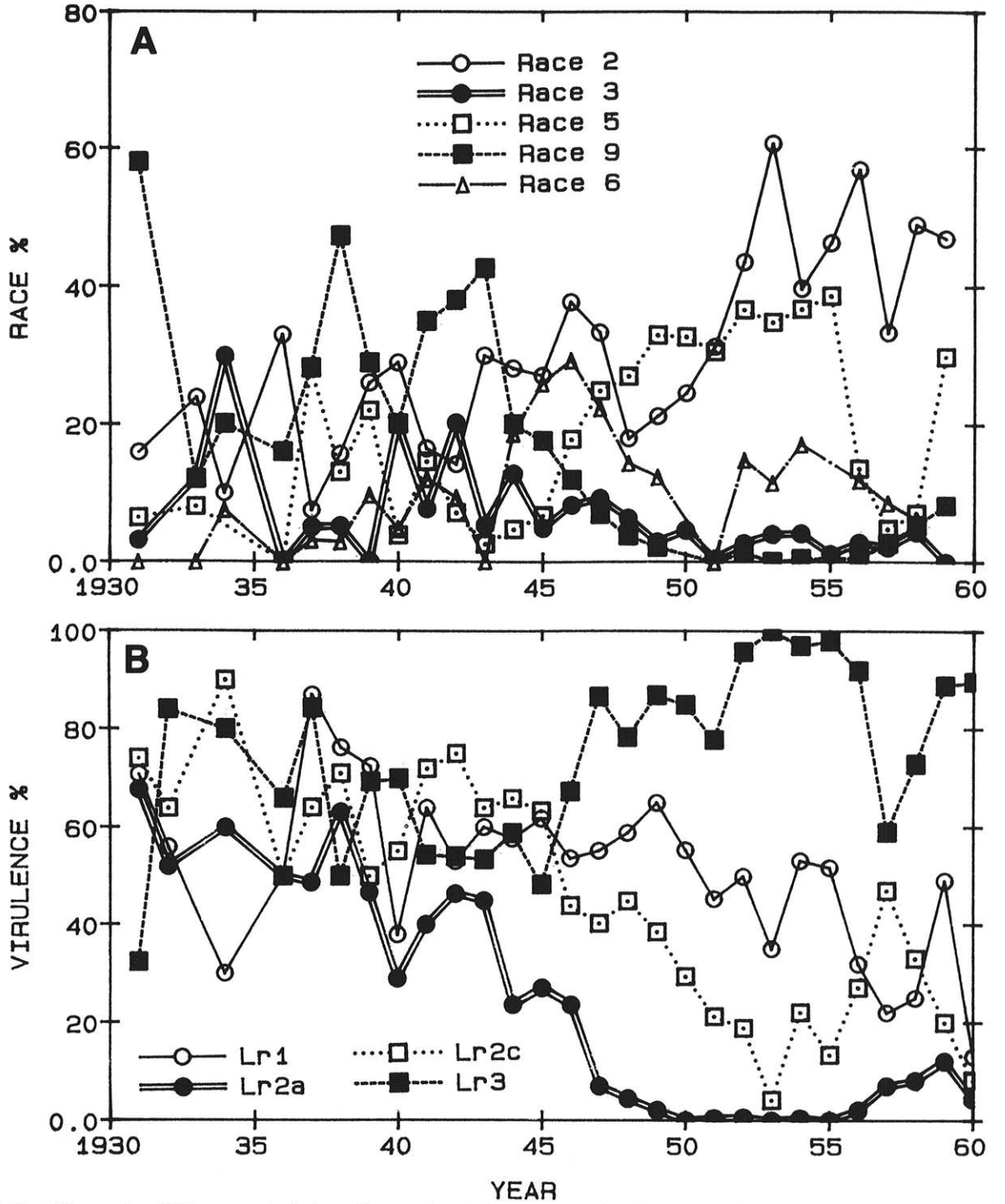

Fig. 1. Unified Numeration (UN) race and virulence frequencies of *Puccinia recondita* f. sp. *tritici* in the prairie region of Canada (Manitoba, Saskatchewan) during 1931–1959. **A,** Percent of UN races. **B,** Percent of isolates of *P. r. tritici* virulent on *Lr1*, *Lr2a*, *Lr2c*, and *Lr3*.

the cultivar Renown with resistance gene *Lr14a* was released for production in Canada. Renown and subsequent derivatives quickly became the predominant cultivars in the prairie region (7). The winter wheat Pawnee with *Lr3* was distributed in the central Great Plains region of the United States in 1943 (22) and rapidly became the dominant cultivar in this region. The cultivars Mediterranean (*Lr3*), Hope (*Lr14a*), and Pawnee were the sources of leaf rust resistance for many cultivars released in this region through at least 1960. Leaf rust-susceptible cultivars continued to be grown in the soft white wheat area of eastern Canada.

The effects of the introduced resistance genes were observed quickly in the prairie leaf rust population. In this population during 1944–1960, race 9 and race 3 declined to very low levels (0–5%), whereas race 2 and race 5 increased to 70 and 50%, respectively (Fig. 1A). Race 5 began to decline in frequency after 1954. In the eastern region, race 3 became the predominant race (25–70%), whereas the other UN races declined in frequency to 0–25% (Fig. 2A).

Frequency of isolates with virulence to *Lr3* in the prairies increased to 80–100% after 1947 (Fig 1B). Virulences to *Lr1* and *Lr2c* in this region gradually declined from 50% in 1947 to less than 10% in 1960. Virulence to *Lr2a* was extremely low (0–5%) after 1947. Forty-three percent of the isolates were virulent on Renown (*Lr14a*) in 1946, the first year this cultivar was included in the differential set. Virulence to *Lr14a* increased rapidly in the prairie region, reaching more than 90% by 1953.

In the eastern population during this period, virulence to *Lr1*

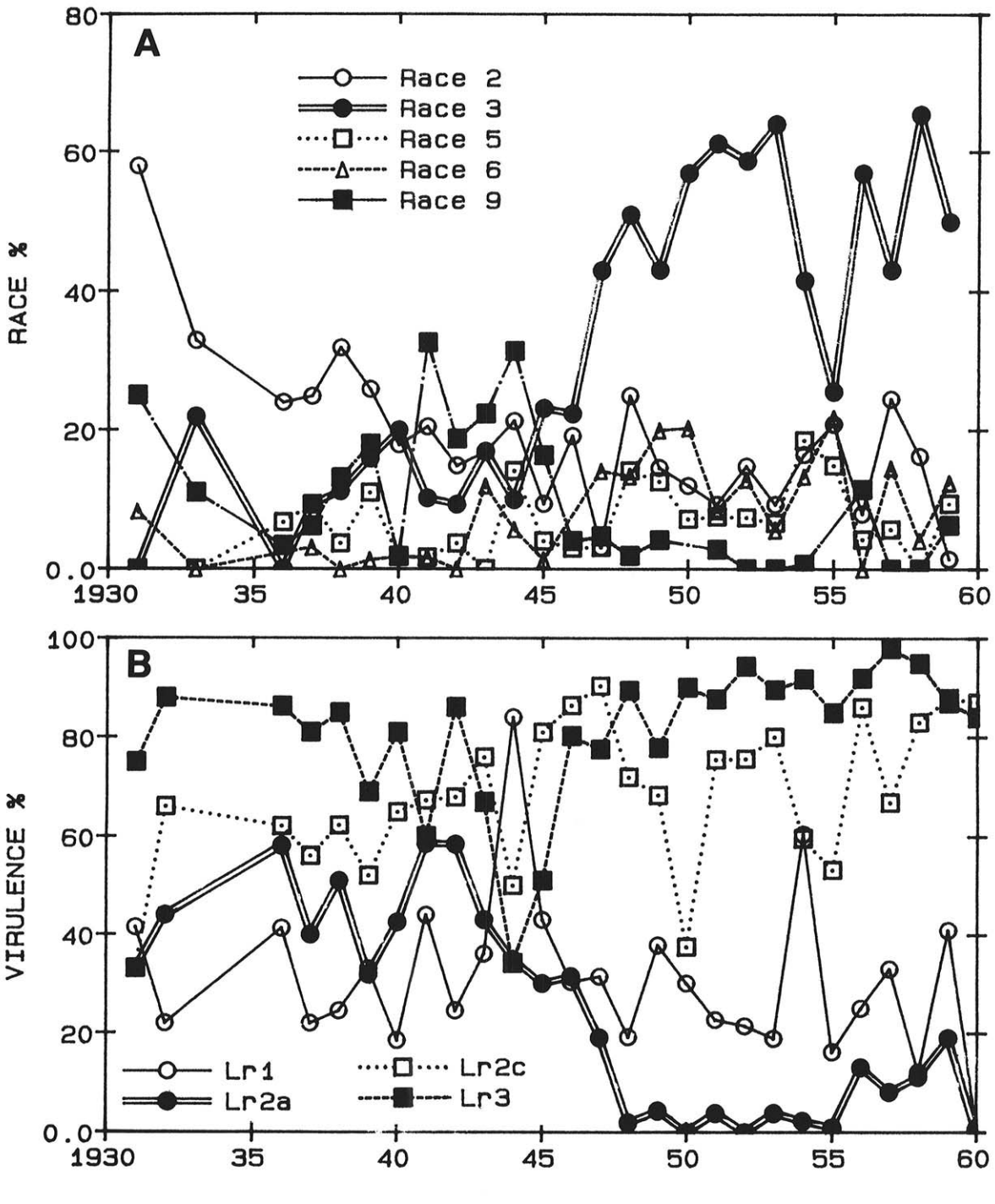

Fig. 2. Unified Numeration (UN) race and virulence frequencies of *Puccinia recondita* f. sp. *tritici* in the eastern region of Canada (Ontario, Quebec) during 1931–1957. **A,** Percent of UN races. **B,** Percent of isolates of *P. r. tritici* virulent on *Lr1*, *Lr2a*, *Lr2c*, and *Lr3*.

was found in 20–50% of the isolates (Fig. 2B). Virulence to *Lr2a* was extremely low (0–15%), whereas virulence to *Lr2c* varied from 45 to 90%. Virulence to *Lr3* was very high, varying from 85 to 95%. Eight percent of isolates in the eastern region were virulent on Renown (*Lr14a*) in 1946. Virulence to this gene increased to 41% by 1949, then declined to 20% by 1954.

Pathogenicity associations. The eastern and prairie populations were characterized by nearly identical pathogenicity associations from 1939 to 1946 (Table 2). For each virulence pair, associations were similar for both the eastern and prairie populations. The largest differences between the two populations were significant pathogenicity associations in the prairie population and nonsignificant associations in the eastern population for certain years and virulence pairs. Virulences to *Lr1* and *Lr2a*, and *Lr1* and *Lr2c* were positively associated in both populations. Virulences to *Lr1* and *Lr3*, *Lr2a* and *Lr3*, and *Lr2c* and *Lr3* were dissociated in both populations. The pathogenicity associations stayed relatively constant during this period, varying only in the eastern population between a significant association in either direction and a nonsignificant association for each virulence pair. Virulence to *Lr14a* in the prairie population from 1946 to 1949 was found predominantly in UN races 2 and 5. Virulence to *Lr14a* in the eastern population during this period most commonly was found in UN races 2, 5, 6, and 14.

Virulence to *Lr10* and frequency of UN race 2. The hard red spring wheat cultivar Lee (*Lr10*) was licensed for production in Canada in 1950 and was widely grown in the prairie region (7). Selkirk (*Lr10* + *Lr16*) was released in 1954 and soon became the predominant cultivar grown in the prairie provinces and the north central states because of its resistance to wheat stem rust race 15B (7). Isolates with virulence to *Lr10* have a selective advantage on Selkirk because resistance gene *Lr16* conditions an avirulent infection type that allows a moderate amount of sporulation. Virulence to *Lr10* in the differential sets was at 3.5% in the prairie leaf rust population in 1955 and steadily increased to more than 80% by 1963 (Fig. 3A). Virulence to *Lr10* arose predominantly in UN race 2, which as a result increased from 45% in 1955 to 87% by 1963. As virulence to *Lr10* and race 2 increased in frequency, races 5 and 6 declined from 45 and 12%, respectively, in 1955 to 10% and 0% by 1963.

In the eastern population, virulence to *Lr10* remained low (<10%) until 1961 when it increased to 35% (Fig. 3B). As in the prairie population, virulence to *Lr10* in the east was found predominantly in race 2 isolates. However, Lee and Selkirk were not widely grown in this region, and virulence to *Lr10* and race 2 did not increase to the high levels observed in the prairie population. As a result, the race composition in the eastern population did not undergo the same degree of change observed in the prairies. Race 2 increased to 40% in 1961 from previous levels of 0–25%; race 6 declined from 22% in 1955 to very low levels (0–3%) after 1960; and race 3 remained at levels between 25 and 65% during this period (Fig. 3B).

TABLE 2. Pathogenicity associations between virulences in the prairie and eastern populations of *Puccinia recondita* f. sp. *tritici* in Canada during 1939–1946

Virulence pair	Pathogenicity association[a]	
	Prairie population	Eastern population
Lr1 Lr2a	+	+[b]
Lr1 Lr2c	+	+[c]
Lr1 Lr3	–	–
Lr2a Lr3	–	–[d]
Lr2c Lr3	–	–[e]

[a] Associations were determined using a *G*-test for goodness of fit. + = significant association ($G > 3.841$, 1 df); NS = nonsignificant association ($G < 3.841$, 1 df); – = significant negative association ($G > 3.841$, 1 df).
[b] NS in 1945, 1946.
[c] NS in 1939, 1945.
[d] NS in 1942, 1945, 1946.
[e] NS in 1940, 1941.

Virulence dynamics in the prairie region, 1986–1989. In 1986, three phenotypes of *P. r. tritici* (virulence formulas 2a, 2c, 3; 1, 3; and 1, 2a, 2c, 3) accounted for 72% of the leaf rust population in the prairie region (Table 3). Virulences to *Lr11*, *Lr24*, and *Lr26* were 0.5, 7.58, and 0%, respectively, in 1986 (Table 4). By 1989 isolates with virulence formulas 2a, 2c, 3, and 1, 3 had declined to 0 and 1.6%, respectively, and isolates with virulence formula 1, 2a, 2c, 3 comprised 23% of the population. Isolates with virulence to *Lr11* or *Lr24* and *Lr26* had increased to 69.7% of the leaf rust population in 1989 from very low frequencies in 1986. In 1989 virulence frequencies to *Lr11*, *Lr24*, and *Lr26* had increased to 43.9, 29.13, and 25.2%, respectively.

The increase of isolates with virulence to *Lr24* and *Lr26* can be attributed to the use of these genes in the hard red winter wheat cultivar Siouxland registered in 1985 and grown from South Dakota to northern Texas (17). The increase of isolates with virulence to *Lr11* possibly can be attributed to the release of Pioneer 2157, a hard red winter wheat cultivar that may have *Lr11* (D. L. Long, *personal communication*).

The recent changes in the prairie leaf rust population are reflected in the three Rogers indexes (8) determined for pairs of succeeding years from 1986 to 1989 (Table 3). The Rogers index for 1986 paired with 1989 is 0.6924, indicating the high degree of overall change in the prairie leaf rust population over 4 yr. Pathogenicity associations in the prairie region from 1987 to 1989 are given in Table 5. Virulences to *Lr24* and *Lr26* were associated with each other and with virulence to *Lr1*, and they were dissociated with virulences to *Lr2a* and *Lr11*. Virulence to *Lr11* was dissociated with *Lr1* and associated with virulence to *Lr2a*. Virulences to *Lr1* and *Lr2a* were dissociated.

Frequencies of virulence to *Lr1* and *Lr2a* changed relatively little (3 and 6%, respectively) from 1986 to 1989 (Table 4). Virulence frequencies to *Lr1* and *Lr2a* should be influenced by the selection and increase of isolates with virulences to *Lr11*, *Lr24*, and *Lr26* because of the pathogenicity associations these virulences have with virulences to *Lr1* and *Lr2a*. Virulence frequency to *Lr1* should be increased by the selection and increase of isolates with virulence to *Lr24* and *Lr26* and should be decreased by the selection and increase of isolates with virulence to *Lr11*. Similarly, virulence frequency to *Lr2a* should be negatively influenced by the increase of isolates with virulence to *Lr24* and *Lr26* and positively influenced by the increase of isolates with virulence to *Lr11*. A simple regression with the net changes in frequencies of virulence to *Lr26* and *Lr11* as they affect virulence frequencies to *Lr1* and *Lr2a* as the independent variables and the changes in *Lr1* and *Lr2a* virulence frequencies as the dependent variables was set up to test the hypothesis that increasing virulence to *Lr24*, *Lr26*, and *Lr11* influenced virulence frequencies to *Lr1* and *Lr2a*. Changes in frequency to *Lr24* were not considered because virulences to *Lr24* and *Lr26* are associated (Table 5). As an example, between 1987 and 1988, virulence to *Lr11* increased by 8.56%, virulence to *Lr26* increased by 20.46% (independent variables), and virulence to *Lr2a* (dependent variable) decreased by 6.80% (Table 4). The net change of the independent variables as they affect frequency of virulence to *Lr2a* is −11.90 (8.56 − 20.46). The same calculations involving changes in virulence frequencies to *Lr1* and *Lr2a*, and *Lr11* and *Lr26* between the years 1986–1987, 1987–1988, and 1988–1989 were performed. The regression of the changes in virulence frequencies to *Lr1* and *Lr2a* on the net change in virulence frequencies to *Lr11* and *Lr26*, as they affect virulence frequencies to *Lr1* and *Lr2a*, was highly significant ($P = 0.0077$, 5 df) with a coefficient of determination (R^2) of 0.8603. The change in virulence frequencies to *Lr1* and *Lr2a* was significantly correlated with the change in virulence frequencies to *Lr11* and *Lr26*.

DISCUSSION

The current distinct populations of *P. r. tritici* in the eastern and prairie regions of Canada most likely arose from a common original population. The regional populations developed through the release of a series of resistant cultivars in the prairie region

starting in 1937 and the continued general use of susceptible cultivars in the eastern region. Before the introduction and spread of resistant cultivars in the Great Plains region of North America, the two leaf rust populations had similar UN race identities and frequencies, similar levels of phenotypic diversity (14), and nearly identical pathogenicity associations. The introduction of host resistance genes in the prairie region has resulted in the continuous selection of *P. r. tritici* isolates with virulence to the introduced resistance genes. In the eastern populations, selection pressure exerted by host resistance genes has not been as prevalent, which has contributed to the fluctuating nature of the virulence and race dynamics and the generally higher levels of phenotypic diversity observed in this population (14).

The release of Renown (*Lr14a*) and Pawnee (*Lr3*) (7,22) was followed by the increase of UN races 2 and 5 in the prairie region because both phenotypes are virulent to *Lr3* and because virulence to *Lr14a* arose mainly in these two races. Race 5 declined after 1954 because virulence to *Lr10* was associated with race 2. The decline of race 5 and increase of race 2 can be attributed only to the increase of virulence to *Lr10* because both races are virulent to *Lr3*, which was still present in many winter wheats grown in the southern plains of the United States. Virulence to *Lr16* (selected by Selkirk) was not detected until 1961 and increased gradually until 1966 when 55% of the isolates were virulent to this gene (22). However, changing levels of virulence to *Lr16* did not cause any major race shifts because UN race 2 accounted

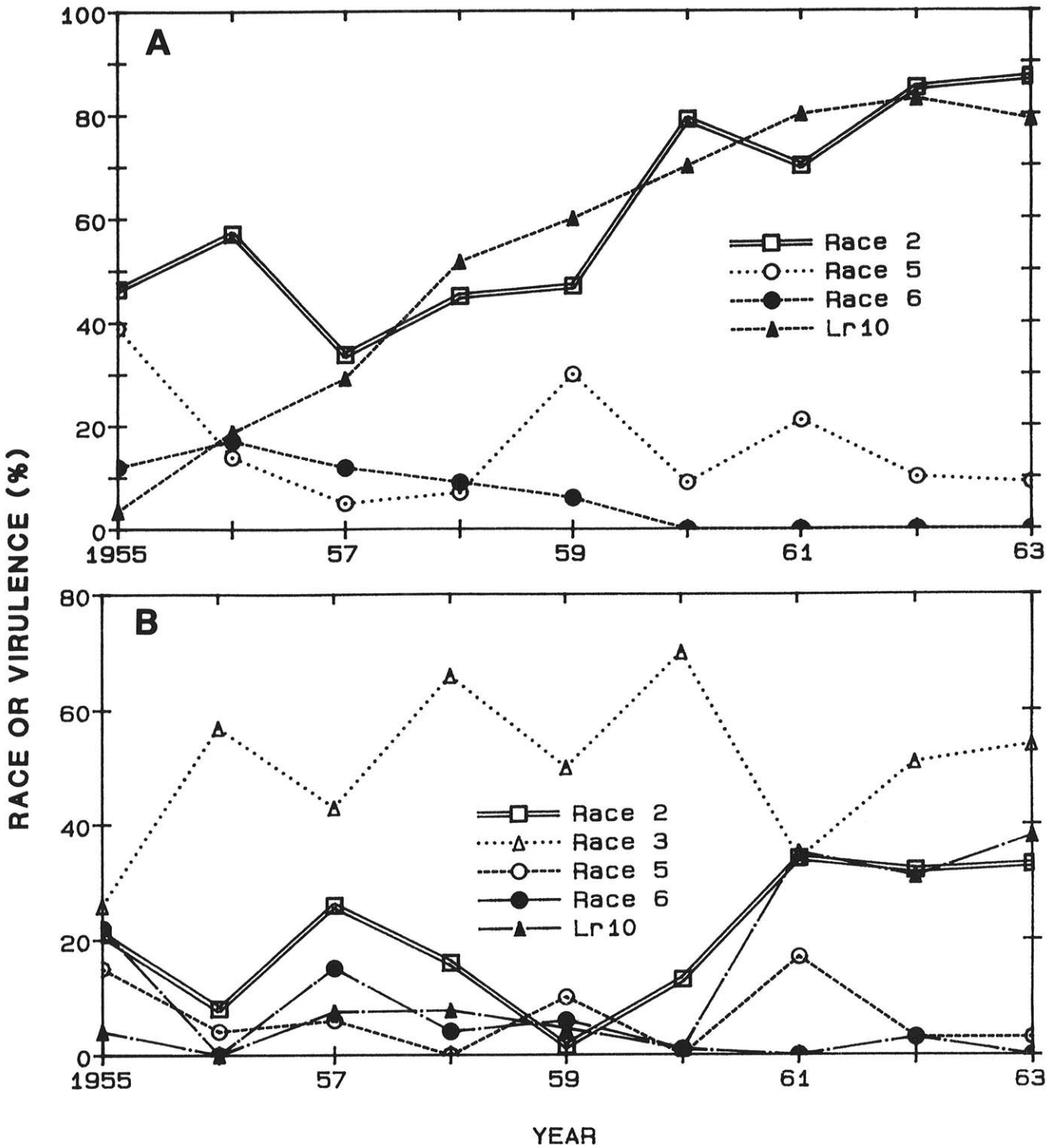

Fig. 3. Unified Numeration (UN) race frequencies and virulence frequencies to *Lr10* of *Puccinia recondita* f. sp. *tritici* in the eastern region of Canada (Ontario, Quebec) and the prairie region (Manitoba, Saskatchewan) during 1955–1963. **A,** Prairie region. **B,** Eastern region.

for 80–100% of the leaf rust population in the prairie region during 1960–1972 (13). Selkirk was replaced with Manitou (*Lr13*) starting in 1966. Virulence to *Lr13* appeared shortly after Manitou was released; however, it occurred in that portion of the leaf rust population avirulent to *Lr16*, resulting in the decline of virulence to *Lr16*. Race 2 dominated the leaf rust population in the prairies until 1978 when cultivars with resistance genes *Lr1*, *Lr2a*, and *Lr2c* were released in Minnesota and North Dakota (17,19,23). Virulence to these genes rapidly increased. Races 5, 13, and 17 increased, and race 2 declined because it was not virulent on the newly introduced resistance.

The history of race dynamics in the eastern population of *P. r. tritici* is more difficult to account for because susceptible soft white winter wheat cultivars generally have been grown in southern Ontario and parts of Quebec. Local populations of *P. r. tritici* may arise in this region due to potential overwintering of the fungus. Leaf rust-resistant soft red winter wheat cultivars grown in the eastern United States also will influence the leaf rust population in eastern Canada. Furthermore, virulence combinations typically found in the prairie population also are found in the eastern population, indicating a common inoculum source for the two populations. The increase of race 2 in 1960 most likely can be attributed to the parallel selection and increase of race 2 in the prairies. Isolates of race 2 may have been wind blown from the southern and central plains of the United States to the susceptible wheats in southern Ontario. The decline of race 2 in the eastern region after 1976 also parallels the decrease of race 2 in the prairies (13). Races 3 and 6 became the most common phenotypes in the eastern populations after race 2 declined. There is no apparant explanation for this other than the hypothesis that isolates of races 3 and 6 were also virulent on an additional *Lr* gene(s) that was used in wheats grown in the eastern United States or eastern Canada. The cultivar Blueboy (3) grown in the southeastern United States has gene *Lr1*, which may explain the increase of race 6 which has virulence to *Lr1*. Races 5 and 13 increased in the eastern population in 1986 and 1987, paralleling the increase of these two races in the prairie region during the same period (13).

Isolates of *P. r. tritici* avirulent to *Lr2a* and virulent to *Lr2c* currently are found in the eastern population but not in the prairies (11). Isolates in the prairie population are either avirulent to *Lr2a* and *Lr2c* or are virulent to both genes. This pathogenicity association between *Lr2a* and *Lr2c* is the most characteristic difference between the eastern and prairie populations. UN races 3 and 6, which are both avirulent to *Lr2a* and virulent to *Lr2c*, were present in intermediate to low (0–20%) frequencies in the prairie region from 1931 to 1960; since then, neither race has been found at a frequency greater than 1%. Races 3 and/or 6 has been present in the eastern population at frequencies between 0 and 65% since the leaf rust survey began in 1931. Races 3 and 6 disappeared in the prairie population after the release of Selkirk and the corresponding increase of virulence to *Lr10* and race 2.

It is unlikely that selection against unneeded virulences in North American populations of *P. r. tritici* significantly influences virulence frequencies in the rust population. The susceptible host population in the eastern region has maintained a population of *P. r. tritici* that has intermediate frequencies of virulence to many resistance genes that have no history of use in this region (13). The release of a series of resistant cultivars in the prairie region has created cycles of selection and displacement of specific virulences similar to Person's idealized model (20). The selective forces arising from directional selection of virulent phenotypes, the effects of pathogenicity associations, and displacement of avirulent isolates are most likely much greater than selective effects due to unneeded virulences. Selection against unneeded virulences would not have a chance to influence the population structure given the overwhelming selective effects of these other factors. Of course, in a leaf rust population where the sexual stage contributes significantly to the epidemiology of the disease, selective forces due to stabilizing selection may have a chance to influence levels of virulence polymorphism. However, this is not the case in North America.

If virulences were randomly distributed throughout the leaf rust population in North America, the selection and increase of specific virulences would not affect frequencies of other virulences. The effects of selection and pathogenicity associations between virulences are seen in the relatively little change in virulence frequencies to *Lr1* and *Lr2a* in the prairie region from 1986 to 1989. This population changed very significantly in terms of distinct phenotypes during this period. However, the virulence frequencies to *Lr1* and *Lr2a* did not reflect this change because the opposing selective forces of increasing virulences to *Lr11*, and *Lr24*, *Lr26* effectively maintained virulences to *Lr1* and *Lr2a* at fairly constant frequencies. If virulences to *Lr11*, *Lr24*, and *Lr26* occurred at random in the rust population, the increase of these virulences would not affect other virulence frequencies. Virulences to *Lr1* and *Lr2a* are also under direct selection themselves because these genes are currently in wheats grown in this area (19,23). Without the balancing effect of opposing selective forces caused by pathogenicity associations, virulences to any genes that are under directional selection would be expected to increase to a frequency of 100% and remain fixed in the population, as has happened with virulences to *Lr3*, *Lr10*, and *Lr14a* in the prairie region (13). Virulences to *Lr1* and *Lr2a* began to increase in 1976 after cultivars with these genes were released in North Dakota and Minnesota (13,19,23). However, after 13 yr of increasing in frequency, virulences to both of these genes have not reached fixation. This can be attributed only to the

TABLE 3. Percentages of virulence formulas of *Puccinia recondita* f. sp. *tritici* in the prairie region of Canada from 1986 to 1989

	Frequency (%)			
Virulence formula	1986	1987	1988	1989
2a,2c,3	13.06	11.02	3.60	0.40
2a,2c,3,11	0.56	0.38	1.60	10.70
1,3	26.70	6.80	3.20	1.60
1,3,24	3.97	28.51	24.30	4.50
1,3,24,26	0.00	2.66	21.50	25.40
1,2a,2c,3	32.95	34.60	31.90	23.00
1,2a, 2c,3,11	0.00	1.14	8.40	29.10
Rogers index	0.3405	0.2930	0.3939	

TABLE 4. Percentages of isolates of *Puccinia recondita* f. sp. *tritici* in the prairie region of Canada virulent on isogenic leaf rust differential lines from 1986 to 1989

	Frequency (%)			
Virulence	1986	1987	1988	1989
Lr1	85.70	87.80	94.40	88.69
Lr2a	60.22	55.80	49.00	66.52
Lr11	0.56	3.04	11.60	43.90
Lr24	7.58	31.55	47.00	29.13
Lr26	0.00	3.04	23.50	25.20

TABLE 5. Pathogenicity associations between virulences in the prairie population of *Puccinia recondita* f. sp. *tritici* in Canada during 1987–1989

Virulence pair	Pathogenicity association[a]
Lr1 Lr2a	−
Lr1 Lr24	+
Lr1 Lr26	+
Lr1 Lr11	−
Lr2a Lr24	−
Lr2a Lr26	−
Lr2a Lr11	+
Lr24 Lr11	−
Lr26 Lr11	−
Lr24 Lr26	+

[a] Associations were determined using a *G*-test for goodness of fit. + = significant positive association ($G > 3.841$, 1 df); − = significant negative association ($G > 3.841$, 1 df).

balancing selective forces caused by pathogenicity associations and directional selection of other virulences. As previously noted (12), virulences to *Lr1* and *Lr2a* have been negatively associated since 1975, which also would contribute to maintaining polymorphism because direct selection for one virulence would decrease the frequency of the other virulence.

Mathematical models based on gene-for-gene systems have concluded that balanced virulence polymorphisms in pathogen populations are not possible without some form of selection against unneeded virulences, or stabilizing selection (9,15,24). In the absence of stabilizing selection, directional selection for virulence or, in the case of finite populations, random drift inevitably will carry a virulence to either fixation or possibly extinction. Genetic equilibrium is maintained by the opposing forces of directional selection for virulence, and selection against unneeded virulences. The models all implicitly assume that single virulences are the selective units in the pathogen population: that is, the virulences are randomly distributed, and changes in frequency of one virulence are independent of the changes in other virulences. However, in the prairie population of *P. r. tritici* in Canada, it is evident that directional selection for virulence can be effectively balanced by the directional selection of dissociated virulences. In this case, an apparent equilibrium is observed due to the combination of directional selection and nonrandom distribution of virulences in the rust population. Although selection for individual virulences is responsible for changes in frequencies of distinct phenotypes, the unit of selection is the entire phenotype itself.

Chester (5) and Arthur (2) have stated that *P. r. tritici* (*P. rubigo-vera tritici*) originated from the same area of southwest Asia where *Triticum* spp. are native. Studies with the alternate hosts have supported the hypothesis that *P. r. tritici* was introduced into North America (18,21). The prairie and eastern populations of *P. r. tritici* in Canada are most likely derived from a common initial population introduced to North America from Europe. The similarity of the geographically separated prairie and eastern populations before resistant cultivars were introduced in the prairies and the paucity of isozyme variation in North American leaf rust isolates (4) suggest a limited range of diversity and small effective population sizes in the initial introductions. *P. r. tritici* probably was introduced first to the eastern seaboard of North America when permanent agricultural settlements were established in the early 17th century. The initial population then would have spread throughout the eastern part of the continent and into the Great Plains region as domestic agriculture became established in North America. Distinct populations of *P. r. tritici* then would have evolved through regional differences in the wheat varieties and cultivars grown.

LITERATURE CITED

1. Alexander, H. M., Roelfs, A. P., and Groth, J. V. 1984. Pathogenicity associations in *Puccinia graminis* f. sp. *tritici* in the United States. Phytopathology 74:1161-1166.
2. Arthur, J. C. 1934. Manual of the Rusts in the United States and Canada. Purdue Research Foundation, Lafayette, IN. 438 pp.
3. Browder, L. E. 1980. A compendium of information about named genes for low reaction to *Puccinia recondita* in wheat. Crop Sci. 20:775-779.
4. Burdon, J. J., and Roelfs, A. P. 1985. Isozyme and virulence variation in asexually reproducing populations of *Puccinia graminis* and *P. recondita* on wheat. Phytopathology 75:907-913.
5. Chester, K. S. 1946. The Cereal Rusts. Chronica Botanica Co., Waltham, MA. 269 pp.
6. Dyck, P. L., and Samborski, D. J. 1968. Genetics of resistance to leaf rust in the common wheat varieties Webster, Loros, Brevit, Carina, Malakof, and Centenario. Can. J. Genet. Cytol. 10:7-17.
7. 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.
8. Groth, J. V., and Roelfs, A. P. 1987. The concept and measurement of phenotypic diversity in *Puccinia graminis* on wheat. Phytopathology 77:1395-1399.
9. Jayakar, S. C. 1970. A mathematical model for interaction of gene frequencies in a parasite and its host. Theor. Popul. Biol. 1:140-164.
10. Johnson, T. 1957. Physiologic races of leaf rust of wheat in Canada. Can. J. Agric. Sci. 36:323-332.
11. Kolmer, J. A. 1989. Physiologic specialization of *Puccinia recondita* f. sp. *tritici* in Canada in 1988. Can. J. Plant Pathol. 11:431-435.
12. Kolmer, J. A. 1989. Nonrandom distribution of virulence and phenotypic diversity in two populations of *Puccinia recondita* f. sp. *tritici* in Canada. Phytopathology 79:1313-1317.
13. Kolmer, J. A. 1989. Virulence and race dynamics of *Puccinia recondita* f. sp. *tritici* in Canada during 1956-1987. Phytopathology 79:349-356.
14. Kolmer, J. A. 1991. Phenotypic diversity in two populations of *Puccinia recondita* f. sp. *tritici* in Canada during 1931–1987. Phytopathology 81:311-315.
15. Leonard, K. J. 1977. Selection pressures and plant pathogens. Pages 207-222 in: The Genetic Basis of Epidemics in Agriculture. P. R. Day, ed. New York Academy of Sciences, New York.
16. Long, D. L., and Kolmer, J. 1989. A North American system of nomenclature for *Puccinia recondita* f. sp. *tritici*. Phytopathology 79:525-529.
17. Long, D. L., Schafer, J. F., and Roelfs, A. P. 1985. Specific virulence of *Puccinia recondita* f. sp. *tritici* on wheat in the United States. Plant Dis. 69:343-347.
18. Mains, E. B. 1932. Host specialization in the leaf rust of grasses, *Puccinia rubigo-vera*. Pap. Mich. Acad. Sci. Arts Lett. 17:289-394.
19. McVey, D. V. 1989. Verification of infection-type data for identification of genes for resistance to leaf rust in some hard red spring wheat. Crop Sci. 29:304-307.
20. Person, C. O. 1966. Genetic polymorphism in parasitic systems. Nature 212:266-267.
21. Saari, E. E., Young, H. C., and Kernkamp, M. F. 1968. Infection of North American *Thalictrum* spp. with *Puccinia recondita* f. sp. *tritici*. Phytopathology 58:939-943.
22. Samborski, D. J. 1985. Wheat leaf rust. Pages 35-39 in: The Cereal Rusts. II. A. P. Roelfs and W. R. Bushnell, eds. Academic Press, Orlando.
23. Statler, G. D. 1984. Probable genes for leaf rust resistance in several hard red spring wheats. Crop Sci. 24:883-886.
24. Vanderplank, J. E. 1968. Disease Resistance in Plants. Academic Press, New York. 208 pp.