INHERITANCE OF RESISTANCE TO SEPTORIA LEAF BLOTCH IN *TRITICUM AESTIVUM* CV. CARIFEN 12

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CHAPTER I

INTRODUCTION

Septoria leaf blotch of wheat, also called speckled leaf blotch, is caused by Septoria tritici Rob. ex Desm. The disease has been reported in more than 50 countries of the world (31). Mature lesions on infected leaves and sheaths of susceptible plants are tan in color and contain numerous light to dark brown pycnidia, hence, the name speckled leaf blotch (37). The disease is often considered to be of minor importance, but serious sporadic epiphytotics develop whenever excessive rains occur during the growing season (30).

In Oklahoma, the disease appears on seedling leaves in late fall, or winter, if moderately warm temperatures and excessive rainfall prevail. Severely infected seedlings are killed prematurely or are predisposed to winter injury. Consequently, both winter grazing potential and grain yield are lowered (4, 22).

Several methods of controlling the disease have been recommended. The application of chemicals give control when appropriately applied. In Illinois, Jacobsen (15) reported that the infection level of flag leaves by S. tritici was reduced 40% when mancozeb (1.65 kg/h), or mancozeb (1.1 kg/h) + benomyl (0.55 kg/H) were sprayed on the cultivar Arthur 71 in the heading stage and again 10 days later.

In Israel, Eyal (8) reported that either maneb (2.8 kg/h), or a powder mixture of 6% maneb + 70% sulfur (30 kg/h) applied to the spring

wheat cultivar 8828-221 increased total yield by 17.3% and 1,000 kernel weight by 15.7%. Although chemical applications successfully control the disease, they may be uneconomical when the cash value of the crop is low.

Also, a number of cultural practices, aimed at reducing the main source of primary inoculum, have been suggested for control of septoria leaf blotch. These are crop rotation, sanitation, deep ploughing, burning the stubble, and destroying volunteer wheat. But these control measures are considered to be uneconomical and only partially effective under the present cropping systems and over large areas (31).

Use of resistant cultivars is the most economical method of controlling the disease, and several sources of resistance are known. At the present time, however, only one cultivar, Oasis, has been developed and released in the United States specifically for its resistance to speckled leaf blotch (20). Oasis is a soft red winter wheat that is not adapted to the Southern Great Plains.

The purpose of this study was to determine the number of genes which condition resistance to S. tritici in the cultivar Carifen 12 of Triticum aestivum L., an introduction from Chile, and to evaluate the resistance for use in breeding new cultivars.

CHAPTER II

LITERATURE REVIEW

S. tritici was first recorded on wheat in 1842 by Desmazieres in France (37). According to Weber (37) Desmazieres presented a complete description of the fungus and the disease.

The perfect stage of the organism was not identified until 1972 when Sanderson (27) reported that a Mycosphaerella sp. on wheat was the sexual stage of S. tritici. Later he (28) named the ascogenous state Mycosphaerella graminicola (Fuckel) Sanderson comb. nov., and cited a report that the sexual stage had been discovered in the United Kingdom and Australia.

The host range of S. tritici is narrow. Weber (37) reported that isolates from wheat, rye, and Kentucky blue grass were able to infect a number of Triticum spp., rye and blue grass when he cross inoculated the plants. However, Hilu and Beaver (14) were able only to obtain infection on wheat, with isolates from wheat, when they inoculated nine genera of the Gramineae. Arsenijevic (1) also showed that of 18 grasses inoculated with S. tritici only wheat was susceptible. But Sprague (32) obtained infection on Poa secunda Presl. and flecks on P. annua L. These results indicated that the host range of S. tritici is either restricted to wheat or to a very few genera of Gramineae.

Only a few attempts have been made to identify physiologic races of the fungus. Morales (18) examined 4 cultures of S. tritici from

different localities in the United States, but could not demonstrate the existance of physiological races on 16 wheat cultivars. Shipton et al. (31) tested the reaction of 28 Western Australian isolates on five wheat cultivars, but observed no physiologic specialization. Eyal et al. (9) claimed evidence for physiological races based on differential production of pycnidia among five isolates on 14 wheat cultivars in the greenhouse. But there is no clear evidence that races really exist in the field because S. tritici is highly dependent upon weather conditions for its development.

According to Weber (37), the disease was first reported in the United States by Pammel in 1901, and since that time, it has been found in every wheat growing region of the country. Dickson (6) noted that speckled leaf blotch occurs over a wide area of the hard red and soft red winter regions, and that it occurred more consistantly than glume blotch. According to Sprague (33), the disease was prevalent in the humid areas of Oregon, Washington, Northern California, and sometimes in the midwestern and eastern states.

In Oklahoma, epiphytotics, or near epiphytotics, have been reported for the years 1941, 1952, and 1974-1975, by Chester (4), Wadsworth and Young (36), and Gough and Smith (11), respectively. Chester (5) reported that S. tritici was able to destroy leaf rust infected wheat leaves faster than the rust could infect new ones. Thus S. tritici reduced the level of wheat leaf rust infections. He suggested that at times septoria leaf blotch is beneficial because it destroys hazardous leaf rust infected wheat leaves, but he also recognized the epidemic capacity of the disease. Wadsworth and Young (36) reported that 70 to 90% of the flag leaves were destroyed by the time of maturity in 1952.

Gough and Smith (11) reported foliage losses of up to 80% in wheat seedlings (rosette stage) in fields of North Central Oklahoma in the winter and spring of 1974-1975.

Rosen (25) reported that wheat acreage in Arkansas declined during the decade of 1937-1947 because of the occurrence of leaf rust, glume blotch, and speckled leaf blotch. Atkins (2) noted that loss of grain yield caused by Septoria was about 4,217,550 bushels in Texas in 1950.

In Indiana, Caldwell and Narvaez (3) reported that in 1957 infection was so severe that it destroyed all leaves shortly after flowering. This led to a reduction in head size, straw strength, and premature ripening. Grain yield comparisons were made among naturally infected plots, artificially inoculated plots, and fungicide sprayed check plots. In naturally infected plots, grain yield was reduced 21.1 to 27.6% and bushel weight was reduced by 1.5 to 3.7 pounds. Artificially inoculated plots showed losses of 25-44.5% in grain yield and 3.1-5.8 pounds in bushel weight.

Heavy losses have been observed since 1939 in Argentina where epidemics occurred in 1943-1944 (31). Schieber and Fumagalli (29) reported that speckled leaf blotch is the most serious disease of wheat in Guatemala, especially in areas at altitudes ranging from 6,000 to 9,000 feet (1,829-2,743 m) and in fields under irrigation.

Eyal et al. (10) reported that the disease has been endemic in Israel during the last 30 years, but severe epiphytotics occurred after the introduction of high yielding, semi-dwarf Mexican varieties which were susceptible to S. tritici. Under the epidemic situation, susceptible wheats showed losses of up to 40.4% in yield.

The disease also has been considered a major problem of wheat in

the coastal regions of the Mediterranean Sea, and in the North and North Eastern part of Africa where annual rainfall exceeds 700 mm. According to Stewart et al. (34) epidemics developed in Morocco, Tunisia, and Turkey in 1968-1969. Severely damaged fields of Northern Morocco, which had a potential yield of 4,000 kg per hectare produced only 500 to 800 kg per hectare. The sudden outbreaks of S. tritici derived from the replacement of tolerant or resistant old local cultivars with susceptible Mexican ones and to wet weather conditions favorable for development of the disease.

In New Zealand and Australia, the disease has been causing moderate or heavy losses for many years (31).

Data on foliage and grain yield losses caused by S. tritici is hard to measure in the field because epiphytotics occur sporadically. However, in greenhouse and growth chamber tests, Gough and Merckle (12) reported that artificially inoculated young winter wheats (3rd and 4th leaf stages) not only reduced the average foliage yields by 19% but also retarded root development by an average of 47%. In Australia, Shipton et al. (31) reported that 12.5% losses in grain yields were caused by moderate infection of S. tritici. In a field test, Eyal (8) estimated that pycnidial coverage of 50% of the upper 5 leaves reduced grain yield approximately 20%.

A number of sources of resistance to speckled leaf blotch have been identified (11, 14, 16, 22, 25, 26, 35, 37), but only a few articles have been published on the inheritance of resistance.

The first study of the inheritable resistance of wheat to S. tritici was reported by Mackie (17). He developed resistant wheat by repeatedly crossing the most resistant selections of hybrid stocks. Later,

these resistant wheats were crossed with susceptible wheats. From these studies, he concluded that resistance was inherited as a single recessive gene.

Narvaez and Caldwell (19) reported that resistance in the winter wheat cultivar Nabob was governed by two independent partially dominant genes with additive effects in crosses with susceptible cultivars Knox and Vermillion. But a single dominant gene conditioned resistance in spring wheats Lerma 52 and P14 in crosses with susceptible Lee and Mayo 54.

Rillo and Caldwell (23) reported that resistance to S. tritici in Bulgaria 88 was controlled by a single dominant gene when three susceptible cultivars were used as parents. In the F_1 and F_2 generations, high initial resistance of heterozygotes declined towards intermediacy as the plants aged. Resistance of the homozygotes did not change.

Rillo et al. (24) found a high level of resistance in a wheat-Agropyron elongatum derivative (Purdue 39120A4), which had chromosome numbers ranging from 52 to 56. He crossed plants of the wheat-A. elongatum derivative with a susceptible sib selection of the hexaploid wheat cultivar Riley. The F_1 plants from reciprocal crosses showed intermediate reactions. The F_2 families segregated in a ratio of seven resistant to nine susceptible. Backcrossed F_1 plants segregated in a ratio of one resistant to three susceptible, which indicated that resistance was on a single Agropyron chromosome transmitted 25% of the time through the male and female gametes.

Gough and Tuleen (13), screened seven lines of Chinese Spring wheat, each of which carried a different pair of chromosomes from A. elongatum (2n=14) in addition to the normal 21 wheat pairs. High

levels of resistance were observed in lines with A. elongatum chromosomes I and VII, while Chinese Spring and the remaining addition lines were highly susceptible.

CHAPTER III

MATERIALS AND METHODS

The wheat (T. aestivum) cultivar Carifen 12 from Chile has shown field resistance to S. tritici in experimental nurseries in Oklahoma when commonly grown cultivars such as Triumph 64 and TAM W 101 have been susceptible (11).

Seed of winter wheat cultivars, Carifen 12, Triumph 64 and TAM W 101 were obtained from the Department of Agronomy, Oklahoma State University. In the spring of 1976, Carifen 12 was used as the female parent in crosses with Triumph 64 and TAM W 101. In early November of 1976, ten F_1 seed from each cross were sown individually in soil filled plant bands (3.5 x 3.5 x 6.5 cm) contained in a common plant flat. The seedlings were grown in the greenhouse until the second leaf appeared, then moved outdoors to a cold frame to vernalize. Six weeks later vernalized plants were moved back to the greenhouse and transplanted singly to 15 cm clay pots. The plants were kept in the greenhouse until the seeds were harvested.

In the fall of 1977, seed of parent cultivars, the F_1, and the F_2 were sown in soil in plant bands (one seed per band) set inside deep (15 cm) plant flats. The plants were grown to maturity in the flats. The deep flats were improvised by "double-layering" ordinary plant flats (7.5 cm). For the first, or lower layer, a common flat was filled to capacity with soil. Side frames of a second flat were then

placed on the first and secured in place by nailing wood lath over the line of contact along the sides and ends of the two. One hundred and thirty plant bands were placed inside the frame of the second (upper) flat and filled with soil. Twenty days after planting, the emerged plants in the flats were placed in the cold frame where the temperature often dropped to near 0° C at night. After 43 days, the plants were returned to the greenhouse where the temperature fluctuated between 10 and 30° C. Just prior to emergence of the flag leaves, the parent cultivars, the F_1 and the F_2 were inoculated with a mixture of conidia from two cultures of S. tritici.

During the fall of 1978, 13 plants of each of 110 randomly selected F_3 families from Carifen 12 x Triumph 64 and Carifen 12 x TAM W 101 were tested for reaction to S. tritici. Also, 13 or more plants of each parent cultivar were tested. As in the F_2 tests, the plants were grown in plant bands in flats. Since it was not intended to advance tested plants to the F_4, the flats were not double-layered, and the plants were not vernalized. The plants were grown in the greenhouse until the third leaf was fully extended, then they were inoculated with a mixture of conidia from the same cultures used to test the F_2.

One of the two cultures (MT-5) used in the inoculum was obtained from A. L. Scharen (SEA, USDA, Plant Pathology, Montana State University, Bozeman, MT 59715). It grew principally by budding to produce a pink yeast-like mass of conidia on yeast extract-malt extract agar. The second culture (ST-22) was isolated from a single pycnidium in an infected wheat plant grown near Stillwater, Oklahoma. Culture ST-22 produced both conidia and mycelium on the yeast extract-malt extract agar.

The method of preparing and applying the inoculum was essentially

the same for both the F_2 and F_3 tests. The cultures were grown singly in 250 ml flasks containing 25 ml of liquid medium [2 g of yeast extract (U. S. Biochemical Corp., Cleveland, OH 44128) and 4 g of malt extract (Difco Laboratories, Detroit, MI 48232) per liter of distilled water] for 8-10 days at a room temperature of 20-25° C. During the incubation period they were shaken once or twice daily either manually or by a powered shaker. Conidia were harvested by straining the liquid medium through a triple layer of cheese cloth. After straining, the conidia from the two cultures were combined on an equal medium-volume basis to produce the inoculum. Counts using a hemacytometer indicated that, on the average, cultures MT-5 and ST-22 produced, respectively, 8.5×10^6 and 2.4×10^6 conidia per ml. of medium used in the F_2 test, and 10.75×10^6 and 5.75×10^6 conidia per ml. of medium used in the F_3 test. Non-flavored gelatin (0.5 g dissolved in 25 ml of warmed distilled water) was added to each 100 ml of inoculum to facilitate adherence of the conidia to the leaf surfaces (19). The plants were sprayed 3 times with inoculum at 24 hour intervals. Each time, 125 ml of inoculum was sprayed onto the plants (four flats containing 130 plants each) with a Devilbiss atomizer using about 5 psi of air pressure. The plants were then kept wet in a moisture chamber (constructed on a greenhouse bench and consisting of an opaque polyethylene film supported by a metal frame) for 96 hr by a time clock-controlled mist blower. The temperature in the chamber varied from 15 to 30° C. Supplemental light was not provided.

The plants were classified for reaction to S. tritici 20 days after inoculation.

The chi-square (χ^2) tests were used for analyzing data from the segregating population.

CHAPTER IV

RESULTS

Reaction of the Parents and F_1 Plants

Small chlorotic lesions, less than 1 mm, were first observed on the inoculated leaves of Triumph 64 and TAM W 101 10-12 days after inoculation. Ten to 17 days after inoculation, the lesions had become elliptical in shape, enlarged to about 3-10 mm in length, and contained pycnidia in their necrotic centers. Often, within 20 days of inoculation the lesions had coalesced to form irregular patterns of chlorosis and necrosis containing numberous pycnidia. No lesions developed in leaves of Carifen 12 or the F_1 plants. However very small chlorotic flecks, less than 0.5 mm, were observed on Carifen 12 and the F_1 plants, but it was not determined whether they were caused by S. tritici or by environmental conditions. The absence of lesion development in the F_1 plants indicated that resistance of Carifen 12 to cultures MT-5 and ST-22 of S. tritici was conditioned by one or more dominant genes.

Reaction of F_2 and F_3 Populations in Carifen 12 x Triumph 64 Crosses

The segregations obtained for S. tritici reaction among F_2 plants and F_3 families of Carifen 12 x Triumph 64 crosses are given in Table I. Of 159 F_2 plants from three F_1 plants of Carifen 12 x Triumph 64, 52

TABLE I

SEGREGATION FOR RESISTANCE TO *SEPTORIA TRITICI* IN THE F_2 PLANTS AND F_3 FAMILIES FROM RESISTANT CARIFEN 12 X SUSCEPTIBLE TRIUMPH 64 CROSSES[a]

		Observed number of plants				Expected number of plants			Values	
Generation[b]		Resistant	Intermediate (F_2) or Segregating (F_3)	Susceptible	Total	Resistant	Intermediate (F_2) or Segregating (F_3)	Susceptible	χ^2 Goodness of fit (1:2:1)	P
F_2	I	14	22	9	45	11.25	22.50	11.25	1.133	0.5-0.7
	II	23	27	15	65	16.25	32.50	16.25	3.831	0.1-0.2
	III	15	25	9	49	12.25	24.50	12.25	1.487	0.3-0.5
	Total (F_2's)	52	74	33	159	39.75	79.50	39.75	5.301	0.05-0.10
	Heterogeneity χ^2								1.152	0.8-0.9
F_3	I	1	14	5	20	5	10	5	4.8	0.05-0.1
	II	6	18	8	32	8	16	8	0.75	0.5-0.7
	III	7	12	1	20	5	10	5	4.4	0.1-0.5
	Total (F_3's)	14	44	14	72	18	36	18	3.56	0.1-0.2
	Heterogeneity χ^2								6.39	0.1-0.2

[a]Approximately one-half of the F_3 families derived from tested F_2 plants were randomly selected for testing.

[b]Roman numerals indicate different F_1 plants from which the tested F_2 plants and subsequent F_3 families derived.

were as resistant as Carifen 12 and the F_1 plants, 74 developed small chlorotic spots or necrotic lesions which in some cases contained a few pycnidia, and 33 developed large coalesing lesions with numerous pycnidia similar to Triumph 64. The value of chi-square for pooled segregation data on the F_2 plants was 6.45 with a probability level of 0.30-0.50. This indicated a good fit to a 1:2:1 ratio expected for a monogenic incompletely dominant mode of inheritance. The chi-square test for homogeneity (P between 0.80 and 0.90) indicated that the three F_2 families may have been samples of the same population.

The test of the 72 F_3 families from 159 F_2 plants confirmed the F_2 results. Of 72 F_3 families tested, 7 were homozygous resistant, 44 were segregated resistant and susceptible plants, and 14 were homozygous susceptible. The pooled value for chi-square was 9.95 with a probability between 0.10 and 0.20. The chi-square test for homogeneity of the three F_3 families gave a value of 6.39 with a probability level between 0.1 and 0.2 indicating the three families could have belonged to the same population.

Reaction of F_2 and F_3 Populations in Carifen 12 x TAM W 101 Crosses

The results of testing 387 F_2 plants and 38 F_3 families from five Carifen 12 x TAM W 101 crosses were similar to those of Carifen 12 x Triumph 64 relative to reaction types and segregation ratios. These results are presented in Table II. Among 387 F_2 plants from Carifen 12 x TAM W 101, 89 were highly resistant, 199 were intermediate to resistance and susceptibility, and 99 were susceptible. The pooled value of F_2 families for chi-square was 8.31 with a probability be-

TABLE II

SEGREGATION FOR RESISTANCE TO SEPTORIA TRITICI IN THE F_2 PLANTS AND F_3 FAMILIES FROM RESISTANT CARIFEN 12 X SUSCEPTIBLE TAM W 101[a]

	Observed number of plants				Expected number of plants			Values	
Generation[b]	Resistant	Intermediate (F_2) or Segregating (F_3)	Susceptible	Total	Resistant	Intermediate (F_2) or Segregating (F_3)	Susceptible	χ^2 Goodness of fit (1:2:1)	P
F_2 I	17	30	19	66	16.5	33.0	16.5	0.667	0.7-0.8
II	14	68	27	89	22.25	44.5	22.25	4.348	0.1-0.2
III	12	16	10	38	9.5	19.0	9.5	1.033	0.5-0.7
IV	18	48	21	87	21.75	43.50	21.75	1.139	0.5-0.7
V	28	57	22	107	26.75	53.50	26.75	1.130	0.5-0.7
Total (F_2's)	89	199	99	387	96.75	193.5	96.75	0.800	0.5-0.7
Heterogeneity χ^2								7.517	0.3-0.5
F_3 (I,II,III,IV,V)	5	23	10	38	9.5	19	9.5	2.999	0.2-0.3

[a]Approximately 1/10 of the F_3 families derived from tested F_2 plants were randomly selected for testing.

[b]Roman numerals indicate different F_1 plants from which the tested F_2 plants and subsequent F_3 families derived.

tween 0.50 and 0.70. These observed ratios were a good fit to a 1:2:1 ratio expected for resistance conditioned by a single incompletely dominant gene. The chi-square test for homogeneity indicated that distribution of reaction types among plants derived from the five F_1 plants was homogeneous (P between 0.3 and 0.5) and that pooling of the data was justified.

Of 38 randomly selected F_3 families from five F_2 plants, five were homozygous resistant, 23 segregated resistant and susceptible plants, and ten were homozygous susceptible. The value for chi-square was 2.99 with a probability between 0.2 and 0.3. These data supported the hypothesis derived from the preceding tests that resistance of Carifen 12 was conditioned by a single gene.

CHAPTER V

DISCUSSION

The reaction of the resistant parent cultivar, Carifen 12, was easily distinguished from reactions of the susceptible parent cultivars, Triumph 64 and TAM W 101. Carifen 12 typically developed no lesions at all or very small ones at the tips of a few leaves. Triumph 64 and TAM W 101 always developed large spreading lesions containing numerous pycnidia. Also, the reaction of F_1 plants presented no classification problem because it was very similar to the reaction of Carifen 12.

Difficulty with reaction classifications were encountered in the segregating populations, because some plants developed large lesions without pycnidia while others developed small- to medium-sized lesions with a few pycnidia. The reaction of these plants were classified as intermediate to the reactions of Carifen 12 and either Triumph 64 or TAM W 101. Also, the reaction of some plants classified first as highly resistant tended to resemble the intermediate class when observed 5 to 10 days later. Although the reactions of individual plants were not always discreet, occasional classification errors of F_2 plant reactions (indicated by reactions of direct descendent F_3 families) did not significantly affect the fit of the F_2 segregation to a 1:2:1 ratio.

The data derived from testing the F_2 and F_3 generations of the crosses strongly indicated that resistance of Carifen 12 was conditioned

by a single incompletely dominant gene. By contrast, the reaction of the F_1, by virtue of its similarity to the reaction of Carifen 12, indicated that resistance was completely dominant. I have no firm explanation for this inconsistency. However, it is suggested that an unknown number of modifier genes present in Carifen 12, but absent from Triumph 64 and TAM W 101, enhances expression of the single major gene for resistance. Assuming that the modifer genes are effective in the heterozygous state, then the reaction of the F_1 would be expected to resemble the reaction of Carifen 12. But in the F_2, segregation would produce genotypes having in common the major gene for resistance but varying in numbers of genes that reinforce its expression. The concept that dominance of resistance can depend upon genetic background has been demonstrated unequivacally for resistance to leaf rust (7) and stem rust (21) of wheat.

The observation that the highly resistant reactions of some plants retrogressed to an intermediate level with time is not without precedent. Rillo and Caldwell (24), studied the inheritance of resistance of the wheat cultivar Bulgaria 88 to S. tritici and noted that "resistant heterozygotes declined towards intermediacy 7-10 days after the first reading, whereas that of homozygotes was maintained". At this time, it is not known why the resistance of wheat leaf tissue decreases as the leaves mature and senesce.

The fact that resistance of Carifen 12 is inherited monogenically is encouraging from the standpoint of breeding resistant wheats adapted to Oklahoma, because a single gene usually can be transferred easily by backcrossing. Neither physiologic races, nor the sexual stage of S. tritici have been shown to exist in the United States. Consequently,

the effectiveness of monogenic resistance may be expected to endure for a longer period of time than that of more specialized pathogens such as the rusts and mildews.

CHAPTER VI

SUMMARY

1. The wheat (*Triticum aestivum* L.) cultivar Carifen 12 from Chile has shown field resistance to *S. tritici* in experimental nurseries in Oklahoma.
2. Carifen 12 was crossed with susceptible winter wheat cultivars, Triumph 64 and TAM W 101.
3. F_1, F_2, and F_3 populations from both crosses were grown in a greenhouse and inoculated with a mixture of conidia from two cultures of *S. tritici*.
4. In both crosses, reaction of the F_1 plants showed a high level of resistance, but F_2 plants segregated into three groups, highly resistant, intermediatly resistant, and susceptible. F_3 families segregated into three groups, homozygous resistant, segregating resistant and susceptible plants, and homozygous susceptible. These F_2 and F_3 segregations were both statistically good fits to 1:2:1 ratios.
5. The F_2 and F_3 reactions indicated that resistance to *S. tritici* in Carifen 12 was conditioned by a single incompletely dominant gene.

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