CHAPTER 4

PHYSIOLOGIC SPECIALIZATION OF *Exserohilum turcicum,* IN KENYA

ABSTRACT

Isolates of *E. turcicum* from Kenya showed variation in growth rates when grown on potato dextrose agar at 22^{o}C. Most colonies reached a diameter of 9 cm in 7 to 11 days. Single-spore colony colors were similar, appearing whitish with a greenish tint that disappeared during and after onset of sporulation. Nineteen isolates were inoculated on *Ht*-bearing differentials of maize. Three isolates were race 0, three were race 1, one race 2, one was race 3, two were race 12; and nine did not fit any known classification and were named k1, k2 and k3, respectively. The isolate designated as k3 was virulent on germplasm with the *HtN* gene, but had a severe chrolotic reaction with *Ht1* gene bearing differentials. The k1 group were virulent to all differentials and k2 group was virulent to *Ht1, Ht2* and *Ht3* gene bearing differentials. Resistant reactions were chlorotic and non-sporulating while susceptible reactions was necrotic with profuse sporulation. This is the first record of racial specialization of *E. turcicum* in Kenya and indicates that the *Ht* gene could be very useful in Kenya for the control of northern leaf blight.

INTRODUCTION

Exserohilum turcicum (Pass.) Leonard & Suggs, the causal agent of maize northern leaf blight is favored by mild temperatures and high humidity (Ullstrup 1970). Heavy dews, cool temperature, and frequent rains create environmental conditions conducive for disease development (Jordan *et al*. 1983). Levy (1991) showed that isolates from different areas were different in parasitic fitness as indicated by infection efficiency, sporulation and lesion size, while isolates from the same location showed less variation. Inoculum in previous crops has been found to be critical in epidemic build up for subsequent cropping especially in non-tillage systems. Four biotypes are known. One biotype is avirulent to lines carrying genes *Ht1, Ht2, Ht3* and *HtN*. The other biotype is avirulent to lines with genes *Ht2, Ht3,* and *HtN* but is virulent to maize carrying genes *Ht1A* or B (Shurtleff 1973). The classification of *E. turcicum* into races is based on the resistant *Ht* gene reactions when inoculated with different isolates. The nomenclature suggested by Leonard *et al.* (1989) is widely used today. In their study, Leonard *et al*. (1989) proposed that evaluations be carried out in temperatures near 20 ^{o}C and light intensities of 25 to 50 lux because reactions associated with *Ht1, Ht2* and *Ht3* are thermal and photo sensitive. Race 0 has the resistance formula, *Ht1, Ht2, Ht3, HtN/,* race 1, *Ht2, Ht3, HtN / Ht1*; race 2, *Ht1, Ht3, HtN/Ht2,* race 3, *Ht1 / Ht2 Ht3,* race 12, *Ht3, HtN / Ht1, Ht2*, race 23, *Ht2, Ht3 / Ht1, HtN* and race 23N, *Ht2, Ht3, HtN / Ht1*. This classification left room for the accommodation of new races that may be encountered in future studies. The *Ht1, Ht2* and *Ht3* resistant gene occurs as chlorotic lesions with minimum sporulation, while the *HtN* induced resistance is expressed as a delay in disease development until after pollination (Leonard *et al.* 1989). Turner and Johnson (1980) reported the presence of race 1 in Indiana that was virulent on *Ht1* but not *Ht2*. Lipps and Hite (1982) reported the presence of race 1 in Ohio that was found also virulent on *Ht1* but avirulent on *Ht2*. Smith and Kinsey (1980) reported a new race designated race 3 with a resistance formula *Ht1/Ht2,Ht3*. Later in 1983, Jordan *at al*. (1983) reported the occurrence of race 1 and race 2 from seven states in the central and eastern USA where race 1 was virulent on B37 only and race 2 virulent on B37Ht1. No isolate was found virulent on B37*Ht2* or Oh43Ht3. Welz *et al.* 1993 indicated the presence of race 0 and race 1 in China, races 23N, 23, and 2N in Mexico, race 23, 23N, and race 0 in Zambia,

and race 0, 2, N, and race 23N in Uganda. The report from Uganda by Adipala (1993a) found that all the isolates tested were virulent on A619 and avirulent on A619*Ht1*, A619*Ht2*, A619*Ht3* and A619*HtN*, hence they were classified as race 0. These observations are at variance with the results of Welz *et al*. (1993) where they reported at a similar time the presence of race 0, 2 and 23N in Uganda. This project was undertaken to examine physiologic specialization of *E. turcicum* in Kenya.

LITERATURE REVIEW

Exserohilum turcicum) (Pass.) Leonard & Suggs, the causal agent of maize northern leaf blight, was first reported in Passerini on maize in Italy in 1876. The disease was reported in New Jersey in the USA in 1878 and a serious outbreak occurred in Connecticut in 1889 (Drechsler C. 1923). The disease is favored by mild temperature and high humidity (Ullstrup 1970). Heavy dews, cool temperature, and frequent rains are environmental conditions conducive for disease development (Jordan *et al*. (1983). Levy and Cohen (1983) reported that the disease is more aggressive in young susceptible plants with an optimum temperature for infection and lesion number at 20^0C. Lesion length and inoculum concentration, increase with extended dew period.

Levy (1989) reported that pathogenic fitness and environmental conditions are important factors in determining disease development and epidemics depend on the ability of *E. turcicum* to infect, grow and sporulate on corn plants. In the continental United States the disease has been effectively controlled by the deployment of dominant *Ht* genes (Smith and Kinsey 1980, Turner and Johnson 1980, Hooker 1961). A new cholorotic halo gene different from *Ht* gene has been reported by Carson (1995a) which may be of limited commercial value alone, but may be useful in combination with *Ht* genes. Pratt e*t al*. (1993) reported a polygenic-based resistance in OhS10 expressed as rate-reducing resistance or low number of lesions. The homozygotes obtained by combining the *Ht1* and *Ht3* genes did not result in significantly less disease than in each parent (Leath and Pedersen 1986). However Dunn and Namm (1970) reported gene dosage effects for the *Ht* gene, and Hooker and Perkins (1980) reported gene dosage effects for *Ht2* gene. Smith and Kinsey (1980) suggested that a combination of *Ht1* and *Ht2* or *Ht3* genes would provide resistance against races 1, 2 and 3.

However, recently Pataky (1994) showed that high levels of partial resistance with or without *Ht* genes presented a spectacular approach in reducing damage from northern leaf blight on sweet corn, which also eliminates the severe yield-depressing chlorosis, associated with *Ht* gene resistance in very susceptible backgrounds. Studies by Carson (1995b) indicated that the latent period is related to partial resistance, which suggested that selection for increased latent period length would be more beneficial than selecting for reduced disease severity. Selection for increased latent period length can be

done in environments without severe disease epidemics, and also breeding material could be assessed as seedlings for latent period length in the greenhouse during the off season. Levy (1991) showed that isolates from different geographic areas were different in parasitic fitness while isolates of the same location showed less variation. Parasitic fitness was measured by infection efficiency, sporulation and lesion size. Inoculum from previous crops has been found to be critical in epidemic build up during subsequent cropping especially in non-tillage systems (Pedersen and Oldham 1992). Leath and Pedersen (1986) found that the progeny from a cross between resistant B37*Ht3* and susceptible B37 had a severe chrolosis associated with the resistant lesions. The presence of intense chlorosis although indicating resistance resulted in leaf damage which was classified as diseased which was reflected as high area under the disease progress curve. These values are normally unusual for resistant inbreds with low sporulation and secondary spread. Four biotypes of *E. turcicum* are known. One biotype is avirulent to lines carrying genes *Ht1, Ht2, Ht3* and *HtN***.** The other biotype is avirulent to lines with genes *Ht2 ,Ht3,* and *HtN* but is virulent to maize carrying genes *Ht1*A or B (Shurtleff 1973). The *Ht1, Ht2* and *Ht3* genes confers resistance to *E. turcicum* as chrolotic lesions with minimum sporulation, while the *HtN* gene is expressed as a delay in disease development until after pollination (Leonard and Levy 1989). The initial designation of races of *E. turcicum* was done by Berquist and Masias (1974) after they characterized an isolate from Hawaii, which was virulent to *Ht1* as race 2. The avirulent isolates were called race 1 but now are called race 0. The genes *Ht2, Ht3* and *HtN* are effective against both race 1 and race 2 (Leonard *et al*. 1989). Smith and Kinsey (1980) reported a new race designated race 3 with a virulent formula *Ht1/Ht2,Ht3.* Race 4, with the virulence formula *Ht1 / Ht2, Ht3, HtN,* was isolated in Texas (Thakur e*t al*.1989)

Turner and Johnson (1980) reported the presence of race 1 in Indiana. Lipps and Hite (1982) likewise reported that an isolate of race 1 in Ohio to be virulent on *Ht1* but avirulent on *Ht2*. Later, Jordan *et al.* (1983) reported the occurrence of race 1 and race 2 from seven states in the central and eastern USA where race 1 was virulent on B37 only and race 2 virulent on B37*Ht1*. None of the isolates were virulent on B37*Ht2* or *Oh43Ht3.* The race classification of isolates of *E. turcicum* is based on resistant genes matched by an isolate in the currently accepted nomenclature (Leonard *et al*.(1989) but

the old classification is still used widely. In this study it was proposed that evaluations be carried out in temperatures near 20 ^{o}C and light intensities of 25 to 50k lux because reactions associated with *Ht1, Ht2* and *Ht3* are thermal and photo sensitive. They also reported that resistance in lines with *Ht2* and *Ht3* was expressed clearly in controlled environmental chambers at 22^{o}C and 18^{o}C night temperatures. Race 0 has the resistance formula, *Ht1, Ht2,Ht3,HtN*/, race1, *Ht2,Ht3,HtN/Ht1;* race 2, *Ht1,Ht3,HtN/Ht2*; race 3, *Ht1/ Ht2/Ht3*; race 12, *Ht3,HtN/Ht1,Ht2*; race 23 as *Ht2,Ht3/Ht1,HtN;* race 23N as *Ht2,Ht3,HtN/Ht1* (Leonard *et al*. 1989). Welz *et al.* (1993) indicated the presence of race 0 and race 1 in China, races 23N, 23, and 2N in Mexico, race 23, 23N, and race 0 in Zambia, and race 0, N, 2 and 23N in Uganda.

According to Gevers (1975) the *HtN* major gene resistance derived from the Mexican maize cultivar Pepitila is reasonably stable but in some parts of the world the effects may fail to be expressed. Genetic segregation may not behave like expected of dominant gene ratios but it does however remain in the tolerable limits of deviation of stability and segregation. He suggested the emergence of biotypes in India able to overcome the *HtN* gene resistance. The *HtN* gene is also background sensitive. Plants were evaluated at both 26 ^{o}C day / 22 ^{o}C night and 22 ^{o}C day/18 ^{o}C night temperatures. There was observable weakened virulence at high temperatures. Another isolate from Hawaii caused disease on B37*HtN*, Oh45*HtN*, B14A*HtN* and B68*HtN* and was designated race 2N with a virulent formula *Ht1, Ht3 / Ht2, HtN* (Windes and Pedersen 1990). Pataky *et al*. (1986) reported that hybrids with or without *Ht2* did not show significant differences in disease severity induced by races 1 and 2 which may have been due to shading of lower leaves because resistance is lowered at low light intensities. Recent studies in Uganda (Adipala *et al.* 1993a) found that the isolates tested were all virulent on A619 and avirulent on A619*Ht1*, A619*Ht2*, A619*Ht3* and A619*HtN*; hence they were classified as race 0. These observations are different from the results of Welz *et al.* (1993) that reported the presence of other races in Uganda in addition to race 0. Adipala *et al*. (1993) found that average disease severity ranged between 0.5 to 25% in Uganda. Ugandan maize germlasm, exhibited necrotic susceptible reactions when inoculated with races 0, 1, 23 and 23N and reacted unlike the known *Ht* gene expressions. Seedling inoculation was useful to identify chlorotic resistance while adult plants were

useful in assessing rate-reducing resistance (Adipala *et al.* 1993b). Levy (1991) showed that isolates from different areas were different in parasitic fitness as was indicated by infection efficiency, sporulation and lesion size, while isolates from the same location showed less variation.

MATERIALS AND METHODS

ISOLATION OF *E. turcicum* AND CULTURE PROPAGATION

Diseased leaves were collected from the field sections cut, washed in distilled water and pieces of 1 cm square containing lesions placed in a beaker half full with sterile distilled water and rinsed. They were picked with forceps and placed in a plate with 0.5% sodium hypochlorite for 1 to 2 minutes and placed between two sterile filter papers to remove excess bleach. The surface disinfected diseased pieces were placed in a petri dish with sterile water pre-moistened filter paper and incubated for 36 hrs.

The sporulating leaf pieces were placed in a test tube containing 5 ml of sterile water and shaken to dislodge the spores. Serial dilutions were made and the final dilution of 10^{-6} used for single spore isolation. One ml of the suspension was pipetted into a clear plate of nutrient agar (Oxoid) and spread evenly. Excess water was sucked out with a sterile glass dropper. Single spores were picked using an alcohol flame sterilized inoculating needle under a dissecting microscope. The single spore was placed in a plate of potato dextrose agar (DIFCO) and incubated at 22^{0} C.

Small blocks of 5 mm square of freshly growing culture were then cut and placed at the center of new plates of potato dextrose agar. The cultures were replicated three times for each isolate and randomized on the incubating bench. Colony growth development was monitored by measuring colony diameter at 2 day intervals for 7 days or longer. A line was marked under the plate as a base for diameter determination. The plates were replicated three times. A total of 45 isolates were established. The cultures prepared for inoculations were grown for 7 to 9 days and then harvested.

MAIZE DIFFERENTIAL SEEDLING INOCULATION AND EVALUATIONS OF REACTIONS

The isolates cultures were grown in petri plates for 6 to 7 days then used for inocultion. The plates were moistened with a few drops of sterile water using a sterile pipette. The conidia were dislodged from the surface of the colonies with a microscope slide and then drained into a beaker with 10 ml of sterile distilled water. The suspension was filtered through double folded cheese cloth and the concentration adjusted to 2.5 x 10^4 spores/ml by use of a hemocytometer. Two to three drops of Tween 20 per liter was added to the inoculum as suggested by Warren (1975). A mixture of sterilized forest loam, peat and gravel in the ratio 4:2:1 was used to grow the test plants in the greenhouse at the National Plant Quarantine station at Muguga near Nairobi. Five seeds per line were planted and then thinned to three plants per pot. The treatment was replicated three times. The seedlings were inoculated eight days after planting using a small hand held sprayer. Inoculum was sprayed to runoff in the greenhouse, the seedlings were placed on a bench covered with polythene sheets dampened with sterile distilled water for 48 hours. The differentials used were A619, A619*Ht1*, A619*Ht2*, A619*Ht3* kindly provided by Dr. Pedersen (Uni. IL. Urbana, IL. 61801), and B73*Ht1*, provided by Asgrow seeds, B37*Ht2*, B37*Ht3* and B14*HtN* were obtained from Dr Pataky (Uni. IL. Urbana-Champain 61801). Illinois Foundation seeds provided B73Ht1rhm and B73Ht1. Plants were evaluated for disease severity 15 days after inoculation using a 0 to 5 scale. The reactions were examined and classified as resistant or susceptible based on lesion types (Hooker 1961, 1963ab). The lesions were examined and sporulating lesions without evidence of chlorosis were taken as susceptible types and non sporulating lesions bearing chlorosis were the resistant types. Length and width of selected lesions was determined at the time of symptom evaluation. The inoculation work was done in the research green houses at the Kenya Agricultural Research Institute (KARI) National Plant Quarantine Station at Muguga near Nairobi. This was because the maize seeds were imported from the United States and could only be tested at the National Plant Quarantine Station. Maize is a quarantine crop in Kenya.

RESULTS

The forty-five isolates evaluated for growth profiles showed variation in rate of growth from the initial inoculation point to the maximum growth of 9 cm. Eleven isolates reached maximum growth after 11 days, 23 isolates after 18 days and 6 isolates after 34 days. The other four isolates reached maturity from 12 to 15 days. The colonies were initially whitish with a greenish tint, becoming brownish to black after 7 days due to abundant sporulation (Figure 2). The growth profiles are provided in Appendix B.

Nineteen isolates were used to inoculate the American differential inbred lines carrying the Ht gene and their reactions were recorded. The Anova for the lesions areas determined after inoculation indicated significant differences among the differentials inoculated at P<.0001 (Appendix A, Table A17). The mean length, width and standard deviations of five lesions per inbred line for eight isolates inoculated on five differentials showed that the inbred lines exhibited variation in their responses as revealed by different lesion dimensions (Figure 1and Table 3).

All plants exhibited small pinhead size spots 48 hrs after inoculation. Resistant plants exhibited chlorotic spots. The small spots developed into typical lesions on susceptible cultivars. Lesions could be distinguished 5 days after inoculation. Resistant reactions were yellowish chlorotic lesions while susceptible lesions were elliptical gray necrotic lesions and sporulating. The inbred lines with many small chlorotic lesions and two or three necrotic ones were considered as moderately resistant. Those with a few chlorotic lesions and more than three necrotic lesions were considered as moderately susceptible and both classes were rated 2 for disease severity. Those with many water soaked necrotic lesions were considered susceptible. The lesions on susceptible plants were either water soaked or had dark margins with profuse sporulation. The types of reactions observed and lesion length and width respectively are shown in Tables 1, 2, and Figures 3, 4, 5 and, Appendix C.

Three isolates from Kitsoeni Chonyi of Coast province, Werugha in Taita Taveta district and Bahati in Nakuru district of the Rift Valley Province, respectively, were; race 0, with the virulence formula *Ht1, Ht2, Ht3, HtN*/. Three were race 12, one from Kisii, one from Runyenjes in Kirinyaga district and the other from Kiptabus in the Uashin Gishu district of the Rift Valley with the virulence formula, *HtN, Ht3 / Ht1, Ht2*. Three

were race 1, one was race 2 , 1 was race 3 and eight isolates did not fit any known races and were designated k1, k2, and k3. Those classified as k1 closely resembled race 4 but were virulent on *Ht1* gene (Tables 1 and 2). The classification system by Leonard *et al*. (1989) was not inclusive and did not satisfy the naming of all the isolates. Thus we relied on the old classification for some of the races (Turner and Johnson 1980). The two isolates designated k1 series had the following virulence formula: /*Ht1, Ht2, Ht3, HtN*, meaning all differentials were susceptible. Five isolates had the formula *HtN, / Ht1, Ht2, Ht3* and were designated k2 isolates. One had the formula *Ht1, Ht2, Ht3 / HtN*, and was designated k3 (Table 1 and 2). The k3 designated isolate from Kapsoit in Kericho District exhibited a severe chlorotic reaction with B73*Ht1* and was rated 3 for disease severity, but was considered a resistant type (Table 1). The eight isolates designated k were from wide geographical areas in Kenya. Three isolates were not tested for *HtN* reaction as the remaining seeds with *HtN* failed to germinate. All resistant reactions exhibited chlorosis and did not produce spores while susceptible reactions were necrotic lesions with heavy evidence of sporulation. The reactions and race classification are illustrated in Tables 1 and 2. Lesions types observed with the inoculated isolates are shown in Figures 3, 4, 5 and and Appendix C. Figure 3 shows the susceptible reaction of inbred line A619. All Isolates caused susceptible reactions on inbred line A619 which were large water soaked lesions that eventually lead to the withering of the leaves. Resistant reactions were always chlorotic spots without visible necrosis or with a very limited necrotic center. The necrotic lesions had extensive sporulation while the chlorotic reactions had no sporulation (Figures 3, 4, 5 and Appendix C).

DISCUSSION

The isolates did not exhibit appreciable variation in appearance or in morphology of the colony. Pedersen and Brandenburg (1986) did not observe any differences in incubation period between isolates, but there was variation in colony diameters between isolates from different locations. Colony radial growth seemed to be influenced by variation in temperature where some isolates had more radial growth than others at 20°C or 28°C. Kenyan isolates showed variation in growth rates between isolates from different zones (Figure 2 and Appendix B). Plant pathologists have not yet agreed on the exact amount of inoculum concentration to be used for inoculation or even the method of inoculum delivery to the test plants. This situation can be explained by many factors like different environments and the pathogens in the experiments. Reuveni *et al*. (1993) reported a rapid new technique of delivering by air pressure a conidial concentration of 100,000 and 200,000 per square centimeter of leaf surface followed by a period of wetness avoiding the masking of susceptibility to *E. turcicum.* Adipala *et al.* (1993) in Uganda used a concentration of 20,000 spores per ml. Leath and Pedersen (1986) found it impossible to accurately assess lesion number due to extreme chlorosis associated with *Ht* genes in line Pa11. In the Kenyan experiment, those lines with acute chlorosis exhibited multitudes of small chlorotic lesions that could not be assessed easily for size or number. Only necrotic lesions were assessed for size. In Uganda, all 215 isolates collected all over the country and tested were of race 0 and hence were all avirulent to the *Ht* gene (Adipala *et al.*1993). This seems to support the hypothesis that the introduction of *Ht* gene in Uganda may have a tremendous impact in maize production but the situation may have changed. The Ugandan report also suggests probably that the *Ht* gene had not been incorporated in Ugandan maize for sufficient exposure to the dynamics of the pathogen host interaction by the time of the study. However, the situation may be very variable presently, or probably, variation was present in low quantities that escaped detection during the sampling. Leath (1984) in an artificially inoculated plot found severities of up to 58%. While the Kenyan isolates do not all seem to fit wholly in the Leonard *et al*. (1989) designations, it is noted that races 0, 1, 2, 3 and 12 occurs. The groups named k1, k2, and k3 may be related to race 23N or race 4 but differs slightly when viewed from the old classification and the new, which are being used variously

today. The studies in Uganda by Adipala *et al.* (1993a) found that *E. turcicum* occurred in all places sampled and was more severe in wet areas than in dry areas. However all the isolates tested were all virulent on A619 and avirulent on A619*Ht1*, A619*Ht2*, A619*Ht3* and A619*HtN*. Hence they were classified as race 0. These observations however, did not agree with those of Welz *et al*. (1993) in Uganda reported at the same time where they reported the presence of races 0, 2 and 23N. After evaluations of Ugandan maize germlasm, Adipala *et al.* (1993b) also reported that all lines had susceptible reactions when inoculated with races 0, 1, 23 and 23N and observed that the reactions had no resemblance to the known *Ht* gene expressions. Average disease severity ranged between 0.5 to 25% in Uganda. In the continental United States northern leaf blight has been effectively controlled by the use of the dominant *Ht* gene (Smith and Kinsey 1980, Turner and Johnson 1980; Hooker 1961). While the Kenyan Maize Improvement Program has done a tremendous job in producing maize varieties for the various ecological zones it is evident the aspect of disease control by genetic resistance must be allotted more attention. This is because all the recommended varieties of 600, 500 series, Coast composite, Pwani series and all land races etc. were found to be very susceptible to two or more races of the pathogens observed during sampling in the fields.

Two isolates from Kisii near the town of Keumbu, differed by their reaction on *Ht3*. One isolate was collected from a field planted with a tall 600 series hybrid and was virulent to *Ht1*, *Ht2* and *Ht3* while the other was collected from a field with weak appearing short maize variety near the same town and was weakly virulent on *Ht3* (Table 1). It was interesting to observe Mzihana, a land race of the coast in Kenya, was resistant to isolates from the highlands while susceptible to Kenya coast isolates mention elsewhere in this report. This tends to suggest that the races in the highlands may be very variable and different from those in the lowlands and may not have migrated to the coast. In the continental United States the disease has been effectively controlled by the use of the dominant *Ht* gene (Smith and Kinsey 1980; Turner and Johnson 1980; Hooker 1961) Smith and Kinsey (1980) suggested that a combination of all *Ht1* , *Ht2* and *Ht3* genes would provide resistance against race 1 , 2 and 3. Pataky (1994) showed that high levels of partial resistance with or without *Ht*-genes presented a spectacular approach in

reducing damage from NLB on sweet corn which also eliminates the severe yield depressing chlorosis associated with Ht gene resistance in very susceptible backgrounds.

Studies by Carson (1995b) indicated that latent period is related to partial resistance which suggested that selection for increased latent period length would be more beneficial than selecting for reduced disease severity. Selection for increased latent period length can be done in environments without severe disease epidemics, and also breeding material could be assessed as seedlings for latent period length in the greenhouse during the off season. Levy (1991) showed that isolates from different areas were variable in parasitic fitness as indicated by infection efficiency, sporulation and lesion size, while isolates of same location showed less variation. Inoculum in previous crop has been found to be critical in epidemic build up for subsequent cropping, especially in non-tillage systems as reported by Pedersen and Oldham (1992) using race 2. While non-tillage is not a common practice in Kenya, the heavy inoculum production means a lot of primary inoculum is available in subsequent plantings. Gevers (1975) reported that the *HtN* major gene of resistance derived from the Mexican maize cultivar *Pepitila* is reasonably stable. In some parts of the world, however, the effects may fail to be expressed and genetic segregation may not behave like expected of dominant gene ratios but does however remain in the tolerable limits of deviation of stability and segregation. He suggested the occurrence of biotypes in India that may able to overcome the *HtN* gene of resistance. The *HtN* gene is also affected by the other genes in the host genome and high temperatures reduced symptom expression on B37*Ht3*. Plants were evaluated at both 26 ^{o}C day/22 ^{o}C night and 22^{o} C day/18^{o} C night temperatures. There was observable weakened virulence at high temperatures. Another isolate from Hawaii was found to cause disease on B37*HtN*, Oh45*HtN*, B14A*HtN* and B68*HtN* and was designated race 2N with a virulent formula *Ht1,Ht3/Ht2,HtN* (Windes and Pedersen 1990). Combining *Ht1* and *Ht3* genes did not result in significantly less disease from those homozygous for each (Leath and Pedersen 1986). It appears that in Kenya the elite hybrids in the maize belt of the western region were of low resistance backgrounds to all the pathogens as evidenced by extensive chrolosis. Dunn and Namm (1980) reported gene dosage effects for *Ht2* gene. Smith and Kinsey (1980) suggested that a combination of *Ht* and *Ht2* or *Ht3* would confer resistance against race 1, 2 and 3. The inoculation

results indicate that *Ht* gene would be very important in Kenya for the management of *E. turcicum* thus there is need for more research on racial specialization and frequencies of occurrence in the various ecological zones (Figures1, 3 and appendix C).

In conclusion it is recommended that plant pathologists have a more near central role in maize breeding by participating in the initial planning and execution of material testing at the initial stages in Kenya. Maize breeding in Kenya must incorporate vigorous field and greenhouse testing of all lines to determine levels of resistance to *E. turcicum*. On the other hand, if major genes were incorporated into the elite hybrids, a program may include studies of pathogen variation to gauge the strength and sustainability of the deployed genes for resistance against *E. turcicum* for each ecological zone. While the *Ht* gene has been beneficial in many maize-growing countries, we note that there are some areas in Kenya which may possibly have evolved or are evolving races that would overcome all the *Ht* genes of resistance i.e. Kericho, Kilifi, Kirinyaga, Uashin Gishu, West Pokot, Kisii, Embu, Nakuru and Keiyo (Table 1 and 2). Further investigations are recommended to completely map the status of *E. turcicum* specialization in each ecological zone.

LITERATURE CITED

Adipala, E., E. P. Lipps and L.V. Madden, 1993a. Occurrence of *Exserohilum turcicum* on maize in Uganda. Plant Dis. 77:202-205.

Adipala, E., E. P. Lipps and L.V. Madden. 1993b. Reaction of maize cultivars from Uganda to *Exserohilum turcicum*. Phytopathology 83:217-223**.**

Berquist, R. R. and O. R. Masias. 1974. Physiologic specialization in *Trichometasphearia turcica f. sp. zea and T. turcica f. sp. sorghi* in Hawaii. Phytopathology 64:645-649.

Carson, M. L. 1995a. A new gene in maize conferring the a chlorotic halo reaction to infection by *Exserohilum turcicum.* Plant Dis. 79:717-720.

Carson, M. L. 1995b. Inheritance of latent period length in maize infected with *Exserohilum turcicum*. Plant Dis.79:581-585**.**

Drechsler, C. 1923. Some graminicolous species of *Helminthosporium.* I. J. Agri. Res. 24:641-739.

Dunn, G. M. and T. Namm. 1970. Gene dosage effects on monogenic resistance to northern corn leaf blight. Crop Sci. 10:352-354.

Gevers, H.O. 1975. A new major gene for resistance to *Helminthosporium turcicum* leaf blight of maize. Plant Dis. Rep. 59:296-299.

Hooker, A. L. 1961. A new type of resistance in corn to *Helminthosporium turcicum.* Plant Dis. Reptr. 45:780-781.

Hooker A. L. 1963a. Inheritance of chlorotic lesion resistance to *Helminthosporium turcicum* in seedling corn. Phytopathology. 53:660-662.

Hooker, A. L. 1963b. Monogenic resistance in Zea mays L.to *Helminthosporium turcicum*. Crop Sci.3:381-383

Hooker, A. L. and J. L. Perkins, 1980. *Helminthosporium* leaf blights of corn- the state of the art . Proc. Ann. Corn sorghum res. Conf. 35:68-87.

Jordan E. G, J. M. Perkins, R. A. Schall, and W. L. Pedersen. 1983. Occurrence of race 2 of *Exserohilum turcicum* on corn in the central United States**.** Plant Dis. 67:1163-1165.

Leath, S. 1984. Qualitative resistance to *Exserohilum turcicum* race 2 in maize. PhD. Thesis University of Illinois, Urbana. 88pg**.**

Leath, S. and W. L. Pedersen. 1986, Effects of the Ht1 and /or Ht3 genes in three maize inbreds on quantitative resistance to *Exserohilum turcicum* race 2 . Plant Dis. 70:529-531.

Leonard. K.J., Y. Levy and D. R. Smith. 1989. Proposed nomenclature for pathogenic races of *Exserohilum turcicum*.. Plant. Dis.79:776-777.

Levy, Y, and Y. Cohen. 1983. Biotic and environmental factors affecting infection of sweet corn with *Exserohilum turcicum*. Phytopathology 73:722-725.

Levy, Y. 1989. Analysis of eoidemics of northern leaf blight on sweet corn in Israel. Phytopathology 79:1243-1245.

Levy, Y. 1991. Variation in fitness among field isolates of *Exserohilum turcicum* in Israel. Plant Dis. 75:163-166.

Lipps. P. E. and R. E. Hite. 1982. *Exserohilum turcicum* virulent on corn with the *Ht* resistant gene in Ohio. Plant Dis. 66:397-398.

Manwiller, A. 1983. Maize genetics. In Kenya Agricultural Research Institute. Record of Research. Annual Report. pg 56-58.

Njuguna, J. G. M., J. C. Kedera, L. Murrithi, S. Songa and B. Odhiambo. 1992. Overview of maize diseases in Kenya. IN KARI, Proceedings of a workshop, Review of the National Maize Research Program, KARI/ISNAR Management Training Linkage Project. 1990.100pg.

Pataky, J. K. , Perkins, J. M., and S. Leath. 1986. Effects of qualitative and quantitave resistance on the development and spread of northern leaf blight of maize caused by E*xserohilum turcicum* races 1 and 2. Phytopathology 76:1349-1352.

Pataky, J. K. 1994. Effects of races 0 and 1 of *Exserohilum turcicum* on sweet corn hybrids differring for Ht- and partial resistance to northern leaf blight. Plant Dis. 78:1189-1193.

Pedersen, W. L. and M. G. Oldham. 1992. Effect of three tillage practices on development of northern corn leaf blight *Exserohilum turcicum* under continuous corn. Phytopathology 76:1161-1164.

Pedersen, W. L. and L.J. Brandeburg, 1986. Mating types, virulence and cultural charateristics of *Exerohilum turcicum* race 2. Plant Dis. 70:290-292.

Pratt, R.C., E. Adipala and P. E. Lipps, 1993. Characterization of race-specific resistance to *Exserohilum turcicum* races 0 and 1 in maize OhS0 S1 progenies. Plant Dis. 77:1227-1232.

Reuven, R., A. Bar-zur, and M. Shimoni. 1993. A rapid detection procedure for the HtN gene under controlled inoculation of maize with *Exserohilum turcicum*. Plant. Dis 77:580-582.

Shurtleff, M. C. 1973. Compendium of corn diseases. American Phytopathological Society. 105pg.

Smith, D. R. and J.G. Kinsey. 1980. Further pyhsiologic specialization in *Helminthosporium turcicum***.** Plant Dis. 64:779-781.

Thakur, R. P., Leonard K. J. and R. K. Jones. 1989. Characterization of a new race of *Exserohilum turcicum* virulent on corn with resistance gene HtN. Plant Dis. 73:151-155.

Turner, M. T. and E. R. Johnson, 1980. Race of *Helminthosporium* not controlled by Ht genetic resistance in corn in the American corn belt. Plant Dis. 64:216-217.

Ullustrup, A. J. 1970. A comparison of monogenic and polygenic resistance to *H. turcicum* in corn. Phytopathology. 60:1597-1599.

Warren H. L. 1975. Temperature effects on lesion development and sporulation after infection by races O and T of *B. maydis.* Phytopathology. 65:623-626.

Welz H. G., R. Wagner, and H. H. Geiger, 1993. Virulence in]*Setosphearia turcica* populations collected from maize in China, Mexico, Uganda, and Zambia. Phytopathology 83:1356.

Windes J. M. and W. L. Pedersen . 1990. An isolate of *Exserohilum turcicum* virulent on maize inbreds with resistance gene *HtN.* Plant Dis. 75:430.