HISTORY AND ETIOLOGY OF THE LETHAL DISEASE OF COCONUT PALM (COCOS NUCIFERA L.) IN TANZANIA

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SUMMARY

In the Bagamoyo District, large coconut plantations were established as early as 1870 and the industry received a further impulse from German settlers in the 1930's. The first report on the occurrence of the lethal disease was in 1911. At present the incidence of the disease is low.

Coconut palms were planted in the Kisarawe and Rufiji Districts during three main periods. The first during the Arab rule mainly between 1870 and 1888, the second during the German administration (1891 - 1914) and the third and most important after the first World War. Although the lethal disease occurred probably as early as 1890, it made little impact on the two first plantings. However, serious outbreaks occurred in the 1940's when the post 1928 groves came into bearing.

The most important outbreak in the Kisarawe District was near Sanga-Sanga in 1941. The disease spread in eastern, then southern directions to Msonga and Magawa. It moved as well to the north across the Luhute valley to Sotele and Binge. In both directions the disease spread over a distance of approximately 40 kilometers during a period of 35 - 40 years. While this general direction of movement over an extended period of years was revealed by the survey, the spread did not occur along an extended front and was often unpredictable. There was no apparent relation between the direction of spread of the disease and the prevailing winds although it spread faster through groves planted along open spaces like valleys or swamps.

Similarly rampant spread of the lethal disease was reported from the Rufiji and Kilwa Districts. Outbreaks of the lethal disease were reported at various places along the coast of mainland Tanzania at various times from the 19th century onwards. Sometimes independent outbreaks occurred simultaneously. It is therefore impossible to recognize one or more sources from which the lethal disease originated. The disease has obviously been indigenous in the coastal areas of mainland Tanzania for a long

time.

Injection of both bearing and non-bearing diseased coconut palms with tetracycline-hydrochloride antibiotic resulted in remission of symptoms in test palms but only when injection was started in sufficiently early stages of disease.

Total remission of symptoms was obtained in only one bearing palm. All the other remitting palms showed delayed symptom development compared with their controls and succumbed later. The temporary symptom remitting bearing palms often produced new healthy inflorescences which blackened later. In non-bearing palms, those which were injected in early stages of disease showed completely unaffected spear leaves for a long time but nonetheless the palms collapsed later. Taken in conjuction with the finding of MLO in diseased palms, these results support the concept of a MLO etiology for the lethal disease.

Insect population studies at seven different lethal disease affected sites revealed that the insects occurring on coconut palms in Tanzania are almost exclusively planthoppers (Fulgoroidea). Twenty-nine different species were identified at CAB Intitute of Entomology of which only seven were common at all sites.These seven species are the most likely candidates as the vectors of the agent causing the lethal disease.

Seasonal peaks in the populations of several species were observed but they were neither clear nor consistent. No variety or hybrid was visited by consistently higher numbers of insects than any other. Removal of shade appeared to result in an increase of insects on the palms.

In transmission trials to find the vector of the lethal disease, only one test palm showed symptoms similar to those of the disease. However, the palm was observed in an advanced stage and not sampled. This possible case of transmission needs confirmation.

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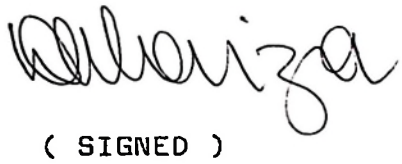
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DECLARATION

I DOMINICK ALFRED KAIZA DO HEREBY DECLARE THAT THIS IS MY ORIGINAL WORK AND THAT IT HAS NOT BEEN SUBMITTED FOR A SIMILAR DEGREE IN ANY OTHER UNIVERSITY



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INTRODUCTION

1:1 THE COCONUT PALM

1:1:1 The Origin of the Coconut Palm

The coconut palm, Cocos nucifera L. is considered to have been domesticated from a wild species growing somewhere in the present optimum range of the modern coconut but with smaller, less satisfactory fruit (Fosberg, 1960). Fosberg thought that as it became domesticated it was spread through the agency of man over an increasingly wide area. Eventually, it replaced its wild ancestor and the original habitat and the center of domestication became obscured. It is now believed that the coconut palm originated in South East Asia or Micronesia, where it was found that the palm and its product were inextricably interwoven with the life and custom of the people to the extent that human life would have been impossible without it (Harries, 1977; Purseglove, 1968, 1972). It is likely that the palm was dispersed westward to India and Sri Lanka and hence to the East African coast and eastwards across the Pacific towards Panama (Harries, 1977).

The way in which the coconut was dispersed to other parts of the tropics is not known (Cobley, 1976). However, the dispersal is believed to be either by floating or by man. Dispersal by floating would not be successful unless the seedling is able to grow where the fruit is washed up. This ability is restricted on the ground that human assistance is needed to overcome competition from other plants and predatory animals. However, both natural and human assisted dispersal have been of equal importance but at different times.

Purseglove (1968), considered that the coconut can be dispersed by sea and establish itself occasionally without the aid of man. This was proved by what happened

on the island of Krakatau, 25 miles south of Java. When the fauna and flora were completely destroyed after a volcanic eruption, the coconuts became reestablished. Another example is the discovery of 41 germinating coconuts on Anak Krakatau, IV, a new volcanic Island which emerged from the sea in 1930. Pureeglove (1968, 1972), also considers that the coconut drifted to the Seychelles Islands and became a natural part of the aboriginal flora along the sea shore.

The idea of dispersal of coconut by ocean currents is supported by Polunin (1964). He considered that the main requirements for dispersal by sea or water are sufficient buoyancy, impermeability and its adaptability to a particular location. Buoyancy in the coconut is not only the result of a thick husk, but also reaults from a large cavity in the nut in which the endosperm is limited to a 12 mm layer lining the shell. The liquid which partially fills this cavity is absorbed as the husk becomes saturated (Harries, 1978). The distance a fruit can float and remain viable is therefore limited by the time it takes to germinate. Natural dissemination over shorter distances for periods up to 400 days particularly in the Indian and Pacific Oceans was considered to be a reasonable assumption (Harries, 1978).

Dormancy is no advantage for the coconut which is a perennial that flowers and sets fruit every month of the year in a constantly warm and humid tropical climate. Successful inter-island or trans-oceanic dissemination will depend on a slow rate of germination. A thick husk, therefore contributes to slow germination as well as to floating ability. Once germination begins, the amount of endosperm determines how long the seedling survives and how well it competes for establishment with the existing flora. When many fruits are produced and individual fruit size is limited and therefore less endosperm is available,

the embryo is small and the resulting seedling grows best where there is little competition (Corner, 1966).

Dissemination of coconut by human interference is an obvious reason for existence of similar varieties in different parts of the world. An example is a variety " Thifow " described in Micronesia which occurs as well on other islands such as Bali and the Andaman Islands. This type also occurs under the name kappadan in south Malabar, in Rangoon as kobbari and in Sri Lanka as kamandala. This variety has been equated with the "Lupisan" and San Ramon in the Philippines and with the Markam in New Guinea (Harries, 1978).

Whitehead (1966), suggested that Polynesian travellers selected and carried coconuts as a source of drink and food for the journey. They could have established them either accidentally or deliberately on arrival on any shore they visited. Also rafts, canoe and other boats loaded with coconuts may have accidentally drifted by ocean currents to new areas and coconuts got established there. Once established, however, cross-pollination between the introduced and the indigenous varieties in subsequent generations and selection resulted in the apparent variable population present today.

1:1:2 History of the Coconut Palms in East Africa

Chinese coins of the eighth and ninth centuries have been discovered at Kilwa and Mogadishu (Revington, 1936) and at Mafia (Robinson, 1937). This suggests that there was commercial contact between Asia and Africa and coconut may have been imported during those commercial transsactions. In any case, when the Arabs arrived in Zanzibar in the tenth century they found coconut palms (Lofchie, 1965). The Arab domination of the colonial scene in East

Africa was interupted early in the sixteenth century by the arrival of the Portuguese. Vasco da Gama recorded the existence of coconut palms at Malindi, north of Mombasa (Kenya) in 1493 (Purseglove, 1968). In 1635 a Portuguese commandant erected a small fort on the east coast of Mafia Island at Kirongwe. During this period, the people in the small islands of Auxole (Chole) and Juani had to pay annual tribute of copal (copra) and coconut fibre to ships sailing to Portugal. However, the Portuguese empire on the East African posts collapsed in 1697 except in Mozambique. The East African coast north of Mozambique was then dominated by the Arabs who settled in Kilwa (Revington, 1936). At this time intensive planting of the coconut palm had not yet begun.

The Europeans to follow the Portuguese were the Germans who arrived in the late nineteenth century. They dominated mainland Tanzania for about thirty years. Some coconut plantations were established between 1906 and 1914 and when the Germans were ousted from Tanganyika the coconut industry was beginning to be established, particularly in the Tanga Region. The expansion of coconut plantations was continued during the British administration. Between the years 1918 and 1946, coconut planting was expanded in the Tanga and Pangani Districts, mainly in the coastal belt of Mkinga and Gombero and along the Pangani River in place of the German and Arab sugarcane plantations (Swynnerton, 1946). This change to coconut production could have been due to high seasonal labour demand of the sugar plantations which could no longer be met following the abolition of forced labour after World War I. By 1934, the coconut palms could be found as far inland as Korogwe.

In contrast to the expansion of the coconut plantations in the Tanga region, the plantations in Bagamoyo District and those south of Dar es Salaam were planted

by German settlers around 1930 as told by Mr. Tessin (1983), one of the Germans involved. He writes that before World War I, there were only a few big plantations in Tanzania, one in Tanga (Moa), the plantations of the government in Dar es Salaam (near the airport - at present given way to industries) and one on Mafia Island. The other groves were smaller, owned by Arabs or Asians and around the Christian Missions. About 1926, German settlers were allowed to the country and planters from the South Sea mainly Samoa started new plantations. The plantations were started at Kitopeni, Mpimbini, Kidagoni, Mbegani and Chambezi near Bagamoyo; Wazo and Mbagala near Dar es Salaam; Kisiju and Kifumangao in Kisarawe District and Salale in Rufiji District. At the same time and during the 1940's and 1950's, villagers near these plantations were encouraged to plant the crop by the colonial administration. This resulted in huge areas of smallholder coconut plantations.

1:1:3 Importance of the Coconut Palms in Tanzania

The coconut palm is an important crop in the coastal areas of mainland Tanzania and the Islands of Zanzibar, Pemba and Mafia. Some coconut palms are found in the inland areas of Morogoro, and as far as Kyera in Mbeya Region, Tabora and Mwanza, where the coconut groves are small. It is predominantly a smallholder crop and provides domestic requirements for freshnuts and oil which are important constituents in the diet of the coastal people. Copra produced by drying the endocarp is used for the extraction of cooking oil and for the soap industry. Industries producing these items are found in Tanga, Dar es Salaam, Mafia, Pemba and Zanzibar. The leaves are used for thatching houses, the water from immature nuts is used as a

refreshment, dry husks and shells are used as fuel and the young unopened inflorescence is tapped for toddy.

1:1:4 Coconut Production in the Main Growing Areas of Tanzania

The optimal growth of coconut occurs between a temperature range of 27 - 32 ^{o} C, with a minimum rainfall of 1500 mm well distributed throughout the year. A rainfall higher than 2000 mm can be tolerated provided drainage is good. Coconut also prefers a light textured, well drained soil, permitting free root development and aeration. In Tanzania, rainfall is the major climatic constraint causing sub-optimal conditions for coconut production (Agrar und Hydrotechnik, 1980). In the north and central part of the coastal belt there is bimodal rainfall pattern but south of the Rufiji river, the distribution is monomodal. Zanzibar and Pemba receives the highest and best distributed rainfall with the least pronounced dry season. The Mafia Island is favoured by relatively high total rainfall, but has one long dry season. The same applies to the Lindi, and Mtwara regions south of the Rufiji river (Agrar und Hydrotechnik, 1980).

Due to a wide variety of parent material and land forms there is a wide range of soils in the coastal belt of Tanzania and the Islands. These soils are described in a land suitability survey carried out for the National Coconut Development Programme by Agrar und Hydrotechnik (1980) and earlier in a FAO report (1975).

Climatic conditions, rainfall patterns, amount of rainfall in a year, soil fertility status and the dominant soil types have been described by Samki and Dewan (1981) and Samki *et al.*, (1982).

The cultivar grown throughout the area is the East African Tall (EAT) and the yield does not exceed an average of 60 nuts per palm per year under good management (Anderson, 1967). This yield was rarely attained even in the earlier years when the crop was in the prime of production. An average of 40 nuts per harvest was an exception (Tessin, 1983).

The amount of copra exported from Tanzania between 1975 and 1983 is given in Table 1.

Table 1: Amount of copra exported from Tanzania between 1975 and 1983

Year	Quantity (m. tonnes)	Value (in '000 T.Shs.)
1975	na	na
1976	na	na
1977	322	362
1978	5247	14648
1979	2177	13691
1980	2069	9573
1981	--	--
1982	--	--
1983	1000	6278

na - figure not given

Source : Bulletin of Crop Statistics 1983/84 Statistic Unit Planning and Marketing Division; Ministry of Agriculture and Livestock Development, Dar es Salaam 1985

Coconut production is at present on the decline particularly on the mainland. This has been attributed to a number of constraints such as :

(i) pests and diseases

(ii) unavailability of selected and improved plant-materials.

(iii) lack of trained and experinced Tanzanian staff to undertake a coconut development programme.

(iv) lack of information for overall planning purpose such as on existing and potential coconut areas.

(v) lack of information to formulate a technical package incorporating recommendations on variety practices.

(vi) lack of information on modern plantation management practices.

(vii) inadequate institutional, legal and organisational facilities required for implementing a coconut developing programme.

(viii) missing concept of policy and decision making for an intergrated development strategy of the coconut industry.

(ix) poor management in smallholder plantations (old age and low yielding palms, little replanting and rarely fertilizing the palm).

The decline of the industry cannot be attributed to one single or major constraint only. It is a combination of the above facts (Speidel, 1981).

Although the coconut industry has declined, it had remained the main source of vegetable oil for the coastal people. The traditional important copra producing areas along the coast are now under over-aged and under-fertilized coconuts of doubtful provenance and despite the low prices of copra offered by the government, the coconut is still important in these areas because all of the coconut produced is used domestically.

There is a single major coconut disease causing heavy losses to coconut palms in Tanzania. This disease has been named the Lethal Disease (LD), because there is no vernacular name for it as those given to coconut diseases in west Africa (Dabek, 1977; Dabek et al., 1976; Dollet and Giannotti, 1976; Dollet et al., 1977). It could not be named lethal yellowing because the causal agent of the disease has not been proved to be the same. In addition it could not be named lethal decline because Howard and Thomas (1980) and McCoy and Gwin (1977) used the term to refer to lethal yellowing affecting species of the palm family other than coconut.

The objectives of this study was to obtain the following additional information on the LD of coconut palms in Tanzania.

(i) To survey the history of the LD

The LD destroying the coconut palms is known to exist along the coast of mainland Tanzania. The areas where the disease is active have been located. In addition, its symptoms have been determined. However, its origin and the date of its first outbreak(s) is not known. It is considered to be of importance to know when and where the disease first broke out. This is relevant to determine the direction of spread and to postulate whether the disease was introduced into Tanzania from outside or the disease originated inside the country.

(ii) To find additional evidence for the mycoplasms-like organism (MLO) etiology of the LD.

In earlier investigations with the fluorescence and electron microscopy, mycoplasma-like organisms (MLO), were associated with the LD. However, as MLO cannot be cultured on artificial media and the vector of LD is not yet known,

it is impossible to prove that MLO causes the disease.

It has been shown that tetracycline antibiotics are effective against MLO resulting in remission of symptoms in the host plant. It is relevant to investigate the effect of tetracyclines when injected in LD affected coconut palms. Remission of symptoms in treated palms would be additional evidence for a MLO etiology of the LD.

(iii) To survey, identify and study populations of putative vectors

Homoptera insects, virtually all leafhoppers and planthoppers have been reported to be vectors of yellows diseases. The survey, identification and study of these insects is considered important to answer the following questions :

(a) Which Homoptera species rest or feed on coconut palms ?
(b) Are these insects abundant everywhere or restricted to particular localities ?
(c) Are there seasonal fluctuations in their numbers ?
(d) Is there a preference of the observed insects for any coconut varieties ?

All of this information must indicate which species are more likely to transmit the LD and therefore which species should receive less emphasis in the transmission trials ?

(iv) To transmit the LD agent by putative insect vectors

Yellows diseases associated with MLO, are known to be transmitted by insects. Observation on the etiology of the LD have indicated that it is associated with MLO Initiation of trials with insects is considered important in implicating the putative insects of the LD.

LITERATURE REVIEW

2:1:0 DISEASES OF THE COCONUT PALM IN EAST AFRICA

2:1:1 History of Diseases of the Coconut Palm in East Africa

Diseases of the coconut palm have been reported along the coast of Tanganyika (mainland Tanzania) since 1905. Stein (1905), described a rot of the heart leaves (die Fäulnis des Herzblattes) in which the lower leaves and the spear leaf became yellow red. The spear leaf could be pulled out easily while the roots and the trunk were completely unaffected. The palm then died after sometime. The incidence of the heart rot was reported to be higher during the rainy season. He postulated that the disease was caused by a fungus transmitted by insects. He considered that the removal and burning of diseased palms was an adequate control method.

Vosseler (1907), reported that palms aged between eight and nine years died in great number at Magogoni (near Dar es Salaam) and at Sadani. He partly attributed this disorder due to flooding by sea water but also observed a rot in the heart.

Morstatt (1911), reported the death of many palms aged between one and eight years at the plantation owned by the Catholic Mission at Bagamoyo which were caused by heart rot. He associated this disorder with the damage caused by the rhinocerous beetle Oryctes monocerous, although he also reported heart rot in palms not damaged by the beetle. Furthermore, Morstatt (1912), observed that this disorder occurred only in young palms.

The earliest record of a disease affecting coconuts in Kenya is of Downson (1921), who reported a budrot of unknown etiology from areas north of Mombasa. Only bear-

ing palms were affected. Palms imported from Sri Lanka were highly susceptible while native palms were comparatively resistant.

De Carvalho and Mendes (1958), reported a disease in coconuts in Mozambique between Pebane and Ruvuma characterized by the following symptoms: yellowing of successively young leaves with breaking of the petiole, nutfall, blackening of inflorescences and bud rot.

Santana-Quadros (1972), reported a similar disorder at Morruba, Mozambique which he associated with soil conditions and waterlogging.

Bock et al., (1970), described lethal bole rot of coconut caused by the fungus Marasmiellus cocophilus Pegler from several areas along the coast of Kenya and Tanzania. This disease is characterized by a primary bole rot followed by frond wilt and crown rot and occurs predominantly on seedlings and young palms of up to eight years old. In Tanzania the disease was reported to be most damaging near Kibiti, Pande and Rushungi. The local authorities have always refered to the disease as lethal bole rot (McCauley, 1970; Ringo, 1968; Thomas, 1975).

However, Steiner et al., (1977), investigated twenty one palms in different stages of a decline around Tanga. They did not detect heart or bole rot. The symptoms included, premature nutfall, drying up of inflorescence, drying up of successively young leaves with the midrib breaking, discolouration of parenchyma and vascular bundles below the growing point. In nearly all palms investigated, very young inflorescences were found to be arrested in growth and rotten. Rickettsia-like organisms were identified by electron microscopy in degenerating cells of discoloured tissues below the growing point and from the base of young inflorescences of three declining palms.

Coconut palms exhibiting symptoms similar to those of lethal yellowing were reported by Steiner (1978), at

Bagamoyo, north of Dar es Salaam. In tissue samples of an unopened inflorescence of one diseased palm, no pathogen could be detected by electron microscopy. By aerial survey, Steiner (1978), further observed several outbreaks of a similar disorder between Kibiti and Rufiji river and at Pande, south of Dar es Salaam. Although there was no opportunity for a closer examination, he strongly suspected that the disease occurring at Bagamoyo and south of Dar es Salaam is a kind of a lethal yellowing. Thus Steiner et al., (1977) and Steiner (1978) postulated that there are two diseases of different etiology in Tanzania.

However, Schuiling et al., (1981), never observed the symptoms like those described by Steiner et al., (1977), in particular the breaking of leaf petiole, the time elapsing from initial symptoms to death of the palm and the arrest in growth of juvenile spathes. They described symptoms very similar to those of lethal yellowing in the Caribbean and West Africa (Carter et al., 1965; Dabek et al., 1976; Dollet et al., 1977; Grylls and Hunt, 1971; Romney, 1983).

Considering that Steiner et al., (1977), visited the Tanga region in the dry periods of 1976 (after the exceptionally dry years of 1974 and 1975), it is possible that the symptoms attributed by these authors to the decline were caused by dry conditions. Furthermore, through extensive surveys along the coast of mainland Tanzania the lethal bole rot as described by Bock et al., (1970), has not been found.

The nursery diseases, blast, dry bud rot and leaf spots also occurs in Tanzania (Wuidart, 1980). These are of minor economic importance.

2:1:2 Lethal Disease of Coconut in Tanzania

Since 1980 when the National Coconut Development Programme began its operation, a lethal disease of the coconut palm has been observed. This occurs virtually along the whole coastal belt of Tanzania with the exception of the far south and the Islands of Zanzibar, Pemba and Mafia. Allowing for minor differences due to the variety of environments, the syndrome of the disease recorded was comsistently the same in all affected areas (Schuiling *et al.*, 1981).

2:1:2:1 Symptomatology of Lethal Disease

The first visible symptoms of the disease is premature nutfall. Medium-sized and large immature nuts from the middle of the crown are shed first (Fig. 1). The smaller nuts from the young inflorescences follow while mature nuts usually remain longest on the palm. Shed nuts rapidly develop a darkbrown discolouration in irregular patches where formerly attached to the calyx. Premature nutfall is not restricted to palms affected by the disease but may be due to natural abortion or adverse environmental conditions.



Fig. 1. Prematurely fallen nuts (deliberately gathered)

Shortly after the onset of premature nutfall the youngest open inflorescence appears blackened (Fig. 2.) This blackening may be restricted to the tips of a few branches or include as much as the whole inflorescence. It generally starts when the spathe is still closed but sometimes an apparently healthy open inflorescence turns black later. The tips of the branches bearing the male flowers turn black or darkbrown in typical necrotic patches. This discolouration extends down the branches first involving all male flowers, then the female flowers and rachis until the whole inflorescence is black. Partial blackening (only the branches) is occasionally observed at the onset of symptoms. As the disease progress, inflorescences tend to be more severely affected and often

split prematurely. Depending on the stage of the disease up to five or six blackened inflorescences may be visible, rarely more because beyond this number spathes do not split anymore.

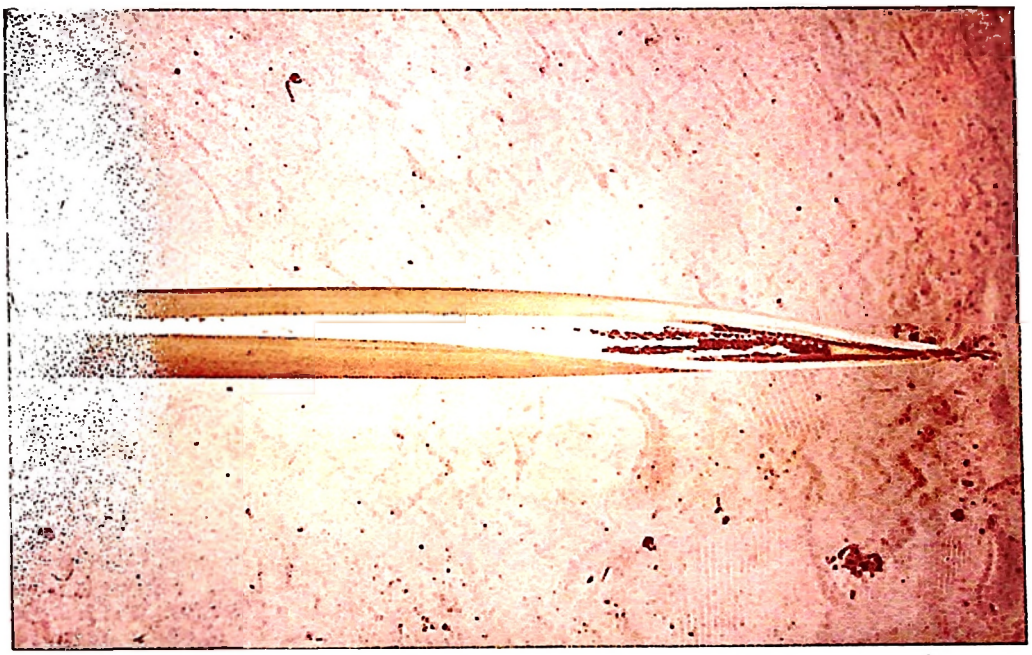


Fig. 2 A Prematurely opened inflorescence showing the start of tips blackening.



Fig. 2 6 Prematurely opened inflorescence showing advanced blackened branches.

In unopened inflorescences necrosis is initially restricted to two or rarely three spathes. Only in advanced stages of the disease the very young spathes are affected by a secondary soft rot descending in the heart of the palm. It affects the outer spathe first progressing from the tips downwards, then the inner spathe and ultimetely the juvenile inflorescence (Fig. 3). This blackening of both opened and unopened inflorescences is considered to be the most characteristic symptom of the lethal disease.



Fig. 3 Abnormal growth and rot of juvenile inflorescences

A healthy palm of the East African Tall variety growing under favourable conditions generally exhibit between 26 and 30 expanded leaves of which one or two of the oldest show discolouration due to advanced senescence. Due to adverse conditions, like old age,drought and malnutrition, palms in Tanzania often exhibit less leaves in the canopy. The pattern of leaf discolouration due to the lethal disease is very different from normal senescence as it progresses rapidly. Simultaneously with symptoms in the inflorescences, the oldest leaves turn bronze (or sometime ochre - yellow). This bronzing starts at the tips of the pinnae at the distal part of the leaf extending proximally. It progresses rapidly to the successively

younger leaves while the oldest ones turn brown and dry up. With advancing disease, dessicated leaves are usually shed resulting in a reduced number of leaves in the canopy but occasionally they remain attached to the palm hanging along the trunk forming a skirt (Schuiling et al., 1981).



Fig. 4 A Initial bronzing in bearing East African Tall variety.

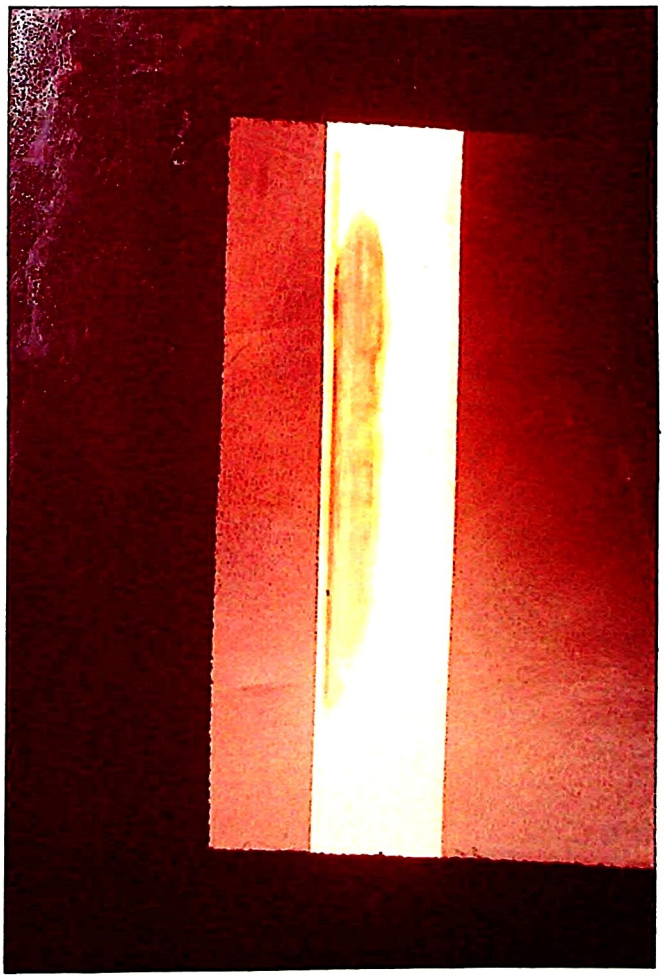


Fig. 4 B Advanced bronzed leaves in lethal disease affeced East African Tall bearing coconut palm variety.

When symptoms in the inflorescences and expanded leaves are already advanced, necrosis starts in unexpanded (spear) leaves (Fig. 5). Patches of grey-brown papery necrosis appear in the apical parts of the spears, usually at the tips of the pinnae, extending from the midrib toward the margins. This necrosis is generally more advanced in the successively younger spear leaves, the oldest spear may escape altogether. It extends rapidly

downwards into the juvenile leaf tissue developing into a characteristic foul smelling rot of the heart. Vascular discolouration is occassionally found in immature stem tissue below the growing point and in the petiole of immature leaves. Only in very advanced stages, when a few green leaves are left on the palm, this rot destroys the growing point and may descend into the soft immature tissues below. It rarely extends into the mature trunk tissues (Fig. 5).

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Fig. 5 A : Initial necrosis in pinnae of spear leaf.

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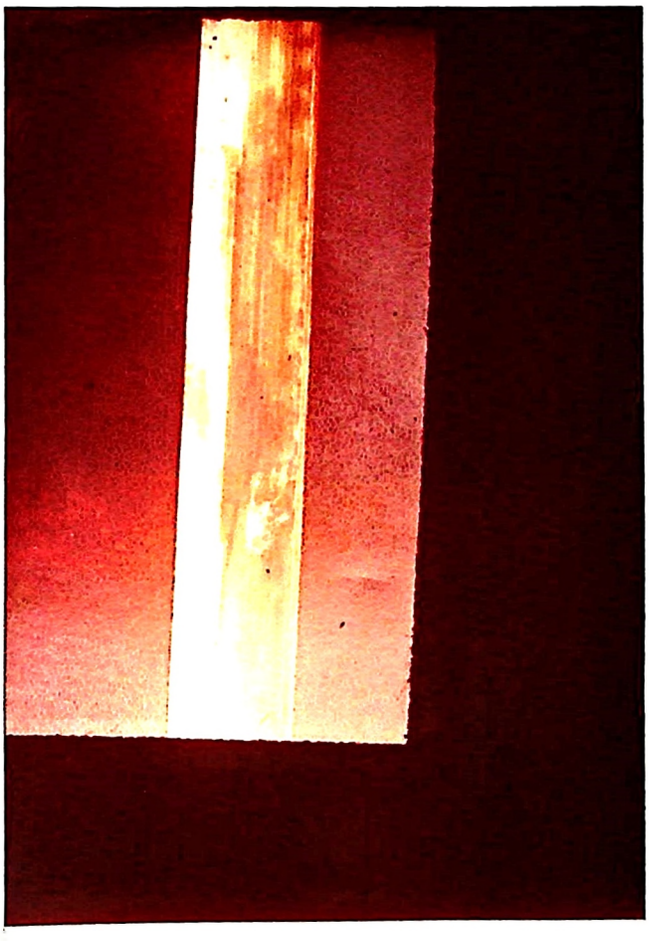


Fig. 5 B Progressing necrosis in the spear leaf

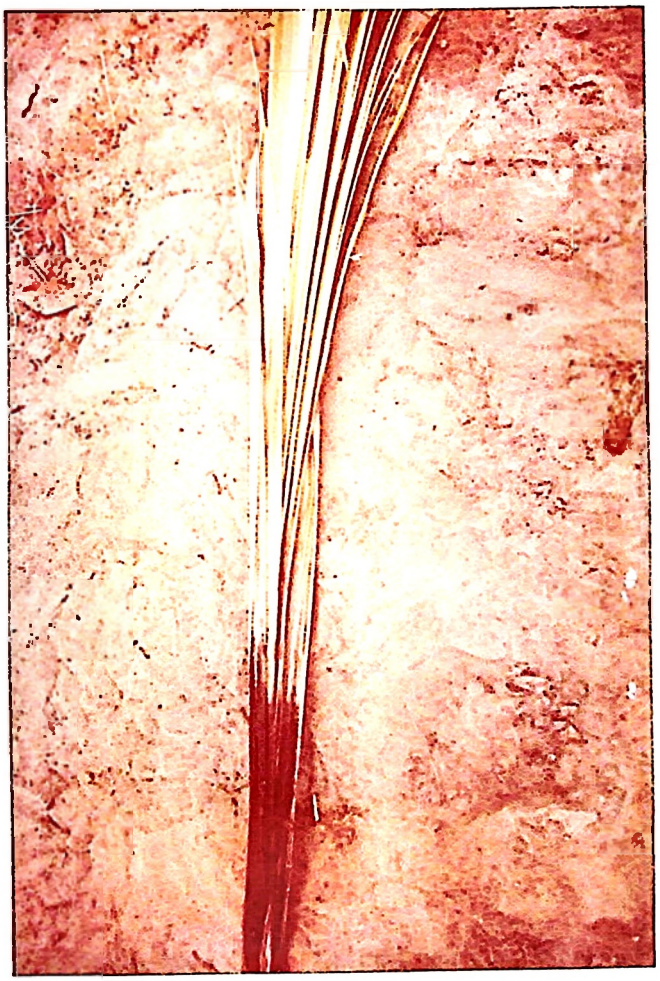


Fig. 5 C Rotten base of the spear leaf

Symptoms in the spear leaves due to the lethal disease can be confused with damage caused by the rhinoceros beetle, Oryctes monocerous. Adults of this insect feed on the spear leaves, tunneling down the growing point. A soft rot descending from the damaged areas often is observed. After heavy beetle attack, the whole cluster of spears may be rotten.

Necrosis and decay of the root system is generally not an early symptom and definitely not a primary symptom. An interval of two to three months usually elapses between early nutfall and the onset of collapse of the root system. Lateral roots are generally affected first with progressive necrosis of first stele and then the cortex spreading from the tips to older tissues. Necrosis in the primary roots usually starts at the tips but occasionally partially rotten roots show healthy tips indicating proximal necrosis. Decay of the root system does not progress rapidly. In palms exhibiting advanced aerial symptoms, normal primary and lateral roots can be found and are often still predominant. Only in very advanced stages of the disease, when only a few green leaves are remaining in the canopy, a general collapse of roots is observed. Even then an occasional health, newly grown root could be detected. New roots are obviously produced until the ultimate stages of the disease. It takes no more than six to eight months from initial nutfall to complete collapse of the crown (Schuiling et al., 1981).

The symptoms in non-bearing palms although restricted in range, are similar to those of bearing palms. These include bronzing and browning of successively young leaves, necrosis and rot of spears (Fig. 6). Unlike in bearing palms, the root decay symptoms appear much earlier. Root decay always starts at the root tips. It takes a period of between two and four months for the young palm to die depending on the age of the palm.



Fig. 6 Bronzing and browning in a young palm affected by the lethal disease

2:1:2:2 Etiology of the Lethal Disease

Considerable research work has been done on the etiology of the lethal disease since 1980. Schuiling et al., (1981) reported DNA accumulation and abnormal callose formation in the phloem elements of the growing point, young leaves and inflorescences of both bearing and non-bearing palms affected by the lethal disease. In the search for the

causative agent of the lethal disease typical MLO structures were found in the same tissues but only when the investigations by electron microscopy were focussed on tissues with a distinct DNA accumulation in the phloem elements demonstrated by fluorescence microscopy using 4-6-diamidino-2-phenylindole (DAPI) as a fluorescence dye (Nienhaus et al., 1982; Deutsch and Nienhaus, 1983). This technique is now used as a simple method of demonstrating DNA in lethal disease affected coconut palms.

2:1:2:3 Epidemiology of the Lethal Disease

Between January and November 1982, the whole coconut producing coastal belt of mainland Tanzania was surveyed for incidence of the lethal disease. A map of disease incidence in the area was completed (Appendix A). No new developments different from these data have been observed. The observations were :-

(a) the disease incidence of the lethal disease in affected areas varies greatly from low (a few widely and irregular scattered diseased palms) to high incidence (many palms are affected).

(b) the pattern of spread in young plantings does not tally with that in mature (old) palm population. On the contrary, at several sites like in the Tanga District (Chongoleani, Bushiri Primary School) and Bagamoyo District (at Chembezi and Pimbini Estate), many diseased young plantings were observed while in mature palms there were a few diseased palms or negligible.

(c) even in areas where the disease incidence was high, the spread of the LD was coperatively low. A few outbreaks occurred far away from the initial foci. This frequent "jump" often of several kilometers resulting in a few secondary foci of disease, compares favourably with the spread of lethal yellowing in the Caribbean and Florida.

2:1:2:4 Resistance to the Lethal Disease

Research carried out in Jamaica on lethal yellowing disease has indicated that, the most important if not the only way of controlling the disease is through the use of resistant varieties (Been, 1980, 1981).

In Jamaica, the Malayan Dwarf and later Malayan x Panama Tall hybrid (maypan) were found to be highly resistant to lethal yellowing (Harries and Romney, 1974). Similar disease resistant trials are under way in the Cameroun and Togo.

The need for a resistant variety favourable to the growing conditions of Tanzania has been realized since 1980. Disease resistant trials including thirty six imported coconut varieties and the local EAT from different origins are now under way. Four of these trials were planted in areas where the lethal disease incidence is high (Kifumangao, Magawa and Sotele in Kisarawe District and Mbwera in Rufiji District), and two trials in areas where the disease incidence is low (Chambezi in Bagamoyo District and Pongwe in Tanga District). In the absence of an inoculation technique, assessment of resistant is based on infection which occurs naturally in the field.

2:2 MYCOPLASMA-LIKE ORGANISMS

2:2:1 The Biology, Morphology and Classification of Mycoplasmas

Mycoplasmas were previously known as pleuropneumonialike organisms (PPLO) because of their similarity to the organisms causing an infectious bovine pleuropneumonia (Edward and Freundt, 1967). There are six genera described Mycoplasma, Acheloplasma, Ureoplasma, Spiroplasma, Thermoplasma and Anaeroplasma. The first mycoplasma species Asterococcus mycoides, was isolated in 1894 from exudates of the destructive cattle disease bovine pleuropneumonia (Friis, 1974). This species was used to study the structure and classification of mycoplasmas. The genus name Mycoplasma was proposed by Nowak in 1929 (Edward and Freundt, 1956), and hence the family name was given as. Mycoplasmataceae. To name the order, Edward and Freundt (1956), proposed two alternatives; Mycoplasmatales and Mollicutes. At present Mycoplasmatales is the Order name and Mollicute the Class name (Edward and Freundt, 1967; Freundt and Edward, 1967).

Mycoplasmas include the smallest known organisms which are able to multiply independently of other living cells. They vary in size, from 300 nm to more than 1 μm in diameter. They are bounded by a triple layered unit or plasma membrabe, 7 - 11 nm thick, distinguishing them from bacteria which have a rigid cell wall (Cole, 1977; Hiruki and Dijkstra, 1973; Maniloff, 1970; Maniloff et al., 1977; Razin, 1978).

Animal species of mycoplasmas are culturable on artificial media of diverse complexity. However, virtually all attempts to culture mycoplasma-like organisms (MLO) associated with plant diseases, aimed at fulfilling Koch's postulates have failed. Giannotti et al., (1975), reported

that they cultured mycoplasma from tips of roots and young inflorescences of coconut palms attacked by Kaincope disease in Togo. It is now believed that the isolated culture was a contamination. The only cultured plant pathogenic member of the class Mollicute is Spiroplasma citri, isolated from citrus infected with "stubborn" disease. It is therefore the only mycoplasma-like organism (MLO) properly identified (Cole et al., 1973; Maramorosch 1974).

Eden-Green and Waters (1981), isolated spiroplasma from healthy and lethal yellowing diseased coconut palms but only in two initial experiments. The results could not be repeated in extensive further tests. Attempt to test the pathogenicity of this spiroplasma to palms and other plants using the suspected vector of lethal yellowing, Myndus crudus were negative. It was concluded that this organisms was not the causal agent of lethal yellowing (Eden-Green et al., 1983).

2:2:2 Mycoplasma and Plant Diseases

In plants, mycoplasmas are considered to be the causal agents of various diseases formally thought to be of viral origin (Hirumi and Maramorosch, 1972; Holmes et al., 1972; Jones et al., 1974; Maramorosch, 1974; Maramorosch et al., 1972; Nayar and Seliskar, 1978). Mycoplasmas of plants are morphologically similar to members of culturable animal Mycoplasmatales. This similarity has given rise to the term Mycoplasma-like Organisms, MLO, (Nienhaus and Sikora, 1979).

Mycoplasma-like Organisms were first associated with plant diseases by Doi et al., (1967), in Japan when they observed them in plants with mulberry dwarf, aster yellows, paulownia witches' broom or potato witches' broom diseases.

Therefore, they postulated that these diseases were not caused by viruses but by MLO.

Diseases associated with MLO, belong to the yellows group. They are of economic importance in temperate and tropical areas where environmental conditions favour multiplication and vector activity. They are divided into four groups based on symptoms; aster yellows (elongation of internodes and leaf yellowing), stolbur (apical dwarfing, stunting, leaf roll, epinasty, wilting and virescence), witches' broom (proliferation of axillary shoots) and decline (degeneration). Under natural conditions MLO may produce symptoms belonging to a number of different types depending on the host species affected, the time and mode of infection, environmental factors and the agent involved (Nienhaus and Sikora, 1979).

Mycoplasma-like organisms affect more than 200 different species in 59 families. The widest host range has been described for the California and New York strains of the aster yellows agent. The stolbur type mainly affects solanaceous plants. The disease affecting tomatoes was named "big bud" in Australia and North America, "stolbur" in Europe and "Mal Azul" in South America. Other disease types are parastolbur and metastolbur in potato. The corn stunt disease, first described in Texas and California in 1945, was differentiated into several types. The disease caused by the Rio Grande strain was found to be associated with spiroplasmas, whereas that incited by the Mesa Central strain seem to have a MLO origin. Among decline diseases caused by MLO, the Western-X disease of peach has a fairly wide host range including other fruits and a number of herbaceous plants. Mycoplasma-like organisms are also found to be associated with a decline of coconut palms and for proliferation diseases of carrots (Nienhaus, 1976, 1981; Nienhaus and Sikora, 1979).

The detection of MLO in diseased plants is not proof of pathogenicity. The main reason for the failure to prove pathogenicity is that attempts to isolate and culture these organisms from plants have failed. The hypothesis that MLO cause plant diseases is supported by their association with diseased plants, their effects on invaded and surrounding plant cells, disease development following vector and graft transmission, and the decrease in their numbers after treatment with antibiotics (Nienhaus and Sikora, 1979).

2:2:3 Localization and Spread of Mycoplasmas in Plant Tissues

Mycoplasma-like organisms are found in sieve elements of phloem and occassionally in the parenchyma and companion cells of phloem. They have been found in the phloem of lethal yellowing infected coconut palms of the cultivar Jamaica Tall (Beakbane et al., 1972; Heinze et al., 1972 b; Plavsic-Banjac et al., 1972). Similar findings on the occurrence of MLO in the phloem of other palm species, Pritchardia and Veitchia affected by lethal yellowing have been reported from Florida (Parthasarathy, 1973). Mycoplasma-like organisms have also been reported from coconut palms having symptoms similar to those of lethal yellowing in West Africa (Dabek, 1977; Dabek et al., 1976; Dollet et al., 1977; Dollet and Giannotti, 1976; Nienhaus and Steiner, 1976) and in Tanzania (Nienhaus et al., 1982; Deutsch and Nienhaus, 1983). In these investigations MLO were detected mainly in the rachis of immature leaves, unopened inflorescences, root tips and near the growing point, the so called "sink" regions of the palm.

Zimmermann (1979), discussed some aspects of translocation of MLO throughout the plant via the long-distance transport channels. He considered that pathogen can only move in the xylem over distances longer than the length of

a vessel if they are smaller than 10 - 20 nm, the approximate size of pores in vessel to vessel pit membranes, or if they can produce wall dissolving enzymes. This requirement makes xylem transport of MLO from roots to leaves unlikely. However, MLO do move in the phloem, because they can be found in sieve tubes of sink areas which are protected from transmission vectors. Sieve tubes are normally under high positive pressure and exude very briefly when punctured. Transmission is therefore an interesting problem; it may be successful only under conditions of slack sieve-tube turgor.

Symptomatology of yellows diseases indicate that sieve tubes can become so plugged with MLO's that phloem sinks are deprived of the nutrients. Excessive callose formation and degeneration of phloem tissue in MLO diseased plants may lead to stunting, wilting or leaf yellowing. They disrupt the normal translocation process and normal growth regulator level resulting in premature development of adventitious buds and altered apical dominance, leading to the proliferation of axillary shoots and the condition known as witches' broom. This imbalance is responsible for phyllody and antholysis in the flowering stage (Nienhaus and Sikora, 1979).

2:2:4 Histological Investigations of MLO Diseases

Since culturing of MLO agents was generally unsuccessful, identification was concentrated on characterization of the agents in thin sections of plant and insect vector material (Dale and Kim, 1969; Giannotii et al., 1968; Heinze et al., 1972 a; Holmes et al, 1972; Maramorosch et al., 1968; Nayar and Seliskar, 1978).

Attempt to detect MLO in either plant or insect tissue have relied upon histochemical technique of staining DNA. These techniques are used as a tool in the preliminary

diagnosis of different mycoplasma diseases (Cousin et al., 1969). Giannotti et al., (1968), experimented with clover phyllody MLO in the plant Trifolium repens L. and an insect vector Euscelis plebejus Fall. They used the Feulgen reaction and methyl green to detect DNA with a complementary DNAase treatment for confirmation. A Feulgen positive reaction was obtained in the phloem of diseased plants. Subsequently, the Feulgen reaction has been used succefully to detect plant cells containing DNA (Cousin et al., 1971; Giannotti et al., 1969; Kartha et al., 1971).

Russel et al., (1975), showed that DAPI, a DNA binding fluochrome could be used to detect mycoplasma contamination of tissue culture. Seemüller (1976), was quick to recognize that DAPI and a benzimidole derivative 33258 Hoechst could be used as a fluochrome to detect DNA in phloem. He used phloem from trees affected by pear decline as well as healthy trees. Positive DNA/DAPI fluorescence was recorded in fourteen out of sixteen samples from diseased trees and none from eighteen healthy trees. The DAPI technique is now employed as a tool in preliminary diagnosis of MLO in lethal disease affected coconut palms (Deutsch and Nienhaus, 1983; Nienhaus et al., 1982; Schuiling et al., 1981). However, it still remain necessary to establish the nature of this DNA related fluorescence by electron microscopy as it can be caused by viruses, MLO and Rickettsia-like Organisms (RLO) (Cousin and Kartha, 1975; Thomas and Norris, 1980).

Phloem elements were reported to respond to injury with the production of wound callose and suggestion was made to detect abnormally high level callose in sieve elements which was thought to be an indirect method of diagnosis of MLO infected plants. Hiruki and Dijkstra (1973), used aniline blue to demonstrate accumulation of callose in the phloem of Vinca plants infected with the sandal spike. Using plants affected by different diseases Gosdziewsk and Perzold (1975), confirmed the observations

made by Hiruki and Dijkstra (1973). The aniline blue method was thus established for detecting differences between the phloem of healthy and MLO affected plants. It cannot be used in isolation because it does not detect MLO. However, callose is formed in degenerating phloem of plants affected by a wide range of diseases and not only by phloem invading organisms alone. The aniline blue technique is thus less specific than the DAPI technique.

2:2:5 Chemotherapy of Diseased Palms Associated with MLO

The mycoplasmas are suppressed by antibiotics of the tetracycline group. These chemicals can therefore be used to distinguish MLO associated diseases from virus diseases. The first report on the effect of antibiotics on yellows diseases was from Japan (Ishie et al., 1967). They applied antibiotics to mulberry dwarf diseased plants and observed that the MLO were partially eliminated. Since then tetracycline therapy has been applied to many MLO associated diseases (Davis et al., 1968; Freitag and Smith, 1969; Igwegbe and Calavan, 1973; Klein and Maramorosch, 1970; Martinez et al., 1970).

Various antibiotics were injected into lethal yellowing diseased coconut palms in Florida (McCoy, 1972; 1973). McCoy (1973), tested oxytetracycline-hydrochloride, tetracycline-hydrochloride, streptomycin-nitrate, potassium penicillin-G and griseofulvin. Remission of symptoms was recorded only in palms injected with the tetracyclines. McCoy (1975), investigated the effect of oxytetracycline dose and the stage of disease development in lethal yellowing infected palms. He reported that doses as small as 50 - 100 mg per tree produced a definite response but doses from 6 - 20 g produced an induced remission of symptoms. The palms treated in the early or pre-yellowing stages of lethal yellowing responded much better than those

in more advanced stages of the disease. Similar results have been obtain by Hunt et al., (1974) and Steiner (1976). Remission of symptoms as a result of antibiotic treatment have been observed as well in the palm species Pritchardia thurstonii, P.pacifica, Trachycarpus fortunei, Arykuryoba schizophylla and Veitchia merrillii (McCoy and Gwin, 1977). McCoy (1976), observed that the level of the antibiotic residue in the coconut milk was always low and that it could not be detected four weeks after treatment.

Tetracycline do not permanently cure MLO diseased plants, but cause temporary remission and symptoms of the disease reappears when no further antibiotic is administered. It has been demonstrated that large scale field application of tetracycline has a definite protective effect on apparently healthy coconut palms in areas of advancing lethal yellowing disease by injecting 3g (apparently healthy trees) or 6g (diseased trees) of antibiotic (McCoy et al., 1976).

McCoy (1976, 1982), reported that direct trunk injection of tetracycline antibiotics was the most effective method in controlling yellows diseases. The criteria for disease remission in plants has not been well defined and chemotherapy not combined with electron microscopy can be misleading in diagnozing MLO infection.

When tetracycline application is followed by MLO destruction in phloem cells of plants as well as remission of disease symptoms, strong evidenve is established for MLO etiology of the disease (Nienhaus and Sikora, 1979).

2:2:6 Attempts to Transmit MLO Associated Diseases of Palms

In initial studies on the lethal yellowing disease of coconut palms in Jamaica, Carter and Suah (1964), noted

that the infectious agent appears to be distributed at random around an initial focus to provide local spread and by wind and convection currents to cause jump spread. The pattern of spread of the disease suggested an airborne vector rather than a soil-borne vector of the pathogen. In the early disease transmission experiments in Jamaica emphasis was placed on the search for air-borne vector.

The transmission trials in Jamaica from 1962 to 1972, were summarized by Johnson and Eden-Green (1978). The insects tested were mainly. Aphididae (aphids); Aleurodidae (whiteflies); Cicadellidae (leafhoppers); Fulgoridae (planthoppers); and Eriophydae (gall mites). In all these experiments, insect proof cages were used which were constructed around the test palms. Insects were collected from diseased palms in the field and introduced into the cages. Although a few transmissions did occur (which could not be repeated), the results of these experiments were generally considered to be negative. It was demonstrated by Heinze et al., (1972 b), that the insect proof cages protected the palms from the disease. Natural infections occurred only after the insect proof cages were removed.

With the discovery of MLO in diseased coconut palms in Jamaica around 1972 (Beakbane et al., 1972; Heinze et al., 1972 a; Plavsic-Banjac et al., 1972), transmission tests were concentrated on Cicadelloidea (leafhoppers) and Fulgoroidea (planthoppers), because most phytopathogenic mycoplasmas with known vectors are transmitted by these insects (Nienhaus, 1976). In field surveys in Jamaica (Schuiling, 1976) and Florida (Howard and Mead, 1980; Woodiel, 1976), several species of Homoptera, in particular Fulgoroidea (planthoppers) were found to be very common on coconut foliage. These planthoppers which are phloem feeders were considered as potential vectors. Waters (1977), demonstrated that these insects were not only visiting the coconut palms but fed on them. He devised a simple method

for the location of stylet tracks.

Eden-Green and Schuiling (1978), tried to transmit the coconut lethal yellowing disease by direct introduction of adult Proarna hilaris Germ (Cicadidae) and Haplaxius crudus (Cixiidae) from diseased areas into test cages and by controlled acquisition feeding of Haplaxius on diseased roots. Over 2400 adult P.hilaris were introduced on 38 test palms and 900 Haplaxius adult from nymphs collected from roots around diseased palms, were transfered to 9 test palms. A further 890 adult Haplaxius were transfered to seven test palms following deliberate acquisition feeding of nymphs on lethal yellowing affected coconut palm roots. The results were negative.

Eden-Green (1979), further tested Cicadellidae by enclosing them on either green or yellow attached fronds of diseased palms for three to four days. They were then incubated for ten to fourteen days on grasses before transfering them to test palms. The species tested were Chlorotettix minima, C.viridius, C.nigromaculata, Graminella sp., Hortensis similis, Poecilocarta histrio, Protalebrella braziliensis and Sanctanus fasciatus. Additional laboratory tests were carried out with C.minima, C.viridius, Spangbergiella vulnerata. Furthermore the planthopper, Haplaxius was injected with an extract from lethal yellowing diseased palms followed by a similar incubation and transmission technique. No transmissions were observed.

Myndus crudus van Duzee (formerly Haplaxius crudus), was experimentally proved to feed on coconut and other palm species such as Veitchia merrillii and Pritchardia sp. (Tsai, 1977, 1980). Tsai (1977), collected M.crudus from diseased palms and fed them on healthy palms. Also partially purified and crude sap from diseased palms was artificially fed to or injected into M.crudus in an sttempt to transmit lethal yellowing. The results were negative.

However, Howard and Thomas (1980) and Howard et al., (1983), successfully transmitted lethal yellowing disease in three of five coconut palms, Cocos nucifera L., five of seven Manila palms, Veitchia merrillii (Becc.) H.E. Moore, and two of three of the palm, Pritchardia thurstonii F. Muell. et Drude, that were planted in cages into which approximately 850 wild planthoppers, Myndus crudus van Duzee, were introduced each month for 34 months. Mycoplasma-like organisms (MLO) were observed in sieve elements in tissues sampled from all palms that developed lethal yellowing disease symptoms, but not in samples from healthy palms. Palms of the same species in the cages in which M.crudus were not introduced remained healthy.

Dabek (1982), experimented with natural and incubated populations of the Cicadellid leafhopper Deltocephalus flavicosta Stal. (Euscelinae). Initially incubated insects on diseased palms failed to provide evidence that D.flavicosta is a vector of coconut lethal yellowing disease. Natural populations of the insects did however transmit MLO associated with phyllody in Catharanthus roseus (L) G. Don (periwinkle), but did not re-transmit the MLO to C.roseus test plants after acquisition feeding and incubation for eight weeks on corn. The transmission tests failed to suggest any relationship between the MLO associated with coconut lethal yellowing and periwinkle phyllody.

3:0 MATERIALS AND METHODS

3:1 Survey on the History of the Lethal Disease of Coconut Palms in Tanzania

The survey on the history of the lethal disease was in the form of a standardized questionnaire (Appendix B), to interview individual farmers in villages where the disease is active. The farmers selected were old people who had been living in the villages for a long time. The main purpose of the survey was to obtain general information about the disease since its first outbreak. No attention was given to coconut growing practices.

The questionnaire included subjects as the presence the lethal disease in the villages; description of the disease symptoms; the possible cause of the disease; the most likely date when the disease appeared in the village and the probable mode and direction of spread of the disease; the possible decrease or increase of the disease incidence and the possible difference between the symptoms observed now and in the past.

An average of ten farmers in the villages of Sotele, Magawa, Kifumangao in Kisarawe District, Mbwera in Rufiji District; Somanga and Pande in Kilwa District and Bagamoyo in Bagamoyo District were selected for interviews. Each farmer was initially interviewed alone but later broader participation of other villagers was encouraged. If a farmer could not remember exactly the date of the first outbreak of the disease, events such as the second World War, famine, pre-independence and post independence periods became useful tools. A total of 165 farmers were interviewed.

3:2 Additional Evidence for a Mycoplasma-like Organism (MLO) Etiology of the Disease of Coconut Palms in Tanzania

Forty bearing palms of the cultivar EAT, varying in age from twenty to sixty years old and showing symptoms of lethal disease were selected at Kifumangao coconut farm of the Kisarawe District Development Corporation. This farm is situated in an area where the lethal disease is very active. The trees were evaluated for severity of symptoms of nutfall, inflorescence blackening and leaf discolouration. On the basis of this evaluation fifteen matching pairs were selected and 10 palms were discarded as unmatchable. Disease stages of the selected pairs ranged from early , moderate to advanced.

Each selected palm was scored for disease severity according to the extent of leaf symptoms, on a scale from 0 (healthy) to 10 (dead). The score for an individual diseased palm was obtained in the following way :
As the number of open leaves in the crown of a coconut palm of the EAT cultivar varies from approximately 30-34, only the first 30 leaves were recorded. Each leaf was given 0-3 points; 0 (green), 1 (initial bronzing), 2 (advanced bronzing) or 3 (totally brown/dry).Where the canopy of a diseased palm had less than 30 leaves, the deficient number were considered to have fallen and therefore recorded as completely brown/dry. A diseased palm would thus have a maximum score of 90 points (when only brown/dry leaves are left) and a minimum of 0 points (when all of the leaves on the palm are green). The total score for each palm was then expressed as a fraction of 90 and placed on a scale 0- 10. The palms were divided into three groups as to severity of symptoms:

Group 1 Early stage of disease; nutfall in progress, 1-2 recently opened inflorescences showing necrosis. The score of leaf symptoms ranged

0 - 2.5

Group 2 Moderate stage of disease; nutfall complete, 2 -4 newly opened inflorescences showing necrosis. The score of leaf symptoms ranged between 2.6 - 4.5.

Group 3 Advanced stage of disease; nutfall complete, all newly formed inflorescences blackened. The score of leaf symptoms was above 4.5.

For each matching pair, one palm was injected with tetracycline hydrochloride and the other palm untreated control. A trunk injection technique used in Jamaica (Hunt et al., 1974), was adopted to administer the tetracycline to palms. A sloping hole drilled almost completely across the base of the trunk was filled with two litres of a 1% aqueous solution of tetracycline hydrochloride (manufactured by Serva, Feinbiochemica, Heidelberg, West Germany), contained in an inverted plastic bottle suspended above the hole (Fig. 7).



Fig. 7 A : Hole boring

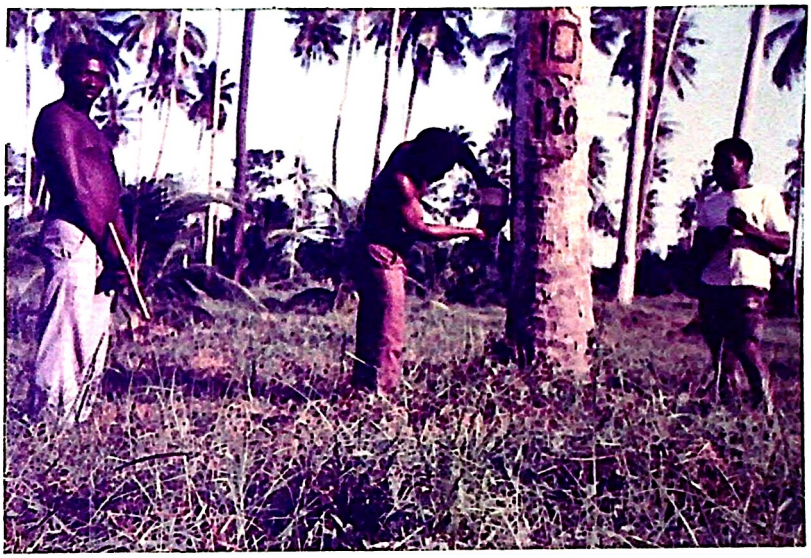


Fig. 7 B Adjustment of the bottle



Fig. 7 C Securing the bottle to the trunk of the palm.

Tetracycline hydrochloride was injected eight times at bi-weekly intervals and records were taken at bi-weekly intervals for both injected and control palms. For each palm the following records were taken : nuts remaining on bunches, the condition of newly opened inflorescences and leaves.

A further thirty non-bearing coconut palms of the hybrid PB 121 (a cross between Malayan Yellow Dwarf and West African Tall) aged between two and three years were selected at Chambezi Agricultural Experimental Station. The diseased palms were evaluated for severity of leaf symptoms as described before. In addition the spear leaf which could be reached in young palms was included in this evaluation; 0 (no necrosis), 1 (initial necrosis), 2 (advanced necrosis), 3 (totally rotten). On the basis of this evaluation fifteen matching pairs were selected in which the symptoms ranged from earliest, moderate to advanced.

Group 1 early stage of disease had a score of leaf symptom ranging between 0 and 2.5

Group 2 moderately advanced stage of disease had a score of leaf symptoms ranging between 2.6 and 4.5.

Group 3 with advanced stage of disease had a score of leaf symptoms ranging between 4.5 and 6.0.

Of each matching pair, one palm was injected with tetracycline hydrochloride and the other palm used as a control. The palms were injected in the same way as the bearing palms. Injecting the young palms with a 1% aqueous solution of tetracycline hydrochloride was proved to be toxic, therefore a lower concentration of the antibiotic was used. Injection was made with one litre of 0.5% solution of the antibiotic nine times at weekly intervals and records were taken at weekly intervals for both injected and non-injected palms. For each palm, records were taken of the

condition of all opened leaves and one or two unopened (spear) leaves.

By analysing the records of successive visits, the progress of nutfall from individual bunches, the blackening of inflorescences and the progress of bronzing and drying of individual leaves was determined for bearing palms. In non-bearing palms, the progress of leaf symptoms and necrosis of the spear leaf was determined.

3:3 Survey, Identification and Population Studies of Putative Insect Vectors.

The survey was restricted to insects of the Order Homoptera, suborder Auchenorryncha (leafhopper and planthopper), as virtually all vectors of MLO-associated diseases are found in this group.

The main coconut growing areas along the coastal belt were visited to find out which members of the Auchenorryncha occur on coconut palms and their relative frequency. The insects observed were collected with small stoppered bottles containing 80% ethanol and separated into different species. They were then sent to CAB International Institute of Entomology in the United Kingdom and identified by Dr. M. R.Wilson.

In observing the relative abundance and seasonal fluctuations, only the common species were used while the rare species were excluded. Six locations were selected: Pongwe in Tanga District, Chambezi in Bagamoyo District where the disease is less active, Kigamboni near Dar es - Salaam, Sotele, Magawa and Kifumangao in Kisarawe District where the disease is very active.

At these locations, the insects were counted at fortnightly intervals on young palms planted in 1981 and 1982. The palms at each location were in one block and

consisted of sixty plants. At five locations, Pongwe, Chambezi, Magawa, Kifumangao and Sotele, the young palms were a mixture of dwarfs, hybrids and talls. Only at Kigamboni was the block consisting of a hybrid PB 121.

3:4 Insect Transmissions of the Lethal Disease

The insects used in the attempt to transmit the lethal disease were restricted to the Order Homoptera, suborder Auchenorrhyncha. In the absence of data on feeding habits and breeding sites of the observed insects occurring on coconut palms, the selection of groups of genera or species of insects to be used in transmission tests was based on morphological features used in identification at CAB International Institute of Entomology and on their relative abundance in the fields.

Preliminary data on the distribution and abundance of different genera or species were obtained in a series of bi-weekly countings on small palms at seven different locations and the more abundant genera or species were then selected for transmission trials.

In attempting to transmit the lethal disease under controlled conditions, the test palms were protected from natural infection by insect proof cages. Four cages measuring 3 m x 3 m x 3 m made of 32 mesh nylon supported by slotted aluminium frames were constructed (Fig. 8). At the bottom of the cages , the mesh was extended about 20 cm into the soil to inhabit the passage of burrowing insects. In addition the cages were dusted at ground level with the insecticide Aldrin 5% to prevent the nylon mesh being destroyed by termites.



Fig. 8 Insect proof bages

Test palms were obtained from Mkuranga nursery where the lethal disease does not occur. Five coconut palms of the cultivar EAT each about thirty months old grown in oil drums which were cut in half were placed in each cage. The palms were irrigated regularly when needed. Weeds were removed from all cages to provide constant contact between the insects and palms. All the insects used for testing were collected with erlenmeyer bottles directly from the foliage of young diseased or adjacent healthy palms. They were then transfered directly to test palms and not given deliberate acquisition feeding.

At the beginning of the experiment starting May 7th, 1984, the insects were relatively abundant. A notable

feature was the preponderance of the species Diostrombus abdominalis (Distant) = D. moerensis Synave syn. nov.; Diostrombus mkurangai sp. nov.; Lydda woodi Muir; Paraphenice mawae sp. nov.; Phenice pongwe sp. nov. and Zorabana vipaku sp. nov.

In the first series of transmission tests D.abdominalis (Distant) was introduced into two cages. An average of 1150 and 1120 insects were introduced each week into the two cages respectively for thirteen weeks. The other insects, D.mkurangai sp. nov.; L.woodi Muir; P.mawae sp. nov.;P. pongwe sp. nov. and Z.vipaku sp. nov. were introduced as a mixture in the other two cages. The average introductions per week for these two cages were 82 and 72 insects for thirteen weeks.

At the end of the insect introduction period, the palms were removed from the cages and placed in an isolated coconut free area (Ruvu Forest Reserve), to observe possible development of disease symptoms.

The procedure for the second series of transmission tests was the same as in the first series. In the first cage a mixture of D.abdominalis (Distant), D. mkurangai sp. nov., were introduced. In the second cage a mixture of P.mawae sp. nov., P. pongwe sp. nov., Kamendaka kordofana Synave, Meenoplus sp. and L.woodi Muir were introduced. In the third cage a mixture of Robigus magawae sp nov., Bandusia erythrostena Walker, Zoraida fuligipennis Muir = Z. nigeriensis Synave syn. nov., Elasmosceles cimicoides Spinola,Diazanus sp. and Z.vipaku sp. nov. were introduced. However, the species B.erythrostena Walker and Zorabana sp. were not abundant during the test period.

It was also considered that conditions within a cage are so different from natural conditions in the open that they could possibly interfere with infection of the test palms. Therefore similar transmission experiments were carried out with test palms planted in the open .

Eight small plots within a dense cashewnut plantation were selected and cleared at Chambezi Agricultural Experimental Station. Twelve 2-3 year old coconut palms of the cultivar EAT were rectangularly planted at 4m x 4 m apart. In addition to the dense growth of the cashewnut trees, a fence made of dry palm leaves was constructed around the test palms with the assumption that this was sufficient to protect these test palms from natural infection. Insects were transfered to these test palms in the same way as to the test palms in cages.

The insects introduced in these experiments were D.abbdominalis (Distant), D.mkurangai sp. nov. repeated thrice; P. mawae sp. nov., P.pongwe sp. nov. L.woodi and Meenoplus sp., repeated twice; Nesodryas antiope Fennah, Amania angustifrons Melichar once; E.cimicoides Spinola R.tortinotatus, Zorabana sp. and K.kordofana repeated twice.

4:0 RESULTS

4:1 Survey of the History of the Lethal Disease of Coconut Palms in Tanzania

The discussions with farmers in the villages surveyed indicated that the outbreak of the lethal disease of the coconut palm is not a recent phenomenon. The farmers became concerned only after the loss of many palms in the 1960's. The discussions further revealed that there appears to be a relationship between the outbreak of the lethal disease and the planting periods of the palms. Therefore the information on the history of the planting of coconut is included in these results as well as the origin of the seednuts used in the different planting periods. The results of the surveys in the four districts are summarized in Table 2 and Appendix C. The results for each of the four districts are presented separately.

Bagamoyo District

Coconut palms have been grown in this district on small farms for a long time. These farms were established along the coast. Large plantations of the crop were started as early as 1870 by the Elza Fathers of the Catholic Mission at Bagamoyo. The importation of seednuts from Zanzibar are recorded in the mission's diaries of 1870.

The palms were first planted around the mission and later large plantations were established at Kimarangombe in 1903 and at Matimbwa in 1912. Some small farms and plantations were started in the villages around the mission. The Elza Fathers distributed the seedlings from their nurseries as early as 1871. When the German administration started there was already a well established coconut industry in the district and the survey did not reveal a special planting effort of the crop during this period.

However, in the 1920's the planting of coconut palms in the district received a new impulse. In this period, the German settlers planted comparatively big coconut estates at Chambezi, Kitopeni, Pimbini, Kidagoni and Gwazi. They obtained seedlings from the Catholic Mission. Therefore most of the coconut palms in Bagamoyo District appear to be offsprings of the palms of the mission whose origin is Zanzibar.

The coconut plantations established by the Elza Fathers did not flourish well without setbacks. Morstatt (1911), reported that many young palms aged between 1-8 years old were dying at Kimarangombe estate. In the mission's diaries of 1916, a loss of 1200 palms in that year alone is recorded. The symptoms of the disorder as described by Morstatt (1911), appears to be the same as those of the lethal disease. This is supported by the information obtained during the survey. The fifteen farmers (aged between 60 and 100 years old) interviewed stated that the disease had been in the area for a long time. In most cases the disease appeared before their birth (information passed on by the former generation). The earliest date mentioned is 1910.

At present the degree of incidence of the lethal disease in the district is low. A few irregular scattered diseased palms can be found at all times and everywhere in the area. The disease appears to be stagnant and does not have an epidemic nature. This explains why the farmers were unable to indicate where the lethal disease occurred first and the pattern of spread.

Kisarawe District

In this important coconut growing district the lethal disease has caused the heaviest losses. Two areas with a high degree of incidence can be distinguished, the area north of the Luhute river including the villages of Binga, Sotele

and Kisiju and the area south of this river including the villages of Kifumangao, Magawa and Msonga.

Most of the farmers interviewed indicated (mainly from information passed on by the former generation) that before the German rule in Tanganyika (1888-1918), there were coconut palms in their district. These were around the Arab settlements along the coast, of which Kisiju was the most important. They also referred to the influence of Sultan Bargash bin Said of Zanzibar who "brought" seednuts to the mainland. This Sultan encouraged the planting of coconut palms (among other crops) and a modest number of seednuts were made available at Kisiju to individual farmers. The farmers at Kisiju claim that these seednuts originated from Mafia Island. However, none of the farmers interviewed considered this Arab influenced coconut planting to have been an important contribution to the coconut industry in the district. The number of palms were too low.

The first coconut planting period of importance was during the German administration. Although the administration lasted from 1888 to 1918, the farmers indicated that most of the planting was done from 1892-1896 and certainly completed before the Maji-Maji up-rising (1905-1907). This is likely because during the Banganazi famine (scrape coconut) in 1907/1908 when the farmers used copra as the principle food, these palms were already 10-15 years old and bearing. The seednuts, which were provided free of charge were distributed by the Swahili or Arab agents (Maakida) of the German administration to small-holder farmers via the chiefs of the village. Sometimes the farmers were forced to plant the coconut palms.

During the discussions, the farmers were of the opinion that the seednuts for planting during this period were imported from Mafia Island. It depended on the authority of the village chiefs on how many of these

seednuts were actually planted. The planting was most successful in the villages of Msonga, Nasibugani and Sanga-Sanga. It is estimated that between 30,000 and 40,000 palms were planted during this period in the area. Quite a few of these palms are still alive but many died due to beetle attack, old age and the lethal disease.

The most important period of coconut planting in the district was between the first and second World Wars with strong emphasis on the late 1920's and early 1930's. Although the British administration encouraged the planting of the crop, it appears that this was mainly a private effort of individual farmers who were not forced. The smallholder farmer became aware of the value of the coconut palm for private use and as a cash crop. However, in some areas the planting of coconuts during this period received a strong boost with the arrival of German settlers.

In the late 1920's and early 1930's, these settlers established comparatively large coconut estates. Around 1930, 400 hectares were established at Kifumangao by C.W. P.Bennent and 1500 acreas were established at Mavunja by H.Schroeder. The seednuts for these plantations were imported from Mafia Island. It was revealed during the survey that many men were forced to work on these plantations in order to earn cash for paying taxes and some seednuts were distributed by the settlers to their workers as an inducement for working. On the other hand the survey made clear that most of the seednuts for the smallholders planted during this period were collected from the existing bearing palms.

The first reference to the outbreak of the lethal disease in the district is from Kerekese. The information passed on by the previous generation indicated that a disease occurred in the palms during the reign of Sultan Bargash of Zanzibar (1870-1888) in an area called Kwewe north east of the village. The farmers claim to be sure about this

event because the people in the area refrained from planting coconut palms in that area during the German and British administrations because of the presence of the disease. In 1967, the Tanzania government encouraged the people in the same area to plant about 30,000 seednuts which were imported from Zanzibar. The lethal disease has reappeared around Kwewe since 1974 but not elsewhere in the Kerekese area. It is likely that the disorder in the late 19th century was the lethal disease. In the palms planted during the German administration, the outbreaks of the lethal disease were reported at Kifumangao North (in the smallholder farms) between 1920 and 1924, at Dagwe in 1925 and Kisiju in 1926. However, these outbreaks made little impact on coconut production and remained isolated. It was in the coconut palms planted after the first World War either with seednuts collected from palms planted before the war or seednuts imported from Mafia Island that the disease really became of economic importance.

Several independent reports indicated that the new groves succumbed to the disease 2-3 years after coming into bearing. At Dagwe this occurred in 1933, at Kifumangao (south and north) both in the smallholder farms and in the settlers farms and at Nyamwage in 1942, at Kisiju in 1943, at Nyamihimbo and Mtongani in 1941/1942 and at Tunduni in 1942.

The outbreak at Dagwe, an isolated coconut growing areas, killed the palms very fast so that by the late 1930's the people started leaving the area. At present no coconut palms can be found there. However, in the nearby settlements of Pehe-Pehe and Ndimni, the lethal disease hardly caused any damage and virtually disappeared. The outbreaks of the lethal disease at Kifumangao/Nyamwage were of moderate consequence as well. The disease moved to the north and wiped out most of the coconut palms, which were planted irregularly to the south of Nagawa village but did not reach the centre of the village. From the outbreak at Kisiju, the disease

spread randomly but remained confined to the area and it is only recently (1984) that a new outbreak was observed at Mavunja north of Kisiju where a dense coconut belt begins

However, the outbreaks of the disease at Nyamihimbo(west of Sanga-Sanga) and at Mtongani (west of Nasibugani), made a great impact on the coconut industry in the area. From Nyamihimbo the lethal disease spread in a north-eastern direction to old Sanga-Sanga, Nyatupa and then south to Pwapwapwa and Mipafu. From Mtongani, the disease spread to the east to Zelele and Mipafu. It is remarkable that in both cases the important coconut growing areas of Sanga-Sanga and Nasibugani were hardly affected. The lethal disease remained on the periphery of the coconut belts, along the low lands of the Luhute river (north of Sanga-Sanga) and the gully between Nasibugani and Sanga-Sanga.

From Mipafu the disease spread southwards along the Luhute river to Msonga where it reached in 1947. The farmers at Msonga village recall that there was an outbreak west of the village as well at Tunduni around 1942. Thus at Msonga the disease came from two directions and caused heavy losses. From Msonga the disease spread in several direction southwards. It moved fastest where coconut groves were near the open land or gullies, to Songarani in 1960 and north as well as west of Magawa in 1960's (Fig. 9). It moved slower into the solid coconut belt north east of Magawa and south west of Mdimni. In the latter area the lethal disease made inroads only recently (1980), and at present it is causing heavy losses.



Fig. 9 Palms killed by the lethal disease at Magawa near Msonga.

In the coconut growing areas north of the Luhute river, the disease was first observed first at Njianne in 1950. The farmers interviewed claimed that the disease originated from Sanga-Sanga (obviously the outbreak at Nyamihimbo). This is not unlikely because the distance between Njianne and Nyamihimbo across the Luhute valley is only eight kilometers. From Njianne the lethal disease spread to the north reaching Mikere in 1958, Mingombe in 1960, Kitomondo in 1960, Sotele in 1962 and Binga in 1972. In the first three villages the coconut palms have been virtually wiped out while in the latter three villages the disease is still rampant killing thousands of coconut palms (Fig. 10).



Fig. 10 Advancing lethal disease at Sotele

Rufiji District

Before the arrival of the Germans in East Africa, coconut palms in the Rufiji delta were not numerous and as in Kisarawe District they were distributed by the Arabs. The planting of the crop on a large scale was started when the Germans arrived. The farmers claimed that this happened as early as 1880. It appears that German traders of the German East African Company rather than personnel of the German administration were involved in these early planting of the crop as Tanganyika came under the German rule only in 1894. One farmer interviewed (claiming to be over 100 years old)came to Mbwera around the turn of the century. He stated that there were already many bearing coconut palms in Mbwera at that time. In any case the planting of the crop was continued during the German rule but completed before the Maji-Maji uprising. All the farmers interviewed at Mbwera claim that the seednuts for planting were imported from

Mafia Island. They were distributed to the smallholder farmers via the chiefs of the village.

As in the Kisarawe District, most of the coconut palms in the district were planted after the first World War. Although the British administration encouraged the planting of the crop, this was a private effort of individual farmers who recognized the importance of the coconut palm. In this district a notable influence of the arrival of the German settlers like in the other forementioned districts was absent. Only one coconut plantation of about 195 hectares was established by the German settler C.W.P.Bennent at Msala around 1928. This is a very isolated area in the northern Rufiji delta near Msala Island. All palms of this plantation have been killed by the disease.

The first outbreak of the lethal disease occurred in Mbwera west in the village of Mtawatawa around 1948. The farmers are sure about the date of this outbreak because they could relate it to a serious famine which occurred in the area after the second World War. One farmer who settled in Mbwera in that year recalls that an earlier outbreak of the disease occurred at Nyanjati (north of Rufiji delta) in 1946. The disease moved from Mtawatawa to Doka but remained stagnant for four to five years, then became active again in the Doka area and spread quickly to the centre of Mbwera village. The disease then became rampant in the whole of Mbwera-West in the 1960's but did not move to Mbwera-East until 1981 although Mbwera-East is only separated from Mbwera-West by a narrow tidal channel. It is assumed that the dense coconut belt of Mbwera-West formed a barrier against infection.

Kilwa District

There are two seriously disease affected areas in the district, Somanga, south of the Rufiji river and Pande

south of Kilwa Kisiwani Island. Both areas are very isolated and were visited once only, hence the results are preliminary.

It appears that in both areas the planting of the coconut palms was done after the first World War although some palms must have existed in the area before. In both areas most of the farmers indicated that the seednuts for planting originated from Mafia Island but also local sources are mentioned.

In Somanga the opinion of the farmers on the date of the first outbreak of the disease varied widely. The years mentioned were 1920, 1945 and in the 1960's although the majority of the farmers claimed that the disease occurred during the campaign for independence. However, it seems likely that the disease became of economic importance in those years while the first outbreaks of the disease may have been earlier.

At Pande the farmers were more consistent in their opinions. The earliest reference to the outbreak of the disease is during the TANU campaign for independence. The majority of the farmers stated that it was shortly after independence. The disease became rampant in the 1970's killing 80% of the original palms to date..

In all of the districts surveyed, the farmers repeatedly commented on the erratic mode of spread of the lethal disease. While the general direction in which the disease moved over an extended period can now be recognized (like in the Nyamihimbo and Mtongani outbreaks), the movement of the disease in certain areas was often unpredictable. Sometimes it jumped over certain areas only to turn around and attack from the other side.

There are reports indicating that the lethal disease disappeared completely from certain areas only to become

active again a few years later. The reason for this behaviour is not understood, obviously there is no relationship between the direction of spread and the prevailing winds. However, there are indications that the lethal disease spreads more quickly through coconut palms established near open spaces like valleys, gullies and along roads. Actually the disease originated from these gullies. Many farmers claimed that the lethal disease made hardly any impact on the older coconut palms planted during the periods of Arab and German rule. The disease started to become serious only when the new coconut palms (post World War I plantings) came into bearing in the early 1940's. The new coconut plantings died much faster than the old stands although complete immunity of the latter was never claimed.

Area	District	Approx.ha under coconut	Villages visited	Present incidence of LD	Year of first report of LD	No. of villagers interviewed
Bagamoyo	Bagamoyo	32000	Sumai Yombo Kiromo Pande Matimbwa Kitunga Mataya Zinga Ukuni Chasimba	low incidence	1911	15
North of Luhute River	Kisarawe	30000	Njianne Mikere Mingombe Kitomondo Sotele Binga Kerekese Kisiju	palms wiped out palms wiped out palms wiped out rampant rampant rampant moderate stagnant	1950 1950 1960 1960 1962 1972 1880 (?) 1926	40

Table 2: Survey of the history for the Lethal Disease in some of the main affected areas of the coastal coconut belt of mainland Tanzania.

Area	District	Approx.ha under coconut	Villages visited	Present incidence of LD	Year of first report of LD	No. of villagers interviewed
South of Luhute River	Kisarawe	15000	Kifumangao Magawa Mdimni Msonga Nasibugani Sanga-Sanga Dagwe	palms wiped out rampant light most palms wiped hardly any LD hardly any LD palms wiped out	1920 - 1924 1960 1981 1946 1942 1941 1933	70
Mbwera	Rufiji	10000		Over 80% of palms wiped out LD still rampant	1948	20
Somanga	Kilwa	4500		Heavily diseased At least 50% of palms wiped out LD still rampant	1920 (?)	10
Pande	Kilwa	5500		Over 80% of palms wiped out. LD still rampant	1954	10

Table 2 : (continued)

4:2 Additional Evidence for a MLO Etiology of the Lethal Disease of Coconut Palms in Tanzania

Bearing Palms

Group 1 In this group of five pairs of palms antibiotic injected in the early stage of the lethal disease, only one injected palm showed complete remission of symptoms. The upward progression of leaf bronzing and leaf fall was arrested and opening inflorescences appeared healthy. The other four injected palms showed temporary remission of symptoms to various degrees. The development of leaf symptoms in the five palms in compared with their controls in Fig. 11.

Initially the progress of leaf symptoms in injected palms was the same as in their controls but later it slowed down and few new affected leaves were produced. There were healthy inflorescences produced regularly at this time but they were often stunted and most turned black 2-4 weeks after opening. There were up to three healthy inflorescences produced after the last injection of the antibiotic.

The slow-down in the progress of leaf symptoms ceased as well at various periods after the last injection of antibiotic and then bronzing and leaf drop advanced again until the death of the palm. The four injected palms with temporary remission of symptoms survived an average of 13 weeks longer than their controls.

Group 2 In this group, injected in a moderate stage of the lethal disease, three injected palms showed temporary remission of symptoms and the other two showed no difference when compared with their controls (Fig. 12).

In palms with temporary remission of symptoms, the rate of leaf bronzing slowed down when compared with their

controls. Some inflorescences appeared healthy but later turned black in less than four weeks after opening. These three injected palms with temporary remission of symptoms survived an average of 9 weeks longer than their controls.

Group 3 In this group antibiotic injected in advanced stage of the lethal disease, no palm showed any remission of disease symptoms. They died at the same time as their controls (Fig. 13).

None of the antibiotic injected palms in this group did produce healthy inflorescences. In most of these palms, injected or controls, no green leaves remained on the palm twelve weeks after the initial injection of the antibiotic. The initial disease status and the development of leaf symptoms in the 15 matching pairs of bearing palms is presented in Table 3.

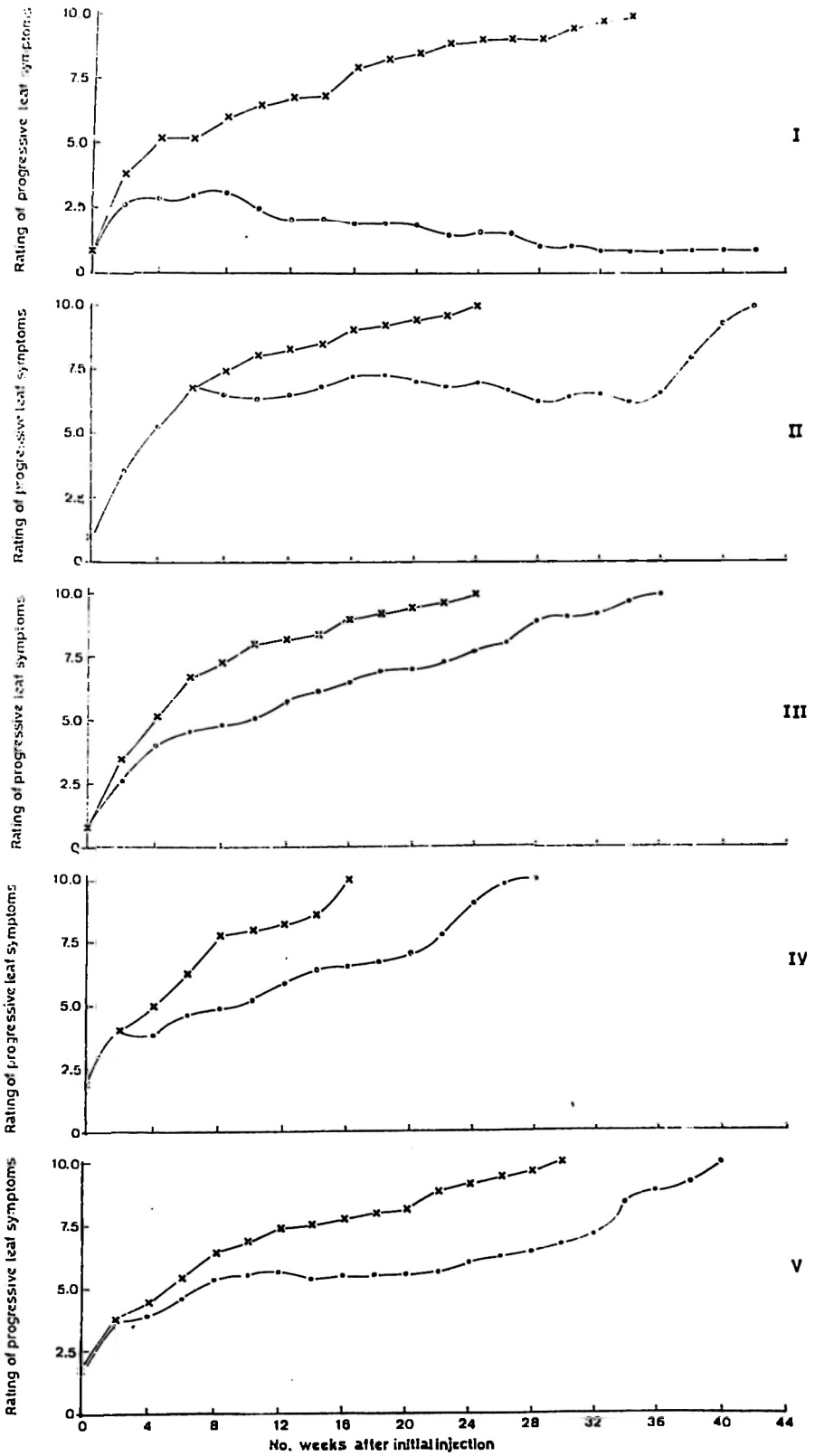


Fig. 11 : Tetracycline – injection trials.

Progressive development of leaf symptoms in bearing Lethal Disease infected coconut palms in early stages of disease. Of five matching pairs (I – V), one palm was injected (-•——•—), the other untreated control (×——×—). Progressive leaf symptoms were rated from 0 (palm healthy, all leaves green) to 10 (palm dead, all leaves completely dry/brown).

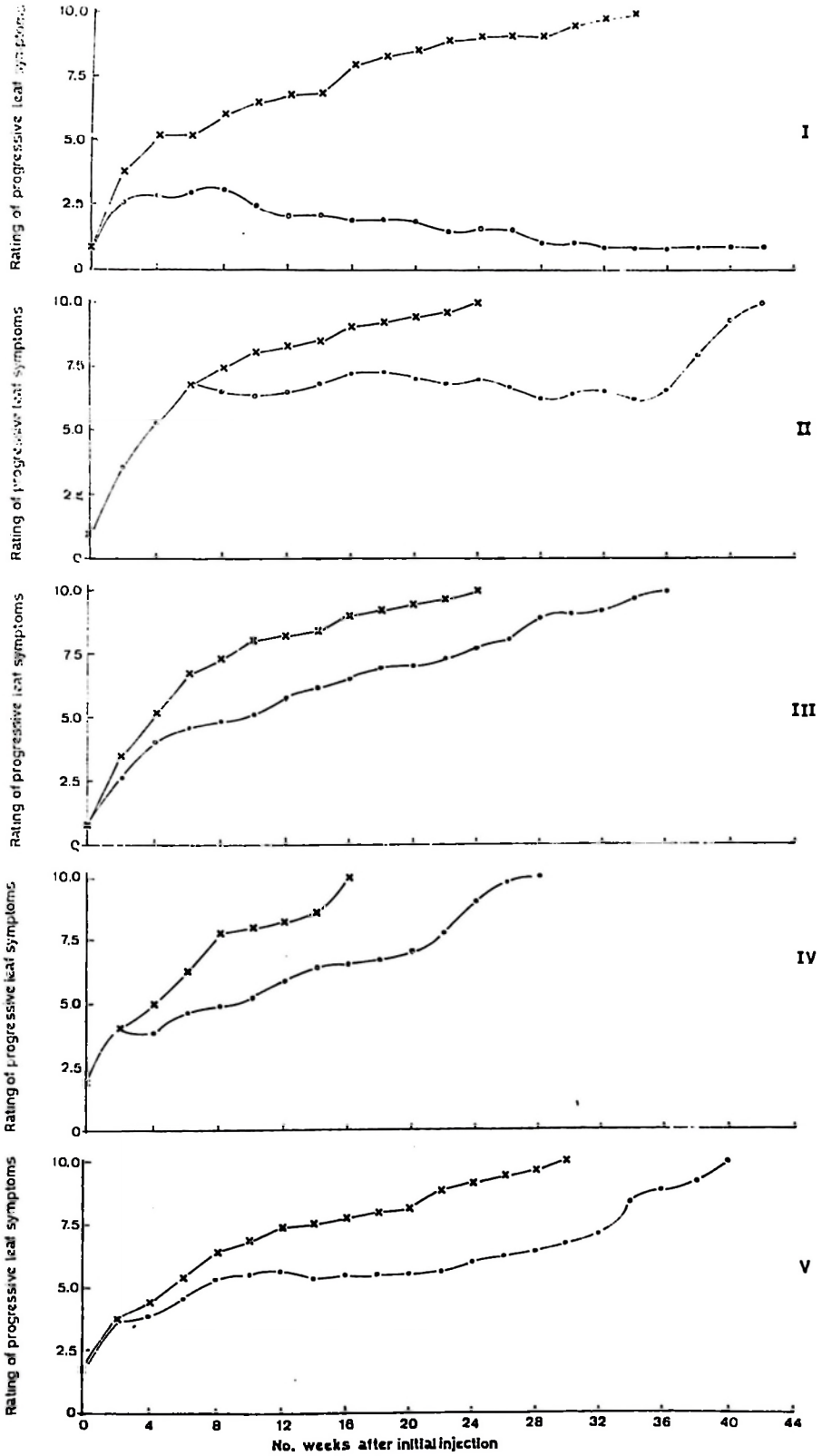


Fig. 11 : Tetracycline – injection trials.

Progressive development of leaf symptoms in bearing Lethal Disease infected coconut palms in early stages of disease. Of five matching pairs (I – V), one palm was injected (–•——•—), the other untreated control (x——x—). Progressive leaf symptoms were rated from 0 (palm healthy, all leaves green) to 10 (palm dead, all leaves completely dry / brown).

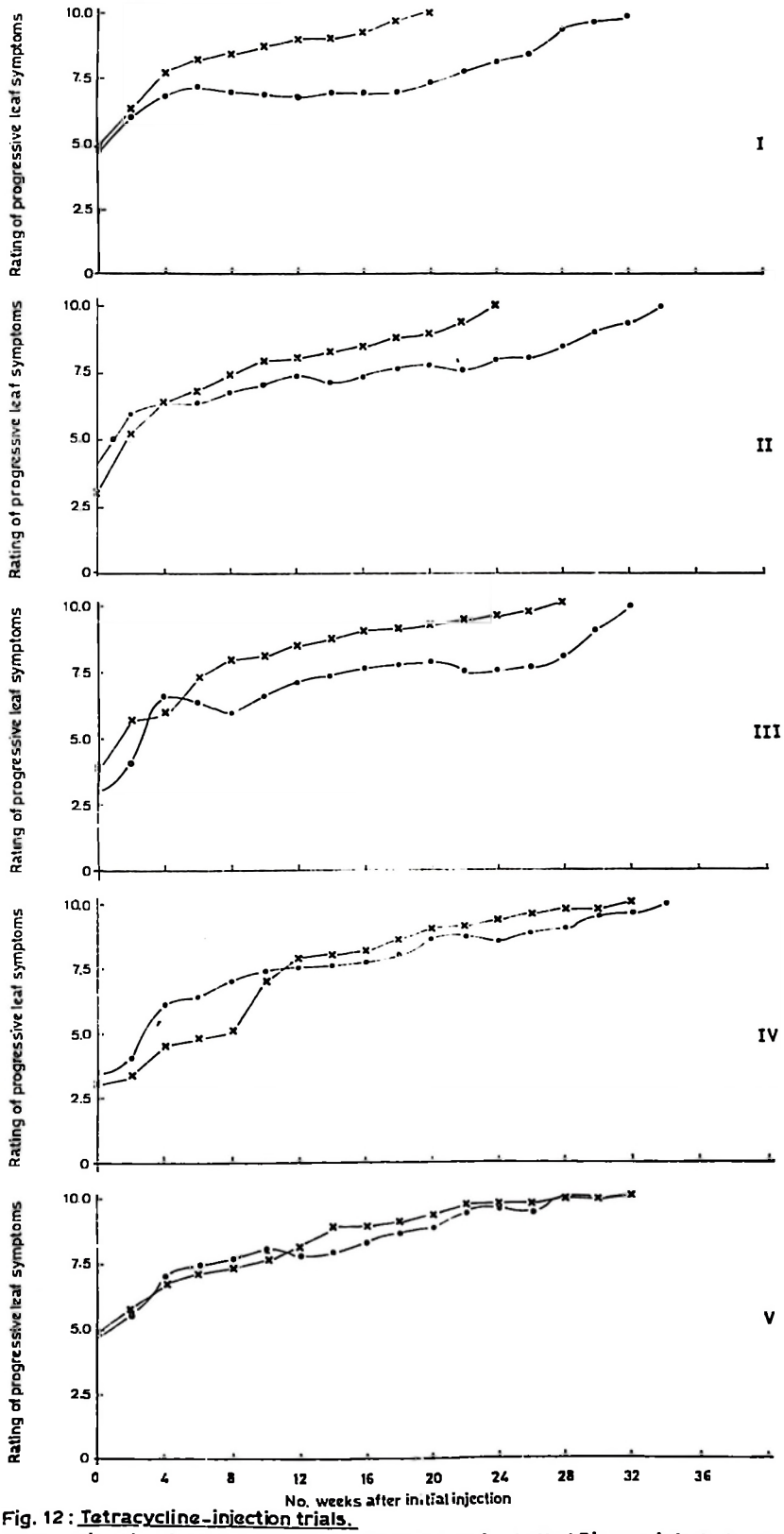


Fig. 12 : Tetracycline-injection trials.

Progressive development of leaf symptoms in bearing Lethal Disease infected coconut palms in moderate stages of disease. Of five matching pairs (I – V), one palm was injected (●——●—), the other untreated control (×——×–). Progressive leaf symptoms were rated from 0 (palm healthy, all leaves green) to 10 (palm dead, all leaves completely dry/br

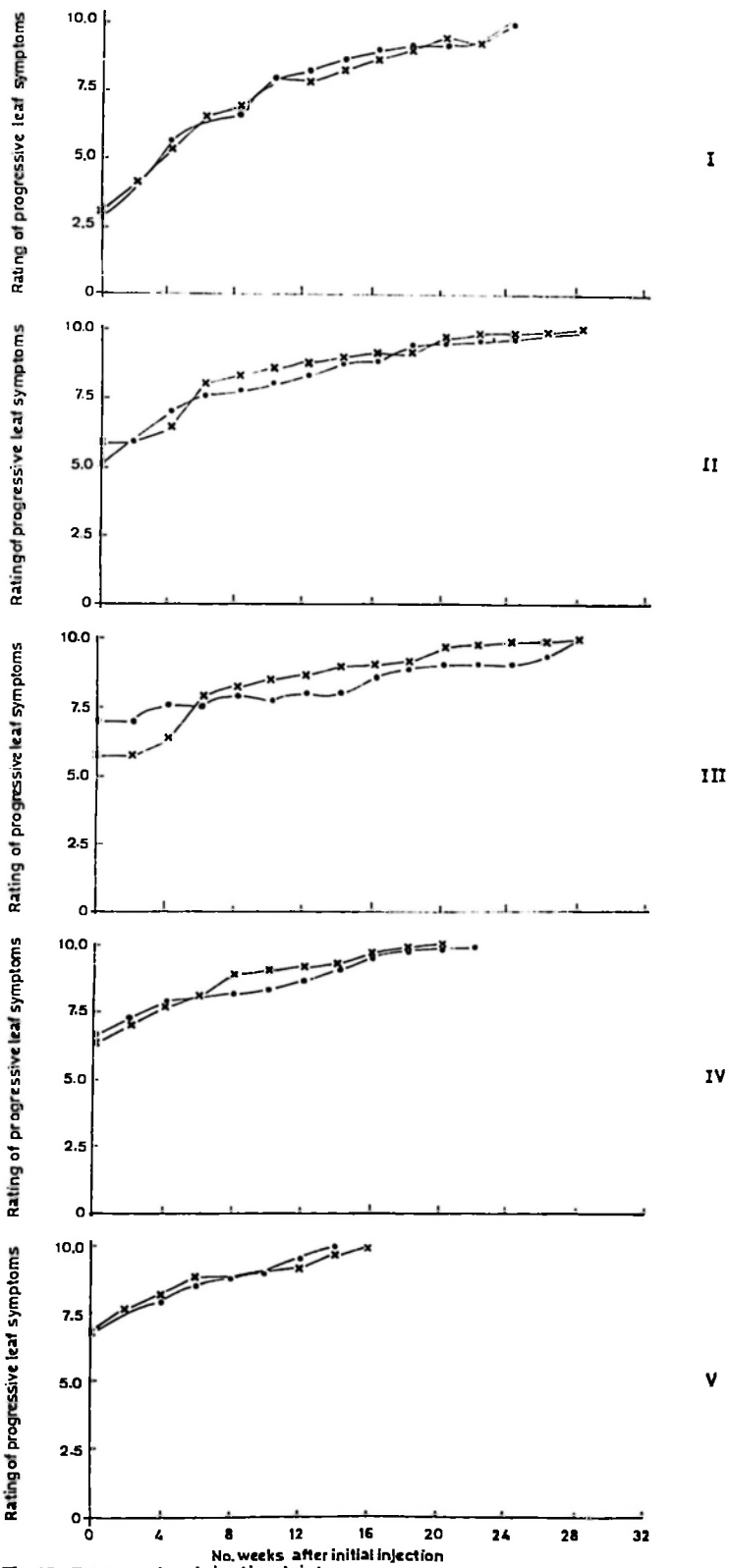


Fig.13 : Tetracycline-injection trials.

Progressive development of leaf symptoms in bearing Lethal Disease infected coconut palms in advanced stages of dis... Of five matching pairs (I–V), one palm was injected (- •——• —), the other untreated control (×——×—). Progressive l... symptoms were rated from 0 (palm healthy, all leaves green) to 10 (palm dead all leaves completely dry / brown).

PALM[a]		BEFORE INJECTION				AFTER INJECTION		RESULTS OF INJECTIONS
		NUT FALL[b]	STATE OF OPEN INFLORESCENCE[c]	NO. OF LEAVES BRONZING	NO. OF GREEN LEAVES	STATE OF INFLORESCENCE[d]	NO. OF GREEN[e] LEAVES	
1	At	-	-	10	24	-	24	Remission
	Au	-	-	2	20	+++	0-98	
2	Bt	+++	-	10	22	+++	0-280	Temporary
	Bu	+	+++	8	23	+++	0-70	remission
3	Ct	-	+	14	22	+++	0-238	Temporary
	Cu	-	+	14	20	+++	0-70	remission
4	Dt	-	+	9	21	+++	0-182	Temporary
	Du	-	+	9	20	+++	0-70	remission
5	Et	+++	++	8	23	+++	0-280	Temporary
	Eu	+++	+++	7	21	+++	0-112	remission

Table 3: Initial disease status and results of injection of 15 bearing , matching pairs of lethal disease infected coconut palms. Tree pairs arranged in order of increasing disease severity at the time injection started.

PALM[a]		BEFORE INJECTION				AFTER INJECTION		RESULTS OF INJECTION
		NUT FALL[b]	STATE OF OPEN INFLORESCENCE[c]	NO. OF LEAVES BRONZING	NO. OF GREEN LEAVES	STATE OF INFLORESCENCE[d]	NO. OF GREEN LEAVES	
1	Ft	+++	+++	8	12	+++	0-210	Temporary remission
	Fu	+++	+++	10	11	+++	0-42	
2	Gt	+++	+++	6	15	+++	0-224	Temporary remission
	Gu	+++	+++	5	16	+++	0-96	
3	Ht	++	++	4	20	+++	0-210	Temporary remission
	Hu	+	+	7	22	+++	0-36	
4	Jt	+++	+++	5	16	+++	0-84	No difference
	Ju	+++	+++	6	16	+++	0-84	
5	Kt	+++	+++	8	11	+++	0-154	No difference
	Ku	+++	+++	9	11	+++	0-96	

Table 3: (Continued)

PALM [a]	BEFORE INJECTION				AFTER INJECTION		RESULTS OF INJECTIONS
	NUT FALL [b]	STATE OF OPEN INFLORESCENCE	NO. OF LEAVES BRONZING	NO. OF GREEN LEAVES	STATE OF INFLORESCENCE [c]	NO. OF GREEN GREEN	
1 Lt	++++	+++	8	5	+++	0-154	No
Lu	++++	+++	9	10	+++	0-56	difference
2 Mt	++++	+++	8	17	+++	0-70	No
Mu	++++	-	7	17	+++	0-70	difference
3 Nt	++++	+++	4	7	+++	0-182	No
Nu	++++	+++	5	6	+++	0-84	difference
4 Pt	++++	+++	7	7	+++	0-56	No
Pu	++++	+++	10	4	+++	0-42	difference
5 Qt	++++	+++	9	10	+++	0-28	No
Qu	++++	-	9	10	+++	0-28	difference

Table 3: (continued)

a A,B,C, etc, identical palm pairs; t-treated, u-control

b Rating scale: -= no nutfall; += 10%; ++ = 11-50%; +++ = 51-99%; ++++= 100% nutfall

c Extent of blackening of the most recently opened inflorescence: - = none; + = 5%; ++ = 6-50%; +++ = 50%

d Extent of blackening of the most recently opened inflorescence, rating as in c.

e Number of green leaves at the end of observation period (366 days). An entry t-1 indicates that there were no green leaves remaining 70 days after injection started.

Non-Bearing Palms

Group 1 These palms were injected with the antibiotic while in early stages of lethal disease. All of the injected palms showed temporary remission of symptoms when compared with their controls. It was observed that the injection of tetracycline antibiotic initially prevented necrosis and rot of the spear leaves almost completely. In three injected palms the spear leaves remained completely healthy for 21 weeks while the two other palms developed slight necrotic lesions in the pinnae of the spear leaves within two weeks after the first injection of the antibiotic. However, these lesions did not enlarge. In all of the injected palms, the spear leaves did not open, thus the number of green leaves in the canopy did not increase. They started drying up between 21 and 25 weeks and completely collapsed between 25 and 30 weeks after the first injection.

In the five control palms necrosis in the spear leaves commenced within 3-4 weeks after the first recording and progressed into an advanced rot within 4-6 weeks. The progress of symptoms in the open leaves of the injected palms slowed down when compared with their controls but did not cease altogether. In the five injected palms, all open leaves were completely brown/dry betwee 23 and 32 weeks after the first injection while in the control palms this happened between 13 and 24 weeks. The progress of leaf symptoms in injected palms and their controls is presented in Fig. 14.

Group 2 The palms were antibiotic injected while in moderate stages of the lethal disease. Of the five injected palms, three showed temporary remission of symptoms and two did not show remission of symptoms when compared with their controls. In the three remitting palms, the spear leaves were unaffected at the time of first injection and the

tetracycline antibiotic prevented necrosis for a period of 16 to 25 weeks after the first injection. However, afterwards their total collapse was fast between 20-26 weeks after the first injection. In the corresponding control palms necrosis in the spears appeared 2-3 weeks after the first recording and progrssed to an advanced rot in 4-5 weeks.

In the three remitting palms the progress of leaf symptoms slowed down but did not cease. All leaves were totally brown/dry between 27 and 28 weeks after the first injection compared with 18-24 weeks in their controls. In two non-remitting injected palms the spear leaves showed moderate necrotic lesions at the time of first injection. It was observed that the tetracycline antibiotic did not prevent further necrosis of the spear leaves and they were totally rotten five weeks after the first injection like their controls. The progress of symptoms in the open leaves did not slow down either. All leaves on these two palms were completely brown/dry 2o-22 weeks after the first injection just like their controls. The progress of leaf symptoms in injected palms and their controls in this group is presented in Fig. 15.

Group 3 The palms were antibiotic injected in advanced stages of the lethal disease. These five injected palms did not show any remission of symptoms. The period from the first injection and recording to complete collapse of the palms ranged from 80-140 days and no difference was observed when compared with their controls (Fig. 16).

The initial disease status and results of antibiotic treatment in the 15 non-bearing palms are represented in Table 4.

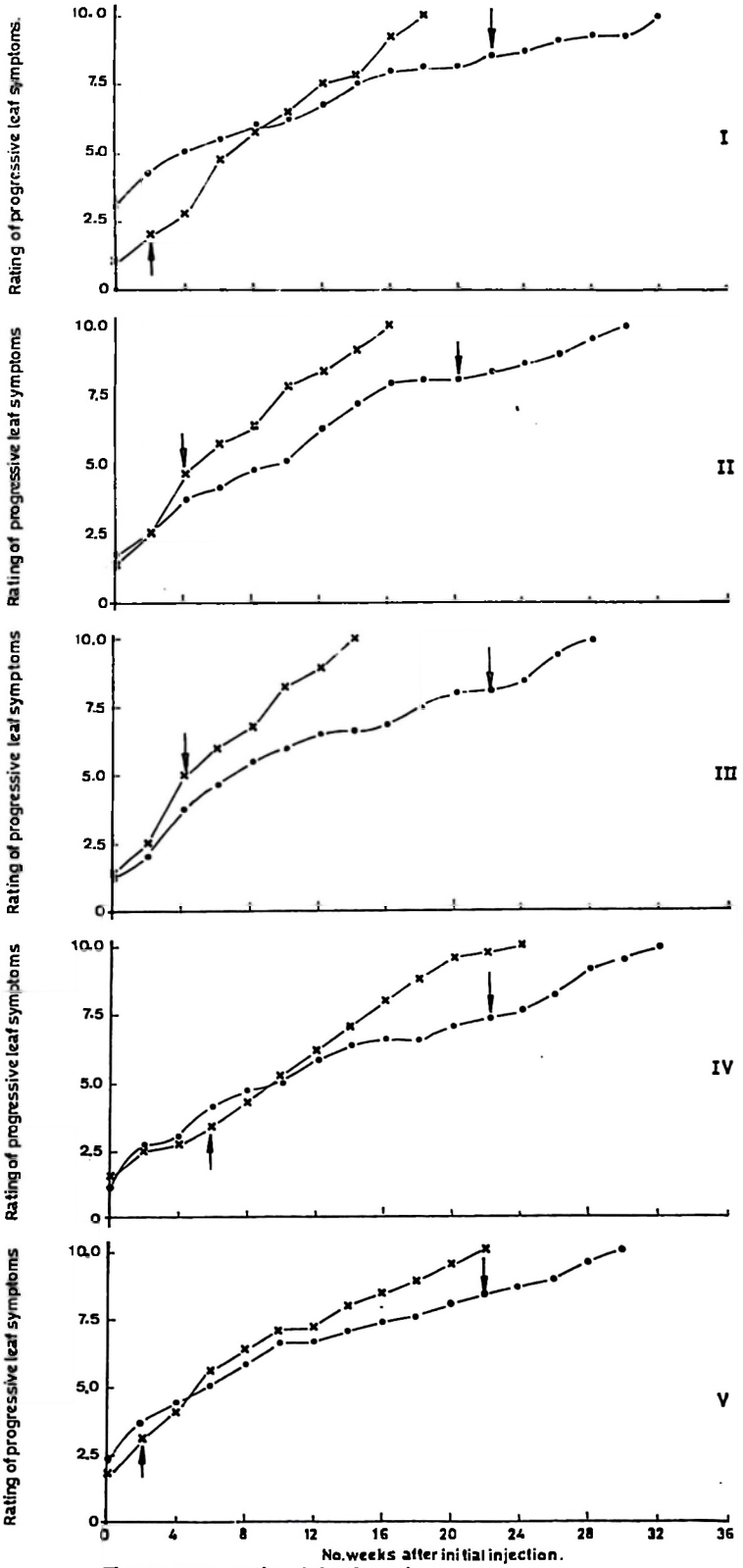


Fig.14 : Tetracycline injection trials.

Progressive development of leaf symptoms in non-bearing Lethal Disease infected coconut palms in early stages of disease. Of five matching pairs (I–V), one palm was injected (•——•—), the other untreated control (×——×—). Progressive leaf symptoms were rated from 0 (palm healthy, all leaves green) to 10 (palm dead, all leaves completely dry/brown). Necrosis in spears starts.

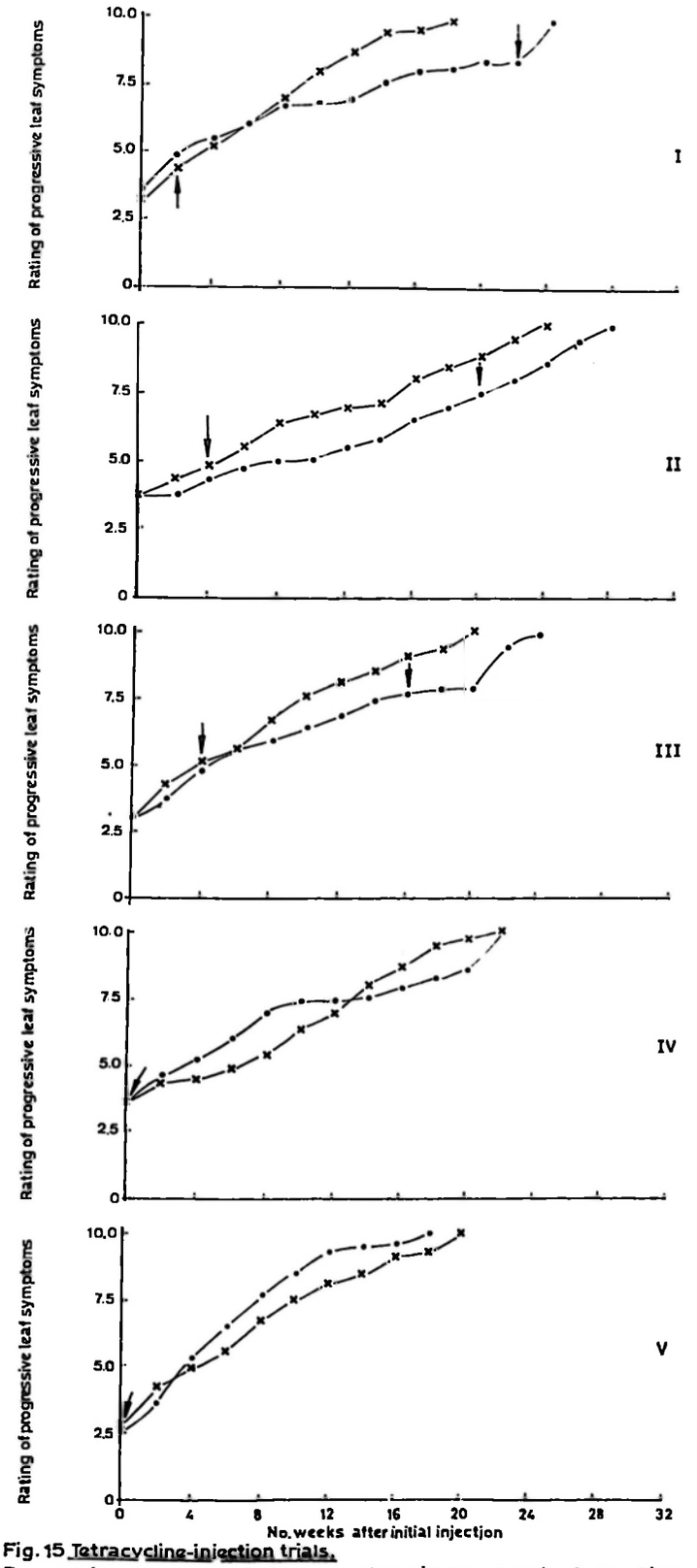


Fig. 15 Tetracycline-injection trials.

Progressive development of leaf symptoms in non-bearing Lethal Disease infected coconut palms in moderate stages of disease. Of five matching pairs (I–V), one palm was injected (- •—— •–), the other untreated control (—×——×—). Progressive leaf symptoms were rated from 0 (palm healthy, all leaves green) to 10 (palm dead, all leaves completely dry/brown).
↑ Necrosis in spear(s) starts

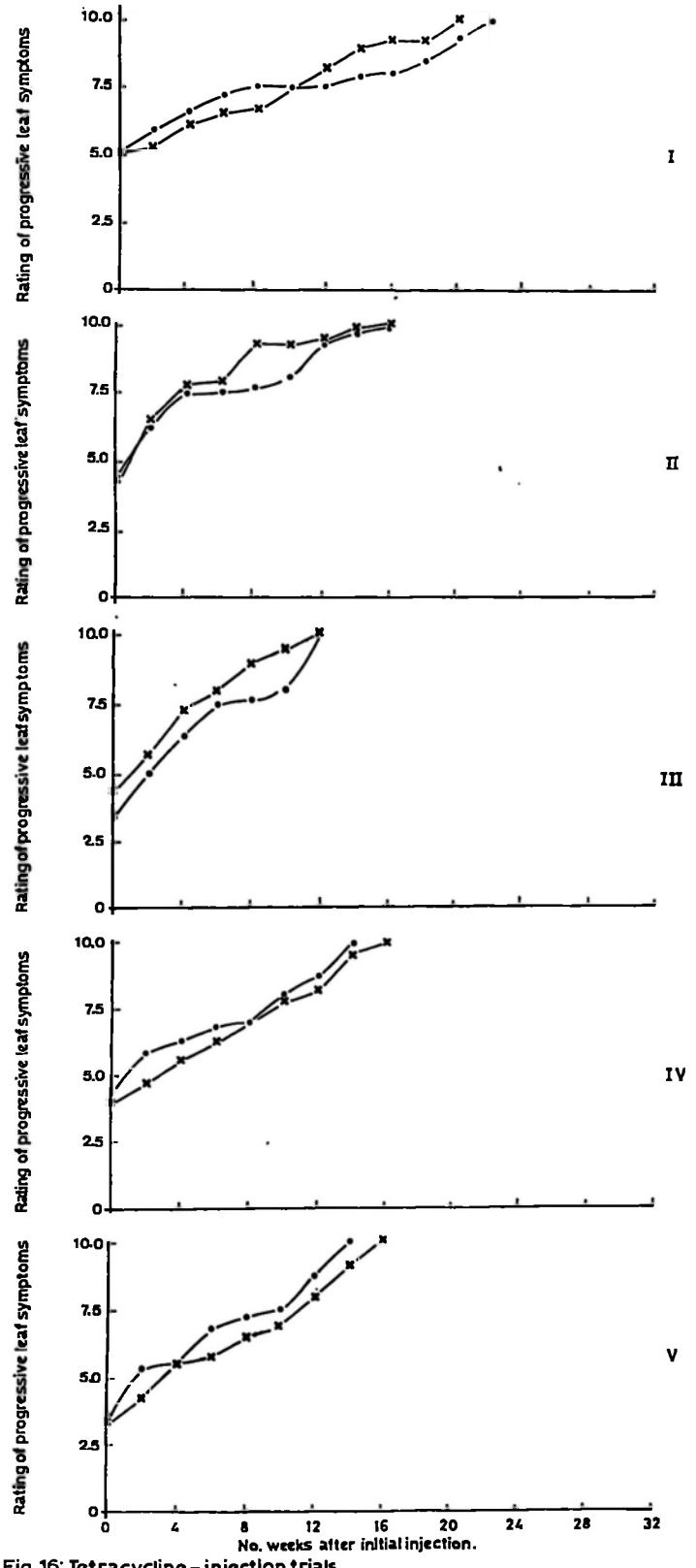


Fig. 16: Tetracycline – injection trials.
Progressive development of leaf symptoms in non-bearing Lethal Disease infected coconut palms in advanced stages of disease. Of five matching pairs (I – V), one palm was injected (– • —— • –), the other untreated control (—x——x—). Progressive leaf symptoms were rated from 0 (palm healthy, all leaves green) to 10 (palm dead, all leaves completely dry/brown). Necrosis in spear already present at initial injection / scoring.

PALM[a]		BEFORE INJECTION			AFTER INJECTION		RESULTS OF INJECTIONS
		START OF SPEAR LEAF[b]	NO. OF BRONZING	NO. OF GREEN LEAVES	START OF SPEARLEAF[c]	NO. OF[d] GREEN LEAVES	
1	At	-	7	14	+++	0-203	Temporary
	Au	-	3	16	+++	0-112	remission
2	Bt	-	4	19	+++	0-175	Temporary
	Bu	-	2	22	+++	0-147	remission
3	Ct	-	4	13	++	0-203	Temporary
	Cu	-	5	13	+++	0-84	remission
4	Dt	-	4	19	+++	0-175	Temporary
	Du	-	2	22	+++	0-147	remission
5	Et	-	4	14	++	0-154	Temporary
	Eu	-	6	16	+++	0-140	remission

Table 4: Initial disease status and results of injection of 15 non-bearing matching pairs of lethal disease infected coconut palms. Tree pairs are arranged in order of increasing disease severity at the time injection started.

PALM[a]	BEFORE INJECTION			AFTER INJECTION		RESULTS OF INJECTION
	START OF SPEAR LEAF[b]	NO. OF LEAVES BRONZING	NO. OF GREEN LEAVES	START OF SPEAR LEAF[c]	NO. OF GREEN LEAVES[d]	
1 Ft	-	7	11	++	0-168	Temporary
Fu	-	3	16	+++	0-96	remission
2 Gt	-	9	9	+++	0-196	Temporary
Gu	-	7	8	+++	0-135	remission
3 Ht	-	5	6	+++	0-168	Temporary
Hu	+	5	9	+++	0-77	remission
4 Jt	++	4	6	+++	0-154	No
Ju	++	6	7	+++	0-98	difference
5 Kt	++	5	8	+++	0-140	No
Ku	++	5	9	+++	0-84	difference

Table 4 : (continued)

PALM[a]		BEFORE INJECTION			AFTER INJECTION		RESULTS OF INJECTION
		START OF SPEAR LEAF [b]	NO. OF LEAVES BRONZING	NO. OF GREEN LEAVES	START OF SPEAR LEAF [c]	NO. OF GREEN LEAVES[d]	
1	Lt	+++	6	8	+++	0-126	No difference
	Lu	+++	7	6	+++	0- 91	
2	Mt	+++	4	9	+++	0- 98	No difference
	Mu	+++	5	9	+++	0- 91	
3	Nt	+++	4	7	+++	0- 91	No difference
	Nu	+++	6	7	+++	0- 91	
4	Pt	+++	6	7	+++	0- 70	No difference
	Pu	+++	6	7	+++	0- 63	
5	Qt	+++	7	6	+++	0- 42	No difference
	Qu	+++	7	6	+++	0- 42	

Table 4 : (continued)

a A,B, C, etc identical palm pairs; t-treated; u- control

b Extent of necrosis in spear leaves - = none; + = 5%; ++ = 6-50%; +++> 50%

c Extent of necrosis in spear leaves. Rating as in b

d Number of green leaves at end of observation period (224 days). An entry 0-77 indicate that there were no green leaves remaining after 77 days when injection started.

4:3 Survey, Identification and Population Studies of Putative Insect Vectors of Lethal Disease

The Homoptera, Auchenorrhyncha recorded on coconut palm foliage were almost all Fulgoroidea (planthoppers). Twenty six different insects which belong to this superfamily were identified some as far as to species and the others to genus. There were only three species recorded on coconut which belong to the superfamily Cicadoidea.

It was not ascertained whether these species were feeding or only resting on coconut palm foliage because of the absence of equipments required to carry out this work. However, the insects were often observed in a typical feeding position with the proboscis pointed down to the leaf surface and it was assumed that they do feed on coconut palm foliage. This is supported by the work carried out in Jamaica where all fulgorids observed on coconut palm foliage were found to be phloem feeders (Waters, 1977).

Among the species recorded only two, Amania angustifrons (Melichar) and Nesodryas antiope Fennah appear to be breeding on coconut palm foliage as nymphs of all stages were observed regularly. The search of the breeding sites of the other species was not done but it is known that the nymphs of most fulgorid species feed on fungi or decaying vegetation in the soil or roots of grasses (Eden-Green, 1978; Eden-Green and Schuiling, 1978).

The twenty nine Auchenorrhyncha species recorded on coconut palm foliage are listed in Table 5. The eleven species marked incidental (inc.) were observed only once or a few times. They were extremely rare and of little consequence in the search for the vector of the lethal disease. They were not included in the transmission trials. For each of the common species the relative abundance, the number of counting sites where they occur and the emphasis

they receive in the transmission trials are outlined in Table 5. A simple description of these common species as observed on coconut palm foliage in the field in given.

Diostrombus (Derbidae)

Three species of this genus were identified. The most common was Diostrombus abdominalis (Distant)= D.moerensis Synave syn. nov. The species has an orange/red thorax and proboscis and paired black spots on the dorsal side of the abdomen. It was very abundant on all counting sites, in particular at Chambezi and Kigamboni. At three counting sites, Chambezi, Kifumangao and Kigamboni, the peaks in its population were observed twice during the driest months of June 1984 to October 1984 and then December 1984 to February 1985. At Magawa, only one peak in its population was observed during the dry months of June 1984 to October 1984 (Fig. 17). Higher numbers of this species were counted on young palms in early stages of lethal disease (especially on lower, bronzing/yellowing fronds) when compared with adjacent healthy palms. However, when symptoms in such diseased palms progressed the number of insects decreased and more were counted on the adjacent healthy palms. This aggregation on yellow or bronzing leaves (not necessarily related to the disease) is a common phenomenon in the Homoptera.

The other two Diostrombus spp., were identified as D.mkurangai sp. nov. and D.schuilingi sp. nov. D.mkurangai sp. nov. is comparatively big, light green to orange coloured with V-shaped ridges on the proboscis. It was abundant at all counting sites but far less numerous than D.abdominalis. The peaks in its population varied at five counting sites. At Chambezi and Kifumangao, only one peak was observed between January 1985 to March 1985; at Kigamboni two peaks were observed, April 1984 to June 1984 and then

fluctuating peaks between November 1984 to May 1985; at Magawa three peaks were observed, April 1984 to August 1984, December 1984 to February 1985 and March 1985 to June 1985; at Sotele two peaks were observed, June 1984 to September 1984 and then fluctuating peaks between December 1984 to June 1985 (Fig. 18).

D.schuilingi is dorsally pitch black, ventrally with white bands and red pigmentation. It does not occur at all sites and is rare.

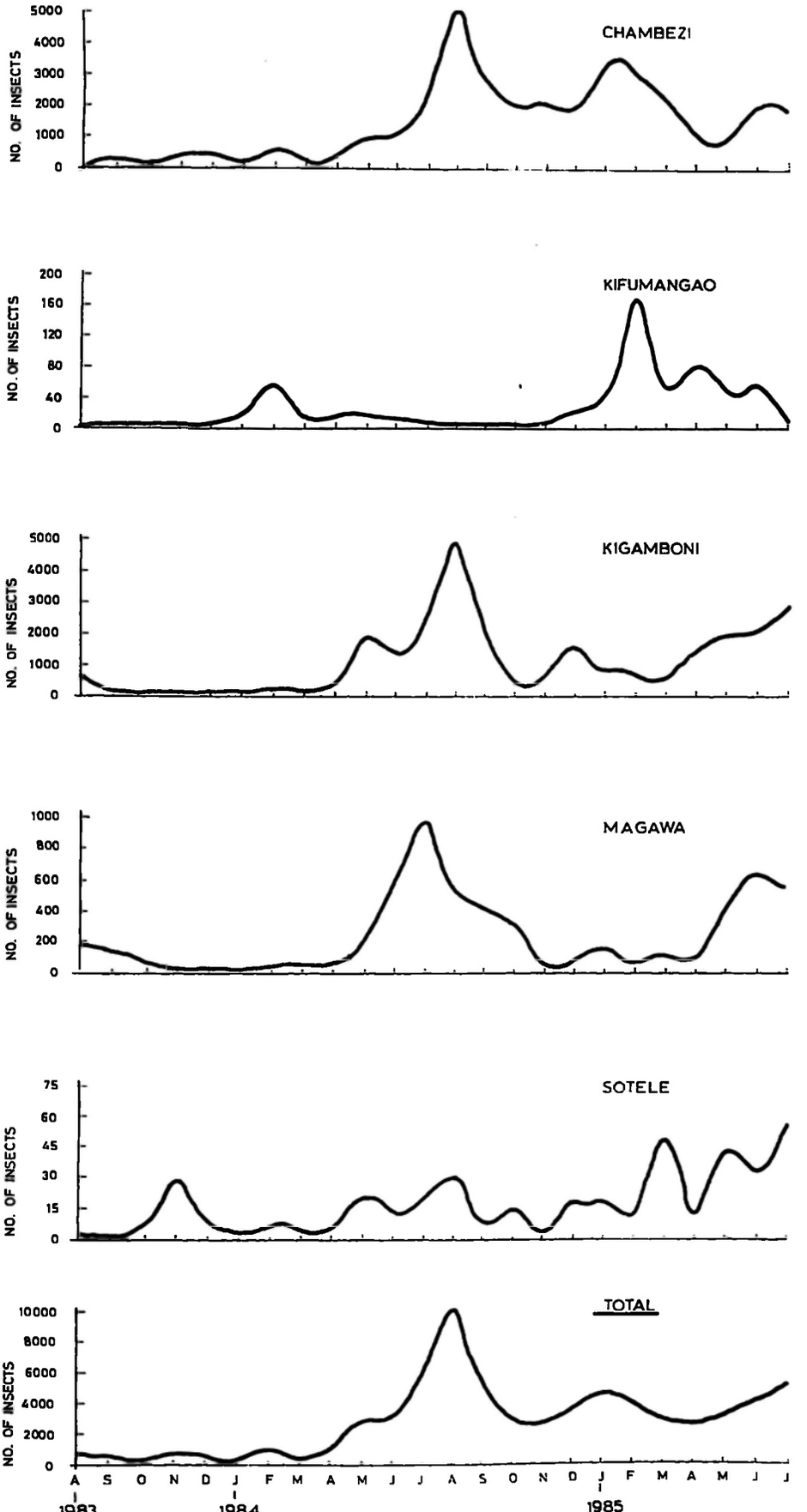


Fig 17 : Fluctuation in adult populations of Diostrombus moerensis at five locations. Insects were counted at fortnightly intervals but presented as monthly totals

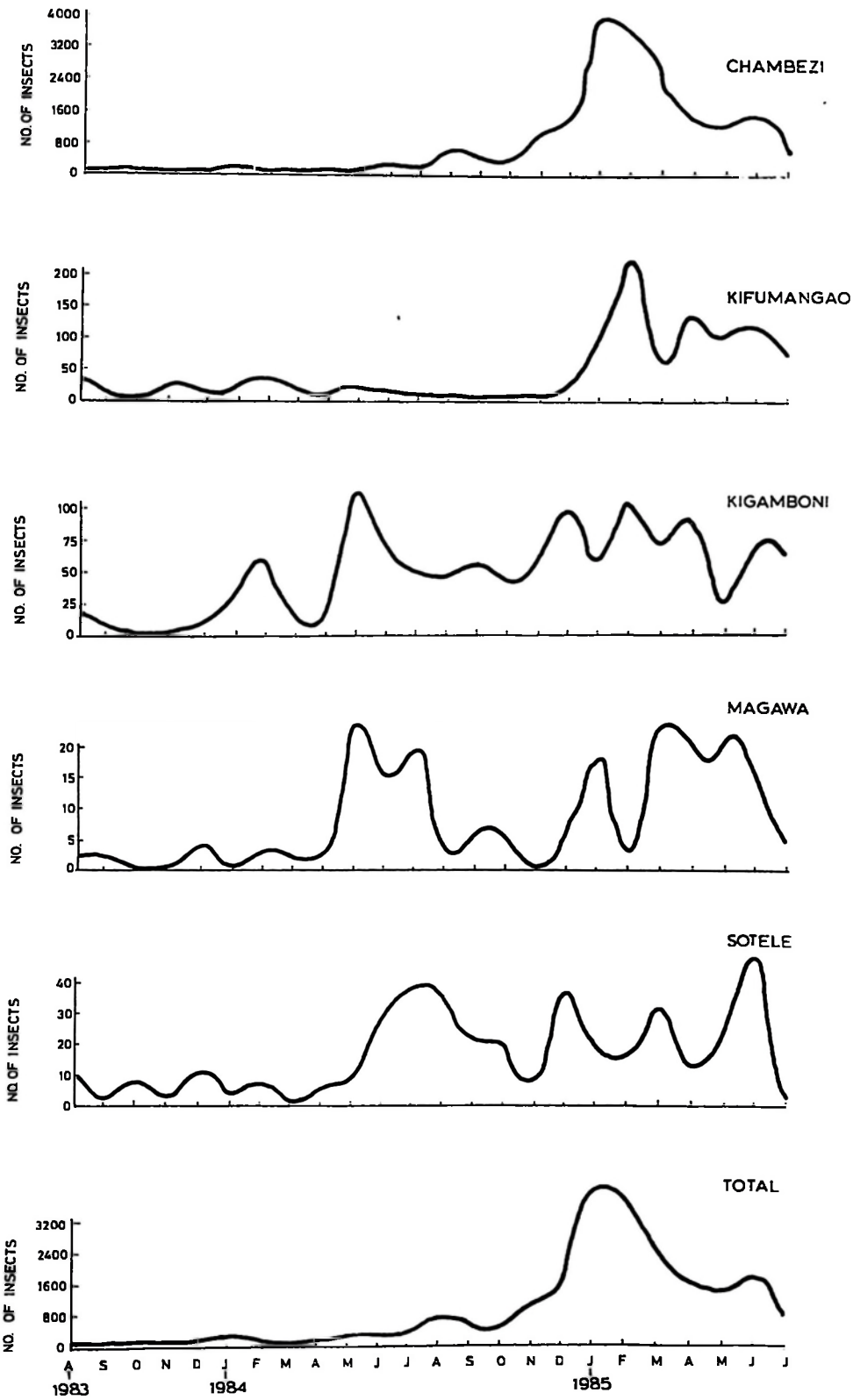


Fig. 18. Fluctuation in adult populations of Diostrombus sp. in five locations
Insects were counted at fortnightly intervals but presented as monthly totals.

Robigus (Derbidae)

This derbid species, was identified as Robigus [illegible] nov. It has orange bands on white forewings, red [illegible] and front legs and large flat protrusion on the [illegible]. It occurred at all counting sites (but only regularly at five) but was not common. At five counting sites where the species occurs regularly, peaks in its populations were observed. At Chambezi during December 1983 to February 1984 and December 1984 to February 1985. At Magawa the peaks were observed from May 1984 to August 1984 and October 1984 (Fig. 19).

Amanin annustifrons (Melichar) (Dersidae)

This is a small pale yellow fulgorid with light [illegible] almost colourless wings. Like Mesocryas antiqua, they appear to be the only [illegible]rhynchous species breeding on coconut palm foliage as nymphs of all stages were regularly found. The species occurred at all sites and was common. Population peaks were observed at Chambezi between October 1984 and January 1985, at Magawa between November 1983 and January 1984; at Sotele between November 1983 and January 1984 (Fig. 2[illegible]).

Lyoda woodi Muir (Derbidae)

This dark coloured fulgorid has yellow spots and bands on the abdomen. It was recorded at all seven counting sites but regularly at five. The species was common. Peaks in its populations were observed during the rainy months. At Chambezi, Mifumangao and Kigamboni the peaks occurred between March 1984 and June 1985; at Magawa the peaks occurred during March 1984 and June 1984 and then March 1985 to June 1985; whereas at Sotele the peaks occurred during March

1985 and June 1985 (Fig. 21).

Phenice/Paraphenice (Derbidae)

These two similar fulgorids species which could not be separated in the field were identified at CAB International Institute of Entomology as two separate species belonging to separate genera, Paraphenice mawae sp. nov. and Phenice pongwe sp. nov.

Under the stereomicroscope the two species could be separated. Paraphenice sp. has red-brown mottled wings while Phenice sp. has wings with dark-red veins. The species were recorded at all counting sites and were rather abundant everywhere. In the transmission trails they are tested as one species.

Kamendaka kordofana Synave (Derbidae)

This is a small grey-white fulgorid with reticulate fore wings and colourless hind wings. The species is abundant at Chambezi only.

Zoraida (Derbidae)

Zoraida is known to be a large genus in Africa. On coconut in Tanzania, the species was identified as Zoraida fuligipennis Muir = Z.nigeriensis Synave syn. nov. It is a red brown fulgorid with long fore wings and reduced hind wings. The species was observed regularly at four of the seven counting sites only but was not common.

Nesodryas antiope Fennah (Derbidae)

This species was first described from coconut palms in Mauritius. A subspecies N.a.seychelles was described from coconut in the Seychelles. In continental Africa it was not

formerly recorded. The species looks very much like A. angustifrons but completely without spots or pigmentation. Like A.angustifrons it breeds on coconut palm foliage. The two insects could not be seperated in the field and were counted and included in the transmission trials as one species(Fig. 20).

Meenoplus (Meenoplidae)

This species was identifed only as far as Meenoplus sensu latu, even the genus being uncertain. The family Meeneplus is currently under investigation by Dr.M.R.Wilson of CAB International Institute of Entomology. The species is a blue grey fulgorid occurring at six of the seven counting sites but not common.

Elasmosceles cimicoides Spinola (Lophodidae)

This is a dark red-brown fulgorid which can be easily distinguished because it has the shape of a horse-shoe. The front legs are flattened. It was recorded at six of the seven counting sites but rare. The peaks in its population as observed at five counting sites are shown in Figure 22.

Diazanus (Nogodinidae)

This species was only described as far as the genus. It is a large dark brown, delta-shaped fulgorid. It occurred at five of the seven counting sites and was not common.

Bandusia erythrostena (Cercopidee)

This is the only Auchenorrhynchus species on coconut palm foliage in Tanzania not belonging to the superfamily Fulgoroidea but to the superfamily Cicadoidea. It is a red

brown beetle-like froghopper with a balloon-like progress on the head. The wings of the males are lighter than those of the females. The species was recorded at all counting sites and rather abundant. It was particularly numerous at Chambezi, Kifumangao, and Sotele. Population peaks were recorded during June/July 1984, and the number declined steeply.

The total number of the more common species counted on palm foliage at the seven counting sites is given in Table 6. At these counting sites, no particular hybrid or variety showed consistently higher numbers of insects.

At Chambezi, there was a considerable increase in the number of insects after the tall palms remaining from the old stand were removed.

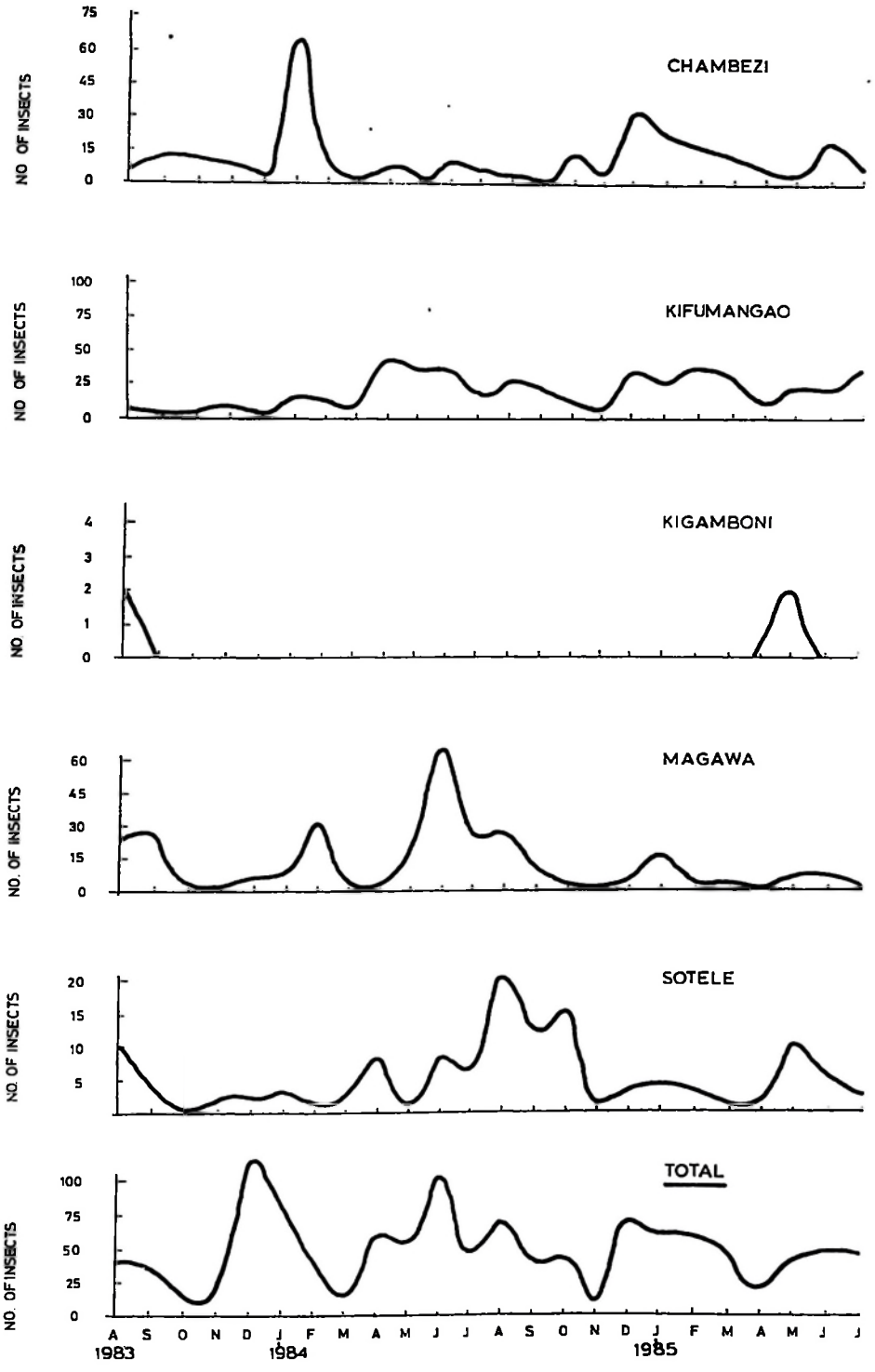


Fig. 19 : Fluctuations in adult populations of Robigus sp. at five locations
Insects were counted at fortnightly intervals but presented as monthly totals.

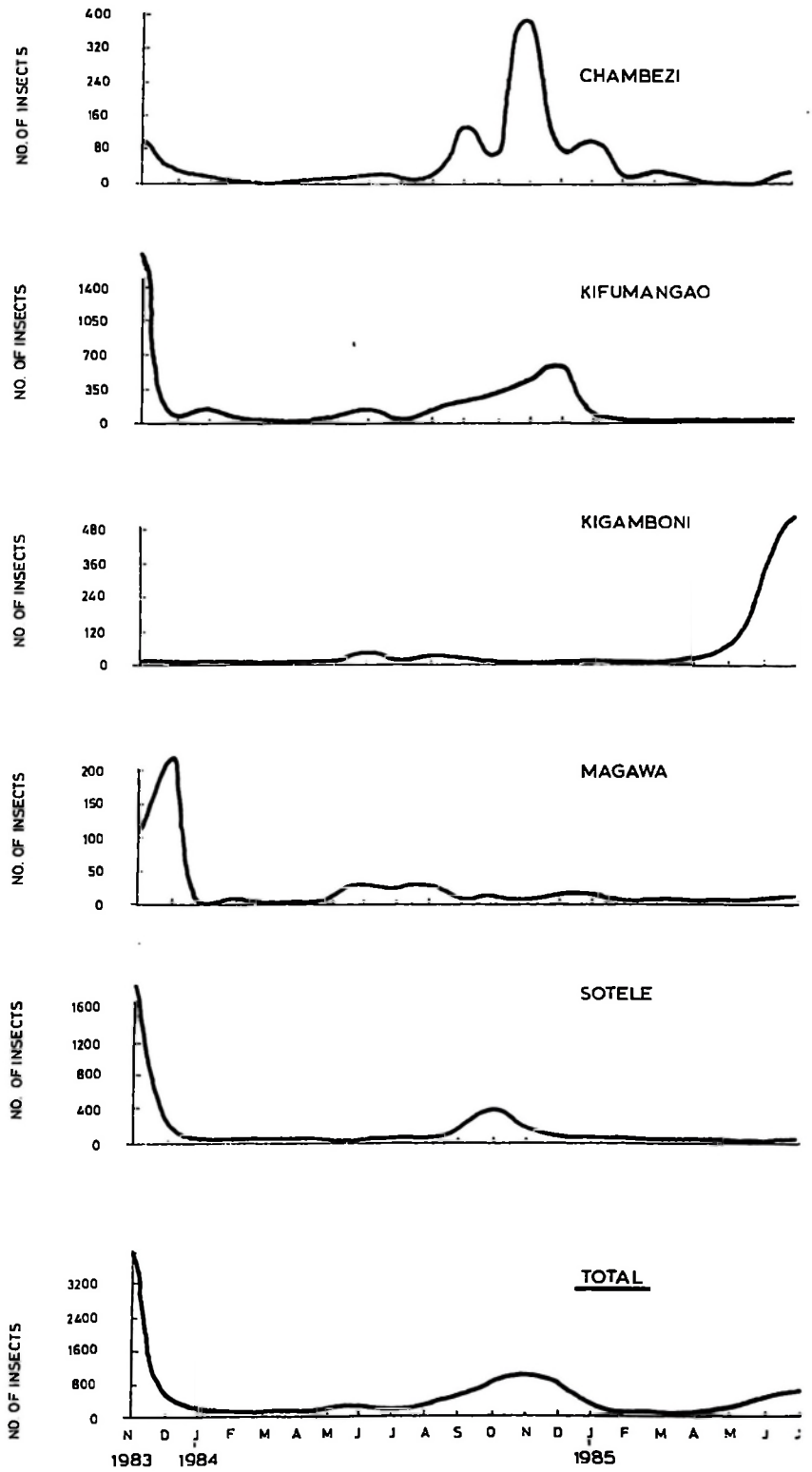


Fig.20: Fluctuations in adult populations of Amania sp. and Nesodryas sp. at five locations
Insects were counted at fortnightly intervals but presented as monthly totals

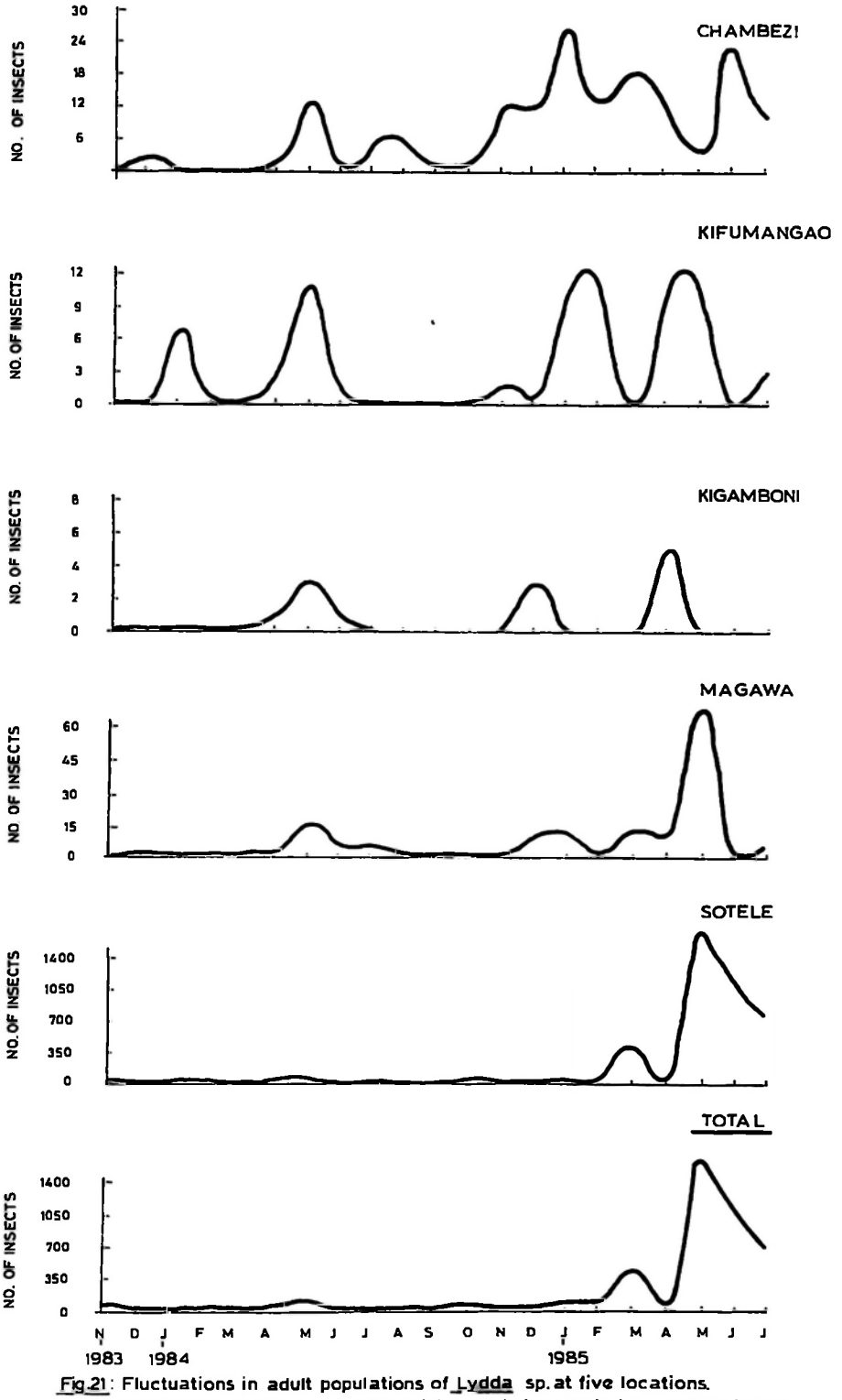


Fig.21: Fluctuations in adult populations of Lydda sp. at five locations. Insects were counted at fortnightly intervals but presented as monthly totals.

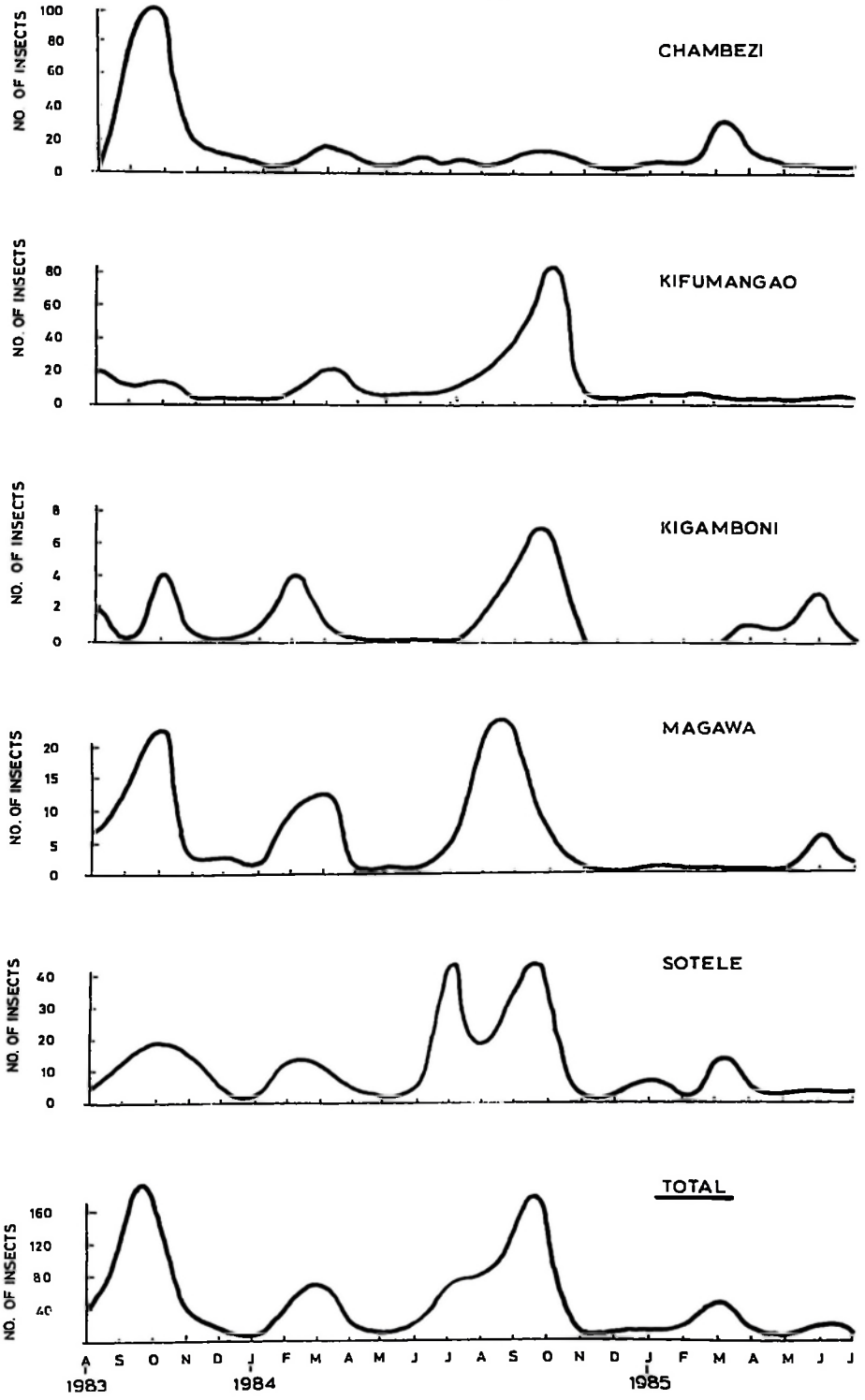


Fig. 22: Fluctuations in adult populations of Elasmoceles sp at five locations. Insects were counted at fortnightly intervals but presented as monthly totals.

FAMILY AND GENERA	No. of counting sites where the species occur	Relative abundance of species	Emphasis in transmission trials
FULGOROIDEA			
FAMILY DERBIDAE			
Diostrombus abdominalis (Distant)	7	v.a.	+++
Diostrombus mkurangai sp. nov.	7	v.a.	+++
Diostrombus schuilingi sp. nov.	5	v.r.	+
Lydda woodi Muir	5	r.	+
Phenice pongwe sp. nov.	7	a	++
Paraphenice mawae sp. nov.	7	a	++
Kamendaka kordofana Synave	4	a	+
Amania annustifrons (Melichar)	7	a	+
Zoraida fuliginennis Muir	4	r	+

Table 5: List of insect species of the order Homoptera, suborder Auchenorrhyncha recorded on coconut palms in Tanzania, their occurrence in the 7 different counting sites, their relative abundance and the emphasis they receive in the disease transmission trials. i.c. = incidental; a = abundant; v.a. = very abundant; r = rare; v.r. = very rare; n.a. = not applicable. +++ = included regularly; ++ = included; + = occasional included; - = not included

FAMILY AND GENERA	No. of counting sites where the species occurs regularly	Relative abundance of species	Emphasis in transmission trials
Rohigus magawae sp. nov.	5	a.	+
Zorabana vipaku sp. nov.	3	v.r.	+
Patara minazi sp. nov.	n.a.	inc.	-
FAMILY *DELPHACIDAE*			
Peregrinus maidis (Ashmead)	n.a.	inc.	-
Nesodryas antiope Fennah	7	a	++
FAMILY *MEENOPLIDAE*			
Meenoplus asasu latu	6	r.	+
Nisia nervosa (Motschulsky)	n.a.	inc.	-
Kermesia sp.	n.a.	inc.	-
FULGOROIDEA			
FAMILY *CIXIIDAE*			
Oliarus lupialensis Synave	n.a.	inc.	-
FAMILY *FLATIDAE*			
Flata sp.	n.a.	inc.	-

(Table 1 continued)

FAMILY AND GENERA	No. of counting sites where the species occurs	Relative abundance of species	Emphasis in transmission trials
FAMILY LOPHODIDAE			
Elasmosceles cimicoides Spinola	7	a	-
FAMILY NOGODINIDAE			
Diazanus sp., new sp.?	5	r	+
FAMILY RICANIDAE			
Pachazoides sp.	n.a.	inc.	-
Paraclusivius sp.	n.a.	inc.	-
CICADOIDEA			
FAMILY CICADELLIDAE			
Molopterus sp.	n.a.	inc.	-
Signoretia sp., n. pacifica Walker	n.a.	inc.	-
FAMILY CERCOPIDAE			
Bandusia erythrostena Walker	7	a	++

Table 5: (continued)

Month	Diestrombus abdominalis	Diestrombus mkurangei	Diestrombus schultzingi	Parapheneice sp. & Phenice sp.	Robigus sp.	Lyada sp.	Hamandaka sp.	Amania sp & Mesocrya sp.	Zoraida sp.	Zorabana sp.	Acanoplus sp.	Plasmoscelea sp.	Libuzanus sp.	Candusia sp.
Aug. 1983	56	33	-	5	6	-	-	-	-	-	-	1	-	-
Sept.	202	21	2	9	11	-	-	-	1	-	-	74	-	-
Oct.	138	23	1	7	10	-	-	-	-	-	-	67	-	3
Nov.	426	25	3	7	9	-	133	100	-	-	-	13	-	3
Dec.	321	25	-	6	3	-	77	31	-	-	-	8	-	1
Jan. 1984	416	17	-	6	6	-	116	11	-	-	-	1	-	-
Feb.	384	13	-	2	-	4	6	-	-	-	-	2	-	-
March	379	22	-	1	1	-	51	2	2	-	1	20	-	-
April	342	31	-	10	4	2	58	1	-	-	2	-	-	-
May	1043	82	1	22	7	13	47	10	-	-	3	-	-	-
June	1236	165	-	15	8	1	6	12	1	-	-	7	2	99
July	2582	209	-	37	5	5	43	14	1	-	3	5	4	65

Table 5 a: Monthly total of different common adult species of planthoppers counted on young palms at Lunsazi.

Month	Diastrombus abdominalis	Diastrombus mkuranzai	Diastrombus schulzinoi	Psraphenice sp. & Phenice sp.	Robigus sp.	Lydda sp.	Ramandaka sp.	Amania sp & Masodryas sp.	Zoraida sp.	Zorabana sp.	Mcenoplus sp.	Elasmoscelos sp.	Mauzanus sp.	Conchsia sp.
Aug. 1983	58	33	-	5	6	-	-	-	-	-	-	1	-	-
Sept.	202	21	2	9	11	-	-	-	1	-	-	74	-	-
Oct.	138	23	1	7	10	-	-	-	-	-	-	87	-	3
Nov.	426	25	3	7	9	-	133	100	-	-	-	13	-	3
Dec.	321	25	-	6	3	-	77	31	-	-	-	8	-	1
Jan. 1984	416	17	-	6	6	-	116	11	-	-	-	1	-	-
Feb.	384	13	-	2	-	4	6	-	-	-	-	3	-	-
March	379	22	-	1	1	-	51	2	2	-	1	26	-	-
April	342	31	-	10	4	2	55	1	-	-	2	-	-	-
May	1043	82	1	22	7	13	47	16	-	-	1	-	-	-
June	1236	165	-	15	8	1	6	12	1	-	-	7	2	99
July	2582	209	-	37	5	5	43	14	1	-	5	5	2	68

Table 5 a: Monthly total of different common adult species of planthoppers collected on yellow pans at Chemezi.

Month	Diostrombus abdominalis	Diostrombus mkurangai	Diostrombus schulline	Paraphenica sp. & Phenicu sp.	Robious sp.	Lydda sp	Komendaka sp.	Amania sp. & Nesodrvas sp	Zoraida sp.	Zorabana sp.	Mesnodius sp.	Elasmoscelioosp.	Diazanus sp.	Bandusia sp.
Aug. 1984	5475	592	2	13	4	5	24	21	-	-	1	3	1	14
Sept.	3115	383	1	11	-	2	12	136	-	-	1	10	5	-
Oct.	2247	416	1	8	12	2	57	70	3	-	1	17	3	-
Nov.	1377	805	1	15	2	12	53	326	6	-	-	2	-	1
Dec.	2227	1434	1	28	30	12	46	86	1	5	-	2	-	-
Jan. 1985	3689	3837	1	77	20	27	47	103	8	2	3	4	2	1
Feb.	3463	3576	1	79	17	13	13	18	9	5	1	5	6	-
March	2492	2167	-	99	12	19	37	26	1	5	-	30	6	2
April	1247	1339	-	111	7	12	8	5	12	4	1	10	8	-
May	1056	1170	-	110	13	16	6	5	4	3	-	2	6	-
June	2030	1460	6	138	17	24	4	5	-	2	-	3	4	-
July	2186	576	-		7	10	-	20	-	-	-	4	-	-

Table 6 a: (continued)

Month	Diastrombus abdominalis	Diastrombus mkurangai	Diastrombus schultzei	Paraphenice sp. & Phenice sp.	Robiqus sp.	Lydda sp.	Amania sp. & Mesodryas sp.	Zoraida sp.	Zorabana sp.	Meenoplus sp.	Elasmoscelis sp.	Diazanus sp.	Bandusia sp.	Kamendaka sp.
Aug. 1983	-	34	3	12	4	nr	nr	-	nr	nr	18	-	-	nr
Sept.	-	1	-	1	1	nr	nr	-	nr	nr	8	-	-	nr
Oct.	-	1	1	2	3	nr	nr	-	nr	nr	11	-	-	nr
Nov.	3	26	13	4	8	nr	1653	2	nr	nr	1	-	-	-
Dec.	6	10	-	2	104	-	54	-	-	-	2	-	-	-
Jan. 1984	23	14	3	7	13	7	181	2	-	5	-	-	3	1
Feb.	34	92	12	-	10	-	36	1	-	-	6	-	2	-
March	10	19	1	1	5	-	3	-	-	-	19	-	-	-
April	14	30	-	3	41	4	24	2	-	1	6	5	8	-
May	19	52	-	8	38	11	69	1	-	4	3	2	35	-
June	10	171	1	11	35	1	137	8	1	1	3	7	222	23
July	6	88	-	8	15	-	45	1	-	2	8	1	18	1

Table 6 b: Monthly totals of different common adult species of planthoppers counted on young palms at Kifumengao.

Month	Diostrombus abdominalis	Diostrombus mkurangai	Diostrombus schulinoi	Paraphenice sp. & Phenice sp.	Robigus sp.	Lydda sp.	Kamendaka sp.	Amania sp. & Nesodrvas sp.	Zoraida sp.	Zorabana sp.	Meenoplus sp.	Elasmosceles sp	Diazanus sp.	Bandusia sp.
Aug. 1984	4	85	2	1	24	-	-	141	2	-	5	20	7	6
Sept.	1	55	-	1	21	-	-	250	-	-	-	37	6	-
Oct.	2	43	2	-	12	-	-	296	-	-	-	76	-	-
Nov.	6	16	-	1	3	2	-	422	-	-	-	-	-	1
Dec.	22	82	-	1	32	1	1	603	2	1	-	-	3	-
Jan. 1985	42	98	1	1	23	10	-	86	2	1	2	2	2	10
Feb.	186	223	36	6	36	11	-	17	3	2	1	3	2	-
March	54	61	3	7	32	-	-	-	2	-	1	-	-	3
April	82	130	6	8	11	10	1	15	3	-	1	2	2	4
May	48	98	10	2	20	11	-	11	-	1	1	1	-	6
July	8	73	2	6	36	3	-	26	-	-	1	1	-	-

Table 6 b: (continued)

Month	Diostrombus abdominalis	Diostrombus ekurangai	Diostrombus schillingi	Paraphenice sp. & Phenice sp.	Reblgus sp.	Kamendaka sp.	Amania sp. & Mesodryas sp.	Zoraida sp.	Zorabana sp.	Maenoplus sp.	Elasmoscelis sp.	Bandusia sp.	Lydda sp	Diazanus sp.
Aug. 1983	593	17	-	11	2	-	nr	nr	-	nr	-	2	-	-
Sept.	88	3	-	-	-	-	nr	nr	-	nr	-	-	-	-
Oct.	20	-	-	-	-	-	nr	nr	-	nr	-	4	-	-
Nov.	100	3	1	-	-	-	-	-	-	nr	-	-	-	-
Dec.	36	8	-	1	-	-	-	-	-	nr	-	-	-	-
Jan. 1984	69	35	-	1	-	-	-	-	-	nr	-	-	-	-
Feb.	138	54	4	1	-	-	-	-	-	nr	-	4	-	-
March	23	13	-	-	-	-	2	1	-	-	-	1	-	-
April	306	19	-	5	-	1	5	1	-	-	-	-	-	-
May	1859	112	-	5	-	3	-	2	-	1	2	-	-	-
June	1436	65	-	6	-	1	-	46	-	-	1	-	-	3
July	2296	49	-	15	-	-	3	14	-	-	2	-	-	25

Table 6 C: Monthly totals of different common adult species of planthoppers counted on young palms at Kigamboni.

Month	Diostrombus abdominalis	Diostrombus mkurangai	Diostrombus schuilingi	Paraphenice sp. & Phenice sp.	Robigus sp.	Lydda sp	Kamendaka sp.	Amania sp.' & Nesodrvas sp.	Zoraida sp.	Zorabana sp.	Meenoplus sp.	Elasmoscelis sp.	Diazanus sp	Bandusia sp.
Aug. 1984	4815	46	-	27	-	-	10	27	-	-	-	-	-	-
Sept.	2051	53	-	1	-	-	-	23	-	-	-	5	-	-
Oct.	434	43	-	-	-	-	-	3	-	-	1	6	-	-
Nov.	nr	nr	nr	nr	nr	nr	nr	nr	nr	nr	nr	nr	nr	nr
Dec.	1426	97	-	3	-	3	-	2	-	-	-	-	-	1
Jan. 1985	829	56	-	1	-	-	-	5	-	-	-	-	8	-
Feb.	594	103	-	21	-	-	1	-	-	-	-	-	-	-
March	436	71	-	-	-	-	-	2	-	-	-	-	-	-
April	1451	89	+	4	-	5	-	28	-	1	2	1	-	-
May	1860	24	-	-	2	-	-	66	-	-	-	1	-	-
June	1979	66	+	3	+	-	-	313	-	-	3	3	-	-
July	2732	64	-	2	+	-	-	545	-	-	-	-	-	-

Table 6 c: (continued) nr = not recorded

Month	Diostrombus abdominalis	Diostrombus mkuranoai	Diostrombus schillingsi	Paraphenice sp. & Phenice sp.	Robigus sp.	Lydda sp.	Kamendaka sp.	Amania sp. & Nesodryas sp.	Zoraida sp.	Zorabana sp.	Neenoplus sp.	Elasmoscelis sp.	Diazanus sp.	Bandusia sp.
Aug. 1983	nr	nr	nr	nr	nr	nr	nr	nr	nr	nr	nr	nr	nr	nr
Sept.	156	2	-	6	20	nr	nr	nr	-	nr	-	18	-	2
Oct.	70	-	-	2	-	nr	nr	nr	-	nr	-	27	-	1
Nov.	13	-	1	2	-	nr	-	101	-	nr	-	3	-	-
Dec.	21	4	1	9	4	nr	-	222	-	nr	-	3	-	-
Jan. 1984	11	-	-	13	5	-	-	1	-	-	1	2	-	-
Feb.	37	3	-	16	24	-	-	7	-	-	3	[illegible]	-	-
March	35	2	2	-	3	-	-	-	-	-	1	15	-	-
April	39	2	-	27	2	-	-	1	-	-	4	-	-	-
May	196	22	-	65	16	14	-	6	-	-	5	1	3	-
June	606	15	1	133	51	3	-	24	2	-	3	1	-	26
July	1025	19	-	95	19	3	-	18	-	2	4	7	2	17
Aug.	567	3	-	10	20	1	-	29	-	-	1	26	6	3

Table 6 d: Monthly totals of different common adult species of planthoppers counted on young palms at Magawa.

Month	Diastrombus abdominalis	Diastrombus mkurannai	Diastrombus schulingi	Paraphenice sp- & Phenice sp.	Robiquss sp.	Lydda sp.	Kamendaka sp.	Amania sp. & Nesodryas sp.	Zoraida sp.	Zorabana sp.	Mcenoplus sp.	Elasmoscelas sp.	Diazanus sp.	Banduria sp.
Sept. 84	419	5	-	1	8	2	-	3	-	-	-	23	-	-
Oct.	314	5	-	1	2	-	-	9	-	-	-	7	-	-
Nov.	11	-	-	2	-	-	-	6	-	-	-	1	-	-
Dec.	45	6	-	76	4	9	-	6	-	-	-	-	2	-
Jan. 85	153	18	-	42	12	9	2	10	1	1	3	1	-	7
Feb.	40	3	5	15	2	-	3	-	1	3	1	-	-	-
March	90	23	6	25	2	11	-	1	18	6	3	1	-	-
April	68	20	-	10	-	8	-	-	4	6	3	-	-	-
May	422	20	-	16	4	64	-	1	-	10	1	-	-	-
June	552	15	3	3	4	2	-	1	-	-	-	2	-	-
July	589	4	-	3	1	3	-	10	-	-	3	-	-	-
Aug.	553	12	3	3	5	4	-	13	-	-	2	2	-	-

Table 6 d : (continued)

Month	Diostrombus abdominalis	Diostrombus mkurangai	Diostrombus schulingi	Paraphenice sp. & Phenice sp.	Robigus sp.	Lydda sp	Kamendaka sp.	Amania sp & Nesodryas sp.	Zoraida sp.	Zorabana sp.	Meenoplus sp.	Elasmosceles sp.	Diazanus sp.	Gandusia sp.
Aug. 1983	3	8	-	140	10	nr	nr	nr	5	-	nr	4	nr	1
Sept.	-	2	-	50	4	nr	nr	nr	-	-	-	12	nr	-
Oct.	6	6	-	13	-	2	nr	nr	4	-	-	17	nr	-
Nov.	29	2	-	34	2	59	-	2078	4	-	-	13	-	-
Dec.	7	5	-	60	2	16	-	163	1	-	-	2	-	-
Jan. 1984	3	3	-	32	3	5	-	21	-	-	-	-	-	-
Feb.	6	6	-	52	1	4	-	6	1	-	1	11	-	-
March	3	1	-	6	3	8	-	1	-	-	2	10	-	-
April	4	4	-	83	8	22	-	-	2	-	3	3	2	4
May	28	6	-	95	1	66	-	2	2	-	8	2	1	6
June	13	22	-	177	8	8	-	8	3	-	-	4	-	14
July	19	30	-	270	6	1	-	8	-	4	3	39	1	49

Table 6 e: Monthly totals of different common adult species of planthoppers counted on young palms at Sotele.

Month	Diastrombus abominalis	Diastrombus mkurangai	Diastrombus schuilingi	Paraphenice sp. & Phenice sp.	Robigus sp.	Lydda sp.	Kamendaka sp.	Amania sp. & Pesodryas sp.	Zoraida sp.	Zorahana sp.	Meenoplus sp.	Elasmoscelis sp	Diazanus sp	Bandusia sp.
Aug. 1964	30	28	-	106	20	2	-	45	1	-	1	16	-	8
Sept.	8	17	-	21	12	-	-	100	-	-	1	35	1	1
Oct.	14	15	-	15	15	53	-	400	2	1	-	27	-	-
Nov.	3	6	-	78	1	2	-	169	-	-	-	-	-	3
Dec.	17	29	-	145	3	4	-	79	4	-	-	2	-	2
Jan. 1965	19	14	-	95	4	31	-	14	4	1	-	5	2	1
Feb.	11	13	-	52	3	59	-	-	6	1	-	1	-	-
March	50	25	-	65	1	420	-	1	1	4	-	12	1	-
April	12	10	-	24	1	51	-	-	-	-	-	2	-	-
May	41	18	-	102	10	1039	-	-	-	-	-	3	-	1
June	33	38	-	39	5	1104	-	8	1	3	-	2	-	2
July	54	3	-	39	2	696	-	5	-	-	-	3	-	-

Table 6 a : (continued)

Month	Diostromus abdominalis	Diostromus mkurangai	Elasmoscelus sp.	Meenoplus sp.	Bandusia sp.	Phenice sp. & Paraphenica sp.	Zoraida sp.	Lydda sp.	Amania sp. & Nesodryas sp.
December 1983	-	-	2	3	-	-	-	-	-
January 1984	1	-	4	2	-	-	-	-	-
February	2	3	7	5	-	-	-	-	-
March	-	-	2	1	-	-	-	-	-
April	-	-	3	2	5	3	-	-	-
May	4	1	4	9	-	-	1	-	-
June	8	3	-	11	24	-	-	-	-
July	8	5	1	7	21	2	-	-	-
August	12	3	13	3	8	-	-	-	-
September	6	3	6	1	1	-	-	-	-

Table 6 f : Montly totals of different common adult species of planthoppers counted on young palms at Sotele B

Month	Diostracombus abdominalis	Diostrombus mkuranga1	Elasmoscales sp.	Meemoplus sp.	Bandusia sp.	Phantice sp. A Paraphenales sp.	Zoraida sp.	Lydda sp.	Mamita sp. A Mesodevas sp.
October 1984	3	2	2	1	2	1	1	1	1
November	11	4	1	1	2	1	1	1	1
December	12	10	1	1	1	1	1	1	1
January 1985	25	6	1	2	1	1	1	1	1
February	21	4	1	1	1	2	1	1	2
March	16	22	3	1	3	2	1	1	1
April	4	5	1	2	1	1	1	2	1
May	15	10	2	1	1	1	1	1	1
June	11	19	2	1	2	1	1	1	1
July	10	8	1	3	1	1	1	1	1

Table 6 f : (continued)

Month	Diostrombus abdominalis	Diostrombus mkurangai	Diostrombus schuilingi	Paraphenice sp & Phenice sp.	Robigus sp.	Bardusia sp.	Zoraida sp.	Elasmosceles sp.	Amania sp. & Mesodryas sp.	Kamendaka sp.	Meenoplus sp.	Lydda sp.	Zorabana sp.
August 1983	1051	2	-	16	7	1	-	-	15	1	-	-	-
September	677	2	-	3	9	-	-	2	2	-	-	-	-
October	634	4	-	9	10	-	-	11	3	5	2	-	-
November	539	10	-	8	3	-	-	12	31	45	-	-	-
December	325	8	-	6	1	-	-	-	28	36	-	-	-
January 1984	39	2	-	3	-	-	-	1	1	21	-	-	-
February	64	15	-	1	-	-	-	6	5	98	-	-	-
March	32	-	-	-	-	-	-	-	-	45	-	-	-
April	7	-	-	-	-	-	-	1	-	44	-	-	-
May	15	-	1	3	-	-	-	-	-	1	-	-	-
June	10	-	-	3	-	2	-	1	-	37	-	-	-
July	11	-	-	3	-	9	-	-	-	8	-	-	-

Table 6 g: Monthly totals of different common adult species of planthoppers counted on young palms at Pongwe

Month	Diostrombus abdominalis	Diostrombus mkurangai	Diostrombus schuilinai	Paraphenice sp. & Phenice sp.	Robligus sp.	Bandusia sp.	Zoraida sp.	Elasmocoelies sp.	Amanta sp. & Mesodryas sp.	Mamendaka sp.	Keenoplus sp.	Lydda sp.	Zorabena sp.
August 1984	72	-	-	14	-	20	-	-	3	41	-	-	-
September	72	-	-	7	3	-	-	8	10	22	-	-	-
October	28	-	-	1	1	-	-	1	9	7	-	-	-
November	86	-	-	19	1	1	-	1	100	13	-	-	-
December	369	9	-	33	1	5	-	1	398	7	-	-	-
January 1985	543	12	-	50	8	-	-	4	94	3	-	-	-
February	113	-	-	15	9	-	-	-	10	-	-	-	-
March	168	24	-	-	-	2	-	-	15	7	-	-	-
April	108	9	-	11	1	-	1	-	1	-	-	-	-
May	343	16	2	28	1	4	-	-	-	[illegible]	-	-	-
June	660	54	4	8	3	-	-	-	-	-	-	-	-
July	288	21	1	2	-	-	-	-	2	-	-	-	-

Table 5 g: (continued)

4:4 Insect Transmissions of the Lethal Disease

The number of insects caught and released in the cages in the first and second attempts to transmit the lethal disease are shown in Tables 7 and 8. The number of insects collected and released in the third transmission attempt under semi-controlled conditions is given in Table 9.

The test palms were exposed to insects in the cages for a period of 106 days in the first series and 205 days in the second series of transmission trials. When they were removed from the cages no symptoms of the lethal disease had yet appeared. In the first series of the transmission tests, one palm developed symptoms similar to those of the lethal disease. This was 280 days after the palm was remove from the cage and placed in an isolated area free from coconut. The lower leaves developed a bronzed discolouration and the spear leaf got necrotic. This palm was exposed to a mixture of the species D.mkurangai sp. nov., P.mawae sp. nov. and L.woodi. Unfortunately the test palm was already in an advanced diseased stage when observed. It was estimated that the test palm developed the initial symptoms of the disease about six weeks earlier that is 240 days after removal from the cage. Furthermore, the diseased palm was not sampled for Ultra Fluorescence (UF) and electron microscopy (EM) studies.

Cage No.	Insect Species	No. Transferred	First Introduction	Last Introduction	Test Palms (infected/ exposed)	Symptoms
1	Diostrombus abdominalis	14900	7/5/84	8/8/84	0/4	-ve
2	As in cage 1	14550	6/5/84	9/8/84	0/4	-ve
3	Mixture of D.mkurangai, Paraphenice sp. Phenice sp. Lydda sp.	850	7/5/84	8/8/84	0/4	-ve
4	As in cage 3	740	8/5/84	9/8/84	0/4	1 doubtfull case early May 1985

Table 7: Transfers of natural populations of planthopper species collected in the field to caged test palms in the first series.

Cage No.	Insect species	No. Transfered	First Introduction	Last Introduction	Test Palms (infected/ exposed)	Symptoms
1	Mixture of *Diostrombus* spp.	32170	29/8/84	27/3/85	0/5	-ve
2	Mixture of *Paraphenice* sp.; *Phenice* sp. *Lydda* sp. *Meenoplus* sp. *Kamendaka* sp.	1280	30/8/84	27/3/85	0/5	-ve
3	Mixture of *Robigus* sp. *Zoraida* sp. *Elasmosceles* sp. *Diazanus* sp. *Zorabena* sp.	760	6/9/84	27/3/85	0/5	-ve

Table 8: Transfers of natural populations of planthopper species collected in the field to caged test palms in the second series.

Plot No.	Insect Species	No. Transfered	First Introduction	Last Introduction	Test Palms (infected/ exposed)	Symptoms
I	Mixture of *Diostrombus* spp.	9925	4/4/85	13/1/86	0/9	-ve
II	Mixture of *Nesodryas* sp. & *Amania* sp.	6275	19/7/85	9/1/86	0/9	-ve
III	Mixture of *Paraphenice* sp. *Phenice* sp. *Lydda* sp. *Meenoplus* sp.	3054	24/4/85	13/1/86	0/9	-ve

Table 9: Transfers of natural populations of planthopper species collected in the field to test palms in semi-controlled environment.

Plot No.	Insect Species	No. Transfered	First Introduction	Last Introduction	Test Palms (infected/ exposed)	Symptoms
IV	Mixture of Rocious sp.; Bandusia sp.; Zoraida sp.; Elasmosceles sp. Kamendaka sp. Diazanus sp.	475	29/6/85	14/1/86	0/9	-ve
V	As in I	7795	8/5/85	14/1/86	0/9	-ve
VI	As in III	3035	13/6/85	8/1/86	0/9	-ve
VII	As in IV	370	13/6/85	8/1/86	0/9	-ve
VIII	As in I	6425	13/6/85	7/1/86	0/9	-ve

(continued)

5:0 DISCUSSION

In the development of the coconut industry in the Kisarawe and Rufiji Districts of mainland Tanzania, three planting periods can be distinguished. The first was under the Arab influence mainly during the reign of Sultan Bargash ibn Said of Zanzibar (1870 - 1888), the second during the German administration (1891 - 1919) and the third and most important between the two World Wars with emphasis on the years around 1930. Although the lethal disease probably existed as early as 1890, it made little impact on the two former plantings. However, serious outbreaks of the disease started in the early 1940's when the post 1920 groves and offsprings of the two older plantings came into bearing.

In the Caribbean and Florida, lethal yellowing was reported to spread through the introductions of planting materials from diseased to disease free areas (Howard, 1983; McCoy *et al.*, 1982). In the case of the lethal disease, the planting materials were imported from Zanzibar and Mafia. In these areas no disorder of coconut was (even at present) ever reported. Thus there is no evidence to suggest that the lethal disease originated from these areas. As it is postulated that the disease is spread by insects, it is obvious that this insect(s) has been indigenous. The insect was not very active because of the presence of the thick bushes which acted as a barrier to its movement and the thinly isolated planted coconut palms available. It is therefore doubtful whether the offsprings of the older plantings became more susceptible to the lethal disease than the populations themselves. However, it is assumed that the massive bush clearing which removed the protective barrier and the post 1920 plantings gave the disease a better chance than the more isolated plantings before.

The survey revealed that the outbreaks of the disease occurred at different times and in different areas often far apart and isolated from each other. Moreover, in the Kisarawe District, in the 1940's, several independent outbreaks were registered at the same time. It is therefore impossible to recognize one or more source from where the disease has spread to other areas. It has probably been indigenous in various places along the coast for a long time. One may postulate that the palm-pathogen relation on the mainland had achieved equilibrium. The disease did not become epidemic until coconut were introduced from elsewhere where palms were never exposed to the disease like Mafia and Zanzibar. This may be an explanation as for the much lower incidence of lethal disease in the Bagamoyo District compared with the affected areas south of Dar es Salaam. In the Bagamoyo District, large coconut plantings were established long before the latter areas. Resistance may have built up in the Bagamoyo groves because through several generations, seednuts were collected from palms exposed to and surviving the lethal disease. On the other hand the original seednuts used for planting in the groves of the Catholic Mission at Bagamoyo, from where most of the palms in the district originated came from Zanzibar while virtually all seednuts for areas south of Dar es Salaam were imported from Mafia. There may be differences in resistance between the EAT from Zanzibar and the EAT from Mafia.

The survey of the history of the lethal disease was carried out by interviewing farmers in four districts. It is realized that the survey should be extended to other areas of Tanga, Pangani, Lindi, Mtwara districts and the islands Mafia and Zanzibar. Even then it is uncertain whether any relevant answers can be expected on the relationship between the lethal disease and the origin of the host plant.

Injection of both bearing and non-bearing diseased coconut palms with tetracycline hydrochloride resulted in remission of symptoms in test palms but only when the injections were started in sufficient early stages of the lethal disease. In bearing palms, those with least inflorescence blackening and less advanced leaf bronzing responded best to the treatment. Similarly, in non-bearing palms, those with least leaf bronzing and necrosis in the pinnae of the spear leaves responded best.

In bearing palms showing temporary remission, bronzing progressed during the 98 days of treatment. Also in non-bearing palms bronzing continued during the 68 days of treatment. In no case was regreening of a bronzed leaf observed. It would seem therefore that certain irreversible processes occur even before symptoms are expressed for which the antibiotic is unable to influence.

In bearing palms, remission of symptoms was not nearly as spectacular as reported in Jamaica (Hunt et al., 1974) and Florida (McCoy, 1972, 1973, 1976; McCoy et al., 1976) as only one antibiotic injected palm showed total remission of symptoms. This may be because the uptake of tetracycline antibiotic in the very tall (50 - 60 years old) palms was inadequate or the antibiotic less effective. On the other hand, the results are in agreement with those obtained in West Africa (Steiner, 1976). This may indicate that the diseases in Africa are not caused by the same MLO as lethal yellowing. Considering that tetracycline acts againsnt MLO, the obtained remission of symptoms in diseased palms is a support for a MLO etiology theory of the lethal disease.

The diversity and number of planthoppers (Fulgoroidea) on coconut palm contrasted strongly with the paucity of leafhoppers (Cicadelloidea). Most of MLO associated diseases are transmitted by leafhoppers. However, the

same observations were made in Jamaica (Schuiling et al., 1976) and in Florida (Howard and Mead, 1980) and moreover a fulgorid was implicated as a vector of the lethal yellowing in Florida (Howard et al., 1984). Therefore it was justified to restrict the transmission experiments to planthoppers. Of the 29 different species of fulgorids occurring on coconut palms in Tanzania, only seven species were recorded at all counting sites. It is obvious that these species had and will have priority in the transmission trials, in particular the Diostrombus spp., which are extremely numerous everywhere.

There are strong indications that the removal of the remaining (shading) tall palms at Chambezi led to a considerable increase in the numbers of planthoppers on young palms. This may have been a seasonal effect as shaded young palms were not kept as controls. However, the observations would agree with a strong suspicion in the NCDP, Disease Control Section, that palms are more easily infected by the lethal disease when they are not planted under shade. This implies that shading interferes with the feeding behaviour of the insects hence with the infection of the lethal disease. No particular hybrid or variety at any of the counting sites was visited consistently by higher numbers of any of the planthopper species. This indicates that all of the varieties and hybrids were equally exposed to the insects if it is one of the common planthoppers recorded in these population studies.

In the disease transmission trials one test palm developed symptoms similar to those of the lethal disease. Unfortunately the palm was in an advanced stage when first observed and was not sampled for UF or EM investigations. Therefore the case must be considered doubtful. In young palms symptoms similar to those of the lethal disease may be produced by blast, a nursery disease. If the lethal

disease was involved the incubation period was between 240 and 340 days. This agrees with data from experiments in Jamaica (Heinze et al., 1972; Heinze and Schuiling, 1973).

It is intended to continue testing planthopper species as mixture by direct introduction of insects collected from diseased palms in the field. This is under the assumption that if a lethal disease occurs on the spot, there must be infectious insects flying around. When a few certain cases of transmission are observed then the transmission trials will be separated to single species and aquistion feeding attempted.

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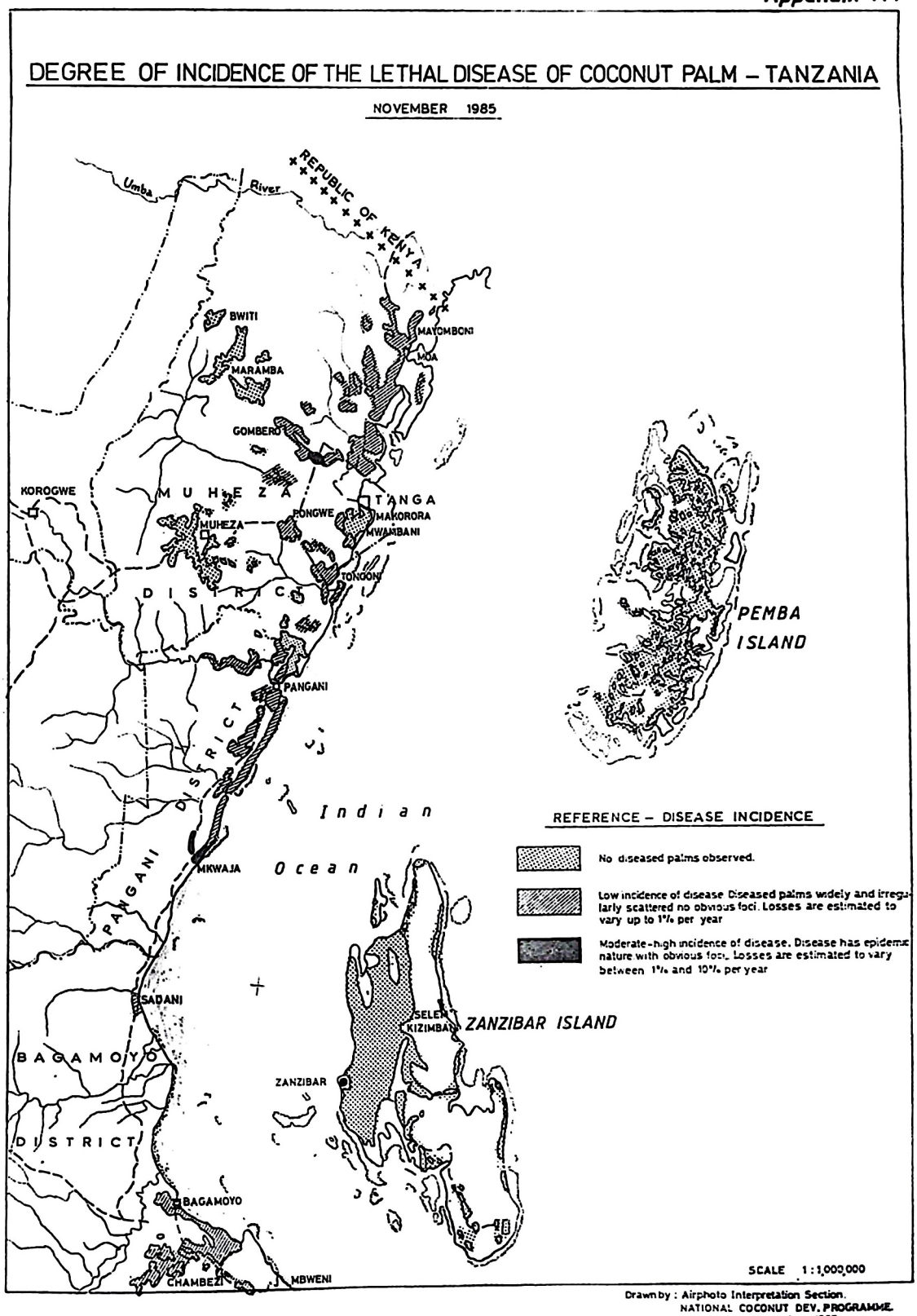
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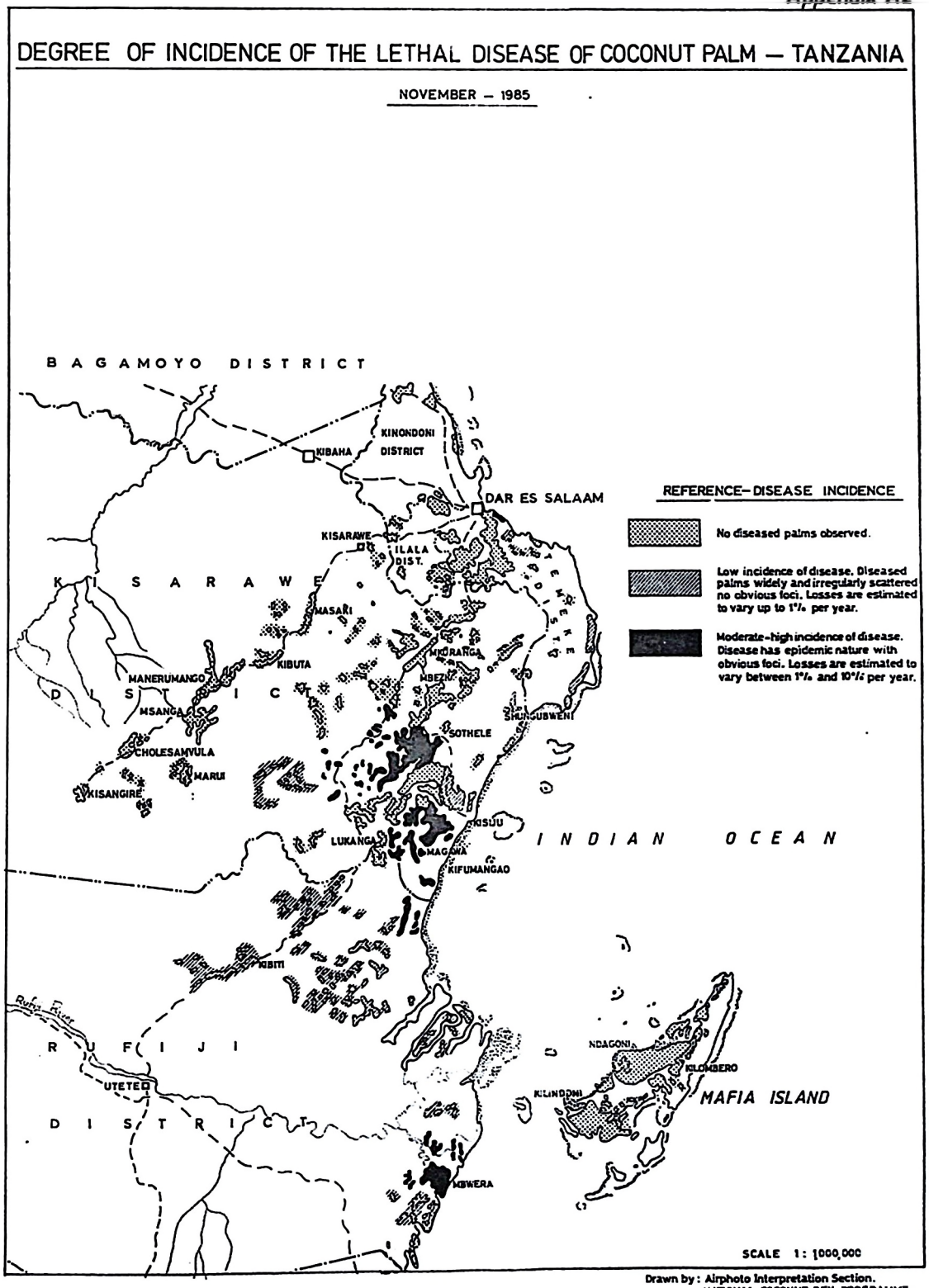
Appendix A 1


DEGREE OF INCIDENCE OF THE LETHAL DISEASE OF COCONUT PALM – TANZANIA
NOVEMBER 1985
REPUBLIC OF KENYA
Umba River
BWITI
MARAMBA
MAYOMBONI
MOA
GOMBERO
KOROGWE
MUHEZA
MUHEZA DISTRICT
TONGONI
TANGA
PONGWE
MAKORORA
MWAMBANI
PANGANI
PANGANI DISTRICT
Indian Ocean
MKWAJA
SADANI
BAGAMOYO
DISTRICT
BAGAMOYO
CHAMBEZI
MBWENI
ZANZIBAR
SELEM
KIZIMBA
ZANZIBAR ISLAND
PEMBA ISLAND
REFERENCE – DISEASE INCIDENCE
No diseased palms observed.
Low incidence of disease Diseased palms widely and irregularly scattered no obvious foci. Losses are estimated to vary up to 1% per year
Moderate-high incidence of disease. Disease has epidemic nature with obvious foci. Losses are estimated to vary between 1% and 10% per year
SCALE 1 : 1,000,000
Drawn by : Airphoto Interpretation Section.
NATIONAL COCONUT DEV. PROGRAMME.
November 1985

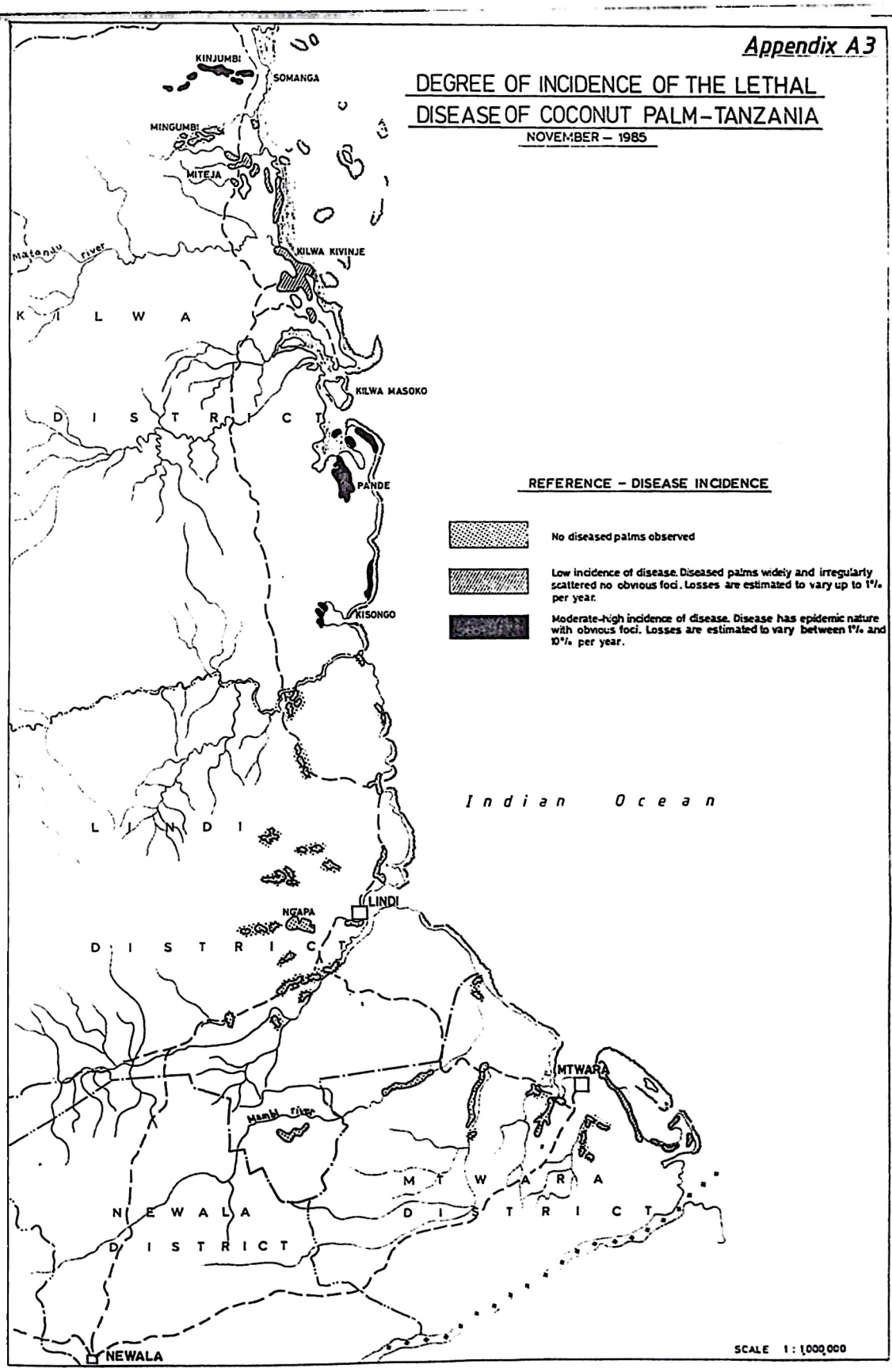
Appendix A2

DEGREE OF INCIDENCE OF THE LETHAL DISEASE OF COCONUT PALM — TANZANIA

NOVEMBER — 1985



Drawn by : Airphoto Interpretation Section.
NATIONAL COCONUT DEV. PROGRAMME.
November 1985.


Appendix A3
DEGREE OF INCIDENCE OF THE LETHAL DISEASE OF COCONUT PALM – TANZANIA
NOVEMBER – 1985
KINJUMBI
SOMANGA
MINGUMBI
MITEJA
Matandu river
KILWA KIVINJE
K I L W A
D I S T R I C T
KILWA MASOKO
PANDE
KISONGO
REFERENCE – DISEASE INCIDENCE
No diseased palms observed
Low incidence of disease. Diseased palms widely and irregularly scattered no obvious foci. Losses are estimated to vary up to 1% per year.
Moderate-high incidence of disease. Disease has epidemic nature with obvious foci. Losses are estimated to vary between 1% and 10% per year.
Indian Ocean
L I N D I
NGAPA
LINDI
D I S T R I C T
MTWARA
Mambi river
M T W A R A
D I S T R I C T
N E W A L A
D I S T R I C T
NEWALA
SCALE 1 : 1,000,000

APPENDIX 6

QUESTIONNAIRE ON THE HISTORY OF THE LETHAL DISEASE OF COCONUT PALMS IN TANZANIA

1. VISIT NO. :----------

2. DATE OF VISIT :----------

3. VILLAGE :----------DIVISION :----------

   DISTRICT :----------

4. AGE OF FARMER :----------(YEARS)

5. WERE YOU BORN IN THIS VILLAGE? YES/NO.

6. IT HAS BEEN REPORTED THAT IN YOUR VILLAGE THERE IS A COCONUT DISEASE,DO YOU KNOW ANYTHING ABOUT IT? YES/NO

7. WHAT ARE/WERE THE SYMPTOMS? ----------

   ----

8. IN YOUR OPINION WHAT IS/WAS THE CAUSE?----------

9.(a) WHEN DID IT START? ----------(YEAR)

(b) CAN YOU RELATE IT TO ANY EVENT WHICH HAPPENED IN THE RECENT HISTORY OF TANZANIA SUCH AS WORLD WARS, FAMINE, INDEPENDENCE CAMPAIGN ETC :-

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10. (a) WHERE DID IT START?----------(PLACE)

(b) WAS THE INCIDENCE AT ANY TIME HIGHER OR LOWER THAN AT PRESENT?----------

(c) WAS IT CONFINED TO ONE AREA OR DID IT SPREAD TO OTHER AREAS? ----------

11. (a) HOW DID IT SPREAD? ----------

(b) WHAT WAS THE MEANS OF SPREAD?----------

(c) WAS THERE ANY PARTICULAR DIRECTION OF SPREAD?

(d) WAS THE SPEED OR SPREAD FAST OR SLOW? ----------

12. WAS IT DISAPPEARING AND REAPPEARING? ----------

13. ARE THE SYMPTOMS OF THE DISEASE OBSERVED EARLIER STILL THE SAME? YES/NO

14. DO YOU THINK THAT THE DISEASE IS INCREASING OR DECREASING NOW? ----------

15. (a) WERE SEEDNUTS OR SEEDLINGS IMPORTED TO THIS

VILLAGE? YES/NO

(b) NAME THE VILLAGE OR PLACE WHERE YOU IMPORTED THEM FROM----------

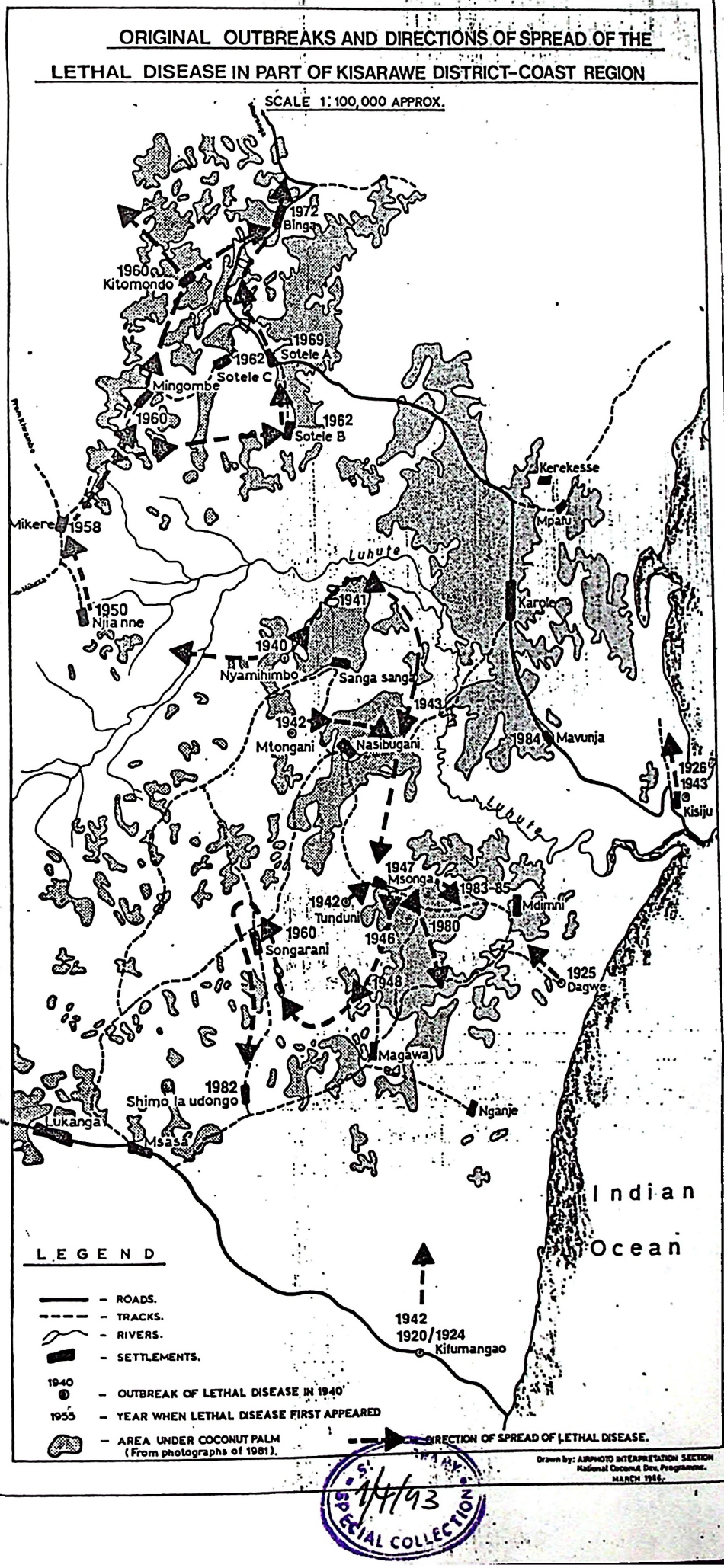
(c) WHAT WAS THE DISEASE SITUATION IN THE AREAS FROM WHERE THEY WERE IMPORTED? ----------

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(d) WERE SEEDNUTS OR SEEDLINGS EXPORTED TO OTHER VILLAGES? YES/NO

(e) NAME THE VILLAGE WHERE YOU EXPORTED THEM TO

(f) WHAT WAS THE DISEASE SITUATION SHORTLY AFTER PLANTING AND WHEN THE PALMS CAME INTO BEARING



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