Differential Suppression of Stripe Rust Resistance in Synthetic Wheat Hexaploids Derived from *Triticum turgidum* subsp. *dicoccoides* and *Aegilops squarrosa*

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We thank G. Jochemsen (CPRO-DLO) for technical assistance in the production of the synthetic hexaploids, R. Johnson (Cambridge Laboratory, JI Centre, Norwich, England) for critically reading the manuscript and useful suggestions for improvement, and R. P. L. A. de Rooij (IPO-DLO) for preparing the figures.

Accepted for publication 12 December 1994.

ABSTRACT

Kema, G. H. J., Lange, W., and Van Silfhout, C. H. 1995. Differential suppression of stripe rust resistance in synthetic wheat hexaploids derived from *Triticum turgidum* subsp. *dicoccoides* and *Aegilops squarrosa*. Phytopathology 85:425-429.

The expression of resistance to stripe rust of wheat, conditioned by four *Aegilops squarrosa* (DD, $2n = 14$) and 11 *Triticum turgidum* subsp. *dicoccoides* (AABB, $2n = 28$) accessions, was studied by testing 22 synthetic hexaploids (AABBDD, $2n = 42$) with five stripe rust races in the seedling stage and two races in the adult plant stage. Resistance in one or both parents was frequently suppressed in the synthetic hexaploids, indicating the presence of suppressor genes on the AB and D genomes. Specificity was apparent because the putative suppressor genes affected the expression of specific resistance genes, although not with all races nor in all growth stages. The results and data from F_1 and F_2 populations derived from crosses between two synthetic hexaploids with the same *T. t. dicoccoides* parent but with different *A. squarrosa* parents revealed that several recessively inherited suppressor genes on the D genome seemed to be involved. A possible mechanism explaining these results and the variable mode of action of suppressors are discussed.

Additional keywords: gene expression, race specificity, resistance mechanism, wild emmer wheat.

Broadening the genetic variation for resistance to plant pathogens in general can be accomplished through introduction of alien genetic material into cultivated crops (22,27). Knott (23) listed the genes in wheat conferring resistance to rust diseases derived from distant relatives of bread wheat, *Triticum aestivum* (AABBDD, $2n = 42$). Closely related species or progenitors of bread wheat such as *Aegilops squarrosa* L. (DD, $2n = 14$) and *T. monococcum* (AA, $2n = 14$) are useful sources of resistance to several cereal diseases and pests (1,5,24,39). Wild emmer wheat, *T. turgidum* (L.) Thell. subsp. *dicoccoides* (Körn) Thell. (AABB, $2n = 28$), another putative progenitor of bread wheat, was comprehensively studied with respect to resistance to stripe rust (*Puccinia striiformis* f. sp. *tritici* Westend.) and powdery mildew (*Erysiphe graminis* f. sp. *tritici*) (12–14,40–42).

A number of resistance genes to cereal rusts originated from alien species, including stripe rust resistance genes *Yr17, Yr15, Yr9, Yr8,* and possibly *Yr7* that were transferred to bread wheat by extensive crossing programs (3,14,28,32). An alternative strategy to promote transfers from the full complement of the progenitors of bread wheat, i.e., wild emmer wheat and *A. squarrosa*, is the production of amphiploids from hybrids of these two species (25,26).

Despite the value of related species with respect to resistance to cereal rusts, breeding for resistance in bread wheat requires the expression of the genes involved at higher ploidy levels. The objective of our study was to elucidate the expression of stripe rust resistance derived from wild emmer wheat and *A. squarrosa* when combined in the form of synthetic wheat hexaploids (SHs).

MATERIALS AND METHODS

Stripe rust isolates. The inoculum for this study was obtained from the stripe rust gene bank at IPO-DLO (Research Institute for Plant Protection, Wageningen, the Netherlands). Five isolates were employed to produce single-pustule and monospore cultures used to evaluate the response of the synthetic hexaploids and their parents (Table 1).

Plant materials. Lange and Jochemsen (25,26) generated 22 synthetic hexaploids from 11 stripe rust-resistant wild emmer accessions and eight *A. squarrosa* accessions (Table 2). Initial triploid ABD embryos were rescued and treated with colchicine to produce functional AABBDD hexaploid plants (25). Seed harvested from the first generation after the colchicine treatment (C_2) was used in most experiments reported here. Seed harvested from the colchicine-treated plants of SH40 (C_1) was used in an additional experiment. Parental wild emmer and *A. squarrosa* accessions were included in each experiment.

SH40 and SH58, which have wild emmer wheat G148-1-2M as a common parent, were crossed to study the effect of segregating suppressors contributed by the parental *A. squarrosa* accessions, Cambridge L and Rennes 33. The derived F_1 and F_2 populations, as well as the parents and the donor accessions involved, were inoculated with race 6 E16. The F_1 and F_2 plants were tested at different times but under similar environmental conditions.

Experimental procedures. The growth conditions and inoculation and incubation procedures were similar to those described by Kema (17). In each seedling experiment 10–15 plants per entry, SHs and parental accessions, were grown. *A. squarrosa* accessions were planted 3 days before the other accessions because the growth rate of this species was low compared to that of wild emmer wheat and the SHs. This facilitated simultaneous inoculations of all plants in a similar growth stage, i.e., optimally developed primary leaves, at 10 and 13 days after planting wild emmer,

SHs, and *A. squarrosa* accessions, respectively. Observations were conducted twice, at 16 and 21 days postinoculation (DPI). Infection types (ITs) were classified according to the 0–9 scale in which 0 represents no symptoms and 9 complete susceptibility (29). Experiments with races 234 E171, 6 E16, and 66 E0 were conducted three times. The experiment with races 32 E0 and 45 E140 was performed once. Each experiment usually included all SHs and their parental accessions. SHs 18, 32, 40, 58, and 91 were tested several additional times in seedling and adult plant stages.

For adult plant experiments, seedlings with known reactions

TABLE 1. Virulence/avirulence characteristics, origins, and race numbers of five *Puccinia striiformis* isolates used to study the expression of resistance in hexaploids synthesized from *Triticum turgidum* subsp. *dicoccoides* and *Aegilops squarrosa*

Isolate	Origin	Race[a]	Virulence/avirulence on differential cultivars[b]																
			1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18
IPO87036[c]	Kenya	6 E16	−	+	+	−	−	−	−	−	−	−	−	−	−	−	+	−	−	−
IPO76033[c]	India	66 E0	−	+	−	−	−	−	+	−	−	−	−	−	−	−	−	−	−	−
IPO60018[c]	The Netherlands	32 E0	−	−	−	−	−	+	−	−	−	−	−	−	−	−	−	−	−	−
IPO88519[d]	The Netherlands	45 E140	+	−	+	+	−	+	−	−	−	−	−	−	+	+	−	−	−	+
IPO85564[d]	The Netherlands	234 E171	−	+	−	+	−	+	+	+	−	−	+	−	−	+	−	+	−	+

[a] Nomenclature according to Johnson et al (16).

[b] + = Virulent and − = avirulent for resistance genes in differential cultivars 1–18. Kema and Lange (18) proposed to incorporate *Yr15* in the differential set of cultivars through extension of the World set cultivars. Such a suggestion for *Yr5* by Wellings and McIntosh (43) has been adopted here. The World set comprises the cultivars 1–10, cultivars 11–18 form the European set. The names of these cultivars and their respective *Yr* genes are: 1, Chinese 166-*Yr1*; 2, Lee-*Yr7*+; 3, Heines Kolben-*Yr6*+; 4, Vilmorin 23-*Yr3*+; 5, Moro-*Yr10*+; 6, Strubes Dickkopf; 7, Suwon/Omar; 8, Clement-*Yr9*+; 9, *T. aestivum* subsp. *spelta* var. *album*-*Yr5*; 10, *T. t. dicoccoides* sel. G25-*Yr15*; 11, Hybrid 46-*Yr4*[b]+; 12, Reichersberg 42-*Yr7*+; 13, Heines Peko-*Yr6*+; 14, Nord Desprez-*Yr3*+; 15, Compair-*Yr8*; 16, Carstens V; 17, Spaldings Prolific; and 18, Heines VII-*Yr2*+.

[c] Monospore isolates.

[d] Single-pustule isolates.

TABLE 2. Composition of 22 synthetic wheat hexaploids (SH numbers) and the stripe rust infection types (ITs) of these hexaploids and their constituent *Triticum turgidum* subsp. *dicoccoides* (male) and *Aegilops squarrosa* (female) parents, after inoculation with five *Puccinia striiformis* races in the seedling stage and with two races in the adult plant stage[a]

Parents of SHs			Infection type							
			Parents		Synthetic hexaploids					
					Seedlings					Adults
T. t. dicoccoides[b]	*A. squarrosa*[b]	Synthetic hexaploids	Male (all)	Female (all)	32 E0	45 E140	234 E171	6 E16	66 E0	6 E16	66 E0
G4M-1M (*A*)[c]	Rennes 33	SH48	*[d]	1–7[e]	*	2	*	7–8	3–6	2–4	1
G4M-1M (*A*)	Rennes 33	SH49	*	1–7[e]	8	5–8	7–8	7–8	5–8	3	1
G4M-1M (*A*)	Gatersleben 473	SH84	*	8–9	2–3	1–3	1–3	7–8	3–6	2	1
G4M-1M (*A*)	Gatersleben 525	SH86	*	1–3	1–2	1–2	2–3	7–8	3–4	1	1
G25-4M (*Yr15*)	Cambridge L	SH39	1	8–9	7	1–3	1–5	5–8	7	1?	...[f]
G90-1-1BM (*C*)[g]	Cambridge G	SH50	1–2	1–3	2	1–2	1	3–5	2–3	1–2	1
G148-1-2M (*C*)[g,h]	Cambridge L	SH58	1	8–9	7	1–4	1–3	7	5–8	3–4	2–8
G148-1-2M (*C*)	Rennes 33	SH40	1	1–7[e]	7	1–2	2–4	7	2–3	4–5	1
G148-1-2M (*C*)	Gatersleben 473	SH87	1	8–9	2	1–2	2	7	2–6	3–5	2–3
G148-1-2M (*C*)	Gatersleben 525	SH89	1	1–3	2–4	1–2	2	4–7	2–3	2–4	1–2
G168-1-2-4BM (*A*)[i]	Cambridge G	SH44	1	1–3	1–2	1–2	1	2–6	2–3	1–2	1–3
G168-1-2-4BM (*A*)	Cambridge L	SH22	1	8–9	2–3	1–3	1	7–8	*	3	2–3
G193-1M (*B*)[j]	Cambridge G	SH66	1–2	1–3	*	1–5	1–3	4–7	2–3	3–5	2–3
G193-1M (*B*)	Cambridge L	SH68	1–2	8–9	2–4	1	1–2	1–3	2–3	1–3	1–2
G306-12M (*D*)	Gatersleben 194	SH189	1–2[k]	8–9[l]	7	1–3	2–5	7	*	2–4	2–3
G315[a]-1M (*E*)[m]	Cambridge L	SH15	1–3[n]	8–9	6–7	1	2–5	7	*	1–2	1–2
G315[a]-1M (*E*)	Rennes 33	SH10	1–3[n]	1–7[e]	*	1–5	*	7–8	2–6	5–7	1–2
G326-1-4-5-3M (*F*)	Gatersleben 189	SH192	...[f]	8–9[l]	2–4	1–2	2–3	3–6	2–3	1–2	1–2
G342-2-2M (*B*)	Cambridge L	SH18	1–2	8–9	*	2	1–5	7	8	2–3	8
G342-2-2M (*B*)	Rennes 33	SH32	1–2	1–7[e]	6–8	*	*	8	*	3	2–8
G342-2-2M (*B*)	Gatersleben 143	SH91	1–2	1–3	2	1–2	1–3	2–3	3	2–3	1–3
G363-4-4BM (*C*)[g]	Cambridge L	SH34	1	8–9	2–3	1	1–3	1–5	2	2–4	...

[a] Observation of seedlings and adult plants were performed at 21 and 28 days postinoculation, respectively. Seedling responses (ITs) of parents were for all races within the indicated ranges. Particular deviations are explained in subsequent footnotes. Adult plant responses were similar to those in the seedling stage, except for *T. t. dicoccoides* G4-1M (SHs 48, 49, 84, and 86), which showed a homogeneous IT 1 to all races in the adult plant stage, and *A. squarrosa* Gatersleben 473 (SHs 84 and 87), which showed IT 3-4 to race 6 E16.

[b] *T. t. dicoccoides* accessions originated from the Volcani Center, Bet Dagan, Israel; *A. squarrosa* accessions originated from genebanks at Cambridge, Gatersleben, and Rennes.

[c] Similar letters refer to identical resistance spectra as observed in studies by Van Silfhout et al (42) with 28 stripe rust isolates representing 25 races of the fungus.

[d] Heterogeneous response, i.e. covering both resistant ITs (1–3) and susceptible ITs (7–9).

[e] *A. squarrosa* Rennes 33 was only susceptible to race 6E 16 (IT 7). Responses to the other races ranged from IT 1 to 3.

[f] Missing value.

[g] No compatible interactions observed to date (41; G. H. J. Kema, *unpublished*).

[h] One dominant gene (G. H. J. Kema and A. Badebo, *unpublished*).

[i] Two dominant genes (12,41).

[j] Two complementary dominant genes (41).

[k] *T. t. dicoccoides* G306-12M was only susceptible to race 66 E0 (IT 7–8). Responses to the other races ranged from IT 1 to 2.

[l] *A. squarrosa* accessions Gatersleben 194 (SH189) and Gatersleben 189 (SH192) had a heterogeneous response to race 32 E0.

[m] One dominant gene (41).

[n] *T. t. dicoccoides* G315[a]-1M had a heterogeneous response to race 66 E0.

to races 6 E16 and 66 E0 were selected and vernalized for 6 wk at 5 C (light intensity 200 μE s^{-1} m^{-2} for 12 h a day and darkness for the rest of the day). After vernalization, the plants (generally three per IT) were transplanted, one plant per pot (experimental units) in a mixture of clay, peat, and sand, and grown for 10 wk in a walk-in climate chamber (light intensity: 175 μE s^{-1} m^{-2} for the whole period; day/night: 4 wk at 12 h/12 h at 10 C, 2 wk at 14 h/10 h at 15 C, and 4 wk at 16 h/8 h at 17 C). Inoculations with spore suspensions of the same isolates in mineral oil (Soltrol 170, Phillips Scientific, Mahwah, NJ) were conducted during anthesis (GS 59-69, [38]) with an ultra-low volume sprayer (Micron-Ulva 8, Micron Sprayers Ltd., Bromyard, England). The plants were incubated individually in narrow plastic bags at 10 C for 48 h in a walk-in climate chamber, which was adjusted to the conditions of the seedling experiments afterward (light intensity: 175 μE s^{-1} m^{-2} for 16 h a day, and darkness for 8 h day; temperature: 18 C during the light period and 16 C during darkness; relative humidity: 70%). Final observations of the flag and second leaves of each stem were performed at 28 DPI.

RESULTS

Responses of parental accessions. The seedling responses of the *A. squarrosa* and wild emmer accessions to the five stripe rust races were generally either uniformly resistant or susceptible. Adult plant responses of both species to races 6 E16 and 66 E0 were similar to those in the seedling stage. Incompatible interactions of the *A. squarrosa* accessions had slightly higher ITs compared to the ITs observed in the wild emmer accessions, which generally showed a very strong hypersensitive response with minute (IT 1) or larger necrotic flecks (IT 2) (Table 2).

Responses of the SHs in the seedling stage. The level of resistance, ITs observed in one or both parents, was reduced in most SHs (Table 2). Parental ITs, either from wild emmer or *A. squarrosa*, were expressed in only 18 of the 110 SH-race combinations. Wild emmer ITs were observed in only five combinations: SH50, SH44, and SH22 inoculated with race 234 E171, and SH68 and SH34 inoculated with race 45 E140. Resistance derived from either the wild emmer or *A. squarrosa* donors or both was only occasionally expressed to all races, as in SHs 91, 68, and 34. Nevertheless, the ITs induced by races 66 E0 and 32 E0 on SH91 were slightly higher than the parental ITs. SH68 and SH34 occasionally showed an intermediate response after inoculation with races 32 E0 and 6 E16, respectively. The majority of SHs, however, showed a susceptible response toward at least one race, irrespective of the resistance in one or both donors to such a race.

Suppression of resistance was particularly evident after inoculations with race 6 E16. The hypersensitive response conferred by *Yr15* (IT 1) in a tetraploid background was suppressed in SH39 after inoculation with pathotypes 32 E0, 6 E16, and 66 E0. Similarly, the hypersensitive response of wild emmer G148-1-2M to race 6 E16 (IT 1) was suppressed in SH87, but was expressed to races 32 E0, 45 E140, and 234 E171, though a slightly higher response (IT 2) was observed. The resistance in *A. squarrosa* Rennes 33 was clearly suppressed after inoculating SH49 with races 32 E0, 45 E140, 234 E171, and 66 E0. The resistance in *A. squarrosa* Gatersleben 525, parental accession of SH86 and SH89, was only suppressed to race 6 E16. Reciprocal suppression, i.e., a susceptible response of the SH in spite of resistance in both donor accessions, was observed several times, as exemplified by the responses of SH32 and SH40 to race 32 E0 and, to a lesser extent, by the responses of SH66 and SH89 to race 6 E16.

These observations provide evidence for the presence of suppressors on the genomes of both *T. t. dicoccoides* and *A. squarrosa*. The suppressors had a specific mode of action toward the resistance genes in the parental genotypes and toward the races involved. This is illustrated by observations of SHs 39, 58, 22, 68, 15, 18, and 34, which have *A. squarrosa* Cambridge L as a parent. The suppressor(s) on the genome of this accession blocks the resistance of accession G168-1-2M in SH22 to race 6 E16 but not to race 234 E171, which is a clear example of specificity toward races. A comparison of the responses of SH18 and SH34, which have wild emmer accessions G342-2-2M and G363-4-4BM as a parent, respectively, to race 66 E0 revealed significantly different ITs, IT 8 versus IT 2, which is a clear example of specificity toward the different resistance genes in these wild emmer accessions.

The development of sporulating phenotypes in most cases was retarded and not completed until 21 DPI, though ITs on the parents developed within 16 DPI and susceptible controls were heavily sporulating (IT 9) at 14 DPI. The eventual responses of some SHs toward certain races, e.g., SHs 22, 189, and 32 to race 66 E0 (Table 2), were very heterogeneous and covered the whole range of ITs in each experiment. Initial responses (<10 DPI) of these SHs resembled the typical hypersensitive response in the parents but increased later in genotypes in which suppression was evident, often resulting in ITs comparable with a mesothetic response. To exclude possible heterogeneity in the C_2 SHs materials, C_1 seedlings, which originated from seed harvested from the colchicine-treated plants of SH40, were challenged with races 32 E0 and 234 E171. Comparison of observations at 16 DPI and 21 DPI revealed a completely altered spectrum of ITs (Fig. 1). The majority of plants that had a resistant or intermediate response to race 32 E0 at 16 DPI were susceptible (IT 7) at 21 DPI. This shift toward susceptibility, though less pronounced, also was observed with race 234 E171 (Fig. 1), which revealed a broader range of ITs than initially observed (Table 2).

Responses of SHs in the adult plant stage. Comparison of the seedling and adult plant responses toward races 6 E16 and 66 E0 generally showed an enhanced expression of resistance in the latter stage, e.g., compare SHs 58, 40, and 87 with race 6 E16. Adult plant resistance to both races was observed in a number of SHs, e.g. SHs 84, 22, and 15. The range of ITs tended to be smaller when suppression of resistance was evident, as in SH66 and SH89 with race 6 E16 and SH10 and SH87 with race 66 E0, and the full expression of symptoms required much more time (28 DPI) compared to the seedling stage (21 DPI). An illustrative example of differential suppression of resistance was provided by the response of SH18, which was susceptible to both races in the seedling stage but resistant to race 6 E16 in the adult plant stage (Table 2).

Genetics of suppression. The inheritance of suppressor genes was dealt with in the seedling stage, since suppression of resistance was most evident in that stage. The resistance of accession G148-1-2M (IT 1) was suppressed in SH40 (IT 7) and SH58 (IT 7) after inoculation with race 6 E16 (Table 2; Fig. 2). The F_1 of the cross between these SHs, however, was resistant (ITs 1–3; Fig. 2). The F_2 population revealed nearly the whole spectrum of ITs (Fig. 2). Parental phenotypes (SH40 and SH58; Table 2) predominated in the entire population. Forty-eight of 142 F_2

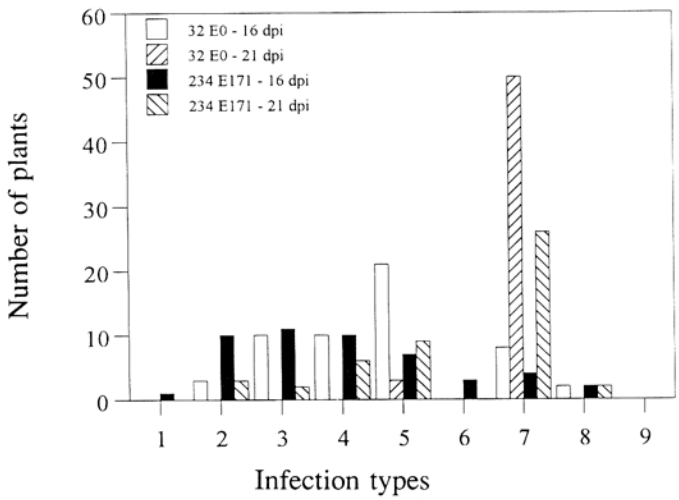

Fig. 1. Frequency of stripe rust infection types on colchicine-treated (C_1) plants of synthetic wheat hexaploid SH40 at 16 and 21 days postinoculation (DPI) with races 32 E0 ($n = 53$) and 234 E171 ($n = 48$).

individuals had higher resistance levels (ITs < 7) than the parents (ITs ≥ 7), but only a few plants approached the phenotype of accession G148-1-2M (IT 1) (Table 2). Since no obvious separation between resistant and susceptible plants was evident in the F_2, the response of the F_1 was considered appropriate for a division in resistant (ITs ≤ 3) and susceptible (ITs ≥ 4) plants, which resulted in a segregation ratio of R:S = 8:134. This is close to a 1:15 ratio ($X^2_{1:15} = 0.092$) for two independent dominant suppressors. However, the suppressors in SH40 and SH58 were shown to inherit recessively. This ratio, therefore, suggests an oligogenic system of independent recessive suppressor genes. If resistance in the F_2 progeny is considered relative to the response of the SH parents, the R:S ratio would be 48:94, which approaches a 7:9 ratio ($X^2_{7:9} = 5.71$) for two independent recessive suppressor genes.

DISCUSSION

Expression of resistance in SHs. The current data provide evidence for active suppression of stripe rust resistance by genes on the genomes of *A. squarrosa* and wild emmer wheat. Resistance genes located on one genome are inhibited by a suppressor(s) located on another genome. This phenomenon was observed previously in *xTritordeum*, triticale, and (amphiploid) wheat (2,4, 10,19,21,30,31,33–35). The emphasis of the present study is on the mode of action of suppressors, which is characterized by specificity. They operate toward specific resistance genes for specific races at specific growth stages, which implies the presence of a number of suppressor genes. This hypothesized specificity is strengthened by the fact that some wild emmer accessions carry single resistance genes (e.g., accession G148-1-2M, G. H. J. Kema, F. Kiriswa, and A. Badebo, *unpublished data*), hence race specificity of the responses of SHs involving this accession cannot be explained by a model with at least two genes for resistance and at least two complementary suppressor genes.

The resistant response of the majority of the SHs in the adult plant stage could be explained by either additional genes for adult plant resistance or nonfunctionality of suppressors in later growth stages. The operation of adult plant resistance genes for which suppressor genes do not have specific recognition evidently will result in a resistant response. The susceptible response of SH18 to race 66 E0 indicated that suppressors in *A. squarrosa* Cambridge L also were operational in mature plants. The resistant response of SH18 to race 6 E16, therefore, might be due to an adult plant resistance gene that is not suppressed rather than to nonfunctionality of suppressors in mature plants. Kerber and Green (21) also observed differences between seedling and adult plant tests for suppression of stem rust resistance. Specificity in the mode of action of suppressor genes to leaf and stem rust resistance genes also was discussed by Bai and Knott (2) but could not be examined due to the application of a single race of each rust species. Kerber and Green (21) demonstrated a dominant suppressor gene on chromosome 7D of cv. Canthatch, which was not operational to stem rust race C64, indicating its specific mode of action.

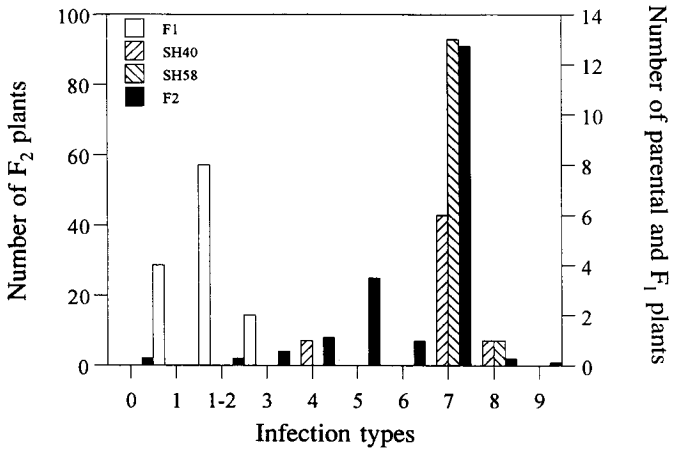

Fig. 2. Stripe rust infection types of race 6 E16 at 21 days postinoculation on synthetic wheat hexaploids SH40 and SH58 and the F_1 and F_2 populations derived from crosses between these hexaploids.

The current evidence for suppressor genes on the *T. t. dicoccoides* genome corresponds with that reported by Kerber (19), who demonstrated the presence of suppressor(s) on the AABB genome of cv. Tetra Canthatch. The suppressor neutralized the resistance of *A. squarrosa* RL5495 to four of nine leaf rust races in a SH, which also suggests a specific mode of action of the suppressors involved.

Genetics and possible mechanism of suppression. The suppressors in the *A. squarrosa* accessions Cambridge L and Rennes 33 are different and are inherited recessively, because the F_1 of crosses between SH40 and SH58 was resistant, and an identical factor would have resulted in a susceptible response. In contrast to the present data, the inheritance of suppressor genes to cereal rusts was reported to be dominant and was demonstrated by susceptible F_1 responses and additional tests with aneuploid stocks, which revealed equal effectiveness of homozygous, heterozygous, and hemizygous states of the loci involved (2,15,19,20,21,47).

The skewed distribution of ITs in the present F_2 can be explained by segregation of at least two independent recessive suppressor genes. The transgressive segregation for enhanced resistance levels in the F_2 is considered to be due to segregating suppressor genes, since segregation for A and B genome characters, including resistance genes, can be excluded from consideration, because both SHs have the same wild emmer parent (G148-1-2M). Both *A. squarrosa* accessions were susceptible to the race involved. The theoretical possibility that the susceptibility of these accessions is due to the suppressor genes they carry provides an alternative hypothesis for transgressive segregation that cannot be excluded, though it is improbable because there would be no selective advantage for plants neutralizing their own resistance. Cytogenetic imbalance in the SHs could have contributed to the development of certain phenotypes, and consequently could have influenced the segregation ratio, but this was not checked because their fertility is normal. The regular phenotypes and growth rates of the F_1 and F_2 progenies exclude the presence of nullisomic individuals. Therefore, cytological imbalance, if of any importance, would be restricted to monosomy, which would only affect the segregation ratio if the suppressor genes are located on the same chromosome. Nevertheless, special attention will be paid to this aspect in further research, and segregation ratios must be confirmed in F_3 tests and analyses of other crosses between SHs, which are currently under way.

In the present study, susceptible responses developed slowly in the seedling as well as in the adult plant stage and were only occasionally beyond IT 7. Suppression of resistance to leaf rust and stem rust, also rarely revealed ITs exceeding IT 3 on the 0–4 scale, but retarded development of ITs was not reported, and heterogeneous- and mesothetic-like responses were quite frequently observed (2,10,19,21,44). Experiments with C_1 seedlings confirmed the data obtained with C_2 seedlings, showing that susceptible ITs can develop from an initial resistant or intermediate response, which might imply that elicitation of resistance is restricted to specific stages of fungal development in the host. Williams et al (44) suggested that environmental fluctuation affected the expression of the stem rust resistance gene and, thus, of the suppressor gene involved in their study. The present data provide evidence that different modes of timing of suppressor and resistance genes (comparing responses at 16 and 21 DPI), rather than environmental fluctuations, are responsible for heterogeneous expression of the resistance genes.

The described results do not seem to comply with the basic ingredient of the gene-for-gene interaction, as discussed by Thompson and Burdon (37), since challenging resistance genes with avirulent races revealed compatible responses. However, this does not necessarily weaken the gene-for-gene hypothesis (6), nor does it require a modification of it as suggested by Knott (23). Tepper and Anderson (36) emphasized the importance of gene

regulation and its possible role in race-cultivar specificity rather than direct interaction between the products of avirulence and resistance genes, elicitors and receptors, respectively, even though it is a primary aspect of host-pathogen interaction models (6–9,11). Suppressors might be analogues of regulator products that control timing and expression of resistance.

Certainly suppressors of resistance do interfere with the expression of resistance genes transferred from alien species. They also could have a more general function in gene expression, which requires further research to elucidate their role in the expression of the hypersensitive response of wheat to cereal rusts.

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