Chapter 13
Diseases of the Oil Palm

As the area under oil palm has expanded, there have been serious, and at times devastating, outbreaks of disease in several parts of the world. Of greatest importance have been the devastation caused by *Fusarium* wilt in several parts of Africa, the considerable losses sustained through dry basal rot (*Ceratocystis*) in Nigeria and associated with *Ganoderma* in old and replanted areas in Asia and the attacks of fatal yellowing and other diseases in plantations in Latin America.

Diagnosis and prevention or cure of some of these diseases have proved difficult. It is not clear in some instances whether a pathogen is involved or whether the symptoms are a disorder caused by some abiotic factor. The fact that some, perhaps most, diseases only become serious under certain predisposing environmental conditions may further complicate matters. Turner (2003) emphasised the importance of nutrition in disease incidence in other crops but noted that few details of the factors which predispose oil palms to infection have been established. One approach to control has been to search for resistance to, or tolerance of, disease, both within *Elaeis guineensis* material and in interspecific hybrids with *E. oleifera*. Good progress has been made in relation to *Fusarium* wilt with *E. guineensis*, and there is scope for this approach with *Ganoderma*. Hybrids may be tolerant to fatal yellowing, but poor fruit set because of pollen sterility limits their value at present. Readers should refer to specialist works if pesticide treatment is considered. We have not given details here, because new pesticides are constantly being developed and older ones withdrawn.

A comprehensive work on diseases, which is still relevant, was provided by Turner (1981). Nutritional disorders are described in Chapter 12; this chapter deals with conditions caused by pathogenic organisms and with important disorders of unknown cause.

It is convenient to deal with diseases according to the stage of growth at which the palm is attacked and the organs affected. However, there is much overlap, and some diseases have been rather arbitrarily listed for a particular stage of growth. Information on a number of 'minor' diseases is summarised in Table 13.1. Some of these caused serious losses at one particular time and place, but have not been a problem since. Several were observed in the early days of oil palm cultivation, when fertiliser inputs were low or absent and poor nutrition was probably an important predisposing factor

In the past, pathologists set much store by Koch's postulates, conditions to be met before the cause of a disease was considered proven. The requirements that typical disease symptoms be obtained after inoculation with a supposed pathogen, and that the pathogen then be reisolated from the inoculated plants, have not been met for several oil palm diseases. Even where Koch's postulates are proven, predisposing environmental factors may still be important, while failure to meet the postulates may be because a predisposing factor is absent, rather than indicating non-pathogenicity. DNA-based detection is now very important and should be attempted whenever possible.

13.1 DISEASES OF GERMINATING SEEDS

The only disease of any consequence is brown germ.

Symptoms and distribution: Brown spots appear on the emerging 'button'. These spread and coalesce as the embryo develops, and the tissues become slimy and rotten. The disease may occur wherever seeds are being germinated.

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Table 13.1 Minor oil palm diseases: diseases either causing little economic damage, or of rare occurrence

Disease	Symptoms	Cause	Location	Ref.	Comments
Nursery diseases					
Anthracnose	Necrotic lesions, slow growth	Various (see Ref. 1)	Widespread	1, 2	
Seedling blight	Elongated spots with yellow halo	*Curvularia eragrostidis* (?)	Malaysia	1	Treatment similar to *Cercospora*
Cylindrocladium	Brown lesions with white centres	*Cylindrocladium macrosporum*	Ivory Coast	1	
Leaf spotting		Various (see Ref. 1)	Widespread	1	See Ref. 1 for control recommendations
Nursery spear rot	Rotting of median leaflets of the spear leaf	*Phytophthora* sp.	Congo	3	Not serious enough to need control
Corticium leaf rot	Rot at base of unopened leaves	*Corticium solani*	Congo, Malaysia	3	See Ref. 1 for control recommendations
Dry bud rot	Yellow patches on leaves, then dry rot of spear	Unknown	Ivory Coast	4	Similar disease transmitted by *Sogatella* in coconut (see also patch yellows, below)
Chlorotic ring	Conspicuous mottling	Potyvirus, related to sugarcane mosaic virus	S. America	5, 6	
Mature palm leaf diseases					
Necrotic spot	Brown spots, orange halo, premature withering	*Cercospora elaeidis*	Africa	7, 9	Different strain of *C. elaeidis* from that causing freckle (Section 13.2.1)
Crusty spot	Orange spots with black crust in centre	*Parodiella circumdata*	Africa	7	Mainly affects oldest leaves
Genetic orange spotting	Yellow or orange leaf spots	Cadang-cadang viroid detected	Widespread but rare	8, 14	Transmission not demonstrated
Algal leaf spot	Pinpoint yellow spots on upper surface	*Cephaleuros virescens*	Widespread	1, 9	Effect on yield unknown
Patch yellows	Pale circular lesions in pairs on opposite leaflets	*F. oxysporum*	Africa	1, 16, 17	Sporadic. Different strain of *F. oxysporum* from wilt (Section 13.4.2)
Finschhafen disorder	Yellow-bronze leaves	Plant hopper *Zophiuma lobulata*	PNG	15	Direct effect of feeding, not a pathogen
Other mature palm diseases					
Armillaria trunk rot	Similar to vascular wilt, but leaf bases rot	*Armillariella mellea*	Congo	10, 11	Incidence has decreased since the 1950s
Basal decay	Sudden death of leaves, following trunk rot	Unknown	Africa, Malaysia	1	Rare
Charcoal base rot	Black rot at base, leaves chlorotic	*Ustulina* sp.	Malaysia	1, 12	Pathogenicity not proven
Stem wet rot	Internal rotting	Unknown	India	13	

References: 1: Turner (1981); 2: Robertson (1956); 3: Kovachich (1957); 4: Renard and de Franqueville (1989a); 5: Rivera *et al*. (1996); 6: Morales *et al*. (2002a); 7: Kovachich (1956b); 8: Hanold and Randles (1991); 9: Robertson *et al*. (1968); 10: Wardlaw (1950a); 11: Moureau (1952); 12: Thompson (1936); 13: Chander Rao (1997); 14: Randles *et al*. (2009); 15: Gitau *et al*. (2011); 16: Bull (1954); 17: Kovachich (1956a).

Cause: Duff identified a variety of *Aspergillus niger* from diseased embryos in Nigeria and demonstrated pathogenicity by inoculation and reisolation (A.G. Prendergast, pers. comm., 2001). This may not be the only cause, though: Turner (1981) listed 27 fungi associated with the disease, of which *Aspergillus* spp. and *Penicillium* spp. were most frequent. Many are secondary invaders, as are bacterial species.

Control: Brown germ develops most readily under moist conditions at a temperature of 38–40°C; use of the wet heat treatment for germination (see Section 8.1) therefore encourages its spread. Although sanitary measures in the germinator may reduce incidence, the best method of control is to adopt the dry heat treatment method of germination, since the seeds are dry when being heated at 39.5°C, and when germinating, they are at around 27°C, a temperature too low to encourage the growth of the organisms.

13.2 SEEDLING DISEASES

Turner (1981) noted the importance of nursery management in minimising disease susceptibility. With adequate water supplies and balanced nutrition, nurseries in many areas remain largely free of serious diseases, and investigations into disease outbreaks may primarily involve examination of growing techniques, rather than a search for a pathogen. This emphasises the importance of predisposing factors in disease development, as mentioned previously.

13.2.1 *Cercospora* leaf spot or freckle

Distribution: *Cercospora* leaf spot is widespread throughout Africa but has not been reported in Asia or America. It is primarily a disease of nursery seedlings which sometimes starts in the prenursery and is frequently carried to field plantings, where it can survive for many years.

Symptoms: The youngest leaves of nursery seedlings become infected, and minute translucent spots surrounded by yellowish-green haloes enlarge and become dark brown. Conidiophores, emerging through the stomata in the centre of the spots mainly on the undersurface of the leaf, produce conidia which give rise to further, surrounding spots. This results in a freckled appearance, but later, the lesions coalesce and the tissue dries out to become greyish brown and brittle. The disease tends to become aggressive as the leaves age, and the process described earlier may proceed very rapidly at certain periods of the year. In West Africa, this is usually the middle or end of the wet season.

Cause: *Cercospora elaeidis*. Proof of pathogenicity was obtained by Kovachich (1954) in Congo and Robertson (1956) in Nigeria. Nitrogen manuring may cause a small increase in the incidence of freckle in the nursery, but potassium substantially reduces it. Small favourable effects of phosphorus have also been noted (Robertson, 1960).

Effect on yield: Duff (1970) showed that *Cercospora* could depress yields by more than 10% over the first 7 years of production, but Jacquemard (1998) describes the disease as depressing nursery growth, but not economically important.

Control: The obvious course is to eradicate the disease in the nursery and to prevent reinfection of the young seedlings in the field. Jacquemard (1998) recommended fungicide spraying, but if this is not done, all old dry leaves and any others badly infected should be removed by pruning in the nursery. In the field, excessive pruning may reduce growth and delay flowering, whereas failure to prune may increase the severity and prolong the incidence of the disease. A compromise suggested by Hartley (1988, p. 585) was to remove and burn any leaf that showed dead or badly necrotic areas over more than one-third of its total surface, but it is not clear whether this would control the disease.

Elaeis oleifera progenies planted in Africa have shown susceptibility to the disease; interspecific hybrids are rather less susceptible. There are significant differences between *E. guineensis* progenies in *Cercospora* susceptibility (Robertson, 1963), and Duff (1970) suggested that breeding for tolerance would be worthwhile. With good control being obtainable with fungicides, however, it is doubtful whether breeding for tolerance can be justified.

13.2.2 Other seedling leaf diseases

Various diseases of minor importance or rare occurrence are listed in Table 13.1. Some nursery diseases, for which many causes (including virus infection) have been suggested but none established, have been constant enough in their symptoms to acquire distinctive names. These include bronze streak, ring spot and infectious chlorosis (which, despite the name, appears not to be infectious). Turner (1981) gave information on these.

Leaf distortions occur in young prenursery and nursery seedlings at the bifurcate leaf stage (Plate 13.1) and have been described as:

- Leaf crinkle, in which the lamina between the veins is folded in lines across the leaf.
- Leaf roll, in which the lamina is rolled under the leaf, giving it a spiky appearance.

(A)

(B)

(C)

Plate 13.1 Certain abnormalities of nursery seedlings. (A) Leaf crinkle; (B) leaf roll and (C) collante.

- Collante, in which the lamina between the veins becomes laterally compressed at a band about halfway along the leaf, forming a constriction (Gunn *et al*., 1961). Mathews *et al*. (2010) showed that this may result from spraying with hexaconazole.

Other abnormal conditions that necessitate culling in the nursery are described in Section 8.2.2.4. Malformed seedlings are not uncommon in the prenursery and are variously attributed to the after effects of brown germ or to incorrect orientation of the germinated seed at planting.

13.2.3 Seedling spear and bud rots

Several nursery spear and bud rots have been recorded, but none appears to be serious (Table 13.1).

13.2.4 Seedling root diseases: blast disease

13.2.4.1 *Distribution*

Blast has been a serious nursery disease throughout West Africa. It was particularly severe in the Ivory Coast and of considerable importance in Nigeria and Cameroon. The disease has also been recorded from Malaysia (Turner, 1966b), Indonesia, Brazil (Cardoso, 1961) and Colombia, and Turner (1981) considered that it could occur in any country where climatic conditions and nursery techniques are likely to favour its development.

13.2.4.2 *Symptoms*

The symptoms of blast disease were described in detail by Bull (1954) and Robertson (1959a). Affected seedlings lose their normal gloss and become dull and flaccid, the leaf colour changing successively to olive green, dull yellow, purple or umber (at the tips), finally drying out to a brittle dark brown and grey (Plate XIA). Necrosis of the central spear is usual, and death occurs in a few days. In a small percentage of cases, the rot may not reach the growing point; the seedling then survives but as a weak and unacceptable plant. In many of the roots of diseased plants, the parenchymatous tissue within the hypodermis has been destroyed from the tip towards the stem base, the stele remaining loose within the hollow cylinder. When the rate of cortical rotting becomes greater than the rate of production of new absorbing roots, desiccation and death follow rapidly.

13.2.4.3 *Causes*

Two quite distinct causes for this disease have been convincingly demonstrated. Robertson (1959b) showed that it was caused by a joint infection of the roots by two fungi and satisfied Koch's postulates in proof of this. Subsequently, it has been demonstrated in the Ivory Coast that an insect vector is involved. Turner (1981) discussed the possibility of there being several causes of blast or of two apparently different causes being linked. The leaf symptoms are essentially those of acute water stress and might be caused by any severe damage to the roots.

Robertson (1959b) isolated *Rhizoctonia lamellifera* from decaying cortical tissue in the roots and a *Pythium* species, probably *P. splendens*, from primary infections of the root tips, where it was shown to penetrate the cells and cause their collapse. In laboratory experiments, Robertson (1959b) showed that the *Pythium* may be parasitised by *R. lamellifera*. A mixed inoculum of *Pythium* and *R. lamellifera* produced more extensive root rotting, and the leaf symptoms were more pronounced than with either species individually. Inoculation with *R. lamellifera* alone was only successful when the roots had been artificially damaged, while with *Pythium* inoculation, damage was confined in the root tips. In all of these cases, pathogenicity was established by reisolation of the organisms. It was concluded that *R. lamellifera* plays an important part in blast disease in the destruction of cortical tissues and that it gains access either through a prior invasion by *Pythium* sp., which it parasitises, or through root damage from some other cause. The *Pythium* species was thought to be important through its role as a primary invader and its ability to penetrate the parenchyma cells and develop within them.

Subsequent to Robertson's work, quite different results were obtained in the Ivory Coast, where the blast problem had always been severe. It was noted that plants grown in metal cages covered with mosquito netting showed less than 1% blast in comparison with 15% outside in unshaded areas. A polybag nursery trial compared a completely closed cage with very fine netting (to give the minimum shading effect), an open-top cage, plots treated twice weekly with parathion and unshaded control plots which had natural grass between the bags (Renard *et al.*, 1975). The results were as follows:

	Treatment:			
	Completely enclosed	Open-top cage	Parathion	No treatment
Blast (%) by end of Dec.	0	6	27	46
Blast (%) by end of Jan.	2	9	35	63

These results led to the hypothesis that an insect vector was involved: the cages excluded insects, while the insecticide reduced numbers, but did not completely exclude them. Later, it was established that the insect was *Recilia mica* (Hemiptera: Delphacidae), for which the grasses *Paspalum* spp. and *Pennisetum* spp. were alternate host plants (Julia, 1979). Julia found that *R. mica* moved to the palm nursery from surrounding grass only in October and November. De Franqueville *et al.* (1991) found that

the insects were most frequent in November and December, and introduction of *R. mica* to caged palms gave the highest disease incidence at that time. The exact connection between *R. mica* and blast disease has not been determined. Renard (1981) showed that tetracycline gave good protection, suggesting the possibility that a mycoplasma is involved, but this has not been confirmed.

13.2.4.4 *Control*

Shade was regarded as the only protection against blast until the role of *R. mica* in transmission of the disease was discovered (Quencez, 1982). It still remains unclear whether blast has the same cause all over West Africa, but in general, shade as a method of control has been abandoned in favour of systemic insecticides.

It has been shown both in the Ivory Coast (Bachy, 1958) and in Nigeria (Robertson, 1959a) that if the seedlings are either very young (1–4 months) or old (11 months or over) at the beginning of the blast season, the casualties are few. De Franqueville *et al.* (1991) showed that seedlings with four or five leaves were more susceptible than older seedlings. In seedlings with two leaves, the disease developed slowly but eventually reached the same level as in plants with four or five leaves.

Desmier de Chenon (1979) found that the removal of grasses in the vicinity of the nursery reduced blast incidence; the application of an appropriate insecticide was also effective and made it possible to eliminate the shade which had always been found necessary in the Ivory Coast (Quencez, 1982). De Franqueville *et al.* (1991) tested several insecticides.

The control measures for blast in West Africa may be summarised as follows:

1. Time the planting of nurseries to ensure that the blast season has passed before the seedlings reach the susceptible stage. This will involve planting well-developed prenursery seedlings early in the rainy season and ensuring their rapid growth.
2. Pay particular attention to irrigation during the short dry season and make sure that polybags have a sufficient, though not excessive, water supply throughout the nursery period.
3. Where *R. mica* is prevalent, spray out host grasses in the vicinity of the nursery, and apply a suitable insecticide monthly.

Clones differ in their susceptibility to blast (IRHO, 1992b), but breeding for resistance would not be a sensible approach to an easily controlled nursery disease.

13.3 ADULT PALM LEAF DISEASES AND DISORDERS

13.3.1 Crown disease

13.3.1.1 *Distribution*

The disease was particularly prevalent in the early Deli plantations but can be found worldwide. All but the most severe cases normally recover during the second year after planting, and effects on yield are then not serious.

13.3.1.2 *Symptoms*

A palm suffering severely from crown disease has many of its leaves bent downwards in the middle of the rachis; at this point, the leaflets are absent or small and ragged (Plate XVA). These symptoms originate in the spear leaf, where the folded leaflets begin to show a rot of their edges or centre (Kovachich, 1957). This rot spreads throughout the central portion of the leaf so that when the leaf unfolds, the leaflets of this section are disintegrating or already missing. The rachis bends at the point where the leaflets are absent. In severe cases, all the leaves surrounding the spear may be bent down, and the spear itself may have a rot of its terminal portion which turns brown and hangs down. Under these extreme circumstances, crown disease may have a severe effect on early development and yields. The disease normally affects palms in the second to fourth year in the field, but instances have been reported in the nursery and up to 10 years of age.

13.3.1.3 *Causes*

No pathogen has been identified, and it was assumed in the early days that the disorder was 'physiological' and might be inherited. The latter assumption proved correct (see Section 13.3.1.5). It was suggested that incidence might be affected by magnesium and potassium manuring (Hasselo, 1959). Breure and Soebagyo (1991) compared two sites and observed lower leaf boron levels (and rather lower magnesium) at the site with more severe incidence of crown disease.

Thompson (1934) stated that the 'decreased rigidity' of the rachis was due to insufficient lignification of the parenchymatous tissue, and Monge *et al.* (1994) found that the fibres of vascular bundles in the rachis were very thin walled in affected leaves. The leaves, however, tend to be quite rigid, although bent. Monge *et al.* considered that crown disease and spear rot might be manifestations of the same disorder. Alvarado *et al.*

(1997) also suggested the two were associated, but in their trials with a susceptible progeny, poor drainage reduced the level of crown disease but increased spear rot incidence (see Section 13.5.1). Boron application also reduced crown disease incidence.

13.3.1.4 *Effects on yield*

Severity of crown disease varies between environments (Breure and Soebagyo, 1991). In Papua New Guinea (PNG), where incidence can be severe, Table 13.2 shows a yield loss of about 6% in the first 6 months of production, and just under 2% over the next 2.5 years. Breure and Soebagyo (1991) estimated losses of 4.5% over the first 3 years of production in North Sumatra, with the greatest loss in the first year. Kohar *et al*. (2002) considered that yield was only affected in very severe cases.

13.3.1.5 *Control*

De Berchoux and Gascon (1963) showed that pure Deli progenies in the Ivory Coast were highly susceptible. La Mé material, free of crown disease, gave crosses with Deli which were also free of crown disease, but Congo material, which showed several cases of the disease, gave Deli × Congo crosses with a quarter to a half of the palms showing the disease. The authors postulated that susceptibility to crown disease is due to a single recessive gene. Some examples from their results are given in Table 13.3, showing close agreement with the expected segregations. Thus, it appeared practicable to select palms that would not produce susceptible individuals in their progeny; in particular, it would be valuable to have *pisifera* shown to be homozygous for absence of crown disease (CC), as the Congo (Sibiti) palm S127P appeared to be.

Blaak (1970b) found that with some palms in Cameroon, the expected inheritance occurred. However, other crosses gave segregations that could best be explained by the presence of an inhibitor gene which, when homozygous, suppressed expression of the recessive gene. Examples from Blaak's results are also given in Table 13.3.

If susceptibility is controlled by only one or two genes, then its elimination from a breeding programme should be easy, although Blaak (1970b) pointed out that the presence of an inhibitor gene complicates selection, since detection of a palm of *cc* genotype (susceptible) is only possible by test crossing with a palm that is known not to have the inhibiting gene. The losses noted by Dumortier (1998) are quite small, but 6% extra crop over the first 6 months of production is clearly worth having. However, Dumortier showed that *pisifera* DM742.207 transmitted crown disease susceptibility to its offspring, and yet it consistently gave the highest yielding progenies in most environments (Dumortier and Konimor, 1999). Thus, it is understandable that some oil palm breeders have not regarded eliminating susceptibility from their programmes as being very important.

13.3.2 Leaf wither, *Pestalotiopsis* leaf spot or grey leaf blight

Distribution: A virulent type of leaf withering has been troublesome in parts of Colombia, Ecuador and Honduras and has caused much defoliation, with significant effects on yield. It is also commonly seen in Colombia on *E. oleifera* palms.

Symptoms: The first symptom is the appearance of small brown spots with yellowish haloes. These spots

Table 13.2 Reduction in yield of palms with crown disease

Class	Severity of symptoms	Palms in class (%)	Yield loss/palm (%)		Yield loss/ha (%)	
			1st 6 months	7–38 months	1st 6 months	7–38 months
0	No symptoms	80.8	0	0	0	0
1	Slight	8.0	15.1	2.7	1.2	0.2
2	Mild	6.3	22.8	3.4	1.4	0.2
3	Severe	4.5	36.1	4.5	1.6	0.2
4	Very severe	0.25	54.3	34.0	1.4	0.8
5	Extremely severe	0.12	43.2	27.1	0.5	0.3
	Total loss/ha (%)				6.1	1.7

Source: From Dumortier (1998).
Loss per hectare = loss/palm × palms in class.

Table 13.3 Incidence of crown disease in various crosses in the Ivory Coast and Cameroon and expected segregation with and without Blaak's inhibitor gene; expectations shown are those if the inhibitor were dominant

Presumed genotypes	Cross	Observed (%)		Expected (no inhibitor) (%)		Expected + inhibitor (%)	
		Healthy	Affected	Healthy	Affected	Healthy	Affected
de Berchoux and Gascon (1963)							
cc × cc	D115D selfed	0.5	99.5	0	100		
CC × cc	L10T × D115D	100	0	100	0		
cc × CC	D115D × S127P	100	0	100	0		
Cc × cc	L219T × D115D	57	43	50	50		
Cc × cc	S7T × D115D	52	48	50	50		
Cc × Cc	L219T × D10D	81	19	75	25		
Cc × Cc	L239T × D128D	71	29	75	25		
Blaak (1970b)							
Cc ii × Cc ii	1.2229 selfed	79	21	75	25	75	25
Cc II × Cc ii	3.417 × 1.2229	77	23	75	25	75	25
Cc II × Cc II	3.417 selfed	100	0	75	25	100	0
Cc ii × Cc ii	5.37 selfed	76	24	75	25	75	25
Cc II × Cc ii	3.415 × 5.37	96	4	75	25	100	0
Cc II × Cc II	3.415 selfed	100	0	75	25	100	0
Cc Ii × Cc ii	15.4624 × 5.1295	85	15	75	25	87.5	12.5

Source: Amended from de Berchoux and Gascon (1963) and Blaak (1970b).

soon coalesce into brown necrotic areas which spread over the leaflet tissue and later become grey and brittle (Plate XIC). There is a sharp line between the brown and grey areas, and in the latter, a species of *Pestalotiopsis* is found, black specks indicating the location of spore-bearing acervuli (C.W.S. Hartley, 1974, unpubl.).

Cause: The disease has been described as *Pestalotiopsis* leaf spot and grey leaf blight in Malaysia, but there, the fungus is only associated with old and near-moribund leaves and is not considered of economic importance (Turner, 1981). The severity of the attacks in Latin America seem to be due to the easy access given to the young leaves by the feeding activities of insects, but it is also possible that the strains of the *Pestalotiopsis* species involved are more aggressive. Genty *et al*. (1975) showed that *Leptopharsa gibbicarina* (Hemiptera: Tingidae) was the principal means of infecting young leaves. This insect punctures the leaflets alongside the midribs, producing whitish spots with their surrounds stained with excrement (Genty *et al*., 1983). In Ecuador, *Peleopoda arcanella* has been implicated in assisting infection of the leaves by *Pestalotiopsis* sp. (Turner, 1981). Two species of *Pestalotiopsis* are the usual entrants, but species of *Helminthosporium*, *Curvularia* and other genera may also gain access to the leaflets (J.A. Aldana, 2003c).

Effects on yield: The disease has caused considerable defoliation on some plantations, and as would be expected, this has been followed by serious yield decline. Bunch production falling from 18–20 to 12–15 t fresh fruit bunch (FFB)/ha in adult areas, and from 11 to 7–8 t/ha in young plantings has been reported (Jiménez and Reyes, 1977).

Control: The attacks on moribund tissue in Africa and Malaysia have often been associated with magnesium deficiency symptoms (Bull, 1961a). In Colombia, the disease is less severe if potassium and magnesium are in balance (P.L. Gomez, pers. comm., 2001).

Control of *L. gibbicarina* by aerial spraying of propoxur, fenitrothion or phosphamidon has had considerable success in checking the disease, but more recently, trunk injection or root absorption of monocrotophos has been advocated; as noted in Section 14.1.4, these methods of application have advantages in terms of selectivity. Monocrotophos injection has also been successful in Honduras (Vessey, 1981). The possibility of biological control has been studied (Genty *et al*., 1983; Guerrero, 1985), and Mendez (2000) described successful management of the disease by encouraging *Crematogaster* ants and using *Beauveria* and *Paecilomyces* fungi to control the insect vector. Aldana (2003c) showed that the

population of *L. gibbicarina* declined after introducing *Crematogaster*, but did not give data for control plots without ants. He also showed that introducing ants costs much less than treatment with monocrotophos.

13.3.3 White stripe

Distribution: The condition is sporadic and, in Asia, is said to be more common on alluvial soils, particularly organic clays or mucks. Affected palms have reduced yield.

Symptoms: Narrow yellowish stripes are found on each side of the leaflet midrib and extending its whole length (Plate X D). The stripes are sharply divided from the adjoining green (often dark green) tissue. Affected palms usually recover, and Rajaratnam (1972b) reported that the chlorotic tissue might turn green after about 7 months and that the symptom was more prominent in young leaves than in old. He also showed that chlorosis was due to failure of the palisade mesophyll cells to elongate and that apparent recovery was through an increase in the chlorophyll content of the spongy mesophyll and not through development of the palisade cells. Turner (1981) stated that symptoms are more severe in Malaysia than elsewhere and that typically they appear at 2–3 years of age, becoming more severe at 3–5 years and then becoming chronic.

Effects on yield: Moderately affected palms yielded 15% less than healthy neighbours and severely affected palms nearly 50% less (Rajaratnam, 1972b). However, Tohiruddin *et al*. (2002) found that yield was sometimes positively correlated with white stripe incidence (see below).

Cause: It has been suggested that the disorder is of genetic origin. A certain *tenera* × *dura* cross showed similar percentages of white stripe when planted in Ivory Coast and in East Cameroon; also, certain Deli selfs in Ivory Coast showed the symptoms, while others did not (Ollagnier and Valverde, 1968; Gascon and Meunier, 1979). However, the general view is that the cause is nutritional: boron deficiency and a high leaf N/K ratio have both been suggested. In boron-deficient seedlings, Rajaratnam (1972a) found chlorotic patches, in which the palisade mesophyll had not developed, but the patches did not form stripes. In one trial, palms showed a degree of recovery when boron was applied, but in another, they did not.

Turner and Bull (1967) considered that a nitrogen/potassium imbalance was the main cause of white stripe, but Rajaratnam (1972b) observed the disorder in palms with N/K ratios well below that suggested as critical by Turner and Bull. Tohiruddin *et al*. (2002) found a positive correlation of white stripe incidence with N/K ratio in only one of five fertiliser trials studied. In that trial, there was a negative correlation with leaf K content, and a stronger correlation with rachis K content (rachis K is more sensitive to applied K than leaf content; see Section 12.4.2.2). In unfertilised palms in that trial, rachis K content was below 0.6%, much lower than in the other trials. The correlation of white stripe incidence and yield was negative and statistically significant.

In two other trials, where leaf N content exceeded 2.8%, white stripe incidence was positively correlated with leaf N content and positively (but not significantly) correlated with yield. There were no correlations with leaf boron content. These results support the N/K imbalance hypothesis, but suggest that the leaf N/K ratio is not an adequate indicator of the imbalance.

Treatment: Turner (1981) suggested substantial applications of potash with reduction of nitrogen applications, but Tohiruddin *et al*. (2002) distinguished between white stripe caused by low K status and by high N status. Where rachis K is low, potassium fertiliser should be applied. Where leaf N exceeds 2.8%, reducing N input may reduce white stripe incidence, but it is also likely to reduce yield. In this situation, symptoms can be expected to disappear with time, since as palms grow older, leaf N content tends to fall, and K reserves increase.

13.3.4 Leaf mottle (mancha anular)

Distribution: This condition, which often leads to death of the palm, has been reported from Ecuador and Peru and is described by Turner (1981). It has been called ring spot, but that term is also used for a nursery disease.

Symptoms: When the spears open, they fail to become fully green, and spots of pale tissue remain. These may be circular or elongated and almost rectangular and may form almost continuous streaks. Younger fronds then become chlorotic. This leaf symptom is followed by the rotting of the root system and spear, although Turner (1981) considered the spear rot to be secondary. Developing bunches may also rot. Palms may die within 3 months of the first symptoms, but some palms continue to grow and yield for several years, despite showing leaf symptoms.

Cause: Martínez (1988) suggested that a virus might be the cause, and Morales *et al*. (2002b) found a *Foveavirus* consistently associated with the disease. Renard and de Franqueville (1989a) described nursery dry bud rot as

being similar (see Table 13.1). A similar disease of coconuts is transmitted by two species of *Sogatella* (Homoptera: Delphacidae) (Julia and Mariau, 1982).

Treatment: Fungicides, insecticides and antibiotics have been tested without effect, but a virus would not be affected by these. Diseased palms tend to be scattered throughout a field, but incidence is much higher with dense grass cover than with a legume cover (Dzido *et al.*, 1978). A good leguminous cover should therefore be maintained in areas subject to this condition.

13.3.5 Minor leaf diseases

The oil palm leaf is susceptible to patchy discoloration and necrosis from a variety of minor pathogens, some of which are listed in Table 13.1, and to surface covering by epiphytic and saprobic organisms. These often cause the older leaves to appear far from healthy, but as the oldest leaves contribute relatively little photosynthetically, the effects may be small.

Black 'sooty mould' is often found to grow on the older leaves of adult palms and occasionally spreads over a large proportion of the leaf surface, giving the palms a blackish-grey appearance. Sooty moulds usually grow on honeydew, the exudate of partially digested plant sap from plant lice, but it is not certain whether this is always the case in oil palm (Wood, 1968a).

Several of the most common fungi to be found in Africa as constituents of the epiphytic flora are listed by Turner (1971). Among these, the Ascomycetes *Apiospora* sp., *Meliolinella elaeidis* and *Meliola elaeis* may be mentioned. *M. elaeidis* is recorded as also being found in Costa Rica on *E. oleifera*. Epiphytic flora may appear on the upper or lower surface of the leaves. In West Africa, the black mould usually found on the upper surface consists of discrete circles of about 5 mm diameter; on the lower surface, the black mould is in irregular patches of less dense material. In Malaysia, sooty moulds of *Brooksia*, *Ceramothyrium* and *Chaetothyrium* spp. develop on insect secretions on the leaves (Williams, 1965; Turner, 1981). *Brooksia tropicalis* is common in Africa.

Lichens are often found among the epiphytic flora on oil palm leaves, forming small grey–green encrustations on the upper surface of the leaflets (Turner, 1971).

13.4 STEM AND ROOT DISEASES

Root and stem diseases are characterised by fracture and drying out of fully developed leaves, leaving the spear leaf and some surrounding leaves standing erect. These early symptoms may be accompanied by a change in colour, drying out or wilting of one of the more erect younger leaves. In contrast, bud and spear rots tend to be characterised by symptoms in the centre of the crown. The spear leaf may be directly affected or the surrounding leaves show a sudden chlorosis. Successive spear leaves may be shortened, have peculiar 'little leaf' formations or cease to develop, leaving a palm with an empty centre. These general symptom differences between stem and root diseases, on the one hand, and bud and spear rots, on the other, give a rough guide when deaths occur or alarming disease symptoms appear, but dissection of the palm must follow to determine exactly where the site of destruction is. The site of decay with the root and stem diseases is the bole, trunk or roots, the disease killing the palm by denial of water and nutrients to the crown. Bud and spear rots, in contrast, kill the palm by growing towards and reaching the single growing point.

13.4.1 Dry basal rot

13.4.1.1 *Distribution*

This disease appears to be confined to West Africa, and although the pathogen is a common soil inhabitant, the disease was not recorded in epidemic form until 1960. One estate in Nigeria was devastated, and thereafter, minor outbreaks occurred in several parts of Nigeria, West Cameroon and Ghana. In the first epidemic, deaths were common, but recovery then became more usual, and further serious outbreaks have not been reported.

13.4.1.2 *Symptoms*

The foliar symptoms, which usually appear at the end of the dry season, are preceded by extensive bunch and inflorescence rot. The rachis of certain leaves then becomes fractured submedianly, although the leaflets remain green for a considerable period before they eventually die (Plate 13.2A). It is quite common for a complete ring of leaves to exhibit the submedian fracture while the upper leaves are still erect, and this gives the newly affected palm its characteristic appearance. Later, the upper leaves and the spear will be similarly affected, and the palm may die. A palm that survives may take several years to come back into bearing.

The characteristic internal symptom of the disease is a dry rot at the base of the trunk (Plate 13.2B). This rot is well established by the time the primary leaf

(A)

(B)

Plate 13.2 Dry basal rot, *Ceratocystis paradoxa*. (A) A severely infected palm showing sub-median fracture of the lower leaves. (B) A palm showing external symptoms of the disease, dissected to expose the dry rot at the base of the trunk.

symptoms are apparent. In the transition zone between rotted and healthy materials, many vascular bundles are necrotic, and it is possible to trace infection from an infected root or leaf base into the base of the trunk.

13.4.1.3 *Cause*

The cause of dry basal rot was shown by Robertson (1962a, c) to be the ascomycete *Ceratocystis paradoxa*, the imperfect stage of which is known as *Thielaviopsis paradoxa*. The latter has been implicated in fatal yellowing (see Section 13.5.2). *C. paradoxa* is a soil inhabitant widely distributed throughout the tropics of Africa and Asia and causes diseases of several other crops. Its sudden appearance in West Africa as the cause of a serious condition was unexpected and led to investigations of predisposing factors. An epidemic at Akwukwu in Nigeria and minor outbreaks at the Nigerian Institute for Oil Palm Research (NIFOR) Main Station occurred on fields with little clay in the profile. This led to the belief that incidence might be connected with soil–climate relationships. A further outbreak at NIFOR in 1967 followed a severe dry season.

At the NIFOR Main Station, incidence in one field of 9-year-old palms was mainly confined to progenies having the same female parent. Robertson found he could infect all seedlings through a root dipping technique; nevertheless, inoculated progeny lines planted in the nursery showed marked differences in disease incidence (Robertson, 1962b). Selection for resistance is therefore a promising line for the future should the disease once more become important.

13.4.2 *Fusarium* wilt or vascular wilt

Since its description by Wardlaw (1946) in Congo, *Fusarium* wilt has been considered one of the most menacing of oil palm diseases. The disease has been observed on plantations in Congo, Nigeria and West Cameroon, in the Ivory Coast and elsewhere in West Africa. It has also been recorded in Brazil and Ecuador. Its effects are serious: in the acute form, the palm rapidly dies, while chronically affected palms yield little or nothing.

13.4.2.1 *Symptoms*

In the commoner, chronic form of the disease in mature palms, the older leaves become desiccated and the rachis breaks near the base or at some distance from the base, the ends of the leaves hanging downwards. This feature has been used to distinguish the disease from *Ganoderma*, in which the leaves collapse at the base and closely cloak the stem. The disease usually proceeds gradually along several leaf spirals, with younger leaves becoming successively affected. The erect and still green leaves in the crown become successively more

reduced in size and are often chlorotic, and the palm may stay in this state for several years before the crown eventually collapses.

Occasionally, a mature palm suffers a rapid death through an acute attack. The leaves dry out and die while still in an erect position and then snap off about 1 m or more from the trunk, usually during strong winds (Plate XIIB). All stages between the acute and chronic forms are encountered.

Symptoms of the disease in young palms in which no trunk has yet been formed are somewhat different. In these palms, the 'lemon-frond' symptom is frequent: a leaf somewhere in the upper middle part of the crown (fourth to 15th leaf) develops a bright lemon-yellow colour before drying out from the tip to the base. Leaves at about the same level then turn yellow and dry out, to be followed by some of the younger leaves, which will die while many of the older ones remain green. Newly developed leaves become successively smaller (Plate XIIA), and death of the whole palm usually takes less than a year. The striking lemon-frond symptom is not always seen, and in southern Congo, a general yellowing of the leaves before death was more usual.

Internal symptoms of the disease are quite distinctive (Plates XII and XIII). The vascular bundles are normally pale yellow or whitish, but when diseased, they become brownish grey or black, and a cross section of the trunk therefore shows a speckled appearance (compare Plates XIIID and E). Discoloration, which is associated with the presence of gum, is confined to the xylem vessels (Plate XIIE); blackened fibre strands do not indicate vascular wilt. Such blackening often occurs in older palms and sometimes in other conditions, and the inexperienced observer can therefore be misled into a wrong field diagnosis. Moureau (1952) pointed out that although discoloration of the xylem vessels is normal in palms over 20 years old, in these palms, the blackening decreases towards the top of the palm instead of becoming accentuated as in the case of wilt. In palms with acute wilt, A.G. Prendergast (pers. comm., 2001) found large cavities, often more than 30 cm across, in the stem within 2 m below the crown; these cavities were filled with a dense mass of *Fusarium* mycelium.

The disease is normally recognised in the field by the external symptoms, but Mepsted *et al.* (1991) showed that the internal symptom of vascular browning could be detected non-destructively by taking tissue samples with an auger (Plates XIIC and D). Using this method, they found that 25% of a sample of palms, classed as healthy by external appearance, showed internal symptoms of the disease (latent infection). Buchanan (1999) used the auger method and found a poor correlation between external and internal symptoms. In one palm family, 54% of apparently healthy palms showed internal symptoms, while, conversely, 40% of palms with external symptoms showed no internal browning. Buchanan noted that simultaneous infection by *Cercospora* and *Ganoderma* may produce external symptoms somewhat similar to those of *Fusarium* wilt, so erroneous identification of wilted palms may partly explain his results. Cooper and Rusli (2014) mentioned a DNA probe specific to *Fusarium oxysporum* f.sp. *elaeidis*, which could be very useful for detecting the pathogen.

Palms may recover, and recovery has become increasingly common as plantings of tolerant material are extended (Renard *et al.*, 1991).

13.4.2.2 *Cause*

F. oxysporum f.sp. *elaeidis* (abbreviated in the following to *Foe*). One isolate from Congo was found to be *F. oxysporum* var. *redolens* (Ho *et al.*, 1985). There is some evidence that differences in aggressiveness of the pathogen may account for the difference between the acute and chronic forms of the disease (R.M. Cooper and M.H. Rusli, pers. comm., 2014). The pathogen is soil borne, and it usually enters the palm through the roots, growing along the stele, which becomes blackened. Infection can take place through wounds in the stem base and through uninjured roots (Kovachich, 1948). Renard (1970) considered that entry of the mycelium was much impeded by lignification even with wounding and that rapid infection was mainly through the transmission of spores in the vascular system. Locke (1972), working with seedlings, showed that the pathogen is confined to the conducting elements of the xylem (Plate XIIIB) and can reach the stele from the tip of a lateral root or the damaged cortical tissue of a pneumathode. From the roots, the mycelium penetrates into the xylem vessels of the vascular strands, where conidia and chlamydospores are also found. Locke considered that the plant had little defence against serious infection in spite of resin formation and tyloses (Plate XIIIC). However, Paul (1995) indicated that in resistant genotypes, gels and tyloses were produced earlier and accumulated to higher levels, and fungal colonisation was restricted compared with susceptible genotypes. In the latter, production of gels and tyloses was delayed, and the fungus rapidly colonised the host.

13.4.2.3 *Distribution*

The first record of the disease was briefly described by Wardlaw (1950b), and pathogenicity was confirmed by Fraselle (1951). Thereafter, *Fusarium* wilt was found on several plantations in Nigeria and West Cameroon, in the Ivory Coast and elsewhere in west and west central Africa. The disease has been recorded in Brazil (van de Lande, 1983) and Ecuador (Renard and de Franqueville, 1989b), and Flood *et al*. (1989) confirmed the pathogenicity of a Brazilian isolate by inoculation of clonal plants. Dossa and Boisson (1991b) showed that, while *Foe* strains from Africa were in many different 'vegetative compatibility' groups, strains from Brazil and Ecuador were in the same group as strains from Benin and the Ivory Coast. This close relationship was confirmed with molecular markers (IRHO, 1992b). It is thus likely that the disease was introduced to South America from Benin or the Ivory Coast, probably on seed, as it has been shown that spores of *Foe* can be spread on seed (Locke and Colhoun, 1973; Flood *et al*., 1990b).

13.4.2.4 *Incidence and spread of the disease*

In southern Congo, the greatest devastation occurred in replants. In West Africa, the disease was largely confined to plantations, particularly replants, and for a long time was not much noticed in the groves. However, Aderungboye (1982) found that it was widespread in the drier Ogun and Ondo states of Nigeria but infrequent or absent in the high-rainfall areas of the south-east. Oritsejafor (1989) found that the average incidence in Nigeria was 0.77% in palm groves and 1.35% in plantations. The disease is less frequent in plantations on forest land than on former savannah (de Franqueville, 1991); in the latter, the disease usually takes the chronic form, whereas after forest, the acute form is more common.

In plantations in southern Congo, and in Nigeria in replants, wilt is commonly found in young palms that have recently come into bearing. However, in West Africa, the disease has also attacked older palms that have been in production for 10 years or more (Prendergast, 1957). Renard and de Franqueville (1989b) indicated that disease development depends on the previous history of the site. In new plantings, the first cases may not be seen until 6–10 years after planting, but in replants in previously infected areas, losses may occur within a year of planting. De Franqueville and Renard (1988) found that wilt incidence in a replant was correlated, not with total losses in the old stand, but with the percentage of previously infected palms which were still living at the time of replanting.

Prendergast (1957) stated that healthy, vigorous palms in good soil suffered little from the disease, and he showed that, in areas of potassium deficiency, incidence was substantially reduced by the application of potassium fertiliser. This finding was confirmed in experiments in both the Ivory Coast and Benin (Ollagnier and Renard, 1976). In the nursery test (Section 13.4.2.9), in contrast, Prendergast (1963) found that nitrogen reduced susceptibility, but potassium had no effect. Ho *et al*. (1985) found that drought stress increased the severity of symptoms in seedlings. Alvarado *et al*. (2014) emphasised that incidence was greatest where palms were under stress and suggested that stress tolerance and disease tolerance were associated.

Prendergast (1957) showed that diseased palms occurred in pairs more frequently than would be expected by chance, indicating infectious spread between neighbouring palms. Dumortier *et al*. (1992) found that palms with missing neighbours, in an area where the main cause of death was *Fusarium* wilt, were more likely to have wilt themselves than those without missing neighbours: of 1600 palms without missing neighbours, 17% had wilt, compared with 24% of 1000 palms with one or more neighbours missing. Only 18 palms had three neighbours missing, but 35% of those had wilt. It has generally been assumed that the disease is soil borne, but Moureau (1952) mentioned aerial spread by spores, and Cooper *et al*. (1989) showed that the pathogen sporulates profusely on male inflorescences and thus could be spread by spores.

13.4.2.5 *Effects on yield*

Acute wilt kills the palm, but Prendergast (1957) observed that infection rates as high as 20% had no apparent effect on yield and suggested that this was due to yield compensation by palms adjacent to vacant points. However, Dumortier *et al*. (1992) compared the yields of apparently healthy palms, with and without missing neighbours, and found that, although yield did increase slightly in palms with one or two neighbours missing (Table 13.4), the increase was not sufficient to compensate for the missing palms. When more than two neighbours were missing, yield was depressed (although the number of palms with more than two neighbours missing was small). If this

Table 13.4 Yield of palms with missing neighbours at Binga, Congo

	Missing neighbours				
	0	1	2	3	4
Yield (kg FFB/palm) for palms without symptoms	93.0	98.5	104.5	77.9	15.7
Yield as % of that of palms without missing neighbours	100	106	112	84	17
Number of palms in class	400	179	23	8	1

Source: From Dumortier *et al*. (1992).
Note: The main cause of vacancies was death from *Fusarium* wilt.

Table 13.5 Effects of chronic *Fusarium* wilt on yield

Class of palm	Yield (kg/ha.year, 1986–1992)	Yield as % healthy	Palms in class (%)	Contribution to yield (t FFB/ha.year) (143 palms/ha)
Cross L2T × D115D (tolerant)				
1. Healthy	111.8	100	75	11.99
2. Chronic wilt	35.6	32	3	0.15
3. Recovered from wilt	90.8	81	13	1.69
4. Latent wilt	107.4	96	8	1.23
5. Dead	0	0	2	0
Weighted mean:	94	94		Total: 15.06
Cross L2T × D10D (susceptible)				
1. Healthy	121.4	100	61	10.60
2. Chronic wilt	26.4	22	4	0.15
3. Recovered from wilt	79.4	65	27	3.07
4. Latent wilt	113.4	93	6	0.97
5. Dead	0	0	2	0
Weighted mean:	85	85		Total: 14.79

Source: Amended from Renard *et al*. (1993).
Note: The weighted mean yield is (yield as % healthy) × (palms in class).

depression is real, an explanation could be that palms with several neighbours missing are themselves infected (see 13.4.2.4). Yield in the year before a palm died from acute wilt was only 54% of that of healthy palms, while palms with chronic wilt gave a yield less than 30% of that of healthy palms. The effects of deliberate and systematic thinning on yield are discussed in Section 10.3.6.2, where it is concluded that, at the planting densities most widely used, the yield increase from palms adjacent to gaps is unlikely to be sufficient to compensate for the palms removed. This is even more likely to be true for the patchy thinning that would result from disease. Thus, it appears likely that any incidence of acute wilt will reduce yield.

With chronic wilt, palms remain alive and may recover. Renard *et al*. (1993) looked at the effects of chronic wilt on the yields of four classes of palms:

- Healthy palms
- Palms with typical chronic wilt symptoms
- Palms which had been diseased but had recovered
- Palms with internal signs of infection (browning of the vascular tissue) but no external symptoms, described as 'latent infection'

They estimated yield losses due to the disease as 15% in a susceptible cross and 6% in a tolerant cross (Table 13.5). The difference was attributable to lower yields from, and a larger number of, recovered palms in the susceptible cross compared with

the tolerant cross. In both crosses, palms with latent infection yielded much the same as healthy palms. Although healthy trees of the susceptible cross gave a slightly greater yield, overall yield was greater from the tolerant cross.

13.4.2.6 *Physiology of diseased palms*

Drying up of the leaves and death of the palm are caused partly by the destruction of the roots and partly by the blocking of the xylem vessels by gels and gums. Diseased vessels may at first occur in only one section of the stem base, and this might account for leaf symptoms being confined at first to certain spirals. Xylem vessels in the centre and at the top of the stem then become diseased, and the symptoms spread across the stem, so that a large proportion of the vessels at the top are affected. In young palms, up to 6 years old, diseased vessels are usually widely dispersed throughout the base.

In nursery seedlings, the main symptoms are a reduction in petiole length and leaf area. Mepsted *et al*. (1995a) observed symptoms of water stress in infected nursery palms (closed stomata, lower leaf water potential, greater resistance to water flow from stem to leaf), but they considered that water stress was not the cause of stunted leaf development, because stressed but uninfected plants did not show stunted growth. Application of a gibberellin inhibitor, paclobutrazol, caused stunting symptoms similar to those of *Fusarium* wilt. Application of gibberellic acid to infected palms restored petiole length to normal, but had no effect on leaf area. The authors concluded that wilt symptoms might be due, at least in part, to an upset in gibberellin metabolism.

Rusli *et al*. (2015) found an increase in chitinase gene expression after infection of a tolerant progeny, but in a susceptible progeny activity decreased. A similar increase in chitinase expression followed infection with *Ganoderma* (see Section 13.4.3.10).

13.4.2.7 *Chronic and acute wilt*

Mepsted *et al*. (1995b) found that water stress symptoms in nursery palms were much more severe in the older leaves, opened before the palms became infected, than in the younger, stunted leaves. They suggested that stunting might be an adaptation by the palm to reduce the water stress caused by occlusion of the xylem vessels and speculated that, in field palms, failure to adapt in this manner might result in the acute form of wilt, while in palms that were able, or had time, to adapt, the chronic form would result. Prendergast (1963) and de Franqueville (1991) found no obvious difference in behaviour in the nursery test between *Foe* strains isolated from acutely infected palms and from palms with chronic wilt. This suggests that the difference between forms of the disease may be in the host reaction. According to de Franqueville (1991), however, the acute form is more frequent in plantations on forest land than on former savannah. Such a difference is more easily explained in terms of pathogen strains than by host reaction, but differences in climate could be a factor. Strains isolated from acute cases tend to be more aggressive than those from chronic cases (R.M. Cooper and M.H. Rusli, pers. comm., 2014).

13.4.2.8 *Control*

In Congo, the destruction by fire of all diseased palms and their neighbours was recommended, and the replanting of areas where *Fusarium* wilt had been prevalent was discouraged (Moureau, 1952). In the Ivory Coast, de Franqueville and Renard (1988) stated that all trees infected with chronic wilt must be removed at least 5 years before replanting. However, they were unsure how early such removal should start and suggested as a compromise the removal of any infected palms not producing at least one bunch per year. *Calopogonium caeruleum* or *Pueraria* as cover increases wilt incidence, and Renard and Quillec (1983) recommended planting grass species such as *Brachiaria* instead, suggesting that competition for nitrogen between the grass and the palm discouraged *Fusarium* infection. Competitive grasses may themselves depress palm yield, though, and the recommendation of de Franqueville and Renard (1988) to keep a strip of bare soil on either side of the row of young palms seems preferable. Turner (1981) considered that any effect of fungicides was likely to be short lived. Renard and de Franqueville (1991) found a significant increase in wilt in plots mulched with EFB and a reduction when potassium fertiliser was applied.

13.4.2.9 *Breeding for resistance*

The most promising method of control is by the breeding of tolerant or resistant lines (with *tolerance* the pathogen invades but has little effect on the host, whereas with *resistance* the pathogen is excluded – Cooper and Rusli, 2014). Prendergast (1963) was the first to develop a technique for the screening of

seedlings for tolerance to the disease at the nursery stage (Plate XIIIA); his method was adopted with little modification by Renard *et al*. (1972). The method involves inoculation by pouring a suspension of *Foe* spores onto the bulb of the seedlings or the exposed roots around the collar. Prendergast (1963) described the symptoms in nursery seedlings in detail and showed that results were not much affected by the size of the seedlings at the start of the test. He also noted the importance of the inoculum level and the time of evaluation: with too light an inoculum, or too early evaluation, few cases might be observed, and differences would be hard to detect. However, if the inoculum was very heavy or evaluation was late, all plants might die.

The standard nursery test requires large numbers of plants of each family and hence large areas of nursery space; Prendergast (1963) used 40 seedlings per family, and Renard *et al*. (1991) up to 160. Locke and Colhoun (1974) developed a method of inoculating very young seedlings grown in compost with two known levels of inoculum and then compared their growth with that of seedlings grown in uncontaminated compost. Determinations were made of the number of propagules in the soil so that subsequent inoculations could be related to normal soil levels. The fungus was recovered from progenies showing both large and small reductions either in weight per plant or in 'leaf area product'. It was demonstrated that some progenies were tolerant of infection in lightly contaminated compost only and some in both lightly and heavily contaminated compost; others showed high susceptibility at both levels. A high degree of repeatability was attained, but this method does not seem to have been adopted by oil palm breeders.

Flood *et al*. (1989), using clonal plants, were able to reduce the numbers required to only 12 plants per clone, by using inocula with a known, constant concentration of fungal spores and by detailed classification of the severity of symptoms on each plant.

Sound statistical analysis of the data from nursery inoculation trials is essential, if reliable results are to be obtained (see Porter, 1989). In all trials, the percentage of infected plants in each family has been recorded. Prendergast (1963) then divided the progenies in a trial approximately into quartiles or classified as resistant those that differed from the mean by at least one standard error. Renard *et al*. (1972) calculated a 'wilt index', as the percentage of wilt-infected plants in a progeny divided by the mean wilt percentage of all the progenies in the trial. The best method appears to be that described by de Franqueville (1984). A wilt index was calculated in the same way as by Renard *et al*., but after angular transformation of percentages for individual plots, data were statistically analysed, and progenies were only accepted as resistant if they had significantly lower losses than either the mean of the trial or standard crosses of known performance.

An alternative to the standard nursery test was developed by Mepsted *et al*. (1995c). This involved inoculation of 2.5 cm sections of the rachis from near the tip, by immersion in a suspension of *Foe* spores under mild vacuum. Within 8 days, rachis sections from susceptible clones turned completely brown internally, whereas those from resistant clones showed little or no browning. This test is much faster than the nursery test, which takes several months. In addition to speed, the method has the great advantage that it can be applied to individual palms. The nursery test is based on the average performance of a group of palms, so the only way to screen individuals is as clones, or by studying the progeny of a self-pollination. Subsequent experience showed that the rachis test only works well on palms that are in good health; nutrient-deficient palms, or palms infected with *C. elaeidis*, showed severe browning in both susceptible and resistant clones (Buchanan, 1999).

The inheritance of resistance is discussed briefly in Section 6.3.5. Prendergast (1963) and de Franqueville (1984) considered that the most susceptible families could be quite consistently and repeatably identified by the nursery test. De Franqueville also found a reasonable correlation between results of the nursery test and disease incidence in the field, in a heavily infested part of Congo. Renard *et al*. (1972, 1980) also showed that tolerant seedlings in the nursery test give rise to palms with a low incidence of wilt in the field.

Porter (1989) described resistance breeding in Congo, where selection was based primarily on performance in disease-infested fields (Plate VID), backed up by the nursery test. He gave examples of parents consistently transmitting resistance or susceptibility to their offspring in field trials. For example, 14 crosses derived from palm 69MAB (see Fig. 6.4) were all more resistant than the trial mean, 12 of them significantly so. Conversely, of seven crosses from palm 2/5710 (see Fig. 6.5), six were more susceptible than the trial mean, five significantly so. Corley (1993) and de Franqueville *et al*. (1995) found differences between clones in susceptibility.

In Nigeria, Rajagopalan *et al*. (1978) found that, among 336 progenies, none was immune but 149 showed sufficient tolerance to be considered valuable for breeding; and certain *pisiferas* consistently gave tolerant crosses with a range of *duras*. Prendergast (1963)

and Locke and Colhoun (1974) also observed that no progenies appeared to be wholly immune to the disease. However, Rosenquist *et al*. (1990) noted that pure Dumpy Deli *dura* material (see Section 6.1.1.2) appeared to be virtually immune: two families gave 0 and 1% wilt in the nursery test in Cameroon, while one family in Congo had suffered no losses after 10 years in the field, a figure recorded in only two other families out of more than 450 in the programme. Renard *et al*. (1980) found strong resistance in some *E. guineensis* × *E. oleifera* hybrids.

There are reports of tolerant material from other countries proving susceptible when imported to Nigeria (Oritsejafor, 1989), but replicated trials were not involved. De Franqueville (1991) tested three strains of *Foe* on 66 different families. There were significant differences between the strains and between the families, but no strain × family interaction. Mepsted *et al*. (1994) tested three isolates of the fungus from different parts of Africa, on 14 clones. The isolates differed in aggressiveness, but as in de Franqueville's trial, the clone × isolate interaction was not significant. These two studies indicate that resistant material selected in one area should remain resistant when transplanted elsewhere. However, Rusli *et al*. (2015) found a progeny which was susceptible to an aggressive strain of *Foe*, but not to a less aggressive but still pathogenic strain.

13.4.2.10 *Symptomless infection*

Ho *et al*. (1985) isolated *F. oxysporum* from roots of healthy palms in Malaysia; these strains were apparently non-pathogenic, causing no disease symptoms. Flood *et al*. (1989) showed that one such strain did cause mild wilt symptoms in a susceptible clone, however.

Mepsted *et al*. (1988) found that inoculation of seedling roots with a non-pathogenic isolate could prevent subsequent infection by pathogenic strains. Diabaté *et al*. (1992) confirmed this 'cross-protection' effect and showed that phenolic compounds accumulated in the palm roots after inoculation, whether this was with a pathogenic or a non-pathogenic strain. Susceptibility appears not to be due to a lack of these phenolics, as both resistant and susceptible palms produced them, at similar levels, in response to the non-pathogenic strain of *F. oxysporum*. Presumably, in susceptible palms, the fungus normally spreads more rapidly than the build-up of phenolics; preinoculation with the non-pathogenic strain may allow sufficient build-up, in advance of infection, to confer resistance. However, Tengoua and Bakoumé (2010) isolated apparently non-pathogenic strains from palms with symptoms in Cameroun; in two instances, pathogenic strains were also isolated from the same palm. Both preformed and induced antifungal compounds were extracted from xylem fluids and petiole tissue by Mepsted *et al*. (1995c), with the effect being particularly pronounced in resistant material. Paul (1995) suggested that preformed antifungals were also present in oil palm roots, but the identity of the compounds in roots or petioles was not determined.

Diabaté *et al*. (1992) suggested that there might also be competition between pathogenic and non-pathogenic strains in the soil. Flood *et al*. (1989) had previously suggested that competition for an ecological niche may be the reason that *Fusarium* wilt is not present in Malaysia: any strains accidentally introduced from West Africa would face competition from native Malaysian strains. Cooper and Rusli (2014) found that infection and disease severity of seedlings in Malaysian soil were greater when the soil was sterilised. Fravel *et al*. (2003) reviewed work on other crops, showing that non-pathogenic strains of *F. oxysporum* may compete with and suppress pathogenic strains in the soil, at the root surface and in plant tissues. In some crops, non-pathogenic strains have been applied as biological control agents, but this would probably not be practicable on a plantation scale. Soil bacteria have also been shown to reduce *Fusarium* infection in other crops (Klein *et al*., 2012).

13.4.2.11 *Plant quarantine*

The demonstration that spores of *Foe* can be carried on oil palm seeds (Locke and Colhoun, 1973), and even on the kernel surface inside the shell (Flood *et al*., 1990b), poses potential problems for plant quarantine. Flood *et al*. (1994) showed that the standard 40°C heat treatment used to break dormancy (see Section 8.1) greatly reduced the level of infection, but some viable spores remained. They developed a method of fungicide application involving vacuum infiltration, which eliminated spores, including any within the shell. This treatment should be applied whenever seeds are exported from areas where wilt occurs.

Spores of *F. oxysporum* were also found in batches of freeze-dried pollen used for oil palm breeding and were shown to be pathogenic (Flood *et al*., 1990b). This contamination can be detected by plating out samples on a *Fusarium*-selective culture medium, but this is laborious if many samples are involved and is specific only for *F. oxysporum*, not for *Foe*. Use of the

pathotype-specific DNA primers developed by Cooper and Rusli (2014) would avoid discarding samples with harmless contamination.

13.4.2.12 *Conclusion*

There is no doubt that *Fusarium* wilt can be devastating, and there are instances of plantations in Africa being abandoned or converted to other crops because of the disease. Given the large quantities of seed and pollen exported from Africa in the past, it is surprising that the Far East has remained free of the disease. Perhaps symptomless infection by other strains of *F. oxysporum* gives a degree of cross protection, but the very strict quarantine measures now enforced by Malaysia and other countries appear sensible (see Section 13.8). Breeding of resistant material is clearly the best approach to controlling the disease, and as a precaution, breeders in the Far East would be wise to gather information on the performance of their materials in areas where the disease occurs.

13.4.3 *Ganoderma* trunk rot or basal stem rot

In severely affected areas in the Far East, over 50% of palms may be infected by *Ganoderma* (Flood *et al*., 2002). *Ganoderma* was at first regarded as a disease of old palms, of little economic importance because such palms would soon be replanted. In the mid-1950s, however, the disease was recognised in much younger palms, particularly in areas planted after coconuts or replanted from oil palms (Turner, 1981). In Sumatra, the disease is typically first seen about 12 years after planting and starts to reduce yields from about 18 years onwards (Flood *et al*., 2002), but much younger palms may also be infected.

13.4.3.1 *Symptoms*

The usual first symptom of infection by *Ganoderma* is similar to that of drought conditions: a failure of the young leaves to open, so that a number of fully elongated but unopened 'spears' are seen in the centre of the crown (Turner, 1966a). This indicates that the stem or root system is already extensively damaged, so that water uptake is restricted; it is apparently a direct response to water shortage and so is not necessarily diagnostic for *Ganoderma* (Turner, 1966a). In old palms, the lower leaves collapse, hanging vertically downwards from the point of attachment to the trunk (Plate XIVC). This is followed by the drooping of younger leaves, which turn a pale olive green or yellowish colour and die back from the tip. Later, the base of the stem blackens, gum may be exuded and the distinctive fructifications of *Ganoderma* sp. appear (Plate XIVA). The whole crown of the palm may then fall off, or the trunk may collapse (Plate XIVB).

Bull (1954) described and illustrated the internal symptoms of old palms exhibiting *Ganoderma* trunk rot. The peripheral tissues are hard and unaffected by the rot, the black fibres in this zone being normal. Within the stem at the base of the palm, the majority of the tissue is yellow coloured and breaks up easily; mycelium can be found extending through the tissue. Roots are also found to be infected, the cortex being brown and decaying, the stele black. Large numbers of sporophores may be formed, the early ones small and rounded, the later ones typical brackets.

13.4.3.2 *Cause*

Ganoderma species. Early work referred to *G. lucidum*, but that is a temperate species (Steyaert, 1967). The general consensus now appears to be that *G. boninense* is the main species pathogenic to the oil palm, at least in South East Asia (Moncalvo, 2000). According to Swinburne *et al*. (1998), three types of *Ganoderma* can be isolated from oil palms in Malaysia; these were subsequently identified as different species by Idris *et al*. (2001). *G. boninense* (type A) was significantly more aggressive than type B (actually two species, *G. miniatocinctum* and *G. zonatum*). *G. tornatum* (type C) appeared to be a non-pathogenic saprobe, although Pilotti (2005) described it as a minor pathogen in PNG. Idris *et al*. speculated that infection by *G. tornatum* might offer some protection against the pathogenic species. In Sumatra, several species were found on fallen palms, but *G. boninense* was the only one isolated from infected living palms (Rees *et al*., 2012). Kinge and Mih (2011) isolated a new species, *G. ryvardense*, from oil palms in Cameroon, but did not confirm that it was pathogenic. In this chapter, we will follow most authors in referring to the pathogen simply as *Ganoderma*.

Navaratnam (1961) confirmed pathogenicity by inoculating both roots and stems of 40-year-old palms with *Ganoderma* mycelium. More recently, palms at the nursery stage have also been successfully inoculated (Amiruddin, 1993; Sariah *et al*., 1994; Ariffin *et al*., 1995b), and the pathogen has been reisolated (Khairudin *et al*., 1993). Rees *et al*. (2007) found that three different isolates from Sumatra differed in aggressiveness after inoculation of nursery seedlings.

Soil conditions appear to be an important predisposing factor, as discussed in Section 13.4.3.3.

13.4.3.3 *Distribution*

In Malaysia, the disease is much more prevalent on low-lying alluvial soils, particularly the coastal clays, than on inland soils, and it is on the former that the most serious attacks on young palms have occurred. Turner (1965a) showed that incidence on areas where the preceding crop was coconuts was much higher (2 fields with 39 and 35%) than where planting followed rubber (4 and 2%) or forest (see also Fig. 13.1). The greatest losses were where old coconut trunks had been buried, to prevent infestation by *Oryctes rhinoceros* (see Section 14.4.1). Turner (1981) considered oil palm tissue to be a less conducive medium for the fungus than coconut tissue; he quoted survey results showing an average of 26% infection in 15-year-old palm to oil palm replants, but 44% in oil palms following coconuts (Turner, 1965b). However, Gurmit (1991) quoted a 15-year-old replant with 67% infection, and Fig. 13.1 suggests little difference in the rate of disease development in plantings after oil palm or coconut. Some live coconut palms were found to contain *G. boninense* as a symptomless endophyte (Idris *et al*., 2001), which could explain the high incidence in plantings following coconuts. On peat soils, a high incidence may be observed whatever the previous crop (Ariffin *et al*., 1990).

The effect of soil type on disease incidence is not yet understood. Gurmit (1991) suggested that the usually high soil moisture of the coastal soils might favour *Ganoderma* over other, antagonistic soil fungi, but on peat, Lim and Udin Wahidin (2011) found lower incidence when a high water table was maintained (but overall incidence was below 2%). Wood (2007) noted that most, but not all, low-lying sites are prone to the disease, and not all sites with high incidence are low lying. Swinburne *et al*. (1998) found that isolates from coastal soils, usually with a history of previous coconut or oil palm planting, were predominantly *G. boninense*, while those from inland soils were 'type B' (see Section 13.4.3.2). This is consistent with the greater incidence on coastal soils and might explain the occasional high incidence on inland soils if *G. boninense* were occasionally found there, but it remains unclear whether it is simply coincidental that these types are usually associated with particular soils.

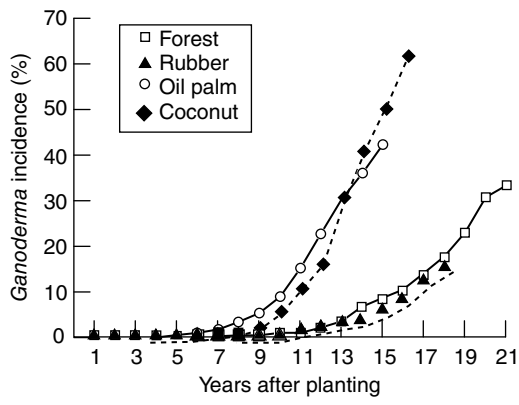

Fig. 13.1 Incidence of *Ganoderma* in oil palm in relation to previous crop. (From data of Gurmit, 1991.)

In a survey of 1061 estates in Malaysia, Idris *et al*. (2011) found that, while 60% of estates reported the disease, the average incidence was only 3.7%, and 8.4% in palms over 16 years old. The estates participating in the survey were self-selected, so data may not be representative, but they do suggest that the disease is not the universal problem that is sometimes portrayed. Surveys of visual symptoms may underestimate the extent of infection, though, as Hasan and Turner (1998) noted that infected palms may remain symptomless for long periods, before the internal damage becomes so severe that external symptoms develop. Ariffin *et al*. (1995a) found, by extraction of trunk samples in a 22-year-old planting, that between 13 and 17% of palms classified as healthy were actually infected.

13.4.3.4 *Spread of the disease*

There is evidence that *Ganoderma* can spread both by root contact and by spores. It is not a 'soil-borne' disease like *Fusarium* (Section 13.4.2), as it is a very weak competitor and will not grow through soil. Wood (2007) suggested that the disease in young palms might differ from that in older palms, there being two distinct phases. Where decaying tissue from the old stand is left *in situ*, it can form a large inoculum, especially if buried, and young palms planted nearby can be infected. This is Wood's 'early phase' disease, and it is clear that infection under these conditions is mainly by root contact with an inoculum source. The 'late phase' of the disease starts to build up from 12 to 15 years after planting. *Ganoderma* may be found in the roots at this stage, and it is assumed that this causes the symptoms, but Wood pointed out the possibility that the fungus invades tissues which are already senescing for some other reason. Other possibilities are that the 'late phase' disease results from infection by spores

rather than root contact, or that earlier infection remains symptomless until the palm is weakened by some other factor.

Infection by root contact: Seedlings planted very close to *Ganoderma*-infected stumps may show disease symptoms within 1 or 2 years (Hasan and Turner, 1998). Other trials in Malaysia and Indonesia also indicate that root contact is important: in the replanting trial described in Table 13.8, trenching around old stumps significantly reduced subsequent infection (Chung G.F., pers. comm., 2009), while Flood *et al.* (2005) obtained 60% infection after 54 months in seedlings planted next to trunk sections covered with soil, but no infection if the trunks were exposed. It has been suggested that infection at the base of the stem might be from spores germinating on cut leaf bases, but Rees *et al.* (2009; also Cooper *et al.*, 2011) showed clearly that infection can occur through the roots, preceding infection of the stem. Anatomical studies of infection were described by Flood *et al.* (2010). Breton *et al.* (2005) described dense knots of *Ganoderma* mycelium surrounding the oil palm roots at the point of infection.

Turner (1981) considered that *Ganoderma* was a weak parasite and that it needed to develop saprobically in large masses of dead palm tissue before it had sufficient 'inoculum potential' to infect live plants. Rees *et al.* (2007) obtained 90% infection of seedlings after 8 months using 430 cm^3 rubber-wood blocks as inoculum, but only 30% with 54 cm^3, showing the importance of inoculum size. Hasan and Turner (1998) showed that poisoned stumps, even of symptomless palms, gave higher infection of adjacent seedlings, presumably because they rotted more quickly and could be invaded by seedling roots more easily. Short (20 cm) stumps gave higher infection rates than 50 cm stumps, perhaps again because they rotted more quickly. Diseased stumps had largely ceased to be sources of infection 2 years after felling. Stumps of healthy palms colonised by *Ganoderma* became sources of infection and rotted more slowly than diseased stumps. Hasan and Turner suggested that they therefore remained infectious for longer.

Infection by spores: Most attempts at control have been based on the assumption that infection is by root contact with an infected palm or other inoculum source. Studies on spatial patterns of the disease have shown that diseased palms in some fields tend to be in clumps, which also suggests spread through root contact. Work with molecular markers has shown that such spread can occur, with the same genotype detected in diseased stumps as in seedlings planted close to the stump (Flood *et al.*, 2000). Several authors have found the same genotype in adjacent palms (Miller *et al.*, 2000; Pilotti, 2005; Cooper *et al.*, 2011), but Rees *et al.* (2012) found different genotypes in diseased palms and adjacent dead palms.

In other studies, the disease appears to be randomly distributed, as might be expected if spread were by spores (Flood *et al.*, 1998). Work with molecular markers (Miller *et al.*, 2000), and mating compatibility studies (Miller *et al.*, 1999; Pilotti and Sanderson, 2001; Pilotti *et al.*, 2003; Pilotti, 2005), has indicated that a range of genotypes may exist within quite a small area. Even a single palm may be infected by more than one genotype of the pathogen. This suggests that basidiospores (which are sexually produced and thus genetically variable) may be an important mode of spread. Clear evidence for involvement of spores comes from occurrence of the disease in oil palms planted on land used for growing rice for 10 years (Sanderson *et al.*, 2000). In PNG, it was concluded that spread by root-to-root contact was not a common occurrence and that spores were the main source of the disease (Pilotti *et al.*, 2003). Apparent attack at the base of the trunk might sometimes actually be from infection of cut leaf bases (Sanderson and Pilotti, 1997; Bridge *et al.*, 2001; Panchal and Bridge, 2005). Rees *et al.* (2012) showed that spores could be sucked up to 10 cm into cut xylem vessels, where they germinated protected from the external environment. However, infection from spores has not been demonstrated, despite several attempts (Flood *et al.*, 2002).

Idris *et al.* (2003) found *G. boninense* fruiting bodies on leguminous cover crops in immature palms, the extent of infection being highly correlated with the level of incidence in the previous oil palm stand. Spores from such fruiting bodies could provide another source of infection for the next generation of palms, but the importance of this is not yet clear.

The life cycle of Basidiomycetes such as *Ganoderma* has two phases: basidiospores, released from the fruiting bodies (brackets), are monokaryotic (haploid) and germinate to form haploid mycelium. When mycelia from two compatible mating types meet, they can fuse to form a dikaryotic mycelium, which has two haploid nuclei per cell. It is the dikaryotic mycelium which forms fruiting bodies, in cells of which the haploid nuclei fuse to give a diploid nucleus and then produce haploid basidiospores by meiosis. Hasan and Flood (2003) and Rees *et al.* (2007) showed that monokaryotic mycelium could grow saprobically in sterile, dead oil palm stem tissue, but did not infect live seedlings. Only dikaryotic mycelium was able to infect living palms and

even that only if a sufficiently large inoculum was used. Rees *et al*. (2007) also showed that soil and the debris from palm leaf axils were only able to support growth of the fungus if they were previously sterilised, emphasising the weak competitive ability of *Ganoderma*. The proposed life cycle is shown in Fig. 13.2.

Conclusion: It seems likely that the disease can be spread in two ways. Root contact with an inoculum source, such as old oil palm or coconut trunks, would result in 'early phase' infection, within a few years after planting. 'Late phase' outbreaks of the disease 15 or 20 years after planting could be from aerial spores or from slow development of infection that took place more than a decade earlier, perhaps triggered by some environmental factor or host senescence.

Growth of the fungus through infected roots may be very slow. Flood *et al*. (2005) found that infection started after 38 months in seedlings planted 1 m away from felled, *Ganoderma*-infected trunk sections. At 0.5 m, infection was observed after 27 months, implying a fungal growth rate of about 0.5 m/year. Rees *et al*. recorded a growth rate of *Ganoderma* in seedling roots of up to 4.4 cm/month, while Ariffin *et al*. (1995b) recorded a growth rate of only 1 cm/month; at the latter rate, the fungus would take 8 years to reach the trunk of a palm one metre from an inoculum source. The trial described in Table 13.8 shows that preventing root contact with infected stumps can reduce the incidence of disease developing between 8 and 12 years after replanting.

For infection to occur from spores, two compatible mating types would have to colonise the same mass of decaying palm tissue, such as leaf bases or the debris in leaf axils, and form a dikaryon, as Hasan and Flood (2003) showed that only the dikaryon is infectious. This would require both build-up of residues and colonisation by compatible mating types, perhaps explaining the time interval before 'late phase' infection is seen. Infection of leaf bases near the base of the trunk could eventually lead to basal stem rot, while in older palms, infection of leaf bases higher up might cause upper stem rot (see Section 13.4.6).

Which of these modes of infection is more important in a particular planting may depend on some environmental factor or on the other competing microorganisms present. Wood (pers. comm., 2013) found that early and late phase incidences were not correlated.

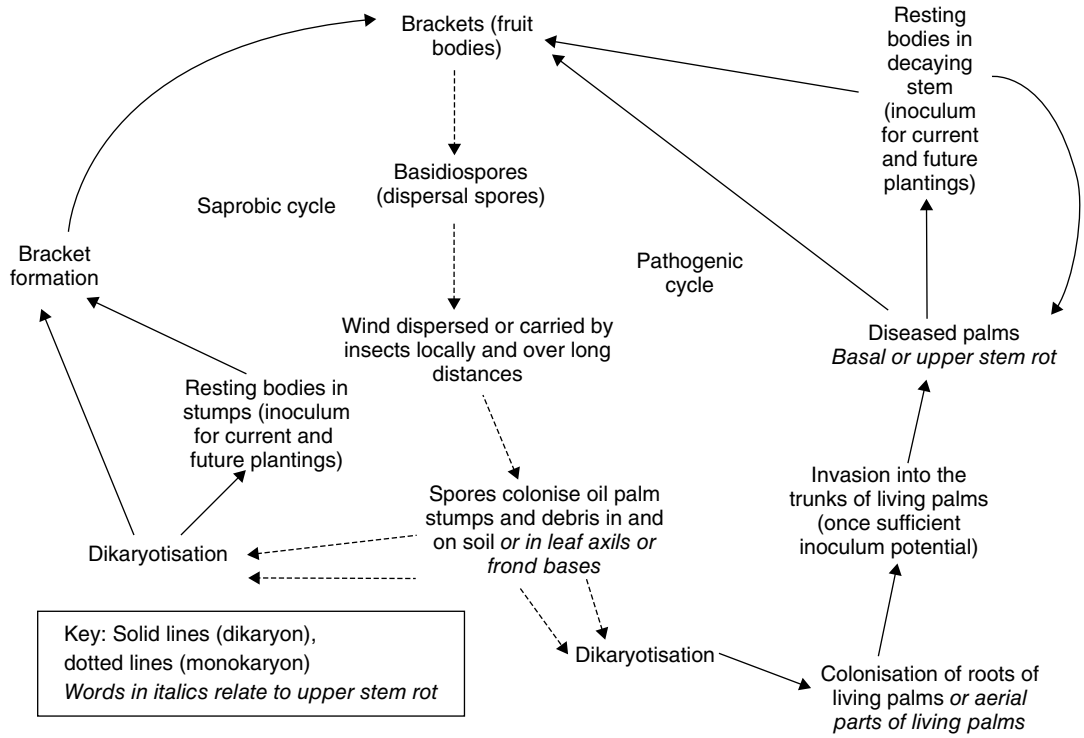

Fig. 13.2 Life cycle of *Ganoderma* (from Flood et al., 2002).

13.4.3.5 *Predisposing factors*

The effects of previous crop and of soil type have already been mentioned. The strong influence of soil type does not appear to have received the attention it merits, but several studies of the effects of fertilisers have been made. Akbar *et al.* (1971) indicated that nitrogen and magnesium may have some role in combating the disease, but more recent trials have given equivocal results. Potassium chloride and urea application have both increased disease incidence in some trials and decreased it in others (Gurmit, 1991). Tayeb Dolmat and Hamdan (1999) found similar conflicting results with phosphorus and potassium in three trials, two on peat and one on a coastal alluvial soil. Gurmit (1991) noted that high soil salinity and low soil pH appeared to discourage the disease. Tohiruddin *et al.* (2010b) found increased incidence in fertiliser trials in North Sumatra, where nitrogen and phosphorus application had reduced leaf copper and zinc levels. Nur Sabrina *et al.* (2012) showed that application of copper sulphate as fertiliser in the nursery reduced incidence and severity in inoculated seedlings. Najihah *et al.* (2015) found a similar effect of silicon application to inoculated seedlings; silicon often improves plant disease tolerance (Epstein, 2009), but soluble silicon measurements are needed to show the significance of this in the field.

Cooper *et al.* (2011) noted a strong effect of shade and soil temperature on inoculated seedlings and suggested that in the field, lack of shade and raised soil temperature before canopy closure might explain the fact that *Ganoderma* is typically a disease of older palms. Before canopy closure, the ground is usually shaded by a vigorous legume cover or other vegetation, though, so this seems unlikely to be important.

13.4.3.6 *Effects on yield*

Yield reduction may occur both from death of palms and from reduced yield in infected but still living palms. Disease losses might be partly compensated for if palms next to gaps gave increased yield, and Turner (1981) stated that there was circumstantial evidence that losses of up to 20% might be compensated for. This may have referred to Prendergast's comments on *Fusarium* wilt (Prendergast, 1957), but other work on that disease suggests that any yield increase is insufficient to compensate for the lost palms (see Section 13.4.2.5). Hasan and Turner (1994) stated that yield compensation ceased at around 10% losses, but again presented no supporting data. The extent to which compensation occurs will depend on the optimal planting density, which varies with soil fertility and with type of planting material (see Section 10.3.3.1). In Sumatra, in plantings from the late 1960s and early 1970s, there was little decline in yield until the surviving stand had fallen to about 115 palm/ha (Fig. 13.3a), but in more recent plantings, any loss of palms was associated with a loss of yield (Fig. 13.3b). In Sabah, Rao *et al.* (2003) found that yield of neighbouring palms increased by 14% in the year after disease was detected; with six neighbours, this would be insufficient to compensate for loss of the central palm.

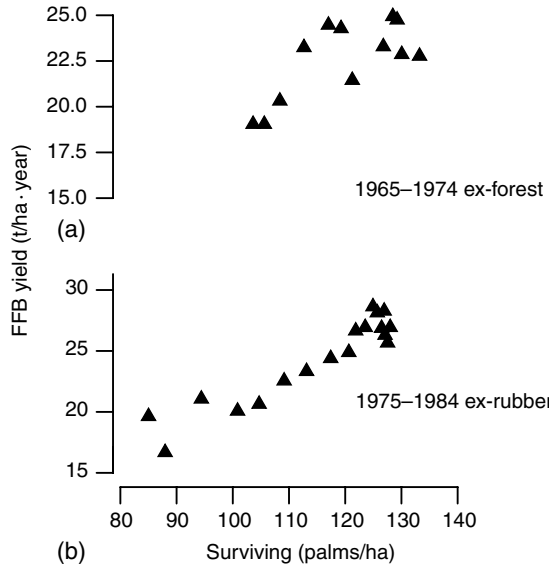

Fig. 13.3 Yield of individual fields in North Sumatra, with different amounts of *Ganoderma* losses. (From London Sumatra, unpubl.)

Khairudin (1995) found that yield of infected palms was reduced by 20–40% in the year before infection was detected. Effects may not always be so large, though: Nazeeb *et al.* (2000) showed that palms with *Ganoderma* yielded between 13 and 21% less than symptomless palms at the same age. Rao *et al.* (2003) noted that yield was 40% below that of symptomless nearby palms in the year the disease was detected and 50–60% below in the year after detection. In the year before detection, there was a yield loss of about 10%.

13.4.3.7 *Control at time of replanting*

It seems to be generally accepted that incidence of *Ganoderma* increases from one generation of oil palms to the next, but there are few published data to support

this contention, the evidence being mainly anecdotal. The 25-year time interval between generations makes reliable comparisons within the same field difficult, and possible genetic differences in susceptibility would further confound comparisons (see *Breeding for resistance*), but a survey of estate records could be very useful in establishing whether there is an increase.

Table 13.6 shows data from three estates in Indonesia; there appears to be an increase in disease incidence over the first three generations in estate 1, and perhaps in estate 2, but not in estate 3. These data are from different fields, recorded at the same time, so could be confounded by the possible effects of soil type already mentioned earlier. As further evidence against a build-up from one generation to the next, Gurmit (1991) found that in fields that ranged from 40 to 60% infection before replanting, all had about 3% disease 9 years after replanting. However, in Sabah, Rao *et al.* (2003) found an average of 31% 14 years after replanting, already higher than in the old stand, which averaged 29% (diseased plus dead) at the time of replanting. The high incidence in first replants, as shown in Fig. 13.1, suggests that a build-up occurs, but there is little evidence for a further increase after the second replant. Despite this, most control measures are aimed at preventing such a build-up, on the assumption that spread is by root contact with an inoculum source.

Table 13.6 *Ganoderma* incidence (% of palms severely affected or dead) in different generations, planted in different blocks at the same time

Estate	Age (years)	Generation			
		1	2	3	4
1	11–15	1.5	7.8	17.9	4.7
2	11–15	–	10.6	10.2	–
	16–20	–	14.9	17.5	–
	21–25	5.1	9.3	17.7	–
	>25	11.4	8.6	–	–
3	11–15	–	11.1	11.1	12.4
	16–20	20.5	20.6	17.8	9.4
	21–25	–	27.6	21.3	–

Note: All blocks were replanted by felling and windrowing; all disease scoring was done in 1999–2000 (de Franqueville, 2000).

The recommended method of reducing *Ganoderma* incidence is to rid the fields of as much oil palm tissue as possible at replanting time. Mechanical methods of 'clean clearing' were outlined by Turner (1981). Stimpson and Rasmussen (1973) described a system which entailed either burning or cutting up and splitting the boles and windrowing the old palms so that they rotted rapidly. The method included prior poisoning of the palms and subsequent root raking and ploughing to bring up and dispose of pieces of palm base and other material that might form a focus for *Ganoderma*. The operations are costly but have been regarded as essential in coastal areas in Malaysia. In inland areas, the incidence of *Ganoderma* is not usually so great, and the emphasis on clean clearing has been less strong.

Table 13.7 shows results of a number of comparisons of replanting methods. Disease incidence may be higher after underplanting, but in most comparisons, the difference was negligible. In Sabah, there was a difference

Table 13.7 Effects of replanting method on *Ganoderma* incidence

Location	Age when surveyed (years)	Disease incidence and deaths (%)			Ref.
		Underplanted	Felled and wind-rowed	Clean cleared	
Inland (Johore)	17	5.7	5.0	–	1
Inland (Johore)	20	5.5	4.5	–	2
Alluvial (Sabah)	11	13.7	6.7	–	5
Sabah (same fields)	14	30.9	31.6	–	5
Alluvial (Selangor)	14	13.2	16.8	6.1	6
Alluvial (Selangor)	15	33.0	17.6	14.0	3
Alluvial (Perak)	15	–	5.4	0.5	4

References: 1: Loh and Rajaratnam (1977); 2: Pamol Plantations Bhd (unpubl.); 3: Khairudin (1990); 4: Gurmit (1991); 5: Rao *et al.* (2003); 6: Chung G.F. (pers. comm., 2009; see also Table 13.8).

Table 13.8 Effect of replanting method on *Ganoderma* incidence 12 years after replanting

	Replanting method	Disease incidence (%)		
		Old stand	Replant	
			Year 8	Year 12
1	Cut at 0.5 m, wind-rowed, stumps left in ground, not ploughed	59	4	30.3
2	As 1, but stumps left but isolated by trench 0.5 m deep	57	1	7.9
3	Pushed over, wind-rowed, complete bole removal, ploughed	64	1	16.8
4	Pushed over, chipped, complete bole removal, ploughed	59	1	13.2
5	Underplanted, wind-rowed, complete bole removal, not ploughed	64	3	13.2
6	Chipped, bole removal, all debris removed from plots, ploughed	64	0	6.1
	Standard error			3.6

Note: Selected treatments from replanting trial on alluvial soil, Selangor (replant means adjusted for old stand differences) (Chung G.F., pers. comm., 2009).

at 11 years, which disappeared 3 years later. In one trial, the difference between felling and windrowing, and the clean clearing advocated by Turner (1981) and others, was also quite small, while in a second trial, incidence was quite low even with windrowing.

In Table 13.8, a number of clearing methods are compared. Complete removal of all old stand material (Tr. 6) gave 6% incidence after 12 years. This treatment involved transporting all material out of the field; this is impracticable but is more or less equivalent to what might be achieved by burning. When palms were felled above ground level and stumps left *in situ* (Tr 1), disease started to build up from 8 years after planting, reaching 30% by year 12. If a trench was dug round each stump (Tr 2), this was reduced to only 8%, indicating that root contact with the old stumps is likely to be a major factor in disease development at this relatively late stage (Chung G.F., pers. comm., 2009). Underplanting with bole removal (Tr 5) was no worse than clearing before planting (Tr 3, Tr 4). Chipping the old trunks (Tr 4) gave no advantage compared to windrowing (Tr 3), but in Sumatra, Virdiana *et al*. (2012) found that complete shredding of trunk and bole gave 11% incidence nine years later, compared to 21% after windrowing old trunks.

Table 13.9 shows a comparison of first replants (the second generation of palms) with second replants (third generation). The first replant was done by felling and windrowing, without removal of stumps; the second replants of the same blocks were done by clean clearing. The almost identical figures in successive generations can be interpreted in several ways:

- Gurmit (1991) concluded that, because clean clearing had not reduced the incidence, the method needed improving.
- If a large increase from one generation to the next is expected, it could be argued that clean clearing has been effective in preventing such a build-up (but the evidence for a build-up is equivocal; see Table 13.6).
- Comparison of clean clearing and windrowing in the same generation does not always show an advantage for clean clearing (Table 13.7), so the similar incidence in the two generations in Table 13.9 could, alternatively, be interpreted as evidence against a build-up from one generation to the next.

There is a danger of arguing in a circle, but considering all the results available, we suggest that:

- Clean clearing does not always reduce disease incidence (although what is meant by clean clearing may differ from one organisation to another).
- Incidence does not increase much from one generation to the next, but is more dependent on the strain of *Ganoderma* prevalent in the area, the soil type or some other predisposing factors.

Hasan and Turner (1994) found that very few seedlings planted over 1.5 m away from infected stumps became infected. One approach to replanting severely *Ganoderma*-infected fields could therefore be to plant down the centre of the interlines (4.5 m from the old stumps). It would probably take the roots of the seedling more than 2 years to reach the stumps, by which time, provided they were poisoned, they should have

Table 13.9 *Ganoderma* incidence in first and second replants of the same blocks

Block	First replant[a] (2nd generation of palms)		2nd replant[b] (3rd generation of palms)	
	Year planted	*Ganoderma* (%)	Year planted	*Ganoderma* (%)
1	1955	4.8 (10 years)	1978	2.8 (10 years)
2	1954	19.5 (12 years)	1976	21.6 (12 years)
3	1955	33.8 (9 years)	1975	32.4 (9 years)
4	1957	2.0 (9 years)	1980	3.5 (9 years)
Mean		15.0		15.1

Source: Amended from Gurmit (1991).
Method: [a]Felled and wind-rowed, stumps left *in situ*; [b]clean cleared by burning and raking to remove root and bole tissue.

rotted enough to be no longer infectious. Hasan and Turner (1994) also suggested that a 2-year fallow period should precede replanting, but this would only be acceptable from the financial viewpoint if some other crop, at least as profitable as oil palm, could be taken. Virdiana *et al*. (2012) found that delaying planting by 1 year after clearing and windrowing reduced disease incidence 9 years later from 32 to 11%. In contrast, Gurmit (1991) showed that 1 year under soya beans before replanting had no effect on subsequent disease incidence. Even underplanting, so that old and new stands overlap, may have little effect on disease incidence (Table 13.7). Wood (1999) made a theoretical comparison of underplanting with 80% *Ganoderma* losses by 20 years and clean clearing with no losses. Table 13.7 shows that this greatly exaggerates any real difference, but even with these unrealistic assumptions, on a discounted cash-flow basis, underplanting would be more profitable than clean clearing, because yield many years after the investment has relatively little value in a discounted cash flow. Virdiana *et al*. (2012) noted that the effect of a year's fallow on cash flow needed study, but in view of Wood's results, it seems clear that fallowing is unlikely to be profitable. Virdiana *et al*. (2012) also found that shredding the old palm material was as effective as fallowing in reducing infection.

If spores are the main mode of spread, rather than root contact (see Section 13.4.3.4), then the important factor would be a source of spores; for instance, infected old palms adjacent to the young palm area or windrowed old trunks. Burying old trunks has been proposed as a method of preventing release of spores (Santa Maria *et al*., 1996), but as noted in 13.4.3.4 (*Infection by root contact*), Flood *et al*. (2005) obtained high infection in seedlings planted next to trunk sections covered with soil, but no infection if the trunks were exposed. Routine removal of brackets (fruiting bodies) has also been advocated to prevent dispersal of spores (Sanderson *et al*., 2000).

Nazeeb *et al*. (2000) suggested planting at a higher than normal density when a high incidence of disease is anticipated. They presented yield data up to the seventh year of harvesting to support this suggestion, but by that stage, deaths were only 3–7%.

13.4.3.8 *Other methods of control*

Various attempts to control the disease with systemic fungicides have been made. Early work was not successful (Loh, 1977; Jollands, 1983), perhaps because massive lesions may already be present by the time external symptoms are seen. Trunk injection of fungicides may have some effect (Ariffin and Idris, 1993; George *et al*., 1996). Idris *et al*. (2004) recommended injection of hexaconazole, but Tey and Ahdly Arbain (2007) found this to be ineffective.

Biological control by inoculating lesions with cultures of various microorganisms was suggested by Varghese *et al*. (1976), and subsequent work was reviewed by Sariah and Zakaria (2000). These authors studied the effects of *Trichoderma harzianum*, alone and in factorial combination with a mycorrhizal preparation, dried palm oil mill effluent and calcium nitrate, on *Ganoderma* development in inoculated seedlings. Table 13.10 is based on their data and suggests that $Ca(NO_3)_2$, dried effluent and mycorrhizal inoculation may all have had some effect, but *Trichoderma* did not. However, Normahnani and Tey (2011) found that treatment with *Trichoderma* spp. or *Pseudomonas*

Table 13.10 Effect of various treatments on *Ganoderma* development in seedlings

Treatment		Foliar symptoms[a] (%)	Roots with lesions (%)	Bole infection (%)
Calcium nitrate	+	18.9	0.0	0.0
	–	41.0	27.6	23.8
Dried palm oil mill effluent	+	22.8	2.1	0.6
	–	37.1	25.5	23.2
Mycorrhiza	+	26.0	3.4	1.1
	–	33.9	24.2	22.8
Trichoderma harzianum	+	28.2	13.9	13.0
	–	31.7	13.8	10.8

Note: Four-month-old seedlings were inoculated with rubber-wood blocks. Data are main effects from a factorial design, from Sariah and Zakaria (2000).

[a] Percentage of leaves that were desiccated or chlorotic.

aeruginosa reduced incidence and severity of disease in seedlings inoculated with *Ganoderma*, and Ili Bazilah and Sariah (2011) found reduced severity after inoculation with *Pseudomonas* sp. Ginting *et al*. (2011) found that *Trichoderma* inoculation of the rubber-wood blocks used to raise *Ganoderma* reduced incidence in nursery tests from nearly 80% to below 40%, but mixing *Trichoderma* with the nursery soil had little effect. Yow and Jamaluddin Nasir (2001) claimed that young palms, inoculated with mycorrhiza in the nursery and planted next to diseased stumps, remained free of infection by *Ganoderma* for at least 3 years, whereas uninoculated plants were 'mostly infected'.

Excision of large, discrete lesions ('surgery') has been practised (Turner, 1968), sometimes successfully, but it is expensive, and treated palms may later collapse. Surgery is less likely to work with young palms than with old palms, but it is with young palms, with a long life ahead of them, where the greater benefit would be obtained from a 'cure'. Gurmit (1991) indicated that a backhoe could be used to do the surgery mechanically and gave figures for costs, but not for efficacy. Hasan and Turner (1994) obtained some benefit from surgery, in terms of better survival and yield 36 months after treatment, but according to Chung (2011a), surgery is no longer done.

Removal of diseased palms is widely practised. Such palms are identified in regular inspection rounds and removed by poisoning, felling, cutting up the trunk and excavating the bole tissue to hasten decay. Gurmit (1991) presented data which showed that this approach only slightly reduced the rate of spread of the disease. Over a 4-year period, incidence increased from 13 to 50% where infected palms were removed and from 12 to 57% without removal. Though small, this difference was statistically significant, but Rao *et al*. (2003) obtained no reduction in disease incidence from this approach. Idris and Ariffin (2005) showed very high infection of replacements after removing infected palms if stump tissue was not completely excavated, but replacement of single palms in mature stands is not worthwhile anyway (see Section 10.1.5).

Lim and Udin Wahidin (2011) showed that on peat in Riau, digging a trench around infected palms reduced infection of neighbouring palms from 25 to 2.5% 4 years later. This implies a fungal growth rate through the roots of 7–8 m in 4 years, much faster than some of the measurements discussed in Section 13.4.3.4. Chung (2011a) noted that with this method, harvesting of diseased palms could be continued until they died.

Mounding of the base of diseased palms with soil, after surgery, was suggested by Lim K.H. *et al*. (1995) (Plate XIVD). Numerous new roots developed from the trunk above the point of surgery, with treated palms suffering fewer deaths and some remission of symptoms. Hasan and Turner (1994) found that surgery was unnecessary, mounding alone being just as effective. Rao *et al*. (2003) observed that mounding of diseased palms did not affect the rate of disease spread, but Ho and Khairudin (1997) found that mounding reduced the death rate from 34 to 2%, over 24 months after treatment. Yields were over 30% higher because of this, the difference being mainly due to increased palm numbers, with little effect on yield per palm. Treatment with dazomet as well as mounding gave only a small additional benefit, insufficient to cover the extra cost. By 36 months, only 14% of mounded palms had died, compared with 71% of untreated palms, and yield was over 50% greater as a result (Ho, 1998). Wood (2007) quoted this in support of the idea that the 'late phase' disease follows prior root senescence. Mounding uninfected palms as a preventative measure does not appear to have been tested.

Sanderson and Pilotti (1997; also Sanderson *et al*., 2000) argued that the disease could be controlled by removing infected palms and ensuring that fruiting bodies were never allowed to develop to the point where spores were released. Whether this is a valid approach will depend on the extent to which spread is by spores, rather than root contact. Rao *et al*. (2003) found that in Sabah removing infected palms every 6 months did not reduce the rate of disease spread. In Riau, removing diseased palms was less effective than digging a trench round them and leaving them *in situ* (Lim and Udin Wahidin, 2011). However, neither of these papers specifically mentioned preventing production of fruiting bodies, and the method needs to be tested further.

13.4.3.9 *Detection of diseased palms*

Early detection of infection is important for some of the proposed control methods; the appearance of fruiting bodies is often the first sign of infection. Mohd As'wad *et al*. (2011) showed that ergosterol could be detected in infected, but not in symptomless, palms. The use of molecular markers was discussed by Hushiarian *et al*. (2013). Tan Y.C. *et al*. (2013) identified differences in gene expression between healthy and infected palms, but it is not clear how these methods could be used on a plantation scale. The remote sensing methods described by Lelong *et al*. (2009), Santoso *et al*. (2011) and Liaghat *et al*. (2014) may be more promising.

13.4.3.10 *Breeding for resistance*

This is an obvious approach, particularly given its success against *Fusarium* wilt (see Section 13.4.2.9). Differences in incidence between West African and Deli material were observed in the field in Indonesia (Akbar *et al*., 1971), and de Franqueville *et al*. (2001) showed significant differences between families in *Ganoderma* incidence in 8 of 12 breeding trials and between clones in six of seven clone trials. In one trial, for example, incidence in individual families ranged from 12 to 75%, 24 years after planting. An index was calculated, in the same way as for *Fusarium* resistance (see Section 13.4.2.9), and showed reasonable consistency for the same parents in different crosses and trials. Durand-Gasselin *et al*. (2005) found that Deli x Yangambi crosses had lower incidence than pure Deli *dura*. They also found that interspecific *E. guineensis* x *E. oleifera* hybrids had lower incidence than pure *E. guineensis* but quoted other work showing little difference between the species. In a clone trial in Sumatra, Durand-Gasselin *et al*. (2011) found two clones with over 35% infection 8 years after planting and another clone with only 5% infection.

For an effective breeding programme, a method of screening plants at the nursery stage is needed, though it should be noted that it has not been established that resistance to 'early phase' infection will also be effective against the 'late phase' disease. Amiruddin (1993) used several different *in vitro* inoculation methods to compare three clones and observed consistent differences among the clones in susceptibility. Rees *et al*. (2007) showed that size of the inoculum was important, that rubber-wood blocks were a superior substrate to oil palm blocks and that attaching the inoculum to the root with parafilm gave high and rapid infection. Infection occurred several months earlier and was more severe if shade was used to keep soil temperature below 35°C. Strains of *G. boninense* differed in aggressiveness, with less aggressive strains only able to infect wounded roots. Breton *et al*. (2006) used methods very similar to those of Rees *et al*. to inoculate germinated seeds, with the first symptoms seen within 3 months. Rahmaningsih *et al*. (2011) gave a brief report of 78 nursery screening trials, each testing from 10 to 50 crosses. There was significant genetic variation in most trials, but no crosses were completely free of infection. Rahmaningsih *et al*. (2013) showed that Deli material was more susceptible than AVROS, with crosses intermediate. At time of writing, we are not aware of any seed suppliers offering *Ganoderma*-resistant seed, but this should only be a matter of time. Grogan and Mosquera (2015) used a model to show that, if resistant material is available, it will not be worth spending on control measures, such as mounding or palm removal, towards the end of the planting cycle.

Naher *et al*. (2011) found increased chitinase gene expression in plants infected with *Ganoderma*, but it is not clear whether expression differed between tolerant and susceptible material. Chitinase also increased following infection with *T. harzianum* and so may be a universal response to fungal infection.

13.4.3.11 *Conclusion*

Ganoderma has been a serious problem in some areas in Malaysia and Indonesia for 50 years. Despite much recent research, our knowledge of the disease still depends heavily on anecdotal evidence, and several aspects remain surprisingly obscure. A great deal has been invested in clean clearing at replanting time, but the evidence that this has had any effect is equivocal.

There is no convincing evidence that the problem is getting worse; it still needs to be confirmed that the disease increases from one palm generation to the next. An attempt to collate the extensive data that must be available in plantation records, in relation both to palm generation and to replanting methods, was proposed by Wood (1999) and might be very informative, but has not been done. More research on predisposing factors also appears to be needed.

Mounding of diseased palms, perhaps combined with fungicide treatment, can delay death and improve yield, but mounding of symptomless palms as a preventative measure has not apparently been tested. Mounding appears to be preferable to the removal of diseased palms, which does little to prevent disease development. Systematic removal of all fruiting bodies, to prevent spread by spores, has yet to be tested as a means of control. It appears to us that, in the longer term, the best approach to controlling the disease in areas where it is prevalent will be to develop tolerant material, in much the same way as has been done for *Fusarium* wilt.

13.4.4 Marchitez sorpresiva, sudden wither or sudden wilt

13.4.4.1 *Distribution*

This disease has been serious on plantations in Colombia, Ecuador and Peru (Plate XID). In Surinam, the disease has been described as 'hartrot' (van Slobbe *et al*., 1978), and a similar condition has occurred in Bahia, Brazil.

13.4.4.2 *Symptoms*

The disease is characterised by a sudden rotting of all developing bunches, a reddish discoloration of the top of the petioles and rapid drying out of the leaves from the oldest ones upwards. This drying out is preceded by the appearance of reddish-brown streaks at the ends and centres of the lowest leaflets. The leaf then becomes successively pale green (as in nitrogen deficiency), yellow, reddish brown and ash grey (Genty, 1981). The palm dies in 2–3 weeks, and as soon as the external symptoms appear, the root system will be found to have rotted and to a large extent dried out. Similar symptoms, though proceeding at a slower rate, are sometimes seen; this has been referred to as 'marchitez progresiva' or 'marchitez lenta'. However, there may be confusion with fatal yellowing (Corrado, 1970). In the typical marchitez symptoms, the spear is initially unaffected. The root cortex decomposes in wet weather but in the dry season tends to become necrotic and to detach itself from the stele. The rot starts from the extremities and moves towards the trunk and the lower roots. The trunk itself usually remains healthy, but cases are reported where the base is rotted sufficiently to form a cavity (Martin, 1970; van den Hove, 1971). Palms have been attacked by marchitez from the age of 1 year.

13.4.4.3 *Cause*

It appears that the cause is infection by a protozoan flagellate, *Phytomonas staheli*. These have been found in several countries in association with the disease, in the phloem of roots, meristem zone, spear base and inflorescence stalks (Dollet *et al*., 1977; Dollet and Lopez, 1978; Dzido *et al*., 1978), and are also present in certain weed species (Dollet, 1982).

A connection between infection and insect attack has been suggested. Lopez *et al*. (1975) considered that the root miner, *Sagalassa valida* (see Section 14.7), might be a carrier. In Colombia, where the disease devastated an area of palms growing in a heavy stand of *Panicum maximum*, the bug *Myndus crudus* (*Haplaxius pallidus*) was found on palm leaves, while the nymphs were present on the roots of *P. maximum*. The use of herbicides and insecticides reduced the incidence of marchitez (Mena Tascon *et al*., 1975), while inoculation experiments (Mena Tascon and Martinez-Lopez, 1977) also suggested that *M. crudus* might be playing a part in the transfer of the disease. However, Martinez *et al*. (2011) considered that the disease studied by Mena Tascon and co-workers was actually marchitez letal (see Section 13.4.5).

Desmier de Chenon (1984) and Perthuis *et al*. (1985) claimed that the bug *Lincus lethifer*, which lives in the axils of the leaves (Dollet *et al*., 1977), was the vector of the flagellate. Symptoms of the disease developed within a few months of the bug being released onto young palms, in the first instance at sites far from any cases of the disease and in the second on a caged palm surrounded by other, healthy palms. It has also been shown that *L. lethifer* and another species, *L. tumidifrons*, can transmit the flagellates to coconut palms, where they cause 'hartrot' (IRHO, 1992b).

13.4.4.4 *Control*

In view of the uncertainty of diagnosis, it is difficult to recommend definite control measures. On the grounds that *S. valida* was likely to be playing a part in the

transmission of the disease, applications of insecticide around the base of the palms have been used to suppress the insect and were strongly recommended in Colombia, Ecuador and Peru (Lopez *et al*., 1975; Genty, 1977). As noted in Section 13.4.4.3, it now appears that *Lincus* spp., not *S. valida*, are probably involved, and Gomez *et al*. (1996) indicated that a combination of insecticide and herbicide (to eliminate alternate host plants for *Lincus*) has proved useful.

Cases of marchitez in *E. oleifera* × *E. guineensis* hybrids have not been recorded until recently, so it is possible that the planting of hybrids may be a method of avoiding the disease. In Surinam, however, some hybrids have suffered from 'hartrot', although wild *E. oleifera* palms have not been observed with the disease (Alexander and Kastelein, 1983). Certain *E. guineensis* palms on a plantation devastated by this disease remained healthy (Hartley, 1988), so it may be possible to select resistant progenies within the species.

13.4.5 Marchitez letal, lethal wilt or Bajo Upia wilt

This disease has caused the loss of several thousand hectares in Colombia (Martinez *et al*., 2011). It was first reported in 1994, but Martinez *et al*. considered that a report of marchitez sorpresiva in 1975 was actually marchitez letal. According to Gomez *et al*. (2003), it affects palms between 15 and 18 years old, but Arango *et al*. (2011) found symptoms on 3-year-old palms.

Symptoms: Drying of older leaves and then younger leaves become successively yellow, bronze and dry. Bunches and inflorescences rot, and roots are found to be rotten and sometimes reddish (Gomez *et al*., 2003). These symptoms appear to be more or less identical to those described for marchitez sorpresiva (Section 13.4.4).

Cause: The pathogen remains unknown, but it appears to be transmitted by an insect, *Myndus crudus* (Martinez *et al*., 2011). It occurs mainly where drainage is poor (Gomez *et al*., 2003).

Control: Interspecific hybrids appear to be tolerant. There are also large differences in susceptibility between different sources of *E. guineensis* (Cenipalma, 2009). As with marchitez sorpresiva, a combination of insecticide and herbicide appears to slow the spread of the disease (Arango *et al*., 2011).

13.4.6 Upper stem rot

Thompson (1931, 1937) described a lethal trunk rot that was serious only on deep peat and inland valley soils. The disease has appeared on other soils in both Malaysia and Indonesia but is usually sporadic and not of major importance.

13.4.6.1 *Symptoms*

Typically, the lower leaves first become yellow, and this symptom gradually extends to the middle leaves and then to the spear. It is evident that spore infection of leaf bases takes place and that from these the fungus gains entry to the peripheral tissues of the stem. The brown decay appears to proceed slowly inwards from the leaf bases, and in many cases, a typical collapse of the stem at one point occurs, usually following high winds (Plate XVB). The rot spreads upwards and downwards in the stem, eventually killing the palm by invading the crown. Two forms of fruiting bodies (normal and resupinate) appear but only on palms where the leaf bases are extensively decayed. These are small, greyish-brown bodies with velvet-brown margins and are inconspicuous among the leaf bases. The disease is confined to the stem and does not enter the roots.

13.4.6.2 *Cause and distribution*

Phellinus noxius; probably also *Ganoderma*. Thompson (1931) described the disease, but little work was done on it until Navaratnam and Chee (1965) and Turner (1969, 1981) gave accounts of its symptoms, incidence and control. *Ganoderma* (see Section 13.1.6.3) is also often found in association with upper stem rot, perhaps as a secondary infection, but Turner (1981) noted the possibility that this fungus might sometimes cause the disease, and isolations made in Sumatra have confirmed the presence of *Ganoderma* in all cases of upper stem rot examined (Flood *et al*., 2002), sometimes together with *Phellinus* sp. (Hasan *et al*., 2005). Hasan *et al*. were unable to cause infection by inoculating with *G. boninense* or *P. noxius*, although Hartley (1988) stated that pathogenicity of *P. noxius* had been proven by inoculation experiments. Hasan *et al*. (2005) suggested that upper stem rot infection only occurs when a sufficiently large inoculum has built up in dead material in frond axils, but Rees *et al*. (2007) found that *G. boninense* failed to grow through organic debris from frond bases. Rees *et al*. (2012) found that infections were all genetically unique, suggesting single spores as the source.

13.4.6.3 *Control*

As there is usually much penetration of the stem by the time sporophores appear, it is desirable to detect the disease at an earlier stage. This can be done on palms of 10 years or older by striking the leaf bases with a wooden

pole to detect the dull sound of an infected base. Incidence is insufficient to justify surveying palms below 10 years old. When the diseased leaf bases are cut away, the extent of the infection can be explored. The lesion is excised from the stem with a harvesting chisel, and the cut surfaces are treated with a preservative such as coal tar (Turner, 1969). Treated palms give as high a yield as untreated palms, so the measures are considered worthwhile wherever incidence is likely to be significant.

In a fertiliser experiment containing different progenies, there was evidence that potassium reduced incidence and also that progeny differences in susceptibility existed (Navaratnam and Chee, 1965).

13.4.7 Red ring disease

13.4.7.1 *Distribution*

Red ring is confined to South America, where it has been found in oil palms in Venezuela, Surinam, Brazil and Colombia, where the similar disease of coconuts is prevalent. In unprotected areas, incidence can increase rapidly; Malaguti (1953) cited a group of 100 palms showing only 16 doubtful cases in January which by August had 22 deaths, nine doubtful or affected cases and only 69 palms remaining healthy.

13.4.7.2 *Symptoms*

The symptoms of this disease have been described by Malaguti (1953). The centre of the crown takes on a dwarfed appearance and the newly opened leaves become bundled together into an erect, compact mass, the leaflets being corrugated, twisted and sometimes adhering to the rachis. Gum is exuded. Later, this crown of leaves turns slowly yellow and dries out, the rachis being a light brown colour with yellow spots. Developing bunches rot and inflorescences fail to set fruit.

The diagnostic internal symptom is the brown cylindrical ring found in the trunk, 7–8 cm from the periphery and 1–2 cm broad (the ring is red in coconut, hence the name). This ring is most distinct towards the base of the palm (Plate 13.3), but the infection proceeds upwards into the petioles and rachis of the leaves in the crown in which, on cross-sectioning, necrotic areas or spots can be found. This infection does not, however, invade the tissues of the stem apex or surrounding very young leaves.

13.4.7.3 *Cause and spread*

The coconut nematode, *Rhadinaphelenchus cocophilus*, seems first to have been recorded on oil palms by Freeman (1925) in Trinidad. Proof that the nematode

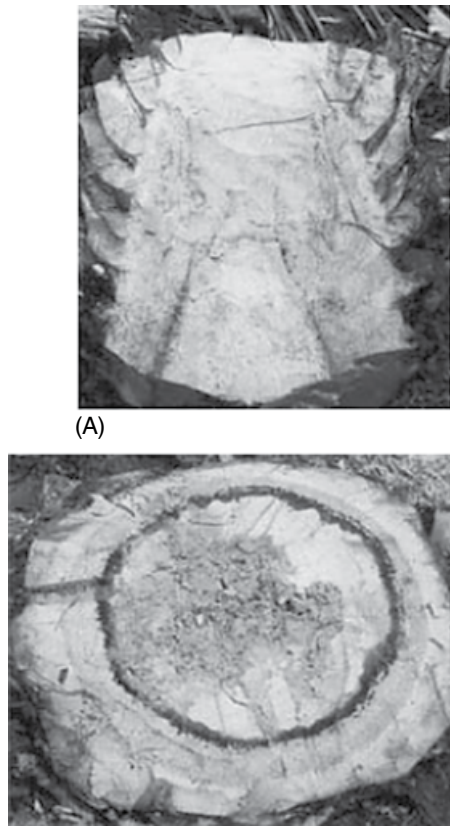

Plate 13.3 Longitudinal (A) and transverse (B) sections of an oil palm suffering from 'red ring' in Venezuela.

was the cause of red ring was obtained by Malaguti (1953), who did tests with inoculum from both the oil palm and the coconut. The disease appeared 2–10 months after inoculation.

Giblin-Davis *et al*. (1989) described the nematode as an obligate plant parasite, which is only able to reproduce in palm tissue but can also parasitise the weevil *Rhynchophorus palmarum*, which thus acts as a vector for the palm disease. Gerber and Giblin-Davis (1990) found that 90% of the weevils emerging from infected palms carried the nematode either internally or externally. According to Gomez *et al*. (1996), *Metamasius hemipterus* is the main vector in Colombia, but Chinchilla *et al*. (1996) considered that the latter species was not a vector in Costa Rica, and Schuiling and Dinther (1982) found that *M. hemipterus* did not carry the nematode in Brazil. Warwick and Bezerra (1992) showed that transmission in coconuts could also occur

by root contact, so this possibility cannot be ruled out for oil palm.

In Brazil, Schuiling and Dinther (1982) found both the nematode and *R. palmarum* on wild *Oenocarpus distichus* palms and suggested that these may form a reservoir for infection of plantation palms.

13.4.7.4 *Control*

Incidence on an affected estate in Venezuela was greatly reduced by regular sanitary measures (Hartley, 1988, p. 629). Diseased palms were poisoned, felled and burnt, with the whole estate being inspected every 2 months for diseased palms. Most important was protection of the palm against the type of wounding that provides sites for *R. palmarum* to lay eggs. A high incidence of red ring in Brazil was preceded by very close leaf pruning which had resulted in wounding of the trunk (Schuiling and Dinther, 1982); care must be taken when removing leaves to make a clean cut sufficiently far up the petiole to avoid this. Circle weeding with herbicides instead of with hand tools may also help to prevent wounding. Regular disinfection of tools has been suggested, together with treatment of the cut leaf and bunch stalk surfaces, but provided that wounding is avoided, it is doubtful whether such precautions are necessary.

Oehlschlager *et al.* (1993) described a pheromone-based trapping method for the vector, *R. palmarum*. Chinchilla *et al.* (1995) showed that after a year of trapping, red ring incidence was reduced by two-thirds. Chinchilla *et al.* (1996) showed that pheromone trapping could also be used against *M. hemipterus*. Biological control of *R. palmarum* has been proposed (Moura *et al.*, 2006).

13.5 DISEASES OF THE BUD OR STEM APEX

Under this heading are grouped diseases occurring in the emerging spear and younger leaves inside the crown. Such diseases normally move towards the growing point through the enclosed developing leaves of the 'cabbage', and when they reach it, the palm is killed. Bud and spear rots have occurred widely in all three continents. Investigation is difficult owing to the position of the transition zone, often in the heart of the palm, the rapid entry of secondary organisms into any rot within the cabbage and the multiplicity of confusing symptoms, some of which may be similar to those of deficiencies or genetic abnormalities.

Turner (1981) suggested that the term 'spear rot' should apply to diseases in which the primary rotting affects the spear, while 'bud rot' should be used only for diseases first destroying the unemerged leaves and the hidden base of the spear and also, usually, the apical meristem. The latter diseases are usually fatal, the former frequently not.

13.5.1 Spear rot–little leaf disease

13.5.1.1 *Distribution*

This disease, previously called bud rot–little leaf, caused serious losses in the oil palm areas of southern Congo, where deaths exceeding 30% were recorded. Elsewhere in West Africa and in Asia, cases rarely exceed a few per cent and are often confined to certain progenies; deaths occur but are rare. Chinchilla (2008) noted that a non-lethal 'common spear rot' can be found at low frequency in all oil palm growing areas, often associated with crown disease (see Section 13.3.1).

13.5.1.2 *Symptoms*

The first sign of attack is a wet, brown rot on the lower part of the unopened spear leaf. Duff (1963) described how in very mild cases only the leaflets may be affected; the leaflet rot is passed from spear to spear until either it develops further or the palm grows out of the attack. Normally, however, the rachis becomes infected, and the spear collapses and hangs down; it is not uncommon to find a spear leaf, in which the infected portion has rotted away altogether, lying on the ground where it has fallen. The spear rot grows downwards and may become a bud rot, but it is only if this reaches the growing point that the palm dies. In other cases, the first leaves to emerge after the spear rot are stumps consisting of the malformed basal portion of the rachis. Subsequent 'little leaves' are very short with a few corrugated shortened leaflets, but each successive leaf will be longer, and the leaflets less abnormal, until fully normal leaves are again produced. Little leaf is therefore a recovery symptom and does not precede rotting. Very similar symptoms may occur after damage by insects such as *Oryctes* (Chapter 14).

13.5.1.3 *Cause*

Many causes have been assigned to the 'little leaf' symptom; for the early history of investigations, Bull and Robertson (1959) should be consulted. One common cause is boron deficiency (Ferwerda, 1954) (see Section 11.4.1), but this is not preceded by spear

rot. Robertson (1960), working in Nigeria on palms of a susceptible progeny having regular cycles of infection, showed that spear rot–little leaf disease was an active pathogen, since the appearance of little leaves and bud rot could be prevented by cutting off the spear below the rotted portion. Although prior insect attack is often suspected, it is not known for certain how spear infection takes place.

A bacterium of the genus *Erwinia*, similar to *E. lathyri*, was consistently isolated in Congo by Duff (1963) from young lesions and from tissue in advance of visible rotting, and inoculation experiments showed that spear rot–little leaf symptoms could be induced by it. Susceptibility appears to have several aspects. In a field in Nigeria, the disease was confined to one progeny. Genetic differences were also found in Congo, where there was an association between rate of growth and disease incidence. The former was judged by the rate of elongation of spears, and in susceptible palms, the elongation rate fell below normal levels 2 or 3 weeks before an attack of the disease. It was believed that these circumstances, encountered in 'unhealthy' palms, allowed susceptible tissues to be exposed to infection for longer periods than normal. Palms in which the growth rate was artificially reduced by root or leaf cutting showed greater than normal susceptibility. In some instances, there has been high incidence at either the beginning or end of the rains (Turner, 1981).

Kochu Babu (1988) described a similar disease from Kerala, southern India, where frequency of the disease increased with proximity to coconuts affected by root wilt or *Areca* palms affected by yellow leaf disease (Kochu Babu and Ramachandran Nair, 1993). Mycoplasma-like organisms have been found in tissues of palms with these diseases, but were not found in oil palms with spear rot (Kochu Babu, 1988). Spear and bud rots in America have different and varying symptoms (see Section 13.5.2).

13.5.1.4 *Control*

Duff (1963) provided growth and health records showing that the more vigorous progenies suffered less from the disease, and he inferred from this that anything interfering with vigorous growth increased susceptibility. While, therefore, the disease is not likely to be serious enough for control measures to be taken in areas where growth conditions, particularly those of water and nutrient supply, are good, in marginal areas, the planting of particularly vigorous progenies might be considered (Hartley, 1988).

13.5.2 Fatal yellowing or lethal bud rot (pudrición del cogollo or PC)

A bud rot with variable symptoms, but not usually including the typical 'little leaf' progression, has caused serious damage on plantations in Central and South America. Some plantations have been totally devastated, while others have suffered serious losses with many palms remaining in a moribund, unproductive condition for long periods. The first outbreak may have been in Panama in 1928 (Richardson, 1995). There has been extensive research on this disease in recent years, much of it reviewed by Gomez *et al.* (2000). Turner (1981) called the disease fatal yellowing, from one of the characteristic symptoms. However, symptoms differ quite considerably between different areas, and the disease is not always fatal (Acosta and Munevar, 2003). There appear to be both 'acute' and 'chronic' forms, which may be different manifestations of the same disease, as with *Fusarium* wilt (see earlier), but it appears more likely that more than one disease is being described under one name (IRHO, 1992b; de Franqueville, 2003) (see Section 13.5.2.2).

13.5.2.1 *Symptoms*

In the Llanos Orientales region of Colombia, typical symptoms were described by Gomez *et al.* (2000) as dry or wet spear rot, accompanied by yellowing of young fronds (Plate XVD) appearing during wet periods but disappearing during the dry season. The rot spreads downwards towards the growing point, but affected palms usually recover. Turner's description (1970) of the symptoms in La Arenosa plantation in northern Colombia was quite similar, but it appears that the disease killed many palms when the rot spread to the growing point. Turner noted a tendency for four to six young spear leaves to remain unopened and stuck together, as a 'baton', but he was not convinced that this was a valid disease symptom. When this symptom was seen, spear rot was said to follow within 10–30 days. The rot spread downwards, and within 1–9 weeks, the spear collapsed, the rot reached the growing point and the palms died (Plate 13.4). This seems to be the typical 'acute' form of the disease.

As already noted, the symptoms and the severity of the disease appear to vary considerably from one country to another. De Franqueville (2003) described the disease as having two phases: in the first phase, which may last for up to 12 years, increase is more or less linear, but then as foci start to develop, it moves into a phase of exponential increase. Table 13.11 lists the

Plate 13.4 Young palm suffering from spear rot, with no central leaves, Panama.

Table 13.11 Symptoms of fatal yellowing in different countries

Symptoms	Colombia La Arenosa	Colombia Llanos Orientales	Ecuador	Surinam	Brazil	Panama	Nicaragua	Costa Rica
'Baton' effect	Often	No	No	–	No	–	–	–
Chlorosis (no. of leaves)	A few	A few, mild	A few	Few initially, later many	Many	Often none	Some	A few
Spear break or collapse	Yes	Yes	Yes	Yes	Yes	–	–	Yes
Spear rot	Yes	Yes	Rapid	Usually	Eventually	Yes	Yes	Often
Leaves reduced in size	No	No	No	–	Yes	–	Yes	No
Spread to meristem	Rapid	Rare	Rapid	Slow	Slow	Slow	Slow	Rare
Bunch rot	No	No	–	No	Sometimes	No	–	No
Root rot	–	–	No	No	Yes	–	–	Malformation
Recovery or remission	Rare	Yes	No	Yes	Yes	Yes	Yes	Yes
Death	Yes	Rare	Yes	Yes	Eventually	–	Yes	Rare
Time from 1st symptom to death	4–5 months	Death rare	1–2 months	1–2 years	1–3 years	–	–	Death rare

Source: Based on Turner (1970, 1981), van de Lande (1993), van Slobbe (1986), Swinburne (1990, 1993), Chinchilla and Duran (1999) and personal observations (R.H.V. Corley). – no information available.

symptoms described by various authors. The main differences are in the extent of chlorosis, the speed with which the spear rot develops and whether or not the rot reaches the growing point, causing death. The disease appears to take its most acute form in Ecuador and southwestern Colombia, while the most extreme chronic form is that seen in Brazil (Para state) (Plate XVC) and Surinam. De Franqueville (2003) noted that more acute symptoms were seen elsewhere in Brazil. Swinburne (1990, 1993) reviewed symptoms in Brazil, Ecuador and Colombia; he noted that chlorosis is a common response to stress in many plants and is poor evidence for a common cause. The symptoms that he described for Brazil are similar to those for leaf mottle (see Section 13.3.4); they are also similar to those described for iron deficiency by Setyobudi *et al*. (1998).

13.5.2.2 *Cause*

Torres *et al*. (2010) reported isolation of *Phytophthora palmivora* from diseased palms in the central and south-western (Pacific coast) regions of Colombia. Nursery seedlings were inoculated and developed symptoms, and the pathogen was reisolated. Thus, it seems clear that *P. palmivora* is involved, confirming the view of O.A. Reinking in 1928 (Richardson, 1995). However, it should be noted that, as symptoms vary from place to place, it is not certain that they are of the same disease or have the same cause. Thus, earlier observations on other possible causes of the disease may still be relevant. Nieto (1992) believed that the Colombian and Brazilian forms were different diseases, as did Swinburne (1990, 1993). Martinez *et al*. (2009) speculated that the different forms of the disease might depend on environmental conditions, or on which opportunistic microorganisms colonised tissues initially affected by *P. palmivora*, while Drenth *et al*. (2012) suggested that secondary invaders and *Rhynchophorus* might kill infected palms. Ochoa and Bustamante (1974) isolated *F. moniliforme* var. *subglutinans* from diseased palms in Colombia, and inoculation of palms grown in high humidity and low light intensity caused spear rot. Nieto (1993) obtained symptoms by inoculating with *F. solani*, but Turner (1981) considered that the symptoms of fatal yellowing were unlike those normally associated with *Fusarium* attack.

It was also reported from Colombia that *Thielaviopsis paradoxa* causes the disease; drying and necrosis of the central leaves was induced by inoculation of seedlings (Nieto, 1993; Gomez *et al*., 2000). Alvarez *et al*. (2012) inoculated seedlings with 111 different *T. paradoxa* isolates from four regions in Colombia, one site in Brazil and one in Ecuador. All caused symptoms, of varying severity. It is possible that the chronic form of the disease seen in the eastern zone of Colombia is caused by *T. paradoxa*, while the acute form elsewhere is caused by *P. palmivora*, or a combined infection by both species. It should be noted that the sexual stage of *T. paradoxa*, *Ceratocystis paradoxa*, is associated with dry basal rot (see Section 13.4.1), which has quite different symptoms. The fungus is described by Turner (1981) as 'one of the most common fungi recorded on the oil palm throughout the world' and is not normally pathogenic. If this is one of the pathogens involved, therefore, it is not yet clear what renders palms susceptible to it in Latin America, but not elsewhere.

Other fungi that have been isolated from diseased spears include *F. oxysporum* and *Botryodiplodia* sp. in Colombia and *F. solani* and *Sclerophoma* sp. in Ecuador. Invasion of bud tissue by many species of bacteria follows the spear rot. Pathogenicity of these organisms has not been demonstrated, but de Franqueville (2003) speculated that a joint infection by a fungus and a bacterium may be involved. In Brazil, a viroid was suggested as the cause (Singh *et al*., 1988), but viroid-like RNA was found in both diseased and healthy palms (Beuther *et al*., 1992).

Insects have also been associated with the disease, but intensive efforts to identify vectors for the disease in Ecuador and Brazil were unsuccessful (IRHO, 1992b; de Franqueville, 2003). *Phytophthora* is soil borne, but Drenth *et al*. (2012) speculated that it might be carried up to the spear leaves of mature palms by grasshoppers (Tettigoniidae), which may lay eggs on the spears.

Wood (quoted by Swinburne, 1990, 1993) found that application of several different herbicides to the roots could cause symptoms similar to fatal yellowing. This suggests the possibility that, if diseased palms were poisoned as a control measure, transmission of herbicide to healthy neighbours might occur through root contact. This could be a contributory factor to the apparently rapid spread of disease sometimes observed, as the neighbours would then show symptoms and would be poisoned in turn. Swinburne noted that this could not be the only cause, as fatal yellowing has been observed on smallholdings never treated with herbicides.

13.5.2.3 *Predisposing factors*

Whatever the pathogen, it is clear that there must be predisposing factors for the disease to develop, and there has been much more work on this aspect than for most other oil palm diseases (Chinchilla, 2008). It has frequently been associated with poor drainage; Alvarado *et al*. (1997) found, in nursery trials with a susceptible progeny, that poor drainage led to significantly higher spear rot incidence than 'excessive drainage'. However, drainage improvements did not slow the spread of disease in Ecuador (de Franqueville, 2003). Acosta *et al*. (2002) noted that incidence is maximal during periods of high humidity. Drenth *et al*. (2012) suggested that the milder form of the disease occurred where there was a dry season, allowing recovery from *Phytophthora* infection which requires wet conditions, but in eastern Colombia, recovery occurs even under irrigation, though it may take longer (I. Ochoa, pers. comm., 2012).

Compacted soils and unbalanced nutrition have been mentioned as predisposing factors: in northern Colombia, areas with compacted ex-pasture soils suffered the

highest casualties. A low potassium/magnesium ratio was also suspected, but corrective manuring did not stop the disease spreading in La Arenosa (Hartley, 1965; Turner, 1981). Munévar *et al.* (2001; also Acosta and Munévar, 2003) recorded higher incidence of the disease on soils with high clay content, on compacted soils and on poorly drained soils, and they found a much slower rate of spread of the disease where drainage was improved. De Franqueville (2003) mentioned several studies on trace elements but noted that differences between diseased and healthy palms may be effects, not causes. In Colombia, leaf levels of phosphorus, copper and particularly potassium were lower in infected than in healthy palms, while calcium and magnesium levels were higher (Munévar *et al.*, 2001). Acosta *et al.* (2002) found that affected palms gave yield responses to higher levels of fertiliser than healthy palms, implying poor nutritional status. They considered that a long period of low fertiliser inputs in the 1990s had increased susceptibility in many plantations.

Chinchilla and Duran (1999) described a 'dry spear rot' in Costa Rica, which appears similar to the milder forms of fatal yellowing in Colombia. They noted particularly that the root systems of both healthy and diseased palms in affected areas were poorly developed, with many malformations (corky texture, abnormal branching), and that in affected palms, vegetative vigour was reduced before symptoms developed. In some areas, recovery occurred after drainage and other management aspects were improved (see *Control*). They concluded that any pathogens were a secondary problem and that the disorder resulted from stress caused by poor soil aeration, in areas with a high water table and inadequate drainage or suffering from soil compaction or with shallow soil overlying gravel. However, de Franqueville (2003) quoted several studies in other countries showing that the root system of affected palms was normal and healthy.

13.5.2.4 *Effects on yield*

In some regions of Colombia and in Costa Rica, affected palms usually recover (Chinchilla and Duran, 1999; Gomez *et al.*, 2000; Acosta *et al.*, 2002). Santacruz *et al.* (2000) found that yields of fruit were depressed by 30–40% in the first 2 years after infection; oil/bunch was also slightly depressed. Acevedo *et al.* (2000) showed that both FFB yield and oil/bunch were reduced, and oil yield was halved in the worst affected palms. Santacruz *et al.* (2000) stated that 80–90% of palms recovered within 1.5–3 years. Surveys in the Llanos Orientales have shown that up to 100% of palms in some blocks have been infected but with complete recovery so that a more or less complete stand remains (I. Ochoa, pers. comm., 2012). Chinchilla and Duran (1999) stated that 2 years after the first symptoms had been seen, yields were still below those expected from palms of that age.

In other areas, the disease may be fatal, and very heavy losses of palms have occurred, with severe effects on yield. The heaviest losses have often been associated with deliberate destruction of diseased palms, and sometimes of healthy palms surrounding diseased points, in an effort to prevent the disease spreading. It appears not always to be appreciated that, if 14% of palms show symptoms (whether fatal or not), and those palms and their six immediate neighbours are destroyed, close to 100% mortality may result. Van Slobbe (1988) noted that 46,000 palms had been 'lost to the disease' on one plantation in Brazil but also that 'all diseased palms have been eliminated within a month after detection'. De Franqueville (2003) later reported that this plantation had been abandoned because of disease losses (but *Fusarium* wilt also occurred there). Van de Lande (1993) recommended that even recovering palms should be destroyed in Surinam, as it was not certain that they were not still infectious. In some regions in Colombia, diseased palms must be destroyed as soon as they are seen (Resolutions 000716 of Feb 2010 and 002854 of Aug 2012 from Instituto Colombiano Agropecuario). With such policies in place, it is impossible to ascertain the true effect of the disease on palm mortality.

13.5.2.5 *Control*

Speculative prophylactic applications of mixed fungicides and insecticides have not been successful (e.g. Gomez *et al.*, 2000). Surgical removal of rotting spear tissues is currently recommended in Colombia (Martinez and Sarria, 2013), although Santacruz *et al.* (2000) stated that recovery was just as good if no surgery was done. It appears that if the disease is detected early enough, then only mild surgery of a few spear leaves is needed, and recovery can be quite rapid.

Destruction of infected or suspect palms has been widely practised but, as noted earlier, this may have contributed as much to the problem as to control. None the less, it is still being recommended on the assumption that it is the best way to control *P. palmivora* (Guest, 2012). Corredor and Gomez (2009) argued that eradication could significantly slow the progress of the disease, giving the plantation time to plan for eventual replanting. They also said that if the disease is less aggressive and palms recover, then eradication can be

suspended – but if eradication is done thoroughly, there will be no opportunity to observe recovery. There is a significant problem here: if the disease is going to prove fatal, as it has in parts of Colombia and Ecuador, then rapid eradication of infected palms may help to control it. If, on the other hand, infected palms will recover anyway, as in Costa Rica and the eastern zone in Colombia, eradication only serves to turn a fairly minor problem into a disaster. Thus, with a new outbreak, the grower needs to know which form of the disease it is, but at present, there is no way of distinguishing.

Chinchilla and Duran (1999) indicated that the disorder could be prevented by improving drainage, mulching with composted empty bunches, and paying particular attention to potassium and phosphate nutrition. Affected blocks more than 20 years old were replanted, after subsoiling to rectify soil compaction. In Colombia, the problem has been greatly reduced where drainage was improved (Munévar *et al*., 2001; Acosta and Munévar, 2003). Deep ploughing to improve soil aeration and fertiliser application to lower the [Ca + Mg]/K ratio also reduced disease incidence (Acosta *et al*., 2002).

13.5.2.6 *Breeding for resistance*

It is possible that there may be tolerant lines within *E. guineensis*: there are several reports of differences between progenies in disease incidence (e.g. Santacruz *et al*., 2000), with Deli × AVROS material apparently being more susceptible than other origins in Colombia. In Ecuador, de Franqueville (2003) reported differences in the rate of disease development between crosses of Deli × La Mé origin. Amblard *et al*. (2009) found differences in susceptibility between crosses and a correlation between resistance to the fatal form of the disease in Ecuador and the milder form in eastern Colombia (also Louise *et al*., 2012). Chinchilla *et al*. (2006) found differences in incidence between origins in Ecuador and Costa Rica, with several origins less susceptible than Deli x AVROS material.

Ayala (1999) described a method for testing the susceptibility of individual palms by inoculation of petiole sections with *T. paradoxa*, thought at that time to be the cause of the disease. Martinez *et al*. (2011) outlined a possible *in vitro* test for resistance to *P. palmivora* using inoculation of leaflet sections. Alvarado *et al*. (2014) suggested that crosses more tolerant of abiotic stress (drought, low temperature, low solar radiation) were also more tolerant to the disease.

On La Arenosa plantation in Colombia, several plots of *E. oleifera* × *E. guineensis* hybrids survived, while large numbers of the surrounding *E. guineensis* palms died. Similar observations have been made elsewhere (IRHO, 1992b). Planting of this hybrid is clearly one method of combating the disease, and large parts of La Arenosa were replanted with hybrids (Turner, 1981). More recently, over 24,000 ha of hybrids have been planted in affected areas in Colombia, and there are also extensive plantings in Ecuador. Provided that assisted pollination is done, acceptable oil/bunch and good oil yields appear to be achievable, and it is clear that *E. oleifera* is a very useful source of resistance (see Section 6.5.1.6 for further discussion of hybrids). Even the hybrids may suffer at least mildly from the disease, though. Romero *et al*. (2013) recorded 28% infection in 10 hybrid crosses in the western region of Colombia, compared to 95% in 19 *E. guineensis* crosses. Gaitán and Romero (2012) showed variation among hybrid crosses in tolerance to inoculation with *T. paradoxa*.

Grogan and Mosquera (2015) investigated the optimum time for replanting with resistant material in relation to disease incidence and the cost of control measures. Their model indicates that, even though resistant material may be available, replanting costs mean that it will often not be economically optimal to adopt the new variety.

13.5.2.7 *Conclusion*

In Ecuador and parts of Colombia, this complex of symptoms is undoubtedly a serious problem, though in some areas, losses have been exacerbated by the measures taken to 'control' the disease. There is little evidence that eradicating infected palms is helpful, and improving agronomic management, particularly drainage and nutrition, should be the first step when the disease occurs. We previously considered that planting of interspecific hybrids was unlikely to be justified (Corley and Tinker, 2003), but good results have been obtained in recent hybrid plantings in areas where the disease has led to heavy losses of *E. guineensis*. In the long term, though, it appears likely that resistant *E. guineensis* material can be developed, which may be preferable to hybrids.

13.6 DISEASES OF THE BUNCHES AND FRUIT

The occasional bunch and fruit rots that are encountered have not been extensively studied. Bunch-end rot has been associated with the Deli palm, particularly in Malaysia (Thompson, 1934). Where neither lack of

pollen nor insect attack is implicated, both this condition and complete bunch failure have been attributed to 'overbearing' (Turner and Bull, 1967): the suggestion being that the number of bunches is more than the palm can support. As discussed in Section 5.4.6, however, the evidence for this is not convincing.

A bunch stalk rot has been connected with an unexplained condition in West Africa known as leaf base wilt (Bull, 1954). The leaves bend down towards the ground, and the stalks of bunches in the leaf axils also bend and may then begin to rot. The disorder seems to be of purely mechanical origin, and provided the rot is not so extensive that the bunch falls, the majority of fruit will develop. The small splits that appear in the stalk are invaded by a variety of saprobic bacteria and fungi.

Marasmius palmivorus is common as a saprotroph on the cut leaf bases and on the decaying debris between these and the trunk. It appears that, under moist conditions in the Far East, sufficient inoculum potential may sometimes build up for healthy bunches to be invaded (Turner, 1965c). For a full discussion of the factors involved in the spread of the disease, Turner (1981) should be consulted. The obvious means of control is to reduce, through sanitary measures, the medium on which the fungus grows on the palm. Rotting bunches should also be removed. Prophylactic spraying against *Marasmius* is not generally recommended, but Turner (1981) considered that on acid sulphate soils in Malaysia, spraying might be economically justified.

13.7 OTHER ABNORMAL CONDITIONS

The oil palm is subject to many abnormal conditions of growth and development, the causes of which are not known. Usually, although not always, these abnormalities are encountered where conditions are in some way adverse: impoverished sandy soils, long dry seasons, excessively wet conditions or intermittent waterlogging, grass competition, pockets of unusual soils, etc. In the more severe conditions, bunch yield is usually negligible (Courtois, 1968). Only a few are mentioned here.

The term plant failure was used for palms that almost ceased to grow. The rate of root and spear production, and the number of green leaves, decreases and the leaves that remain are erect and crowded. This, in turn, leads to a tapering of the trunk and progressive deterioration of the leaves, which become chlorotic, dry out prematurely and become brittle. There has been much speculation on the reasons for such palms being found dotted about among normal ones. The condition rarely occurs in Asia. In Africa, it is considered to be of genetic origin or may be associated with severe potassium and magnesium deficiency or where soil depth varies sharply from point to point.

A condition known as choke, or dwarfed crown, has been encountered in fields in America suffering from red ring disease (see Section 13.4.7), but does not appear to have the same cause (Malaguti, 1953). It has been referred to as 'hoja pequeña' (small leaf), but the term 'little leaf' should be reserved for the recovery symptom of spear rot–little leaf. In this condition, all the leaves are smaller than normal, green, erect, bunched together and twisted with varying amounts of atrophy or corrugation of the leaflets. A sudden recovery from the condition is frequent, a tall cluster of normal new leaves being produced in the centre of the deformed ones, giving the palm a two-tier appearance. This type of deformity is not unknown elsewhere, and the term 'choke' has been used in Malaysia to describe a similar condition.

The oil palm is occasionally killed by lightning strike. Young palms can collapse rapidly and wither, but a sublethal condition known previously as rachis internal browning is now also believed to be caused by lightning (Turner, 1981). In older palms, the trunk base is often charred. Lightning strike can usually be distinguished from other causes of death because surrounding palms show scorching of the leaves facing towards the strike.

Oil palms are quite tolerant of short-term flooding and have been successfully established in river floodplains. However, if young palms are flooded to a level above the leaf axils, so that silt is deposited in the axils, extensive inflorescence abortion, and sometimes death of the palm, may follow. The risk diminishes as the palms grow taller, but significant losses have sometimes occurred in young plantations (Teoh *et al.*, 2001). Siburat *et al.* (2003) recommended removing rotting spears and treating with a fungicide (Thiram) after floods receded, but provided no data on effectiveness.

13.8 PLANT QUARANTINE

The existence of serious diseases in some areas but not others justifies strict plant quarantine measures. Fungal spores are the most likely contaminant, but nematodes (unidentified but in this case apparently harmless) have been found on germinated seeds (Kushairi and

Rajanaidu, 2000). There are already examples of diseases spreading: as noted in Section 8.1.5, *Fusarium* wilt was spread from Africa to South America with oil palm seeds. The precautions adopted in Malaysia were described by Kang (1986), and included prohibitions on import of seed or pollen from areas where diseases of unknown aetiology occur, prohibition of import of secondary hosts or insect vectors of disease, and limits on the quantities of seed imported. Precautions must start in the country of origin, with inspection of parent palms for disease and thorough cleaning of materials. Where possible, seed and pollen should be screened for spores of important fungal diseases at an intermediate quarantine centre, between the country of origin and the importing country. Seed treatment to control disease is discussed in Section 8.1.5.