1982

Genetics of Resistance to Hessian Fly Derived from Dawn Winter Wheat in Spring Wheat Crosses

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GENETICS OF RESISTANCE TO HESSIAN FLY DERIVED FROM DAWN WINTER WHEAT IN SPRING WHEAT CROSSES

BY

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A thesis submitted
in partial fulfillment of the requirements for the

degree Master of Science
Major in Agronomy

South Dakota State University
1982

GENETICS OF RESISTANCE TO HESSIAN FLY DERIVED FROM DAWN WINTER WHEAT IN SPRING WHEAT CROSSES

This thesis is approved as a creditable and independent investigation by a candidate for the degree, Master of Science, and is acceptable for meeting the thesis requirements for this degree. Acceptance of this thesis does not imply that the conclusions reached by the candidate are necessarily the conclusions of the major department.

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To Kathy Sellers who without her continual support and encouragement this project would never have been completed. It has been a great privilege and honor working with her these past four years. I thank you for your willingness to share your knowledge, and the joy you have brought to me in this profession.

ACKNOWLEDGEMENTS

The author wishes to express her sincere appreciation to Dr. Fred A. Cholick, my thesis advisor, for the patience, friendship, and valuable guidance he has provided in the preparation of this paper and to Dr. Don L. Keim for his guidance in the early phases of this research.

Special thanks are expressed to Dr. Jim H. Hatchett, USDA-ARS, for providing facilities and Hessian flies necessary for the screening of the large numbers of plants in this study. Appreciation is also extended to Ken Oppenlander for his proficient management of the experimental materials. This work would not have been possible without their total cooperation and guidance in the screening of plants.

Thanks to Dr. Charles L. Lay, my major advisor, Dr. Maurice L. Horton, Dr. David D. Walgenbach, and Dr. Duane Sander for their critic of this paper and for serving on my committee.

Appreciation is also extended to Mary and Valerie Sellers for their many long hours of dedicated work in the care and classification of plants.

The author is grateful for the financial assistance provided by the South Dakota Wheat Commission and to the USDA for their specific cooperative agreement.

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INTRODUCTION

Hessian fly, Mayetiola destructor (Say), has been a serious insect pest of common wheat (Triticum aestivum L. em Thell.) since it was first identified in the United States over 200 years ago (32). Losses are difficult to estimate due to geographical variability in populations but are large enough to characterize this pest as one of economic importance. Dahms (18) in 1967 estimated the direct average annual loss in the United States at 15 million bushels. The last serious outbreak in the United States was reported in 1978 in the spring wheat producing regions of north central and northeastern South Dakota where conservative loss estimates exceeded 10 million bushels (90).

As a result of the outbreak in South Dakota spring wheats, all lines in the spring wheat breeding program were screened for Hessian fly resistance. Resistance was found in spring by winter crosses involving 'Dawn' winter wheat. The resistance appeared to be operating as a single dominant gene based upon reactions of F_2 derived families.

The objective of this study was twofold:

1) to characterize the inheritance of resistance to Hessian fly derived from Dawn winter wheat in crosses with spring wheats;

2) to determine which gene(s) in Dawn winter wheat confer resistance to Hessian fly.

REVIEW OF LITERATURE

The Hessian fly and Its Hosts

The Hessian fly is a member of the Cecidomyidae midge family. The egg is deposited by an adult female on the upper suface of younger leaf blades in the longitudinal grooves. It is minute (0.4 to 0.5mm in length), cylindrical, glossy, translucent, and a pale yellowish red in color. Free moisture is essential for hatching and survival of emerging larvae and is a major factor in egg hatch variability which lasts from 4 to 12 days (46, 49, 61).

The larval stage causes plant damage and has been extensively studied. First instar larva orients its direction of movement opposite its position in the egg and begins migrating down the leaf to the base of the plant. Only the second instar larva feeds on the plant with its head pointed downward between the culm and leaf sheath (46). The second instar larvae feed on liquid plant content. Refai (73) was able to demonstrate by both visual and audiometric means that the larva obtains food through an intermittant sucking action. Asavanich and Gallun (5) have found that the larva must feed on susceptible plant seedlings for at least three days before the plant is affected and for five days before permanent stunting results.

At this time the old larval skin hardens and becomes reddish-brown in what is called a flaxseed (actually a puparium). The third instar larva overwinters in a puparium in infested plants, plant debris,

and on the soil surface. It can survive dryland conditions and temperature extremes of -27 to 38°C. In some cases the flaxseed has remained viable for three years (48, 49).

Pupation occurs within the flaxseed very early in the spring. The pupa is pearly white changing gradually to a reddish color. The wing pads become black and the abdomen blackish; the female's abdomen becomes tinged with red due to the presence of eggs. One to six days later the adult fly will emerge if humidity is relatively high, greater than 60%, and temperatures are moderate, 16 to 21°C (49, 91).

The adult fly is a mosquito-like, long-legged, dark-colored insect which lives two to three days without feeding. The female is slightly larger than the male, and the ovipositor is conspicuous. Initial spring emergence generally occurs when spring wheat is at the one to three leaf stage. The actual date varies with temperature, humidity, and latitude. Adult flies mate almost immediately after emergence, and the female begins ovipositing shortly afterward (49). The adult is fragile and incapable of long flight; hence, wind dispersion is a major factor in movement. A study conducted in Kansas showed that adult females were carried up to two miles with no injury. Males were never found more than 30.5 meters from where they had emerged (47).

The entire life cycle can be completed in 28 days if temperatures are moderate, 16 to 21°C, and humidity is above 60%. Each female is capable of depositing from 25 to 388 eggs (49, 76, 92). With the possibility of three to five generations each crop season depending

upon geographical location, it becomes apparent how quickly the population can increase given favorable climatic conditions.

In Kansas, where the Hessian fly has been studied extensively on winter wheats, there are two principal generations, namely, the spring brood from overwintering puparia and the fall brood which emerges from mid-September through October (49). In the South Dakota infestation which occurred in 1978 on spring wheats there was an initial spring brood in early May, a second brood in mid-June, and a partial third brood infesting volunteer spring wheat (90). A small portion of each generation remains in the flaxseed to emerge at a later date. For this reason adults emerging during any period may represent two or more generations (49).

The principal host of economic importance is common bread wheat (Triticum aestivum L. em Thell); however, the Hessian fly is able to feed on many different species of the tribe Hordeae. The insect can develop on certain varieties of barley (Hordeum) and rye (Secale) (13, 65). Little barley (Hordeum pusillum) is one of the foremost native alternate grass hosts and is prevalent in most wheat producing regions. Other commonly reported grass hosts are species of Aegilops, Agropyron, Bromus, Elymus, Lolium, Phleum, and Agrostis (41, 42, 43).

Reaction of Susceptible Wheat Cultivars to the Larval Feeding of the Hessian fly

Injury to susceptible wheat plants is caused by the feeding of the second instar larva between the leaf sheath and culm. Characteristic

symptoms of fly damage are the stunting of leaves after infestation and retardation of younger leaf initials (17, 65). Asavanich and Gallun (5) found that duration of feeding by the larva directly influences the degree of stunting. Two days of feeding or less did not cause significant stunting of susceptible plants. Larvae feeding for three days had an inhibitory effect, but no permanent plant damage resulted; whereas, four or five days of feeding caused permanent stunting of seedlings. Byers and Gallun (8) found that the most likely cause of stunting of winter wheats is due to a toxic secretion by the larva as it feeds. They found more plant growth inhibitors present in infested plants than in uninfested plants. The identity of the inhibitory substances secreted by the larva is unknown; however, Refai (72) found biochemical factors involved in the resistance reaction of the wheat plant. In vitro studies showed that larvae secrete hemicellulase as well as a substance which caused a decrease in plant phosphorylase action. There was a direct relationship between the hemicellulose content of the plant and the degree of resistance.

The unnatural deep bluish-green color of infested plants is believed to be due to increased chlorophyll concentration as a result of stunted growth. Miller et al. (53) found that younger stunted leaf initials had a higher percentage of the lipid soluable pigments chlorophyll, carotene, and xanthophyll than the older, outer leaves of infested plants or uninfested control plants. Further evidence of increased chlorophyll content in central leaves of infested plants was

presented in a study of chloroplast numbers by Robinson et al. (75). The number of chloroplasts per gram of fresh weight was higher in stunted leaves than in uninfested leaves. Toxic secretions of the insect inhibited elongation of leaves, but chloroplast and chlorophyll production were unaffected.

A susceptible winter wheat seedling may be killed by the developing larvae or it may be so greatly weakened that it becomes more susceptible to disease and winterkill. The hard red spring wheat plant may die if infested when very young or it may attempt to recover by producing more tillers. If infested after jointing, the affected tiller will be weakened at the point of attack and may lodge before harvest (65). Painter (65) in 1951 estimated that grain yields may be reduced by 25 percent due to incomplete filling of the spike alone. Quality of the grain is greatly reduced by shrivelling even if the culm has not lodged.

Possible Mechanisms of Resistance of the Wheat Plant to Infestation by the Fly

Several investigations have been conducted in an attempt to determine the reason for the plant's resistance to Hessian fly infestation. The precise morphological or biochemical factor or factors which confer resistance are still unknown, but several hypotheses have been formulated. In a study using P^{32} labelled resistant and susceptible wheat cultivars, Gallun and Langston (23) found that the larvae fed on resistant wheats but only for a limited time. They suggested a number

of causes including repellant action of the plant, a toxic effect of the plant, deficiency of nutrients to the larvae, and morphological characteristics of the plant.

Earliest observations suggested that silica and ash content may have an effect on Hessian fly resistance. Enock (19) and Slingerland (80) suggested that wheat plants with coarse and siliceous stems enabled them to resist damage by the feeding larvae. McColloch and Salmon (50) showed that resistance could be obtained in susceptible cultivars when the plants were grown in Pfeffer's solution containing a small amount of sodium silicate. The degree of resistance increased with the amount of silica added to the solution. Haseman (30) found a direct relationship between ash content of the plant and level of infestation. The results were not significant and only three cultivars were tested. Refai et al. (72) refuted all previous findings relating to silica content when they found no significant correlation between the total amount of silica in the lower stem portion of the plant and degree of resistance. They did, however, find that cultivars which have tough sheaths and stem tissue are more resistant to the fly. Arrangement of silica deposits could contribute to this additional toughness. Miller et al. (54) found, although not conclusively, that silica is deposited in rod-shaped masses arranged in spaced rows on susceptible varieties which may allow the larvae to feed between rows of silica. They further suggested that in some resistant varieties there may not be enough space between silica deposits to permit unrestricted feeding.

At one time it was thought that resistance was related to cellulose concentration of the plant. Painter (62) counted the number

of pupae resulting from eggs laid on the outer to inner leaves and found that a decrease in larval survival occurred on outer leaves where cellulose concentration was higher.

Painter (65) later theorized that an enzymatic system may be involved whereby the larvae secreted some toxic or enzymatic substance that stopped plant growth. This was supported in a study by Refai et al. (72) when they found that higher levels of hemicellulose were positively correlated with resistance. They suggested that as the larvae first begin to feed they must secrete hemicellulase since resistant wheats must have sufficient hemicellulose to withstand a normal quantity of the insect's enzyme and still remain stiff. Evidence of toxic larval secretions was presented by Haseman (31) and Painter (65) when they studied larval feeding. While the toxic substance has not been identified, research continues to support evidence of larval secretions. Most convincing evidence was presented by Byers and Gallun (8). They showed by use of benzene extracts of wheat that uninfested plants contained fewer plant growth inhibitors than infested plants.

Morphological characteristics have not explained the basis for resistance. Jones (44) showed that some resistant wheat cultivars appeared to have coarser and more tightly spaced vascular bundles. He theorized that the larvae were physically killed by greater tissue pressure exerted by resistant plants. Anderson and Brown (4) in a study of characteristics of the wheat culm found no relationship between breaking strength, diameter of the culm, or weight of the culm and fly resistance. McColloch and Yuasa (46) reported that the larvae were affected by mechanical obstacles during their migration to the base of

the wheat leaf. Roberts et al. (74) studied leaf pubescence and found that it was responsible for both reduction in oviposition by the female and the survival of the larvae in both field and greenhouse studies.

The biochemical or morphological factor(s) responsible for Hessian fly resistance are still unknown, and to date, no attempt has been made to correlate these factors with any of the genetic systems identified in resistance sources. Painter et al. (63) suggested that the characteristics of resistant wheat plants involved the mechanisms of antibiosis, tolerance, and nonpreference. It becomes readily apparent that the factor(s) conferring resistance in wheat is highly complex and cannot be explained by an single plant characteristic.

Control Methods for the Hessian fly

Many methods of control work quite well in suppressing populations of the fly, especially on winter wheats. Earliest preventive measures involved planting after the "fly-free date" in the fall, burning the stubble and threshing debris, planting narrow trap strips, crop rotation, and application of fertilizer to encourage vigorous plant growth (49, 93). Later, treatment with insecticides such as coal-oil emulsion, lime and paris green, and Bordeaux mixtures were attempted on winter wheats (29). These first treatments met with varying degrees of success, but as the life history of the fly became better understood, control measures became more efficient and refined.

Cultural controls work well for winter wheats but are less successful for the protection of spring wheats. The single most important cultural control on winter wheat is to plant after the "fly-free date" which is calculated for each latitude and each state. Planting after this date will generally avoid most of the fall emergence of the fly as well as minimizing populations overwintering in growing winter wheats (18).

Other methods work well if used in combination. Crop rotation of wheat with a row crop reduces potential population increase. Deep plowing is effective for spring wheats; however, this method also increases the potential for soil erosion. The field must be plowed at least 6 inches deep in the fall or early spring to prevent the adult fly from crawling through the soil after emergence. Destruction of volunteer wheat is important in the fall since it provides an excellent host plant source for the fall generation of the fly (49, 76, 78).

Research has shown that application of higher levels of nitrogen fertilizer increases the loss due to the fly and is no longer recommended as a cultural control. Okigbo and Gyrisco (59, 60) concluded that the additional tillering resulting from higher levels of nitrogen fertilizer increases the number of infested plants when the period of fly emergence is prolonged. The greatest grain losses were observed at the highest levels of nitrogen while the lowest losses occurred where no nitrogen had been applied.

Insecticidal control has been effective for the control of the fall brood on winter wheat. Carbofuran, Phorate, and Disulfoton have

proven most effective (6, 7, 55). Use of insecticides is an alternative to late planting and use of resistant varieties. The cost of chemical control on wheat may be prohibitive.

Climatic conditions have been the most effective natural control. Generally, low humidity (less than 60%), hot dry winds, drought, heavy rains at the time of larval migration down the stem, and lack of snow cover adversely affect fly survival. Actual population reductions are difficult to determine, but field observations indicate that potentially destructive populations can be reduced to below economic thresholds by adverse climatic conditions.

Parasites and predators are a factor in natural control, but their populations are reduced in proportion to that of the Hessian fly. Platygaster vernalis Myers is the primary parasite attacking the spring generation of the fly (40); whereas, Platygaster hiemalis attacks the fall generation (45). These parasites are single-brooded and lay their eggs on the larva of the fly directly through the flaxseed. Many other parasites of minor importance have also been identified.

Recent studies have shown that effective control can be achieved by releasing the dominant but avirulent Great Plains (GP) biotype of the fly into a virulent race. Offspring of the GP fly x any other biotype will not be able to attack wheats having any source of genetic resistance. More research is needed for this type of autocidal control program, but preliminary results indicate a good potential for success (21, 22).

The most effective and economic control of Hessian fly has been the development and use of resistant cultivars. As early as 1792, Havens (32) reported that 'Underhill' was resistant to the fly. It was not until the 1930's that plant breeders began to actively incorporate resistance to Hessian fly into wheats (52).

Biotypes of the Hessian fly

Eight biotypes of the Hessian fly, GP, A, B, C, D, E (26), and J, L (85), are now present in fields in the United States. In addition, two other biotypes, F and G, have been isolated in the greenhouse (95). Biotypes are differentiated from one another solely on their ability to infest and develop on wheat cultivars having different genes for resistance (Table 1). The evolution of new biotypes is concurrent with the use of resistant wheat cultivars. There is a complimentary relationship between wheat and insect; each gene for resistance in wheat has a complimentary gene for survival in the insect. Gallun and Hatchett (24) theorized that virulence, or the ability of a biotype to survive on a cultivar with a specific gene for resistance, is inherited in a recessive condition in the fly. This gene-for-gene specificity is similar to the flax and flax rust system described by Flor (20).

Biotypes of Hessian fly have been crossed and new biotypes derived in the laboratory to be used to screen for new sources of resistance before the biotypes evolve in the field. This type of work is made possible through the use of the single-egg-per-plant

Table 1. Virulence of specific Hessian fly biotypes on wheat cultivars with different genes for resistance.

Biotypes of Hessian fly	Wheat cultivars and genes for resistance				
	Triumph (None)	Seneca H_7H_8	Monon H_3	Knox 62 H_6	Abe H_5
GP	S†	R	R	R	R
A	S	S	R	R	R
B	S	S	S	R	R
C	S	S	R	S	R
D	S	S	S	S	R
E	S	R	S	R	R
F	S	R	R	S	R
G	S	R	S	S	R
J	S	S	S	R	S
L	S	S	S	S	S

† R = resistant; S = susceptible.

biotype purification technique developed by Sosa and Gallun (82) and the knowlege of paternal chromosome elimination (25).

The GP biotype possesses dominant avirulent genes which means it cannot attack cultivars with any gene for resistance. It is present in the Great Plains states and can infest only wheats, such as 'Triumph', which are universally susceptible. Biotype A is the predominant race in the eastern soft wheat region. Biotype B is the predominant race in Indiana and is present in the eastern and some midwestern states. Biotypes C and D have not yet been found in appreciable numbers in the field (26). Biotype E was first isolated in 1969 from samples of wheat collected in Georgia (34). Biotypes F and G were developed through selection in the greenhouse (95). Biotypes J and L were recently discovered in an Indiana wheat field (85).

There is tremendous genetic variability in the Hessian fly for virulence (25, 33). Using a resistant versus susceptible reaction to the Hessian fly (Y) and four wheat differentials (X), $Y^X = 2^4$ or 16 possible biotypes of Heesian fly may be classified (3). It seems likely that as acreage of cultivars with genes for resistance increases, there will be an increase in selection pressure favoring variants of the fly capable of surviving on these cultivars.

Resistance and Inheritance of Resistance in Wheat to the Hessian fly

In 1931, Painter et al. (63) presented the first evidence that resistance to the Hessian fly was heritable. It was not until 1936 that

resistance was determined to be under genetic control (14). As of 1981, twelve genes for Hessian fly resistance in wheat have been described and designated H_1 through h_{12}. Two other sources of indeterminant inheritance, 'Kawvale' and 'Marquillo', are recognized (Table 2). Genes conferring resistance, except for Kawvale and Marquillo, are qualitative in expression.

Cartwright and Weibe (14) in 1936 using the susceptible spring wheat cultivars 'Poso' and 'Big Club' in crosses with the resistant winter wheat 'Dawson' showed that Dawson possessed two dominant genes for resistance. In doing so they established the precedent of using F_3 families as a technique to determine F_2 progeny genotypes. Later, Noble and Suneson (57) identified two independent dominant genes and designated them as H_1 and H_2. Both genes were about equal in their ability to impart resistance but inferior to the combination of the two as in Dawson itself.

Separation of the dominant genes H_1 and H_2 into genetic testers made possible the designation of other genes for resistance. Noble et al. (56) established that gene(s) for resistance in 'W38' were different from those in Dawson when a cross of Dawson x W38 produced susceptible progeny in the F_3 generation. A more complete genetic analysis of W38 revealed an incompletely dominant gene which was designated H_3 (10).

Suneson and Noble (89) differentiated the resistance in 'Java' spring wheat from H_1, H_2, or H_3. They reported that resistance was due to a recessive gene and was designated as h_4. Allan et al. (2)

Table 2. Genes for resistance to Hessian fly, their source, and year designated.

Genetic Designation	Source of Resistance	Year Genetics of Resistance Designated
H_1H_2	Dawson (winter wheat)	1943
H_3	W38 (spring wheat)	1946
h_4	Java (spring wheat)	1950
H_5	Ribiero (spring wheat)	1953
H_6	PI 94587 (durum)	1959
H_7H_8	Seneca (winter wheat)	1973
H_9	Elva (durum)	1980
H_{10}	Elva (durum)	1980
H_{11}	PI 94587 (durum)	1980
h_{12}	Luso (common wheat-Portugal)	1981
Kawvale	Kawvale (winter wheat)	††
Marquillo	Iumillo (durum)	††

††Genetic basis for resistance has not been determined.

suggested that the h_4 factor of Java was an identical allele of or closely linked to the H_3 factor of W38.

The H_5 gene was differentiated by Shands and Cartwright (79) in the resistant spring wheat cultivar 'Ribiero'. This resistance was transferred to a Purdue release, 'Arthur 71', which at the time of its release was resistant to all biotypes of Hessian fly occurring in the field. Recent research using growth chamber tests indicates that H_5 resistance breaks down at high temperatures (83).

Allan et al. (2) in a genetic analysis of ten sources of Hessian fly resistance used in Kansas showed that a wheat derived from PI 94587 (durum) possessed a single partially dominant factor, H_6. Crosses involving the durum parent with H_3 and H_5 tester lines suggested there may be more factors for resistance in PI 94587. Caldwell et al. (11) crossed PI 94587 with three Ethiopian durum cultivars. They concluded from F_3 families that as many as four dominant genes may be present in PI 94587.

Patterson and Gallun (68) in tests with biotype E of the Hessian fly determined that resistance derived from 'Seneca' winter wheat was due to two partially dominant factors which they designated H_7 and H_8. These factors have not been separated from each other.

The resistance of 'Elva' (T. turgidum L. durum group) was transferred to three common wheat lines. Resistance appeared to be controlled by two linked dominant genes in two lines and a single dominant gene in the third, Purdue 822-34 (12). Stebbins et al. (86) concluded that 822-34, now released as 'Ella' common wheat germplasm

line (70), possessed a single dominant gene designated as H_9. In further investigations of these derived wheat lines, Stebbins (87) found that line 812-24, now released as 'Stella' germplasm line (70), possessed two dominant genes, H_9 and H_{10}, and that they were independently inherited.

A second dominant gene from PI 94587 durum was transferred to three Purdue soft red winter wheat selections. This gene was designated H_{11} and may be a non-adjacent duplicate of H_5 (87).

The h_{12} gene was recently identified in 'Luso', a common wheat cultivar from Portugal. It is the second resistance gene expressed in a recessive manner, the other being h_4. In the analysis of F_1 plants of the cross 'Abe' (H_5) x Luso, it was concluded that the effectiveness of resistance of the H_5h_5 genotype is significantly enhanced by the presence of the h_{12} gene in the heterozygous condition (58).

Patterson and Gallun (69) presented the first evidence of linkage of genes governing resistance to Hessian fly when they determined that the H_3 and H_6 genes were linked 9.0 map units apart on chromosome 5A (28). H_3 and H_9 are linked 15.5 map units apart (86) and H_6 and H_9 are linked 2.02 ± 2.01 map units apart in Stella. Thus, the gene order on chromosome 5A of wheat is H_3-H_6-H_9 (87). Carlson et al. (12) postulated that H_9 and H_{10} are linked 36 map units apart; however, Stebbins (87) in a more comprehensive analysis refuted those findings. She did conclude that H_9 and H_{10} appeared on chromosome 5A but were greater than 50 map units apart. In further studies of testcross progeny, she determined that the transferred H_{11} gene was linked 4.396 ± 1.775 map units from the H_5 gene in Abe wheat.

The genetic factor or factors conditioning resistance in Kawvale winter wheat have not been determined (27). Kawvale as a source of resistance has not been extensively studied, but information available suggests there may be several interrelated genes involved. Painter (65) reported that in crosses between Kawvale and 'Tenmarq' (susceptible), resistance appeared to be dominant. The dominance of resistance in Kawvale was later confirmed by Suneson and Noble (89), but they were not able to differentiate the genes responsible. Conversely, Allan et al. (2) reported the 'Pawnee', which has the Kawvale resistance, probably has two recessive factors conferring resistance to the GP biotype.

Marquillo is a spring wheat derived from the cross of "Iumillo' durum and 'Marquis' spring wheat. The resistance appears to be due to several different mechanisms: low larval survival (antibiosis), low oviposition, and tolerance (ability of plants to survive infestation) (64). Inheritance studies have shown that the Marquillo resistance behaved as a recessive and was complex (64, 89). It has been shown that some crosses involving Marquillo result in F_1 plants that die in the three to four leaf stage indicating the presence of one or more genes for lethality (9, 38, 64). Marquillo resistance has also been reported to be sensitive to temperature (51, 65, 67). Powers (71) has shown that Marquillo has greater cytological variability than some other wheats. It also has a more marked tendency to outcross in the field, and F_1 plants of some crosses involving Marquillo tend to be susceptible (64). These characteristics of the Marquillo type resistance further complicate attempts to characterize and identify

the resistance factors involved. Maas in 1982 (51) used 'Parker 76' (Marquillo resistance) and Marquillo in crosses with susceptible wheats to study the inheritance of the resistance. He ruled out theories of complete dominance and complete recessiveness as well as the possibility of one or two nondominant factors operating. Because of the heterogeneity in the F_3 tests, statistical comparisons to genetic models were not valid. There is still no conclusive evidence characterizing the Marquillo resistance.

Recently, Hatchett et al. (35, 36, 37) described at least three sources of resistance from Triticum tauschii (Coss) Schmal, formerly Aegilops squarrosa L., the donor of the D-genome in common wheat (77). The resistance has been transferred to synthetic hexaploid wheats. Preliminary results of inheritance studies indicate completely dominant resistance, but these have not yet been given 'H' designations. Sources of resistance from the D-genome donor of hexaploid wheats represent a relatively unexplored germplasm pool for broadening the genetic base of resistance to Hessian fly.

The Effect of Temperature on the Expression of Resistance

Temperature can affect the expression of resistanct to Hessian fly. Cartwright et al. (16) were the first to test specifically the effects of temperature when they grew plants under two temperature regimes (18 - 21°C and 24 - 27°C). On all cultivars tested, especially W38 (H_3), with the exception of PI 94587, the number of larvae per

infested plant surviving and the percentage of plants infested increased as temperature increased. The effects of temperature were similar for susceptible and resistant cultivars, but the relative magnitude of the effect was much smaller on susceptible cultivars. They were not able to determine whether the effects of temperature were due to the plant's response alone or to the combined response of both the host plant and the insect.

The phenotypic response of resistant plants is affected by host genes, Hessian fly biotype, length of time exposed to high temperatures, and the stage of plant growth when infested (84). Sosa and Foster (83) used biotypes GP, B, C, and D to infest wheats carrying the H_3, H_5, H_6, and H_7H_8 genes. Infestations increased with temperature regardless of biotype, but H_3 showed significant temperature sensitivity to GP biotype, and H_5 had a high level of resistance breakdown when attacked by biotype D at high temperatures. Wheats carrying the Marquillo resistance also showed a breakdown of resistance at higher temperatures (67).

The genotypic condition of the plant may allow the effects of temperature to be more pronounced. Painter et al. (63) and Abdel-Malek et al. (1) have shown that wheat plants heterozygous for resistance factors are affected more by temperature than plants homozygous for resistance. F_1 plants of resistant x susceptible lines had nearly as many larvae per plant as the susceptible parent.

MATERIALS AND METHODS

Dawn (CI 17801), a hard red winter wheat cultivar, was selected from a cross made by the Colorado Agricultural Experiment Station in 1970 (Co 701733) and subsequently released by South Dakota State University (94). Dawn was derived from the cross II21031/'Trapper'//Co 652363. II21031 is a Mexican spring wheat. The pedigree of Co 652363 is 'Warrior'/3/'Kenya 58'/'Newthatch'/2/2 * 'Cheyenne'/Tenmarq/'Mediterranean'/'Hope'/4/'Parker'. The Hessian fly resistance is apparently derived from Parker. The pedigree of Parker is 'Quivira'/3/'Kanred'/'Hard Federation'//'Prelude'/4/Kawvale/Marquillo//Kawvale/Tenmarq.

Experiment 1: Inheritance Study

Resistant parents were 3583 ('Eureka'/Dawn) and 3383 ('James'/Dawn). Both were F_5 spring wheat families derived from F_3 head selections. Selections were screen in the F_3 and F_4 generations for resistance to Hessian fly collected in South Dakota. Susceptible spring wheat parents were Eureka and James. All crosses including reciprocals were made by hand emasculation and the 'approach method' of pollination inside closed dialysis tubing.

Parents, F_1 plants, families from randomly selected F_2 plants, and families from randomly selected BC_1 plants were grown to maturity

in the greenhouse. Plants and families were bagged before anthesis to ensure self-pollination. The number of parent plants, progenies, and families tested are presented in Appendix Table 1.

Progenies were grown in 54 x 36 x 8cm wooden flats filled with prepared greenhouse soil mixture. Flats were fertilized twice during the testing period and sprayed with Bayleton to control mildew. Varying numbers of flats were tested at one time from November, 1981 through March, 1982 at Kansas State University, Manhattan, Kansas. Each flat had a resistant and susceptible parent row. In addition, flats of family rows had three resistant and one susceptible standard cultivar check row to determine uniformity of infestation. 'Larned' (H_3), 'Knox 62' (H_6), Seneca (H_7H_8), and Triumph were used. Flats and rows of progenies or families were randomized. F_1, F_2, and BC_1 plants were planted 10 rows per flat, 50 seeds per row.

Flats were grown in growth chambers at 20 ± 2°C with a 12-hour light period after infestation in the greenhouse. Family tests were grown in flats with 20 family rows per flat, approximately 25 seeds per row on benches in a temperature controlled greenhouse with supplemental light. Temperatures ranged from 7°C (night) to 24°C (day), but seldom exceeded 21°C for more than three to four hours.

Flats were infested with Hessian fly collected from an infested spring wheat field in Spink county, South Dakota, thus, the designation SD fly. The population used was the third generation increase from the field-collected population.

Methods of infestation and determination of resistance or susceptibility of individual plants were similar to those described by Cartwright and LaHue (15). A susceptible plant was identified by its stunted appearance and broadened deep bluish-green central leaf. Growth had essentially stopped. A resistant plant, although sometimes damaged, was continuing growth. All F_1, F_2, and BC_1 resistant progeny were examined under a stereoscopic microscope (30x) for presence of dead larvae. Phenotypically resistant plants with no dead larvae present were regarded as escapes and not included in the analysis. Chi-square analysis was used to compare observed with expected ratios for goodness of fit to one-gene, two-gene, or three-gene theories and tests of independence throughout the study (81).

Experiment 2: Identification of Gene(s) Conferring Resistance

Two separate techniques were used to identify which gene(s) conferred resistance in Dawn.

Part A. Winter x Winter Test

Individual plants of Dawn were identified as resistant in Kan Kansas and transplanted in South Dakota, vernalized, and increased. These selections of Dawn and winter wheat cultivars with known genes for resistance were planted in flats, vernalized, and transplanted into the greenhouse. Dawn was crossed with 'Newton' (None), 'Arthur' (H_3), Knox 62 (H_6), Seneca (H_7H_8), and Ella (H_9). Seed of parents, F_1, and

F plants was tested for resistance to SD fly. The flats were placed in a cold room at 2°C for five days following seeding to synchronize seedling emergence. When plants were 3 to 5cm tall, they were infested. After egg hatch and larval migration, flats were moved into growth chambers at 20 ± 2°C. Temperature in the chambers was reduced to 16°C after five days and then to 13°C six days later to slow growth of plants. When symptoms were clearly evident, resistance or susceptibility of plants was determined. Phenotypically resistant plants were examined microscopically to verify presence of dead larvae.

Part B. Reaction of Cultivars to Specific Biotypes

Two tests were conducted to determine the reaction of selected cultivars to specific biotypes. The first test had SD fly and biotypes A, B, and C. A second test used GP and D biotypes in addition to the biotypes in the first test. Biotypes of Hessian fly used, resistant and susceptible cultivars, and expected plant reactions are presented in Appendix Table 2. SD 8014 ('Coteau'/Dawn) and SD 8015 (Eureka/Dawn) are germplasm lines released by South Dakota State University (88). SD 8011 (James/Dawn) is an advanced breeding line.

Flats were planted 12 rows per flat, 50 seeds per row with a row of susceptible check cultivar ('Hyslop') in the first and twelfth rows. Flats and rows of cultivars were randomized. Four replications (one flat per replication) in each test were subjected to selected biotypes of the fly. Methods of infestation and determination of plant reaction were similar to those used in previous tests.

Experiment 3: Differentiation of SD fly and GP Biotype

It had been assumed that the SD fly was of the GP biotype population; however, differences in cultivar resistance were observed when infested with SD fly or GP biotype. A study was conducted to determine if the SD fly is a new biotype of Hessian fly or of the GP biotype. Data was supplied by Dr. Jim Hatchett, USDA-ARS, Kansas State University, Manhattan, Kansas from a routine screening of resistance of new cultivars identified in the 1981 Annual Wheat Newsletter (39).

One test involving SD fly and GP biotype was conducted using techniques decribed in experiment 2, part B. In this screening test there were two replications for each of the "biotypes" of Hessian fly.

RESULTS AND DISCUSSION

Figures 1-3 illustrate typical reactions of resistant and susceptible parents, F_3's, and progenies of BC_1 when infested with SD fly. It was observed that Eureka exhibited a much broader central leaf than did James (Figure 1). However, both parents had the stunted and dark bluish-green appearance typical of susceptible plants. Resistant parents were more vigorous than either susceptible parent. The etiolation of leaves was attributed to having been grown under greenhouse rather than field conditions. The phenotypic difference between susceptible and resistant plants has been described as a clear-cut distinction. However, differences were not distinct in the progeny examined in this study, and at times the classification of plants into resistant and susceptible classes became a matter of degrees. This becomes obvious with examination of resistant and susceptible F_3 plants of the crosses 3583/Eureka and 3383/James (Figure 2). As with the parents, the susceptible reaction of the BC_1 progeny was more pronounced in the Eureka cross (Figure 3).

A chi-square test of independence comparing the reaction of the two crosses, 3583/Eureka and 3383/James, in F_1, F_2, and BC_1 generations produced chi-square values of 49.13, 610.51, and 29.98, respectively (Appendix Table 3). Values of chi-square this large occur with a probability of <0.001 (one degree of freedom) indicating that the crosses had different reactions to Hessian fly infestation. Further evidence of the distinctiveness of the crosses came from the application of the same test to F_3 and F_2 BC_1 families (Appendix Table 4). Chi-square

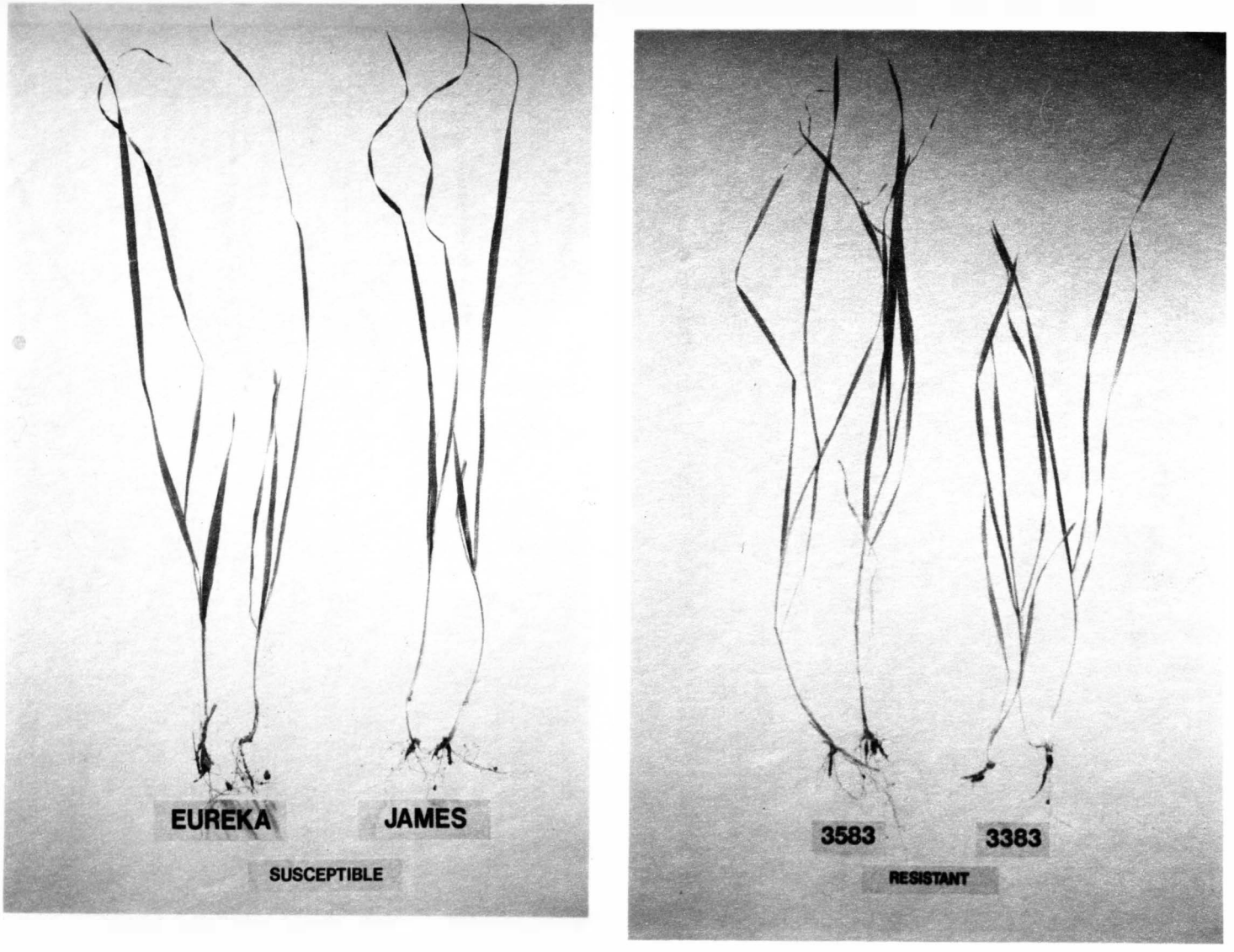

Figure 1. Phenotypic reaction of susceptible and resistant parents when infested with SD fly under greenhouse conditions.

Figure 2. Phenotypic reaction of resistant and susceptible F_3 plants of the crosses 3583/Eureka and 3383/James when infested with SD fly under greenhouse conditions.

Figure 3. Phenotypic reaction of resistant and susceptible BC_1 plants of the crosses 3583/2*Eureka and 3383/2*James when infested with SD fly under greenhouse conditions.

values of 2.55 and 42.19 were produced with associated probabilities of .50-.25 and <0.001, respectively (two degrees of freedom). Evidence that the two crosses were not independent occurred only in the F_3 families. Thus, it was assumed that factors other than the relationship between the crosses produced this non-significant chi-square value in the F_3 families test. Therefore, the two crosses were not pooled, and the reactions of the crosses will be presented and discussed separately.

Experiment 1: Inheritance Study

3583 and Eureka

No maternal cytoplasmic effects were detected in the reciprocal crosses of Eureka x 3583 in the F_1, F_2, or BC_1 using a chi-square test of independence (Appendix Table 5). Chi-square values of 1.99, 0.19, and 0.008 were produced with associated probabilities of .25-.10, .75-.50, and >.90, respectively (one degree of freedom). A similar test was conducted on the reciprocal crosses using F_3 and F_2 BC_1 families (Appendix Table 6). The test indicated reciprocal effects only in the F_2 BC_1 families with a chi-square value of 14.62 and associated probability of <0.001 (two degrees of freedom). The F_3 reciprocal families produced a chi-square value of .73 with an associated probability of .75-.50 (two degrees of freedom). Because evidence of reciprocal effects were found only in one of five tests, it was assumed that factors other than cytoplasmic effects produced this significant chi-square value. Therefore, plants from reciprocal crosses were pooled for

further analysis. Maternal inheritance appeared to have little or no effect on the progeny response to Hessian fly in these crosses.

Results of the F_1, F_2, and BC_1 tests are presented in Table 3. Infestation on these tests was light. There were 14% escapes in the susceptible check, Hyslop. In addition, 27% of the 3583 resistant parent plants and 10% of the Eureka susceptible parent plants were uninfested and considered escapes. The Eureka parent had 13.9% resistance in these tests; however, in the families' tests it had an average of only 3% resistance. It was assumed that the resistance exhibited by Eureka was due to the lighter infestation or faster growth habit of Eureka spring wheat where the migrating larvae may have been pushed out of the plant before becoming established. The percentage of susceptible plants (21.4%) in the F_1 also indicated that the resistance observed in the Eureka parent was not genetically induced.

Growing these tests in temperature controlled growth chambers eliminated the possibility that the susceptible reaction of some F_1 plants was temperature induced. The presence of susceptible phenotypes in the F_1 plants indicated that the resistance derived from Dawn is not inherited as a dominant gene. Likewise, the F_2 and BC_1 data do not support a dominance hypothesis because a higher level of resistance would be necessary (Table 3). A theory of complete recessiveness was also rejected based on the number of resistant plants in the F_1 and the level of resistance in the F_2 and BC_1. Complete recessiveness would require a ratio of 1 resistant:3 susceptible in the F_2 and a 1:1 ratio in the BC_1.

Table 3. Reaction of F_1, F_2, BC_1, parents, and check plants from the cross Eureka by 3583 to SD fly.

Progeny, parent, or check plants	Ratio Expected	Number of progenies or plants		% Plants Resistant	Assoc. X^2 Probability†
		Resistant	Susceptible		
Progenies:					
F_1: Total Observed		66	18		
Expected	1:0	84	0		.10-.05*
F_2: Total Observed		1197	464		
Expected	3:1	1245.75	415.25		.10-.001*
BC_1: Total Observed		48	83		
Expected	1:1	65.5	65.5		.005-.001*
Parents and check plants:					
Eureka		50	311	13.85	
3583		294	0	100.0	
Hyslop		3	651	.46	

† One degree of freedom.

The F_3 families and F_2 BC_1 families were classified into three catagories based on the proportions of resistant and susceptible phenotypes. Families having 90% or more resistant plants were class as 'R' denoting a homozygous resistant line. Families having 10% or fewer resistant plants were classed as 'S' denoting a homozygous susceptible line. Families having between 10% and 90% resistant plants were classed as 'H' denoting a segregating line derived from a heterozygous F_2 plant (Table 4). These same limits were used in a recent M.S. thesis study conducted at the same laboratory where these plants were tested (51).

Seed numbers were adequate to permit testing of a relatively large number of F_3 and F_2 BC_1 families (Appendix Table 1). There was an average of 28 plants per F_3 family and 25 plants per F_2 BC_1 family.

Combining the results of check plants from both families' tests, Triumph was 99% susceptible, Larned (H_3) was 90% resistant, and Knox 62 (H_6) and Seneca (H_7H_8) were greater than 98% resistant. These tests were more uniformly infested and as a result only 0.9% escapes were observed in the susceptible Triumph check rows.

Reactions of F_3 and F_2 BC_1 families supported the rejection of theories of complete dominance or recessiveness. The expected ratio 1:2:1 (R:H:S) for either theory was tested and the resulting chi-square values were 70.77 and 25.47, respectively. Chi-square values that large or larger would be expected with a probability of <0.001, two degrees of freedom (Table 4). Clearly some form of nondominance or complex inheritance is operating in this cross. Possible nondominance hypotheses will be discussed later.

Table 4. Reactions of F_3 and F_2 BC_1 families of the cross Eureka by 3583 when infested with SD fly under greenhouse conditions.

	Ratio Expected	Number of families			Assoc. X^2 Probability†
		R	H	S	
F_3 Families:					
Total Observed		60	187	11	
Expected	1:2:1	64.5	129.0	64.5	<0.001*
Expected	3:10:3	48.38	161.24	48.38	<0.001*
Expected	10:44:10	40.31	177.38	40.31	<0.001*
F_2 BC_1 Families:					
Total Observed		15	105	33	
Expected	1:2:1	38.25	76.5	38.25	<0.001*
Expected	3:10:3	28.68	95.63	28.68	.025-.01*
Expected	10:44:10	23.91	105.18	23.91	.05-.025*

† Two degrees of freedom.

3383 and James

Maternal cytoplasmic effects were detected in reciprocal crosses of James x 3383 in the F_2 plants and F_3 families (Appendix Tables 7 and 8). Chi-square values of 5.58 and 8.0 were produced with associated probabilities of .025-.01 (one degree of freedom) and .025-.01 (two degrees of freedom), respectively. Tests of independence for the F_1, BC_1, and F_2 BC_1 families produced chi-square values of .08, .04, and 1.6, respectively. The associated probability for the F_1 and BC_1 tests was .90-.75 (one degree of freedom) and .50-.25 for the F_2 BC_1 families. While the F_2 plants and F_3 families tests indicate the possibility of maternal effects, the hypothesis was rejected based upon the strong evidence of no maternal influence in the other three tests and the unpredictable resistance reaction of the 3383 parent. Therefore, data from the reciprocal crosses was pooled for analysis.

Results of the F_1, F_2, and BC_1 are presented in Table 5. Approximately 30% of the 3383 resistant parent plants and 17% of the James susceptible parent plants were uninfested and considered escapes. The 3383 resistant parent had only 71% resistance in these tests. Selections of 3383 were tested for resistance to SD fly as F_3's and F_4's, and it had been assumed that 3383 was homozygous resistant before crosses were made. Either 3383 underwent a breakdown in the expression of resistance, or it was not homozygous resistant. However, in the families' tests 3383 was 84% resistant which may have been indicative of undetermined environmental interactions in the progenies' tests. A higher percentage of susceptible plants was observed in all tests of

Table 5. Reaction of F_1, F_2, BC_1, parents, and check plants from the cross James by 3383 to SD fly.

Progeny, parent, or check plants	Ratio Expected	Number of progenies or plants		% Plants Resistant	Assoc. X^2 Probability†
		Resistant	Susceptible		
Progenies:					
F_1: Total Observed		16	68		
Expected	1:0	85	0		<0.001*
F_2: Total Observed		355	979		
Expected	3:1	1000.5	333.5		<0.001*
BC_1: Total Observed		15	145		
Expected	1:1	80	80		<0.001*
Parents and check plants:					
James		2	201	.99	
3383		128	52	71.11	
Hyslop		3	507	.59	

† One degree of freedom.

James x 3383 than Eureka x 3583 indicating the importance of the choice of parents when dealing with resistance derived from Dawn winter wheat.

Hypotheses of complete dominance or recessiveness of resistance were rejected based upon F_1, F_2, BC_1, F_3 families, and F_2 BC_1 families' results in the cross James x 3383 (Tables 5 and 6). In all cases chi-square values produced were large enough that the associated probability was <0.001. As in the cross Eureka x 3583, the results suggest some form of nondominance or complex inheritance.

Consideration of Nondominance Genetic Hypotheses

The interaction of environmental and hereditary factors is an important consideration in a nondominance hypothesis. Even though F_1's, F_2's, and BC_1's were grown in a temperature controlled environment, the F_3 and F_2 BC_1 families data must be considered more reliable indicators of possible genetic hypotheses if nondominance was operating. This is partially due to the unknown response of heterozygous plants under greenhouse testing conditions (51).

If a single nondominant factor was controlling the resistance, a 1:2:1 (R:H:S) ratio would be expected among the F_3 families (Tables 4 and 6). Chi-square values produced for Eureka x 3583 and James x 3383 were 70.77 and 223.4, respectively with an associated probability of <0.001 (two degrees of freedom). The large number of segregating families observed in the F_3 families rules out the possibility of a single nondominant factor acting alone. The number of susceptible families in each cross was also too small to support such a hypothesis.

Table 6. Reactions of F_3 and F_2 BC_1 families of the cross James by 3383 when infested with SD fly under greenhouse conditions.

	Ratio Expected	Number of families			Assoc. X^2 Probability†
		R	H	S	
F_3 Families:					
Total Observed:		35	150	12	
Expected	1:2:1	49.25	98.5	49.25	<0.001*
Expected	3:10:3	36.9	123.2	36.9	<0.001*
Expected	10:44:10	30.8	135.4	30.8	.01-.001*
F_2 BC_1 Families:					
Total Observed		1	56	75	
Expected	1:2:1	33	66	33	<0.001*
Expected	3:10:3	24.75	82.5	24.75	<0.001*
Expected	10:44:10	20.63	90.74	20.63	<0.001*

† Two degrees of freedom.

A two factor model was considered in which one major nondominant factor in the homozygous condition and a minor nondominant factor in either the homozygous or heterozygous condition would confer resistance. The expected F_3 and F_2 BC_1 families' ratios would be 3:10:3 (R:H:S). This hypothesis depends upon a large proportion of plants heterozygous for the minor factor and homozygous for the major factor conferring resistance (51). Chi-square values produced for the F_3 families were 35.78, Eureka x 3583, and 22.7, James x 3383. The chi-square values of the respective backcross F_2 families were 8.09 and 133.33. Associated probabilities were <0.001, <0.001, .025-.01, and <0.001 (Tables 4 and 6). Based on the reactions of these two crosses such a two factor model of nondominance was rejected.

A simple three factor model where any two of the nondominant factors in the homozygous resistant condition would confer resistance was considered. The model gives an expected F_3 and F_2 BC_1 families ratio of 10:44:10 (R:H:S) (51). With such a model higher levels of resistance would be expected in the F_2 and BC_1 plants than were observed (Tables 3 and 5). Chi-square values for the F_3 families of the Eureka x 3583 and James x 3383 crosses and their respective backcross F_2 families were 31.45, 13.62, 6.78, and 175.33. The associated probabilities were highly significant: <0.001, <0.001, .05-.025, and <0.001, respectively (Tables 4 and 6). Thus, this three factor model was rejected as an explanation of the inheritance of resistance factors derived from Dawn.

Based on the observed complexity of the resistance and earlier inheritance studies (2, 51, 64), it appears that the resistance derived

from Dawn winter wheat is some form of the Marquillo resistance. The results of this study do not permit us to determine if all the factors involved were fixed in Dawn. The greater degree of susceptibility found in the James x 3383 cross versus the Eureka x 3583 cross indicates that more factors were fixed in the Eureka cross. The differential reaction between crosses also supports the conclusion that Dawn derived its resistance from Marquillo. Painter et al. (64) reported differences in degree of resistance between crosses with Marquillo as well as within crosses as they were grown through six generations in the field.

It has been reported that the Marquillo resistance tends to be temperature sensitive (51, 67) and that F_1 plants tend to be susceptible (64). The temperature threshold of the Marquillo resistance has not been determined. If temperatures during testing exceeded the critical limit, a greater than expected number of phenotypically susceptible plants would result. This would account for some of the increased susceptibility in the James cross if the temperature threshold of the James cross was lower that the Eureka cross. Another possible explanation of the differences between crosses relates to the greater cytological instability that has been observed in selection of Marquillo (71). The author has observed greater variability than expected in crosses of Dawn with spring wheats. There was greater difficulty in fixing characteristics such as height, winter versus spring growth habit, and awned versus awnlessness. Advanced generation breeding material was still segregating for these characteristics when they would be expected to be fixed by the fifth generation.

'Ponca' winter wheat was derived from the cross of an F_3 plant of Kawvale/Marquillo to a sister selection of Pawnee (Kawvale resistance) from the Kawvale/Tenmarq cross. Painter et al. (66) reported that Ponca probably carried the Kawvale and the Marquillo resistance. Allan et al. (2) later reported three levels of resistance among ten Ponca selections. Dawn has this same Kawvale/Marquillo//Kawvale/Tenmarq cross in its parentage. Since the inheritance of this cross or of Kawvale has never been fully described, any number of indeterminable gene interactions may have taken place in conferring resistance to Hessian fly that was derived from Dawn.

To account for the possible effect of the spring growth habit on expression of resistance to Hessian fly, a winter x winter cross was made to serve as a control. One plant selection of Dawn (Dawn 3) was crossed to Newton (susceptible); the reciprocal did not produce sufficient seed for testing. Reactions of the F_1, F_2, and parents to SD fly are presented in Table 7. Dawn 3 was 100% resistant in this test and Newton was 100% susceptible. Hyslop, susceptible check, had 0.6% escapes.

The F_1 and F_2 reactions of this winter x winter cross also led to the rejection of any complete dominance or recessiveness hypotheses. If the spring growth habit had affected the expression of resistance, we would have expected a lesser degree of resistance in a winter x winter cross. Instead the results suggest, just as the spring x spring crosses, some form of nondominance or complex inheritance.

Table 7. Reaction of F_1 and F_2 plants from Dawn 3/Newton cross, parent plants, and check plants to SD fly.

Progeny, parent, or check plants	Ratio Expected	Number of progenies or plants		% Plants Resistant	Assoc. X^2 Probability†
		Resistant	Susceptible		
F_1 progenies:					
Total Observed		19	10		
Expected	1:0	29	0		.10-.05*
F_2 progenies:					
Total Observed		101	67		
Expected	3:1	126	42		<0.001*
Parents and check plants:					
Dawn 3		115	0	100.0	
Newton		0	105	0.0	
Triumph		0	89	0.0	

† One degree of freedom.

Experiment 2: Identification of Genes Conferring Resistance

Part A. Winter x Winter Test

Crosses were made with individual plant selections of Dawn and five winter wheats with known genes for resistance to Hessian fly: Arthur (H_3), Knox 62 (H_6), Seneca (H_7H_8), Ella (H_9), and Newton (None). Segregation in the F_2's would be expected in the Dawn 3/Newton cross. If segregation in the F_2's occurred in a cross of Dawn and any of the cultivars with known genes for resistance, it would indicate that the two parents did not have the same gene(s) in common.

An average of 0.6% Hyslop, susceptible check, plants were classified as escapes. Susceptible plants throughout the test had several larvae and flaxseed when infested with SD fly. Phenotypically resistant plants had 2 to 18 dead larvae as determined microscopically. Reactions of parent, F_1, and F_2 plants are presented in Table 8.

It was assumed that the SD fly was of the GP biotype population. Parents with any genes for resistance should have conferred 100% resistance to the SD fly or at least equal resistance. Since biotypes can be differentiated solely on the basis of their ability to survive on cultivars having different genes for resistance (27), this was our first indication that the SD fly was distinct from the GP biotype. Differentiation of these "biotypes" will be discussed later.

Phenotypically resistant and susceptible plants were observed in the F_1 plants except in the cross with Seneca where there were no susceptible plants. Crosses with cultivars with known genes for

Table 8. Reaction of parent, F_1, and F_2 plants in winter by winter crosses when infested with SD fly.

Parent or progeny (resistance)	Number of Plants		% Plants Resistant
	Resistant	Susceptible	
Dawn 3 (?)	115	0	100.0
Newton (None)	0	105	0.0
F_1	19	10	65.5
F_2	101	67	60.1
Dawn 2 (?)	112	0	100.0
Arthur (H_3)	84	44	65.6
F_1	18	3	85.7
F_2	36	18	66.7
Dawn 1 (?)	108	0	100.0
Knox 62 (H_6)	66	47	58.4
F_1	22	5	81.5
F_2	249	82	75.2
Dawn 8 (?)	111	2	98.2
Seneca (H_7H_8)	88	2	97.8
F_1	27	0	100.0
F_2	225	88	71.9
Dawn 7 (?)	103	0	100.0
Ella (H_9)	54	63	46.2
F_1	47	6	88.7
F_2	211	99	68.1

resistance should have produced F_1 populations with 100% resistance to SD fly if it was of the GP population.

Segregation of F_2 plants would normally indicate that different genes from each parent were conferring resistance. Tests of independence were conducted on the F_2 populations to determine if Dawn had any genes in common with the cultivars used in the crosses. Independence chi-square values of 11.43 and 6.40 were produced for the Knox 62 (H_6) and Seneca (H_7H_8) crosses versus the Newton (None) cross, respectively (Appendix Table 9). These values correspond to probabilities of <0.001 and .025-.01 (one degree of freedom) indicating no association between genes for resistance in these two crosses versus the Newton (None) cross. It can be concluded that Dawn does not possess either the H_6 or the H_7H_8 genes. Similar tests of independence did not differentiate the H_3 and H_9 genes from those in Dawn in these tests (Appendix Table 9).

Although this test was conducted in temperature controlled growth chambers, the temperature sensitivity of the incompletely dominant H_3 gene may have affected the resistance of the F_2 plants. The moderate resistance of Ella (H_9) to SD fly in this test made it impossible to differentiate the H_9 gene from the Dawn resistance in the F_2's. However, if conclusions were based upon the reaction of parents alone, all genes tested except H_7H_8 would be differentiated from the genes conferring resistance in Dawn.

Part B. Reaction of Cultivars to Specific Biotypes

Gene(s) for resistance to Hessian fly in a cultivar may be identified by comparing its reaction to reactions of cultivars with known genes for resistance when infested with different biotypes of the fly. The results of this test are presented in Table 9.

An average of 248 plants per specific biotype was infested. The level of infestation was excellent with an average of 0.4% escapes in Hyslop (susceptible check) rows. Data of the two tests was combined for discussion. Comparison of expected cultivar reactions (Appendix Table 2) with the reactions observed in this test (Table 9) showed that there was no deviation of observed from expected resistance reactions.

Determination of gene(s) conferring resistance in Dawn was done by comparing the percentage of resistant plants in any or all of the sources of Dawn resistance with the percentage of resistant plants of a specific cultivar to one specific biotype. Using this technique, gene(s) which Dawn could not possess were identified and eliminated from further consideration. More than one biotype may identify gene(s) not possessed by Dawn. In this particular test, SD fly and GP biotypes are used only as checks since any cultivar with resistance should be resistant to the avirulent GP biotype. They also served to check purity of seed used and to detect elevated temperatures in the test which would create breakdown of resistance in cultivars with temperature sensitive genes.

Comparisons of cultivar reactions when infested with biotype A indicated that Dawn does not possess H_7H_8 or Kawvale. Biotyp B

Table 9. Percentage of plants resistant to specific Hessian fly biotypes on cultivars with different genes for resistance.

Cultivars (resistance)	Biotypes of Hessian fly					
	SD	GP[1]	A	B	C	D[2]
James (None)[1]	--	3	3	5	0	--
Coteau (None)[1]	--	0	0	.5	0	--
Eureka (None)	1	16	2	15	1	2
Seneca (H_7H_8)	95	98	6	15	5	5
Monon (H_3)	84	99	63	4	93	.6
Knox 62 (H_6)	99.7	99	91	97	3	1
Ella (H_9)	72	98	59[1]	99[1]	30	93
Stella (H_9H_{10})[2]	--	--	99	100	--	--
Parker (Marquillo)[2]	97	87	59	4	67	52
Pawnee (Kawvale)[2]	96	69	9	12	10	6
SD 8011 (Dawn)	70	60	42	22	16	25
SD 8014 (Dawn)	82	81	75	33	51	59
SD 8015 (Dawn)	96	97	95	37	79	87
Dawn[2]	99.5	99	91	21	75	85

1. Data from first test only.

2. Data from second test only.

infestations presented weak evidence that Dawn does not have H_3, Marquillo, or Kawvale and good evidence that it does not have the H_6, H_9, or H_9H_{10} genes. Biotype C infestations indicated that Dawn does not have H_7H_8, H_3, H_6, H_9, or Kawvale resistance. Comparisons of infestations of cultivars to biotype D confirmed that Dawn does not have H_7H_8, H_3, H_6, H_9, or Kawvale resistance.

The inheritance study had indicated a complex form of nondominance operating in conferring resistance to Hessian fly derived from Dawn winter wheat. Comparisons of percentage resistant plants with Dawn resistance to cultivars with known dominant resistance genes confirmed these findings. Dawn apparently carries some or all of the resistance derived from Marquillo. Since SD 8011, SD 8014, and SD 8015 were in all probability derived from crosses with different individual Dawn plants, it cannot be concluded that the differential reactions of these lines were due entirely to the spring parent involved. It is more likely that the original Dawn parents varied in the number of factors for resistance derived from their Marquillo and/or Kawvale ancestry. SD 8015 had a greater percentage of resistant plants than SD 8011 in all cases. This confirms the results of the inheritance study since SD 8015 and SD 8011 were derived from the resistant parent lines used in the inheritance study.

Experiment 3: Differentiation of SD fly and GP Biotype

The SD fly was first collected in the field in 1978 in Spink county, South Dakota. It has, to date, not been found on winter wheats

to any degree; however, it is equally capable of infesting susceptible spring or winter wheats in the greenhouse. Preliminary observations in greenhouse screening tests indicated that the SD fly and GP biotype were of the same population. In a recent study of the Marquillo resistance using SD fly and GP biotype, Maas (51) reported that a chi-square test comparing the ability of the two fly cultures to infest F_1's produced a value of 0.42 with an associated probability of 0.50. At that level, any differences between fly cultures were assumed to be random. His study was conducted using the same facilities and fly cultures as utilized in this study.

The first indication of a difference in fly cultures was observed in the winter x winter test when Dawn resistance to SD fly was nearly 100%, but Arthur (H_3), Knox 62 (H_6), and Ella (H_9) were only 65.6%, 58.4%, and 46.2% resistant, respectively (Table 8). Results were similar in the biotype test when differences in the percentage resistant plants were observed for Monon (H_3) and Ella (H_9) when infested with either SD fly or GP biotype (Table 10). In this test Knox 62 (H_6) resistance to both fly cultures was nearly perfect. Parker (Marquillo) and Pawnee (Kawvale) also showed differential reactions to the SD fly and GP biotype. Eureka and SD 8011 continued to exhibit unpredictable reactions to infestation. Eureka had only 1% resistance to SD fly, whereas in the inheritance study, it had almost 14% resistance.

Differences between fly cultures could not be detected when using and source of Marquillo resistance, including any spring wheats derived from spring x winter crosses involving Dawn. However, because

Table 10. Evaluation of selected cultivars for resistance to the SD fly and GP biotype of Hessian fly.

Cultivars (resistance)	Biotypes of Hessian fly	
	SD	GP
James (None)	3†	--
Coteau (None)	0	--
Eureka (None)	1	16
Seneca (H_7H_8)	95	98
Monon (H_3)	84	99
Knox 62 (H_6)	99.7	99
Ella (H_9)	72	98
Parker (Marquillo)	97	87
Pawnee (Kawvale)	96	69
SD 8015 (Marquillo)	96	97
SD 8014 (Marquillo)	82	81
SD 8011 (Marquillo)	70	60
Dawn (Marquillo)	99.5	99

† Percentage of plants resistant.

of differences in resistance to the fly cultures observed in the biotype test, it became apparent that cultivars could be found that would differentiate the SD fly and GP biotype.

Information was provided by Dr. Jim Hatchett, head of the USDA Hessian fly project at Kansas State University, whereby we were able to present convincing evidence that the SD fly was distinctly different from the GP biotype in its ability to infest cultivars (Table 11). Once again it was noted that Dawn could not be used as a cultivar to differentiate the biotypes. Of the cultivars presented here, only the genetics of the resistance of Dawn winter wheat has been studied.

The SD fly can be called a distinct biotype only if homozygous progeny of the fly can be isolated that can infest a particular cultivar and those from the avirulent GP biotype population that cannot. In most instances where a difference in infestation has been observed, cultivars appeared to be more resistant to the SD fly than to the GP biotype. These results suggest that the SD fly is less virulent than the avirulent GP biotype. Two possible explanations may be postulated. The GP biotype has been present in Kansas for many years, and for many years there has been evidence of the presence of biotypes A, B, and C in Kansas wheat fields (26). The GP culture used in these studies may by evolving to a new biotype of the fly. Precautions are taken in the greenhouse laboratory to prevent intermating of the biotypes, but the GP culture is replenished with field collections on a regular basis. The frequency of virulent biotypes may have been greater than expected in the GP culture, since it had not been selected for avirulence to the

Table 11. Evaluation of selected cultivars for resistance to SD and GP biotypes of Hessian fly.

Cultivar	Biotype of fly SD	GP	Class	Origin
Sandy	85[2]	33	HRW	Colorado
PB 835	56	7	HRW	Northrup King
Tam W-106	59	0	HRW	Texas
Mit	56	0	HRW	Texas
Dawn	74	77	HRW	South Dakota
MD 55-286-21	28	0	SRW	Maryland
ND 575	86	0	HRS	North Dakota
ND 585	80	0	HRS	North Dakota
Centa	44	0	HRS	South Dakota
Martonvasari 4	56	3	HRW	Hungary
Fargo	33	0	Durum	North Dakota

1. Screening conducted by Dr. J. H. Hatchett, USDA-ARS, Kansas State University, Manhattan, KS for Dr. R. L. Gallun, Leader of Hessian fly investigations, Purdue University.

2. Percentage of resistant plants.

Marquillo resistance. This does not, however, account for differences observed when cultivars with other resistance genes were used.

A second and less likely explanation is that the Hessian fly infesting spring wheats in South Dakota and now in Montana and Washington (37) is the true avirulent biotype. Populations of the spring wheat infesting fly are necessarily small, even marginal, except in the occasional year that emergence of the first brood happens to niche well with the growth of spring wheat seedlings, and climatic conditions are favorable for the development of the fly. Winter wheats are relatively unaffected by the fly because of the powerful controlling effect of climate on the fly. The cool, wet years that favor the survival of the fly also result in the planting of larger acreages of spring wheat. Thus, the life cycle of the fly is more in niche with the production of spring rather than winter wheats in South Dakota.

CONCLUSIONS

Wheat breeders are constantly searching for new sources of resistance to Hessian fly that can be incorporated into their breeding programs. The transfer of resistance from Dawn winter wheat into spring wheats represents the first attempt to develop resistant hard red spring wheats adapted to the Northern Great Plains. This study was initiated to characterize the inheritance of Hessian fly resistance derived from Dawn and to determine which gene(s) in Dawn confer the resistance.

Two F_5 head selections involving Dawn, designated 3583 and 3383, from the spring wheat breeding nursery at South Dakota State University identified as resistant to SD fly as F_3's and F_4's were crossed with susceptible spring wheats. Parents, F_1's, F_2's, BC_1's, F_3 families, and F_2 BC_1 families were screened for resistance to Hessian fly collected from South Dakota spring wheat fields. Reactions of progenies indicated that resistance was complex and some form of nondominance.

The resistance derived from Dawn was identified as a Marquillo type resistance through reactions of cultivars with identified genes for resistance to specific biotypes of Hessian fly. The complexity of the Marquillo type resistance observed in this study verified the results of Allan et al. (2), Painter et al. (64), and Maas (51).

When this study was initiated, it had been assumed that the SD fly was of the GP biotype population. Differential reactions of cultivars with known genes for resistance to the fly cultures indicated the possibility of two distinct biotypes. The SD fly has been found predominantly on spring wheats in South Dakota with little infestation of winter wheats. Further research into the differentiation of these populations will be required before the SD fly can be classified as a distinct biotype.

LITERATURE CITED

LITERATURE CITED

1. Abdel-Malek, S.H., E.G. Heyne, and R.H. Painter. 1966. Resistance of F_1 wheat plants to greenbugs and Hessian fly. J. Econ. Entom. 59:707-710.

2. Allan, R.E., E.G. Heyne, E.T. Jones, and C.O. Johnston. 1959. Genetic analysis of ten sources of Hessian fly resistance, their interrelationships and association with leaf rust reaction in wheat. Kans. Agr. Exp. Sta. Tech. Bull. 104:51pp.

3. Allard, R.W. 1956. Formulas and tables to facilitate the calculation of recombination values in heredity. Hilgardia 24:235-278.

4. Anderson, D.C. and H.M. Brown. 1936. Studies on Hessian fly infestation and some characters of the wheat culm. J. Am. Soc. Agron. 28:479-483.

5. Asavanich, A.P., and R.L. Gallun. 1979. Duration of feeding by larvae of the Hessian fly and growth of susceptible wheat seedlings. Annals Ent. Soc. Am. 72(2):218-221.

6. Biggar, J.H., P.E. Johnson, and R.O. Weibel. 1965. Controlling Hessian fly with Phorate and Disulfoton. J. Econ. Ent. 58(6):1083-1085.

7. Brown, H.E. 1960. Insecticidal control of the Hessian fly. J. Econ. Ent. 53:501-503.

8. Byers, R.A. and R.L. Gallun. 1972. Ability of Hessian fly to stunt winter wheat: paper chromotography of extracts of freeze-dried larvae and wheat plants. J. Econ. Ent. 65:997-1001.

9. Caldwell, R.M. and L.E. Compton. 1938. Wheat breeding for the combined resistance to disease and Hessian fly. Purdue Univ. Agr. Exp. Sta. Ann. Rept. 51:41-42.

10. Caldwell, R.M., W.B. Cartwright, and L.E. Compton. 1946. Inheritance of Hessian fly resistance derived from W38 and durum PI 94587. J. Amer. Soc. Agron. 38(5):398-409.

11. Caldwell, R.M., R.L. Gallun, and L.E. Compton. 1966. Genetics and expression of resistance to Hessian fly, Phytophaga destructor (Say). Proc. 2nd Int. Wheat Genetics Symp. Lund, 1963. Hereditas, Suppl. 2:462-463.

12. Carlson, S.K., F.L. Patterson, and R.L. Gallun. 1978. Inheritance of resistance to Hessian fly derived from Triticum Turgidum L. Crop Sci. 18:1011-1014.

13. Cartwright, W.B. 1922. Host plant selection by Hessian fly (Phytophaga destructor Say). J. Econ. Ent. 15:360-363.

14. Cartwright, W.B. and G.A. Weibe. 1936. Inheritance of resistance to the Hessian fly in wheat crosses Dawson x Poso and Dawson x Big Club. J. Ag. Res. 52:691-695.

15. Cartwright, W.B. and D.W. LaHue. 1944. Testing wheats in the greenhouse for Hessian fly resistance. J. Econ. Ent. 37:385-387.

16. Cartwright, W.B., R.M. Caldwell, and L.E. Compton. 1946. Relation of temperature to expression of resistance to Hessian fly. J. Econ. Ent. 65:997-1001.

17. Cartwright, W.B., R.M. Caldwell, and L.E. Compton. 1959. Responses of resistant and susceptible wheats to Hessian fly attack. Agron. J. 51:529-531.

18. Dahms, R.G. 1967. Insects Attacking Wheat in Wheat and Wheat Improvement. Am. Soc. Agron. Monograph. Vol. 13:411-443.

19. Enock, F. 1891. The life history of the Hessian fly, Cecidomyidae destructor (Say). Trans. Ent. Soc. of London 26:329-366.

20. Flor, H.H. 1956. The complementary genic systems in flax and flax rust. Adv. in Genet. N.Y. Academic Press 8:29-54.

21. Foster, J.E. and R.L. Gallun. 1972. Populations of the eastern races of Hessian fly controlled by the release of the dominant avirulent Great Plains race. Am. Ent. Soc. Am. 65:750-754.

22. Foster, J.E. 1977. Suppression of a field population of the Hessian fly by release of the dominant avirulent Great Plains biotype. J. Econ. Ent. 70:775-778.

23. Gallun, R.L. and R. Langston. 1963. Feeding habits of Hessian fly larvae on P^{32}-labeled resistant and susceptible wheat seedlings. J. Econ. Ent. 56:702-706.

24. Gallun, R.L. and J.H. Hatchett. 1968. Interrelationship between races of Hessian fly, Mayetiola destructor (Say), and resistance in wheat. Proc. Third Int. Wheat Genetics Symp. Canberra, 1968, Australia Acad. Sci. :258-262.

25. Gallun, R.L. and J.H. Hatchett. 1969. Genetic evidence of elimination of chromosomes in the Hessian fly. Ann. Ent. Soc. Am. 62(5):1095-1101.

26. Gallun, R.L. and L.P. Reitz. 1971. Wheat cultivars resistant to the Hessian fly. U.S. Dept. Agr., ARS, Prod. Res. Rept. 134:16pp.

27. Gallun, R.L. 1977. Genetic gasis of Hessian fly epidemics. Ann. of the N.Y. Acad. of Sci. 287:223-229.

28. Gallun, R.L. and F.L. Patterson. 1977. Monosomic analysis of wheat for resistance to Hessian fly. J. Hered. 68:223-226.

29. Garman, H. 1903. The Hessian fly in 1902-1903. Ky. Ag. Exp. Sta. Bull. 111:212-224.

30. Haseman, L. 1916. An investigation of the supposed immunity of some varieties of wheat to the attack of the Hessian fly. J. Econ. Ent. 9:291-294.

31. Haseman, L. 1930. The Hessian fly larvae and its method of taking food. J. Econ. Ent. 23:316-321.

32. Havens, J.N. 1792. Observations of the Hessian fly. N.Y. Soc. Prom. Agr., Arts and Manufactures Trans. 1:89-107.

33. Hatchett, J.H. 1969. Genetic interactions between resistance in wheat, Triticum aestivum L., and races of Hessian fly, Mayetiola destructor (Say). Ph.D. Thesis, Purdue Univ., W. Lafayette, IN. 129pp.

34. Hatchett, J.H. 1969. Race E, sixth race of Hessian fly, Mayetiola destructor, discovered in Georgia wheat fields. Ann. Ent. Soc. Am. 62:677-678.

35. Hatchett, J.H. and B.S. Gill. 1981. D-genome sources of resistance in Triticum tauschii to Hessian fly. J. Hered. 72:126-127.

36. Hatchett, J.H., T.J. Martin, and R.W. Livers. 1981. Expression and inheritance of resistance to Hessian fly in synthetic hexaploid wheats derived from Triticum tauschii (Coss) Schmal. Crop Sci. 21:731-734.

37. Hatchett, J.H. 1982. Professor of entomology. KSU, Manhattan, KS. USDA-SEA. Personal communication.

38. Heyne, E.G., G.A. Weibe, and R.H. Painter. 1943. Complementary genes in wheat causing death of F_1 plants. J. Hered. 34:243-245.

39. Heyne, E.G. and A.B. Campbell, eds. 1981. Annual Wheat Newsletter. Vol 27. Ag. Press, Manhattan, KS. 175pp.

40. Hill, C.C. 1923. Platygaster vernalis Myers, an important parasite of the Hessian fly. J. Ag. Res. 25:31-42.

41. Jones, E.T. 1936. Hordeum grasses as hosts of the Hessian fly. J. Econ. Ent. 29(4):704-710.

42. Jones, E.T. 1938. Infestation of grasses of the genus Aegilops by the Hessian fly. J. Econ. Ent. 31(3):333-337.

43. Jones, E.T. 1939. Grasses of the tribe Hordeae as hosts of the Hessian fly. J. Econ. Ent. 32:505-510.

44. Jones, E.T. 1940. A discussion of Hessian fly resistance in certain wheat varieties. Trans. Kans. Acad. Sci. 43:234-265.

45. Leiby, R.W. and C.C. Hill. 1923. Twinning and monembryonic development of Platygaster heimalis, a parasite of the Hessian fly. J. Ag. Res. 25:337-350.

46. McColloch, J.W. and H. Yuasa. 1917. Notes of the migration of the Hessian fly larvae. J. An. Behavior 7:307-323.

47. McColloch, J.W. 1917. Wind as a factor in the dispersion of the Hessian fly. J. Econ. Ent. 10:162-170.

48. McColloch, J.W. 1919. Variation in the length of the flaxseed stage of the Hessian fly. J. Econ. Ent. 12(3):252-255.

49. McColloch, J.W. 1923. The Hessian fly in Kansas. Kans. Exp. Sta. Tech. Bull. 11:96pp.

50. McColloch, J.W. and S.C. Salmon. 1923. The resistance of wheat to Hessian fly, a progress report. J. Econ. Ent. 16:293-298.

51. Maas, F.B., III. 1982. The inheritance of the Marquillo type resistance in wheat to the Great Plains biotype of the Hessian fly. Unpublished M.S. Thesis, Kansas State Univ., Manhattan, KS. 29pp.

52. Maxwell, F.G. and P.R. Jennings, eds. 1980. Breeding Plants Resistant to Insects. John Wiley & Sons, New York. 683pp.

53. Miller, B.S., H.L. Mitchell, J.A. Johnson, and E.T. Jones. 1958. Lipid soluable pigments of wheat plants as related to Hessian fly infestation. Plant Phys. 33:413-416.

54. Miller, B.S., R.J. Robinson, J.A. Johnson, E.T. Jones, and B.W.X. Ponnaiya. 1960. Studies on the relation between silica in wheat plants and resistance to Hessian fly. J. Econ. Ent. 53:995-999.

55. Nelson, L.R. and W.L. Morrill. 1978. Hessian fly control in wheat by suppression of fall generations with Carbofuran. Agron. J. 70:139-141.

56. Noble, W.B., W.B. Cartwright, and C.A. Suneson. 1940. Inheritance of resistance to Hessian fly. J. Econ. Ent. 33:580-581.

57. Noble, W.B. and C.A. Suneson. 1943. Differentiation of the two genetic factors for resistance to Hessian fly, Phytophaga destructor (Say), in Dawson wheat. J. Agr. Res. 67:27-32.

58. Oellerman, C.M., F.L. Patterson, and R.L. Gallun. 1981. Inheritance of resistance in 'Luso' wheat to Hessian fly. Agron. Abs. Am. Soc. of Agron. p. 69.

59. Okigbo, B.N. and G.G. Gyrisco. 1962. Effects of fertilizers on Hessian fly infestations. J. Econ. Ent. 55:753-760.

60. Okigbo, B.N. 1963. Evaluation of losses caused by Hessian fly in a wheat fertilizer experiment. Agron. J. 55:117-119.

61. Packard, C.M. 1928. The Hessian fly in California. USDA Tech. Bull. 81:26pp.

62. Painter, R.H. 1930. Observations on the biology of the Hessian fly. J. Econ. Ent. 23(2):326-329.

63. Painter, R.H., S.C. Salmon, and J.H. Parker. 1931. Resistance of varieties of winter wheat to Hessian fly, Phytophaga destructor (Say). Kans. Ag. Exp. Sta. Tech Bull. 27:58pp.

64. Painter, R.H., E.T. Jones, C.O. Johnston, and J.H. Parker. 1940. Transference of Hessian fly resistance and other characteristics of Marquillo spring wheat to winter wheat. Kans. Ag. Exp. Sta. Tech. Bull. 49:55pp.

65. Painter, R.H. 1951. Insect Resistance in Crop Plants. Macmillan, New York, N.Y. 520pp.

66. Painter, R.H., E.T. Jones, and E.G. Heyne. 1952. Hessian fly resistance of Ponca wheat. J. Econ. Ent. 45:778-783.

67. Painter, R.H. 1954. Some ecological aspects of the resistance of crop plants to insects. J. Econ. Ent. 47:1036-1040.

68. Patterson, F.L. and R.L. Gallun. 1973. The inheritance of resistance of Seneca wheat to race E of Hessian fly. Proc. 4th Int. Wheat Genetics Symp., Missouri Ag. Exp. Sta., Columbia, MO. pp. 445-449.

69. Patterson, F.L. and R.L. Gallun. 1977. Linkage in wheats of the H_3 and H_6 genetic factors for resistance to Hessian fly. J. Hered. 68:293-296.

70. Patterson, F.L., R.L. Gallun, S.K. Carlson, and N.B. Stebbins. 1981. Registration of Stella and Ella common wheat germplasm lines. Crop Sci. (In press).

71. Powers, L. 1932. Cytological and genetic variability of strains of wheat derived from interspecific crosses. J. Ag. Res. 44:797-831.

72. Refai, F.Y., E.T. Jones, and B.S. Miller. 1955. Some biochemical factors involved in the resistance of the wheat plant to attack by the Hessian fly. Cereal Chemistry 32:437-451.

73. Refai, F.Y., B.S. Miller, E.T. Jones, and J.E. Wolfe. 1956. The feeding mechanism of the Hessian fly larvae. J. Econ. Ent. 49(2):182-184.

74. Roberts, J.J., R.L. Gallun, F.L. Patterson, and J.E. Foster. 1979. Effects of wheat leaf pubescence on the Hessian fly. J. Econ. Ent. 72:211-214.

75. Robinson, R.J., B.S. Miller, H.L. Mitchell, H.C. Mussman, J.A. Johnson, and E.T. Jones. 1960. Chloroplast number in leaves of normal wheat plants and those infested with Hessian fly or treated with maleic hydrazide. J. Econ. Ent. 53:560-562.

76. Rockwood, L.P. and M.M. Reecher. 1933. The Hessian fly in the Pacific Northwest. U.S. Dept. Ag. Tech. Bull. 361:23pp.

77. Sears, E.R. 1948. The cytology and genetics of the wheats and their relatives. Adv. in Genet. 2:239-270.

78. Severin, H.C. 1916. The Hessian fly in South Dakota. S.D. Ann. Corn and Grain Show and School, 1916:117-122.

79. Shands, R.G. and W.B. Cartwright. 1953. A fifth gene conditioning Hessian fly response in common wheat. J. Am. Soc. Agron. 45:302-307.

80. Slingerland, M.V. 1901. Facts about fly-proof wheat. Rural New Yorker. 60:627.

81. Snedicor, G.W. and W.G. Cochran. 1967. Statistical Methods. 6th Ed. ISU Press. Ames, Iowa.

82. Sosa, O., Jr. and R.L. Gallun. 1973. Purification of races B and C of the Hessian fly by genetic manipulation. Ann. Ent. Soc. Am. 66:1065-1070.

83. Sosa, O., Jr. and J.E. Foster. 1976. Temperature and the expression of resistance in wheat to the Hessian fly. Env. Ent. 5:333-336.

84. Sosa, O., Jr. 1979. Hessian fly: resistance of wheats as affected by temperature and duration of exposure. Env. Ent. 8:280-281.

85. Sosa, O. Jr. 1981. Biotypes J and L of the Hessian fly discovered in and Indiana wheat field. J. Econ. Ent. 74:180-182.

86. Stebbins, N.B., F.L. Patterson, and R.L. Gallun. 1980. Interrelationship among wheat genes for resistance to Hessian fly. Crop Sci. 20:177-180.

87. Stebbins, N.B. 1981. Interrelationships among wheat genes H_3, H_5, H_6, H_9, H_{10}, and H_{11} for resistance to Hessian fly. Ph.D. Thesis, Purdue Univ. W. Lafayette, IN. 82pp.

88. Steiger, D.K., D.L. Keim, and J.H. Hatchett. 1982. Registration of SD 8011 and SD 8014 Hessian fly resistant spring wheat germplasm (Reg. Nos. GP195 and GP196). Crop Sci. (In press--Jl-Aug., 1982 issue).

89. Suneson, C.A. and W.B. Noble. 1950. Further differentiation of genetic factors in wheat for resistance to the Hessian fly. U.S. Dept. Ag. Tech. Bull. 1004:8pp.

90. Walgenbach, D.D. and D.K. Steiger. 1979. Unpublished Hessian fly report on infestations of spring wheat in South Dakota, 1978-79. South Dakota State Univ. Brookings, SD. 9pp.

91. Walkden, H.H. 1936. Pupal period of the Hessian fly. J. Econ. Ent. 29(4):701-704.

92. Walkden, H.H. 1945. Reproductive capacity of the Hessian fly. J. Econ. Ent. 38:56-58.

93. Webster, F.M. 1899. The Hessian fly. Ohio Ag. Exp. Sta. Bull. 107:257-288.

94. Wells, D.G., J.J. Bonneman, W.S. Gardner, and K.F. Finney. 1982. Registration of Dawn wheat (Reg. No. 654). Crop Sci. 22:162.

95. Wootipreecha, S. 1971. Studies of genetic synthesis of new races of Hessian fly, Mayetiola destructor (Say). Ph.D. Thesis, Purdue Univ., W. Lafayette, IN. 187pp.

APPENDIX

Table 1A. Number of parent plants, progenies, and families tested against SD fly.

	Pedigree	Plants	Families
3583	Resistant Parent (Eureka/Dawn)	294	
Eureka	Susceptible Parent	361	
F_1	Eureka/3583	55	
	3583/Eureka	39	
F_2	Eureka/3583	730	
	3583/Eureka	931	
F_3 Families	Eureka/3583	3732	132
	3583/Eureka	3443	126
BC_1	Eureka/3583//Eureka	58	
	3583/Eureka//Eureka	73	
F_2 BC_1 Families	Eureka/3583//Eureka	1779	72
	3583/Eureka//Eureka	2109	81
3383	Resistant Parent (James/Dawn)	180	
James	Susceptible Parent	203	
F_1	James/3383	42	
	3383/James	42	
F_2	James/3383	638	
	3383/James	795	
F_3 Families	James/3383	2847	105
	3383/James	2902	92
BC_1	James/3383//James	84	
	3383/James//James	76	
F_2 BC_1 Families	James/3383//James	1843	70
	3383/James//James	1725	62

Table 2A. Virulence of specific Hessian fly biptypes on wheat cultivars with different genes for resistance.

Cultivars (Resistance)	Biotypes of Hessian fly					
	SD	GP	A	B	C	D
James (None)	S†	S	S	S	S	S
Coteau (None)	S	S	S	S	S	S
Eureka (None)	S	S	S	S	S	S
Seneca (H_7H_8)	R	R	S	S	S	S
Monon (H_3)	R	R	R	S	R	S
Knox 62 (H_6)	R	R	R	R	S	S
Ella (H_9)	R	R	R	R	MR	R
Stella (H_9H_{10})	R	R	R	R	R	R
Parker (Marquillo)	R	R	MR	S	MR	MR
Pawnee (Kawvale)	R	MR	S	S	S	S
SD 8015 (?)	R	R	?	?	?	?
SD 8014 (?)	R	R	?	?	?	?
SD 8011 (?)	R	R	?	?	?	?

† R = resistant; MR = moderately resistant; S = susceptible.

Table 3A. Test of independence of the crosses Eureka/3583 and James/3383 using F_1, F_2, and BC_1 data.

Postulated Cross	Observed (Expected) Numbers Resistant	Susceptible	Totals	Ratio	X^2	Assoc. X^2 Probability†
F_1:						
Eureka/3583	66 (41)	18(43)	84	.5		
James/3383	16 (41)	68(43)	84	.5		
Totals	82	84	168		49.13	<0.001*
F_2:						
Eureka/3583	1197 (861.36)	464 (800.87)	1661	.555		
James/3383	355 (690.64)	979 (642.13)	1334	.445		
Totals	1552	1443	2995		610.51	<0.001*
BC_1:						
Eureka/3583//Eureka	48 (28.35)	83 (102.6)	131	.45		
James/3383//James	15 (34.65)	145 (125.4)	160	.55		
Totals	63	228	291		29.98	<0.001*

† One degree of freedom.

Table 4A. Test of independence of the crosses Eureka/3583 and James/3383 using data of F_3 and F_2 BC_1 families.

Postulated Cross	Observed (Expected) Families R	H	S	Totals	Ratio	X^2	Assoc. X^2 Probability†
F_3 Families:							
Eureka/3583	60 (53.87)	187 (191.08)	11 (13.04)	258	.567		
James/3383	35 (41.13)	150 (145.92)	12 (9.96)	197	.433		
Totals	95	337	23	455		2.55	.50-.25
F_2 BC_1 Families:							
Eureka/3583//Eureka	15 (8.59)	105 (86.46)	33 (58)	153	.537		
James/3383//James	1 (7.41)	56 (74.54)	75 (50)	132	.463		
Totals	16	161	108	285		42.15	<0.001*

† Two degrees of freedom.

Table 5A. Test of independence of the cross Eureka/3583 and its reciprocal for maternal cytoplasmic effects using F_1, F_2, and BC_1 data.

Postulated Cross	Observed (Expected) Numbers: Resistant	Susceptible	Totals	Ratio	X^2	Assoc. X^2 Probability†
F_1:						
aa x AA	38 (35.38)	7 (9.65)	45	.536		
AA x aa	26 (30.62)	11 (8.35)	39	.464		
Totals	66	18	84		1.99	.25-.10
F_2:						
aa x AA	530 (525.48)	200 (203.7)	730	.439		
AA x aa	667 (671.52)	264 (260.3)	931	.561		
Totals	1197	464	1661		0.19	.75-.50
BC_1:						
(aa x AA) x aa	21 (21.26)	37 (36.77)	58	.443		
(AA x aa) x aa	27 (26.74)	46 (46.23)	73	.557		
Totals	48	83	131		0.008	>0.90

† One degree of freedom.

Table 6A. Test of independence of the cross Eureka/3583 and its reciprocal for maternal cytoplasmic effects using F_3 and F_2 BC_1 families.

Postulated Cross	Observed (Expected) Families R	H	S	Totals	Ratio	X^2	Assoc. X^2 Probability†
F_3 Families:							
aa x AA	30 (30.72)	95 (95.74)	7 (5.63)	132	.512		
AA x aa	30 (29.28)	92 (91.26)	4 (5.37)	126	.488		
Totals	60	187	11	258		.73	.75-.50
F_2 BC_1 Families:							
(aa x AA) x aa	13 (7.07)	50 (49.46)	9 (15.54)	72	.471		
(AA x aa) x aa	2 (7.93)	55 (55.54)	24 (17.46)	81	.529		
Totals	15	105	33	153		14.62	< 0.001*

† Two degrees of freedom.

Table 7A. Test of independence of the cross James/3383 and its reciprocal for maternal cytoplasmic effects using F_1, F_2, and BC_1 data.

Postulated Cross	Observed (Expected) Numbers: Resistant	Susceptible	Totals	Ratio	X^2	Assoc. X^2 Probability†
F_1:						
aa x AA	9 (8)	33 (34)	42	.5		
AA x aa	7 (8)	35 (34)	42	.5		
Totals	16	68	84		0.08	.90-.75
F_2:						
aa x AA	192 (172.53)	456 (475.79)	648	.486		
AA x aa	163 (182.47)	523 (503.21)	686	.514		
Totals	355	979	1334		5.58	.025-.01*
BC_1:						
(aa x AA) x aa	7 (7.88)	77 (76.13)	84	.525		
(AA x aa) x aa	8 (7.12)	68 (68.87)	76	.475		
Totals	15	145	160		.04	.90-.75

† One degree of freedom.

Table 8A. Test of independence of the cross James/3383 and its reciprocal for maternal cytoplasmic effects using F_3 and F_2 BC_1 families.

Postulated Cross	Observed (Expected) Families R	H	S	Totals	Ratio	X^2	Assoc. X^2 Probability†
F_3 Families:							
aa x AA	16 (18.66)	78 (79.95)	11 (6.4)	105	.533		
AA x aa	19 (16.34)	72 (70.05)	1 (5.6)	92	.467		
Totals	35	150	12	197		8.0	.025-.01*
F_2 BC_1 Families:							
(aa x AA) x aa	0 (.53)	28 (29.68	42 (39.75)	70	.53		
(AA x aa) x aa	1 (.47)	28 (26.32)	33 (35.25)	62	.47		
Totals	1	56	75	132		1.6	.50-.25

† Two degrees of freedom.

Table 9A. Test of independence of F_2 plants of Dawn 3/Newton cross versus other winter crosses involving Dawn.

Cross	Observed (Expected) Numbers Resistant	Susceptible	Totals	Ratio	X^2	Assoc. X^2 Probability†
Dawn 3/Newton	101 (103.71)	67 (64.35)	168	.757		
Dawn 2/Arthur	36 (33.29)	18 (20.65)	54	.243		
Totals	137	85	222		.49	.50-.25
Dawn 3/Newton	101 (117.95)	67 (50.21)	168	.337		
Dawn 1/Knox 62	249 (232.05)	82 (98.79)	331	.663		
Totals	350	149	499		11.43	<0.001*
Dawn 3/Newton	101 (113.77)	67 (54.1)	168	.349		
Dawn 8/Seneca	225 (212.23)	88 (100.9)	313	.651		
Totals	326	155	481		6.40	.025-.01*
Dawn 3/Newton	101 (109.51)	67 (58.27)	168	.351		
Dawn 7/Ella	211 (202.49)	99 (107.73)	310	.649		
Totals	312	166	478		2.69	.25-.10

† One degree of freedom.