Virulence Genes and Virulence Gene Frequencies of *Blumeria graminis* f. sp. *tritici* in Ohio

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ABSTRACT

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Field isolates of *Blumeria graminis* f. sp. *tritici* were collected from 36 counties in Ohio in 1992 and 1993. Single colony progeny of the 199 isolates were tested against 11 powdery mildew resistance genes (*Pm* genes) using 8-day-old seedlings of differential host cultivars and lines. Virulence against nine of the resistance genes was found in 1992 while virulence against all 11 resistance genes was found in 1993. All isolates tested were virulent on *Pm*7 and *Pm*8 both years and a high percentage (>40%) of the isolates were virulent on *Pm*2, *Pm*3a, *Pm*3b, *Pm*3c, *Pm*4a, *Pm*5, and *Pm*6 in each year. However, a low percentage (< 5%) of the isolates were found to be virulent on *Pm*l, *Pm*3b, and *Pm*17 both years. Results also suggested that virulence matching resistance gene pairs *Pm*2 + *Pm*6 and *Pm*3c + *Pm*5 always occurred together in the pathogen population. Approximately 92% of the isolates tested gave a compatible reaction with four to eight *Pm* resistance genes. Therefore, complex races occurred frequently in the pathogen population.

Powdery mildew, caused by *Blumeria graminis* (DC.) E. O. Speer f. sp. *tritici* Ém. Marchal (4,10) (*Bgt*) is an important disease of wheat (*Triticum aestivum* L.) throughout wheat-growing areas of the world (7,14,16,37,41) and significant yield losses have been reported in various countries (12,23,46,50). In the United States, powdery mildew is considered one of the most important diseases of wheat in New York (38), South Carolina (22), Maryland (18), Pennsylvania (45), and Ohio (31). Yield losses as high as 12, 27, and 37% have been reported in the Midwest, East, and Southeast, respectively (9,24,25,31).

Powdery mildew is managed by using resistant cultivars, crop rotation, delayed planting, and occasionally fungicide applications. Of these, cultivar resistance is considered the most practical, effective, and economical means of managing powdery mildew (5,37,38). Currently, major genes for powdery mildew resistance have been described at 17 different loci in wheat (17,35,47,48,49) and most of these genes exist in wheat cultivars throughout the world (14,15,25,35). However, the deployment of single-gene resistance may be short-lived if variability of the pathogen is great.

Any powdery mildew management program that includes the use of host resistance will require information on the virulence genes that exist, and the frequency at which they exist, in the pathogen population. Five recent studies provided information on the pathogen population in the United States, and Canada (1,26,27,36,38, 44). These surveys indicated virulence to most of the widely used resistance genes. High frequencies of virulence, between 30 and 80%, were observed for resistance genes *Pm*2, *Pm*3c, *Pm*4a, *Pm*5, and *Pm*6 in the United States (38,44). In Europe, similar high virulence frequencies were observed. Frequencies of 67 to 90% on *Pm*2 and 50% on *Pm*4b in England, and 80 to 90% on *Pm*8 in Germany have been reported (30). Combined virulence to powdery mildew resistance genes also has been observed against *Pm*2 and *Pm*6 in England (2). In North America, races virulent against two resistance genes were observed in approximately 40% of isolates tested in Pennsylvania (44) and 13% in Canada (36), while races virulent against four and five resistance genes were observed in 24 and 14%, respectively, in Pennsylvania (44).

Powdery mildew is currently considered the most important disease of wheat in Ohio (31); however, no information is available on the variability of the *Bgt* population in the state. The objectives of this study were to determine the virulence spectrum of the *Bgt* population in Ohio and the frequency at which virulence to resistance genes occurred in the pathogen population.

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MATERIALS AND METHODS

Collection of mass isolates. Isolates of *Bgt* were collected from commercial wheat fields in 17 and 26 wheat-growing counties of Ohio during April to June of 1992 and 1993, respectively. Fewer counties were sampled in 1992 than in 1993 because of low disease incidence due to unfavorable environmental conditions for powdery mildew development. Thus, 35 of the 89 isolates recovered in 1992 were collected from experimental plots and surrounding fields near Wooster in Wayne County. The other 54 isolates were collected from 16 counties, with from one to 16 isolates being collected from each county. Two to three commercial fields per county were sampled in 1993 and a maximum of four isolates were taken from each field. Thus, in 1993, 110 isolates were recovered and tested.

A 5-cm section of the diseased leaf was taken from each plant sampled in the field. Leaf sections were placed in 50 × 200 mm test tubes containing 8-day-old wheat seedlings of the susceptible cultivars Becker or Chancellor. Seeds of these cultivars were planted in tubes filled to approximately 1/3 their volume with Baccto plant mix (Michigan Peat Co., Houston, Tex.). Seeds were watered at the time of planting, then tubes were closed with sterile foam plugs (Jace Industries, Inc., North Tomawanda, N.Y.) and placed in the greenhouse under natural light. Tubes with diseased leaf sections were then placed in a growth chamber and maintained at 16°C, a relative humidity of at least 85%, and a photoperiod of 12 h with a light intensity of 180 $\mu E\ m^{-2}\ s^{-1}$ at the top of the tubes. Mildew colonies sporulated profusely within 8 to 10 days. These conditions were used for all the following experiments. Mass isolates were made by transferring conidia from each field isolate into a tube containing a seedling of either Becker or Chancellor. These seedlings were then placed in the growth chamber and when the plants developed sporulating colonies, single colonies were transferred from each mass isolate.

Purification of single-colony isolates. Single-colony isolates were made from each mass isolate by transferring a few conidia from a single pustule to a noninfected seedling in a tube using a sterile dissecting needle. This process was repeated three to four times, successively, in order to reduce the chance of isolate mix-

tures. All isolates were maintained throughout the course of this study on Becker or Chancellor seedlings in test tubes at 4°C. Isolates were transferred to new plants in tubes every 18 days using sterile, camel-hair brushes. Each time transfers were made the same procedure was repeated on control plants; however, the needle or brush used in the inoculation was placed on a disease-free plant instead of an infected one. Controls were used to ensure contamination of isolates did not occur.

Inoculum production. Approximately 15 Becker or Chancellor seedlings were grown in 10-cm-diameter plastic pots containing Baccto potting mix. Pots were placed in enclosed glass cases except that one side was partially open to permit access to pots. The partial opening was covered with two layers of cheesecloth held tightly to the case by Velcro strips. Plants were inoculated 7 to 8 days after planting when seedlings had 1 to 2 leaves. Just before inoculating, each pot was watered with 30 ml of 50 ppm chloromequat chloride (Cycocel, American Cyanamide Company, Wayne, N.J.) to retard plant growth. Pots were then fitted with 19-cm-tall clear plastic cylinders to prevent contamination. Cylinders were covered at the top with a 0.5-cm-thick piece of cotton held in place by a rubber band. Cylinders were held in place by wrapping the cylinder bottom to the top of the pot with Parafilm. The room was disinfested with ultraviolet light for 2 h prior to performing inoculations. Conidia of each isolate were transferred by tapping the tube containing the isolate culture over the seedlings in the pot, or by using a sterile, camel-hair brush in a clean airflow hood. Inoculated plants were then placed in the growth chamber. Inoculum was ready for use in 8 to 10 days. Control plants were used as previously described to ensure that contamination of isolates did not occur.

Identification of virulence genes. Three seeds of each of eleven differential isogeneic lines and Chancellor (Table 1) were planted in radial rows in a 21-cm-diameter plastic pot half filled with Baccto planting mix. After planting, each pot was covered with a 60 × 26 cm plastic bag to prevent contamination of the seedlings. Plastic bags were supported by a thin stake placed in the center of the pot. A rolled piece of cotton was placed at the top of the stake and the plastic bag was bound with a rubber band around the cotton at the top of the stake to allow aeration.

Eight-day-old seedlings (one- to two-leaf stage) of the differentials and Chancellor were inoculated with conidia from a single isolate. Differentials were inoculated by shaking the pot containing infected plants over the pot as described above. After inoculation, pots were placed in growth chambers. The set of differentials in a single pot was inoculated with each isolate, and the procedure was repeated at least three times. Inoculum was produced on new seedlings for each repetition of the test. Control pots containing all the differentials and Chancellor were included in each repetition of the test. The same inoculation procedure was performed on the control plants, except noninfected plants were shaken over this pot.

Table 1. Accession number and *Pm* gene symbols for differential cultivars and lines used in the assessment of *Blumeria graminis* f. sp. *tritici* virulence

Cultivar or line	Accession No.	*Pm* genes
Chancellor	CI 12333[a]	. . .[b]
Axminster /8* cc[c]	CI 14114	*Pm*1
Ulka /8* cc	CI 14118	*Pm*2
	CI 14119	*Pm*2(+)[d]
Tyler	CI 17899	*Pm*3a
Chulu /8* cc	CI 14121	*Pm*3b
Sonora /8* cc	CI 14122	*Pm*3c
Khapli /8* cc	CI 14123	*Pm*4a
Hope /8* cc	CI 14125	*Pm*5[e]
	PI 405718[f]	*Pm*6+2
Tranzec	CI 14189	*Pm*7
Kavkaz	PI 361879	*Pm*8
Amigo	CI 17609	*Pm*17

[a] Indicates cereal investigation number.
[b] No known gene for powdery mildew resistance.
[c] 8* cc indicates that the line to the left of the slash was crossed to Chancellor and seven subsequent backcrosses to Chancellor were then made (6,21).
[d] This gene reported to be same as *Pm*2 (6).
[e] Only recessive powdery mildew resistance gene known (2).
[f] Indicates plant investigation number.

Table 2. Frequencies (%) of *Blumeria graminis* f. sp. *tritici* races collected in Ohio in 1992 and 1993[a]

	Frequency (%)	
Race[b]	Observed	Expected[c]
1992		
7,8	3.37	0.0957
3a,7,8	2.25	0.0855
2,6,7,8	5.62	0.5615
3c,5,7,8	5.62	1.4897
3b,4a,7,8	1.12	0.0035
3a,4a,7,8	1.12	0.5286
2,3b,6,7,8	1.12	0.0334
2,4a,6,7,8	2.25	0.3471
3c,4a,5,7,8	5.62	0.9209
3a,3c,5,7,8	4.50	1.3312
2,3a,4a,6,7,8	1.12	0.0185
2,3c,5,6,7,8	18.00	8.7415
3b,3c,4a,5,7,8	1.12	0.0548
3a,3c,4a,5,7,8	4.50	0.8229
2,3a,3c,5,6,7,8	21.35	7.8112
2,3c,4a,5,6,7,8	6.74	6.3231
2,3b,3c,4a,5,6,7,8	2.25	0.3246
2,3a,3c,4a,5,6,7,8	12.36	4.8283
1993		
7,8	8.18	0.3041
4a,7,8	1.81	0.3960
3a,7,8	0.91	0.0941
2,6,7,8	7.27	0.4071
3c,5,7,8	10.00	4.9654
2,4a,6,7,8	1.81	0.5077
3c,4a,5,7,8	13.64	6.0681
3a,3c,5,7,8	3.64	1.5063
2,3c,5,6,7,8	10.00	6.5134
3b,3c,4a,5,7,8	1.81	0.1703
3a,3c,4a,5,7,8	5.45	1.8754
1,2,3c,5,6,7,8	0.91	0.0592
2,3a,3c,5,6,7,8	3.64	2.0165
2,3c,4a,5,6,7,8	19.10	8.1235
3a,3b,3c,4a,5,7,8	0.91	0.0527
2,3a,3c,4a,5,6,7,8	9.10	2.5149
2,3c,4a,5,6,7,8,17	1.81	8.1235

[a] Sample size = 89 for 1992 and 110 for 1993.
[b] Races described in terms of ineffective host *Pm* genes inwhich isolates were tested against *Pm*1, 2, 3a, 3b, 3c, 4a, 5, 6, 7, 8, and 17.
[c] Calculated as the product of the frequencies of virulence genes in a given race and the complement of the frequencies of the gene not in the race.

Eight days after inoculation, seedlings were rated for their reactions to each isolate on an individual plant basis. A 0 to 9 rating scale was used (25,37,38): 0 = no visible sign of infection; 1 to 3 = highly resistant, increasing from 1) a few flecks with no necrosis to 2) more flecks and tiny necrotic spots visible, to 3) larger necrotic spots to some chlorosis; 4 to 6 = intermediate, increasing from larger chlorotic areas with moderate amount of mycelium to some sporulation evident; 7 to 9 = susceptible, increasing in amount, size, and density of mycelium and conidial production representing a fully compatible reaction. A score of 0 to 6 indicated a resistant reaction of the host to the particular isolate, while a score of 7 to 9 indicated a susceptible reaction. An isolate that gave a rating of 7 to 9 on a particular differential line or cultivar was assessed as having the virulence gene that gave a compatible reaction with a corresponding resistance gene in the host. The virulence test was repeated at least three times and the average rating used as a basis for interpreting the results.

The expected frequency of each race was calculated as the product of the virulence gene frequencies for the genes identified in the race times the complement of the virulence gene frequencies that were not in the race (44). Expected frequencies were calculated based on the assumption that the virulence genes occurred independently and that there was no differential selection for genes or gene combinations (38,44). The data obtained on virulence to *Pm*2 and *Pm*2+ was used, in some cases, to highlight the observed consistent association of virulence to these genes. All other analyses conducted for this study treated virulence to *Pm*2 and *Pm*2+ as being the same.

RESULTS

The majority of reactions classified as resistant were within reaction types 1 to 3 whereas susceptible reactions were within reaction types 7 to 9. Only a small percentage of the reaction types were classified as intermediate, being types 4 to 6. Differentials with *Pm*1, *Pm*3b, and *Pm*17 consistently had highly resistant reaction types (0 or 1). Reactions by differentials with *Pm*2, *Pm*2+, *Pm*6, *Pm*3c, and *Pm*5 were type 3 in almost all cases; those for *Pm*3a and *Pm*4a represented reactions from 0 to 3. Highly consistent results were obtained for nearly all the isolates; however, in a few cases in which the results were not consistent the test was repeated an additional two to three times. Reactions of Chancellor plants indicated that the inoculation technique provided a uniform distribution of inoculum. Noninoculated control plants used to monitor the entire procedure did not become infected, indicating that there was no contamination.

The isolates tested both years represented 22 different races (Table 2). Of these, thirteen races were detected both years, five detected in 1992 only and four detected in 1993 only. Races that occurred both years differed in their frequency of occurrence from one year to the next by a range of approximately 0.4 to 18%. Those races detected in 1992 but not in 1993 and vice versa were observed at very low frequencies. In almost all cases, the observed frequencies of races were greater than the calculated expected frequencies both years.

A high percentage of isolates were virulent on differentials carrying *Pm*2, *Pm*2+, *Pm*3c, *Pm*5, *Pm*6, and *Pm*7 in both years (Fig. 1). All isolates were virulent on *Pm*7 and *Pm*8. Virulence to *Pm*3c and *Pm*5 occurred together and approximately 80% of all isolates were virulent to these two genes both years. Virulence to *Pm*2, *Pm*2+, and *Pm*6 also occurred in association and the frequency of virulence to these genes was high both years. Fifty percent fewer isolates were virulent on *Pm*3a in 1993 than in 1992 and 30% more isolates were virulent on *Pm*4a in 1993 than in 1992. Very low frequencies of virulence were detected for *Pm*1 and *Pm*17 among isolates tested in 1993 and none were virulent on differentials with these two genes in 1992. The observed virulence to *Pm*3b was low both years.

None of the isolates tested had less than two genes for virulence (Fig. 2). Actually, 91.5% of all the isolates tested had four or more genes for virulence. Isolates with seven genes for virulence occurred at the highest frequency both years.

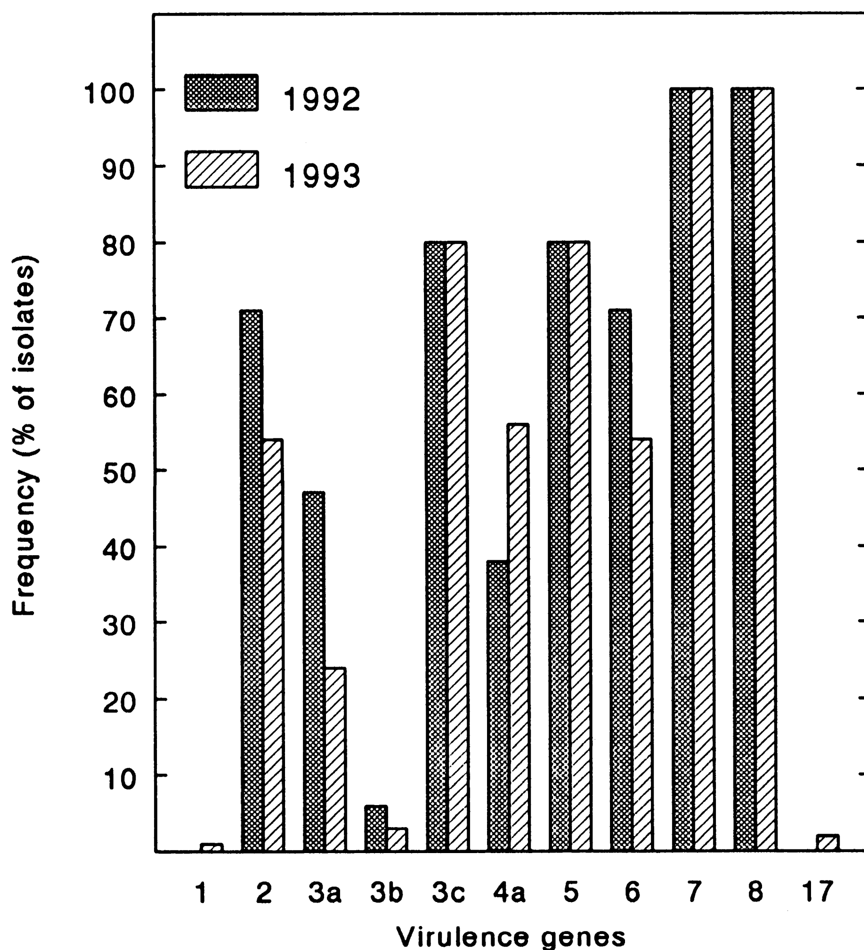

Fig. 1. Frequency of virulence genes capable of overcoming wheat powdery mildew resistance genes (*Pm*) in 89 and 110 *Blumeria graminis* f. sp. *tritici* isolates collected in Ohio in 1992 and 1993, respectively.

DISCUSSION

In this study, virulence was detected on lines carrying any of the major powdery mildew resistance genes tested. Variation in the observed virulence gene frequencies between isolates tested in 1992 and those tested in 1993 was probably due to differences in sampling locations between these 2 years rather than to shifts in the pathogen population. Most of the isolates tested in 1992 were collected from counties in central and north central Ohio. However, most of the isolates tested in 1993 were collected from counties in northwestern and southwestern Ohio. Only five counties were sampled in 1992 and 1993. Small shifts in the pathogen population have been very difficult to detect (43,44) and a large difference in the pathogen population was not expected within one growing sea-

son, since there was no known significant change in the host genotypes deployed in the field (54).

The calculated expected frequencies for virulence gene combinations were greater than the observed frequencies in almost all cases, indicating that either virulence genes do not occur independently or there is differential selection for genes or combinations of genes (38,44). For organisms that reproduce sexually, there is no simple relationship between individual gene frequencies and those of combined virulence gene frequency (43). There is evidence that virulence genes are not independent (43,53) and that linkages may occur (21, 29,43,52,53). Selection forces also may be continuously acting on virulence genes in the pathogen population (54). Vanderplank's (51) concept of stabilizing selection indicates that all virulence genes in the pathogen are necessary for the pathogen survival and when not necessary the virulence genes are lost. This apparently does not apply to the wheat-*Bgt* system. However, factors other than genes for virulence may be responsible for competition and ability of a race to survive in nature (32,52). Thus, it is possible that once a race of a pathogen has acquired the necessary virulence genes to overcome the resistance genes present in the host population, any other "unnecessary" genes for virulence will not affect its fitness characteristics or its frequency in the pathogen population (11,19,39,43) Sexual recombination appears to be a major contributor to genetic variability in *Bgt* and most likely is responsible for the diversity and predominance of complex races detected in this study.

The observed uniform association of virulence matching *Pm*2 and *Pm*2+ was expected because the line (CI 14119) used to test for *Pm*2+ did not carry this gene but instead carried *Pm*2. Royer et al. (44) also found near absolute association for virulence to the isoline carrying *Pm*2 and CI 14119 in the pathogen population. The parent, CI 12632, used to develop CI 14119 was reported to have *Pm*2+ (20). However, Briggle (6) reported that CI 14119 has only the *Pm*2 gene and that the other gene reported in CI 12632 was not transferred to this isoline. The results obtained from our study agreed with that reported by Briggle (6).

It is generally accepted that the widespread planting of cultivars with certain resistance genes has aided in the proportional increase of pathogen races capable of overcoming those genes (19,30). Our results indicate that host-driven selection pressure may explain the frequency of isolates capable of overcoming certain *Pm* genes, but not others (54). Very few isolates were virulent to *Pm*1, *Pm*3b, and *Pm*17. Selection pressure necessary to increase the population of isolates virulent on these three resistance genes has been absent because these resistance genes have not been deployed in host genotypes in Ohio. The cultivar Amigo, containing gene *Pm*17 (17), was reported to give good resistance to races of powdery mildew in Maryland (33) and New York (38). Virulence to *Pm*1 and *Pm*3b was rare in studies in New York (38), North Carolina (26,27) Pennsylvania (44), and Ontario, Canada (1,36).

There is no report of *Pm*3c, *Pm*4a, or *Pm*7 ever being deployed in Ohio or to any great extent in the United States, yet a high percentage of isolates tested in our study were virulent to these resistance genes. Relatively high frequency of virulence to *Pm*3c and *Pm*4a has been reported in Pennsylvania (44), New York (38), North Carolina (26,27), and Ontario (36). Virulence to *Pm*7 has been reported at a low level in North Carolina (26,27), but it appears to be common in Germany (13). It is known that both *Pm*7 and *Pm*3c are not fully expressed at an early seedling stages, but are expressed at later growth stages (2, 6,8,16). Thus, the frequency of virulence to *Pm*7 and *Pm*3c may be due to the restricted expression of these genes at the seedling stage. *Pm*4a is expressed in seedlings and virulence was readily detected in our tests. It appears that there is no evidence for host-driven selection for virulence against *Pm*3c, *Pm*4a, or *Pm*7 in the *Bgt* population.

Few cultivars with *Pm*3a or *Pm*8 have been deployed in Ohio. Yet frequencies of virulence to *Pm*3a were relatively high and all of the isolates were virulent to *Pm*8. The cultivar Tyler, with the gene *Pm*3a (35), was grown to a limited extent in Ohio in the mid 1980s and still occupies some area in northeast Ohio and northwest Pennsylvania. The cultivar Kavkaz, known to have *Pm*8 (2), has been used in public and private wheat-breeding programs and is present in the pedigree of two commercial cultivars grown to a limited extent in Ohio (3). However, this cannot explain the level of observed virulence to *Pm*3a or *Pm*8 in the Ohio *Bgt* population. High frequencies of virulence to *Pm*3a were observed in North Carolina (26,27) and low frequencies were reported in New York (38), Pennsylvania (44), and Ontario (1, 36). Nearly 20% of the isolates tested in New York were virulent on *Pm*8 (38), but this gene was found to be highly effective against the pathogen population in North Carolina (26,27). In Europe, cultivars carrying *Pm*8 have been known to become

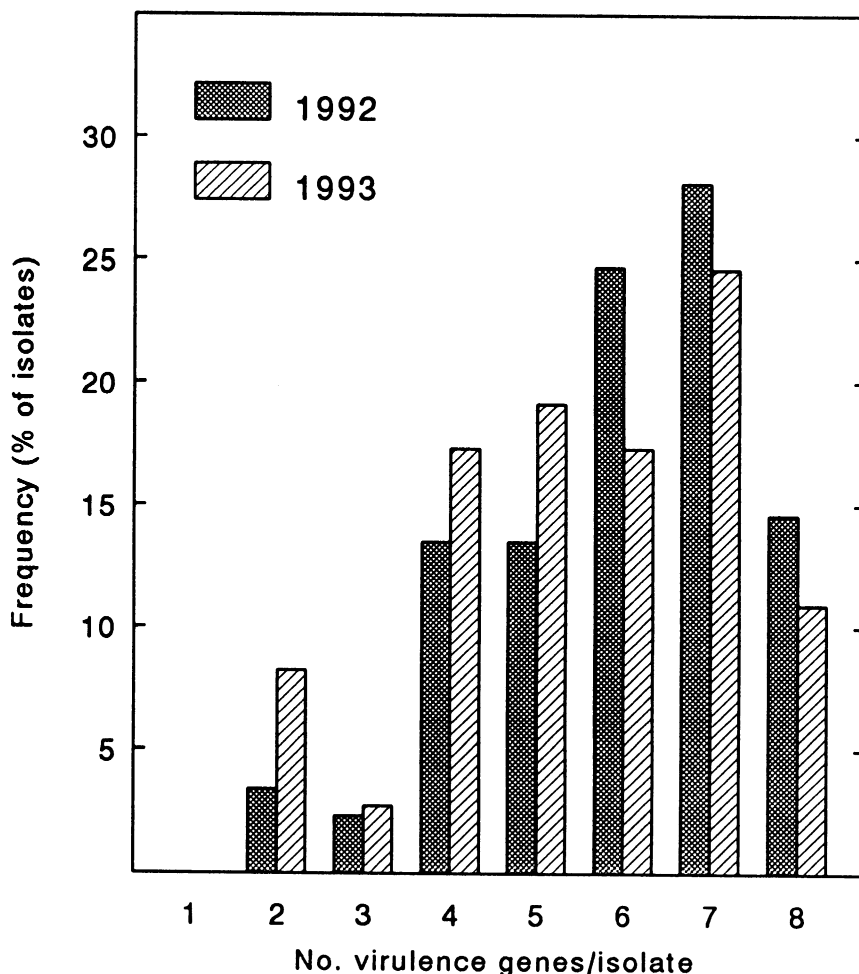

Fig. 2. Frequency of *Blumeria graminis* f. sp. *tritici* isolates capable of overcoming from one to eight wheat powdery mildew resistance genes (*Pm*); 89 and 110 isolates collected in Ohio in 1992 and 1993, respectively.

completely susceptible to mildew despite being grown only on a small scale (2). High frequencies of virulence to *Pm*8 (80 to 90%), also have been reported in Germany and some other European countries (13,30). Selection for virulence in a small, but resistant, host population and wind dispersal of conidia are reasons usually cited as possible causes for high frequency of virulence in pathogen populations (13, 19,30,38).

Virulence to *Pm*2, *Pm*5, and *Pm*6 appears to occur frequently in North America (26,27,28,36,38,44). Some genes for rust (*Puccinia graminis* f. sp. *tritici* and *Puccinia recondita* f. sp. *tritici*) resistance are found on the same chromosomes as powdery mildew resistance genes (34), or are closely linked to powdery mildew resistance genes (21). The cultivar Hope, with gene *Pm*5, has been used extensively in breeding programs in the United States and throughout the world as a source of rust resistance (2). Gene *Pm*5, the only recessive powdery mildew resistance gene (2), has been reported to be present in the cultivars Caldwell and Arthur (25). The cultivars Arthur (2) and Abe are reported to carry *Pm*2 and *Pm*6 (2), and probably obtained these genes from CI 12633 present in their pedigrees (21,40,42). Arthur and Abe were grown extensively in Ohio in the 1960s and 1970s and Caldwell was planted to approximately 30% of the wheat acreage in the mid 1980s. High frequencies of virulence to *Pm*2 and *Pm*6 (reported as *Pm* MA) also have been reported in New York (38), Pennsylvania (44), and Europe (55), and to *Pm*6 in Ontario (36). Thus, the presence of these resistance genes in commercial cultivars grown in Ohio over a long period may have created a positive selection pressure for virulence to *Pm*2, *Pm*5, and *Pm*6 in the pathogen population.

Our study indicated a uniform nonindependent occurrence of virulence to *Pm*3c + *Pm*5 and *Pm*2 + *Pm*6 in the *Bgt* population in Ohio. There are no reports of the nonindependent association of virulence to *Pm*3c + *Pm*5, but a high frequency of virulence matching *Pm*2 + *Pm*6 has been reported in Europe (55). It is possible that these genes may be linked or may have formed an adaptive complex (38) relative to the host or other selective forces that act on the pathogen population in Ohio.

Contrary to Vanderplank's concept of stabilizing selection (51), the *Bgt* population in Ohio is mainly composed of complex races capable of having compatible reactions with all major resistance genes tested and the majority of races carry from four to eight virulence genes. The pathogen virulence spectrum indicates that success in breeding for disease resistance using only major resistance genes or pyramiding resistance genes will be ephemeral at best. Quantitative resistance sources exhibiting high degrees of field resistance need to be incorporated into breeding programs to obtain durable *Bgt* control. *Bgt* survives between wheat crops as cleistothecia (19). Apparently, sexual recombination is responsible for creating, resorting, and maintaining relatively high numbers of virulence genes in the pathogen population over an extended period of time (19). Our study indicates that resistance genes present in the host population have little influence on virulence genes or the number of virulence genes maintained by the *Bgt* population.

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