

The genetics of resistance of hexaploid wheat to the wheatgrass powdery mildew fungus

Y. TOSA AND K. SAKAI¹

Kochi University, Faculty of Agriculture, Nankoku, Kochi 783, Japan

Corresponding Editor: A. J. F. Griffiths

Received July 12, 1989

Accepted November 16, 1989

TOSA, Y., and SAKAI, K. 1990. The genetics of resistance of hexaploid wheat to the wheatgrass powdery mildew fungus. *Genome*, **33**: 225–230.

The avirulence of *Erysiphe graminis* f.sp. *agropyri*, Ak-1, on *Triticum aestivum* 'Norin 4' and 'Norin 10' and *T. compactum* 'No.44' is conditioned by four genes; three operate singly against each cultivar and one operates against all three cultivars. If the forma specialis – genus specificity follows the gene-for-gene relationship, four major genes should be involved in the resistance of the three cultivars to Ak-1, one carried only by 'Norin 4', one carried only by 'No.44', one carried only by 'Norin 10', and one carried by all three cultivars. The first and second genes were considered to be the previously reported genes *Pm10* and *Pm11*, respectively. The third and fourth genes were successfully detected using F₁ hybrid cultures between Ak-1 and *E. graminis* f.sp. *tritici*, Tk-1. They were located on chromosomes 6B and 7D and designated *Pm14* and *Pm15*, respectively. These results strongly support the assumption that the forma specialis – genus specificity follows the gene-for-gene relationship. It is, therefore, concluded that this type of specificity belongs to cultivar specificity rather than plant-species specificity and that the resistance to inappropriate formae speciales is essentially cultivar resistance and not nonhost resistance.

Key words: powdery mildew, *Erysiphe graminis*, wheat, wheatgrass, resistance.

TOSA, Y., et SAKAI, K. 1990. The genetics of resistance of hexaploid wheat to the wheatgrass powdery mildew fungus. *Genome*, **33** : 225–230.

Chez le *Triticum aestivum* (Norin 4) et (Norin 10) et le *T. compactum* (No.44), l'avirulence d'*Erysiphe graminis* f.sp. *agropyri*, l'Ak-1, est conditionnée par quatre gènes; trois d'entre eux opèrent individuellement à l'encontre de chacun des cultivars et l'autre opère à l'encontre des trois cultivars. Si la spécificité «forme spéciale – genre» suit une relation «gène pour gène», les quatre gènes principaux devraient être impliqués dans la résistance des trois cultivars à l'Ak-1, soit un premier présent seulement chez le (Norin 4), un second présent seulement chez le (No.44), un troisième présent seulement chez le (Norin 10), et un quatrième présent chez les trois cultivars. Le premier et le second gènes ont été considérés respectivement comme étant les *Pm10* et *Pm11* rapportés antérieurement. Le troisième et le quatrième gènes ont été détectés avec succès, suite à l'emploi de cultures d'hybrides F₁ entre l'Ak-1 et l'*E. graminis* f.sp. *tritici*, le Tk-1. Ces gènes ont été localisés sur les chromosomes 6B et 7D et respectivement désignés *Pm14*, *Pm15*. Ces résultats appuient fortement l'hypothèse que la spécificité forme spéciale – genre suit la relation gène pour gène. La conclusion se dégage donc, que ce type de spécificité est une spécificité de cultivar plutôt que d'espèce, et que la résistance aux formes spéciales inappropriées est essentiellement une résistance de cultivar et non pas une résistance de non-hôte.

Mots clés : le blanc, *Erysiphe graminis*, blé, agropyre, résistance.

[Traduit par la revue]

Introduction

In nature very complicated host–parasite specificity can be recognized between higher plants and phytopathogenic microorganisms. Ellingboe (1976), Day (1976), and Heath (1981a) suggested that this specificity could be divided into two types, which are controlled by different mechanisms. The first type determines whether a plant is a host for a microorganism or, in Heath's words, determines host species range of a pathogen. In this type incompatibility is the rule and the specific interaction resides in compatibility. Heath (1981a) referred to this type of specificity and the resistance involved in it as plant–species specificity and nonhost resistance, respectively. The second type is what is called race–cultivar specificity, which in Heath's words, determines cultivar range within a given host species. In this type, compatibility is the rule and the specific interaction resides in incompatibility. Heath (1981a) referred to this type of specificity and the resistance involved in it as cultivar specificity and cultivar resistance, respectively. The three authors assumed that the second type of specificity is

superimposed on "basic compatibility" resulting from the first type of interaction.

In the *Erysiphe graminis* – gramineous plant system, forma specialis – genus specificity is recognized in addition to race–cultivar specificity (Tosa *et al.* 1987). Some questions then arise: the first is whether the forma specialis – genus specificity belongs to plant–species specificity or cultivar specificity; the second, whether the resistance to inappropriate formae speciales is nonhost resistance or cultivar resistance. Cultivar specificity or resistance has been demonstrated to follow the gene-for-gene relationship (Flor 1956) in many host-parasite systems (Day 1974; Vanderplank 1984). On the other hand, plant-species specificity or nonhost resistance is unlikely to be governed by gene-for-gene interactions (Heath 1981b, 1982). Answers to the preceding questions can be attained by examining whether the forma specialis – genus specificity (or the resistance to inappropriate formae speciales) is governed by gene-for-gene interactions.

We conducted genetic analyses of the forma specialis – genus specificity. Tosa (1989a) reported that four major genes are involved in the avirulence of *E. graminis* f.sp. *agropyri* (wheatgrass mildew fungus), Ak-1, on three

¹Present address: Fukuoka branch of Moji Plant Protection Station, 1-22 Okihama-cho Hakata-ku, Fukuoka 812, Japan.



TABLE 1. Wheat materials used, and their infection types with *Erysiphe graminis* f.sp. *tritici*, Tk-1, *Erysiphe graminis* f.sp. *agropyri*, Ak-1, and their F₁ hybrids, Gw-34, Gw-180, and Gw-121

Wheat	Infection type with:				
	Tk-1	Gw-34	Gw-180	Gw-121	Ak-1
<i>T. aestivum</i> 'Norin 4' (N4)	4	0	4	0	0
<i>T. aestivum</i> 'Norin 26' (N26)	4	0	4	0	0
<i>T. aestivum</i> 'Shin-chunaga' (Sch)	4	0	4	0	0
<i>T. macha</i> var. <i>subletschumicum</i> (Mch)	4	0	2	0	0
<i>T. compactum</i> 'No.44' (Cmp)	4	2	0	0	0
<i>T. aestivum</i> 'Chinese Spring' (CS)	4	3	0	0	0
<i>T. aestivum</i> 'Norin 10' (N10)	4	2	0	0	0
<i>T. aestivum</i> 'Kokeshi-komugi' (Kks)	4	2	0	0	0
<i>T. aestivum</i> 'Akabozu' (Akb)	4	0	0	0	0
<i>T. spelta</i> var. <i>duhamelianum</i> 'Splt'	4	0	0	2	0
<i>T. aestivum</i> 'Red Egyptian' (RE)	4	2	3	4	0

NOTE: Infection types are as follows: 0, no mycelial growth or sporulation; 2, reduced sporulation; 3, slightly reduced sporulation; 4, heavy sporulation.

TABLE 2. Distribution of infection types with Gw-180 in F₂ populations derived from crosses between wheat cultivars

Cross	No. of F ₂ seedlings							Total	Resistant:susceptible	χ^2 (ratio)	P
	0 to 0 ⁺ ^a	1 ⁻	1	1 ⁺	2 ⁻	2	2 ⁺ to 4				
N10 × RE	113	13	6	0	0	9	37	178	132:46	0.07 (3:1)	0.75-0.90
Kks × RE	88	29	9	0	2	5	43	176	126:50	1.09 (3:1)	0.25-0.50
Akb × RE	115	13	2	0	0	5	42	177	130:47	0.23 (3:1)	0.50-0.75
N10 × Kks	353	8	0	0	0	0	0	361	361:0	—	—
Akb × N10	298	58	7	1	0	0	0	364	364:0	—	—
CS × N10	472	30	25	13	0	1	2	543	540:3	30.09 (15:1)	<0.01

^aInfection type. 0 to 1⁺, resistant; 2⁻ to 4, susceptible.

hexaploid wheat cultivars, *Triticum aestivum* 'Norin 4' (N4) and 'Norin 10' (N10) and *T. compactum* 'No.44' (Cmp). One (*A_c*) operates against all three cultivars, and the other three genes, *A_{N4}*, *A_{N10}*, and *A_{Cmp}*, operate against N4 only, N10 only, and Cmp only, respectively. Subsequently, Tosa (1989b) suggested that the forma specialis - genus specificity follows the gene-for-gene relationship. If this suggestion is correct, four major genes should be involved in the resistance of the three wheat cultivars to Ak-1, one carried by all three cultivars, one carried by N4 only, one carried by N10 only, and one carried by Cmp only.

The objective of this study was to identify the four resistance genes. In the light of our results, genetic systems and the nature of the resistance to inappropriate formae speciales were discussed.

Materials and methods

Plant and fungal materials

Wheat materials tested were *Triticum aestivum* (L.) Thell. 'Norin 4' (N4), 'Norin 10' (N10), 'Norin 26' (N26), 'Shin-chunaga' (Sch), 'Chinese Spring' (CS), 'Kokeshi-komugi' (Kks), 'Akabozu' (Akb), and 'Red Egyptian' (RE), *T. compactum* Host 'No.44' (Cmp), *T. macha* Dakapur et Monad. var. *subletschumicum* (Mch), and *T. spelta* L. var. *duhamelianum* (Splt). They were all highly susceptible to *E. graminis* f.sp. *tritici* (wheat mildew fungus), Tk-1, and highly resistant to *E. graminis* f.sp. *agropyri*, Ak-1 (Table 1). However, they showed various responses to Gw-34, Gw-180, and Gw-121 (Table 1), which are F₁ hybrid cultures

derived from the cross Ak-1 × Tk-1 (Tosa 1989a). These cultures were maintained on an accession of einkorn wheat, *T. urartu* Thum. (Urr), which had been used to collect them (Tosa 1989a).

Determination of infection types

F₂ seeds derived from crosses between the wheat cultivars were sown in soil in 1.8 × 35 cm glass test tubes with paper plugs. On the same day conidia of cultures to be tested were transferred to Urr seedlings growing in test tubes. Eight days after sowing, primary leaves of the F₂ seedlings were inoculated with conidia from the 8-day-old colonies by using writing brushes. The seedlings were grown in a controlled-environment room under fluorescent lighting (2000-4000 lx) before and after inoculation. The temperature in the room was 22 ± 1°C during the light cycle (13 h) and 20 ± 1°C during the dark cycle (11 h). Eight days after inoculation, infection was rated using 13 progressive grades from 0 to 4: 0, no mycelial growth or sporulation; 0⁺, mycelial growth or conidiophore formation with no visible sporulation; 1⁻, 1, 1⁺, scant sporulation; 2⁻, 2, 2⁺, reduced sporulation; 3⁻, 3, 3⁺, slightly reduced sporulation; and 4⁻, 4, heavy sporulation. Taking frequency distribution of infection types into consideration, the F₂ seedlings were rated as resistant or susceptible. In this report resistant and susceptible are considered only as relative. Thus, seedlings showing infection type 1 may be judged to be susceptible in some cases.

Results

Resistance to Gw-180

N10, Kks, and Akb were highly resistant to Gw-180, whereas RE was susceptible (Table 1). When F₂ seedlings



TABLE 3. Distribution of infection types with Gw-121 in F₂ populations derived from crosses between wheat cultivars

Cross	No. of F ₂ seedlings							Total	Resistant:susceptible	χ^2 (3:1)	P
	0 to 0 ⁺ ^a	1 ⁻	1	1 ⁺	2 ⁻	2	2 ⁺ to 4				
N4 × RE	137	0	0	0	1	7	31	176	137:39	0.76	0.25-0.50
N26 × RE	124	2	0	1	7	21	15	170	126:44	0.07	0.75-0.90
Sch × RE	131	0	0	0	0	8	30	169	131:38	0.57	0.25-0.50
Cmp × RE	177	22	0	9	12	13	18	251	199:52	2.46	0.10-0.25
CS × RE	173	25	0	10	10	10	36	264	198:66	0	>0.90
N10 × RE	128	0	0	1	2	5	31	167	128:39	0.24	0.50-0.75
Kks × RE	133	1	0	0	8	12	21	175	134:41	0.23	0.50-0.75
Akb × RE	140	0	0	0	2	6	31	179	140:39	0.99	0.25-0.50
N26 × N4	253	0	0	0	0	0	0	253	253:0	—	—
Sch × N4	255	0	0	0	0	0	0	255	255:0	—	—
Cmp × N4	247	0	0	0	0	0	0	247	247:0	—	—
CS × N4	261	0	0	0	0	0	0	261	261:0	—	—
N10 × N4	262	0	0	0	0	0	0	262	262:0	—	—
Kks × N4	210	0	0	0	0	0	0	210	210:0	—	—
Akb × N4	261	0	0	0	0	0	0	261	261:0	—	—

^aInfection type. 0 to 1⁻, resistant; 1⁺ to 4, susceptible.

derived from the crosses N10 × RE, Kks × RE, and Akb × RE, were inoculated with Gw-180, they showed discontinuous distributions of infection types, with one break at 1⁺ (Table 2). Assuming that seedlings showing infection types 0 to 1⁺ and 2⁻ to 4 are resistant and susceptible, respectively, their ratios fitted a 3:1 ratio. Crosses between the resistant cultivars (i.e., N10 × Kks, Akb × N10) yielded no susceptible F₂ seedlings (Table 2). These results showed that the resistance of N10, Kks, and Akb to Gw-180 is controlled by a common, dominant major gene.

The expression of this gene was easily affected by environmental conditions, especially temperature. The temperature of our room for incubating inoculated plants was 21 ± 2°C during the season from October to May, as described in Materials and methods, and 24 ± 2°C in the summer (June–September). When experiments were conducted in the summer season, the resistance allele was recessive, or sometimes resistance was not expressed.

The resistance of Cmp, CS, and Splt to Gw-180 is controlled by *Pm11* (Tosa *et al.* 1988), the expression of which is not affected by the increase of temperature. Therefore, the gene detected in N10, Kks, and Akb appeared to be different from *Pm11*. When F₂ seedlings from CS × N10 were inoculated with Gw-180, susceptible seedlings occurred, but in a ratio that was significantly lower than 1 in 16 (Table 2). This result suggested that the gene in N10 is different from, but closely linked to *Pm11*. Since the latter is located on chromosome 6B (Tosa *et al.* 1988), the former was also considered to be located on chromosome 6B. This gene is different from the previously described series of genes, *Pm1–Pm13*, that control the resistance of wheat to *E. graminis*; none of *Pm1–Pm10* or *Pm12–Pm13* is located on chromosome 6B (McIntosh 1988; Ceoloni *et al.* 1988). The gene detected here is, therefore, designated *Pm14*.

Resistance to Gw-121

N4, N26, Sch, Cmp, CS, N10, Kks, and Akb were highly resistant to Gw-121, whereas RE was highly susceptible (Table 1). When F₂ seedlings from crosses between the resistant cultivars and RE were inoculated with Gw-121, they

showed discontinuous distributions of infection types, with one break at 1 (Table 3). The segregation of resistant (infection types 0 to 1⁻) and susceptible (infection types 1⁺ to 4) seedlings fitted a 3:1 ratio. Crosses between the resistant cultivars yielded no susceptible F₂ seedlings (Table 3). These results showed that the resistance to Gw-121 of N4, N26, Sch, Cmp, CS, N10, Kks, and Akb is controlled by a common, dominant major gene.

The nulli-tetrasomics of CS were available except nulli-2A-tetra-2B, nulli-2A-tetra-2D, nulli-4A-tetra-4B, nulli-4B-tetra-4A, nulli-4B-tetra-4D, nulli-6B-tetra-6D, nulli-4D-tetra-4A, and nulli-5D-tetra-5B. When the available 34 lines were inoculated with Gw-121, nulli-7D-tetra-7A and nulli-7D-tetra-7B were susceptible (infection type 2⁺), whereas the other lines were highly resistant (infection type 0). Among the ditelocentrics of CS, ditelo-7DS was available, but ditelo-7DL was not. The former was resistant to Gw-121. These results suggested that this gene is located on the short arm of chromosome 7D. This gene is different from *Pm1–Pm14*, since none of them is located on chromosome 7D (McIntosh 1988; Ceoloni *et al.* 1988). It is, therefore, designated *Pm15*.

Resistance to Gw-34

Akb was highly resistant to Gw-34, whereas RE was susceptible (Table 1). When F₂ seedlings from Akb × RE were inoculated with Gw-34, they showed a discontinuous distribution of infection types, with one break at 1⁻ (Table 4). The segregation of resistant (infection types 0 to 0⁺) and susceptible (infection types 1 to 4) seedlings fitted a 3:1 ratio. This result showed that the resistance of Akb to Gw-34 is controlled by a dominant, major gene. The gap between the two groups of seedlings occurred on the resistant side compared with those in Tables 2 and 3, which seemed to be attributable to the low susceptibility (infection type 2) of the susceptible RE parent.

The resistance of N4, N26, Sch, and Splt to Gw-34 is controlled by *Pm10* (Tosa *et al.* 1987, 1988). In the F₂ population from Akb × N4 no susceptible seedlings occurred (Table 4). This result suggested that the gene in Akb is *Pm10*.



TABLE 4. Distribution of infection types with Gw-34 in F₂ populations derived from crosses between wheat cultivars

Cross	No. of F ₂ seedlings								Total	Resistant:susceptible	χ^2 (3:1)	P
	0 ^a	0 ⁺	1 ⁻	1	1 ⁺	2 ⁻	2	2 ⁺ to 4				
Akb × RE	65	3	0	8	6	2	4	2	90	68:22	0.01	>0.90
Akb × N4	212	13	0	0	0	0	0	0	225	225:0	—	—

^aInfection type. 0 to 0⁺, resistant; 1 to 4, susceptible.

Discussion

All wheat lines tested are highly susceptible to the wheat mildew fungus, Tk-1, which is one of the parents of Gw-180 and Gw-121. *Pm14* and *Pm15* are, therefore, considered to be involved in resistance to the wheatgrass mildew fungus, Ak-1.

Major genes for resistance to the wheatgrass mildew fungus carried by the common wheat lines tested are summarized in Table 5. N4, N26, and Sch carry *Pm10* and *Pm15*, but not *Pm11* or *Pm14*, since they were susceptible to Gw-180 (Table 1). Cmp and CS carry *Pm11* and *Pm15*, but not *Pm10*, since they were susceptible to Gw-34. They also do not carry *Pm14*, since their resistance to Gw-180 was controlled by one gene, *Pm11*. Similarly, N10 and Kks carry *Pm14* and *Pm15*, but not *Pm10* or *Pm11*. Akb carries three genes, *Pm10*, *Pm14*, and *Pm15*, but not *Pm11*. Splt carries *Pm10* and *Pm11*, but not *Pm15*, since it is susceptible to Gw-121. It also does not carry *Pm14*, since its resistance to Gw-180 is controlled by one gene, *Pm11*. RE carries none of the four genes, since it is susceptible to all three hybrid cultures.

Table 5 shows that the four resistance genes postulated in the Introduction are *Pm10*, *Pm11*, *Pm14*, and *Pm15*. *Pm10* is carried by N4, but not by Cmp or N10, and is considered to correspond to pathogen gene *A_{N4}*. *Pm11* is carried by Cmp, but not by N4 or N10, suggesting that it corresponds to *A_{Cmp}*. *Pm14* is carried by N10, but not by N4 or Cmp, suggesting that it corresponds to *A_{N10}*. *Pm15* is carried by all three cultivars, suggesting that it corresponds to *A_c*. It is, therefore, concluded that the resistance of wheat to the wheatgrass mildew fungus is governed by gene-for-gene interactions. Tosa (1989b) designated *A_{N4}* and *A_{Cmp}* as *Ppm10* and *Ppm11*, respectively. Now, we designate *A_{N10}* and *A_c* as *Ppm14* and *Ppm15*, respectively.

The gene-for-gene theory enables inference of number and identity of resistance genes carried by a plant cultivar from the segregation pattern of avirulent and virulent fungal cultures on the cultivar (Flor 1956; Hiura *et al.* 1961; Moseman 1959, 1966). When N4, N26, and Sch were inoculated with 240 F₁ cultures from Ak-1 × Tk-1, the F₁ population showed bifactorial segregation (3:1) of avirulent and virulent cultures (Tosa 1989a). This suggests that these cultivars carry no more than the two resistance genes appearing in Table 5. On Mch the fungal F₁ population also showed bifactorial segregation, the pattern of which was identical with those on N4, N26, and Sch (Tosa 1989a). This suggests that Mch also carries the two resistance genes, *Pm10* and *Pm15* (Table 5). On Akb the F₁ population showed trifactorial (7:1) segregation (Y. Tosa, unpublished), suggesting that this cultivar carries no more than the three resistance genes appearing in Table 5. However, on Splt the F₁ population showed tetrafactorial (15:1) segregation (Y. Tosa, unpubl-

ished). Presumably Splt carries two unknown resistance genes in addition to *Pm10* and *Pm11*.

Table 6 shows the genotypes of the mildew cultures used, and the resistance genes involved in their interactions with the three representative wheat cultivars, N4, Cmp, and N10. Ak-1 carries all of the four avirulence genes, *Ppm10*, *Ppm11*, *Ppm14*, and *Ppm15*. When Ak-1 is placed on N4, *Ppm10* and *Ppm15* match *Pm10* and *Pm15*, respectively, and incompatibility results. On Cmp *Ppm11* and *Ppm15* permit resistance to be expressed by *Pm11* and *Pm15*, respectively. On N10 *Ppm14* and *Ppm15* permit resistance to be expressed by *Pm14* and *Pm15*, respectively. On the other hand, Tk-1 carries no avirulence genes, and when it is placed on N4, Cmp, or N10, compatible interactions result, since there are no corresponding gene pairs that produce incompatibility. Hybridization between Ak-1 and Tk-1 produces all combinations of the avirulence genes. Gw-34 carries *Ppm10* but not the other three genes. When it is placed on N4, *Ppm10* matches *Pm10*, resulting in an incompatible interaction. When it is placed on Cmp or N10, there are no gene pairs producing incompatibility, so compatible interactions result. On the other hand, Gw-180 carries *Ppm11* and *Ppm14* but not the other two genes. When it is placed on Cmp and N10, the corresponding gene pairs, *Ppm11-Pm11* and *Ppm14-Pm14*, respectively, cause incompatible interactions. On N4 no gene pair produces incompatibility. Gw-121 carries only *Ppm15*. However, its corresponding resistance gene, *Pm15*, is carried by all three cultivars, so an incompatible interaction results on each of the three cultivars.

The results of the present study strongly support the concept that the forma specialis - genus specificity follows the gene-for-gene relationship (Tosa 1989b). We therefore conclude that this type of specificity belongs to cultivar specificity rather than plant-species specificity. This means that the resistance to inappropriate formae speciales is essentially cultivar resistance, not nonhost resistance. Presumably the genes involved in the resistance to inappropriate formae speciales are not essentially different from those controlling interactions between races and cultivars, and the durability of the resistance, if any, would be mainly attributed not to the involved genes themselves, but to their "stacking." These ideas also apply to the cereal rust systems (Johnson 1976). It is thus not surprising that resistance genes introduced to wheat from different genera were rendered ineffective by the occurrence of new races (e.g., Negulescu and Ionescu-cojocaru 1974). Such races could easily occur within the wheat-attacking forma specialis by mutation of the avirulence genes corresponding to the introduced resistance genes, without introducing virulence by hybridization from the formae speciales that attack the donor genera of the resistance genes.



TABLE 5. Major genes for resistance to the wheatgrass mildew fungus, Ak-1, carried by various common wheat lines

Wheat	Resistance gene			
	<i>Pm10</i>	<i>Pm11</i>	<i>Pm14</i>	<i>Pm15</i>
N4	+ ^a	—	—	+
N26	+ ^a	—	—	+
Sch	+ ^a	—	—	+
Mch	+ ^b	—	—	+ ^b
Cmp	—	+ ^c	—	+
CS	—	+ ^c	—	+
N10	—	—	+	+
Kks	—	—	+	+
Akb	+	—	+	+
Splt	+ ^c	+ ^c	—	—
RE	—	—	—	—

NOTE: +, present; —, absent.

^aReported by Tosa *et al.* (1987).^bInferred from the segregation pattern of F₁ hybrids between Ak-1 and Tk-1 (Tosa 1989a).^cReported by Tosa *et al.* (1988).TABLE 6. Genetic mechanisms of the interactions between cultures of *E. graminis* and representative cultivars of common wheat

Wheat	<i>E. graminis</i> culture				
	Tk-1	Gw-34	Gw-180	Gw-121	Ak-1
	[—]	[<i>Ppm10</i>]	[—]	[—]	[<i>Ppm10</i>]
	[—]	[—]	[<i>Ppm11</i>]	[—]	[<i>Ppm11</i>]
	[—]	[—]	[<i>Ppm14</i>]	[—]	[<i>Ppm14</i>]
	[—]	[—]	[—]	[<i>Ppm15</i>]	[<i>Ppm15</i>]
N4 [<i>Pm10</i> ; — ; — ; <i>Pm15</i>]	S	R (<i>Pm10</i>)	S	R (<i>Pm15</i>)	R (<i>Pm10</i> , <i>Pm15</i>)
Cmp [— ; <i>Pm11</i> ; — ; <i>Pm15</i>]	S	S	R (<i>Pm11</i>)	R (<i>Pm15</i>)	R (<i>Pm11</i> , <i>Pm15</i>)
N10 [— ; — ; <i>Pm14</i> ; <i>Pm15</i>]	S	S	R (<i>Pm14</i>)	R (<i>Pm15</i>)	R (<i>Pm14</i> , <i>Pm15</i>)

NOTE: R, resistant; S, susceptible. Square brackets and parentheses indicate genotypes of hosts or parasites, and resistance genes involved in their interactions, respectively. *Ppm10*, *Ppm11*, *Ppm14*, and *Ppm15* are avirulence genes corresponding to *Pm10*, *Pm11*, *Pm14*, and *Pm15*, respectively. —, virulent or susceptible allele.

Acknowledgments

The authors thank Dr. U. Hiura, emeritus professor of Okayama University, Okayama, and Dr. K. Tsunewaki, professor of Kyoto University, Kyoto, for providing the common wheat lines. The authors also thank Dr. K. Nishikawa, professor of Gifu University, Gifu, for providing the nulli-tetrasomics of 'Chinese Spring', and Dr. H. Tsujimoto, Kihara Institute for Biological Research, Yokohama, for providing the ditelocentrics of 'Chinese Spring'. Special thanks are due to Dr. H. Ogura, professor of Kochi University, Kochi, for constant encouragement throughout the course of this study.

CEOLONI, C., DEL SIGNORE, G., PASQUINI, M., and TESTA, A. 1988. Transfer of mildew resistance from *Triticum longissimum* into wheat by *ph1* induced homoeologous recombination. Proceedings of the 7th International Wheat Genetics Symposium, July 13-19, 1988, Cambridge, U.K. Agricultural and Food Research Council, Institute of Plant Science Research, Cambridge, U.K. pp. 221-226.

DAY, P.R. 1974. Genetics of host-parasite interaction. Freeman, San Francisco.

———. 1976. Gene functions in host-parasite systems. In *Specificity*

in plant disease. Edited by R.K.S. Wood and A. Graniti. Plenum Press, New York. pp. 65-73.

ELLINGBOE, A.H. 1976. Genetics of host-parasite interactions. *Encycl. Plant Physiol.* 4: 761-778.

FLOR, H.H. 1956. The complementary genetic systems in flax and flax rust. *Adv. Genet.* 8: 29-54.

HEATH, M.C. 1981a. A generalized concept for host-parasite specificity. *Phytopathology*, 71: 1121-1123.

———. 1981b. Nonhost resistance. In *Plant disease control: resistance and susceptibility*. Edited by R.C. Staples and G.H. Toenniessen. John Wiley & Sons, New York. pp. 201-217.

———. 1982. The absence of active defense mechanisms in compatible host-pathogen interactions. In *Active defense mechanisms in plants*. Edited by R.K.S. Wood. Plenum Press, New York. pp. 143-156.

HIURA, U., HETA, H., and TSUSHIMA, T. 1961. Pathogenic variability attributed to hybridization in *Erysiphe graminis hordei*. Studies on variation in pathogenicity. II. *Nogaku Kenkyu*, 48: 107-115. (In Japanese.)

JOHNSON, R. 1976. Genetics of host-parasite interactions. In *Specificity in plant disease*. Edited by R.K.S. Wood and A. Graniti. Plenum Press, New York. pp. 45-62.

MCINTOSH, R.A. 1988. Catalogue of gene symbols for wheat. 1988 ed. Proceedings of the 7th International Wheat Genetics Symposium, July 13-19, 1988, Cambridge, U.K. Agricultural



- and Food Research Council, Institute of Plant Science Research, Cambridge, U.K. pp. 1225-1323.
- MOSEMAN, J.G. 1959. Host-pathogen interaction of the genes for resistance in *Hordeum vulgare* and for pathogenicity in *Erysiphe graminis* f.sp. *hordei*. *Phytopathology*, **49**: 469-472.
- _____. 1966. Genetics of powdery mildews. *Annu. Rev. Phytopathol.* **4**: 269-290.
- NEGULESCU, F., and IONESCU-COJOCARU, M. 1974. The outbreak of a new form of race 77 of *Puccinia recondita* f.sp. *tritici* on wheat cultivar Aurora in Romania in 1973. *Cereal Rusts Bull.* **2**: 19-22.
- TOSA, Y. 1989a. Genetic analysis of the avirulence of wheatgrass powdery mildew fungus on common wheat. *Genome*, **32**: 913-917.
- _____. 1989b. Evidence on wheat for gene-for-gene relationship between formae speciales of *Erysiphe graminis* and genera of gramineous plants. *Genome*, **32**: 918-924.
- TOSA, Y., TSUJIMOTO, H., and OGURA, H. 1987. A gene involved in the resistance of wheat to wheatgrass powdery mildew fungus. *Genome*, **29**: 850-852.
- TOSA, Y., TOKUNAGA, H., and OGURA, H. 1988. Identification of a gene for resistance to wheatgrass powdery mildew fungus in the common wheat cultivar Chinese Spring. *Genome*, **30**: 612-614.
- VANDERPLANK, J.E. 1984. *Disease resistance in plants*. 2nd ed. Academic Press, Inc., London.

