Introgression of Powdery Mildew Resistance from Rye into Wheat

M. Heun and B. Friebe

Institute of Agronomy and Plant Breeding, 0-8050 Freising-Weihenstephan, Federal Republic of Germany.
We would like to thank the Deutsche Forschungsgemeinschaft (DFG) for financial support.
Accepted for publication 30 August 1989 (submitted for electronic processing).

ABSTRACT

Heun, M., and Friebe, B. 1990. Introgression of powdery mildew resistance from rye into wheat. Phytopathology 80:242-245.

A recently developed set of isogenic wheat-rye addition, substitution, and translocation lines has been analyzed for its potential to offer new sources of powdery mildew resistance originating from rye. These lines were compared with a set of cultivars/lines carrying the resistance genes *Pm*1 to 9 and *Mlk*. 4× and 6× Thatcher wheat reacted highly susceptible to wheat powdery mildew, whereas the triticale line derived from 4× Thatcher and Prolific rye showed a high level of resistance. The rye chromosomes 1R, 4R, and 7R do not condition any powdery mildew resistance against the isolates used here. The powdery mildew resistance of the 2D/2R substitution line was highly effective, whereas *Pm*7, assumed to originate from 2R, gave mainly susceptible reactions. Cytological analyses, done by using the Giemsa C-banding technique for chromosome identification, of Transec wheat carrying *Pm*7 imply that the "Transec" translocation consists of a complete short arm of chromosome 4B of wheat and about half of the proximal region of the long arm of that chromosome and a rye segment derived from the distal part of the long arm of chromosome 5R of rye. The chromosome 2R of the 2D/2R substitution line showed the typical C-banding pattern after differentially staining the constitutive heterochromatin. The rye chromosome 6R conditioned absolute resistance to all powdery mildew isolates tested and is shown to be located at the long arm of that chromosome. For this resistance, we propose the temporary gene symbol *Ml*P6L. In addition, we present a way of transferring this resistance gene using a 6BS/6RL wheat-rye translocation line.

Additional keywords: gene identification.

Powdery mildew of wheat (*Triticum aestivum* L.) caused by *Erysiphe graminis* DC. ex Merat f. sp. *tritici* Em. Marchal is one of the most economically important wheat diseases in Europe and other parts of the world and has been studied intensively by scientists over the past 40 years. Several powdery mildew resistance genes have been identified; most of them have been incorporated into high-yielding cultivars. However, the plant breeders' successes are often of limited durability because powdery mildew has the ability to overcome new resistance genes easily. For example, some of the 59 winter wheat cultivars currently registered in West Germany carry the powdery mildew resistance genes *Pm*2, *Pm*4b, *Pm*5, *Pm*6, and *Pm*8 (1,19), but only *Pm*2 and *Pm*6 combinations are still effective throughout the country (18). Even virulences against powdery mildew resistance genes that have never been used, for example, *Pm*1 or *Pm*4a, occur. In the United Kingdom, the *Pm*2 and *Pm*6 combination has been widely overcome (29). Therefore, it is only a matter of time and intensity of use of cultivars resistant to *Pm*2 and *Pm*6 before the corresponding virulence combination also becomes common in Germany. New sources of powdery mildew resistance are essential if new cycles for the development of resistant cultivars are to be initiated.

Wild species of wheat, such as *Triticum dicoccoides* (Koern. ex Asch. & Graebner) Aarons., were used as new sources of resistance (17); rye was another source. Part of the rye powdery mildew resistance has been used extensively because the resistance gene *Pm*8 is assumed to be located on the short arm of rye chromosome 1R and has been incorporated into widely grown East and West German wheat cultivars. Consequently, virulence for *Pm*8 has become very common in the German pathogen population (18); the same is true in Switzerland (27) and New York (23) but not in the United Kingdom (29) or North Carolina (21). Because earlier results imply that rye may offer some additional powdery mildew resistances (22,24), a set of isogenic wheat-rye addition, substitution, and translocation lines recently developed by Friebe and Larter (14) was analyzed to determine if it contains new genes for powdery mildew resistance.

MATERIALS AND METHODS

The wheat cultivars/near-isogenic lines containing previously identified genes for powdery mildew resistance (3,9,20) were provided by J. G. Moseman, Beltsville, MD, or by German plant breeders (provided via the Bundessortenamt, Hannover, FRG), or were taken from our institute's collection. The addition, substitution, and translocation lines developed by Friebe and Larter (14) and their parents also were used. The seven single-pustule-derived powdery mildew isolates used to identify powdery mildew resistance genes were selected by Heun and Fischbeck (20); their virulence pattern is shown in Table 1. The inoculation experiments and the disease assessments were carried out according to the methods of Heun and Fischbeck (19,20). Within each inoculation experiment with one specific powdery mildew isolate, three 3-cm-long leaf segments were inoculated; six inoculation experiments were performed with each of the seven powdery mildew isolates used. Based upon the assessed infection grade (0–9, where 0 = no visible disease symptoms and 9 =

50–100% leaf area covered with mycelium), infection type (0–4, where 0 = no pustules visible and 4 = large pustules without hypersensitive host reaction), and pustule size (ranging from very small to normal size pustules) of all six replications, three major classes of host reaction were formed: r = resistant, i = intermediate, and s = susceptible reaction. However, in some cases, this classification was unsatisfactory in explaining the observed variation, and therefore a combined classification was introduced. For example, r,i indicates that, although mainly resistant reactions occurred, intermediate reactions also were observed. Chromosome identification was carried out according to the Giemsa C-banding technique described by Giraldez et al (16).

RESULTS AND DISCUSSION

Wheat powdery mildew isolates. Seven well-defined wheat powdery mildew isolates were chosen to characterize the powdery mildew resistance of the isogenic Thatcher addition, substitution, and translocation lines. The characterization of these powdery mildew isolates in relation to a set of 18 cultivars/near-isogenic lines carrying known powdery mildew resistance genes is given in Table 1. More than one wheat line was used to determine the resistance patterns of some *Pm*/*Ml* genes to take into account genetic background effects on the expression of major resistances. It is evident that the *Pm*7 resistance is not effective against six powdery mildew isolates and can be distinguished only by an intermediate, susceptible (i,s) reaction with isolate W72/27 from wheats with no powdery mildew resistance. In contrast to *Pm*7, the resistance of *Pm*3b has not been overcome by any of the powdery mildew isolates used. Because *Pm*3b is located on 1AS, the high level of resistance of some wheat-rye addition, substitution, and translocation lines is not due to *Pm*3b if the used Thatcher lines are highly susceptible, as will be shown later.

Comparing the resistance patterns of all resistance genes used, that is, *Pm*1 to 9 and *Mlk*, it becomes evident that each powdery mildew resistance gene can be distinguished from all others by a characteristic resistance pattern (Table 1); this was the precise reason for choosing these specific powdery mildew isolates.

Thatcher and some descendants. Table 1 shows the powdery mildew resistance pattern of 4× and 6× Thatcher. Because both forms of Thatcher are highly susceptible to all seven powdery mildew isolates, it can be concluded that the resistance genes *Pm*1 to 9 and *Mlk* are not present in these wheats. The triticale line used is highly resistant to all seven isolates (Table 1). Because this triticale line (6× = 42, genomically AABBRR) was derived from 4× Thatcher carrying the A- and B-genome and because it is highly susceptible, its good resistance can be traced to its rye chromosomes which originated from an inbred line of the rye cultivar Prolific (2n = 14). The same is true for the addition (2n = 44), substitution (2n = 42), and translocation (2n = 42) lines shown in Table 1 because all are isogenic for the A-, B-, and D-genome chromosomes of Thatcher (14). Analyzing the resistance patterns of these lines, it is evident that the rye chromosomes 1R, 4R, and 7R do not condition any powdery mildew resistance against the isolates used here (Table 1). This agrees with Lind's (22) results for the chromosomes 4R (small variable effects when tested with the two powdery mildew isolates used by Lind [22]) and 7R but disagrees with respect to 1R.

TABLE 1. Disease reactions of wheat cultivars/lines (*Triticum aestivum*) after inoculation with seven well-defined powdery mildew isolates (*Erysiphe graminis* f. sp. *tritici*)

Wheat cultivar/line	Resistance gene	Reaction[a] of powdery mildew isolate number:						
		6	85135	W72/27	2	5	9a	4a
Cultivar/near-isogenic lines[b]								
Axminster/8CC[c]	*Pm*1	s	s	s	s	r	r,i	s,i
Avalon, Galahad	*Pm*2	s	s	s	r	r	s	r
Asosan/8CC	*Pm*3a	i	i,r	r	r	r	s	r
Chul/8CC	*Pm*3b	r	r	r	r	r	r	r
Sonora/8CC	*Pm*3c	s	r	···	···	···	s	s
Khapli/8CC, Yuma/8CC	*Pm*4a	s	s	r	s	i,r	s	s
Orbis, Olymp	*Pm*4b	s	s	r	s	r	s	r
Rektor, Wattines	*Pm*5	i,s	i	i	s	s	s	s
Timgalen	*Pm*6	s	s	i	s	i	s	s
Transec	*Pm*7	s	s	i,s	s	s	s	s
Disponent, Götz	*Pm*8	r,i	r,i	r,i	s	s	s	s
Mephisto	*Pm*9,1,2	r	s	s	r	r	r	r
Ralle	*Mlk*	s	r	r	r	r	r	s
Thatcher and Thatcher crosses[d]								
4× Thatcher	···	s	s	s	s	s	s	s
6× Thatcher	···	s	s	s	s	s	s	s
Triticale	some	r	r	r	r	r	r	r
1D/1R sub	···	s	s	s	s	s	s	s
2D/2R sub	*Pm*7?	r	r	r	i	r	r	r
7D/4R sub	···	s	s	s	s	s	s	s
6R add	*Ml*P6L	r[e]	r[e]	r[e]	r[e]	r[e]	r[e]	r[e]
6D/6R sub	*Ml*P6L	r[e]	r[e]	r[e]	r[e]	r[e]	r[e]	r[e]
6RS add	···	s	s	s	s	s	s	s
6D/6RS trans	···	s	s	s	s	s	s	s
7R add	···	s	s	s	s	s	s	s
Wheat-rye derivatives[f]								
TAM-104	?	s	s	s	i	r,i	s	s
Zorba	*Pm*6	r,i	r,i	r,i	s	s	s	s
ST-1	*Pm*8	r	r	r	s	s	s	s

[a] s = susceptible, r = resistant, and i = intermediate reaction.
[b] Wheat cultivars/lines carrying known powdery mildew resistance genes (*Pm*1 to 9, *Mlk*).
[c] Eight times backcrossed with cultivar Chancellor (3).
[d] The disease reactions of hexaploid (6× = 42, genomically AABBDD) and tetraploid (4× = 28, AABB) cultivar Thatcher and of a hexaploid triticale line (6× = 42, AABBRR) derived from crossing tetraploid Thatcher with an inbred line of the spring rye cultivar Prolific (2× = 14, RR) are given. In addition, the disease reactions of addition, substitution, and translocation lines developed by crossing 6× Thatcher and this triticale are shown.
[e] Necrotic spots are observed frequently.
[f] The resistance pattern of TAM-104 carrying a 6BS/6RL translocation and of Zorba and ST-1 carrying a 1B/1R substitution are given.

Chromosome 1R conditioned a high level of powdery mildew resistance in the experiments of Lind (22) but did not in our experiment. This deviation may be explained by the fact that the 1R chromosomes used are of different origin. Consequently, they may possess different genes/alleles. Lind (22) used only two powdery mildew isolates, only one of which is virulent for *Pm*8. Thus, 1R addition lines carrying *Pm*8 should be resistant to one of these isolates but not to both, as was observed by Lind (22). Therefore, these results are worth verifying.

The use of our well-defined powdery mildew isolates allows us to show that Zorba reacts like a *Pm*8 resistant wheat. This cultivar possesses a 1R chromosome instead of chromosome 1B of wheat (25,30) and is considered to be the source of all 1BL/1RS translocations in German winter wheat cultivars (13,31). Some of the cultivars carrying this 1BL/1RS translocation showed the specific resistance pattern conditioned by *Pm*8 (see Disponent and Götz, Zorba, and ST-1 in Table 1), but others did not (13). In these cases, the origin of the rye segment 1RS is the same as the origin of typical *Pm*8 resistant cultivars (13). Therefore, the assumption that the expression of *Pm*8 is suppressed by other genes or that this *Pm*8 gene has been lost or changed in some West German cultivars is justified. It is of scientific interest to show that 1R substitution lines exist that do not react like a *Pm*8 resistant genotype.

Rye chromosome 2R. The rye chromosome 2R conditions a high degree of resistance. This can be seen from the resistant reactions of the 2D/2R substitution line (Table 1) after inoculation with powdery mildew isolates 6, 85135, W72/27, 5, 9a, and 4a; an intermediate reaction was observed with isolate 2. This resistance pattern is different from that of Transec, which is susceptible to most of these powdery mildew isolates (Table 1). Transec carries the powdery mildew resistance gene *Pm*7. This gene is assumed to be located on a 4B/2R (formerly 4A/2R) translocation (2,5–10) (according to the 7th International Wheat Genetics Symposium, 1988, chromosomes 4A and 4B have been exchanged). Without cytological analyses, it would be normal to assume that the 2R chromosome used here carries a more efficient *Pm*7 allele and/or some further powdery mildew resistance genes. The three different 2R chromosomes analyzed by Lind (22) also condition a high degree of powdery mildew resistance to all the resistance parameters measured.

Giemsa C-banding which allows the identification of specific chromosomes or chromosome arms in wheat and rye was used for the further characterization of the Transec translocation. This technique allows one to differentially stain chromosome regions which consist of constitutive heterochromatin; however, the underlying mechanisms are not fully understood. Because the C-banding pattern is highly chromosome specific, it allows the identification of individual chromosomes or chromosome segments even in those cases where other morphological differences are absent. The C-banding pattern of the Transec translocation is shown in Figure 1F. The short arm of that chromosome is characterized by a large C-band adjacent to the centromere and a terminal band, whereas the long arm shows two C-bands adjacent to the centromere, two faint interstitial bands, and one pronounced and one faint band in the subterminal region.

The wheat chromosome involved in this translocation can be identified as being chromosome 4B. Compared with the corresponding chromosome 4B of the hexaploid wheat cultivar Chinese Spring, the Transec translocation differs in the location of the C-bands adjacent to the centromere. However, it is known that chromosome 4B of Chinese Spring carries a pericentric inversion (15) not present in other cultivars of wheat—for example, Poros (12).

Thus our results agree with earlier reports indicating that the Transec translocation involves chromosome 4B (formerly 4A). The C-banding pattern of the Transec translocation differs from a normal chromosome 4B in the distal part of the long arm. The two faint interstitial C-bands observed in the long arm of the Transec translocation are also typical for a normal chromosome 4B, whereas this is not true for the pronounced subterminal C-band found in the long arm of the Transec translocation, which is of different origin. This band closely resembles the subterminal marker band which is characteristic for the long arm of rye chromosome 5R.

The C-banding pattern observed indicates that about half of the long arm of 4B is still present in the Transec translocation. This disagrees with the mapped translocation point, which was found to be located within less than five crossover units from the centromere (2,7). Furthermore, evidence is presented that shows that the distal part of the rye chromosome 5R is present in the Transec translocation, whereas no evidence was found to show that rye chromosome 2R is involved as assumed by Driscoll (5). This chromosome usually shows large telomeric C-bands in one or both arms (26,28) (see Fig. 1A). The existence of a segment derived from rye chromosome 5R in Transec is possible because Driscoll and Sears (11) showed that the Transec ancestor line 82al-2-4-7 possesses a 4B/5R translocation carrying a dominant gene for pubescent peduncle (*Hp*), also called "hairy-neck." This gene is now known to be located in the long arm of rye chromosome 5R (4).

Combining all data, we suggest that the Transec translocation consists of a complete short arm of chromosome 4B of wheat and about half of the proximal region of the long arm of that chromosome and a rye segment derived from the distal part of the long arm of chromosome 5R of rye. The whole arm of 5L may not be involved because Transec does not show the "hairy-neck" character. It is possible that an unbanded intercalary segment of rye chromosome 2R also may be present. However, it never has been shown in crosses with the appropriate wheat-rye addition lines and analyses of meiotic pairing in the obtained hybrids that the telocentric chromosomes present in the Transec ancestor line 82al-2-4-7 are derived from the rye chromosome 2R. The powdery mildew resistance reaction of *Pm*7 greatly differs from the high degree of resistance observed in the 2D/2R Thatcher/Prolific substitution line analyzed here. Irrespective of any further cytological analysis of Transec, the high level of powdery mildew resistance of rye chromosome 2R has not yet been used effectively in wheat-breeding programs.

Resistance conditioned by 6R. The rye chromosome 6R conditions absolute resistance to all seven powdery mildew isolates used (Table 1), irrespective of whether the 6D/6R substitution line (see Fig. 1B) or the 6R addition line is considered. We also tested the powdery mildew isolates 1, 3, 7, and 8 (described by Heun and Fischbeck [20]) and again observed only highly resistant reactions. All these lines showed typical small necrotic spots on the leaves after inoculation with powdery mildew. Such necrotic spots do not occur with any other *Pm*/*Ml* resistance gene previously analyzed (19,20).

Our evidence suggests that the whole resistance of 6R is located in the long arm of that chromosome. First, the analyzed 6RS addition line ($2n = 42 + 2t_S$) and the 6DL/6RS translocation

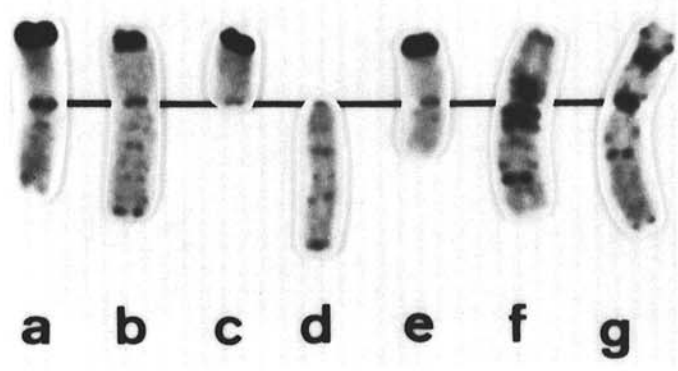

Fig. 1. C-banding patterns of the rye chromosomes/chromosome arms present in the isogenic wheat/rye D-genome substitution lines: **A,** 2D/*2R*, **B,** 6D/*6R*, **C,** 6D/*6RS*, **D,** 6D/*6RL*, and **E,** the 6DL/*6RS* translocation. Also shown are: **F,** the C-banding patterns of the Transec translocation, and **G,** the 6BS/*6RL* translocation present in the line TAM-104.

line (Fig. 1E) were susceptible to all seven powdery mildew isolates (Table 1). Second, within the self-pollinated progeny of a monosomic 6D/6RS substitution line ($2n = 40 + 1t_S$) (Fig. 1C), all plants having $2n = 40$, $2n = 40 + 1t_S$ or $2n = 40 + 2t_S$ reacted highly susceptible to powdery mildew isolate 6. Third, both susceptible and resistant plants occurred among the self-pollinated progeny of a monosomic 6D/6RL substitution line ($2n = 40 + 1t_L$) (Fig. 1D). The cytological analysis of these plants revealed that all 22 susceptible plants possess $2n = 40$ chromosomes, whereas 13 of the 14 resistant plants showed $2n = 40 + 1t_L$ and one plant showed $2n = 40 + 2t_L$. Thus, the conclusion that the total resistance of rye chromosome 6R to the above-mentioned powdery mildew isolates is located on the long arm of that chromosome is justified. In crosses with normal wheat lines or cultivars carrying no 6RL segment, this resistance will be inherited as a single factor because homoeologous chromosome pairing and crossing over are normally suppressed. Therefore, introduction of the temporary gene symbol *Ml*P6L is justified for this new powdery mildew resistance gene originating from Prolific rye and being located in 6RL.

We also analyzed a line called TAM-104, which was developed at Texas A&M University, College Station. This line carries a 6BS/6RL translocation (Tuleen, *personal communication*), but it does not exhibit the high degree of resistance shown by the 6R lines mentioned above (Table 1). C-banding analysis confirms the presence of a 6BS/6RL translocation in that line (Fig. 1G). The short arm of that translocation shows the characteristic marker bands of chromosome 6B of wheat, whereas the long arm shows the characteristic banding pattern of the long arm of rye chromosome 6R. This indicates that the translocation results from centric breakage and fusion. The difference in the C-banding patterns of the long arm of rye chromosome 6R in the TAM-104 translocation and the corresponding chromosome arm in the isogenic Thatcher/Prolific lines is within the range of polymorphic changes known to occur between different cultivars of rye (16,26).

No data currently exist on the genetic relationship between the TAM-104 resistance and that of the other 6R lines. Nevertheless, line TAM-104 allows a specific transfer of the powdery mildew resistance because, in crosses between TAM-104 and the 6R Thatcher lines, crossing over between the different 6RL arms can occur. Moreover, such crosses allow testing whether the resistance gene *Ml*P6L controls the whole 6R resistance pattern alone or in combination with other genes. However, in crosses with normal wheat, a single factor segregation for the 6R resistance of Thatcher lines would occur as discussed above.

The results presented show conclusively that useful powdery mildew resistance provided by rye chromosomes 2R and 6R has not yet been used effectively in wheat breeding. The incorporation of these resistances in wheat cultivars is desirable; therefore, the authors will provide small quantities of seed of the 2R and 6R Thatcher lines to interested plant breeders.

LITERATURE CITED

1. Anonymous. 1989. Beschreibende Sortenliste 1989. Getreide, Mais, Ölfrüchte, Leguminosen, Hackfrüchte, Bundessortenamt. 216 pp.
2. Barlow, K. K., and Driscoll, C. J. 1981. Linkage studies involving two male-sterility mutants in hexaploid wheat. Genetics 98:791-799.
3. Briggle, L. W. 1969. Near-isogenic lines of wheat with genes for resistance to *Erysiphe graminis* f. sp. *tritici*. Crop Sci. 9:70-72.
4. Chang, T. D. 1975. Mapping of the gene for hairy peduncle (*Hp*) on rye chromosome 5R. Can. J. Genet. Cytol. 17:127-128.
5. Driscoll, C. J. 1968. Alien transfer by irradiation and meiotic control. Pages 196-203 in: Proc. Int. Wheat Genet. Symp., 3rd, Canberra, Australia.
6. Driscoll, C. J., and Anderson, L. M. 1967. Cytogenetic studies of Transec—a wheat-rye translocation line. Can. J. Genet. Cytol. 9:375-380.
7. Driscoll, C. J., and Bielig, L. M. 1968. Mapping of the Transec wheat-rye translocation. Can. J. Genet. Cytol. 10:421-425.
8. Driscoll, C. J., and Jensen, N. F. 1963. A genetic method for detecting induced intergeneric translocations. Genetics 48:459-468.
9. Driscoll, C. J., and Jensen, N. F. 1964. Characteristics of leaf rust resistance transferred from rye to wheat. Crop Sci. 4:372-374.
10. Driscoll, C. J., and Jensen, N. F. 1965. Release of a wheat-rye translocation stock involving leaf rust and powdery mildew resistances. Crop Sci. 5:279-280.
11. Driscoll, C. J., and Sears, E. R. 1965. Mapping of a wheat-rye translocation. Genetics 51:439-443.
12. Friebe, B., and Heun, M. 1989. C-banding pattern and powdery mildew resistance of *Triticum ovatum* and four *T. aestivum-T. ovatum* chromosome addition lines. Theor. Appl. Genet. 78:417-424.
13. Friebe, B., Heun, M., and Bushuk, W. 1989. Cytological characterization, powdery mildew resistance and storage protein composition of tetraploid and hexaploid 1BL/1RS wheat-rye translocation lines. Theor. Appl. Genet. 78:425-432.
14. Friebe, B., and Larter, E. N. 1988. Identification of a complete set of isogenic wheat/rye D-genome substitution lines by means of Giemsa C-banding. Theor. Appl. Genet. 76:473-479.
15. Gill, B. S. 1987. Chromosome banding methods, standard chromosome band nomenclature and applications in cytogenetic analysis. Pages 243-254 in: Wheat and Wheat Improvement. Monograph 13. E. G. Heyne, ed. American Soc., Madison, WI. 765 pp.
16. Giraldez, R., Cermeno, M. C., and Orellana, J. 1979. Comparison of C-banding pattern in the chromosomes of inbred lines and open pollinated varieties of rye, *Secale cereale* L. Z. Pflanzenzuecht. 83:40-48.
17. Groenewegen, L. J. M., and van Silfhout, C. H. 1988. The use of wild emmer in Dutch practical wheat breeding. Pages 184-198 in: Cereal Breeding Related to Integrated Cereal Production. M. L. Jorna and L. A. J. Slootmaker, eds. Pudoc, Washington, DC. 244 pp.
18. Heun, M. 1987. Virulence frequencies influenced by host resistance in the host-pathogen system wheat-powdery mildew. J. Phytopathol. 118:363-366.
19. Heun, M., and Fischbeck, G. 1987. Identification of wheat powdery mildew resistance genes by analyzing host-pathogen interactions. Plant Breed. 98:124-129.
20. Heun, M., and Fischbeck, G. 1987. Genes for powdery mildew resistance in cultivars of spring wheat. Plant Breed. 99:282-288.
21. Leath, S., and Murphy, J. P. 1986. Use of mobile nurseries to assess the *Erysiphe graminis* f. sp. *tritici* population in North Carolina. Phytopathology 76:1059-1060.
22. Lind, V. 1982. Analysis of the resistance of wheat-rye addition lines to powdery mildew of wheat (*Erysiphe graminis* f. sp. *tritici*). Tagungsber.-Akad. Landwirtschaftswiss. D. D. R. 198:509-520.
23. Namuco, L. O., Coffman, W. R., Bergstrom, G. C., and Sorrells, M. E. 1987. Virulence spectrum of the *Erysiphe graminis* f. sp. *tritici* population in New York. Plant Dis. 71:539-541.
24. Riley, R., and Macer, R. C. F. 1966. The chromosomal distribution of the genetic resistance of rye to wheat pathogens. Can. J. Genet. Cytol. 8:640-653.
25. Sastrosumario, S., and Zeller, F. J. 1970. Chromosomensubstitution und Desynapsis in der Weizensorte Zorba (W 565). Z. Pflanzenzuecht. 63:185-195.
26. Schlegel, R., Melz, G., and Mettin, D. 1986. Rye cytology, cytogenetics and genetics—Current status. Theor. Appl. Genet. 72:721-734.
27. Streckeisen, P., and Fried, P. M. 1985. Virulenzanalyse im Weizenmehltau in der Schweiz in den Jahren 1981 bis 1983. Schweiz. Landwirtsch. Forsch. 24:261-269.
28. Sybenga, J. 1983. Rye chromosome nomenclature and homeology relationships. Z. Pflanzenzuecht. 90:297-304.
29. Wolfe, M. S., and Slater, S. E. 1988. Mildew of wheat. Pages 6-16 in: 1987 Annual Report. U.K. Cereal Pathogen Virulence Survey. V. Silvey and R. A. Bayles, eds. NIAB Press, Cambridge. 52 pp.
30. Zeller, F. J., and Fischbeck, G. 1971. Cytologische Untersuchungen zur Identifizierung des Fremdchromosoms in der Weizensorte Zorba (W 565). Z. Pflanzenzuecht. 66:260-265.
31. Zeller, F. J., and Hsam, S. L. K. 1983. Broadening the genetic variability of cultivated wheat by utilizing rye chromatin. Pages 161-173 in: Proc. Int. Wheat Genet. Symp., 6th, Kyoto, Japan.