

# Virulence dynamics, phenotypic diversity, and virulence complexity in two populations of *Puccinia triticina* in Canada from 1987 to 1997<sup>1</sup>

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**Abstract:** Populations of *Puccinia triticina* (causal agent of wheat leaf rust) in Ontario, Quebec, Manitoba, and Saskatchewan were analysed for frequency of isolates with virulence to leaf rust resistance genes *Lr1*, *Lr2a*, *Lr2c*, *Lr3*, *Lr9*, *Lr16*, *Lr24*, *Lr26*, *Lr3ka*, *Lr11*, *Lr17*, and *Lr30* for the years 1987–1997. Winter wheats in the southern plains of the United States with resistance genes *Lr24*, *Lr26*, *Lr11*, *Lr3ka*, *Lr17*, and *Lr16* selected virulent *P. triticina* phenotypes in the Manitoba and Saskatchewan population. The *P. triticina* population in Quebec and Ontario was comparatively stable, showing no directional trends in selection of virulence phenotypes. In the Manitoba and Saskatchewan population, diversity of virulence phenotypes changed relatively little despite the selection that occurred in this population. The average number of virulences per isolate in the Manitoba and Saskatchewan population increased from 1987 to 1994 during the period of selection for virulence to *Lr24*, *Lr26*, *Lr3ka*, and *Lr11*, and declined from 1995 to 1997 when selection for virulence to *Lr16* and *Lr17* occurred. The most complex virulence phenotypes were not necessarily selected in the Manitoba and Saskatchewan population because of how the resistance genes were deployed in the host population and the nonrandom distribution of virulences in the *P. triticina* population.

**Key words:** *Puccinia recondita* f.sp. *tritici*, specific resistance, wheat leaf rust, *Triticum aestivum*, specific virulence.

**Résumé :** Les auteurs ont analysé les populations du *Puccinia triticina* (agent causal de la rouille des feuilles du blé) dans les provinces de l'Ontario, du Québec, du Manitoba et de la Saskatchewan, afin de déterminer la fréquence des isolats virulents envers les gènes de résistance à la rouille des feuilles *Lr1*, *Lr2a*, *Lr2c*, *Lr3*, *Lr9*, *Lr16*, *Lr24*, *Lr26*, *Lr3ka*, *Lr11*, *Lr17* et *Lr30*, pour les années 1987–1997. Les blés d'hiver des plaines du sud des États-Unis, avec résistance aux gènes *Lr24*, *Lr26*, *Lr11*, *Lr3ka*, *Lr17* et *Lr16*, sélectionnent des phénotypes virulents du *P. triticina* dans la population du Manitoba et de la Saskatchewan. La population du *P. triticina* au Québec et en Ontario est comparativement stable, ne montrant pas de tendances directionnelles quant à la sélection de phénotypes virulents. Dans la population du Manitoba et de la Saskatchewan la diversité des phénotypes de virulence change relativement peu en dépit de la sélection qui s'effectue dans cette population. Le nombre moyen de virulences par isolat dans la population du Manitoba et de la Saskatchewan augmente de 1987 à 1994 au cours de la période de sélection pour la virulence aux gènes *Lr24*, *Lr26*, *Lr3ka* et *Lr11*, et diminue de 1995 à 1997 lorsque la sélection pour les gènes *Lr16* et *Lr17* s'effectue. Les phénotypes de virulence les plus complexes ne sont pas nécessairement sélectionnés dans la population du Manitoba et de la Saskatchewan, dû à la façon selon laquelle les gènes de résistance se déploient dans la population hôte, et la distribution non-aléatoire des virulences dans la population du *P. triticina*.

**Mots clés :** *Puccinia recondita* f.sp. *tritici*, résistance spécifique, rouille des feuilles du blé, *Triticum aestivum*, virulence spécifique.

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## Introduction

North American populations of *Puccinia triticina* Erikss. (syn. *Puccinia recondita* Roberge ex Desmz. f.sp. *tritici* E. Henn.) (Anikster et al. 1997), the causal agent of wheat leaf

rust, are highly variable, consisting of many different physiologic races or virulence phenotypes (Kolmer and Liu 1997; Long et al. 1992). Leaf rust survives on winter wheats in the southern plains of the United States, and urediniospores are carried to the northern prairies of Manitoba and Saskatchewan on the southerly winds in spring and early summer. The southern, central, and northern plains of North America can be considered as a single epidemiological unit for *P. triticina* (Leonard et al. 1992). Virulence phenotypes that occur in the southern plains can also be found in the northern plains. The *P. triticina* population in Ontario and Quebec survives on the susceptible winter wheats that are grown within the region. Virulence phenotypes of *P. triticina* collected from the susceptible winter wheats in Quebec and Ontario are different compared with the phenotypes collected

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from the resistant spring wheats in Manitoba and Saskatchewan (Kolmer 1992b). Some migration of leaf rust from adjacent regions in the United States into Ontario and Quebec most likely also occurs.

Annual surveys of physiologic specialization in *P. triticina* in Canada have been conducted at the Cereal Research Centre in Winnipeg since 1931. The continual surveys are a unique database to examine selection and evolution in a plant pathogen population over a comparatively long period of time and in a large geographic area. Johnson (1956) summarized the results of the surveys from 1931 to 1955 using the differential cultivars in the International Register of Standard Races. Kolmer (1989, 1991b) summarized the surveys from 1931 to 1987 using the differential cultivars in the Unified Numeration system with leaf rust resistance genes *Lr1*, *Lr2a*, *Lr2c*, and *Lr3*. Since 1987, 12 near-isogenic lines of 'Thatcher' wheat with single leaf rust resistance genes (i.e., *Lr1*, *Lr2a*, *Lr2c*, *Lr3*, *Lr9*, *Lr16*, *Lr24*, *Lr26*, *Lr3ka*, *Lr11*, *Lr17*, and *Lr30*) have been used as differentials in the wheat leaf rust nomenclature system (Kolmer 1988; Long and Kolmer 1989). This paper summarizes the results of the *P. triticina* virulence surveys in Canada from 1987 to 1997 using these 12 differentials. General attributes of plant pathogen populations including diversity of virulence phenotypes, virulence complexity, and non-random associations between virulences were also examined.

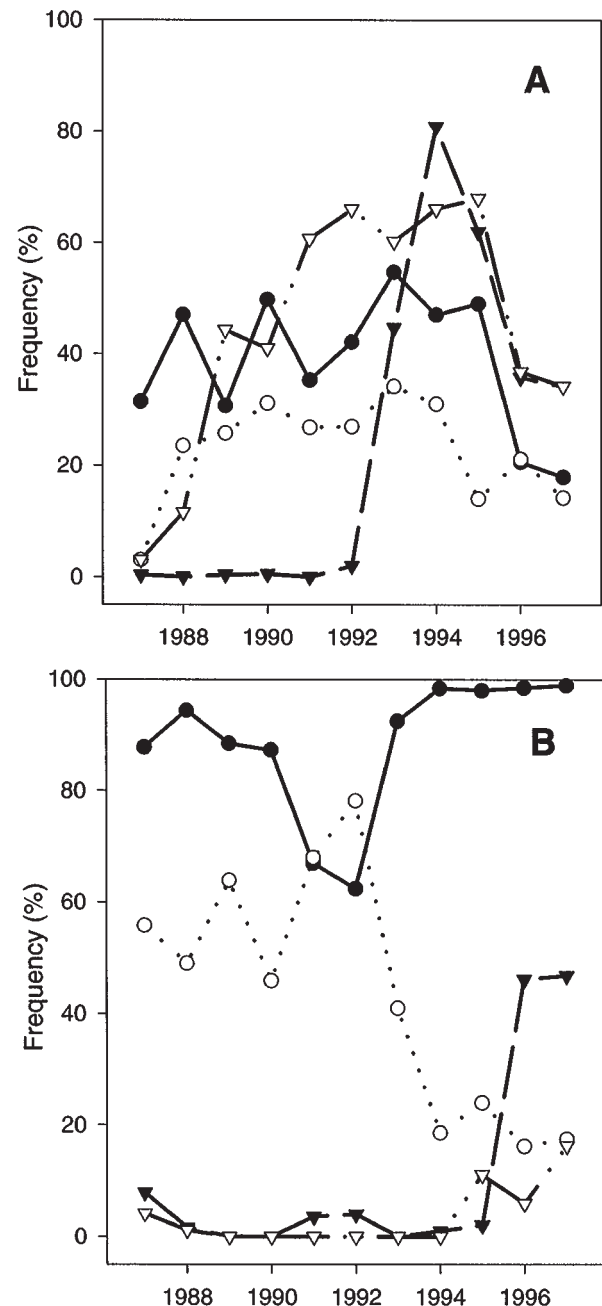
## Materials and methods

Data on virulence to specific leaf rust resistance genes and virulence phenotype frequencies from 1987 to 1996 were taken from the annual surveys of physiologic specialization of *P. triticina* in Canada, which were published in the *Canadian Journal of Plant Pathology*. Data from the 1997 survey were also included. The frequency of isolates with virulence to the 12 near-isogenic lines of 'Thatcher' wheat was determined each year in the survey. Isolates with infection types of 0 (immune response) to 2+ (medium-size uredinia with chlorosis) were rated as avirulent; isolates with infection types of 3 to 4 (medium to large uredinia without distinct chlorosis) were rated as virulent. The methods used in processing leaf rust collections for determination of virulence phenotypes were as previously described (Kolmer and Liu 1997). Approximately 200–250 *P. triticina* single-uredinial isolates from Manitoba and Saskatchewan were tested for virulence phenotype, and 25–75 isolates from Quebec and Ontario (eastern Canada population) were tested. Virulence phenotypes were determined by the infection types for each isolate on the 12 'Thatcher' differential lines. Data on the leaf rust resistance genes postulated to be in winter wheat cultivars grown in the U.S. were obtained from the USDA-ARS Cereal Disease Laboratory in St. Paul, Minn. (D.L. Long, personal communication). Diversity of virulence phenotypes in the eastern and western Canada *P. triticina* populations was determined with the Shannon index as

$$[1] \quad H_w = - \sum p_i \log_e(p_i)$$

where  $p_i$  is the frequency of the  $i$ th phenotype. The Shannon index [1] reflects the number of phenotypes and the relative evenness of their frequencies (Kolmer 1991a). Associations between virulences in the Manitoba and Saskatchewan population were measured using the  $G$ -test of independence at the 5% confidence level (Sokal and Rohlf 1981).

**Fig. 1.** (A) Frequency (%) of *Puccinia triticina* isolates in Manitoba and Saskatchewan with virulence to 'Thatcher' wheat lines near-isogenic for resistance genes *Lr24* (●), *Lr26* (○), *Lr11* (▽), and *Lr3ka* (▼) from 1987 to 1997. (B) Frequency (%) of *Puccinia triticina* isolates in Manitoba and Saskatchewan with virulence to 'Thatcher' wheat lines near-isogenic for resistance genes *Lr1* (●), *Lr2a* (○), *Lr16* (▽), and *Lr17* (▼) from 1987 to 1997.



## Results

### Manitoba and Saskatchewan

In 1987 virulence to *Lr24* was found in 31% of isolates (Fig. 1a). Virulence to this gene increased to near 60% in 1993 and then declined to less than 20% by 1997. Virulence

**Table 1.** Four selected virulences and virulence phenotypes of *Puccinia triticina* in Manitoba and Saskatchewan from 1987 to 1997.

Selected virulence	Year selected	Frequency (%)	Initial selected phenotype	Frequency (%)	Year	Frequency (%)	Number of phenotypes with virulence
<i>Lr24</i>	1987	31	<i>1, 3, 24</i>	24	1994	47	14 phenotypes
<i>Lr26</i>	1988	24	<i>1, 3, 24, 26</i>	21	1994	31	8 phenotypes
<i>Lr11</i>	1989	44	<i>1, 2a, 2c, 3, 11</i>	29	1995	68	12 phenotypes
			<i>2a, 2c, 3, 11</i>	11			
<i>Lr3ka</i>	1993	48	<i>1, 3, 3ka, 11, 30</i>	19	1995	62	12 phenotypes
			<i>1, 3, 24, 26, 3ka, 30</i>	19			

**Table 2.** Virulence associations to pairs of leaf rust resistance genes in wheat in the Manitoba and Saskatchewan population of *Puccinia triticina* from 1987 to 1997 as measured by the *G* statistic.

Virulence pair	1987	1988	1989	1990	1991	1992	1993	1994	1995	1996	1997
<i>Lr1, 2a</i>	-	-	-	-	-	-	-	ns	ns	ns	ns
<i>Lr1, 24</i>	+	+	+	+	+	+	ns	ns	+	ns	ns
<i>Lr1, 26</i>			+	+	+	+	ns	ns	-	ns	ns
<i>Lr1, 3ka</i>							+	+	+	ns	ns
<i>Lr1, 11</i>			-	-	-	-	ns	ns	ns	ns	ns
<i>Lr1, 16</i>									ns	ns	ns
<i>Lr1, 17</i>										ns	ns
<i>Lr2a, 24</i>	-	-	-	-	-	-	ns	+	+	+	ns
<i>Lr2a, 26</i>		-	-	-	-	-	-	ns	ns	ns	+
<i>Lr2a, 3ka</i>							-	-	-	-	-
<i>Lr2a, 11</i>		+	+	+	+	+	+	ns	ns	ns	-
<i>Lr2a, 16</i>									ns	ns	+
<i>Lr2a, 17</i>										-	-
<i>Lr24, 26</i>		+	+	+	+	+	+	+	+	ns	ns
<i>Lr24, 3ka</i>							ns	-	-	ns	+
<i>Lr24, 11</i>			-	-	-	-	-	-	-	ns	+
<i>Lr24, 16</i>									ns	-	-
<i>Lr24, 17</i>										ns	ns
<i>Lr26, 3ka</i>							+	ns	ns	ns	-
<i>Lr26, 11</i>		-	-	-	-	-	-	-	-	-	-
<i>Lr26, 16</i>									ns	ns	+
<i>Lr26, 17</i>										ns	ns
<i>Lr3ka, 11</i>							ns	ns	+	+	+
<i>Lr3ka, 16</i>									-	-	-
<i>Lr3ka, 17</i>										-	-
<i>Lr11, 16</i>									-	-	-
<i>Lr11, 17</i>										-	-
<i>Lr16, 17</i>										-	-

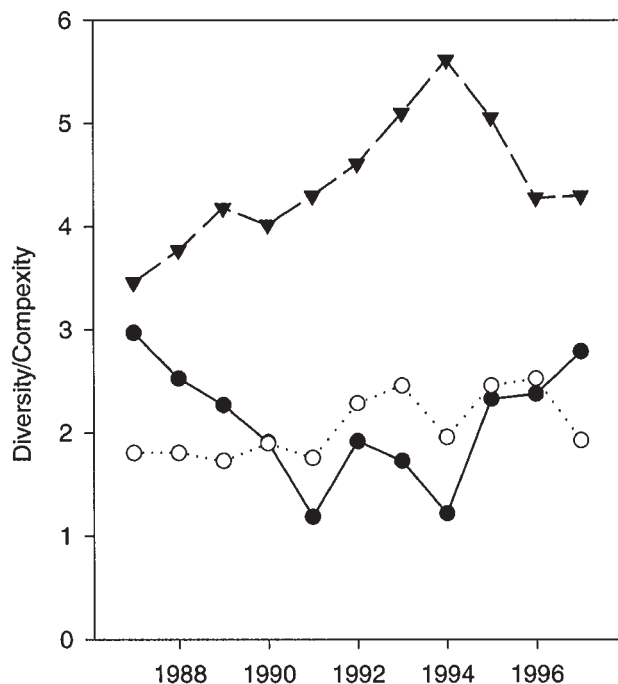
**Note:** The *G* statistic is the contingency table test (Sokal and Rohlf 1981). -, significant negative association ( $P < 0.05$ ); ns, nonsignificant association ( $P > 0.05$ ); +, significant positive association ( $P < 0.05$ ).

to *Lr26* increased from less than 5% in 1987 to over 20% in 1988 and remained between 20 and 30% through 1997. During the late 1980s and early 1990s the cultivar Siouxland, postulated to have *Lr24* and *Lr26*, was grown from Texas to South Dakota. This cultivar, or other cultivars with the same genes, most likely selected *P. triticina* phenotypes with virulences to *Lr24* and *Lr26*. Virulence to *Lr11* increased from near 0% in 1987, to 11% in 1988, and to over 40% in 1989. By 1991 virulence to *Lr11* was over 60%. In 1996–1997 virulence to *Lr11* declined to slightly less than 40%. *Lr11* was postulated to be in the cultivar Newton that was grown in Kansas in the late 1980s. Virulence to *Lr3ka* was near 0% from 1987 to 1992. In 1993 virulence to this gene increased to over 40% and reached 80% in 1994. During

1995–1997, virulence to *Lr3ka* declined to less than 40%. *Lr3ka* may be present in the Kansas wheat ‘Karl 92’ (postulated to have genes *Lr3ka* and *Lr11*) that was widely grown by 1992. Virulence to *Lr30* changed frequencies in the same manner as virulence to *Lr3ka* (data not shown in Fig. 1). Virulences to *Lr3ka* and *Lr30* are tightly linked (Kolmer 1992a). Isolates with virulence to *Lr30* are almost invariably also virulent to *Lr3ka*.

Virulence to *Lr16* was less than 10% in 1987 in Manitoba and Saskatchewan and declined to 0% by 1989 (Fig. 1b). Cultivars with *Lr16*, such as ‘Probrand 812’, were grown in Texas in the early 1980 and later declined in acreage. Virulence to *Lr16* increased in 1995 to 10% and reached 16% in 1997. The wheats ‘Tomahawk’ (*Lr16*), ‘Vista’ (*Lr16*), ‘Red-

**Fig. 2.** Shannon indexes of phenotypic diversity of *Puccinia triticina* populations in Manitoba and Saskatchewan (○) and Quebec and Ontario (●), and average number of virulences per isolate in the Manitoba and Saskatchewan population (▼) from 1987 to 1997.

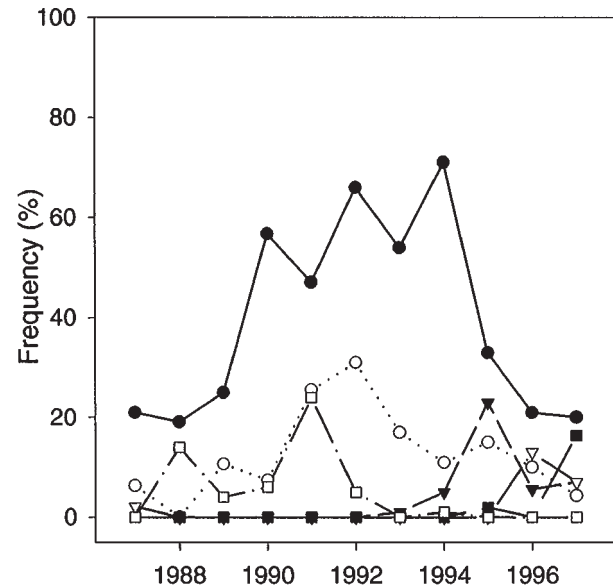


land' (*Lr16*), and 'Arapahoe' (*Lr16*, *Lr24*) were released and grown in the southern Great Plains states starting about 1994. A number of Canadian spring wheats with *Lr16* (AC 'Domain', AC 'Majestic', AC 'Barrie', AC 'Karma') were also released and grown in Manitoba and Saskatchewan in the mid to late 1990s. Virulence to *Lr17* was between 0 and 3% from 1987 to 1995 and increased to over 40% in 1996–1997. The Kansas wheat 'Jagger', released in 1996, may have *Lr17*. Virulence to *Lr1* was near 90% from 1987 to 1990, declined to 60% by 1992, and then increased to nearly 100% from 1994 to 1997. Virulence to *Lr2a* was between 40 and 60% from 1987 to 1990, increased to 80% in 1992, and then declined to 20% from 1994 to 1997. Virulence frequencies to *Lr2c* were nearly identical to *Lr2a* since the same gene in *P. triticina* conditions virulence to both resistance alleles (Samborski and Dyck 1968). From 1987 to 1997, isolates with virulence to *Lr3* were near 100%, and isolates with virulence to *Lr9* were near 0%.

When virulences to *Lr24*, *Lr26*, *Lr11*, and *L3ka* were initially selected, they were predominantly found in one or two phenotypes (Table 1). After a few years the selected virulences became more widely distributed in other phenotypes throughout the rust population. In 1987 virulence to *Lr24* was at 31%, and most of the isolates with virulence to this gene were virulence phenotype *Lr1*, *Lr3*, *Lr24*, which occurred at 24%. By 1994 virulence to *Lr24* was at 47% and was found in 14 different phenotypes. Almost all of the isolates with the selected virulences to *Lr26*, *Lr11*, and *Lr3ka* occurred in one or two different phenotypes (Table 1) the first year they increased in frequency.

The association of virulences with only one or two phenotypes during the initial years of selection can also be seen in

**Fig. 3.** Frequency (%) of the predominant *Puccinia triticina* virulence phenotypes in Quebec and Ontario from 1987 to 1997. ●, virulence phenotype *Lr1*, *Lr2c*, *Lr3*, *Lr3ka*; ○, virulence phenotype *Lr1*, *Lr3*, *Lr11*; ▼, virulence phenotype *Lr1*, *Lr3*, *Lr3ka*, *Lr11*, *Lr30*; ▽, virulence phenotype *Lr1*, *Lr3*, *Lr17*; ■, virulence phenotype *Lr1*, *Lr3*, *Lr24*, *Lr3ka*, *Lr11*, *Lr30*; □, virulence phenotype *Lr2c*, *Lr3*, *Lr3ka*.



the contingency table tests of virulence pairs in Table 2. Nonrandom associations between virulences to *Lr1*, *Lr2a*, *Lr11*, *Lr24*, and *Lr26* were stable between 1987 and 1992, the period when virulences to *Lr11*, *Lr24*, and *Lr26* were increasing. Starting in 1993, when virulence to *Lr3ka* began to increase, three of five pairs with virulence to *Lr3ka* were nonrandomly associated, while some of the associations with virulences to *Lr1*, *Lr2a*, *Lr11*, *Lr24*, and *Lr26* changed to nonsignificant. By 1996 most associations between *Lr1*, *Lr2a*, *Lr3ka*, *Lr11*, *Lr24*, and *Lr26* were nonsignificant. In 1997 virulences to *Lr16* and *Lr17* were nonrandomly associated with most of the other virulences.

The diversity of virulence phenotypes in Manitoba and Saskatchewan changed relatively little from 1987 to 1997 despite the major increases in different virulences that occurred during this time (Fig. 2). From 1987 to 1991, the Shannon index [1] values were less than 2.0 and varied between 2.0 and 2.5 from 1994 to 1997. The average number of virulences per isolate in Manitoba and Saskatchewan increased from 3.5 in 1987 to 5.5 in 1994 (Fig. 2). The average virulence complexity declined to slightly over 4.0 in 1996–1997. The Shannon indices of diversity and the average virulence complexities for each year were not significantly correlated (5% confidence level). In 1997, only 6 of 28 virulence pairs were positively associated (Table 2), which indicated that the most complex isolates were not necessarily being selected.

#### Quebec and Ontario

The *P. triticina* phenotypes in Quebec and Ontario changed relatively little from 1987 to 1997 compared with the population in Manitoba and Saskatchewan (Fig. 3). The most common phenotype in Quebec and Ontario during this



period had virulence to *Lr1*, *Lr2c*, *Lr3*, and *Lr3ka*. This phenotype was found at 20–70% of the Quebec and Ontario population but was never found in the Manitoba and Saskatchewan population. A phenotype with virulence to *Lr1*, *Lr3* and *Lr11* was detected every year at 2–30% in Quebec and Ontario and was sporadically detected in the Manitoba and Saskatchewan population at less than 3%. A phenotype virulent to *Lr2c*, *Lr3*, and *Lr3ka* was found in Quebec and Ontario between 2 and 15% from 1987 to 1992. This phenotype was never detected in the Manitoba and Saskatchewan population. Two phenotypes with virulence to *Lr1*, *Lr3*, *Lr3ka*, *Lr11*, *Lr30* and to *Lr1*, *Lr3*, *Lr17* occurred at 9–22% from 1995 to 1997. These phenotypes were between 0 and 38% in the Manitoba and Saskatchewan population during these 3 years. A phenotype with virulence to *Lr1*, *Lr3*, *Lr24*, *Lr3ka*, *Lr11*, *Lr30* was the second most common phenotype at 17% in Quebec and Ontario in 1997. This phenotype was at 10% in the Manitoba and Saskatchewan population in 1997. Diversity of *P. triticina* phenotypes declined in Quebec and Ontario from a Shannon index [1] value near 3.0 in 1987 to slightly over 1.0 in 1991 (Fig. 2). From 1992 to 1997 the index of diversity in this population varied between 1.2 to 2.3. Virulence associations and virulence complexities were not calculated for *P. triticina* isolates in Quebec and Ontario since the predominant virulence phenotypes changed relatively little.

## Discussion

From 1987 to 1997 the virulence of *P. triticina* collections from wheat in Manitoba and Saskatchewan changed on an almost annual basis because of the introduction of leaf rust resistance genes in the winter wheats grown in the Great Plains region of the U. S. This is a classic example of single resistance genes causing rapid selection of virulence phenotypes in a rust population. Kolmer (1992a) noted previously that almost all *P. triticina* isolates from Manitoba and Saskatchewan in the 1988 survey that were avirulent to *Lr3ka*, *Lr11*, and *Lr17* were most likely heterozygous for virulence alleles to these genes. In this case, only a single mutation affecting one avirulence allele would be needed for the avirulent isolates to gain virulence to one of the resistance genes. The *P. triticina* population that overwinters in the U.S. southern plains is relatively large since many winter wheat cultivars in that region lack effective resistance. Random mutation of avirulence alleles to virulence alleles would be a frequent event in this large population.

In contrast, the *P. triticina* population in Quebec and Ontario has been relatively stable, with no obvious directional shifts in virulences. The *P. triticina* collections from this region were made from breeding plots and farm fields of susceptible soft white winter wheats, as well as hard red spring wheats in rust nurseries and farm fields. The predominant virulence phenotypes that are found on a yearly basis in this region most likely overwinter on the susceptible winter wheats that unlike the winter wheats in the U.S. southern plains, have not imposed an obvious selection pressure on the *P. triticina* population. The predominant virulence phenotypes found in Quebec and Ontario also have distinct molecular backgrounds compared with isolates found in Manitoba and Saskatchewan (Kolmer et al. 1995). Certain

virulence phenotypes collected from Quebec and Ontario were also found in Manitoba and Saskatchewan in the same years. These phenotypes may have migrated to Quebec and Ontario from wheat growing areas in the southern plains or the eastern U.S states (Long et al. 1992). The roughly parallel occurrence of these phenotypes in both eastern Canada and Manitoba and Saskatchewan suggests a common overwintering source of *P. triticina*.

In plant pathogen populations that are subject to continuous directional selection for increasing virulence, it is inevitable that the most complex phenotypes will eventually predominate if no other selective forces are present. The average virulence complexity of the Manitoba and Saskatchewan population increased from 3.5 to 5.5 during 1987–1994, and then declined to slightly greater than 4.0 from 1995 to 1997. Although this rust population was under continuous selection for increasing virulence, the most complex virulence phenotypes were not necessarily selected in the last 3 years. In 1997 there were only six pairs of virulences that were positively associated. The majority of virulence pairs were randomly or negatively associated. A greater number of positive virulence associations would be expected if selection always favoured the most complex virulence phenotypes.

Other factors may also affect virulence complexity in *P. triticina* populations. The most important may be the deployment of resistance genes in the winter wheat populations coupled with the nonrandom distribution of virulence in the *P. triticina* population. The winter wheat ‘Karl 92’ (*Lr3ka*, *Lr11*) selected phenotypes with virulence to *Lr1*, *Lr3*, *Lr3ka*, *Lr11*, and *Lr30*, while ‘Jagger’ (*Lr17*) selected phenotypes with virulence to *Lr1*, *Lr3*, and *Lr17*. Since virulence to *Lr17* arose in isolates that were avirulent to *Lr3ka* and *Lr11*, the virulence complexity of the *P. triticina* population decreased when virulence to *Lr17* began to increase and virulence to *Lr3ka* and *Lr11* decreased. Cultivars with combinations of different resistance genes, i.e., ‘Siouxland’ (*Lr24*, *Lr26*), ‘Arapahoe’ (*Lr16*, *Lr24*), and ‘Redland’ (*Lr16*), would also be expected to select different phenotypes. The diversity of leaf rust resistance genotypes in winter wheat has helped to maintain a diversity of *P. triticina* phenotypes. Østergård and Hovmøller (1991) modelled associations between virulences in barley powdery mildew with various arrangements of host resistance genes. Their models predicted that hosts with two or more combinations of resistance genes would select complex pathogen phenotypes, while host populations with different resistance genes in different lines would select simpler phenotypes.

Kolmer (1993, 1995) selected a sexually derived heterogeneous population of *P. triticina* on mixtures of different near-isogenic ‘Thatcher’ lines. After 12 uredinial generations of selection, phenotypes with an intermediate number of virulences were the predominant isolates. Selection had not favoured either isolates with the fewest unneeded virulences, as would be predicted from Vanderplank’s (1968) original description of “stabilizing selection,” or phenotypes with virulence to all resistance genes. In these experiments the multilines and the susceptible host ‘Thatcher’ selected the same predominant *P. triticina* phenotypes, suggesting that the fitness differences between the isolates was the major selective force. Intrinsic fitness differences favouring iso-

lates with intermediate numbers of virulences may also affect levels of virulence complexity in field populations of *P. triticina*.

Gene *Lr16* has been present in U.S. winter wheats and Canadian spring wheats since before 1993, but virulence to this gene in the Manitoba and Saskatchewan *P. triticina* population has not increased at the same rapid rate as virulences to *Lr11*, *Lr3ka*, and *Lr17*. Kolmer (1992a) previously noted that virulence heterozygosity to *Lr16* was very low in the 1988 Manitoba and Saskatchewan population. This may partly explain why isolates with virulence to this gene have not increased at a faster rate. Virulence to *Lr16* was common in Texas and Oklahoma in the early 1980s, reaching 17% of the total U.S. *P. triticina* population in 1986 (Schafer and Long 1988) and up to 10% in the Manitoba and Saskatchewan population (Kolmer 1991b). However, virulence to *Lr16* declined in the late 1980s to very low levels in the U.S. and Canada when winter wheats with this gene were replaced. Virulence to *Lr16* in Manitoba also increased in the mid 1950s with the introduction of 'Selkirk' wheat (Kolmer 1991b). Virulence to *Lr16* declined with the replacement of 'Selkirk' in the mid 1960s with wheats that lacked *Lr16*. The apparent selection against virulence to *Lr16* in the absence of the resistance gene fits the original definition of stabilizing selection as used by Vanderplank (1968).

The diversity of the virulence phenotypes in the Manitoba and Saskatchewan *P. triticina* population remained relatively stable from 1987 to 1997 despite the constant directional selection for different virulences. If directional selection resulted in only a few complex virulence phenotypes in the population, then it would be expected that diversity would decrease. However, selection of different phenotypes with intermediate numbers of virulences resulted in a stable level of diversity in the Manitoba and Saskatchewan population. Andrivon and de Vallavieille-Pope (1993) determined that there was no correlation between virulence complexity and diversity as measured by the Shannon index in three populations of *Erysiphe graminis* on barley in France from 1986 to 1990. Andrivon and de Vallavieille-Pope (1995) also found a general lack of correlation between virulence complexity and diversity for other rust or mildew diseases.

The *P. triticina* population in Manitoba and Saskatchewan and Quebec and Ontario have had distinct virulence phenotype populations since the mid 1940s when cultivars with *Lr14a* were introduced in Manitoba and cultivars with *Lr3* were introduced in winter wheats in the U.S. southern plains (Kolmer 1991b). The two populations remain quite distinct, with few virulence phenotypes found in common. These populations will likely remain differentiated, since the respective host populations have different selective effects on the *P. triticina* population and they are epidemiologically somewhat isolated.

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