DISEASES OF UNDETERMINED CAUSES, WITH SPECIAL REFERENCE TO THE ROOT (WILT) DISEASE OF SOUTH INDIA

by

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INTRODUCTION

The coconut palm, in spite of its hardy nature, is subject to a number of diseases the aetiology of many of which are still unknown and present a complicated problem. The very nature of its hardiness makes the progress of work on these diseases slow, in spite of the concentrated efforts of those working in the field. These diseases are all economically important, affecting vast tracts of fertile land and upsetting the economy of millions of people who solely depend on this crop for livelihood. While investigations on the disease of any plantation crop can naturally only be slow in progress, it is slower still in a crop which does not lend itself to vegetative propagation. Perhaps, one saving factor is that these diseases are not very fast spreading and this might, in all probability, be due to the absence of vegetative propagation in coconut.

Six diseases of unknown origin are reported so far. Table I gives details of their distribution, synonyms and general symptoms. It might be noted that the diseases can be grouped generally into five distinct types: (1) Lethal yellowing, (2) Kainecopé disease, (3) Bronze Leaf Wilt, (4) Cadang-Cadang, and (5) Root (Wilt) disease. The diseases that have more or less the same symptoms are the "lethal yellowing" or "unknown disease" of Central America, the Caribbean Islands, Jamaica and Florida in U.S.A. and the "Kainecopé disease" of French Togoland. Premature shedding of nuts followed by necrosis and blackening of inflorescence is the primary symptom in this. Shortly after, the lower leaves develop a conspicuous rich yellow colour, which eventually spreads over the whole crown. The "Bronze leaf wilt", prevalent in Trinidad, British Guiana and Tobago is similar to the above showing yellowing of outer whorl of leaves followed with a bacterial rotting of the developing cabbage. However, while this disease is considered to be caused by adverse environmental conditions leading to "physiological drought" (Martin, 1934; Bain, 1937 and Briton Jones, 1940) investigators on lethal yellowing consider it to be caused by a biological entity (Leach, 1946; Martin, 1949).

Perhaps no other disease of coconut has received the attention that cadang-cadang has, especially in recent years, because of the economic importance of the crop in the Philippines. Unlike the three diseases mentioned above, the cadang-cadang is slow in progress and kills the tree only after several years. The characteristic symptoms of this disease

are yellow or orange spots on the younger leaves, yellow mottling of older leaves with a distinctive bronzing of veinlets and rounding and scarification of the fruit. The leaves become brittle and dwarfed, the fruits become smaller in size until fruit production stops altogether. While the "bronze leaf wilt" and "lethal yellowing" are similar in symptoms, the wilt disease is more akin to the cadang-cadang in that the latter two kill a tree only slowly. But there the resemblance between the two diseases ends. Judging from the literature (Fajardo, 1953; 1955; Price, 1958; McWhorter, 1959) the symptoms of the cadang-cadang seem to be different from those of the wilt disease. The main symptom of the latter is flaccidity of leaflets accompanied by slow wilting. Yellowing of outer whorl of leaves, bending of petioles and abnormal nutfall also accompany the above symptoms. Like the cadang-cadang disease, the tree becomes finally barren although it continues this unproductive existence for a long time.

NAME

The name root (wilt) disease itself is suggestive of the nature of the disease, especially its complex nature. The slow wilting of the foliage originally observed was later found to be associated with a gradual rotting of the root system, which seems to be a characteristic feature of the disease.

HISTORY

Although all the diseases of unknown origin have been known to be prevalent for at least 25 years or more, the root (wilt) disease perhaps has the longest case-history. Records show that the disease was present more than 85 years ago, three foci of infection about 30 miles away from each other in central Travancore being noted. It is not clear how and from where this infection could have occurred. The disease became significant after a severe flood in 1882 when the land was water-logged for a considerable time. Water-logging still remains a pre-disposing factor in many areas. Investigations, started in 1900, were intensified with the visit of Butler in 1908 to the affected tracts. He correlated the rotting of the roots with the foliar symptoms of wilt which, according to him, was caused by drought and poor nutritional status of the soil. The cortical cells of many of the roots were attacked by a species of the fungus Botryodiplodia, resulting in collapse and death of the root tip. The constant occurrence of this fungus with the roots made him suggest that probably this root rot was the primary cause of the disease. Investigation on this disease was discontinuous until 1937, when the seriousness of the disease, which by that time had invaded wider tracts, demanded closer and more intensified work on it.

SYMPTOMS

The most distinguishing symptoms are (1) general slow wilting of the leaves; (2) flaccidity of the leaflets accompanied with an abnormal bending and consequent breaking of the petiole; (3) marginal and tip necrosis of leaflets and (4) an abnormal button shedding. In the early stages

of the disease the symptoms may appear singly or in combination, being accompanied by a chlorosis of the older leaves in the majority of cases. As the disease progresses, these symptoms get acute, the leaves are shed in quick succession, the general growth rate of the plant is retarded so that the number of leaves produced becomes smaller, the leaves themselves becoming short and stunted. The production of female flowers is curtailed, the spathes become much smaller in size and accompanied by premature nutfall, the yield is gradually reduced. In advanced stages of the disease, the highly stunted spathes fail to open normally.

The foliar symptoms generally appear on the younger leaves first. Thus although the yellowing appears to be on the outer whorl, it starts as small specks or dots on the 6th or 7th leaf, later spreading and merging with each other in the older leaves. Yellowing and sudden drying of intermittent leaves are also not uncommon just as blackening and shrivelling of spathe are met with as the first and only symptoms in some cases. The natural resistance of the tree is broken and the foliage becomes susceptible to the attack of secondary parasites like fungi and bacteria that cause severe leaf rot.

A high percentage of pollen produced is sterile and the viability of those that are good is low. The quality of nuts produced is adversely affected. The husk gets thinner, the fibres weaker and the shell does not harden normally. The kernel or endosperm becomes thinner which fails to dry up normally when converted into copra so that the latter remains soft and flexible.

Progressive deterioration of the root system takes place with the drying up of the majority of roots and rootlets from the tip backwards. The newly formed rootlets also get rotted due to the attack of weak soil parasites. A hard hypodermis is formed in many apparently healthy roots, thus preventing their normal functioning. Increased root production takes place in some cases to counteract the abnormal root loss by rotting, but as the natural resistance of the tree is gradually lost, these newly formed roots are affected by soil parasites. Root production is highly curtailed in the late stages.

These symptoms are very slow in developing so that an affected tree may continue its life for 10-15 years after the first appearance of symptoms. Generally palms that have just started bearing are most susceptible, infection on palms younger than 4 years being rather rare.

DISTRIBUTION AND LOSS

About 100,000 acres of the best coconut growing tracts of South and Central Kerala are at present affected by the disease. Very heavy infection occurs along the coastal districts and along the banks of the rivers. The disease is also found in districts situated more to the interior like Kottayam and Trichur. Recent surveys show that it is slowly spreading to the South and the North, irrespective of the type of soil.

It is difficult to estimate the loss caused by the disease because

actual data on this do not exist. However, it might be pointed out that in the heavily infected tracts about 80 per cent of the bearing palms are affected, nearly half of which has only 10-20 per cent of the original yield.

The disease is found to spread gradually and continuously into new areas. However, in an infected tract, pockets of healthy areas and patches where infection has just started are not uncommon. Although the actual rate of spread into new areas has not been determined the percentage increase of diseased trees within an infected plot is found to be 3-5 per annum where the mode of spread is rather at random (Table II). These observations find their parallel in the Philippines where within an infected plantation the disease appears more or less at random but has a continuous spread into new areas.

WORK DONE ON THE DISEASE

Attempts have been made to tackle the problem on (1) nutritional and biochemical studies in relation to soil conditions; (2) pathogenic aspects especially mycological and virological, and (3) diagnostic tests.

1. Nutritional and biochemical aspects

Environmental factors, both soil and atmosphere, are known to exert profound influence on the initiation, intensity and spread of a disease. Of this, soil is a major dynamic factor with its physical, chemical and biological constituents, each with its direct and indirect influence on the crop plant. To facilitate the study of interaction of these various factors by the accepted methods of scientific studies, where all contributing factors can be maintained under controlled conditions, coconut is perhaps the most difficult test material encountered. With the limited resources at hand, this proved to be a definite handicap and therefore field studies had to be resorted to. The work was carried out under three different heads: (1) Survey of soil, its composition and nutrient status; (2) Chemical composition of plant material; (3) Nutritional studies in relation to disease incidence.

Soil survey

The major soil types in Kerala as indicated by the survey are: (1) sandy soil of the coastal tracts; (2) reclaimed clayey soil of the backwater areas; (3) sandy loam between the backwater and the hill slopes; (4) laterite soil of the hill slopes and (5) alluvial soils of the river beds. Coconut grows in all these soil types and the disease is observed in all. However, the disease is more acute in areas with poor soil aeration such as waterlogged areas and soils with poor moisture retentive capacity, a high water table, shallow depth and poor drainage (Menon and Mair, 1951). It is interesting to note in this connection that other diseases of coconut under discussion here have a more or less similar distribution as the wilt disease. The distribution of cadang-cadang in the Bicol region is said to be the distribution of coconut plantations in that region (Price, 1958).

Similarly, bronze leaf wilt is reported to occur in waterlogged soils, soil with poor retentive capacity, in areas where the top soil is friable and the sub-soil layer intolerant and finally in well drained soils as well. The "unknown disease" is prevalent on a variety of soils although not very serious on lime stone soils (Martyn, 1948). An important point that one can infer from these observations is that these diseases cannot be caused by unfavourable soil conditions, but that they provide an environment conducive to "infection" by a biological factor, for vast stretches of healthy coconut tracts with the same soil conditions are found elsewhere - for instance in North Kerala. Indeed, that waterlogging is harmful to root growth is well known. Thus, although salycilic aldehyde which accumulates in soil under semi-anaerobic conditions did not influence the growth of sugar-cane in the absence of the fungus Pythium arrhenomanes and also the growth of the fungus in pure culture, it apparently so predisposed the plant to infection by the fungus as to cause a loss in weight 2 to 7 times greater than that caused by the fungus alone (Rands and Dopp, 1938)

The nutrient status of the soil was determined by analysing chemically representative samples of soil collected from a large number of places, both healthy and diseased. Moisture, organic matter, nitrogen, calcium, magnesium, phosphoric acid, potash, iron and also available potash and phosphoric acid were estimated in these samples (Sankara Subramoney et al., 1954, 1955, 1956 and Pandalai et al., 1958,1959). In general, in both healthy and diseased tracts, the essential plant nutrients were much below the minimum required for balanced nutrition of the coconut palm. The soils from diseased tracts have comparatively lower values for total calcium, available potash, total exchangeable base and pH. This finding is of considerable importance since, as one may notice, all these factors are inter-related - for instance, low calcium and potassium result in low base exchange capacity and a low percentage base saturation, which ultimately leads to acidity of soil. The normal physiological functioning of the palm, which requires large amounts of potash for a balanced nutrition, is thereby impaired bringing about a predisposing environment for the attack of facultative organisms. Although no details of chemical analysis on the nutrient status of the soil in relation to the other diseases are available, it has been reported that potash deficiency is associated with the bronze leaf wilt and Briton Jones (1940) states that his observations in Trinidad and St. Lucia suggest very strongly that the primary cause is a soil factor. Analysis of the various chemical and physical factors of the soil throughout Trinidad suggests that unbalanced nutrition coupled with the physical conditions of the soil and its water relationship constitute the primary cause of the disease (Bain, 1937). A compact subsoil which retards proper growth of roots or even drought or waterlogging resulting in a physiological drought have also been suggested. "Lethal yellowing" was considered to be caused by some toxic material probably strontium, present in the soil (Hansen, 1952). Innes (1949) reported inconclusive results as far as manganese deficiency is concerned with this disease while it is also reported that it is not associated with any particular soil condition, mineral excess or deficiency. Thus while contradictory statements are made in the case of "lethal yellowing" similar hypotheses of one or more deficient elements in the Philippines soils being responsible for the cadang-cadang are also reported to be put farward (Price, 1958).

Tissue analysis

The standard procedure of leaf sampling accepted for analysis was to take 40th to 50th leaflets from $(\frac{N}{2} + 1)$th leaf of a tree, N being the total number of leaves. Comparative analysis of leaf tissues indicated that the diseased leaves contained more of nitrogen, phosphoric acid, potash and ash (insoluble in hydrochloric acid) to the extent of 5.0 to 13%, 0 to 13.0%, 5 to 39.0% and 22.0 to 134.0% respectively (Table III).

As far as Ca and Mg were concerned, the results were inconclusive (Verghese et al., 1959). This imbalanced nutrition was noticed in diseased samples irrespective of the soil type from where they were collected. The insoluble ash was not further analysed but presuming it to be silica, the consistently higher concentration of ash (insoluble in hydrochloric acid) in diseased samples raises the suggestion that perhaps it might be toxic to the plant and that the lower N/insoluble ash ratio may be directly or indirectly connected with disease incidence. No difference was noticed in the percentage oil content of copra of diseased and healthy palms. The accumulation of the major nutrients in leaf suggests a defective metabolism or an impaired translocation.

Nutritional studies

Since most of the soil of different types analysed proved to be of low nutrient status, nutritional studies were started in order to gain an insight into the effect of different nutrients, both major and minor, on plant growth and disease incidence. Although there are indications that nutrient deficiency might be a predisposing factor for the disease, the nutritional studies were expected to throw more light on this aspect. For, if the deficiency of one or more elements in the soil was responsible for the disease, the incorporation of deficient element(s) in an available form into the soil is bound to restore the palms into normal health. The experime conducted come under four groups : (1) effect of major nutrients like N, P and K; (2) effect of individual nutrients like S and Cd and testing of proprietory fertilizers that appear in the market from time to time; (3) effe of trace elements (micro nutrients) like Mg, Ca, Fe, Mo, etc., and (4) pot culture experiments to study different macro and micro element deficiency symptoms on coconut seedlings.

Preliminary demonstration trials conducted in various plots representative of infective areas showed some favourable response of infected palms towards judicial manuring (Menon and Nair, 1952). The results of a manuring cum spraying experiment conducted at the Coconut Research Station, Kayangulam, during 1951-1956 showed a general improvement in the condition of the treated trees over that of the control. The manures in this case were applied at the rate of 0.227 kg. N, 0.227 kg. P_2O_5 and 0.907 kg. K_2O per tree per annum. The visual disease symptoms however persisted but in a lesser degree.

It was found that the application of Chilean nitrate on diseased trees at the rate of 0.907 kg. N/tree/year, with a basal dressing of 0.342 kg

P_2O_5 and 0.681 kg. K_2O had no appreciable effect on foliar condition of tree or on yield (John et al., 1959). This might be due to the beneficial effect of nitrogen on vegetative characters, an increased dose of which is known to increase the disease proneness of a plant. A randomised and replicated field experiment is in progress now to study the effect of high doses of NPK manures on incidence and development of the disease on trees at the prebearing stage. Twelve NPK combinations with three levels each, viz. no dose, normal and double dosage of the major nutrients are being tried. Effect of applying major and micro nutrients as foliar sprays is also being studied.

Since it was found that the soils were generally deficient in Ca and available potash, a field experiment was conducted for five years where 3.632 kg of lime and 27.25 kg. of ash per tree were applied per year over and above the basal dressing of NPK at 1:1:2. Here again, no favourable effect was noticed on disease incidence although the calcium content of leaves was increased showing almost normal uptake of this nutrient (Chettiar et al., 1959). Table IV shows the Ca and K content of leaves of palms growing under different conditions. The data show that the lime and potassium contents and the K_2O/CaO ratio of leaflets of coconut palms growing under varying soil conditions are not appreciably different whether the trees are healthy or diseased. Although comparatively low values are noticed for Ca in the leaf at the pretreatment stage, the Ca contents and K_2O/CaO ratio were almost normal in the final post treatment analysis.

It was noticed that the introduction of a 2% solution of cadmium chloride through cut roots caused sudden wilting due to cadmium toxicity, symptoms of which were not like those of root (wilt) disease. Spectographic analysis showed no trace of cadmium in the tissues of healthy and diseased palms. The effect of the following materials on diseased incidence is being studied at present: Chemotherapeutant 1182F (4 Chloro 3, 5-Dimethyl phenoxy - ethanol); the hormones B naphthoxy acetic acid, phenyl acetic acid, 2-4, dichlorophenoxy acetic acid (2-4, D), 2-4, methyl, 4 chloro-phenoxy acetic acid, indolacetic acid, indolbutyric acid and indol propionic acid, nutrients like urea and $CaCl_2$, and sequestrene compounds namely Fe, Cu, Na and Ca in organic form as 330 Fe, Na Cu, NaFe and Na_2Ca. Of these, urea and 2-4, D reduced yellowing considerably whereas the calcium injections slightly improved the flaccid nature of leaflets.

An experiment is in progress at the Central Coconut Research Station, Kayangulam, to study the effect of sulphur on incidence and development of the disease. A number of observational experiments are also in progress to study the effect of proprietory activated chemical fertilizers on the disease incidence.

An extensive statistically planned micro nutrient manurial experiment was started in 1953 with 384 single tree treatments to study the effect of B, Cu, Mn, Fe, Mo, Zn and Mg at a concentration of 25.9 g., 58.1 g., 55.8 g., 45.4 g., 1.0 g., 51.3 g. and 45.4 g. respectively singly and in combination. The nutrients, applied over a basal dressing of NPK fertilizers at 0.34: 0.34:0.68 kg./tree, lime at the rate of 101.6 kg./acre and a green manure crop of sannhemp grown in situ, were given as soil application for the first

five years. Subsequently and in order to avoid the absorption of these nutrients by other trees, the micronutrients were applied as solutions by the root injection method. In this method, the cut end of a healthy root is dipped in the nutrient solution kept in a suitable container buried in the soil. A single functioning root absorbs about 400 ml. of solution in 24 hours. While applying nutrients by the root injection method, the concentration is reduced to 1/1000 of the field dose, above which they are generally found to be toxic. The vast amount of data collected on morphological characters and yield are under statistical scrutiny. On an average, the yield of these 384 palms have been appreciably increased.

Sand culture experiments are in progress on one-year-old dwarf seedlings to study the deficiency symptoms of major elements and their probable relationship to the wilt disease.

In the case of cadang-cadang a nutritional experiment in which $MgSO_4$, KH_2PO_4 and $CuSO_4$ at 250 g., $MnSO_4$ and $ZnSO_4$ at 200 g., $NaMoO_4H_2O$, Na_3Bo_3 and $FeSO_4$ at 125 g. each per tree were applied at the base of diseased trees showed the majority of the trees so treated deteriorated further (Price, 1958; Subido, 1957). Similar trials with these salts applied as foliar spray also yielded only negative results (Price, 1958) thereby suggesting that the disease is not caused by any nutrient deficiency.

2. Pathogenic studies

One common feature of all diseases of unknown origin on coconut is that all seem to be infectious - at least, all these diseases have spread from the original foci of infection thereby suggesting to the majority of workers the pathogenic origin of these. Again, two more features common to all these diseases are: (1) the presence of a deteriorating root system and (2) chlorosis of leaves in one form or the other. Only a thorough survey was able to reveal that the root rot is associated with unfavourable soil conditions such as waterlogging, the presence of some impermeable layer or soils with poor retentive capacity. In association with these rotting roots, a number of saprophytic fungi and bacteria were found which were unable to reproduce the disease in the case of lethal yellows, cadang-cadang and wilt disease. This constituted a factor which was against the hypothesis of the biological origin of the disease. A survey and discussion of work done in this field on the three important diseases might yield useful information towards the future working of this problem.

Mycological work

The main difference that exists between the root (wilt) disease and the other diseases of unknown origin is that ever since investigations were started, it has been fairly understood that the former is of an infectious nature whereas theories as to the origin of the others have been many and varied.

Butler (1908) who visited the infected area suggested, after an exhaustive survey, that the disease manifests itself chiefly through alterations produced in the leaves and the roots. According to him, the leaf tissue becomes pale and flaccid probably due to a condition of drought and starvation. The lateral roots affected by rot were invaded by a parasitic fungus which was responsible for the death of the cortical cells. A species of Botryodiplodia isolated from the rotting roots was considered to be the causal agent of the disease. Systematic work on the disease which was started in 1937, however, revealed the complexity of the disease which was found to owe its origin not to any single fungal organism but to some unknown factor(s) (Menon and Nair, 1951).

A distinct deterioration of the roots of the infected trees is seen -- a deep brown discolouration of the cortex develops which later dries up in flakes. From a large number of isolations made from (1) rotting roots, (2) roots where rotting has just set in and (3) root tissues which were actually found to be attacked by intracellular hyphae, Botryodiplodia theobromae, Rhizoctonia bataticola and R. solani were found to be the organisms constantly associated with the root rot. A number of pathogenicity trials on 1-2 year old seedlings were conducted using the fungi and bacteria associated with root rot. A higher percentage occurrence of these three fungi, caused by the addition of artificially multiplied inocula in potted soil prevented the successful establishment of seedlings whereas other fungi and bacteria remained purely saprophytic. The root tips of 4-5 year old and mature palms however took up only local infections when inoculated by these fungi in situ and the foliar symptoms of the disease failed to develop in them. This type of restricted growth of these fungi inside living root tissue of healthy palms has been consistently observed (Menon and Nair, 1951; Menon, 1951-1961). However, it has been observed of late that when a tree is already diseased the roots lose much of their resistance, almost 100 per cent infection being met with in such cases. This strongly points out that these fungi are only secondary parasites and being soil inhabitants cause enough damage to the trees by root rot alone when other predisposing conditions are favourable or when the host resistance is reduced. This same phenomenon explains the infection in transplanted potted seedlings reported by Menon and Nair in 1951. Again, as reported by Menon et al. (1952), these fungi caused heavier infection on roots under waterlogged conditions, an environment detrimental to normal root growth. Such instances of heavy damage caused to crops are reported in the case of internal root rot of tea in Ceylon which formerly was considered to be caused by Botryodiplodia theobromae and which later was found to be caused by the lack of starch reserves in the root, due to excessive plucking and pruning, the fungus having only a secondary role (Gadd, 1928 and 1929). Indeed, Radha and Menon (1954) reported the cosmopolitan occurrence in large numbers of B. theobromae in healthy palms in both healthy and diseased areas. Although R. bataticola occurred in all samples, only fewer numbers of this were isolated from the healthy areas than from the diseased. R. solani, however, was isolated only from the diseased samples.

A study of the root system and its deterioration in relation to the manifestation of external foliar symptoms becomes imperative at this

stage. The root system of coconuts affected by cadang-cadang was reported to be rotted, certain fungi and bacteria sssociated with this being considered to be only secondary (Ocfemia, 1937; Price, 1958). Similarly, no root-invading organism was reported as being associated with the root decay found extensively in the case of bronze-leaf wilt by Briton-Jones (1940) who considered the disease occurring in Trinidad as a non-parasitic wilt. In Jamaica, the trees affected by the "unknown disease" had roots which were becoming rapidly moribund but in the early stages of disease no parasitic organism was found to be associated (Martyn, 1949). Isolates of Rhizoctonia sp. and other saprophytic soil fungi obtained from the decaying roots of palms affected by the above disease produced, on inoculatio only local infection (Leach, 1946), a phenomenon comparable to the one found in the case of wilt disease of South India.

Detailed studies on the root system of palms at different stages of disease in comparison with that of the healthy showed that root decay is more or less a normal phenomenon (Table V). Trees in the early stages of disease have a root system similar to that of the healthy, the percentage of trees showing above 50 per cent root decay being the same in both. It was found that as the disease progresses root decay also progresses with the percentage of trees showing above 50 per cent root decay increasing from 50 in trees in the middle stage of disease to 91.7 per cent in those of the advanced stage (Nagaraj and Menon, 1955). Recent work showed that 38 per cent of the rotting or damaged roots were replenished by branching in healthy while only 7 per cent were replenished in the diseased.

A study of the rhizosphere and general soil microflora of healthy and diseased tracts revealed a consistently lower rhizosphere effect in diseased palms than that in the healthy in sandy soils. Within an infected tract of sandy soil, the rhizosphere effect, being highest in healthy trees, progressively becomes lower in the diseased (Table VI). The factors influencing the concentration of microflora in the rhizosphere of any plant are too well known and as such the results described above do not require further explanation. The rhizosphere microflora is comparatively low in diseased plants in many crops and in a number of cases varietal susceptibili also is correlated with it (Timmonin, 1940; Agnihotrudu, 1953). In fact the potential parasite present in the rhizosphere may be expected to become aggressive when the general competition from other micro-organisms is low, especially at a time when the resistance of the plant is lowered by other environmental factors. It is worth noting in this connection that recent investigations show a comparatively low microbiological activity, as indicat by soil respiratory studies, in soils at the base of diseased trees affected by the wilt (unpublished data).

Virological studies

Menon and Nair (1951) suggested that in view of the spreading nature of the disease and considering that the fungi causing root rot are only secondary, some infectious virus might be involved in the wilt disease. The systemic type of symptoms and the nature of its manifestation from the younger leaves downwards support this suggestion.

The nature of the crop again was a handicap in the successful investigation of this aspect because vegetative propagation of any type is, except in some abnormal cases, practically unknown in coconut. Thus grafting, the primary and yet the most important test to demonstrate the presence of an infectious virus of any plant, is not possible on coconut and all attempts to get even a few successful root grafts failed.

After several attempts at transmission by different kinds of insects and by different mechanical means such as the pin prick, abrasion and leaf insertion, Nagaraj and Menon (1956) succeeded in reproducing the disease on a few healthy plants in the field by the abrasion method and by means of the insect Stephanitis typicus, commonly known as the banana lace wing bug (Table VII). Thus, the presence of a sap transmissible virus with an active insect vector was indicated, although the experimental set up had its drawbacks which delimited the value of the results obtained. Due to the absence of proper controlled conditions, especially an insect proof house, the experiments were conducted in the field in an already infected tract where other contributing factors were present. This drawback was sought to be compensated for, by maintaining an equal number of uninoculated palms under identical conditions (Table VII). Similar results were consistently obtained in the later experiments conducted in the field. While testing the susceptibility of the plants of different age groups, it was found that the 8-20 years age group was most susceptible, a finding conforming to the general field observations. Generally seedlings below 4 years were seldom affected.

A high percentage infection was obtained in the field by maintaining a continuous flow of the sap from the root of a diseased palm to that of a healthy for 1-2 months by the method perfected and adopted by Nagaraj et al (1954) as a substitute for grafting which was not possible in coconut (Table VIII).

A number of workers have suggested a viral origin for the other diseases of unknown origin. Thus Martyn (1949) suggested that the "unknown disease" of Jamaica might be caused by some toxic substance present in the soil or by a virus, pointing out that the storage in large quantities of starch in the cortex of the root might be due to the absorption of some substance which is of a viral or toxic nature through the roots. Although the nature or irregular spread was suggestive of the action of an insect vector, no control to the spread was obtained by spraying of DDT. Therefore some soil inhabitant was suggested as the vector which was responsible for the rapid spread of the disease in certain types of soil. The frequent appearance of double or more nuclei in the cells of leaf, petioles, bases of the developing nuts and sometimes even in the roots of palms affected by lethal yellowing, the presence of one or more substances capable of acting as antigens and the epidemiological data were pointed out by Nutman and Roberts (1955) to support the virus theory. Van Weerdt and Martinez (1959) suggested after considering the different factors involved in the disease that "lethal yellowing" might be caused by some ectoparasitic nematodes or by a virus. Celino (1947) reported successful transmission of the cadang-cadang by needle pricks into healthy palms. Although Kent (1953) reported

the reproduction of the symptoms through the insect vectors Cicadella unimaculata and C. bipunctiformis, Price (1958) presented data to show that none of the 9 insects tried, including the above two, were vectors of the disease. Several parasitic forms of nematodes were obtained from the roots of cadang-cadang affected trees and Price (1958) pointed out that this in itself was confusing because of the unlikelihood of more than one parasitic form of nematode being involved in a disease of this type. However, it must be mentioned that none of the nematodes isolated from the healthy trees belong to the known parasitic species. McWhorter (1959) reported the presence of the plant Pandanus coplandii M. in large numbers in the Philippines with foliar symptoms similar to cadang-cadang. A serological test, suggestive of the presence of a specific protein matter is said to be developed in the Philippines (Holmes, 1961). Production of specific symptoms on seedlings of maize by mechanical inoculation and by feeding of mealy bugs collected from diseased palms suggest that the infectious agent is likely to be a virus.

Since comparative histopathological studies revealed binucleate cells in the tender tissues of both healthy and wilt diseased palms, this cannot be taken as a phenomenon specific to the disease. Indeed, binucleate cells in tissues of normal healthy coconut palms have been reported by Patel (1938). However, proliferation of phloem in the tender leaves of diseased palms have been noticed, a phenomenon associated with many known plant virus diseases (unpublished data). Chlorosis of leaves is found to be caused by a disruption and disintegration of the chloroplastids caused perhaps by the accumulation of some toxic material.

The presence of a sap transmissible virus in the wilt diseased palms was further demonstrated by the successful transmission of a virus from diseased palms into seedlings of cowpea (Shanta and Menon, 1960). This was considered to be of definite advantage because cowpea is easy to be handled and because in this host the virus has an incubation period of only 4-9 days as against 8-12 months in coconut, so that further studies on the virus were facilitated by the use of cowpea as an indicator plant. The insect Stephanitis typicus, generally feeding and breeding in large numbers on leaves of coconut in the infected tract, was confirmed as a vector of the virus.

Transmission trials are in progress at the Central Coconut Research Station, Kayangulam, on potted 4-year old coconut seedlings grown in sterilised soil in the insect-proof house. Unless and until symptons are reproduced in these, the role of the virus as a primary causal agent cannot bo finally established.

The virus involved in the wilt disease is fairly stable (about 3 weeks) in extracted leaf sap at room temperature, has a longevity of 8-9 weeks at -4° C, and a thermal inactivation point of 76° C. The titre of the virus ranges from 10^{-2} to 10^{-4} in the leaves of diseased palms. It has a fairly wide host range confined to the natural orders Leguminosae, Solanaceae and Palmae. It has been isolated from the roots of a number of common weeds occurring in coconut gardens and is found to be soil transmiss:

mainly by root contact. Since the virus also occurs in the clay and fine silt fractions of infective soil, it is quite probable that it spreads through flood water and through rivers. In this connection, it might be pertinent to point out that this disease occurs to a considerable extent along the banks of rivers and canals. Work is in progress to find out whether any soil inhabitant acts as an active vector of the virus. Also work is in progress to see whether any parasitic soil nematodes are associated with the disease. Although the virus has been isolated from roots, root sap and leaves of diseased trees, only in two cases was it obtained from anthers and endosperm (unpublished data). Therefore nothing definite can be said at present whether the virus is transmitted to the progeny or not.

Another limitation of these studies has been that only from 19 of the 29 trees, in the early stage of disease tested, was the virus isolated on cowpea. This might be due to (1) that the concentration of the virus in the coconut at the early stage of disease was not enough to produce symptoms on cowpea or (2) that the virus was not present in the coconut at the time of the test since it might not be the primary cause of the disease. This can be clarified only by serological studies and by further transmission trials on coconut under controlled conditions.

An observation trial plot of 3.67 acres of underplanted seedlings was used for 4 years to study the effect of spraying 0.2% DDT at fortnightly intervals. It was seen after the experimental period that 21 per cent of the treated and 34 per cent of the control trees were diseased, a finding which supports the virus hypothesis especially when one considers that there was every possibility of the disease spreading by other means, viz. through soil or by root contact with mature palms in the same plot.

The phenomenon of accumulated nutrients especially nitrogen in the leaves and tender tissues of affected palms, which may be either due to the inadequate translocation or due to an impaired physiological functioning is comparable to the accumulated nutrients common to many plant virus diseases, viz. sandalspike and potato leaf roll (Smith, 1957).

3. Diagnostic tests and physiological studies

Whatever be the aetiology of the disease, diagnosis of it in the early stage is essential especially since it is known to be spreading to new areas. The limitations in using cowpea as the indicator plant have already been discussed, but for the time being this is the best test plant met with. A colour test based on the dehydrogenase activity of tender leaves is being standardised since it is found that trees in the early stage of disease have higher dehydrogenase activity.

The walls of bundle sheaths and of the fibres below the epidermis of leaves of affected palms are generally highly thin due to the lack of lignin resulting in the drooping of affected leaves (unpublished data). This phenomenon is also made use of in identifying diseased palms.

Finally, work on the antigenic properties of the virus has been started with a view to develop a specific diagnostic test to identify the disease in the very early stages.

Physiological studies indicate a high rate of transpiration in the affected leaves which is supported by the observation of an increased number of stomata per unit area in the diseased leaves.

INHERITANCE OF THE DISEASE

An observation field trial to study the performance of seedlings raised from healthy and diseased parents indicated a higher percentage infection in the progeny of the diseased mothers. Another trial is under way in which the performance is being studied of the progeny raised after artificial crossing between 4 father and 25 mother palms that seemed to be apparently resistant to the disease.

CONTROL

Since a review of work done so far shows that no specific agent has been found to be the primary cause of the wilt disease, no definite control program can be drawn out at this stage. Nevertheless, the finding that the soils of the diseased tract are predominantly of a low nutrient status, which no doubt is one of the contributing factors of the disease points out by itself that better manurial practices would augment the disease resistance of the trees. Similarly since soil conditions detrimental to the healthy growth and normal functioning of roots are found to favour disease incidence, cultural practices that would set these factors right as far as possible should be taken up. Thirdly, good crop hygiene which is an essential step to be adopted in the effective control of any infectious d would, to a large extent, help in the control of the disease. While these are simple but effective measures that should be followed closely by cultiva themselves, the best method of control that should be scientifically worked out is the introduction of resistant trees that have withstood the ravages of the disease and from imported forms that are found to be resistant; for the effective working of which, in a crop like coconut, public cooperation is essential. While this is being done, small scale trials on the use of soil fungicides, insecticides or nematicides may be tried, but as pointed out by Garrett on the control of root disease fungi of plantation crops, this might be of only limited value.

A few practical measures in an already infected tract would be: (1) cutting down and removal of trees in the advanced stage of disease since these do not give an economic yield and immediate destruction of the stump and root system by the injection of some poisonous chemical such as sodium arsenite; (2) replanting with quality seedlings; (3) manuring all palms with NPK fertilizers at the rate of 0.34 kg. N, 0.34 kg. P_2O_5 and 0.681 kg. K_2O per tree per year, the manures being applied in August-September and judicial liming of the soil in view of the low pH of the diseased soils; (4) adopting regular cultural practices that are generally practised depending on the different soil types so as to retain general

fertility status of the soil and "Cordon sanitaire" and (5) regular extension work so as to give the general public a scientific awareness.

The last step mentioned above would by itself make the enforecement of quarantine measures most effective in preventing the spread of the disease into new areas. In case the disease is introduced into new areas, quarantine and immediate and proper use of the suitable chemical, whether it be fungicide, insecticide or nematicide, should prevent the further spread of it.

DISCUSSION

As suggested by Martyn (1955) a study of the symptomatology, epidemiology, etc. of the four important infectious diseases indicate a close relationship between the diseases of West Africa and the West Indies on the one hand and those of Philippines and South India on the other. Stunting of leaves and retardation of general growth resulting in a tapering stem with a small crown are features common to both cadang-cadang and the wilt disease. Progress of both the diseases are slow, an infected tree living long after it has become completely barren. In the absence of any positive experimental data no definite conclusion as to the aetiology of the disease can be arrived at.

Although it is seen that the disease is prevalent in all types of soil, areas with poor soil aeration such as waterlogged or highly porous soils favour the disease, that is, soil conditions unfavourable to normal root growth in general. Thus, root decay is an imperative factor commonly met with in all wilt diseased palms. Yet, root decay is not associated with this disease alone. Thus a palm with tapering stem, a phenomenon generally associated with senility of the palm, has very few normal functioning roots. Similarly any impermeable layer below the surface or even a poorly drained soil causes root decay and subsequent yellowing of leaves indicating thereby that root decay is a natural outcome of the physiological conditions of the surrounding medium, and not the cause of the disease. That this is so is further proved by the fact that no disease is observed in North Kerala where the soil conditions are more or less the same as are obtained in the affected tracts. Therefore, these unfavourable soil conditions form an environment conducive to infection to a biological factor, the severity of which is intensified by the enhanced disease proneness of the host caused by the same unfavourable soil conditions.

Again, it is found that soils on which the disease occurs are predominantly of low nutrient status, especially total calcium, available potash, base exchange capacity, percentage base saturation and pH. In this connection it would be pertinent to quote Garrett (1944) who exercises caution regarding interpretation of observations made in field or environmental factors - "field observations may be misleading owing to the common association between soil acidity, lightness of texture and poverty in plant nutrients. Conclusions are more trustworthy for diseases of great economic importance; field observations on such diseases have usually been supplemented by experiments. Such experiments are more easily performed with diseases of annual or field crops than with diseases of plantation crops; for this

reason, evidence available for correlating incidence of disease with soil conditions is both more abundant and more reliable for field crops than for plantation crops". Rectification of the soil reaction by application of lime and of any nutrient deficiency caused by low potash status by manuring has so far failed to actually cure the disease. However, these practices have served to improve the nutrient status of the soil and also the general condition of the tree which again tend to point out that the poor nutrient status of the soil is another factor that favours disease incidence.

The occurrence of Botryodiplodia theobromae, Rhizoctonia bataticola and R. solani in association with root rot presents an interesting problem. In view of their cosmopolitan occurrence and because of their presence on dead roots of even healthy palms, B. theobromae and R. bataticola can be only considered as secondary invaders of a non-living medium or of one already very low in vitality. Indeed that this is so in the case of internal root rot of tea is shown by Gadd (1928-1929) who reported the secondary invasion by B. theobromae of tea roots deprived of their starch by heavy pruning of the bushes. Similarly, Napper (1939) observed a fairly close relationship between the rate of loss of starch from the roots and the rate of invasion by saprophytic fungi. Thus, while B. theobromae and R. bataticola remain as saprophytes, the role of R. solani seems to be more specific in relation to the root rot of this disease. However, since studies show that root decay is only the effect and not the cause, it might be possible that the role of R. solani is only secondary.

From the infectious and systemic nature of symptoms, it becomes apparent that the biological entity that is involved might be a virus if not a parasitic nematode. All preliminary trials failed to indicate the presence of a parasitic nematode, work on which is still in progress but the probable occurrence of a sap transmissible virus was soon demonstrated, the presence of which was later confirmed. One factor pointing out that this virus might be the causal agent is that a higher percentage infection is consistently obtained on healthy palms in the field by inoculation of the virus than is normally obtained by natural infection (Table VII). However, till the disease is reproduced experimentally under controlled conditions, this theory cannot be confirmed although it is quite probable that, given the relevant soil conditions, the symptoms might be reproduced, for which necessary, controlled, nutritional experiments have to be done. The finding that the virus is soil transmissible, especially by root contact is interesting in view of the slow mode of spread of the disease. The presence of an insect vector which actually breeds on coconut and the occurrence of a wide host range for the virus seem only to complicate the problem further. Search for the presence of a soil vector(s), and if any, its/their role in spreading the disease is in progress.

TABLE I. Comparative data on distribution, synonyms and general symptoms of the different diseases of unknown origin

	Lethal yellowing or Unknown disease	Kainecope disease	Bronze leaf wilt	Cadang-cadang	Root (wilt) disease
Distribution	The Caribbean Islands, North Caribbean Coast of Panama and Central America, Florida Coast in U.S.A. and Jamaica	French Togoland.	British Guiana, St. Lucia, Tobago and Trinidad in West Indies and Nigeria.	Philippines.	Kerala in South India.
Synonyms	West-end Bud Rot, Pester field disease, Unkonwn disease.	Unknown disease.	West-end Bud Rot Maturation wilt.	..	..
Symptoms	Palms of all ages are susceptible. Premature shedding of nuts, necrosis and blackening of inflorescence, yellowing of outer whorl of leaves accompanied with soft rot of the central spike. Unilateral development of nut fall and root decay also is noticed. Affected trees die within 4-6 months after symptoms first appear.	Bacterial rot. Symptoms resemble the unknown disease of Jamaica.	Palms 15-30 years old are affected. Yellowing and later bronzing of outermost leaves spreading centripetally. Bacterial rotting of cabbage takes place subseqently. Nut fall takes place and the spathes turn brown from tip backwards. Root decay also takes place. Affected trees die within 4 to 6 months.	4-year old seedlings onwards are affected although bearing palms are most susceptible. Development of yellow or orange spots on young leaves, mosaic mottling on outer whorl of leaves, a bronzing of veinlets of affected leaves and rounding and scarification of nuts. Nuts become small in size, leaves fall off in quick succession and the tree remains barren. Root rot also is noticed. Affected tree dies only 5-10 years after symptoms appear.	4-5 years old seedlings onwards are affected; pal are most susceptible when they just start bearing, i.e. 6-10 years old ones. Flaccidity of leaves and abnormal bending of petio marginal and tip necrosis of leaflets, abnormal nut fall, yellowing of leaves and stunting of the crown are the major symptoms. Progressive deterioration of root system accompanie the development of foliar symptoms. Affected trees although barren may remai alive for 10-15 years aft appearance of symptoms.

TABLE I. Comparative data on distribution, synonyms and general symptoms of the different diseases of unknown origin (Cont.)

	Lethal yellowing or Unknown disease	Kainecope disease	Bronze leaf wilt	Cadang-cadang	Root (wilt) disease
Soil	Varied soil types both acidic and alkaline		Soils where the surface is of a close texture, where soil and subsoil are of open texture and where top soil is friable and sub-soil layer intolerant. Also in well drained areas.	Varied soil types.	Soils of all types.

TABLE II : Disease incidence in an already infected plot for the period 1954-1958

Block No.	1954				1955				1956				1957		1958	
	I	V	VI	VII	I	V	VI	VII	I	V	VI	VII	V	VI	V	V
Total number of trees	416	351	458	397	406	330	452	388	395	326	427	386	314	413	313	410
Healthy	127	73	88	68	131	61	85	67	130	61	81	65	48	66	49	58
Wilt	247	234	318	273	231	225	317	269	204	232	300	267	228	298	242	318
Yellowing with nut fall	22	27	31	41	23	32	37	40	43	24	31	42	27	37	12	22
General chlorosis	4	7	14	6	5	2	7	5	4	2	8	5	4	5	3	4
Tapering disease	16	10	7	9	16	10	6	7	14	7	7	7	7	7	7	8
Number of trees cut due to wilt					10	21	5	7	9	1	25	2	12	14	1	3
☆ % of wilt-affected palms	59	67	69	69	58	70	70	70	54	72	72	70	75	75	79	80

☆ Percentage of wilt is calculated in respect to the total number of trees taken for observation originally in 1954, so as to find out its actual increase every year.

TABLE III. Average values for the nutrient content of leaves of healthy and diseased trees

	Sandy Soil		Loamy Soil		Clayey Soil		Laterite Soil	
	H	D	H	D	H	D	H	D
N %	1.88	2.12 (+12.76)	1.75	1.97 (+12.57)	1.81	2.00 (+10.52)	1.92	2.01 (+ 4.69)
P_2O_5 %	0.39	0.47 (+ 2.05)	0.41	0.41 ..	0.31	0.35 (+12.90)	0.39	0.40 (+ 2.56)
K_2O %	1.53	1.67 (+ 9.15)	0.98	1.45 (+ 4.80)	1.51	1.61 (+ 6.62)	1.17	1.63 (+39.31)
CaO %	0.44	0.49 (+11.36)	0.46	0.42 (- 8.70)	0.35	0.49 (+45.17)	0.59	0.59 ..
MgO %	0.34	0.26 (-25.53)	0.24	0.28 (+16.66)	0.31	0.47 (+45.31)	0.39	0.34 (-12.82)
Percentage of ash insoluble in HCl.	2.67	5.22 (+95.50)	1.94	4.53 (+133.60)	3.09	3.78 (+22.32)	3.44	5.46 (+58.72)

H = Healthy D = Diseased. The figures in brackets represent the percentage increase (+) or decrease (-) of the nutrient element in the diseased leaf over that of the healthy.

TABLE IV. Calcium and potassium content of leaves of healthy and diseased palms

Description of sample	Healthy			Diseased			Authority
	CaO	K_2O	K_2O/CaO	CaO	K_2O	K_2O/CaO	
aring tree	0.28	0.56					Sampson, 1923
rees growing in ifferent types f soil, including andy soil	0.46	1.30	2.83	0.50	1.59	3.18	Verghese et al. 1959
rees from sandy soil	0.44	1.53	3.48	0.49	1.67	3.41	- do -
rees treated with lime and ash							
re-treatment values ost-treatment values				0.24 0.54	1.03 2.08	4.29 3.85	Chettiar et al. 1959
ntreated control							
re-treatment values ost-treatment values				0.22 0.055	1.26 2.29	5.70 4.16	- do - 1959

TABLE V. Percentage root decay in diseased trees of early, middle and advanced stage

	Healthy		Diseased					
			Early		Middle		Advanced	
	No.	% age of total	No.	% age of total	No.	% age of total	No.	% age of total
Total number of trees exposed	45		19		10		12	
Trees without root decay	12	26.7	6	31.6	..	..	..	..
Trees with root decay								
Root decay 1-10%	11	24.4	2	10.5	1	10	..	..
Root decay 10-20%	1	2.2	3	15.8	1	10	..	..
Root decay 20-30%	1	2.2	1	5.3	1	10	..	..
Root decay 30-40%	4	9.0	1	5.3	2	20	..	..
Root decay 40-50%	3	6.7	1	5.3	..	..	1	8.3
Root decay 50-60%	3	6.7	2	10.5	2	20	1	8.3
Root decay 60-70%	3	6.7	..	..	1	10		
Root decay 70-80%	2	4.4	..	..	2	20	2	16.7
Root decay 80-90%	3	6.7	1	5.3	..	..	3	25.0
Root decay 90-100%	2	4.4	2	10.5	..	..	5	41.7

TABLE VI. Rhizosphere flora of diseased and apparently healthy palms

Condition of trees		Number of			Rhizosphere effect on		
		Fungi	Bacteria	Actinomycetes	Fungi	Bacteria	Actinomycetes
Healthy	R	375	530	2.5	63.98	252.6	4.16
	S	5.86	2.1	0.6			
Early stage of disease	R	250	620	1.5	51.64	258.3	3.0
	S	6.03	2.4	0.5			
Advanced stage of disease	R	137.5	340	0.3	30.09	120.4	1.6
	S	4.57	2.8	0.5			

R = Rhizosphere S = Soil

TABLE VII. Results of virus transmission trials conducted in the field up to 1955

Expt. No.	Treatment	No. of plants infected	Percentage infection	Incubation period
I	Sap inoculation	5/7 *	71	8-9 months
	Uninoculated control	-/6	-	-
II	Sap inoculation	2/5	40	8-10 months
	Uninoculated control	-/5	-	-
III	Sap inoculation	3/5	60	9-11 months
	Uninoculated control	1/5	20	11 months
IV	Sap inoculation	4/9	49	11-12 months
	Uninoculated control	1/9	11	16 months
V	Sap inoculation	3/5	60	8-9 months
	Uninoculated control	1/5	20	9 months
VI	Feeding infective S. typicus	5/9	55	8-9 months
	Uninoculated control	-/8	-	-
VII	Feeding infective S. typicus	6/10	60	8-10 months
	Uninoculated control	1/10	10	14 months

Numerator indicates the number of palms infected and the denominator the number used for each treatment.

TABLE VIII. Results of root sap transfusion trials

Age of Palms in years	Number of palms		Incubation period
	Inoculated	Infected	
3-4	2	-	
6-10	6	3	9-10 months
12-20	6	5	9-10 months
40-50	6	3	9-10 months

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