CHAPTER 2

OCCURRENCE OF NORTHERN LEAF BLIGHT, *Phaeosphaeria LEAF SPOT* , SOUTHERN LEAF BLIGHT, RUST AND MAIZE STREAK VIRUS IN KENYA.

ABSTRACT

A survey of maize diseases was conducted in the 1995/96 seasons in Kenya where 65 farms were sampled representing all ecological zones. *Exserohilum turcicum (Pass.)* Leonard and Suggs the causal agent of northern leaf blight, *Phaeosphaeria maydis* (P. Hennings) Rane, Payak and Renfro (leaf spot*), Bipolaris maydis (*Nisik.) *Shoemaker (*southern leaf blight) *Puccinia polysora Underw.(*lowland rust*),* and *Puccinia sorghi* Schwz. (*common rust*) and maize streak virus were found to be the most important pathogens in Kenya. Disease estimation of 0 to 5 scale was used to estimate percent leaf area diseased (PLAD). Each scale unit was assigned a specific PLAD range for each disease. A total of 28 plants were examined per site of sampling. *E. turcicum,* characterized by lesions of up to 400 mm long by 25 mm in width, was observed in all areas. Severities of over 45% for northern leaf blight was recorded. Southern leaf blight was more serious in the highlands west of the Rift Valley where it occurred alone or with *Phaeosphaeria maydis* leaf spot producing spectacular epiphytotics. Severities of over 75% were recorded for southern leaf blight and *P. maydis* leaf spot in Western Kenya and over 85% for *P. maydis* leaf spot in Central Province. *P. sorghi induced* rust was very important especially in the areas around Molo, Njoro and Bahati in Nakuru of the Rift Valley Province with severity levels of over 65%. *Puccinia polysora* rust was important in the coastal areas reaching over 75% in Kwale district but trace amounts were identified in Kirinyaga District. Maize streak virus was important in the Lake Victoria region, Western, Central and Coastal regions. Serious incidences were observed where Napier grass (*Pennisetum pupureum* L.) was grown near maize plots. This is the first time these data set have been provided for Kenya.

INTRODUCTION

In order to develop rational and economical crop disease control measures, either by breeding resistant cultivars, or application of fungicides, it is not sufficient to state that a specific disease cause losses. This has been the case in Kenya. Ideally, the magnitude of the loss must be evaluated so that it can be related to economic gains. Only by disease loss appraisal is it possible to determine the economical loss due to different amounts of disease. Crop disease appraisal represents a basic essential step facilitating loss determination, as the pivot to articulate and implement management schemes aimed at economic control. Field disease assessment is the only way of determining the amounts and variation in distribution of diseases and pathogens in crops and the significance of the results and conclusions have far reaching effects (James 1968). There are no standard protocol for disease assessment reporting, although many forums have been set to look for practical efficient and accurate assessment of disease severity.

A globally accepted standard method would encourage data comparison through exchanges, improvement of communication and interpretations of results between professionals and ultimately to the consumer the farmer (Watson *et al.* 1990). The lack of reliable data to define the importance of diseases in World agriculture may well have retarded the progress of plant pathology as much as any other single factor. Losses due to disease are substantially higher in developing countries and unfortunately more severe in countries that can least afford them. Assessment of disease presents the initial data critical in plant protection programs. In order to have and sustain sound planning or management of plant pathology investment, we need estimates of crop losses. Priorities in resource allocations must be established during planning stages (James 1968).

The Food and Agricultural Organization (FAO) of the United Nations has developed publications offering guidelines on disease assessment methods (Botrel 1979). Despite the need for a standard, there has not been a universally accepted formula for disease measurement. Disease assessment generates a large data base which is expensive to collect and therefore should be fully interpreted and feedback given to the farming community (James 1968). This project was undertaken to document for the first time in Kenya the occurrence of northern leaf blight, *Phaeosphaeria* leaf spot, southern leaf blight, rust and maize streak virus in Kenya. This is the first comprehensive survey in

Kenya to provide the essential statistics of disease incidence, severity and distribution for maize.

LITERATURE REVIEW

A commonly used system for disease loss assessment, that of Horsfall and Barratt (HB) (1945) is rooted in the Weber-Fechner law which states that the eye distinguishes according to the logarithm of the light intensity. Therefore, the grading was to be based on equal ability to distinguish but not on equal disease. Below 50% the eye sees the amount of diseased tissue and above 50% it sees the amount of disease free tissue. They proposed a 12-category scheme of disease assessment from 0 to 100%. The underlying assumption in the HB classification has been challenged because not all estimates that depend on visual perception would obey the Weber-Fechner law (Herbert 1982).

In his study Slopeck (1989) used a 1-5 scale containing ranges from 0 to 100 as percent leaf area diseased (PLAD) categories and noted that visual assessments in some situations may be valuable. Field disease measurements in varietal trial and general production can help estimate reductions in yield and quality (Large 1965). Duveiller (1994) developed pictorial disease assessment keys for bacterial leaf streak determined by measuring diseased sample of leaves in cereals and corresponding to 1, 5, 10, 25, 50, and 75% diseased area. In Uganda, Adipala *et al*.(1993) used a (zero) 0, 0.5, 1, 5, 10, 25, 50, and >75% severity scale for evaluation of maize northern leaf blight where 20 plants were sampled along geographical cardinal points. Farms were selected and a site chosen where 5 plants each to the North, South, East and West were visually examined and assessed for disease severity. Nicholson and Warren (1975) rated disease severity or the extent of tissue coverage by lesions on a scale of 0 to 5. Elliot and Jenkins (1946) assessed *H. turcicum* severity on a 0 to 5 scale as follows: 0.5= very slight infection, one or two restricted lesions on the lower leaves, 1= slight infection, a few scattered lesions on the lower leaves, 2= light infection, moderate number of lesions on lower leaves, 3= moderate infection, abundant lesions on lower leaves and few on middle leaves, 4= heavy infection, lesions abundant on all leaves, and extending to upper leaves, 5= very heavy infection, lesions abundant on all leaves, plants may be prematurely killed.

Pataky (1992), in a study of resistant and susceptible sweet corn used a rating scale of 2% to 90% generated from a computer program DISTRAN developed by Tomerlin and Howell (1988). The evaluation was based on the primary ear leaf and top and bottom leaves which accounted for 33 to 40% of the total leaf area in sweet corn.

Susceptible corn consistently showed yield losses unlike those with *Ht* genes of resistance. Pedersen *et al.*(1986) using a 1-9 scale adopted from Perkins and Hooker (1981) evaluated levels of resistance to *E. turcicum* by inoculating inbred lines B37, B37*Ht1*, B37*Ht2* and Oh43*Ht3* in the greenhouse and in plots. They found some lines classified resistant in the greenhouse were susceptible in different locations, probably due to temperature, relative humidity, and light variations. Manwiller *et al*. (1985) rated maize at Muguga for reactions to rust and Northern maize blight but did not challenge the lines. Darrah and Mukuru (1977-1980) evaluated maize crosses for hybrid production for rust and northern leaf blight incidence on a 0-5 scale where 0-represented no symptoms and 5 very severely attacked based on natural field infection, without artificially inoculating the progenies in Kenya. During this period rust scores had a mean value of 3.07 while blight was even lower at 1.93. Scores of 3.8 and 3.3 were recorded for rust and northern leaf blight respectively, in Trans Mara region but the overall average was remarkably low and were even lower in earlier reports (Darrah 1974, Darrah *et al*. 1975, 1976). Nicholson and Warren (1975) used disease severity or the extent of tissue coverage by lesions rating on a scale of 0 to 5. O'Brien and Brugg (1992) used ten participants and three severity scales to test accuracy and precision for corky root of lettuce. One the scales which they called mature plant scale had seven levels from 0 to 6, the other scale known as seedling scale had ten levels from 0 to 9 and the third one, the H-B scale had 12 levels, 1=0%, 2=0-3%, 3=3-6%, 4=6-12%, 5=12-25%, 6=25-50%, 7=50-75%, 8=75-87%, 9=87-94%, 10=94-97%, 11=97-100%, 12=100%. They found that no scale was better than the other in each situation but the scale for mature plants was more precise and accurate. Saghaai Maroof *et al* (1993) used seven inbreds to evaluate gray leaf spot in Virginia. They used two methods namely; disease index and disease severity. The disease index was a scale of 1-5 where 1=no symptoms; 2=moderate lesion development below the leaf subtending the ear; 3=heavy lesion development on and below the leaf subtending the ear and a few lesion above it; 4=severe lesion development on all but the uppermost leaves, which may have a few lesions; and 5= all leaves dead. They expressed disease severity as diseased leaf area divided by total leaf area multiplied by 100 assessed on the ear-1 leaf and all leaves above it. This study found that the disease index is a better method for general resistance screening of

germplasm with multiple ratings. Gaunt (1995) proposes that remote sensing, imaging and positioning hardware and software have provided new technologies for assessing disease severity and for sampling. Nutter and Forrest (1995) reported that use of computerized disease assessment training programs such as Disease. Pro, can greatly enhance accuracy and precision of visual disease assessments.

MATERIALS AND METHODS

Farms were selected with the assistance from the office of District Agricultural Officers who also provided an officer to aid in the purpose. The maize crop was examined during the post milk stage equivalent to growth stage 8-9 as illustrated by Hanway (1979) when all leaves were green. The farms were selected randomly per district and evaluated thoroughly for disease occurrence and severity as a whole and rated whether present or absent with a binomial 1 for presence and 0 for absence. Disease severity at a particular farm was then rated on a 0 to 5 modified 0-5 scale reported by Slopeck (1989). The disease assessment was done in respect to northern leaf blight (*E. turcicum), P. maydis* leaf spot , southern leaf blight (*B. maydis),* lowland rust (*P. polysora),* and common rust (*P. sorghi).* Where particular disease incidences were very low, the number 1 was assigned. When the appraisal within a farm was complete, a random site inside the farm was selected and an individual plant marked as the starting point for detailed examination. Seven plants were examined to the North, South, West and East resulting in a total of 28 plants per site, eight plants more per site than reported by Adipala (1993) in Uganda. The distance between plants ranged from 0.75 to 4 meters. The plot sizes ranged from small peasant plots of about 0.5 hectares in Central Kenya to over 50 hectares in the highlands. Detailed examination and rating was done on the plants for rust, northern leaf blight, *P. maydis* leaf spot and southern leaf blight as follows; the total number of leaves per plant, the number of leaves with lesions of *E. turcicum*, total number of lesions of *E. turcicum* per plant, total number of leaves with *Puccinia sorghi*, or *P. polysora* for the coastal region, number of leaves with *B. maydis* - induced lesions and the total number of leaves with *P. maydis* infection and then severity rating for each disease was recorded per plant. All diseases were evaluated on each plant or as they occurred per site. The disease estimation was based on a modified 0 to 5 scale reported by Slopeck (1989) and was used to estimate percent leaf area diseased (PLAD). Each scale was assigned a specific PLAD range for each disease as indicated by lesion size, intensity and distribution on an individual plant.

E. turcicum blight severity rating was done as follows; 0 = no disease - No lesions identifiable on any of the leaves, 1 = 0.5 to 1.0 % of leaf surface diseased – (a few restricted lesions on a few leaves); 2 = 5 to 10 % of leaf area diseased – (several small or

big lesions on many leaves); 3 = 10 to 15 % of leaf surface diseased – (numerous small and large lesions on many leaves); 4 = 20 - 35 % of leaf surface diseased – (many large and coalesced lesions on many leaves) and 5 = 45-75 % of leaf surface diseased ;representing multitudes of coalesced lesions resulting in leaf wilting and tearing and blotching. The lesions were long elliptical brownish necrotic zones more or less aligned to the main axis of the leaf. Individual lesions were more or less oblong shaped.

P. maydis leaf spot severity was rated as follows: 0 - no disease ; 1 = 0.1 to 0.5 % of leaf surface diseased – (a few lesions scattered on the leaf surfaces); 2 = 5 to 10 % of leaf surface diseased – (numerous lesions on the leaf surfaces); 3 = 30 to 45 % of leaf surface diseased – (many lesions on the leave surfaces); 4 = 50 to 65 % of leaf surface diseased – (many lesions on the leaf surfaces with some coalescing); 5 = over 75 % of leaf surface diseased – (many lesions on the leaf lamina with many coalesced leading to leaf wilting or blighted areas). The lesions were whitish necrotic spots with dark brown borders.

P. polysora and *P. sorghi* rust severity was rated on a modified method reported by Groth (1992): 0 - no disease –No rust pustules seen. 1 = 0.5 to 1 % of leaf surface diseased – (a few non erumpent pustules scattered on the leaf surface); 2 = 10 to 15 % of leaf surface diseased –(numerous pustules on the leaf surfaces some erumpent); 3 = 30 to 40% of leaf surface diseased – (many erumpent pustules over the leaf surfaces); 4 = 45 to 65 % of leaf surface diseased- (many erumpent pustules surrounded with huge blighted and sometimes rusty chlorotic zones). 5 = over 75 % of leaf surface diseased – (many huge dry pustules surrounded by dead rusty wilted and blighted areas on the leaves). Rust pustules were small rusty spots surrounded by a yellowish halo with or without torn leaf cuticle.

Southern leaf blight was like wise assessed as follows; 0 = No disease -No lesions observed; 1 = 0.1 to 0.5 % of leaf surface diseased- (only a few longitudinal lesions with or without chlorotic margins), 2 = 5 to 10 % of leaf surface diseased – (several lesions on the leave surface); 3 = 15 to 30 % of leaf surface diseased – (many longitudinal lesions on the leaf lamina), 4 = 45 to 55 % of leaf surface disease – (many lesions with some coalescing); 5 = over 75 % of leaf surface disease- (many coalesced lesions on the leaf lamina with some wilted zones). The spots were small, elliptical, brownish, necrotic

lesions between and parallel to the leaf veins and were about 15mm long and 2mm in width. Severity of maize streak virus, headsmut, and any other disease was assessed on the frequency of diseased plants observed in a particular field. Data for these two diseases are only entered as occurrence and severity.

STATISTICAL ANALYSIS

Summary statistics of incidence and severity are presented for each disease in as figures and tables in the results and appendix. Random sampling of fields have been used elsewhere (Levy 1989). The General linear model procedure (SAS, Institute, Cary, NC) was used for analysis of variance of all data. Arcsine transformation was done on percentages and square root transformations were performed for leaf and lesion counts. Tests for significance were done at $p<.0001$ for ANOVA. Means were grouped using Tukeys Stundentized Range (HSD) at alpha =0.05 and generated from the GLM model. The means are presented in descending order of magnitude. Disease severity analysis is presented to show the Min or minimum rating per sampling point, Q1 or first quartile, Me or median, Q3 or third quartile, Max or maximum rating, the Std or standard deviation, the mean and the rating for the entire farm per location.

RESULTS

Maize plants were examined in the post milk stage before senescence. The map of Kenya is presented indicating the districts which were visited within the country as a whole (Figure 1). These districts consist the important maize growing areas of Kenya. It is important to note that these areas are well served with all weather roads but access roads are very poor being either bare earth or gravel surfaces which tended to limit how far one can penetrate into the farm lands. We observed very severe maize disease status in Kenya with epiphytotics of one or two of the four common diseases in all maize producing areas. The diseases encountered were those incited by *E. turcicum*, *P. maydis*, *B. maydis, P. polysora* and *P. sorghi*. Table 1 presents disease occurrence and severity as observed per site visited. Table 1 uses a binomial classification as 1 for presence and 0 for absence followed and a severity rating for each disease. The data sets are presented sequentially as the information was gathered in the field. The proportion of diseased leaves is presented as percentages in descending order as an estimate of the disease pressure per area. By analyzing the fraction of percentage number of leaves diseased one can deduce quickly which disease was important in what location or the probable disease combinations in each area (Tables 3, 5, 7, 9).

These tables show the percentage diseased leaves, means comparisons for the sampling points. Analysis of variance (ANOVA) for each disease is also provided and indicated significant differences among locations at $P<0.0001$ (Tables 2, 4, 6, and 8). Analysis of variance for total number of leaves and diseased leaves indicated significant differences among locations (Appendix A). The comparison of the means for total number of leaves and diseased leaves for each disease showed significant differences among locations (Appendix A). Figures 2, 3, 4 and 5 provide comparisons for total and diseased number of leaves for the four diseases. While the highest number of leaves for individual plants were recorded in Taita Taveta (Table 14), Kilifi had the most diseased leaves by *E. turcicum (*Table 14). West Pokot District showed the highest mean of diseased leaves by *P. maydis* (Table 15). Keiyo District had the highest mean for leaves infected with both *P. sorghi* and *B. maydis (*Figure 16 and 17*).*

Kilifi District had the highest percentage of infected leaves by *E. turcicum* followed by Kakamega and Thika, respectively, while Nakuru District at Njoro had the

lowest. It is noted that Mtwapa and Keumbu had no significant differences despite the fact that the two places are over 600 miles apart. Mtwapa is at the sea shore of the Indian Ocean while Keumbu is in the western Kenya in the Gusii highlands near Lake Victoria. It can also be observed that the chief maize producing areas of the western highlands have comparatively high disease pressure (Table 3).

Table 10 is a severity rating presentation for *E. turcicum* showing the minimum, 25th percentile, median, the mean, the 75th percentile the maximum, the and the overall assessed severity value for the sampling farm. The severity data in general indicate that *E. turcicum* was a national problem. *E. turcicum* was less severe in Nakuru District at Njoro where rust was very severe.

From the lake Victoria basin, including Kisii, Siaya and Kakamega, to the sea coast encompassing Kwale, Taita Taveta and Kilifi regions, *E. turcicum* was seen as an important pathogen with levels of northern maize leaf blight of up to 45% (Tables 2 and 9). The foliar destruction was very severe considering the size of lesions which ranged around 250 mm long and 15 mm in width on leaves approximately 1000 mm in length and 100 mm in width. This suggests that to cover the entire leaf area, the pathogen would need to induce approximately five lesions. Lesions of over 400mm long and 25mm in width were recorded in Njoro. When *E. turcicum* lesions occurred at the base of the leaf they rendered the leaf functionally inoperative. The photo (Figure 8) taken in Trans Nzoia in the fertile Kenyan high potential highlands shows an example of a plant with leaves infected at the base almost isolating them from the rest of the plant. Northern maize leaf blight was distributed throughout Kenya with serious levels and foci around the Lake Victoria region, Kiminini in Trans Nzoia, West Pokot, Bahati in Nakuru and Taita Taveta. The lesion distribution appeared diffused throughout the plants from near top to bottom with a reducing gradient of severity upwards (Figures 6, 7, 8, 9, and 10).
P.maydis always infected the upper most leaves in the Western Rift highlands while *B. maydis* appeared to favor the shaded leaves. These pathogens tended to occur in combination which was a frequent phenomenon but very variable. In the region of the highlands west of the Rift valley, *P. maydis* was more severe on the mid and top leaves, showing a reverse severity gradient to that of *E. turcicum*, a scenario that was not seen in Central Kenya where *P. maydis* leaf spot occurred in a uniform pattern on an individual

plant from bottom to top (Figures 14, 15, and 16). *P. maydis* was observed as an important disease in the western and central Kenya with nine locations situated in west Pokot, Kirinyaga, Embu, Siaya, Muranga, Nyeri and Trans Nzoia exhibiting the highest disease pressure as observed in the comparisons of means (Table 5).

P. maydis leaf spot was found to be in epidemic proportions from the Lake Victoria region to Taita Taveta but was not observed in Kilifi or Kwale districts. The occurrence of *P. maydis* in Kirinyaga, Nyeri, Embu, West Pokot, Kericho, Keiyo, and Uashin Gishu Districts was severe. In these areas over 65 to 75% of the leaf surface was destroyed and it posed a serious threat to maize production. This high disease severity means that each individual leaf per plant was covered by densely packed lesions. In the majority of farms examined in central Kenya, *P. maydis* was the dominant pathogen. In Nyeri, Embu, Kirinyaga and Muranga Districts 85% of the leaf tissue was destroyed. These districts have favorable environments that are conducive to disease development. The pathogen was also observed to be very competitive even seen thriving on already established lesions caused by other pathogens notably *B. maydis*.

Analysis of variance indicated significant difference between areas (Table 4). It is noted that West Pokot in the Rift Valley had the highest percent diseased leaves followed by Kirinyaga and Embu in the mid altitudes (Table 5). *P. maydis* was found in all areas except near the sea in the Mombasa and Kilifi areas and was very important in West pokot and Central Kenya in areas where *E. turcicum* occurred (Table 12). In West Pokot, *P. maydis* leaf spot was the most important disease followed by northern maize leaf blight in both the highly elevated Kapenguria area and the deep valley near Chapareria where the maize crop generally appeared yellowish. It was noted that the critical dry matter producing middle third portion of the canopy was severely infected.

Epiphytotics induced by *B. maydis* were observed in, Kakamega, Siaya, Keiyo, Uashin-Gishu and Trans Nzoia all very important maize producing areas. The disease occurrence was greater than 75% with a uniform cover from top to bottom (Table 7, Figures 17 and 18). Kakamega District exhibited the severest incidence of *B. maydis* with a minimum rating at 40% and maximum at >75%. In 1995 and 1996 all of the West Kenyan maize exporting areas, had at least two of the four diseases in epidemic proportions. These areas were observed for two seasons where one observation was

initiated to prepare the keys for assessment. It was noted that *B. maydis* was an important pathogen in the high rainfall zones situated generally in the western region of Kenya. Two locations in Kakamega District had the highest fraction of infected leaves (Table 7).

Southern maize leaf spot (*B. maydis)* which was found scattered throughout the maize growing areas of Kenya had serious incidences in Kwale, Kakamega, Kericho, Keiyo, Uashin Gishu and Trans Nzoia Districts (Table 11). While *B. maydis* was dispersed throughout the country we noted that the western areas exhibited the most severe disease pressure with the top ten locations with the highest means being located in Kakamega, Bungoma, Keiyo, Trans Nzoia, and Uashin Gishu (Table 7). Some *Bipolaris carbonum* infection was seen in Kapenguria and Kakamega but not serious as other diseases.

In Laikipia, and Nyandarua Districts, all diseases were serious even on young plants. Around Njoro area towards Mau, and in the vicinity of Egerton University, head smut was observed to be of high incidence. However, common rust incited by *P. sorghi* was the dominant disease in the Molo-Njoro region of Nakuru district and also in the fertile and high rainfall highland districts of Trans Nzoia, Uashin Gishu, and Keiyo (Table 9). Lowland rust (*P. polysora*) was very important in Kwale District where the highest percentage of infected leaves were recorded. Lowland rust was also observed severe at Mtwapa in Kilifi District occurring on sorghum. Rust was not important in mid altitude areas of central Kenya but occurred sparingly. The highlands to the West of Kenya had the highest rust incidence especially where the blights and leaf spots were less severe (Table 13).

While rust was the most important disease in Nakuru District, *P. maydis* leaf spot was also very severe in Molo reaching over 65% severity (Figures 11 and 12). *P. polysora* was mainly found in the coast on maize and sorghum although some traces were seen in Kirinyaga District (Figure 13). Lowland rust was observed as very important in Kwale, Nakuru, Nyandarua, Keiyo and Trans Nzoia Districts (Table 9). Charts of means of total number of leaves and number of infected leaves are provided for the four main diseases (Figures 2, 3, 4 and 5). Rust occurred as numerous pustules on the leaves resulting in extensive leaf blotching, curling and tearing, large areas appearing blighted and rusty brown from top to bottom (Figure 11). Table 13 shows the severity for rust in

twenty three locations. The table shows that rust was important in these locations with maximum incidences between 65 and 75 % in most of the sample points. The first column, which is identified as farm, describes the farm general severity assessment and the rest of the entries are for the evaluation point inside the farm.

Maize streak virus infection was observed in all areas but was severe in Siaya, Coast, Bahati area, and Embu and Central Kenya, especially in areas where Napier grass (*Pennisetum purpureum*) is cultivated for fodder near maize fields. In areas around Thika, Kiambu, Muguga maize streak was intense. This may be due to the fact that maize is found in all stages of growth providing an all year round ideal habitat for the leaf hopper vector (C*icadulina mbila).* This may be due also to the fact that the farmers have to use their small plots continuously and intensively for survival. Due to the land inheritance in some parts of Kenya some people own strips less than one acre. Plots observed in Thika District were surrounded with Napier grass and all plants exhibited symptoms of maize streak virus infection (Figures 19, 20, 21, and 22). Maize streak-virus like symptoms were observed in Napier grass near the infected plants. In all three growing seasons nearly 100% of maize plants exhibited symptoms of the maize streak virus and this pattern was widespread and was observed in other areas where Napier or another member of the *gramineae* was found thriving near or along the edges of the maize crop. In Embu district, seedlings as young as four leaf (GS 1) stage were infected and inevitably develop into sterile individuals (Figure 22).

DISCUSSION

Foliar diseases induced by *E. turcicum*, *P. maydis*, *B. maydis* and *Puccinia* spp were severe in Kenya and posed a great threat to maize production (Tables 3, 5, 7, 9, 10, 11, 12, 13 and Figures 2 to 22). At least two diseases were in epidemic proportion in the high production areas of Western Kenya (Tables 1, 10, 11, 12, and 13). ANOVA for percentage leaves diseased indicated significant differences between the various locations (Tables 2, 4, 6 and 8). The means comparisons for percentage leaf diseased show differences between locations but does highlight the fact that the fertile and high rainfall areas had more severe infections than the rest of the country (Tables 3, 5, 7 and 9).

E. turcicum, the northern leaf blight pathogen, had a nationwide distribution with disease severity levels reaching over 45% and higher in the wetter areas of the Kenyan grain producing highlands (Table 10). These are the areas that have high annual rainfall of about 1100 mm with high humidity and cool temperatures of between 11 to 27^0C that create ideal conditions for infection and dispersal of inoculum. Recent studies in Uganda by Adipala *et al*. (1993) found that *E. turcicum* occurred in all areas sampled and was more severe in wet areas than in dry areas. Reductions in yield are associated mainly with the amount of leaf tissue damaged in the upper two thirds of the plant canopy (Levy and Pataky (1992). Yield losses of about 18% were reported when severity of NLB ranged between 1% and 38% (Perkins and Pedersen (1987). The situation as observed in Kenya was serious because most of the infection occurred in mid and upper plant canopy where most yield depressing effect is known to occur (Levy and Pataky 1992).

P. maydis leaf spot a relatively unknown disease that has been reported in Brazil, India, Colombia, Ecuador and Mexico (De Leon 1984 was very serious with disease severity levels greater than 85% in extensive areas of Central, Rift valley, highland coast and Nyanza. The infected leaves were all heavily blotched and pale and diseased tissue almost covered the entire leaf lamina especially in the mid altitudes and the highlands (Figures, 14 and 16). *P. maydis* leaf spot was not important along the lowland coast. The disease incidence and severity levels observed in Kenya and recorded comprehensively for the first time here are alarming especially for *P. maydis*, previously a minor pathogen with little or no data in Kenya (Njuguna *et al.*1992). In Kenya, *P. maydis* emerged as a

serious pathogen requiring high priority research in all important maize production areas.

B. maydis, the causal agent of southern maize leaf blight, was serious in the highlands west of the Rift Valley where severity levels of over 75% were observed. Infected tissue was extensively covered with spots, and chlorosis rendering them non productive. *B. maydis* was found to have a higher saprophytic ability (Blanco and Nelson, 1972) and hence high primary inoculum levels will be likely to be found in areas with high disease occurrence. *B. maydis* shows little or no specificity to plant cytoplasms; it can infect a wide range of plant genotypes and cytoplasms giving access to invade the various varieties developed. It infects leaves, producing smaller lesions with parallel sides and little chlorosis. It tends to be limited by temperature and climate to the warmer part of the US (Hooker *et al*, 1970). Spore production is influenced by temperature and more lesions formed at 30^{o} C than at 15 or 22.5^{o} C (Warren 1975). The large lesions observed in Kenya are an indication of the host susceptibility and the prevailing physical conditions because lesion size increases in almost a straight line relationship with increasing dew periods and temperature (Nelson and Tung 1973).

Common rust caused by *P. sorghi* was serious in areas within the Rift Valley inducing heavy leaf rusty blotching and tearing. Once infection has been induced, the disease progresses as the plant develops. *P. sorghi* infects the leaves and sheath and the disease is severe on late planted maize. Seeds planted in a three bi-weekly regime had 4, 23 and 45% yield loss, respectively (Roduel Rodriguez *et al*. 1980). Epidemics resulting from uredospores as the primary source of inoculum can cause up to about 60% loss in yield. During the 1950-51 epidemic, high levels of southern rust in West Africa occurred (Rhind *et al*. 1952). Rusted maize leaves in Kenya appeared dirty brown, blotched and torn, occasionally with intense chlorosis. Maize lines with small pustules surrounded by chlorosis or necrosis were rated as resistant while well developed pustules were considered susceptible (Hulbert *et al*. 1991). Line B37R is resistant, being homozygous for *Rp9* (Roduel Rodriguez *et al*. 1980). The Kenyan maize, especially in the important growing areas of the Rift Valley did not show appreciable resistance to *P. sorghi* although there was observable extensive chrolosis in some areas.

Analysis of variance for percentage diseased leaves for all locations indicated that there were significant differences at $p<.0001$ (Tables 1, 3, 5, and 7). All means comparisons grouped with Tukey's Studentized Range (HSD) indicated that there were significant differences between areas examined at $\alpha = 0.05$ (Tables 3, 5, 7, and 9). All maize grown in E. Africa and tested in the 1950s had no resistant reaction, but those from Mexico had major gene resistance and an isolate of *P. polysora* collected in Kenya from maize cultivar with a B17 background was found to be virulent to *Rp1* gene (Storey and Ryland 1954). Resistant lines normally show small chlorotic or necrotic flecks with no sporulation. Highly resistant genotypes have smaller uredosori than moderately resistant or susceptible ones. Few uredosori rupture late in the resistant combinations (Subrahmanyam *et al.*1983). Disease develops rapidly in warm weather and uredospores comprise both primary and secondary inoculum (Bailey *et al.* 1987). The pathogen has the potential of being destructive even if infection comes after anthesis (Raid *et al.* 1988). Melching (1975) rated *P. polysora* as the most destructive of the rusts of maize. Severe losses can occur especially if infection occurs early (Zummo 1988).

The high rainfall areas of western Kenya had very severe levels of all the four diseases encountered. At least two diseases were in epidemic proportions. When the percentage of damage or severity is evaluated for each disease, we concluded that the maize disease situation in Kenya has not received the attention deserving for a national crop of first priority and therefore more research is urgently needed (Tables 10, 11,12 and 13). All four diseases were observed to cause damage in the critical canopy zones of the plants examined during this study in Kenya. Field assessments of disease severity is compounded by the fact that diseases do not occur singly in individual plants at any sample point.

Perhaps, maize varieties released to farmers were thought to carry a strong polygenic base for disease tolerance. However, evidence is deficient to show that intensive greenhouse inoculations were carried out during the earlier stages of varietal development to confirm genetic basis for the assumed resistance. The situation currently observed in fields throughout Kenya indicates that disease incidence and severity during the initial evaluations were chronic and underestimated. Hence, the mature maize in the absence of pathogens is essentially agronomically robust crop, but lacking the genetic

resistance to withstand infection. The end result is dangerous vulnerability under field disease pressure as was observed in Kenya. In addition to the underestimation's, and endemic nature of these pathogens in Kenya, the pioneer field disease evaluations were too narrow to be acceptable or applicable because pathogens do not occur in isolation.

Plant Pathologists should have a central role in maize breeding programs by participating in the initial planning and execution of future programs where germplasm would be exhaustively tested at the initial stages. Maize breeding in Kenya must incorporate vigorous field and green house testing of all cultivars to determine the kind and levels of resistance to the four most important pathogens. If major genes are incorporated into elite cultivars, the programs should study variation of pathogens to gauge the strength and sustainability of the deployed genes, especially for *Puccinia* spp., *E. turcicum* and *Bipolaris* spp which are known to express variability and races (Shurtleff 1973).

Nevertheless, the prevailing field disease status calls for immediate re-evaluations of germplasm and the addition of multiple genes of resistance. The variation and ecology of these pathogens need to be studied to determine aggressiveness of each. Kenyan scientists need to determine the yield losses associated with pathogens alone and in combinations through on-farm based research. To increase production of maize we have to rely on crop improvement rather than area expansion (Karanja and Oketch 1992). Kenya policy makers must develop strategies in agricultural extension to advise farmers on maize diseases and demonstrate on-site disease effect on yield. There is a need to educate farmers so they can associate signs of pathogens and symptoms of disease on maize and demonstrate that intervention is beneficial.

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