Chapter 5

Effects of White Root Rot Disease on *Hevea brasiliensis* (Muell. Arg.) – Challenges and Control Approach

Victor Irogue Omorusi

Additional information is available at the end of the chapter

http://dx.doi.org/10.5772/54024

1. Introduction

Monoclonal *Hevea brasiliensis* (Willd. ex Adr. De Juss) Muell. Arg.)is principally valued for its latex content, the latex or Natural Rubber (NR) is very significant in world's industrialization. This importance has been expressly emphasized in the production of elastomers, the use of which is indispensable in space, water, and ship technologies (Jacob 2006). The dependence of world industrialization on NR production is further underscored especially now considering the diminishing reserves of petroleum with increasing environmental hazards.

The rubber tree is subject to a plethora of economically important pathological problems, mainly of fungal origin (the basisdiomycetes) (Igeleke, 1998). In Nigeria, the most serious diseases of rubber seedlings and budded plants in the nursery are leaf diseases (Begho, 1990), while In mature plantation, the most devastating leaf disease is the South American Leaf Blight (SALB), and *Corynespora* Leaf Fall Disease (CLFD) appears to be next to SALB. In field plantations, root diseases pose a serious problem especially in the first few years after planting. In Nigeria, the white root rot disease of rubber is the most serious. It accounts for about 94% of incidences of all root diseases and kills up to five *Hevea* trees/ha (Otoide, 1978). Over a period of time, half of the rubber trees in a plantation are lost to the disease. The infective fungal organism of the white root rot disease is *Rigidoporus lignosus* (klotzsch) Imazeki. The brown root rot disease (*Phellinus noxius*) Corner-Cunn., unlike the white root rot, is most the most serious root disease in *Hevea* plantations in Liberia while *R. lignosus* and Armillaria root rots occur to a lesser extent (Nandris *et al*. 1987). Similarly, in Cote d´lvoire, *R. lignosus* is the main cause of *Hevea* tree losses with 40-60% of the trees destroyed over a period of up to 21 years (Nandris *et al*. 1987). The white root rot incidence is absent in

India, however, it is serious in Malaysia, Sri Lanka and Congo (Rajalakshmy and Jayarathnam 2000), aside its severe occurrences in Nigeria and Cote d´lvoire.

Rubber tree exhibits natural resistance to invading root pathogens. Resistance often breaks down due to effect of pathogens that colonize living tissues of the tree to obtain nutrients as a result of the damaging and weakening of the plant with toxins or by preventing the plants defense mechanism (Jayasuriya 2004). A number of certain defense mechanisms in *Hevea* against *R. lignosus* and *P. noxius* have been identified. These include cellular hypertrophy and hyperplasia, cambium activity stimulation, lignifications and suberification of certain cell walls (Jayasuriya 2004., Nicole *et al* 1985).

The process of pre- infection involves pathogen breaking down the host cuticle and cell wall. Plants respond to infection process by producing anti-microbial compounds of low molecular weight (phytoalexins) (Darvill *et al* 1984). *Hevea* plants produce anti-microbial phenolic compounds such as coumarins, flavonoids, triterpenes among others (Jayasuriya 2004) that can partially or completely inhibit microbial infection.

The growth and spread of infective fungal pathogens from existing population have been on the increase with great virulency and inflicting damages even to resistant genotypes. The impact of fungal pathogens results in crop losses. The production of phenylalanine ammonia-lyase (PAL) is implicated as key enzyme in the plant phenyl propanoid pathway to catalyse the synthesis of phenyl lignin and phytoalexin from L-phenylalanine (Jones 1984). The synthesis of these anti-microbial compounds and the subsequent increase in PAL concentration are often useful resistance indicator in the host (Nicholson and Hammerschmidt 1992). Also, oxidases and peroxidases are known to be actively involved in polymerization of phenolic compounds in the lignin formation. In resistance mechanism action of peroxidase is related to initiation of hypersensitive cell collapse (Simons and Rose 1971).

Pathogenesis-related proteins(PR) is yet another defense response of rubber against pathogen infection (Narasimhan *et al* 2000) The PR protein induced by polyacrylic, acetyl salicylic (aspirin) and salicylic acids are known to increase resistance to pathogens (Gianinazzi 1984).

There is variation among high yielding genotypes in disease tolerance level. In this regard, some clones are resistant to virtually most of the diseases but are susceptible to few diseases. However, certain clones exhibit tolerance to few diseases but some others are susceptible to many diseases. There are few clones which tolerate characteristics of pathogens, and as a result of abiotic factors, produce new strains, and these strains can be more aggressive against rubber clones. Mutation is seen to be responsible for variability in pathogens, which involves changing sequence bases in the nuclear DNA, either by way of substitution or addition or deletion of one or many base pairs.

The *Hevea* root fungal parasites- *R. lignosus*, and *P. noxius* are polyporaceae major causes of rubber tree losses in plantation causing the decay of lignified root tissues (Geiger *et al* 1986). *R. lignosus* is partially involved lignin consumption where as *P. noxius* degrade the polysaccharide fraction of but not lignin (Cowling 1961., Kirk 1971), however, findings by

Geiger *et al* (1986) showed that *R. lignosus* and *P. noxius* degrade both the lignin and polysaccharide fractions of the wood. Although *P. noxius* exhibits preferential degradation of polysaccharides, whereas, *R. lignosus* degrades both the lignin and polysaccharides in a relatively balanced manner but with slight preference for lignin.

The *H. brasiliensis* belongs to the family Euphorbiaceae of laticiferous plants. The tree growth can reach a height of over 20 m. Root decaying pathogens, *Rigidoporus lignosis* and *Phellinus noxius* are considered economically important in Africa and Asia due to the fact that they are most dangerous root parasites of the Hevea trees. The R. lignosus is most notorious in its destructive effects on rubber plants.

2. Mechanism of disease infection cycles

The host-parasite interactions involve attacks by *R. lignosus* on the tap root of the *Hevea* tree. The process of disease infection is basically through three stages namely penetration, colonization and degradation. The pathogen penetrates the root system and colonize the tissues. The mycelium of the pathogen, there after degrade the host's cell structures. The root rot pathogen of *R. lignosus* must repeatedly carry out penetration and colonization of their host cell wall. *R. lignosus* carries out its disease infection activities either by enzymatic digestion of the tissues characterized by differentiation of specialized structures (Nicole *et al*. 1986), or by mechanically penetration through colonized natural openings or wounds. Affected host tissues colonized by *R. linosus* through perforation and digestion of cell walls or by penetration through pores and pits of the vascular tissues (Nandris *et al* 1987). Usually, fungal hyphae can be observed as intra and inter interactions of the cell wall. Such observations have revealed some distortions of the cell wall, as well as digestion of the middle lamella and of the cell walls. The implication of these distortions is that enzymatic actions are involved in degradation of cell wall polymers. The root rot path-produce a host range of enzymes which are cell wall degrading enzymes (CWDE) that may correspond to the diverse polymers in plant cell wall of the host and parasite. Infected tissues parasitized are said to contain three enzymes as CM-cellulose, pectinase and Laccase for which their involvement in the pathogenic activity may be presumed (Geiger *et al* 1986). Enzymatic actions in infected tissues are much higher when compared to enzyme activities in healthy tissues. In defense response, the host reacts to parasite infection with increased enzymes from the stimulation of the biosynthesis of enzymes that are present in healthy tissues (Geiger *et al* 1986). Pegg (1977) claimed the enzymes involved take part in host defense responses to the parasite by degrading structural polymers in the mycelia wall. A hypothesis most frequently proposed and verified (Albershiem *et al* 1969) relates to excretion of parasite produced enzymes into the host tissues. The enzymes would be involved in pathogenesis by degrading the polymers in the invaded tissues. Studies of some β-(1-3) glucanases when synthesized by tomato plantlets, they degrade the β-(1-3) glucans of the invading parasite. *Verticillium alboatrum* (Pegg & Young 1981).

Analysis of root tissues (Geiger *et al* 1986) indicated that some enzymes – (CM – cellulose, pectinase, laccase) are present only in parasite tissues. It is explained that these enzymes are

biosynthesized by the parasite and not by the host. However, it is yet to be shown that the fungi are able to perform biosyntheses of those enzymes.

3. Degrading enzymes of cell wall effects in pathogenicity

Involvement of CWDE goes through certain criteria. The synthesis of CWDE is not a proof of involvement in disease. Pathogens with low potentials for the production of CWDE, the ability of cells to reduce the viscosity of a polysaccharide or to grow on a polysaccharide implies synthesis of the relevant polysaccharide, and lack of growth can sometimes reflect inability to metabolize the end product.

Studies of transmission electron microscopy show valuable indications to the participation of CWDE. Microcopic alterations of walls of the infected tissue is displacement of wall fibrils indicating mechanical penetration, whereas extensive wall dissolution signifies freely diffusible extracellular CWDE. Loss of the middle Lamella implies action by pectic enzymes (Baker *et al* 1980) which may also be revealed by lost affinity for the specific stains, ferric hydroxylamine. Alteration or loss of wall microfibrils resulting from cellulose activity is also obvious as reduced birefringence under polarized light.

In wood degradation by enzymes of white root rot fungi the structural elements cellulose, hemicelluloses and lignin synthesized and deposited in the plant cell walls reinforce the mechanical strength and rigidity of the stems of higher plants.

In the host specificity of wood rotters (Tuor *et al* 1995), hard and softwood are distinguished by structural elements building the phenylypropane backbone of the lignin component. Lignin is a three dimensional, optically inactive phenyl propanoid polymer randomly synthesized from coniferyl, p-coumaryl and sinapyl alcohol precursors (Sarkanen and Ludwig 1971). Soft wood on the other hand is referred to as guaiacyl lignin, having over 95% coniferly alcohol (4-hydroxy-3-methoxy-cinnamyl alcohol) units.

4. Pathogenicity of *rigidoporus lignosus a*nd mechanism infection cycle

Hevea trees are usually killed by root rot pathogens infecting the plants and detection is difficult at the early stages of disease development. Symptoms of root diseases in the above ground level are somewhat similar but differ in the below ground level (Farid *et al* 2009). Generally, the presence of above ground symptoms shows that the trees are now untreatable due to the fast and increased rate of disease infection their make death of plants imminent.

Infected trees show a general foliage discoloration, proceeded sometimes by premature flowering and fruiting. Affected tree branches die back until the whole canopy is destroyed and the tree eventually dies. In Nigeria, the foliage symptoms appear only when the tree is beyond treatment and recovery. The pathogen *R. lignosus,* forms large firm semi fleshy, often tiered brackets, on the collar of infected trees in the advanced stage of the disease. Normally formations of fructification come up only and after the trees have been dead for a

while. Distinctive features of fructification (basidiocarps) (Fig.1), show the upper surface of concentric zones that is brownish-orange with a bright yellow margin when fresh, while the lower surface is reddish-brown.

Figure 1. Basidiocarps of *R.lignosus* on dead *Hevea* tree.

When roots of infected are exposed, profusely branched white rhizomorphs are readily seen. The rhizomorphs are flattened mycelia strands of 1-2 mm thick that grow firmly attached to the surface of infected roots (Fig. 2).

The rhizomorphs grow rapidily and ahead of the rot and extend many meters through the soil freely hindrances from woody substrate. The internal progression of the development of the root rot pathogen is rather an ectotrophic growth characteristic. At infection point, the parasite penetrates the taproot down the soil. Nandris *et al.* (1987) explained for infection to take place, the rhizomorphs are subjected to morphogenetically state into infectious hyphae

Figure 2. Rhizomorph strands on the collars of root of *Hevea* tree.

exhibiting degrading extracellular enzymes capable of wood rotting. The authors further stated that this mechanism is strictly regulated by partial anoxia conditions in the soil. Following root infection, colonization within the taproot progresses towards the collar region and other parts of the root. Newly killed wood is brownish thereafter turns cream and soft. This shows fading of coloration along a gradient from the progression front of the parasite toward the tissues that were colonized before now. The effect of *R. lignosus* causing white root rot extracellular enzymes degradation of lignin in the cell walls of the root system. The basidiocarps seen at the collar region of the tree produce a large number of basidiospores especially during rainy season, but appear less functional in the dissemination of the disease. According to Nandris *et al.*(1987), this role is one of the most controversial points in the biology of *R. lignosus.* The spores are viable, however, John (1965) asserted that there is agreement now that the probability of a spore germinating in situ on a receptive substrate is rather very low. In newly established *Hevea* plantation after clearing of a forest, mycelia of *R. lignosus* do cause infection to take place. However, in the second planting, spores can become inoculum source for infecting the stump surfaces of old rubber trees that are existing between the planting rows. In plate cultures, *R. lignosus* growth on potato dextrose agar (PDA) or malt agar (MA) forms a superficial, extensive white fluffy myceilia (Fig.3).

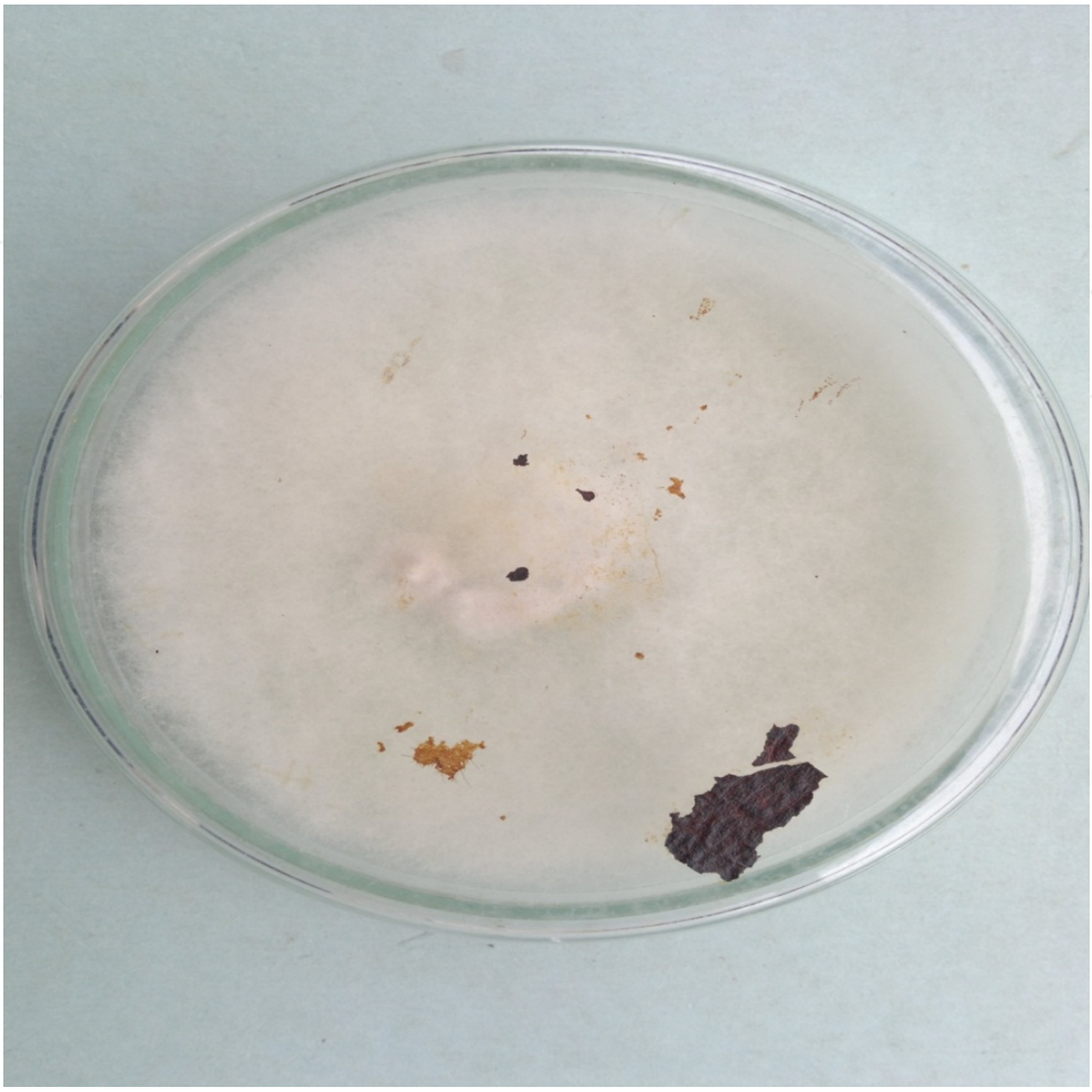

Figure 3. Fluffy growth of *R.* lignosus Disease Symptom Expression

Foliage of infected trees shows general discoloration, often preceded by premature flowering and fruiting. Branches of infected tree die back until the whole canopy is destroyed and the tree eventually dies. In Nigeria, usually the foliage symptoms appear only when the tree is no longer treatable. Exposure of roots around the collar infected trees is carried out by searching the collars with wooden like spades for the presence of rhizomorph filaments. The stick-trapping method is also a useful tool in detecting the growth of *R. Lignosus*. The sticks of *Hevea* wood are poked down the soil around the collar of the tree. The developments of mycelium on the stick are checked after three weeks of insertion into the soil. Another technique is the use of mulch around the collar for three weeks to provide a damp microclimate for the superficial mycelium growth on the collars of the trees. After three weeks the mulch is removed, mycelia growth of *R. lignosus* can be seen.

Generally, two phases characterize the spatial spread of root rot disease in *Hevea* plantations (Nandris *et al* 1985). In the disease cycle of white root rot (Fig. 4), the processes involve infection and colonization by mycelia of the root system of young rubber trees growing

from stumps of infected forest tree or otherwise referred to as primary inoculums. The other aspect is progress of the pathogen along roots from infected trees (second inoculums) toward healthy rubber trees around (Fig. 4). The mycelia growth length of *R. Lignosus* is about 2.5 m while it is 0.7 m for *P. noxius* per year (Nandris *et al*. 1987). This clearly shows comparative growth rates of the two root pathogens of *Hevea* that reveals differences in rapidity of spread. According to Nandris *et al.* (1985), disease development and death of *Hevea* trees are most rapid during the first few years after planting. Infection cycle development in diseased trees usually results in sudden death, delayed death or survival of infected trees.

5. Assessment of effect of white root rot disease on quantity and quality of rubber production

The level of incidence of white root rot disease and its effect on *Hevea* latex were evaluated at the Rubber Research Institute of Nigeria, main station, Iyanomo (Table 1). The plantation was previously cropped with cassava, yam, and plantain. The plantation consists of 36 sub plots of one hectare each with nine clones planted out in a completely randomized block designed. Six *Hevea* test clones comprising three local clones – NIG 800, 801, 802, 803, 804, 805, and exotic clones – GT1, PR 107, and RRIM 700 were assessed for the study. Assessment of the severity of the white root rot disease was based on disease index method by Parry (1990) on a scales of 0 = no infection, 1 = light infection, 2 = moderate infection, and 3 = severe infection. Infection was then calculated using the following formula, thus:

$$\text{Disease index(DI)} = \frac{(0 \times a)+(1 \times b)+(2 \times c)+(3 \times d)}{a+b+c+d} \times \frac{100}{x}$$

where,

0, 1, 2, and 3 are infection categories
a, b, c, and d are plants that fall into the infection categories
x is the maximum disease category which is 4

	***Hevea* clones**					
Rep.	NIG 800	NIG 800	NIG 800	GT 1	PR 107	RRIM 707
1	25.10	23.18	28.00	9.34	25.30	22.83
2	31.72	21.10	20.13	10.75	27.47	26.48
3	28.13	29.19	29.90	10.61	33.61	37.11
4	26.53	34.00	32.00	28.14	35.80	22.00
Mean	27.87	26.87	27.51	14.71	30.55	26.86

Table 1. Disease indices of incidence of white root rot of six *Hevea* test clones.

Disease indices recorded showed highest susceptibility score in PR 107 (30.55) and lowest score in GT 1 (14.71). Intermediate scores among other clones were equally high compared

with PR 107. The lowest susceptibility in GT 1 indicated that GT 1 showed a significant level of resistance to the white root rot disease.

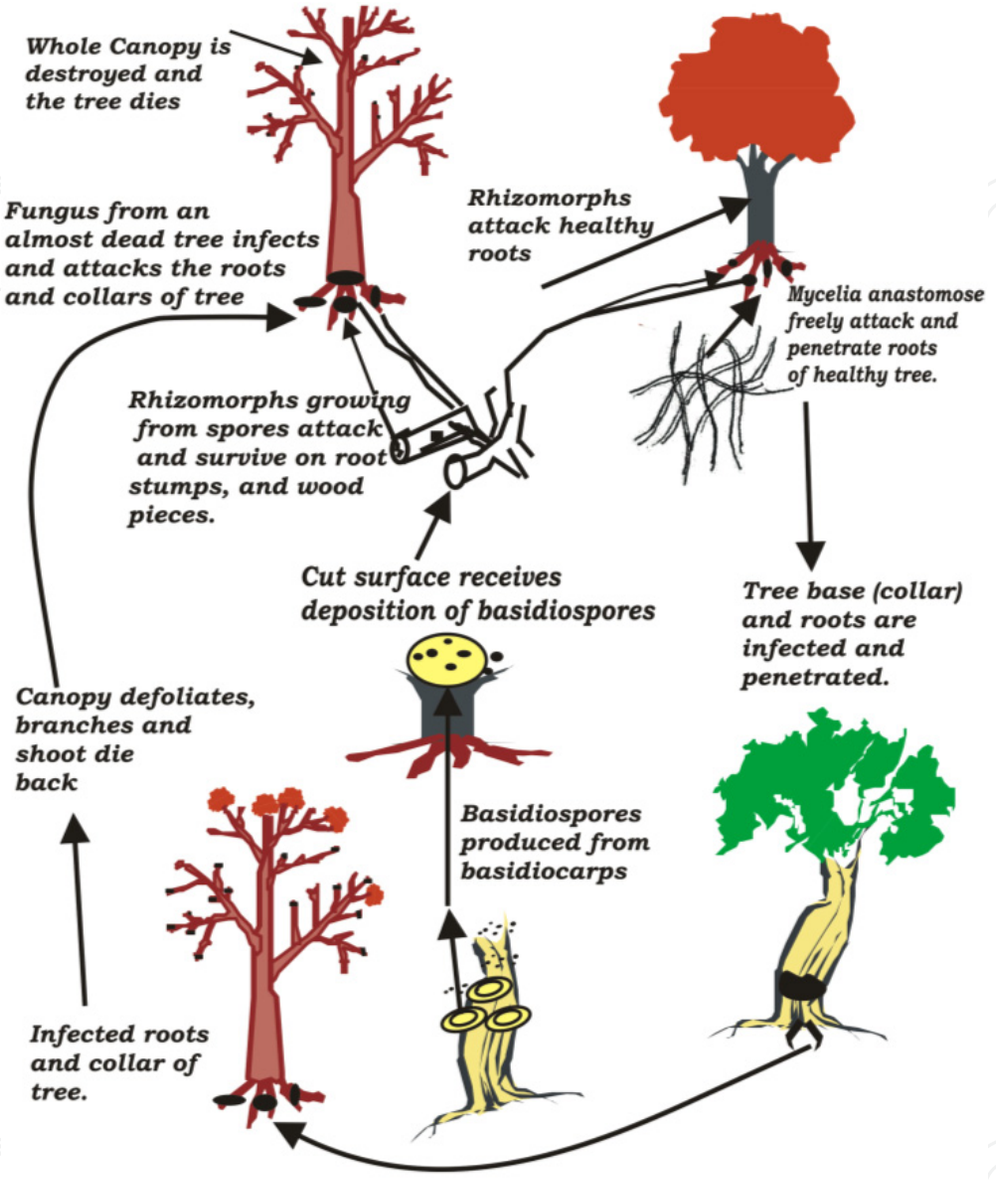

Figure 4. Diagramatic illustration of white root rot disease cycle.

The result of the white root rot incidence in six clones are shown in Table 1.

Based on the susceptibility status recorded, the GT 1 Lowest susceptibility status was assessed for the volume of latex and dry weight of cup lumps from effects at different infection categories of white root rot incidence (Table 2). The 'O' infection category had the highest volume of latex (228.93 cm^3), however, showed no significant difference with the rest clones. The highest infectivity category gave the highest dry weight of cup lumps (118.88g), followed by '0' '1' and '2' categories but no significant difference among the clones was obtained. This may be attributed to increase in DRC in response to effect of the white root rot disease.

Infection category	Volume of latex (cm^3)	Dry weight of cup lumps (g)
0	228.93	114.00
1	192.44	103.71
2	191.17	94.00
3	224.18	118.88
Cv (%)	26.91	28.24

Table 2. Volume of latex and dry weight of cup lumps under infection categories in GT 1

6. White root rot observations of *hevea* clones

Observations recorded at Rubber Research Institute of Nigeria (RRIN), Iyanomo, on the roots of some *Hevea* clones after ten years of planting are shown in Tables 3 & 4. Results of root rot observations showed that the white root rot accounted for almost all the root rots investigated indicating that there was high incidence of white root rot disease. Inspite of the high incidence of the white root rot, only a small proportion of the trees with root rot infection were actually killed by the disease. From this study, sixteen of the RRIN clones ('C' clones) examined recorded no loss due root disease infection. However, the remaining fourteen 'C' clones and PB 86 had losses due to root rot infection.

Clone	No. Planted Out	No. 9 Survival	No. with White Root Rot	No. of Dead From Root Rot	% Infection	% Survival
RRINSC*1	9	5	3	1	44.44	55.55
3	9	7	5	1	55.55	77.77
8	9	8	6	1	66.66	88.88
11	9	7	5	1	55.55	77.77
21	9	9	6	-	66.66	100.00
27	9	9	7	-	77.77	100.00
28	9	8	6	1	66.66	88.88
29	9	6	4	3	44.44	66.66
42	9	9	5	-	55.55	100.00
43	9	8	4	1	44.44	88.00
47	9	9	8	1	88.88	100.00
51	9	8	8	1	88.88	88.88
52	9	9	7	-	77.77	100.00
54	9	9	6	-	66.66	100.00
55	9	6	6	3	66.66	66.66
58	9	8	6	1	66.66	88.88
75	9	8	6	1	66.66	88.88
76	9	7	4	2	44.44	77.77
82	9	7	5	1	55.55	77.77
83	9	9	6	-	66.66	100.00

Clone	No. Planted Out	No. 9 Survival	No. with White Root Rot	No. of Dead From Root Rot	% Infection	% Survival
85	9	9	8	-	88.88	100.00
86	9	9	8	-	88.88	100.00
87	9	9	6	-	66.66	100.00
97	9	9	6	-		
98	9	5	4	4	44.44	55.55

*RRINSC - Rubber Research Institute of Nigeria 'C' Clones

Table 3. Root Rot Observation after 10years of Planting at RRIN

Clone	No. Planted Out	No. 9 Survival	No. with White Root Rot	No. of Dead From Root Rot	% Infection	% Survival
RRINSC						
104	9	6	1	2	11.11	66.66
105	9	9	7	-	77.77	100.00
106	9	9	5	-	55.55	100.00
114	9	6	5	1	55.55	66.66
118	9	9	4	-	44.44	100.00
PB86	162	121	87	25	53.70	74.69

* Rubber Research Institute of Nigeria 'C' Clones

Table 4. Impact of *Rigidoporus lignosus o*n *H. brasiliensis* clones-root rot observations after 10 years of Planting.

7. Fungicide curative approaches

Reduction of root diseases is performed through some control approaches. Proper clearing of land is recommended especially mechanically clearing is the most effective clearing method. In the recent past in Nigeria, treatment of infected roots with root rot diseases involved excavating the soil around the roots of infected trees, and removal of surface rhizomorphs and roots sections penetrated by the pathogen. A long-lasting collar protectant dressing fungicides such as pentachloronitrobenzene (PCNB) is then smeared around the collar, tap root and basal portion of the main laterals of the tree, before replacing the soil. The same treatment is also given to immediate tree neighbors, especially those along the same row. This approach is rather labor intensive and quite difficult to apply on a large scale plantation.

Results of investigation on the effectiveness of calixin fungicide (a.i. tridemorph) at the RRIN mainstation, Iyanomo (Table 5) revealed significant reduction of white root rot disease of *Hevea* trees. Virtually all clones treated with calixin recovered satisfactorily. Of the fourteen test clones treated with calixin, seven of the clones were over 80% cured of the *R.lignosus* infection while five were over 70% cured. However, these percentages of cured plants were highly significantly different at $P<0.01$. Only a minor proportion of two clones with over 50 and 66% were cured and significantly different at $P<0.05$.The percentages of cured trees related to the number of clones that survived from the number of dead clones against the number of clones planted out.

Clones	Dead/ No. Planted	No of Survivors	Pecentage of Plants cured
PRINSC 1	2/9	7	77.77
3	1/9	8	88.88
8	1/9	8	88.88
11	1/9	8	88.88
28	1/9	8	88.88
55	3/9	6	66.66
58	2/9	7	77.77
75	1/9	8	88.88
76	2/9	7	77.77
82	1/9	8	88.88
98	4/9	5	55.55
104	2/9	7	77.77
114	2/9	7	77.77
PB 86	30/162	133	82.09

P< 0.01 (Clones with over 70-80% cured)
P<0.05 (Clones with over 50-60% cured)
* RRIN SC – Rubber Research institute of Nigeria 'C'clones

Table 5. Clones with White Root Rot treated with Calixin P< 0.01 (Clones with over 70 -80% cured)

8. Preventive measures against incidence of *r.lignosus*

Foliage discoloration of *Hevea* trees is external indicator of the presence of white root rot incidence when the foliage is watched of three months intervals. Notice of foliage discoloration is often preceded by premature flowering and fruiting. There after branches begin to die back and eventually the whole canopy is destroyed and the tree is dead. This phenomenon above ground described serves as a warning sign of the presence of white root rot disease. Below ground level reveals the presence of rhizomorphs, is usually by inspection of the diseased collar region as well as neighboring trees.

Clearing methods such as uprooting and poisoning of old trees are found to reduce root disease incidence in replanting. The stumps from felled trees should be poisoned and the cut surface painted with creosote. This will prevent the root fungi of food sources.

Planting of legume cover crops helps to reduce root disease since they cause roots and stumps to rot faster (Malaysian Rubber Board 2000). It also asserted that about 30g of powdered sulphur should be amended into the planting hole. Sulphur is known to promote fungal growth antagonistic to incidence of fungal root diseases.

The use of collar protectant to paint the collar part of the tree is said to prevent root disease infection for few years (Malaysian Rubber Board 2000). Suitable fungicidal wound dressing should be applied to any damaged part of the tree. This prevents infective spores from gaining entry into the plant tissues. The digging of isolation trenches of one meter between infected and healthy trees prevents the spread of disease from infected plants to health trees. This method is rather difficult and may not be satisfactorily effective.

9. Treatment measures

On exposure of infected root system, all dead roots are removed, bulked and burnt. Soil particles attached to the roots are shaken off. After exposure, root surface are dried and suitable long lasting collar protect ant fungicide pentachloronitrobenzene (PCNB) is painted over the exposed infected root system, including collar, tap root and basal portion of the main laterals of the tree, before replacing the soil. The same treatment is carried out for the immediate tree neighbors, especially those along the same row. This method of exposing the root system and subsequent application of a collar protect ant fungicide is quite labour intensive exercise and uneconomical. The PCNB use is hazardous, being carcinogenic and has been banned (Rajalakshmy and Jayarathnam 2000). This method has been abandoned in most rubber growing regions of the world.

A less laborious approach is digging a slight furrow around the base of the infected tree and drenching the collar region with two liters of 0.5% calixin (a.i.tridemorph). Infected trees as well as two direct neighbors in the same row are treated every six months. Other suitable fungicides for the treatment of white root rot disease is the use of Bayleton (a.i. triadimefon), Bayfidan (a.i. tridimenol), Anvil (a.i. hexaconazole), Folicur (a.i. tebuconazole), Contaf (a.i. hexaconazole) and Daconil (a.i. chlorothaconil) (Jayaratne *et al* 2001). The use of these chemical fungicides as described above involving loosening the soil and forming funnel – like furrow around the base of the trunk, and then drench the chemicals into the furrow along the trunk of the tree with about 10-20 ml at every four to six months is highly effective.

The purpose of controlling root diseases is to remove their sources of infection or inocula at the very young stages of the trees in order to prevent the rest trees from being infected as they mature. Due to possible sources of infection it is necessary to put in place adequate management measures. In embarking on preparations for the management, the role of tapers is necessary since tapers know the locations of the diseased trees in the plantations and this however, simplifies task of locating the diseased trees. This explains the need to train tapers on how to recognize root diseases and other *Hevea* disease symptoms. Treating diseased plants is to curtail the spread of the disease since it takes a higher cost of treating older trees and which may be abandoned and isolated by trenches to reduce the spread of root disease to healthy trees.

Author details

Victor Irogue Omorusi
Plant protect Division, Rubber Research Institute of Nigeria, PMB, Iyanomo, Benin City, Nigeria

10. References

Begho, E.R. 1990. Nursery diseases of *Hevea brasiliensis* in Nigeria and their control. 103—106 in : *Proceedings of a Natural Workshop on fruit / tree crop seedling Production* held at NIFOR, Benin City, Aug 14-17, 1990. 160 PP.

Cowling, E.D. 1961. Comparative Biochemistry of the decay of sweet gum sapwood by white rot and brown rot fungi. *U.S. Department of Agriculture, Technical Bulletin* No. 1258, 79p.

Darvill , A.G., and Albersheim, P. 1984. Phytoalexins and their elicitors – A defence against microbial infection in plants. *Annual Review Plant Physiology* 35:243-275.

Geiger, J.P., Rio, B., Nicole, M., and Nandris, D. 1986. Biodegradation of *Hevea Brasiliensis* wood by *Rigidoporus lignosus* and *Phellinus noxius*. *Eur.J . For. Path;* 16:147-159.

Gianinazzi, S.1984. Genetic and molecular aspects of resistance induced by infections or chemicals. In : *Plant –microbe Interactions Molecular and genetic perspectives*. Vol 1. (Eds; Kosuge, T and Nester, E.W), 321-342 PP. Macmillan, New York.

Igeleke, C.L. 1988. Diseases of Rubber (*Hevea brasiliensis)* and their control. Paper presented at the 10th Annual conference of the Horticultural society of Nigeria, 6-8 Nov. 1988

Jacob, C.K 2006. Corynespora leaf disease of *Hevea brasiliensis:* A threat to natural rubber production (9-16pp) in Corynespora leaf disease of *H. brasiliensis:* strategies for management (Ed. C.K. Jacob). Rubber Research institute of India.

Jayasuriya, K.E. 2004. Factors affecting disease tolerance of rubber tree and research needs for developing disease tolerant genotypes for the sustainability of rubber industry. *Bulletin of the Rubber institute of Sri Lanka,* 45 : 1-10.

Jayaratne, R, Wettasinghe, P.C., Siriwardene, D., and Peiris D. 2001. Systemic fungicides as a drench application to control white root disease of rubber. *Journal of the Rubber Research Institute of Srilanka*, 84:1-17.

Jones, D. H. 1984. Phenylanine ammonia – lyase: regulation of its induction, and its role in plant development. *Phytochemistry,* 23:1349-1369.

Kirk, T.K . 1971. Effect of microorganisms on ligin. *Annual Review of Phytopathology,* 9:185-210.

Malaysian Rubber Board. 2000. *Treatment of maladies and injuries and control of pests.* In: Rubber Plantation and Processing technologies. Malaysian Rubber Board, 2000.

Nandris, D., Nicole, M. and Geiger, J.P. 1987. Root Rot Diseases of Rubber Trees. *Plant Disease,* 71 (4) : 298-306.

Narasimhan, K.T and Kothandaraman, R. 2000. Detection of pathogenesis related proteins in *Hevea brasiliensis* infected by *Phytophthora meadii*. *Indian Journal of Natural Rubber*, 13:30-37.

Nicholson, R.L,. and Hammerschmidt, R. 1992. Phenolic compounds and their role in disease resistance. *Annual Review of Pyhtopathology* 30:369-389.

Nicole, M., Geiger, J.P., and Nandris, D. 1985. Variability among Africa populations of *Rigidoporus lignosus and Phellinus noxius, Eur. J. for. Pathol*. 15: 293 -300.

Otoide, V.O. 1978. Further observations on the pre-treatment of forest trees for root disease control in *Hevea* plantings. Paper presented at RRIN Seminar, 1978, 7Pp.

Rajalakshmy, V.K, and Jayarathnam, K.2000. Root diseases and non- microbial maladies. In: *Natural Rubber: Agro-management and crop processing* (Eds. P.J George and C. Kuruvilla Jacob) Rubber Research Institute of India, Kohayam, Kerala, India.

Simons, T.J., and Rose, A.F. 1971. Metabolic changes associated with systemic induced resistance to tobacco mosaic virus in Samsun -NN tobacco. *Phytopathology,* 61:293 -300.