AN ABSTRACT OF THE THESIS OF

Akhtar Ali for the degree of Doctor of Philosophy in Horticulture presented on August 29, 1984

Title: Inheritance of Resistance to Head Smut Disease in Maize (Zea mays L.).

Abstract approved: James R. Baggett

The inheritance of head smut disease of corn caused by the fungus Sphacelotheca reiliana (Kühn) Clinton was studied in the field in crosses of N6, a resistant inbred line of dent type, with two susceptible inbred lines of sweet type, SD-1 and SM7. The infection, measured as percentage of infected plants, was consistently low in the resistant parent and consistently high in susceptible parents. Differences in percent infection among reciprocal crosses for percent infection in F_1 and F_2 were non significant. However, in backcrosses to either the resistant or susceptible parent, the infection was higher when F_1 and F_1R (F_1 reciprocal) were used as male parent than when F_1 and F_1R were used as female. When dent and sweet kernel types were extracted from segregating populations and planted separately, infection was higher in sweet types than in dent types of the same segregating populations of F_2 and backcrosses to the sweet susceptible parent (BC_s). Clipping plants at the 4- to 5-leaf stage to predispose seedlings to infection, almost doubled percent infection compared to that of the

unclipped plots. Mean plot plant height reduction due to infection was not correlated with percentage of infected plants.

Among genetic populations, percent infection in F_1 and backcrosses to resistant parent (BC_r) was similar to that in the resistant parent in unclipped plots and higher than the resistant parent in clipped plots. Percent infection in BC_s was higher than in the F_1 and lower than in the susceptible parent for both clipped and unclipped plots. This showed partial dominance of resistance. In graphical comparisons, the relationship of the parents and progeny generations approached a linear configuration for clipped plots. As the infection increased, the graphs tended to show additive gene action. Deviations of the F_1 progenies from mid-parent values were significant and almost twice as large in unclipped plots as in clipped plots. Deviations of BC_r from mid-parent values were generally non-significant. In 1982 (high rates of natural infection), all deviations of BC_s from mid-parent values were positive while in 1983 (lower infection rates) deviations were negative. This showed dominance of resistance at lower levels of infection and suggested a tendency toward additivity with a slight indication of dominance of susceptibility at higher levels of infection. Chi square tests indicated that ratios of uninfected:infected plants fit theoretical single dominant gene ratios only at certain levels of infection, suggesting that resistance was polygenic. The estimations of gene effects indicated that besides dominance and additive gene action, resistance was also subject to epistatic gene effects. The average number of

genes involved was relatively small (12.9) and the average heritability estimates were relatively high (78.8%). Breeding by reciprocal recurrent selection and modified mass selection was proposed for efficacy of achieving head smut resistance in Zea mays L.

Inheritance of Resistance to Head Smut Disease in Maize (*Zea mays* L.)

by

Akhtar Ali

A THESIS

Submitted to

Oregon State University

in partial fulfillment of
the requirements for the
degree of

Doctor of Philosophy

Completed August 29, 1984

Commencement June, 1985

APPROVED:

Professor of Horticulture in charge of major

Head of Department of Horticulture

Dean of Graduate School

Date thesis presented August 29, 1984

Typed by Karla Sorensen for Akhtar Ali

DEDICATIONS

This thesis is dedicated to my parents, CH. Abdul Malik and Mrs. Abdul Malik, who gave me moral support in my goals to achieve an education. Without the love and encouragement of my parents, I would have never come this far.

ACKNOWLEDGEMENTS

I would like to thank Dr. James R. Baggett, major professor, for his guidance, encouragement, and help during the course of my studies. Thanks are also due to Dr. Richard O. Hampton for his valuable suggestions and guidance in the areas of plant diseases and to Dr. Warren E. Kronstad for guidance in the field of plant breeding and genetics. I wish to thank my graduate committee, Dr. James R. Baggett, Dr. Richard O. Hampton, Dr. Warren E. Kronstad, Dr. Francis J. Lawrence, and Dr. Glenn Fisher for guidance in the completion of my degree. The assistance of Dr. Kenneth E. Rowe, professor of statistics, for his help in analyzing the data and reviewing the manuscript is greatly appreciated. I wish to thank fellow graduate students, especially Janice Tibbs, and technical assistant Deborah Kean for cooperation and help in my research work.

The author also wishes to express appreciation to the Chairman, Pakistan Agricultural Research Institute, Islamabad, Pakistan for providing the opportunity and financial support during the course of these studies, and to the Director General, Ayub Agricultural Research Institute, Faisalabad, Pakistan, for sparing me from my duties to make these studies possible.

Finally, the author highly appreciates the patience and encouragement of parents, wife, children, relatives, friends, and colleagues during my period of study abroad.

TABLE OF CONTENTS

LIST OF FIGURES

LIST OF TABLES

INHERITANCE OF RESISTANCE TO HEAD SMUT DISEASE IN MAIZE (*ZEA MAYS* L.)

I. INTRODUCTION

Head smut, caused by *Sphacelotheca reiliana* (Kühn) Clinton, affects maize (corn), sorghum, and sudangrass (19). Head smut of corn, caused by *S. reiliana* var. *zeae*, is found in Africa, Asia, Australia, Europe, and North, Central, and South America (1). In tropical and subtropical areas, the disease is very destructive (33). In the United States, the disease occurs in corn in Texas, Kansas, Ohio, California, Washington, Oregon, Idaho, and Minnesota (5,14,15,19,40).

The first definite symptom of head smut is the appearance of sori (fungal spore cluster) in the male or female inflorescence, although at that time the growth of infected plants may already be somewhat reduced. Leaf sori are rare. The infection is systemic and may induce floret sterility and growth aberrations, such as bizarre proliferation of the inflorescence (phyllody) (14,38).

Corn is an important crop used for both human and livestock consumption. Infection causes destruction of the ear which is the most important part of the plant. In Texas, an incidence of 30 to 80 percent head smut disease was noted in corn fields with histories of intensive corn culture (14). Infection incidence ranged from traces to approximately 40 percent smutted plants in fields in Idaho and California (18,38). In South Africa, on certain lands where corn has been grown continuously for many years, an infection incidence of 25 percent and more has been reported (26).

Sorghum yields, in crop loss models, were inversely proportional to the percentage of smutted plants (42).

Thus far, no satisfactory control measure for head smut has been adopted in commercial production. Treatment of smut-contaminated seed has been reported effective in eliminating this source of infection (38), but soil-borne infection, a vastly more important source of disease, has been only moderately suppressed by application of fungicides (13). Head smut control by soil fumigants (24) is currently economically infeasible. Early infection and the systemic character of the disease (33) virtually preclude practical therapy, thus inviting investigations of host resistance.

Corn breeders have identified strains of both sweet and field corn with a high degree of resistance to head smut. Incorporating resistance into inbred lines therefore promises to produce adapted, high-quality hybrids as an efficient and environmentally advantageous means of head smut control. Knowledge of the inheritance of resistance would facilitate breeding programs, improving efficiency and possibly result in higher levels of resistance in released cultivars. As yet only preliminary studies on the inheritance of resistance to head smut of corn have been reported.

The nature of resistance to head smut of sorghum has never been elucidated. Since head smut of corn is closely related to head smut of sorghum, assuming that the resistance mechanism in both species are the same, an inheritance pattern determined for corn and breeding methods proposed for corn might also be of value

for development of head smut resistant sorghum cultivars. Identification of characters, especially those expressed before anthesis, associated with the resistance to head smut disease should greatly facilitate breeding for resistance. Past investigations of the inheritance of resistance have possibly been hampered by the difficulty of consistently achieving artificial epidemics for screening genetic populations. The present study made use of field inoculation methods (2) previously developed at Oregon State University. These methods greatly increased the chances of getting sufficient infection for reliable evaluations.

The principal purpose of this study was to determine the mode of inheritance of resistance to head smut in corn. Additional objectives were to determine the association of levels of resistance with the degree of height reduction caused by head smut infection, and the association of resistance with dent versus sweet endosperm.

II. LITERATURE REVIEW

The head smut disease

The fungus Sphacelotheca reiliana belongs to sub-division basidiomycotina; class teliomycetes; order ustilaginales; and family ustilaginaceae. The fungus produces basidiospores and teliospores which are infecting and resting spores, respectively.

In classical studies of spore germination, Hanna (20) found that each teliospore (chlamydospore) contains a single large nucleus which divides into four nuclei, one of which remains in the spore, while the other three are distributed in the promycelium (basidium). Cross walls are then laid down in the promycelium and four sporidia (basidiospores) are budded off from the promycelial cells. Meanwhile, the nucleus of each promycelial cell divides and one of the daughter nuclei migrates into the sporidium, while the other one remains in the promycelial cell. In this way, the promycelium is able to produce more than one generation of sporidia. The haploid sporidia are able to penetrate the epidermal cells but the delicate haploid hyphae lack the vigor necessary for further parasitic development. It is only when the haploid hyphae of opposite sex (plus and minus type) unite to form a diploid mycelium that the pathogen becomes actively parasitic. Thus, S. reiliana is heterothallic. Apparently the paired condition of the nuclei (dikaryotic condition) is associated with an increase in vigor which enables the fungal hyphae to invade the host plant.

Several studies have elucidated the effect of environmental conditions on spore germination and disease development. Christ-

ensen (10) concluded that soil temperature and soil moisture determine to a great extent the occurrence and severity of head smut of sorghum. He observed that S. reiliana infected sorghum seedlings at temperature ranging from 16 to 36°C. The infection was not favored either by relatively high or low temperature. High soil temperature (20-30°C) during germination of seed and emergence of the seedling favored infection. The highest percentage of infection was obtained at 28°C. He further observed that low soil moisture (15 percent on an oven dry basis) was much more favorable for infection than high soil moisture (25 percent). Kruger (26) investigated the effect of temperature (16°C to 36°C) and soil pH (2.0 to 9.0) on spore germination of S. reiliana on corn. He found that the optimum temperature was 23°C to 29°C and there was a sharp decline in germination when the incubation temperature increased to 32°C. On prune agar, good general germination was recorded in the pH range 4.0 to 6.0 with pH 5.4 as the optimum. He further investigated the effect of temperature (22, 25, 30, and 35°C), soil moisture (30, 50, and 70 percent water holding capacity), and soil pH (4.5, 5.5, 7.0, and 8.0) on seedling infection in corn. He found that optimum conditions for infection were a temperature of 25 to 30°C, a soil moisture content of 30 to 50 percent, and an acid base. Simpson (38) also found that corn plants grown in the greenhouse at approximately 25°C had the most infection as compared to smut infections at other temperatures.

Many studies have shown that infection takes place in the seedling stage. Kruger (26) found that corn seedlings may be infected through the roots as well as the coleoptile. Miller (32) has reported that the sorghum seedling becomes vulnerable to infection shortly after the seed coat is ruptured by the emerging radicle. Seedlings remain vulnerable until they are approximately nine weeks old, but most infection occurs before the first true leaves unfold. Potter (33) and Reed, Swabey, and Kolk (34) have also reported that infection in corn and sorghum is a "seedling-infecting type" resulting in a systemic distribution of mycelium in the apical primodial tissues. The parasite continues its development, keeping pace with the host plant as it increases in size. Potter (33) concluded from a histological study of sorghum that the lateral buds carry the infection in their meristematic tissue apparently from the time of their formation when the culm is starting to differentiate nodes.

Halisky (18) found that sporulation by S. reiliana occurs in corn ears independently of tassel sporulation. Tassel sori, in contrast, are invariably accompanied by smutted ears or rudimentary abortive leaf buds that may harbor spores internally. Occasional appearance of a tassel-smutted plant with a normal ear is an indication of incomplete systemic infection of the host by smut mycelium.

Fungal infections do not always result in sporulation. Jacks (24) observed some corn plants with abnormal flowering parts (phyllody) which lacked spore masses along with a number of in-

fected plants with stunted growth and black powdery spore masses on tassels and ears, in both glasshouse and field conditions.

In the sporulation process, the inflorescence of the host is transformed into a sorus. This sorus is covered by a shiny, white membrane-peridium. The membrane splits easily at maturity and the teliospores are disseminated by wind and rain and overwinter in soil where they may retain viability for a considerable period of time. This provides a source of inoculum for succeeding corn crop and completes the life cycle of the pathogen.

In studies of the initial sources of head smut infection, Potter (33) found that teliospores of S. reiliana produced on smutted heads of sorghum are wind-distributed in the locality in which disease occurs. Distribution from one district to another by seed contamination was not the determining factor in the occurrence of general field infection. In field studies on seed treatment of sorghum, he applied hot water treatment with different temperatures for different periods and fungicide treatments with formalin, copper sulphate, cresol, and potassium sulphide. The treatments did not reduce the infection. In another test, some of these treatments caused inviability of the spores. He concluded that infection commonly takes place from sources other than seed-borne spores. Similarly, Simpson (38) found that teliospores produced on smutted plants of corn are scattered by wind and rain. These spores overwinter in the soil and become the main source of infection. Greenhouse tests also indicated that head smut was not seed-borne. He selected seed of susceptible sweet corn lines

infected with head smut. Kernels of these lines were surface-sterilized and planted into sterilized soil in a greenhouse. There was no smut development from the surface-sterilized seeds. When a new lot of seed was surface-sterilized and planted in soil contaminated with smut, a high percentage of plants showed head smut at maturity. He concluded that infection resulted only from the smut inoculum applied in the soil.

Artificial epiphytotics

Many inoculation techniques have been studied to produce high and consistent incidence of disease to screen larger populations for resistance to head smut.

Previous studies on the life cycle of S. reiliana have indicated that the presence of the parasite in the soil at germination time is necessary for successful infection. Kruger (26) secured a reasonably high and consistent rate of infection in corn under greenhouse conditions by using mixed smut spores at the rate of 0.1 gm spores per cubic decimeter of dry soil. Potter (33) inoculated dry seed, germinating seed, and older plants of sorghum under greenhouse conditions using inoculating material of dry spores, suspension of spores with few spores germinated, and conidia. He used a light application of inoculating material, heavy application of inoculating material, smutted heads raked into the soil, heavily inoculated soil at planting, and other modifications. He found that germinating seeds inoculated with soil containing large amounts of dry spores at planting produced the most consistent positive results.

Spores have also been mixed with carriers other than soil to prepare inoculum. Baier and Kruger (3) used soil, sand, soil and sand mixture, and vermiculite as carriers for smut spores. The spores were mixed at the rate of 0.1 gm spores per cubic decimeter of carrier. The corn seed was planted in furrows, 5 cm deep and each seed was covered with 50 ml of the spore-carrier mixture. A sufficiently high rate of infection was obtained only when the soil was used as a carrier. Recently Baggett and Koepsell (2) investigated five inoculation treatments for head smut in sweet corn: 1) 0.7 liters of inoculum (50 cc spores/liter of vermiculite) per 10.6 m plot applied with the seed in a V-belt planter; 2) same as treatment 1, with 25 cc spores/liter of vermiculite; 3) 100 cc of spore-vermiculite mixture (50 cc spores/liter of vermiculite) placed in a hole with the seed and covered with 2-3 cm of soil; 4) seeds heavily coated with spores and methylcellulose; and 5) noninoculated control. Application of a mixture of teliospores and slightly dampened vermiculite (25 cc and 50 cc spores/liter of vermiculite) with the seed by means of a V-belt planter resulted in a 95% disease incidence in the most susceptible cultivars. Application of this mixture by hand in individual planting holes resulted in greater disease incidence but required much more labor and inoculum.

Individual plants have also been inoculated directly by hypodermic injection. Edmunds (11) conducted field inoculations of sorghum by injecting sporidia of S. reiliana into the growing point. Plants to be inoculated had 4 to 5 expanded leaves with

the growing point 3 to 4 cm above soil. The inoculations resulted in 65-90% smut infection in susceptible checks and 0-90% smut infection in selections. Differences between smut incidence from natural infection and sporidial inoculations were highest among partially resistant selections. A line 57M1250, which had no smut in naturally infected fields and as high as 27% by hypodermal inoculations, showed that the mechanism of natural resistance was partially overcome by hypodermal inoculations.

As previously reviewed, infection occurs in the seedling stage. Based on the hypothesis that infection incidence may be increased if the susceptible stage is prolonged, Banyra and Baggett (unpublished thesis research) have recently found that clipping of sweet corn plants at 4- to 5-leaf stage markedly increases infection. They hypothesized that clipping retards growth, keeps the growing point below ground level longer, and increases the length of the period of susceptibility.

Infection readings

Resistance to head smut disease has generally been treated as a threshold trait in published studies. Hartl (21) proposed that threshold traits are quantitative and are influenced by the environment and the aggregate action of several loci. A more recent approach to the study of threshold traits involves the use of conceptual tools developed for dealing with quantitative traits. Unlike quantitative traits, however, the phenotype cannot be measured directly. All that can be observed about the individual plant's 'liability' is whether the individual has the condition or

trait in question. Falconer (12) explained that the 'liability' is intended to express not only the individual's innate tendency to develop the disease, i.e. its susceptibility in the usual sense, but also the whole combination of external circumstances that make an individual more or less likely to develop the disease. The point on the scale of 'liability' above which all individuals are affected and below which all are normal is the 'threshold' level. These concepts of 'liability' and 'threshold' are developed quantitatively so the terms of quantitative traits are used.

To overcome the communication difficulties attributable to inconsistent use of terms by plant pathologists and plant breeders, Cooper and Jones (9) have proposed to reserve the terms resistant and susceptible to denote the opposite ends of a scale covering the effects of an infectible individual on pathogen infection, multiplication, and invasion and the terms tolerant and sensitive to denote the opposite ends of a scale covering the reaction of the plant to disease.

In almost all studies reviewed, infection incidence was expressed as the percentage of smutted plants irrespective of the type or severity of symptoms observed. Stromberg (41), however, screened 161 corn hybrids and 14 inbreds in the field with artificially infested soil, for resistance to head smut. At maturity, incidence of smutted ears, tassels, ears and tassels, partially smutted ears, ear proliferation and dwarfness were recorded. Resistance was expressed as the percentage of smutted plants, with

0% resistant; >0-5% moderately resistant; >5-10% moderately susceptible; >10% susceptible. Baggett and Koepsell (2) determined disease incidence by counting infected plants after sweet corn reached full edible maturity. A plant was counted as infected if sori were found on an ear or tassel. Similarly, Kruger (26) also determined the infection incidence in corn by the appearance of smut in the ears or tassels.

Inheritance studies

A search of the literature has revealed only preliminary work recently done on inheritance of resistance to head smut in corn. Therefore, studies on the inheritance of resistance to some related diseases, and literature relating to the mathematical determinations used in our inheritance studies of head smut of corn, are also reviewed.

Inheritance of resistance to head smut in corn

Recently, Ma et al. (27) made a preliminary study on the genetics of resistance to head smut in corn, in China. They screened 750 inbred lines, hybrids, and local cultivars and identified 177 with varying degrees of resistance to S. reiliana. They found that resistance was controlled by dominant genes, recessive genes, or the interaction of several nonallelic genes. Additive gene effects were important. There was a significant positive correlation between degree of infection in the parents and in the F_1s, and a negative correlation between susceptible F_1s and resistant F_2s, indicating that at least one resistant parent was needed to produce a resistant hybrid.

Mei et al. (31) have also studied the pattern of inheritance of resistance to head smut in corn, in China. They found that resistance to S. reiliana in the F_1 populations derived from a diallel cross of 10 inbred lines, using highly resistant inbred lines as maternal parents, was not significantly different from that in F_1s of the reciprocal crosses involving maternal parents with low resistance. They further found that resistance in the F_1 was positively correlated with the average of the two parents; it was transgressive and incompletely dominant.

Inheritance studies involving related diseases

Studies of the inheritance of resistance to head smut of sorghum, loose and covered kernel smut of sorghum, and common smut of corn have been reported.

Steward and Reyes (39) tested sorghum cultivars and hybrids commonly grown in the Coastal Bend area, for resistance to head smut of sorghum caused by Sphacelotheca reiliana var. reiliana, under uniformally infested field conditions. They found that excellent resistance was already present in certain commercially available cultivars and hybrids. They further found that a hybrid derived from the susceptible female parent 'Combine Kafir-60' (9.3% infected) and resistant male parent 'Combine White Feterita' (0.0% infected) was as resistant as its resistant male parent. This showed complete dominance for resistance in this cross. On the other hand, hybrids Texas 601, Texas 660, and RS 590 did not differ significantly from their common female parent 'Combine Kafir-60'. Their male parents TX 07, SA 7000, and SA 386 showed

1.2, 0.0, and 7.8 percent infection, respectively. This showed incomplete dominance for susceptibility in these crosses. Another type of resistance was also observed where only one hybrid RS 610 (17.8% infected) was significantly more susceptible than its female parent 'Combine Kafir-60' (9.3% infected) because it had a very susceptible male parent 'Combine 7078' (37.9% infected). Whereas hybrids RS 650 (5% infected), Texas 620 (5.1% infected), and Texas 611 (5.5% infected) were significantly more resistant than their common female parent 'Combine Kafir-60' (9.3% infected), while their male parents Plainsman 7005, TX 07, and TX 74 showed 3.4, 0.7, and 22 percent infection, respectively. From these studies, they speculated that the mode of inheritance was not a qualitative character and proposed an extension of the study by including further generations to resolve the question of inheritance.

Marcy (29,30) studied the inheritance of resistance to loose smut caused by Sphacelotheca cruenta (Kuhn) Potter and covered kernel smut caused by Sphacelotheca sorghi (Link) Clinton, in sorghum. When resistant 'Dwarf Yellow Milo' was crossed with susceptible cultivars, approximately one-fourth of the F_2 plants were susceptible. It was suggested that 'Dwarf Yellow Milo' carried a factor for resistance (R) completely epistatic to a factor for susceptibility (S) in the susceptible cultivars. When resistant 'Feterita' was crossed with susceptible cultivars, the results indicated the interaction of the factor for resistance (B), and a factor for susceptibility (S). Under environmental

conditions highly favorable for infection, S was epistatic to B, with an approximated ratio of 13 smutted to 3 normal plants for F_2 segregation reconstructed from F_3 data. Under environmental conditions less favorable for infection, F_2 ratios were nearly reversed, approaching the ratio of 3 resistant to 1 susceptible. It was concluded that hybrid plants containing both the S and B factors were extremely unstable in their infection reactions, and that epistasis of S over B, or B over S, was determined solely by environmental conditions during the germination period. When 'Dwarf Yellow Milo' and 'Feterita' were crossed, approximately one-sixteenth of the F_2 plants were susceptible indicating that 'Dwarf Yellow Milo' and 'Feterita' possessed different genes for resistance.

Infection results with loose smut indicated that both 'Dwarf Yellow Milo' and 'Feterita' maintained their resistance. When 'Dwarf Yellow Milo' was crossed with susceptible cultivars there was a two factor interaction similar to that of 'Feterita' hybrids inoculated with the covered smut. When 'Feterita' was crossed with susceptible cultivars there was clear-cut dominance of resistance. No infected plants were obtained from crosses of 'Feterita' with 'Dwarf Yellow Milo', indicating that these two cultivars possessed at least one common factor for resistance. It was suggested that the factors governing reaction to covered smut may also determine the reaction of loose smut, but that their effect is reversed. The presence of an additional factor influencing loose smut reactions has complicated the ratios, so addi-

tional data would be necessary for a more precise analysis of these results.

Casady (6) studied the inheritance of resistance to physiologic races 1, 2, and 3 of Sphacelotheca sorghi causing covered kernel smut in sorghum. Data for 217 F_3 lines of a cross between 'Spur Feterita' and 'Pink Kafir' reconstructed to represent the F_2 generation, demonstrated a 1:3 segregation of resistance to susceptibility to each of the three races. This established the existence of three single factor pairs designated as Ss_1ss_1, Ss_2ss_2, and Ss_3ss_3 for reaction to races 1, 2, and 3, respectively. The reaction of the F_1 progenies of 'Combine Kafir-60' and 'Pink Kafir' crossed with 'Spur Feterita' indicated an incomplete dominance of resistance. The three genes for resistance to the races 1, 2, and 3 appeared to be linked with gene order and approximate crossover percentages as Ss_2 (6.94), Ss_1 (37.95), and Ss_3. When only Ss_2 and Ss_3 were considered, the data gave no indication of linkage. However, the data definitely showed both these genes linked with a common gene, Ss_1.

Bojanowski (4) studied inheritance in corn of reaction to common smut caused by Ustilago maydis. The inbreds chosen for genetic experiments represented a wide range of variability, both in origin and in reaction to smut. The distribution of F_3 progenies in classes of percentage of smutted plants indicated that reaction to smut was polygenic in inheritance, conditioned by several morphological and physiological characters and segregating according to a quantitative model. He further found that resist-

ance was controlled by both additive and non-additive genes. Estimates of variance due to general and specific combining ability for total smutted plants indicated that character was entirely additive. If, however, the location of smut galls was taken into account, then differences among lines, as well as specific, non-additive effects of some hybrid combinations became apparent.

Genetic parameters

Examples of determinations of the following genetic parameters were reviewed: the deviation of hybrid from mid-parent value, gene effects, number of genes, and heritability.

(1) Deviations of hybrids from mid-parent values

Kim and Brewbaker (25) adapted generation mean analysis to compare hybrids with the limits set by their parents for inheritance of general resistance to Puccinia sorghi Schw in corn. They used 110 populations including 10 parents, 25 F_1 hybrids, 25 F_2 populations, and 50 backcrosses, for average rust rating. Rust ratings of the 25 F_1 hybrids were significantly less than the mid-point between resistant and susceptible parents. The 25 F_2 populations were slightly more susceptible than the F_1 hybrids. Backcross progeny averages of OH 545 for BC_r (backcross to resistant parent) and BC_s (backcross to susceptible parent) were more resistant than predicted from the average of the F_1 and the respective parents. The data suggested partial dominance for factors controlling resistance.

(2) Gene effects

Generation means have also been used to determine gene effects. Hayman (22,23) has given three parameter and six parameter models. The simplest experiment supplying information on additive, dominance, and the three kinds of epistatic variation contains two inbred lines and their F_1, F_2 and first backcross generations. When epistasis is absent the three-parameter model estimates a constant, additive, and dominance effects. When epistasis is present, the data fitted to a six-parameter model estimate constant, additive, dominance, and three kinds of epistatic (additive x additive, additive x dominance, and dominance x dominance) gene effects. However, additive and dominance gene effects cannot be uniquely measured when significant epistasis is present. These models also lack orthogonality. On the other hand, estimates of the parameters do provide an indication of the relative importance of the various types of gene effects affecting the total genetic variation of a plant attribute.

Similarly, Gamble (16,17) outlined a procedure for the separation of gene effects into six parameters effecting genetic variation of a quantitative trait. To estimate these parameters he used six inbred lines, and all possible F_1, F_2, P_1F_1 (backcross to P_1), and P_2F_1 (backcross to P_2) crosses among them. The estimations of these six parameters require the means of only six populations, that is, $\overline{P_1}$, $\overline{P_2}$, $\overline{F_1}$, $\overline{F_2}$, $\overline{P_1F_1}$, and $\overline{P_2F_1}$. The gene effects were calculated by using different contrasts of these

populations depending on the expectations of the means of these populations.

To estimate these gene effects he used data on yield of shelled corn, plant height, and certain yield components. The estimates of gene effects for corn yield indicated that dominance gene effects were quite important. Estimates of additive gene effects were of low magnitude and many were non-significant. Epistatic gene effects were more important than additive gene effects. The additive x additive and additive x dominance gene effects were relatively more important than dominance x dominance effects, in the inheritance of yield in corn.

(3) Number of genes

Castle (7) gave a formula, corrected by Wright, to calculate the number of genes controlling a trait. This formula is comprised of the difference between the means of parental races, variance of the F_2 (total variance), and variance of the F_1 (environmental variance). Later, Kim and Brewbaker (25) estimated environmental variance by taking the cube root of variance of resistant parent x variance of susceptible parent x variance of the F_1 instead of using variance of the F_1 alone. The number of genes were calculated by dividing the squared difference of parental means by genetic variance (total variance - environmental variance). He estimated the minimum number of genes controlling resistance to Puccinia sorghi Schw, causing common rust in corn. The OH 545 crosses with three susceptible inbreds averaged 2.08 gene pairs with high consistency among different progenies.

(4) Heritability

Information concerning heritability of quantitative characters is useful for predicting the efficiency of selection in segregating populations. Mahmud and Kramer (28) and Weber and Moorthy (43) explained that the extent to which genetic segregation is expected in later generations of a cross is largely a reflection of the heritability of the character in question. The consideration of total genetic variance (additive + non-additive) determines broad-sense heritability. The data permit the calculation of broad-sense heritability in several ways. The simplest one is by utilizing the variability among spaced F_2 plants in relation to the variability among spaced plants of the non-segregating populations (parents and F_1). It has been shown that the expected variance of any non-segregating population is environmental variance; thus total variance of segregating population would be genetic variance plus environmental variance. Broad-sense heritability is the ratio of genetic variance to the total variance.

Reifschneider and Arny (35) based broad-sense heritability estimates on variance of parental, F_1, and F_2 populations in the studies of inheritance of resistance to Kabatiella zeae causing eye spot disease in corn. Parental and F_1 populations estimated environmental variance and the F_2 populations estimated total variance. The broad-sense heritability estimates for resistance to eye spot were approximately 75%.

III. MATERIALS AND METHODS

The resistant parent in these studies was an inbred line of dent type, Nebraska 6 (N6). Two sublines were involved. One, designated N6-79-17-47 (N6A) was obtained from Illinois Foundation Seed Corporation and used for crossing in the greenhouse in spring 1981, and a second subline N6-2164-C277 (N6B) was obtained from Del Monte Corporation and used for crossing along with N6A in the field in 1981. Progenies derived from these two sublines were maintained separately through all field tests. It was determined by preliminary analysis that the behavior of the separate crosses was identical. Data from the two crosses and N6 sublines were thus combined prior to computation of plot means. It had previously been observed that the two sublines of N6 were indistinguishable in the field. Susceptible parents were SM7, an early maturing sweet corn inbred line obtained from W. Crookham Seed Company, and SD-1 a sweet corn inbred derived by self pollination of a highly susceptible commercial F_1 hybrid, 'Sugar Daddy'. SD-1, which was used to produce all progeny seed tested in 1982 was the result of two generations of selfing. The SD-1 which was used to produce all progeny seed tested in 1983 was the result of three generations of selfing. Parent SD-1 seed used in tests each year was that obtained from three generations of selfing. Although it was not as inbred or as phenotypically uniform for general characteristics as desired, it was highly and uniformly susceptible to S. reiliana.

The original crosses between N6 and the two susceptible lines were made in the greenhouse, using the standard technique of bagging tassels and ears before pollination. The F_1 plants were grown in the field for F_2 and backcross seed production for 1982 disease tests. At this time, additional F_1 combinations were made to provide a supply for evaluation in head smut tests, and to provide the second set of progenies for 1983 disease tests.

For each cross, the following populations were studied.

POPULATIONS	PARENT COMBINATIONS
Resistant Parent (P_r)	
Susceptible Parent (P_s)	
F_1	$P_r \times P_s$
F_1 Reciprocal (F_1R)	$P_s \times P_r$
F_2	F_1 ($P_r \times P_s$) Self
F_2 Reciprocal (F_2R)	F_1R ($P_s \times P_r$) Self
Backcrosses to Resistant Parent (BC_r)	
BC_r1	$P_r \times F_1$
BC_r2	$F_1 \times P_r$
BC_r3	$P_r \times F_1R$
BC_r4	$F_1R \times P_r$
Backcrosses to susceptible parent (BC_s)	
BC_s5	$P_s \times F_1$
BC_s6	$F_1 \times P_s$
BC_s7	$P_s \times F_1R$
BC_s8	$F_1R \times P_s$

Field plantings

Plantings were made in an alluvial silty clay loam soil at the Vegetable Research Farm, Corvallis. The area used had been infested artifically with S. reiliana spores for the previous one or more years for screening sweet corn cultivars. In seedbed preparation, thorough tillage was followed by a band application of 600 kg/ha of 8N-10.3P-6.6K/100 kg commercial fertilizer. A hand powered V-belt planter was used for sowing. About 0.4 liters of inoculum (50 cc teliospores/liter of slightly dampened vermiculite) per 6 m plot was applied to the planting furrow along with the seed by the V-belt planter over the banded row of fertilizer. The rows were 1.1 m apart.

A randomized complete block design with four replications was used. In each replication, parents, F_1's, and BC_r (backcrosses to the resistant parent) were planted in 2 plots each. The populations were planted on double the number of plots so that the clipping treatment could be applied to half of each population. The dent and sweet kernels from populations segregating for dent and sweet kernels (xenia effect), F_2 and BC_s (backcross to susceptible parent), were sorted to extract dent and sweet populations and planted in 8 plots (4 dent and 4 sweet) and 4 plots (2 dent and 2 sweet), respectively. F_2's were planted on double the number of plots to increase population size. Data from F_2 plots of the same treatment and from the same replication were pooled for analyses. Dent and sweet counterparts were always planted in the same row next to each other.

A light irrigation by sprinkler was used immediately after planting to obtain uniform germination. Otherwise, irrigation was avoided during early growth stages to increase infection potential by providing a favorably dry condition for spore germination and infection. Water was applied by overhead sprinkler irrigation about every 10 days or as needed, after the infection stage was well past.

After planting, a pre-emergence application of atrazine [2-chloro-4-(ethylamino)-6-(isopropylamino)-s-triazine] and alachlor [2-chloro-2′,6′-diethyl-N-(methoxymethyl) acetanilide] herbicides at the rate of 2.24 and 1.68 kg active ingredient per hectare, respectively, were applied to control weeds during early stages of growth. Mechanical cultivations and hand hoeings were employed as needed. After germination, plants were thinned to 15 to 20 cm apart, resulting in a stand of 30 to 40 plants per plot.

At the 4- to 5-leaf stage, in each population and each replication, half of the plots were clipped at ground level. These plots were identified in the design of the experiment, and were randomized within blocks.

Observations

After crop maturity, when complete expression of infection had occurred, infection incidence was recorded by counting the infected and uninfected plants. A plant was counted as infected if sori were found on an ear or tassel. Since plants were classified as healthy or infected on the basis of presence of the pathogen, the terminology of resistant and susceptible instead of

tolerant and sensitive was adapted. Resistance was considered as a threshold trait and there was no attempt to record severity of infection on each infected plant. The observation unit was a single plot instead of a single plant in a plot. In each plot, infection percentage was calculated as the proportion of infected plants to total plants.

In 1982, at maturity, plant height was measured from ground level to tassel tip. The plant height of up to 10 each of healthy and infected individual plants in each plot was recorded. The height reduction due to infection, expressed as percentage, was calculated as the mean height of healthy minus mean height of infected plants divided by mean plant height of healthy plants in each plot.

The natural values of both percent infection and percent height reduction were transformed by the following relationship: Transformed Value = Radian x Arc sine x Square Root of Natural Value.

Arc sine transformation, also called angular transformation, is used when there is a wide range of proportions to spread out the proportions and increase their variances. The natural data are presented but significance tests were made on transformed data.

Data analysis

The data recorded were used for analyses of variance, generation mean analysis, estimation of minimum number of genes, and broad-sense heritability.

Analyses of variance

Analyses of variance were done for both percent infection and percent height reduction due to infection. Because of an unbalanced design due to different numbers of plots in different populations, the analyses were done in several steps. In each step described below, analyses were applied with years separate. For the combined dent sweet data, analysis of variance across years was also applied. To determine the effect of dent and sweet kernel type on infection, the populations segregating for dent and sweet (F_2 and BC_s) were analyzed separately. Then to determine the effect of clipping on infection, dent and sweet plots were analyzed separately and combined. Finally, differences between populations for infection and height reduction were tested by the analyses of populations segregating for dent and sweet, dent and sweet plots separately, dent and sweet plots combined, and clipped and unclipped plots (with dent and sweet plots combined) separately.

Correlations between different variables were calculated.

Relationship among genetic populations

The means of percent infection in parents, F_1, F_2, BC_r, and BC_s were tabulated and plotted to illustrate the mode of inheritance (dominance or additive gene action) involved. The occurrence of additive gene action was determined by whether the measure of the slope of the regression line (b_1) was equal to 1.0.

Deviations of F_1, BC_r, and BC_s means from their respective mid-parent values

The deviation in percent infection of F_1, BC_r, and BC_s means from their respective mid-parent values (MP) were analyzed as a test of additivity. The mid-parent values of F_1, BC_r, and BC_s were determined using an additive model by taking the average of parents, F_1 and resistant parent, F_1 and susceptible parent, respectively. Differences were tested against the standard error calculated by the contrast of different populations used in the calculations of deviations, assuming common experimental error, using the following models adapted from Kim and Brewbaker (25):

DEVIATIONS	POPULATIONS	S.E.
F_1 - MP	$\frac{F_1 + F_1R}{2} - \frac{P_r + P_s}{2}$	$\sqrt{\frac{MSE}{n}}$
BC_r - MP	$\frac{BC_r1\ BC_r2 + BC_r3 + BC_r4}{4} - \frac{1/2(F_1 + F_1R) + P_r}{2}$	$\sqrt{\frac{10\ MSE}{16\ n}}$
BC_s - MP	$\frac{BC_s5 + BC_s6 + BC_s7 + BC_s8}{4} - \frac{1/2(F_1 + F_1R) + P_s}{2}$	$\sqrt{\frac{10\ MSE}{16\ n}}$

Gene effects

The gene effects were calculated adapting the models derived by Gamble (16). The means of percent infection in six populations, that is, $\overline{P_1}$ $\overline{(P_r)}$, $\overline{P_2}$ $\overline{(P_s)}$, $\overline{F_1}$, $\overline{F_2}$, P_1F_1 (BC_r), and P_2F_1 (BC_s) were used to obtain the estimates of the following six parameters: 1) mean effects (m), 2) additive effects (a), 3) dominance effects (d), 4) additive x additive epistasis (aa), 5) additive x dominance epistasis (ad), and 6) dominance x dominance epistasis (dd).

The calculations of these gene effects were done by using the models given by Gamble (16):

GENE EFFECTS	POPULATIONS	S.E.
m	$\overline{F_2}$	$\sqrt{MSE/2n}$
a	$\overline{P_1F_1} - \overline{P_2F_1}$	$\sqrt{MSE/2n}$
d	$-1/2\overline{P_1} - 1/2\overline{P_2} + \overline{F_1} - 4\overline{F_2} + 2\overline{P_1F_1} + \overline{P_2F_1}$	$\sqrt{11\ MSE/n}$
aa	$-4\overline{F_2} + 2\overline{P_1F_1} + \overline{P_2F_1}$	$\sqrt{10\ MSE/n}$
ad	$-1/2\overline{P_1} + 1/2\overline{P_2} + \overline{P_1F_1} - \overline{P_2F_1}$	$\sqrt{MSE/n}$
dd	$\overline{P_1} + \overline{P_2} + 2\overline{F_1} + 4\overline{F_2} - 4\overline{P_1F_1} - 4\overline{P_2F_1}$	$\sqrt{20\ MSE/n}$

The gene effects were tested against their standard errors calculated by taking a square root of their error variances. The error variances of these gene effects were calculated in the usual manner by using the error variances (error mean squares) of populations used in the calculation of each gene effect with the assumption of common experimental error. For example, the error variance for dominance (Vd) was calculated as follows:

$$Vd = 1/4\ \overline{VP_r} + 1/4\ \overline{VP_s} + \overline{VF_1} + 16\ \overline{VF_2} + 4\ \overline{VP_rF_1} + 4\ \overline{VP_sF_1}$$

Minimum number of genes

The estimates of minimum number of genes were made by following the Castle-Wright formula (7).

$$N = \frac{D^2}{8(VF_2 - VE)}$$

Where D = mean difference between parents, VF_2 is the total variance (genetic variance + environmental variance) and VE is the environmental variance (non-genetic variance). Following Kim and Brewbaker (25), the environmental variance was estimated by taking the cube root of $VP_r \times VP_s \times VF_1$ (variance of resistant parent x

variance of susceptible parent x variance of F_1) instead of using the variance of F_1 alone as originally adopted by Castle-Wright.

Broad sense heritability

Broad-sense heritability was calculated using the variances of parents, F_1 and F_2 populations. The estimates of broad-sense heritability were made by adapting the following formula suggested by Mahmud and Kramer (28).

$$B.S.H. = \frac{VF_2 - VE}{VF_2} \times 100$$

The VF_2 is the total variance (genetic + non-genetic). The VE is the environmental variance (non-genetic). As mentioned before, the environmental variance was calculated from the variances of non-segregating populations of the resistant parent, susceptible parent, and F_1.

IV. RESULTS AND DISCUSSION

Incidence of head smut disease

Incidence of head smut infection was adequate for genetic analysis of resistance in both 1982 and 1983. The analysis of variance across years (Table 28) showed that percent infection in 1982 and 1983 was statistically different in N6 x SD-1 but not in N6 x SM7. The interaction between years and population was significant because F_2 and BC_s populations were more susceptible to infection in certain environments than other populations. The interaction between years and clipping was also significant because unclipped plots were more susceptible to infection in certain environments than the clipped plots. Overall, infection was higher in 1982 than in 1983 when the same genetic populations were compared (Table 1). This difference was only slightly evident in the parents, F_1 and BC_r, but pronounced in the F_2 and BC_s. This differential response, will be discussed in a later section. A chi square test of the overall numbers of infected versus uninfected plants in 1982 and 1983 also showed that infection was significantly higher in 1982 than in 1983 (Table 2). Overall infection was 25.2% in 1983 and 39.8% in 1982. The environmental conditions which resulted in greater infection in 1982 probably involved differences in temperature and soil moisture during the seedling period.

Effect of clipping on infection

In all analyses of variance in which the effect of clipping was included, except the analyses across years, there was a signif-

Table 1. Incidence of head smut in genetic populations in 1982 and 1983; N6 x SD-1 and N6 x SM7 combined.

Populations	Percent Infection	
	1982	1983
N6	1.9	1.8
SD-1 + SM7	92.7	92.6
F_1	17.8	17.0
F_2	31.8	17.2
BC_r	6.8	6.3
BC_s	64.7	44.7

Table 2. Incidence of head smut in 1982 and 1983; N6 x SD-1 and N6 x SM7 and genetic populations combined.

Year	Disease Incidence			
	No. Uninfected	No. Infected	% Infection	X^2
1982	5891	3898	39.8	652.8**
1983	14015	4717	25.2	

**Significant at 1% probability.

icant F value for difference in percent infection in clipped and non-clipped plots (Tables 16-23,28). When data for the two crosses and two years were combined for each population, percent infection was higher in clipped plots of all progeny populations, but not in either the resistant parent or SD-1 and SM7 (Table 3). Thus the effect of clipping was greater on populations with intermediate levels of resistance, while the populations with high levels of resistance, or susceptibility were not affected. Edmunds (11) found that differences between smut incidence from natural infection and artificial inoculations were highest among partially resistant lines. The probable reason for this differential response of populations to clipping will also be discussed in later section.

The small effect of clipping on infection in the parents compared to the other populations resulted in some significant clipping x population interaction [Tables 16 (1983), 17 (1982), 18 (1983), 22, 23, 28]. Most clipping x population interactions were non-significant.

The overall effect of clipping on the ratio of infected/uninfected was also tested by chi square (Table 4). Infection incidence was higher in clipped plots (38.5%) than in unclipped plots (23.3%). Correlations between clipping and percent infection were also significant (Table 35).

The increase in disease incidence observed in clipped plants confirms the findings of Banyra and Baggett (unpublished thesis research) who have also found an increase in infection by clipping

Table 3. Effect of clipping on head smut infection in genetic populations; N6 x SD-1 and N6 x SM7 and 1982 and 1983 combined.

	Percent Infection	
Populations	Unclipped	Clipped
N6	2.1	1.5
SD-1 + SM7	93.0	92.1
F_1	5.3	30.3
F_2	14.1	32.3
BC_r	2.0	11.6
BC_s	41.1	65.0

Table 4. Effect of clipping on head smut incidence; populations, crosses, and years combined.

	Disease Incidence			
Treatments	No. Uninfected	No. Infected	% Infection	X^2
Unclipped	11968	3639	23.3	775.3**
Clipped	7938	4976	38.5	

**Significant at 1% probability.

sweet corn seedlings. The clipping effect presumably involves a slowing in the growth of the plant and or a prolongation of the growth period during which the plant is susceptible.

The correlation of plant height reduction in infected plants with the percent infection in the plot was computed to determine if there is a relationship. In all analyses of the 1982 data, differences among populations for percent height reduction due to infection were non-significant (Tables 29-34). The infected plants in populations with a low percent infection were as stunted by disease as the infected plants in populations with high percent infection. In most of the cases, the effect of clipping on percent height reduction due to infection was also non-significant. However, in some cases the clipped plants showed a greater percent height reduction from infection than did unclipped plants [Tables 30 (N6 x SM7), 31]. There was also a correlation between clipping and height reduction (Table 35). There was apparently a dual effect of clipping on height reduction and percent infection. There were no significant interactions between genotype and clipping, and seed type and clipping for percent height reduction.

The correlation on an entry mean basis of percent infection and percent height reduction, obtained from all plots in the 1982 study were very small and were non-significant (Table 35). In the absence of any association between disease incidence and percent height reduction due to infection, the height measurements were not taken in 1983.

Effect of seed type on infection

In each cross, the analyses of F_2 and BC_s (the only populations segregating for dent and sweet) showed that dent and sweet types differed significantly for percent infection except in N6 x SM7, 1982 (Tables 20 and 21). The interaction of seed type (dent and sweet) with genotypes and clipping were non-significant. As shown in a summary of all tests of F_2 populations and all BC_s (Table 5), percent infection of sweet plants was about 7% higher than for dent plants. This difference was also shown to be significant (Table 6) where the overall effect of dent and sweet seed type on the ratios of infected:noninfected plants were compared. The average infection of all dent plots was 33.2 percent compared to 41.1 percent in all sweet plots of the same segregating populations. Correlations between seed type and percent infection were also significant (Table 35).

Since equal numbers of dent and sweet plots were planted for all F_2's and backcrosses to the susceptible parent, percent infection data for dent and sweet plots were pooled in the genetic analyses.

Reciprocal cross differences

Reciprocal cross differences for percent infection in the F_1 and F_2 were non-significant (Tables 7 and 8) which is in agreement with Mei *et al*. (31). However, in backcrosses to either the susceptible or resistant parent, the infection was higher when F_1 and F_1 reciprocal were used as male parent than when F_1 and F_1 reciprocal were used as female. Since there was no differences

Table 5. Disease incidence in dent and sweet plots of segregating populations; N6 x SD-1 and N6 x SM7, and 1982 and 1983 combined.

Populations	Percent Infection	
	Dent	Sweet
F_2	19.4	26.6
BC_s	48.1	55.4

Table 6. Effect of dent and sweet seed type on infection; all segregating populations of N6 x SD-1 and N6 x SM7, and 1982 and 1983 combined.

Seed Type	Disease Incidence			X^2
	No. Uninfected	No. Infected	% Infection	
Dent	6634	3303	33.2	122.0**
Sweet	4968	3472	41.1	

**Significant at 1% probability.

Table 7. Percent infection in reciprocal progenies of N6 x SD-1 in 1982 and 1983.

Populations	1982		1983	
	% Infection[y]	Reciprocal Difference[z]	% Infection	Reciprocal Difference
F_1	22.8	----	15.9	4.2 NS
F_1R	----		20.1	
F_2	32.3	1.5 NS	14.3	4.5 NS
F_2R	33.8		18.8	
N6 x F_1 and F_1R	8.2	1.6†	7.8	4.0**
F_1 and F_1R x N6	6.6		3.7	
SD-1 x F_1 and F_1R	75.2	10.0†	50.4	9.3**
F_1 and F_1R x SD-1	65.2		41.0	

[y] Mean of 4 replications.
[z] Reciprocal difference tested by t test.
** Significant at 1% probability.
† Higher than S.E.
NS Non-significant.

Table 8. Percent infection in reciprocal progenies of N6 x SM7 in 1982 and 1983.

Populations	1982		1983	
	% Infection[y]	Reciprocal Difference[z]	% Infection	Reciprocal Difference
F_1	----	----	17.4	1.4 NS
F_1R	6.9		18.8	
F_2	30.3	2.5 NS	19.3	1.6 NS
F_2R	32.8		20.9	
N6 x F_1 and F_1R	7.9	2.6†	12.5	5.5*
F_1 and F_1R x N6	5.4		7.0	
SM7 x F_1 and F_1R	64.0	10.8**	54.4	13.8**
F_1 and F_1R x SM7	53.1		40.6	

[y] Means of 4 replications.
[z] Reciprocal difference tested by t test.
* Significant at 1% probability.
** Significant at 5% probability.
† Higher than S.E.
NS Non-significant.

between F_1 and F_1 reciprocal, the average of backcrosses of F_1 and F_1 reciprocal was used to test the differences of the crosses where F_1 and F_1 reciprocal were used as male or female.

From these studies it can only be speculated that there may have been a selective advantage of F_1 pollen carrying genes for susceptibility over pollen carrying genes for resistance.

Since these reciprocal differences were found in both backcrosses to resistant parent and backcrosses to susceptible parent, the reciprocals were combined to determine the difference among different populations in the genetic analyses.

Inheritance of resistance

Incidence of head smut in unclipped and clipped plots of N6 x SD-1 and N6 x SM7 is given as the number and percentage of infected plants in Table 9. Disease incidence in the resistant parents was consistently low (0-3.5%) and consistently high (89-96%) in the susceptible parents. In both unclipped and clipped plots, percent infection was higher in SD-1 than in SM7 in 1982, but higher in SM7 than in SD-1 in 1983. The small differences in susceptibility between these two parents were not considered important.

Relationships among genetic populations

In all the analyses of variance for percent infection, the differences among populations were significant (Tables 16-28). Comparison of all unclipped and clipped populations by analyses of variance (Tables 10 and 11) showed that F_1 progenies were similar in percent infection to the resistant parent under conditions of

Table 9. Incidence of head smut in unclipped and clipped plots of all populationa of N6 x SD-1 and N6 x SM7, 1982 and 1983.

		1982						1983					
		Unclipped			Clipped			Unclipped			Clipped		
Cross	Populations	No. Uninf.	No. Inf.	% Infection	No. Uninf.	No. Inf.	% Infection	No. Uninf.	No. Inf.	% Infection	No. Uninf.	No. Inf.	% Infection
N6 x SD-1	N6	109	4	3.5	94	0	0	349	6	1.7	298	6	2.0
	SD-1	7	222	96.9	13	148	91.9	20	187	90.3	9	123	93.2
	F_1	106	11	9.4	75	50	40.0	430	28	6.1	301	126	29.5
	F_2	666	219	24.8	497	339	40.6	1509	135	8.2	1092	359	24.7
	BC	435	17	3.8	353	42	10.6	975	13	1.3	778	85	9.8
	BC_s^r	305	574	65.3	173	586	77.2	1181	549	31.7	599	878	59.4
N6 x SM7	N6	109	4	3.5	94	0	0	349	6	1.7	298	6	2.0
	SM7	17	151	89.8	10	77	88.9	9	147	94.2	3	60	95.2
	F_1	79	1	1.2	72	10	12.2	439	19	4.2	249	117	32.0
	F_2	695	210	23.2	474	321	40.4	1402	137	8.9	872	382	30.5
	BC	469	13	2.7	422	50	10.6	789	12	1.5	544	89	14.1
	BC_s^r	431	429	49.9	186	420	69.2	1088	545	33.4	432	692	61.6
N6 x SD-1	N6	218	8	3.5	188	0	0	698	12	1.7	596	12	2.0
+	SD-1 x SM7	24	373	94.0	23	225	90.7	29	334	92.0	12	183	93.8
N6 x SM7	F_1	185	12	6.1	147	60	29.0	869	47	5.1	550	243	30.6
	F_1	1361	429	24.0	971	660	40.5	2911	272	8.6	1964	741	37.7
	BC	904	30	3.2	775	92	10.6	1764	25	1.4	1322	184	12.2
	BC_s^r	736	1003	57.7	359	1006	73.7	2269	1094	32.5	1031	1570	60.4

lower disease incidence in the unclipped plots, while the percent infection in the F_1 was significantly higher than that of the resistant parent under higher disease incidence in the clipped plots. Likewise, the unclipped BC_r was similar in percent infection to the resistant parent, while infection was higher in clipped BC_r than in the resistant parent. Percent infection in BC_r and in the F_1 was similar in unclipped plots, except in the N6 x SD-1 test in 1983. In clipped plots, percent infection in BC_r was lower than in the F_1, with the exception of BC_r from N6 x SM7 in 1982. This behavior of F_1 and BC_r generations indicated that resistance may be inherited as a dominant character under conditions of lower disease incidence in unclipped plots and as a partially dominant character under conditions of higher disease incidence in clipped plots.

Percent infection was lower in the F_1 than in the F_2 in unclipped plots of both crosses in both years, but differences were significant only during 1982 (Tables 10 and 11). This was due to dominance of resistance in the F_1 and segregation for resistance in the F_2 in unclipped plots. With the exception of N6 x SM7, 1982, percent infection in the clipped plots was similar in the F_1 and F_2 of both crosses in both years. This was indicative of dominance and additivity of resistance in unclipped and clipped plots, respectively.

Percent infection in backcrosses to susceptible parents (BC_s) was in all tests significantly higher than in the F_1 and lower

Table 10. Percent infection in unclipped and clipped plots of populations of N6 x SD-1 with reciprocals combined, 1982 and 1983.

	1982				1983			
	Unclipped		Clipped		Unclipped		Clipped	
Populations	% Infection[y]	Significance test[z]	% Infection	Significance Test	% Infection	Significance test	% Infection	Significance test
N6	4.0	a	0	a	1.8	ab	0.0	a
BC_r	3.7	a	11.4	b	1.3	a	10.2	b
F_1	9.3	a	36.3	c	6.5	bc	29.5	c
F_2	25.6	b	40.4	c	8.3	cd	24.8	c
BC_s	63.6	c	76.6	d	31.5	e	60.0	d
SD-1	96.3	d	91.3	e	90.7	f	92.9	e

[y]Mean % infection in 4 replications.

[z]Multiple comparisons of means by Newman-Keuls method.
The means with different letters were significantly different at 5% probability.

Table 11. Percent infection in unclipped and clipped plots of populations of N6 x SM7 with reciprocala combined, 1982 and 1983.

	1982				1983			
	Unclipped		Clipped		Unclipped		Clipped	
Populationa	% Infection[y]	Significance teat[z]	% Infection	Significance Teat	% Infection	Significance Teat	% Infection	Significance Teat
N6	4.0	a	0	a	1.8	a	0.9	a
BC_r	2.8	a	10.4	b	1.5	a	18.1	b
F_1	1.3	a	12.5	b	4.2	ab	32.1	c
F_2	22.8	b	40.3	c	9.0	b	31.3	c
BC_s	50.2	c	66.8	d	33.6	c	61.3	d
SM7	90.3	d	83.3	e	95.0	d	96.0	e

[y]Mean % infection in 4 replicationa.

[z]Multiple compariaona of meana by Newman-Keula method.
The meana with different lettera were significantly different at 5% probability.

than in the susceptible parents. This behavior may also be due to partial dominance of resistance.

The present findings of dominance and partial dominance of resistance supports the previous findings of incomplete dominance for genes controlling resistance to head smut of corn, made by Mei et al. (31).

Partial dominance might be due to additive and non-additive (dominance and/or epistasis) gene action. A graphical comparison of population percent infection (Figures 1-6) indicated that resistance is strongly but not entirely dominant over susceptibility. Generally, at the lower disease incidence obtained in unclipped plots, the graphs tend to support a hypothesis of complete dominance. In the clipped plots, where disease incidence was higher in the F_1, F_2, and BC_s but little changed in the parents and BC_r, the graphs approach a linear configuration and are more indicative of additive gene action. In clipped and unclipped plots of both crosses, additivity was further indicated by the determination that the measure of the slope of the regression line (b_1) was equal to 1.0. All the measures of coefficients of determination (R_2) were significant. This showed, overall, in all populations, that additive gene effects were important. These results confirm the previous findings of Ma et al. (27).

Deviations of F_1, BC_r, and BC_s means from their respective mid-parent values

Percent infection of the F_1 progenies was significantly lower than their respective mid-parent values in both unclipped and

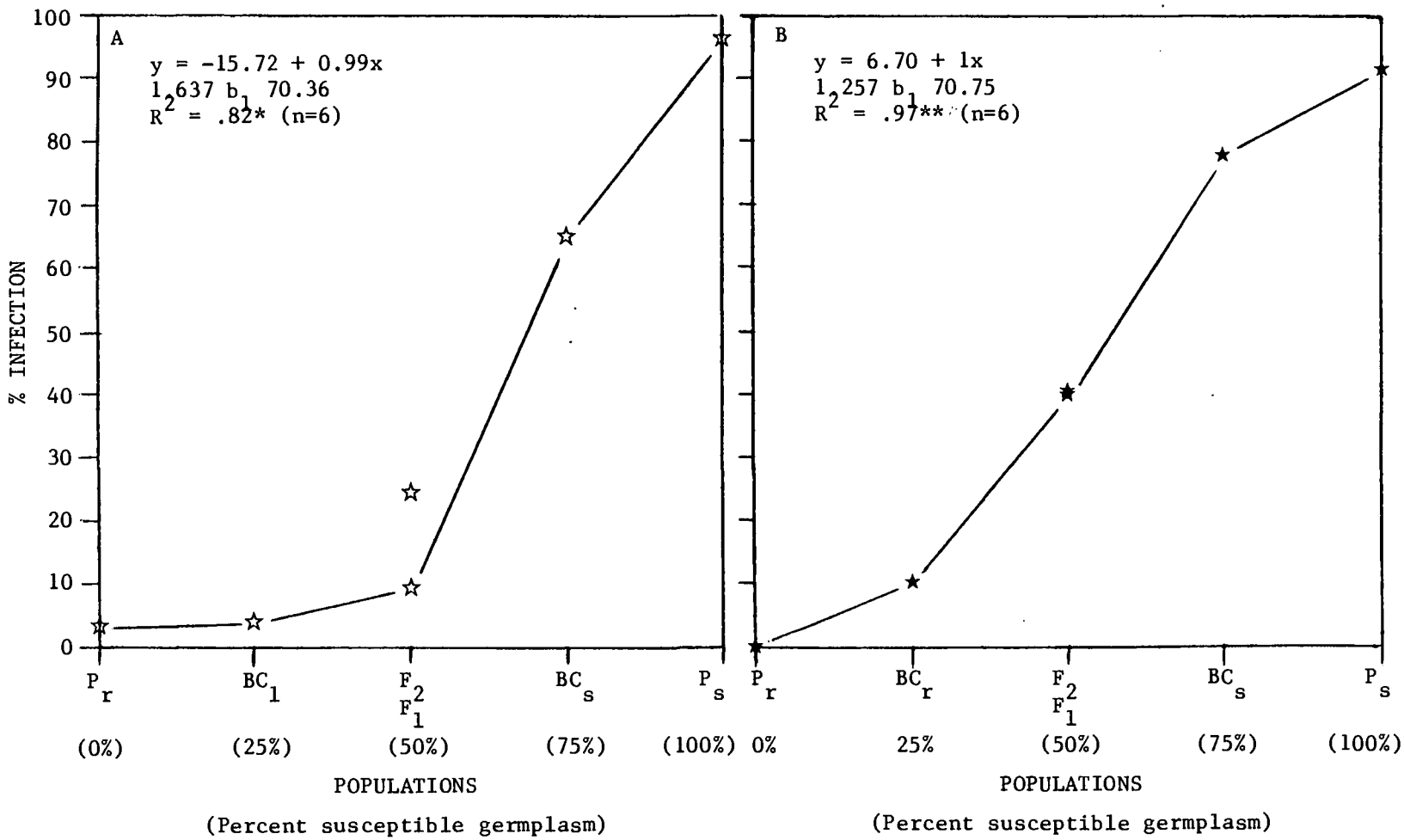

Fig. 1. Incidence of infection in parents and progeny generations of N6 x SD-1, 1982.
A: Unclipped plots, B: Clipped plots (* or ** significant at 5% or 1% probability).

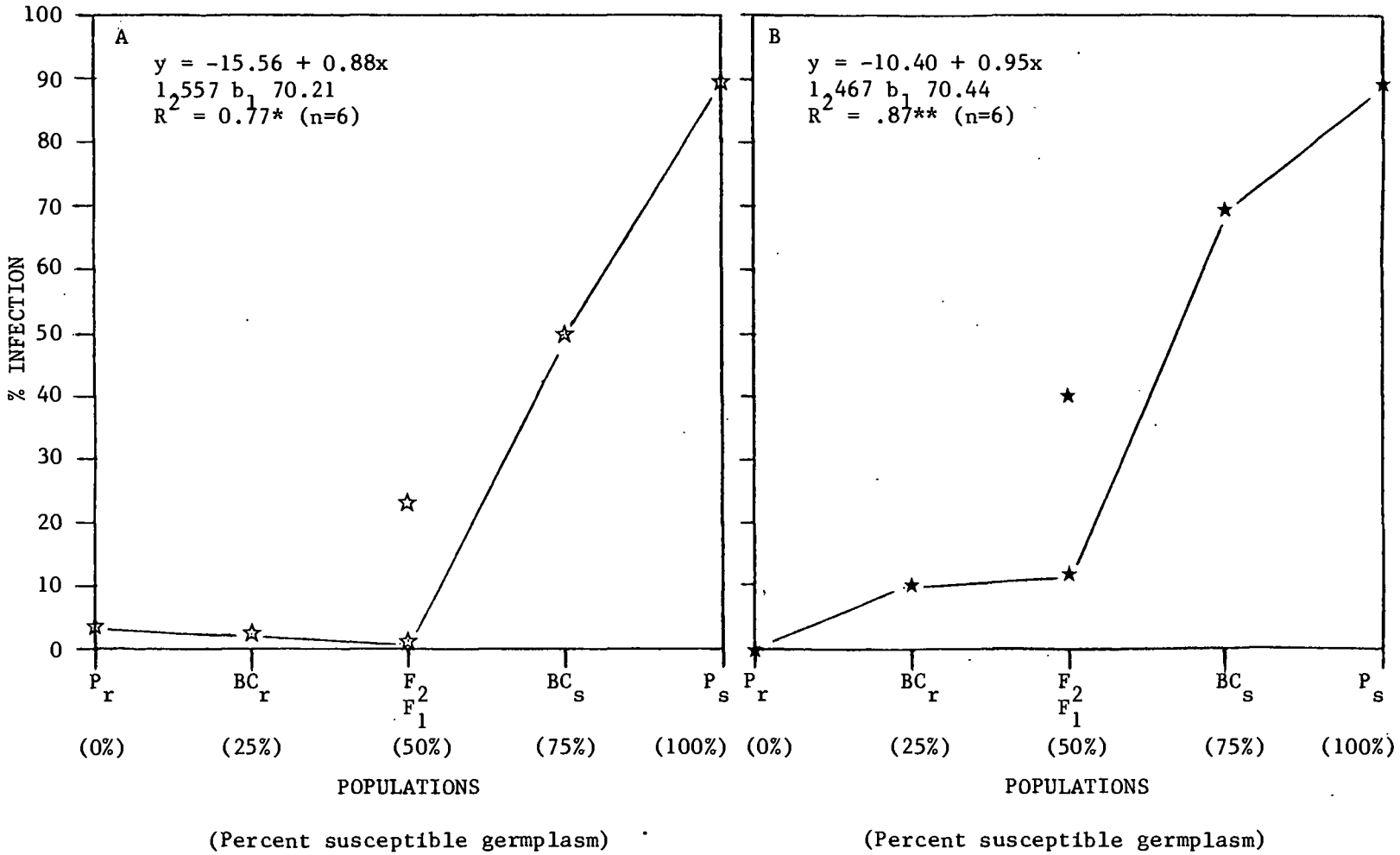

Fig. 2. Incidence of infection in parents and progeny generations of N6 x SM7, 1982.
A: Unclipped plots, B: Clipped plots (* or ** significant at 5% and 1% probability).

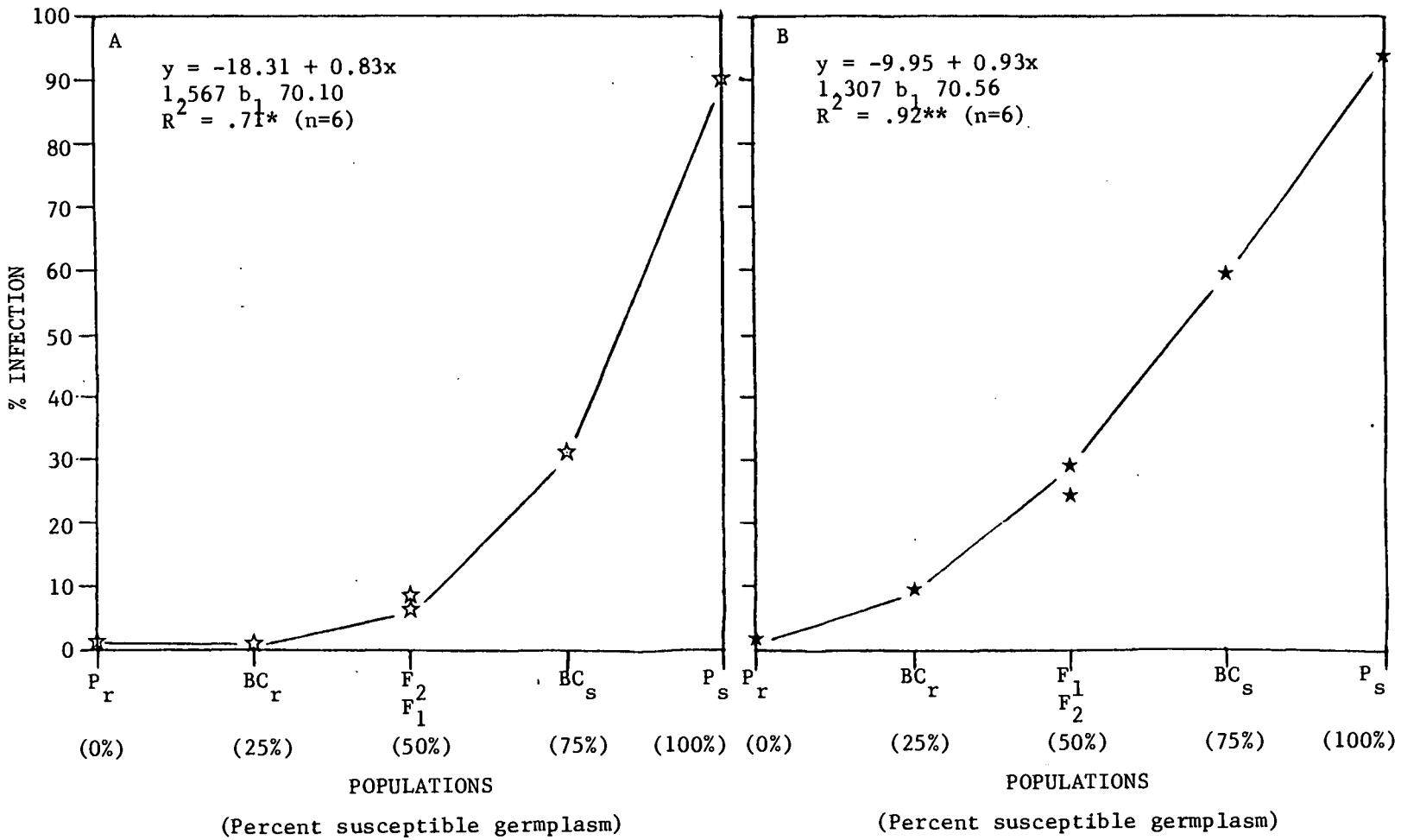

Fig. 3. Incidence of infection in parents and progeny generations of N6 x SD-1, 1983. A: Unclipped plots, B: Clipped plots (* or ** significant at 5% or 1% probability).

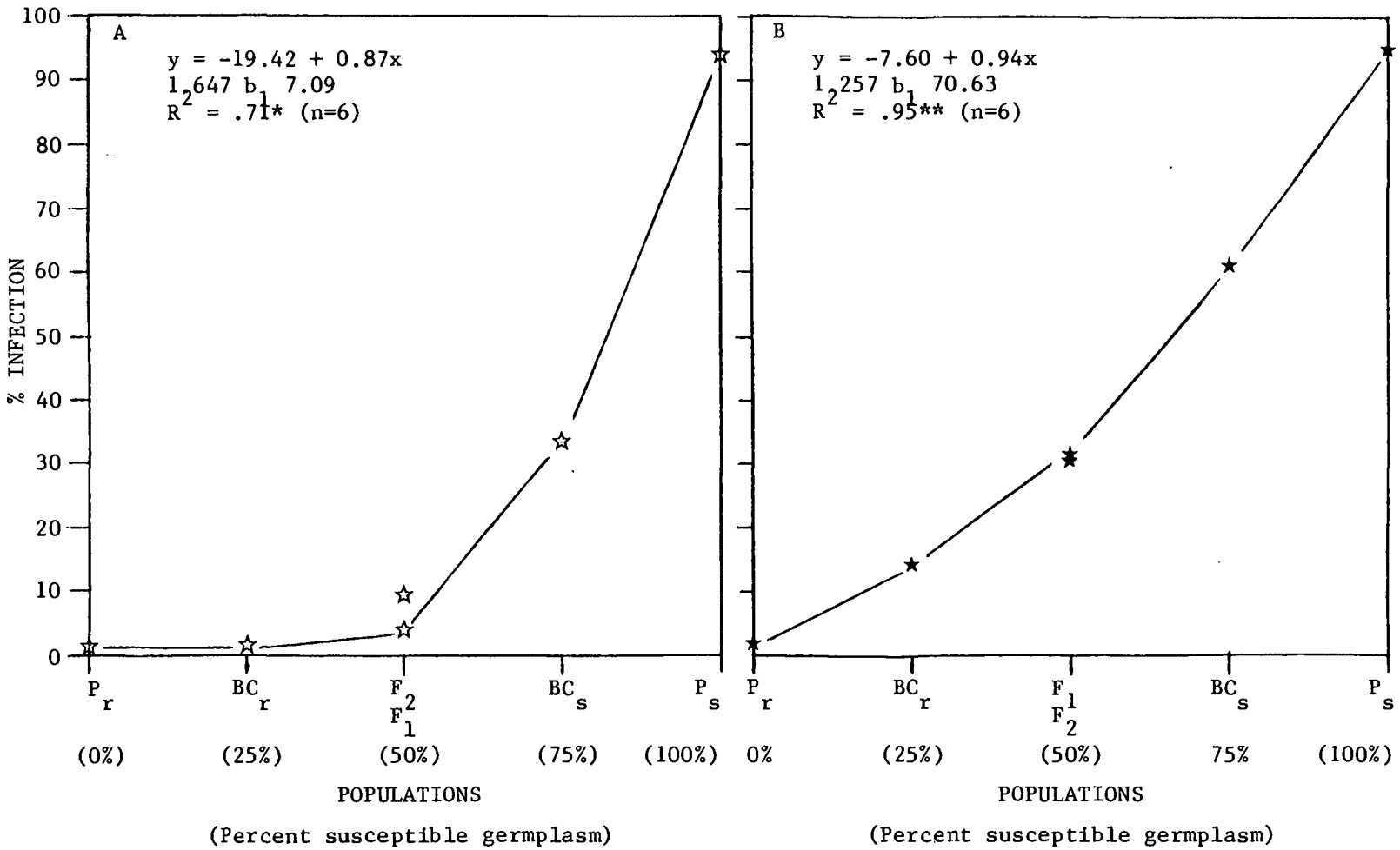

Fig. 4. Incidence of infection in parents and progeny generations of N6 x SM7, 1983.
A: Unclipped plots, B: Clipped plots (* or ** significant at 5% or 1% probability).

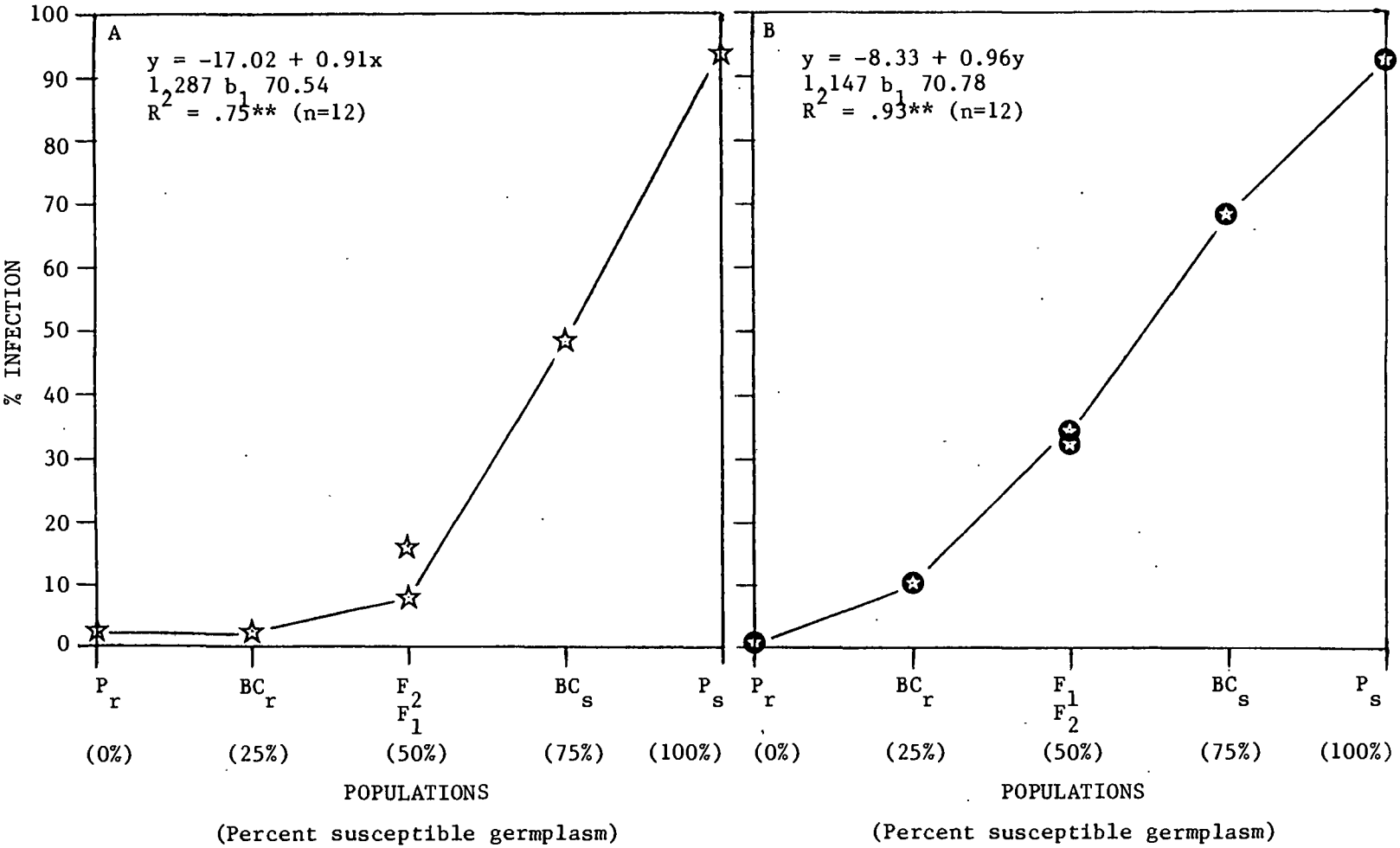

Fig. 5. Incidence of infection in parents and progeny generations of N6 x SD-1, 1982 and 1983 combined. A: Unclipped plots, B: Clipped plots (** significant at 1% probability).

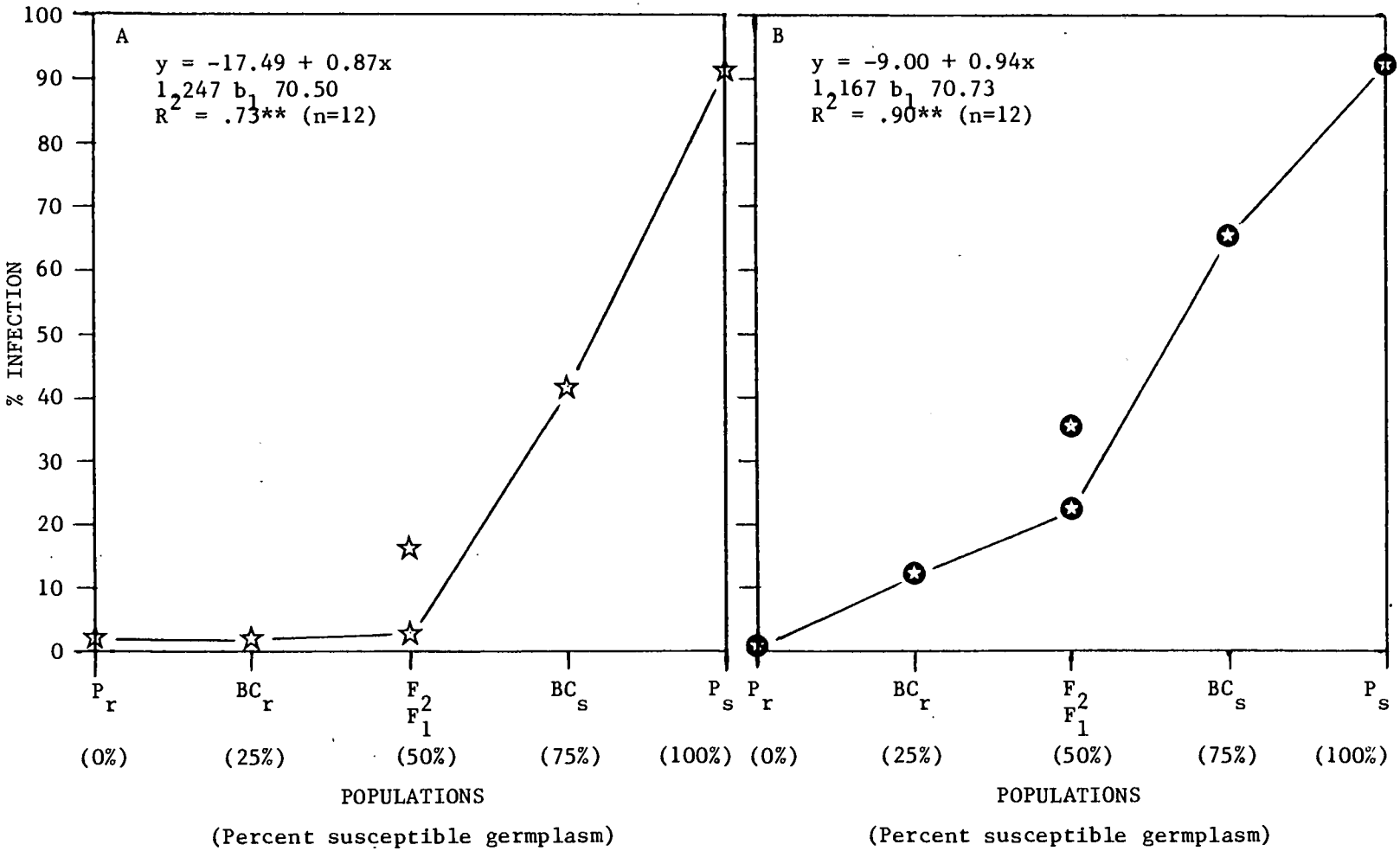

Fig. 6. Incidence of infection in parents and progeny generations of N6 x SM7, 1982 and 1983 combined. A: Unclipped plots, B: Clipped plots (** significant at 1% probability).

clipped plots (Table 12). In unclipped plots, the deviations were almost double those in clipped plots, indicating greater importance of additive gene action in clipped plots. Significant deviations in clipped plots further indicated that the partial dominance expressed was the result of both additive and non-additive gene action.

Deviations in percent infection of BC_r progenies from their mid-parent values were mostly non-significant in both unclipped and clipped plots. The small deviation reflects the generally small difference between resistant parent, BC_r, and the F_1 because of dominance to partial dominance of resistance. Because BC_r was similar to P_r in not responding much to clipping with an increase in disease incidence, deviations from mid-parent values were not greatly different in clipped and unclipped plots.

Deviations of BC_s from mid-parent values were significant with the exception of the unclipped BC_s of N6 x SM7 in 1982, and the clipped plots of both crosses in 1983. In 1982, the year of higher disease incidence, all of the deviations of BC_s from mid-parent values were positive, i.e., percent infection was higher in BC_s than their mid-parent values. In 1983, the year of lower infection, the deviations of BC_s from their mid-parent values were negative, i.e., the percent infection in BC_s was lower than the mid-parent values but the differences were significant in unclipped plots and non-significant in clipped plots, showing dominance in unclipped plots and additivity in clipped plots. This indicated partial dominance of resistance leading to additiv-

Table 12. Deviation in percent head smut infection, of the F_1 generation and backcrosses from their mid-parent values, N6 x SD-1 and N6 x SM7.

		1982			1983		
		Deviation			Deviation		
Treatment	Pedigree	F_1 - MP	BC_r - MP	BC_s - MP	F_1 - MP	BC_r - MP	BC_s - MP
Unclipped	N6 x SD-1	-40.8**	-3.0 NS	10.8**	-39.8**	-2.8 NS	-17.1**
	N6 x SM7	-45.8**	0.2 NS	4.4 NS	-44.2**	-1.5 NS	-16.0**
Clipped	N6 x SD-1	-9.4*	-6.8†	12.8*	-17.4**	-5.0**	-1.2 NS
	N6 x SM7	-29.2**	4.2†	18.9**	-16.4**	1.6 NS	-2.7 NS
Unclipped + Clipped	N6 x SD-1	-25.0**	-5.0†	11.9**	-28.6**	-4.0*	-9.2**
	N6 x SM7	-37.5**	2.2†	11.7**	-30.4**	0.02 NS	-9.4**

*Significant at 5% probability (greater than .05 x S.E.).

**Significant at 1% probability (greater than .01 x S.E.).

†Higher than S.E.

NS Non-significant.

ity when disease incidence was higher, and a slight dominance of susceptibility when disease incidence was severe. These results appear to be similar to those of Ma et al. (27) who obtained the effects of additive genes, dominant genes, or the interaction of non-allelic genes in their preliminary study on the genetics of resistance to head smut in corn.

Chi square tests of fit to qualitative gene ratios

Because of the strong dominance of resistance in some cases, e.g., unclipped plots in 1982 and 1983, the ratios of uninfected: infected plants in the F_2 and BC_s generations were tested for goodness of fit to one and two-gene mendelian ratios using a two-class system (healthy and infected), used by Scott and Rosenkranz (37). In these tests, (Tables 36 and 37) unclipped plants of F_2 progenies of N6 x SD-1 and N6 x SM7 in 1982, and the clipped plants F_2 progenies of N6 x SD-1 in 1983 fit 3:1 expected ratios. Observed ratios of uninfected:infected plants in the unclipped BC_s of N6 x SM7 in 1982 fit the expected ratio of 1:1. These fits occurred because the percent infection in the unclipped plots in 1982, the year of higher infection, and in the clipped plots in 1983, the year of lower infection, was close to 25% in the F_2 and 50% in BC_s. Thus, in these particular populations the observed ratios indicated control of resistance by a single dominant gene. Probably, resistance to head smut is not a qualitative character controlled by a single major gene with complete dominance. This level of infection is only one of the levels observed in the same

populations. The other ratios in F_2 and BC_s populations did not fit the qualitative expected ratios for either one or two genes.

It is important to recognize that the varying disease incidence, as affected by environment and factors which might predispose the corn plant to infection, can determine the apparent inheritance pattern in a given test. All corn genotypes inoculated in the study, including N6, can be infected. In this program, N6 has been infected readily by injection (unpublished M.S. thesis research of L. Banyra), and thus is not immune. If infection pressure is high enough, no resistance would be demonstrated. However, in our field studies even cv. N6 at the highest level of infection (clipping, 1982) remained almost free of infection.

In this study, the level of disease incidence obtained in the clipped plots probably exceeded levels normally encountered in commercial corn production. Thus, from an economic viewpoint, the dominance of resistance observed at the lower disease incidence of the unclipped plots may be more realistic than the trend toward additive gene action expressed under a higher disease incidence.

Although the chi square test indicated that qualitative inheritance could be involved, these studies have generally shown quantitative dominance of genes conditioning resistance under moderate disease incidence and partial dominance including additive gene action at higher disease incidence. Another point favoring quantitative inheritance is the variability for susceptibility found among commercial corn cultivars. Evidence is greater that resistance to head smut is not inherited by simple

mendelian inheritance but is a polygenic character inherited in a quantitative manner. It should be noted, however, that even if major mendelian genes were involved in control of resistance, the expression of resistance would tend to be quantitative because of the interactions between plant genotype and the environment. Infection of any particular plant would depend on its genotype and whether the infection potential, comprised of environmental factors affecting the host and pathogen, and inoculum density, exceeded a threshold level. Normal field variations in soil conditions and other factors affecting soil moisture, for example, preclude uniform infection, even in a genetically uniform corn line or cultivar. In this study, it was also noted that genotypes in the middle range of susceptibility, e.g., the F_1, F_2, and BC_s progenies, appear to be more sensitive to environment, with respect to variations in disease incidence, than the extreme resistant or susceptible genotypes.

Gene effects

To quantify the components of partial dominance, all gene effects were calculated using Gamble's generation mean analysis (16). This analyses indicated that resistance to head smut is determined by both additive and non-additive gene effects (Table 13), as summarized below.

Mean effects (m): The mean gene effects were significant in all populations.

Additive effects (a): Additive effects were significant in all of the populations of both unclipped and clipped plots and

were in the same direction. This again showed more importance of additive gene effects than non-additive gene effects. The magnitude of additive gene effects was higher in clipped plots than in unclipped plots. These results confirmed the previous indication, by analyses of the deviation from mid-parent values, that additivity becomes more important in clipped plots.

Dominance effects (d): The dominance effects were significant in N6 x SM7 in 1982 where differences of F_1 and mid-parent values were also higher. The dominance effects expressing resistance (negative signs) were mostly higher in unclipped than in clipped plots, which is in agreement with the results obtained previously by other methods.

Additive x additive epistasis (aa): Additive x additive epistatic gene effects were lower in magnitude in 1982 when additive effects were higher. In 1983, additive x additive epistatic gene effects were higher in magnitude while additive effects were lower.

Additive x dominance epistasis (ad): Additive x dominance epistatic effects were negative and higher in magnitude in 1982 while positive and lower in magnitude in 1983. Mostly, the additive x dominance epistatic effects were higher in magnitude when dominance effects were non-significant.

Dominance x dominance epistasis (dd): With few exceptions, dominance x dominance epistatic effects were higher when dominance effects were also higher.

Table 13. Estimates of the six gene effecta for N6 x SD-1 and N6 x SM7, 1982 and 1983, by Generation Mean Analyses.

		1982						1983					
		Gene effects[1]						Gene effecta					
Cross	Treatment	m	a	d	aa	ad	dd	m	a	d	aa	ad	dd
N6 x SD-1	Unclipped	27.0**	-60.0**	-8.8 NS	32.1**	-13.8**	-47.9**	8.3**	-30.2**	-7.4 NS	32.4**	14.3**	7.6 NS
	Clipped	40.4**	-65.3**	5.0 NS	14.4†	-19.6**	-26.5†	24.8**	-49.8**	24.0**	41.4**	3.8†	-28.9**
	Unclipped + Clipped	33.0**	-62.8**	-2.4 NS	22.7*	-17.0**	-36.4*	16.6**	-40.0**	8.0†	36.6**	5.2**	-10.3†
N6 x SM7	Unclipped	22.8**	-47.4**	-30.9**	15.0†	-4.2†	-24.2†	9.0**	-32.0**	-10.0†	34.3**	14.6**	0.7 NS
	Clipped	40.3**	-56.4**	-35.8**	-6.6 NS	-14.7**	-40.0*	31.3**	-43.2**	17.2†	33.6*	4.3†	-31.3†
	Unclipped + Clipped	31.6**	-51.9**	-33.4**	4.1 NS	-9.5*	-32.0**	20.1**	-37.7**	3.7 NS	34.0**	9.4**	-15.4†

[1] m = mean effecta; a = additive; d = dominance; aa = additive x additive; ad = additive x dominance; dd = dominance x dominance.

* Significant at 5% probability.

** Significant at 1% probability.

† Higher than S.E.

NS Non-significant.

To simplify statistical procedures, all genetic models used in the study of quantitative inheritance have been based on certain assumptions. Gamble (16) makes the following assumptions in the genetic models used in the present studies for the calculations of gene effects: 1) multiple alleles absent; 2) linkage absent; 3) lethal genes absent; 4) constant viability for all genotypes; and 5) environmental effects additive with the genotypic values. No serious bias would be expected in the estimates of the parameters from assumptions 1, 3, and 4, because the only segregating populations in this study were F_2 and first backcross generations of the cross between two homozygous lines. The assumption that environmental effects are additive with the genotypic value would probably not be expected. Robinson and Comstock (36) suggest that estimates of additive genetic variance may be biased upward because of genotype x environment interaction. This bias applies to these studies because the experiment was conducted at a single location. Perhaps this bias resulted in higher additive gene effects than other gene effects in both unclipped and clipped plots. Since only early generations of crosses were used, an equilibrium of linkage relationships is improbable; if linkage occurs, however, epistasis would be biased.

In summary, the analyses of gene effects indicates that resistance to head smut is controlled by dominant, additive and epistatic gene effects. Additive gene effects were more important than other gene effects under conditions of higher infection while dominance was expressed under conditions of lower infection.

Overall, in all populations, additive gene effects were more important than non-additive gene effects. In individual epistatic gene effects, additive x additive appeared to contribute more to the resistance of head smut in the crosses studied while additive x dominance contributed least. According to available information, gene effects had not been calculated for head smut of corn and sorghum and other related diseases.

Minimum number of genes

The minimum number of genes calculated by using the formula of Castle and Wright (7) ranged from about 6 to 25 (Table 14). The gene numbers were almost the same for the two crosses. The high infection, both in the year 1982 and in clipped plots, tended to show a smaller number of genes. This appeared to be the result of an increase in genetic variance in clipped plots in segregating F_2 populations by an increase in total variance due to higher infection, while infection in non-segregating populations of parents and F_1 was not increased. The average gene number for different treatments and different seasons was 12.9.

This estimate of minimum number of gene pairs would help decide the number of plants to be included in breeding schemes to cover all the variation in resistance present without using an excessive number of plants. According to these estimates of minimum number of genes, moderate population sizes are sufficient.

On the basis of estimates of a relatively small number of genes controlling resistance to head smut, higher heritability estimates were expected. The heritability estimates of a trait

Table 14. Minimum number of gene pairs[1] controlling resistance in N6 x SD-1 and N6 x SM7, 1982 and 1983.

Cross	Treatment	Number of genes
Year 1982		
N6 x SD-1	Unclipped	6.8
	Clipped	14.2
Mean	Unclipped + Clipped	15.5
N6 x SM7	Unclipped	9.2
	Clipped	8.1
Mean	Unclipped + Clipped	8.7
Year 1983		
N6 x SD-1	Unclipped	25.2
	Clipped	8.4
Mean	Unclipped + Clipped	16.8
N6 x SM7	Unclipped	25.5
	Clipped	5.9
Mean	Unclipped + Clipped	15.7
Mean (1982 + 1983)		12.9

[1]Using method of Castle and Wright (7).

under selection facilitates estimation of the gain which can be expected by making selection of resistant plants in a segregating population.

Broad-sense heritability

The estimates of broad-sense heritability of resistance were similar in the two crosses. However, heritability estimates were higher in 1982 than in 1983 (Table 15). Similarly, in most cases, heritability estimates were higher in clipped plots than in unclipped plots. This suggested that higher disease incidence in 1982 and from clipping increased heritability due to an increase in genetic variance due to an increase in total variance in F_2 populations. These results indicated that selections made under higher disease incidence caused by clipping would have a higher proportion of resistant plants during the next generation. The heritability estimates ranged from 60.7% to 92.2%. These estimates are relatively high as compared to the broad-sense heritability estimates of other quantitative characters of corn.

Table 15. Broad-sense heritability estimates for resistance to head smut in N6 x SD-1 and N6 x SM7, 1982 and 1983.

Cross	Treatment	Broad-sense heritability[1] (%)
Year 1982		
N6 x SD-1	Unclipped	92.2
	Clipped	84.9
Mean	Unclipped + Clipped	88.5
N6 x SM7	Unclipped	81.2
	Clipped	82.8
Mean	Unclipped + Clipped	82.0
Year 1983		
N6 x SD-1	Unclipped	60.7
	Clipped	72.1
Mean	Unclipped + Clipped	66.4
N6 x SM7	Unclipped	71.5
	Clipped	84.7
Mean	Unclipped + Clipped	78.1
Mean (1982 + 1983)		78.8

[1]Using variance of segregating and non-segregating populations.

V. GENERAL DISCUSSION

These studies have indicated that at a low disease incidence the genes conditioning resistance show dominance, while less dominance is shown with a trend toward additivity at higher levels of infection. At high levels of disease incidence resulting from predisposing treatment of seedlings (clipping), dominance of susceptibility starts to become apparent. Under high infection incidence promoted by high inoculum and predisposition, the mechanisms of resistance are overcome in plants which exhibit resistance under more natural and more moderate levels of infection. It is anticipated that a very severe disease pressure, such as could be obtained by injection of seedlings in the greenhouse, would result in a high disease incidence in the most resistant lines such as N6. This plasticity of gene action expressed as the gradual decrease in levels of resistance with the increase in the levels of infection can be due to the plant's survival mechanism.

Under commercial production conditions, where head smut incidence in infested fields is most often in the range of 0-20%, the hypothesis that resistance is dominant would be reasonable and commercially applicable. When disease incidence is higher, as it occasionally is, even under commercial conditions, the dominance of resistance would break down, and because partial dominance to additivity would be expressed, plants previously classified as resistant would have an increased chance for infection. Breeding methods capitalizing on both additive and non-additive gene action

should be adopted to develop varieties resistant to head smut of corn.

Breeding methods

Reciprocal recurrent selection. The breeding procedure reciprocal recurrent selection, proposed by Comstock et al. (8) appears to be the best available to meet the requirements. Sufficient resistant plants should be selected in each generation to avoid inbreeding and to maximize genes conditioning resistance by taking advantage of variability present in populations used. Reciprocal recurrent selection makes maximum use of both general and specific combining abilities, using both additive and non-additive genes, respectively.

Modified mass selection. Another simple method of breeding, modified mass selection, can also be used in populations heterogeneous for resistance to head smut. Usually this method requires two years per cycle, the first year for selfing selected healthy plants and the second year for pollinating selfed seed progenies with the pollen collected from all selected plants. If techniques could be developed to detect infection in plants before anthesis, it would be possible to cross the selected plants during the same year. However, at present infection can usually be detected only after pollination is complete. This mass selection procedure takes advantage of additive gene action. The resulting inbred lines can then be tested for specific combining ability which takes advantage of non-additive gene action.

VI. SUMMARY AND CONCLUSIONS

1. Disease incidence was adequate for determination of inheritance of resistance in both 1982 and 1983. The disease incidence was significantly higher in 1982 than in 1983.

2. Disease incidence was significantly higher in clipped plots than in unclipped plots because of predisposition of clipped plants to infection.

3. There was no association between disease incidence and percent height reduction due to infection. The infected plants in populations with a low percent infection were as stunted by disease as the infected plants in populations with high percent infection. In some cases the clipped plants showed a greater percent height reduction from infection than did unclipped plants.

4. Percent infection was significantly higher in sweet endosperm plants than in dent endosperm plants.

5. Reciprocal cross differences for percent infection in F_1 and F_2 were non-significant. However, in backcrosses to either the resistant or susceptible parent, the infection was higher when F_1 and F_1R were used as male parent than when F_1 and F_1R were used as female.

6. Resistance to head smut is a polygenic character inherited in a quantitative manner.

7. The comparison of genetic populations graphically and by analysis of variance showed dominance of genes conditioning resistance at lower levels of infection in unclipped plots and partial dominance of resistance at higher levels of infection in

clipped plots. Overall, additive gene effects appeared to be more important.

8. Similarly, the deviation of F_1, BC_r, and BC_s from their respective mid-parent values indicated dominance of resistance at lower levels of infection, additive gene action at higher levels of infection, and slight dominance of susceptibility at severe infections.

9. The calculations of gene effects indicated the existence of both additive and non-additive (dominance and epistasis) gene effects. The magnitude of additive gene effects was higher in clipped plots while the magnitude of dominance effects was higher in unclipped plots. Among epistatic gene effects, additive x additive effects were more important and additive x dominance effects were least important in determining level of resistance.

10. The average number of genes involved in conditioning resistance were relatively small (12.9) and the average heritability estimate of resistance was relatively high (78.8%).

11. Breeding by reciprocal recurrent selection and modified mass selection was proposed.

It was concluded that resistance to head smut is a polygenic character segregating in a quantitative manner. At lower levels of infection, genes conditioning resistance showed dominance; at higher levels of infection genes tended to show additivity; and, at the highest disease incidence obtained in these studies, susceptibility showed slight dominance. The mode of inheritance

apparently was dependent on plant genotype, environment, and factors which predisposed plants to infection.

VII. LITERATURE CITED

1. Ainsworth, G. E. 1965. Sphacelotheca reiliana. C.M.I. Descriptions of pathogenic fungi and bacteria. No. 73.

2. Baggett, J. R., and P. A. Koepsell. 1983. Field inoculation of sweet corn with the head smut pathogen (Sphacelotheca reiliana). HortScience 18:67-68.

3. Baier, W., and W. Kruger. 1962. Sphacelotheca reiliana of maize. II. Field studies on the effect of soil conditions. S. Afr. J. Agric. Sci. 5:183-190.

4. Bojanowski, J. 1969. Studies of inheritance of reaction to common smut in corn. Theoret. Appl. Genetics 39:32-42.

5. Bressman, E. N., and H. P. Barss. 1933. Experiments with head smut of corn in western Oregon. Phytopathology 23:396-403.

6. Casady, A. J. 1961. Inheritance of resistance to physiologic races 1, 2, and 3 of Sphacelotheca sorghi in sorghum crop. Crop Sci. 1:63-68.

7. Castle, W. E. 1921. An improved method of estimating the number of genetic factors concerned in cases of blending inheritance. Science 54:223.

8. Comstock, R. E., H. F. Robinson, and P. H. Harvey. 1949. A breeding procedure designed to make maximum use of both general and specific combining ability. Agron. J. 41:360-367.

9. Cooper, J. I., and A. T. Jones. 1983. Responses of plants to viruses: Proposals for the use of terms. Phytopathology 73:127-128.

10. Christensen, J. J. 1926. The relation of soil temperature and soil moisture to the development of head smut of sorghum. Phytopathology 16:353-357.

11. Edmunds, L. K. 1963. Use of sporidial hypodermic injection to test sorghum for head smut resistance. Plant Dis. Rep. 47:909-913.

12. Falconer, D. S. 1965. The inheritance of liability to certain diseases, estimated from the incidence among relatives. Ann. Hum. Genet., Lond. 29:51-76.

13. Fenwick, H. S., and W. R. Simpson. 1967. Suppression of corn head smut by in-furrow application of pentachloronitrobenzene. Plant Dis. Rep. 51:626-628.

14. Foster, J. H., and R. A. Frederiksen. 1977. Symptoms of head smut in maize seedlings and evaluation of hybrids and inbreds. Texas Agric. Exp. Sta. PR-3432, pp. 4.

15. Frederiksen, R. A., R. W. Berry, and J. H. Foster. 1976. Head smut of maize in Texas. Plant Dis. Rep. 60:610-611.

16. Gamble, E. E. 1962. Gene effects in corn (Zea mays L.). I. Separation and relative importance of gene effects for yield. Can. J. Plant Sci. 42:339-348.

17. _____. 1962. Gene effects in corn (Zea mays L.). II. Relative importance of gene effects for plant height and certain component attributes of yield. Can. J. Plant Sci. 42:349-358.

18. Halisky, P. M. 1962. Prevalence and pathogenicity of Sphacelotheca reiliana causing head smut of field corn in California. Phytopathology 52:199-202.

19. _____. 1963. Head smut of sorghum, sudangrass, and corn, caused by Sphacelotheca reiliana (Kühn) Clint. Hilgardia 34:287-304.

20. Hanna, W. F. 1926. Studies in the physiology and cytology of Ustilago zeae and Sorosporium reilianum. Phytopathology 19:415-441.

21. Hartl, D. L. 1977. Quantitative and threshold traits. IN: Our uncertain heritage, pp. 404-424. J. B. Lippincott Company, New York.

22. Hayman, B. I. 1958. The separation of epistatic from additive and dominance variation in generation means. Heredity 12:371-390.

23. _____. 1960. The separation of epistatic from additive and dominance variation in generation means II. Genetics 31:133-146.

24. Jacks, H. 1951. Soil disinfection X. Preliminary report on control of head smut (Sorosporium reilianum (Kühn) McAlpine) of maize (Zea mays L.). New Zealand J. Sci. Tech. (A) 33:37-44.

25. Kim, S. K., and J. L. Brewbaker. 1977. Inheritance of general resistance in maize to Puccinia sorghi Schw. Crop Sci. 17:456-461.

26. Kruger, W. 1962. Sphacelotheca reiliana on maize. I. Infection and control studies. S. Afr. J. Agric. Sci. 5:43-56.

27. Ma, B. Y., Y. L. Li, and S. K. Duan. 1983. Preliminary study on the genetics of resistance to head smut in maize. Scientia Agricultura Sinica. No. 4:12-17.

28. Mahmud, I., and H. H. Kramer. 1951. Segregation for yield, height, and maturity following a soybean cross. Agron. J. 43:605-609.

29. Marcy, D. E. 1937. Inheritance of resistance to the loose and covered kernel smuts of sorghum. I. Dwarf Yellow Milo hybrids. Bull. Torrey Club 64:209-228.

30. _____. 1937. Inheritance of resistance to the loose and covered kernel smuts of sorghum. II. Feterita hybrids. Bull. Torrey Club 64:245-267.

31. Mei, Z. B., G. Y. Yu, H. C. Wang, G. D. Hou, and J. Y. Li. 1982. Pattern of inheritance of resistance to head smut in maize. Shanxi Nongye Kexue No. 11:10-13. (Pl. Breed. Abstr., 1983, 8734).

32. Miller, R. C. 1978. Inoculation techniques with Sphacelotheca reiliana, the cause of head smut. Texas Agric. Exp. Sta. MP-1373:39-40.

33. Potter, A. A. 1914. Head smut of sorghum and maize. J. Agric. Res. 2:339-368.

34. Reed, G. M., M. Swabey, and L. A. Kolk. 1927. Experimental studies on head smut of corn and sorghum. Bull. Torrey Club 54:295-310.

35. Reifschneider, F. J. B., and D. C. Arny. 1983. Inheritance of resistance in maize to Kabatiella zeae. Crop Sci. 23:615-616.

36. Robinson, H. F., and R. E. Comstock. 1955. An anlysis of genetic variability in corn with reference to probable effects of selection. Cold Spring Harbor Symposia on Quant. Biol. 20:127-135.

37. Scott, G. E., and E. Rosenkranz. 1982. A new method to determine the number of genes for resistance to maize dwarf mosaic in maize. Crop Sci. 22:756-761.

38. Simpson, W. R. 1966. Head smut of corn in Idaho. Plant Dis. Rep. 50:215-216.

39. Stewart, R. B., and L. Reyes. 1958. Head smut of sorghum and varietal reaction. Plant Dis. Rep. 42:1133-1140.

40. Stromberg, E. L. 1981. Head smut of maize, a new disease in Minnesota. Phytopathology 71:906. (Abstr.)

41. _____. 1982. Evaluation of maize hybrids and inbreds for resistance to Sphacelotheca reiliana. Phytopathology 72:943: (Abstr.)

42. Tuleen, D. M., and R. A. Frederiksen. 1982. Evaluating a crop loss model for head smut of sorghum. Phytopathology 72:1278-1280.

43. Weber, C. R., and B. R. Moorthy. 1952. Heritable and nonheritable relationships and variability of oil content and agronomic characters in the F_2 generation of soybean crosses. Agron. J. 44:202-209.

APPENDIX

Table 16. Analysis of variance for transformed percent head smut infection in dent plots, N6 x SD-1.

Source	1982			1983		
	D.F.	M.S.	F value	D.F.	M.S.	F value
Reps	3	192.8	1.7 NS	3	105.8	3.9*
Population	11[z]	3163.6	27.1**	12	1362.2	50.7**
Clipping	1	3115.4	26.7**	1	5129.4	190.8**
Population x Clipping	11	209.8	1.8 NS	12	62.0	2.3*
Error	69	116.7		75	26.9	
Total	95			103		

* Significant at 5% probability.

** Significant at 1% probability.

NS Non-significant.

[z] In 1982 F_1R missing.

Table 17. Analysis of variance for transformed percent head smut infection in dent plots, N6 x SM7.

Source	1982			1983		
	D.F.	M.S.	F value	D.F.	M.S.	F value
Reps	3	895.6	2.8*	3	613.9	11.4**
Population	11[z]	5979.2	18.9**	12	1388.1	25.9**
Clipping	1	6584.9	20.8**	1	7727.5	144.0**
Population x Clipping	11	1538.8	4.9**	12	74.3	1.4 NS
Error	69	316.9		75	53.6	
Total	95			103		

* Significant at 5% probability.

** Significant at 1% probability.

NS Non-significant.

[z] In 1982 F_1 missing.

Table 18. Analysis of variance for transformed percent head smut infection in sweet plots, N6 x SD-1.

Source	D.F.	1982 M.S.	1982 F value	1983 M.S.	1983 F value
Reps	3	59.8	1.5 NS	116.1	3.9*
Population	6	1720.6	44.2**	2276.9	76.1**
Clipping	1	269.4	6.9*	3445.3	115.1**
Population x Clipping	6	72.6	1.9 NS	108.3	3.6**
Error	39	38.9		29.9	
Total	55				

* Significant at 5% probability.

** Significant at 1% probability.

NS Non-significant.

Table 19. Analysis of variance for transformed percent head smut infection in sweet plots, N6 x SM7.

Source	D.F.	1982		1983	
		M.S.	F value	M.S.	F value
Reps	3	152.5	1.5 NS	223.9	4.3*
Population	6	1169.8	11.6**	2551.0	48.7**
Clipping	1	842.0	8.3**	2983.7	57.0**
Population x Clipping	6	112.4	1.1 NS	81.2	1.6 NS
Error	39	101.3		52.4	
Total	55				

* Significant at 5% probability.

** Significant at 1% probability.

NS Non-significant.

Table 20. Analysis of variance for transformed percent head smut infection in F_2 and backcrosses to susceptible parent (BC_s), N6 x SD-1.

Source	D.F.	1982 M.S.	1982 F value	1983 M.S.	1983 F value
Reps	3	113.6	2.7 NS	66.5	2.1 NS
Population	5	2399.6	58.0**	1870.4	59.4**
Clipping	1	1899.0	45.9**	6387.0	203.0**
Population x Clipping	5	66.0	1.6 NS	57.4	1.8 NS
Dent/Sweet	1	634.1	15.3**	951.5	30.2**
Population x Dent/Sweet	5	30.0	0.7 NS	34.9	1.1 NS
Clipping x Dent/Sweet	1	152.1	3.7 NS	48.5	1.5 NS
Population x Clipping x Dent/Sweet	5	33.0	0.8 NS	19.3	0.6 NS
Error	69	41.4		31.5	
Total	95				

** Significant at 1% probability.
NS Non-significant.

Table 21. Analysis of variance for transformed percent head smut infection in F_2 and backcrosses to susceptible parent (BC_s), N6 x SM7.

Source	D.F.	1982 M.S.	1982 F value	1983 M.S.	1983 F value
Reps	3	283.8	4.5**	585.8	13.6**
Population	5	1498.9	23.6**	1627.0	37.9**
Clipping	1	2770.9	43.7**	6973.6	162.5**
Population x Clipping	5	25.2	0.4 NS	31.5	0.7 NS
Dent/Sweet	1	172.0	2.7 NS	538.2	12.5**
Population x Dent/Sweet	5	90.2	1.4 NS	13.8	0.3 NS
Clipping x Dent/Sweet	1	31.2	0.5 NS	3.2	0.1 NS
Population x Clipping x Dent/Sweet	5	80.5	1.3 NS	10.2	0.2 NS
Error	69	63.4		42.9	
Total	95				

** Significant at 1% probability.
NS Non-significant.

Table 22. Analysis of variance for transformed percent head smut infection in clipped and unclipped plots with dent and sweet plots combined, N6 x SD-1.

Source	1982			1983		
	D.F.	M.S.	F value	D.F.	M.S.	F value
Reps	3	206.8	2.0 NS	3	111.6	5.0**
Population	12[z]	4444.6	43.7**	13	3029.9	136.0**
Clipping	1	1961.3	19.3**	1	5027.5	225.7**
Population x Clipping	12	225.1	2.2*	13	95.4	4.3**
Error	75	101.6		81	22.3	
Total	103			111		

* Significant at 5% probability.

** Signficiant at 1% probability.

NS Non-significant.

[z] In 1982 F_1R missing.

Table 23. Analysis of variance for transformed percent head smut infection in clipped and unclipped plots with dent and sweet plots combined, N6 x SM7.

Source	1982			1983		
	D.F.	M.S.	F value	D.F.	M.S.	F value
Reps	3	93.5	1.9 NS	3	484.7	9.5**
Population	12[z]	3793.4	77.1**	13	3246.0	63.8**
Clipping	1	1932.8	39.3**	1	6798.5	133.6**
Population x Clipping	12	98.1	2.0*	13	116.7	2.3*
Error	75	49.2		81	50.9	
Total	103			111		

* Significant at 5% probability.

** Significant at 1% probability.

NS Non-significant.

[z] In 1982 F_1 missing.

Table 24. Analysis of variance for transformed percent head smut infection in unclipped plots with dent and sweet plots combined, N6 x SD-1.

Source	1982			1983		
	D.F.	M.S.	F value	D.F.	M.S.	F value
Reps	3	21.2	0.4 NS	3	42.5	1.8 NS
Population	12[z]	2552.3	53.6**	13	1555.7	67.6**
Error	36	47.6		39	23.0	
Total	51			55		

** Significant at 1% probability.

NS Non-significant.

[z] In 1982 F_1R missing.

Table 25. Analysis of variance for transformed percent head smut infection in unclipped plots with dent and sweet plots combined, N6 x SM7.

Source	1982			1983		
	D.F.	M.S.	F value	D.F.	M.S.	F value
Reps	3	38.9	0.9 NS	3	67.2	2.4 NS
Population	12[z]	2020.5	45.9**	13	1804.7	64.6**
Error	36	44.0		39	27.9	
Total	51			55		

** Significant at 1% probability.

NS Non-significant.

[z] In 1982 F_1 missing.

Table 26. Analysis of variance for transformed percent head smut infection in clipped plots with dent and sweet plots combined, N6 x SD-1.

Source	1982			1983		
	D.F.	M.S.	F value	D.F.	M.S.	F value
Reps	3	451.4	3.2*	3	93.6	4.4**
Population	12[z]	2114.4	14.9**	13	1569.7	73.4**
Error	36	141.8		39	21.4	
Total	51			55		

* Significant at 5% probability.

** Significant at 1% probability.

[z] In 1982 F_1R missing.

Table 27. Analysis of variance for transformed percent head smut infection in clipped plots with dent and sweet plots combined, N6 x SM7.

Source	1982			1983		
	D.F.	M.S.	F value	D.F.	M.S.	F value
Reps	3	82.3	1.5 NS	3	529.3	7.7**
Population	12[z]	1871.0	33.3**	13	1558.0	22.5**
Error	36	56.3		39	69.1	
Total	51			55		

** Significant at 1% probability.

NS Non-significant.

[z] In 1982 F_1 missing.

Table 28. Analysis of variance for transformed percent head smut infection in clipped and unclipped plots with dent and sweet plots combined in 1982 and 1983.

Source	D.F.	N6 x SD-1		N6 x SM7	
		M.S.	F value	M.S.	F value
Year	1	3175.1	17.9**	95.2	0.34 NS
Reps (Year)	6	177.4	2.9*	282.5	5.6**
Population	12	7471.1	31.9**	7062.2	36.0**
Clipping	1	6189.6	24.0 NS	7283.0	13.6 NS
Year x Population	12	234.1	3.8**	196.0	3.9**
Year x Clipping	1	257.4	4.2*	536.7	10.7**
Population x Clipping	12	235.6	3.8**	207.0	4.1**
Year x Population x Clipping	12	87.6	1.4 NS	7.9	0.16 NS
Error	150	61.2		50.2	
Total	207				

* Significant at 5% probability.
** Significant at 1% probability.
NS Non-significant.

Table 29. Analysis of variance for transformed percent height reduction by head smut infection in dent plots, 1982.

Source	D.F.	N6 x SD-1		N6 x SM7	
		M.S.	F value	M.S.	F value
Reps	3	16.2	0.4 NS	108.6	2.6 NS
Population	11	47.8	1.3 NS	27.2	0.6 NS
Clipping	1	91.2	2.5 NS	90.6	2.2 NS
Population x Clipping	11	25.0	0.7 NS	26.0	0.6 NS
Error	69	36.3		42.0	
Total	95				

NS Non-significant.

Table 30. Analysis of variance for transformed percent height reduction by head smut infection in sweet plots, 1982.

Source	D.F.	N6 x SD-1		N6 x SM7	
		M.S.	F value	M.S.	F value
Reps	3	10.5	0.5 NS	43.2	2.2 NS
Population	6	18.8	0.9 NS	27.6	1.4 NS
Clipping	1	69.5	3.4 NS	167.0	8.6**
Population x Clipping	6	11.4	0.6 NS	16.2	0.8 NS
Error	39	20.4		19.4	
Total	55				

** Significant at 1% probability.

NS Non-significant.

Table 31. Analysis of variance for transformed percent height reduction by head smut infection in F_2 and backcrosses to susceptible parent (BC_s), 1982.

Source	D.F.	N6 x SD-1 M.S.	N6 x SD-1 F value	N6 x SM7 M.S.	N6 x SM7 F value
Reps	3	1.8	0.1 NS	55.2	3.1*
Population	5	20.1	1.2 NS	41.8	2.3 NS
Clipping	1	257.0	14.8**	331.8	18.4**
Population x Clipping	5	9.6	0.6 NS	24.8	1.4 NS
Dent/Sweet	1	3.5	0.2 NS	44.6	2.5 NS
Population x Dent/Sweet	5	3.7	0.2 NS	8.2	0.5 NS
Clipping x Dent/Sweet	1	9.8	0.6 NS	0.0	0.0 NS
Population x Clipping x Dent/Sweet	5	7.4	0.4 NS	1.9	0.1 NS
Error	69	17.3		18.0	
Total	95				

* Significant at 5% probability.
** Significant at 1% probability.
NS Non-significant.

Table 32. Analysis of variance for transformed percent height reduction by head smut infection in clipped and unclipped plots with dent and sweet plots combined, 1982.

		N6 x SD-1		N6 x SM7	
Source	D.F.	M.S.	F value	M.S.	F value
Reps	3	12.8	0.4 NS	87.6	2.3 NS
Population	12	45.2	1.4 NS	26.9	0.7 NS
Clipping	1	56.0	1.7 NS	98.6	2.6 NS
Population x Clipping	12	20.2	0.6 NS	25.2	0.7 NS
Error	75	32.2		38.4	
Total	103				

NS Non-significant.

Table 33. Analysis of variance for transformed percent height reduction by head smut infection in unclipped plots with dent and sweet plots combined, 1982.

Source	D.F.	N6 x SD-1		N6 x SM7	
		M.S.	F value	M.S.	F value
Reps	3	25.8	0.9 NS	51.0	1.3 NS
Population	12	35.7	1.2 NS	21.1	0.6 NS
Error	36	29.4		38.4	
Total	51				

NS Non-significant.

Table 34. Analysis of variance for transformed percent height reduction by head smut infection in clipped plots with dent and sweet plots combined, 1982.

Source	D.F.	N6 x SD-1		N6 x SM7	
		M.S.	F value	M.S.	F value
Reps	3	95.8	3.4*	88.8	2.4 NS
Population	12	29.7	1.0 NS	31.0	0.8 NS
Error	36	28.6		37.2	
Total	51				

* Significant at 5% probability.

NS Non-significant.

Table 35. Correlations between variables in all populations of N6 x SD-1 and N6 x SM7 on an entry mean basis.

Factor	Correlation			
	N6 x SD-1 (1982) n=192	N6 x SM7 (1982) n=192	N6 x SD-1 (1983) n=392	N6 x SM7 (1983) n=384
Clipping and % infection	0.18*	0.22**	0.36**	0.39**
Seed type and % infection	0.50**	0.51**	0.45**	0.42**
Clipping and % ht. reduction	0.20**	0.24**		
% Infection and % ht. reduction	-0.05 NS	0.14 NS		

* Significant at 5% probability.

** Significant at 1% probability.

NS Non-significant.

Table 36. Ratios of uninfected:infected plants tested for goodness of fit to one and two gene mendelian ratios, unclipped and clipped plots, N6 x SD-1, 1982 and 1983[1].

	Unclipped								Clipped							
	Observed		Expected						Observed		Expected					
			One gene			Two genes					One gene			Two genes		
Populations	No. uninf.	No. inf.	No. uninf.	No. inf.	X^2	No. uninf.	No. inf.	X^2	No. uninf.	No. inf.	No. uninf.	No. inf.	X^2	No. uninf.	No. inf.	X^2
1982																
P_r	109	4	113	0	---	113	0	---	94	0	94	0	---	94	0	---
P_s	7	222	0	229	---	0	229	---	13	148	0	161	---	0	161	---
F_1	106	11	117	0	---	117	0	---	75	50	125	0	---	125	0	---
F_2	666	219	664	221	0.02 NS	830	55	513.7**	497	339	627	209	107.0**	787	52	1626.0**
BC_r	435	17	452	0	---	452	0	---	353	42	395	0	---	395	0	---
BC_s	305	574	440	440	81.7**	659	220	246.8**	173	586	380	380	223.6**	569	190	1100.5**
1983																
P_r	349	6	355	0	---	355	0	---	298	6	304	0	---	304	0	---
P_s	20	187	0	207	---	0	209	---	9	123	0	132	---	0	132	---
F_1	430	28	458	0	---	458	0	---	301	126	427	0	---	427	0	---
F_2	1509	135	1233	411	246.2**	1541	103	10.5**	1092	359	1088	363	.04 NS	1360	91	843.6**
BC_r	975	13	988	0	---	988	0	---	778	85	863	0	---	863	0	---
BC_s	1181	549	865	865	230.2**	1298	432	41.5**	599	878	738	738	52.3**	1108	369	932.8**

[1] Assuming complete dominance;
* Significant at 5% probability;
** Significant at 1% probability;
NS Non-significant.

Table 37. Ratios of uninfected:infected plants tested for goodness of fit to one and two gene mendelian ratios, unclipped and clipped plots, N6 x SM7, 1982 and 1983[1].

	Unclipped								Clipped							
	Observed		Expected						Observed		Expected					
			One gene			Two genes					One gene			Two genes		
Populations	No. uninf.	No. inf.	No. uninf.	No. inf.	X^2	No. uninf.	No. inf.	X^2	No. uninf.	No. inf.	No. uninf.	No. inf.	X^2	No. uninf.	No. inf.	X^2
1982																
P_r	109	4	113	0	---	113	0	---	94	0	94	0	---	94	0	---
P_s	17	151	0	168	---	0	168	---	10	77	0	87	---	0	87	---
F_1	79	1	80	0	---	80	0	---	72	10	82	0	---	82	0	---
F_2	695	210	679	226	1.5 NS	848	57	441.1**	474	321	596	199	99.4**	745	50	1574.3**
BC_r	469	13	482	0	---	482	0	---	422	50	472	0	---	472	0	---
BC_s	431	429	430	430	0.0 NS	645	215	282.7**	186	420	303	303	89.6**	454	152	632.1**
1983																
P_r	349	6	355	0	---	355	0	---	298	6	304	0	---	304	0	---
P_s	9	147	0	156	---	0	156	---	3	60	0	63	---	0	63	---
F_1	439	19	458	0	---	458	0	---	249	117	366	0	---	366	0	---
F_2	1402	137	1154	388	211.8**	1443	96	18.0**	872	382	940	314	19.7**	1176	78	1250.4**
BC_r	789	12	801	0	---	801	0	---	544	89	633	0	---	633	0	---
BC_s	1088	545	816	816	179.9**	1225	408	60.6**	432	692	562	562	59.7**	843	281	799.6**

[1] Assuming complete dominance;
* Significant at 5% probability;
** Significant at 1% probability;
NS Non-significant.