Causal Relationship Between Cucumber Mosaic Cucumovirus and Kava Dieback in the South Pacific

R. I. Davis, Former Junior Research Fellow, and **J. F. Brown,** Associate Professor, Department of Botany, University of New England, Armidale NSW 2351 Australia; and **S. P. Pone,** Plant Protection Adviser, South Pacific Commission, Private Bag, Suva, Fiji

ABSTRACT

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The major constraint to kava (*Piper methysticum*) production in the South Pacific is a dieback disease of previously unknown etiology. The development of a mosaic symptom on young leaves plus certain leaf growth distortions frequently precedes the dieback. Data are presented that show that cucumber mosaic cucumovirus (CMV) is either the direct cause of kava dieback or a significant component of a disease complex. Double antibody sandwich–enzyme-linked immunosorbent assay (DAS-ELISA) showed that CMV is geographically widespread in kava plants with leaf and dieback symptoms throughout the four main kava-producing nations of Fiji, Vanuatu, Tonga, and Western Samoa. An isolate of CMV originally obtained from a dieback-affected plant caused similar dieback symptoms in 80% of mechanically inoculated kava plants. Aphid transmission was also demonstrated. CMV was not fully systemic in all mechanically inoculated plants. DAS-ELISA indicated that the virus was not always present in every stem on multi-stemmed plants and 56% of subsequent new stem growth was uninfected.

Kava (*Piper methysticum* Forster f.) is an important traditional and cash crop in the Pacific Island nations of Fiji, Tonga, Vanuatu, Western Samoa, and parts of Micronesia (Ponape). It has considerable export potential as a source of pharmaceutical compounds. Kava is used to prepare an intoxicating, but non-alcoholic ceremonial and recreational beverage by mixing ground or masticated roots and stem bases with water.

Kava provides growers with higher economic returns than most alternative crops (2,8). Kava is a hardy, slow-growing perennial plant that reaches 2 to 3 m in height at the time of harvest, 3 to 5 years after planting. Plants develop a stump or caudex from which several monopodial stems arise.

The most serious disease of kava in the South Pacific is a dieback in which a portion or all of the stems rot from the tip or from the nodes progressively to the stem base (R. I. Davis et al., *unpublished*). This disease was first reported in Fiji in 1932 (12) and current crop losses have been estimated to reach 60% in Fiji (Jainend Kumar, Fiji Ministry of Agriculture, Fisheries and Forests, *personal communication*). The disease is characterized by a black discoloration of the affected tissue and stems, which usually disintegrate. New shoots often arise from the stem base and cycles of dieback followed by regrowth are common.

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A symptom less frequently observed is wilting prior to stem rot and death. In recent years, entire plantings have been almost destroyed in Tonga, Fiji, Western Samoa (R. I. Davis et al., *unpublished*), and Vanuatu (Victor Tiollier, Vanuatu Department of Agriculture and Horticulture, *personal communication*) The disease was previously of unknown etiology.

Over a period of several years, a number of field trials involving selective chemicals (several different fungicides, insecticides, nematicides, and an antibiotic) and many inoculation tests (using a diverse selection of organisms isolated from the roots and foliage of diseased plants) failed to implicate fungi, bacteria, and nematodes in the dieback syndrome (R. I. Davis et al., *unpublished*). Studies on the pattern of spread of the disease within kava monocultures suggested that the pathogen involved was disseminated as airborne inoculum or was transmitted by an airborne vector (R. I. Davis et al., *unpublished*).

Detailed monitoring of plants from the time they were transplanted into the field showed that certain symptoms often preceded the stem rot. These included mosaic symptoms (numerous small patches of chlorotic tissue with defined boundaries) along with one, two, or three different types of leaf distortions (puckering along larger leaf veins, crinkling of leaves, and blistering of leaves) (Fig. 1). Sometimes however, only a general chlorosis accompanied by regions of necrotic tissue was found on leaves prior to dieback. Small regions of internal necrosis associated with vascular tissue were often observed inside stems that showed these leaf symptoms but no other externally visible signs of infection.

Cucumber mosaic cucumovirus (CMV) has been shown to be present in leaves showing mosaic, mottling, and chlorotic symptoms, by immunoelectron microscopy and by dsRNA analysis (10). Preliminary surveys conducted in Tonga using double antibody sandwich–enzyme-linked immunosorbent assay (DAS-ELISA) and dot blot immunoassay techniques to detect the virus (R. I. Davis et al., *unpublished*) showed that CMV was most often detected in leaves showing mosaic symptoms plus one or more of the leaf distortion symptoms (75% of 125 leaves examined) and less frequently in leaves showing veinbanding symptoms (49% of 104 leaves examined), crinkling (41% of 39 leaves examined), or general chlorosis (47% of 55 leaves examined). However, CMV was also present in some leaves that showed no viruslike symptoms (38% of 122 leaves examined).

This paper reports results of (i) studies to determine the relationship between mosaic and growth-distortion symptoms and the subsequent dieback of the plants, (ii) a survey in Fiji, Vanuatu, and Western Samoa to determine if the disease there is the same as that in Tonga, (iii) pathogenicity tests to determine whether dieback is caused by CMV, and (iv) aphid transmission tests.

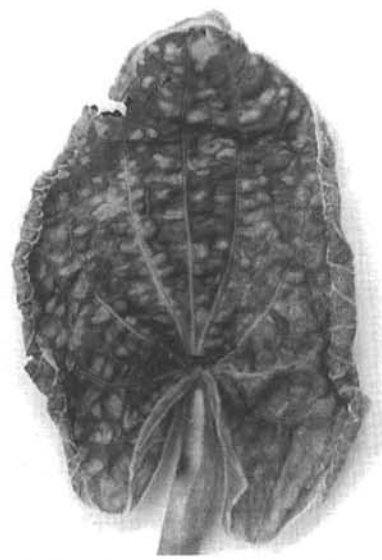

Fig. 1. Viruslike leaf symptoms that often preceded dieback of stems.

MATERIALS AND METHODS

Cultivation of plants. Each kava plant used in inoculation experiments was vegetatively propagated from a portion of stem more than 2 cm in diameter and about 4 cm long, with a single node. Cut ends of the stem pieces were coated in wax to reduce desiccation and infection by fungi. The sets were planted in trays containing potting mix or field soil previously fumigated with methyl bromide. When new shoots were 1 to 5 cm high, they were transplanted into field soil contained in 3-liter polythene bags. Plants were grown in an insect-free greenhouse under partial shade at 21 to 28°C.

Serological tests for CMV. Direct DAS-ELISA was used to test for the presence of CMV. Procedures and chemical preparations were similar to those described by Clark and Adams (3). Antiserum was obtained from Agdia Inc., Elkhart, IN. Two wells in a 96-well Nunc or Imulon immunoplate were used for each sample. Colorimetric responses were recorded as the absorbance at 405 nm read on a Bio-Tek El 307 EIA microtiter plate reader (Bio-Tek Instruments Inc. Winooski, VT) after at least 1 h of incubation at 25 to 30°C.

Samples were interpreted as positive when the absorbance values exceeded twice the mean of the five negative controls included on each plate. In some tests, many absorbance values were obtained that were considerably larger than that of the negative control mean reading, but still less than twice this figure. In these cases, a category of marginal positive was recognized. Readings that fell into this category were less than twice the mean of the controls but greater than the mean plus four times the standard deviation.

The negative controls consisted of leaves, petioles, or stems taken from kava plants that were grown in an insect-proof greenhouse, showed no symptoms of infection, and had given negative DAS-ELISA results in at least 10 previous tests to detect CMV. Positive controls included on each plate were one or more fresh kava leaves showing mosaic and leaf-growth distortions (obtained from the field on the day of testing) and *Nicotiana tabacum* L. leaves infected with CMV originating from kava.

Isolation of CMV from kava. The isolate of CMV used for inoculation experiments was originally obtained from a kava leaf showing mosaic and distortions. The stem also showed symptoms of active dieback. The mechanical sap inoculation procedure used consisted of triturating infected leaf tissue (at a ratio of about 1 g to 5 ml) in 0.168 M phosphate buffer, pH 8.0, containing 0.25% (wt/vol) Na_2SO_3, 10% (wt/vol) polyvinylpyrolidone, and a small amount of the abrasive celite. The inoculum was rubbed onto the expanding leaves of the recipient plant, and the leaves were then immediately rinsed with water. The CMV isolate selected for inoculation tests was first passed through aphids (*Aphis gossypii* Glov.) from *N. tabacum* to *N. tabacum*. The isolate was then serially transferred by mechanical inoculation five times from *N. tabacum* to zucchini (*Cucurbita pepo* L.) and back to *N. tabacum* In addition, five serial single-lesion transfers were made using the local lesion host *Chenopodium quinoa* Willd. before passage back to *N. tabacum*.

Relationship between viruslike symptoms on leaves and subsequent development of dieback symptoms. An experiment was initiated in 1991 in a field of about 500 kava plants at Vaini Agricultural Research Station in Tonga. As each of 50 stems on different plants developed mosaic plus leaf distortion symptoms on one or more leaves, it was labeled with a weatherproof tag. Each tagged stem as well as stems on an adjacent "symptomless control" plant on the day of tagging were examined at weekly intervals for 12 weeks. Leaf and dieback symptoms were recorded on each plant at each examination. A similar trial was conducted in 1992.

Incidence of CMV and dieback symptoms in Tonga, Fiji, Vanuatu, and Western Samoa. The incidence of dieback and CMV infection was assessed in two and six kava plantings, respectively, on the islands of Tongatapu and 'Uta Vava'u in Tonga, and in one, three, and three plantings, respectively, on the islands of Espiritu Santo, Tanna, and Efate in Vanuatu. Likewise, two plantings on the island of Viti Levu in Fiji, and five and nine plantings, respectively, on the islands of Upolu and Savai'i in Western Samoa were observed.

At each site in which kava was suspected to be infected with CMV, two leaves from each of five plants were selected for testing by DAS-ELISA. Some leaves with other leaf symptoms (crinkling, mottling, or vein banding alone) were also collected. Leaves were taken from plants with and without visible signs of dieback on stems. In addition, two leaves were selected from each of five plants at each site that showed no visible symptoms of CMV infection on leaves or of dieback on stems. These leaves served as "healthy" controls. Similar samples of 10 symptomless leaves (from five plants) were also taken from some sites where no visible signs of CMV infection or dieback were found. Leaves were taken to Vaini Agricultural Research Station, Tonga, and DAS-ELISA was used to test for the presence of CMV. Leaves were held over ice in a cool box or were desiccated when DAS-ELISA could not be conducted within 8 days.

Pathogenicity tests. Planting material of cultivar Kula was selected from the island of 'Eua in Tonga, where dieback was not widespread. Stems were examined for signs of disease and three leaves were tested for CMV by DAS-ELISA. Only stems that were virus free were used.

At planting, each single node set was individually labeled to indicate the stem from which it was derived and its position on the stem. At 12 weeks and 1 week before pathogenicity tests were conducted, the youngest three leaves gave negative results in DAS-ELISAs for CMV. Thirty pairs of plants were selected when plants were 5 months old. In order to reduce genetic and other forms of variability, both members of each pair were derived from adjacent nodes on the same parent stem. One member of each pair was mechanically inoculated with CMV. The other served as the control.

Extracts from leaves of *N tabacum* that contained the biologically "purified" isolate of CMV were used to mechanically inoculate the youngest three leaves of each plant. Plants were grown in darkness for 24 h immediately before inoculation. Control plants were inoculated with the buffer rubbed onto the leaf surface. The experiment was conducted in an insect-free greenhouse at 21 to 28°C.

Plants were monitored for disease symptoms for 14 weeks. When leaf symptoms or dieback lesions developed, affected tissue was tested for the presence of CMV using DAS-ELISA. Five weeks postinoculation, stems that did not develop symptoms were tested one or more times for virus, using DAS-ELISA, on a composite sample of the three youngest leaves. When an inoculated plant was tested, a composite sample from the three youngest leaves of the uninoculated control member of the pair was also tested. All surviving stems (including all new sprouts over 2 cm in length) were subjected to a destructive DAS-ELISA 14 weeks postinoculation.

Aphid transmission. *Aphis gossypii,* the aphid most commonly found feeding on kava, was tested for its ability to transmit CMV. Colonies were established that were free of nonpersistent viruses (aphids were starved overnight and reared on taro [*Colocasia esculenta* var. *esculenta* (L.) Schott], which is not known to be a host of CMV). After a 1-h fasting period, aphids were given an acquisition access period of 1 to 5 min on kava leaves that had mosaic, had been mechanically inoculated with CMV, and had tested positive for CMV. Groups of 50 aphids were then transferred to each of five healthy kava plants and allowed to feed for 2 h. Equal numbers of aphids that had not been allowed to feed on infected kava leaves were also transferred to equal numbers of kava plants for the same period of time. The kava plants used in this experiment had given negative DAS-ELISA results for the presence of CMV 2 weeks previously and were derived from the same source of planting material as those used for mechanical inoculations. Plants were main-

tained in an insect-free greenhouse at 21 to 25°C. Plants were tested using DAS-ELISA for the presence of CMV if symptoms of infection developed and at the termination of the experiment, 14 weeks postinoculation.

RESULTS

Relationship between viruslike symptoms and development of dieback symptoms. In 1991, there was a clear relationship between leaf mosaic/distortion and development of dieback symptoms. Over the 12-week period, 30 of the 50 stems showing leaf symptoms developed dieback symptoms 3 to 4 weeks after the leaf symptoms first appeared. In 1992, however, a correlation was less clear, in that only 9 of the 50 stems showing leaf symptoms developed dieback.

In 1991, stems on 14 of the 50 symptomless control plants (28%) developed dieback and died. In 1992, none of the symptomless control stems developed dieback.

Incidence of CMV in Tonga, Fiji, Vanuatu, and Western Samoa. CMV was detected in kava at all sites where leaf mosaic/distortion symptoms were present in Tonga, Fiji, and Western Samoa and in Vanuatu at four of the five sites where leaf symptoms were found (Table 1). The virus was detected in 60% of the 95 plants showing various viruslike leaf symptoms. Although the virus was detected in 5% of the 155 plants without symptoms, a positive result was duplicated (CMV detected in both leaves) in only one symptomless plant. CMV was detected in 81 of 113 leaves tested that showed mosaic/distortions (72%). The majority of the remaining 32 leaves in which CMV was not detected had become necrotic before testing and this may have affected the result. CMV was also present, but at lower frequencies, in leaves showing other symptoms (1 of 22 crinkled leaves, 3 of 35 mottled leaves, and 3 of 14 chlorotic leaves), but not in any of 6 leaves showing vein banding.

Pathogenicity tests. Kava plants that were infected with CMV by mechanical sap inoculations developed dieback symptoms similar to those observed in the field.

CMV was detected by DAS-ELISA in all 30 mechanically inoculated plants. Twenty-four of the 30 infected plants (80%) developed symptoms of dieback 5 to 12 weeks postinoculation. Forty of 49 inoculated stems on the 30 plants (82%) developed a rapidly spreading blackening and soft rot of stem tissue that destroyed the stem (Fig. 2). In two stems, the rot commenced at the base of the stem; in the rest, the rot originated from upper petioles

Table 1. Presence of cucumber mosaic cucumovirus (CMV) in kava leaf samples collected in Tonga, Fiji, Vanuatu, and Western Samoa as determined by double antibody sandwich–enzyme-linked immunosorbent assay

Location	Active dieback[a]	Viruslike leaf symptoms[b]	Number CMV-postive[c]			
			Symptomatic[d]		Symptomless[e]	
			Leaves[f]	Plants[f]	Leaves[f]	Plants[f]
Vava'u, Tonga						
Leimatu'a	+	+	5	4	0	0
Longomapu	+	+	8	5	0	0
Tu'anuku	+	+	7	4	0	0
Tu'anuku	+	+	1	1	1	1
Tefisi	–	+	4	3	1	1
Longomapu	–	–	–	–	1	1
Tongatapu, Tonga						
Anana	+	+	10	5	1	1
Toloa	+	+	7	5	2	1
Viti Levu, Fiji						
Naqali	+	+	6	4	0	0
Koronivia	+	+	8	5	0	0
Tanna, Vanuatu						
Imaru	+	+	5	3	0	0
Lenaken	+	+	3	2	0	0
North	–	–	–	–	0	0
Efate, Vanuatu						
Epulet	–	–	–	–	0	0
Pangpang	+	+	1	1	1	1
Tagabe	–	+	0	0	0	0
Espiritu Santo, Vanuatu						
Valeteruru	+	+	1	1	0	0
Upolu, Western Samoa						
Taelefaga	–	–	–	–	0	0
Samamea	–	–	–	–	0	0
Samamea	–	–	–	–	0	0
Aufaga	+	+	8	5	1	1
Togitogica	–	–	–	–	0	0
Savai'i, Western Samoa						
Asau	–	–	–	–	0	0
Asau	–	–	–	–	0	0
Vaisala	+	+	7	4	0	0
Vaisala	–	–	–	–	0	0
Neiafu	–	+	2	1	0	0
Neiafu	–	–	–	–	0	0
Aopo	+	+	1	1	0	0
Aopo	+	+	3	3	0	0
Safotu	–	–	–	–	0	0
Total of all sites			87 (46%)	57 (60%)	8 (3%)	7 (5%)

[a] Dark lesions and a soft rot of tissue were visible on one or more stems on one or more plants (+) or were not visible on any stems (–)

[b] Symptoms thought to be associated with infection by CMV (mosaic symptoms and leaf-growth distortions or chlorosis) or other unusual symptoms (crinkling, mottling, or vein banding) were visible on some leaves (+) or were not visible on any leaves examined (–)

[c] Absorbance values were means of two wells per sample. Those greater than twice the mean of five uninfected control leaves that were included on each plate were considered to be virus positive.

[d] Ten leaves with virus symptoms were collected from each site (two from each of five different plants).

[e] Ten asymptomatic leaves were collected from each site (two from each of five different plants).

[f] In each case, the number of leaves presented was taken from the plants indicated in the adjacent right hand column.

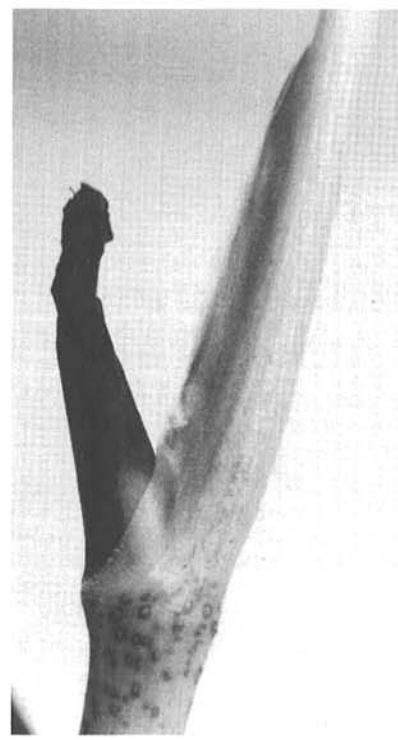

Fig. 2. Black soft rot of the apex of the stem of a young kava plant that developed following mechanical inoculation with cucumber mosaic cucumovirus.

or the stem apex. Leaves that exhibited viruslike symptoms and rotting were usually the first to emerge after inoculation. Twenty of the inoculated plants (67%) were killed by the virus.

Fifteen stems developed wilt symptoms before or during the early stages of the stem rot. In eleven stems (28%), the rot was preceded by development of mosaic symptoms plus leaf-growth distortions on young leaves. In 18 stems (45%), young leaves rotted before they unfolded. Necrosis of leaf margins was the only symptom on leaves preceding stem death in seven stems (18%). Six CMV-positive stems did not die but remained turgid and most leaves remained alive, showing only a general chlorosis and a necrosis of leaf margins. Five of these six stems also developed a black rot at the apex that did not progress. However, at the final destructive testing, necrotic vascular tissue was visible in all six stems.

CMV was not fully systemic in infected plants. The virus was not detected in three inoculated stems although positive results were obtained from other stems on each of these plants. Some new postinoculation growth was not infected with the virus. Of 36 new sprouts over 2 cm in length, produced by 10 of the inoculated plants (Fig. 3), 20 (56%) did not develop symptoms and gave negative DAS-ELISA results. Eight new sprouts developed viruslike symptoms on leaves and tested positive for CMV. However, only seven new stems (three that tested positive and four that showed viruslike leaf symptoms but only gave marginal positive results) rotted and died during the experimental period.

None of the 30 uninoculated control plants tested positive for CMV, nor did they develop viruslike leaf or dieback symptoms.

Aphid transmission. Symptom expression and serological tests indicated that CMV was transmitted by *A. gossypii.* Two of the five plants exposed to aphids previously fed on infected kava developed mosaic. These leaves also tested positive for CMV and the stems developed symptoms of dieback and died. Control plants did not develop leaf or dieback symptoms and gave negative tests for CMV.

DISCUSSION

These studies show that CMV is widely distributed in kava in production areas of the South Pacific and that symptoms of dieback developed on plants that were inoculated with CMV. The association of symptoms of CMV in leaves with development of dieback disease appears to be the same in Tonga, Fiji, Vanuatu, and Western Samoa.

Dieback and necrotic symptoms are not typical of CMV infection in most plant species. The virus usually causes mosaic and stunting of plants. In some situations, however, CMV causes necrotic reactions in plants. In lupin seedlings, for example, CMV can induce a spreading necrosis and plant death (15). The symptomatology of kava dieback is similar to that of lethal necrosis disease of tomato, which is caused by CMV in association with a satellite RNA (7).

Symptoms of dieback in kava following artificial inoculation with CMV were variable. Some plants died rapidly, some wilted prior to dying, and others survived, showing only chlorosis and marginal necrosis on leaves. Dissection of infected stems 2 to 7 weeks after mechanical inoculation with CMV indicated that these three different conditions were all preceded by the development of internal necrosis of vascular tissue and serological tests confirmed that this cell death is linked with the presence of CMV (R I. Davis et al., *unpublished*). In red clover (*Trifolium pratense* L.) infected by bean yellow mosaic virus, enhanced levels of the enzyme peroxidase were associated with virus localization, and lethal systemic necrogenesis was accompanied by a several-fold increase in peroxidase activity (14). It is possible that a virus-induced massive overproduction of this or other enzymes involved in resistance mechanisms may be responsible for the necrotic reaction of kava to infection by CMV.

Dieback may be caused by an interaction between CMV and another virus. There are many reports of specific diseases caused by interactions between different viruses. Tip necrosis disease of passionfruit is caused by CMV in combination with passionfruit woodiness virus (11). Poolpol and Inouye (13) showed that multiplication of CMV in cucumber is enhanced by coinfections with zucchini yellow mosaic virus. The lethal necrotic disease of taro in the Solomon Islands known as Alomae (5) appears to be caused by an interaction between two bacilliform viruses (6). Plants infected with either virus alone developed mild symptoms. In addition to CMV, the authors have observed other spherical, viruslike particles (50 to 70 nm in diameter) in cell nuclei of kava by transmission electron microscopy. The significance of these particles is unknown. dsRNA analyses of kava leaves with viruslike symptoms associated with dieback (10) and of *N. tabacum* mechanically inoculated from at least five different diseased kava plants (M. N. Pearson, *personal communication*) have produced no evidence for the existence of other RNA viruses.

It is possible that two or more strains of CMV might interact to cause kava dieback. Different strains of a given virus can interact to produce more severe disease symptoms. For example, severe maize chlorotic dwarf disease occurred only when two strains of maize chlorotic dwarf virus interacted (4). Each strain alone produced only mild symptoms. More studies are needed to clarify the strain situation of CMV in kava.

Another possibility is that dieback disease of kava results from an interaction between CMV and a nonviral pathogen. For

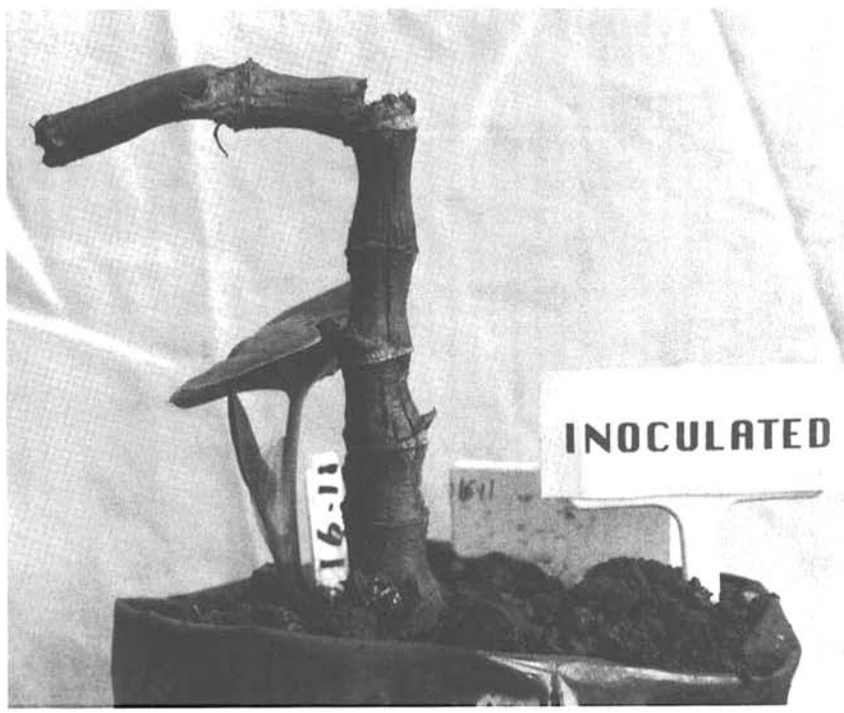

Fig. 3. Regrowth sprout that developed at the base of a young kava stem that was destroyed by cucumber mosaic cucumovirus after mechanical inoculation.

example, CMV is known to interact synergistically with fungal pathogens (1,9). *Colletotrichum* spp. and *Glomerella* spp. have been found to be consistently associated with black lesions on kava stems (2; R. I. Davis et al., *unpublished*). The severity of symptoms induced by these pathogens might be increased in plants that are pre-infected with CMV, resulting in dieback symptoms. However, we have conducted field trials that compared the effects of a range of different biocides on kava dieback. The chemical treatments included the broad spectrum fungicide Prochloraz, which has good activity against *Colletotrichum* spp. and *Glomerella* spp. No consistent statistically significant differences were found between any chemical treatments and dieback disease incidence or severity (R. I. Davis et al., *unpublished*). Pathogenicity tests using CMV on kava plants cultivated under axenic conditions (e.g., grown from tissue culture) would remove the possibility that other pathogens may be involved. So far, attempts to develop a tissue culture system for kava have been unsuccessful. If an attempt succeeded, it could confirm whether CMV alone or CMV in combination with another pathogen or pathogens is responsible for kava dieback.

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