Virulence Dynamics of *Puccinia graminis* f. sp. *avenae* in Canada, 1921–1993

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ABSTRACT

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The virulence dynamics in the oat stem rust fungus, *Puccinia graminis* f. sp. *avenae*, in Canada from 1921 to the present are documented. The reidentification of isolates of *P. g. avenae* stored since 1953 was used as the basis to relate virulences in older and current populations. Compared to other cereal rust fungi in North America, virulence in *P. g. avenae* appears highly stable. Common pathotypes (races) of *P. g. avenae* have tended to dominate populations for 25 yr or longer—some races have been isolated for about 40 yr. In the prairie region, virulence to genes *Pg9* and *Pg13*, currently important resistance sources, was relatively common (races NA3 and NA7 were the most frequently identified from stored isolates of races 1/5-C1 and 2-C2, respectively), then declined with the emergence and dominance of race 6AF/C10/NA27 in the 1960s. However, because races NA3 and NA7 are avirulent to cultivars carrying gene *Pg2*, and given the asexual nature of the prairie *P. g. avenae* population, the maintenance of *Pg2* resistance in contemporary cultivars should reduce the threat to *Pg13* resistance in this region. The frequency of virulence to *Pg15* was very high across Canada but declined in the prairie region along with virulence to *Pg9* and *Pg13*. Virulence to *Pg16* has occurred in only one year, and virulence to gene *Pga* was found once in this study. Virulence to all of the *Pg* resistances has occurred at some time in the North American *P. g. avenae* populations, regardless of the exposure of the populations to these resistances.

Additional keywords: historical virulence, race dynamics.

Stakman et al (19) first recognized physiologic specialization in the oat stem rust fungus, *Puccinia graminis* Pers. f. sp. *avenae* Eriks. & E. Henn. In Canada, identification of virulence pathotypes (races) of *P. g. avenae* began during 1923, using samples collected as early as 1921 (15). The original differential set used in Canada consisted of the lines White Tartar (*Pg1*), Richland (*Pg2*), and Joanette (*Pg3*) (1,15). Lines with gene *Pg4* were added in 1952, with *Pg8* in 1959, and with *Pg9* in 1965. The currently used differential set, including the above genes with the addition of genes *Pg13, Pg15, Pg16,* and *Pga*, was derived in 1979 (14).

A continuous record of annual virulence surveys in Canada is available based on collections of *P. g. avenae* made from 1921 to the present. An important advantage for historical assessment of races of *P. g. avenae* is that the differential genotypes originally used were single-resistance gene lines, and these resistance genotypes are still in use as differentials today. Summaries of incidences and changes in virulence in *P. g. avenae* in Canada have been published for the years 1921–1943 (15), 1944–1959 (3), 1960–1968 (13), and 1969–1977 (12). In the United States, summaries were published for the period 1923–1970 by Stewart and Roberts (20) and for 1948–1977 by Roelfs et al (18).

Isolates of *P. g. avenae*, collected from annual field surveys across Canada or isolated from greenhouse studies, have been stored at the Agriculture and Agri-Food Canada Research Centre in Winnipeg since 1953. These were identified either as the older "standard" races (14,18; identified in Canada prior to 1963), C races (11; identified between 1964 and 1978), or NA races (14; identified since 1979). These stored isolates all were recovered and reidentified (6) using the contemporary NA nomenclature (14). This provided an opportunity to relate the identity of isolates from previous surveys to the present NA race designations and to estimate their longevity as distinct races.

This study has two relevant implications. One is an analysis of the long-term stability of the *P. g. avenae* populations in Canada, with implications for present and future strategies for resistance breeding. The second is to judge the long-term effectiveness of current resistance sources. Gene *Pg13* is now the most important component of the stem rust resistance genotype of oat cultivars released for the rust area of the Canadian prairies. It is important to learn more of the past history and potential of virulence to this and other genes used in oat breeding.

MATERIALS AND METHODS

Summary of occurrence of races and virulence. The documentation and references used in this report are from Newton and Johnson (15) for the period 1923–1943, the annual survey reports from the Plant Pathology Laboratory (now Winnipeg Research Centre) 1944–1960, *Canadian Plant Disease Survey* volumes 41–59 (1961–1978), and *Canadian Journal of Plant Pathology* volumes 2–8, 10–12, 15, and 16 (1979–1994). Specific references relating to these reports generally are not quoted.

Although it is recognized that virulence or avirulence is expressed as a reaction by the host plant, virulence to specific genes is used here to simplify the narrative. The frequencies of races and virulences to the individual *Pg* genes between 1921 and 1963 were calculated as percent occurrence in 5-yr intervals, beginning with the first year of publication (15). The periods during which the C and NA race nomenclatures were used are relatively short; thus, these were averaged on a biennial basis. Only the more frequently occurring races are shown in the figures.

Continuity between nomenclature systems. Previous race designations have been cross-referenced by Martens et al (13) and Stewart and Roberts (20). The races identified in Canada prior to 1963 are referred to in this paper as "standard" races, those between 1964 and 1978 as "C" races (11), and those since 1979 as "NA" races (14). The recovery and identification of isolates of *P. g. avenae* stored at the Winnipeg Research Centre since 1953 has been described (6). This was used as the basis to assess the continuity of race designations from older to current nomenclature systems. Of the stored isolates, 930 had the original standard or C-race designations. Of these, 807 isolates that could be interpreted unambiguously in terms of the NA differentials were used in this study (Table 1). The majority of the stored isolates were from field collections (host cultivar usually not indicated). A small proportion (<10%) came from trap nurseries planted at various locations in Canada. Any isolates from artificially inoculated nurseries were not included.

Estimates of conformity between isolates identified by the various nomenclature systems were made from the number of times a previously identified pathotype could be related to a current NA race (Table 1). The frequencies of occurrence of isolates in storage for particular survey periods generally conformed to the frequencies of occurrence of these races according to the original survey data. The stored isolates, however, did not represent all of the races identified in any one year; thus, it was possible to relate only the most commonly occurring races from 1953 to 1978 to the current NA nomenclature. Although a number of equivalent standard race designations could be related to the current NA system, these associations could not be extended prior to 1953, because no representative samples were available in storage for verification.

In this paper, the regions of Canada are defined as: eastern—the maritime provinces, Quebec, and Ontario; prairie—Manitoba and Saskatchewan; and Pacific—Alberta and British Columbia. Alberta is a transitional zone between the prairie and Pacific zones that may yield isolates characteristic more or less of the prairie and/or Pacific *P. g. avenae* populations (5), depending on the prevailing weather patterns. The annual survey reports indicate that the Alberta *P. g. avenae* population is more characteristic of the Pacific population, therefore frequencies for these two regions were combined.

The original designations (1,15) of races 5, 10, 12, and 13 were based on a mesothetic (infection type X) reaction by lines with gene *Pg3*. Current binary nomenclature systems (17), however, can accommodate only virulent or avirulent interactions. In a previous review (3), broader race groupings were made (e.g., races 1/2/5), but such groupings ignored virulence or avirulence to gene *Pg3*. In other reports (13,20), the mesothetic reaction was interpreted as susceptible, resulting in race groupings such as 2/5, 7A/12A, 8A/10A, and 6A/13A. The *Pg3*-mediated reaction may vary considerably to some races and is environmentally unstable as well (10). In the key for the standard races (1), the reactions by the *Pg3* line to race 5 ranged from 1^c to X^{++} infection types, with a mean of X^{+-}. The infection types produced by races 10, 12, and 13 were somewhat higher, with minimums of X^- and X^+ or X. These may have been classed as either virulent or avirulent. Given the historically lower reactions on the *Pg3* differential by race 5 and the preponderance of virulent type reactions by the NA representatives of races 10 and 13 (Table 1), the groupings used in this study are races 1/5, 6/13, 6A/13A, 7/12, 7A/12A, and 8/10, 8A/10A.

Virulence frequencies to *Pg* genes. Because genes *Pg1, Pg2,* and *Pg3* were originally used as differentials, virulence frequencies to these genes since 1923, to *Pg4* since 1953, and to all other genes used as differentials since 1978 were calculated directly from survey data. Estimates of the levels of virulence to genes *Pg8, Pg9, Pg13,* and *Pg15* back to 1953 were made from the frequency of identifications as NA races (Table 1). Because there was only a single occurrence of virulence in Canada to gene *Pg16* in 1984 and virulence to *Pga* is very infrequent, these genes were not included in the virulence-frequency analysis.

RESULTS

Distribution and continuity of races. The standard and C races and the numbers of isolates of these races subsequently identified as NA races are listed, along with the regions in which they predominated, in Table 1. For most isolates, the avirulence/virulence formulae of the originally identified standard or C races corresponded to those of the NA races. When identifications were ambiguous, they were not included in Table 1, except when they were repeatedly made. For example, nine isolates of race 6AFH (C20) were identified as NA25, and three of race 7 (C16) were identified as NA32 (Table 1). The ambiguous host genes in these cases were *Pg8* and *Pg2*/*Pg4* respectively. The reactions by lines with these genes normally are stable and easily recognized, and it is not known why these variant identifications occurred. There were frequent instances of variant identifications based on either gene *Pg3* or *Pg9*. The reasons for differing interpretations of reactions by the *Pg3* differential were given above. Similarly, gene *Pg9* is thermolabile (10), and reactions by lines with this gene may be highly variable and difficult to interpret. In my experience, the expression of resistance by *Pg9* also may vary considerably

TABLE 1. Equivalent standard[v] (Std) and C[w] races of *Puccinia graminis* f. sp. *avenae* stored since 1953 at the Agriculture and Agri-Food Canada Research Centre, Winnipeg, the frequencies of their identification as NA[x] races, and the regions of Canada where the races primarily were found

Std no.	C no.	Formula[y]	NA no.	Formula[y]	No. of times identified	Region[z]
1/5	1	1,2,3,4,8/9				
1		1,2,3/	1	1,2,3,4,8,9,13,16,a/15	3	P/EP
			2	1,2,3,4,8,13,16,a/9,15	5	EP
			3	1,2,3,4,8,16,a/9,13,15	15	EP
5		1,2/3X	1	1,2,3,4,8,9,13,16,a/15	1	P/EP
			3	1,2,3,4,8,16,a/9,13,15	1	EP
			7	1,2,4,8,16,a/3,9,13,15	1	EP
2	2	1,2,4,8/3,9	5	1,2,4,8,9,13,16,a/3,15	7	P
			6	1,2,4,8,13,16,a/3,9,15	7	EP
			7	1,2,4,8,16,a/3,9,13,15	21	EP
3	–	2,3,4,8/1	65	2,3,4,8,13,16,a/1,9,15	4	E/EP
4	–	3,4,8/1,2	62	3,4,8,13,16,a/1,2,9,15	5	P
4A	8	3,8/1,2,4,9	19	3,8,9,13,16,a/1,2,4,15	3	E
			20	3,8,13,16,a/1,2,4,9,15	8	E
			25	8,13,16,a/1,2,3,4,9,15	15	E
			54	3,8,16,a/1,2,4,9,13,15	1	E
6A	14	8,9/1,2,3,4	24	8,9,13,16,a/1,2,3,4,15	8	E
	9	8/1,2,3,4,9	25	8,13,16,a/1,2,3,4,9,15	119	E
	9M		26	8,16,a/1,2,3,4,9,13,15	6	E
13A		8/1,2,3X,4	19	3,8,9,13,16,a/1,2,4,15	5	E
			20	3,8,13,16,a/1,2,4,9,15	20	E
			24	8,9,13,16,a/1,2,3,4,15	3	E/EP
			25	8,13,16,a/1,2,3,4,9,15	23	E
			54	3,8,16,a/1,2,4,9,13,15	1	E
6	13	4,8/1,2,3,9	22	4,8,9,13,16,a/1,2,3,15	12	E
			58	4,8,13,15,16,a/1,2,3,9	1	U
			61	4,8,13,16,a/1,2,3,9,15	12	E
6F	5	4,9/1,2,3,8	23	4,9,13,15,16,a/1,2,3,8	57	EP
			63	4,9,13,16,a/1,2,3,8,15	5	EP
6AF	10	9/1,2,3,4,8	27	9,13,15,16,a/1,2,3,4,8	168	EP/E
			28	9,13,15,16/1,2,3,4,8,a	1	EP
			29	9,13,16,a/1,2,3,4,8,15	6	EP
			30	13,16,a/1,2,3,4,8,9,15	2	EP
6AFH	20	/1,2,3,4,8,9	25	8,13,16,a/1,2,3,4,9,15	6	E
			27	9,13,15,16,a/1,2,3,4,8	8	EP
			30	13,16,a/1,2,3,4,8,9,15	9	EP
			68	13,15,16,a/1,2,3,4,8,9	10	EP
7	16	2,4,8/1,3,9	32	1,8,16,a/2,3,4,9,13,15	3	E
			35	2,4,8,13,16,a/1,3,9,15	24	E
			45	2,4,8,13,15,16,a/1,3,9	1	E
7A/12A	3	2,8/1,3,4,9	36	2,8,13,16,a/1,3,4,9,15	56	EP
			57	2,8,9,13,16,a/1,3,4,15	3	EP
8		1,4,8/2,3	10	1,4,8,9,13,16,a/2,3,15	11	P
			33	1,4,8,13,16,a/2,3,9,15	9	E/EP
			42	1,4,8,16,a/2,3,9,13,15	3	EP
8AF	C7	1/2/3/4/8/9	13	1,13,16,a/2,3,4,8,9,15	1	E
10		1,4,8/2,3X	33	1,4,8,13,16,a/2,3,9,15	8	EP/E
			34	1,3,4,8,16,a/2,9,13,15	1	E
			42	1,4,8,16,a/2,3,9,13,15	12	EP
			64	1,3,4,8,13,16,a/2,9,15	1	E
8A/10A	6	1,8/2,3,4,9	9	1,3,8,13,16,a/2,4,9,15	3	E
			11	1,8,9,13,16,a/2,3,4,15	2	E
			12	1,8,13,16,a/2,3,4,9,15	33	E
11		1,3,4,8/2	34	1,3,4,8,16,a/2,9,13,15	4	EP/E
			64	1,3,4,8,13,16,a/2,9,15	3	E
11A	17	1,3,8/2,4,9	9	1,3,8,13,16,a/2,4,9,15	6	E
			11	1,8,9,13,16,a/2,3,4,15	1	E
			12	1,8,13,16,a/2,3,4,9,15	1	E
			31	1,3,8,16,a/2,4,9,13,15	1	E
			32	1,8,16,a/2,3,4,9,13,15	1	E
			69	1,3,8,9,13,16,a/2,4,15	1	E
–	7	1/2,3,4,8,9	13	1,13,16,a/2,3,4,8,9,15	1	E
–	21	1,8,9/2,3,4	12	1,8,13,16,a/2,3,4,9,15	2	E
–	23	2,4,9,/1,3,8	16	2,4,9,13,15,16,a/1,3,8	30	EP
			18	2,4,9,13,16,a/1,3,8,15	6	EP
–	24	1,2,8/3,4,9	4	1,2,3,8,16,a/4,9,13,15	1	EP
			8	1,2,8,16,a/3,4,9,13,15	1	EP

[v] Newton and Johnson (15).
[w] Martens and Green (11).
[x] Martens et al (14).
[y] Effective/ineffective *Pg* genes.
[z] P = Pacific region (Alberta and British Columbia); EP = eastern prairies (Manitoba and Saskatchewan); E = eastern Canada (all of Canada east of Manitoba); U = undetermined.

under similar environmental conditions with different isolates of a single phenotype, such as NA27. From this study, it is apparent that interpretations of reactions by the *Pg3* and *Pg9* differentials at different times by different workers have been inconsistent.

The occurrences of the most common races identified in each of the three nomenclature periods for the eastern and prairie regions are illustrated in Figures 1 and 2, respectively. The percent occurrences for each nomenclature period were calculated directly from survey data. When particular standard, C, or NA races were very frequently identified as common races (Table 1), they were given the same symbols in the figures to indicate their relatedness (6A/13A-C9-NA25 and C10-NA27 in Fig. 1; and 7A-C3, 6F-C5, 6AF-C10-NA27, and C23-NA16 in Fig. 2). There were insufficient collections from the Pacific region during the period of C-race nomenclature to provide useful data; thus, for this region the most commonly occurring standard and NA races are illustrated independently in Figure 3. Other races also were continuous through the nomenclature periods, but at one or more times were too infrequent to be shown graphically. The latter are indicated in Tables 1 and 2.

Long-term occurrences of races of *P. g. avenae*. Of the 31 NA races listed in Table 2, 12 have been identified from isolates collected over a period of 30 yr or longer, 12 between 20 and 29 yr, four between 10 and 19 yr, and three less than 9 yr. A number of the NA races were identified from the earlier periods of isolate storage through to recent times. The standard races, 1/5, 2, 3, and 4 were identified over a period of about 40 yr (Table 2). However, these identifications were based on only three differentials; thus, how many phenotypes would have been identified had more differentials been available is unknown. Three NA races were identified from collections of race 1 (Table 1). Of these, NA1, originally derived from race 1, was identified as recently as 1993 in a collection from British Columbia (D. E. Harder, *unpublished data*).

The historic pattern of race occurrence in Canada has been one of periods of prominence of only a few races for particular time periods (Figs. 1–3), but many of the races continue to be identified for much longer periods (Table 2). The prominence of particular races is normally attributable to the deployment of resistance genotypes in the host populations (7). Races 1/5 and 2 dominated for about 30 yr during a time when there was no resistance in the host populations and, thus, no selection for particular virulences. These races, avirulent to genes *Pg1* and *Pg2*, declined in response to the introduction of cultivars such as Vanguard, Ajax, and Exeter that had gene *Pg2* resistance and later, during the 1950s, to Bond-derived cultivars with gene *Pg1* resistance (11). Race 6 in eastern Canada and races 8/10 and 7 across Canada existed at low levels for about 30 yr, then became prominent for a short period during 1950–1960 (Figs. 1 and 2). These races, avirulent to gene *Pg4*, declined with the widespread use of the cultivar Rodney (with *Pg4* resistance) beginning in 1954. Races 6, 7, and 8/10 were commonly identified as races NA22, NA35, and NA33, respectively (Table 1). The latter races have been identified in survey collections into the 1980s; thus, races equivalent to races 6, 7, and 8/10 during their period of prominence have continued to exist at low levels in the *P. g. avenae* populations.

Race 7A, identified mainly as C3-NA36 (Table 1), virulent to gene *Pg4*, was first identified in 1955. This race was prominent in the prairie region for a short period during the late 1950s and early 1960s (Fig. 2). The widespread cultivation of Rodney in western Canada probably provided the opportunity for the selection and increase of this race and will have been responsible for the decline in virulence to gene *Pg2* (Fig. 4; [12]) during this period. The subsequent decline of race NA36 will have been due to the common use of cultivars such as Garry and Harmon, which combined *Pg2* and *Pg4* resistance. Race NA36 was identified as recently as 1987 (Table 2); thus, this phenotype has continued to persist at a low level in the prairie population.

Race 6A/13A, subsequently identified most commonly as C9-NA25 (Table 1), was first identified in 1958 and quickly rose to prominence in eastern Canada. Race C9 predominated from 1964 to 1977, and race NA25 until about 1990 (Fig. 1). Although NA25 has declined in frequency in recent years (Fig. 1), it remains a common component of the eastern population. Although genes

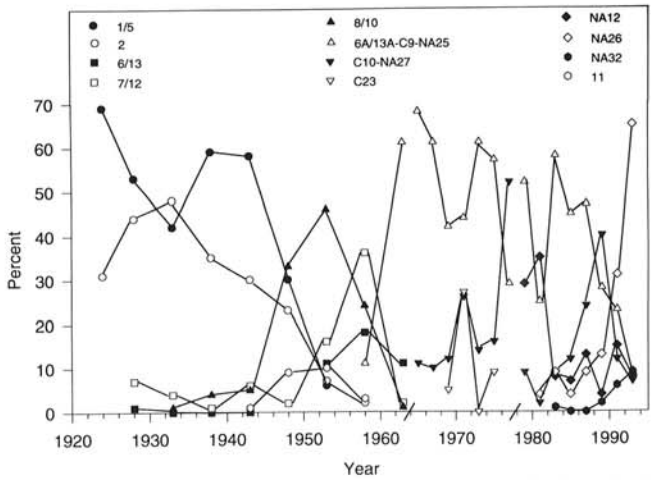

Fig. 1. Frequencies of occurrence of the standard (1925–1963), C (1964–1978), and NA (1978–1993) races of *Puccinia graminis* f. sp. *avenae* in eastern Canada. Each nomenclature period is separated by a slash on the x-axis. The most common equivalent races from the various nomenclature periods are shown by common symbols.

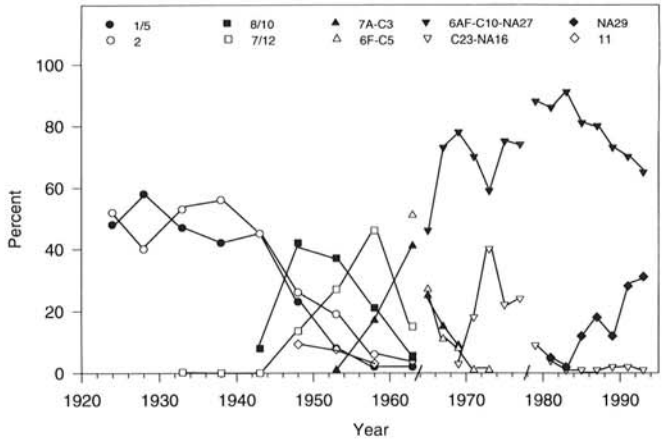

Fig. 2. Frequencies of occurrence of the standard (1924–1963), C (1964–1977), and NA (1978–1993) races of *Puccinia graminis* f. sp. *avenae* in the eastern prairie region of Canada. Each nomenclature period is separated by a slash on the x-axis. The most common equivalent races from the various nomenclature periods are shown by common symbols.

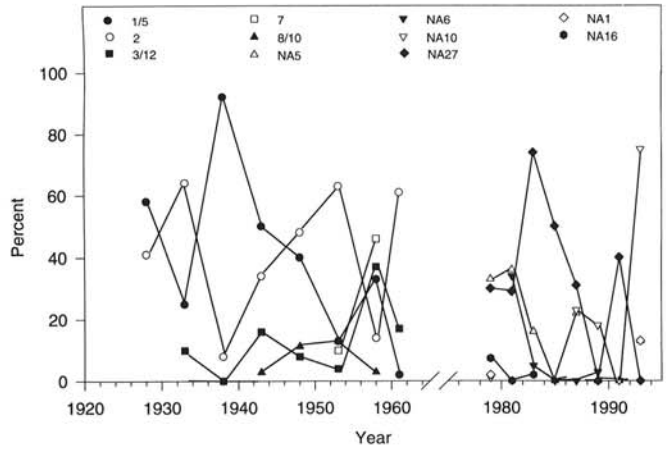

Fig. 3. Frequencies of occurrence of the standard (1928–1963) and NA (1978–1993) races of *Puccinia graminis* f. sp. *avenae* in the Pacific region of Canada. There was no data for C races for this region.

Pg1, Pg2, and *Pg4* were used in breeding programs in eastern Canada as in the rest of North America, the extent of their deployment in oat production in this region is uncertain. The occurrence of these resistances in cultivars in this region may have contributed to the predominance of 6A/13A-C9-NA25, but there has been no change in resistance-gene deployment; thus, it is unclear why NA25 has declined and *Pg13*-virulent races such as NA26 and NA32 have increased.

Race NA27 has predominated in the prairie *P. g. avenae* population (Fig. 2) since the NA nomenclature was introduced and has been common in the eastern and Pacific (Alberta) populations as well (Figs. 1 and 3). This race may not be an indigenous component of the eastern Canadian *P. g. avenae* population, because it could arrive there annually via the prevailing west to east movement of weather systems in Canada. Its irregular occurrence in Alberta also is likely due to prevailing weather systems in any given year. Race NA27 is equivalent to 6AF and C10 (Table 1) and to race 31 of the Stewart and Roberts (20) nomenclature. Race 6AF was first identified in 1963, and race C10 subsequently became predominant during 1964–1976 (Fig. 1). At the same time, there also was a rapid rise to prominence of race 31 in the United States (18). This phenotype increased in frequency very quickly, has maintained this position for 30 yr, and has shown little indication of decline. Roelfs et al (18) indicated a selective advantage for race 31 due to its combined virulence to the commonly used sources of resistance, genes *Pg1, Pg2,* and *Pg4*. In Canada this race replaced 7A-C3-NA36, which differs by avirulence to gene *Pg2*.

The interpretation of Alberta as a transitional zone between the prairie and Pacific populations (5) is further indicated by the erratic occurrence of NA27 (Fig. 3) and by the irregular occurrence of virulences to genes *Pg1, Pg2,* and *Pg8* (Fig. 5). During some years, this race has been nearly absent from Alberta and during other years has predominated. Races NA5, NA6, and NA10, the most commonly isolated races from British Columbia (Fig. 3 and survey data), also commonly occur in Alberta.

Other races have held lower but stable long-term positions within regional populations. Race NA12 (8A-C6), first identified in 1957, never became prominent but persists as a component of the eastern *P. g. avenae* population. Race C23-NA16 has occurred since about 1970 in the prairie region, reaching a peak of 40% occurrence during 1972–1973 (Fig. 2). This race is avirulent

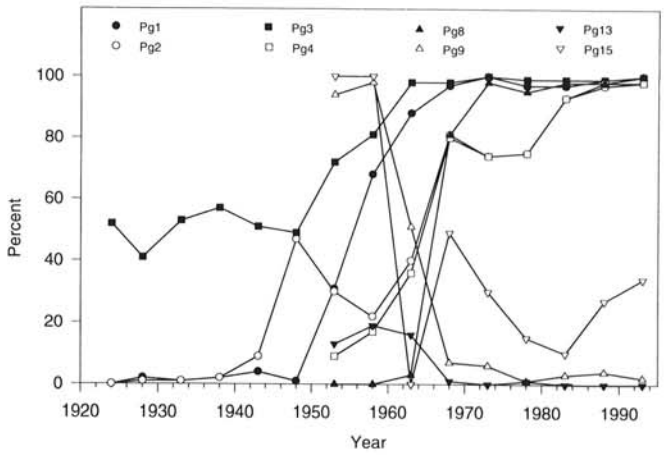

Fig. 4. Percent virulence frequencies in *Puccinia graminis* f. sp. *avenae* to resistance genes (*Pg*) in *Avena sativa* L. in the eastern prairie (Manitoba and Saskatchewan) region of Canada, 1923–1993.

TABLE 2. NA races[v] of *Puccinia graminis* f. sp. *avenae*, the C[w] and/or standard (Std) race[x] storage isolates from which they were identified, and the first and last known dates of identification

NA race	Years[y]	Total no. of years	C race	Years	Std race	Years[z]
1	1964–1993	30	1	1964–1975	1/5	1921/1923–1962
2	1964–1983	20			1/5	
3	1956–1982	27			1/5	
7	1962–1980	19	2	1964–1977	2	1921/1923–1960
6	1957–1981	25			2	
65	1957–1988	32			3	1925–1963
62	1954–1988	35			4	1925–1960
19	1962–1983	22	8	1964–1977	4A	1960–1963
19	1954–1983	30	9	1964–1977	13A	1957–1963
20	1957–1989	33	8	1964–1977	13A	
22	1955–1983	29			6	1926–1962
61	1957–1988	32			6	
24	1963–1990	28	14	1965–1975	6A	1957–1963
25	1958–1992	35	9	1965–1977	6A	
26	1974–1992	19	9/9M	1974–1976	6A	
23	1961–1988	28	5	1964–1975	6F	1961–1963
27	1963–1992	30	10	1964–1977	6AF	1963
35	1953–1988	36	15		7/12	1928–1963
36	1954–1987	34	3	1964–1971	7A	1952–1963
33	1957–1980	24	16	1970	8	1929–1963
42	1957–1963	7			8	
12	1965–1992	28	6	1964–1975	8A	1957–1963
33	1956–1980	25			8/10	1935–1963
9	1959–1990	32			11A	1957–1963
11	1963–1980	18			11A	
32	1959–1992	34			11A	
13	1975	1	7	1964–1975		
16	1969–1992	24	23	1969–1977		
18	1969–1991	23	23			
8	1971–1980	10	24	1971–1975		
55	1984	1				

[v] Martens et al (14).
[w] Martens and Green (11).
[x] Newton and Johnson (15).
[y] Year of first and last known identification, from stored isolates or contemporary surveys.
[z] The latest year (1963) in this column represents the shift from the standard- to the C-race nomenclature.

to genes *Pg2* and *Pg4*, but cultivars with these genes were grown at that time; thus, it is unclear why this race became as common as it did. Race NA16 is equivalent to race 61, which was relatively common in the United States (18) during the same period. The inoculum for this race was generated primarily from wild *Avena fatua* (18), and this may have influenced its occurrence in Canada. Race NA16 continues to be identified at low levels.

Race NA55 is an example of a race identified for a very short period (Table 2). This race was isolated several times from Ontario during 1984 but has not been found since. It is possible that larger sample sizes would reveal the occasional occurrence of this and other sporadically occurring races (not listed in Tables 1 or 2).

Virulence frequencies to *Pg* genes. The virulence frequencies, as measured from actual race occurrences, are given for each of the main regions in Figures 4–6. The data for genes *Pg1, Pg2,* and *Pg3* represent the virulence levels to these genes both historically and currently. The levels shown to gene *Pg3* in Figures 4–6 are lower for the period 1925–1963 than previously published (12,20) because race 5 was combined with race 1 in this study. Gene *Pg3* has not provided effective resistance in North America historically or currently. Genes *Pg1, Pg2,* and *Pg4* have been ineffective in the eastern and prairie regions since 1960 (Figs. 4 and 6) but remain effective to more of the Pacific population (Fig. 5).

Estimates of virulence to genes *Pg8, Pg9, Pg13,* and *Pg15* in the eastern region from 1955 to about 1975 and actual virulences from the late 1970s to the present are shown in Figure 6. There has been no virulence to gene *Pg16* in this region, except for a few isolates in 1984, and none to gene *Pga* from eastern Canada was detected. Although no representatives of races 1/5 or 2 from eastern Canada were available, the remaining common races, 6, 7, or 8/10, collected from this region (Fig. 1) were virulent to genes *Pg9* and *Pg15* (Table 1), indicating that virulence to these genes historically was high (Fig. 6). The *P. g. avenae* population in eastern Canada is generally avirulent to *Pg8*, except for that represented by race NA27 (C10). Races NA32 and NA26, virulent to gene *Pg13*, have become increasingly prevalent in recent years (Fig. 1), corresponding with increased virulence to *Pg13* (Fig. 6). No oat cultivars with gene *Pg13* resistance are known to be grown in eastern Canada.

In the prairie region of Canada, none of the isolates of *P. g. avenae* collected and stored before the early 1960s were virulent to *Pg8*, whereas all of those isolates appeared to be virulent to *Pg9* and *Pg15* (Table 1; Fig. 4). After this time, virulence to *Pg8* rose sharply and virulence to *Pg9* and *Pg15* declined, coinciding with the appearance of races 6F-C5-NA23 and 6AF-C10-NA27. During the period 1955–1959, races 1/5, 2, 7/12, 8/10/11, and 7A were the most common (3). From the proportion of those identified as NA races (Table 1), up to about 20% of isolates during this period were estimated as virulent to *Pg13*. Virulence to *Pg13* was the highest (70%; Table 1) in races 1/5 (C1) and 2 (C2). Although it was not possible to confirm the occurrence of this virulence in isolates of these races prior to the 1950s, it is possible that virulence to *Pg13* may have been higher in the prairie region during the times that these races predominated.

DISCUSSION

The regional *P. g. avenae* populations in Canada appeared to be stable with respect to virulence until about 1940, then there was a period of race changes until the mid-1960s, after which the populations again have been stable. This is particularly apparent in the prairie region with the dominance of race NA27. The widespread use of genes *Pg1, Pg2,* and *Pg4* as resistance sources in North American oat cultivars was the main cause of race shifts in *P. g. avenae* during the middle period. Other resistance genes have been used more recently. Gene *Pg9* occurs in the cultivar Hudson, which was grown on a limited scale for a short period in Manitoba during the 1970s, and in Dumont, which is currently widely grown in Manitoba. Gene *Pg13* occurs in all cultivars that have been recommended for the rust area of the Canadian prairies since 1981, and in some cultivars (e.g., Steele) in the northern plains of the United States. Gene *Pg2* also occurs in most cultivars now grown in Manitoba. Because Manitoba is at the northern end of the annual northward migration of inoculum, the use of these genes in the northern plains region should have little effect on selection of virulence phenotypes in *P. g. avenae* collected in this region.

The historic pattern of race distribution in Canada has been one of dominance by one or a few phenotypes, typically for 20–30 yr, declines in their occurrence, then continued persistence at lower levels. Other races, although never very prominent, have persisted for long periods. Although it was not possible to ascertain the identity of isolates as NA races prior to the earliest stored collections, it is likely that some (NA races) have been components of the *P. g. avenae* populations for longer periods than is indicated in Table 2. Some of the longer occurring races, such as NA35 and NA36, have continued to be identified to recent times. Thus, the data indicates high levels of stability in *P. g. avenae* in North America. This is illustrated by the persistence of avirulences to genes *Pg1* and *Pg4* (e.g., NA16 and NA36) despite selection against these avirulences, and virulence to *Pg3* despite no selection for this virulence, from at least 1921 to the present. Similarly, virulence to genes *Pg9* and *Pg15* has persisted at high levels in the eastern Canadian population for over 35 yr despite no known presence of these genes in this region.

Martens (10) indicated that changes in races in the pre-1960s period probably were due to the advantageous selection of virulence phenotypes that existed previously. The apparent stability

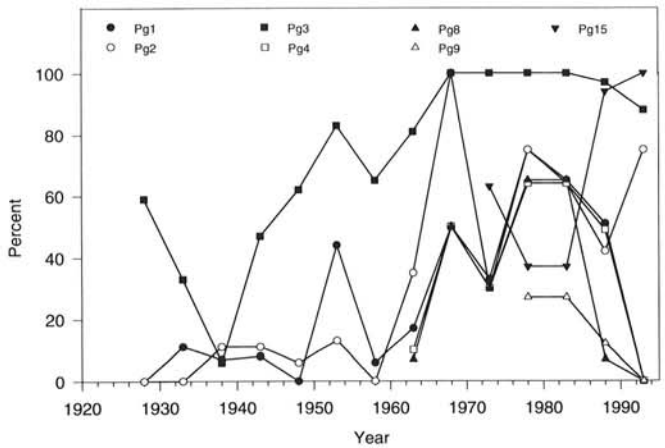

Fig. 5. Percent virulence frequencies in *Puccinia graminis* f. sp. *avenae* to resistance genes (*Pg*) in *Avena sativa* L. in the Pacific (Alberta and British Columbia) region of Canada, 1928–1993.

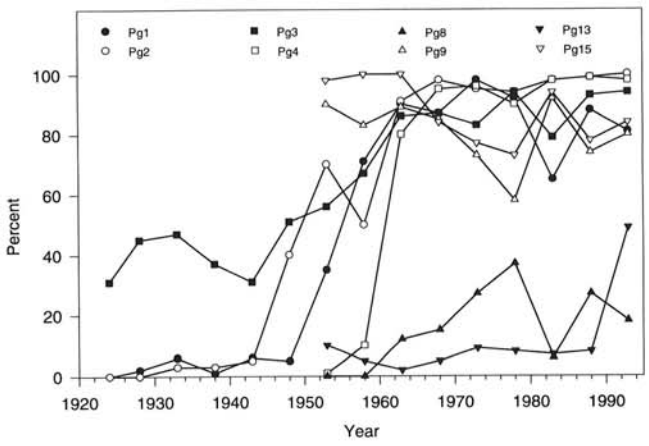

Fig. 6. Percent virulence frequencies in *Puccinia graminis* f. sp. *avenae* to resistance genes (*Pg*) in *Avena sativa* L. in eastern Canada (Maritime provinces, Quebec, and Ontario), 1924–1993.

of virulence phenotypes in *P. g. avenae* and occurrence of races over long periods as determined in the present study tend to support this conclusion. Most of the 74 NA races have likely been in existence for some time, and new virulence combinations appear to be infrequent. The relative stability of virulence phenotypes in *P. g. avenae* is further indicated by the few virulence combinations that occur in North America as compared to other cereal rusts. For example, *P. recondita* f. sp. *tritici* (8) and *P. coronata* f. sp. *avenae* (2) very rapidly develop new virulence biotypes and show high levels of diversity. It appears that the more common races of *P. g. tritici* have tended to dominate for periods of about 10–15 yr (S. L. Fox, *unpublished data*) as compared to 25 yr or longer for *P. g. avenae*. This may in part be due to a wider diversity of host resistance genes used in wheat breeding than in oat breeding in North America but also could indicate inherently greater stability. As for *P. g. avenae*, the prairie *P. g. tritici* population is asexual.

The occurrence and frequencies of virulences in *P. g. avenae* in North America do not appear to have been influenced by resistance genes other than *Pg1, Pg2,* and *Pg4*. Genes *Pga* and *Pg8* have not been used in resistance breeding in North America, and genes *Pg13, Pg15,* and *Pg16*, derived from exotic sources (10), are recent introductions. However, virulences to all of these genes have occurred at various times and levels in the North American populations. It is unclear how new virulence arose in the past, or how it may occur in the future. Martens et al (13), noting the decline in virulence to *Pg2* in the 1950s, indicated that this was the only known case of decline in virulence in the history of oat stem rust in Canada. The present study has shown that declines in virulence to *Pg9, Pg13,* and *Pg15* also occurred in the prairie region of Canada. As for *Pg2*, discussed above, these declines are explained by the association of these virulences in races (e.g., NA27) whose prevalence was affected by the commonly used resistance genes *Pg1, Pg2,* or *Pg4*.

Genes *Pg9* and *Pg13* currently provide the main effective resistance in oat cultivars released for the rust area of western Canada, and gene *Pg2* occurs in all of these cultivars as well. Virulence to genes *Pg9* and *Pg13*, however, historically was higher than at present in the prairie region, and races (NA3 and NA7) with this virulence have continued to be identified. Is this significant? With the current dominance of race NA27, virulence to genes *Pg9* and *Pg13* is very low. Race NA30, similar to NA27 but virulent to *Pg9* and *Pg15*, has been identified in surveys of the prairie population but has remained at low levels. Considering the current stability of virulence in the pathogen, the effectiveness of genes *Pg9* and *Pg13* is not immediately threatened. Also, races NA3 and NA7 are avirulent to gene *Pg2*. Given the asexual nature of the *P. g. avenae* population in the plains region of North America, crossover of virulence from one 'virulence cluster' (17) in the population to another should be rare. Thus, although gene *Pg2* is ineffective against the current prairie *P. g. avenae* population, it is important to retain this gene in breeding programs to reduce the probability of reoccurrence of those races that had associated *Pg9* and/or *Pg13* virulence. Nevertheless, new virulence combinations, although apparently rare in *P. g. avenae*, could occur, and it would be prudent to devise alternate resistance-gene combinations.

In several papers, Martens and colleagues (10,12,13) reviewed data relating to the concept of stabilizing selection and virulence in *P. g. avenae* and emphasized that this fungus, on a worldwide basis, carries virulence factors considerably in excess of those needed for survival and that they are not a factor in the ability of races to survive under natural conditions. The present study has further substantiated the observations that *P. g. avenae* may carry large numbers of virulence genes in various populations and that many of these are unrelated to the occurrence of host resistance. In an experiment with sexually derived populations to isolate the effects of avirulence/virulence factors on survival, Leonard (9) provided data that indicated that the ability of races virulent to particular resistance genotypes to survive was depressed as compared to respective avirulent races. Leonard concluded that an important barrier to the build up of "complex" races in a population was their inability to compete with "simple" races. This appears to be at variance with the current population-based data that *P. g. avenae* carries virulence factors unrelated to selection and unnecessary for survival. It is possible that although virulences may have an effect on survival, under natural conditions other more complex environmental or physiological (4) factors probably have a more significant effect. The alternate host, *Berberis vulgaris*, occurs in a small area near Sunbury, Ontario, and part of the Ontario population may be sexual. However, the occurrence and stability of virulences in this region also appear unrelated to resistance-gene deployment, and they apparently show patterns similar to the asexual prairie population. Further analysis of the relative diversity of the *P. g. avenae* populations in these regions is needed. Although the *P. g. avenae* populations in North America appear quite stable with respect to virulence phenotypes, there is more diversity for virulence resident in these populations than is apparent from annual survey data, and the appropriate virulences are available for selection should the corresponding host-resistance genotypes be widely deployed.

The theory of genetic polymorphism in parasitic systems (16) indicates that unnecessary virulence alleles will eventually decline when the corresponding host genes are no longer available. As discussed by Martens et al (13), this should have resulted in the predominance of races virulent to genes *Pg2* and *Pg4* but to no others in the 1960s. However, this did not occur. The *P. g. avenae* populations have shown much greater buffering capacity in the retention of virulences or avirulences. Some selection of virulences has occurred as a result of resistance-gene deployment, but the increase or decrease of frequencies of other unrelated virulence alleles, at least in the asexual populations, appears to be influenced more by the 'virulence clusters' in which they occur and how these are selected by resistance genotypes or possibly other factors related to survival.

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