

Inheritance of Resistance to *Stagonospora nodorum* Leaf Blotch in Kansas Winter Wheat Cultivars

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ABSTRACT

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Stagonospora nodorum blotch can cause serious yield and quality losses of wheat (*Triticum aestivum*) in many countries worldwide. Although there are other control methods, host resistance is the most desirable. Three recent Kansas winter wheat cultivars (Betty, Heyne, and 2163) have been developed with moderate levels of resistance to the leaf phase of *Stagonospora nodorum* blotch. To determine inheritance of resistance and allelism, these cultivars were crossed with one of three susceptible lines (Larned, KS96WGRC39, or Newton) and intercrossed in all possible combinations, including reciprocals. The parents, F₁, F₂, and F₃ generations were tested for resistance to *S. nodorum* in the greenhouse as 4-week-old seedlings. Cytoplasmic effects were not detected in any cross. The mean levels of infection in the F₁s of the two crosses Betty × Larned and Heyne × KS96WGRC39 indicated resistance was dominant. The observed phenotypic ratios of F₂ plants for both crosses were not significantly different from the expected ratio for a single dominant gene. The ratio observed for F₃ lines in the Betty × Larned cross fit that expected for a single dominant gene. However, the observed ratio of the F₃ lines from the cross Heyne × KS96WGRC39 did not fit the ratio expected for a single dominant gene. The allelism test for Betty and Heyne indicated that they have different resistance genes. The F₁ mean rating of the cross 2163 × Newton was intermediate between the two parents, indicating the absence of dominance for resistance in 2163. The phenotypic ratio observed in the F₂ plants from this cross did not fit the ratio expected for a single dominant gene. The simple genetic control of resistance in cv. Betty makes it a useful source of resistance for wheat breeding programs.

Stagonospora nodorum blotch, caused by *Stagonospora nodorum* (Berk.) Castellani & E.G. Germano (= *Septoria nodorum* (Berk.) Berk. in Berk. & Broome, teleomorph: *Phaeosphaeria nodorum* (E. Müller) Hedjaroude) can cause serious yield and quality losses of wheat (*Triticum aestivum* L.) in many countries worldwide (3,14,20). Yield losses caused by this disease have been reported as high as 40% in severe epidemics (8,18,31). Under severe epidemics, the kernels of susceptible wheat cultivars are shriveled and are not fit for milling (11).

The fungus causes both leaf and glume blotch of wheat. Although glume blotch was stated to be the main cause of yield reduction (16,18), foliar infection can be as

detrimental to yield as head infection (11). For example, Walther and Bohmer (34) reported that *Stagonospora nodorum* blotch severity on leaves had the highest correlations with yield loss, whereas head infection was poorly correlated with yield.

Although there are several control methods, including crop rotation and foliar fungicides, the preferred method of control is the use of resistant cultivars (4,36). However, breeding for resistance to *S. nodorum* is difficult because resistance can be correlated with undesirable agronomic traits such as tall plant height and late maturity (32).

Resistance to the leaf and head phases may be under separate genetic control (2,15,35). Fried and Meister (13) also reported evidence for independent segregation of genes controlling resistance to leaf and glume blotch. Resistant genes for reaction on the flag leaf were found on chromosomes 3A, 4A, and 3B, while those for the head phase were located on the same chromosomes and on 7A (15).

The testing of wheat seedlings in the greenhouse has been reported to be an effective strategy for identifying reactions to *S. nodorum* because the susceptibility of seedlings was correlated with that of the adult plants in the field (10,20,28). This

type of testing permits use of quantitative inoculation techniques and eliminates the influence of undesirable agronomic traits such as tall plant height and late maturity on selection (37).

Many sources of resistance to *Stagonospora nodorum* blotch have been identified in species related to wheat: *Triticum timopheevii* (23,33), *T. monococcum* (22), *Aegilops tauschii* (22,27), *A. speltoides* (5), and *A. longissima* (6). In addition, resistance also has been identified in several wheat cultivars and their genetic control studied. However, most studies were conducted for resistance to glume blotch and indicated that the resistance was polygenically controlled (7,13,17,25,26,37). A few studies were undertaken to study resistance to leaf blotch. In recent studies, Wilkinson et al. (37) and Wicki et al. (35) reported that the resistance to the leaf phase is inherited polygenically in winter wheat cultivars commonly grown in the United States and Europe, respectively. However, Frecha (12) reported a single dominant gene for seedling resistance to *Stagonospora nodorum* blotch in the wheat cv. Atlas 66. This gene was located on chromosome 1B (19). Wong and Hughes (38) also reported monogenic control for resistance to the leaf phase of *Stagonospora nodorum* blotch in three winter wheat cultivars (81IWWMN 2095, Coker 76-35, and Red Chief). In addition, Scharen and Eyal (30) provided evidence that resistance in highly resistant cultivars might be governed by major resistance genes. Although simple inheritance of resistance to leaf blotch has been reported, use of the resistance has lagged because of lack of supporting reports for additional single-gene resistances. The reason for failure to identify other resistances controlled by single genes is because many studies were conducted in the field where other foliar diseases may confound disease ratings.

In recent years, several winter wheat cultivars adapted to Kansas have been released with improved resistance to the leaf phase of *Stagonospora nodorum* blotch (1). These were developed by recurrent-selection for green leaf duration in numerous field nurseries during the breeding period. These cultivars have been grown extensively in Kansas with one cultivar (Jagger) occupying 43% of the seeded acreage in 2002. Because of their

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commercial importance and usefulness as sources of resistance to *Stagonospora nodorum* blotch, it would be desirable to know more about the inheritance of resistance in these cultivars. The objective of this study was to determine the number of genes controlling resistance to the leaf phase of *Stagonospora nodorum* blotch in selected Kansas winter wheat cultivars and to identify whether the cultivars have the same or different genes for resistance.

MATERIALS AND METHODS

Plant material. Three winter wheat cultivars with moderate resistance to the leaf phase of *Stagonospora nodorum* blotch and three susceptible lines were selected based on their observed reaction in the field and results from preliminary experiments in the greenhouse. Their reaction in the greenhouse was further quantified during the time it took to produce F₂ and F₃ seed. Crosses involving several resistant parents usually are made using the same susceptible parent. However, we did not do this because, at the beginning of our experiments, we were not sure of the relative usefulness of our susceptible parents for a greenhouse inheritance study. In retrospect, Larned would have been the best choice for greenhouse inheritance studies because of its more uniform distribution of highly susceptible plants (Table 1). The moderately resistant cultivars were crossed with one of three susceptible lines and intercrossed in all possible combinations, including reciprocals, to examine the significance of reciprocal effects. Certified seed of all cultivars was used to make a total of 12 crosses (Table 2). Cv. Betty was included instead of Jagger because it is a sister line of Jagger and a newer release. The F₁ progenies of the 12 crosses were selfed to produce F₂ generations. F₃ lines were obtained from 192 and 128 F₂ plants of Betty × Larned and Heyne × KS96WGRC39 crosses, respectively. In each generation, seed were sown in small square pots (5 by 5 by 4 cm). After 7 to 10 days, the seedlings were vernalized for 7 to 8 weeks in a growth chamber (4°C) before being transplanted into larger pots (15.5-cm top diameter, 13-cm bottom diameter, and 15.5 cm tall) containing pasteurized soil (Kennebeck silty clay loam) in the greenhouse.

The F₁ plants were tested for resistance to assess dominance, and the F₂ plants and F₃ lines were tested to determine the phenotypic ratios, which were used to estimate the number of genes controlling resistance. For testing, seed of F₁, F₂, and F₃ generations, and their parents, were placed on moistened filter paper (Whatman no. 1) in petri dishes and incubated at room temperature for 3 to 4 days to synchronize germination. One germinated seed was planted in a plastic tube (2.5 cm in diameter by 16 cm long) containing 50% soil by volume (Kennebeck silty clay loam) and 50% vermiculite. The tubes were completely randomized in the appropriate number of racks (100 tubes/rack). Testing of F₁s and F₂s involved three runs (except for Heyne [female] crossed with KS96WGRC39, which had two runs) of about 40 seed of each reciprocal cross and each parent in each run. All runs of experiments produced similar results and were combined. In the F₃ generation, 10 tubes per line and eight lines per rack with their parents were planted. In all, 192 lines of Betty × Larned (three runs) and 128 lines of Heyne × KS96WGRC39 (two runs) were tested and the reaction of each F₃ line determined by the mean of 10 plants. The F₃ lines of the Betty × Larned cross were tested twice. All experiments were conducted in the greenhouse (20 ± 5°C) with supplementary lighting from high-pressure sodium lamps (400 W) to provide a 16-h photoperiod (about 200 μE m⁻² s⁻¹). All seedlings were watered everyday and fertilized once with water-soluble N-P-K (20:20:20) within 1 week after planting.

Inoculum preparation. A single-spore, virulent isolate of *S. nodorum* (NOD-99), obtained from diseased wheat leaves col-

lected from a commercial field in 1999 and stored in soil at 4°C, was used throughout this study. Fungal spores from 7- to 10 day-old cultures on V8 juice agar (150 ml of V8 juice, 3 g of CaCO₃, 15 g of agar, and 850 ml of distilled water) were streaked onto fresh V8 agar in petri dishes at room temperature (22 ± 2°C) and placed 40 cm below four cool-white fluorescent tubes (40W, about 36 μE⁻² s⁻¹). After 7 days of growth, spores were harvested by blending a single agar plate in 200 ml of distilled water with a commercial blender and resulting suspensions were filtered through two layers of cheesecloth. Unflavored gelatin (0.5 g) was dissolved in 20 ml of warm distilled water and added per 100 ml of spore suspension as a spreader sticker. Spores were counted with a hemacytometer and suspensions adjusted using distilled water to 4 × 10⁵ spores/ml.

Inoculation procedure. Plants (four-leaf stage) were inoculated 4 weeks after planting by spraying the fungal spore suspensions (35 ml/120 plants) onto leaves with an atomizer (De Vilbiss Co., Somerset, PA) operated at 69 kPa. Inoculated plants were air dried for about 30 min to allow spores to adhere to the leaves. After inoculation, the plants were placed in a plastic-covered mist chamber in a greenhouse (25 ± 5°C) for 72 h to maintain continual leaf wetness and then returned to the greenhouse bench. Mist was provided by two centrifugal atomizing humidifiers electrically controlled to operate for 1 min every 10 min.

Disease assessments. Although plants at the four-leaf stage were inoculated, symptoms only occurred on the bottom three leaves. Even on susceptible cultivars, young leaves are highly resistant. Therefore, the bottom three leaves of each plant

Table 2. Crossing scheme for three moderately resistant (MR) and three susceptible (S) winter wheat cultivars used in greenhouse tests to assess the inheritance of resistance

Male parent	Female parent					
	Betty	Heyne	2163	Larned	KS96WGRC39	Newton
Betty (MR)	...	X	X	X
Heyne (MR)	X	...	X	...	X	...
2163 (MR)	X	X	X
Larned (S)	X
KS96WGRC39 (S)	...	X
Newton (S)	X

Table 1. Leaf reactions to *Stagonospora nodorum* for Kansas winter wheat lines in the seedling stage in the greenhouse

Parent	Pedigree ^a	Disease (%) ^b	Reaction ^c
Betty	Jagger (KS82W418/Stephens) (KS, 1994) 'Sib' selection (KS, 1998)	26.5 (9)	MR
Heyne	Plainsman V/KS75216//SWM 754308/3/Plainsman V/Lindon//KS82W422 (KS, 1998)	21.8 (17)	MR
2163	Pioneer line W558/5/Etoile de Choisy//Thorne/Clarkan/3/C115342/4/Purdue 4946A4-18-2 (KS, 1989)	27.7 (11)	MR
Larned	Scout*5/Ottawa (KS, 1976)	54.2 (18)	S
KS96WGRC39	TAM 107*3/TA2460 (<i>Aegilops tauschii</i>)	52.8 (1)	S
Newton	Pitic 62/Chris sib//2*Sonora 64/ Klein Rendidor/4/Scout (KS, 1977)	49.3 (9)	S

^a State and year of release in parentheses; KS = Kansas.

^b Percentage leaf area affected. Numbers in parentheses indicate number of replicated experiments conducted in the greenhouse to determine reaction to *Stagonospora nodorum* leaf blotch with 40 plants per experiment.

^c MR = moderately resistant and S = susceptible.

were rated for percentage of leaf area covered by chlorosis and necrosis 14 days after inoculation. Each leaf was placed into one of the following categories: 0, 1, 5, 10, 25, 50, 75, and 100% of leaf area affected (11). For each plant, the percentages of infection on three leaves were averaged. To give a final disease score for a plant, the average percentage of infection was divided by the average percentage of infection of the most susceptible plant in a rack. These final disease scores were used in the analysis of variance. For the F₃ generation, 30 leaves per line were averaged.

Genetic analysis. To determine the phenotypic ratio, the F₂ plants for all crosses were placed into two groups: (i) a susceptible group (S), consisting of plants with the percentage of infection greater than the mean of the susceptible parent (or the susceptible control for the cross between two moderately resistant cultivars) minus two standard errors; and (ii) the remaining group, consisting of resistant and intermediate (R + I) plants. The F₃ lines were classified into three groups: (i) an S group, consisting of plants with the percentage of infection greater than the mean of the susceptible parent minus two standard errors; (ii) an R group, consisting of plants with the percentage of infection less than the mean of the resistant parent plus two standard errors; and (iii) the remaining lines, grouped into an I group.

Statistical analysis. Statistical analyses were conducted using PC SAS (SAS version 8.0; SAS Institute, Cary, NC). To determine differences between the parents and the F₁s, as well as F₂s for moderately resistant × moderately resistant crosses, the mean values of *Stagonospora nodorum* blotch infection were compared by the general linear model analysis of variance (ANOVA) followed by mean separation by least significant difference (LSD, *P* = 0.05). For the F₃ lines of the cross Betty × Larned, the correlation coefficient *r* was calculated to test the strength of the relationship between experiments 1 and 2. For the F₂ and F₃ generations, χ^2 analyses were carried out to test the goodness-of-fit of the observed distribution with expected segregation ratios. To compare the difference between the percentage of F₂ plants of the cross Betty × Heyne and the two parents in

the susceptible group, a one-tailed *t* test was performed.

RESULTS

The mean values of *Stagonospora nodorum* blotch infection in the F₁ plants and parents indicated a wide separation between Betty and Larned (Table 3). Similarly, the range of *Stagonospora nodorum* blotch infection on individual plants also showed that Betty (0 to 58%) and Larned (51 to 100%) had minimal overlap (Fig. 1). According to the ANOVA, the means of the F₁ plants from the cross Betty × Larned were not significantly different from that of Betty (Table 3). Therefore, resistance in cv. Betty was dominant. The means of each reciprocal indicated that there was no cytoplasmic effect (Table 3). The F₂ generation of Betty × Larned segregated in a ratio of 3.8:1 (R+I:S). The observed phenotypic ratio was not significantly different from the ratio expected for a single dominant gene (Table 4). In the first test of the Betty × Larned F₃ lines (Fig. 2, Exp. 1), 38 lines were classified as R, 39 as S, and 90 as I. The observed ratio was not significantly different from the ratio expected for a single dominant gene ($\chi^2 = 1.02$, *P* > 0.50; experiment 1; Table 5). In the second test (Fig. 2, Exp. 2), there were 53 R, 41 S,

and 97 I lines. Again, the observed ratio was not significantly different from the ratio expected for a single dominant gene ($\chi^2 = 1.56$, *P* > 0.25; experiment 2; Table 5). The means of F₃ lines from experiment 1 were highly correlated (*r* = 0.77, *P* < 0.0001) with those of experiment 2.

Mean separation for Heyne and KS96WGRC39 was relatively narrow in the experiment involving F₁s, because the level of resistance in Heyne was lower than that of Betty or 2163, and KS96WGRC39 was not as highly susceptible as Larned (Table 3). The frequency distribution of *Stagonospora nodorum* blotch infection on individual plants showed that there was broad overlap between Heyne and KS96WGRC39 (Fig. 3). According to the ANOVA, the means of the F₁ plants from the cross Heyne × KS96WGRC39 were not significantly different from that of Heyne (Table 3). Therefore, resistance in cv. Heyne was dominant. The means of each reciprocal indicated that there was no cytoplasmic effect (Table 3). The F₂ generation of Heyne × KS96WGRC39 segregated in a ratio of 3.1:1 (R+I:S). The observed phenotypic ratio was not significantly different from the ratio expected for a single dominant gene (Table 4). In the F₃ test of the Heyne ×

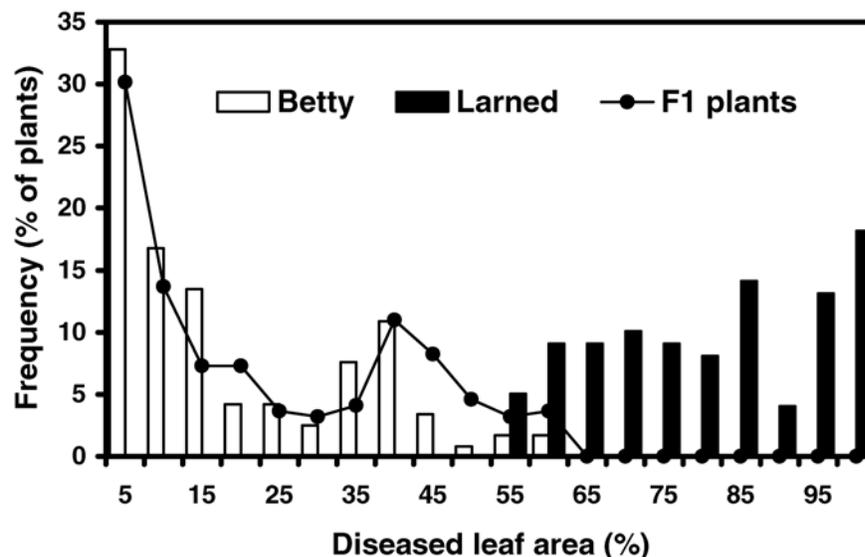


Fig. 1. Frequency distribution of percentage diseased leaf area for *Stagonospora nodorum* leaf blotch in winter wheat cvs. Betty and Larned and their F₁ progeny.

Table 3. Mean percentage of diseased leaf area for *Stagonospora nodorum* leaf blotch for moderately resistant (MR) and susceptible (S) parents and their F₁ progeny^a

Cross	Parent		F ₁ plants		LSD ^d
	MR	S	MR × S ^b	S × MR ^c	
Betty (MR) × Larned (S)	21.1 (119)	81.3 (99)	20.5 (111)	20.0 (109)	6.4
Heyne (MR) × KS96WGRC39 (S)	31.6 (80)	53.3 (79)	31.0 (79)	34.3 (80)	8.1
2163 (MR) × Newton (S)	20.3 (118)	52.8 (117)	39.5 (118)	41.7 (117)	6.9

^a Number in parentheses = total number of plants tested.

^b MR parent was used as a female in the cross.

^c S parent was used as a female in the cross.

^d Least significant difference.

KS96WGRC39 cross (Fig. 4), 52 lines were classified as R, 27 as S, and 37 as I. The observed ratio of the F₃ lines failed the test for goodness-of-fit for the 1:2:1 ratio expected for a single dominant gene ($P < 0.001$; Table 5).

A broad overlap between 2163 and Newton was observed in the frequency distribution of percent infection of individual plants (Fig. 5). The means of the F₁ plants from the cross 2163 × Newton were intermediate between the two parents and were significantly different from that of both parents (Table 3). This indicated that there was no dominance to resistance in the cv. 2163. The ratio observed for the F₂ plants from the cross 2163 × Newton did not fit the ratio expected for a single dominant gene ($P < 0.001$; Table 4).

To determine whether the moderately resistant parents shared any resistance genes, three moderately resistant cultivars were intercrossed in all possible combinations. When crossing two resistant parents, each carrying an independent single dominant gene for resistance, the following patterns potentially could occur: (i) if the genes in both parents are the same, no segregation will be observed and (ii) if the genes in both parents are different and unlinked, the F₂ populations will segregate with 1 out of 16 F₂ plants carrying no resistant genes. Therefore, 6.25% of the individual F₂s should be significantly more susceptible than either parent.

Compared with both moderately resistant parents, the mean percentage of *Stagonospora nodorum* blotch infection in the F₃s from all moderately resistant × moderately resistant crosses was not significantly different from the mean of both parents (Table 6). Cytoplasmic effects were not detected. In the F₂ generation, the mean values of the F₂ plants from the cross Betty × Heyne were not significantly different from the mean of both parents. However, the frequency of plants with disease infection greater than 73.5% (mean of the susceptible control Larned minus two standard errors) in the F₂ populations was 8.4%. The frequencies of Betty and Heyne were 3.4 and 3.6%, respectively (Fig. 6). According to the one-tailed *t* test, the difference between the F₂ plants and the parents in the susceptible group was significantly different from zero. Further allelism tests for other crosses were not analyzed because of the failure to estimate the number of resistance genes in cv. 2163.

DISCUSSION

Selecting for green leaf duration has significantly improved the level of resistance in popular Kansas cultivars to *Stagonospora nodorum* leaf blotch (1). Our data quantified the magnitude of this improvement in the greenhouse environment where moderately resistant cultivars showed 20 to 32% diseased leaf area compared with 81% for a susceptible cultivar (Table 3). The main goal of this research was to determine how this moderate level

of resistance was inherited and whether certain Kansas cultivars share a common gene for resistance.

All of our experiments were conducted in the greenhouse with seedling plants at the four-leaf stage. Several parameters can be used to estimate resistance to *Stagonospora nodorum* blotch, including incubation period; latent period; infection frequency; size, shape, and rate of lesion growth; and spore production and its rate of increase (9,21,24). To eliminate the

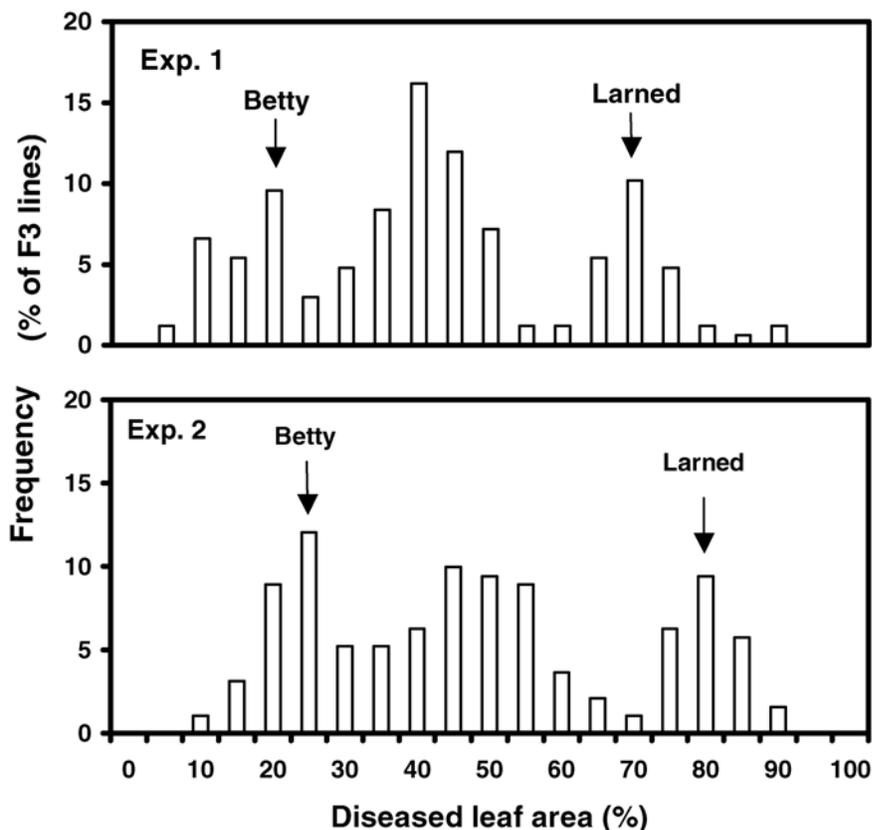


Fig. 2. Frequency distribution of F₃ lines for percentage *Stagonospora nodorum* leaf blotch for Betty crossed with Larned in two separate experiments. The level of the parents also is indicated.

Table 5. Phenotypic ratios for reaction to *Stagonospora nodorum* leaf blotch in F₃ lines from moderately resistant (MR) × susceptible (S) crosses and their goodness-of-fit tests with the ratio expected for a single dominant gene

Cross	Observed ratio (no. of F ₃ lines) ^a			Expected ratio (1:2:1)	
	R	I	S	χ^2	<i>P</i> value
Betty × Larned (experiment 1)	38	90	39	1.02	0.75–0.50
Betty × Larned (experiment 2)	53	97	41	1.56	0.50–0.25
Heyne × KS96WGRC39	52	37	27	26.0	<0.001

^a R = resistant and I = intermediate.

Table 4. Phenotypic ratios for reaction to *Stagonospora nodorum* leaf blotch in the F₂ plants from moderately resistant (MR) × susceptible (S) crosses and their goodness-of-fit tests with the ratio expected for a single dominant gene

Cross	Observed ratio (number of F ₂ plants)			Expected ratio (3:1)	
	R + I ^a	S	Segregation ratio	χ^2	<i>P</i> value
Betty × Larned	190	50	3.8:1	2.20	0.25–0.10
Heyne × KS96WGRC39	99	32	3.1:1	0.02	0.90–0.75
2163 × Newton	106	114	0.9:1	84.4	<0.001

^a R + I = resistant and intermediate.

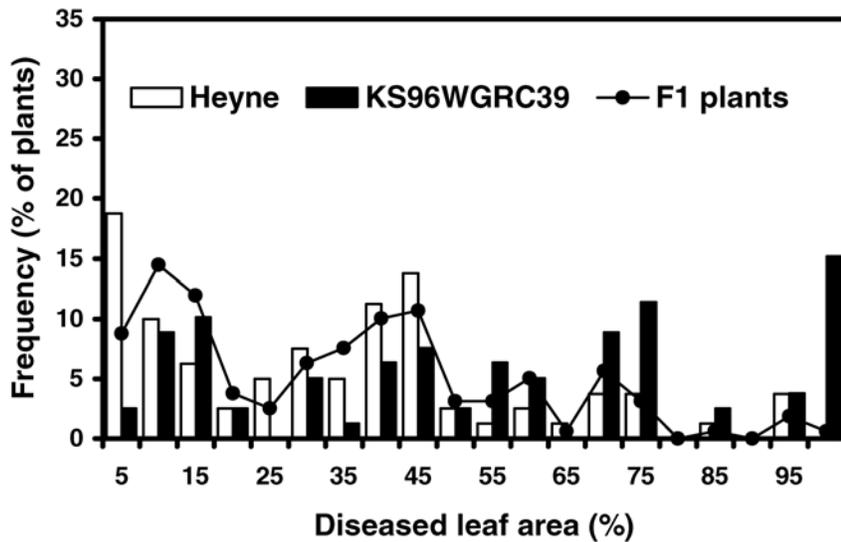


Fig. 3. Frequency distribution of percentage diseased leaf area for *Stagonospora nodorum* leaf blotch in winter wheat cvs. Heyne and KS96WGRC39 and their F₁ progeny.

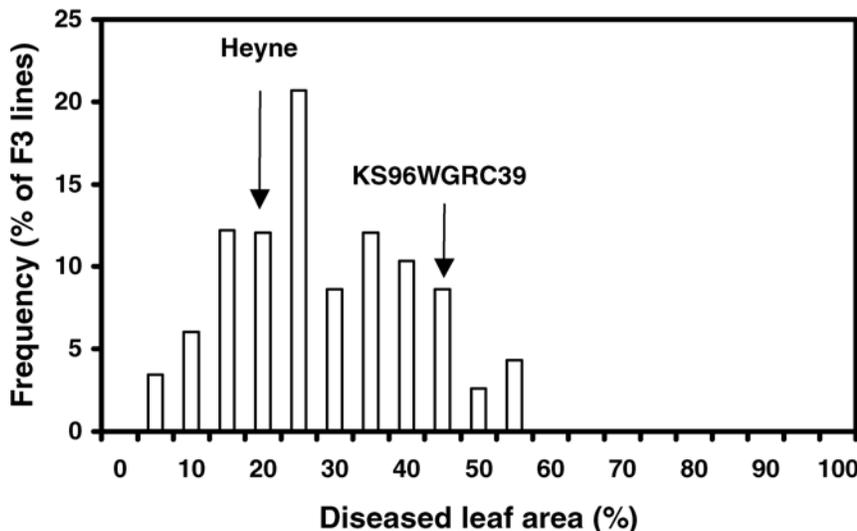


Fig. 4. Frequency distribution of F₃ lines for percentage *Stagonospora nodorum* leaf blotch for Heyne crossed with KS96WGRC39. The level of the parents also is indicated.

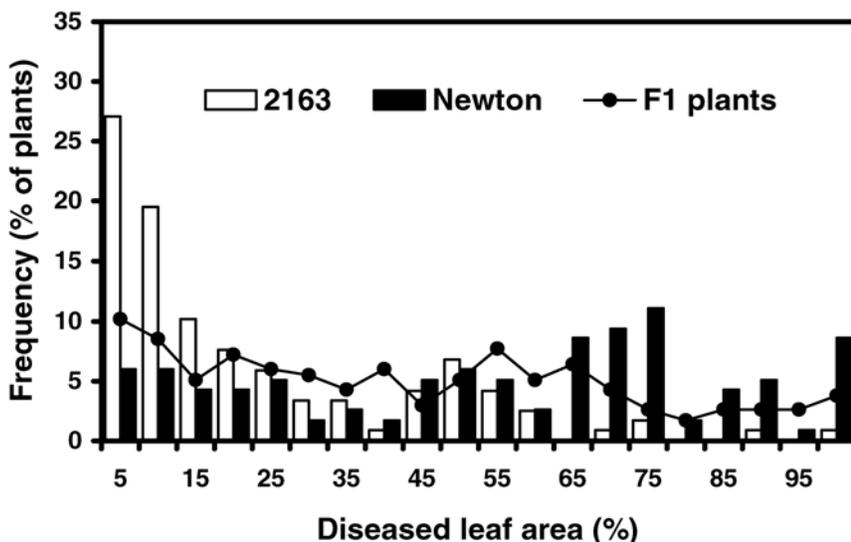


Fig. 5. Frequency distribution of percentage diseased leaf area for *Stagonospora nodorum* leaf blotch in winter wheat cvs. 2163 and Newton and their F₁ progeny.

influence of plant height and maturity, seedling tests in a controlled environment such as a greenhouse or growth chamber often are used by measuring the percentage of infected leaf area (29,37). Seedling tests in the greenhouse are reported to be highly correlated with field tests (10,20,28). However, seedling test data reported here cannot be used for determining reactions to *Stagonospora nodorum* blotch on the heads because resistance to the leaf and head phases are under separate genetic control (13,15,35).

Our experiments differed from those of Wong and Hughes (38) in scoring disease development for classification of segregating populations. They rated the disease severity on a 0-to-9 scale and classified 0 to 4 as a resistant group and 5 to 9 as a susceptible group. However, in this study, plants were rated by using percentage of leaf area affected (0 to 100%) and disease scores were continuous, with overlap between resistant and susceptible parents. Significant overlap existed between the parents of most crosses we tested. This phenomenon also was detected in the field (17) and in the greenhouse (2). In preliminary testing of the parental lines, the disease scores among the parents were separable without any overlap when at least 10 plants per line were averaged. In this case, plants with little, or no, disease (escapes) are compensated for by plants that may produce above-average disease. However, for our data, the disease reaction of individual plants in the F₂ generations showed large variation because of segregation and the lack of this 10-plant replication.

The disease reactions of F₁ plants from the two crosses Betty × Larned and Heyne × KS96WGRC39 demonstrate dominance for resistance. Cytoplasmic effects were not observed in all crosses we tested. Many other studies also have found no cytoplasmic effects (2,24) even though Nelson (25) suggested that cytoplasmic effects might be involved in some sources of *Stagonospora nodorum* blotch resistance.

Testing of F₂s from the cross Heyne × KS96WGRC39 indicated that Heyne may carry a gene of large effect. However, the phenotypic ratio obtained from the 116 F₃ lines did not fit a ratio for single dominant gene segregation. There are several possible reasons for the failure to estimate the number of resistant genes from the F₃ testing. First, the number of F₃ lines was somewhat small to separate the segregating lines from the R group compared with that of Betty × Larned cross (192 F₃ lines). Second, to increase the probability up to 99% to correctly categorize the segregating lines, 16 plants per line, instead of 10, would be needed. Although the segregation ratio obtained from the F₃ lines (52:37:27 R:segregating:S) did not fit the ratio expected for a single dominant gene (1:2:1), when the R and segregating lines were

Table 6. Mean percentage diseased leaf area for *Stagonospora nodorum* leaf blotch for moderately resistant parents and their F₁ progeny^a

Type of cross (A × B)	Parent ^b				F ₁ plants		LSD ^e
	A	B	Mean	S	A × B ^c	B × A ^d	
Betty × Heyne	37.0 (40)	48.8 (40)	42.9 (80)	66.7 (40)	39.0 (40)	41.2 (40)	10.7
Betty × 2163	34.0 (40)	40.7 (40)	37.3 (80)	61.4 (40)	36.5 (40)	37.1 (40)	11.1
Heyne × 2163	25.6 (40)	40.1 (40)	32.8 (80)	63.3 (40)	32.5 (40)	30.7 (40)	11.0

^a Number in parentheses = total number of plants tested.

^b A = moderately resistant parent A, B = moderately resistant parent B, and S = susceptible control; Larned for the crosses Betty × Heyne and Betty × 2163, and Newton for Heyne × 2163.

^c Moderately resistant parent A was used as a female in the cross.

^d Moderately resistant parent B was used as a female in the cross.

^e Least significant difference.

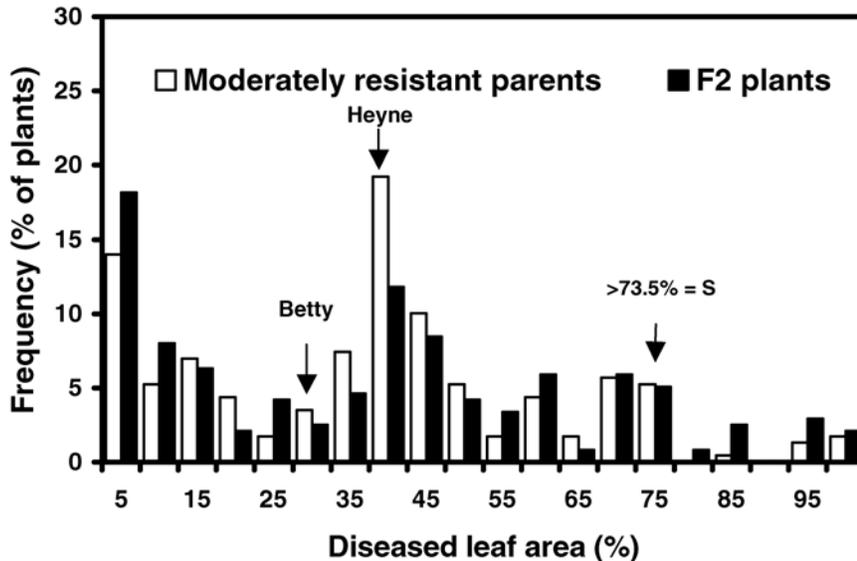


Fig. 6. Frequency distribution of F₂ plants for percentage *Stagonospora nodorum* leaf blotch in the cross of two moderately resistant cultivars (Betty and Heyne) and the average of the two parents. The susceptible class is any value greater than 73.5% (mean of the susceptible control Larned minus two standard errors). The level of the parents also is indicated.

combined, the ratio (3.3:1) was close to 3:1. Finally, the disease ratings of the parents Heyne and KS96WGRC39 were relatively close to each other. The difference of *Stagonospora nodorum* blotch infection between Heyne and KS96WGRC39 was only 23.8% compared with 60.2% between Betty and Larned. In addition, some overlap existed between Heyne and KS96WGRC39, which made the separation of each category difficult (23). Nevertheless, genetic results obtained from the cross Heyne × KS96WGRC39 suggested that Heyne may carry a gene of large effect on resistance to the leaf phase of *Stagonospora nodorum* blotch.

In the F₂ testing from the cross 2163 × Newton, the observed segregation ratio failed the test for a single gene for resistance. Because of the large variances within the parents and a wide overlap between the parents, the number of genes controlling resistance to *Stagonospora nodorum* blotch could not be estimated. Another reason for failure to determine the number of genes is because the level of resistance in cv. 2163 was not as high as that of Betty or Heyne in most experiments (Tables 1 and 6), and cv. Newton was not

as highly susceptible as Larned (Table 3). Otherwise, resistance in 2163 may be polygenic.

In the allelism tests, the F₂ mean from the cross Betty × Heyne was not significantly different from that of either parent. However, the frequency comparison between the F₂ populations and the average of both parents revealed that the resistance gene in cv. Betty probably is not an allele of the gene in Heyne.

Breeding for resistance to *Stagonospora nodorum* leaf blotch can reduce the secondary infection and slow disease development (2). Resistance to *Stagonospora nodorum* leaf blotch in the Kansas winter wheat cv. Betty appears to be controlled by a single dominant gene. Although Scharen and Eyal (30) reported that resistance to *Stagonospora nodorum* blotch might be controlled by the additive action of several genes in moderately resistant cultivars, our results with Betty were similar to those of Frecha (12) with cv. Atlas 66. Further work is needed to identify the chromosomal location of the resistance gene. Additionally, it appears that a gene different from that in Betty is responsible for the resistance observed in Heyne. Having more

than one gene deployed in Kansas is important in case a new race of the fungus develops. This information should be useful to wheat breeding programs interested in development of resistance to *Stagonospora nodorum* leaf blotch.

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