CHAPTER 1

INTRODUCTION

Maize (*Zea mays* L.) is the most important food crop in Kenya with a national production of 2.4 million tons in a total area of 1.6 million hectares (Gebrekidan *et al*. 1992). Shortage of maize in Kenya always results in famine among the poor urban and rural people. Among the biotic stresses that are a constraint to production are diseases and pests. In the review of the National Maize Research Program, Gebrekidan *et al*. (1992) highlighted *Exserohilum turcicum (Pass.)*Leonard and Suggs, *Pucinia sorghi Schw*.**,** *Bipolaris maydis* (Nisikado)Shoemaker, and *Puccinia polysora* Underw. as the main pathogens among other numerous important abiotic and biotic constraints. Lack of sufficient moisture is the chief constraint among the abiotic category. The economy of Kenya is based on agriculture and losses due to plant pathogens on the staple crop maize may be enormous. Since almost all the arable land is under cultivation in Kenya, future increases in maize production will heavily depend on yield improvement rather than expansion in area (Karanja and Oketch 1992**).** A comprehensive approach must then be undertaken and disease control given priority. Over 90% of the crop diseases are reported in research and extension annual reports but very little is known about disease incidence and severity, pathogen distribution, epidemiology, yield losses and physiologic specialization which are vital basic data in plant breeding.

Breeding for maize disease resistance in Kenya was done without sufficient field data resulting in a scenario where hybrids were produced without any field and green house disease challenges. Thus, Kenya has produced hybrids of high disease risk. This may be explained by several factors including the lack of technology. Since our maize came from the Americas, the use of American technology and inbred lines as differentials has great value in understanding the pathogens' variation and genetics. The most important maize diseases in Kenya are rusts caused by *P. sorghi* and *P. polysora*; leaf blight incited by *E. turcicum, B. maydis, Phaeosphaeria maydis* leaf spot and maize streak virus. While these diseases are known to be present, there is no information on incidence and severity, distribution, variation and yield loss. Hence it is practically impossible to implement sound breeding practices in maize improvement programs as has been done in the West. All these pathogens can occur on one individual plant.

Information about races of pathogens among the Kenyan resource-poor farmers is non-existent or manifestly fragmented.

Low yields in developing countries are due to disease and largely to ineffective control strategies (Ayub-Takem and Chheda 1982). Another reason for low yields is the use of selfed or sowing previous season diseased seed, which is in various stages of degeneration. Different maize varieties in Kenya are grown as determined by the prevailing conditions characteristic of the ecological zones. Moisture stress and altitude influence the varieties for each ecological zone . There are two seasons ranging from 4 to 5 months in the relatively dry zones and the coast to 8 months in the western highlands and the Rift Valley. Therefore maize breeders in Kenya have always taken this into account resulting in maize lines developed for the arid zones, characterized chiefly by faster maturity and drought tolerance. Varieties for the mid-altitudes and others for the cool highlands have the longest growing seasons of about 8 months (Gebrekidan *et al*. 1992).

Kenya depends on the agricultural sector for food requirements but due to climatic variations, total self-sufficiency is not achieved every year. The main staple food is maize which as a crop is the most important in both land area and value. Over 90% of the maize is produced in small holdings of less than 5 hectares (Heyer *et al*. 1976). Whenever there is a severe drought in Kenya the maize crop fails which result in food shortage because maize is the primary source of nutrition for the majority of Kenyans. About 26 diseases caused by fungi, bacteria, nematodes, and viruses have been reported in Kenya (Njuguna *et al.* 1992). However, no comprehensive study has ever been undertaken to document the incidence, distribution and the severity of these diseases in the maize growing areas as a tool to aid in prioritization of research in maize disease management country wide. The main reason is the lack of funds and trained personnel. This research project was undertaken as an attempt to fill this gap of information and establish a foundation in our National Maize Research Program in the area of disease resistance. The project is an evaluation of the diseases of maize in the ecozones and an attempt to determine the pattern of pathogenic variation as impacted by altitude and other environmental factors. The research was carried out during the short and long rain seasons of 1995 and 1996 in the months of July 1995 through October 1996. **The**

Objectives of this study were as follows: 1. Determine rust and blight incidence and severity in the maize producing regions in Kenya, 2. Collect isolates of *E. turcicum* from all corn growing areas and study their cultural characteristics, 3. Determine the physiologic races of *E. turcicum* by inoculation on American inbred differentials and 4. Determine the reactions of selected Kenyan maize lines to inoculation with *E. turcicum*.

LITERATURE REVIEW

The most important diseases are rust (*Puccinia* spp.), and leaf blights in the maize-growing regions of Kenya (Manwiller 1983). Foliar diseases have a direct influence on the amount of dry matter stored in the grain and leaf blights can induce up to 50% losses in yield (Ullstrup and Miller 1957). Disease severity is often assessed using various methods that utilize scales, keys, visual estimations and measurements (Adipala *et al*.1993, Horsfall and Barratt 1945, Saghai Maroof *et al.* 1993, Slopeck 1989, Nicholson and Warren 1975, Robbins and Warren 1993, James 1968, Solomonovitz, Levy and Pataky 1992, Levy and Pataky 1992, Traut and Warren 1993).

COMMON RUST -*Puccinia sorghi* Schw.

Common rust caused by *P. sorghi,* an obligate pathogen of maize and a macrocyclic heteroecious fungus, occurs in all areas of the world where maize is grown. The fungus thrives in high humidity and moderate temperature conditions (Kim and Brewbaker 1976). Production of maize all year round increases the severity of epiphytotics (Brewbaker 1974). Uredospores are the primary source of inoculum and secondary spread (Kim and Brewbaker 1976). *P. sorghi* infects the leaves, 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). Resistance to rust (*Rp)* is expressed as hypersensitive or fleck-like reactions and sources of this resistance has been identified (Hooker *et al.* 1955, Hooker and LeRoux 1957, Hooker 1969, Hagan and Hooker 1965, Hooker and Saxena 1971). Resistance for *P. sorghi* in the United States has been found in maize strains from Australia, Africa, Guatemala,Turkey, Uruguay, Yugoslavia and the United States. The resistance is expressed as chlorotic to necrotic flecks with small uredia (Hooker and Le Roux 1957, Hooker 1962). Two sources from Kenya were identified as Kitale flat white and Njoro flat white (Hooker 1962). A race able to infect maize with Rp genes has been described (Bergquist and Pryor 1984). Common rust has not been very damaging in the United States largely due to the incorporation of resistance genes (Hooker 1969, Melching 1975). Headrick and Pataky (1986) reported that rust developed rapidly at night temperatures of 24^{o}C and 16^{o}C and that on nights with 32^{o}C very few uredia formed but

necrotic lesions formed without sporulating. Pathogenesis in resistant and susceptible combinations have been studied and reported (Hilu 1965). Resistant varieties is the only feasible means for the control of common rust (Bergquist and Pryor 1984).

LOWLAND RUST - *Pucinia polysora* Underw

P. polysora was first observed in Western Africa in 1949, and assumed to have come from the Western Hemisphere probably with maize germplasm. Epidemics resulting from uredospores as the primary source of inoculum can cause up to 60% loss in yield. During the period 1950-51 epidemic, high levels of southern rust in West Africa was reported (Rhind *et al.* 1952). Southern corn rust (*P. polysora*) differs from common rust (*P. sorghi)* in pustule size, shape and color, but the most pronounced variation is that it kills the host, unlike *P. sorghi*. Raid *et al*.. (1988) found that even if infection comes as late as after anthesis losses occur and the pathogen may cause heavy losses when the conditions are conducive. Lowland rust is a warm weather disease being favored by temperatures of 12-27^{o} C (Hollier and King 1985, Melching 1975). Highly resistant genotypes have smaller uredosori than moderately resistant or susceptible ones. Few uredosori rupture late in the resistant combinations (Subrahmanyam *et al.* 1983). Once infection has been induced the disease progresses as the plant develops. 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 due to lowland rust can occur especially if infection occurs early (Zummo 1988). All maize grown in E. Africa and tested in the 1950s had no resistant reaction, but those from Mexico had major gene resistance (Storey and Ryland 1954). An isolate of *P. polysora* collected in Kenya from maize cultivar with a B73 background was found to be virulent to *Rp1* gene of resistance (Hulbert *et al.* 1991). Resistant lines normally show small chlorotic or necrotic flecks with no sporulation. 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). Ullstrup (1965) reported a dominant gene of resistance and designated it as Rpp9. In Kenya, three physiologic races known as EA. 1, EA. 2 and EA.

3 have been reported (Ryland and Storey 1955, Storey and Ryland 1954, 1961). Six races were reported in the United States with the possibility of many others thought to occur (Robert 1962). The search for physiologic specialization in plant pathogens should be a continuous process as variation is present and keeps on evolving as the foregoing review implies.

SOUTHERN LEAF BLIGHT -*Bipolaris maydis*

Bipolaris maydis (Nisik.) Shoemaker, *Helminthosporium maydis* Nisik. (Syn. *Dreschlera maydis* (Nisik.) Subram. & Jain), teleomorph *Cochliobolus heterostrophus* (Drechs) occurs as races 0, T and C . In an overwintering experiment, race O was found to have a higher saprophytic ability than race T; only about 4% of the recovered spores were race T (Blanco and Nelson 1972). Race C was reported in China infecting maize with C-cytoplasm (Wei *et al*. 1988). Race T is characterized as specific for certain cytoplasms such as the widely used T (Texas) type for male sterility. The P-cytoplasm from South America and a few other cytoplasms are also known to be susceptible (Hooker *et al*. 1970); is a weak parasite on resistant plants in the field; seedlings are more easily infected; produces a pathotoxin; attacks the leaf, leaf sheath, husk, shank, ear, and stalk tissue of the plant; reproduces rapidly in susceptible plants and ; may have a lower temperature optimum than race O; race O shows little or no specificity to plant cytoplasms; produces only limited amounts of a non specific phytotoxin; infects leaves mainly, producing smaller lesions with parallel sides and little chlorosis; seems to reproduce less rapidly than race T on susceptible plants; 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 in both races with race T being most sensitive. More lesions formed at 30°C than at 15 or 22.5° C (Warren 1975). Lesion size increases in almost a straight line relationship with increasing dew periods and colonization temperatures (Nelson and Tung 1973). In the 1970s, *B. maydis* induced southern maize blight epidemic caused losses in maize with cytoplasmic male sterility gene (CMS-T) of more than US Dollar 1 Billion (Ullstrup 1972).

Phaeosphaeria maydis LEAF SPOT

*Phaeosphaeria maydis (*P. Hennings) Rane, Payak and Renfro as a pathogen of maize has been reported in India, Brazil, Colombia, Equador and Mexico (De Leon 1984). It was identified as *Phaeosphaeria maydis* in India where it was observed in West Bengal and Uttar Pradesh expressed as round, elongate or oblong bleached spots with brownish colored margins occurring on the leaves (Rane *et al*. 1966). Perithecia and pycnidia containing conidia of *Phyllosticta* were observed on the lesions. Primary inoculum was found in diseased crop debris (Rane *et al*. 1966). In Kenya the disease has been reported as a minor pathogen of maize needing only medium level attention and hence is considered not important (Njuguna *et al.* 1992).

NORTHERN LEAF BLIGHT -*Exserohilum turcicum*

Exserohilum turcicum (Pass.) Leonard and Suggs, *Bipolaris turcica* (Pass.) Shoemaker, *Dreschlera turcica* (Pass.) (Subram. and Jain). teleomorph *Trichometasphaera turcica* Luttrell, (Syn. *Setosphaeria turcica* (Luttrell) Leonard and Suggs) is the cause of maize northern leaf blight first reported in Passerini on maize in Italy in 1876. It was first reported in the USA in New Jersey in 1878, this was followed by a serious outbreak of northern leaf blight in Connecticut in 1889 (*Drechsler* 1923). Northern maize blight is favored by mild temperature and high humidity (Ullstrup 1970). Heavy dews, cool temperature and frequent rains create good environmental conditions for disease development (Jordan *et al.*1983). Levy and Cohen (1983) reported that disease is more aggressive in young susceptible plants with an optimum for infection and lesion number at 20°C, lesion length increases with increasing length of dew period. Levy (1989) reported that pathogenic fitness and environmental conditions are very important in determining severity as the epidemics depend on the ability of *E. turcicum* to infect, grow and sporulate on maize plants. In the continental United States the disease has been effectively controlled by the use of the dominant *Ht* gene (Hooker 1961, Smith and Kinsey 1980, Turner and Johnson 1980). A new cholorotic halo gene for resistance of limited commercial value but which may be useful in combination with *Ht* genes has been reported (Carson 1995a). Pratt e*t al*. (1993) reported a polygenic based resistance in OhS10 expressed as rate reducing resistance or low number of lesions using a 0-5

severity rating scale. Combining *Ht1* and *Ht3* genes did not result in significantly less disease from those homozygous for each *Ht1* or *Ht3* (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 the *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. 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. 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 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. 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. Pataky (1992) found that yield losses were significant when disease severity was high on the upper leaf canopy which is in agreement with the studies of Levy *et al.* (1990), Raymundo (1978) and Solomonovich (1992) who found that plants defoliated of the lower third of all the leaves showed no yield losses. Leath and Pedersen (1986) found that a cross between resistant B37*Ht3* and susceptible B37 had a severe chrolosis associated with resistant lesions resulting in a high area under the disease progress curve (AUDPC) value for resistant inbreds with low sporulation and secondary spread. 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).

Classification of isolates of *E. turcicum* into races is based on the resistant genes marked by an isolate in the widely used nomenclature as suggested by Leonard *et al*. (1989). They proposed that evaluations be carried out in temperatures near 20°C and

light intensities of 25 to 50 lux because reactions associated with *Ht1, Ht2* and *Ht3* are thermal and photo sensitive. This was demonstrated by Leath *et al*. (1987) when they showed that resistance in lines with *Ht2* and *Ht3* was expressed clearly in controlled environment chambers at 22°C and 18°C night temperatures. 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 HtN*, race 12, *Ht3, HtN /Ht1, Ht2*, race 23 as *Ht2, Ht3 / Ht1, HtN* race 23N as *Ht2, Ht3, HtN / Ht1*. This classification left room for the accommodation of new races that could be encountered in future studies. The *Ht1, Ht2* and *Ht3* resistance occurs as chrolotic lesions with minimum sporulation, while the *HtN* induced resistance is expressed as a delay in disease development until after pollination (Leonard and Levy 1989). Bergquist and Masias (1974) reported the first race of *E. turcicum* . Lipps and Hite (1982) reported the presence of race 1 in Ohio and was virulent on *Ht* and *Ht1* but avirulent on *Ht2*. Turner and Johnson (1980) reported a similar race in Indiana. Smith and Kinsey (1980) reported a new race designated race 3 with a virulent formula *Ht1/Ht2,Ht3*. Thakur *et al* (1989*)* reported the presence of yet another race named race 4. Jordan e*t al*. (1983) reported the occurrence of races 1 and 2 from seven states in the Central and Eastern USA where race 1 was virulent on B37 only and race 2 virulent on B37*Ht*; no isolate was found virulent on B37*Ht2* or Oh43*Ht3*. A report by 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, 23N and race 2 in Uganda.

According to Gevers (1975) the *HtN* major gene of resistance derived from the Mexican maize variety *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 genes ratios, but does, however remain in the tolerable limits of deviation of stability and segregation. He suggested the occurrence of biotypes in India which were able to overcome the *HtN* gene of resistance. The *HtN* gene in some backgrounds was sensitive and at high temperatures symptom expression was reduced on B37*Ht3*. Plants were evaluated at both 26^{o} day / 22 ^{o}C night and 22^{o} 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). 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 may be reduced at low light intensities. Recent studies in Uganda by Adipala *et al.* (1993) found that *E. turcicum* occurred in all maize growing areas and was more severe in wet areas. However, all isolates tested were virulent on A619 and avirulent on A619*Ht1*, A619*Ht2*, A619*Ht3* and A619*HtN*, hence were classified as race 0. These observations are at variance with the results in Uganda by Welz *et al*. (1993). Average disease severity ranged between 0.5 to 25% in Uganda. After evaluations of Ugandan maize germlasm, Adipala *et al*.. (1993b) also reported that all had necrotic susceptible reactions when inoculated with races 0, 1, 23 and 23N and did not express symptoms typical of the *Ht* gene. Seedling inoculation was useful to identify chlorotic resistance, while adult plants were useful in assessing rate reducing resistance.

GENETICS OF RESISTANCE TO *E. turcicum*

Hooker (1961) reported the unique chlorotic lesion type of resistance on maize lines characterized by chlorotic lesions, and late developing lesions with small necrotic center surrounded by a light green margin. These lesions produced fewer spores compared to the rapidly developing necrotic susceptible lesions. This type of resistance was found to be controlled by a single dominant gene *Ht*. Homozygous dominant plants rarely have lesions. Ullstrup (1963) reported similar results on line P.I. 217407 where small lesions were surrounded by chlorotic halos with very limited sporulation in resistant genotypes. Further work by Hooker (1963) also concluded that the resistant chlorotic lesion type was conditioned by a single dominant gene in the dent corn inbred line GE440.

Hilu and Hooker (1963, and 1965) showed that symptoms were similar for susceptible and resistant lines, from 2-7 days, which appeared as minute white to light green flecks after inoculation of inbred and hybrid seedlings of GE440 with *E. turcicum*. On susceptible lines these flecks developed into lesions that wilted before developing necrosis. No wilting was seen on resistant cultivars. Disease development may take

about 15 days. Sporulation was delayed 50-80 hrs and the population of spores per unit area may be reduced 60 times in the resistant lesions as compared to susceptible lesions. This is a situation normally seen in monogenic chlorotic lesion resistance but not in multigenic resistance. Ceballos *et al., (*1991) reported that development of new races shorten the durability of the chlorotic resistant reactions which are controlled by single monogenic resistance genes. Polygenic resistance is normally expressed by reduced number of lesions and decrease in lesion size and amount of sporulation (Ullstrup 1970). Singulas *et al*. (1988) reported that the average level of resistance, mean lesion area, the rate of increase in lesion size and the shape of the lesion are strongly influenced by host gene makeup as determined by contributions of each parent.

PLANT DISEASE ASSESSMENT

There has been no standard protocol for disease assessment, although many forums have been devised to look for practical, efficient and accurate assessment of disease intensity. A globally accepted standard method would encourage data comparison, improvement of communication and interpretations of results between professionals and ultimately to the consumer, the farmer (Watson, *et al.* 1990). Despite the great need for a standard, there has not been a universally accepted formula for disease measurement. A commonly used system, that of Horsfall and Barratt (1945) suggests that according to Weber-Fechner law, 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. However, Hebert (1982) indicates that the underlying assumption of the Horsfall-Barratt system is false and that we are devoid of data supporting one scale over the other or even the superiority of a scale over direct visual disease assessment. Slopek (1989) stated that the Horsfall and Barratt system is more rapid than direct visual estimates because one need only determine severity of disease is within the percent leaf area diseased (PLAD) range boundaries of a rating category. In his study, Slopeck (1989) used a 1-5 scale containing ranges from 0 to 100

as percent leaf area diseased categories, but also noted that visual assessments in some stuations may be most valuable. Field disease measurements in varietal trial and measurements 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 leaves in cereals from 1 to 75% diseased area. In Uganda Adipala *et al.* (1993a) used 0 to 75% severity scale for maize northern leaf blight and observed 20 plants along the geographical cardinal points. On selected farms, Adipala *et al*. (1993a) examined five plants to the North, South, East and West resulting in a sample of 20 plants per site and then visually assessed disease severity. Disease severity or the extent of tissue coverage by lesions rating on a scale of 0 to 5 was used by Nicholson and Warren (1975). O'Brien and Brugg (1992) used ten participants and three severity scales to test accuracy and precision for corky root of lettuce. One of 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 was the H-B scale which 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. Maize crop for

assessment is found in the field in many stages of growth as illustrated by Hanway (1971). Assessment depends on the pathogen involved, the crop that is diseased and its growth stage. It is critical to time the time of survey to coincide with the pathogen peak activity and at the time the host shows most evidence of attack.

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