Euphytica **70**: 151–157, 1993.

Resistance to Septoria nodorum blotch in several *Triticum* species

H. Ma[1] & G.R. Hughes
Department of Crop Science and Plant Ecology, University of Saskatchewan, Saskatoon, Saskatchewan, Canada, S7N 0W0; [1] present address: CIMMYT, Apartado Postal 6-641, CP. 06600, Mexico, D.F., Mexico

Received 6 April 1993; accepted 23 August 1993

Key words: interspecific hybridization, *Leptosphaeria nodorum, Septoria nodorum,* rate-reducing resistance, *Triticum* spp., wheat, durum wheat

Summary

Resistance to septoria nodorum blotch in *Triticum monococcum, T. tauschii, T. timopheevii, T. dicoccum* and *T. durum* was evaluated on plants at the three-leaf stage in greenhouse tests. A high frequency of resistant genotypes was found in *T. monococcum, T. tauschii* and *T. timopheevii,* but not in *T. dicoccum* and *T. durum.* The resistance of F_1 plants of crosses of resistant *T. monococcum* (PI 289599) and *T. timopheevii* (PI 290518) accessions with susceptible common wheat cv. Park and durum wheat cv. Wakooma, respectively, was evaluated on the basis of percentage leaf necrosis, lesion number, lesion size and incubation period. No dominance was found for long incubation period, but various dominance relationships occurred for low percentage leaf necrosis, low lesion number and small lesion size, depending on the cross. Multiple regression analysis showed that lesion number contributed more to percentage leaf necrosis than lesion size or incubation period. Resistance to septoria nodorum blotch was transferred successfully from *T. timopheevii* to cultivated durum wheat. Resistant BC_1F_7 lines, recovered from the *T. timopheevii* (PI 290518) × Wakooma cross, showed normal chromosome behaviour at meiosis (14 bivalents) and were self-fertile. However, an effective level of resistance was not recovered in lines derived from the other interspecific crosses.

Introduction

Septoria nodorum blotch of wheat (*Triticum aestivum* L.), caused by *Leptosphaeria nodorum* E. Müller (= *Phaeosphaeria nodorum* (E. Müller) Hedjaroude) (anamorph *Septoria nodorum* (Berk.) Berk. in Berk. & Broome = *Stagnospora nodorum* (Berk.) Castellani and E.G. Germano), has become a problem in many wheat growing regions of the world. Alone or in association with other *Septoria* species pathogenic on wheat, *S. tritici* Rob. ex Desm. (teleomorph *Mycosphaerella graminicola* (Fuckel) Schroeter) and *S. avenae* Frank f.sp. *triticea* T. Johnson (teleomorph *Leptosphaeria avenae* f.sp. *triticea* T. Johnson), this pathogen has caused significant yield losses in many countries (King et al., 1983). In Saskatchewan, this septoria complex is widespread (Bailey et al., 1992), and severe epidemics have occurred during the last decade when *L. nodorum* was the predominant casual species. Average annual yield losses caused by the septoria complex are estimated at 15% in the Parkland region of Saskatchewan (unpublished data). Increased use of early maturing, high yielding, semidwarf wheat cultivars has contributed to the septoria problem (Wiese, 1977). Agronomic practices such as greater use of nitrogen fertilizers, continuous wheat rotations and minimum tillage resulting in large amounts of infected stubble accumulating

on the soil surface may also promote development of epidemics (Eyal, 1981; Sumner et al., 1981).

Breeding for resistance is the most economical approach to disease control, but resistant cultivars adapted to Saskatchewan are not yet available. Progress in improving resistance to septoria nodorum blotch in cultivated wheat, especially in spring wheat, has been hindered by lack of usable genetic variability for resistance (King et al., 1983). Sources of resistance to septoria nodorum blotch have been reported in alien *Triticum* species (Krupinsky, 1972; Scharen & Eyal, 1980; Tomerlin et al., 1984), but successful transfer of the resistance identified to cultivated wheat was not indicated.

More work is needed to enlarge the pool of available sources of resistance to septoria nodorum blotch. Therefore, this study was undertaken to identify sources of resistance to *L. nodorum* in various *Triticum* species, to determine the expression of resistance in interspecific F_1 hybrids and to transfer resistance to cultivated durum and common wheat cultivars.

Materials and methods

Evaluation of germplasm

Plant material. A total of 109 accessions of diploid and tetraploid *Triticum* species, including *T. monococcum* L., *T. tauschii* (Coss.) Schmal., *T. timopheevii* (Zhuk.) Zhuk. and *T. dicoccum* Schubl. were studied. These accessions, obtained originally from the National Plant Germplasm System, U.S. Department of Agriculture, were supplied to the authors by Dr. A. Limin, Department of Crop Science and Plant Ecology, University of Saskatchewan. One hundred cultivated lines of *T. durum* Desf., representing genotypes from the USA, Mexico, Italy and Canada, were provided by Dr. D.R. Knott, Department of Crop Science and Plant Ecology, University of Saskatchewan.

Inoculation procedure. Three isolates of *L. nodorum*, cultures PW, BL, and Y-16, collected from different locations in Saskatchewan, were used as sources of inoculum. Single-pycnidial cultures of each isolate were grown on V8-juice agar (150 mL V8-juice, 1.5 g $CaCO_3$, 15 g agar and 850 mL H_2O) at 20 ± 2° C under continuous cool-white fluorescent light. A spore suspension, adjusted to $2–4 \times 10^6$ spores/mL, was prepared for each isolate from 14-day-old cultures. Plants were inoculated at the three-leaf stage using inoculum produced by mixing equal amounts of the spore suspension of each isolate. This inoculum, with Tween 20 surfactant added (0.4 mL/L), was sprayed onto the plant leaves with a hand-sprayer until runoff. After inoculation, the plants were placed in a mist-chamber for 48 h.

The seedling disease tests were conducted in a greenhouse with a 16 h photoperiod and a 18–24° C temperature range. Each test was arranged in a completely randomized design with replication varying with the availability of seed. The hexaploid spring wheat cv. Park was used as the susceptible control in all tests. Resistant genotypes were tested at least twice to confirm their resistant reaction.

Disease assessment. Disease severity was measured by percentage leaf necrosis (percentage leaf surface covered by visible lesions) 10 days after inoculation. Ratings for percentage leaf necrosis were based on a 0 to 5 scale: 0 = immune (leaf necrosis = 0%); 1 = highly resistant (leaf necrosis = 1–3%); 2 = resistant (leaf necrosis = 4–10%); 3 = intermediate (leaf necrosis = 11–20%); 4 = susceptible (leaf necrosis = 21–30%); and 5 = highly susceptible (leaf necrosis > 30%).

Interspecific hybridization

Two highly resistant accessions, *T. monococcum* (TM), PI 289599, and *T. timopheevii* (TT), PI 290518, were selected as resistant parents for the interspecific crosses on the basis of good crossability with cv. Park (Ma, 1988). These accessions, used as the female parent, were crossed with susceptible cvs. Park wheat and Wakooma durum, respectively. The F_1 hybrid plants of each cross were tested for resistance to septoria nodorum blotch using a completely randomized design with 10 replications and one plant per treatment. Incubation period and percentage leaf necrosis, lesion number and lesion size,

Table 1. Mean leaf necrosis rating and frequency of disease reactions of the *Triticum* species tested for resistance to *L. nodorum* at the three-leaf stage under greenhouse conditions.

Species	Genome	Frequency of disease reaction[a]			Mean leaf necrosis rating	Genotypes tested
		R	Int	S		
T. tauschii	D	17	1		1.6 ± 0.14	18
T. monococcum	A	37	4	1	1.8 ± 0.11	42
T. timopheevii	AG	15	1	1	1.7 ± 0.19	17
T. dicoccum	AB	12	10	10	3.0 ± 0.23	32
T. durum	AB	4	45	51	3.8 ± 0.21	100

[a] R = resistant (ratings 1 and 2); Int = intermediate (rating 3); S = susceptible (ratings 4 and 5).

10 days after inoculation, were measured. Lesion number and lesion size were rated using a 0 to 3 scale: 0 = nil; 1 = few lesions or small lesion size; 2 = intermediate lesion number or lesion size; and 3 = many lesions or large lesion size. Incubation period was recorded as days from inoculation to appearance of the first symptoms.

Table 2. Sources of resistance to *L. nodorum* identified in *T. tauschii, T. monococcum, T. timopheevii, T. dicoccum* and *T. durum* rated as highly resistant (rating 1) in greenhouse tests of plants at the three-leaf stage.

Genotype	Source	Genotype	Source
	T. tauschii		
CI 5	Afghanistan	RL 5534-3	Azerbaijan
CI 16	Iran	RL 5536-4	Azerbaijan
CI 18	Iran	RL 5548	Azerbaijan
CI 52		RL 5561	Azerbaijan
RL 5523	Azerbaijan		
	T. monococcum		
PI 167591	Turkey	PI 286068	Poland
PI 167621	Turkey	PI 289599	England
PI 190942	Spain	PI 289605	England
PI 221329	Yugoslavia	PI 355529	Switzerland
PI 221393	Yugoslavia	PI 355546	Switzerland
PI 254195	Turkey	CI 13963	USA
PI 272562	Hungary	CI 14090	USA
PI 277140	Germany		
	T. timopheevii		
PI 119442	Turkey	PI 352510	Switzerland
PI 289752	Australia	CI 11802	USA
PI 290518	Hungary	CI 14133	
PI 341802	USSR	CI 15590	Greece
	T. dicoccum		
PI 109024	Spain	PI 266842	England
PI 221403	Yugoslavia	CI 07042	Portugal
	T. durum		
Vernal (emmer wheat)		M 75	Mexico
S 76190	Canada	CPB 144	

Results and discussion

Evaluation of germplasm

Most accessions of *T. tauschii,* 37 of *T. monococcum* and 15 of *T. timopheevii* were rated as resistant (Table 1). Their mean leaf necrosis ratings were significantly lower (more resistant) than those of the *T. dicoccum* or *T. durum* accessions tested, where only 12 (38%) of the *T. dicoccum* and 4 (4%) of the *T. durum* accessions were resistant. No accessions with an immune reaction were identified. Those accessions which consistently rated 1 in repeated testing are listed in Table 2.

The diploid progenitors of common wheat and other related species have been recognized as good sources of disease resistance (Sprague, 1980). As this present study indicates, this is true also for resistance to *L. nodorum.* The high frequency of resistant genotypes in *T. tauschii* and *T. monococcum* indicates that both the A and the D genomes possess gene(s) for seedling resistance. Thus direct transfer of resistance to *L. nodorum* from these two species to common wheat appears feasible. A similar high frequency of resistant genotypes was found also for the *T. timopheevii* accessions, indicating that either the A or the G genome, or both, possess gene(s) for resistance. However, Frauenstein & Hammer (1985) found no genotypes resistant to *L.*

nodorum in diploid and tetraploid *Aegilop* species including *Ae. speltoides,* a suspected source of the G genome. This suggests that the resistance gene(s) in *T. timopheevii* is most likely present on the A genome. Similar findings were reported by Krupinsky et al. (1977), Jahier & Trottet (1980) and Tomerlin et al. (1984). Since the A genome of *T. timopheevii* is believed to be derived from *T. monococcum,* which is also the source of the A genome of *T. aestivum* (Kimber & Sears, 1987), it should be possible also to directly transfer the resistance identified in *T. timopheevii* to hexaploid wheat.

The lack of resistant genotypes in *T. dicoccum* and *T. durum,* which also possess the A genome, could be due to a low frequency of resistance genes in the sample tested. These two species were usually grown in low rainfall areas where septoria nodorum blotch would not normally be a major disease (Feldman & Sears, 1981). Thus, a low frequency of resistance genes in those populations could be expected as the result of low selective pressure imposed by low disease pressure. The presence of susceptible modifier genes or resistance inhibiting genes in the B genome would also result in a low frequency of resistant genotypes in these two species. Helena & Dzieglo (1985) reported evidence for a resistance inhibiting gene located on chromosome 5B in hexaploid winter wheat.

Resistance in F_1 hybrids

F_1 hybrid means for the resistance components measured fell either between the resistant parent and mid-parent values, or were similar to the resistant parent or mid-parent (Table 3). The mean lesion number of the F_1 hybrids of the crosses involving Park was significantly different (P = 0.05) from, and

Table 3. Mean percentage leaf necrosis (PLN), lesion number (LN), lesion size (LS) and incubation period (IP) of parents and F_1 hybrids tested with *L. nodorum* at the three-leaf stage under greenhouse conditions.

Genotype	Resistance component[a]			
	PLN (%)	LN[b]	LS[b]	IP (days)
		TM × Wakooma		
PI 289599 (TM)	2.0 *	0.5	0.5	9 **
Mid-parent	7.0 **	1.3 **	0.8 **	7
F_1	2.9	0.6	0.5	7
Wakooma	12.1 **	2.1 **	1.1 **	4 **
		TT × Wakooma		
PI 290518 (TT)	2.1 **	0.5 **	0.5	8 *
Mid-parent	7.1	1.3	0.8 *	7
F_1	4.6	0.8	0.5	5
Wakooma	12.1 **	2.1 **	1.1 **	4 **
		TM × Park		
PI 289599 (TM)	2.0 *	0.5 **	0.5	9 **
Mid-parent	10.0	1.3 **	0.8 **	6
F_1	6.3	0.9	0.6	6
Park	18.9 **	2.1 **	1.1 **	3 **
		TT × Park		
PI 290518 (TT)	2.1 **	0.5 **	0.5	8 *
Mid-parent	11.0	1.3 **	0.8 **	4
F_1	5.5	0.9	0.8	5
Park	18.9 **	2.1 **	1.1 **	3 **

[a] Leaf necrosis data were transformed using $x' = \log_{10}(x+1)$, and lesion number, lesion size and incubation period were transformed using $x' = (x+1)^{1/2}$ before statistical analysis.

[b] Based on a 0–3 scale where 0 = no lesions and 3 = many lesions or large lesion size.

*, ** Significantly different from the F_1 mean at P = 0.05 and 0.01, respectively, as determined by single degree of freedom contrasts.

fell between, the resistant parent and the mid-parent means. However, the mean lesion number of the TM × Wakooma F_1 was not significantly different (P = 0.05) from the resistant parent mean and that of the TT × Wakooma F_1 was not significantly different (P = 0.05) from the mid-parent mean. Thus low lesion number can behave as an incompletely dominant or dominant trait, or may show no dominance, depending on the parental genotypes.

For lesion size, three of the F_1 hybrids were significantly different (P = 0.05) from their mid-parent means, but not from their resistant parent means (Table 3). This indicated that small lesion size was dominant. F_1 means for incubation period were significantly different (P = 0.05) from the resistant, but not the mid-parent mean. Similar results were obtained for F_1 means for percentage leaf necrosis. The absence of dominance for incubation period and percentage leaf necrosis suggests these traits are determined by genes with additive effects.

Dominant, incompletely dominant, recessive and additive gene action for resistance have been reported in studies of resistance to septoria nodorum blotch in wheat (Frecha, 1973; Laubscher et al., 1966; Nelson & Gates, 1982; Wong & Hughes, 1989). The various dominance relationships are likely due to the use of different parental genotypes or disease assessment methods. For example, Frecha (1973) reported a dominant gene for low percentage leaf necrosis, but Wong & Hughes (1989) found that resistance was recessive when infection type was assessed on the basis of lesion size and the associated amount of chlorosis.

In this study, a host genetic background effect on dominance was suggested by the different dominance relationships observed for a resistance component, depending on the cross. Within a cross, different dominance relationships for the various components studied were also found. This may imply that the components of resistance are controlled by different genetic systems. Studies of components of resistance to *L. nodorum* suggested that percentage leaf necrosis could be interpreted as resistance to fungal toxin, lesion size as resistance to pathogen growth, and lesion number and incubation period as resistance to colonization by the pathogen (Jeger et al., 1983; Lancashire & Jones, 1985). These were considered to be independent factors controlling resistance to *L. nodorum.*

Incubation period was significantly and negatively correlated with lesion number (r = − 0.86), lesion size (r = − 0.82) and percentage leaf necrosis (r = − 0.93), while percentage leaf necrosis was significantly and positively correlated with lesion size (r = 0.88) and lesion number (r = 0.97). Thus, short incubation period, high lesion number and large lesion size will result in high percentage leaf necrosis.

The relationship between percentage leaf necrosis and lesion number, lesion size and incubation period was investigated further by multiple regression analysis. The regression equation obtained was:

$Y = -0.33 - 0.43X_1 + 1.39X_2 - 0.06X_3$; ($R^2$ = 98.5%)

where, Y = percentage leaf necrosis, X_1 = lesion size, X_2 = lesion number and X_3 = incubation period. Only the regression coefficient associated with lesion number was significant (P = 0.01). Lesion number had a greater effect on percentage leaf necrosis than did the other two components. Baker & Smith (1979) obtained similar results in a field study of hexaploid winter wheat. Therefore, low lesion number should be an important criterion when selecting for resistance to *L. nodorum* and studies are needed to determine the heritability of this trait.

Production of resistant lines

Hybrid seed set in the crosses TM (PI 289599) × Park, TT (PI 290518) × Park, TM (PI 289599) × Wakooma and TT (PI 290518) × Wakooma was 15%, 12%, 28% and 50%, respectively. Approximately 75% of the hybrid seed of the Park crosses and 100% of the Wakooma crosses was viable. Since the F_1 plants were completely male, and partially female, sterile, they were backcrossed with Park or Wakooma. The resulting seed set was very low and ranged from 1.4% to 5.2% with many BC_1 seeds possessing non-viable embryos (characteristically torpedo-shaped) and highly shrivelled endosperm. These seeds rarely germinated even when embryo rescue was attempted. However, embryo rescue of shrivelled seeds with normal-shaped embryos was successful.

BC_1F_1 plants of each cross segregated for resistance. Since all BC_1F_1 plants of the crosses involving Park were self-sterile, the resistant plants were backcrossed to Park. However, most BC_1F_1 plants of the Wakooma crosses were partially self-fertile and BC_1F_2 families were produced from resistant BC_1F_1 plants.

Selection for seedling resistance, based on low percentage leaf necrosis, and for self-fertility in each family continued until the F_5 generation in all crosses. In the BC_1F_6 generation of the TT × Wakooma cross, seed from resistant plants in a family were bulked and 16 BC_1F_7 lines were produced. Of these, 15 lines were homozygous for a level of resistance similar to that of the *T. timopheevii* parent, produced 14 bivalents at meiosis and had 70 to 90% seed set when selfed. Preliminary results from field disease nurseries indicate that these lines possess a good level of resistance to septoria nodorum blotch at the adult-plant stage (unpublished data). Three randomly selected lines were crossed with Wakooma and *T. dicoccum* (PI 266842). Fourteen bivalents were observed at meiosis of the F_1 plants, indicating that these resistant lines possess all AB-genome chromosomes (Ma, 1993). Attempts to recover homozygous lines with the parental level of resistance from the other crosses was not successful.

The success or failure of interspecific hybridization to directly transfer alien genes depends on at least four distinct factors, crossability, germination and subsequent survival of the hybrid, the fertility of the hybrid plants and the frequency of desirable hybrid plants. Failure to obtain resistant families from both *T. monococcum* crosses and the *T. timopheevii* × Park cross was due mainly to the low hybrid production rate (i.e. % hybrid seed set × % culturable embryos × % green plants obtained). This rate for crosses *T. monococcum* × Park (5.0% in F_1 and 0.6% in BC_1), *T. timopheevii* × Park (6.7% in F_1 and 0.6% in BC_1) and *T. monococcum* × Wakooma (11.7% in F_1 and 0.6% in BC_1) was much lower than for the cross *T. timopheevii* × Wakooma (30.5% in F_1 and 2.6% in BC_1) because of the very low production of F_1 and BC_1 hybrid seeds. In addition, a low number of bivalents (mean 5.7) was observed at meiosis in the F_1 hybrids of crosses *T. monococcum* × Park and *T. monococcum* × Wakooma. This low frequency of chromosome pairing would reduce the chance of obtaining resistant segregants from the crosses involving *T. monococcum* and thus hinder the recovery of homozygous resistant families. Reduced pairing between A genome homologues in F_1 hybrids of *T. monococcum* × *T. durum* and *T. monococcum* × *T. aestivum* has been reported (The & Baker, 1975).

Creation of resistant durum wheat lines has two advantages for breeding for resistance to septoria nodorum blotch. They can be used directly as sources of resistance in durum wheat breeding programs and, indirectly, as 'bridge' sources of resistance in common wheat breeding. Identification of seedling resistance in *T. tauschii* indicates that other gene(s) for resistance exist in the D genome. This provides the opportunity to produce resistant synthetic hexaploid wheat lines by crossing the resistant durum lines with resistant *T. tauschii* accessions. These synthetics would contain combinations of different resistance genes and may represent sources of resistance superior to those based on resistance from a single genome.

Acknowledgements

The authors wish to acknowledge the financial support received from the Hantleman Trust and the Ada M. Graham Estate, University of Saskatchewan, and from the Saskatchewan Agriculture Development Fund, Canada.

References

Baker, E.A. & I.M. Smith, 1979. Components of *Septoria nodorum* infection in winter wheat: Lesion number and lesion size. Trans. Br. Mycol. Soc. 73: 57–63.

Bailey, K.L., L.J. Duczek, M.R. Fernadez, G.R. Hughes, D. Kaminski, C. Kirkham, K. Mortenson & S. Boyetchko, 1992. Saskatchewan wheat disease survey, 1991. Can. Plant Dis. Surv. 72: 62–63.

Eyal, Z., 1981. Integrated control of *Septoria* diseases of wheat. Plant Dis. 65: 763–768.

Feldman, M. & E.R. Sears, 1981. The wild gene resources of wheat. Sci. Am. 244: 102–112.

Frauenstein, K. & K. Hammer, 1985. Testing of *Aegilops* species for resistance to powdery mildew (*Erysiphe graminis* DC.),

brown rust (*Puccinia recondita* Rob. ex. Desm.) and glume blotch (*Septoria nodorum* Berk.). Kulturpflanze 33: 155–163 (Engl. Abst.).

Frecha, J.H., 1973. The inheritance of resistance to *Septoria nodorum* in wheat. Bol. Inst. Fitotec. Castelar 8: 29–30.

Helena, W. & Dzieglo, 1985. Monosomic localization of genes that are resistant to *Septoria nodorum* (Berk.) in summer wheat (*Triticum aestivum* L.) variety Sappo. Hodowla Rosl. Aklim. Nasienn. 29: 11–20 (Engl. Abstr.).

Jahier, J. & M. Trottet, 1980. Consequences of an attack of *Septoria nodorum* on the accumulation of dry matter in grain of an accession of *Aegilops squarrosa.* Cereal Res. Comm. 8: 325–330.

Jeger, M.J., D.G. Jones & E. Griffiths, 1983. Components of partial resistance of wheat seedlings to *Septoria nodorum.* Euphytica 32: 575–584.

Kimber, G. & E.R. Sears, 1987. Evolution in the genus *Triticum* and the origin of cultivated wheat. p. 154–164. In: E.G. Heyne (Ed). Wheat and Wheat Improvement. Am. Soc. Agron. Madison, Wisconsin, USA.

King, J.E., R.J. Cook & S.C. Melville, 1983. A review of *Septoria* disease of wheat and barley. Ann. Appl. Biol. 103: 345–373.

Krupinsky, J.M., 1972. Resistance in wheat to *Septoria nodorum.* Crop Sci. 12: 528–530.

Krupinsky, J.M., J.C. Craddock & A.L. Scharen, 1977. *Septoria* resistance in wheat. Plant Dis. Reptr. 61: 632–636.

Lancashire, P.D. & D.G. Jones, 1985. Components of partial resistance to *Septoria nodorum* in winter wheat. Ann. Appl. Biol. 106: 541–553.

Laubscher, F.X., B. Von Wechmar & D. Von Schalkwyk, 1966. Heritable resistance of wheat varieties to glume blotch (*Septoria nodurum* Berk.). Phytopathology 56: 260–264.

Ma, H., 1988. *Septoria nodorum* resistance in wild *Triticum* species and its transfer to cultivated tetraploid and hexaploid wheat. M.Sc. thesis. Univ. of Saskatchewan, Saskatoon, Canada. p. 137.

Ma, H., 1993. Genetic and cytogenetic studies of resistance to *Septoria nodorum* in tetraploid and hexaploid wheat. Ph.D. thesis. Univ. of Saskatchewan, Saskatoon, Canada. p. 144.

Nelson, L.R. & C.E. Gates, 1982. Genetics of host plant resistance of wheat to *Septoria nodorum.* Crop Sci. 22: 771–773.

Scharen, A.L. & Z. Eyal, 1980. Measurement of quantitative resistance to *Septoria nodorum* in wheat. Plant Dis. 64: 492–496.

Sprague, G.F., 1980. Germplasm resources of plants: Their preservation and use. Ann. Rev. Phytopathol. 18: 147–165.

Sumner, D.R., B. Doupnik Jr. & M.G. Boosalis, 1981. Effects of reduced tillage and multiple cropping on plant diseases. Ann. Rev. Phytopathol. 19: 167–187.

The, T.T. & E.P. Baker, 1975. Basic studies relating to the transference of genetic characters from *T. monococcum* L. to hexaploid wheat. Aust. J. Biol. Sci. 28: 189–199.

Tomerlin, J.R., M.A. El-morshidy, J.G. Moseman, P.S. Baenziger & G. Kimber, 1984. Resistance to *Erysiphe graminis* f.sp. *tritici, Puccinia recondita* f.sp. *tritici,* and *Septoria nodorum* in wild *Triticum* species. Plant Dis. 68: 10–13.

Wiese, M.V., 1977. Compendium of wheat diseases. Am. Phytopathol. Soc., St. Paul MN. p. 106.

Wong, L.S.L. & G.R. Hughes. 1989. Genetic control of seedling resistance to *Leptosphaeria nodorum* in wheat. p. 136–137. In: P.M. Fried (Ed). Septoria of Cereals. Proc. Workshop, July 4–7, 1989. Zurich, Switzerland.