Crown Dieback in Eucalypt Forests

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1. INTRODUCTION

This review describes conditions of eucalypt crown dieback based on submissions from forestry personnel in Western Australia, South Australia, Victoria, Tasmania, New South Wales and the Australian Capital Territory.

The location and extent of the problem, history, symptoms and possible economic implications are examined and a definition of the problem is attempted.

2. **DEFINITION**

For the purpose of this review, crown dieback is defined as a conditions where the primary crown shows a disorder or has been replaced by a secondary crown and if the cause of the disorder persists, leads to tree death.1

To the forest manager, the significant feature of crown dieback is that it is a symptom of change in health or growth potential of the trees. It can be detected by ground inspection or from air photointerpretation and represents a disorder with a potential to adversely influence forest production, protection or amenity values. The impact of crown dieback is dependent on its extent in area and time, its total influence on forest values, and the nature of the agent responsible.2

3. RECORDED PROBLEMS

The disorders are grouped on a State basis according to history, symptoms, locations and extent.

3.1 Western Australia

3.1.1 Crown deterioration in jarrah—Prior to 1952 foresters expressed concern at progressive crown deterioration in cut-over, mature jarrah (Eucalyptus marginata Sm.) in the northern sector of its natural range (Wallace and Hatch, 1953 and Hatch, 1953; Fig. 1). In certain areas the dense primary crowns remaining after selection cutting, were observed to thin out and develop a secondary crown leaving much exposed dead wood. This crown deterioration following logging occurs among mature and overmature trees and is a permanent condition. The condition could be caused by excessive exposure, this fact has never been explained and has largely been ignored following the association of jarrah dieback with Phytophthora cinnamomi (Podger, et al. 1965). However some crown deterioration has developed in Western Australia in the absence of P. cinnamomi, and the degrade of crowns due to exposure following canopy disturbances (Hayes and Buell, 1955) is worth further investigation.

As jarrah is sensitive to waterlogging (Podger, 1967; Batini, 1968), environmental disturbance causing fluctuations in soil moisture and water table levels (Peck and Hurle, 1972) could induce crown deterioration. Jarrah also suffers severe crown thinning during heavy seeding, which occurs approximately every four years. Crown losses during seed production, in 1965-67, decreased basal area increment by more than 40 per cent. (Forests Department Western Australia, Annual

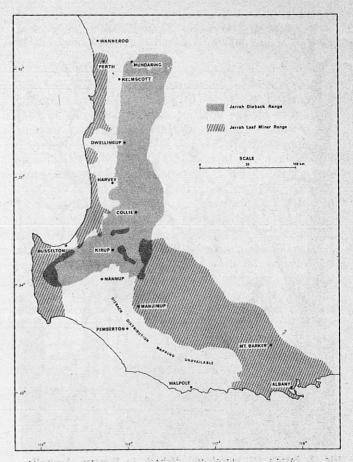


Fig. 1. Dieback areas in Western Australia.

Report, 1967). However the damage is temporary and occurs in all age classes.

3.1.2 Jarrah dieback—This condition refers to crown dieback and eventually mortality that occurs in virtually all situations within jarrah's natural range (Podger, 1972; Batini and Hopkins, 1972). Initial records of unexplained jarrah death in 1921 refer to small, discrete patches near Karragullen in the northern forest. Further small pockets of dead and dying jarrah were observed in 1928 some 80 km further south. By 1948 the disease had reached sufficient magnitude to be considered an important problem and resulted in a joint State-Commonwealth investigation.

The first symptoms of the disease (Figs. 2, 3) are usually yellowing and deaths of many understorey species and shrub layer plants, notably bull banksia (Banksia grandis Willd.) blackboys (Xanthorrhoea pressii Endl.) and Zamia palms (Maxrozamia reidlei C. A. Gardn.). The jarrah trees show symptoms at a later stage, often after all the understorey has died,3 and their foliage begins to thin out and branches die back. Damage becomes progressively more severe until the tree dies. Deaths in the jarrah overstorey appear to be random and sporadic. Some individuals or small

¹ Ed. note: For a more detailed definition see review by F. D. Podger.

² Ed. note: For detailed treatment of causes of dieback see review by F. D. Podger.

3 c.f. 3.2.2, Brisbane Ranges, Victoria.

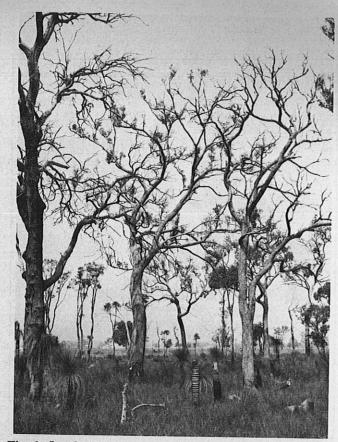


Fig. 2. Jarrah (E. marginata) dieback in Western Australia. Note the epicormic shoots on the dying trees. (Hopkins)



Fig. 3. A healthy jarrah (E. marginata) forest in Western Australia. (Hopkins)

groups may survive for many years and then succumb

quite rapidly.

Diseased areas usually expand slowly, though the rate of movement varies with the site, the forest type and the season. Some mature trees, marri (Eucalyptus calophylla R.Br.), blackbutt (Eucalyptus patens Benth.), bullich (Eucalyptus megacarpa F. Muell), wandoo (Eucalyptus wandoo Blakely) and karri (Eucalyptus diversicolor F. Muell) are tolerant to the disease.

Mapping of 0.7 million ha of the northern jarrah forest has revealed that 36,000 ha or 5 per cent of the area is affected by the disease (Batini and Hopkins, 1972). It is however, not possible to map dieback accurately from aerial photographs in southern forests. Allowing for dieback in southern areas and a measured rate of 4 per cent increase in the diseased area per annum (Batini, 1973), the current area infected is estimated to be about 80,000 ha.

The more severely affected areas are located in the northern and western margin of the jarrah forest concentrated in a belt from Mundaring to Collie parallel with the Darling Scarp (Fig. 1). In this area, up to 30 per cent of the forest is affected to some degree. Proceeding inland, the extent of infection gradually decreases to less than one per cent in the eastern section of the State Forest (Batini and Hopkins, 1972). It is obvious that disease development is affected by a large number of inter-related ecological factors.

It is closely associated with watercourses, and strong relationships exist between the disease and roads, soil type, cutting history and rainfall (Batini, 1973).

3.1.3 Jarrah leaf miner—The jarrah leaf miner (Perthida glyphopa Common) infests flooded gum (Eucalyptus rudis Endl.) and jarrah in the south-west of Western Australia (Wallace, 1970). Flooded gum is attacked by the leaf miner throughout the southwest, except on the Darling Range. The tree is confined to watercourses and other moist situations.

Newman and Clark (1926) first described outbreaks of leaf miner in jarrah around the ports and coastal townships and recorded an infestation near Perth as early as 1914. In the late 1950s outbreaks of the leaf miner were noted in jarrah forest east of Manjimup and these spread rapidly. In 1971 moderate to heavy infestation covered approximately 1.4 million ha of which approximately 0.4 million ha were in State forest, the remainder in partly-cleared farmland (Mazanec, pers. comm.). Surveys have revealed that the prime jarrah forest still remains relatively free from the disease, and that the major damage is associated with cleared farm areas (Wallace, 1970).

The moths emerge in April-May from the soil and deposit their eggs singly under the lower epidermis of the leaves. The larvae excavate blotch-shaped mines within the leaves and, on maturation in September-October, each larva constructs a cell. The cell is cut out leaving an oval hole in the leaf. The encased larva drops to the ground and aestivates in the soil until the following April. Up to 30 larvae may reach maturity in one jarrah leaf (Wallace, 1970).

Infested leaves become discoloured early in September and many of those in which the tissue has been destroyed are shed. The less damaged leaves are retained longer, but many drop off during January-March in the following year.

Up to 23 per cent of jarrah trees are resistant to leaf miner attack. The annual loss in girth increment has been assessed by Mazanec (pers. comm.) to be in the order of 70 per cent in the infected forest area and

40 per cent on the coastal plain.

It is suspected that parasitism and bird activity may control numbers of leaf miners in certain areas (Wallace, 1970). Controlled burning and tree density may also be relevant in the control of the disorder.

3.1.4. Karri dieback—To date there has been little cause for concern with karri crown dieback. Several years ago an isolated patch of virgin karri in Warren Block was killed, apparently as a result of association with Armillaria mellea.* No further occurances have been reported and although the fungus is present in the southern forest, inexplicable karri mortality is unusual.

3.1.5 Psyllid damage to flooded gum—In certain seasons extensive psyllid attack occurs on E. rudis. Attack can lead to complete defoliation and, in association with leaf miner activity, may result in tree mortality.

The damage appears to be identical to that reported for E. camaldulensis in South Australia and Victoria.

3.2 Victoria

3.2.1 Dieback in mixed hardwood forests of eastern Victoria—Lee (1962) described a rapid decline and dieback of patches of several eucalypt species in eastern Victoria. It appears that the disease may have originated in the late 1930s but was first recorded soon after the heavy rains of 1952—53 and increased further during the wet summer of 1956. Lee's inspections in 1958—59 showed that the largest patches did not exceed 2 ha and that a total area of approximately 25 ha was damaged. The disease was associated with road construction and heavy logging on sites with impeded drainage. During the past 13 years the disease expanded rapidly and has been associated with the presence of P. cinnamomi (Marks et al. 1972), (Fig. 4).

Observations made in 1971 based on ground survey and aerial reconnaissance show that the larger dieback patches exceed 40 ha in area. Patches of dieback ex-

^{*} Ed. note: Serious decline and dieback of eucalypt species have been recorded at Beaufort and Daylesford in Victoria. This disease is associated with Armillaria mellea and resembles regrowth dieback in Tasmania.

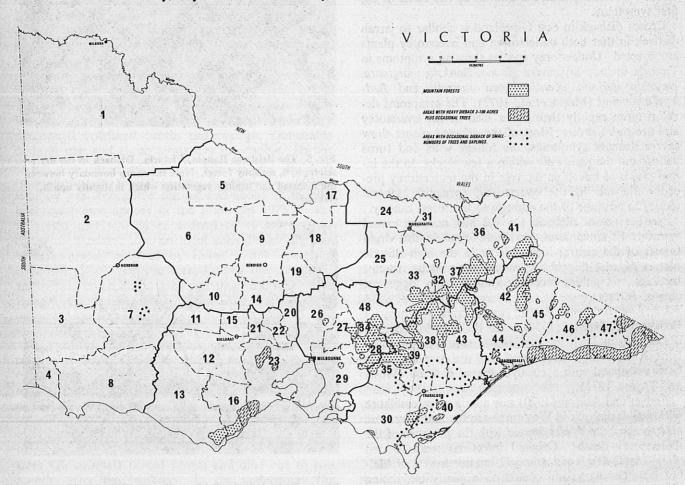


Fig. 4. Distribution of *Phytophthora cinnamomi* in Victoria. Most of the dieback is found in coastal forests on soils with impeded drainage that are heavily infected with *P. cinnamomi*.

tend from the far east coast of Victoria to Yarram and into the low foothills of the eastern highlands. A total of 4900 ha shows severe dieback, and large areas in eastern Victoria are lightly affected. All damaged areas are either close to roads or have been logged in the past. Identification of the disease is often complicated by past fire damage.

The disease appears to be worst on relatively infertile soils where drainage is impeded, although damage has been observed among old trees growing in well-drained soils subject to occasional periods of excessive moisture. After periods of heavy summer rain, severe and extensive epidemics of dieback occur (Marks et al. 1972).

All dominance classes of trees are affected by dieback. Macrantherous species show resistance while the renantherous species are susceptible. Lightly affected stands usually show poor crown development, with dieback in few of the major limbs. In severe cases a considerable part of the crown is lost and there are many dead limbs. On severely-affected sites most trees are killed. Natural regeneration in affected stands is stunted and spindly, with thin, chlorotic flat-topped crowns. When dieback assumes epiphytotic proportions the foliage of trees of all sizes suddenly loses its sheen and begins to wilt. The leaves turn yellow, then brown, and the tree dies within 2-3 months of the onset of the first symptoms.

Crown dieback in east Gippsland is similar to jarrah dieback in that both understorey and overstorey plants are affected. Understorey species showing symptoms in Victoria include Stypandra glauca, Banksia marginata, Daviesia latifolia, Xanthorrhoea australis and Bedfordia salicina (Marks et al., 1972). The symptoms develop more rapidly than those seen in the overstorey and are more severe. Most of the affected plants show severe drought symptoms, the foliage wilts and turns yellow and the plants die within a few weeks. In the initial stages of infection, damage in the understorey precedes dieback in the overstorey, spreading several metres in advance of the damage to the forest canopy.

Neither crown dieback (> 0.2 ha) nor the causal associate *P. cinnamomi*, has been found in the virgin forests of the central highlands or in the high-altitude forests of east Gippsland, except near roads, tractor landings and gullies receiving run-off from logged or cleared forests.

3.2.2 The Brisbane Ranges and Western Otways—Podger and Ashton (1970) were the first to record a disorder in understorey species and shrubs in the Brisbane Ranges in Victoria, and this disorder has since been associated with a eucalypt crown dieback (Weste and Taylor, 1971).

Podger and Ashton (1970) saw a striking resemblance with jarrah dieback in Western Australia and they were able to associate *P. cinnamomi* with the presence of the understorey deaths. Original infections were located along Marshall's Road, some 72 km north-west of Melbourne. The woodland occurs on a gently undulating plateau with a mean annual rainfall of 630 mm. Soils are lateritic, gravelly, grey and yellow sands overlying shallow, dense mottled clays. *E. obliqua*, *E. mac-*

rorhyncha and E. baxteri are the main overstorey species. There is a dense evergreen shrub cover dominated by Xanthorrhoea australis.

Podger and Ashton recorded four diseased areas, less than 0.5 ha in extent, to be of recent origin. Weste and Taylor (1971) and Weste et al. (1973) in detailed studies showed that the total affected area in 1970 was > 60 ha, consisting of small patches scattered over a wide area. All diseased sites showed appreciable increase in area over a five-month study interval (Weste and Taylor, 1971). In poorly-drained soils the spread of the disease symptoms was twice the lateral rate (10.4m) and three times the downhill rate (29.4m) of that in moderately drained soils (5.2m and 10.2m respectively). The maximum uphill spread was 1.5m on waterlogged sites. Lateral spread on well-drained sites was zero.



Fig. 5. The Brisbane Ranges, Victoria. Dieback in the dry sclerophyll, shrubby forest. Note the sharp boundary between the diseased and healthy vegetation which is slightly uphill. (Weste)



Fig. 6. Dieback in the Brisbane Ranges, Victoria. The eucalypts affected are *E. baxteri* and *E. macrorhyncha*. The shrubs are replaced by grasses. The tea trees remain alive but are chlorotic. (Weste)

Two years later Weste et al. (1973) found that the lateral rate of spread on these sites over a period of three years was about 171m per year. Extension of disease by run-off measured over the same period was > 400m per year. The disease always extended from an infected site. In the absence of P. cinnamomi no disease developed on disturbed sites with similar topography, soil and flora.

Visual symptoms appear first in the understorey and the grass tree (X. australis) is a useful indicator (Fig. 5). The leaves turn yellow (the outermost first), then a rich cinnamon brown, and finally the umbrella-shaped top collapses and the grass tree dies over a period of 2-5 months. Symptoms in eucalypts (Fig. 6) depend on the extent of droughting and fine root destruction. Initially, the crown loses foliage due to leaf abcission and this is followed by dieback of the branchlets. Epicormic buds may develop but then die with the tree. The process is gradual, occurring over a period of twelve months. When trees die rapidly their leaves lose their sheen and turn grey-green in colour. The veins project to form prominent ridges. The crown then dries out rapidly, the leaves becoming a pale-grey-green and brittle. Epicormic shoots may develop and finally the foliage turns brown and remains attached long after death. Trees usually die four weeks from the appearance of initial symptoms. 3.2.3 Wilson's Promontory—This valuable national park covering about 60 000 ha, rich in plant species, is located about 240 km south-east of Melbourne. Diseased vegetation was first reported from the northern slopes of the Vereker Range at Wilson's Promontory in September 1970 (Weste, 1972; Weste and Law, 1973). The pathogen P. cinnamomi was found associated with the disease in this region and has since invaded dry sclerophyll woodland, heaths and swamps. Dominants of both tree and shrub strata are susceptible. Shrubs of the understorey such as X. australis and Isopogon ceratophyllus are destroyed first, followed 6-12 months later by less susceptible shrubs, and about 12 months later symptoms appear in the trees. In 1973 it was estimated that 400 ha of a total of 60 000 ha was diseased. The area and extent of each disease site has been accurately mapped by Helen Veitch (personal communication). Disease spread along the contour is very much slower (18m per year) than in the Brisbane Ranges (171m per year). Spread by water run-off occurred at a similar rate i.e. > 400m per year (Weste et al. 1973).

The disease evidently originated from infected soil carried by tracked vehicles used in 1962 to assist fire control on the Vereker Range. The symptoms in both understorey and overstorey are similar to those described for the Brisbane Ranges. The dominant tree in the shrubby woodland, Banksia serrata, exhibits marked chlorosis. Death usually occurred within two months from the onset of of symptoms. Associated trees (E. obliqua) turned brown and died one to two months after the banksias. In the understorey the extreme susceptibility of the swamp-heath, Sprengelia incarnata was useful in delineating disease sites (Weste and Law, 1973).

3.3 Tasmania

3.3.1 High altitude dieback of Eucalyptus delegatensis R. T. Baker—This condition, studied in detail by Ellis (1964, 1972; Fig. 7), consists of a decline in vigour followed by mortality of mature, dominant *E. delegatensis* (Fig. 8).

Dieback can be seen in aerial photographs of the Camden Plateau taken in 1932. It has been recorded on the Surrey Hills, on Maggs Mountain and on the Camden Plateau where Ellis (1964) estimated that 1800 ha of 5000 ha of eucalypt forest was affected in 1962. The dieback is severe on this high plateau en-

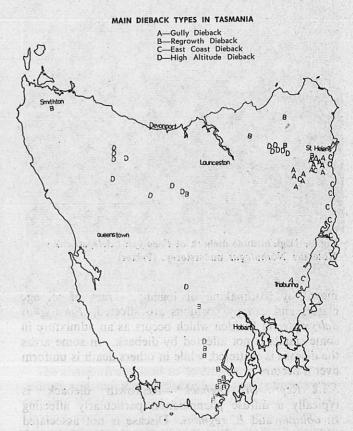


Fig. 7. Main dieback areas in Tasmania.

closed roughly within the triangle bounded by Trenah, Ben Nevis, and Mt Scott. It is restricted to altitudes above 760m and becomes severe above 900m. A general estimate of the total area affected by the disorder is 2500 ha (Felton, personal communication).

The forest trees do not show the symptoms where the understorey has been maintained in an open condition by repeated burning. Deaths occur in forests with dense understorey (Felton, 1972 (a) & (b). Ellis (1972) considers that this type of dieback, which is restricted to the dominant trees, is due to micro-climatic changes in the soil following the invasion of the stands by rain-forest understorey species.

Dieback symptoms begin with a general thinning of the foliage followed by death of the smaller branchlets and inhibition of new growth; leaves do not wilt or shrivel prior to falling. Epicormic shoots may develop but are not generally vigorous except after scrub re-

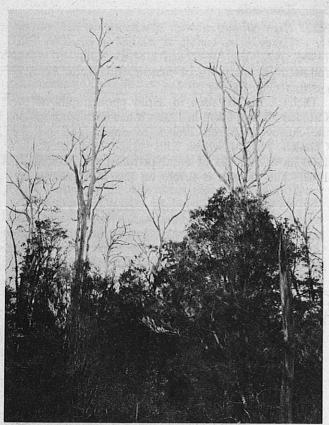


Fig. 8. High altitude dieback of Eucalyptus delegatensis. Note the Nothofagus understorey. (Palzer)

moval by roadmaking or logging. Trees of all age classes and canopy positions are affected. *Eucalyptus dalrympleana* Maiden which occurs as an admixture in some stands is not affected by dieback. In some areas the disease is scattered, while in others death is uniform over a hectare or so.

3.3.2 Regrowth Dieback*—Regrowth dieback is typically a diffuse phenomenon particularly affecting E. obliqua and E. regnans. Disease is not associated with a progressing front but rather distributed through the stand, and where more severely affected patches occur, boundaries are not discrete. The symptoms resemble those due to suppression though generally death appears to occur more rapidly. As the primary crown dies back it may be partially replaced by a secondary epicormic crown which may be retained for a variable period. No instances of complete crown recovery are known but some respite may be obtained when competing trees die. Growth is retarded before crown loss is noticed (Felton and Bird, 1972).

Dieback as such was first recognised in the south of Tasmania in 1964 (Bowling and McLeod, 1968) when the first unexplained deaths of co-dominant eucalypts were recorded on a permanent yield plot. An earlier record of declining trees dates from 1956 when descriptions were made of crown dieback symptoms in *E. obliqua* on two temporary assessment plots in a stand of regrowth which now has severe dieback. Between 1956 and 1964 regrowth dieback occurred in

the south at a much lower level, but since 1964 dieback has increased noticeably in amount and severity (Felton and Bird, 1972).

In the north-east of Tasmania regrowth dieback was first noticed in 1956 in *E. obliqua* and *E. regnans* after a very wet summer. Some of these original patches have since increased slightly in size. The same is true for a patch of about 40 ha in the north-west, first noticed in 1945. By 1947 many trees were dead, but the patch did not enlarge and in 1970 still contained live trees which had epicormic crowns (Felton, 1972 (a) & (b)).

Most regrowth dieback studies have been undertaken in southern Tasmania where appreciable areas of regrowth resulted from wildfires associated with the commencement of logging at the end of the last century. The important eucalypts in regrowth stands are E. obliqua, E. regnans and E. globulus. E. globulus appears to be relatively tolerant to dieback compared with E. obliqua or E. regnans, but dead trees of this species can be found. There is no obvious association between the occurrence of regrowth dieback and factors such as soil type, topography or forest age. The understorey below the eucalypts is dense, usually the wet sclerophyll type tending towards rain forest types in the moister habitats. With the exception of very young roadside regeneration or that in burnt areas, deaths of understorey species have not been observed (Felton, 1972 (a) & (b)).

South of Hobart, dieback affects about 16 000 ha of regrowth bounded on the south by the Catamaran River and on the north by the middle reaches of the Huon River. Except for a few patches in the lower catchment of the Huon, these regrowth forests are generally healthy as are those on Bruny Island, Tasman Peninsula and in the Florentine Valley.

3.3.1 Gully dieback—Gully dieback was recognised as a problem in 1969 following a major upsurge of dead and dying forest in eastern Tasmania. This upsurge coincided with high populations of leaf-eating larvae of the seedling gum moth (*Uraba* sp.), and with a severe drought. Both the gum moth and the drought must have had some effect on the disease, although other agencies were involved. Subsequent deaths have continued since 1969 when rainfall has been high and gum moth populations low (Felton, 1972 (a) & (b)).

Gully dieback occurs where annual rainfall is less than 1000mm and summers are drier than those within the regrowth and high altitudinal dieback areas. The area affected extends from Buckland in the south, to west of St. Helens in the north. Gully dieback occurs infrequently in the southern part of this area, the Eastern Tiers; the area most severely affected is on the lower eastern slopes of the north-eastern Highlands. Here the predominant vegetation type is dry sclerophyll forest, tending to wet scleropyll in most gullies. The forest consists of well defined ridges and gullies, Eucalyptus sieberi L. Johnson is characteristic of the drier ridge tops and upper north-facing slopes and

Similar areas have been discovered in Victoria at Beaufort and Daylesford.



Fig. 9. Typical gully dieback, Tasmania. (Palzer)

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Eucalyptus amygdalina Labill and E. obliqua of gully bottoms and south-facing slopes, the latter being the only renantherous species found in gullies. E. viminalis and E. globulus are scattered through the moister situations. Soil drainage is good in all topographic positions (Felton, 1972 (a) & (b)).

Of 60 000 ha assessed to date by aerial photographic interpretation (Fig. 10), approximately 2400 ha were found to be affected by dieback—most of this occurring within the western section of the Upper Scamander (Felton, 1972 (a) & (b)). Palzer (1973) reports that a total of 2800 ha is affected by gully dieback. Comparisons of photographs taken in 1950 and 1969 clearly show that both the area affected by the disease, and the disease intensity increased substantially in this period (Felton, 1972 (a) & (b)).

Recognition of early symptoms of gully dieback is made difficult by crown defects due to periodic fires. Where the disease is well developed all older trees die leaving a few acacias. Where disease is less developed, trees with dead or dying primary crowns but apparently healthy epicormic shoots are common. Rapid deaths are more frequent than with regrowth dieback.

The change from dead to healthy trees is usually well defined and, on the north-facing slopes, often corresponds to the sharp change from E. obliqua to E. sieberi. Healthy eucalypts occur upslope. E. amygdalina, E. delegatensis and E. obliqua are killed by gully dieback, E. viminalis shows some tolerance, but E. ovata shows great tolerance, living in gullies where other eucalypts are dead. E. sieberi rarely occurs in gullies but where it does it may be killed.

Wet sclerophyll understorey is unaffected. In the majority of gullies there are no appreciable deaths of understorey plants in the families *Papilionaceae* and *Epacridaceae*. It is only in the presence of *P. cinnamomi* in a few gullies close to roads, that patchy deaths in the understorey have been observed (Palzer, personal communication).

Healthy eucalypt regeneration, about four-years-old, may be found below the dead trees in gullies where the understorey is sparse. Seedling deaths occurred in autumn 1971 in an artificial regeneration trial established in 1969. P. cinnamomi was recovered from these dead seedlings (Felton, 1972 (a) & (b)), but the association of P. cinnamomi with gully dieback is not clear (Palzer, personal communication).

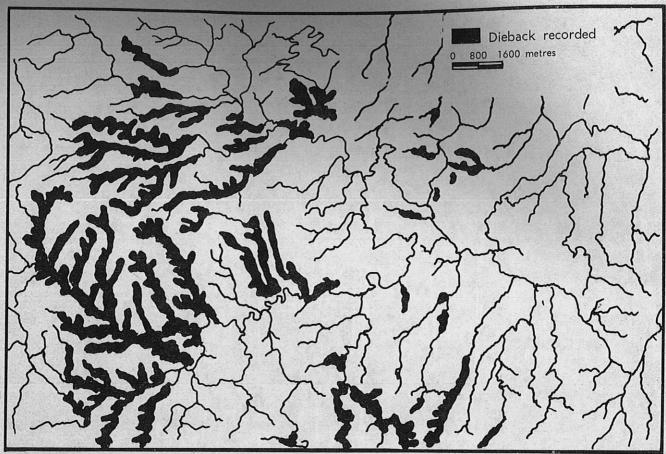


Fig. 10. Map of the upper Scamander forests affected by gully dieback. This is an actual plot of the distribution of the disease reproduced from a colour photograph and it illustrates the general way the disease spreads along gullies. (Felton)

3.3.4 East coast dieback—Along the east coast, especially around Scamander and Coles Bay, there is an extensive decline in E. sieberi and E. amygdalina and many understorey plants susceptible to P. cinnamomi (Fig. 11). There are also some poorly-drained river flats and plateaux where E. amygdalina and sometimes E. obliqua and understorey plants are declining.

3.3.5 Insect defoliation—Greaves (1966) reported sporadic defoliaiton of 2-8m high regrowth eucalypts in central Tasmania. The damage, principally to young regrowth of *E. regnans* and *E. viminalis*, is caused by a leaf beetle *Chrysophtharta bimaculata*. *E. delegatensis* is also attacked while *E. obliqua* and *E. simmondsii* appear to be resistant under natural conditions.

Outbreaks could be associated with above-average temperatures during December and January and the availability of host eucalypts within a critical height range. Kile (1973 (b)) reports that this insect destroyed more than 40 per cent of the foliage of 60-year-old E. obliqua and E. regnans in some areas during the 1972-73 growing season. He suggests that such damage may influence the growth and health of large trees.

3.4 New South Wales and Australian Capital Territory

Extensive defoliation of alpine ash by phasmatids is the greatest crown dieback problem in New South Wales. Crown dieback is common in woodland formations but is relatively rare in coastal scleropyll formations (Fig. 12).

The Eden district is regarded as an important area because of its close proximity to dieback areas in Victoria. Eucalypt crowns have been extensively firedamaged but there is no sign of deterioration of the understorey. Occasional scattered deaths in seedling regeneration have been associated with the presence of *P. cinnamomi* which is suspected to be native over most of the area (Forestry Commission, New South Wales, 1973, Anon. rept.).

In New South Wales Pratt and Heather (1973) recovered *P. cinnamomi* from eucalypt forest, with disease symptoms in the overstorey, at Green Cape, Eden, Bega, east Batemans Bay, Penrose, Ourimbah, Coffs Harbour and Orara. These workers consider *P. cinnamomi* to be widespread in eastern Australian forests and a common component of the soil microflora.

3.4.2 Newcastle district—At Ourimbah, some 80 km north of Sydney, severe crown dieback has occurred in mixed eucalypt forest. This dieback appeared to be due chiefly to psyllid attack (Moore, 1962). P. cinnamomi and other fungi have only been found in some places in association with the dying trees. Similar psyllid-induced deaths have been recorded in the Glen



Fig. 11. Typical east coast dieback in Tasmania associated with infection by P. cinnamomi. (Palzer)

Innes, Wauchope, Coffs Harbour and Taree districts.

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Hartigan (1969) reported that the dieback described by Moore in the Wyong forestry sub-district, developed insidiously, the symptoms being partially masked by seasonal variations in forest cover. Species involved were E. saligna, E. deanei, E. microcorys, E. propinqua, E. paniculata and Syncarpia laurifolia. It was possible in 1969 to detect the disorder with a fair degree of accuracy and the disease spread about 6 km in 10 years. The affected area covers about 30 km² between Wyong and Gosford. These forests have been exploited heavily for timber, fragmented by land development and small farms and damaged by fire. There has been no spread of the disorder to the better areas of Ourimbah State Forest. The disorder appears in patches of approximately 100 ha originating from about half-way up a hill to the ridge itself and then spreading along the ridge. It is assumed this pattern is due to distribution of susceptible species which in turn reflect soil conditions. It would appear that the cause of the disorder is complex, psyllids being primarily responsible for the damage.*

3.4.3 Other areas—All areas in which P. cinnamomi has been located (Pratt and Heather, 1973) are being

investigated by the New South Wales Forestry Commission. At present very little serious damage has occurred, crown dieback of eucalypts in New South Wales is regarded however, as the most important single threat to the forest estate.

3.4.4 Australian Capital Territory—Some form of dieback is found throughout the wet and dry sclerophyll eucalypt forests of the A.C.T. These are most likely to be caused by drought, insects, fire and wood rottingfungi (McArthur, personal communication).

Jehne (1970) sampled areas of disordered trees throughout the A.C.T. In general, the selection of a sampling site was guided by visible symptoms of dieback. Although these symptoms were mainly confined to individual trees, no case of complete death of vegetation on any appreciable area was encountered. *P. cinnamomi* was found in half the eucalypt sites with dieback symptoms. Most dieback patches were found on soils waterlogged after high rainfall.

Jehne (1972) described death and dieback among scattered trees and in patches of mature *E. dives* in dry sclerophyll forest at Blue Range. *Eucalyptus dalyrm*-

^{*} Ed. note: See Gerettson-Cornell, 1973.



pleana was occasionally affected. The dieback patches, up to one hectare in extent, were on a steep, rocky slope with an easterly aspect at an elevation of 100m. Most of the crown dieback appeared to be less than 2 years old although isolated dead trees showed that there had been earlier deaths. He suggested that the disorder must be largely due to a high level of root parasitism by Exocarpus cupressiformis.

Numerous deaths of recently-established plants in the Botanical Gardens on Black Mountain have been attributed to *P. cinnamomi* activity by Pratt and coworkers.

3.4.5 Phasmatid defoliation of alpine ash-Since the early 1950s a phasmatid, Didymuria violescens Leach, has become a principle defoliator of eucalypts in the mountain forests of south-eastern Australia (Readshaw, 1965). The first outbreak was noted in Bago State Forest in 1952—53 and subsequently other outbreaks moved progressively southwards with each successive generation of D. violescens. Initially its favoured habitat appeared to be the E. delegatensis-E. dalrympleana association but later alpine ash (E. delegatensis) and mountain ash (E. regnans) associations were also heavily infested. The least favoured habitats were dry sclerophyll forests and snow gum communities. Approximately 1700 km² of forest were defoliated in 1962-63 and further defoliation occurred in 1964-65 (Mazanec, 1966). The most characteristic feature of the phasmatid outbreaks is the occurrence of synchronised defoliation during odd calendar years (1955, 1957, 1959, etc.) with no defoliation in the even years (Readshaw, 1965). This corresponds with the two-year life cycle of the insect.

The effect of defoliation on the growth and survival of the mountain eucalypts varies considerably according to the species of the host. E. radiata Sieb, E. dives Schauer, E. dalrympleana, E. viminalis and E. maculosa R. T. Baker survive repeated defoliation mainly by producing large numbers of epicormic shoots. Eucalyptus delegatensis and E. regnans are more sensi-

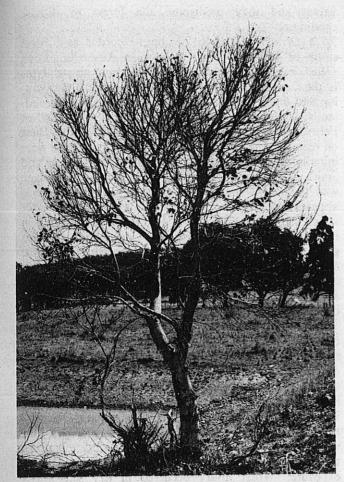
tive to damage and both species have suffered considerable mortality after only one complete defoliation. Shepherd (1957) studied the effects of severe defoliation of alpine ash at Bago State Forest and showed that serious mortality and reduction in diameter growth occurred. Mortality of mountain ash defoliated by phasmatids during 1960—61 and again in 1962—63 reached 83 per cent within two years of the second defoliation (Mazanec, 1967). The effect of successive defoliation on current annual radial increment ranged from an estimated reduction of one per cent in 1951 to 56 per cent in 1962 and averaged 20 per cent over the outbreak period of 16 years (Readshaw and Mazanec, 1969).

Crown dieback of eucalypts in woodlands and plantations—Biennial surveys of insects infesting eucalypt trees bordering a 100 km route extending from Canberra to Euroa in Victoria over an 8 year period (Carne, 1965), showed that most dieback appears to have resulted from insect attack.

(i) Saw-flies (Perga spp.) are among the most important defoliators of woodland eucalypts (Figs 13, 14), and are often present in sufficient numbers to destroy the entire crown. The hosts include E. melliodora A. Cunn., E. blakelyi Maiden and E. camaldulensis Dehn. Perga affinis is one of several species likely to prevent either



Fig. 13. Colony of Perga affinis on E. blakelyi. (Carne)



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Fig. 14. Defoliation of Eucalyptus blakelyi by the saw-fly Perga affinis, near Duntroon (A.C.T.). A colony is visible on branch at left. (Carne)

growth or successful establishment of plantations of these and several other eucalypt species. Related saw-flies are responsible for severe and repeated damage to eucalypts dominant in alpine or sub-alpine catchment areas.

The principal area occupied by P. affinis lies between the 540 mm and 760 mm isohyets, delineated by a 50—100 km-wide corridor from Seymour in Victoria to Coolac in New South Wales. To the east of Coolac, the insect becomes progressively less abundant and has not been found east of Gunning. The wetter and colder limit is defined with remarkable precision by the Hume Highway from Seymour to Canberra (Carne, 1962).

The saw-fly is an inhabitant of river valley woodland, rarely abundant in other situations and absent from sclerophyll forest formations. The susceptibility of trees to infestation is influenced by seasonal production of new foliage. Those growing in sites where the water table is high, and whose leaf production is to a large extent independent of rainfall patterns, may be subject to chronic attack (Carne, 1965).

(ii) Christmas beetles (Anoplognathus montanus)

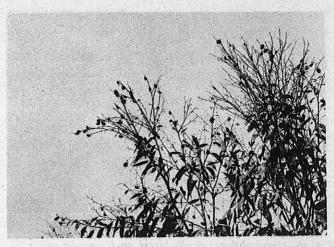


Fig. 15. Anoplognathus hirsutus and A. saturalis defoliating Eucalyptus pauciflora at Braidwood, N.S.W. (Carne)

seriously damage E. albens in the Cootamundra district. Extensive dieback and mortality followed a series of seasons with very high beetle populations. This insect also causes extensive dieback of roadside or ornamental plantings of E. rubida, E. lindleyana (syn. E. elata) and other species in the Australian Capital Territory (Carne, personal communication, Fig. 15). The species Anoplognathus porosus and A. chlorophyrus defoliate plantations of E. grandis Hill in the Coffs Harbour region (Carne, et al., 1973). Severe defoliation is limited to young trees planted on old grasslands. Leaf beetles. (Paropsis and Chrysophtharta spp.), a sap-sucking bug (Eurymela sp.) and psyllids also attack these plantations.

- (iii) Psyllid damage has caused dieback of E. blakelyi, E. camaldulensis and E. melliodora in many parts of southern New South Wales and northern Victoria (Carne, personal communication).
- (iv) Paropsine beetles and their larvae are prominent among the insects that defoliate woodland eucalypts in south-eastern Australia. They are active from late spring until early winter and can cause dieback or even death (Carne, 1966). One of the most abundant paropsines is Paropsis atomaria. In the Australian Capital Territory the eucalypts most commonly attacked are E. blakelyi, E. melliodora, E. polyanthemos Schau and E. fastigata F. Muell. In the A.C.T. and New South Wales P. atomaria was abundant only on young trees or on coppice growth, both in savannah woodland and in disturbed sclerophyll forest. Mature trees never supported sufficient numbers of the insect to cause appreciable defoliation (Carne, 1966). However, in extensively-cleared areas in Victoria, occasionally severe defoliation of healthy shelter belts of E. cladocalyx occurs (C. J. Irvine, personal communication).

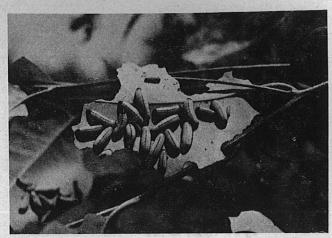


Fig. 16. Chrysophtharta cloelia larvae on E. grandis near Bonville, N.S.W. (Carne)

3.5 Queensland

There are few reports of eucalypt dieback in Queensland. Of particular interest in recent years is a wide-spread decline and death of *E. drepanophylla* (grey ironbark) on gully and ridge-top sites around Brisbane, Nambour, Gympie and Jimna. Death appears to be confined mainly to trees of the larger girth classes.

In the Mackay region death of patches of *E. tereti-cornis*, *E. intermedia*, *E. creba*, *Tristania saureolens*, and *Glochidion ferdinandi*, up to 2 ha in extent, have been reported. *E. carnea* has shown the greatest resistance to the disorder. No pathogens have yet been recovered from these areas. Features of these deaths are that there are proportionately more dead trees in the gullies than on ridges and that most of the deaths appeared to have occurred over a relatively short period of time.

In general, stag-headed eucalypts are evident in many commercial forests and causes of the disorder are unknown. Though growth increments may be affected, the disorder has no effect on tree selection in forest management (Queensland Department of Forestry, 1973).

3.6 South Australia and Northern Territory

No report was received concerning eucalypt crown dieback from these areas, with the exception of sporadic defoliation of *E. camaldulensis* in South Australia by psyllids.

3.7 Miscellaneous

3.7.1 Fire—Under present management methods it is usual to omit reference to the serious effect that uncontrolled fire has on promoting crown dieback in eucalypt forest. For the purpose of this seminar such an omission is unacceptable. In Western Australia at least, uncontrolled fires have devastated several national parks and water catchments in dry sclerophyll forest within the past few years. Mortality and crown dieback resulting from the relatively recent Dwellingup, Dandenong, Hobart and Blue Mountain fires represent greater changes in forest values than can be accredited to the numerous instances of dieback already noted. Fires represent a direct loss in wood increment and aesthetic

values and may predispose the forest to disease conditions.

3.7.2 Drought—Drought has been recorded as a direct cause of eucalypt crown dieback in Australia. Pook (1967) reported effects of the 1965 drought on eucalypts in the Canberra district that produced widespread wilting and death of eucalypts in native forests and woodlands. Water stress first became apparent at the end of February, 1965, the first quarter of 1965 being the driest on record in the Canberra area. Wilting symptoms were initially obvious in shallow soils on E. rossii Baker and Smith and E. macrorhyncha F. Muell where drainage was poor. By the end of March water stress was apparent over a wide area. The first symptoms of water stress in mature trees was a uniformly dull colour in the foliage. With increasing desiccation leaves gradually changed colour, became yellowish or chlorotic and in extreme cases turned brown and brittle as they died. Two and three year-old leaves were shed during a period of intense leaf-fall in the early stages of the drought.

On many sites, there was at first a tendency for the more exposed foliage to desiccate more rapidly than those protected by the lower levels of the tree canopy or on the cooler aspects of the tree crown. This meant that there was often a superficial 'scorching' of crowns in forest stands having a more or less continuous canopy. Lateral 'scorching' was seen on the exposed sides of trees growing on westerly and northerly slopes and where desiccation was intensified by dry winds. Damage was aggravated by stony ground, shallow soils and exposure. Worst injury occurred in dry sclerophyll forests and this extended occasionally into adjacent savannah woodlands.

Desiccation and shrinkage of the wood caused the bark to split and shed to varying degrees depending on the dryness of the habitat, tree age and species. Progressive dehydration ultimately led to exposure and destruction of the cambium of drought affected trees. This type of injury was frequently limited to the exposed sides of tree trunks and main branches. Drought injury assisted invasion by bark beetles.

Eucalyptus rossii was the most drought-sensitive of the species studied. Stringybarks usually sustained severe wilting with less obvious bark injury and low mortality. Red box (E. polyanthemos Schau.) was rarely affected visibly and suffered least damage. Other minor species which wilted or died in some stands include E. goniocalyx, E. dives, and E. bridgesiana. In some high mountain forests E. pauciflora, E. stallulata and E. delegatensis suffered some injury. Injury and death was observed in all size-classes of drought-susceptible species.

Most stands recovered rapidly after substantial rainfall in July and August 1965. In the following spring, shoots developed from epicormic buds on all but the most seriously decorticated trees. During a dry spell in January 1966 this epicormic growth wilted on several damaged trees in areas where mortality had been high. Some of these trees finally died (Pook, 1967).

A series of drought years between 1968 and 1973 in

the south-west of Western Australia also caused dieback of some eucalypts in dry sclerophyll forest. This coincided with a record drought in the eastern goldfield woodlands in which many unique species suffered extreme damage and mortality (Forests Department, Western Australia).

4. DISCUSSION

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Only a few reports provided useful observations on possible agents causing dieback. However, it is obvious that the economic implications of eucalypt crown dieback are considerable.

To summarise, it may be possible to associate dieback with the following conditions:

- 4.1 Known soil pathogen*—Jarrah dieback (Podger, 1972), the Victorian diebacks (Mark et al., 1972 and 1973; Weste and Taylor, 1971; Weste and Law, 1973), and Tasmanian east coast dieback (Palzer, 1973). The epidemiology of these diseases is not completely understood so that methods of controlling them have not been worked out. P. cinnamomi is, however, suspected in some cases and has proved to be causal in others.
- 4.2 Insect damage—Dieback associated with jarrah leaf miner (Wallace, 1970), phasmatid defoliation (Readshaw and Mazanec, 1969) and the various seedling and woodland defoliations (Greaves, 1966; Kile 1973b; Carne 1962; 1965; 1966; Carne et al., 1973) have identified causal agencies and, in some instances, acceptable partial control techniques have been developed.
- 4.3 Insufficiently studied diseases—Under this vague heading I have grouped jarrah crown deterioration (Wallace and Hatch, 1953), high altitude dieback of E. delegatensis (Ellis, 1964), gully dieback (Felton, 1972), Ourimbah dieback (Hartigan, 1969) and regrowth dieback (Felton, 1972 (a) & (b)). At present the factors causing these diebacks are only partly understood. It is suggested that the potential threat posed by these unknown disorders requires thorough investigation. The existence of these disturbances, possibly associated with man's activities, makes it imperative that there is improvement in the present ecological information if future forestry in Australia is to be soundly based.

The validity of the generalisation that many diebacks are due to physical intervention by man was questioned by one of the delegates. Many recent occurrences of Jarrah, gully, regrowth and high altitude dieback have been recorded in areas undisturbed by man for many decades. In other places vigorous and healthy regeneration grows on sites which have experienced severe physical disturbance in the past. In the latter areas causal agents of dieback must have been absent or remained under very powerful environmental control. Undoubtedly there are areas on which physical disturbance is likely to accelerate the activity of pathogens such as P. cinnamomi. In such places forest managers must exercise considerable restraint, but extending this restraint to areas in which such pathogens do not occur or are controlled by environmental conditions is unwarranted and will unnecessarily reduce the choice of silvicultural options available to forest managers.

4.4 Fire and Drought-Damage by these two agents is common in all eucalypt forests. The delicate balance that exists between natural fires and the distribution and vigour of a tree is illustrated by high altitude dieback (Ellis, 1972). This clearly shows the need for a better knowledge of fire ecology. Too little or too frequent burning may jeopardise the balance of the eco-system. The effect drought has on disease expression, especially in the case of trees injured by P. cinnamomi and its role in gully dieback (Felton, 1972 (a) & (b)) is poorly understood. The influences of major drought on eucalypts (Pook, 1967) have not been adequately studied.

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^{*} Pratt & Heather (1973) stated that some oomycetous fungi found in eucalypt forests may be potential pathogens.

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^{**} Reports marked in this manner are internal departmental reports used in preparation of this review.

THE LABBARY
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Eucalypt Dieback in Australia

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