EPPO Datasheet: *Cocadviroid cadangi*

Last updated: 2021-07-28

IDENTITY

Preferred name: *Cocadviroid cadangi*
Taxonomic position: Viruses and viroids: Viroids: Pospiviroidae
Other scientific names: *CCCVd, Coconut cadang-cadang cocadviroid, Coconut cadang-cadang viroid, Palm cadang-cadang viroid*
Common names: cadang cadang, yellow mottling of palms
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EPPO Categorization: A1 list
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EU Categorization: Quarantine pest ((EU) 2019/2072 Annex II A)
EPPO Code: CCCVD0

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Notes on taxonomy and nomenclature

The name Cadang Cadang comes from a local Philippine dialect word meaning death or dying and was used to describe a decline and death of coconut palms. The causal agent remained elusive for a long period until it was demonstrated that the disease is caused by a viroid, which was therefore named coconut cadang cadang viroid (CCCVd, Hanold & Randles, 1991a). Identification of CCCVd led to the development of detection techniques and in particular of molecular hybridization assays. Use of such assays led in turn to the identification of nucleic acid sequences cross-hybridizing with CCCVd probes in a range of plants. Such sequences cross-hybridizing with CCCVd probes have been detected in palm materials from the South-West Pacific region from Indonesia to Vanuatu as well as from South America and Africa (Hanold & Randles, 1991b; Vadamalai *et al.*, 2009). Despite significant efforts, these nucleic acids have never been convincingly shown to be CCCVd and their precise nature and potential pathogenicity remain uncertain. CCCVd cross-hybridizing nucleic acids have also been reported in herbaceous plants. These cross-hybridizing sequences have never been extensively characterized or sequenced and are probably not the same as CCCVd. Over the years, the existence of such CCCVd-related nucleic acids has led to confusion about the host range and geographic distribution of CCCVd.

In Guam, a disease similar to cadang cadang, the 'tinangaja disease' (Boccardo *et al.*, 1981) has been shown to be caused by a different, but related viroid, coconut tinangaja viroid (CTiVd), which has 64% sequence homology with CCCVd (Keese *et al.*, 1988).

HOSTS

All known natural or experimental hosts are in the Palmaceae family. Coconut (*Cocos nucifera*), oil palm (*Elaeis guineensis*) and buri palm (*Corypha elata*) are the only natural hosts of CCCVd for which solid evidence exists (Randles *et al.*, 1980; Zelazny *et al.*, 1982). In the case of coconut and oil palm isolates, the full sequence of the infecting CCCVd variants has been determined (Haseloff *et al.*, 1982; Vadamalai *et al.*, 2006).

In addition to the natural hosts listed above, a range of Palmaceae species have been shown to be experimental hosts of CCCVd, including betel nut palm (*Areca catechu*), Manila palm (*Adonidia merillii*), palmera (*Dypsis lutescens*), and royal palm (*Roystonea regia*) (Anon., 1985; Imperial *et al.*, 1985). There is significant uncertainty about the status of date palm (*Phoenix dactylifera*), Macarthur palm (*Ptychosperma macarthurii*) and, to an even larger extent, anahaw palm *(Livistona rotundifolia)*. These species are listed as experimental hosts in several reviews (Hanold & Randles 1991a for date palm only; Randles and Rodriguez, 2003 and Vadamalai *et al.*, 2017 for date and Macarthur palm) on the basis of the Imperial *et al.* (1985) publication. This later publication is however inconclusive in this respect since it only indicates that '*Current work on the host range of CCCV includes inoculation of other palm species such as date (Phoenix dactylifera), anahaw (Livistona rotundifolia) and Macarthur (Ptychosperma macarthuri) palms.*' without providing any experimental results. As a consequence, it is probably

safer to consider the experimental host status of all three species as not formally established.

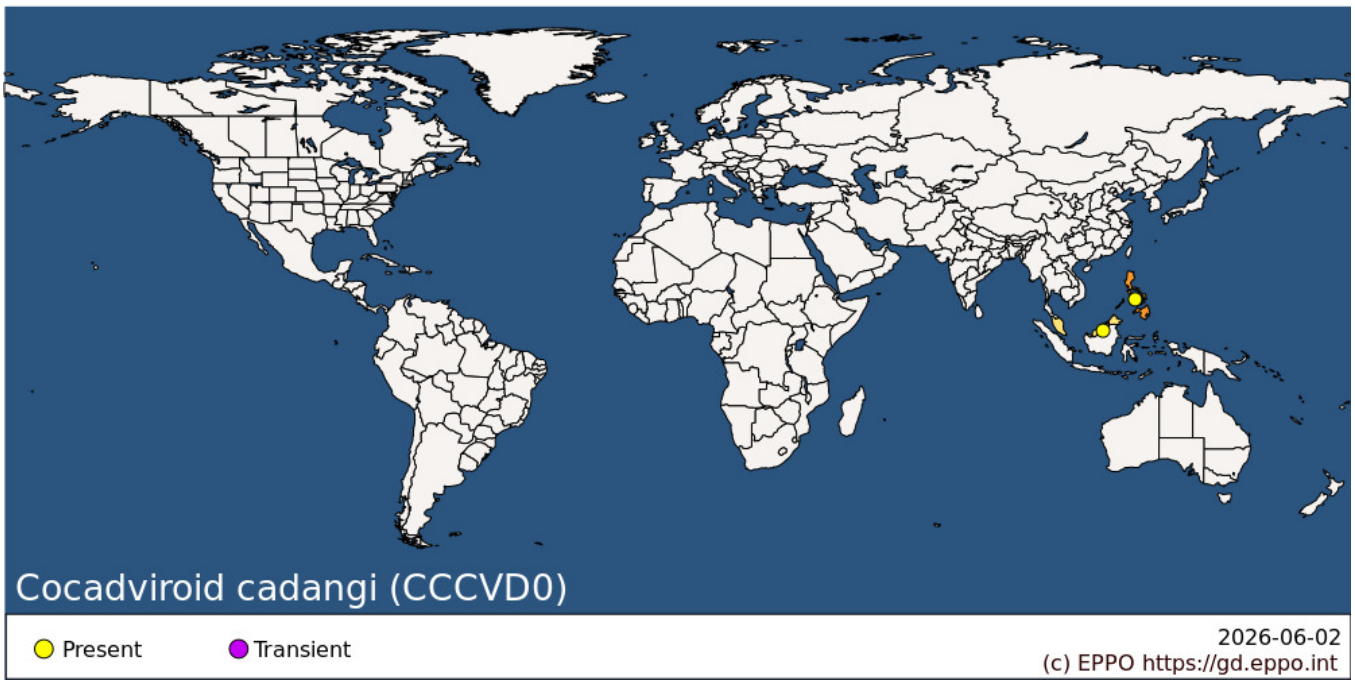
Possibly as a consequence of the existence of CCCVd-cross hybridizing nucleic acids in some palm species, a number of other Palmaceae species have sometimes been reported as hosts of CCCVd, but since it is unclear whether these sequences correspond to CCCVd or to a related but distinct agent, the corresponding Palmaceae species should not be considered as natural hosts of CCCVd until further research is carried out. Similarly, CCCVd-like sequences have also been identified in some symptomless herbaceous monocotyledonous species growing near CCCVd-infected coconut palms (Hanold & Randles, 1991b) but in the absence of confirmatory information, these herbaceous plants cannot be considered as natural hosts.

Host list: *Cocos nucifera, Corypha utan, Elaeis guineensis*

GEOGRAPHICAL DISTRIBUTION

CCCVd is present in Central and Eastern Philippines, with higher incidence in the central islands (Vadamalai *et al.*, 2017 and references therein). It has also been reported in Malaysia.

CCCVd-cross hybridizing sequences have been detected in *Palmaceae* material from the South-West Pacific region from Indonesia to Vanuatu, and also from South America and Africa (Hanold & Randles, 1991b; Hanold and Randles 1998a; Hanold and Randles 1998b; Vadamalai *et al.*, 2009). These sequences are likely not to be CCCVd and CCCVd presence has never been unambiguously confirmed in the corresponding countries.



Asia: Malaysia, Philippines

BIOLOGY

CCCVd spreads systemically within infected plants; this is thought to be via the phloem as with other viroids.

The disease is rarely seen in palms before they commence flowering. After onset the disease incidence in a plantation increases with the age of plants up to 40 years, with the number of diseased palms in the field increasing in a range from 0.1 to 1% per year (Randles *et al.*, 1988; Pacumbaba *et al.*, 1994).

Diseased palms in a field are not clustered and the mode of spread of CCCVd is still unclear, with several routes possibly involved. Above-ground or aerial movement of the viroid could account for new infections arising up to 500 m ahead of the disease front. Transmission of CCCVd to coconut palms at a low rate through pollen and seeds has

been experimentally demonstrated (Pacumbaba *et al.*, 1994). On the contrary, transmission by insects was never confirmed (Zelazny *et al.*, 1982; Randles and Rodriguez, 2003; Vadamalai *et al.*, 2017). Although mechanical transmission by scythes or machetes has not been demonstrated, it cannot be excluded (Randles and Imperial, 1984; Randles *et al.*, 1977; Randles and Rodriguez, 2003).

DETECTION AND IDENTIFICATION

Symptoms

Symptoms of coconut cadang-cadang disease do not usually appear before palm flowering (up to 10 years) and develop through increasing stages of severity.

- - Early stage (lasting 2-4 years): Yellow leaf spots appear water-soaked in reflected light and translucent yellow in transmitted light; inflorescence may be stunted, with necrotic tips; nuts become small and rounded, with characteristic equatorial scars.
- - Medium stage (lasting around 2 years): Leaf spots become numerous, giving the lower two-thirds of the crown a yellowish appearance. Inflorescences become necrotic and infertile; subsequently nut production declines and then ceases. Inflorescence and frond sizes decline.
- - Late stage (lasting around 5 years): Leaf spots become almost confluent; fronds are much reduced in size and number and the whole crown turns yellow/bronze-coloured; leaflets become brittle; the crown and trunk diameter diminish; finally, the palm dies.

Time from appearance of first symptoms to tree death ranges from around 8 to 16 years; this is generally more prolonged in older palms (Zelazny *et al.*, 1982; Hanold & Randles, 1991a).

CCCVd variants associated with symptoms of lamina depletion and a more rapid coconut palm decline (brooming) have been identified (Rodriguez and Randles 1993).

In African oil palm (*E. guineensis*), CCCVd has been associated with symptoms of orange spotting on leaves. Interveinal orange spots appear in young fronds as 2-3 mm long spots of different shape. Spots may coalesce and leaflets in older fronds may show distal necrosis. Palms with orange spotting may be stunted and assume a bronze colour when observed at a distance (Vadamalai *et al.*, 2017). The orange spotting disorder is quite widespread and the possible presence of the viroid in the areas where this disease is present should be considered (Rodriguez *et al.*, 2017).

Morphology

The viroid is presumed to adopt a rod-like structure including partially double-stranded regions separated by small single-stranded loops (Haseloff *et al.*, 1982). A typical central conserved region (CCR) and the terminal conserved hairpin also found in other viroids of the family *Pospiviroidae* are contained in this structure. CCCVd is the representative member of the genus *Cocadviroid*, whose members share a CCR that differs from that of representative viroids classified in the other *Pospiviroidae* genera (slight divergence from the CCR of members of the genera *Pospiviroid* and *Hostuviroid*, and larger divergence from that of members of the genera *Apscaviroid* and *Coleviroid*).

CCCVd has a number of molecular forms; the smallest infectious form is 246 nucleotides (nt) in size, but an additional cytosine may be inserted at position 197. A variable region exists at the right-hand end of the molecule, at which reiteration of either 41, 50 or 55 nt can occur, producing larger forms ranging in size from 287 to 301 nt. The existence of such molecular variants is a feature near specific to CCCVd, as almost all other known viroids have more uniform sizes. Dimeric CCCVd forms with the same sequence variations as their respective monomers are also associated with the disease, but are always isolated in smaller amounts than the corresponding monomers (Randles *et al.*, 1988). A detailed description of the sequences and structures of CCCVd is given by Haseloff *et al.* (1982).

Only the lower molecular weight forms and their dimers are present in fronds of infected palms at the early stage of the disease. Fronds produced subsequently contain progressively larger amounts of the large 287-301 nt forms and

smaller amounts of the 246/247 nt forms. The nature of viroid replication and pathogenesis is not yet understood, but the increase in size of CCCVd with disease progress may be related to the development of more severe symptoms. Alternatively, these molecular changes may be the product of faulty viroid replications, induced by cell metabolic changes (Randles *et al.*, 1988). CCCVd variants associated with lamina depletion and a more rapid coconut palm decline (brooming) have been identified (Rodriguez and Randles 1993). In the oil palm, variants of 246, 270, 293, or 297 nt associated with orange spotting symptoms have been reported, which share more than 90% sequence identity with those from coconut isolates (Vadamalai *et al.*, 2006; Wu *et al.*, 2013).

Detection and inspection methods

Symptomatology is not reliable for disease diagnosis. Indeed, symptoms generally appear only several years after inoculation. Moreover, they may be confused with those caused by coconut tinangaja viroid (CTiVd) or by physiological abiotic disorders (Randles and Rodriguez, 2003). Molecular tests are available for CCCVd detection. Leaves or root tips are used for nucleic acid extraction, with leaves being considered more reliable than roots (Randles and Rodriguez, 2003). CCCVd can be detected by separating nucleic acid preparations by one- or two-dimensional polyacrylamide gel electrophoresis followed by silver staining (Imperial *et al.*, 1985; Hanold & Randles, 1991a), or by transfer to a nylon membrane and molecular hybridization with labelled complementary RNA or DNA probes (Hanold and Randles, 1991b). Labelled probes can also be used in dot-blot assays to detect CCCVd or CCCVd-related sequences (Imperial *et al.*, 1985). However, molecular hybridization assays may not conclusively discriminate between CCCVd and CCCVd-related viroid-like sequences that have been reported in oil palm and in some other *Palmaceae* (Randles *et al.*, 1980; Hanold and Randles 1991a; Hanold *et al.*, 1998a and 1998b). Improved nucleic acids extraction protocols and reverse transcription-polymerase chain reaction (RT-PCR) (Rodriguez and Randles, 1993; Vadamalai *et al.*, 2006; Roslan *et al.*, 2016), ribonuclease protection assay (RPA) (Vadamalai *et al.*, 2009) and reverse transcription loop-mediated isothermal amplification (RT-LAMP) (Thanarajoo, 2014, Madihah *et al.*, 2020) have been developed for the specific and sensitive detection of CCCVd. RT-PCR detection methods allowing discrimination of CCCVd from CTiVd have also been developed (Hodgson *et al.*, 1998). Cloning and sequencing of RT-PCR amplification products is needed for the detection of specific viroid variants (Vadamalai *et al.*, 2006; Wu *et al.*, 2003). Spectral screening using a hyperspectral spectroradiometer has been recently tested as a potential alternative detection method in oil palm seedlings (Golhani *et al.*, 2019).

PATHWAYS FOR MOVEMENT

Trade in plants for planting, including seeds, provides the main pathway for the long distance movement of CCCVd within and between countries (EFSA, 2017). Natural spread of CCCVd and the rate of disease increase vary depending on the site, but incidence and distribution patterns of diseased plants did not help to identify a possible vector (Randles and Rodriguez, 2003). CCCVd can be transmitted by seeds or pollen with low efficiency (Pacumbaba *et al.*, 1994), providing possible pathways for local, short distance movement.

PEST SIGNIFICANCE

Economic impact

The disease results in the premature decline and death of coconut palms (Hanold and Randles, 1991a). In the Philippines, since the first reports of the disease at the beginning of the last century more than 40 million trees have been infected and killed. Disease incidence was estimated at 2.5 million palms in 2006 and about 700,000 in 2012-2013 over an area of about 650,000 ha (Philippine Coconut Authority, 2014 cited by Rodriguez *et al.*, 2017). Stunting and 25% to 50% yield reduction compared to neighbouring healthy plants have been reported in infected oil palms affected by the orange spotting disorder, although a correlation between symptoms' severity and the infecting CCCVd variants has yet to be shown (Rodriguez *et al.*, 2017 and references therein). The impact of the coconut lamina-depletion (brooming) disease, associated with CCCVd variants with a specific mutation, has also not been estimated yet but, given the severity of this symptom, the impact is expected to be in the same range or higher than for typical CCCVd infection.

Control

Besides exclusion of infected plants, no control measures exist for CCCVd in the field. Specific control recommendations cannot be developed until the epidemiology of CCCVd is more clearly understood. Eradication of diseased plants has been unsuccessful in the Philippines, which is likely to be due to the difficulties of early diagnosis. However, roguing of infected plants and replanting healthy ones succeeded in limiting yield losses in the infested areas (Randles 1987). Attempts to identify genetic resistance in *C. nucifera* were unsuccessful (Orolfo *et al.*, 2000), although existence of field resistance in some populations is not excluded (Randles and Rodriguez, 2003).

Phytosanitary risk

In the EPPO region, there are no clear ecoclimatic limitations besides those applying to the host (EFSA, 2017). CCCVd has the potential to cause significant damage in coconut and in oil palm. The potential for damage in other Palmaceae species (whether they have been found to be experimental hosts or not) and that are grown as outdoor or indoor ornamentals is less clear. Since the spread mechanism is unknown, there is uncertainty about how and with what efficiency CCCVd would be able to spread in European palms.

PHYTOSANITARY MEASURES

Appropriate phytosanitary measures to import palm plants for planting (including seeds and embryo cultures) into the EPPO region could require that these plants are produced in a pest free area, or shown to be free from CCCVd by appropriate molecular diagnostic methods.

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ACKNOWLEDGEMENTS

This datasheet was extensively revised in 2021 by Francesco di Serio and Thierry Candresse. Their valuable contribution is gratefully acknowledged.

How to cite this datasheet?

EPPO (2026) *Cocadviroid cadangi*. EPPO datasheets on pests recommended for regulation. Available online. https://gd.eppo.int

Datasheet history

This datasheet was first published in 1997 in the second edition of 'Quarantine Pests for Europe', and revised in 2021. It is now maintained in an electronic format in the EPPO Global Database. The sections on 'Identity', ‘Hosts’, and 'Geographical distribution' are automatically updated from the database. For other sections, the date of last revision is indicated on the right.

CABI/EPPO (1997) *Quarantine Pests for Europe (2nd edition).* CABI, Wallingford (GB).

Co-funded by the European Union