Principes, 37(4), 1993, pp. 187–196

The Threat of Cadang-Cadang Disease

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ABSTRACT

In 1975 the viroid cause of cadang-cadang disease of coconut and other palms became established in the Philippines and Guam. The suspected occurrence of this disease on other Pacific inlands requires confirmation. Replanting with the early maturing Mawa cultivar, widely advocated in the Philippines, merely provides a temporary but inadequate remedy because this cultivar is susceptible to viroid infection. The spread of the disease could be controlled by proper extension work, requiring the thorough decontamination of tools used by plantation workers. Concentrated sodium carbonate solution can degrade RNA viroids, remaining stable and retaining its RNA-destroying activity under tropical conditions. An extensive testing program for resistance to cadang-cadang is imperative. Such a long-term program will require proper funding, commitment, trained personnel and quarantined importation of coconut cultivars from all parts of the world.

Cadang-cadang disease of palms, the most devastating viroid disease known, is always fatal. In the Philippines, where the disease has killed more than 30 million coconut palms, currently 1,000,000 palms succumb every year. The Philippines are the world's largest producer of coconuts and half of the country's export income is derived from copra, the dried coconut meat from which coconut oil is extracted. This oil provides the main source of fat in the diet of the local population. Elsewhere coconut oil is used in soaps, margarines, synthetic rubber, cosmetics and moisturizing compounds. Coconut palms also provide lumber, while leaves are used for thatching, brooms, baskets and hats. The husk fiber is transformed into mats and carpets, and ropes are made from the yarn. On many Pacific atolls the only potable liquid is provided by the coconut water (coconut "milk") and the palms provide the only shade and building material.

Cadang-cadang disease occurs and spreads currently on Luzon and a number of other Philippine islands, as well as on Guam. The disease has been suspected on a few other Pacific islands recently.

Historical Background

Cadang-cadang means "dying-dying," or slowly dying in Bicolano, the language spoken in southeastern Luzon. The first well-documented outbreak of the disease was noticed on San Miguel Island off Luzon in 1928. By 1933 nearly 25% of the coconut palms had died there and the plantation owners approached Prof. Gerardo Ocfemia, a U.S. trained plant pathologist and head of the Plant Pathology Department at the College of Agriculture in Los Banos, to help control the disease. Since no fungi or bacteria were found associated with dying palms, Ocfemia (1937) concluded that the disease was infectious and most likely caused by a virus. Shortly thereafter the disease reached the mainland of Luzon Island. Before the Japanese invasion in 1941, nearly half of the original 250,000 coconut palms on San Miguel had died and the disease appeared in several provinces in the Bicol area of Luzon, as well as on some nearby islands. During World War II no further research was carried out, but after the war the Philippine Government obtained active help from the United States, through the International Cooperation Administration (ICA). By that time cadang-cadang was devastating plantations in the provinces of Albay,

Sorsogon, Camarines Sur, and Camarines Norte, as well as on the islands of Samar, Masbate, and Catanduanes. On San Miguel 90% of the palms were dead and the remaining ones died soon afterwards. Elsewhere on Luzon the disease incidence ranged from 10% to 60%. ICA assigned Dr. Donald De Leon, an entomologist who had earned his Ph.D. at Cornell University in 1933 and specialized in insect transmission of viruses and taxonomy, to study the disease in the Philippines (De Leon and Bigornia 1953). In 1953 the ICA abruptly cancelled the technical assistance program for cadang-cadang and permanently abandoned it under the pressure of U.S. soybean and corn oil producers' lobby. The Philippine Government turned for help to the United Nations' Food and Agriculture Organization (FAO). During the following four decades, experts were sent by FAO to the Philippines to study the devastating disease and to determine its cause, manner of spread, and possible means of control. Indian, U.S., Australian, German, and Italian experts were assisted by Philippine scientists from the Bureau of Plant Industry.

By 1960 the disease reached Bondoc Peninsula in Quezon Province, only a short distance from the solid stand of coconut palms of Laguna Province (Maramorosch 1961). Less than 100 palms of the original quarter million on San Miguel were still standing and slowly dying. Strip surveys indicated that more than a million trees became infected every year (Maramorosch 1964).

Until 1967 plant pathologists did not know of the existence of autonomously replicating low-molecular-weight RNA species that could infect and damage plants. In 1967 Diener and Raymer (1967) found that potato spindle tuber disease is caused by a free RNA. The following year Diener (1968) demonstrated that this RNA is sensitive to ribonuclease. In 1971 Diener coined the name "viroid" as a generic term for pathogenic nucleic acids of low molecular weight. Viroids were subsequently found as causative agents of other plant diseases (Romaine and Horst 1974; Van Dorst and Peters 1974; Diener 1979, 1987). In 1975, Randles provided evidence for the association of two RNA species with cadang-cadang disease. The establishment of the viroid etiology was followed by intensive studies on the epidemiology of the disease and rate of spread as well as on certain measures to control cadang-cadang. Surprisingly, by 1992 the disease has not yet entered the main coconut growing areas in Laguna province. Its spread in some areas appears to be very slow, yet in parts of Camarines Sur, where the incidence in 1956 was only 3%, some areas are now heavily affected and up to 70% of the palms are dying.

Symptomatology

The cadang-cadang disease cannot be diagnosed authoritatively by symptomatology, but in an endemic area symptoms can assist in the detection of affected palms.

On young leaves the earliest symptoms are tiny, circular, translucent spots on the veins. On mature leaves the number and size of the spots increases so as to impart a characteristic chlorotic pattern of the spots (Fig. 1). On the lower surface of mature coconut leaves appear irregular "water-soaked spots." Mature coconut leaves display yellow streaks as a result of enlargement and fusion of adjacent spots. The veins and veinlets become cleared in a very characteristic, although not very pronounced, way. The basal portion of the petiole of a healthy tree differs strongly from those of a diseased one, because the stipules of diseased trees remain attached to the base of the petiole, giving it a winged appearance. Nuts are produced only during the early stages of the disease, but they are smaller, rounded at the base or misshaped and scarified with brown streaks (Fig. 2). Nut bearing of diseased trees can stop within 18 months, but occasionally the period is extended to 4–5 years. A

comparison of healthy and diseased inflorescences shows an abundance of female florets (buttons) on the healthy and fewer on the diseased, that is reduced in size and retains a characteristic upward position of its branches (Fig. 3).

The fronds of diseased palms gradually assume an erect position in the crown (Fig. 4), dry up, die, and drop off. This results in a gradual reduction in the total number of fronds until a small, yellowish-green or yellow tuft is left at the apex of the trunk. Finally, the bud dies, falls off, and leaves the crownless trunk standing. A plantation in this stage looks like a field of telephone poles (Fig. 5).

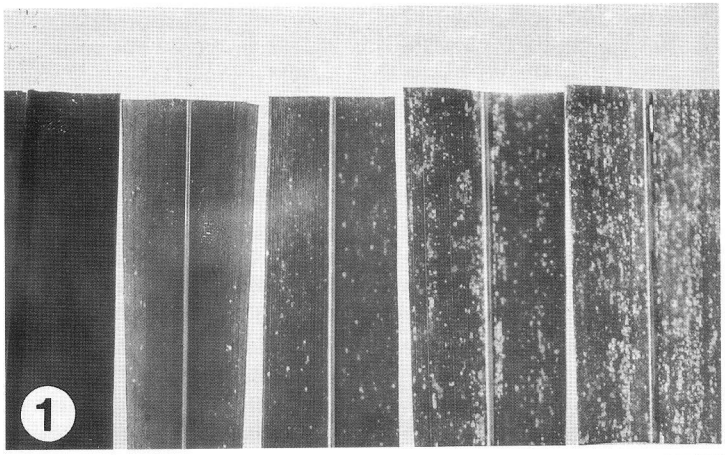
Coconut palms seldom become diseased before flowering. In rare cases the disease strikes before flowering sets in. The progress of the disease is slow, with a five to ten year interval between the appearance of first symptoms and the death of younger trees. In older palms this period may extend to 15–18 years.

The proper recognition of typical symptoms of the disease helps in field diagnosis but it requires long practice and experience and cannot prove accurately cadang-cadang etiology. In an epidemic area, symptomatology is still adequate for recognizing the disease. However, the symptoms may differ slightly, depending on the coconut variety and other factors. While the progression of symptoms is constant in the Philippines, observations in Guam, where the disease is called tinangaja, revealed slightly different nut symptoms there (Weston 1918, Maramorosch 1961, Boccardo et al. 1981, Boccardo 1985). The outbreak of cadang-cadang on Guam preceded the appearance of the disease on San Miguel Island by more than 20 years (Weston 1918). No recovery has ever been observed and the disease is always fatal. Therefore cadang-cadang presents one of the most serious threats to coconut palms not only in the Philippines but also to some Pacific islands, and, if not controlled, to all of southeast Asia.

Epidemiology

The mode of spread of cadang-cadang has not been well elucidated. An aerial vector has been suspected, but none ever found or incriminated. Viroids that cause potato spindle tuber, chrysanthemum stunt, hop stunt, citrus exocortis, and other viroid diseases are mechanically transmissible and do not require vectors to infect susceptible plants (Diener 1979). Man is the actual vector in those diseases. Experimentally, the cadang-cadang viroids have been transmitted to coconut and other palms mechanically (Randles 1985). There is no doubt that mechanical transmission could be responsible for the spread of the disease under natural conditions. The fact that young palms that have not yet commenced bearing nuts are very seldom infected might support this assumption. On the other hand, pollen transmission or long incubation periods would also fit this observation. However, pollen transmission could in no way explain the very rare infection of palms before flowers are formed. Pollen transmission could hardly be responsible for the very slow, perhaps not more than 500 meters, spread of the disease in certain areas, while in others cadang-cadang seemed to have been able to jump from island to island.

The mechanical transmission hypothesis seems supported by observations made by me on Luzon Island (Maramorosch 1987*a*, *b*, 1992). A consistent correlation was found between the destruction of coconut palms owned by Bicolanos and the lack of infection on plantations owned by Tagalogs. This correlation, at first, appeared to be absurd. However, if we accept that viroids are primarily or exclusively transmitted mechanically by man, the observed correlation can easily be explained. Most likely the cadang-cadang viroids are being carried from infected to healthy palms by tools, called bolos, the machetes used by plantation workers. Steps are first cut at the base of a palm to facilitate climbing.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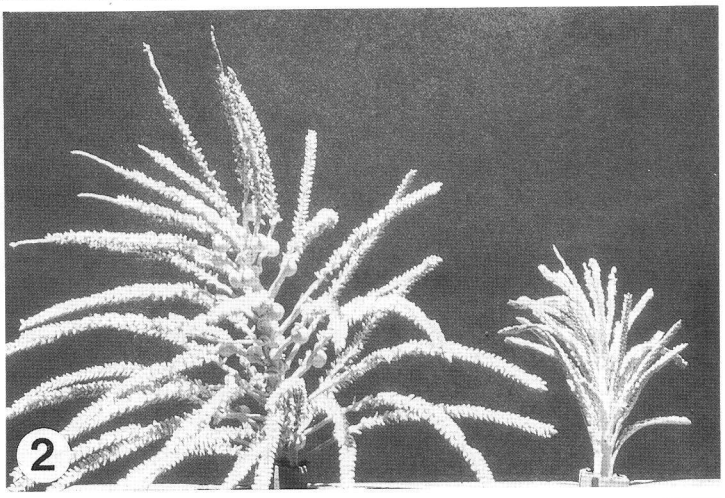
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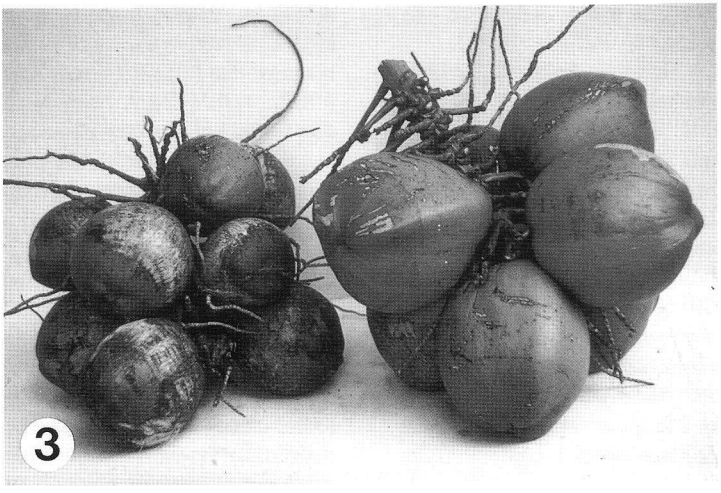
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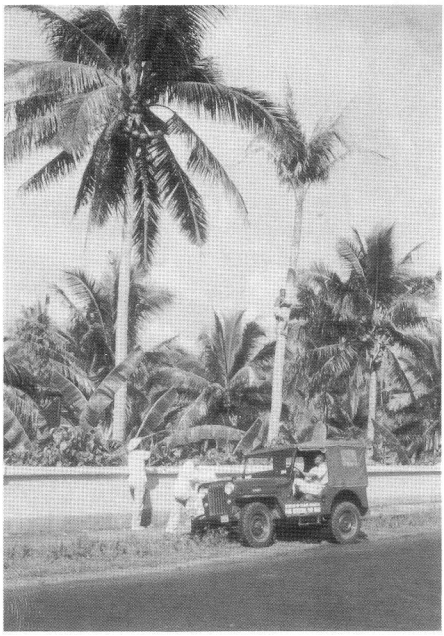
Afterwards, the same knives are used in the crowns to dislodge the nuts. In addition, flower sap is often being collected for "tuba," the sugary sap that ferments and provides a cheap alcoholic beverage in the Philippines. For tuba collection, crowns of several palms are usually linked with bamboo planks so workers can move from palm to palm high up in the crowns, without descending each time. This practice easily explains why clumps of diseased palms often occur at considerable distance from other diseased palms. If a worker uses his bolo on an infected palm, the cadang-cadang viroid can be transmitted to the next palm when steps are cut into the base, when nuts are being collected, or when the inflorescences are being tapped for tuba. Bicolano plantation owners prefer to hire Bicolano workers, while Tagalog owners prefer mainly Tagalogs for work in their plantations. Workers employed decades ago on San Miguel island were Bicolanos from Tabaco, the first locality where subsequently the disease appeared on Luzon Island. The link with Bicolano language seems to make sense on Luzon, but it does not pertain to Guam. There the disease was actually noticed earlier than in the Philippines (Weston 1918) and it spread all over the island in later years (Boccardo 1985).

It is generally accepted that all viroid diseases are of recent, twentieth-century origin (Diener 1979). Despite the fact that all viroids of plants can be transmitted mechanically, attempts to find other means of transmission are often being made. Attempts to find an insect vector of cadang-cadang viroids have been continued until now, even though this seems a complete waste of time and funds. If an insect were responsible, the localization of the disease in a very small pocket area in Quezon Province, at the locality of Mulaney, could not be explained. This small pocket is surrounded by healthy plantations. Neither can the hypothesis of pollen transmission be sustained—the disease would have moved out of Mulaney town since 1958 if the causative viroids were pollen-borne, wind-borne, or carried by insects. The mechanical transmission by contaminated machetes of plantation workers remains the most plausible explanation for cadang-cadang.



4. Left: healthy coconut palm. Right: diseased palm with erect fronds and leaflets reduced in size.

Cadang-cadang viroids have a narrow host range, limited to the palm family. No

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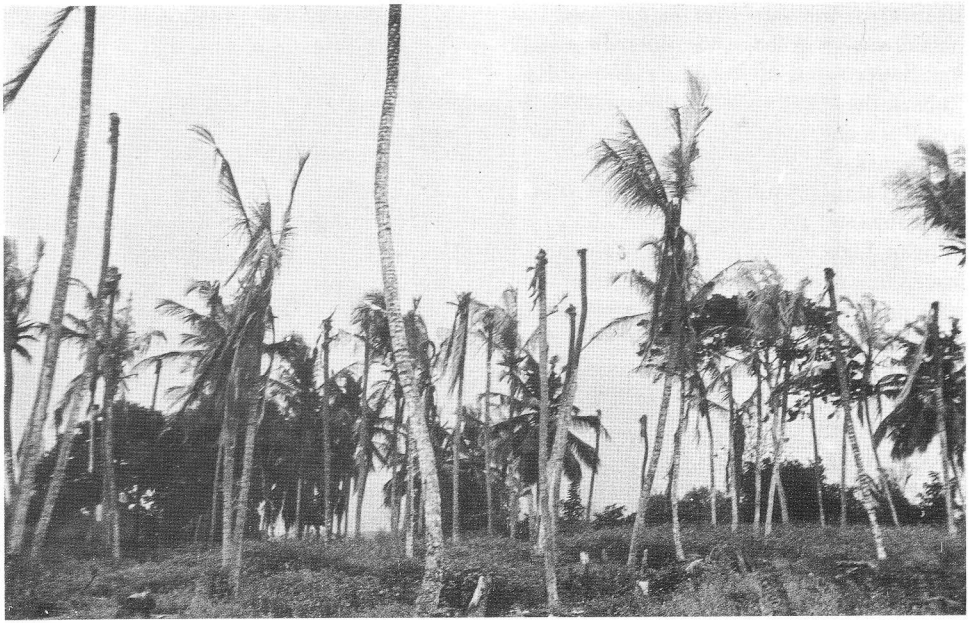
1. Portions of diseased leaflets in transmitted light, with progressive appearance of spots. 2. Left: healthy female inflorescence with an abundance of buttons. Right: inflorescence from diseased palm, lacking buttons and reduced in size. 3. Left: scarified small nuts from cadang-cadang diseased coconut palm. Right: healthy nuts.



5. Coconut plantation on Luzon island, totally destroyed by cadang-cadang disease.

herbaceous hosts are known to be susceptible. In addition to coconut, *Cocos nucifera*, several other members of the palm family, growing in the Bicol provinces, were found to exhibit symptoms similar to cadang-cadang (Maramorosch 1961). The African oil palm, *Elaeis quineensis* Jacq., the Anahau (*Areca catechu* Linn.), the Bunga de China (*Adonidia merrilli* Becc.), and the pugahan fish tail palm (*Caryota cumingii* Lodd.) have all been found to display the yellow mottle symptoms, olivaceous spots, decline in fruit production followed by sterility, brittle leaves, stunting, and a general slow decline and death, characteristic of cadang-cadang.

It is reasonable to assume that cadang-cadang disease is of fairly recent origin in coconut palms. Its ability to spread from plantation to plantation and from one island to another, demonstrated clearly in its progress during the past decades, makes cadang-cadang a dangerous potential threat to all the islands of the Philippine Republic and to other coconut growing areas of southeast Asia. In the Philippine Republic, with its population increasing very fast, the loss of so many million coconut palms is catastrophic.

Etiology

Randles (1975) discovered two viroid-like ribonucleic acid species, associated with cadang-cadang affected coconut palms. This finding determined the viroid etiology of cadang-cadang. Consequently, viroid association of tinangaja-affected palms on Guam was also demonstrated (Boccardo et al. 1981, Boccardo 1985).

When coconut seedlings were inoculated with crude nucleic acid extracts by high power injection and by razor slashing (Randles et al. 1979), typical cadang-cadang symptoms appeared in the mechanically inoculated seedlings. Nucleic acid probes (Randles and Hatta 1979) were used in hybridization assays, permitting the analysis of the kinetics and comparison of percentage homology. This method defi-

nitely identified the presence of cadang-cadang viroids in the African oil palm and the buri palm, as well as the Manila palm grown in the Philippines. It also confirmed that tinangaja, the coconut palm disease of Guam, was cadang-cadang because homologous ccRNA was detected there as well (Boccardo et al. 1981). The viroid nature of cadang-cadang disease was further confirmed by using highly purified RNA and demonstrating that it was infectious and reproduced the disease in mechanically inoculated palm seedlings. Nucleic acid hybridization probes are now being used in the Philippines to detect infected coconut palms long before they show symptoms of the disease—perhaps two years before visual symptoms appear. This allows removal of trees before they could become a potential source of infection to neighboring palms.

Control

Sound and successful strategies to control cadang-cadang should make use not only of the available manpower in the Philippines but also of facilities of international organizations. Simple, reliable and cost efficient measures of control will require long-term and costly efforts.

Replanting has been suggested as early as the 1950s, because the disease spreads comparatively slowly and kills slowly, so that copra production can be maintained in affected areas by new plantings. The use of early maturing varieties, such as Mawa, permitted coconut production to continue on Luzon Island despite considerable losses. Unfortunately, Mawa palms are not resistent to cadang-cadang infection and they die just as do later maturing palms.

Usually eradication is recommended as a standard procedure to prevent the spread of a plant disease. Eradication has failed to control cadang-cadang, probably because of the long period between the actual contraction of the cadang-cadang viroids and the appearance of symptoms that would permit the identification and removal of diseased trees.

At present, eradication can be carried out much earlier. It was hoped that this early eradication would prevent further spread of the devastating disease. However, it has not been proven that the infected and as yet symptomless trees were unable to contaminate the tools of plantation workers. In fact, eradication has not been able to prevent further spread of cadang-cadang and disease continues to appear in places where all infected trees were cut and removed.

There is another possibility to control cadang-cadang. Viroids form mild strains that barely affect plants but protect them from infection by a virulent strain (Fernow 1967, Niblett et al. 1978). If a mild strain of the cadang-cadang viroid could be found in nature or developed artificially, such strain could be introduced into healthy seedlings to protect the palms from the killer form. Long-term tests will be required to implement this approach.

The decontamination of bolos used by coconut workers could provide a simple control measure, by dipping the knives into a solution of concentrated sodium carbonate solution (Na_2CO_3). This solution would degrade the contaminating RNA viroids and retain its stability and its RNA-degrading ability in the tropical environment. Although this suggestion has been made repeatedly (Maramorosch 1987*b*, 1992), it has yet to be implemented.

Hopefully, cadang-cadang will be curtailed eventually by a combination of control measures—selection of tolerant or resistant cultivars, introduction of mild strains for cross-protection, and decontamination of tools.

Discussion and Conclusions

Cadang-cadang, like all other viroid diseases, originated in the twentieth century. Where did it come from? A possible source

of the viroid could have been an ornamental plant introduced to Guam, and later to San Miguel Island around the turn of the century. Perhaps Spanish boats, sailing from Acapulco to Guam, carried such a plant on board? Cadang-cadang was already well established on Guam when Prof. Weston described the disease in 1917, and it appeared on San Miguel Island a decade later, but since the incubation period and slow progress of the disease are now well established, the first contacts of coconut palms with the infectious viroids must have preceded the observed destruction by several years. No plant quarantine existed then and even today quarantine is comparatively lax in tropical countries.

Cadang-cadang is unquestionably an extremely serious palm disease that has caused huge devastation and great economic losses. If we look at the problem as a "local," Philippine disease, the present economic and political instability in that country makes any suggested solutions highly problematic. The rapid and constant population growth in the islands is hardly conducive to long-term breeding for disease resistance, the best practical solution. Preventing the spread by decontamination of machetes has been suggested but not implicated. Replanting gives temporary relief only. The disease, endemic in certain parts of the Philippines and Guam, apparently has been noted on a few remote Pacific islands recently. Besides, cadang-cadang is not limited to coconut palms. In the Philippines other palms have been diagnosed—first only by symptomatology and later by viroid analysis—to be infected by cadang-cadang viroids. Therefore the problem is not, and must not be, considered as of local importance only.

Large trials have been initiated in Albay Province to find whether natural selection for resistance would reveal a resistant coconut population among native palms. This attempt has been based on the assumption that cadang-cadang disease may have existed in Bicol long before it was reported by Ocfemia from San Miguel Island in 1930 and thus natural selection may have started quite early. I consider the assumption as incorrect and the tests as ill-conceived for the following reasons. Ocfemia, a native of Guinobatan, used to spend his yearly vacations and holidays in that locality in Bicol. A very well trained plant pathologist and a keen observer, he never saw a cadang-cadang diseased palm in or around Guinobatan, as he told me when he visited Rockefeller University in the late 1950s. If the disease had existed in Albay earlier, resulting in the survival of resistant palms, the subsequent severe outbreaks could hardly be explained.

Attempts to import a large number of coconut varieties from other parts of the world were initiated by me and were also attempted by the late F. O. Holmes 30 years ago in the hope that a resistant variety would be found. Shipments were made possible by a joint effort of several people and financial support obtained from the Franklin Baker Company. When sacks with seed nuts arrived by air in Manila, they were "accidentally" destroyed or "forgotten" while in the customs area of the airport. At present only local varieties are being tested. The Mawa variety, owned by a former close friend of President Marcos, the "coconut king" Eduardo Cojuangco, was hailed as the best solution to the cadang-cadang problem. It grows faster than the tall varieties but is susceptible to the disease. Instead of depending on such temporary remedies, an effort should be made to plant both local and imported varieties in isolation, on San Miguel Island, where the disease has never ceased to exist and where seedlings could be mechanically inoculated. Even if such tests were performed and a resistant variety found, the results would benefit the Philippines in 20–50 years at the earliest. Who could, and would be willing, to finance such a long-term effort?

There seems to be no question that cadang-cadang, similarly to all other viroid

diseases, is of recent, twentieth-century origin. Human activities have contributed to the origin of viroids in cultivated plants but they might have existed earlier in a latent state in as yet unknown natural hosts in which they cause no symptoms. Through human activities, such as grafting, handling or cutting, viroids have been transmitted to susceptible cultivated plants. We can expect that new viroids will continue to appear unexpectedly, just as did those known today which all appeared in the twentieth century (Diener 1987).

The discovery of viroids has opened new vistas in plant pathology as well as in medicine and molecular biology, becoming the first RNA pathogens whose molecular structure became completely known. Even though the natural mode of transmission and possible natural reservoirs of cadang-cadang and other viroids are still an enigma, they are among the most thoroughly understood host-pathogen systems.

In terms of dollars lost in copra production, the cadang-cadang disease represents the most important viroid disease known today and one of the most devastating diseases of plants anywhere. Efforts to curtail the disease have not yet been successful and cadang-cadang continues to post a serious threat to the economy of the Philippine islands and to other tropical areas of the world.

Acknowledgments

The author's work was supported by the Food and Agriculture Organization, the United Nations Development Fund, and the New Jersey Agricultural Experiment Station, Publication No. D-08420-15-92, supported by State funds.

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