ILLEGIBLE DOCUMENT

THE FOLLOWING
DOCUMENT(S) IS OF
POOR LEGIBILITY IN
THE ORIGINAL

THIS IS THE BEST COPY AVAILABLE

EVALUATION OF CORN GERMPLASM TO FUSARIUM MONILIFORME STALK ROT

Ъу

KAUSHAL KISHORE LAL

B.Sc. Agri. (HONS), University of Udaipur, India, 1970

A MASTER'S THESIS

submitted in partial fulfillment of the

requirements for the degree

MASTER OF SCIENCE

Department of Agronomy

KANSAS STATE UNIVERSITY Manhattan, Kansas

1981

Approved by:

Major Professor

SPEC COLL LD 2668 T4 1981 L34 c. 2

All200 093305

TABLE OF CONTENTS

	Page
LIST OF TABLES	· ii
INTRODUCTION	- 1
LITERATURE REVIEW	• 3
MATERIALS AND METHODS	. 12
RESULTS AND DISCUSSION	. 15
CONCLUSION	- 28
LITERATURE CITED	. 29
ACKNOWLEDGEMENTS	. 36
APPENDIX	. 38
ABSTRACT	

LIST OF TABLES

Tab	le	Page
1.	Analysis of variance of infection lengths, Experiment No. 1	- 15
2.	Duncan's Multiple Range Test for Mean Infection lengths, Experiment No. 1	- 16
3.	Analysis of Variance of infection lengths, Experiment No. 2	- 17
4.	Mean infection lengths of damage resulting from inoculation at two stages of growth (Experiment No. 2)	- 18
5.	Combined analysis of Variance of infection lengths, Experiment No. 3	. 19
6.	Mean infection lengths of entries at two different locations, Experiment No. 3	· 21
7.	Duncan's Multiple Range Test for mean infection lengths, Combined Experiment No. 3	- 22
8.	Analysis of variance of infection lengths, irrigated, Experiment No. 3	- 23
9.	Analysis of variance of infection lengths, non-irrigated, Experiment No. 3	• 23
10.	Duncan's multiple Range Test for mean infection lengths, Experiment No. 3, irrigated	- 24
11.	Duncan's Multiple Range Test for mean infection lengths, Experiment No. 3, non-irrigated	· 25
12.	Analysis of Variance of infection lengths, Tlaltizapan, Mexico, Experiment No. 4	- 26
13.	Duncan's Multiple Range Test for mean infection lengths, Experiment No. 4	- 27

INTRODUCTION

Stalk rots are disease complexes caused by numerous species of fungi and bacteria and affect a multitude of crops. Stalk rot damage is of variable importance from region to region and season to season and occasionally, epiphytotics occurs over wide areas. Severity of stalk rot varies greatly as temperature, rainfall, soil drainage, soil type, available nutrients and other conditions change and interact. Production practices such as the genotype of seed planted, date of planting, crop sequence, fertilizer treatment and plant population have marked effects on severity of infection. Mechanical injury and insect damage generally enhances stalk rot severity.

In recent years, changes in cultural practices involving high plant populations and the liberal use of fertilizers has come about. These developments have resulted in greater yields and thus greater corn stalk strength is required. Stalks weakened by rot causing losses due to broken stalks and lodging. Therefore stalk rot is an important consideration in any corn improvement program.

In 1914, Pammel (37) described <u>Fusarium</u> disease of corn in Iowa. In many cases stalks were lodged, many were barren, and the pith of diseased corn stalks was soft and essentially destroyed. Tissues were brownish or reddish in color. It was stated that Fusarium disease was likely the most important problem in the corn growing area in Iowa. Today, <u>Fusarium</u> species, particularly <u>Fusarium</u> graminearum schwabe and <u>Fusarium</u> moniliforme (Sheldon), are considered among the most destructive fungi on corn causing stalk rot, seedling blight, root rot and ear rot. Stalk rot

is particularly noticeable as corn matures during September and October. Most frequently the infection progresses upward from the adventitious roots and crown into the stalk causing premature necrosis, chaffy or rotted ears, shank breakage, ear dropping and stalk breakage. This results in yield losses, poor grain quality and problems with harvesting. Losses due to stalk rot in Kansas are variable and were 8%, 13%, 7.5%, and 9% in 1977, 1978, 1979 and 1980, respectively (51).

The primary objective of this study was to ascertain tolerance in genotypes that can be utilized in breeding programs. To achieve this objective a considerable number of genetic sources were collected and screened by inoculating genetic sources with \underline{F} , moniliforme under field conditions.

LITERATURE REVIEW

Pathogens and Symptoms

As early as 1896 and 1904, Moore (34) and Peters (42) reported on a wide spread disease of cattle and other animals known as the "stalk rot" disease. This disease occurred in the fall and early winter when cattle were grazing on corn stalks. Peters (40) suggested that the disease might have been caused by <u>Fusarium spp.</u> and the organism was described (47) as <u>Fusarium moniliforme</u> sheldon in Nebraska in 1904.

The confirmation of <u>F. moniliforme</u> as one of the major causes of stalk rot and seedling blight began with Valleau's work (57) in 1920. He isolated it from ears of corn showing a pink mold, and described it as "sporodochium, subeffuse, salmon-pink; sporophores, simple or branched, usually opposite microconidia, continuous, oblong - and generally with three septate, 25-40 \(\mu\) long.

Factors that influence stalk rot development

Moisture and temperature

Stove (54) at Wisconsin studied temperature effects on growth of <u>F. moniliforme</u> in culture and reported that the optimum growth was obtained at $26^{\circ}\text{C} - 33^{\circ}\text{C}$. Higher temperatures decreased growth with minimal growth at 37°C . Growth response was slight at $5^{\circ} - 7^{\circ}\text{C}$, optimum at 30°C and slight at $36^{\circ} - 36.5^{\circ}\text{C}$. (13).

<u>Diplodia zea</u> is another fungal irritant of corn stock rot and is most destructive in regions of heavy rainfall in late summer or during the late growing season (8). For stalk rot to develop, other conditions besides abundant precipitation must occur and they include: (a) sufficient

nutrients in the corn plant; (b) rapid growth of tissue; and (c) loosened leaf sheath. Presence of water between the leaf sheaths and the stalks is essential for germination of spores and growth of \underline{D} . \underline{zea} .

Although moisture is one of the most important factors, temperature also plays a significant role. Michaelson (32) increased the incidence of stalk rot in corn grown in the green house at a temperature of 30° C more than at 18° C when the plants were inoculated with <u>Diploidia</u> <u>zeae</u> and <u>Giberella</u> <u>zeae</u>.

Additionally, less stalk rot developed in the field in corn stalks inoculated with <u>Diplodia zeae</u> and <u>Gibberella zeae</u> when the plants were growing on wet soil, than on relatively dry soil (32). The plots were flooded with 7 to 10 cms of water about 2 weeks before inoculation. Plants inoculated and growing on non-flooded plots died 2 to 3 weeks after inoculation, whereas, those on wet ground remained green almost as long as the non-inoculated plants.

Soil Fertility

Soil fertility greatly influences the susceptibility of corn to stalk rot. Many workers now agree that stalk rot is more severe when nitrogen is in excess in relation to potassium (39, 34, 25, 26, 1). Accordingly, nitrogen tends to increase stalk rot severity and potassium tends to decrease it.

Spencer and McNew (51) found that excess nitrogen and deficient potassium greatly increased bacterial wilt in sweet corn. Low phosphorous levels resulted in necrotic lesions, and at high levels dwarfing and necrosis resulted. Phosphorous has not been reported, in general, as an important factor affecting development of stalk rot. In a green house study, Thayer and Williams (56) found that phosphorous decreased severity of stalk rot

and concluded that high levels of phosphorous would protect corn against the disease. Koehler (27) concluded that potassium chloride fertilizer decreased stalk rot, but this was not true when potassium sulfate or potassium metaphosphate was used. It was suggested that the decrease in disease resulted from applying chloride and not from applying potassium.

Hoffer and Carr (14) found that an accumulation of aluminum and iron in the corn plant rendered the stalks more susceptible to invasion by stalk rot organisms. Lime did not influence the percentage of broken stalks, but did greatly decrease the percentage of leaning stalks (25). Otto and Everett (36) reported differences in stalk rot in corn hybrids growing in fertility plots though it was known that corn hybrids differ in their ability to utilize nutrients (29). The various studies on the influence of soil fertility on stalk rot have been made with naturally occurring stalk rot or with stalk rot resulting from inoculation with different stalk rotting organisms. None of the studies have attempted to show that the response to fertilizer might vary because of the pathogens. However, certain applications of fertilizer resulted in stalk rot being more severe when some corn hybrids were inoculated with one of the pathogens but not when inoculated with the other (5). White et al (59) reported that stalk rot from natural infection and stalk rot following inoculation with Diplodia maydis or Colletotrichum graminicola decreased with increasing nitrogen rates which he contributed to the continuous supply of nitrogen throughout the growing season.

Isolation, Inoculation and Data Collection

Foley (10, 12) reported that Fusarium moniliforms can be isolated from

kernels, roots, leaf sheaths, axillary buds and stalks of corn with the highest frequency of isolates from leaf sheaths. Similar results were reported by Kucharek and Kommedahl (28).

Numerous techniques utilized tolerance. Genotypes determined to be resistant to stalk rot when artificially inoculated may not necessarily maintain the resistance under natural infection. Zuber et al (65) found an inbred line resistant to stalk rot when inoculated with Gibberella zeae, but was susceptible to natural infection in Illinois (27). Others (2, 23, 30, 47, 52) reported progress in breeding for resistance to stalk rot on the basis of artificial stalk inoculation. Data obtained from inoculated genotypes is greatly influenced by the final reading date.

Although most workers record stalk rot notes 3 to 4 weeks after inoculation, Koehler (27) and Hooker (17) concluded that final data on stalk rot ratings should not be taken until 3-4 weeks after inoculation.

Resistance

Resistance to stalk rot involves many physiological, morphological, and perhaps functional characteristics, which in turn may be influenced by many factors. No inbred or hybrid of corn has been reported to be immune to stalk rot. Certain hybrid varieties are now grown in the corn belt that appear to be moderately tolerant to stalk rot. Progress has been made on techniques for testing lines and varieties of corn, although there is no general agreement on the method of selecting for resistance. The development of varieties tolerant to stalk rot is the only practical method for controlling the disease (36). Numerous investigators (22, 47, 55) have reported pronounced variation in reactions of inbreds and hybrids.

THIS BOOK CONTAINS **NUMEROUS PAGES** WITH THE ORIGINAL PRINTING BEING SKEWED DIFFERENTLY FROM THE TOP OF THE PAGE TO THE BOTTOM.

THIS IS AS RECEIVED FROM THE CUSTOMER.

Resistance to one or more pathogens has been frequently reported, but there is no evidence that any variety is resistant to all stalk-rotting organisms. Varieties considered resistant to <u>Diplodia</u> stalk rot were reported to be very susceptible to bacterial stalk rot in Egypt (43). Kirmelasvili (24) reported that varieties resistant to <u>F. moniliforme</u> in eastern Georgia were susceptible in western Georgia. Mesterhazy (31) reported a highly significant correlation between root rot and basal stalk rot disease indices, based on natural infection, indicating that selection for resistant roots gives resistant or tolerant stalks. He also concluded that the stalk splitting method was less laborius than artificial inoculation. Sprague (52) stated that reaction of corn to <u>Diplodia zeae</u> provided a measure of resistance to stalk rot in general but Hooker (16) reported that varieties resistant to one pathogen were not necessarily resistant to other pathogens and resistant in one part of a plant does not insure resistance in other tissues. Similar results were reported by others (7, 41, 60, 62).

Open-pollinated corn varieties also differ in susceptibility to stalk rots. Sidorov et al (48) tested 38 inbreds, hybrids and varieties of corn for resistance to <u>Diplodia zeae</u> in Russia and reported marked difference in susceptibility. Flint corns were more resistant than dent corns. Sansom (44) reported that all local varieties in Rhodesia were susceptible to stalk rot, but improvement has been made by hybridization and selection.

According to Sprague (52), there are marked differences in frequency of genes for resistance to stalk rot in different varieties. Smith et al (50) indicated that dominant factors imparted resistance to <u>Diplodia zeae</u>. Jugenheimer (22) studied responses of several inbreds, single crosses and

top crosses of corn and reported that top cross progenies were more resistant than the inbreds involved. He also found some crosses were less resistant than the parental inbred line and concluded that, although resistance to stalk rots was complex and partially dominant, it appeared to be due to many factors. In addition, he reported that some inbreds were more potent in transmitting resistance than others.

Resistance to F. moniliforme appears to be polygenic in inheritance (19). Sarca et al (45) from Romania reported the type of inheritance of resistance to F. moniliforme as additive in nature. Younis et al (64) studied F₁ and F₂ and BC₁ and concluded that the two major gene pairs control reaction to the pathogen with resistance completely dominant over susceptibility and he also reported the heritability estimate for resistance as 0.73. Younis (62) also studied the interaction of "pathogenicity genes" in F. moniliforme and "reaction genes" in Zea mays and reported that the host genotype changes towards increased resistance, the pathogen races differ in different locations with the result that resistant lines in one location are susceptible in another. He further reported a significant host parasite interaction and suggested selecting resistant lines to the prevalent races of the pathogen only.

There are conflicting reports on the anatomical nature of resistance.

Durrell (8, 9) indicated that resistant inbred lines contained more liquified tissue than did susceptible inbreds, especially in the lower nodes. Loesch et al (3) concluded that rind thickness was not affected by <u>Diplodia zeae</u> infection, but that the crushing strength of stalks from lodged and susceptible crosses was reduced. Black (3) concluded that standability of corn

was associated with a high number of vascular bundles in the fourth node above the soil. Even when the plants were partly rotted by <u>Gibberella zeae</u>, the number of vascular bundles appeared to give strength to the stalks. Pappelis (29) found that at the end of the growing season, the spongy pith tissue of lodged and stalk rot susceptible varieties was dead, but similar tissue in resistant, non-lodged varieties was alive. Foley (11) reported that cellulose occurred in corn plants whether susceptible and resistant to <u>F. moniliforme</u>.

Determination of Resistance

Various methods of testing to determine resistance of maize to stalk rot have been reported. Zwartz (66) reported that resistance should be determined by correlating the degree of stalk disintegration with average yield loss while Mesterhazy (31) concluded that selection for resistant roots would result in concomitant selection for resistant or tolerant stems.

Numerous investigators have reported that stalk rot is a disease of corn which develops as plants approach maturity (8, 15, 20, 25). Michaelson (32) obtained infection on all dates, but the earlier inoculations were much less effective than the later ones. He also showed that infection could occur long before pollination, and the fungi remain more or less dormant until the silking period. In addition, he suggested that after the plant has reached a certain stage of maturity, the resistance does not change. Sprague (52) and Jugenheimer (22) concluded that more stalk rot developed when inoculations were made at silking than at later dates. Sprague (52) stated that the greatest disease severity occurred when the plants are inoculated at the time of pollination. Hooker (18) made similar tests and

inoculated at the interval of 1 to 4 weeks after silking and obtained similar results. Results indicate that the exact timing of inoculation is not critical, hence one can inoculate a group of hybrid or inbred lines on the same date, even though they differ by as much as 10 days in flowering date. This assumes that the amount of diseased tissue is used as a basis of measuring resistance, and not premature dying of the stalks.

There is considerable evidence that rot severity depends on the internode in which the inoculation is made, but there is no general agreement on the exact internodes to inoculate in order to obtain the most efficient results, though most workers inoculate internodes below the ear. Hooker (18) inoculated four inbreds in the first, second, third, fourth, and fifth elongated internodes. Not all inbreds reacted alike to the inoculations at the different internodes. In the susceptible line, the diseased tissue was equally severe in the five inoculated internodes. In the resistant line, rot was least severe in internodes one and two, but increased progressively in the next three internodes, and in the fifth internode the rot was as severe as in the susceptible inbred. Cappellini (4) in New Jersey obtained progressively greater amount of rot with the distance up the stalk. Koehler (27) obtained the same amount of rot in all the three lower internodes. In another test he obtained more rot in the fourth internode than in the first. Christenson et al (5) reported that rot becomes progressively more severe in the internodes from the bottom to the top of the plant. Differences in susceptiblity among internodes may be due to differences in carbohydrate content. De Turk et al (6) found consistantly in single cross hybrids, less carbohydrates and lower total sugars in the lower part of the

stalk than in the middle part of normal plants.

Splitting the stalk and observing the rot is the most reliable method of determining both the amount of stalk rot and whether or not it resulted from natural infection or inoculation. Koehler (27) and others (21, 58, 4) measured both the extent of discolored pith and the number of rotten internodes. Hooker (18) used a disease scale from 1 to 6. Scores of 1 to 4 denoted rot confined to one internode; 5 indicated rot had spread into adjacent internodes; and 6, plants were killed.

MATERIALS AND METHODS

Field experiments were conducted during the 1979 and 1980 growing seasons at the KSU Agronomy Farm test site No. 1, the KSU Ashland Agronomy farm test site No. 2, Manhattan, Kansas and the CIMMYT research station at Tlaltizapan, Mexico, test site No. 3. The Kansas State University Agronomy farms are situated at about 39° 11' north latitude at about 310 M elevation with silty loam soils. Tlaltizapan, Mexico is located at 19°N latitude, about 950 M elevation and has a loamy clay soil type with an alkaline reaction. Sources and origin of the entries are presented in the Appendix.

Field Trials, 1979

In 1979, two experiments were conducted at the KSU Agronomy Farm, test site No. 2, under irrigated conditions.

Experiment No. 1

Over seventy genotypes including open pollinated varieties, inbred lines and hybrids were evaluated for tolerance to <u>F. moniliforme</u> with data collected from thirty-three entries. The experimental design was a randomized complete block design with two replications. Single row plots six meters long with seventy-five centimeters between rows and 25 cms between plants were used. Four plants were inoculated in each row at the 50% silking stage. Inoculations were performed at different dates because of the differences in silking time among genetic sources.

Experiment No.2

Twenty-five experimental hybrids were included in this study. Two row plots 3 meters long were planted in a randomized complete block design.

The plants were inoculated at the 14-leaf and 50% silking stages of growth.

 $[\]frac{1}{C}$ IMMYT - International Corn and Wheat Improvement Center

One row of each plot was used for 14-leaf stage inoculation and the second row was used for 50% silking stage inoculation. Data were, therefore, analyzed as a split-plot design with growth stage comprising the subplot.

Field Trials, 1980

In 1980, experiments were conducted at three test sites. Sufficient seeds of the 1979 experiments No. 1 and 2 sources were not available, therefore, different genotypes were included in the 1980 study.

Experiment No. 3

Twenty-eight maize sources were used in this study, including open pollinated cultivars and experimental hybrids and was planted at Agronomy Farms No. 1 and No. 2 under non-irrigated and irrigated conditions respectively. Serious drought and high temperature climatic conditions were encountered in 1980, therefore plants were exposed to severe stress conditions throughout the growing period. Inoculations were done at the 50% silking stage.

A randomized complete block design with 3 replications and single row plots 5 meters in length were used.

Experiment No. 4

This experiment was conducted at the CIMMYT station at Tlaltizapan in Mexico. Twenty-eight maize germplasms were included in this test comprising most of the composite corn varieties from Nepal. This study utilized a randomized complete block design with two replications. The individual plots were two rows 5 meters long. Plots were irrigated as necessary.

Inoculations were done at 50% silking stage.

Inoculation Method

Fusarium moniliforme was isolated from infected corn stalks. The isolation and purification of the fungus was done by L. E. Claflin in the Dept. of Plant Pathology. The fungus was cultured at room temperature on potato dextrose agar (POA) plates. Mycelia and spores were lifted from the medium and suspended in distilled water. Spore suspension concentration was ascertained with a Hemacytometer and adjusted to 2 x 10⁴ spores per milliliter with distilled water. The inoculum was prepared 30-60 minutes prior to inoculation. A B-D Cornwall leur-lok 10 ml syringe equipped with 16 gauge needle was used for inoculation. The tip of the needle was soldered shut and two holes were drilled in both sides of the needle near the tip. This partially eliminated clogging of the needle with stalk tissue. The syringe was fitted with a continuous pipetting device and 2 ml inoculum was injected into the second elongated internode above the brace roots. Four to 10 plants in each row of each experiment were inoculated.

At Tlaltizapan the inoculation was done by a CIMMYT technician and injections were made into the 4th internode from the base of each plant.

Evaluation

The stalk rot evaluation was done at physiological maturity. The inoculated plants were split lengthwise through the inoculated internode and the length of the infection was measured. Measurements for individual plants were averaged to obtain plot scores and analyzed statistically. Duncan's multiple range test was used for mean separation.

RESULTS AND DISCUSSION

Experiment No. 1

The reaction among entries to <u>Fusarium moniliforme</u> differed significantly as shown by analysis of variance, Table 1, in length of damage in the stalk due to infection. The mean infection lengths for each of the entries are shown in decreasing order in Table 2. Entry means ranged from 10.79 cms to 28.50 cms. Those entries designated by the same letter in Table 2 were not significantly different from each other at .05 probability level. No significant differences were found among entries 1 to 11, 2 to 15, 3 to 17, 4 to 25 and 8 to 33, Table 7. Nepal 103 had highest infection length of 28.50 cms while Fla 73-74:15 x 11 had the lowest infection length of 10.79 cms. The entries denoted by the letter "e" were classified as less susceptible and merit further testing.

Table 1. Analysis of Variance of Infection Lengths, Experiment No. 1.

Source of Variation	D.F.	SS	MSS	F Value
Entries	32	1475.0799	46.4087	2.87**
Replication	1	7.2800	7.2800	1.45
Error	32	517.9243		
TOTAL	65	2010,2842		

** = Statistically significant at the 1% level.

Although entries from serial numbers 8 to 33 do not differ significantly from each other, entries Nepal 104, Nepal 108, Fla $73-14:18 \times 11$, Gemiza 7421 and Fla $73-14:15 \times 11$ should be considered for further testing on the basis of

Table 2. Duncan's Multiple Range Test for Mean Infection Lengths, Experiment No. 1.

		
	Entries	Mean Infection Length 1
1.	Nepal 103	28.50a
2.	Nepal 203	26.71 ab
3.	Nepal 207	25.25 abc
4.	Nepal 304	23.63 abcd
5•	Nepal 202	23.27 abcd
6.	Nepal 206	22.71 abcd
7.	Nepal 105	22.71 abcd
8.	Nepal 209	20.25 abcde
9.	SJ1072-1x	19.88 abcde
10.	Rampur 7433	19.83 abcde
11.	Nepal 114	19.14 abcde
12.	FLA 73-74:80 x 71	19.08 bcde
13.	SJ 109-1 x	17.46 bcde
14.	Nepal 210	17.35 bcde
15.	FLA 73-74:70 x 61	17.31 bcde
16.	Nepal 301	17.12 cde
17.	Nepal 211	. 16.83 cde
18.	Nepal 303	15.89 de
19.	Amarillo BAJ10	14.87 de
20.	FLA 73-74:50 x 41	14.83 de
21.	Khumal (1)-7642	14.71 de
22.	Amarillo TYFD	14.46 de
23.	Nepal 107	14.44 de
24.	Pirsabak 7447	14 _• 25 de
25.	FLA 73-74:19 x 11	14 . 13 de
26.	Rampur 7434	12.69 e
27.	Nepal 305	12.50 e
28.	FLA 73-74:69 x 61	12.50 e
29.	Nepal 104	12.34 e
30.	Nepal 108	11.56 e
31.	FIA 73-74:18 x 11	11.23 e
32.	Gemiza 7 ⁴ 21	10.81 e
<u>33.</u>	FLA 73-74:15 x 11	10.79 e

 $[\]frac{1}{M}$ Means followed by the same letter are not significantly different at the 0.05 level.

relatively lower infection length among the groups. Generally open pollinated cultivars from Nepal were found relatively more susceptible than the US sources and CIMMYT varieties.

Experiment No. 2.

Entries in this experiment were inoculated at two different stages; 50% silking and 14-leaf. The analysis of variances showed a significant difference between the stages (Table 3). The mean infection lengths of both stages of inoculation is presented in Table 4. The incidence of stalk rot was relatively higher at 50% silking stage than 14-leaf stage. A high incidence of stalk rot development was also reported by Jugenheimer (22) and Sprague (52) at 50% silking stage. The interaction between stages and

Table 3. Analysis of Var	riance of	Infection Leng	ths, Experime	nt No. 2.
Source of Variation	D.F.	SS	MSS	F Value
Entries	24	1130.1828	47.0909	2.24**
Replication x Ent.	72	1510.5972	20.9805	
Stage	1	269.5842	269.5842	18.29**
Stage x Ent.	24	385.8408	16.0787	1.09
Error (b)	75	1105.6050	14.7414	

entries was not significant indicating that certain entries were relatively highly susceptible or less susceptible irrespective of the inoculation stages, which can be shown by the mean infection lengths presented in Table 4.

The mean infection lengths of both stages are presented in decreasing order in Table 4. Means ranged from 9.64 cms to 18.10 cms in length. The experimental hybrid No. 3 was found highly susceptible while No. 6 was the least. No significant differences were found among the entries from serial

^{** =} Statistically significant at the 1% level.

Table 4. Mean infection-lengths of damage resulting from inoculation at two stages of growth, Experiment No. 2.

Ent	ries				14 Leaf Stage	50% Silking Stage	Mean 1
1.	Experimental	hybrid	No.	3	15.50	20.70	18.10a
2.	Mo 17 x B68				13.80	22.25	18.03ab
3.	Experimental	hybrid	No.	16	13.87	20.75	17.31abc
4.	VA 26 B73				15.55	17.13	16.34abcd
5•	Experimental	hybrid	No.	22	12.47	18.35	15.41abcde
6.	Experimental	hybrid	No.	21	14.38	15.70	15.04abcdef
7.	Experimental	hybrid	No.	8	13.72	15.10	14.4labcdef
8.	Experimental	hybrid	No.	9	12.27	16.43	14.35abcdef
9.	Experimental	hybrid	No.	15	10.75	16.70	13.73abcdef
10.	Experimental	hybrid	No.	19	16.00	10.48	13.24abcdef
11.	Experimental	hybrid	No.	2	11.33	14.00	12.66bcdef
12.	Experimental	hybrid	No.	13	12.05	13.23	12.64bcdef
13.	Experimental	hybrid	No.	12	10.97	13.75	12.36cdef
14.	Experimental	hybrid	No.	7	12.65	12.00	12.33cdef
15.	Experimental	hybrid	No.	10	11.65	12.88	12.26cdef
16.	Experimental	hybrid	No.	5	10.70	13.30	12.00cdef
17.	Experimental	hybrid	No.	16	9.70	14.28	11.99cdef
18.	Experimental	hybrid	No.	4	11.27	12.55	11.91cdef
19.	Experimental	hybrid	No.	18	11.28	12.48	11.88cdef
20.	Experimental	hybrid	No.	1	10.90	12.45	11.68def
21.	Experimental	hybrid	No.	23	11.08	10.75	10.91def
22.	Experimental	hybrid	No.	17	9.85	10.68	10,26ef
23.	Experimental	hybrid	No.	20	9.45	11.08	10.26ef
24.	Experimental	hybrid	No.	11	9.28	11.08	10.18ef
25•	Experimental	hybrid	No.	6	9.40	9.88	9.64f

 $[\]frac{1}{M}$ Means followed by the same letter are not significantly different at the 0.05 level.

numbers 1 to 10, 2 to 12, 3 to 19, 4 to 21, 5 to 24 and 6 to 25. However entries denoted by letter "f" in Table 4 were classified as less susceptible genotypes and should be considered for further testing. Though a nonsignificant difference was found among entries from serial number 6 to 25, only the entries having the relatively low incidence of stalk rot should be considered relatively a less susceptible one. Thus experimental hybrids No. 17, No. 20, No. 11 and No. 6 can be rated as relatively resistant than other entries among the groups. The check hybrids (Mo 17 x B68) and (Va 26 x B 72) were found relatively more susceptible than other sources tested.

Experiment No. 3.

The incidence of stalk rot was relatively higher under irrigated conditions as compared to the non-irrigated condition at the Agronomy Farm, test site No. 1. This may have been attributable to higher soil moisture and higher humidity in the corn field coupled with high temperatures in the post inoculation period. Twenty-eight entries were grown at both locations but data from only twenty entries were used for the combined analysis. The analysis of variance of combined data is shown in Table 5.

Table 5. Combined Analysis of Variance of Infection Lengths, Experiment No. 3.

Source of Variation	D.F.	SS	MSS	F Value
Location	1	344.5257	344.5257	41.82**
Entries	19	978.7936	51.5155	2.43**
Rep (Loc)	1_	32.9504	8.2376	
Entries x Location	19	363.1709	19.1143	0.90
Error (b)	76	1608.4310	21.1636	

^{** =} Statistically significant at the 1% level.

Significant differences in infection lengths were obtained between locations. The entry x location interaction was not significant, which can be seen from means presented in Table 6. The combined means of both locations are presented in decreasing order in Table 7. Means ranged from 10.91 cms to 20.92 cms. O's Gold SX 5500 A was highly susceptible while (OH7B x Hy) x 1522 was the least. No significant difference was found among entries 1 through 7, 2 to 14, 4 to 15 and 5 to 20, Table 7. Thus the entries denoted by "d" were classified at least susceptible and probably should be considered for further testing. Entries from serial number 5-20 do not differ significantly, but entries (K55 x H28) x 1505, (k731 x OH7B) x 1518, (K41 x K731) x 1524, (SD10 x ZAP) x 1505 and (OH7B x Hy) x 1522 were considered less susceptible on the basis of low incidence of stalk rot found in them, Table 7.

The data of individual locations were also analyzed separately. The analysis of variance for the irrigated test is presented in Table 8 and of non-irrigated in Table 9. Entries were found highly significant at both locations. The mean infection lengths under irrigated condition ranged from 11.66 cms to 26.97 cms; P1270093 being the most highly susceptible and the P1270071 the lowest, Table 10. Under non-irrigated condition the mean infection length ranged from 8.67 cms to 21.23 cms; Asgrow Rx 901 was highly susceptible and (SD10 x Zap) x 1505 the least, Table 11. Experiment No. 4.

Genotypes were found significantly different in infection length at 10% level of significance, Table 12. The mean infection lengths of twenty-eight sources are presented in Table 13 in decreasing order and ranged from 7.5 cms to 15.13 cms. Those genotypes denoted by the same letter do not differ significantly at 10% level of significance. Those falling in the group

Table 6. Mean Infection Lengths of Entries at Two Different Locations in centimeter, Experiment No. 3.

	Entries	Irrigated	Non-irrigated
1.	O's Gold sx 5500A	22.61	19.24
2.	PAG sx 333	24.97	13.73
3.	Asgro RX 901	17.04	21.23
4.	Acco UC 8951	22.10	15.62
5.	(SD10 x 2 AP) x 1527	18.00	14.30
6.	Northrup King x Pa-74	16.34	14.43
7.	Pioneer Brand 3183	18.37	11.97
8.	PI 270076	15.17	14.80
9.	PI 270082	14.13	15.18
10.	BoJac 923	14.02	14.46
11.	Cargil 967	16.05	11.80
12.	Prairie V818	16.46	10.61
13.	Ring Around-RA 1502	15.23	11.60
14.	(K64A x Kl2) x 1516	17.57	9.23
15.	(H28 x K64) x 1511	12.07	13.23
16.	(K55 x H28) x 1505	13.90	10.50
17.	(K731 x OH7B) x 1518	14.20	9.40
18.	(K41 x K731)x 1524	13.53	9.30
19.	(SD10 x ZAP) x 1505	13.40	8.67
20.	(ОН7В х Ну) х 1522	11.87	9.96

Table 7. Duncan's Multiple Range Test for Mean Infection Lergths Combined, Experiment No. 3.1/

	Entries	Mean Infection Length 2/
1.	O's Gold SX 5500A	20.92 a ³ /
2.	PAG SX 333	19.35 ab
3.	Asgro R x 901	19.14 ab
4.	Acco UC 8951	18.86 abc
5•	(SD 10 x 2 AP) x 1527	16.15 abcd
6.	Northrup King x Px-74	15.39 abcd
7.	Pioneer Brand 3183	15.17 abcd
8.	PI 270076	14.98 bcd
9.	PI 270082	14.55 bcd
10.	BoJac 923	14.24 bcd
11.	Cargil 967	13.93 bcd
12.	Prairie V818	13.53 bcd
13.	Ring Around-RA 1502	13.42 bcd
14.	(K64A x K12) x 1516	13.40 bcd
15.	(H28 x K64) x 1511	12.65 cd
16.	(K55 x H28) x 1505	12.20 d
17.	(K731 x OH7B) x 1518	11.80 d
18.	(K41 x K731) x 1524	11.42 d
19.	(SD10 x ZAP) x 1505	11.03 d
20.	(ОН7В х Ну) х 1522	10.91 d

 $[\]frac{1}{2}$ Twenty entries which were common at both locations are included in the combined analysis.

 $[\]frac{2}{}$ Mean of two locations.

 $[\]frac{3}{}$ Means followed by the same letter are not significantly different at the 0.05 level.

Table 8.	Analysis	of	Variance	of	Infection	Lengths,	Irrigated,	Experiment	No.	3.
----------	----------	----	----------	----	-----------	----------	------------	------------	-----	----

Source of Variation	D.F.	SS	MSS	F Value
Replication	2	14.5995	7.2997	0.33
Entries	27	1197.2837	44.3438	1.98**
Error	54	1208.1861	22.3738	
TOTAL	83			

** = Statistically significant at the 1% level.

Table 9. Analysis of Variance of Infection Lengths, Non-irrigated, Experiment No. 3.

Source of Variation	D.F.	SS	MSS	F Value
Replication	2	22.5397	11.2698	0.84
Entries	19	639.1901	33.6416	2.49**
Error	38	512.66796	13.4913	
TOTAL	59	1174.3978		A A A A A A A A A A A A A A A A A A A

** = Statistically significant at the 1% level.

designated by the letter "f" were placed in the less susceptible category and require further testing. Although entries 9 through 28 do not differ significantly, only the entries Hetauda composite, Amarillo BAJIO, Suwan S.9, (VPI x SU) x Mal composite, Pirsaba, 7442 and Khumal (1) 7633 were considered relatively less susceptible on the basis of low incidence of stalk rot.

Table 10. Duncan's Multiple Range Test for Mean Infection Lengths, Experiment No. 3, Irrigated.

1	Entries	Mean Infection Length $\frac{1}{}$ in Centimeter
1.	PI 270093	26.97 a
2.	PAG SX 333	24.97 ab
3.	O's Gold sx 5500A	22.61 abc
4.	Acco UC 8951	22.10 abc
5.	Pioneer Brand 3183	18.37 abcd
6.	PI 270085	18.33 abcd
7.	PI 270075	18.10 abcd
8.	(SD10 x AP) x 1527	18 00 abcd
9•	PI 270077	17.57 bcd
10.	(K64A x K12) x 1516	17.57 bcd
11.	Asgro Rx 901	17.04 bcd
12.	Prairie V818	16.46 bcd
13.	Northrup King x Px-74	16.34 bcd
14.	Cargil 967	16.05 bcd
15.	к55 х н28	15.67 bcd
16.	Ring Around RA 1502	15.23 cd
17.	PI 270076	15.17 cd
18.	PI 270096	15.16 cd
19.	(K731 x OH7B) x 1518	14.20 cd
20.	PI 270082	14.15 cd
21.	BoJac 923	14.02 cd
22.	(K55 x H28 x 1505	13.90 cd
23.	(K41 x K737) x 1524	13.53 cd
24.	(SD10 x ZAP) x 1505	13.40 cd
25.	(H28 x K64) x 1505	12.07 d
26.	(ОН7В х Ну) х 1522	11.87 d
27.	(K55 x H28) x 1511	11.80 d
28.	PI 270071	11.66 d

 $[\]frac{1}{2}$ Means followed by the same letter are not significantly different at the 0.05 level.

Table 11. Duncan's Multiple Range Test for Mean Infection Lengths, Experiment No. 3, Non-irrigated.

Entries	Mean Infection Length in Centimeter
Asgro R x 901	21.23 a
O's Gold sx 5500A	19.24 ab
Acco Uc 8951	15.62 abc
PI 270082	15.18 abc
PI 270076	14.73 abc
BoJac 923	14.46 abc
Northrup King Px-74	14.43 abc
(SD10 x 2 AP) x 1527	14.30 bc
PAG sx 333	13.73 bc
(H28 x K64) x 1511	13.23 c
Pioneer Brand 3183	11.96 c
Cargil 967	11.80 c
Ring Around RA 1502	11.60 c
Prairie V818	11.61 c
(K55 x H28) x 1505	10.50 c
(ОН7В х Ну) х 1522	9.96 c
(K731 x OH7B) x 1518	9.40 c .
(K41 x K731) x 1524	9.30 c
(K64A x K12) x 1516	9•23 c
(SD10 x ZAP) x 1505	8.67 c
	Asgro R x 901 O's Gold sx 5500A Acco Uc 8951 PI 270082 PI 270076 BoJac 923 Northrup King Px-74 (SD10 x 2 AP) x 1527

 $[\]frac{1}{}$ Means followed by the same letter are not significantly different at the 0.05 level.

Source of Variation	D.F.	SS	MSS	F Value
Replication	1	1.2421	1.2421	0.43
Entries	27	141.2111	5.2300	1.79***
Error	27	78.8386	2.9199	

221.2918

Table 12. Analysis of Variance of Infection Lengths, Tlaltizapan, Mexico.

TOTAL

It is suggested that a procedure to upgrade resistance among those materials rated least susceptible would be to grow a large number of rows, inoculate at 50% silking stage, and sib pollinate among resistant plants. Considerable improvement in the level of resistance might be expected in one or two cycles. This procedure would also permit retention of the original genotypes without much dilution.

55

A general occurrence of corn borer larvae tunnels were found in the inoculated internodes. It is suspected that holes punched during inoculation by the needle might have provided easy entry of the larvae to the stalk. Most of the early maturing inbred lines and some of the PI lines were found totally rotten and as a result, no infection lengths were recorded.

In general the inoculated plants were not severely damaged by natural infection of any other corn diseases. Plate I and II of the Appendix illustrate the corn stalk reactions to artificial inoculation under field conditions.

^{*** =} Statistically significant at the 10% level.

Table 13. Duncan's Multiple Range Test for Mean Infection Lengths, Experiment No. 4.

	Entries	Mean Infection Length $\frac{1}{2}$ in Centimeter
1.	Kakani Yellow (Local)	15.125 a
2.	Rampur Mix	13.040 ab
3.	Kathmandu Yellow (Local)	12.725 abc
4.	Obregon 7443	11.855 bcd
5.	Khumal Yellow	11.725 bcd
6.	Kakani Yellow	11.725 bcd
7.	Ganesh-2	11.375 bcde
8.	PI 175334	11.350 bcde
9.	PI 172333	10.750 bcdef
10.	UNCAC	10.675 bcdef
11.	Rampur Yellow	10.660 bcdef
12.	Sarlahi Seto	10.650 bcdef
13.	Rampur Composite	10.550 bcdef
14.	Mix Composite	10.550 bcdef
15.	Ganesh-2	10.475 bcdef
16.	Suwan-1 S10	10.225 bcdef
17.	CIMMYT Mix	10.100 bcdef
18.	Mankamna	9.950 bcdef
19.	Pirsabak 7447	9.725 bcdef
20.	Janaki	9.450 cdef
21.	Pozarica 7525	9.425 cdef
22.	Amarillo Pakisatan	9.400 cdef
23.	Hetauda Composite	9.050 def
24.	Amarillo BAJ10	8.775 def
25.	Suwan S. 9.	8.750 def
26.	(VPI x SU) x Mat Comp. (11)	8.425 ef
27.	Pirsabak 7442	8.174 ef
28.	Khumal (1) 7633	7.500 f

^{1/}Means followed by the same letter are not significantly different at the 0.10 level.

SUMMARY AND CONCLUSIONS

- 1. Several corn germplasm sources including open pollinated varieties, composites, hybrids and inbred lines were tested for resistance to stalk rot <u>Fusarium moniliforme</u>, at Kansas State University, Manhattan, Kansas and Tlaltizapan, (Mexico).
- 2. Experiments were conducted in a randomized complete block design replicated two and three times depending on the availability of seed. Four to ten plants in each row were inoculated at 2nd internodes from the brace roots at the rate of 20,000 spores per millilitre.
- 3. Evaluation was done by splitting the stalk and measuring length of infection in centimeters.
- 4. On the basis of one year's data twenty of the entries were classified as relatively resistant (less susceptible) to \underline{F} . $\underline{moniliforme}$ under Manhattan, Kansas and Tlaltizapan, Mexico conditions.
- 5. Stalk rot development was found to be higher under irrigated conditions than non-irrigated.
- 6. Comparatively higher infection of stalk tissue was observed when plants were inoculated at 50% silking stage than 14-leaf stage.
- 7. Further testing of the entries classified as resistant (less susceptible) is needed to confirm their level of resistance, to study the mode of inheritance, and to increase levels of resistance through breeding.
- 8. Among various Nepalese germplasms, Nepal 104, Nepal 108 and Hetauda composite were found relatively resistant to Fusarium stalk rot.

LITERATURE CITED

- 1. Andrew, R. H. 1954. Breeding for stalk rot resistance in Maize. Euphatica 3: 43-48.
- 2. Barnes, J. M. 1960. Investigation on stalk rot of corn caused by <u>Gibberella zeae.</u> Part I. A comparison of two methods of evaluating the severity of stalk rot in several corn varieties. Part II. Aspects of the biochemical nature of stalk rot resistance. Ph.D. thesis, Cornell University.
- 3. Blaak, G. 1957. Histological studies of seedlings and older plants of several varieties of corn infected by <u>Gibberella</u>. M.S. thesis, Cornell University.
- 4. Cappellini, R. A. 1959. A comparison of techniques and sites of inoculation in field corn artificially inoculated with <u>Gibberella zeae</u> (schw.)

 Petch. Plant. Dis. Reptr. 43: 177-179.
- 5. Christensen, J. J. and R. D. Wilcoxson 1966. Stalk rot of corn. Monograph No. 3, published for The American Phytopathological Society. Heffernan Press, Worchester, Mass. 59 p.
- 6. Deturk, E. E., E. B. Earley, and J. R. Holbert 1937. Resistance of Corn Hybrids related to carbohydrates. Illinois Agr. Exp. Sta. Ann. Rept. 49: 43-45.
- 7. Devay, J. E., R. P. Covey, and D. B. Linden 1957. Methods of testing for disease resistance in the corn disease nurseries at St. Paul and comparisons of 110 lines of corn for resistance to diseases important in the North Central region. Plant Dis. Reptr. 41: 699-702.
- 8. Durrell, L. W. 1923. Dry rot of corn. Iowa Agr. Exp. Sta. Res. Bull. 84.

- 9. Durrell, L. W. 1925. A preliminary study of fungus action as the cause of down corn. Phytopathology 15: 146-154.
- 10. Foley, D. C. 1959. Systemic infection of corn by <u>Fusarium moniliforme</u>. Phytopathology 49: 538 (Abstr.).
- 11. Foley, D. C. 1959. The presence of cellulose in corn stalks infected with <u>Fusarium moniliforme</u>. Phytopathology 49: 538 (Abstr.).
- 12. Foley D. C. 1962. Systemic infection of corn by <u>Fusarium moniliforme</u>. Phytopathology 52: 870-872.
- 13. Henry A. W. 1923. The pathogenicity of <u>Fusarium moniliforme</u> shel. on cereals. (Abstract) Phytopathology 23 (1) 52.
- 14. Hoffer, G. N., and R. H. Carr 1923. Accumulation of aluminum and iron compounds in corn plants and its probable relation to root rots. J. Agr. Res. 23: 801-823.
- 15. Holbert, J. R., P. E. Hoppe and A. L. Smith 1935. Some factors affecting infection with and spread of <u>Diplodia zeae</u> in the host tissue. Phytopatholgy 25: 1113-1114.
- 16. Hooker, A. L. 1956. Association of resistance to several seedling, root, stalk, and ear diseases in corn. Phytopathology 46: 379-384.
- 17. Hooker, A. L. 1956. Correlation of resistance to eight <u>pythium</u> species in seedling corn. Phytopathology 47: 196-199.
- 18. Hooker, A. L. 1957. Factors affecting the spread of <u>Diplodia zeae</u> in inoculated corn stalks. Phytopathology 47: 196-199.
- 19. Hooker, A. L. 1978. Inheritance of disease resistance in corn (Abstract).

 3rd International Congress of Plant Pathology, Munchen 16;23.
- 20. Johann, H., and A. D. Dickson 1945. A soluable substance in cornstalks that retards growth of <u>Diplodia zeae</u> in culture. J. Agr. Res. 71: 89-110.

- 21. Jugenheimer, R. W., and A. A. Bryan 1938. Developing inbred lines of corn resistant to stalk and ear rots. Iowa Agr. Exp. Sta. Ann. Rept. 1937-1938: 33-36.
- 22. Jugenheimer, R. W. 1940. Resistance to <u>Diplodia</u> infection in inbred lines and hybrids of maize. Ph.D. Thesis, Iowa State University.
- 23. Jugenheimer, R. W. 1958. Hybrid maize breeding and seed production. F.A.O. Agric. Development Paper No. 62.
- 24. Kirmelasvili, N. S., and M. I. Dolidze 1973. Materials for study of the toxicity of <u>Fusarium moniliforme</u>, and discovery of resistant varieties of maize. Referativny Zhurnal 2: 55 364.
- 25. Koehler, B., and J. R. Holbert 1930. Corn diseases in Illinois, their extent, nature and control. Illinois Agr. Exp. Sta. Bull. 354.
- 26. Koehler, B. 1950. Corn stalk and ear rot studies. Improved techniques in hybrid seed corn production. Rept. 5th Hybrid Corn Ind. Res. Conf. Amer. Seed Trade Assn. Pub. 5: 33-46.
- 27. Koehler, B. 1960. Corn stalk rots in Illinois. Illinois Agr. Exp. Sta. Bull. 658.
- 28. Kucharek, T. A., and T. Kommedahl 1966. Kernel infection and corn stalk rot caused by Fusarium moniliforme, Phytopathology 56: 983-984.
- 29. Koesch, P. J., Jr., O. H. Calvert, and M. S. Zuber 1962. Interrelations of <u>Diplodia</u> stalk rot and two morphological traits associated with lodging of corn. Crop Sci. 2: 469-472.
- 30. McNew, G. L. 1937. Crown infection of corn by <u>Diplodia zeae</u>. Iowa Agr. Exp. Sta. Res. Bull. 216.
- 31. Mesterhazy, A. 1979. Stalk splitting as a method for evaluating stalk rot of corn. Plant disease reporter 63(3) 227-231.

- 32. Michaelson, M. E. 1957. Factors affecting development of stalk rot of corn caused by <u>Diplodia zeae</u> and <u>Zibberella zeae</u>. Phytopathology 47: 499-503.
- 33. Moore, V. A. 1896. Corn stalk disease and rabies in cattle. U.S. Dept. Agr. Bur. Anim. Ind. Bull. 10.
- 34. Otto H. J. 1956. The influence of nitrogen and potassium fertilization on the incidence of stalk rot of corn in New York. Ph.D. thesis, Cornell University.
- 35. Pammel, L. H. 1914. Serious root and stalk diseases of corn. Iowa Agr. 15: 156-158.
- 36. Pammel, L. H., C. M. King, and J. L. Seal, 1916. Studies on a <u>Fusarium</u> disease of corn and sorghum. Iowa Agr. Exp. Sta. Bull. 33.
- 37. Pappelis, A. J. 1957. Nature of resistance to <u>Diplodia</u> stalk rot of corn. Ph.D. thesis, Iowa State College.
- 38. Pappelis, A. J., and F. G. Smith 1960. Nature of resistance to <u>Diplodia</u> stalk rot of corn. Phytopathology 50: 650 (Abstr.)
- 39. Parker, D. T., and W. C. Burrows 1959. Root and stalk rot in corn as affected by fertilizer and tillage treatment. Agron. J. 51: 414-417.
- 40. Peters, A. T. 1904. A fungus disease in corn. Nebraska Agr. Exp. Sta. 17th Ann. Rept. p. 13-22.
- 41. Reece, O. E. 1949. Inheritance of reaction of root and stalk rot in maize. Ph.D. thesis, Univ. Minnesota.
- 42. Russell, W. A. 1961. A comparison of five types of testers in evaluating the relationship of stalk rot resistance in corn inbredliness and stalk strength of the lines in hybrid combinations. Crop. Sci. 1: 393-397.
- 43. Sabet, K. A., A. S. Samra, M. K. Hingorani, M. G. Abdel Rahim, H. A.

- El-Shafey, I. M. Mansour, Ikbal Khalil, F.A.M. Fadl, and Nadiadawood 1960.

 Annual Report of the project entitled "Stalk and root rots of maize in the United Arab Republic" for the period July 1961 to June 1962.
- 44. Sansom, T. K. 1940. Breeding <u>Diplodia</u> resistant varieties of maize. Rhodesia Agr. J. 37: 442-444.
- 45. Sarca, T., O'Cosmin, E. Muresan, N. Bica, C. Negut, V. Vlinict, and C. Tusa 1978. Studies on the resistance of maize to <u>Helminthosporium</u> and Fusarium. ICCPT Fundulea, Romania.
- 46. Semeniuk, G. 1941. Development of <u>Diplodia zeae</u> and <u>Gibberella</u>

 <u>saubinetii</u> in maize pith following stalk inoculations. Phytopathology

 31: 20 (Abstract).
- 47. Sheldon, J. L. 1904. A corn mold (<u>Fusarium moniliforme</u> n. sp.) Nebr. Agr. Exp. Sta. 175th Ann. Rept. p. 23-32.
- 48. Sidorov, F. F., V. I. Potlaichuk and G. G. Aiba 1962. Assessment of the collection of selfed lines for resistance to diplodiosis. Kukuruza 7:57 (Rev. Appl. Mycol. 41:654).
- 49. Sim, T. 1980. Summary of plant disease condition in Kansas, Kansas State Board of Agriculture, Division of Entomology, Kansas State University, Manhattan, Kansas.
- 50. Smith, A. L. P. E. Hoppe, and J. R. Holbert, 1938. Development of a differential inoculation technique for <u>Diplodia</u> stalk rot of corn. Phytopathology 28: 479-504.
- 51. Spencer, E. L., and G. L. McNew 1938. The influence of mineral nutrition on the reaction of sweet-corn seedlings to Phytomonas.org stewarti.

 Phytopathology 28: 213-223.

- 52. Sprague, G. F. 1954. Breeding for resistance to stalk rot. Amer. Seed Trade Assn. Pub. 9: 38-43.
- 53. Sumner, D. R. 1968. The effect of soil moisture on stalk rot.

 Phytopathology 58: 761-765.
- 54. Stover, W. G. Relation of soil temperature to the development of certain fungus seedling blight of corn. (unpublichsed paper).
- 55. Tayler, G. S. 1953. Stalk rot development in corn following the European corn borer. Iowa State Coll. J. Sci. 27: 265-266 (Abstract).
- 56. Thayer, P., and L. E. Williams 1960. Effect of nitrogen, phosphorous, and potassium concentrations on the development of <u>Gibberella</u> stalk and root rot of corn. Phytopathology 50: 212-214.
- 57. Valleau, W. D. 1920. Seed corn infection with <u>Fusarium moniliforme</u> and its relation to the root and stalk rots. Kentucky Agr. Exp. Sta. Bull. 226.
- 58. Wernham, C. C. 1949. Techniques for inoculating corn with disease producing organisms. Pennsylvania Agr. Exp. Sta. Prog. Rep. 5: 1-6.
- 59. White, D. G., R. G. Hoeft, and J. T. Touchton 1979. Effect of nitrogen and nitripyrin on stalk rot, stalk diameter and yield of corn. Phytopathology 68: 811-814.
- 60. Wood, L. S. 1951. Development of root and stalk rot in certain dent corn single-crosses. M. L. Thesis, Ohio State Univ.
- 61. Younis, S. E., M. K. Abo-El-Eahab, and G. S. Mallan 1969. Genetic studies of the resistance to <u>Fusarium</u> stalk rot in maize. Indian Journal of Genetics and Plant breeding. 29(3) 418-425.
- 62. Younis, S. E. 1970. Interaction of pathogenicity genes in Fusarium

- moniliforme and reaction genes in <u>zea mays</u>. Alexandria Journal of Agric. Science 18(1) 142.
- 63. Young, H.C., Jr. 1949. Resistance in corn to several pathogens causing seedling blights and stalk rots. Ph.D. thesis, Univ. Minnesota.
- 64. Younts, S. E., and R. B. Musgrave 1958. Chemical composition, nutrient absorption, and stalk rot incidence of corn as affected by chloride in potassium fertilizer. Agron. J. 50: 426-429.
- 65. Zuber, M. S., C. O. Grogan, M. E. Michaelson, C. W. Gehrke, and J. F. Monge. 1957. Studies of the interrelation of field stalk lodging, two stalk rotting fungi, and chemical composition of corn. Agron. J. 49: 328-331.
- 66. Zwatz, B. 1978. A new method for determining the resistance of maize to stalk rot. 3rd International Congress of Plant Pathology. Muchen, 16-23.

Acknowledgements

I want to express a heartful appreciation to Dr. Clyde E. Wassom, major professor, for his critical advice and kind support throughout the field study and in writing of this thesis. I am also indebted to Dr. Larry E. Claflin, Head, Plant Pathology Department for helping me in obtaining pathogen cultures and without whose sincere help this study could have not been completed. Special thanks goes to Dr. Malegi and Dr. Simon Manzo for helping me in preparing cultures for inoculation in the field.

Grateful thanks are also due to Department of Agriculture and Integrated Cereal project NEPAL for providing me with the scholarship for completion of this study. A sincere thanks goes to Dr. Wayne H. Freeman for his kind help and encouragement throughout my study.

I am also very much grateful to Mr. B. B. Khadika, Secretary, Ministry of Agriculture, the then Director General Mr. Shiva Bahadur Nepali, Mr. P. N. Rana, Director General, Department of Agriculture, Mr. Gopal Rajbhandari, Chief Corn Development Officer, and to Mr. A. M. Pradhanang, Deputy Director General, for their kind recommendation without whose support I may not have got this opportunity to get this degree.

To my parents, Mr. and Mrs. Suryanarayan Lal, the greatest of their kinds, I wish to extend deepest gratitude for their faith and loving support and for taking care of my family while I was in U.S.A.

I wish to extend my deepest appreciation and love to my wife Anita Lal, for allowing me to complete this study while she was taking care of all my beloved children back home. Deep appreciation also goes to my son Dipendra, my daughter, Anju, Manju and Bobby.

I owe a special debt of gratitude to the members of my committee, Dr. Clyde Wassom, Dr. Larry E. Claflin, Dr. R. Ellis, and to Dr. D. A. Whitney for their

encouragement and valuable suggestions.

I am also very much thankful to Dr. Johnson in helping me in calculation of the data and finally to all the members of International Agricultural Development Service, New York involved in my study program.

Last but not the least, I also want to thank Dr. Carlos De León and his group at Tlaltizapan (CIMMYT, Mexico), without whose support the Experiment No. 4 would have not been completed.

APPENDIX

Table 1. Pedigrees, origin and source of seed of entries in experiment No. 1.

ENTRY NO.	PEDIGREE	ORIGIN	SOURCE
1.	NEPAL 103	NEPAL	CIMMYT
2.	NEPAL 203	g I	It
3∙	NEPAL 207	11	11
4.	NEPAL 304	11	11
5•	NEPAL 202	11	11
6.	NEPAL 206	!!	***
7•	NEPAl 105	11	***
8.	NEPAL 209	"	II
9•	SJ1072-1 x	11	11
10.	RAMPUR 7433	u	11
11.	NEPAL 114	11	11
12.	FLA 73-74 : 80 x 71	KANSAS	KSU MAIZE PROJ.
13.	SJ 109-1	11	"
14.	NEPAL 210	NEPAL	CIMMYT
15.	Fla 73-74 : 70 x 61	KANSAS	KSU MAIZE PROJ.
16.	NEPAL 301	NEPAL	CIMMYT
17.	NEPAL 211	11	u
18.	NEPAL 303	"	u
19.	AMARILLO BAJIO	CIMMYT	n
20.	FIA 73-74 : 50 x 41	KANSAS	KSU MAIZE PROJ.
21.	KHUMAL (7642)	NEPAL	CIMMYT
22.	AMARILLO TYED	CIMMYT	11
23.	NEPAL 107	NEPAL	11
24.	PIRSABAK 7447	CIMMYT	II
25.	FIA 73-74 : 19 x 11	KANSAS	KSU MAIZE PROJ.
26.	RAMPUR 7434	NEPAL	CIMMYT ¹ /
27.	NEPAL 305	KANSAS	KSU MAIZE PROJ.
28.	FLA 73-74 : 69 x 61	11	11
29.	NEPAL 104	NEPAL	CIMMYT
30.	NEPAL 108	11	11
31.	FLA 73-74: 18 x 11	KANSAS	KSU MAIZE PROJ.
32.	FLA 73-74: 15 x 11	KANSAS	KSU MAIZE PROJ.

 $[\]underline{1}_{\text{International Maize}}$ and Wheat Improvement Centre, Mexico.

Table 2. Sources of seed of experimental hybrid in experiment No. 2.

ENTRY NO.	ENTRIES	SOURCE		
1.	Experimental hybrid	No. 1	7-9292 x 7-9338 Ear 1	
2.	Experimental hybrid	No. 2	7-9292 x 7-9338 Ear 2	
3•	Experimental hybrid	No. 3	7-9292 x 7-9338 Ear 3	
4.	Experimental hybrid	No. 4	7 - 9292 x 7 - 9338 Ear 4	
5•	Experimental hybrid	No. 5	$7-9289 \times 7-9290$ Bulk	
6.	Experimental hybrid	No. 6	$7-9274 \times 7-9247$ Bulk	
7•	Experimental hybrid	No. 7	7-9276 x 7-9247 Ear 1	
8.	Experimental hybrid	No. 8	7-9208 x 7-9234 Ear 1	
9•	Experimental hybrid	No. 9	7-9251 x 7-9257 Ear 1	
10.	Experimental hybrid	No. 10	7-9233 x 7-9236 Ear 1	
11.	Experimental hybrid	No. 11	7-9233 x 7-0236 Ear 2	
12.	Experimental hybrid	No. 12	7-9233 x 7-0236 Ear 4	
13.	Experimental hybrid	No. 13	7-9205 x 7-9207 Ear 2	
14.	Experimental hybrid	No. 14	7-9205 x 7-9208 Ear 1	
15.	Experimental hybrid	No. 15	7-9282 x 7-9274 Ear 3	
16.	Experimental hybrid	No. 16	7-9200 x 7-9203 Ear 1	
17.	Experimental hybrid	No. 17	7-9200 x 7-9203 Ear 2	
18.	Experimental hybrid	No. 18	7-9269 x 7-9285 Ear 1	
19.	Experimental hybrid	No. 19	7-9269 x 7-9285 Ear 2	
20.	Experimental hybrid	No. 20	7-9269 x 7-9285 Ear 3	
21.	Experimental hybrid	No. 21	7-9277 x 7-9280 Ear 1	
22.	Experimental hybrid	No. 22	7-9277 x 7-9280 Ear 2	
23.	Experimental hybrid	No. 23	7-9285 x 7-9339 Ear 3	
24.	VA 26 x B 73			
25.	MO 17 x B 68			

Table 3. Pedigree, origin and sources of seed of entries in experiment no. 3.

Entry No.	Pedigree	Origin
1.	PI 270093	West Pakistan
2.	Pag SX 333	USA
3.	O's Gold SX 5500A	11
4.	Acco UC 8951	n
5.	Pioneer Brand 3183	11
6.	PI 270085	West Pakistan
7.	PI 270075	ri .
8.	(SD10 x ZAP) x 1527	USA
9•	PI 270077	West Pakistan
10.	(K64A x K12) x 1516	Kansas and Mississi
11.	AC x 901	USA
12.	Prairie Valley V 818	USA
13.	Northrup-King PX-74	11.
14.	Cargill 967	"
15.	K55 x 428	LI
16.	Ring a Round RA 1502	rt
17.	PI 270076	West Pakistan
18.	PI 270096	If
19.	(K731 x OH7B) x 1518	Mississippi
20.	PI 270082	West Pakistan
21.	Bojac 923	USA
22.	(K55 x H28) x 1505	Mississippi
23.	(K41 x K737 x 1524	Mississippi
24.	(SD10 x 2AP) x 1505	n
25.	(H28 x K64) x 1505	11
26.	ОН7В x НY) x 1522	11
27.	(K55 x H28) x 1511	11
28.	PI 270071	West Pakistan

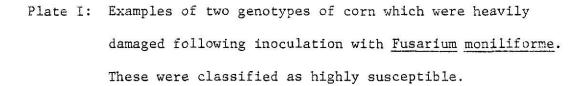
Table 4. Pedigree, origin and sources of seed of entries in experiment No. 4.

(Tlaltizapan Mexico, 1980)

Entry No.	Pedigree	Origin	Source
1.	Kakani Yellow (local)	Nepal	Nepal
2.	Rampur Mix	TT.	t f
3.	Kathmandu Local (yellow)	II	11
4.	Obregon 7443	"	- 11
5•	Khumal Yellow	11	11
6.	Kakani Yellow	tt.	**
7•	Ganesh-2 (1978)	rr .	11
8.	PI 175334	Australia	Pi Amex Iowa
9•	PI 172333	H	11
10.	Uncac	Nepal	Nepal
11.	Rampur Yellow	11	11
12.	Sarlahi Seto	<u>u</u>	**
13.	Rampur Composite	TI .	"
14.	Met. Composite	п	11
15.	Ganesh-2	11	н
16.	Suwan-1 SlO	11	Thailand
17.	Cimmyt Mix	ш	Nepal
18.	Mankamna	TT.	"
19.	Pirsabak 7447	Nepal	Cimmyt
20.	Janaki	ш	Nepal
21.	Pozarica 7525	Nepal	Cimmyt
22.	Amarillo Pakistan	11	11
23.	Hetauda Composite	11	Nepal
24.	Amarillo Bajio	Nepal	Nepal
25.	Suwan S9	Nepal	Thailand
26.	(VPI x SU) x Mal Comp. (11)	11	Cimmyt
27.	Pirsabak 7442	11	ū
28.	Khumal (1) 7633	.11	11

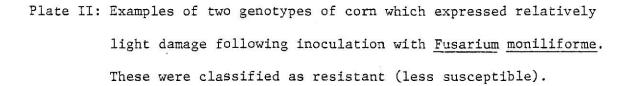
THIS BOOK CONTAINS NUMEROUS PAGES THAT WERE BOUND WITHOUT PAGE NUMBERS.

THIS IS AS
RECEIVED FROM
CUSTOMER.













EVALUATION OF CORN GERMPLASM TO FUSARIUM MONILIFORME STALK ROT

by

KAUSHAL KISHORE LAL

B.Sc. Agri (Hons), University of Udaipur, India, 1970

AN ABSTRACT OF A MASTER'S THESIS

submitted in partial fulfillment of the

requirements of the degree

MASTER OF SCIENCE

Department of Agronomy

KANSAS STATE UNIVERSITY Manhattan, Kansas

Several corn germplasm sources including open pollinated varieties, composites, hybrids and inbred lines were tested for resistance to stalk rot, <u>Fusarium moniliforme</u>, at Kansas State University, Manhattan, Kansas and Tlaltizapan (Mexico). Experiments were conducted in a randomized complete block design replicated two and three times depending on the availability of seed. Four to ten plants in each row were inoculated at 2nd internode from the brace roots at the rate of 20,000 spores per millilitre. Evaluation was done by splitting the stalk and measuring lengths of infection in centimeters. On the basis of one year's data, twenty of the entries were classified as relatively resistant (less susceptible) to <u>Fusarium moniliforme</u> under Manhattan, Kansas and Tlaltizapan, Mexico conditions.

Plants grown under irrigated conditions had a higher stalk rot development than those grown on non-irrigated land. The mean infection lengths were found to be higher in the plants innoculated at the 50% silking stage than at the 14-leaf stage. Further testing of the entries classified as resistant (less susceptible) is needed to confirm their level of resistance, to study the mode of inheritance, and to attempt to increase levels of resistance.