LETHAL YELLOWING OF COCONUT

Introduction

Lethal yellowing (LY) disease is not only destructive on coconut but on at least 30 other species of palms. It is particularly aggressive on the tall varieties grown almost exclusively in the Caribbean region prior to the 1970s-80s, when dwarf varieties began to be introduced as replacements for the talls devastated by LY, especially in Jamaica, Cuba and the Dominican Republic. The economic impact is multifaceted, in keeping with the many uses of the plant. A major commodity of international trade is copra, the dried endosperm, which yields oil that is extensively used in the production of soap, margarine, cooking oil, cosmetics and a variety of processed foods, ice cream and pastries. Coconut is unrivalled in the versatility of its use to humankind.

Identity

Authority : Nutman & Roberts, (1955)
Classification
- Kingdom : Procaryotae
- Phylum : Tenericutes
- Class : Molicutes
- Family : Spiroplasmataceae
- Common names : Lethal yellowing disease, awka disease, Cape St. Paul wilt, kaincope disease, Kribi disease, pudrición del cogollo.
- Role : Pest

Note on classification

Phytoplasmas (formerly mycoplasma-like-organisms, MLO) are prokaryotes lacking a true cell wall. Each cell is bounded by a trilaminar unit membrane ca. 8-10 nm thick. They are limited to the phloem of infected plants, are transmitted in nature by phloem-feeding leafhoppers, and may be experimentally transmitted by dodder and by grafting. They are sensitive to heat treatment in diseased plants and insect vectors and treatment of diseased plants with tetracycline antibiotic results in remission of symptoms. Phytoplasmas have not yet been cultivated *in vitro* and, as a consequence, early studies to identify the causal agent of the disease utilized electron microscopy to demonstrate the presence of the bacterium in host tissue and in the vector if it was known. Symptom remission after applying tetracycline antibiotics was associated with the absence of phytoplasmas in tissues that were formerly positive.

In recent times DNA hybridisation analysis using cloned DNA probes derived from various phytoplasmas have permitted the study of genetic relationships among them and has led to the recognition of 14 groups of the bacteria. The LY pathogen has been placed in group IV, subgroup IV-A. The members of any Group share extensive DNA sequence homology with each other and are distinct from phytoplasmas in another group. Genotype differentiation among members in a Group is achieved through restriction fragment length polymorphism (RFLP) analysis of each phytoplasma's DNA by using selected DNA probes.

Difficulties in performing these analyses because of low concentrations of phytoplasma DNA in host cells are overcome by the application of PCR assay which achieves sensitive detection and differentiation of phytoplasma strains. PCR has been

developed for amplifying 16S rDNA and 16S rRNA sequences from several phytoplasmas (including the LY pathogen) from infected plants and RFLP analysis of the amplified 16S rDNA used in their differentiation and classification (Lee et al., 1994). Applying these techniques, Harrison et al., (1994) demonstrated a close genetic relationship between LY isolates from Florida and "lethal disease"(LD) isolates from Tanzania, although the data indicated that the two pathogens are not genetically identical.

Signs & Symptoms

Premature nut-shedding

One of the early and striking symptoms of LY is the premature shedding of nuts. This usually occurs in a unilateral pattern, with the fall of all the nuts from one side of the tree. This is followed by a general shedding lasting 2-12 weeks; there is sometimes a break in the nutfall of about 2 weeks. Water nuts (from the middle of the crown) generally fall first, then younger nuts and finally mature nuts. The nuts exhibit a dark coloured area under the bracts that extends into the husk, the nut and occasionally the endosperm. The water tends to be slimy and lacks taste and the fallen nuts decay rapidly. This characteristic pattern of nutfall distinguishes the LY situation from other causes of shedding such as rat damage, drought and natural thinning (Carter et al., 1965).

Necrotic inflorescences

The presence of necrotic inflorescences is diagnostic for LY disease. Brown-black necrosis may first appear on the tips of the spikelets of male flowers on a newly opened, apparently normal inflorescence whose colour is creamy-gold. The necrosis, involving a few or all male spikelets, moves into the female flowers and in 4 weeks engulfs the whole inflorescence. As the disease progresses, immature spathes may open to reveal completely necrotic inflorescences. However, it is more usual for spathes to change from green to brown without opening. The collapse of spathes accelerates, with each appearing black as it emerges. The bases are rotted and have a markedly unpleasant smell.

Leaf chlorosis

Leaf chlorosis (yellowing) due to LY occurs only on the "Jamaica Tall variety"; on infected yellow and green cultivars of Malayan Dwarf the leaves are brownish-bronze in colour. Yellowing of coconut leaves may result from normal abscission; from plants growing in swampy land or from growth in soils of high calcium content.

Yellowing symptoms normally appear first on older leaves and progress toward the younger leaves near the middle of the crown. Sometimes a young leaf turns yellow, out of sequence and, thus, stands out among the normal – looking leaves. This is termed a "flag leaf" and, when present, is presumptive evidence of LY. The leaflets near the tips of fronds turn yellow at the stage of the disease just after nut shedding. In about 2 weeks the older, chlorotic leaves turn light brown, some tending to hang around the trunk (Fig.1), others abscising and falling to the ground. Desiccated fronds are readily pulled off the tree.

Bud death

The leaves in the upper one-third of the tree may remain upright; they turn yellow, then brown, until the bud dies. The death of the bud is terminal for the tree and is expressed as a collapse of the youngest, still unfolded, leaf in the desiccating crown. Within a few days the whole area rots from the activity of secondary microorganisms and insects. The entire crown breaks off, falls to the ground, leaving what is known as the "telephone pole" remains of the dead tree (Fig.2). Tree death occurs 3-6 months after the first symptoms of nut shedding. A cross-sectional cut through the trunk just below the dead bud reveals a foul-smelling area of decay (Fig. 4).
In addition to differences in colour, the leaves of infected Malayan Dwarfs are folded at the midvein and exhibit marked wilt symptoms.

Morphology

Observations on the LY phytoplasma from Jamaica showed it to be typically pleomorphic in shape, displaying ovoid, elongate and filamentous forms (Plavsic-Banjac et al., 1972). The bodies were bounded by a three-layered membrane ca. 10nm thick. Some of the elongated bodies measured up to 2μm in length.
The average size of ovoid forms of phytoplasmas is 0.3 to 0.8 μm in diameter. They are the smallest bacteria capable of self-reproduction, with one of the smallest genomes among the prokaryotes. Since they do not have a cell wall and thus lack the ability to synthesize peptidoglycan precursors, they are penicillin resistant. They are sensitive to tetracycline antibiotics which, when applied to diseased plants, cause remission of symptoms. If treatment ceases, disease symptoms reappear.

Biology & Epidemiology

LY is spread by the plant hopper, *Myndus crudus* (Fig.3). Transmission studies in Florida demonstrated that vector insects allowed to feed on diseased trees were able to infect caged healthy plants. Subsequently the phytoplasma was demonstrated in phloem cells of the host by electron microscopy (Denselman, 1981).
M. crudus requires an acquisition period of several hours or days to become infective. The phytoplasma is acquired more effectively when it is fed on young leaves rather than on older ones. Before the pathogen can be transmitted there is a 10-45 day incubation period which is shortest at 30C and longest at 10C. The bacterium multiplies first in the vector's intestinal cells, moves into the haemolymph, the internal organs and finally into the brain and salivary glands. Actual transmission occurs when the bacterial concentration reaches a certain level and is maintained throughout the life of the insect. Both nymphal and adult stages acquire the phytoplasma efficiently, but there is no passage from the adult to the eggs.
Additional, circumstantial evidence supporting *M. crudus* as the vector of LY is:

1) high incidence of LY is always associated with high numbers of *M. crudus* on the host palm
2) reduction in numbers of *M. crudus* is accompanied by a reduction in disease incidence
3) coconuts (and other palms) are favoured hosts of the insect
4) the phytoplasma pathogen has been detected by DNA - based methods in specimens of *M. crudus* that have been allowed to feed on diseased coconut palms.

5) similar evidence implicating any other candidate vector of LY disease does not exist.

Dispersal / vectors

There has been considerable speculation on how the pathogen is disseminated over long distances. This is due to a lack of evidence from detailed epidemiological studies. In their description of LY symptoms found in Jamaica, Carter et al. (1965) observed no abnormality in plants developed from nuts shed by infected trees. Roca de Doyle (2001) associates the appearance of the disease in certain areas in Honduras with expansion in the tourist industry. She suggests that the vector could have been introduced on lawn grasses (on which the nymphal stages of the vector feed) brought in as landscaping plants. On the other hand, areas designated as off-limits to tourist development remained disease-free for several years despite being close to well-developed tourist centres. She further noted the association between the LY epiphytotic in Mexico and the development of tourism. Another possibility was the arrival of the vector on strong wind currents or even hurricanes, e.g., Mitch that devastated the country in 1998.

Management

LY is an important disease from two aspects – its impact on both the coconut industry and the landscaping industry. Increasingly, its management is being approached by employing integrated strategies to reduce the incidence on coconut as well as on ornamental plants in the landscape. In the absence of solid epidemiological data, it can be assumed that reservoirs of the phytoplasma and its vector in susceptible palms and grasses in close proximity to or within coconut plantations could lead to higher disease levels in the coconut crop. The epidemic losses that occurred in ornamental plants after LY moved from the Keys into 'mainland' Florida was strong evidence that the reverse is possible.

Resistant varieties

Management of LY in coconut has been mainly through the use of resistant varieties, primarily various forms of Malayan dwarf and the Maypan hybrid. This strategy has been successful in Jamaica and other islands in the Western Caribbean since the 1960s. Certified seed is available from Jamaica and Costa Rica.

Antibiotic application

The known sensitivity of phytoplasmas to oxytetracycline drugs has been utilized in the development of antibiotic treatments involving direct infection into the trunks of diseased palms. The treatment has to be repeated every three months to ensure remission of symptoms. The technique is only feasible when applied to small numbers of selected coconut trees or favoured ornamental palms. Another, very likely, impediment to long-term use of antibiotics would be the development of resistant strains of the pathogen.

Vector management

Experiments on vector management by using insecticides have shown that numbers can be significantly reduced. However, this strategy is viewed as being too costly, damaging to the environment and likely to lead to resistance in vector populations (Howard and Harrison, 1998). An important factor is the inability of small farmers in many Caribbean countries to afford either the chemical or the equipment costs, especially where the target pest is located at a height of 10 m. Insecticide application may be feasible as a component in an IPM programme, which includes a bio control agent. Natural enemies in the form of predators, parasitoids and fungi are known, but investigative work is still to be conducted.

Another approach to managing the LY vector is through the use of ground cover species on which the vector is unable to develop. Howard and Harrison (1998) reported on studies conducted in Florida that have identified dicotyledonous species that do not support the development of *M. crudus*. Several grass species that are non-hosts were found to be unsuitable for use as turf grasses. The use of non-host ground covers is viewed as a strategy best suited for an IPM programme, with the principal component being the introduction of resistant varieties of coconut.

Host Notes

Cocos nucifera; 34 palm species, including *Veitchia merrillii; Phoenix Dactylifera; Pritchardia pacifica; Caryota mitis*.

There are differences in susceptibility to LY among members of the Palmaceae (Simone, 1993). Further, differences in susceptibility to LY are also exhibited by different cultivars of coconut, *Cocos nucifera*, (Howard & Harrison, 1998). The most susceptible is 'Jamaica/Atlantic Tall', recognized as being significantly more so than the 'Pacific/Panama Tall'. Several dwarf cultivars - Malayan green dwarf, Malayan yellow dwarf, Fiji dwarf, Chowghat green dwarf and others - have shown high levels of resistance. However, this resistance is variable, from 95% in some locations to 50% in others. In Jamaica, the Malayan dwarf and the Maypan hybrid (Malayan dwarf x Panama tall) were selected for the replanting programme, established in the 1960s to manage LY disease. Within the last few years Malayan dwarf and Maypan hybrid trees in Jamaica have been declining from LY – an indication of possible new races of the Phytoplasma appearing in one of the original sites of epidemic losses to the disease.

Distribution

Early recognition of LY came from the Cayman Islands in 1834, Jamaica and Cuba in the 1870s, Haiti (1920), Dominican Republic (1925), the Bahamas (1946) and, in the last 50 years, it has expanded its range to the Florida Keys, lower eastern and western areas of Florida, the Rio Grande Valley in Texas and Mexico, from where it is threatening Central America (Simone, 1993). It has arrived in Honduras with devastating effect, destroying 70% of that country's original coconut population in a 6-year rampage beginning in 1995 (Roca de Doyle, 2001). The disease is present in West Africa (Cameroon, Nigeria, Ghana and Togo) in Tanzania (East Africa) and in the Indian States of Kerala and Karnataka.

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Web Resources:-

http://www.ftld.ufl.edu/lyfacts.htm
http://www.ecoport.org/EP.exe$EntNameSrc

Fig. 1: Coconut plant showing advanced symptoms of lethal yellowing.

Fig. 2: Field of coconut showing trees in various stages of decline due to lethal yellowing disease.

Fig. 3: ***Myndus crudus***, vector of lethal yellowing phytoplasma.

Fig. 4: Cross section of coconut stem showing internal decay associated with death of the terminal bud due to lethal yellowing.