

GENETIC STUDIES OF FIELD REACTION TO
WHEAT SOILBORNE MOSAIC VIRUS

by

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INTRODUCTION

Wheat soilborne mosaic is one of the most destructive diseases of wheat Triticum aestivum (L.) in Kansas.

The virus disease was first reported in Illinois in 1919 (23). McKinney (27) reported wheat soilborne mosaic in Kansas in 1949. The disease has spread from eastern into south-central and central Kansas, where most of the wheat is grown.

In Kansas the area infested with wheat soilborne mosaic virus is around 810,000 hectares, as estimated by Nykaza et al. (33). The estimated wheat loss in Kansas due to the disease in 1979 was 167,000 tons, according to the Kansas State University plant pathologists. At the current wheat prices, this amounts to more than 25 million dollars.

Wheat soilborne mosaic is present in many states in the United States (1, 12, 20, 27, 35, 38), Japan (29), Brazil (7) and other countries as reported by Johnson (17).

Many studies have been done on the etiology of the disease and possible control measures. It is well known that the best and most economic means of controlling wheat soilborne mosaic is through cultivar resistance. However, the inheritance of the disease is not sufficiently understood. One reason is that few genetic studies have been conducted, despite the economic importance of the disease. Most rapid progress can be made in a breeding program designed to produce resistant cultivars to wheat soilborne mosaic if we have a better knowledge of the

genetics of the disease reaction.

The objective of this work is to further elucidate the mode of inheritance of field reaction to wheat soilborne mosaic in some Kansas wheat cultivars.

LITERATURE REVIEW

1- Description of the Disease

Wheat soilborne mosaic (WSBM) is caused by wheat soilborne mosaic virus (WSBMV) which lives firmly adsorbed or inside the zoospores and resting spores of its fungus vector Polymyxa graminis Led. (10, 37). Polymyxa graminis is an obligate parasite found in roots of some plants.

Rao and Brakke (37) reported that the fungus has no visible effect on the host and its presence is not recognized by any external symptoms.

Electron microscopic work in the United States (3, 15, 36, 43) and Brazil (6), described the virus particles found in juice from wheat plants infested with the WSBMV. The studies revealed the presence of rod-shaped particles with a diameter of 20 nm and peaks of particle length distribution of 160 nm and 300 nm.

Tsuchizaki et al. (43) reported that the infection of plants only was observed when both short and long particles were present. It appears that the soilborne virus does not have a broad host range. However, Rao and Brakke (37) found Bromus spp. infested with wheat soilborne mosaic virus. The virus also infects barley, rye and other wheat species (22).

Koehler et al. (19) reported that repeated cropping to susceptible wheat in an area with initial low percentage of infested soil, tends to increase the mosaic virus infestation.

Although the infection on winter wheat occurs in the fall, soon after planting (3, 24), field symptoms, as a rule, are noticeable only in the spring when the wheat plants start growing vigorously (8, 11, 17, 19, 40). Nevertheless, some workers have occasionally observed symptoms when long growing periods occur in the fall (19, 24, 34).

Koehler et al. (19), Sill (40) and Johnson (17) have given detailed description of the field symptoms of the disease. Wheat soilborne mosaic is usually concentrated in lower parts of the fields with less drainage. But topography does not seem to be decisively important as infested fields occur on both low and high ground. The diseased areas vary in dimensions from a few inches to 50 or more feet across and have marginal limits rather sharply defined, clearer than those caused by unfavorable soil conditions. Sometimes the yellowish to light-green areas of irregular shape are spread involving whole fields. Severity of the disease varies. Very susceptible cultivars show a distinct yellow color and the growth may be severely retarded. Less susceptible cultivars show some change in color to a light green but the plants recover rapidly. The symptoms on the leaves are predominantly characterized by small green islands of tissue on a yellowish to light green background. The leaf edges and tops tend to show a bronze-like color. Depending on the environmental and cultivar factors, the diseased plants may be able

to recover the normal green color completely. Wheat soilborne mosaic infested plants also can be recognized by other symptoms such as delayed maturity, fewer heads, shorter heads and shriveled grains. The late ripening is responsible for striking green spotting in the fields at the harvest time and during wet seasons weeds become abundant in the disease-infected areas.

McKinney (23, 24, 25) described the two symptoms expressions of the disease in Illinois, 1) the mosaic mottling of the leaves causing stunting of the plants and 2) the mosaic rosette expression producing excessive tiller proliferation in some cultivars and a rather dark-green color on the leaves. He suggested that the wheat mosaic in Illinois represented a mixture of two distinct types, the yellow and the green forms respectively.

Although workers (19, 40) reported no severe symptoms of WSBM on spring wheat sown in the United States, Caetano et al. (6, 7) stated that the occurrence of the rosette expression of the disease on spring wheat seeded in soilborne mosaic virus infested area in Brazil was a serious problem to the crop.

The rosette expression of the disease was not observed in the field in Kansas, as reported by Sill (40).

McKinney (25), Koehler et al. (19), Campbell et al. (8), reported that adequate temperature, between 10°C to 16°C , ample rainfall during the growing season and adequate sunlight favor the infection. Under low soil-moisture conditions the symptoms are prevented from expression. However, abundant moisture in the spring helps the plants to recover from infection.

Nykaza et al. (33) studied the effects of WSBM on winter wheat and concluded that when the temperature in early spring remained below 17°C for long periods the symptoms persisted longer and the losses were greatest. They considered temperature in early spring as the single major factor in determining wheat yield losses due to wheat soilborne mosaic.

2- Economic Importance

Many workers have studied the effects of WSBM on wheat yield (2, 11, 19, 20, 23, 33, 35). All of them considered the mosaic disease a serious problem when susceptible cultivars are planted in infested areas.

McKinney (23), in 1923, when he first described the wheat soilborne mosaic symptoms, reported yield losses up to 40 percent due to the green mosaic infection.

Soil infestation, environment, cultivar resistance, all have an effect on the incidence of the disease and yield reduction.

The wheat soilborne mosaic was so severe in certain areas of Illinois that only resistant cultivars could be grown in some areas of the state, as reported by Koehler et al. (19).

The yield of several cultivars grown in areas heavily infested with WSBMV and virus-free soil were compared by Bever and Pendleton (2). The average yield reduction was 80.7 percent and 18.5 percent for the wheat cultivars showing severe rosette symptoms and severe leaf mottle, respectively, as compared with the healthy ones.

Fellows et al. (11) reported that although the rosette expression does not occur in Kansas, the losses due to the yellow mosaic virus infection cause severe damages in favorable years and that the disease was spreading to new areas of the state.

Kucharek and Walker (20) conducted tests in commercial fields in Florida infested with WSBMV. They reported yield losses of 42.5 percent and 52.5 percent due to the disease.

Palmer and Brakke (35) conducted surveys from 1972 to 1974 in fields of winter wheat infected with WSBMV. They reported losses caused by the virus as high as 44 percent with an average yield reduction of 20 percent in the 13 fields studied.

Campbell et al. (8) and Nykaza et al. (33), in Kansas, studied the effects of WSBM on yield and other characteristics of winter wheat. They reported that the mosaic disease reduced significantly the kernel weight, tiller number, test weight and plant height. The authors concluded that the yield reduction on susceptible wheat cultivars was up to 48 percent and 45.2 percent, respectively.

Caetano and Caetano (4) stated that the use of susceptible wheat cultivars in WSBMV infested areas caused significant yield reduction on spring wheat grown in Rio Grande do Sul, Brazil.

3- Control

McKinney (23), in 1923, stated that the disease could be controlled through the use of resistant cultivars.

The mosaic virus was reported to persist in the soil for

many years, even if the fields are not planted to susceptible cultivars (19, 23). Therefore, crop rotation is not recommended for controlling the disease. Late fall planting tends to reduce the damage from mosaic, but it is not a practical control measure.

Several workers (18, 21, 23, 24, 28, 34) have tried to eliminate the virus from the soil by physical and chemical means, such as, by applying formaldehyde, steam or other disinfecting chemicals. Although McKinney (23) and Johnson (18) presented some positive results, Kucharek et al. (21) reported that elimination of the virus in the field by soil fumigation was not possible because the P. graminis is at a depth not penetrated by the fumigant. Natural soil infestation with mosaic virus at a depth of two feet was also reported by Koehler et al. (19).

Soil fumigation can also modify the microbial activity in the soil and it is not a feasible control method for the disease.

The use of cultivar resistance has been demonstrated to be successful and it remains the best means of controlling wheat soilborne mosaic.

Due to the economic importance of WSBM on wheat production and the possibility of breeding new resistant cultivars, many workers are screening the breeding material on infested areas in order to select suitable new lines displaying resistance to the disease (4, 5, 9, 14, 21, 33, 38, 39, 41). In Kansas, for example, the WSBM was first reported in 1949 and since 1950 the wheat breeding lines have been tested to mosaic virus (40, 41).

Many workers tried to explain the mechanism of resistance to wheat soilborne mosaic, but it appears that the opinions are

not in complete agreement. While McKinney (26), Palmer and Brakke (35) indicated that the resistance was to the fungus vector because field resistant and field susceptible cultivars were susceptible when artificially inoculated, Kucharek et al. (21) concluded that the resistance was to the virus because cystosori of P. graminis were also found in roots of resistant cultivars.

Campbell et al. (8) reported that some wheat cultivars were resistant to the fungus and others to the virus when the experiments were conducted in the growth chamber. In the field, the resistance appeared to be to the vector P. graminis.

4- Genetic studies on wheat soilborne mosaic reaction

McKinney (24), in 1925, through head selection studies, observed that certain wheat cultivars can be homozygous for botanical and agronomic characters but not necessarily homozygous for the mosaic reaction.

Some genetic studies on wheat soilborne mosaic virus were done in Japan (29, 31, 32) and in the United States (9, 30, 39). However, the results are not in complete agreement, suggesting that more study should be done in this field in order to better understand the inheritance of soilborne mosaic virus. A thorough knowledge of this matter would be useful to breeders trying to create new wheat cultivars resistant to the disease.

In Japan, Miyaki (29) studied the reaction to the yellow and green mosaic of the F_1 and F_2 progeny from crosses of wheat cultivars. He reported the resistance to both forms of the disease to be monogenic dominant over susceptibility.

Nakagawa et al. (31, 32), also in Japan, studied separately the inheritance of reaction to WSBMV in F_1 and F_2 generations. They reported that three genes were involved in the reaction to the green and yellow strains. Two major genes H and M determined susceptibility and the A gene modified the action of the H gene.

A study in Kansas by Shaalan et al. (39) observed the reaction of F_3 plants of winter wheat to the yellow mosaic. They concluded that two factors were responsible for resistance: a major factor with partial dominance for resistance and the other gene acted as a modifier to the major factor. The second factor alone had no visible effect. However, when both genes were present, the resistance was complete.

Dubey et al. (9), in Illinois, studied the reaction to the mosaic and mosaic rosette phase of the disease in the F_1 , F_3 and backcross F_2 families. They reported a single dominant gene conditioning resistance to both strains of the virus. In crosses between resistant and susceptible cultivars the F_1 always was resistant.

Modawi (30), working with F_1 , F_2 , and F_3 generations of a seven parent complete diallel crosses, studied the inheritance of field reaction to wheat soilborne mosaic in Kansas. He reported resistance to be dominant over susceptibility and established a three gene hypothesis to explain the F_3 reaction to the disease: A and B genes conditioning resistance and the third gene C inhibiting the effect of the B locus.

Dubey et al. (9) reported no cytoplasmic effect for reciprocal

crosses made in Illinois. Nevertheless, Modawi (30) suggested a possible cytoplasmic factor in some cultivars because the F_1 plants had a different reaction to the disease when some reciprocal crosses were involved.

McKinney (24) recommended special selection studies within a cultivar in order to obtain homozygous material for the reaction to wheat soilborne mosaic. In his work, Modawi (30) also reported some unexpected reaction of the progeny derived from the Centurk cultivar and suggested that the Centurk population used was not homogeneous for WSBM reaction.

MATERIALS AND METHODS

The five winter wheat parental cultivars used in this study are listed in Table 1 together with some identifying characteristics. Four of them, Centurk, Eagle, Gage and Shawnee are hard red winter wheats. KS73256 is a hard white wheat. Shawnee and KS73256 were considered resistant to wheat soilborne mosaic. Eagle is susceptible, Gage moderately susceptible in the sense that it often is severely infected but recovers faster than Eagle. Centurk in the past gave various reactions not clearly identified and was considered moderately resistant. Normally Centurk recovers well from WSBM infection.

The five wheat cultivars were crossed in a diallel series including the reciprocals. The F_1 generation was grown in the field and greenhouse in 1976-77. The F_2 generation was space planted in the fall of 1977 in 8-foot rows at the Ashland area,

Table 1. Some characteristics of the 5 wheat parents used in the WSBM study. Riley County, Kansas. 1976-1980.

Cultivar	Parent-age*	Glume color	Grain color	Reaction to WSBM
Centurk	1	White	Red	Moderately resistant (mixture of resistant and susceptible)**
Eagle	2	White	Red	Susceptible (slow recovery)
Gage	3	White	Red	Susceptible (fast recovery)
KS73256	4	Brown	White	Resistant (some susceptible)**
Shawnee	5	Brown	Red	Resistant

* 1-Kenya 58/2/Newthatch/3/Hope/2*Turkey/4/Cheyenne/5/Parker

2-Selection from Scout

Scout: Nebred/2/Hope/Turkey/4/Cheyenne/3/Ponca

3-Ponca/3/Mediterranean/Hope//Pawnee

4-CIMMYT/Scout

5-Selection from Ottawa

Ottawa: Mediterranean/Hope/Pawnee/3/Oro/Illinois #1//Comanche

** Based on the 1979-1980 observation.

Riley County. An average of 70 plants were harvested and threshed individually from each cross. In the fall of 1978, 25 seeds from a random sample of F_2 plants were seeded in 3-foot rows in WSBMV infested area at the Agronomy farm, Ashland, Riley County. This study was part of a previous report (30). When the plants matured completely, part of the F_3 lines were harvested and threshed for the 1980 WSBM study.

In order to study the differences for reciprocal crosses to WSBM reaction as reported by Modawi (30), Centurk, Gage and KS73256 were crossed among each other in 1979, including the reciprocals. The harvested seed was space-planted in the fall of 1979 in WSBMV infested area at the unit #3 of the Agronomy farm, Ashland field, Riley County. The parental checks were seeded with the F_1 hybrids. One hundred seeds from the F_1 plants set aside in 1976 were randomly sampled and space-planted in the fall of 1979 in 8-foot rows on WSBMV infested soil also at the unit #3, Ashland area, among with the respective parents.

From the F_3 plants harvested in 1979 two random samples of 6 grams of seeds were taken. They formed the F_4 generation of the 1980 study. At the same time two random samples of 25 seeds were made of the F_2 plants harvested in 1978. These seeds provided lines for the F_3 generation study in 1980.

The F_3 and F_4 lines were seeded in the fall of 1979 on WSBMV infested field at two locations: 1) at unit #3 of the Ashland area, Riley County, 2) at Hesston, located in south-central Kansas. The parental checks were included every 20th and 21st rows.

In the summer of 1979, 226 heads of Gage, 218 of Centurk and 212 of Rocky, a selection from Centurk, were randomly selected and threshed individually. In the fall of 1979 seeds from each head were planted in a 3-foot row in WSBMV infested area at Ashland, Riley County. The objective of this work was to determine whether these cultivars were homogeneous for wheat soilborne mosaic reaction.

The F_4 lines were seeded with a nursery seeder in a 3-row plots, 6 feet long. All the other seedings were done with a one-row hand machine suitable for small amount of seeds. The fertilization and other cultural practices were those recommended for commercial fields and uniform for all the experiments.

The dry weather that characterized the early fall of 1979 caused difficulties in seeding. At Riley County the wheat was seeded September 28 and 29 in the dry soil and most of the germination occurred a month later when rain was received on October 19. However, some F_4 machine planted plots had early and late germination. Early emerged plants had a greater fall growth and most of them in the spring appeared to have less infection with WSBM.

The excess rainfall that came in the fall delayed the seeding at Hesston till December 7. The wheat at Hesston emerged in the spring of 1980 and had poor crown root development and poor growth. Although the wheat soilborne mosaic symptoms were present, it was not possible to make adequate readings and consequently the Hesston experiments were abandoned.

All the 1980 studies are restricted to the results obtained at Riley County, Kansas. The scale used to classify the WSBM reaction of the parents and their progeny was:

Resistant (R): leaves free of mottling, no plant stunting.

Moderately Resistant (MR): leaves with slight mottling, slight plant stunting.

Moderately Susceptible (MS): leaves with clear mottling, clear plant stunting.

Susceptible (S): leaves with severe mottling, severe plant stunting.

In order to ascertain that the field was uniformly infested, besides the parental checks, the susceptible cultivar Sage was seeded in rows transversely to the normal planting except for the F_4 plots, (Fig. 1). Also the susceptible cultivar Red Chief was seeded as border surrounding the experiments.

The readings for WSBM infections were made on April 1, April 5 and April 12. A general check was made on April 18.

The agreement between observation and hypothesis was tested by means of the Chi-square distribution as suggested by Harris (16). The probability of a greater X^2 value, P was given by Snedecor and Cochran (42). As a result the smaller the X^2 the more closely the observed frequency agrees with that expected. The 5 percent level of probability was the limit of significant deviation for all Chi-squares tested.

Due to some difficulties in differentiating between segregating and susceptible lines, the statistical analysis was

applied on two classes as suggested by Fisher (13). The healthy class consisted of resistant lines and the infected class was represented by the segregating and susceptible lines.

RESULTS AND DISCUSSION

The good moisture conditions in late fall together with the cool spring weather greatly favored the disease expression. The severity of the symptoms was observed not only at the site of the experiments but throughout the major wheat producing area of south and south-central Kansas, despite the large acreage sown to the resistant Newton cultivar. In the farms where susceptible cultivars were seeded on infested soil, the WSBM symptoms were considered the most severe during the last decade.

At Riley County the first symptoms were visible in early March when the wheat resumed spring growth. The disease severity reached its peak in the first week of April and by the end of the month, when the temperature had warmed up, the symptoms were decreasing in intensity. However, symptoms could be seen until mid May.

As illustrated in Fig. 1 and 2, the symptoms on the susceptible cultivars and borders were clear and evenly distributed throughout the whole field. No healthy plants were observed in the susceptible borders. This greatly reduced the possibility of escapes occurring.

Some infected lines, besides the color symptoms, suffered

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Figure 1. F_3 lines segregating for WSBM reaction, seeded in 3-foot rows. Note the susceptible border Sage (yellow rows 'L' shaped).

Figure 2. General view of the F_4 lines showing the reaction to WSBM and the uniformity of the virus distribution throughout the field.



Fig. 1

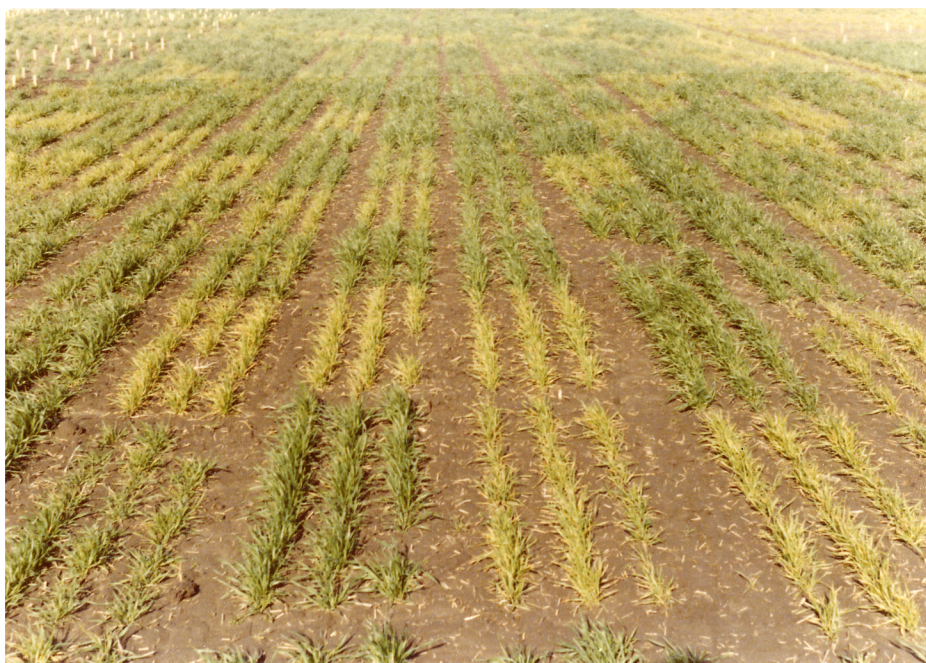


Fig. 2

Figure 3. F_4 lines segregating for WSBM reaction, showing the dwarfing of some susceptible (yellow) lines as compared to the healthy genotypes.

Figure 4. Reaction to WSBM of two F_3 lines. The susceptible on the right had all the color symptoms but the growth rate was not affected.



Fig. 3



Fig. 4

Figure 5. Four lines of heads selected from Centurk showing the heterogeneity to WSBM reaction. The yellow row on the bottom is the susceptible border Sage.

Figure 6. Two lines of heads selected from Rocky classified as segregating moderately resistant (GMR).



Fig. 5



Fig. 6

dwarfing as illustrated in Fig. 3. Other genotypes although clearly infected and showing the yellow mottling on the leaves, had vigorous growth, similar to healthy ones. Fig. 4 illustrates this type of reaction.

I- Analysis of head row progeny

Table 2 lists the field reaction to WSBM of the heads selected from Centurk, Rocky, and Gage. This study clearly showed that a cultivar can be homogeneous for other agronomic characteristics and heterogeneous for WSBM reaction. That is, it can be a mixture of resistant and susceptible plants.

Of the 218 selected ^{spike}heads from the cultivar Centurk, 19 (8.7 percent) were resistant (R); 136 (62.4 percent) were susceptible (S); 48 (22.0 percent) had one or more plants susceptible and were classified as segregating moderately resistant (GMR); 15 (6.9 percent) had all plants with a milder infection than the susceptible plants and were classified as moderately resistant (MR). The reactions of four head progeny selected from Centurk are illustrated on Fig. 5. This study confirms that Centurk is not homogeneous for WSBM reaction. I concluded that Centurk is a mixture of resistant and susceptible genotypes. This explains, in part, some of the reciprocal differences to WSBM reaction observed in crosses involving Centurk.

Rocky has a good level of resistance to WSBM, better than Centurk, but it also showed symptoms of the disease. From 212 selected heads of Rocky, it was found 39 (18.4 percent) resistant

Table 2. Field reaction to WSBM of head selections from 3 winter wheat cultivars. Riley County, Kansas. 1979-1980.

Cultivar	Reaction*				Total
	R	GMR	MR	S	
Centurk	19	48	15	136	218
Rocky	39	170	3	0	212
Gage	0	0	0	226	226

* R = All plants in the head row progeny resistant to WSBM

GMR = One or more plants infected but most plants resistant

MR = All plants with a mild infection

S = All plants severely infected.

(R); the progeny of no head selection was totally susceptible. However, 3 heads (1.4 percent) had all plants with a mild infection and were characterized as moderately resistant (MR); 170 (80.2 percent) of the head row progeny showed a few plants infected within the row and were classed as segregating moderately resistant (GMR). The GMR reaction of head row progeny of Rocky is illustrated in Fig. 6.

This GMR reaction of the selected heads from Centurk and Rocky reinforced the strong relationship between genetic and environmental factors on the manifestation of the disease expression.

As wheat is a self-pollinated crop, it is expected that each head progeny selected would have a uniform reaction to the disease. This was not the behavior of Rocky and Centurk. It is suspected that all plants may be infected with virus and that some plants show clear symptoms while others maintain the normal green color, depending upon certain unknown factor(s) of the environment.

We need more information on the etiology of the virus and its vector in relationship to the host in order to better understand the conditions in which the wheat plants are infected and show symptoms.

The susceptible cultivar Gage was homogeneous in its reaction to WSBM. This was evidenced by the uniformity of infection of all 226 head row progeny of Gage selected heads.

II- Observations of F₁ plants

Table 3 shows the reaction to WSBM of the F₁ plants from crosses made in 1979 with the objective of studying the possible cytoplasmic inheritance related to disease response.

The small number of seed set and winter killing of some of the space seeded plants reduced the number of F₁ surviving plants.

All F₁ plants from the crosses Gage/Centurk, Centurk/Gage and Centurk/KS73256 were susceptible to WSBM. Through the head selection study it was evident that Centurk was a mixture of resistant and susceptible genotypes. So it is suggested that the Centurk plants used in these crosses were susceptible to the disease.

Gage/KS73256 (Table 3) gave 1 resistant and 3 susceptible F₁ plants. As KS73256 showed about 5 percent of the population to be susceptible to WSBM, it is probable that a susceptible plant was used as male parent in the cross. However, if so, the resistant plant obtained was unexpected. The selfing hypothesis cannot be accepted because Gage is susceptible to WSBM. An eventual escape is a possibility or maybe the plant was infected but the symptoms were not visible.

KS73256/Gage gave 6 resistant and 4 susceptible F₁ plants, that is close to a backcross ratio. The KS73256 plant used in this cross might have been heterozygous for WSBM reaction.

KS73256/Centurk gave 23 susceptible and 2 resistant plants. If this KS73256 plant was also susceptible, as evidenced by the great number of susceptible F₁ progeny, then escape was the only

Table 3. F_1 field reaction to WSBM from crosses of 3 winter wheat cultivars. Riley County, Kansas. 1979-1980.

Cross	Observed Plants*			Total
	R	MS	S	
Gage/Centurk	-	-	2	2
Centurk/Gage	-	2	14	16
Gage/KS73256	1	1	2	4
KS73256/Gage	6	-	4	10
Centurk/KS73256	-	-	9	9
KS73256/Centurk	2	-	23	25

* R = Resistant

MS = Moderately Susceptible

S = Susceptible

possible explanation for the resistant plants. At maturity the F_1 plants will be checked for glume color and other characteristics in order to make sure that they were true crosses.

The susceptible and resistant F_1 plants from a single cross could have been caused also by some kind of reaction similar to the cultivar Rocky which was segregating for resistant and moderately resistant plants within most of the selected heads.

Although more extensive reciprocal crosses, including backcrosses for at least two generations should be done in order to confirm or refute the extra chromosomal inheritance to WSBM reaction, it appears from this study that there is no true cytoplasmic effect involving the reaction to the disease among these winter wheat cultivars.

It is believed that some of the reported differential F_1 reactions were caused by using parental plants that were genetically different for WSBM response. The Centurk head selection study supports this hypothesis.

III- F_2 , F_3 and F_4 Generations Study

The segregation ratio of F_2 populations and F_3 - F_4 lines used in this study suggested that WSBM reaction is controlled by one or two genes, depending on the cultivar and the specific parental wheat plant.

Resistance was dominant or partially dominant over susceptibility. This result agrees, in part, with the studies reported by Shaalan et al. (39) and Dubey et al. (9), but does not support the findings of Modawi (30) and Nakagawa et al. (31) who reported three

genes controlling WSBM reaction.

Factors such as diversity of virus strains, different host response and changes in environmental conditions over years and locations might cause the apparent contradictory results observed on some of the studies related to the inheritance of wheat soilborne mosaic.

The decision to study the reaction to the disease on F_2 plants and F_3 - F_4 lines in the same year and location was made in one attempt to clarify some suggestions that modifying factors could alter the segregation ratio of the F_3 or subsequent generations in relation to the F_2 ratio (30, 39). However, despite of the excellent infestation of WSBM, the results obtained in the F_2 populations were as inconsistent that in some cases it was difficult to establish the relationship between the F_2 segregation ratio and that of later generations. Maybe this erratic response was caused by the fact that although the F_2 populations and the F_3 - F_4 lines were derived from the same crosses, most of the F_2 plants of this study came from different F_1 plants than that reported by Modawi (30). That is, the F_1 ancestors of the F_3 - F_4 generations were not all the same plants that gave the F_2 populations of the 1980 study. This fact together with the problem of small size of some populations and the difficulty of grading into a distinct class in some of the progenies studied, might have been responsible for some of these apparent discrepant results. Even resistant cultivars are not completely immune from the disease.

Although there was no problem in distinguishing between the resistant and susceptible lines, in some cases it was difficult to determine whether a line should be classified as truly susceptible or as segregating. Most of the susceptible lines, especially in the F_4 generation, showed resistant plants. In some cases an excess of susceptible lines and a less number of segregating lines were found than the expected. On the other hand, susceptible lines in less number and an excess of segregating lines were found than the expected. These observations suggested that segregating lines could have been classified as susceptible or vice versa and emphasizes the difficulty of conducting WSBM genetic studies.

Some F_2 plants were slightly infected in early growth stages and were classified as MR. However, as they showed good recovery, for statistical test they were grouped together with the resistant class. The MS and S plants made the susceptible class.

Crosses of resistant x resistant cultivars were reported to give always resistant progeny. Modawi (30) found some resistant lines in the progeny from two susceptible wheat cultivars suggesting transgressive segregation. However, in this study no resistant progeny was observed from cross of two susceptible parents.

Eagle/Gage and Gage/Eagle. The wheat cultivars Eagle and Gage are considered susceptible to WSBM, even though Gage is able to recover faster than Eagle.

The F_2 and F_3 field reaction of Eagle/Gage and Gage/Eagle crosses are listed on Tables 4 and 5, respectively. All 114 F_2 plants and the 112 F_3 lines from Gage/Eagle and reciprocal cross were susceptible to the disease. The F_3 progeny had a rate of recovering similar to Eagle but clearly slower than the Gage parent. Although in this study it was not possible to explain genetically the difference in WSBM response, there are clear indications that Gage can recover better than Eagle and may have factors for good recovery not present in Eagle.

Eagle/Centurk and Centurk/Eagle. All 117 F_2 plants and all 117 F_3 lines from the crosses Eagle/Centurk and reciprocal were susceptible to WSBM, Tables 4 and 5, respectively.

Centurk has been reported to be moderately resistant to WSBM. Nevertheless, through the head selection study it was clear that it is a mixture of genotypes with different expression to the disease. Therefore, the WSBM reaction of the progeny from the above crosses could be explained on the fact that the Centurk parents used were susceptible to the disease which when crossed with the susceptible Eagle gave all susceptible progeny.

It was observed that KS73256 is not totally resistant to WSBM and might be a mixture of resistant and susceptible genotypes like Centurk. Due to the fact that some of the parents used were heterogeneous for disease response, the remaining reciprocal crosses are not grouped but analysed individually for their reaction to the disease.

Table 4. F_2 field reaction to WSBM of crosses between winter wheat cultivars. Riley County, Kansas. 1979-1980.

Cross	Observed plants *		Expected ratio	χ^2	Range of P **
	R	S			
Eagle/Gage	0	61	-	-	-
Gage/Eagle	0	53	-	-	-
Eagle/Centurk	0	63	-	-	-
Centurk/Eagle	0	54	-	-	-
Eagle/KS73256	18	33	$\begin{Bmatrix} 9 : 7 \\ 1 : 3 \end{Bmatrix}$	$\begin{matrix} 9.10 \\ 2.88 \end{matrix}$	$\begin{matrix} <.01 \\ .10-.05 \end{matrix}$
KS73256/Eagle	73	10	(self)	-	-
Shawnee/Gage	22	10	3 : 1	.67	.50-.25
Gage/Shawnee	59	26	3 : 1	1.42	.25-.10
Shawnee/Centurk	55	28	3 : 1	3.38	.10-.05
Centurk/Shawnee	46	20	3 : 1	.99	.50-.25
Gage/Centurk	35	20	3 : 1	3.79	.10-.05
Centurk/Gage	0	68	-	-	-
Gage/KS73256	29	30	$\begin{Bmatrix} 3 : 1 \\ 9 : 7 \end{Bmatrix}$	$\begin{matrix} 21.01 \\ 1.21 \end{matrix}$	$\begin{matrix} <.01 \\ .50-.25 \end{matrix}$
KS73256/Gage	33	38	$\begin{Bmatrix} 3 : 1 \\ 9 : 7 \end{Bmatrix}$	$\begin{matrix} 30.80 \\ 2.75 \end{matrix}$	$\begin{matrix} <.01 \\ .10-.05 \end{matrix}$
Centurk/KS73256	17	37	1 : 3	1.20	.50-.25
KS73256/Centurk	11	41	1 : 3	.41	.75-.50

* R = Resistant; S = Susceptible

** P = Probability of a higher Chi-square value, Snedecor and Cochran (42).

Table 5. F_3 field reaction to WSBM of crosses between winter wheat cultivars. Riley County, Kansas. 1979-1980.

Cross	Observed lines*			Observed**		Expected ratio	χ^2	Range of P***
	R	Seg.	S	H	I			
Eagle/Gage	0	0	66	0	66	-	-	-
Gage/Eagle	0	0	46	0	46	-	-	-
Eagle/Centurk	0	0	61	0	61	-	-	-
Centurk/Eagle	0	0	56	0	56	-	-	-
Eagle/KS73256	5	43	12	5	55	1 : 15	.44	.75-.50
KS73256/Eagle	40	27	0	40	27	(self)	-	-

* R = Resistant; Seg. = Segregating; S = Susceptible

** H = Healthy; I = Infected

*** P = Probability of a higher Chi-square value, Snedecor and Cochran (42).

Table 6. F_4 field reaction to WSBM of crosses between winter wheat cultivars. Riley County, Kansas. 1979-1980.

Cross	Observed lines*			Observed**		Expected ratio	χ^2	Range of P***
	R	Seg.	S	H	I			
Shawnee/Gage	23	29	14	23	43	1 : 3	3.41	.10-.01
Gage/Shawnee	20	32	9	20	41	1 : 3	1.97	.25-.10
Sahwnee/Centurk	20	40	12	20	52	1 : 3	.30	.75-.50
Centurk/Shawnee	13	54	5	13	59	1 : 3	1.85	.25-.10
Gage/Centurk	13	38	6	13	44	1 : 3	.14	.75-.50
Centurk/Gage	0	16	17	0	33	-	-	-
Gage/KS73256	14	42	8	14	50	1 : 3	.33	.75-.50
KS73256/Gage	6	50	15	6	65	1 : 15	.59	.50-.21
Centurk/KS73256	7	28	8	7	36	1 : 3	1.74	.25-.10
KS73256/Centurk	5	28	8	5	36	1 : 3	3.58	.10-.01

* R = Resistant; Seg. = Segregating; S = Susceptible

** H = Healthy; I = Infected

*** P = Probability of a higher Chi-square value, Snedecor and Cochran (42).

Eagle/KS73256 and KS73256/Gage. The F_3 segregation ratio of resistant to susceptible lines from Eagle/KS73256 (data on Table 5) and the F_4 segregation of KS73256/Gage (Table 6), suggested that resistance to WSBM was controlled by two partially dominant factors. When tested for goodness of fit, the F_3 - F_4 progeny gave a genotypic ratio close to 1:15 resistant to susceptible lines. The Chi-square values were .44 and .59 with probability of a higher X^2 in the range of .75-.50 and .50-.25, respectively. Shaalan et al. (39) reported similar results on crosses between the wheat cultivars Ottawa/Bison.

The above segregation ratio is expected if we consider the following assumptions:

<u>Cultivars</u>	<u>Genotype</u>	<u>Reaction</u>
Eagle and Gage	aabb	Susceptible
KS73256	AABB	Resistant

Crossing the resistant KS73256 with a susceptible Eagle or Gage, we have:

AABB x aabb

F_1 AaBb - Resistant

		<u>F_2 reaction</u>	<u>F_3-F_4 reaction</u>
F_2	1 AABB	resistant	resistant
	2 AABb	resistant	3 resistant : 1 moderately susceptible
	1 AAbb	moderately susceptible	moderately susceptible
	2 AaBB	resistant	3 resistant : 1 moderately susceptible

4	AaBb	resistant	9 resistant : 6 moderately susceptible : 1 susceptible
2	Aabb	moderately susceptible	3 moderately susceptible : 1 susceptible
1	aaBB	" "	moderately susceptible
2	aaBb	" "	3 moderately susceptible : 1 susceptible
1	aabb	susceptible	susceptible

This reaction is possible if we consider two partially dominant factors A and B responsible for resistance to WSBM. The expected F_2 genotypic ratio of 1:2:1:2:4:2:1:2:1 was modified to 1 resistant : 14 segregating or moderately susceptible : 1 susceptible, by interaction between the two genes. Plants possessing either or both of the recessive factors (aa or bb) in homozygous condition will show symptoms of the disease.

The F_2 study (data given in Table 4) was conducted to clarify the speculated modifying factors. However, the results neither support nor entirely reject the hypothesis.

The F_2 progeny from Eagle/KS73256 gave 18 resistant to 33 susceptible plants. This ratio does not fit the hypothesis of 9 resistant : 7 susceptible due to the excessive number of infected plants. The data are closer to the 1:3 ratio. When tested for goodness of fit the X^2 was 2.88 with .10 and .05 probability of a higher Chi-square value.

The progeny from KS73256/Gage had 33 resistant and 38 susceptible plants. Again the susceptible plants outnumbered the resistant. The segregation ratio appeared to be close to a 7:9

ratio. Although, when tested for the 9 resistant to 7 susceptible ratio, it gave a X^2 value of 2.75 and P between .10 and .05. This result would support the hypothesis of two partially dominant genes responsible for resistance with modifying factors. Nevertheless, due to the inconsistency of the F_2 segregation, the digenic hypothesis was based most on the F_3 and F_4 segregation ratio.

KS73256/Eagle. The small number of susceptible plants compared to the resistant in the F_2 population (Table 4) suggested the possibility that KS73256/Eagle was not a cross. That suspicion was confirmed by the analysis of the F_3 segregation ratio, data given on Table 5, which gave 40 resistant and 27 segregating lines. There was no F_3 line from KS73256/Eagle cross completely susceptible to WSBM. In this study it was observed that KS73256 is not homogeneous for WSBM reaction. Therefore, the susceptible F_2 plants and the segregating F_3 lines could be the result of selfing and propagating the female parent KS73256.

Shawnee/Gage. Despite of the severity of WSBM infection in the 1979-1980 study, the Shawnee cultivar had a strong resistance and showed almost no infected plants. In the F_2 study of the cross Shawnee/Gage (Table 4) 22 resistant and 10 susceptible plants were observed. Tested for the 3:1 ratio it gave a Chi-square value of .67 with .50 and .25 probability of a higher X^2 .

The segregation ratio of the F_4 lines, given in Table 6, showed 23 resistant, 29 segregating and 14 susceptible. When combined the segregating and susceptible lines, the data gave a close fit to the 1:3 ratio. The calculated X^2 being 3.41 and P within the range of .10-.05, indicating that the resistance of Shawnee used in this cross was controlled by a single dominant gene.

Considering a single dominant gene A responsible for resistance and the recessive a conditioning susceptibility, then the resistant Shawnee would have the genotype AA and the susceptible Gage would possess the recessive alleles aa.

Crosses between Shawnee and Gage should give a F_1 progeny of Aa and be resistant. The F_2 generation would segregate into 1 AA : 2 Aa : 1 aa, giving 3 resistant and 1 susceptible plants. The F_3 and F_4 generations would give 1 resistant, 2 segregating and 1 susceptible lines, that is, 1 healthy : 3 infected. The data obtained on the F_2 and F_4 generations from Shawnee/Gage support the above hypothesis.

Gage/Shawnee. The F_2 segregation from Gage/Shawnee, given in Table 4, fits the hypothesis of 3 resistant to 1 susceptible plants. The Chi-square test was 1.42 with a probability between .25-.10 of a higher X^2 value.

Data of the F_4 generation from the cross Gage/Shawnee are given in Table 6 and shows 20 healthy and 41 infected lines which also fits the hypothesis of 1:3 genetic ratio. The calculated X^2

was 1.97 with a probability between .25-.10. This result agrees with the data obtained from the reciprocal cross and supports the same genetic hypothesis.

Shawnee/Centurk. In the F_2 study of the cross Shawnee/Centurk (data given in Table 4) 55 plants were resistant and 28 susceptible. This fits a 3:1 ratio. The Chi-square was 3.38 with P within the range of .10-.05. The F_4 generation study (data in Table 6) gave 20 healthy and 52 infected plants. Tested for goodness of fit for the 1:3 ratio the X^2 value was .30 with .75-.50 probability of a higher Chi-square. If we consider that the male Centurk used in this cross was susceptible to WSBM, then the Shawnee parent would have also a single dominant factor for resistance.

Centurk/Shawnee. The progeny study of the cross Centurk/Shawnee (data given in Tables 4 and 6) suggested the same system of inheritance of resistance to WSBM as the reciprocal cross. The F_2 population had 46 resistant and 20 susceptible plants, again close to the 3:1 ratio. The calculated X^2 was .99 with the range of P being .50-.25. The data of the F_4 generation study (given in Table 6) shows 13 healthy and 59 infected lines. The X^2 test for goodness of fit of the 1:3 genetic ratio was 1.85 and P within the range of .25-.10, indicating a good fit to the hypothesis.

Gage/Centurk. From the head selection study it was clear that Centurk is not homogeneous for WSBM reaction but rather a mixture of resistant and susceptible plants and maybe a third type that is intermediate with a behavior similar to Rocky is present. In the former crosses it is believed that the Centurk used was susceptible to WSBM. As Gage is homogeneous susceptible to the disease, the male Centurk used in this cross must have been a resistant plant.

The F_2 data from Gage/Centurk cross (given in Table 4) showed 35 resistant and 20 susceptible plants, similar to the Shawnee/Centurk cross and fits a 3:1 ratio. The calculated Chi-square being 3.79 with P within the range of .10-.05.

From 57 lines in the F_4 study (Table 6) it was observed 13 healthy and 44 infected lines. This result fits the 1:3 ratio and gave a X^2 value of .14 with P within the range of .75-.50.

Therefore, it is suggested that the resistance of the Centurk plant used in this cross was controlled by one dominant factor similar to that of Shawnee cultivar.

Centurk/Gage. Data in Table 4 show that all 68 F_2 plants were susceptible to the disease. The F_4 data (in Table 6) gave 16 segregating and 17 susceptible lines. As the F_2 population and the F_4 lines came from different F_1 plants, there is a possibility that these F_1 plants were different from each other and thus gave different progeny. Despite of the fact that no resistant

F_4 lines were observed, the result suggests that the segregating lines were brought about by some kind of genetic recombination. However, no definite genetic system could be found to explain this behavior other than some genetic-environment interaction known to play an important role in the WSBM expression.

Gage/KS73256. The ratio of 29 resistant to 30 susceptible plants in the F_2 population from the cross Gage/KS73256 (given in Table 4) had an indefinite pattern and suggested a 1:1 segregation ratio. When tested for the 9:7 ratio the X^2 value was 1.21 with P within the range of .50-.25.

However, the F_4 study (data in Table 6) showed 14 healthy and 50 infected lines. This agrees with the 1:3 genetic ratio and the calculated Chi-square was .33 with .75-.50 probability of a higher value.

Therefore, based on the F_4 segregation ratio, it is believed that one single dominant gene was responsible for the resistance of KS73256 used in the above cross.

Centurk/KS73256. The data given in Table 4 suggested that the F_2 population of Centurk/KS73256 segregated 1 resistant to 3 susceptible plants. When tested for goodness of fit the above ratio, it gave a X^2 value of 1.20 with P within the range of .50-.25.

The F_4 lines appeared to segregate into the same 1:3 ratio. From Table 6, we observed that 7 lines were healthy and 36

infected. The calculated X^2 was 1.74 and P within the range of .25-.10, indicating a good agreement with the hypothesis.

KS73256/Centurk. In Table 4 the data of the F_2 study from the cross KS73256/Centurk are given. Eleven resistant and 41 susceptible plants were counted. This fits the hypothesis of 1:3 ratio and the Chi-square was .41 with P within the range of .75-.50.

The F_4 study of the cross KS73256/Centurk (data in Table 6) suggested also the same 1:3 genetic ratio. From a total of 41 lines 5 were healthy and 36 infected. The calculated X^2 was 3.58 and P within the range of .10-.05. Although P is within the 5 percent range of acceptability, the excessive number of susceptible lines appeared to have contributed to the high Chi-square value. In fact, when tested for the 1:15 ratio the X^2 was 2.47 and $P = .25-.10$. The small number of lines being tested in this case may have contributed to these discrepant results.

A question arises as to which parent contributed the genes for resistance. As we don't have a clear idea of the genetic composition of KS73256 and as we know that Centurk is a mixture of susceptible and resistant genotypes, and KS73256 is generally more resistant than Centurk, it is speculated that the resistance of the progeny from this cross was provided by KS73256. Based on the F_4 lines it is suggested that a single dominant gene was responsible for resistance. However, due to the unique behavior of these two wheat cultivars, this conclusion is only a tentative

and further studies, along with the etiology of the disease are necessary in order to have a better idea of their mode of inheritance to wheat soilborne mosaic.

SUMMARY AND CONCLUSIONS

A field study was conducted involving 5 winter wheat cultivars and their progeny with the objective of further elucidate the inheritance of reaction to wheat soilborne mosaic.

Through the observation of the reaction of 656 heads selected from Centurk, Rocky and Gage seeded on WSBMV infested area it was concluded that a wheat cultivar can be homogeneous for agronomic characters but heterogeneous for WSBM reaction. Centurk is a mixture of resistant and susceptible lines; most of the Rocky selected head progeny showed resistant and moderately resistant plants within the lines; Gage was homogeneous susceptible to the disease.

The F_1 study, although not conclusive, failed to show extra chromosomal effects for disease reaction. Some of the reciprocal differences in WSBM response could be explained by the heterogeneity of the parents utilized.

The fact that the 1980 F_2 population and the F_3 - F_4 generations did not have common F_1 ancestors was believed to be the major factor for the lack of correlation between the response of some F_2 populations and the segregation in subsequent generations.

Resistance in the parents was found to be simply inherited and was monogenic dominant or digenic partially dominant over susceptibility, depending on the cultivar and/or specific reaction of the parental plant used. Crosses between susceptible x susceptible cultivars gave all susceptible progeny. The response of F_3 - F_4 lines and part of the F_2 populations from the crosses Shawnee/Gage, Gage/Shawnee, Shawnee/Centurk, Centurk/Shawnee, Gage/Centurk, Gage/KS73256, Centurk/KS73256 and KS73256/Centurk can be explained by assuming a single dominant gene conditioning resistance to the disease.

The F_3 - F_4 lines from two crosses, Eagle/KS73256 and KS73256/Gage segregated into 1:15 genetic ratio of resistant to susceptible. This can be explained by assuming two partially dominant genes A and B responsible for resistance. The 1:2:1:2:4:2:1:2:1 genetic ratio was modified by the action of the recessive alleles (aa, bb) which caused susceptibility when in the homozygous condition. If this is true, then these modifying factors were only carried out when the resistant parent was KS73256. However, the difficulty in differentiating between the distinct classes may have contributed to these conflicting results. More studies, utilizing larger number of lines, are recommended in order to clarify this matter. Also more work on the etiology of the virus and vector associated with genetic studies would be of great value in trying to answer some of the questions related to this important wheat disease in Kansas.

This investigation supports, in part, the report of Shaalan et al. (39) that described two partially dominant genes responsible for resistance and also Dubey et al. (9) that reported one dominant gene for resistance. But disagree with the tri-genic system described by Nakagawa et al. (31) and Modawi (30). Differences in virus strains, cultivar response and mainly environmental factors may have caused these contradictory results.

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GENETIC STUDIES OF FIELD REACTION TO
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by

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ABSTRACT

Progeny from crosses of 5 winter wheat cultivars, along with selected heads of three cultivars, were studied for their field reaction to wheat soilborne mosaic. The parents used in the crosses were Shawnee, KS73256, Centurk, Gage and Eagle. The head selections were made in the cultivars Centurk, Rocky and Gage. Shawnee and KS73256 were considered resistant; Centurk, moderately resistant; Gage and Eagle are susceptible to the disease.

From the analysis of head row progeny it was concluded that the Centurk cultivar is a mixture of resistant and susceptible genotypes; Rocky had good level of resistance but showed a few infected plants within most of the rows; Gage was homogeneous susceptible to WSBM.

Observation of F_1 progeny of Centurk, Gage and KS73256, crossed among each other, including reciprocals, failed to show extra-chromosomal effects for WSBM reaction. It was concluded that the differential F_1 response was caused by the heterogeneity of the cultivars used as parents.

Crosses between susceptible x susceptible cultivars gave all susceptible progeny. The analysis of progeny from crosses between resistant x susceptible genotypes suggested that the resistance in the parents was simply inherited. Most segregating populations suggested a monogenic dominant factor and in some cases digenic factors for partially dominance of resistance.

The segregation ratio of the progeny from Shawnee/Gage, Gage/Shawnee, Shawnee/Centurk, Centurk/Shawnee, Gage/Centurk, Gage/KS73256, Centurk/KS73256 and KS73256/Centurk indicated that a single dominant factor was responsible for resistance to the disease.

Two partially dominant factors A and B were assumed to be responsible for WSBM resistance of the progeny from Eagle/KS73256 and KS73256/Gage. The F_2 genetic ratio was modified by the action of the recessive alleles (aa, bb) which caused susceptibility when in the homozygous condition.