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Geographical distribution of genes for resistance to formae speciales of *Erysiphe graminis* in common wheat

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Abstract The geographical distribution of *Pm10*, *Pm11*, *Pm14*, and *Pm15* wheat genes for resistance to inappropriate formae speciales of *Erysiphe graminis* was investigated using gene-for-gene relationships. *Pm10* and *Pm15* were very common among many indigenous accessions of common wheat collected from various areas in the world. The diversity of genotypes, which consisted of allelic combination at those loci, was high near the center of origin of common wheat and decreased with increasing distance from the center. In Europe, an apparent contrast of predominant genotypes occurred between the south and the north, suggesting that these genes are useful markers for revealing the routes by which common wheat spread in Europe. On a whole, the genes for resistance to inappropriate formae speciales were observed to be widely distributed throughout the world. We suggest that the difference between these genes and the genes for resistance to races of an appropriate forma specialis may only be in their distribution and that of their corresponding avirulence genes.

Key words *Erysiphe graminis* · Forma specialis · Resistance · Wheat ·

Introduction

Erysiphe graminis, the causal agent of powdery mildews of gramineous plants, is widely distributed throughout the world. It comprises several formae speciales, e.g., f.sp. *tritici* parasitic on *Triticum*, f.sp. *secalis* parasitic on *Secale*, and f.sp. *agropyri* parasitic on *Agropyron*. The genetic basis of the differences among formae speciales was established by Hiura and his co-workers. Hiura (1962) found that different formae speciales could be hybridized and that there were various levels of reproductive isolation among them. He observed that f.sp. *tritici* and f.sp. *agropyri* were inter-fertile and, when crossed, produced germinable F_1 ascospores (Hiura 1964). Based on analyses of F_1 populations between the two formae speciales Hiura (1978) concluded that a number of genes were involved in the avirulence of f.sp. *agropyri* on wheat cultivars and that some of them operated commonly on many wheat cultivars. The latter conclusion led him to an assumption that many wheat cultivars might have common genes for mildew resistance.

Genetic analyses of the resistance to inappropriate formae speciales were also initiated by Hiura and his co-workers. Hiura et al. (1981) reported that wheat genes for resistance to f.sp. *agropyri* could be detected by using hybrids between f.sp. *agropyri* and f.sp. *tritici*. Using this method, Oku et al. (1986) described a total of eight genes controlling the resistance of wheat to f.sp. *agropyri* and f.sp. *secalis*. Tosa and his co-workers (Tosa et al. 1987, 1988; Tosa and Sakai 1990) detected four genes that were involved in the resistance of wheat to f.sp. *agropyri*. These were located on chromosomes 1D, 6B, 6B, and 7D, and were designated as *Pm10*, *Pm11*, *Pm14*, and *Pm15*, respectively. *Pm15* was also involved in the resistance of wheat to f.sp. *secalis*, but *Pm10*, *Pm11*, or *Pm14* were not (Tosa 1994).

How many wheat cultivars share these resistance genes? Are there common resistance genes as assumed by Hiura (1978)? In order to answer these questions we examined the geographical distribution of those genes using about 360 indigenous wheat accessions collected from various areas throughout the world. Such an analysis would be a time-consuming and laborious task if carried out by crossing plants. In the *E. graminis* – gramineous plant system, however, gene-for-gene relationships (Flor 1956) can be used to identify resistance genes. Tosa and his co-workers (Tosa 1989a,b; Tosa and Sakai 1990) identified avirulence genes corresponding to *Pm10*, *Pm11*, *Pm14*, and *Pm15*,

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and designated them as *Ppm10*, *Ppm11*, *Ppm14*, and *Ppm15*, respectively. These reports demonstrated that the forma specialis – genus specificity follows the gene-for-gene relationship. Therefore, if resistance gene *Ra* carried by accession A corresponds to, for example, *Ppm10*, we can conclude without crossing plants that *Ra* is *Pm10*. This method was successfully applied to the identification of *Pm10* and *Pm15* among accessions of *Aegilops squarrosa* (*Triticum tauschii*), the donor of the D genome of common wheat (Tosa and Sakai 1991). In the investigation presented in this paper, this method was applied to making a survey of resistance genes among a number of common wheat accessions.

Materials and methods

Fungal cultures

The fungal cultures used are listed in Table 1. Parental field isolates were *Erysiphe graminis* DC. f.sp. *agropyri* Em. Marchal, Ak-1, f.sp. *tritici* Em. Marchal, Tk-1, and f.sp. *secalis* Em. Marchal, Sk-1. Gw cultures, comprising 240 cultures from Gw-1 to Gw-273, were F_1 hybrids derived from the cross Ak-1 × Tk-1 (Tosa 1989a). Rw cultures, comprising 60 cultures from Rw-301 to Rw-360, were F_1 hybrids derived from the cross Sk-1 × Tk-1 (Tosa 1994). Dh cultures, comprising 45 cultures from Dh-1 to Dh-45, were hybrids derived from a cross between two representative Gw cultures, Gw-34 and Gw-180 (Tosa 1989b). These cultures were maintained at 3° ± 1 °C on susceptible seedlings growing in 2 × 35-cm glass test tubes with paper plugs.

Determination of infection types

Seeds of the test plants were sown in soil in 2 × 35-cm test tubes. On the same day conidia of cultures to be tested were transferred to seedlings of a common host of all cultures, *Triticum urartu* Thum. accession 199-1, growing in the test tubes. Eight days after sowing, primary leaves of the test plants were inoculated with conidia from the 8-day-old colonies using writing brushes. The seedlings were grown in a controlled-environment room under fluorescent lighting (2000–4000 lux) before and after inoculation. The temperature in the room was 23° ± 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 without sporulation; 1−, conidiophore formation without visible powder-like conidia; 1, 1+, scant sporulation; 2−, 2, 2+, reduced sporulation; 3−, 3, 3+, slightly reduced sporulation; 4−, 4, heavey sporulation. The frequency distribution of infection types among Dh cultures that had been observed in the previous study (Tosa 1989b) suggested that infection types 0 to 0+ and 1 to 4 should be considered as incompatible and compatible, respectively, but that infection type 1− was difficult to be sorted. In this study, infection types 0–1− were considered as incompatible (resistant or avirulent), and infection types 1–4 as compatible (susceptible or virulent).

Plant materials

Plant materials tested (IL, JL, CL, SL, and CP) were indigenous accessions of *Triticum aestivum* (L.) Thell. collected from various areas throughout the world. First, a total of 366 accessions were inoculated with Tk-1. Four accessions were resistant to Tk-1, and the others were susceptible. The 4 resistant accessions were excluded from further analyses since they were presumed to be resistant to all of the hybrid cultures mentioned above. The remaining 362 accessions were inoculated with Ak-1 and Sk-1. Two are susceptible to Ak-1, 3 to Sk-1, and 1 to both. The remaining 356 accessions were resistant to both Ak-1 and Sk-1. Sk-1 carries *Ppm15* and induces a resistant reaction in plants carrying *Pm15* (Table 1). Therefore, the accessions susceptible to Sk-1 were considered to be non-carriers of *Pm15*. Similarly, the accessions susceptible to Ak-1 were considered to carry none of the resistance genes *Pm10*, *Pm11*, *Pm14*, or *Pm15* since Ak-1 carried all of their corresponding avirulence genes (Table 1).

Experiment I

The 356 accessions resistant to Ak-1 and Sk-1 were inoculated with Gw-34, Gw-180, and Gw-121, representative F_1 hybrids derived from the cross, Ak-1 × Tk-1. Accessions susceptible to Gw-34 were considered to be non-carriers of *Pm10* since Gw-34 carried *Ppm10* (Table 1). Similarly, those susceptible to Gw-180 were considered to carry neither *Pm11* or *Pm14* since Gw-180 carried both *Ppm11* and *Ppm14*. Accessions resistant to Gw-180 and/or Gw-34 might carry *Pm11*, *Pm14*, and/or *Pm10*. However, there is the possibility that these cultures carry unknown avirulence genes and that their resistance is due to their corresponding, unknown genes. Further analysis was carried out in Experiment II. Accessions susceptible to Gw-121 were considered to be non-carriers of *Pm15* since Gw-121 carried *Ppm15* (Table 1). Those resistant to Gw-121 were subjected to Experiment VI to test whether or not the resistance was due to *Pm15*.

Table 1 Cultures of *E. graminis* used and their infection types with representative wheat cultivars

Cultures		Infection type [a] on		
		Norin 4 (N4) [*Pm10*; − ; − ;*Pm15*][b]	Chinese Spring (CS) [− ;*Pm11*; − ;*Pm15*]	Kokeshi-komugi (Kks) [− ; − ;*Pm14*; *Pm15*]
Ak-1 (f.sp. *agropyri*)	[*Ppm10*; *Ppm11*; *Ppm14*; *Ppm15*]	0 (*Pm10*, *Pm15*)	0 (*Pm11*, *Pm15*)	0 (*Pm14*, *Pm15*)
| Gw cultures (hybrids between Ak-1 and Tk-1)				
Gw-34	[*Ppm10*; − ; − ; −]	0 (*Pm10*)	3	2
| Dh cultures (hybrids between Gw-34 and Gw-180)				
Gw-180	[− ;*Ppm11*; *Ppm14*; −]	4	0 (*Pm11*)	0 (*Pm14*)
Gw-121	[− ; − ; − ;*Ppm15*]	0 (*Pm15*)	0 (*Pm15*)	0 (*Pm15*)
Tk-1 (f.sp. *tritici*)	[− ; − ; − ; −]	4	4	4
| Rw cultures (hybrids between Sk-1 and Tk-1)				
Sk-1 (f.sp. *secalis*)	[− ; − ; − ;*Ppm15*]	0 (*Pm15*)	0 (*Pm15*)	0 (*Pm15*)

[a] 0, no mycelial growth or sporulation; 2, reduced sporulation; 3, slightly reduced sporulation; 4; heavy sporulation

[b] Square brackets indicate genotypes of hosts or parasites. Parentheses following infection types indicate resistance genes involved in the interactions. *Ppm10*, *Ppm11*, *Ppm14*, and *Ppm15* are avirulence genes corresponding to resistance genes, *Pm10*, *Pm11*, *Pm14* and *Pm15*, respectively; −, virulent or susceptible allele.

Experiment II

Cultures used in Experiment II were 20 hybrids (Dh-26 to Dh-45) derived from the cross Gw-34 × Gw-180 (Table 2). As avirulence genes, Gw-34 carries *Ppm10*, while Gw-180 carries *Ppm11* and *Ppm14* (Table 1); among their F_1 progenies, *Ppm10*, *Ppm11*, and *Ppm14* should segregate. The genotypes of the Dh cultures were determined from their reaction on three representative wheat cultivars (*T. aestivum*), 'Norin 4' (N4), 'Chinese Spring' (CS), and 'Kokeshi-komugi' (Kks) (Table 2), which carry *Pm10* and *Pm15*, *Pm11* and *Pm15*, and *Pm14* and *Pm15*, respectively (Table 1, Tosa and Sakai 1990). Although these three cultivars all carry *Pm15*, this gene is not involved in their reaction to Dh cultures. This is because its corresponding avirulence gene, *Ppm15*, is absent in Gw-34 and Gw-180, from which the Dh cultures are derived. Dh cultures incompatible with N4, CS, and Kks were considered to carry *Ppm10*, *Ppm11*, and *Ppm14*, respectively (Table 2). Based on the reaction to these Dh cultures with different genotypes we were able to determine the genotypes of the wheat accessions. Some examples are shown in Table 2. IL59, an indigenous accession from Ethiopia, was resistant to Gw-34 and susceptible to Gw-180. When inoculated with Dh cultures, this accession was resistant to all cultures with *Ppm10* and susceptible to all cultures without *Ppm10*. In other words, the pattern of incompatibility/compatibility corresponded with the segregation of *Ppm10* among the Dh cultures. This result indicated that IL59 carried *Pm10*. On IL30 the pattern of reactions corresponded with the segregation of *Ppm11*, indicating that IL30 carried *Pm11*. IL196 was resistant to both Gw-34 and Gw-180. However, segregation among Dh cultures also occurred on this accession; cultures with *Ppm10*, *Ppm11*, or both were incompatible, whereas those without *Ppm10* nor *Ppm11* were compatible. This result suggested that IL196 carried both *Pm10* and *Pm11*. On IL67, cultures with at least one of the *Ppm10*, *Ppm11*, and *Ppm14* genes were incompatible, and compatible cultures were only those that carried none of them. This result suggested that IL67 carried *Pm10*, *Pm11*, and *Pm14*. On IL238, incompatible and compatible cultures segregated in a ratio of 1:1, suggesting that a single avirulence gene was involved in the reactions. However, the pattern did not correspond with those of *Ppm10*, *Ppm11*, or *Ppm14*, suggesting that IL238 carried an unknown resistance gene. On CP45, incompatible and compatible cultures segregated in a ratio of 3:1, suggesting that two avirulence genes were involved in the reactions. Although the pattern did not correspond with any typical patterns deduced from the combination of *Ppm10*, *Ppm11*, or *Ppm14*, all cultures with *Ppm10* were incompatible. These results suggested that one of the two avirulence genes was *Ppm10*, the other unknown, and that CP45 carried *Pm10* and an unknown resistance gene.

This method is very useful but accompanied by risks. Let us assume that accession X carries resistance gene *Rx* and that its corresponding avirulence gene, *Ax*, is closely linked to *Ppm10*. If the set of Dh cultures does not contain recombinants between *Ax* and *Ppm10*, the analysis described above will lead us to the wrong conclusion that X carries *Pm10*. The possibility of such a misjudgement will increase with the number of avirulence genes involved in the segregation. Thus, accessions that were thought to carry more than one gene were further tested in Experiments III, IV, and V.

Experiments III, IV, and V

Experiment III was conducted to check the involvement of *Pm10*. Eleven cultures were chosen from Gw cultures (Table 3). They were all incompatible with N4 but compatible with CS and Kks, and therefore, were considered to carry *Ppm10* but not *Ppm11*, *Ppm14*, or *Ppm15*. Accessions that are susceptible to at least 1 of these cultures should be non-carriers of *Pm10*. Some examples are shown in Table 3. Experiment II indicated that IL139 carries both *Pm10* and *Pm11* and

Table 2 Cultures of *E. graminis* used in Experiment II (identification of *Pm10*, *Pm11*, and *Pm14* using Dh cultures)

Culture	Reaction[a] with			Genotype	Infection type with						
	N4	CS	Kks		IL59[b]	IL30	IL196	SL43	IL67	IL238	CP45
Gw-34	I	c	c	[*Ppm10*; – ; – ; –]	0	2	0	2	0	0	0
Gw-180	c	I	I	[– ;*Ppm11*;*Ppm14*; –]	4	0	0	0	0	2	0+
Dh-26	I	c	I	[*Ppm10*; – ;*Ppm14*; –]	0+	3–	0	0	0	1–	0
Dh-27	I	I	I	[*Ppm10*;*Ppm11*;*Ppm14*; –]	0	0+	0	0	0	0	0
Dh-28	c	I	I	[– ;*Ppm11*;*Ppm14*; –]	3+	0	0+	0	0	0	0+
Dh-29	I	I	I	[*Ppm10*;*Ppm11*;*Ppm14*; –]	0	0	0	0	0	0	0
Dh-30	I	c	c	[*Ppm10*; – ; – ; –]	0	4	0	1+	0	0+	0
Dh-31	c	c	I	[– ; – ;*Ppm14*; –]	4–	3–	2–	0	0	2	0
Dh-32	c	I	I	[– ;*Ppm11*;*Ppm14*; –]	3+	0	0+	0	0	0	0
Dh-33	c	c	c	[– ; – ; – ; –]	3+	3+	1+	4	3	3–	3–
Dh-34	c	I	c	[– ;*Ppm11*; – ; –]	3–	0+	0	0	0	3–	1+
Dh-35	I	c	c	[*Ppm10*; – ; – ; –]	0	3+	0	2–	0	0	0
Dh-36	I	I	I	[*Ppm10*;*Ppm11*;*Ppm14*; –]	0	0	0	0	0	0	0
Dh-37	c	I	I	[– ;*Ppm11*;*Ppm14*; –]	3	0	1–	0+	0	2–	3+
Dh-38	c	I	I	[– ;*Ppm11*;*Ppm14*; –]	3	0	0+	0	0	1+	0
Dh-39	c	c	I	[– ; – ;*Ppm14*; –]	3	3+	1+	0	0	1	2–
Dh-40	I	c	c	[*Ppm10*; – ; – ; –]	0	3+	0	1	0	4	0
Dh-41	I	I	I	[*Ppm10*;*Ppm11*;*Ppm14*; –]	0	0+	0	0	0	2+	0
Dh-42	I	I	c	[*Ppm10*;*Ppm11*; – ; –]	0	0	0	0+	0	1+	0
Dh-43	c	I	I	[– ;*Ppm11*;*Ppm14*; –]	4–	0	0+	0	0	3	2
Dh-44	c	c	c	[– ; – ; – ; –]	3	4–	1+	4–	2–	3+	2+
Dh-45	I	c	c	[*Ppm10*; – ; – ; –]	0	4–	0	2	0	0	0
Resistance genes involved					*Pm10*	*Pm11*	*Pm10* + *Pm11*	*Pm11* + *Pm14*	*Pm10* + *Pm11* + *Pm14*	Unknown	*Pm10* + Unknown

[a] I, incompatible (infection type 0–1 –); c, compatible (infection type 1–4)
[b] Wheat accession

Table 3 Cultures of *E. graminis* used in Experiment III (check of involvement of *Pm10* using Gw cultures)

Culture	Reaction[a] with			Genotype	Infection type with	
	N4	CS	Kks		IL139[b]	IL184
Gw-45	I	c	c	[*Ppm10*; – ; – ; –]	0	0
Gw-50	I	c	c	[*Ppm10*; – ; – ; –]	0	0
Gw-64	I	c	c	[*Ppm10*; – ; – ; –]	0	0
Gw-77	I	c	c	[*Ppm10*; – ; – ; –]	0	0
Gw-80	I	c	c	[*Ppm10*; – ; – ; –]	0	0
Gw-91	I	c	c	[*Ppm10*; – ; – ; –]	0	0
Gw-116	I	c	c	[*Ppm10*; – ; – ; –]	0	0
Gw-199	I	c	c	[*Ppm10*; – ; – ; –]	0	0
Gw-204	I	c	c	[*Ppm10*; – ; – ; –]	0	0
Gw-211	I	c	c	[*Ppm10*; – ; – ; –]	0	0
Gw-226	I	c	c	[*Ppm10*; – ; – ; –]	0	1
Involvement of *Pm10*					Yes	No

[a] I, incompatible (infection type 0–1 –); c, compatible (infection type 1–4)
[b] Wheat accession

that IL184 carries *Pm10*, *Pm11*, and *Pm14*. When inoculated with the 11 cultures, IL139 showed resistance to all cultures, suggesting that this accession carried *Pm10*. On the other hand, IL184 was susceptible to a culture carrying *Ppm10* (Gw-226), indicating that this accession did not carry *Pm10*. Presumably the other 10 cultures carry an avirulence gene that is different from, but closely linked to, *Ppm10*, and IL184 carries its corresponding resistance gene. Similar analyses were conducted for *Pm11* in Experiment IV and for *Pm14* in Experiment V.

Experiment VI

The cultures used in Experiment VI were 39 F_1 hybrids (Rw cultures) derived from the cross, Sk-1 × Tk-1 (Table 4). Sk-1 carries *Ppm15* but none of the other three avirulence genes, while Tk-1 carries none of these genes (Table 1). Among their F_1 progenies only *Ppm15* should segregate. The genotypes of the Rw cultures were determined from their reactions on Kks. Although Kks carries *Pm14* and *Pm15*, *Pm14* is not involved in its interactions with the Rw cultures. This is because its corresponding avirulence gene, *Ppm14*, is absent in Sk-1 and Tk-1, from which Rw cultures are derived. Cultures incompatible and compatible with Kks were considered to be carriers and non-carriers of *Ppm15*, respectively. Some examples of analyses are shown in Table 4. On IL159 all of the cultures with *Ppm15* were incompatible, suggesting that IL159 carried *Pm15*. The cultures without *Ppm15* were thus expected to be compatible, but some of them were incompatible. Such a case as this has been frequently observed in Rw cultures and is considered to be due to minor genes (Tosa 1994). On IL41, incompatible and compatible cultures segregated in a ratio of 1:1, suggesting that a single avirulence gene was involved in the interactions. However, as the pattern of segregation did not correspond with that of *Ppm15*, IL41 was considered to carry a resistance gene other than *Pm15*.

Results

Carriers of *Pm10*, *Pm11*, *Pm14*, and *Pm15* accounted for 62.2%, 22.4%, 8.3%, and 68.0% of the 362 accessions tested, respectively. The geographical distribution of various genotypes is summarized in Table 5. For simplification, *Pm14*, which showed the lowest occurrence, was excluded from this table. Various genotypes occur-

Table 4 Cultures of *E. graminis* used in Experiment VI (identification of *Pm15* using Rw cultures)

Culture	Reaction[a] with Kks	Genotype	Infection type with	
			IL159[b]	IL41
Rw-322	c	[– ; – ; – ; –]	1	0
Rw-323	I	[– ; – ; – ;*Ppm15*]	0	1+
Rw-324	c	[– ; – ; – ; –]	2	0
Rw-325	c	[– ; – ; – ; –]	2	2+
Rw-326	c	[– ; – ; – ; –]	2–	2
Rw-327	I	[– ; – ; – ;*Ppm15*]	0	0
Rw-328	c	[– ; – ; – ; –]	3	1
Rw-329	I	[– ; – ; – ;*Ppm15*]	0	1
Rw-330	c	[– ; – ; – ; –]	0	0
Rw-331	c	[– ; – ; – ; –]	1–	0
Rw-332	I	[– ; – ; – ;*Ppm15*]	0	0
Rw-333	c	[– ; – ; – ; –]	2	1
Rw-334	I	[– ; – ; – ;*Ppm15*]	0	0
Rw-335	c	[– ; – ; – ; –]	2	0
Rw-336	I	[– ; – ; – ;*Ppm15*]	0	1–
Rw-337	I	[– ; – ; – ;*Ppm15*]	0	1
Rw-338	c	[– ; – ; – ; –]	2–	0
Rw-339	c	[– ; – ; – ; –]	1	0
Rw-340	c	[– ; – ; – ; –]	1	0
Rw-341	I	[– ; – ; – ;*Ppm15*]	0	1
Rw-342	I	[– ; – ; – ;*Ppm15*]	0	0
Rw-343	I	[– ; – ; – ;*Ppm15*]	0	0
Rw-344	c	[– ; – ; – ; –]	1+	2
Rw-345	I	[– ; – ; – ;*Ppm15*]	0	0
Rw-346	c	[– ; – ; – ; –]	0	3+
Rw-347	I	[– ; – ; – ;*Ppm15*]	0	1–
Rw-348	I	[– ; – ; – ;*Ppm15*]	0	1
Rw-349	I	[– ; – ; – ;*Ppm15*]	0	2
Rw-350	I	[– ; – ; – ;*Ppm15*]	0	1+
Rw-351	I	[– ; – ; – ;*Ppm15*]	0	2
Rw-352	c	[– ; – ; – ; –]	3	1
Rw-353	c	[– ; – ; – ; –]	1	1+
Rw-354	I	[– ; – ; – ;*Ppm15*]	0	0
Rw-355	c	[– ; – ; – ; –]	2	2
Rw-356	c	[– ; – ; – ; –]	4	3
Rw-357	I	[– ; – ; – ;*Ppm15*]	0	2+
Rw-358	I	[– ; – ; – ;*Ppm15*]	0	0
Rw-359	c	[– ; – ; – ; –]	1	1+
Rw-360	c	[– ; – ; – ; –]	2	0
Resistance gene involved			*Pm15*	Unknown

[a] I, Incompatible (infection type 0–1 –); c, compatible (infection type 1–4)
[b] Wheat accession

red near the center of origin of common wheat (Georgia, Armenia, Azerbaidjan, Iran, Turkey); especially in Iran, where all of the eight genotypes possible were found. With increasing distance from this area, however, the diversity decreased. In the east, *Pm10* and *Pm15* prevailed widely, and genotype [*Pm10* + *Pm15*] was predominant while *Pm11* was rarely found. A similar pattern was also found in the west (Greece, Italy, and Egypt – the Mediterranean countries). On the other hand, the northern areas were characterized by a low occurrence of *Pm15*: most accessions carried *Pm10* alone, *Pm11* alone, or neither of them. Particularly in Sweden, the pattern was extremely uniform; most accessions carried *Pm11* alone. Diverse genotypes occurred in Ethiopia even though it is distant from the center of origin.

Table 5 Geographical distribution of genes for resistance to formae speciales of *E. graminis*

Area	Distribution[a]								Number of accessions (%)			
	Pm10 *Pm11* — [b]	— *Pm11* —	— — —	*Pm10* — —	*Pm10* — *Pm15*	— — *Pm15*	— *Pm11* *Pm15*	*Pm10* *Pm11* *Pm15*	Total of *Pm10* carriers	*Pm11* carriers	*Pm15* carriers	Total
Japan (Kyushu)				..	:::::::				16 (97.0)	0 (0.0)	14 (81.8)	16
(Other islands)			.	...	::::::.				16	0	13	17
Korea				.	...				4	0	3	4
China (Dongbei)			.	.	.....	.			6	0	6	8
(Jangsu)				.	::::...		.	..	14	3	14	15
(Shandong)					.....	.			5 (77.3)	0 (9.1)	6 (93.9)	6
(Zhejang, Fujian)					:::::..				12	0	12	12
(Zhongbu of China)					..	.	..		2	2	5	5
(Sichuan)					....	.....	.		4	1	11	11
(Unknown)					....	.			4	0	5	5
Bhutan					::.....		.	....	13 (90.6)	5 (50.0)	14 (96.9)	14
Nepal			.		.....		.	:::....	16	11	17	18
Tadzhikistan						..	..	.	1	3	5	5
Pakistan				.	....	.	.	.	6 (52.9)	2 (29.4)	7 (88.2)	8
Afghanistan	.			..	:.....	.....	....		11	5	18	21
Iran	.	...	..	..	:::::::	:::....	.....	.....	24 (54.5)	16 (36.4)	36 (81.8)	44
Transcaucasia (South)[c]	.....	...	:::::...	.....	.....		.		17 (53.1)	9 (28.1)	6 (18.8)	32
Turkey	.	..	.....	.	.....	....			8 (33.3)	3 (9.1)	10 (39.4)	19
Iraq			.		...				3	0	3	4
Greece					..	.			2	0	3	3
Italy					..				2 (90.0)	0 (0.0)	2 (100.0)	2
Egypt					.....				5	0	5	5
Ethiopia		..	.	.	::::::.	.....	.	...	17 (65.4)	6 (23.1)	22 (84.6)	26
Turkmenistan			.						0	0	0	1
Uzbekistan			...	.		.			1	0	1	5
Kazakhstan			...	.					1	0	0	4
Transcaucasia (North)[d]		....	...	..					2 (29.6)	4 (24.1)	0 (7.4)	9
Ukraine	.		:.....	....	.				6	1	1	14
Moldova		..	....	.....		..			5	2	2	13
Sweden		.....	.	.					1	6	0	8
America			...			...	.	.	1	2	5	8
Total									225 (62.2)	81 (22.4)	246 (68.0)	362

[a] One dot represents one accession
[b] Combination of genes
[c] South of Caucasus Mts (Georgia, Armenia, Azerbaidjan)
[d] North of Cuaucasus Mts (Daghestan, North Ossetian)

Discussion

In the present study the gene-for-gene relationship was successfully applied to a rapid survey of resistance genes among a large number of accessions. This method enables us to identify resistance genes without crossing plants. Its advantages can be summarized as follows. (1) Very rapid. One cycle (sowing – inoculation – determination of infection types) takes only 16 days, and about 3000 seedlings can be tested per month by one person. (2) Comparison of resistance genes is possible even between plant species that are different in genome constitution or ploidy level.

The diversity of genotypes was high near the center of origin of common wheat (Transcaucasia) and decreased with increasing distance from the center. This is a typical pattern that follows Vavilov's gene center theory. An exception was Ethiopia where high diversity was observed in spite of its location distant from Transcaucasia. Ethiopia is known to be a secondary center of diversity of tetraploid wheat and other crops that were first domesticated in the Near East (Harlan 1975). It should be noted that, in Europe, there was an apparent contrast of predominant genotypes between the south (Greece and Italy) and the north (Sweden) and that the pattern in Sweden was similar to that in Moldova. Tsunewaki (1970) and Zeven (1980) investigated the geographical distribution of necrosis genes in wheat for the purpose of revealing its origin and routes of spreading. We suggest that the genes for resistance to formae speciales are also very useful markers for this purpose, especially for elucidating routes of spreading in Europe. More accessions, especially from Central Europe, must be tested.

Pm10 and *Pm15* were observed to be widely distributed over the world and very common in common wheat accessions. This result supports Hiura's assumption (Hiura 1978) that many wheat cultivars carry common genes for mildew resistance. Those genes may be comparable to *Sr18* in the stem rust system (McIntosh 1988; Loegering and Powers 1962; Loegering and Harmon 1969). *Pm11* and *Pm14* occurred at relatively low frequencies. However, this may be because the accessions tested in the present study include many from China, Japan and few from Europe. If more accessions from Europe, especially from the north of the Alps, were included, we may have obtained different results. Most accessions from Sweden carried *Pm11* (Table 5) and *Pm14* (data not shown).

Among the 366 accessions tested in the preliminary experiment, only 4 were resistant to *E. graminis* f.sp. *tritici*, Tk-1. These accessions are considered to carry genes for resistance to races of the wheat mildew fungus. What is the difference between resistance genes to races and those to formae speciales? Both are under the control of gene-for-gene relationships (Tosa 1989b; Tosa and Sakai 1990; Tosa and Tada 1990) and both are considered to govern hypersensitive cell death (Tosa et al. 1985, 1990), which suggest that there is no functional difference between the two types of resistance genes. The difference may be only in the distribution of the genes and their corresponding avirulence genes. The resistance genes to inappropriate formae speciales are widely distributed throughout the world, and their corresponding avirulence genes scarcely occur in the population of an appropriate forma specialis. On the other hand, the resistance genes to races of an appropriate forma specialis occur at very low frequencies, and their corresponding avirulence genes are widely distributed in the population of an appropriate forma specialis.

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References

Flor HH (1956) The complementary genic systems in flax and flax rust. Adv Genet 8:29–54

Harlan JR (1975) Geographic patterns of variation in some cultivated plants. J Hered 66:182–191

Hiura U (1962) Hybridization between varieties of *Erysiphe graminis*. Phytopathology 52:664–666

Hiura U (1964) Pathogenicity of the haploid progeny cultures from crosses between cultures of *Erysiphe graminis* f.sp. *tritici* and *Erysiphe graminis* f.sp. *agropyri* (preliminary note). Ber Ohara Inst Landwirtsch Biol Okayama Univ 12:131–132

Hiura U (1978) Genetic basis of formae speciles in *Erysiphe graminis* D.C. In: Spencer DM (ed) The powdery mildews. Academic Press, London, pp 101–128

Hiura U, Heta H, Uno H (1981) Genes of wheat for resistance to powdery mildew (in Japanese). Ann Phytopathol Soc Jpn 47:368 (abstr)

Loegering WQ, Harmon DL (1969) Wheat lines near-isogenic for reaction to *Puccinia graminis tritici*. Phytopathology 59:456–459

Loegering WQ, Powers HR (1962) Inheritance of pathogenicity in a cross of physiological races 111 and 36 of *Puccinia graminis* f.sp. *tritici*. Phytopathology 52:547–554

McIntosh RA (1988) Catalogue of gene symbols for wheat. In: Miller TE, Koebner RMD (eds) Proc 7th Int Wheat Genet Symp. Bath Press, Bath, UK, pp 1225–1323

Oku T, Yamashita S, Doi Y, Hiura U (1986) Genetic analysis of resistance of wheat cultivars to races and some formae speciales of *Erysiphe graminis* DC. Ann Phytopathol Soc Jpn 52:700–708

Tosa Y (1989a) Genetic analysis of the avirulence of wheatgrass powdery mildew fungus on common wheat. Genome 32:913–917

Tosa Y (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 (1994) Gene-for-gene interactions between the rye mildew fungus and wheat cultivars. Genome 37:758–762

Tosa Y, Sakai K (1990) The genetics of resistance of hexaploid wheat to the wheatgrass powdery mildew fungus. Genome 33:225–230

Tosa Y, Sakai K (1991) Analysis of the resistance of *Aegilops squarrosa* to the wheatgrass mildew fungus by using the gene-for-gene relationship. Theor Appl Genet 81:735–739

Tosa Y, Tada S (1990) Operation of resistance genes in wheat to *Erysiphe graminis* f.sp. *tritici* against *E. graminis* f.sp. *agropyri*. Genome 33:231–234

Tosa Y, Mise K, Shishiyama J (1985) Recognition of two formae speciales of *Erysiphe graminis* by wheat cells. Ann Phytopathol Soc Jpn 51:223–226

Tosa Y, Tsujimoto H, Ogura H (1987) A gene involved in the resistance of wheat to wheatgrass powdery mildew fungus. Genome 29:850–852

Tosa Y, Tokunaga H, 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

Tosa Y, Akiyama T, Ogura H (1990) Cytological aspects of interactions between formae speciales of *Erysiphe graminis* and genera of gramineous plants, and their evolutionary implications. Can J Bot 68:1249–1253

Tsunewaki K (1970) Necrosis and chlorosis genes in common wheat and its ancestral species. Seiken Ziho 22:67–75

Zeven AC (1980) The spread of bread wheat over the old world since the neolithicum as indicated by its genotype for hybrid necrosis. J Agric Trad Bot Appl 27:19–53