20(3), 58-68, 1972.

The Possible Cause of Lethal Yellowing Disease of Coconut

K.G. Heinze,[1] FAO entomologist/virologist, *M. Schuiling*, FAO associate expert, and *D.H. Romney*, Coconut Industry Board, Kingston, Jamaica

The devastating disease of coconut palm known as lethal yellowing in the Caribbean area has been reported in the Bahama Islands (18), Cayman Islands (29), Cuba (8,21), Dominican Republic (28), Haiti (18), Jamaica (5, 10, 18) and Key West, Florida (20). In Togo this disease is known as kaincopé (9,24), in Ghana as Cape St Paul wilt (19, 31) and, in Surinam, Coronie wilt is believed to be similar but with a different strain of the causal agent involved. Although no definite conclusion has been reached concerning the inciting factor for the syndrome of lethal yellowing, it has been regarded by most investigators as an infectious disease caused by either bacteria, fungi or viruses (1, 4, 5, 11, 12, 13, 14, 15, 17, 26). Information on the ecological conditions favouring the development of the disease is also limited.

On the basis of symptomatology, typical lethal yellowing can be distinguished from other leaf spot disease and also from bud rot. In young palms with lethal yellowing disease, the first symptoms of yellowish discoloration on distal parts appear on the oldest leaves, whereas bud rot is characterized by brown discoloration and decay in the youngest leaves and the spear while the older leaves remain green. Decay of the spear and the partly folded leaves are symptoms of the last stage of lethal yellowing disease. This period of slow decay may last for several months. The symptoms in bearing palms are premature nut fall, blackening of young inflorescences, premature opening of spathes and root rot. The discoloration of the inner part of the cabbage is accompanied by a typical odour, which is a good diagnostic feature (4).

The present paper reports the results of investigations that were undertaken to determine whether a virus might be the causal agent of lethal yellowing disease.

Mode of disease spread

In an experimental plot containing both San Blas (now called Panama Tall) and Jamaica Tall palms and a few Malayan Dwarf hybrids at Silverstock, near Buff Bay, Jamaica, the progress of lethal yellowing was observed over a period of about three years, beginning in October 1963, when only two palms showed symptoms of lethal yellowing (Figure 1). In 1964-65, seven new cases of the disease were observed, five near the first outbreak and two at some distance from it. Subsequently, lethal yellowing spread like a vector-transmitted disease through the plot, sometimes jumping across several rows of coconut palms (Figure 2). At the end of 1966, nearly half of the palms at Silverstock were diseased or dead (Figure 2), with about 30 to 40 palms being infected each year; further increased incidence of infection was noted toward the end of the observation time. The diseased palms in this plot were presumably the sources of infection, but new introductions of lethal yellowing into the plot from outside during the observation period cannot be excluded. The manner in which the disease spread strongly indicates a vector-transmitted disease.

Evidence for an insect vector

Although experimental transmission trials at Caenwood with suspected insect vectors

[1] Present address: Biologische Bundesanstalt, 1 Berlin 33, Dahlem, Königin-Luisestr. 19, Federal Republic of Germany.

11. POULSEN, K.K. Parents used in the development 1969 of Mexican wheat varieties. *Information Bulletin on the Near East Wheat and Barley Improvement and Production Project*, 6(2): 33-37.

12. SANTIAGO, J.C. *Resultado das observacoes effectuadas em Marrocos e na Tunisia respeifantes as doencas e pragas dos cereais, principalmente em relação aos trigos Mexicanos cultivados nestes paises: missoes de estudo realizadas em 1969 a expensas das Missoes Americanas de auxilio tecnico a Marrocos e Tunisia.* Elvas, Portugal, Estaçao de Melhoramento de Plantas. 12 Junho de 1970. 22 p. (Mimeographed)

13. SEARS, E.R.,Wheat cytogenetics. *A. Rev. Genet.*, 1969 3: 451-468.

14. STEWART, D.M., GILMORE, E.C. JR. & AUSEMUS, 1968 E.R. Resistance to *Puccinia graminis* derived from *Secale cereale* incorporated into *Triticum aestivum.Phytopathology*,58(4):508-511.

15. STEWART, D.M. *Research and control of pathogens* 1971 *and pests. Improvement of field crops productivity in the U.A.R.* United Nations Special Fund Project U.A.R. 30, April 1971. 51 p. (Mimeographed)

Corrigenda

Vol. 20, No. 2, April 1972 — Mycoplasma-like bodies in the leaves of orange trees infected with greening disease.

Cover and page 28, title: *for* Roger Buvat *read* J.M. Bové

Page 30, column 2, line 28: *after* disease *add* is 32° C. whereas that of greening

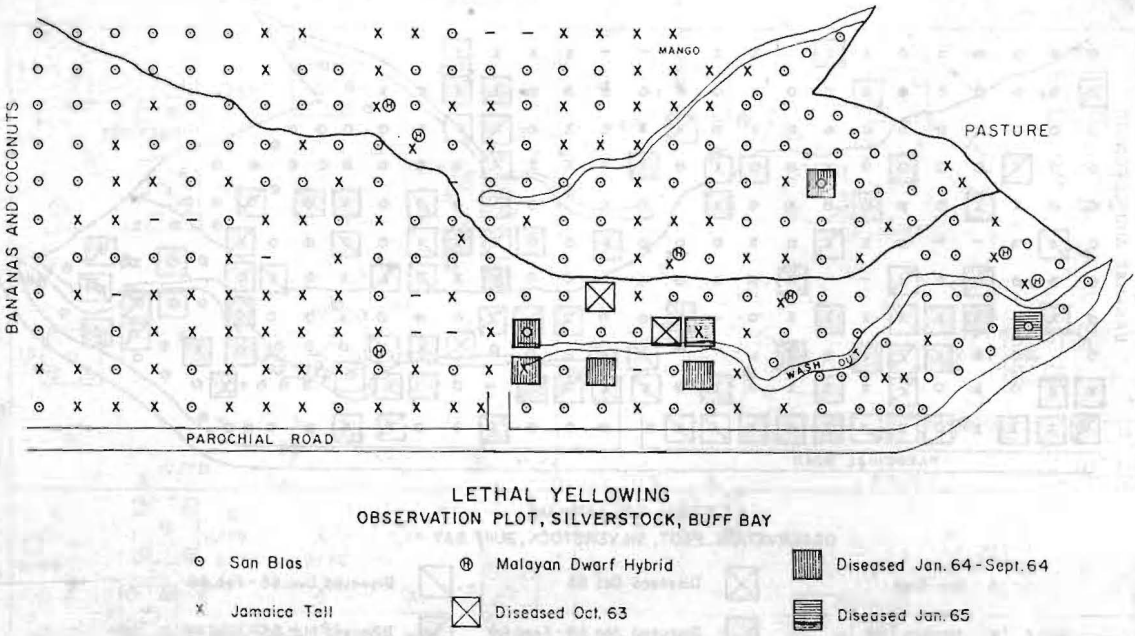

Figure 1. The first outbreak of lethal yellowing in a coconut grove at Silverstock/Buff Bay in 1963 and the spread of the disease during the following year.

failed, it was concluded that lethal yellowing is an infectious disease (with an airborne vector), the causal organism probably being a virus or a mycoplasma-like body.[2] Eighteen insect-proof cages (3 × 3 × 3 metres) supported by slotted aluminium frames were erected and five young healthy Jamaica Tall palms were planted in each one, either in drums or in the soil (Figure 3). One year later, five more similar cages were erected, each enclosing five palms, with the centre palm three years old and the outer four one year old. In two cases the centre palm was diseased and in the remaining three cases it was healthy (Figure 3). A number of palms planted between the cages remained unprotected (most were one or two years old by 1970 but some were three or four years old) and were thus exposed to natural infection. To test whether the fauna of the undergrowth had any influence on the transmission of lethal yellowing, the area was divided into four subplots, all of the undergrowth was carefully removed and the subplots were maintained clean, whereas the undergrowth was allowed to grow on the other two subplots and was merely slashed when it became too luxurious.

Fifteen of the older unprotected palms were infected with lethal yellowing, showing distinct symptoms by April 1970. In May-June of the same year, six more of the older palms were diseased, three more in July-August and another by the end of November. None of the experimental coconut palms in cages showed any signs of infection up to October 1970.

In November 1969 the cage was removed from one group of palms and one of the palms was diseased by June 1970. A second cage was removed in February 1970 and two of the palms were diseased by November 1970. Insect transmission trials were also carried out during this time, but as they were unsuccessful it was concluded that the correct vector species was not being utilized. However, it was evident that the cages gave full protection against the vector species moving around in the area. The

[2] In research institutes in Yonkers, New York, East Malling (England) and Berlin mycoplasma-like bodies were found to be associated with lethal yellowing disease.

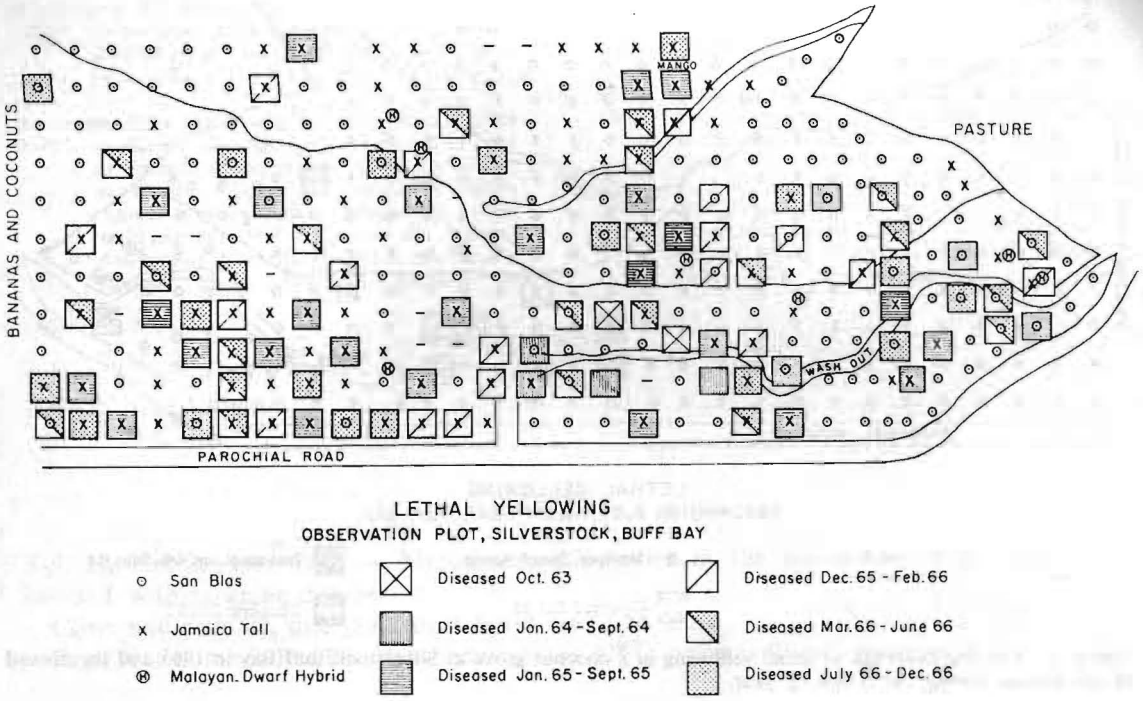

Figure 2. The progress of lethal yellowing at Silverstock/Buff Bay in 1965 and 1966.

results also exclude the possibility that soil organisms are vectors of lethal yellowing (17), since some of the palms in the cages were planted in the soil.

Among the one- and two-year-old unprotected palms no disease was detected in July-August, but seven palms showed symptoms in September, as did five more in October (Figure 3). This suggests that very young palms have some resistance to the disease, as the first symptoms did not appear before September, whereas 25 of the 28 older palms were displaying symptoms by October-November 1970.

Incubation period of the disease

With reference to the cases in which the palms became infected after the cages were removed, on both occasions the trees were exposed for a period of seven to nine months before the first symptoms were observed. If infection occurred soon after cage removal, the results imply that an incubation period of seven to nine months is required in this disease system.

In a "transfer experiment," sets of healthy palms in drums were transferred from Kingston to a diseased area, Fair Prospect, for two to three months and then returned to Kingston for further observation. The first set was transferred in May 1969 and subsequent transferals were made every one and a half to two months. One palm of the first set which was exposed to infection for two months developed leaf symptoms at the end of August and necrosis at the beginning of September 1969. The syndrome was fairly typical of lethal yellowing and quite unlike bud rot. The period between initial exposure and the appearance of symptoms was therefore about three and a half months. One palm in set 4 which was exposed for three months showed symptoms of lethal yellowing after five and a half months, but died during the observation period. All other sets of palms were exposed for a period of three months, and the period of time between exposure and symp-

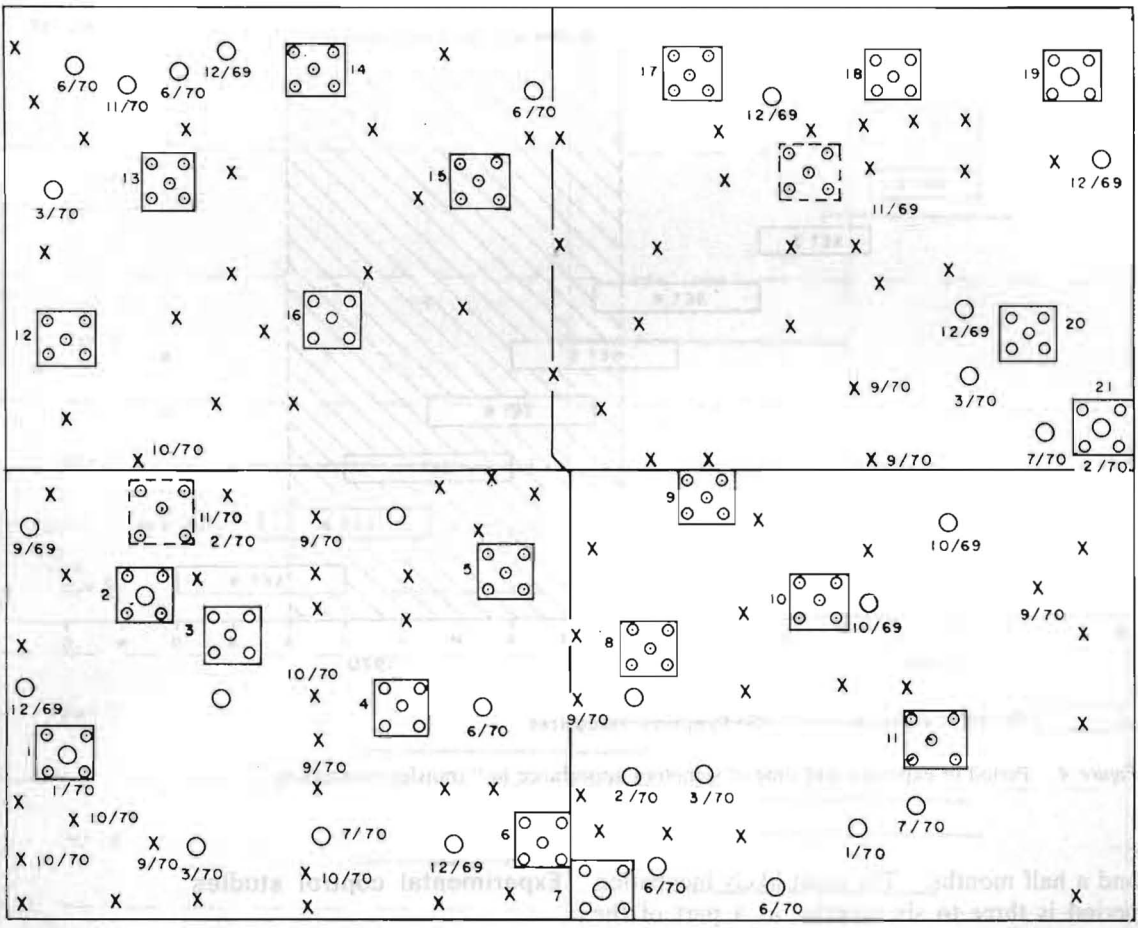

Figure 3. Experimental plot at Caenwood, groups of coconut palms in cages, and interplanted uncovered palms.

Insect cages covering five coconut palms (⊙ — in drum, ○ — in soil)
Cages 1, 21 were erected in 1/70 around a diseased three-year-old palm, and four one-year-old palms
Cages 2, 7, 19 were erected in 1/70 around a healthy three-year-old palm, and four one-year-old palms
All other cages were erected in 1/69 (five one-year-old palms in each)

Cage removed, date indicated

○ Palm planted between 6/66 and 6/67. Date indicates when symptoms of lethal yellowing were observed

× Palm planted between 10/69 and 4/70

tom development varied from four and a half to nine and a half months, within and between sets (Figure 4). Control sets of palms not taken to the diseased area were placed adjacent to the sets brought back from the diseased area, and they did not become diseased. The apparent incubation time on young nonbearing palms could therefore be between one and a half and nine

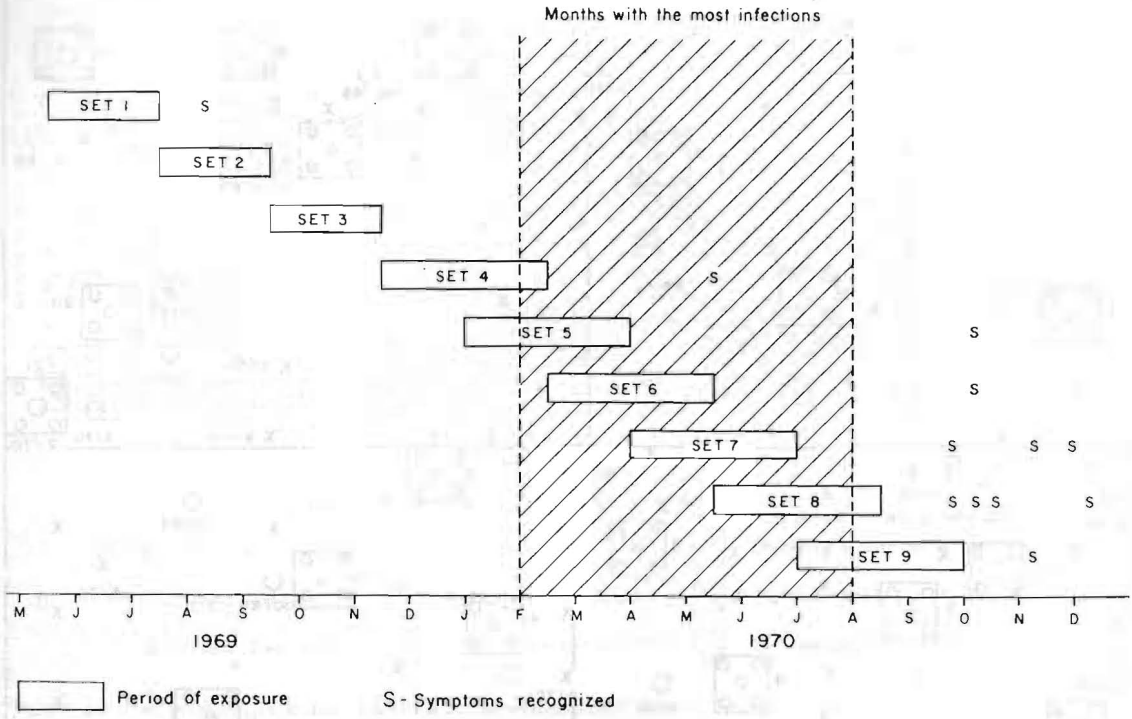

Figure 4. Period of exposure and time of symptom appearance in " transfer experiment."

and a half months. The most likely incubation period is three to six months, as a part of the exposure time occurred in that period in all cases of disease except one.

All but one of the cases, set 5, fit into this range (Figure 5). It would appear that the incubation period for sets 5, 6 and 7, exposed from January to July, was longer than for sets 1, 8 and 9 exposed from May to September (Figure 4). Sets 2 and 3 exposed from July to November did not become diseased. In spite of the fact that the percentage of infection was low in all sets, symptoms were expressed within the limited period August to December, irrespective of the months of exposure. As the experiment continues, the development of a definite pattern, i.e., February to August inclusive (Figure 4), might indicate the period of activity of the vector. If this period corresponds to a particular insect group found in traps, this group can be strongly suspected as the vector.

Experimental control studies

Although proof is lacking concerning the cause and method of transmission of lethal yellowing disease, its rapid spread in some parts of Jamaica warranted experimenting with control measures in areas which were free of the disease (26). Only when from one to four diseased palms are concentrated in a small area, remote from the main diseased area, such as the primary outbreak shown in Figure 6, is eradication of the source of infection likely to be effective. Healthy palms within a radius of approximately 100 metres of the outbreak were treated with malathion, chlordane and white oil using a mist blower (see Table 1). In later experiments the palms were injected with a systemic insecticide, usually dimethoate, which was introduced into a hole bored in the tree trunk to kill infective vectors on and around the diseased palms. Ten to fourteen days later,

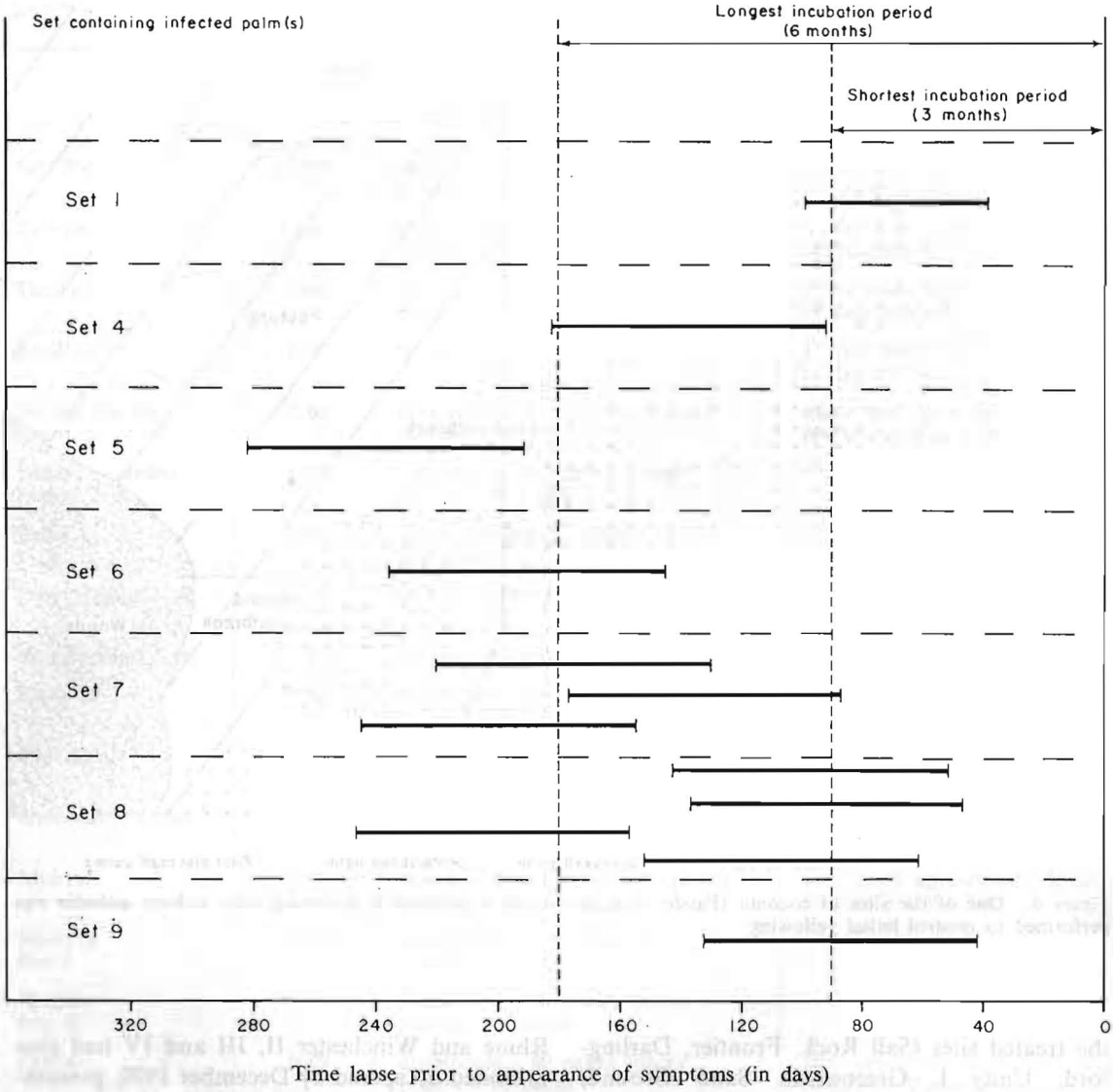

Figure 5. Parameters of incubation periods of lethal yellowing disease in individual palms after exposure in the "transfer experiment." Length of each bar shows duration of exposure.

the same diseased palms were poisoned by injecting sodium arsenite into the trunk, and palms were also treated within a 50-metre radius.

At Passley Garden (Figure 6), the first case was treated in February 1969 and the next two cases in August 1969 (although several poisoned palms failed to die). However, by 23 March 1970, 56 palms were newly infected. On 15 May, 80 diseased palms were scattered across the field, mostly in the vicinity of the first outbreak, but some were isolated and at a considerable distance from the outbreaks. Infection of some of the palms probably occurred from outside, and perhaps a few were infected before poisoning. The treatments appear to have had no effect on limiting or eradicating the disease.

The reappearance of the disease at nine of

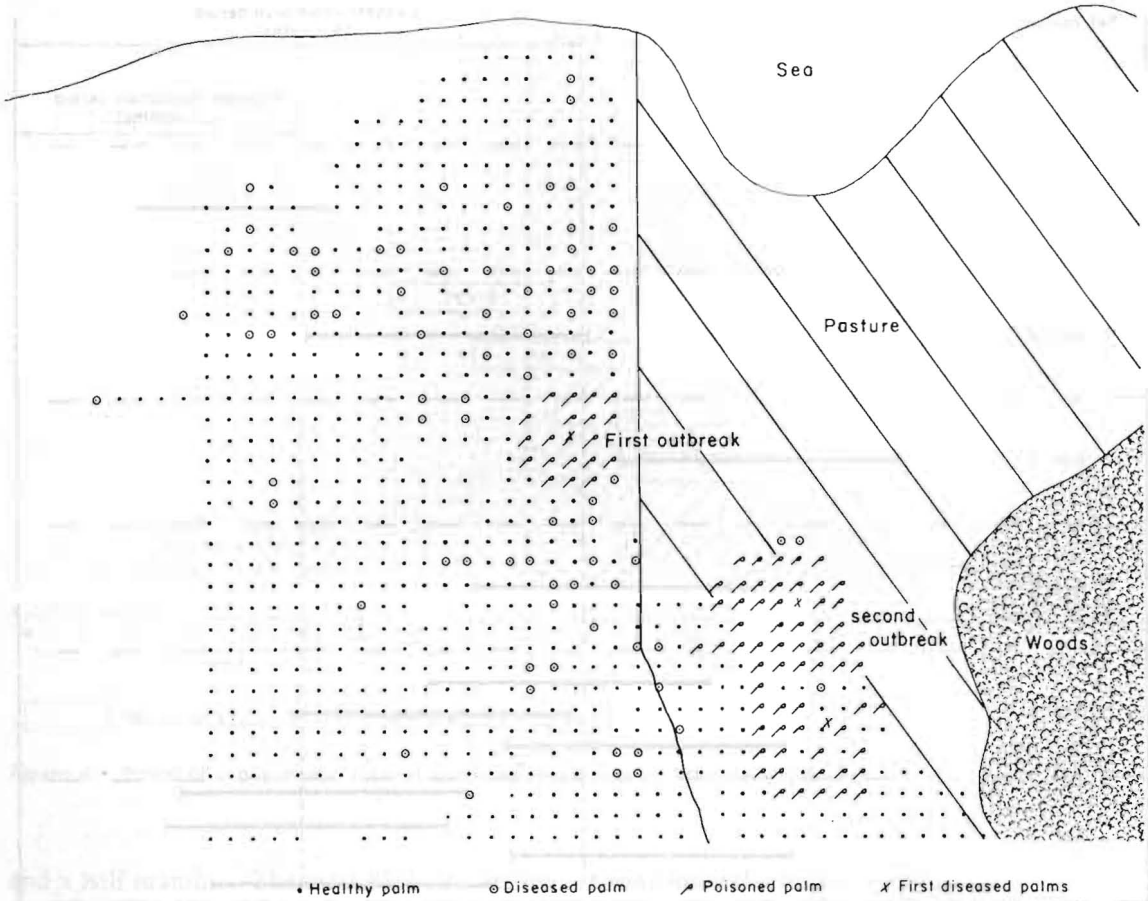

Figure 6. One of the sites of coconut (Passley Garden) where experimental poisoning with sodium arsenite was performed to control lethal yellowing.

the treated sites (Sail Rock, Frontier, Darlingford, Unity I, Greencastle, Sand Ground, Hordley, Winchester I and Church Corner, as shown in Table 1) occurred after periods varying from 7 to 15 months, and then only in a comparatively few trees. It is doubtful whether the low number of new cases can be attributed to the control treatments used. No new cases occurred at four untreated sites (Sandside, Bowden I, Harbour Head and Springfield) until 10 to 17 months later. The other four untreated single tree outbreaks at Suffolk, Duckenfield, Bowden II and West Rhine had no new cases 11 months after the first case appeared. The six treated sites at Holland, Arcadia, East Rhine and Winchester II, III and IV had also exhibited no spread by December 1970, presumably because the incubation period was not yet complete. All of these 23 outbreaks are considered therefore to differ from the Passley Garden outbreak and similar ones. Certainly, the ecological conditions at the eight untreated sites do not favour rapid spread of the disease. It was not possible at the time of writing to make a final judgement as to whether the treatments used have resulted in a degree of control, as not enough time had elapsed for possible new cases to occur around the treatment sites, but in general the treatments had no obvious effect. By January 1970, several treatment sites

TABLE 1. — TRIALS TO ERADICATE FIRST OUTBREAKS. TREATMENTS AND RESULTS TO DECEMBER 1970

Site	First disease	Treatment [1]	Results
	Month/Year		
Sail Rock	11/68	mist blower 0.8 hectares (twice) poison - 12 palms	4 new cases 10/69 (11 months later)
Frontier	12/68	mist blower 1.2 hectares (twice) poison - 27 palms	4 new cases 3/70 (15 months later)
Darlingford	1/69	mist blower 0.6 hectares (twice) poison - 18 palms	2 new cases 10/69 (9 months later)
Sandside	2/69	no treatment	12 new cases 7/70 (17 months later)
Passley Garden (palm 1)	2/69	mist blower 0.8 hectares (twice) poison - 18 palms	many new cases occurred every few weeks after 8/69
Passley Garden (palms 2, 3)	8/69	dimethoate - 30 palms (twice) poison - 77 palms	
Unity I	3/69	felled - 28 palms; crowns misted and burnt	2 new cases 6/70 (15 months later)
Derry (palms 1-4)	4/69	poison - 140 palms	subsequent cases appeared every few months
Derry (palms 5, 6)	7/69	poison - 48 palms	
Unity II	7/69	dimethoate - 50 palms (twice) poison - 35 palms	no new case by 10/70
Greencastle	4/69	poison - 23 palms (no insecticide)	no new case by 3/70; many cases 7/70 (7-11 months later)
Greencastle Coast	8/69	dimethoate - 25 palms (twice) poison - 28 palms	subsequent cases appeared every few months
Muirton	5/69	poison - 48 palms (no insecticide)	new cases appeared 11/69 and frequently thereafter
Muirton Pen I	6/69	dimethoate - 178 palms (twice) poison - 92 palms	
Muirton Pen II	9/69	dimethoate - 50 palms (twice) poison - 21 palms	
Quebec	9/69	dimethoate - 80 palms (once) poison - 34 palms	new cases appeared 3/70 and frequently thereafter
Sand Ground	9/69	dimethoate - 75 palms (twice) poison - 65 palms	4 new cases 7/70 (10 months later), 2 in 8/70 and 1 in 10/70
Holland	12/69	bidrin, azodrin or dimethoate - 200 palms (twice) poison - 91 palms	no new case by 12/70
Arcadia	12/69	dimethoate - 66 palms (twice) poison - 65 palms	no new case by 12/70
East Rhine	12/69	dimethoate - 61 palms (twice) poison - 39 palms	no new case by 12/70
Hordley	12/69	dimethoate - 107 palms (twice) poison - 53 palms	several new cases 11/70 (11 months later)

TABLE 1. — TRIALS TO ERADICATE FIRST OUTBREAKS. TREATMENTS AND RESULTS TO DECEMBER 1970 (*concluded*)

Site	First disease	Treatment[1]	Results
	Month/Year		
Winchester I-IV	12/69	4 separate single-palm outbreaks 275-365 metres apart. All outbreaks injected, but IV not poisoned	new case at Winchester I 12/70 (12 months later)
Church Corner	1/70	dimethoate - 32 palms (twice) poison - 68 palms	symptoms 12/70 (11 months later) in poisoned palms which did not die
Bowden I	1/70	no treatment	1 new case 12/70 (11 months later)
Harbour Head	1/70	no treatment	2 new cases 12/70 (11 months later)
Suffolk	1/70	no treatment	no new case by 12/70
Duckenfield	1/70	no treatment	no new case by 12/70
Bowden II	1/70	no treatment	no new case by 12/70
West Rhine	1/70	no treatment	no new case by 12/70
Springfield	2/70	no treatment	3 new cases 12/70 (10 months later)

[1] Treatments consisted of: Mist blower - 0.5% malathion, 0.5% chlordane and 2.5% white oil; Insecticide injection - azodrin (120 cc of 5% pellets); or bidrin (25 cc of 24% solution); or dimethoate (100 cc of 7.5 % solution). Poison injection - sodium arsenite (100 cc of 15% As_2O_3). Where treatment was carried out more than once, it was repeated at an interval of two to three weeks.

showed further cases of disease. In view of the cost involved, it was decided that no more experiments would be performed until the existing ones had time to show conclusive results.

Conclusions

Since mechanical transmission of lethal yellowing disease of coconut palm, including the use of buffers and an abrasive (22, 25), was not successful in Jamaica, one of the essential proofs for the viral nature of this disease is missing. It is not known which organism was transmitted from palm to palm by Roberts *et al.* in Florida (22, 25), but it does not seem to be the agent responsible for the development of the "Jamaica" lethal yellowing syndrome. The lethal yellowing disease pathogen is very active (4, 5), killing a tall palm in 7 to 15 months and a young one in 3 to 6 months. The climates of both Key West, Florida, and Jamaica are well adapted to coconut growing; any slight differences between the two territories are not likely to influence transmissibility, whether the causal agent is a nonpersistent, mechanically transmissible virus (2), or a semipersistent or persistent one which is not transmissible by mechanical inoculation methods, or a mycoplasma.

Other proof was therefore sought to confirm the viral nature of lethal yellowing disease. The mode of spread of the disease in the field, as demonstrated in Figures 1, 2 and 6, is quite typical of viral diseases, although this alone is not conclusive proof of a causal virus. However, the experiment with cages at Caenwood and the transfer experiment Kingstone-Fair Prospect (diseased area)- Kingston seem to leave no doubt that a disease requiring a transmitter is the cause of lethal yellowing. Nematodes would seem to be excluded as a cause of lethal yellowing (5, 17). The present study supports this con-

clusion, since the transferred palms were in drums and many of the palms under cages at Caenwood were not protected against soil nematodes. The cages were able to protect palms against transmitter of lethal yellowing, but the unprotected palms of the plot were infected. Cage removal was followed by infection of some of the previously protected palms. The only disease agents with a specialized vector are viruses. (Some exceptions are a few typical fungal and bacterial diseases which produce unmistakable symptoms and well recognized stages of their life cycle in or on the plants. In many such cases, the insects operate only as an additional transmitter of the airborne spores.)

A further proof of the viral nature of the disease is the incubation period after infection. There are indications that the incubation period in a young coconut palm ranges between 3 and 6 months, and in an old and bearing palm not less than 7 to 15 months. These long incubation periods are typical of viral infections, but normally not of infections by bacteria or fungi.

It was not possible to prevent the spread of lethal yellowing disease by insecticidal sprays or injections, together with poisoning or felling, against the supposed vector. Failure may have been due partly to difficulties in the application of insecticides; sprays by mist blower do not reach all of the leaves, especially in very tall trees. It has even been found that a systemic insecticide applied to the upper side of a leaflet does not pass through to the lower surface. The amount of insecticide injected into tall palms must be extremely high to have an effect on the insect population, and this tends to be an uneconomic practice.

Summary

Research studies on the cause and mode of transmission of lethal yellowing disease of coconut in Jamaica demonstrated that the vector is airborne. Insect-proof cages protected palms from the disease: natural infection occurred only after cage removal. The time interval between cage removal and first symptoms, together with early data from an experiment in which coconut plants in drums were transferred to the diseased area for a definite period and then returned to a healthy area for observation, indicated an incubation period of approximately three and six months. Experiments on control of new natural outbreaks of disease in mature palms appeared to be unsuccessful, but they indicated an incubation period of not less than 7 to 15 months.

LITERATURE CITED

1. ANONYMOUS. Progress in lethal yellowing research. 1969 *Farmer*, 74(1): 45-49.
2. BHARGAVA, K.S. & KHURANA, S.M.P. Papaya 1969 mosaic control by oil sprays. *Phytopath. Z.*, 64(4): 338-343.
3. BOR, N.A. Coronie wilt, a coconut disease of 1970 unknown etiology in Surinam. *Caribbean Plant Prot. Commiss., Q. Rep. FAO, Port of Spain, Trinidad and Tobago*, 3:6.
4. CARTER, W., LATTA, R.K. & SUAH, J.R.R. The 1965 symptoms of lethal yellowing disease of coconut palms. *FAO Plant Prot. Bull.*, 13(3): 49-55.
5. CARTER, W. & SUAH, J.R.R. Studies on the 1964 spread of lethal yellowing disease of the coconut palm. *FAO Plant Prot. Bull.*, 12(4): 73-78.
6. CASPAR, R. Mykoplasmen als Erreger von Pflan- 1969 zenkrankheiten. *NachrBl. dt. PflSchutzdienst, Braunschweig*, 21: 177-182.
7. CIFERRI, R. & CICCARONE, A. Observaciones 1949 sobre la enfermedad de la "hoja bronceada" del cocotero en Venezuela. *Rev. Fac. nac. Agron., Medellín*, 9: 17-26.
8. DE LA TORRE, C. La enfermedad de los cocoteros. 1906 *Revta de Letras y Ciencias, Univ. la Habana*, 2(3): 269-281.
9. DRING, B.M. *Rapport au gouvernement du Togo sur* 1963 *la maladie de kaincopé du cocotier*. Rome, FAO. 29 p. Rapport FAO/PEAT N° 1610.
10. FAWCETT, W. *Report on the coconut disease at* 1891 *Montego Bay*. Kingston, Jamaica, Botanical Department. Bulletin No. 23.
11. GRYLLS, N.E. *Report to the Government of Jamaica* 1959 *on lethal yellowing disease of coconut*. Rome, FAO. 29 p. FAO/EPTA Report No. 2659.
12. GRYLLS, N.E. & BOR, N.A. *Investigations on the* 1968 *aetiology of lethal yellowing disease of coconuts in Jamaica. 2. The role of insects as possible vectors*. Document, Third Session of the FAO Technical Working

Party on Coconut Production, Protection and Processing, Jogjakarta, Indonesia, 9-19 September 1968. 11 p.

13. GRYLLS, N.E., HUNT, P. & BOR, N.A. 1968 *An approach to the study of the aetiology of coconut lethal yellowing disease.* Paper presented at the first International Congress of Plant Pathology, London, July 1968.
14. HARRIES, H.C. 1967 *Seventh report, July 1966-June 1967*, Research Department, Coconut Industry Board, Jamaica, W.I., p. 11.
15. HEINZE, K. [& SCHUILING, M.] 1970 Investigations on etiology and control of lethal yellowing disease. *Ninth report, July 1968-June 1969*, Research Department, Coconut Industry Board, Jamaica, W.I., p. 71-74.
16. HOYLE, I.C. 1969 Suspected outbreak of bronze leaf wilt in coconuts in Trinidad. *J. agric. Soc. Trin.*, 69(1): 539-562.
17. LATTA, R. 1966 Attempts to relate nematodes to lethal yellowing disease of coconut palms in Jamaica. *Trop. Agric., Trin.*, 43(1): 59-68.
18. LEACH, R. 1946 The unknown disease of the coconut palm in Jamaica. *Trop. Agric., Trin.*, 23(3): 50-60.
19. LEATHER, R.J. 1959 Further investigations into the Cape St. Paul wilt of coconuts at Keta, Ghana. *Empire J. exp. Agric.*, 27: 67-78.
20. MARTINEZ, A.P. 1965 Lethal yellowing of coconut in Florida. *FAO Plant Prot. Bull.*, 13(2): 25-29.
21. MIJAILOVA, P.T. 1967 Informe sobre la investigación de la enfermedad "Pudrición dei cogoilo del cocotero." *Revta Agric., Habana*, 1(1): 74-110.
22. MILLER, M.E. & ROBERTS, D.A. 1970 Mechanical transmission of the coconut palm lethal-yellowing pathogen from frozen or fresh inocula prepared in two buffers. *Phytopathology*, 60(9): 1304.
23. OHLER, J.G. 1969 Broken leaf, a new coconut disease in the Caribbean area. *J. agric. Soc. Trin.*, 69(1): 569-590.
24. OLLAGNIER, M. & WESTSTEIJN, G. 1963 Coconut diseases in the islands of the Caribbean: comparison with the kaincopé disease in Togo. *Indian Cocon. J.*, 16(1): 23-35, 1962 (received 1963).
25. PRICE, W.C., MARTINEZ, A.P. & ROBERTS, D.A. 1967 Mechanical transmission of coconut lethal yellowing. *FAO Plant Prot. Bull.*, 15(6): 105-108.
26. ROMNEY, D.H. 1970 Attempts to control lethal yellowing. *Ninth report, July 1968-June 1969*, Research Department, Coconut Industry Board, Jamaica W.I., p. 75-76.
27. RUSSELL, L.M. 1965 A new species of *Aleurodicus* Douglas and two close relatives (Homoptera: Aleyrodidae). *Fla Ent.*, 48: 47-55.
28. SCHIEBER, E. & HICHEZ FRIAS, E. 1970 Lethal yellowing disease of coconut palms in the Dominican Republic. *Phytopathology*, 60 (11): 1542.
29. SEAL, J.L. 1928 *Coconut bud rot in Florida.* Gainesville, Florida Agricultural Experiment Station. Bulletin No. 199. 27 p.
30. SHIKATA, E., MARAMOROSCH, K. & LING, K.C. 1969 Presumptive mycoplasma etiology of yellows diseases. *FAO Plant Prot. Bull.*, 17(6): 121-128.
31. WESTWOOD, D. 1953 *Coconut diseases, Keta, Ho.* Gold Coast, Department of Agriculture. 25 p.