STUDIES ON TURCICUM BLIGHT OF SELECTED EARLY AND EXTRA EARLY MAIZE (*Zea mays* L.) LINES INDUCED BY *Helminthosporium turcicum* Pass IN ZARIA

BY

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B.Sc. Microbiology (UNIBEN) 1990

A THESIS SUBMITTED TO THE POSTGRADUATE SCHOOL, AHMADU BELLO UNIVERSITY, ZARIA
IN PARTIAL FULFILMENT OF THE REQUIREMENT FOR THE DEGREE OF MASTER OF SCIENCE IN CROP PROTECTION

DEPARTMENT OF CROP PROTECTION
FACULTY OF AGRICULTURE
AHMADU BELLO UNIVERSITY
ZARIA, NIGERIA

OCTOBER 2003

DECLARATION

I hereby declare that this thesis has been written by me and that it is a record of my own research work. It has not been presented before in any application for higher degree.

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Major Supervisor

DEDICATION

This thesis is sincerely dedicated to my wife, Nana Fatimah in recognition of the sacrifice she made through out the course of this work.

CERTIFICATION

This thesis titled “STUDIES ON TURCICUM BLIGHT OF MAIZE (*Zea mays* L.) INDUCED BY *Helminthosporium turcicum* Pass” by Omo-Eboh Mamudu Eragbai meets the regulations governing the award of the degree of Master of Science, Crop Protection, of Ahmadu Bello University, Zaria and is approved for its contribution to knowledge and literary presentation.

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ACKNOWLEDGEMENTS

All praise is due to Allah the Lord and sustainer of the worlds. I wish to express my deepest gratitude to my Major supervisor, Dr. A.A. Adeoti, for his active supervision, guidance, support and tolerance throughout the period of this work. The same goes to my other two supervisors, Dr. P.S. Marley and Dr. A.D. Akpa for their suggestions and constructive criticisms especially during the thesis write-up.

I wish to extend my appreciation to the Department of Crop Protection, Institute for Agricultural Research, Ahmadu Bello University, Zaria for the opportunity and assistance given to me to carry out this research. I am particularly grateful to Dr. I. Onu, Dr. A.D. Akpa, the Head of Department and Postgraduate Co-ordinator 1996/97 session respectively; Dr. M.C. Dike and Dr. Alabi, the Head of Department and post graduate coordinator 2001/2002 session respectively.

I would like to extend my gratitude to Mr. Abdullahi, Mr. Auta, Mallam Taneemu and all other staff of the Plant Pathology Unit, Crop Protection Department, for their technical assistance. I am indebted to my senior brother, Omo-Eboh Amedu, and a host of others for their invaluable moral and financial support, love and encouragement during the cause of my study. I am particularly indebted to Mr. and Mrs. M.S. Ahmed, Mr. Abdukareem Agunu, Dr. Yakubu Itai, Mr. Gbogi Umoru, Mr. Dominic Isunnoya, Mr. Wesley Emmanuel, Mr. Tunde Jatto, Mr. Tunde Adepoju, Mr. Yahaya, Dr. Ogunwale, Mr. Martins Royal Nwanosike, Mr. Oyakhire Otoikhila, just to mention a few.

My sincere appreciation also goes to my friend Lee Obomeghie for his assistance in the production of the soft and final copies of this thesis. May Allah shower his infinite blessings and mercy on each and every one of you. Amen.

ABSTRACT

During the 1997 and 1998 cropping seasons, studies on turcicum blight of maize (*Zea mays* L.) induced by *Helminthosporium turcicum* Pass were carried out in Zaria using selected early and extra-early maize lines. The pathogenicity test carried out in the glass house confirmed *Helminthosporium turcicum* as the causative agent of the disease.

Field and glasshouse screening of fifteen early and eleven extra-early maize lines for their resistance to turcicum blight was conducted in 1997 and 1998. Seven early lines (AK 9331-DMSR, $SynE_2$, ACR 92TZE Comp. 5-W, Dorke-SR, Farakoba 90 pool 16DT (HD), NAES pool 16DT, DT-E-Y SR) and five extra-early lines, TZE-W-$SRBC_5$, CSP-$SRBC_5$, KEB, TZEF-Y-SR, 95.TZEE-W1 were moderately resistant while CSP x local Raytiri, CSP-SR x TZEE-Y among the extra-early lines and AB 11, Kamboinse 88 Pool 16DT RE among the early lines were moderately susceptible.

Sowing date trials at 14-day intervals were conducted in 1997 and 1998 using early line AB 11 and extra-early line CSP-SR x TZEE-Y. The sowing dates were June 26, July 10, July 24 and August 7 in 1997; June 29, July 13, July 27 and August 10 in 1998. In both lines, disease incidences were recorded at 30, 45 and 60 days after sowing (DAS) while severity were recorded at 50, 60 and 70 (DAS). The maximum disease severity (12.71 % and 26. 04%) was recorded on early maize lines planted on July 24 and 27 (i.e. third sowing date) in 1997 and 1998 respectively. The highest grain yields in both years (1997 and 1998) (2592.6kg/ha and 1592.6kg/ha) respectively were recorded on maize planted on June 26 and 29 (i.e., first sowing date) that were least affected with turcicum blight. For the extra – early lines, the maximum disease severity (9.68% and 38.34%) were recorded on maize planted in July 24 and 27 in 1997 and 1998 respectively. Least severity (7.61% and 17.08%) was recorded on maize planted

on June 26 and 29 in both years with subsequent significantly (p= 0.05) highest yield (2555.6 kg/ha) in 1997.

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CHAPTER ONE

1.0 INTRODUCTION

Maize, Zea mays L., is an annual plant of the family Gramineae like all the other cereal crops. Cultivated maize is a fully domesticated plant (Dowswell *et al*., 1996), and very high in grain yielding capacity probably exceeding all other cereal crops.

Maize is susceptible to numerous diseases and pests all over the world. Variation of these diseases and pests as noted by Effron (1985), is a major cause of yield instability with yield losses reaching 100% under epidemic conditions. Ullstrup (1977), had noted that parasitic diseases which may be caused by bacteria, fungi, viruses and mycoplasma provide severe constraints on tropical maize production frequently causing 30-40% yield loss.

According to Effron (1985), maize diseases are categorized as follows:

i. Local-spot foliar diseases;
ii. Systemic foliar disease; and
iii. Stalk and ear rots.

Local-spot foliar diseases: These affect primarily the leaves and sometimes other green tissues. Notable examples are Puccinia rust (Puccinia spp), Helminthosporium leaf blights (Helminthosporium spp), Curvularia leaf spot (Curvularia pallenscens) and brown leaf spot (Physoderma maydis). The local spot foliar diseases destroy the leaves and result in significant yield reduction when susceptible plants are infected early in their growth stage. Amongst these, *Helminthosporium turcicum* blight is most destructive as the disease can develop into epiphytotic proportions under favourable environmental conditions such as cool to moderate temperatures and adequate moisture (Purseglove, 1976; El-Shafey, 1978).

Systemic foliar diseases: These include maize streak and maize mottle viruses and downy mildew. The first two are caused by two different viruses but are transmitted by the same insect vector, *Cicadulina sp.* Downy mildew caused by fungus *Peronosclerospora sp.* is a threat to maize production in some countries of tropical Africa including Nigeria, Ivory Coast and Sudan (Effron, 1985).

Stalk and ear rots: Bacteria stalk rot incidence is low; attacks maize during the active growth stage causing losses usually. Fungal stalk rots are incited by *Fusarium moniliforme, Diploidia maydis, Cephalosporium maydis, Botriodiploidia* and *Rhizoctonia sp.* The later two are most important in low land rainforest and moist savanna. All the stalk rot pathogens cause kernel rot, resulting in grain deterioration and loss of quality especially in soft endosperm genotypes (Effron, 1985). Development of resistant varieties is the best means of controlling stalk and ear rots.

West and Central Africa Maize Research Network (WECAMAN) have released early and extra-early maize lines that could fit into the semi arid tropics. These lines are generally susceptible to foliar diseases in the tropics (Misovic, 1985). They are also very stable over years under low rainfall and dry land conditions with insufficient precipitations.

The interest on the studies on turcicum blight of maize in Samaru - Zaria was elicited due to:

a. Introduction of these early and extra early maize line vis-à-vis the short period of rainfall experienced in the northern savanna regions;
b. The severity of turcicum blight consistently reported by Adeoti (1982, 1995, and 1996) with other previous report by Fajemisin and Osunlaja (1977) and Cammack (1956);

c. High yield potential reported by Adeoti and Iwuafor (1997) in the regional trial experiment with the early and extra early maize lines from (WECAMAN) in Zaria and Minjibiri research stations.

The specific objectives of this study are as follows:

i. To confirm the identity of the causal organism;

ii. To screen early and extra-early maize lines in the glass house and on the field for their reaction to turcicum blight. This will help in identifying sources of resistance;

iii. To study the effect(s) of sowing date on the incidence and severity of turcicum blight with a view to determining the most appropriate time for sowing to avoid high incidence and severity of the disease; and

iv. To identify those germplasm that are high yielding which may be useful for breeding programme in future.

CHAPTER TWO

2.0 LITERATURE REVIEW

2.1 History and Nomenclature of the Organism

Helminthosporium turcicum Pass; *Drechslera turcicum* (Pass) Subram and Jain (perfect stage: *Trichometasphaeria turcica* Luttrel), the causal agent of northern corn leaf blight or turcicum blight of maize was first described on maize in Italy in 1876 (Tarr, 1962). Two years later, a similar fungus was described on maize in New Jersey, U.S.A. and was named *Helminthosporium inconspicum* (Tarr, 1962). These two species were generally identical as discussed by Drechsler (1923). Luttrell (1958) identified the perfect (Ascomycete) state in culture but it is not known to occur naturally.

Nisikado (1928), divided *Helminthosporium* into two subgenera: *Cylindro helminthosporium* and *Euhelminthosporium*. Ito (1930), raised *Cylindro-helminthosporium* to generic rank as *Drechslera*. This change was ignored until Hughes (1958) and Shoemaker (1959) recognised Ito's genus. Shoemaker (1959) raised the subgenus *Eu-helminthosporium* to generic rank as *Bipolaris* but did not provide a taxonomic treatment of these species. The key difference between *Drechslera* and *Bipolaris* is the difference in germination of the conidia. In *Drechslera*, the germ tubes are lateral and usually amphigenous; in *Bipolaris*, they are semi axial and polar (Luttrell, 1963). Also in *Drechslera*, the first septum delimits the basal cell, whereas in *Bipolaris* the first septum forms near the middle of the conidia.

Helminthosporium has been applied to the species with pseudopleurogenous conidia in the subgenera *Cylindro-Helminthosporium* and *Euhelminthosporium* (Luttrell, 1964). The name *Helminthosporium* based on *Helminthosporium maydis* as the type species should be conserved for the species in *Cylindro-Helminthosporium* and *Euhelminthosporium* in accordance with the provision in Article 14 of the International Code of Botanical Nomenclature or this

provision might later be abandoned (Lanjouw, 1961).

The subgenus *Helminthosporium* differs from the subgenera *Cylindro-Helminthosporium* and *Euhelminthosporium* in that the conidial germ tubes are percurrent. Furthermore the conidiophores proliferations when present are percurrent with the conidiophores usually grouped on a stroma. Luttrell (1964) however showed that the three subgenera are superficially alike as the conidia are produced at the tips and along the sides of the conidiophores. Based on this, the presentation of this thesis will be restricted to the use of *Helminthosporium turcicum* to refer to the pathogen and turcicum blight to mean the disease.

2.2 Distributions and Host Range

Turcicum blight of maize occurs worldwide; virtually everywhere maize is grown (Hooker, 1975). It is particularly favoured in warm wet subtropical and tropical regions. Such areas include Brazil, Nicaragua, Uruguay, Colombia, Argentina, Panama, U.S.A., Mexico, Italy, France, Austria, Eastern Europe, China, Egypt, South Africa, Nigeria, India, Japan, Turkey, Israel, the Philippines, New Guinea, Australia, New Zealand and Canada (Tarr, 1962).

Under natural conditions the fungus *Helminthosporium turcicum* appears to be largely confined to species of *Zea, Sorghum,* and *Euchlaena* (Tarr, 1962). Although Sprague (1950) has stated that fungi referred to as *Helminthosporium turcicum* are sometimes isolated from the roots of various grasses in northern America; the exact identity of these isolates however, remained in some doubt.

Tarr (1962) reported Ghimpu (1939) to have recorded *Helminthosporium*. *turcicum* on tobacco seedlings in Romania. It occurs on maize, most type of sorghum (Doidge *et al*., 1953; El-Shafey *et al*., 1982), Sudan grass, Johnson grass, *Paspalum conjugatum* (Hughes, 1952); and teosinte *Euchlaena*

mexicana. Tarr (1962) wrote that *H. turcicum* has been successfully transmitted to wheat, barley, oats, sugar cane and (doubtfully) to rice in India through inoculation experiment. In America, similar experiment gave negative results with Marquis wheat and Bonda Oats while Barbless barley was resistant (Robbles, 1949). There appears to be no reports of this fungus occurring on these hosts under natural conditions.

2.3 Symptoms

The symptoms of turcicum blight may be seen on nearly all aerial parts of the corn plant, but the most conspicuous lesions are found on the leaves (Gordon *et al*., 1959). On susceptible maize leaves, the symptoms first appear as chlorotic spots elongating and becoming straw-coloured. They finally become grey-brown in colour. The blotch is sharply demarcated from the surrounding healthy tissue by a thin dark brown line. The surface of the blotch becomes covered with grey-green conidiophores and if sufficient blight patches are present, the leaf shrivels and dies (Cammack, 1956). The disease first develops on the lower leaves and progresses upward on the plant's upper leaves of 2.5 - 15cm in the length of the lesions. In damp weather, large numbers of grayish-black spores are produced on the leaves often in concentric or target-like zones (Anonymous, 1973). The number, size and types of lesions have been reported to depend on the environmental weather conditions, susceptibility of the host plant and races of the pathogen.

In Florida, Cox and Wolf (1955), attributed a crown rot of sweet corn (maize) plants up to two months old to *Helminthosporium turcicum*. Leaves of affected plants suddenly wilted and necrosis started at the leaf margins and progressed inwards to the midribs with death occurring within 2-5 days. The initial discoloration of the vascular bundles subsequently extended throughout the crown area and sometimes upwards through the second node. However, the roots appeared to remain healthy until advanced stage of the disease. *Helminthosporium turcicum* was consistently isolated from diseased tissue and

caused crown rot symptoms when inoculated into potted plants, and typical leaf blight symptoms on leaves of maize plants. Known isolates of *Helminthosporium turcicum* produced crown rot symptoms when inoculated into crowns of maize plants. This disease is in some ways similar to seedling blight of maize previously attributed to *Helminthosporium species* in the United States (Stover, 1922), but differs in not being restricted to seedlings. Jennings and Ullstrup (1957) suggests that leaf blight is fundamentally a localized wilt disease.

2.4 Description of the Fungus

Helminthosporium turcicum Pass. is generally accepted as the name of the fungus inciting northern corn leaf blight or turcicum blight. Detailed descriptions have been published by Drechsler (1923); Nisikado and Miyake (1926); Nisikado (1928); Mitra (1923).

Spores of this fungus are olive-grey, spindle-shaped, slightly curved, three-to eight-septate, 20 x 105µ to 250µ (Anonymous 1973). Ellis (1971), described the conidia to be straight or slightly curved, ellipsoidal to obclavate, pale to mid straw-coloured, smooth, 4-9 - pseudoseptate, 40 - 144 (115)µ long, 18-23 (commonly 20-24)µ thick in the broadest part; hilum distinctly protuberant. Conidiophores emerging singly or in groups of 2-6 through stomata, straight or flexuous, brown up to 300µ long, 7-11 (mostly 8-9)µ thick.

Considerable variation in size of the conidia has been reported; no doubt this is affected by environmental factors and perhaps by the existence of several strains of the pathogen (Tarr, 1962). According to Tarr (1962), Drechsler (1923) reported 15-25 x 45 - 132µ in U.S.A., 20-24 x 80 - 100µ in Madagascar (Boriquet, 1946), 16-24 x 56 - 115µ in French Equatorial Africa (Saccas, 1954), 18-24 x 62-130µ in Argentina (Muntanola, 1954), 14 - 24 x 66 - 128µ in Israel (Keneth, 1958). The conidia are widest near the middle and taper towards both ends. The basal portion of the conidia tending to be rounded off

with a hilum and the apex always rounded, i.e. approximately fusoid (El-Shafey *et al*., 1982).

Trichometasphaeria turcica, the sexual stage, occurs rarely in nature but produces black, globose perithecia in the laboratory. The asci are cylindrical with a short stipe and contain one to six but usually two to four ascospores, which are hyaline, straight or slightly curved, typically three-septate and 13 to 17 x 42 to 78µ in size (Anonymous, 1973).

The fungus grows well on various standard media (Drechsler, 1923; El-Shafey *et al*., 1982). Luttrell (1958) reported that it produces small sclerotia in culture. The fungus can form chlamydospore within conidia after 4-5 weeks of sporulation (El-shafey *et al*., 1984).

El-Shafey *et al*. (1984) studied the effect of temperature and humidity on sporulation. They reported that the optimum temperature for sporulation of *Helminthosporium turcicum* on leaf lesion fell between 20 and 23^{o}C while optimum atmospheric humidity fell between 90 and 100%. Cox and Van Nostram (1957) in Florida reported an isolate of *Helminthosporium turcicum* with optimum temperature for growth in culture at 30^{o}C as compared with 20^{o}C for isolates from another area. El-shafey *et al*. (1982) reported 30^{o}C as optimum temperature for spore germination.

Considerable variation in growth characteristics - growth rate, growth density, colony colour and spore production - have been reported by El-shafey and El-fangary (1982); Ullstrup (1954); Hillu (1964). El-shafey and El-fangary (1982), established a correlation between the growth density, colony colour and sporulation. Colonies with high growth density and dark colour produced higher numbers of spores. In a foliar pathogen, such as *Helminthosporium turcicum* the spore production ability undoubtedly has a great influence on pathogenicity. Hillu (1964) showed that definite correlation exists between the

number of conidia in the inoculum and disease development. Robert (1952), reported that there appears to be no relationship between virulence and rate of growth in culture.

2.5 Disease Development

Knox-Davis (1974) studied the penetration of maize leaves by *Helminthosporium turcicum.* He reported that penetration is direct and a fine infection hyphae developed from the lower surface of the appressorium, penetrate the cuticle and the outer epidermal cell wall. It becomes thickened at the site of penetration. An intercellular vesicle is formed at the end of the infection hyphae and stout colonization hyphae extended to adjacent cells. Initial subcuticular growth of the fungus and development of more than one infection hypha from an appressorium were occasionally observed. In the susceptible maize, Gelic *et al*. (1987), showed that the plasmalema and tonoplast of mesophyll cells were affected first, followed by disorganisation and alteration of organells, chloroplasts being particularly sensitive. Bundle sheet cells were affected later and to a lesser extent the mesophyll cells. In the resistant Ht_1 line, cytoplasmic residues of prematurely dead cells surrounded by healthy mesophyll cells protect them and stimulate the activity, halting the infection process 36-48 hours after inoculation. Soon after infection has taken place, the pathogen becomes established in the xylem where mycelial growth is profuse in susceptible leaves. The profuse growth results in plugging of the xylem vessels, with subsequent wilting and necrosis of the chlorenchyma. Hyphae move from the xylem into the moribound parenchymatous tissues after lesion have formed (Jenings and Ullstrup, 1957).

2.6 Factors Affecting Disease Development

Generally disease development is determined essentially by four factors: Infection, latent period, pathogen sporulation and loss of infectious tissues (Van der Plank, 1963). According to Buddenhagen (1985), there are six

factors other than host resistance that restrict or affect maize disease distribution and severity. These are: location of origin of the pathosystem; ease of pathogen dissemination with seed or by wind; climatic conditions during the maize growing season(s); climatic conditions during the off season; contiguity of maize plantings in time and space and their genetic uniformity; and presence of alternative hosts for the vector as well as for the vectored pathogens.

Helminthosporium turcicum over winters locally in infected maize leaves as dormant mycelia (Hooker, 1975). The succeeding season, spores developing on old leaf tissue are wind dispersed to the new crop. These spores, under adequate moisture, germinate and penetrate maize leaves within a few hours. It is believed that infection on new crop mainly originate from the locally built up inoculum although spores can travel over long distances through the air (El-shafey *et al*., 1984). Cool to moderate temperature and free moisture favours infection (Raymundo and Hooker, 1981). Moderate temperatures (20 – 25^{o}C) and high relative humidity (90-100%) favours the development of turcicum blight (Levy, 1989; Levy and Cohen, 1983). Adipala *et al* (1993) reported turcicum blight to have caused extensive damage on maize in the humid (>80% RH), warm (24^{o}C) and wet areas (>1200mm rainfall) in Uganda.

Leach (1976), proposed electrostatic mechanism to explain the violent liberation of spores by *Helminthosporium turcicum*. He suggested that spores, sporophores, and surrounding surfaces accumulate charges of like signs creating a repulsive force that breaks a spore from its sporophores and propels the spore into the air. He further explained that the magnitude of the voltage across the leaves, increase with increasing relative humidity. Donald and Kyaw (1980) on evaluating the forces caused by electrostatic charges on spores concluded that although electrical effects may play some still unappreciated role in the weakening of the bond between spore and sporophore, the electrostatic force encountered out doors are not large

enough to propel spores of *Helminthosporium turcicum* into the air as reported by Leach (1976).

Turcicum blight has been reported to be most prevalent and damaging when cool to moderate temperatures and moist conditions prevail during the growing season, particularly during the plant's grain filling period (Smith and White, 1988; Gowda *et al*., 1989). Both laboratory studies and field studies have revealed turcicum blight incidence and severity to be affected by relative humidity - low relative humidity reduce the incidence while severity varies with cultivar, leaf position and plant growth stage (Gowda *et al*. 1989; Solomonvitz *et al*., 1992). Thakur *et al*. (1989) reported heavy damage on the popular corn hybrid pioneer Brand 3165 and Funks 4673A - both lack the Ht gene for resistance. Tillage practices have been reported to have effect on the severity of turcicum blight (Pedersen and Oldham, 1992). They reported that turcicum blight was more severe under no till than mulch till for susceptible hybrid. Solomonvitz *et al*. (1992) noted that turcicum blight severity varied with cultivar. They recorded highest yield loss when the middle third of the leaf canopy was defoliated or infected with *Helminthosporium turcicum*. They concluded that leaf position, cultivar and plant growth stage is important for evaluating losses caused by northern leaf blight and may explain the failure of various models to describe yield loss due to northern leaf blight on maize.

2.7 Host Resistance/Biological Races of the Pathogen

Turcicum blight first develops on the lower leaves and progresses upward to the plant's upper leaves. The characteristic symptoms are large boat-shaped, grayish lesions developing in a few days at each infected spot (Aldrich, 1967). The edges of the lesions are usually well defined. As the infection progresses, the fungus will sporulate profusely in the centre of the lesions, giving them brown black dusty appearance (Gordon *et al*., 1959), severe infection causes premature death of the leaves.

Various levels of polygenic resistance are available in maize inbreds as is monogenic resistance (Bowen and Pedersen, 1988). Polygenic, rate reducing or partial resistance effectively reduces rate of disease increase (Hughes and Hooker, 1971; Perkins and Pedersen, 1987; Mayer *et al*., 1991; Raymundo and Hooker, 1982). It is expressed by a reduction in lesion number and size without change in lesion type. Genotype with rate-reducing resistance range from highly resistant, in which few lesions form to susceptible, in which many large sporulating lesions are present (Elliot and Jenkins, 1946; Meyer *et al*., 1991).

Monogenic resistance (Geevers, 1975; Windes and Pedersen, 1991; Leonard *et al*., 1989) or race specific resistance Van dar Plank 1963) under the control of Ht_1, Ht_2 and Ht_3 genes, is characterized by chlorotic lesions with minimal sporulation that delays lesions epidemic onset or as in the case of gene Ht, delays lesion formation until well after anthesis (Geevers, 1975; Raymundo *et al*., 1981). El-shafey (1978) has described the chlorotic type of resistance under local conditions in Egypt. The susceptible type lesions are chlorotic and surrounded by a yellow halo. Necrosis if developed in the centre of lesion was usually delayed requiring about 5 days longer for development than in lesions of susceptible plants. This type of lesions is expressed both at seedling and adult field-grown plants (El-shafey, 1978; Hooker 1963). The single dominant gene conferring chlorotic lesion type of resistance can be incorporated into the parental inbred lines of the hybrid by back crossing (Hooker, 1963). Both the lesion number and lesion type of resistance interact together with a resultant effect of more favourable degree of resistance than that of either of them.

Another type of resistance (Chlorotic fleck) described by El-shafey *et al*. (1976) is very much localized occurring only on green house inoculated seedlings. Adult plants in the field are almost symptom less as the symptoms are generally not visible. He noted that very few spores are produced on chlorotic lesions and none on chlorotic fleck form of resistance.

Carson (1995) has reported another type of unique reaction in maize to infection by *Helminthosporium turcicum* - chlorotic halo which he referred to as simply “disease reaction”. It is not a resistant character as it differs from chlorotic-lesion type caused by the dominant genes Ht1, Ht2, Ht3 and HtN. Furthermore it differs in its apparent inheritance.

Existence of parasitic races of the fungus *Helminthosporium turcicum* on maize and different sorghum has been reported in India, USA, Egypt, China and other countries (Robert, 1960; Rhowmik and Prasada, 1970; El-shafey *et al*., 1982; Abadi *et al*. 1989; Thakur *et al*. 1989). Gene-for-gene relationship has been demonstrated for *Helminthosporium turcicum* on maize. Thus five physiological races have been demonstrated (Leonard *et al*., 1989; Thakur *et al*., 1989; Windes and Pedersen, 1991). Race 0 has the virulence formula Hti, Ht2, Hr3; HrN/ is widely distributed in many parts of the world (Abadi *et al.*, 1989). Race 1 was first detected in Hawaii with the virulence formula Ht2, Ht3, HtN/Ht1 (Berquist and Masias, 1974). Race 23, with the virulence formula Ht1, HtN/Ht2, Ht3, and Race 23N, with virulence formula Ht1/Ht2, Ht3, HtN, appears restricted to the continental United States (Thakur *et al.,* 1989). Race 2N, with the virulence formula Ht1, Ht3/Ht2, HtN, was reported in 1991 in Hawaii (Windes and Pedersen, 1991).

2.8 Seasonal Persistence, Spread and Control

Helminthosporium turcicum can survive as dormant mycelium in infected maize leaf tissue for at least two years when kept at room temperature (El-shafey *et al*., 1984). They noted that survival was greater in susceptible than in resistant leaves. Robert and Findley (1952) earlier reported survival of mycelia in dried leaf tissue for at least a year. They also reported that inoculum might over winter in infected corn leaves as dormant mycelium on which spores are produced the following season. The occurrence of the

disease in isolated locations suggests that inoculum may be wind borne and deposited as spore showers.

Turcicum blight is usually not controlled with fungicides in hybrid corn crops because of low crop value relative to high application cost. Results of field experiment carried out to control the disease by spraying with fungicides were not encouraging (Samara *et al*., 1974). Aldrich (1969) reported that it can be controlled by manzate or parzote dust spray (at 5 to 7-day intervals from knee-high to roasting ear stage), but this is too expensive to be practical except in high-value breeding nurseries. Bowen and Pedersen (1988) reported an excellent control of southern and northern leaf blight (turcicum blight) with propiconazole used in protectant application schedule. They noted that propiconazole has no currative effect when applied 24 hours after inoculation of corn with *Helminthosporium turcicum*, nor did fungitoxicity persist beyond 17 days after application. Fungistatic effect of propiconazole against *Helminthosporium turcicum* as noted by Bowen and Pedersen (1988), were inversely proportional to the rate of application; fewer and smaller lesions resulted from progressively higher doses of *Helminthosporium turcicum*.

The most effective control measure against turcicum blight is the use of disease resistant varieties (El-shafey, 1978; Hooker, 1963). Destruction of maize residue by incorporating it into the soil and a two-year rotation of maize has been recommended to reduce the infection in the absence of resistant varieties (El-shafey *et al*., 1984).

As the disease is known to affect sorghum and Sudan grass, Johnson grass, it is obvious that maize should not be planted too close to plots where these crops are grown in order to check the spread of the disease.

2.9 Economic Importance

Turcicum blight has been reported to be one of the most destructive diseases of maize. This has been attributed to the ability of the disease to develop into epiphytotics under favourable environmental conditions on susceptible maize plants (Purseglove, 1976; Perkins and Hooker, 1981; Ullstrup and Miles, 1957). Ullstrup and Miles (1957) reported the disease to be one of the most serious leaf diseases in the US Corn Belt and can cause yield losses of more than 50%. Yield losses appears to be influenced by environmental conditions, the level of disease resistance in the maize plant, races of the pathogen occurring in that region, the stage of plant growth when infection occurs. Perkins and Hooker (1981) reported yield loss of 30% or more when susceptible hybrids are grown under severe disease conditions. Long *et al*. (1978) reported a 66% reduction under favourable conditions for infection. Highest yield loss has been observed when infection occurred before silking, however, when infection occurs 6-8 weeks after silking, no yield reductions occurred (Raymundo and Hooker, 1981). Pataky *et al*. (1988) reported 12-20% yield reductions by turcicum blight, severity ranging from 15-58%. In Nigeria, turcicum blight is found to be widely distributed causing extensive damage to maize in Ibadan, Agege, Abeokuta, and Ilorin (Cammack, 1956). Fajemisin and Osunlaja (1977), classified turcicum blight as important disease of maize in areas with cool climate, destructive in the highland areas of Benue and Plateau states of Nigeria. They reported grain yield loss of up to 80%.

Adeoti (1982, 1985 and 1996) has consistently reported high incidence of turcicum blight during field surveys for crop diseases carried out on farmers' fields within Jos Plateau, Kabba, Zaria, Bauchi, Sokoto, Kaduna and parts of Katsina. He reported that the incidence of the disease was particularly high (up to 100%) on early and extra early maize lines planted late with complete loss of the foliage in some parts of Zaria in 1996.

CHAPTER THREE

3.0 IDENTIFICATION OF CAUSAL ORGANISM

3.1 Introduction

True knowledge of any plant disease must begin with the identification of the causal organism. The symptoms, cultural and morphological studies are useful in identifying a particular disease and the causal agent. The growth stage of plant and the time of the season during which the disease is observed will be quite useful in the overall control strategy.

3.2 Materials and Methods

3.2.1 Observations on Symptom Development and Collection of Samples

Studies on symptom development of turcicum blight on maize were carried out in 1997 on maize fields at the Institute for Agricultural Research (IAR), Zaria Research farm. Visits were made to maize fields and observation taken on symptoms of the disease at different stage of its development. Infected leaves were collected in paper envelopes and taken to the laboratory for isolation of the pathogen.

3.2.2 Preparation of Media: potato dextrose agar (PDA) medium.

The PDA used for this study was prepared according to the recipe given below:

Peeled Irish potato	200g
Dextrose	20g
Agar	20g
Distilled Water	1000ml

The peeled Irish potato were cut into small pieces, washed and boiled for one hour in 1000ml of water. Thereafter, the material was filtered through muslin clot. 20g each of dextrose and powdered agar-agar was added to the filtrate. The volume was made up to 1000ml and re-boiled to dissolve the agar-agar completely. Five hundred milliliters (500ml) each was dispensed into 1000ml

Erlenmeyer flask (Pyrex). Their mouths were closed with a stopper (plug of cotton wool) and autoclaved at 1.2kg/cm^2 steam pressure for 15 minutes. Petri dishes were earlier washed, dried and packed in steel canisters, covered and sterilized in an oven set at 120^{o}C for 6 hours to ensure adequate sterilization.

Before pouring the PDA into petri dishes, 5ml of 0.5% streptomycin sulphate solution (5mg/ml solution) was pipetted into each 500ml of the molten autoclaved PDA and rotated gently to mix properly. This is to prevent bacterial growth.

Pouring was done when the PDA-streptomycin (PDAS) had cooled to about 45 - 50^{o}C. The cotton wool plug was removed and the mouth passed through a flame of spirit lamp before carefully pouring about 15ml aliquots - enough to cover the base of the Petri dish into the sterilized Petri dishes. The poured plates were left for 24 hours to set on a clean bench in a clean room previously fumigated with potassium permanganate solution.

The PDA slants were prepared in MacCartney bottles. Using a measuring cylinder, 10ml aliquots of the dissolved agar were dispensed into each MacCartney bottle. The metal screw caps were screwed up so that it is not very tight to allow free flow of air and steam. The bottles were autoclaved at 1.2kg/cm^2 steam pressure for 15 minutes. The bottles were allowed to cool and while the PDA is still in the molten state, the bottles were arranged in a slanting position to solidify making sure the molten PDA medium does not reach the end of the bottles. Thereafter they were preserved in a refrigerator until when needed.

3.2.3. **Isolation Method**

Avoiding the necrotic central portions of the lesions which might have been invaded and colonized by saprophytes, the leaf with lesion(s) were cut off (.5cm) to include the margin of the advancing lesion and healthy portions. Some quantity of 1% sodium hypochloride (NaOCl) solution from the laboratory stock

solution was added to cut pieces of the diseased spot in a MacCartney bottle. The bottle was closed and shaken vigorously to achieve surface sterilization. The sodium hypochloride solution was drained off and the cut pieces were rinsed with two exchanges of sterile distilled water. With the aid of a forceps, flamed and cooled in ethanol, three pieces were carefully transferred unto the PDAS in Petri dish by opening the Petri dish carefully and covering the dish with the lid immediately after transferring the cut pieces. The whole process was carried out under sterile conditions in a Laminar Flow Chamber. The plates were kept on the bench in a previously fumigated clean room at room temperature for 72 hours.

3.2.4 Purification and Examination of Culture.

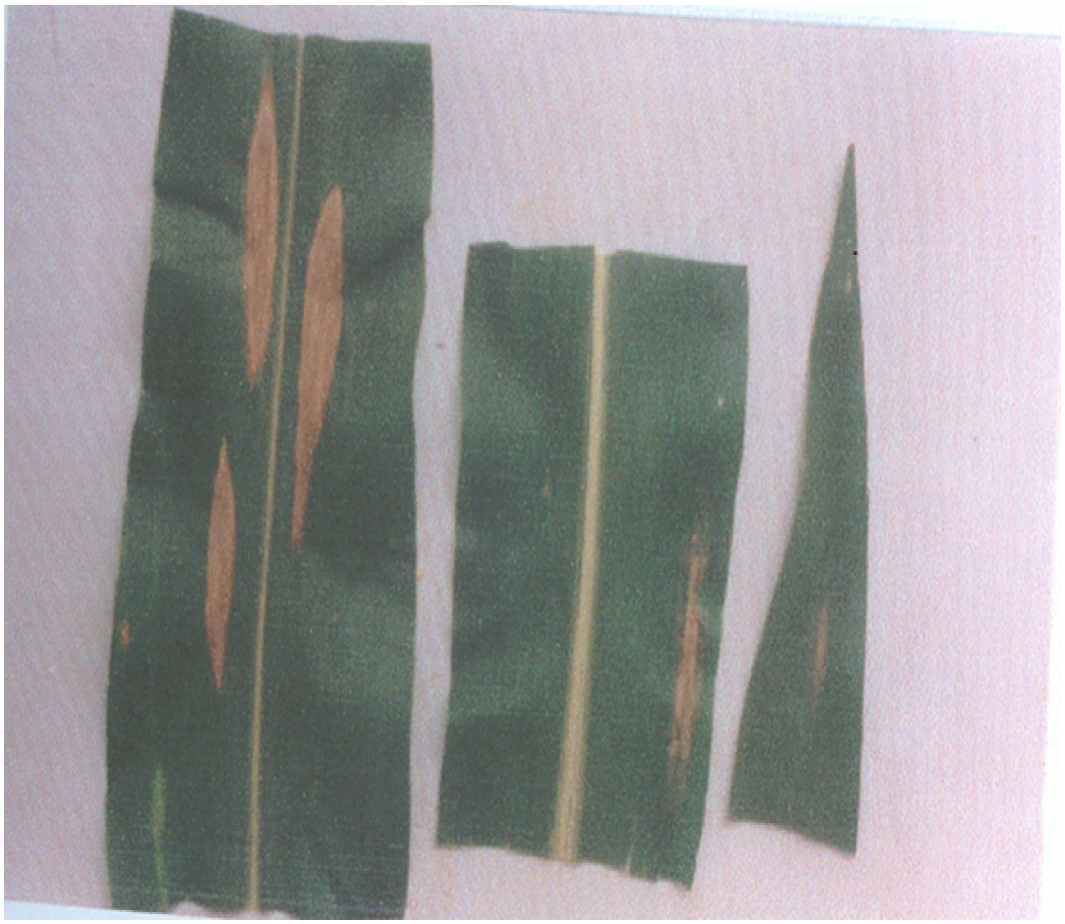
Pure culture of the fungus was obtained through the sub-culturing technique. After 72 hours, characteristic fungal hyphae growing out from the incubated leaf material into the agar were transferred unto fresh PDAS plates with the aid of an inoculating needle flamed first and cooled in ethanol. The process was carried out under sterile condition. The plates were again incubated at room temperature for 3 days. Thereafter, fungal hyphae growing out of the colonies were transferred unto fresh PDAS and incubated for 10 days at room temperature. Colony characteristic of the fungus were observed daily during the growth period until when the cultures have sporulated. Pure culture for further works was maintained on PDA slants in MacCartney bottles.

Detailed microscopic studies were carried out during the active growing stage on 8-day and 14-day old cultures to reveal any differences in the hyphae of actively growing culture and old culture. In each case, temporary slides were prepared and viewed under x 10 objective lens of a compound microscope. Spore size measurement was done using an ocular micrometer previously calibrated with a stage micrometer. In all, 30 spores were selected randomly and measured for the length, width and type of septation to obtain the size of the spores.

3.3 Results and Discussion

3.3.1 Symptomatology

Symptoms were observed when the maize was at their vegetative stage about 4 weeks old. The first noticeable symptom was a chlorotic spot on the leaf, which gradually elongated into a boat-shaped scald lesion. The lesions observed on different maize plants were not all equal in length. The lesion became longer and wider becoming brownish in colour. The matured lesion was sharply demarcated from the surrounding healthy tissue by a dark brown line (Plate 1). Lesion measuring 12cm long and 1.5cm wide was observed, typical lesion was 7-8cm long and 0.5-1cm wide. Some lesions were covered with grey-green colour, which reveals conidiophores and conidia when the surface was carefully scraped with a scalpel unto a clean slide, with a drop of lactophenol-in-cotton blue and viewed under x 10 lens of a compound microscope. Some of the leaves became blighted in appearance where several lesions coalesce together at the tasselling stage in some plants (Plate 2). These observations agreed with the description of turcicum blight disease on maize reported by Fajemisin (1973), and El-shafey (1978). El-shafey (1978) reported variation in size of lesions. Usually 4-6cm long and about 1cm wide on inoculated plants - fifteen days after inoculation. On adult field grown plants, the lesions were first seen on the lower leaves, spreading higher up the plant as the season progresses becoming up to 20cm long and 3cm wide. There were instances of reduction in the entire plant growth in a heavily infected plant with reduced cob (Plate 3). The ear leaf and the three leaves above were heavily infected. Pataky (1992) reported that the primary ear leaf and the leaves immediately above and below accounted for 33-40% of the total leaf area. Thus photosynthetic activity will be reduced when such leaves are heavily infected by turcicum blight. This is probably why there is reduced growth and reduced cob size in relation to other maize stands which were not severely infected.



late 1: Showing turcicum blight symptoms on maize leaf

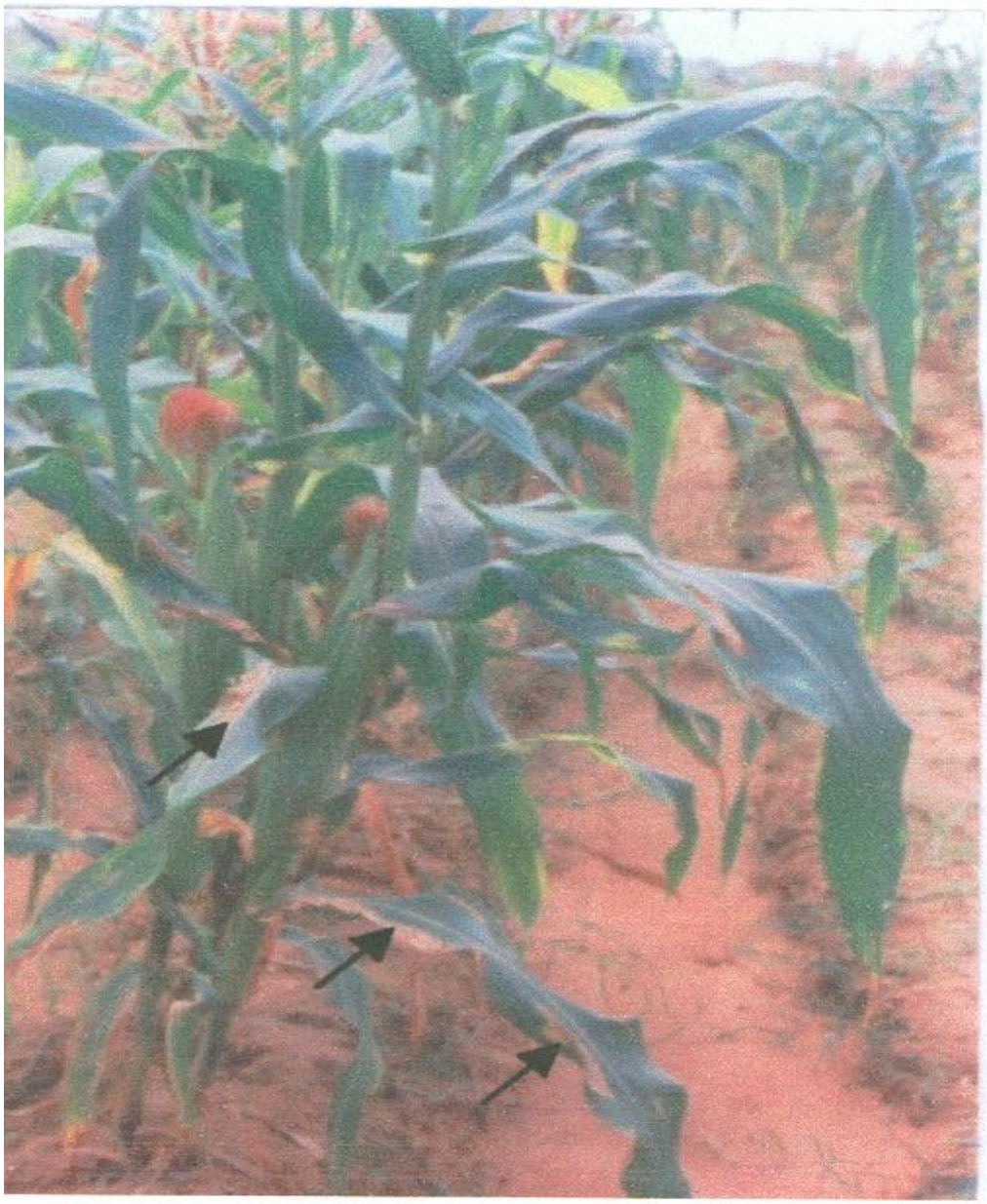


Plate 2: Showing maize leaves with coalesced lesions of turcicum blight

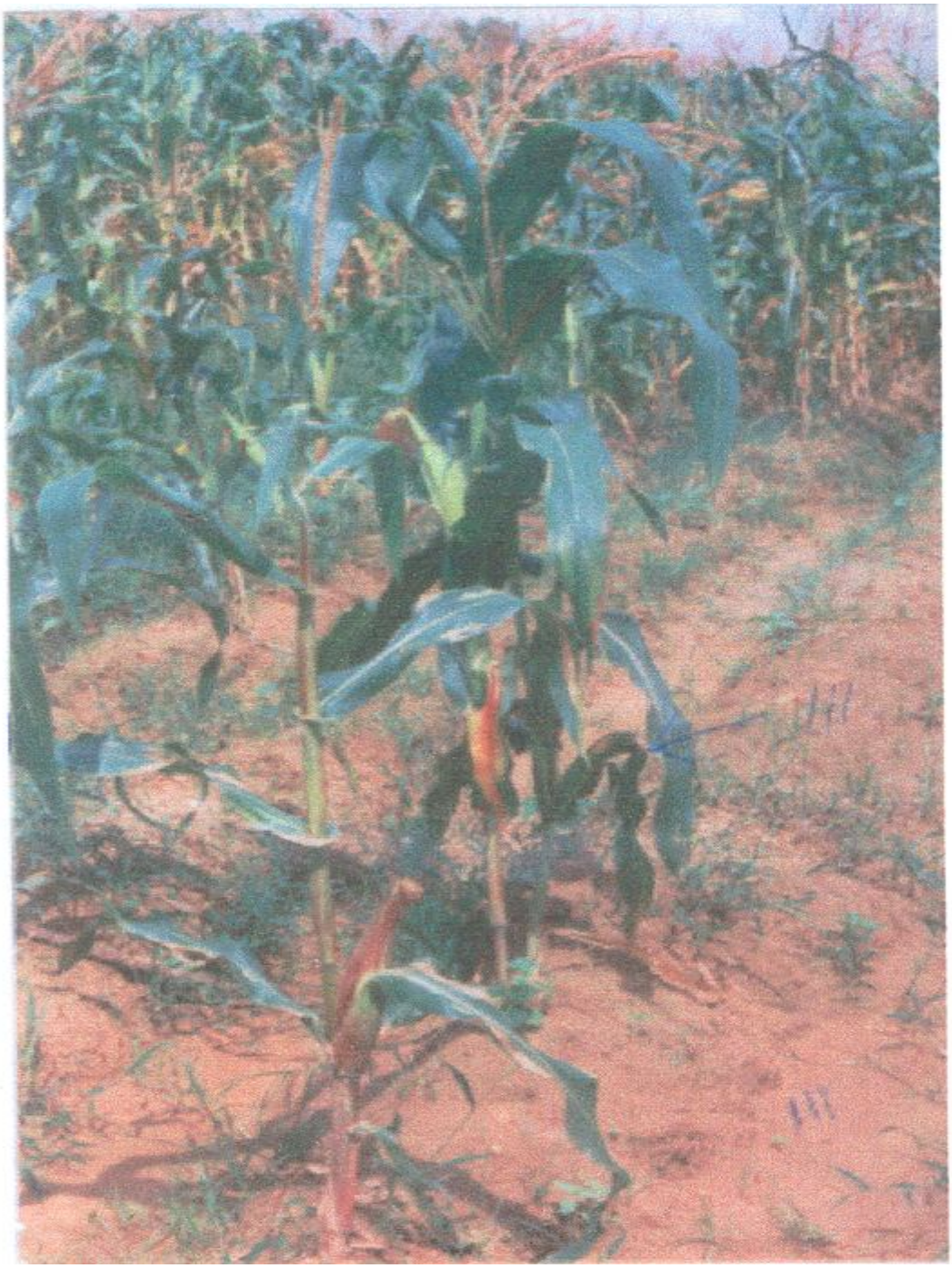


Plate 3: Plant suffering from heavy infection with reduced cob size

3.3.2 Isolation and Morphological studies of the Pathogen.

Helminthosporium turcicum Pass was isolated in pure culture from the infected leaf that was surface-sterilized, plated out and sub-cultured on PDAS at room temperature. *Helminthosporium turcicum* mycelia grew out of the incubated diseased material within 48 hours after inoculation and the colony was whitish. After 72 hours, the colour and nature of the colony changed to light gray, raised center becoming fluffy with a dark background at the center - that was the point of inoculation - when viewed from behind the culture plate. The dark colour became more evident on the fifth to seventh day with faint rhizoid irregular margin.

Microscopic examination of temporary slides prepared from advancing hyphae of actively growing culture reveals hyaline, narrow and widely septate hyphae. Hyphae from around the middle of the colony of 8-day-old culture reveal broad, sub-hyaline septate hyphae. These observations compared favourably with that of El-Shafey and El-Fangary (1982). These hyphae gradually become thick, wide and light yellowish brown in colour as the culture aged. Conidia were observed within 9 to 11 day-old cultures. The conidia were fusoid, straight to slightly curved, broader at the middle tapering towards each end with a hilum rounding off the basal portion (Plates 4 & 5). Evidence of conidia borne singly at the apex of the conidiophore was observed (Plate 6). The young conidia were hyaline and later became olive to yellowish brown colour, 5-9 septate. The minimum length of conidia was 55.00µm while the maximum length was 93.50µm with a mean length of 75.33µm.

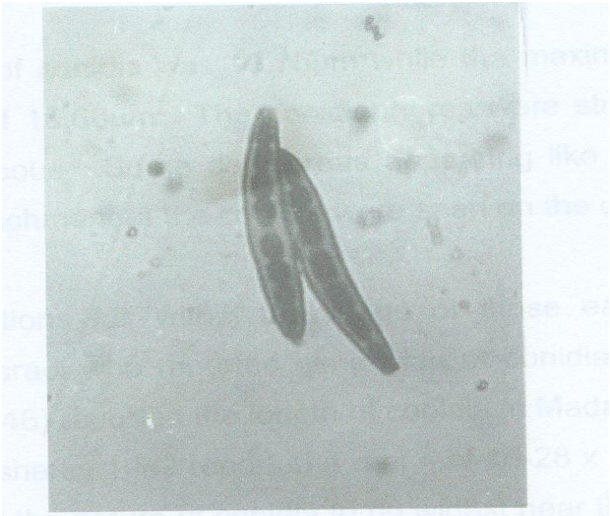


Plate 4

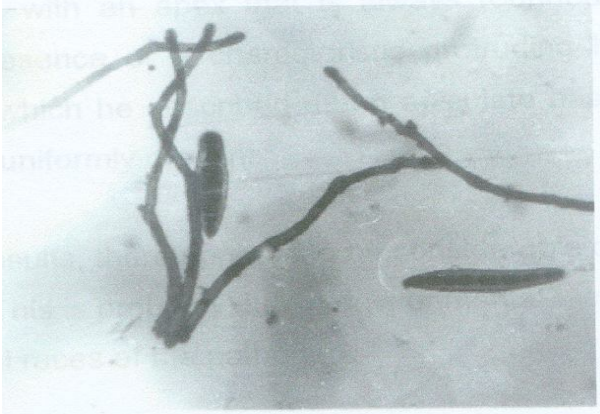


Plate 5

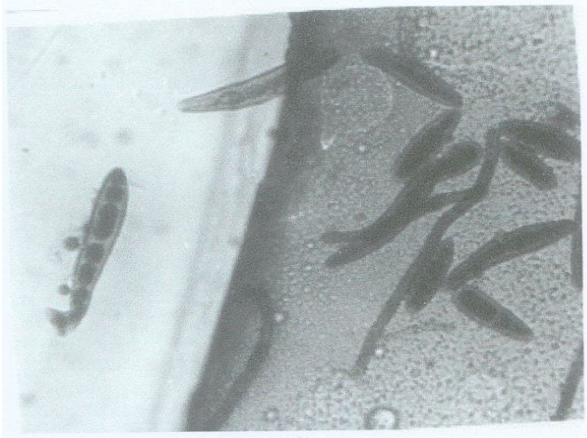


Plate 6

Plates 4, 5 & 6: Conidia and Conidiophore of *Helminthosporium tuccicum*

The minimum width of conidia was 13.75µm while the maximum was 19.25µm with a mean width of 15.68µm. The conidiophores were straight, or flexuous, brownish and olivaceous. Some dark areas appearing like scars marking the original places of attachment of the conidia were seen on the conidiophores.

The above observations fall within the range of those earlier described by Kenneth (1958) in Israel who reported dimension of conidia to be 14-24 x 66-125µm, Boriquet (1946) reported the length of conidia in Madagascar to be 20-24 x 80-100µm and El-shafey 1982 reported a range of 10-28 x 28-153µm in Egypt. He further described the nature of conidia to be widest near the middle and taper towards both ends, with an apex that is always rounded. Drechsler (1923) emphasized the presence of a characteristic protruding hilum as a valuable diagnostic feature, which he described as an apiculate basal protuberance that although minute, is uniformly present.

From the present results, there appears to be considerable difference or variation in size of conidia. This is probably due to environmental factors, and perhaps the existence of several races of the pathogen.

Table I: Dimension of Conidiospores and septation of *Helminthosporium turcicum*

S/No	Conidial (length (µm)	Conidial width (µm)	No of Septae
1.	82.50	16.50	8
2	82.50	16.50	7
3	66.00	13.75	7
4	77.60	16.50	7
5	82.50	13.75	8
6	77.00	13.75	7
7	55.00	16.50	7
8	77.00	13.75	5
9	71.50	16.50	8
10	82.50	13.75	8
11	77.00	16.50	9
12	88.00	13.75	9
13	82.50	16.50	9
14	66.00	13.75	7
15	93.50	16.50	8
16	60.50	16.50	8
17	71.50	16.50	7
18	55.00	19.25	7
19	71.50	16.50	9
20	60.50	13.75	8
21	82.50	16.50	8
22	71.50	13.75	8
23	77.00	13.75	8
24	66.00	19.25	8
25	82.50	16.50	7
26	71.50	16.50	6
27	88.00	13.75	6
28	77.00	13.75	7
29	77.00	16.50	7
30	88.00	16.50	6
X	75.35	15.68	
SED	2.49	0.42	

CHAPTER FOUR

4.0 SCREENING OF EARLY AND EXTRA-EARLY MAIZE LINES FOR RESISTANCE TO TURCICUM BLIGHT

4.1 Introduction

The use of resistant lines has been appraised as the most feasible, most economic, safest and most effective means of disease control (Agrios, 1997). This is an axiom as the use of resistant lines relieves the farmers of the cost of pesticides and it poses no health and environmental hazards to the users. Sourcing for resistance is the first step in developing resistance lines. This experiment was therefore designed to identify early and extra-early maize lines that could serve as source(s) of resistance to turcicum blight. Such lines could be recommended to the farmers or it could serve as sources of resistance in breeding against turcicum blight. The screening was carried out both on the field and in the screen house.

4.2 Materials and Methods

4.2.1 Field Screening

The early and extra-early maize lines screened for turcicum blight originated from West and Central Africa Maize Research Network (WECAMAN) and were obtained from the Cereals Research Programme of the Institute for Agricultural Research, Samaru, Ahmadu Bello University, Zaria, Nigeria.

Fifteen entries of the early (AB 11, KPB, AK 9331-DMRSR, ACR 92 TZE Comp. 5-W, Farakoba 90 Pool 16DT (CH), TZE Comp. 4DMR BC_2, DT-E-Y. SR, NAES Pool 16DT, Syn E2, TZE Comp C_1, Kamboinse 88 POD 16DT (RE), EV DT 94 C_2, TZE Comp. $4C_2$, Dorke-SR, and KPJ) and eleven entries of the extra-early (95 TZEE-Y1, TZEE-Y-SR BC_5, CSP x Local Raytiri, 95 TZEE-W1, TZEF-Y-SR, KEB, Csp-SR x TZEE-Y, TZE-W-SR B C_5, TZESR-W X Gua 314 BC_5, KEJ, and CSP-SR B C_5) maize lines were screened in the 1997 and 1998 cropping

seasons for their resistance to turcicum blight under natural field conditions in the IAR, Samaru, Zaria (Latitude $11^{o}11'N$, $7^{o}38'E$, altitude 686m) research farm. The experiment was set up in a randomized complete block design (RCBD) with each entry serving as a treatment; there were three replicates per treatment on a plot measuring 3m by 2 ridges spaced 75cm apart. Each plot was separated from another by two bare-ridges while replicates were separated by a two-metre gap. The plots for early and extra-early maize lines were separated by a 10-metre gap to minimize cross-pollination. Planting was done on the 26th of June 1997 and 29th June 1998 when the rains had established in both years. Three undressed maize seeds were planted per hole ridges spaced 75cm apart in a plot with intra-row spacing of 30cm and later thinned to 2 plants/stand after seedling has established.

Compound fertilizer (N.P.K. 15:15:15) was applied in two splits at the rate of 120kg N_2/ha: 60kg P_20_5/ha: 60kg K_20/ha. The first application was done three weeks after sowing at the rate of 60kg N_2/ha, 60kg P_20_5/ha and 60kg K_20/ha (0.18kg/plot) by spot application while the remaining 60kgN_2 was applied as top dressing from Urea five weeks after sowing. Hoe weeding was done before the first fertilizer application at 3 weeks after sowing. The ridges were molded up six weeks after sowing.

Observations were made on disease incidence and severity at different growth stages of the plant as well as the yield of each line.

4.2.2 Turcicum blight assessment

4.2.2.1 Incidence

The incidence of turcicum blight was visually assessed in all the plots at 30, 45, and 60 days after sowing (i.e. 15-day intervals) for the two lines. For each plot, the number of infected maize plants were counted and expressed as a percentage of the total number of maize plants in that plot. The mean percentage disease incidence for each treatment was obtained from the three

replicates. The data were subjected to analysis of variance and means were compared using LSD.

4.2.2.2 Severity

The method described by Fajemisin (1973) was adapted in assessing the severity of turcicum blight in each plot. Assessment for percentage disease severity was delayed until the maize plant reached silking stage as the ear leaf was used for severity assessment at 10 day-intervals. The procedure was as follow; first the lesion length on the ear leaf was determined by means of a ruler and given a numerical disease rating as provided by the key below adapted from Fajemisin (1973).

Table 2. Numerical disease rating, severity and reaction group.

Numerical disease rating	Lesion length (mm)	Reaction group
1	≤ 8mm	HR
2	8.1mm-16mm	MR
3	> 16mm - 24mm	MS
4	> 24mm - 32mm	MS
5	> 32mm - 40mm	S
6	> 40mm - 48mm	HS
7	> 48mm - 56mm	HS
8	> 56mm	HS

HR = Highly Resistant, MR = Moderately Resistant, MS = Moderately Susceptible, S = Susceptible, HS = Highly Susceptible.

A total of 10 plants were selected and assessed in each plot given the corresponding numerical disease rating. The percentage disease severity was estimated using the formula given below:

$$\text{Percentage Severity} = \frac{\Sigma X\,(100)}{8N}$$

Where X = numerical rating for each maize stand, N = the total number of plants assessed, and 8 = the highest numerical rating. The mean percentage disease severity for each treatment was calculated from the three replicates. The data obtained were subjected to analysis of variance and means were compared using LSD.

Fajemisin (1973) employed a scale of 1-7 in which 1 represent lesions up to 3mm in length, and 7 represent lesion of 21mm or longer with intermediate classes at 3mm interval. In his scale, lesions up to 3mm were referred to as resistant while lesions above 3mm were designated susceptible. This scale was modified in this work for three reasons: 1) the size of lesion found in this environment, 2) no known resistant variety was used as standard, and 3) no known races of the pathogen was used to classify the type of lesion that will be formed.

4.2.2.3 Yield Assessment

Yield for each treatment was assessed in each of the two years (1997 and 1998). Each plot was harvested when the maize were completely dried on the field and packed in labeled bags. Thereafter, threshing was done manually before drying the grains to constant weight. The final weighing was done to determine the actual yield in kg/plot. The data were extrapolated to kg/ha by multiplying by a constant (2222.22) obtained from the ratio of the area of a hectare ($10000m^2$) to the area of the plot per treatment ($4.5m^2$). The data was subjected to analysis of variance and the means were compared using LSD.

4.3 Glass House Screening

The glass house screening was carried out with the same fifteen and eleven entries of early and extra-early maize lines respectively. Seedlings were raised in plastic pots (12.5cm x 9cm) perforated at the bottom containing 800g of heat sterilized sandy clay soil. These were arranged on the bench in a glass house using Randomized Complete Block Design (RCBD). Each treatment was two maize seedlings per pot replicated six times, with a separate pot serving as control for each treatment.

Fertilizer application was done using N.P.K. 20:10:10 at the rate of 120kgN_2/ha, 60kgK_20/ha, and 60kg P_20_5/ha (.24g per pot) at two weeks after planting. 0.24g of N.P.K. 20:10:10 was carefully placed in two shallow holes scooped beside the maize seedling stands in each pot.

4.3.1 Inoculation of the plants in the screen house with conidial suspension

Conidia suspension used for inoculation was obtained from 14-day old PDAS plate cultures. The conidia (spores) from six plates were collected by carefully scraping the culture into 200ml sterile distilled water in a conical flask, using a sterile scalpel. This was thoroughly mixed with sterile glass rod, shaken vigorously and filtered through two layers of muslin cloth. The resulting suspension was further diluted with more sterile distilled water before it was adjusted to approximately 1 x 10^4 spore/ml using a haemocytometer and a compound microscope.

A hand sprayer was used for the inoculation. The spore suspension of the pathogen was sprayed unto the leaves of the maize plants at the 4-5-leaf stage of growth until run off. Just before spraying, the control pots were set aside, and sprayed with sterile distilled water before they were replaced. Thereafter all the pots were covered with transparent polyethylene bags for 48 hours to sustain

enough humidity for infection to take place. After 48 hours, the polyethylene bags were removed. Sterile distilled water was used to spray the leaves of the inoculated maize plants once daily after sun set for five days to enhance disease development.

4.3.2 Turcicum blight assessment

Disease assessment was carried out 15 days after inoculation. Severity was based on total plant leaf affected by visual observation of each treatment and the average for the six replicates was taken. Visual scale of 1-5 was used according to the description given below:

Table 3: Numerical scale, description of severity and reaction group of maize

Numerical Scale	Description of Severity	Reaction type
1	All leaves free from symptoms	Resistance
2	1-20% of leaves affected	Moderately resistance
3	21-40% of leaves affected	Moderately susceptible
4	41-60% of leaves affected	Susceptible
5	61% and above	Highly susceptible

4.4 Results and Discussion of Field Screening

4.4.1 Early Maize Lines 1997

The result (Table 4) showed that there was infection of turcicum blight on all the early maize lines in 1997 cropping season. The lines expressed different

reaction to the disease under same environmental conditions in the field. This was probably due to differences in their genetic make up. The result also showed that percentage disease incidence and severity increases with increasing days after sowing. This is a reflection of an overall increase in disease development as the season progressed. There was no significant (P = 0.05) difference in disease incidence among the lines at 30 DAS. However, the highest incidence (8.33%) was observed on lines EV DT 94 C_2 and TZE Comp 4 C_2 while the least (0.00%) was observed on Farakoba 90 pool 16 DT (HD). At 45 DAS, there was significant difference (P = 0.05) in disease incidence among the lines. The highest incidence (26.67%) was observed on TZE Comp. 4 C_2 and Syn E_2 while the least (10.00%) was observed on AB 11, AK 9331-DMRSR and ACR 92 TZE Comp 5-W. Line TZE Comp 4 C2 at 60 DAS sustained the highest incidence of 45.0%. This was significantly (P = 0.05) higher than that of EV DT 94 C_2 (23.33%) with the least incidence. There was no significant difference (p = 0.05) among all other treatments. Farakoba 90 Pool 16 DT (HD), which was free from infection at 30 DAS suffered 30.00% disease incidence at 60 DAS.

Disease severities among most of the lines at 50 DAS were not significantly different (P = 0.05). However, AK 9331-DMRSR (10.84%) was significantly (P = 0.05) higher than TZE Comp. 4 DMR BC_2 (7.92%), DT-E-Y.SR (7.92%), NAES pool 16D (7.71%) KPJ (7.92%) and Syn E_2 (6.67%) and Syn E_2 (7.29%). At 60 DAS, Kamboinse 88 POD 16 DT (RE) suffered the highest severity (18.96%). This was significantly (P = 0.05) higher than the least (9.38%) observed on Syn E_2. At 70 DAS, the lines significantly differ in disease severity. The highest severity (21.67%) was observed on KPJ while the least (10.84%) was observed on Syn E_2.

None of the lines was immune from turcicum blight infection neither was there any line that was highly susceptible. Wingard (1953) emphasized the fact that the different degrees of disease resistance displayed by plants are not fixed or absolute. Environmental factors may modify them greatly. The lines can be

grouped into two categories based on their reaction to turcicum blight. These are moderately resistant and moderately susceptible groups.

Syn E_2, Farakoba 90 Pool 16 DT (HD), DT - E-Y. SR, TZE Comp 4 DMR BC_2, ACR 92 TZE Comp. 5-W, NAES Pool 16 DT, KPB, AK 9331-DMRSR, TZE Comp. C_1 and Dorke-SR were observed to be moderately resistant. Lines KPJ, Ev DT 94 C_2, AB 11, TZE Comp. 4 C_2, and Kamboinse 88 POD 16 DT (RE) were observed to be moderately susceptible to turcicum blight.

There was significant difference (P = 0.05) in the yield of the lines (Table 4). The highest grain yield (3185.2kg/ha) was observed in EV DT 94 C_2, which was moderately susceptible. This was significantly higher than the least grain yield (1703.7kg/ha) obtained on TZE Comp C_1 that was moderately resistant. It thus implies that EV DT 94 C_2 is tolerant to turcicum blight. It could therefore, be a line from the moderately susceptible group that can be recommended for breeders to improve its reaction to the level of resistance to turcicum blight. Similarly EV DT 94 C_2 significantly (p = 0.05) out yielded AB 11 (2296.3kg/ha) and DT-E-Y.SR (2444.4 kg/ha) but with no significant difference with all other lines.

Table 4: Reaction of fifteen early maize lines to turcicum blight under field Conditions in Samaru during the 1997 Cropping Season.

Lines	Percentage mean disease incidence			Percentage mean disease severity at silking state			Yield kg/ha	Reaction type
	30 DAS	45 DAS	60 DAS	50 DAS	60 DAS	70 DAS		
AB 11	1.67	10.00b	31.67^{abc}	9.38abc	18.34^{ab}	21.25^{abc}	2296.3^{cdefg}	MS
KPB	5.00	21.67^{ab}	36.67^{abc}	9.59^{ab}	16.25^{abc}	17.50^{abcdefgh}	2888.9^{abcd}	MR
AK 9331-DMRSR	3.33	10.00^{b}	33.33^{abc}	10.84^{a}	15.63^{abc}	17.92^{abcdefgh}	3037.0^{ab}	MR
ACR 92 TZE COMP 5-W	3.33	10.00^{b}	30.00^{abc}	8.55^{abc}	12.71^{cd}	15.84^{defgh}	2740.7^{abcdef}	MR
Farakoba 90 Pool 16 SR (HD)	0.00	11.67^{b}	30.00^{abc}	8.34^{abc}	12.71^{cd}	13.96^{ghij}	2963.0^{abcd}	MR
TZE COMP 4DMR BC_2	3.33	21.67^{ab}	38.33^{abc}	6.67^{bc}	12.09^{d}	15.21^{ghi}	2963.0^{abc}	MR
DT-E-Y.SR	5.00	21.67^{ab}	40.00^{ab}	7.92^{bc}	12.09^{d}	15.00^{ghij}	2444.4^{bcdef}	MR
NAES Pool 16 DT	5.00	21.67^{ab}	41.67^{ab}	7.71^{bc}	13.13^{cd}	16.46^{defgh}	2592.6^{abcdef}	MR
Syn E_2	6.67	26.67^{a}	33.33^{abc}	7.29^{bc}	9.38^{d}	10.84^{ghij}	2592.6^{abcdef}	MR
TZE Comp. C_1	5.00	20.00^{ab}	41.67^{ab}	9.38^{abc}	13.13^{cd}	18.13^{abcdefg}	1703.7^{cdefg}	MR
Kamboinse 88 POD 16 DT (RE)	1.67	13.33^{b}	35.00^{abc}	9.59^{ab}	18.96^{a}	20.42^{abcd}	2666.7^{abcdef}	MS
EV DT 94 C_2	8.33	16.67^{ab}	23.33^{bc}	9.59^{ab}	18.33^{ab}	21.46^{ab}	3185.2^{a}	MS
TZE Comp 4 C_2	8.33	26.67^{a}	45.00^{a}	10.00^{ab}	16.05^{abc}	20.00^{abcde}	2814.8^{abcde}	MS
Dorke-SR	5.00	21.67^{ab}	30.00^{abc}	8.34^{abc}	16.25^{abc}	19.17^{abcdef}	3037.0^{ab}	MR

KPJ	1.67	13.33^{b}	36.67^{ab}	7.92^{bc}	17.50^{ab}	21.67^{a}	2888.9^{abcd}	MS
			^{c}		^{c}			
X	4.22	17.78	35.44	8.74	14.84	17.65	2720.99	
LSD	10.45	13.54	15.23	2.91	5.16	4.61	690.4	
SED	5.1	6.6	7.4	1.4	2.5	2.2	337.1	

DAS = Days after Sowing; MS = Moderately susceptible; MR = Moderately resistance. Means followed by the same letters in the same column are not significantly different at P = 0.05; $\overline{x}$ = overall mean; SED = standard error of difference. The values of mean disease incidence and severity are transferred lesion length into percentage

4.4.2 Early Maize Lines 1998

From the result (Table 5), turcicum blight incidence was lower at the vegetative stage (30 DAS) in 1998 than in 1997. The average incidence in 1997 at 30 DAS was 4.22% while in 1998 it was 3.78%. There was no significant difference (P = 0.05) among the lines. The least incidence (0.00%) at 30 DAS was observed on NAES pool 16 DT while the highest (10.00%) was observed on KEB. At 60 DAS, the highest incidence (40.00%) was observed on TZE Comp. 4 DMR BC_2. This was not significantly (P = 0.05) higher than the least (26.67%) observed on Dorke-SR.

Severity was low at 50 DAS. Line AB 11 sustained the highest severity (11.67%). This was significantly (P = 0.05) higher than Syn E_2 (6.88%), Dorke-SR (7.08%), AK 9331-DMRSR (6.88%), Farakoba 90 pool 16DT (HD) (8.13%), DT-E-Y.SR (8.13%) and TZE Comp. 4 DMR BC_2 (6.88%). At 60 DAS, the highest severity (22.50%) was observed on AB 11. This was significantly (P = 0.05) higher than the least (8.75%) observed on Syn E_2. Kamboinse 88 POD 16 DT (RE) (17.92%) was significantly (P = 0.05) higher than Syn E_2. At 70 DAS there was significant difference between them. Furthermore, AB 11 (26.05%) was significantly (P = 0.05) higher than Syn E2 (11.46%), NAES Pool 16 DT (15.00%), DT-E-Y.SR (15.63%), AK 9331-DMRSR (15.63%) ACR 92 TZE Comp. 5-W (16.46%) and Farakoba 90 Pool 16 DT (15.63%).

The early lines in 1998 were grouped into two categories - moderately resistant and moderately susceptible. The moderately susceptible groups are: AB 11, EVDT94 C2, KPB and Kamboinse 88 POD 16 DT (RE). The moderately resistant group are AK 9331-DMRSR, ACR 92 TZE Comp. 5-W, Farakoba 90 Pool 16 DT (HD), TZE Comp. 4 DMR BC_2, DT-E-Y.SR, NAES Pool 16 DT, Syn E_2, TZE Comp. C_1, KPJ, Dorke-SR TZE Comp. 4 C_2.

It was observed that KPB with severity 17.5% was moderately resistant in 1997 but became moderately susceptible in 1998. On the other hand, KPJ (21.67%)

and TZE Comp. 4 C_2 (20.00%), which were moderately susceptible in 1997, became moderately resistant in 1998. This change in reaction by the same cultivar could probably be due to environmental weather conditions and existence of different races of the pathogen involved (*Helminthosporium turcicum*). Shurtleff *et al* (1970) observed that diseases of corn, like those of other crops, vary in severity from year to year and from one locality or field to another, depending on the presence of the pathogen, weather and soil conditions and relative resistance or susceptibility of the corn. Levy (1989) reported that the epidemics of turcicum blight depend on the ability of *Excerohilum turcicum* to infect, grow and sporulate on corn plants. This infection process is affected by light, dew temperature, dew period, plant age, and inoculum concentration (Berger, 1970; Levy and Cohen (1983).

The result (Table 5) showed no significant difference (P = 0.05) in the grain yield of the lines in 1998. The highest grain yield (2259.3kg/ha) was observed on EV DT 94C_2, which was moderately resistant in 1998. The least grain yield (1407.4kg/ha) was observed on TZE Comp. C_1.

From the ongoing results (Table 4 and 5), EV DT 94 C_2, Dorke-SR, Farakoba 90 Pool 16 DT (HD), TZE Comp. 4 DMR BC_2 can be recommended to farmers for the following reasons: EV DT 94 C_2 gave the highest grain yield (3185.2kg/ha and 3259.3kg/ha) in 1997 and 1998 cropping season respectively. Although, it was moderately susceptible in 1997 and in 1998, it could be said to be tolerant because of its high yield potential.

Dorke-SR was observed to be more stable-moderately resistant in both years (1997 & 1998) with grain yield 2814.8kg/ha and 3037kg/ha in 1998 and 1997 respectively. Similarly Farakoba 90 Pool 16 DT (HD) was stable in its reaction and yield (2963kg/ha) in 1997 and 1998.

Line Syn E_2 could be recommended to breeders as a possible source of

resistance as it was observed to consistently sustain the least severity (10.84% and 11.46%) respectively in 1997 and 1998.

Table 5: Reaction of fifteen early maize lines to turcicum blight under field conditions in Samaru during the 1998-cropping season.

Lines	Percentage Mean Disease Incidence			Percentage Mean Disease Severity at silking state			Yield kg/ha	Reaction Type
	30 DAS	45 DAS	60 DAS	50 DAS	60 DAS	70 DAS		
AB 11	1.67	8.33	35.00	11.67a	22.50a	26.05a	2296.3a	MS
KPB	10.00	21.67	38.33	9.59abc	16.04abc	21.46ab	2814.8a	MS
AK 9331-DMRSR	1.67	23.33	31.67	6.88c	10.63cd	15.63bcd	2740.7a	MR
ACR 92TZE Comp.5-W	5.00	20.00	30.00	9.17abc	12.36bcd	16.46bcd	2666.7a	MR
Farakoba 90 pool 16DT(HD)	3.33	16.67	31.67	8.13bc	11.04bcd	15.63bcd	2963.0a	MR
TZE Comp. 4DMR BC2	5.00.	30.00	40.00	6.88c	12.92bcd	17.50abcd	2888.9a	MR
DT-E-Y-SR	1.67	23.33	33.33	8.13bc	12.09bcd	15.63bcd	2592.6a	MR
NAES pool 16 DT	0.00	23.33	33.33	8.75abc	11.46bcd	15.00bcd	2518.5a	MR
SynE2	6.67	28.33	35.00	6.88c	8.75d	11.46d	2444.4a	MR
TZE Comp C1	3.33	21.67	35.00	7.92bc	12.92bcd	17.7abc	1407.4a	MR

						d		
Kamboinse 88 POD 16DT(RE)	1.67	18.33	31.67	10.42ab	17.92ab	20.42abc	2814.8a	MS
EVDT94 C2	6.67	16.67	36.67	10.21abc	15.63abcd	20.00abcd	3259.3a	MS
TZE Comp 4C2	3.33	25.00	28.33	8.54abc	12.50bcd	18.54bcd	2592.6a	MR
Dorke SR	3.33	16.67	26.67	7.08bc	11.46bcd	16.04abcd	2814.8a	MR
KPJ	3.33	16.67	36.67	8.55abc	12.71bcd	19.17abcd	2592.6a	MR
X	3.78	20.67	35.56	8.59	13.40	17.78	2627.16	
LSD	10.17	19.06	21.99	3.46’	7.05	8.80	975	
SED	4.97	9.31	10.73	1.69	3.44	4.30	476.06	

DAS = Days after sowing; Means followed by the same letter in the same column are not significantly different at P = 0.05 LSD;

MR = moderately resistant; MS = Moderately susceptible; x = overall mean; SED = standard error of difference. Values of means disease incidence and severity are transformed lesion length into percentage.

4.4.3 Extra-early Maize Lines 1997

The result (Table 6) showed that all the extra-early maize lines were infected by turcicum blight. The lines reacted differently to the disease under same environmental conditions. However most of the lines were moderately resistant. The incidence and severity increased with the age of the crop similar to what was observed with the early lines. At 30 DAS, CSP-SR X TZEE-Y showed the highest incidence (10.00%). This was significantly (P = 0.05) higher than KEB (0.00%) and TZE-W-SR BC_5 (0.00%) but significantly different from all other lines. At 45 DAS, Csp-SR x TZEE-Y (40.00%) was significantly higher (P = 0.05) than all other lines except CSP x Local Raytiri (28.33%) with no significant difference. At 60 DAS, the highest mean disease incidence was observed on CSP-SR x TZEE-Y (55.00%) while the least (25.00%) was observed on KEB. There was significant difference (P = 0.05) between these two. Further more CSP-SR BC_5 and TZESR-W x Gua 314 BC_5 (30.00%) respectively were not significantly (P = 0.05) different with the least incidence (25.00%) observed on KEB. Also lines TZEE-Y-SR BC_5 and CSP x Local Raytiri (46.67%) were not significantly different (P = 0.05) from the highest incidence (55.00%) observed on CSP-SR x TZEE-Y.

Severity was low at 50 DAS than at 60 DAS and 70 DAS. This is a reflection of an overall increase in disease development as the season progressed. The highest severity at 50 DAS (17.09%) was observed on CSP-SR x TZEE-Y. This was significantly (P = 0.05) higher than the least (7.71%) observed on CSP-SR BC_5. Further more TZEE-Y-SR. BC_5 (14.59%), KEJ (13.93%), CSP x Local Raytiri (13.75%) were significantly (P = 0.05) higher than CSP-SR BC_5. At 60 DAS, the least severity (12.71%) was observed on TZESR-W- x Gua 314 BC_5 while the highest (23.13%) was observed on CSP-SR x TZEE-Y. There was significant difference (P = 0.05) among the lines. CSP-SR-BC_5 also exhibited significantly (P = 0.05) low severity of 12.92%. At 70 DAS, there was significant difference (P = 0.05) among the line in terms of disease severity.

Table 6: Reaction of eleven extra-early maize lines to turcicum blight under field conditions in Samaru during the 1997-cropping season.

Varieties	Percentage mean disease incidence			Percentage mean disease severity at silking state			Yield kg/ha	Reaction type
	30 DAS	45 DAS	60 DAS	50 DAS	60 DAS	70 DAS		
95 TZEE-Y1	5.00a	20.00b	35.00bc	10.00bcd	14.59b	18.75bc	2000.0b	MR
TZEE-Y-SR.BC_5	b	cd	46.67ab	e	cd	d	2222.2b	MS
CSP x Local Raytiri	3.33a	18.33b	46.67ab	14.59ab	19.80a	21.04ab	22963b	MS
95 TZEE-W_1	b	cd	38.33bc	13.75abc	b	20.63ab	2518.5b	MR
TZEF-Y-SR	8.33a	28.33a	31.67c	d	16.67b	17.29bc	2592.6b	MR
KEB	b	b	25.00c	10.63bcd	cd	d	2222.2b	MR
CSP-SR x TZEE-Y	5.00a	23.33b	55.00a	e	15.00b	18.13bc	2518.5b	MS
TZE-W-SRBC_5	b	c	31.67c	9.17cde	cd	d	3629.6a	MR
TZEST-W x	3.33a	11.67c	30.00c	8.13e	16.05b	17.92bc	2296.3b	MR
Gua314BC_5	b	d	33.33bc	17.09a	cd	d	2370.4b	MR
KEJ	0.00b	8.33d	30.00c	11.05bcd	16.67b	24.59a	2370.4b	MR
CSP-SR BC_5	10.00	40.00a	36.67	e	cd	17.50bc	2457.91	
$\bar{x}$	a	18.33b	14.15	9.59cde	23.13a	d	761.3	
LSD	0.00b	cd	6.78	13.93abc	14.59b	13.55d	364.94	
SED	5.00a	23.33b		7.71e	cd	19.38bc		
	b	c		11.42	12.71d	15.42cd		
	5.00a	20.00b			18.54a	18.56		

	b	cd	4.97	bc	5.11
	6.67a	16.67b	2.38	12.92d	2.45
	b	cd		16.42	
	4.70	20.76		5.49	
	8.57	14.04		2.64	
	4.12	6.73			

DAS = Days after sowing

Means followed by the same letter in the same column are not significantly different at P = 0.05 (LSD)

MR = moderately resistance; MS = Moderately susceptible; $\bar{x}$ = over all mean; SED = Standard error of difference.Mean disease incidence and severity are transformed lesion length into percentages.

The highest severity (24.59%) was observed on CSP-SR x TZEE-Y while the least (13.55%) was observed on TZESR-W X Gua 314 BC_5. Furthermore CSP x Local Raytiri (20.63%) and TZEE-Y-SR.BC_5 (21.04%) were significantly higher than TZESR-W x Gua 314 BC_5 (13.55%). The lines can be grouped into moderately resistant and moderately susceptible.

The moderately resistant group is made up of 95 TZEE-Y1, 95 TZEE-W1, TZEE-Y-SR, KEB, TZE-W-SR BC_5, TZESR-W x Gua 314 BC_5, KEJ and CSP-SR BC_5. The moderately susceptible groups are CSP-SR x TZEE-Y, TZEE-Y-SR BC_5, and CSP x Local Raytiri.

There was significant difference (P = 0.05) in the yield of the lines. TZE-W-SR BC_5 that was moderately resistant gave the highest grain yield (3629.6kg/ha) that was significantly (P = 0.05) higher than all other lines. However, the least yield 2000kg/ha was observed from 95 TZEE-Y1.

4.4.4 Extra-early Maize Lines 1998

The result of 1998 (Table 7) showed that there was low disease incidence at 30 DAS. However, the mean disease incidence for the lines at 30, 45 and 60 DAS (5.45%, 28.94% and 39.09%) respectively were higher than those of 1997 (4.70%, 20.76% and 36.67%). This was probably due to higher disease inoculum and better weather condition for infection (Appendix 1). The highest incidence at 45 DAS and 60DAS was observed on CSP-SR x TZEE-. The result also showed that CSP x Local Raytiri showed the highest incidence (11.67%) at 30 DAS. Furthermore it sustained the highest severity (17.10%, 26.47% and 31.90%) at 50, 60 and 70 DAS. CSP-SR BC_5 sustained significantly low disease incidence, and severity throughout the rating period.

All the lines were infected by turcicum blight by 30 DAS. The highest incidence (11.67%) observed on CSP x Local Raytiri was significantly (P = 0.05) higher than the least (1.67%) observed on CSP-SR BC_5, TZEE-Y-SR and TZEE-Y-SR BC_5. All other

Lines showed no significant difference (P = 0.05) in disease incidence. At 45 DAS, the highest incidence (55.00%) was observed on CSP-SR x TZEE-Y. This same Line sustained the highest incidence and severity throughout the rating dates in 1997. However, it failed to record the highest severity in 1998.The least incidence (16.67%) was observed on TZEE-Y-SR. There was significant difference (P = 0.05) in disease incidence between these two Lines. At 60 DAS, the least incidence (23.33%) was observed on CSP-SR BC_5. This was not significantly (P = 0.05) different from all other Lines except that KEB (31.67%) CSP-SR x TZEE-Y (60.00%) KEJ, CSP x Local Raytiri (50.00%) respectively were significantly (P = 0.05) higher.

Disease severity was significantly different (P = 0.05) among the lines at 50, 60 and 70 DAS. The lowest severity at 50 DAS (6.90%) was observed on KEB while the highest (17.10%) was observed on CSP x Local Raytiri. At 60 DAS, there was no significant difference (p = 0.05) in disease severity observed on 95 TZEE-Y1 (21.87%) and CSP-SR x TZEE-Y (21.67%). At 70 DAS the least severity (14.77%) was observed on CSP-SR BC_5 while the highest (31.90%) was observed on CSP x Local Raytiri. There was significant difference (P = 0.05) between these two lines. 95 TZEE-Y1 (26.27%) was significantly (P = 0.05) higher than 95 TZEE-W1 (16.03%), KEB (17.30%), TZEF-Y-SR (18.97%) and TZE-W-SR BC_5 (17.07%). Furthermore CSP-SR x TZEE-Y (23.13%) was significantly (P = 0.05) higher than CSP-SR BC_5 (14.77%).

In terms of disease reaction, the extra-early maize lines were grouped into moderately resistant and moderately susceptible. The moderately resistant groups are 95 TZEEW1, TZEF-Y-SR, KEB, TZE-W-SR BC_5 and CSP-SR BC_5. The moderately susceptible are 95 TZEE-Y1, TZEE-Y-SR BC_5, CSP x Local Raytiri, CSP-SR x TZEE-Y, TZESR-W x Gua314BC5 and KEJ.

Table 7: Reaction of eleven extra-early maize lines to turcicum blight under field conditions in Samaru during the 1998-cropping season.

Varieties	Percentage mean disease incidence			Percentage mean disease severity at silking state			Yield kg/ha	Reaction type
	30 DAS	45 DAS	60 DAS	50 DAS	60 DAS	70 DAS		
95 TZEE-Y1	5.00ab	23.33c	30.00b	9.20b	21.87ab	26.27ab	2000.0	MS
TZEE-Y-SR.BC_5	1.67b	28.33bc	46.67ab	12.10ab	18.13abc	22.07bcd	2296.3	MS
CSP x Local Raytiri	11.67a	35.00abc	50.00ab	17.10a	26.47a	31.90a	2148.1	MS
95 TZEE-W_1	6.67ab	28.33bc	36.67ab	7.93b	11.70c	16.03cd	2518.5	MR
TZEF-Y-SR	1.67ab	16.67c	28.33b	8.17b	14.20bc	18.97bcd	2518.5	MR
KEB	6.67ab	18.33c	31.67b	6.90b	12.73c	17.30cd	2222.2	MR
CSP-SR x TZEE-Y	5.00ab	55.00a	60.00a	11.67ab	21.67ab	23.13bc	2444.4	MS
TZEW-$SRBC_5$	3.33ab	21.67c	36.67ab	10.03b	12.73c	17.07cd	2814.8	MR
TZESR-W x	6.67ab	28.33bc	36.67ab	8.97b	18.57abc	21.27bcd	2148.2	MS
Gua314BC_5	10.00ab	45.00ab	50.00ab	10.63ab	16.07bc	20.20bcd	2518.5	MS
KEJ	1.67b	18.33c	23.33b	7.136b	11.47c	14.77d	2222.2	MR
CSP-$SRBC_5$	5.45	27.94	39.09	9.98	16.87	20.82	2350.17	
$\bar{x}$	8.33	19.08	23.36	6.02	7.80	7.06	1124NS	

LSD	4.00	9.15	11.20	2.88	3.74	3.39	538.90
SED							

DAS = Days after sowing

Means followed by the same letter in the same column are not significantly different at P = 0.05 (LSD)

MR = moderately resistance; MS = Moderately susceptible; $\bar{x}$ = over all mean; SED = Standard error of difference.

Mean disease incidence and severity are transformed lesion length into percentages.

It was observed that 95 TZEE-Y1, KEJ and TZESR-W x Gua 314 that were moderately resistant in 1997 were moderately susceptible in 1998. Changes in disease reaction i.e. moderately resistant to moderately susceptible by the same cultivar is probably due to environmental conditions for disease development and existence of different races of the pathogen involved. Wingard (1953) emphasized that the different degrees of resistance displayed by plants are not fixed or absolute. Environmental factors may modify them greatly. Levy (1989) observed that severity varies between years and locations. He further reported that severity depend greatly among isolates in Israel.

There was no significant difference (P = 0.05) in the yield of the Lines. However the highest grain yield (2814.8kg.ha) was observed on TZE-W-SR BC_5 while the lowest yield (2000kg/ha) was observed on 95 TZEE-Y1. The yield of 1997 was higher than that of 1998. The average yield in 1997 and 1998 were 2457.91 kg/ha and 2350.17kg/ha respectively. The lower yield in 1998 could probably be accounted for by a period of drought in the month of July when the total rainfall was 196.4mm (Appendix I). This was followed by excessive rain in August and September with total monthly rainfall was 473.6 and 321.8mm respectively. There was water logging in the field. There was also higher disease incidence and severity in 1998 than what obtained in 1997.

From the ongoing result, the moderately resistant lines with high yield potentials to be recommended to farmers are TZE-W-SR BC_5, TZEF-Y-SR, 95 TZEE-W1. Also the moderately susceptible lines with high yield potentials (tolerant) are CSP-SR x TZEE-Y and KEJ.

4.5 GLASS HOUSE SCREENING

4.5.1 Extra-early Maize Lines.

The result (Table 8) showed that the lines were all infected by turcicum blight. There was significant difference (P = 0.05) in the severity among the lines. Symptoms (chlorotic spot) were first observed in all the treatments in response to inoculation after 72 hours. At 15 days after inoculation (plate 7, 8), the highest severity (3.33) was observed on 95 TZEE-W1, TZEF-Y-SR and CSP-SR x TZEE-Y respectively. These were significantly (P = 0.05) higher than severity observed on all other lines but at par with CSP x Local Raytiri (3.00). Furthermore, CSP x Local Raytiri was significantly (P = 0.05) higher than CSP-SR BC_5 (2.00), KEJ (2.17) , 95 TZEE-Y1 (2.17), TZE-W-SRB5 (2.33) and TZESR-W x Gua 314 BC5 (2.33). The least severity (2.00) was observed on CSP-SR BC_5.

On the basis of disease reaction, CSP X Local Raytiri, 95 TZEE-W1, TZEF-Y-SR and CSP-SR x TZEE-Y were moderately susceptible while 95TZEE-W1, TZEE-Y-SR BC_5, KEB, TZE-W-SR BC_5, TZESR-W x Gua 314 BC_5, KEJ and CSP-SR BC_5 were moderately resistant.

4.5.2 Early Maize Lines

The result (Table 9) showed that there was significant difference (P = 0.05) in disease severity score among the lines. The highest severity (2.67) observed on Syn E_2 was significantly (p = 0.05) higher than the least severity (2.0) observed on KPJ, Dorke-SR and TZE Comp. C_1 respectively.

All the early maize lines were moderately resistant in the glass house (Table 9). The results (Tables 8 and 9) were slightly different from the field result (Tables 4 and 5). This observed differences in disease reaction in the field and glass house by same cultivar would be expected for the following reasons. Variation in environmental conditions i.e. rainfall; relative humidity, temperature are factors that affect various phases of the disease cycle. The genetic make up of the different cultivars and fitness of the pathogen could also be responsible.

Table 8. Reaction of eleven extra-early maize lines to turcicum blight in glass house fifteen days after inoculation.

Lines	Mean disease severity index	Reaction type
95 TZEE-Y1	2.17cd	MR
TZEE-Y-SR.BC_5	21.50bcd	MR
CSO x Local Raytiri	3.00ab	MS
95 TZEE-W_1	3.33a	MS
TZEF-Y-SR	3.33a	MS
KEB	2.67bc	MR
CSP-SR x TZEE-Y	3.33a	MS
TZE-W-$SRBC_5$	2.33cd	MR
TZESR-W x Gua314BC_5	2.33cd	MR
KEJ	2.17cd	MR
CSP-$SRBC_5$	2.00d	MR
$\overline{x}$	2.65	
LSD	0.65	
SED	0.32	

Means followed by the same letters in the same column are not significantly different at P = 0.05 (LSD)
MR = Moderately resistant; MS = Moderately susceptible

Scale for classifying the reaction was 1-5 in which 1 = Resistant, 2 = moderately resistant, 3 = moderately susceptible, 4 and 5 are susceptible and highly susceptible

Plate 7: Showing an over view of maize seedlings inoculated with Conidial suspension (1 x 10 spore/ml) of *Helmithosporium turcicum*

Plate 8: Shows maize seedlings inoculated with sterile water (a) and (b) inoculated with *Helmithosporium turcicum* Conidial suspension (1 x 10^4 spore/ml)

Infection process of turcicum blight is affected by light, dew temperature, dew period, plant age and inoculum concentration (Berger 1970; Levy and Cohen 1983). Levy 1989 in Israel reported that intensity of turcicum blight was dependent mainly on fitness of the population of *Helminthosporium turcicum* at a particular location. Thakur *et al*. 1989 observed isolates of race 2 of *Helminthosporium turcicum* to be virulent on maize inbreds B37 -Ht1 at both day/night temperature 22^{o}C/18^{o}C and 26^{o}C/22^{o}C but avirulent on inbred H4460 Ht1 at 26^{o}C/22^{o}C. Leath *et al*. 1990 observed that effect of environmental conditions on the expression of *Helminthosporium turcicum* cause problems on screening for resistance.

Table 9: Reaction of eleven early maize lines to turcicum blight in glass house fifteen days after inoculation.

Lines	Mean disease severity index	Reaction type
AB 11	2.50ab	MR
KPB	2.17ab	MR
AK 9331-DMRSR	2.50ab	MR
ACR 92TZE Comp.5-W	2.17 ab	MR
Farakoba 90 pool 16DT(HD)	2.33 ab	MR
TZE Comp. 4DMR BC_2	2.17 ab	MR
DT-E-Y-SR	2.33 ab	MR
NAES pool 16 DT	2.50 ab	MR
$SynE_2$	2.67 a	MR
TZE Comp C_1	2.00b	MR
Kambomse 88 POD 16DT	2.17 ab	MR
(RE)	2.33ab	MR
EV DT 94 C_2	2.17ab	MR
TZE Comp. $4C_2$	2.0b	MR
Dorke-SR	2.0b	MR
KPJ	2.27	
$\overline{x}$	0.59	
LSD	0.30	
SED		

Means followed by the same letters in the same column are not significantly different at P = 0.05 (LSD)
MR = Moderately resistant; MS = Moderately susceptible

Scale for classifying the reaction was 1 – 5 in which 1 = resistant, 2 = moderately resistant, 3 = moderately susceptible, 4 and 5 are susceptible and highly susceptible respectively.

CHAPTER FIVE

5.0 SOWING DATE EXPERIMENT

5.1 Introduction

Disease surveys carried out on farmers field (Adeoti, 1996) showed that turcicum blight was particularly high on extra-early maize lines planted late. High disease severity has been reported to precipitate economic yield loss. The highest yield loss has been observed when infection occurred before silking (Raymundo and Hooker, 1981). Prevailing factors such as temperature, rainfall, relative humidity and the age of the maize plant at the time of the attack can influence the incidence and severity of turcicum blight. Maize planted at different times during the growing season will not be affected equally by the presence of *Helminthosporium turcicum (Pass)*. This is because the factors affecting the incidence and severity vary throughout the cropping season. A trial was therefore set up to determine the most appropriate time for planting these extra-early and early maize lines to avoid or minimize the incidence and severity of turcicum blight thereby avoiding economic yield loss.

5.2 Materials and Methods.

One extra-early maize line CSP-SR x TZEE-Y and one early maize line AB 11 were used for the sowing date experiment. The two, lines have been previously observed to be susceptible in the screening experiment carried out in this study. The experiment was conducted in the Institute for Agricultural Research, Samaru, Zaria research field.

There were four sowing dates and each sowing date being a treatment. The land was ploughed, harrowed and ridged by a tractor. Each line replicated three times in a completely randomized block design. The size of each plot was 3m x 4 ridges .75 cm apart. Each plot was separated from the next by two meters gap. Sowing started when the rain has established in 1997 and 1998. The sowing dates for 1997 were June 26; July 10; July 24; and August 7 while in 1998, sowing dates were June 29; July 13; July 27; and August 8. Three seeds were sown per hole at 30cm between two holes. These

were thinned to one plant per stand two weeks after sowing giving a population of forty stands per plot.

Fertilizer application was at the rate of 120kgN_2/ha, 60kgP_20_5/ha and 60kg K_20/ha. The first application was carried out at three weeks after sowing using NPK 20:10:10 at the rate of 60kgK_20/ha, 60kgP_20_5/ha and 60kgN_2/ha (0.36kg/plot) by spot application and the second application of 60kgN_2/ha was done at five weeks after planting as top dressing using Urea. Hoe weeding was the same as in the field screening experiment section 4.2.1.

5.2.1 Turcicum blight Assessment

Incidence of turcicum blight was visually assessed in all the plots at 30 days (vegetative), 45 days (tarselling) and 60 days (silking stages) of growth (i.e. 15 days intervals). For each plot, the numbers of infected maize plants were counted and the total was expressed as a percentage of the total number of maize stands in that plot. The data obtained was subjected to analysis of variance based on randomized complete block design (RCBD). The means were separated using LSD.

Percentage severity of turcicum blight was assessed at silking stage, using the ear leaf. The procedure was the same as previously described in section 4.2.2.2, except that the two central roles in the 4-row plot were used for assessment. The data obtained was subjected to analysis of variance based on completely randomized block design. The means were separated using LSD.

5.2.2 Yield Assessment

The yield for 1997 and 1998 were assessed for each treatment as described in section 4.3. The data obtained was subjected to analysis of variance based on completely randomized block design. The means were separated using LSD.

5.3 Results and Discussion

5.3.1 Extra-early maize line, 1997

The results (Table 10) showed that there was significant difference (P = 0.05) in

turcicum blight incidence between the sowing dates at 30 DAS, 45 DAS and 60 DAS. The highest disease incidence (16.67%, 40.83% and 67.50%) at 30 DAS, 45 DAS and 60 DAS respectively was observed on maize sown in July 24 while the least was observed on maize planted on June 26. At 30 DAS, there was no significant difference (p = 0.05) in disease incidence between maize planted on July 10 (7.50%), July 24 (16.67%) and August 7 (14.17%). At 45 DAS, the highest incidence (40.83%) was observed on July 24 plantings. This was significantly higher (p = 0.05) than June 26 and July 10 (15.00% and 19.17%) respectively. At 60 DAS, July 24 plantings significantly showed higher disease incidence (67.50%) than those sown in August 7 (43.33%), July 10 (28.33%) and June 26 (26.67%). The least disease incidences observed on late June planting at 30, 45 and 60 DAS is an advantage of early planting over late planting. Oyekan (1977) observed that early season maize escaped severe infections of major diseases such as *Curvularia* leaf spot and *Helminthosporium* blight.

The result (Table 10) also revealed that severity at 50 DAS was low for all the treatment (sowing dates). Disease severity observed on maize sown in June 26 (4.69%), July 10 (5.10%) and August 7 (4.38%) showed no significant difference (P = 0.05) but the two were significantly lower than July 24 sown crop (6.88%). At 60 DAS, July 24 planting was significantly (P = 0.05) higher than other planting dates while the least severity was observed on June 26 (7.08%) and August 7 (7.61%). At 70 DAS, the severity differs significantly (P = 0.05). July 24 planting showed the highest severity (9.68%). This was significantly higher (P = 0.05) than all other planting dates in which there were no significant difference.

The result (Table 10) showed that there was significant difference (P = 0.05) in the yield between the sowing dates. The highest grain yield (2555.6kg/ha) was observed on maize planted in June 26 while the least (1296.3kg/ha) was observed on maize planted on July 24. Maize planted in August 7 (2111.11kg/ha) and July 10 (2037kg/ha) was not

significantly different (P = 0.05) from that of July 24.

5.3.2 Extra-early Maize Line 1998

The results of the sowing date experiment using extra-early maize line are represented in Table 11. The result showed that turcicum blight incidence was higher in 1998 than in 1997. The average incidence at 30 DAS and 60 DAS in 1998 were 11.25% and 45.21% while in 1997 it was 11.04% and 41.46% respectively. This is probably due to higher disease inoculum present in the field in addition to more favourable weather conditions for disease development. Gowda *et al*. (1989) indicated that turcicum blight intensity depend on location, meteorological factors (temperature, relative humidity and rainfall), cultivar susceptibility.

At 30 DAS, the lowest disease incidence (4.17%) was observed on maize planted in June 29 while the highest (18.33%) was observed on those planted on July 27. There was no significant difference (P = 0.05) between the sowing dates. At 45 DAS, there was no significant difference (P = 0.05) in disease incidence between the sowing dates. However the highest incidence (43.33%) was observed on July 27 planting while the least (16.67%) was observed on July 13 planting.

At 60 DAS, incidence was high (80.83%) on maize planted in July 27. This was significantly (P = 0.05) higher than the other sowing dates. The least incidence (25%) was observed on June 29 planting. Also the incidence on maize planted on August 10 (46.67%) was significantly higher (p = 0.05) than June 29 planting but at par with maize sown on July 13 (28.33%).

Table 10: Effect of incidence and severity of turcicum blight on extra-early maize line CSP-SR X TZEE-Y sown on different dates in Samaru in 1997 cropping season.

Sowing date	Percentage mean disease incidence			Percentage mean disease severity			Yield kg/ha
	30 DAS	45 DAS	60 DAS	50 DAS	60 DAS	70 DAS	
26/06/97	5.83[b]	15.00[b]	26.67[b]	4.69[b]	7.08[b]	7.61[b]	2555.6[a]
10/07/97	7.50[ab]	19.17[b]	28.33[b]	5.10[b]	7.09[b]	7.81[b]	2037.0[b]
24/07/97	16.67[a]	40.83[a]	67.50[a]	6.88[a]	8.95[a]	9.68[a]	1296.3[b]
07/08/97	14.17[ab]	23.17[ab]	43.33[b]	4.38[b]	6.56[b]	7.61[b]	2111.1[b]
$\bar{x}$	11.04	24.54	41.46	5.26	7.42	8.18	2000.00
LSD	10.26	21.13	19.06	0.83	1.48	1.21	677.2
SED	4.19	8.71	7.79	0.34	0.61	0.50	276.75

DAS = Days after sowing

Means followed by the same letter in the same column are not significantly different at P = 0.05 (LSD)
$\bar{x}$ = Overall mean; SED = Standard error of difference.

Mean disease incidence and severity are transformed lesion length into percentages.

Table 11. Effect of incidence and severity of turcicum blight on extra-early maize line CSP-SR X TZEE-Y sown on different dates in Samaru in 1998 cropping season.

Sowing date	Percentage mean disease incidence			Percentage mean disease severity			Yield kg/ha
	30 DAS	45 DAS	60 DAS	50 DAS	60 DAS	70 DAS	
29/06/98 June	4.17	17.50	25.00^{c}	7.50^{c}	13.13^{c}	17.08^{b}	1702.0
13/07/98 July	8.33	16.67	28.33^{bc}	11.67^{bc}	21.46^{bc}	26.25^{ab}	1259.3
27/07/98 July	18.33	43.33	80.83^{a}	23.55^{a}	37.09^{a}	38.34^{a}	1111.1
10/08/98 August	14.17	19.17	46.67^{b}	18.54^{ab}	31.67^{ab}	34.34^{a}	1074.1
$\bar{x}$	11.25	24.17	45.21	15.32	25.84	29.00	1286.63
LSD	15.95	27.46	21.37	7.67	12.67	12.99	918.3
SED	6.52	11.22	8.73	3.14	5.18	5.31	375.29

DAS = Days after sowing

Means followed by the same letter in the same column are not significantly different at P = 0.05 (LSD)

$\bar{x}$ = Overall mean; SED = Standard error of difference.

Mean disease incidence and severity are transformed lesion length into percentages.

At 50 DAS, there was significant difference (P = 0.05) in disease severity between

the sowing dates. Least severity (7.5%) was observed on maize sown in June 29 while the highest (23.55%) was observed on maize sown in July 27. There was no significant difference (P = 0.05) between the severity observed on August 10 planting (18.54%) and July 27 (23.55%), which was significantly (P = 0.05) higher than that of June 29 (7.5%). At 60 DAS, there was no significant difference (P = 0.05) in severity between July 27 (37.09%) and August 10 (31.67%) but both were significantly higher than June 29 planting. Furthermore, there was no significant difference (P = 0.05) between June 29 (13.13%) and July 13 (21.46%). At 70 DAS, the highest severity (38.34%) was observed on July 27 planting. This was not significantly (P = 0.05) different from August 10 (34.34%) and July 13 (26.25%) but significantly higher than June 29 (17.08%). There was also no significant difference (P = 0.05) between severity observed on maize planted in June 29 and July 13 (Table 9).

The results (Tables 10 and 11) showed that, the incidence and severity of turcicum blight was significantly (P = 0.05) low on maize planted early i.e. late June planting while significantly higher disease incidence and severity were observed on maize planted in late July. Thus when planting is done early enough, the severity of turcicum blight will be substantially reduced.

The result (Table 11) showed that there was a low yield in 1998. There was no significant difference (P = 0.05) in yield between the sowing dates. Highest yield (1702.0kg/ha) was observed on June 29 planting while the least (1074.1kg/ha) was observed on August 10 planting. The low yield obtained from maize planted in June 29 could be attributed to destruction caused by grazing cows in some plots with the June 29 and July 13 planting before harvest. Furthermore, there was water logging resulting from excess rainfall in the month of August and September (473.6mm and 321.8mm respectively) Appendix 1. Grain yield of cereals especially depends on duration of green leaf area and the specific activity of assimilation organs Shonbeck (1995). Infection with *Helminthosporium turcicum* will lead to a reduction of assimilating leaf area and damage of photosynthetic organs. Water logging could

also result in premature senescence of the maize leaves as observed on the August planting when there was water logging in the field.

5.3.3 Early Maize Line 1997

The results (Table 12) showed that there was no significant difference (P = 0.05) in percentage disease incidence at 30 DAS between the different sowing dates in 1997. However, the highest incidence at 30 DAS (15.00%) was observed on maize sown on the July 24 while the least (5.83%) was observed on those sown in June 26 and July 10, i.e., the earlier the sowing date, the less the disease incidence on the early variety cultivar.

At 45 DAS and 60 DAS, there was no significant difference (P = 0.05) observed on incidence between the sowing dates. Incidence on maize sown in July 24 was consistently higher while maize sown in June 26 consistently showed the least incidence.

This is an advantage of early sowing of maize. There is less disease incidence probably due to low relative humidity. Gowda *et al*. 1989 studied the variation of incidence of turcicum blight on maize at 2 weekly intervals. He recorded no incidence at 45 days after sowing which he attributed to low relative humidity. Oyekan (1977) observed that early season maize escaped severe infections by major diseases such as *Curvularia* leaf spot and *Helminthosporium* blight.

Disease severity observed on maize planted in July 24 at 50 DAS (8.86%) was not significantly different (P = 0.05) from that of August 7 (8.11%). However it was significantly (P = 0.05) higher than that of June 26 (4.07%) and July 10 (5.63%). At 60 DAS, maize sown in July 24 recorded 11.25% disease severity.

It was followed by August 7 (9.90%) and July 10 (8.02%) respectively. There was no significant difference (P = 0.05) between these three sowing date. They were

however significantly higher than June 26 planting (i.e. 5.31%). At 70 DAS, the highest severity (12.71%) was observed on maize planted in July 24. This was significantly (P = 0.05) higher than the severity observed on June 26 and July 10 (5.94% and 8.65%) respectively but statistically at par with August 10 (11.04%) planting. Furthermore, June 26 planting sustained severity 5.94% that was significantly (P = 0.05) lower than July 10 planting (8.65%); the earlier the planting the lesser the severity.

The highest grain yield (2592.6kg/ha) was observed on maize planted in June 26. This was statistically not different from July 10 (2111.1kg/ha) but significantly (P = 0.05) higher than July 24 (1629.6kg/ha) and August 7 (1925.9kg/ha).

Table 12.Effect of incidence and severity of turcicum blight on early maize line AB11 sown on different dates in Samaru in 1997 cropping season

Sowing date	Percentage mean disease incidence			Percentage mean disease severity			Yield kg/ha
	30 DAS	45 DAS	60 DAS	50 DAS	60 DAS	70 DAS	
26/06/97	5.83	14.17	25.83	4.07^{b}	5.31^{c}	5.94^{c}	2592.6^{a}
10/07/97	5.83	23.33	44.17	5.63^{b}	8.02^{ab}	8.65^{b}	2111.1^{ab}
24/07/97	15.00	27.50	45.00	8.86^{a}	11.25^{a}	12.71^{a}	1629.6 ^{b}
07/08/97	14.17	25.83	36.67	8.11^{a}	9.90^{ab}	11.04^{ab}	1925.9^{b}

$\bar{x}$	10.21	22.71	37.92	6.67	8.62	10.34	2064.80
LSD	12.87	14.39	20.01	1.93	2.63	2.70	579.1
SED	5.26	5.88	9.40	0.79	1.08	1.10	236.67

DAS = Days after sowing

Means followed by same letter in the same column are not significantly different (P =0.05 using LSD. SED = Standard error of difference, $\bar{x}$ = overall mean. Values of mean disease incidence and severity are lesion length (mm) transformed into percentage.

5.3.4 Early Maize Line 1998.

The results (Table 13) showed that there was significant difference (P = 0.05) in percentage disease incidence between the sowing dates only at 30 DAS. Unlike 1997, maize sown in August 10 had the highest incidence (13.33%) at 30 DAS. This was significantly higher (P = 0.05) than June 29 and July 13 (4.17% and 3.33%) planting respectively. At 45 DAS, highest incidence (17.50%) was observed on July 27 planting while the least (12.50% was observed on July 13 planting. At 60 DAS, maize planted on July 27 had the highest disease incidence (47.50%) while the least was observed in June 29 planting. There was however no significant difference (P = 0.05) in severity on maize between the two sowing dates. Incidence and severity were consistently higher on maize sown in July 27 throughout the rating dates. As a result of increased inoculums from infected plants in adjacent plots, plants from the third planting date were expected to develop the highest disease severity. Highest severity at 70 DAS was 26.04% obtained on maize sown on July27 while the lowest (17.09%) was observed on maize planted in June 29.

There was no significant difference (P = 0.05) in the yield of the sowing dates. The

highest yield (1592.6kg/ha) was obtained on maize planted on June 29 while the least 963kg/ha was from on August 10 planting. Maize planted on July 27 with the highest severity (26.04%) gave a low yield 1074.1kg/ha.

From the above results (Table 12 and 13), maize sown in June 26 and 29 were least affected by turcicum blight and subsequently gave the highest yield (2592.6kg/ha and 1592.6kg/ha) respectively. Those planted in July 10 and 13 followed these. The disease was more severe on maize planted on July 24 and 27.However the least yield (963kg/ha) observed on maize sown on August 10 (Table 13) 1998 was due to the destruction caused by cows that invaded the field before harvest. Also there was a rapid drop in rainfall in October (48.3mm) during grain filling period.

Table 13: Effect of incidence and severity of turcicum blight on early maize line AB11 sown on different dates in Samaru in 1998 cropping season.

Sowing date	Percentage mean disease incidence			Percentage mean disease severity at silking state			Yield kg/ha
	30 DAS	45 DAS	60 DAS	50 DAS	60 DAS	70 DAS	
29/06/98	4.17[bc]	21.67	26.67	8.96	14.59	17.09	1592.6
13/07/98	3.33[c]	12.50	43.33	13.13	20.21	22.71	1555.6
27/07/98	10.00[ab]	17.50	47.50	12.30	22.71	26.04	1074.1
10/08/98	13.33[a]	13.33	38.33	9.17	22.09	24.80	963.0

$\bar{x}$	7.71	16.25	38.96	10.89	19.9	22.66	1296.30
LSD	6.39	12.25	33.12	7.64	12.08	10.16	891.8
SED	2.61	5.82	13.54	3.12	4.94	4.15	364.46

DAS = Days after sowing

Means = Followed by same letter in the same column are not significantly different P = 0.05 using LSD.

SED = Standard error of difference, $\bar{x}$ = overall mean. Values of mean disease incidence and severity are lesion length (mm) transformed into percentage.

Tologbonshe (1983) obtained a higher fruit yield on eggplant, *Solanum species* and attributed this to escape of the early sown crop from attack by *Cercospora* leaf spot. Anaso (1989) also showed that early planting of maize reduced the incidence of infection of sorghum downy mildew with subsequent increased grain yield compared with late planting.

CHAPTER SIX

6.0 SUMMARY AND CONCLUSION

Sympotmatological studies carried out through observation on the field revealed that the first visible sign of turcicum blight was a chlorotic spot on the leaf, which gradually elongated into a boat-shaped scald lesion. This later becomes brownish in colour and demarcated from the healthy tissue by a dark brown line. Conidiophores and conidia were formed on the surface of the lesion.

When plated in the laboratory *Helminthosporium turcicum* grew well on PDAS. The colony was light grey, raised centre, fluffy with a dark background. Microscopic observations showed that the hyphae were widely septate, broad and yellowish brown in colour bearing conidiophores. The conidiophores were straight or flexuous. The conidia were broader at the middle tapering towards each end, septate with a hilium rounding-off the basal portion.

Reproduction of the disease symptoms by artificial inoculation was achieved by spraying the leaves of young maize plants to run-off with conidia suspension obtained from pure culture of the isolate of *Helminthosporium turcicum*.

Resistance screening carried out in 1997 and 1998 cropping season under field conditions, showed that the fifteen early and eleven extra-early maize lines reacted differently to turcicum blight infection. It was observed that the early lines syn E_2, Farakoba 90 pool 16 DT (HD), DT-E-Y.SR, TZE Comp 4DMRBC$_2$, ACR 92 TZE Comp. 5-W, NAES pool 16DT, AK 9331-DMRSR, TZE Comp. C_1, and Dorke-SR were moderately resistant in 1997 and 1998 respectively. On the other hand, AB 11, Kamboinse 88 pod 16DT (RE) were moderately susceptible. KpB, TZE Comp. 4 C_2, KpJ were erratic in their reaction.

The results indicated that EVDT94C$_2$, Dorke-SR, Farakoba 90pool 16DT (HD), TZE

Comp. 4 DMRBC_2 can be recommended to farmers as they have high yield potential and tolerant to turcicum blight. Further more the result indicated that synE_2 can be a promising genotype for resistance to turcicum blight.

The extra-early lines 95.TZEE-W_1, TZEF-Y-SR, KEB, TZE-W-SR BC_5, CSP-SRBC_5 were moderately resistant in 1997 and 1998 cropping season respectively. On the other hand CSp x local Raytiri, CSP-SR x TZEE-Y were moderately susceptible. 95 TZEE-Y_1, TZE-SR-W x Gua 314 BC5, KEJ and TZEE-Y-SR B5 were erratic in their reaction. The result indicated that TZE-W-SRBC_5, TZEF-Y-SR and 95 TZEE-W_1 could be recommended to farmers. The result also indicated that CSP-SRBC_5 is a promising genotype for possible source of resistance to turcicum blight.

When the cultivars were directly inoculated under glass house conditions, the extra-early maize lines CSP x local Raytiri, CSP-SR x TZEE-Y were moderately susceptible, similar to the field screening result. The early lines were all moderately resistant.

Sowing date trials conducted in 1997 and 1998 cropping seasons revealed that, turcicum blight disease was most severe on maize sown in late July i.e. 24th and 27th of July; those planted in August 7 and 10 followed this. It was observed that maize planted in June ending i.e. 26th or 29th of June were significantly less affected with turcicum blight with subsequent significantly higher grain yield.

Weather conditions appeared to be an influencing factor in the development of the disease. Maize planted in late July and August, had higher disease incidence and severity when the weather was damp. There was probably higher inoculum density of the disease from previously infected maize plants sown early in June and mid-July. The high rainfall and relative humidity in July and August (Appendix I) favours the development of the disease hence the high incidence on the maize sown in July ending and August. The early-planted maize in June attains maturity stage before

the build up of the disease reaches economic damaging level. Thus, sowing susceptible Lines early in the season, to attain the growth stage before weather condition becomes favourable for severe disease development will be a good control measure against turcicum blight.

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Appendix 1: **Meteorological data during the 1997 and 1998 cropping seasons at Zaria.**

1997	**Total**	**Mean Temperature (%)**		**Mean Relative humidity (%)**	
	Rainfall (mm)	**Max ^{o}C**	**Min ^{o}C**	**10 a.m.**	**4 p.m.**
May	86.4	32.1	21.1	74.9	50.5
June	155.2	30.1	20.5	79.6	65.6
July	213.8	28.8	19.9	84.5	68.5

August	290.2	28.8	19.9	83.4	73.3
September	182.6	30.0	19.1	80.8	24.8
October	82.4	31.2	20.0	79.6	65.9
November	0.0	32.9	14.8	43.3	30.1
1998					
May	69.5	33.2	22.5	76.67	57.23
June	129.6	32.00	19	82.4	64.90
July	196.4	31	21	84.3	73.55
August	473.6	31	23	87.6	81.81
September	321.8	30.6	22.5	86.1	73.00
October	48.3	31.5	22.4	78.8	69.00
November	0.0	33.9	20.2	34.63	24.3

Source: Meteorological unit, Soil Science Department, IAR/ABU, Zaria

APPENDIX 2: BIOGRAPHY

Name:	Mamudu Eragbai **OMO-EBOH.**
Date and Place of Birth:	May 22, 1964, Ihievbe, Owan-East LGA, Edo State.
Nationality:	Nigerian.
Business Address:	P.O. Box 477, Samaru – Zaria, Kaduna State.
Home Address:	Oshodi Compound, Emabu-Ihievbe Owan-East L.G.A., Edo State.

Educational Institutions Attended with Dates and Qualifications

Institutions Attended	From	**To**	**Cert./Degree**
University of Benin, Benin City	1986	1990	B.Sc. (Honours) Microbiology 2nd Class Lower Division.
Ihievebe Grammar School, Ihievbe, Owan-East	1981	1985	WASC/GCE `O' Level.
Oboikhoduma Primary School, Emabu-Ihievbe, Owan-East	1971	1976	First School Leaving Certificate

Post Held Since the Award of First Degree

Organization	**From**	**To**	**Position**
General Hospital Laboratory, Jalingo, Taraba State (NYSC)	1990	1991	Microbiologist
Ihievbe Grammar School, Ihievbe Owan-East L.G.A., Edo Sate	1992	1996	Biology Teacher

Year of Admission for Postgraduate Studies: 1995 - 96

Admission Number: M.Sc./Agric./11049/1995-96