

Full Length Research Paper

Review of scientific research into the Cape Saint Paul Wilt Disease (CSPWD) of coconut in Ghana

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A comprehensive review of the literature on scientific research into the Cape Saint Paul Wilt Disease (CSPWD) of coconut in Ghana was carried out for a three-fold objective: (1) to highlight past scientific research efforts and achievements; (2) so as to provide a reasonably objective basis for evaluating current research efforts at containing the menace of the disease in Ghana, with the view to making an informed proposition on the future course of CSPWD research in the country and finally; (3) to provide a succinct 'one-stop' reference information on all scientific research works carried out since the first incidence of the disease in the country in 1932. Farm hygiene, by way of felling infected coconut palms immediately upon detection is useful only to slow the spread of the disease, but not to eliminate it. The search for phytoplasma vectors has yet to provide any empirical evidence of the existence of such pestiferous vectors in Ghana. Even though, one indigenous grass species, *Desmodium adscendeus* has been found to harbour lethal yellowing phytoplasma, no causal evidence has been established between the presence of this grass and the incidence of CSPWD in coconut farms. Only breeding of coconut germplasms (seed nuts and pollen) for CSPWD resistance has thus far yielded some apparently tolerant coconut varieties and their hybrids. As a result, the profitability of coconut cultivation in Ghana, and its competitive position on the vegetable oils market, will largely depend on the progress made in research, particularly in breeding research.

Key words: Scientific review, epidemiology, Cape Saint Paul Wilt Disease (CSPWD), phytoplasma, breeding research, coconut germplasm.

INTRODUCTION

The coconut palm (*Cocos nucifera* L) exists throughout the humid inter-tropical zone, where it is mostly grown along the coasts. It is planted up to a height of 1 000 m above sea level. Some of the plants can live for more than a century. The economic life span is estimated at fifty years (Bourdeix et al., 2005).

The exact origin of the coconut palm is unknown, but the plant is believed to be disseminated by fruits floating on sea currents and much later, by human travel and migration. There are several varieties and hybrids of coconut in cultivation across the world. The botanist Hugh Harries explained the diversity of coconut varieties as resulting from an INTROGRESSION (repeated crossing and backcrossing) between the ancestral "Niu kafa" and the modern "Niu vai" varieties. His theory is based on observation of the coconut fruit shape and composition, disease resistance, windstorm tolerance,

speed of germination and geographical distribution (Harries, 1995).

The coconut palm consists of a trunk/stem topped with a broad crown of fronds. In the axil of each frond, there is usually an inflorescence that develops into a bunch loaded with coconut fruits. It is possible to distinguish between tall and dwarf varieties, by the distance between successive leaf scars. In tall palms, the distance between two leaf scars is more than 5 cm, whereas it does not exceed 2.5 cm in dwarf palms. The stem, fronds, and flowers all grow from a single bud. The bud functions continuously, so the palm never stops growing, until it dies (Bourdeix et al., 2005)!

The coconut palm is believed to have been brought to West Africa by the Portuguese missionaries over 500 years ago. The early missionaries introduced the crop into the south-eastern part of Ghana (Keta area). The

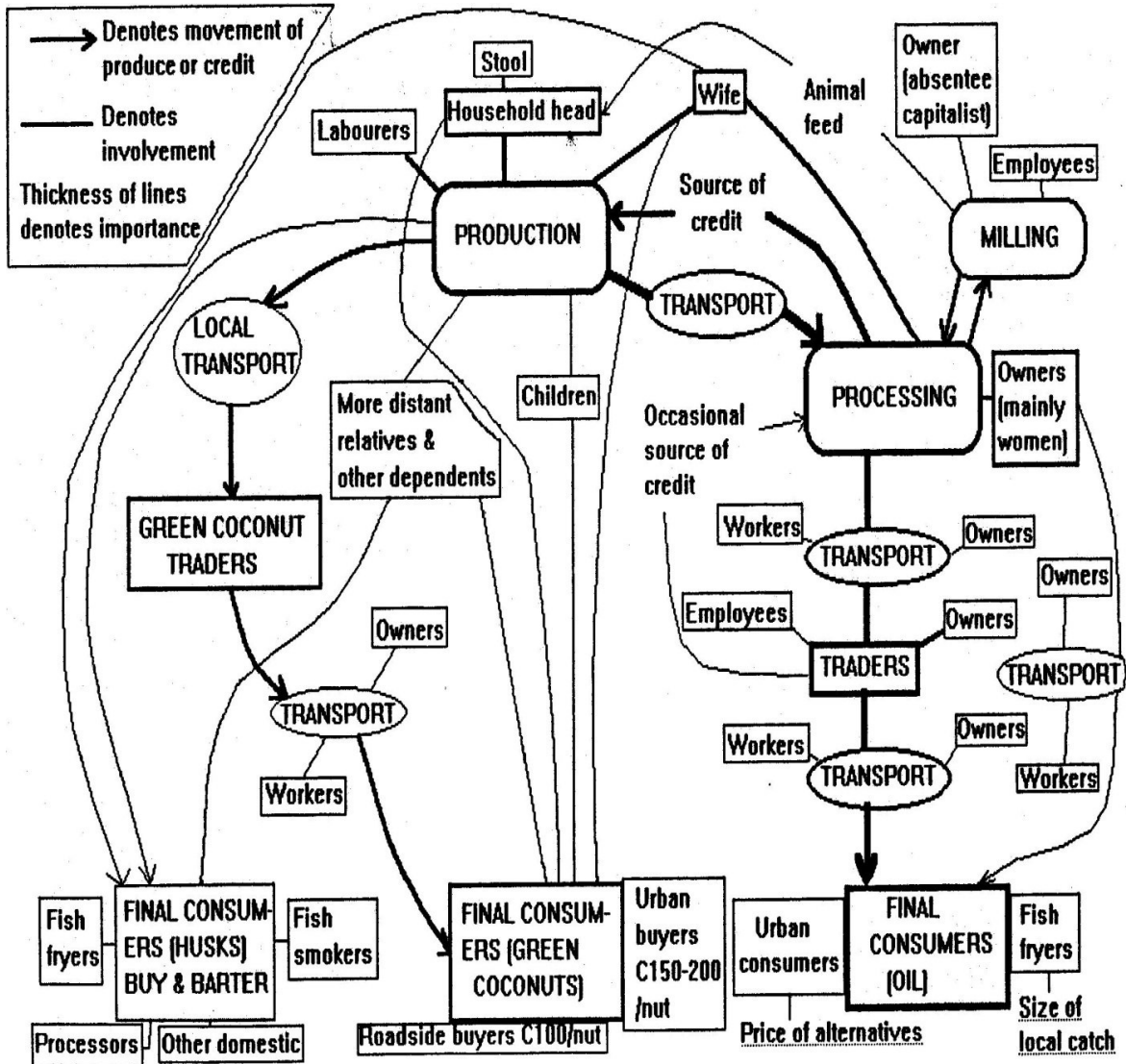


Figure 1a. Diagram of the coconut economy (Credit: Dr. S.K. Dery; CSIR-OPRI, Ghana.)

then British colonial government in Ghana showed interest in the crop, and in 1910 promoted its large-scale cultivation along the coastal strip of the country (Figure 1a).

Coconut thrived at this part of the country whose soil type (mostly sandy soil) did not appear to be very suitable for other cash crops, and therefore, assumed an unprecedented importance in the economy of this area. It is cultivated both as a cash crop and as a food crop. Its cultivation, processing, and marketing, offer employment opportunities to disadvantaged groups such as the landless poor, street children (sale of fresh drinking fruits), and rural women.

World coconut plantings amount to more than a billion palms, two-thirds of which are more than 60 years old. Yields in the ageing plantations are in decline. Serious

diseases such as Lethal Yellowing (Cape Saint Paul Wilt Disease) are devastating the coconut palms in some regions (Bourdeix et al., 2005).

Lethal yellowing is a disease that affects nearly 40 species of palm world-wide. Many of the disease symptoms are caused by plant pathogens called phytoplasmas. Phytoplasmas are wall-less and phloem-limited organisms that lie between bacteria and viruses. They can neither be cultured in-vitro nor viewed by light microscopy (Dollet et al., 2008). They are transmitted by insect vectors. Known vectors of phytoplasma diseases are species of leafhoppers, plant hoppers and psyllids (Tsai, 1979).

A planthopper, *Myndus crudus* van Duzee (Homoptera: Cixiidae), had been shown to be the vector of lethal yellowing disease in Florida (Howard et al.,

1983). *M. crudus* was also found to transmit the palm lethal disease responsible for the decline of the Christmas palm *Veitchia* sp. (Howard et al., 1980). *Myndus* species has been found to be the vector of foliar decay of coconut in Vanuatu (Julia, 1982). Yet, to date, the vector(s) of the phytoplasma responsible for coconut lethal yellowing-like disease in West Africa is unknown (Philippe, 2007).

In Ghana, the first incidence of the lethal yellowing-like disease occurred in 1932, at Cape St. Paul, Woe, in the Volta Region, hence the local name CSPWD. It was detected later in 1964, at Cape 3 Point in the Western Region, and by 1983, the disease had reached Ayensudo in the Central Region of Ghana. New reports of CSPWD incidence in parts of the Eastern Region of Ghana have emerged over the past two years (since 2008).

Several thousands of hectares of coconut plantings in the Western, Central, and Volta Regions, the three main coconut producing regions in Ghana, have since been devastated by the disease and it is still spreading (Ofori and Nkansah-Poku, 1995; Dery and Arthur, 1996).

The disease presently, has no cure. Limited prevention is achieved through breeding coconut germplasms for CSPWD-resistance, and by the adoption of farm hygiene. Control of disease spread is by felling of diseased palms, upon confirmation of visual diagnosis by PCR analysis. Periodic insecticidal hot-fogging is used to reduce the population of putative vectors on coconut plantations.

The history, occurrence, and epidemiology of the disease in Ghana have widely been reported by Addison (1974, 1978), Ofori and Nkansah-Poku (1995), and Dery et al. (1999). Attempts by Researchers to find a cure to the disease have been going on for decades. For instance, in 1942, investigations into the nature, causes and control of the disease, was initiated by the Crops Research Institute (CRI) of Ghana. But there was no breakthrough at that time (Chona and Andoh, 1970).

Presently, a new hybrid developed from a genetic cross between the Sri Lanka Green Dwarf (SGD) and the Vanuatu Tall (VTT) parents have under experimental conditions, proven more resistant to the CSPWD than all hybrids tested so far. The superiority of this hybrid is established, both by resistance values from a predictive additive genetic model (GLM) and observed/ actual field data on CSPWD tolerance/resistance. The hybrid has subsequently been released to coconut farmers for large scale cultivation as far back as the year 2008.

This paper reviews the relevant literature on previous scientific research attempts at containing the CSPWD, evaluates the literature on current research strategies adopted to contain the menace, and finally proposes a way forward for scientific research on the epidemiology of Cape St. Paul Wilt Disease of coconut in Ghana. The socioeconomic cost of the disease in Ghana is also

illustrated.

THE ECONOMIC COST OF CSPWD IN GHANA

The coconut palm occupies an important place in the coastal belt of Ghana. Its cultivation, processing and marketing, offers employment opportunities to disadvantaged groups such as the landless poor, street children (sale of fresh drinking fruits) and rural women.

CSPWD completes its cycle within six months from the date of manifestation of first symptoms. Under healthy physiological and normal growth conditions, a mature coconut palm produces one inflorescence per month. A fruiting diseased (LYD) palm aborts on the average 8 buttons per opened inflorescence per month, depending on the stage and severity of the disease.

This will amount to 48 coconut buttons per palm per CSPWD cycle, all things being equal. The standard standing crop (number of palms per hectare) is 160 coconut palms per hectare (i.e., for MYD x VTT hybrids). At a conservative CSPWD infection rate of 5%, 8 fruiting palms per hectare will have been affected, giving a total of 384 coconut buttons per CSPWD cycle (within 6 months). A mature fresh coconut fruit costs approximately GHC 0.3. Therefore, 384 mature fresh fruits will cost $GHC\ 0.3 \times 384 = GHC\ 115.20$.

The standard standing crop (number of palms per hectare) is 160 coconut palms per hectare (that is, for MYD x VTT hybrids). At a conservative CSPWD infection rate of 5%, 8 fruiting palms per hectare will have been affected, giving a total of 384 coconut buttons per CSPWD cycle (within 6 months). The computation for various CSPWD infection rates/levels is presented in Table 1.

Socioeconomic calculation of loss to CSPWD in Ghana

Given that about 4.2% of Ghana's population (estimated at 22 million) depends on coconut for their livelihood (Adams et al., 1996), and assuming half or 2.1% are direct coconut farmers, each cultivating at least 1 ha coconut farm, the computed financial loss will be 462,000 farmers x GHC 1,152 per ha per CSPWD cycle. That is a whopping GHC 532,224,000. Incredible!

The socioeconomic cost is rather dire, considering the fact that loss of coconut to CSPWD is not just the fruits or their derivatives but also loss in terms of labour cost and man-hours employed in the production process. Figure 1b illustrates the complex interlocking web of the coconut industry.

CSPWD CONTAINMENT STRATEGIES

The CSPWD integrated management strategy adopted,

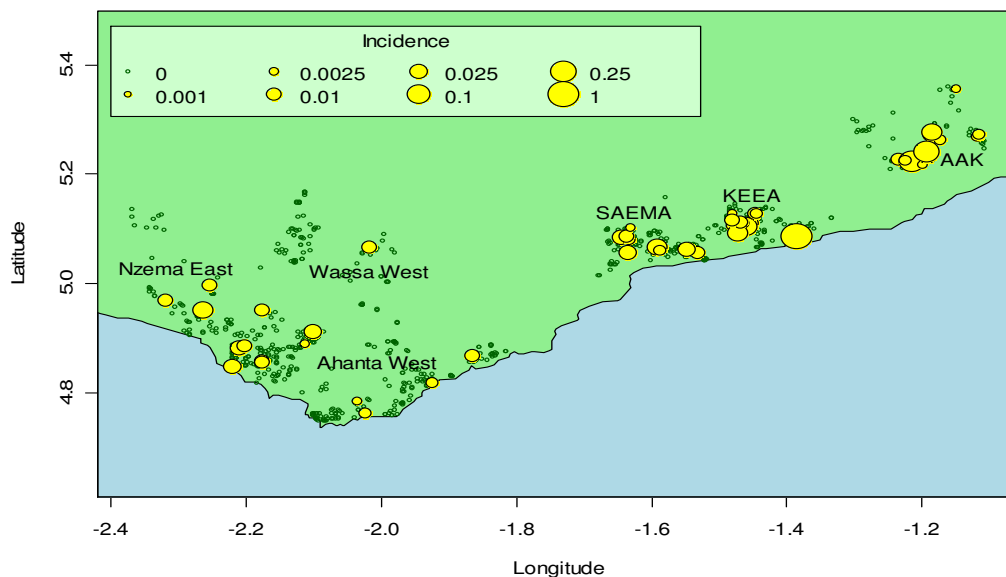


Figure 1b. Geographical distribution of CSPWD of coconut in the Western and Central regions of Ghana as at December, 2008. Each yellow circle represents an infected farm.

Table 1. Hypothetical coconut revenue loss to CSPWD at various infection rates.

Rate of Infection (%)	Number of diseased palms per hectare	Number of aborted buttons per hectare	Market value of fruits at GHC 0.3 per fruit
5	8	384	115.20
10	16	768	230.40
20	32	1,536	460.80
40	64	3,072	921.60
50	80	3,840	1,152.00

Ghana cedi: American dollar exchange rate (~ GHC 1.4: \$ 1) as at 31st December, 2010.

utilizes host-plant resistance, together with CSPWD vector control, and farm hygiene in harmony with the natural regulatory factors of the ecosystem. CSPWD presently has no cure. The containment strategies employed in the fight so far are:

1. Breeding coconut germplasms (seed nuts and pollen) for CSPWD resistance;
2. Adoption of strict farm hygiene/phytosanitation;
3. Putative vector survey, identification, population studies, and control and;
4. Search for secondary or alternative hosts of CSPWD phytoplasma.

Epidemiological studies on CSPWD of coconut

Lack of quantitative historical information on biological events associated with host plants and their pathogens, hinder our ability to make reliable forecasts of disease potential, and estimate crop loss associated with plant

pathogens (Chatterjee, 1997). In this regard, epidemiological studies are of major importance in understanding the determinants of plant diseases in order to control the risk of spread (Bonnot et al., 2009).

The earliest recorded study on the nature, causes, and control of the disease was carried out in the Volta Region by the Ministry of Agriculture (MoA) and the Crops Research Institute (CRI) in 1942 and 1965 (Chona and Andoh, 1970). Other areas of investigations were association of disease with soil type, salinity, lethal substances in soil, bacteria, fungi, and nematodes. The history, occurrence and epidemiology of the disease in Ghana have widely been reported by Johnson and Harries (1976), Ofori and Nkansah-Poku (1997), and Dery et al. (1999).

The symptoms of the LYD in Ghana are: pre-mature nut drop with or without yellowing of fronds. This is followed by progressive yellowing or, sometimes browning of the crown from the older fronds below towards the younger fronds upwards. Eventually, the whole crown turns yellow, dries up and then falls off,

leaving only the stipe standing. The symptoms may vary according to coconut variety. In some of them, including the MYD x VTT hybrids, the leaves may take a bronze colour instead of yellow (Dery et al., 2008)

Breeding coconut germplasms for CSPWD resistance

The cultivation of resistant coconut varieties has long been identified as the best way of controlling coconut lethal yellowing-like disease and lethal yellowing diseases in general (Dery et al., 1995, 2000, 2005; Dery and Arthur, 1996; Harries, 1995; Mariau et al., 1996). The first documented resistance screening trials were set up in 1956/1957 by the Crops Research Institute of Ghana (Chona and Adansi, 1970) using Malayan Green Dwarf and Malayan Yellow Dwarf varieties imported from Jamaica. The local West African Tall variety was planted as control in these trials. All three varieties succumbed to the disease.

In 1977, the Crops Research Institute again set up a resistance screening trials at Cape Three Point, in the Western Region of Ghana. This trial too, was wiped out by the disease over time. Other palm species included in the trial such as *Phoenix canariensis* and *Veitchia merrilli* however, survived.

A more concerted effort at finding a resistant coconut progeny to CSPWD started in 1981 with the establishment of the France-Ghana-Cote d'Ivoire Coconut Project. With funding from the French government, two seed gardens were set up at Ainyinase and Bamiankor in the Western Region to produce hybrids. Seven trial plots were also established to screen 27 coconut progenies/ecotypes for resistance to CSPWD by the Ministry of Food and Agriculture (MoFA). Four of the seven trials were planted in a region already devastated by the disease: Discove, Akwidae, Cape Three Points, and Princess Town. The other three trials at Agona Junction, Dadwen and Axim were planted in a disease-free zone, towards which the disease was spreading. Each trial tested Dwarf ecotypes, Tall ecotypes and Dwarf x Tall and/or Tall x Tall hybrids, using the West African Tall as the control. In all, there were 5 Dwarf varieties, 6 Tall, and 16 hybrids.

In 1993, MoFA initiated the international EC STD III Coconut Lethal Yellowing Project. In conjunction with the Oil Palm Research Institute of Ghana (OPRI), the scope of the research was broadened to include disease epidemiology studies, and disease containment studies. Later, with collaboration of the Department of Crop Science, University of Ghana, the development of disease diagnostic techniques as well as DNA probes which could be used for screening ecotypes and insect vectors was initiated.

The EC STD III Coconut Lethal Yellowing Project

increased the number of coconut ecotypes under test for CSPWD resistance to 39. The success of this project led to on-farm trials with two most promising hybrids: VTT x SGD and VTT x MYD, in the Western, Central, and Volta Regions in 1996. A year earlier, an additional screening trial was set up at Tumentu and Cape Three Points, to test the performance of 7 ecotypes imported primarily from the Philippines, Indonesia and India.

When the 1996 adaptive trials of the VTT x SGD and VTT x MYD hybrids arising out of the 1981 to 1983 resistance screening trials showed that the hybrids were at least tolerant to the disease, and could also be produced commercially, the Ministry of Food and Agriculture (MoFA) with funding from Agence Francaise de Developpement (AFD), launched a replanting programme under the Coconut Sector Development Project (CSDP), in 1999.

The project, covering a 1 300 ha of land surface area was planted to the MYD x VTT hybrids. About six years later, however, the MYD x VTT hybrids succumbed under intense pressure of the disease.

Presently, a new hybrid developed from a genetic cross between the Sri Lanka Green Dwarf (SGD) and the Vanuatu Tall (VTT) parents have under experimental conditions, proven more resistant to the CSPWD than all hybrids tested so far. The superiority of this hybrid is established, both by resistance values from a predictive additive genetic model (GLM) and observed/ actual field data on CSPWD tolerance/resistance. The hybrid has subsequently been released to coconut farmers for large scale cultivation as far back as the year 2009. Table 2 shows some coconut varieties that been genetically screened for CSPWD resistance since 1956.

Adoption of strict farm hygiene/phytosanitation

Most secondary spread of the disease occurs within 100 m of a new focus and eradication of this focus is useful if practiced rigorously in the early stages of an outbreak (McCoy et al., 1976). Felling diseased palms eliminates or at least, reduces the quantity of initial inoculum available to elicit disease. Felling is particularly effective when done immediately upon detection of diseased palm, in concert with strict farm hygiene culture (Figure 2).

Phytosanitary practice such as weed control is important in curbing the incidence of the disease, since it ultimately eliminates the reservoir of potential alternate hosts. Also, many insects probably including vectors of CSPWD phytoplasma are known to lay their eggs in grass species such as *Rottboelia cochinchinensis*, *Panicum maximum*, *Paspalum scrobiculatum* and *Pennisetum polystachion*. Removal of these grasses therefore, eliminates their breeding sites and so helps control the disease.

Table 2. Some varieties of coconut used in CSPWD resistance trials in Ghana.

Full name	Fruit characteristics	Origin
Andaman Tall (ADOT)	Large pale green fruits	India (West India)
Catigan Green Dwarf (CATD)	Large, green, round, nipple- tip	Philippines
Cameroon Red Dwarf (CRD)	Medium, pale orange, pear-shaped fruits	Cameroon/P. Oceanic
E. G. Green Dwarf (EGD)	Medium, green, round fruits	Philippines-Indonesia-Brazil
Laccadive Tall (LCT)	Small, green, oval fruits	India (East India)
Malayan Red Dwarf (MRD)	Medium, orange, round fruits	Philippines
Malayan Yellow Dwarf (MYD)	Medium, yellow, oval fruits	Philippines (now cosmopolitan)
Malayan Tall (MLT)	Large, green, round fruits	Philippines
Niu Leka Dwarf (NLD)	Large, oblong/round, green/brown fruits	Fiji Archipelago
Panama Tall Aguadulce	Medium, pale green, round fruits	Tropical America (East Coast)
Panama Tall Monagre	Medium, pale green, round fruits	Tropical America
Rennell Island Tall (RIT)	Large, greenish-brown, varied shapes	Polynesia and Melanesia
Sri Lanka Green Dwarf (SGD)	Medium, grayish-green, oblong fruits	Sri Lanka
Tacunan Green Dwarf (TACD)	Large, bright green, round	Philippines
Tagnanan Tall (TAGT)	Large, reddish-brown, round fruits	Philippines
Tahiti/Polynesian Tall (TAT)	Small, deep orange, round/oblong fruits	Tahiti
Tahiti Red Dwarf (TRD)	Small, deep orange, round/oblong fruits	Tahiti
Vanuatu Tall (VTT)	Small, pale green, near-round fruits	Vanuatu/French Polynesia
W. A. Tall Benin Type WAT-ex Benin*	Large, greenish-brown, oblong	Mozambique (originally)
W. A. Tall Ghana Type WAT-ex Ghana*	Large, greenish-brown, oblong	Mozambique

* W.A. = West Africa; *E.G. = Equatorial Guinea.

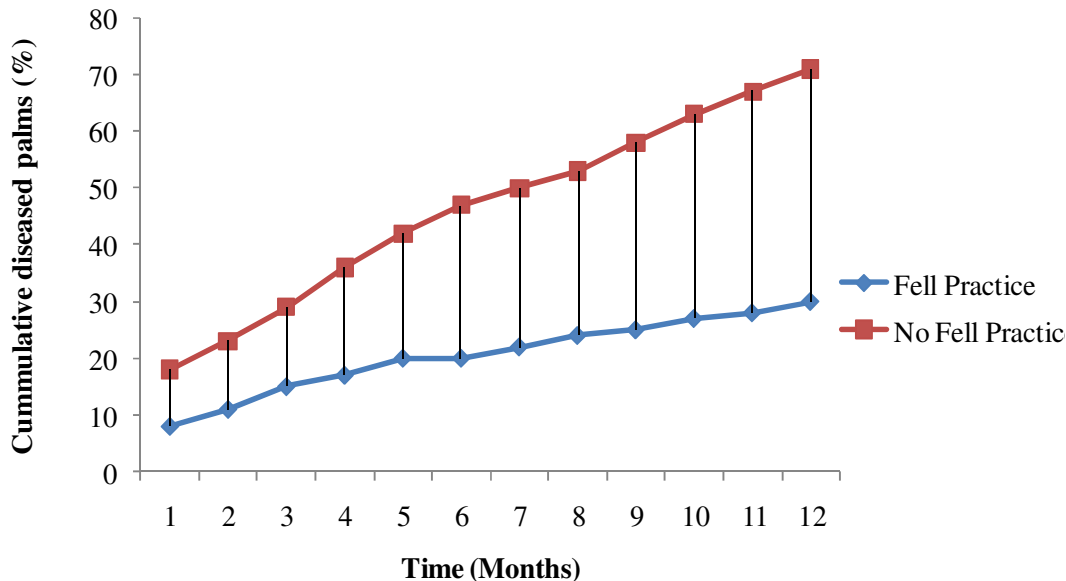


Figure 2. CSPWD progress under the indicated control regimes in the WAT variety.

Putative vector identification and population studies

Phytoplasma diseases are invariably transmitted by Auchenorrhyncha. In particular, leafhoppers, plant hoppers, and psyllids, which are able to inject the

phytoplasma into the phloem (Tsai, 1979). But, the presence of phytoplasma in insect does not necessarily prove its capacity to transmit the CSPWD disease. It only helps us to focus on those insects harbouring the CSPWD phytoplasma in our search for vectors in

Table 3. Duration of lethal yellowing disease cycle in the WAT coconut variety.

LYD stage	Symptom	Duration (weeks)
1	Premature nut/button drop	4 to 6
2	Yellowing of lower fronds	4 to 6
3	Yellowing of entire crown	4 to 6
4a	Death by wilting of crown	6 to 8
4b	Crown fall/Pole	Not determined

transmission trials (Dollet et al., 2008).

In Ghana, studies on putative insect vectors of CSPWD begun in 1990 (Dery et al., 1996) and were given a boost in 1991, when the World Bank, under its Oil Palm Development Phase II, approved a line of credit to fund investigations into the vector of the disease (Dery et al, 1996). The major research activities included:

1. Survey, identification, and population dynamics of putative insect vectors;
2. Detection of phytoplasma DNA in putative insect vectors;
3. Transmission trials with putative insect vectors (Cage studies);
4. Putative insect vector population control (Hot-Fogging).

When this funding ran out in 1993, the EEC approved funding under the STD III programme to continue the vector studies and to develop in collaboration with the Natural Resources Institute and Rothamsted Experimental station both in the UK, diagnostic techniques for Mycoplasma-like organisms and carry out insects of the Order: Homoptera, suborder: etiological investigations on CSPWD of coconut.

Search for alternative hosts of CSPWD phytoplasma

The recurrence of lethal yellowing disease (or CSPWD) in replanted devastated coconut farms, coupled with the isolation of lethal yellowing-type phytoplasmas in grass species associated with coconut farms such as, *Emelia forsbegii*, *Synedrella nodiflora*, and *Stachytarpheta jamaicensis*, in the Caribbean (Myrie et al., 2008), necessitated flora analysis in Ghana. If haply, we might find indigenous grass species that harbour the coconut phytoplasma, and that might serve as alternate hosts in the absence of the primary host (Bonnot et al., 2009).

The study involved the sampling of all flora associated with coconut farms; in and around the farms, identifying and classifying them taxonomically, and then subjecting them to molecular assay for the detection of coconut lethal yellowing disease phytoplasma, using polymerase

chain reaction (PCR).

RESULTS

Epidemiological studies

The Cape Saint Paul Wilt Disease (CSPWD) is caused by a phytoplasma. They are transmitted by insect vectors. The disease almost always starts by infecting a few coconut palms, or even a single palm in a healthy plot (localized infections), then spreads in leaps and bounds over variable distances, in any direction (jump infection). Disease foci occur in patches that can sometimes merge.

All stages in the disease cycle preceding total death, that is, nut drop; yellowing of lower fronds; and generalized yellowing, span approximately six weeks each. Time lapse between stage 1 (premature nut drop) and stage 4 (crown fall) is approximately 22 to 24 weeks (Table 3). When infected, a disease palm normally dies within six months of manifestation of first symptoms of the disease.

Breeding coconut germplasms (seed nuts and pollen) for CSPWD resistance

Only 3 ecotypes, namely, Sri Lankan Green Dwarf (SGD), Vanuatu Tall (VTT), and Malayan Yellow Dwarf (MYD) showed significant tolerance to the Cape Saint Paul Wilt Disease (CSPWD). A new hybrid developed from a genetic cross between the Sri Lanka Green Dwarf (SGD) and the Vanuatu Tall (VTT) parents have under experimental conditions, proven more resistant to the CSPWD than all pure lines and hybrids tested so far. The superiority of this hybrid is established, both by resistance values from a predictive additive genetic model (GLM) and observed/ actual field data on CSPWD tolerance/resistance.

But the strategy has proven rather lengthy and difficult with no truly resistant coconut breed yet. Current strategy has therefore, integrated the use of molecular techniques for genetic improvement with large-scale

Table 4. Putative vectors identified in CSPWD studies in Ghana.

Name of insect vector	Family
<i>Diostrombus luteus</i>	Derbidae
<i>Diostrombus</i> sp.	Derbidae
<i>Diostrombus nitidus</i>	Derbidae
<i>Diostrombus dilatatus</i>	Derbidae
<i>Diostrombus mayumbensis</i>	Derbidae
<i>Myndus adiopodoumeensis</i>	Cixiidae
<i>Metaphenice stellulata</i>	Derbidae
<i>Patara amata</i>	Derbidae
<i>Nzinga palmivora</i>	Typhlocybinæ

Credit: J. Nkansah-Poku, CSIR-OPRI, Coconut Research Programme, Sekondi, Ghana.

production and distribution of promising material (s) by mixed seed garden production.

Adoption of strict farm hygiene/phytosanitation

The treatment of infected farms with insecticide by hot fogging followed by cutting out the diseased and contact palms, immediately upon detection, slows down the disease and in some cases completely holds the disease in abeyance for few years, either by eliminating the reservoir of host palms or reducing the amount of initial inoculum available to elicit the disease subsequently. Figure 2, illustrates the usefulness of the felling practice in CSPWD containment strategy.

Putative vector survey, identification, population studies, and control

Although, the vector(s) of CSPWD could not be identified in the 1992 World Bank assisted project, the resulting data on coconut entomofauna was important in narrowing down the putative vectors to 2 families. The search for putative vectors of CSPWD in Ghana, encountered mainly Auchenorrhyncha species, predominantly species of derbids, *Nzinga palmivora* Wilson (Homoptera: Typhlocybinæ) and *Myndus adiopodoumeensis* Synare (Homoptera: Cixiidae). Other Homoptera species occurred infrequently (Table 4).

So far, only *M. adiopodoumeensis* and *Diostrombus* spp. have tested positive for CSPWD phytoplasma even though, they failed subsequently to elicit the disease in cage transmission trials. Sadly, therefore, to date, the insect vector(s) of the phytoplasma responsible for the CSPWD in Ghana remains unknown.

Search for secondary or alternative hosts of CSPWD phytoplasma in Ghana

The list of plants screened so far, for their capacity to

harbor coconut lethal yellowing disease (or CSPWD) phytoplasma is presented in Appendix 1. At least, 3 grass species (*Emelia forsbegii*, *Synedrella nodiflora*, and *Stachytarpheta jamaicensis*) of the plant species so far sampled are those cited as harbouring the lethal yellowing phytoplasma in the Caribbean. Only one indigenous grass species, *Desmodium adscendeus* has so far tested positive for CSPWD phytoplasma using PCR.

DISCUSSION

The manner of disease initiation and spread can be explained by the hypothesis that the number of vectors transporting the inoculum at the onset of disease is small (Plate 1a, b and c). As the disease develops, many more insects acquire the inoculum through feeding and therefore, infection increases. Although the vector seems to display a sedentary nature, it is possibly transported long distances by the wind, or during a dispersal phase, it may migrate long distances. This behaviour may explain the “leaps and bounds” spread of the disease, particularly the leap from Cape Saint Paul in the Volta Region in 1932, to Cape Three Points in the western Region in 1964, a distance of 350 km!

The variation in disease incidence and severity among the same coconut varieties in different environments may be due to genetic variation, especially where the planting materials were obtained by open pollination. The search for high-yielding disease tolerant/resistant coconut germplasms will therefore, require new performance trials, perhaps, using assisted (closed) pollination instead of open pollination. Although, the coconut seed nut is not known to transmit coconut lethal yellowing phytoplasma or its vector, the risk of vector transfer into areas of healthy coconuts might be reduced if irradiation treatments could control the pestiferous vector (and the pathogen) without harm to the coconut (and other plants) or risks to the environment.

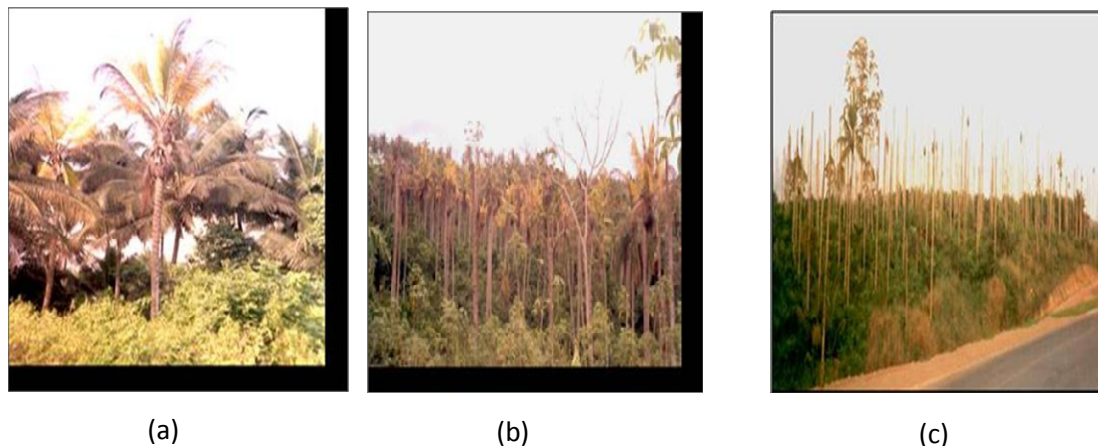


Plate 1. a) Disease initiation: generalized yellowing of fronds; b) intense disease focus: wilting of entire coconut crown; c) devastated coconut farm: dead coconut trunks (Credit: Dr. S.K. Dery, CSIR-OPRI, Coconut Research Programme, Sekondi, Ghana).

Early felling of first diseased palms often reduces disease development in infected plots (for a year or more), confirming that the disease spreads by dispersal from the first palm(s) to be affected. But felling, though useful, can be ineffective when farmers fail to fell diseased palms immediately upon detection, often due to socioeconomic and labour constraints. For example, because CSPWD cycle lasts approximately 6 months, many 'smart' farmers will rather wait for their set bunches to mature for harvest before they fell. Felling with chainsaw is easier than with axe or machete, but many farmers are unable to afford the operational cost. They are also prohibited by law to possess chainsaw machines! This obviously, aggravates the situation.

It has to be pointed out also, that the utility of phytosanitation (removal of infected palms and weeds) as a crop protection practice is not unique to the Cape Saint Paul Wilt Disease. It is an old-age conventional crop protection practice and not a recent innovation. Fact is that, weeds as competitors for soil nutrient, deprive target crops of much needed food leading to the symptoms of disorder. When they act as harbourages of pests and disease-inciting pathogens, they predispose crops to diseases.

The failure of CSPWD vector-transmitted trials to provide conclusive results as to the role played by Homoptera; the most likely species being *Myndus adiopodoumeensis*, calls for re-evaluation of transmission protocols applied in the transmission trials. If, or when the actual vector(s) of the CSPWD is identified in Ghana, reduction of natural vector population by sterile-male release methods should be a practical means of delaying or stopping the spread of CSPWD into new areas. Similar radioactive tracer experiments on vector host grasses should help identify alternate hosts of the CSPWD phytoplasma. Indeed,

identification of CSPWD vectors by radioactive tracing of phytoplasma infected insects in concert with modern sophisticated PCR techniques was done in Jamaica (Eskafi, 1982).

Bonnot et al. (2009), in a study on the role of environmental factors in the dynamics of CSPWD epidemics in Ghana, found significant effects of the presence of some grass species and intercrops in or around coconut farms on the incidence of CSPWD. Indeed, CSPWD incidence correlated positively, with the presence of *Lycopersicum* sp (tomato) inside the farm, and *Citrus* sp (orange) or *Saccharum* sp (sugarcane) around the farm, suggesting that these crops could be potential hosts of the pathogen or its vector(s). This suggestion should, however, be considered with much caution.

CONCLUSION

The coconut palm is not just of interest to millions of small holders, to whom it supplies food and drink, receptacles, fibres braided into rope, frond roofs, and even fuel for cooking, but also, of a global economic importance as a source of fats and oils and numerous industrial products.

However, the profitability of its cultivation, and its competitive position on the vegetable oils market, largely depend on the progress made in research, particularly in breeding research. New coconut palms need to be chosen with care so that they suit to the ecological conditions of the growing zone. They will also have to be appropriate for the intended uses of the plant's products.

Apart from enriching the global literature on alternative hosts, the isolation and elimination of reservoirs of alternative hosts in and around coconut farms, together with breeding resistant coconut germplasm, vector

identification and population dynamics, as well as disease containment through field surveillance and prompt felling of infected palms, remain vital practices in finding a lasting remedy to the menace of CSPWD.

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APPENDIX 1

Catalogue of flora associated with coconut farms in Ghana

Genus	Species	Family
<i>Asystacia</i>	<i>gangetica</i>	Acanthaceae
<i>Crinum</i>	<i>ornatum</i>	Amaryllidaceae
<i>Spondias</i>	<i>mombin</i>	Anacardiaceae
<i>Synedrella</i>	<i>nodiflora</i>	Asteraceae
<i>Ageratum</i>	<i>conizoides</i>	Asteraceae
<i>Chromolaena</i>	<i>odurata</i>	Asteraceae
<i>Vernonia</i>	<i>cinerea</i>	Asteraceae
<i>Aspilia</i>	<i>africana</i>	Asteraceae
<i>Baphia</i>	<i>nitidia</i>	Asteraceae
<i>Canna</i>	<i>indica</i>	Cannaceae
<i>Cleome</i>	<i>ciliate</i>	Cleomate
<i>Commelina</i>	<i>bengalensis</i>	Commelinaceae
<i>Ipomea</i>	<i>cairica</i>	Convolvaceae
<i>Ipomea</i>	<i>involucata</i>	Convolvaceae
<i>Boerhavia</i>	<i>diffusa</i>	Cucurbitaceae
<i>Cyperus</i>	<i>difformis</i>	Cyperaceae
<i>Mariscus</i>	<i>spp</i>	Cyperaceae
<i>Kyllingia</i>	<i>spp</i>	Cyperaceae
<i>Nephrolepis</i>	<i>biserrata</i>	Davalliaceae
<i>Croton</i>	<i>lobatus</i>	Euphorbiaceae
<i>Phyllanthus</i>	<i>amarus</i>	Euphorbiaceae
<i>Euphorbia</i>	<i>heterophylla</i>	Euphorbiaceae
<i>Mallotus</i>	<i>oppositifolius</i>	Euphorbiaceae
<i>Alchornia</i>	<i>cordifolia</i>	Euphorbiaceae
<i>Securinegia</i>	<i>virosa</i>	\ Euphorbiaceae
<i>Centrosema</i>	<i>pubescens</i>	Fabaceae
<i>Momordica</i>	<i>charantia</i>	Fabaceae
<i>Desmodium</i>	<i>spp</i>	Fabaceae
<i>Abrus</i>	<i>preparatorius</i>	Fabaceae
<i>Indigofera</i>	<i>hirsute</i>	Fabaceae
<i>Triclisia</i>	<i>subcordata</i>	Filiaceae
<i>Flagellaria</i>	<i>guineensis</i>	Flagellariaceae
<i>Asparagus</i>	<i>warneckii</i>	Liliaceae
<i>Abutilon</i>	<i>mauritanum</i>	Malvaceae
<i>Malvastrum</i>	<i>coromandelianum</i>	Malvaceae
<i>Momosa</i>	<i>pudica</i>	Mimosaceae
<i>Acacia</i>	<i>spp</i>	Mimosaceae
<i>Digitaria</i>	<i>ascendens</i>	Nyctaginaceae
<i>Ludwigia</i>	<i>spp</i>	Onagraceae
<i>Jussiae</i>	<i>spp</i>	Onagraceae
<i>Axonopus</i>	<i>compressus</i>	Poaceae
<i>Setaria</i>	<i>barbata</i>	Poaceae
<i>Brachiara</i>	<i>deflexia</i>	Poaceae
<i>Melanthera</i>	<i>scandens</i>	Poaceae
<i>Paspalum</i>	<i>spp</i>	Poaceae
<i>Panicum</i>	<i>maximum</i>	Poaceae
<i>Imperata</i>	<i>cylindrica</i>	Poaceae

APPENDIX 1 . Contd.

<i>Sporobolus</i>	<i>pyramidalis</i>	Poaceae
<i>Brachiria</i>	<i>lata</i>	Poaceae
<i>Rottboellia</i>	<i>exaltata</i>	Poaceae
<i>Talinum</i>	<i>triangulare</i>	Portulacaceae
<i>Portulaca</i>	<i>oleracea</i>	Portulacaceae
<i>Pentodon</i>	<i>pentandras</i>	Rubiaceae
<i>Oldenlandia</i>	<i>corymbosa</i>	Rubiaceae
<i>Cardiospermum</i>	<i>grandifloru</i>	Sapindaceae
<i>Malacantha</i>	<i>alniflora</i>	Sapotaceae
<i>Solanum</i>	<i>nigrum</i>	Solanaceae
<i>Physalis</i>	<i>angulata</i>	Solanaceae
<i>Waltheria</i>	<i>indica</i>	Sterculiaceae
<i>Fleurya</i>	<i>aestuans</i>	Urticaceae
<i>Lantana</i>	<i>camara</i>	Verbenaceae
<i>Priva</i>	<i>lappulacae</i>	Verbenaceae
<i>Stachytarpheta</i>	<i>indica</i>	Verbenaceae
<i>Stachytarpheta</i>	<i>cayennnsis</i>	Verbenaceae
<i>Clerodendron</i>	<i>capitalum</i>	Verbenaceae
