2nd Sess. FAO Wks. Pkg. Coco. Prod. Prot. & Process. Colombo, 1964.

WORKING PAPER NO. XXIV

THE CADANG-CADANG OF COCONUTS IN THE PHILIPPINES AND THE COCONUT WILT IN INDIA

by

A. N. NAGARAJ
F.A.O. Virologist, Guinobatan Experiment Station
Guinobatan, Albay

I. THE CADANG-CADANG DISEASE:

Introduction:

In the Philippines, the cadang-cadang disease of coconut has destroyed a large number of plantations in the Bicol region on Luzon island and in a few surrounding islands. It was first noticed in 1926 on San Miguel island, which is an island adjacent to the Bicol region in which the disease was absent at that time (27). By 1947 the disease was widely prevalent in the Bicol region (7). It was reported from Quezon province in 1957 (22); and from Laguna province in 1964 (5).

On the island of San Miguel the disease had destroyed 80% of the initial 260,000 trees by 1953 (11), and at the present time there are only a few hundred trees left (2). In the Bicol region also, within a span of 25-30 years, more than 12 million trees have been affected, causing an annual loss amounting to more than 50 to 60 million pesos (24).

The disease, which was considered to be one of the most destructive plant diseases by Price (30) may be expected to cause much greater losses now that it has invaded two major coconut growing provinces of the country namely Quezon and Laguna.

Symptomatology:

The field symptoms of the disease consist of yellowing and mottling of outer leaves and stunting of younger leaves; on closer examination such leaves exhibit a large number of yellow and 'water-soaked' or olivaceous spots... The nuts of affected palms are smaller in size, lose their angular shape and become rounded, and are scarified with necrotic streaks and patches. In early stages of disease there are

a larger number of nuts, but in later stages fewer nuts are produced. The older leaves are shed faster than in healthy trees and in advanced stages of disease only a few young leaves are left. Ultimately the growing point dies in about 5-10 years after the appearance of first symptoms. (6, 7, 11, 27, 30, 33).

The symptomatology of the disease is complicated by the fact that there are various kinds of yellow and 'water-soaked' spots on both healthy and cadang-cadang affected palms, and that many of the other field symptoms are individually present in cadang-cadang-free areas. There is, therefore, a difference of opinion among various workers as to the nature of leaf-spots specific to cadang-cadang. Calica and Bigornia (6) suggested that none of the 6 symptoms they described was diagnostic by itself, but that the whole symptom syndrome was essential for diagnosis. Others have maintained that it is not possible to accurately diagnose the disease in the field (19) and that there is a lack of agreement as to the typical symptoms of the disease (39, 43).

Recognizing the need for clarifying this situation, Sill and Protacio (34) conducted the first quantitative study of the frequency of occurrence of various crown symptoms. This study was followed by that of Nagaraj and Pacumbaba (25), who after a detailed examination of 730 trees from cadang-cadang-free and affected areas concluded that a particalar kind of leaf-spot was specific to cadang-cadang. These spots were described by them as irregular, olivaceous or water-soaked in appearance, standing out prominently from the adjacent lamina because of their color and distinct margins. The basic spots were approximately 1/8-1/2 mm in diameter, and did not possess brown centers; these spots together with tiny yellow spots were aggregated in most affected palms to form streaks parallel to the veins. In bearing palms rounding of nuts (which were scarified in most cases with necrotic streaks and patches) was an additional and valuable diagnostic symptom. All other symptoms studied by them were found in healthy areas also and therefore were of no diagnostic value.

Cause of Disease:

Analyses of soils from affected and non-affected areas (21, 29-43, 45), and sand culture experiments on the effect of various nutritional deficiencies and aluminum toxicity (23, 44) failed to provide evidence to indicate that the disease was caused by a nutritional deficiency or toxicity. Application of N, P, K, and various micronutrients to the soil around diseased trees (4, 36) was not effective in curing the disease. In fact the fertilized diseased trees deteriorated faster than unfertilized controls (36). Spraying of diseased palms with various micronutrients also failed to reduce the intensity of disease (31).

Isolations of soil microorganisms from cadang-cadang affected and unaffected areas and from roots and leaves of affected palms (5, 15, 29, 38) and inoculations of coconut seedlings with such isolates (5, 29) failed to implicate a fungus or bacterium as the cause of cadang-cadang.

Studies on nematode populations in soils, and roots of coconut palms in healthy and affected areas (37, 46), indicated that nematodes are probably not involved as causal agents.

Bigornia et al, (3) studied the spread of the disease in 7 epidemiological plots and in strip surveys over a period of 8 years, they found that the spread of the disease was slow and erratic although there was a certain degree of adjacency of new infections. Such a spread is typical of a virus disease transmitted by a sluggish insect vector, and does not resemble that of a root disease. This well-conceived and elaborate study is a valuable contribution in favour of the virus theory.

Many workers consider that the absence of association of pathogenic fungi or bacteria with diseased tissues, the distribution and spread of disease, absence of response to application of macro and micronutrients, and the symptomatology indicate that cadang-cadang is a virus disease (20, 22, 27, 30).

Celino (8) reported transmitting the disease by pin-prick inoculation to four-year-old seedlings in which symptoms appeared within 30-220 days. De Leon (10) and Kent (19) pointed out that the symptoms obtained by Celino on inoculated seedlings were not similar to the field symptoms, that no serial transfers were made, and that inoculated seedlings did not continue to exhibit disease symptoms. Celino himself (9) suggested that these results were inconclusive and has again claimed transmission of field symptoms of cadang-cadang in recent trials to coconut seedlings by pin-prick inoculation of leaf extracts adjusted to pH 7.3-7.4. The incubation period in this case was 8-20 months as against 1-11 months reported in his first paper (8). Del Rosario and Quiaoit (13) also claimed transmitting the disease to coconut seedlings within 40-120 days after inoculation, and to corn seedlings in which disease symptoms appeared within 3-20 days. It was pointed out by Celino that the disease transmitted by himself and co-workers in recent years was different from that transmitted by Del Rosario and Quiaoit in symptomatology, incubation period, etc. It also appears to be different from the disease Celino transmitted in 1947. An examination of Celino's test plants by the author showed that only two of them showed cadang-cadang specific spots as described by Nagaraj and Pacumbaba (25).

Calica and Pableo (5) on the other hand were unable to transmit the disease by pin-prick inoculations, rubbing of tissue extracts with carborundum, petiole and root grafting, insertion of diseased tissues, etc. They observed 1012 coconut seedlings thus inoculated and an equal number of controls for more than 10 years, but secured no transmission.

These results, as well as differences in symptomatology, incubation period etc. between three diseases transmitted by Celino (8), Celino and co-workers (9), and Del Rosario and Quiaoit (13) suggested that the etiology of cadang-cadang still remains to be solved.

Electron micrographs of tissue extracts from affected palms were obtained by Ocfemia and Bustrillos (28), and Del Rosario and Quiaoit (13). The published micrographs show particles which were considered to be virus-like but which are not all of the same shape or size.

By injecting clarified leaf extracts, an antiserum was obtained by Del Rosario and Quiaoit (13) which reacted with tissue extracts from diseased palms in most cases. Del Rosario (12) has suggested that the technique needs further improvement. Obtaining such an antiserum indicates the presence of an antigen in diseased trees which is absent in healthy trees. This antigen may be bacterial, viral or an abnormal metabolite.

Anatomical Studies:

Anatomical studies of the typical cadang-cadang leaf spots by Calica and McWhorter (22) showed that such spots were caused by a process called white cell necrosis which converted the protoplasts of chlorenchyma into homogeneous white masses. McWhorter (22) considers that such necrosis is typical of virus diseases and, therefore, is a strong evidence indicating that cadang-cadang is due to a virus. Hypertrophy of cells of phloem and xylem which caused obliteration and necrosis of adjacent cells, and hyperplasia in phloem were noticed by Erriquez (14) in leaves of affected palms. Quantitative studies are needed to confirm these interesting findings.

Protacio (32) reported the presence of intracellular inclusion bodies in epidermal cells of infected leaves; he examined 5 trees from cadang-cadang-free areas, and 28 diseased trees and 5 apparently healthy trees from cadang-cadang-affected areas. This offers to be a promising additional diagnostic test if these results can be confirmed by examination of a larger number of samples from disease-free areas.

Natural Alternate Hosts:

Palms: In nature, diseases similar to cadang-cadang have been reported on several other palms, such as Areca catechu, Adonidia merrilli, and Livistona rotundifolia (10), Corypha elata, Caryota Cumingii, Elaeis guineensis (11) and Pandanus coplandii (22).

Weeds: The epidemiological data of Bigornia et al (3) were carefully analyzed by Holmes (16) who suggested that the more or less constant percentage of new infections obtained year after year indicated that the disease does not spread from diseased coconut to healthy coconut. He hypothesized that the disease is spread to coconuts from a fixed reservoir outside of coconuts,

possibly a weed. In later studies (17, 18) he correlated the incidence of a disease in Elephantopus mollis, characterized by yellowing and sometimes purpling and reddening of leaf margins, to that of cadang-cadang. Such positive correlations were obtained in 19 out of 20 observations and in the exceptional case where there was a high incidence of cadang-cadang he found that there was a large amount of disease in two other weeds--Stachytarpheta jamaicencis and Passiflora foetida which, therefore, were considered to be either supplementary reservoirs of disease or supplementary indicators of disease incidence. He also found that in well-weeded kitchen gardens there was considerably less cadang-cadang than in adjacent plots which had a large number of diseased E. mollis plants

Bigornia (1), who was acquainted with the disease of E. mollis correlated by Holmes with that of cadang-cadang, found that yellowing of leaf margins could be produced by the feeding of the leafhopper Empoasca formosana. These plants under conditions of inadequate water supply and intense sunlight also developed reddening of leaf margins described by Holmes (17). Also, symptoms induced by leafhopper feeding, hopper-burn, (1) were not transmissible by grafting and did not appear in the new growth appearing after removal of leafhoppers. He concluded that hopper-burn was not likely to be linked with the occurrence of cadang-cadang, but conceded that plants having such symptoms could possibly harbor another disease which may be related to cadang-cadang.

Nagaraj and Pacumbaba grafted healthy E. mollis plants with scions from E. mollis affected with hopper-burn symptoms plus purpling and reddening of leaf margins; those infected with yellows, a graft transmissible disease (5) characterized by stunting of plants, chlorosis, and smalling of leaves; and from plants showing both hopper-burn and yellows. They confirmed that yellows is graft-transmissible and that hopper-burn (plus reddening of leaf margins) is not. In certain areas hopper-burn and yellows existed together in the same plants and in other areas only hopper-burn or yellows was found. The former was noticed in large numbers in Davao, a cadang-cadang-free area. Yellows also was noticed in Los Baños, where cadang-cadang was absent. The results of a recent survey indicated that in palms 30 years and older, the percentage of disease incidence was essentially similar in weeded and unweeded gardens. In three of these gardens there was a history of consistent weeding for a period of at least 8 years; yet, in all of them several trees were found to be in very early stages of disease. These findings and those of Bigornia (1) indicate that Holmes (17, 18) was probably not correlating either hopper-burn or yellows of E. mollis with cadang-cadang. The curves for disease incidence which were presented by Bigornia, et al (3) and which were interpreted by Holmes as indicating that cadang-cadang did not spread from coconut to coconut may also be interpreted to mean that diseased coconut trees become ineffective sources of inoculum with the progress of disease possibly because the hypothetical vector may prefer not to feed on such trees. This may be either because of the physiological changes in the tissues, lack of succulence or because of

reduced number of flowers which would adversely affect a vector that is a floral insect. Under these conditions, with increasing new infections the ones infected earlier become less efficient sources of inoculum thereby keeping such sources more or less constant.

Control Measures:

Quarantine regulations: Even though the disease has been reported from Quezon and Laguna, the leafspotting in Laguna appears to be slightly different from the specific leaf-spotting found in Bicol. This raises the question whether the disease in Laguna is caused by a different strain of cadang-cadang. Until this matter is clarified, quarantine regulations prohibiting movement of coconut plant parts out of the Bicol region should not be relaxed. Also, there is a possibility that the disease in Laguna and Quezon can be contained or eliminated by strict enforcement of eradication measures.

Eradication measures: Recent work by Nillos and Silva (26) has shown that spraying, cutting and burning of infected trees, and suspected alternate hosts, can greatly reduce the spread of disease. On the basis of a survey conducted by a team headed by Bigornia, it was estimated that there are probably only 6,000 diseased trees in Quezon, Laguna, and Batangas (24). It has been suggested that these trees, weeds around them, and any other possible alternate hosts, should be sprayed with an insecticide, cut and burnt. If such a program is consistently practiced the spread of disease may be checked. Meanwhile, further studies should be conducted in isolated plots in the Bicol region to accurately assess the value of such eradication measures, (24).

Replanting: Although replanting of devastated areas has been suggested by many scientists, it has acquired greater significance with the findings of Sill et al (35) who found that the incidence of disease was much higher in older trees than in younger ones. Even in the 21-30 year group there was less than 25% disease. In view of these findings they suggested that regular replanting of affected areas should be undertaken.

Nagaraj and Bigornia (24) suggested that a ten year program be instituted in which a total of 20 million trees should be replanted in the Bicol region to replace trees that have already become diseased, dead or too old, and those that can be expected to be attacked by the disease. When these trees start bearing within the next 20 years they should save a minimum of 50 million pesos annually. They also recommended that in the rest of the Philippines particularly in Laguna and Quezon where the disease has been recently discovered, replanting should be undertaken to replace all trees above 40 years of ago which are likely to be first attached by the disease. They estimated that 128 million trees will have to be replanted in the whole of the Philippines and advocated that seed nuts for such replanting should be obtained from mother palms selected throughout the country for high yield and other desirable characters. If replanting is spread out over a period of 10 years and a selected tree yields 80 nuts a year it will be necessary to select 160,000 mother trees. The total cost of such selection for ten years was estimated to be approximately 2 million pesos, (24).

Breeding for disease resistance: Such a breeding program is extremely desirable, but time consuming and is now in progress at the Guinobatan Experiment Station. (2).

Research activities: Research work should be intensified to determine the nature of the disease, mode of spread, alternate hosts, and the efficacy of various control measures such as roguing, spraying, weeding and other cultural practices.

The cost of a well financed 5-year program of research, selection of mother palms for obtaining better seed nuts, replanting throughout the country as mentioned earlier was estimated to be 5.6 million pesos. During this 5-year period the disease would have caused a loss of about 250 million pesos in the Bicol region alone (24). If one adds to those the expected losses in newly infected areas in Quezon and Laguna, the suggested expenditure would be well worth while. If the etiology of the disease is not solved and the disease spreads to the island of Mindanao and other coconut growing areas, the losses for the premier dollar earning industry of this country would indeed be great.

II. THE WILT DISEASE:

Introduction:

The disease was first noticed about 80 years ago on the south west coast of India and appears to be confined to this region until now. It was considered to be primarily a root rot (49), but later work showed that root decay was probably a secondary effect of disease (52). Therefore, the name wilt disease is more appropriate for the disease. In 1951, the estimated annual losses caused by the disease (49) were about 10 million rupees (approximately 8 million Philippine pesos). Accurate and more recent estimates of losses are needed.

Symptomatology:

The symptoms of the disease consist of a paling and wilting of the youngest leaves sometimes accompanied by a necrosis. The wilting gradually spreads to the outer leaves, which in some cases become yellowed. The leaflets become characteristically flaccid and are curled downwards and inwards giving a ribbed appearance (49,55). Such necrosis, flaccidity and ribbing of leaflets is absent in cadang-cadang affected palms. An examination of a large number of leaf samples from wilt diseased trees did not show any leaf-spots similar to the cadang-cadang specific spots (54).

Shedding of immature nuts was considered an early symptom of disease (49), but in trees to which the disease was reproduced by mechanical inoculation (53) this symptom was rarely observed (51). The nuts of infected palms do not consistently show either rounding or scarification

so typical of cadang-cadang. In both diseases the nuts become smaller, lesser in number, have thinner husks, but unlike in cadang-cadang, nuts of wilted palms have kernels that produce rubbery copra (49).

Root rotting was also considered to be an important symptom (49). Nagaraj and Menon (52) conducted a quantitative study of the association of root-rotting with symptoms of wilt and three other diseases. They found that in early stages of wilt the percentage of root decay was essentially similar to that in healthy trees and that it gradually increased in middle and advanced stages of disease; it was also common in trees showing general chlorosis, tapering stem symptoms and yellowing of outer leaves. They concluded that root decay was probably not the cause of wilt and suggested that the name wilt disease was more appropriate for the disease.

The progress of wilt disease appears to be slower than in cadang-cadang. Some of the trees in which the disease was produced artificially in 1954, were in advanced stages of disease in 1964, but still had a few nuts (51).

After having been associated with both the diseases the author feels that the wilt disease is milder than cadang-cadang. The spread of wilt is also probably slower but unfortunately epidemiological studies similar to those of Bigornia et al. (47), have not been conducted for wilt.

Cause of Disease :

Earlier work was concentrated on isolating organisms from diseased roots as the disease was believed to be caused by root decay. In 1908 Butler isolated Botryodiplodia sp. and thought that root-rotting caused by this organism could account for the crown symptoms. Later, Menon and Nair consistently isolated 3 fungi from roots of infected palms; these were Rhizoctonia solani and R. bataticola, and Botryodiplodia theobromae. They reported producing wilting in 9-18 month old coconut seedlings by inoculation with these fungi particularly if the seedlings were water-logged at the time of inoculation; the inoculated fungi were reisolated in all cases of infection. However, they found when they inoculated roots of 4-5 year old palms root-rotting was localized and no disease symptoms developed on the leaves of inoculated plants. They were also unable to obtain infection by pin-prick inoculation and by transference of soil and roots from affected palms to the base of healthy trees (49).

No qualitative differences were noticed in the rhizosphere flora of diseased and healthy palms, and quantitative differences were not consistent in different soil types (61). Of the 3 fungi associated with the disease (49) R. solani was obtained only from diseased roots whereas the others could be obtained from healthy roots as well. Inoculations of roots of healthy palms with 7 isolates of these 3 fungi were conducted by Nagaraj and Menon (52); they obtained infection of young living roots only with one isolate of R. solani but not with the other fungi; even in this case only the root tip was affected and infection did not progress beyond 4 inches from the tip.

Results of detailed chemical analyses of diseased and healthy tissues, and soils from affected and unaffected areas (50, 57, 59, 61, 65, 70, 71) indicated that soils of diseased areas had lower available and exchangeable potassium. In the other nutrients studied the differences between healthy and diseased areas were not consistent in different types of soils examined. The infected trees showed an accumulation of nitrogen, phosphorus, and potassium, (62, 70). Application of N, P and K together with spraying of infected palms with micronutrients improved the condition of diseased palms (56).

Nagaraj and Menon (53) reported transmitting the disease by mechanical inoculation in 5 experiments conducted in the field, involving 31 trees of which 17 got infected, whereas, among the 30 controls there were only 3 infections. The results of transmission trials with five species of insects, indicated that only Stephanitis typicus, transmitted the disease in 2 experiments involving 19 trees of which 11 got infected; and among 18 controls 1 tree developed disease symptoms. The incubation period in both mechanical inoculation and insect transmission trials was 8-12 months from the date of first inoculation. These results indicated that the wilt disease of coconuts is infectious, probably a virus disease transmissible by mechanical inoculation, and that Stephanitis typicus is probably a vector of the disease. Definite conclusions as to the nature of the infectious principle could not be drawn because in these preliminary experiments the inoculum was not passed through bacterial-proof filters.

The results on mechanical and insect transmission of the disease were confirmed by Shanta et al. (68) by using a larger number of test plants. They also reported transmitting the disease to 2-3 year old coconut seedlings, whereas Nagaraj (54) was unable to do so in his earlier work, even after 3 years of repeated inoculations.

Shanta and Menon (66, 67) reported transmitting the disease to 5 varieties of cowpea, Vigna sinensis, obtaining more than 70% transmission both by mechanical inoculation and by Stephanitis typicus. Symptoms were observed within 4-12 days after inoculation and consisted of vein-clearing, malformation and curling of leaves. The disease was also transmitted to Phaseolus mungo, Capsicum annuum, and Areca catechu. All these hosts had been repeatedly inoculated by Nagaraj and Menon (53) but the inoculated plants did not develop any disease symptoms (51). The reason for these discrepancies is unknown especially in view of the fact that methods of inoculation used in these cases appear to be quite similar.

Control Measures:

Manurial and cultural treatments have been recommended (48, 56) to minimize the damage from the disease. The fact that the disese was transmitted by mechanical and insect inoculations whereas the controls under similar conditions of soil remained healthy, clearly indicates that an infectious principle is the cause of disease and that control measures should, therefore, be based on these data.

Because the disease is infectious, quarantine regulations to prevent the spread of disease to non-infected areas, eradication of diseased coconuts and alternate hosts in newly infected areas, and breeding for disease resistance are some of the possible suggested control measures.

Quantitative studies similar to that of Sill et al (69) should be conducted to determine the incidence of disease in various age groups. The results of such a study can be used to determine whether replanting of diseased areas would be profitable or not.

Since the disease is mechanically transmissible it should be possible to test a large number of varieties for disease resistance and recommend a resistant variety for replanting in diseased areas. In diseased areas a large number of palms above 50 years of age appear to remain healthy. Shanta, et al (68) found that 40-50 year old palms were apparently resistant to mechanical inoculation. These results indicate that such healthy palms in heavily diseased areas are probably resistant to the disease. It should be easy to confirm whether these are symptomless carriers or are actually resistant to wilt, by back inoculations on younger susceptible palms. It is also possible that age confers resistance to these palms but that their progenies may become susceptible to the disease when they are around 7 years old. Only after studying these problems would it be possible to decide whether such palms could be used as parents in a breeding program to obtain a variety resistant to the wilt disease.

ACKNOWLEDGEMENTS

My thanks are due to the Director of the Bureau of Plant Industry for permission to quote unpublished data and to Messrs. A.E. Bigornia and C.A. Calica for kindly providing information on their unpublished work. The author had useful discussions with Dr. S.B. Lal and with Dr. K. Radha.

BIBLIOGRAPHY

I. Cadang-cadang disease

1. Bigornia, A. E. (1963). Texomic symptoms on some weeds caused by the leafhopper Empoasca formosana in the Philippines. F.A.O. Plant Protection Bull. 2 (5) :

2. Bigornia, A. E. (1964). Personal communication.

3. Bigornia, A. E., Calica, C. A. and Pableo, G. O. (1960). Epidemiological studies on the coconut cadang-cadang disease. Department of Agriculture and Natural Resources, Bureau of Plant Industry, Manila. 11 pp.

4. Briones, A. Q. (1963). Nutrient deficiencies as a possible cause of cadang-cadang. Proceedings of the symposium on cadang-cadang of coconut :40-44.

5. Calica, C. A. (1964). Personal communication.

6. Calica, C. A. and Bigornia, A. E. (1960). The symptomatology of the yellow mottle decline. Department of Agriculture and Natural Resources, Bureau of Plant Industry :17 pp.

7. Celino, M. S. (1947a). A preliminary report on a blight disease (cadang-cadang) of coconut in San Miguel Estate, Albay province. Philippine J. Agric. 13 (2-4): 31-35.

8. Celino, M. S. (1947b). Progress report on experimental transmission of cadang-cadang disease of coconut. Philippine J. Agric. 13 (2-4): 109-111.

9. Celino, M.S. (1963). Cadang-cadang disease, a virus malady on coconut in the Philippines. Proceedings of the symposium on cadang-cadang of coconut :115-133.

10. De Leon, D. (1951). Report on studies of a disease (yellow mottle or infectious Kadang-kadang) of the coconut tree in the Philippines. U.S. Technical Cooperation Mission: 19 pp. mimeographed.

11. De Leon, D. and Bigornia, A. E. (1953). Coconut Kadang-kadang disease in the Philippines and experimental control program. U.S. Mutual Security Agency, Special Technical and Economic Mission, Philippines :17 pp. mimeographed.

12. Del Rosario, M. S. (1963). Serological detection of cadang-cadang infection. Proceedings of the symposium on cadang-cadang of coconut :134-138.

13. Del Rosario, M.S. and Quiaoit, A. R. (1962). Studies on the virus aspect of cadang-cadang disease of coconut. I. Transmission and serology. The Philippine Agriculturist 45 (9):-477-469.

14. Emriquez, C. M. (1962). Anatomical studies of healthy and cadang-cadang infected leaves of coconut palms. Third Prog. Report. Univ. Philippines College Agric. Cadang-cadang Research Foundation, Inc. Manila :3-5.

15. Garcia, M. P. and Valera, C. L. (1963). Fungi in coconut soils. The Philippine Agriculturist 46 (8): 634-645.

16. Holmes, F.O. (1961a). Does cadang-cadang disease spread from diseased to healthy coconut trees? F.A.O. Plant Protection Bull. 9 (8) :134-143.

17. Holmes, F. O. (1961b). Correlation of cadang-cadang disease in coconut and certain diseases in weeds. F.A.O. Plant Protection Bull. 9 (8) :148-152.

18. Holmes, F. O. (1962). Cadang-cadang disease of coconut.. Report to the government of the Philippines. F.A.O. Report (1574) : 31 pp.

19. Kent, G. C. (1953). Cadang-cadang of coconut. The Philippine Agriculturist 37 (5-6) :228-240.

20. Maramorosh, K. (1961). Cadang-cadang disease of coconut. Report to the government of the Philippines. F.A.O. Report (1333) : 26 pp.

21. Marferi, R. T. (1963). The chemical analysis of some soil and coconut leaf samples in relation to the cadang-cadang disease of coconut. Proceedings of the symposium on cadang-cadang of coconut: 85-89.

22. McWherter, F. P. (1959). Cadang-cadang of coconut. Report to the Government of the Philippines. F.A.O. Report (1107) : 14 pp.

23. Mercado, B. T. and Velasco, J. R. (1961). Progress Report: Effect of aluminum on the growth of coconut and other plants. The Philippine Agriculturist 45 (5) :268-274.

24. Nagaraj, A. N. and Bigornia, A. E. (1964). A discussion of possible measures to control the spread of cadang-cadang. Paper read at the cadang-cadang symposium in May 1964. 6 pp. mimeographed.

25. Nagaraj, A. N. and Pacumbaba, R. P. (1964). Specific diagnostic symptoms of cadang-cadang disease. To be published.

26. Nilles, P. N. and Silva, A. B. (1964). Unpublished data.

27. Ocfemia, G. O. (1937). The probable nature of "Cadang-cadang" disease of coconut. The Philippine Agriculturist 26 (4) : 338-340.

28. Ocfemia, G. O. and Bustrilles, A. D. (1957). Electron microscopy of the unpurified extracts of coconut infected with the cadang-cadang disease. The Philippine Agriculturist 40 (10) :553-558.

29. Pacumbaba, R. P. (1964). Personal communication.

30. Price, W. C. (1957). The cadang-cadang disease of coconut. Araneta J. Agric. 4 (2) :19-27.

31. Protacio, D. B. (1959). A study on the application of basic and minor elements as foliar sprays to control the cadang-cadang disease of coconuts. Philippine J. Agric. 24 (3-4) :99-106 .

32. Protacio, D. B. (1962). Intracellular inclusions observed in epidermal strips from foliage of cadang-cadang infected coconut treesin the Philippines. Plant Disease Reporter 46 (9) :686-687.

33. Reinking, O. A. (1950). Preliminary report on cadang-cadang disease and soil deficiency troubles in the Philippines. Plant Disease Reporter 34 : 300-304.

34. Sill, Jr., W. H. and Protacio, D. B. (1964). Unpublished data.

35. Sill, Jr. W. H. Bigornia, A. E. and Pacumbaba, R. P. (1963). Incidence of cadang-cadang in coconut trees of different ages. F.A.O. Plant Protection Bull. 11 (3) : 49-58.

36. Subido, P. S., Aquino, D. I. and Catral, L. T. (1960). A preliminary report on the role of fertilizers in the control of coconut cadang-cadang. 7th International Congress of Soil Science. Madison, Wisconsin, U.S.A. 3 : 329-335.

37. Valdez, R. B. and Ferino, M. O. (1963). The possible relationship of nematodes to cadang-cadang of coconuts. Proceedings of the symposium on cadang-cadang of coconut : 101-106.

38. Valera, C. L. (1963). Relationship of soil microbiological population to cadang-cadang. Preceedings of the symposium on cadang-cadang of coconut : 94-100.

39. Velasco, J. R. (1961). A re-examination of the coconut cadang-cadang question. The Philippine Agriculturist 45 (4) : 145-164.

40. Velasco, J. R. and Fertig, S. N. (1956). Progress Report: Sand culture experiment with coconut. The Philippine Agriculturist 39 : 540-548.

41. Velasco, J. R., Palmquist, E. M. and Mamawal, E. B. (1957). Copper content of some soil samples from coconut groves of Laguna and Quezon provinces. The Philippine Agriculturist 41 (4) : 241-243.

42. Velasco, J. R., de la Fuente, R. K., Pantastico, E. B., de la Peña, R. S. and Alejar, A. A. (1959a). Chemical composition and physical properties of soil samples from coconut groves. The Philippine Agriculturist 43 (7) : 433-445.

43. Velasco, J. R., Holaso, A., de la Pena, R. S., Pantastico, E. and Guevarra, V. F. (1959b). Aluminum and its possible relationship to the cadang-cadang of coconut. The Philippine Agriculturist 53 (2) : 177-199.

44. Velasco, J. R., de Guzman, E. V., Mercado, B. T. (1960). Growth of young coconut plant in sand culture. The Philippine Agriculturist 43 (8) : 548-576.

45. Velasco, J. R. Caney, C. S., Alejar, A. A. and Guevarra, D. B. (1963). The soil aspect of the cadang-cadang of coconut. Proceedings of the symposium on cadang-cadang of coconuts : 77-84.

46. Velasquez, C. C. (1963). Nematode associated with cadang-cadang infected coconuts. Proceedings of the symposium on cadang-cadang of coconut : 107-110.

II. Wilt disease

47. Bigornia, A. E., Calica, C. A. and Pableo, G. O. (1960). Epidemiological studies on the coconut cadang-cadang disease. Department of Agriculture and Natural Resources, Bureau of Plant Industry, Manila : 11 pp.

48. Menon, K. P. V. (1963). Some diseases and pests of the coconut palm in India. Proc. 8th Pacific Sc. Cong. 6-B : 313-317

49. Menon, K. P. V. and Nair, U. K. (1951). Scheme for the investigation of the root and leaf diseases of the coconut palm in South India. Coconut J. 5 (1) : 5-19.

50. Menon, K. P. V., Sankarasubramoney, H. and Pandalai, K. M. (1950). Investigations on diseases of the coconut palm in Travancore-Cochin State. Indian Coconut J. 3 : 81-94.

51. Nagaraj, A. N. (1964). Unpublished data.

52. Nagaraj, A. N. and Menon, K. P. V. (1955). Observations on root decay in coconuts, its cause and its relation to the foliar symptoms of disease in the disease belt of Travancore-Cochin. Indian Coconut J. 8 (3) : 97-105.

53. Nagaraj, A. N. and Menon, K. P. V. (1956). Note on the etiology of the wilt (root) disease of coconut palms in Travancore-Cochin. Indian Coconut J. 2 (9) : 161-165.

54. Nagaraj, A. N., and Pacumbaba, R. P. (1964). Specific diagnestic symptoms of cadang-cadang disease. To be published.

55. Nagaraj, A. N., Davis, T. A. and Menon, K. P. V. (1954). Sap-transfusion, a new device for virus transmission trials in palms. Indian Coconut J. 7 (3) : 91-95.

56. Nair, U. K. and Radha, K. (1959). Manuring-cum-spraying for the control of coconut palm diseases. Proc. 1st Conf. Coconut Res. Workers. The Indian Central Coconut Committee :250-256.

57. Pandalai, K. M., Sankrasubramoney, H. and Menon, K. P. V. (1958a). Studies on soil conditions in relation to the "Root" and "Leaf" diseases of the coconut palm in Travancore-Cochin. Part IV. Total and exchangeable calcium and magnesium contents of coconut soils. Indian Coconut J. 11 (2) : 49-66.

58. Pandalai, K. M., Sankarasubramoney, H. and Menon, K. P. V. (1958b). Studies on soil conditions in relation to the "Root" and "Leaf" diseases of the coconut palm in Travancore-Cochin. Part. V. Exchangeable cations, cation exchange capacity and pH of coconut soils. Indian Cocoonut J 11 (3) : 87-102.

59. Pandalai, K. M., Sankarasubramoney, H. and Menon, K. P. V. (1959). Studies on soil conditions in relation to the "Root" and "Leaf" diseases of the coconut plam in Travancore-Cochin. Part VI. The combined water, hygroscopic water, loss on ignition and water-table aspects of coconut soils. Indian Coconut J. 12 (3-4) : 87-100.

60. Radha, K. and Menon, K. P. V. (1954). Studies on the wilt (root) disease of the coconut palm. Indian Coconut J. 7 (3) : 99-106.

61. Sankarasubramoney, H., Pandalai, K. M. and Menon, K. P. V. (1951). The manganese content of soil and plant tissue in relation to the root and leaf diseases of the coconut palm. Indian Coconut J. 5: 165-170.

62. Sankarasubramoney, H., Pandalai, K. M. and Menon K. P. V. (1952). On the nutritive contents of the leaf tissues of the coconut palm in health and in disease. Indian Coconut J. 6 (1) : 7-18.

63. Sankarasubramoney, H., Pandalai, K. M. and Menon, K. P. V. (1954). Studies on soil conditions in relation to the "Root" and "Leaf" diseases of coconuts in Travancore-Cochin. Part I. Nitrogen, Organic matter content and Carbon/Nitrogen ratio of coconut soils. Indian Coconut J. 8 (1) : 5-25.

64. Sankarasubramoney, H., Pandalai, K. M. and Menon, K. P. V. (1955). Studies on coconut soil in relation to the root and leaf disease of the coconut in Travancore-Cochin. Part II. Total phosphoric acid, available phosphoric acid and iron content of coconut soils. Indian Coconut J. 9 (1) : 20-29.

65. Sankarasubramoney, H., Pandalai, K.M., and Menon, K.P.V. (1956). Studies on coconut soil in relation to the root and leaf disease of the coconut palm in Travancore-Cochin. Part III. Total available and exchangeable potassium content of coconut soils. Indian Coconut J. 9 (2) : 90-100.

66. Shanta, P. and Menon, K. P. V. (1960). Cowpea (Vigna sinensis Endl.) an indicator plant for the coconut wilt virus. Virology 12 : 309-310.

67. Shanta, P. and Menon, K. P. V. (1961). Studies on some properties of the coconut wilt virus. Indian Coconut J. 15 (1) : 36-46.

68. Shanta, P., Menon, K. P. V. and Pillai, K. P. (1960). Aetiology of the wilt (root) disease : Investigations on its virological nature. Indian Coconut J. 13 (2) : 56-66.

69. Sill, Jr., W. H., Bigornia, A. E. and Pacumbaba, R. P. (1963). Incidence of cadang-cadang in coconut trees of different ages. F.A.O. Plant Protection Bull. 11 (3) : 49-58.

70. Verghese, E. J.,Sankaranarayanan, M. P. and Menon K. P. V. (1959a). Chemical studies on the leaf and root (wilt) diseases of coconuts in Travancore-Cochin. II. Nutrient Content of leaves of healthy and diseased trees. Proc. 1st Conf. Coconut Res. Workers. Indian Central Coconut Committee : 310-319.

71. Verghese, E. J.,Sankaranarayanan, M. P. and Menon K. P. V. (1959b). Chemical studies on the leaf and root (wilt) diseases of coconuts in Travancore-Cochin. VII. Cadmium content of coconut soils and coconut leaves. Proc. 1st Conf. Coconut Res. Workers. Indian Central Committee : 366-370.