# A review of the early years of jarrah dieback research in Western Australia

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# Introduction

From 12-16 November 1973 representatives of Australian forestry departments, leading researchers with an interest in eucalypt health, together with timber producers, attended a seminar at Lakes Entrance, Victoria, to discuss eucalypt dieback in Australia (Marks and Idczak 1973). The main focus of this seminar was the introduced soil-borne pathogen *Phytophthora cinnamomi* which was believed to cause the mortality of jarrah (*Eucalyptus marginata*) in the south west of Western Australia, and the decline and death of several eucalypt species in Victoria. There were presentations on detection, economic impact, management and control. The seminar ended with a set of recommendations to be put to the Pests and Diseases Sub-Committee of the Standing Committee of the Australian Forestry Council. Summaries of the discussions that followed the presentations are included in the proceedings, and nowhere in these discussions is there any doubt that *P. cinnamomi* was the cause of these problems.

The conclusion that *P. cinnamomi* was a eucalypt killer was based on the work of Frank Podger in Western Australia which was accepted in good faith by foresters at that time(Podger 1968; 1972). However, what was not realised was that Podger's investigations did not support his conclusion that *P. cinnamomi* killed jarrah trees, although they did support its role in the death of some mid- and under-storey plants (Davison 2014; 2015). In this paper I review the early days of research into jarrah dieback, based largely on unpublished documents held in the library of the Western Australian Forests Department (now the library of the Department of Parks and Wildlife) and at the former CSIRO Forest Research Station, Kelmscott, Western Australia. These documents were first viewed in 1980, and some may no longer be in existence. A somewhat similar review has already been published (Davison 2015).

# Early commercial exploitation of jarrah

Jarrah (*E. marginata*) originally covered an area of about 39 000 km<sup>2</sup> in the south west of Western Australia (Abbott and Loneragan 1986). The area has a Mediterranean climate with cool wet winters and hot dry summers; about 90% of the rainfall occurring between April and October. On the best sites, which have deep, well-drained lateritic gravel soils, jarrah is dominant, reaching a height of at least 27 metres. Jarrah grows in association with marri (*Corymbia calophylla*) and other eucalypts including *E. patens* and *E. megacarpa* which replace it on wetter sites.

Annexation of what is now Western Australia by Britain in 1829 was triggered by reports of immense forests of large trees in the south west. The Admiralty needed to re-build its Navy after the Napoleonic Wars, and jarrah (also known as Swan River mahogany) appeared to be a suitable

substitute for oak (Lane Poole 1920). Large volumes of jarrah timber were exported from Western Australia during the 19<sup>th</sup> and 20<sup>th</sup> centuries, and the timber industry was extremely important for the economic development of the state (Figure 1).



Figure 1: Jarrah log landing, undated. *Source:* ©Western Australian Department of Parks and Wildlife, image 971429, serial no. F.D. 5188.

The timber industry was not regulated before the formation of the Western Australian Forests Department in 1918. Timber millers had the right to remove and export timber from concessions and leases granted by the state government for a nominal rent (Lane Poole 1920). By 1920 it was estimated that almost 1 million acres [4 600 km<sup>2</sup>] had been logged, mainly in the high quality stands of jarrah in the northern jarrah forest, more that 750 million cubic feet [21 million m<sup>3</sup>] had been removed, and the forest canopy had been reduced by almost 50% (Wallace 1966). These changes resulted in rising superficial water tables with free water being present in winter in areas that had previously been dry. Jarrah crowns deteriorated becoming 'a gaunt framework of limbs which were dead some 20 feet [6 m] from their extremities' (Stoate and Bednall n.d.). This crown deterioration was believed to be the result of the rising water tables, greater exposure of the crowns, and severe fire damage fuelled by logging debris (Stoate and Bednall n.d.; Wallace and Hatch 1953; Wallace 1966) (Figure 2). The newly formed Western Australian Forests Department attempted to reverse these effects through developing working plans, tree marking and fire management (Williamson et al. 2005).



**Figure 2:** Plate 4 from Podger 1959b. The caption reads: 'Banksiadale Compartment 6. Stagheaded condition in a jarrah tree ... The crown is almost entirely composed of secondary epicormics growth. This is typical of the nature of veteran and tree crowns in many places. Where this condition is not associated with dieback it would be described as "crown deterioration" by Western Australian foresters.'

## Jarrah dieback

## **Early investigations**

During the 1940s there was a new problem that concerned the Forests Department. This was groups of dying jarrah trees, a problem that became known as jarrah dieback, with the sites where they occurred labelled as dieback sites (Figure 3). Groups of dying jarrah trees had been first reported in 1921, but by the mid-1940s they were much more numerous (Wallace and Hatch 1953). It was uncertain whether this was a further development of crown deterioration or something quite different. Although individual groups of dying trees were small, the patches were numerous, and the problem was seen as being of considerable economic importance for maintaining the supply of jarrah timber for export. At this time, just after World War II, money and manpower would have been in short supply, but the Forestry and Timber Bureau in Canberra, and the Forests Department in Western Australia considered that these deaths were of such importance that they established a joint research programme at Dwellingup to determine their cause (Forests Department 1947).



Figure 3: Dieback area, 1968. *Source:* ©Western Australian Department of Parks and Wildlife, image 970819, serial no. F.D 5374.

The first investigations were conducted by Charlie Hamilton between November 1947 and July 1948 (Hamilton 1951). He conducted a survey which showed that the patches of dying jarrah trees occurred on previously logged sites in the northern jarrah forest. They were in specific topographical positions: on saddles, in gully heads and along the upper sections of drainage channels. They were in poorer quality forest, on black, heavy lateritic gravels and yellow brown gravels below lateritic caps. The deaths occurred rapidly, with all size classes and ages affected, with the trees appearing to have run out of water. The only large tree that died was jarrah; marri was unaffected and appeared to colonise areas where jarrah deaths had occurred. Other plants also died, including the mid-storey tree *Banksia grandis*, and affected sites showed significant floristic change. Hamilton also established a transect at Teesdale for future intensive monitoring and mentions that there was free water on the soil surface of this transect during winter. Hamilton's notes, field books and maps were left at Dwellingup after his departure; they appear to have been destroyed when the records store was burned down in the 1961 Dwellingup fire (J. B. Sclater, pers. comm. 7 April 2015).

Hugh Waring (1950) confirmed Hamilton's observations and commented on the death of *Allocasuarina fraseriana* in addition to *B. grandis*; he suggested that the deaths might be the result of toxic soil conditions. Further observations were made by Owen Loneragan (1961) who suggested that waterlogging might be important because of the association of deaths with exposed ridges and flooded gullies.

Alongside these field observations there were also several investigations into possible causes of these jarrah deaths. The first was by John Harding (1949a) who examined trees from foliage to roots to determine whether pests or pathogens were involved. He found no evidence that this was the case; the only symptom was that there were more tyloses and gummosis in the sapwood of roots

from affected trees compared with unaffected ones (Harding 1949b). Tyloses are ingrowths into the xylem vessels of the sapwood which seal off these capillaries so that they are no longer able to conduct water. Harding was unable to interpret these symptoms; however it is now known that they form as a result of waterlogging damage (Davison and Tay 1985; Davison 1997), although there may be other causes.

There was also a very large survey comparing the physical and chemical properties of soils from affected and unaffected areas to see whether the deaths might be associated with a nutritional deficiency or toxicity, but no substantial differences were found (Wallace and Hatch 1953). By the early 1950s the investigations into a possible cause had stalled, and forest research at Dwellingup took other directions.

Further work on jarrah dieback was initiated in 1959 by the Forestry and Timber Bureau who appointed Frank Podger, a forester in the Western Australian Forests Department, as a research officer based at the Dwellingup Research Station. In order to assist Podger, the Forestry and Timber Bureau also sent their forest pathologist Bill Stahl, and forest entomologist Ron Greaves to Western Australia to again check whether pests and/or pathogens were involved (Podger 1959a). The three of them inspected several affected stands examining the foliage, stems and roots of jarrah as well as *B. grandis, Macrozamia reidlei* and *Persoonia longifolia* (Stahl and Greaves 1959). No consistent fungal lesions were found and there was no sign that the deaths resulted from insect damage. The only unusual symptom was the presence of excessive numbers of tyloses in the jarrah sapwood, similar to Harding's observation (Harding 1949b) and a thin sheet of kino just below the cambium in jarrah stems. They concluded that, 'with the possible exception of a virus, there is no evidence that a pathogen is the sole cause of the disorder'.

Podger started his work by reviewing the possible causes of jarrah dieback (Podger 1959b). He confirmed earlier observations of the association of jarrah deaths with previously logged sites although there were some occurrences in unlogged forest (Figure 4) and concluded that the most likely cause was environmental change resulting from previous heavy cutting, the exclusion of fire from some areas, or the increase in the incidence of severe fires. These environmental changes would be reflected in changes to the water relations of the site, decreases in nutrient availability, or increases in soil salinity or ion toxicity. Over the next four years he investigated all of these changes, using both pot experiments and field studies.

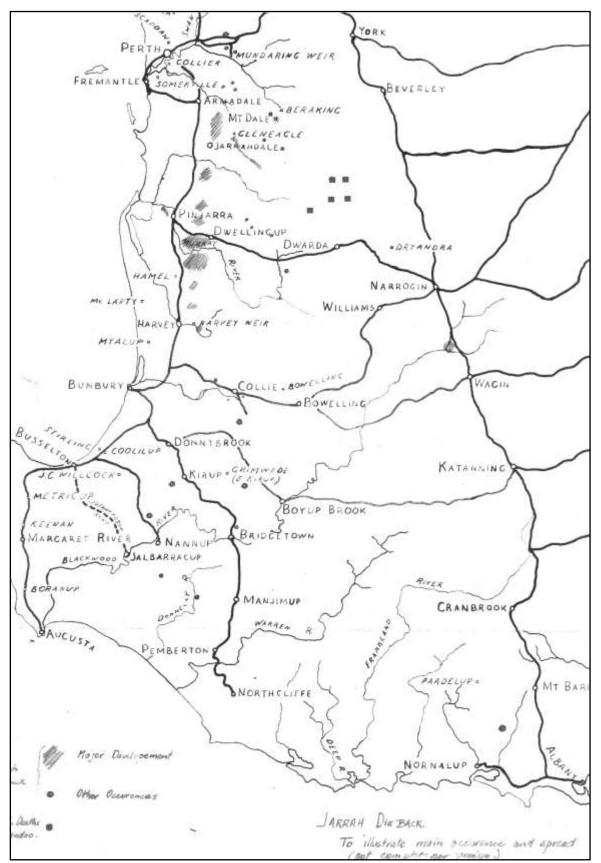


Figure 4: Map from Podger, 1959b. The original caption reads 'Jarrah Dieback. To illustrate main occurrence and spread (not complete or precise)'. The legend on the left reads 'Jarrah Dieback Major Development [coloured grey], Other Occurrences [grey spots], Patch Deaths Wandoo [grey squares].

The possibility that jarrah deaths resulted from either increased salinity or metal ion toxicity was quickly dismissed based on his research and the observations of others (Podger 1966a). He thought that jarrah deaths were unlikely to result from drought because of its survival during the severe summers of 1960-61 and 1961-62 (Podger 1962). Pot experiments conducted in 1963 supported this conclusion because they showed that jarrah was less sensitive to water deficiency than other jarrah forest trees (*C. calophylla, E. accedens, E. megacarpa, E. patens, E. rudis* and *E. wandoo*) (Podger 1967a). In 1963 he also conducted pot experiments on jarrah's waterlogging tolerance and showed that it was far less tolerant than other forest eucalypts (Podger 1963), however he rejected waterlogging as a cause of jarrah dieback because it had recently appeared in undisturbed forest, and on well-drained soil on steep (28°) slopes, and did not explain the death of many mid- and under-storey species (Podger 1967b). Also, by 1963 he had started on another line of investigation.

### Search for Phytophthora

In 1962 there were several cases of *Pinus radiata* shelterbelt mortality on the Swan Coastal Plain and Podger was asked to investigate (Podger 1962). His attention was drawn to similar mortalities in *P. radiata* shelterbelts in New Zealand (Newhook 1959) and little leaf disease of *P. echinata* in the south east of the United States (Campbell 1951; Zak 1961). Both of these problems were associated with *Phytophthora* spp., soil-borne pathogens with a wide host range, which were difficult to isolate, and attacked fine roots. Podger wondered whether a phytophthora might be the cause of jarrah dieback (Podger 1962; 1968; 1972).

Podger started with a series of pot experiments in which he compared the growth of jarrah seedlings in soil from affected and unaffected forest areas. He found that seedlings in unsterilized soil from dieback areas developed root rot and some died (Podger 1963). He quickly realised that he would need an experienced plant pathologist to isolate and identify *Phytophthora* spp., and so he arranged for Ralph Doepel, the diagnostic plant pathologist from the Western Australian Department of Agriculture, to assist. During the winter and spring of 1963 they excavated mature jarrah trees from three stands where jarrah had symptoms of crown decline, but was not dead. At each stand they examined the trunk, traced out major roots and smaller roots, and sampled pieces of apparently healthy and dying roots (R. F. Doepel pers. comm. 13 August 1980). The samples were plated out onto a range of agars including 3P *Phytophthora* spp. (Podger et al. 1965). All cultures they obtained were sent to the Commonwealth Mycological Institute in London for authoritative identification; no known pathogens were identified (Doepel and Podger n.d.).

Undeterred, Podger and Doepel continued to work with pot experiments which showed that there was a biological factor in the soil from dieback sites that caused deaths of jarrah and *B. grandis* seedlings. This was because these deaths could be reduced by soil steaming, treating the soil with a formalin drench, or treating it with thiram fungicide, however they were unable to isolate a *Phytophthora* (Podger 1964; Podger et al. 1965).

In October 1964 the *Phytophthora* specialist George Zentmyer (Professor of Plant Pathology, University of California, Riverside) who was on sabbatical leave at the Waite Agricultural Research Institute in South Australia, visited Podger and Doepel in Western Australia. Zentmyer had plates of 3P *Phytophthora*-selective agar with him. He plated seedling roots from the pot experiments onto the agar, returned to South Australia, and found that he had isolated *P. cinnamomi* (Zentmyer 1964).

Using similar techniques to those of Zentmyer (plating onto 3P agar, and baiting soil with avocado fruit), Doepel was able to isolate *P. cinnamomi* from both soil and plant roots from the pot experiments and from the jarrah forest (Podger 1964). Pathogenicity tests on seedlings showed that

it could infect and kill jarrah and *B. grandis*, but not marri (Podger 1965; Podger et al. 1965). *P. cinnamomi* appeared to be an introduced pathogen.

Over the following months, and using lupin seedlings rather than avocado fruit, Podger showed that *P. cinnamomi* could be isolated from soil from 27 affected jarrah stands and three affected banksia woodlands, but not from 344 soil samples from unaffected stands. He also isolated it directly from 31 species from the forest including jarrah (Podger 1965). In early 1965 Podger was saying that he had solved the problem of jarrah dieback and that forestry practices would have to change to minimise the spread of *P. cinnamomi* (R. J. Underwood pers. comm. 30 July 2014). By 1966 Podger 1966b). He had shown that soil types throughout the whole jarrah forest were suitable for *P. cinnamomi*, with conditions of both soil temperature and moisture being suitable for sporulation between July and late spring (probably November). He stated that this pathogen would be spread by the movement of infested soil, and suggested that modifications to logging practice and road building would need to be made to minimise spread. He had also deliberately infested 26 unaffected forest plots with either cultures or infested soil and he reproduced symptoms of jarrah dieback in 14 of these; no symptoms occurred in 26 control plots (Podger 1966b).

Podger's claim that *P. cinnamomi* was killing jarrah trees was novel, especially as previous investigations had found no evidence of consistent pathogenic lesions (Harding 1949; Stahl and Greaves 1959). The Forestry and Timber Bureau invited Erik Björkman (Professor of Forestry, Royal College of Forestry, Stockholm, Sweden) to comment on Podger's investigations. Björkman and Stahl visited the jarrah forest in March 1966, but unfortunately Podger was overseas at that time and was not able to answer Björkman's questions in person. Björkman accepted that *P. cinnamomi* was involved in the death of jarrah and banksia, but suggested that waterlogging and other soil or silvicultural conditions might also be important (Björkman 1966). He also questioned whether *P. cinnamomi* was introduced, suggesting that it might be widespread but dormant in the unaffected forest. Podger's response was vigorous, rejecting the suggestion that *P. cinnamomi* was not the primary cause of jarrah dieback, and he used his extensive soil sampling results to support his contention that it was an introduced pathogen (Podger 1966c). His arguments won the day.

The Forests Department accepted Podger's explanation that *P. cinnamomi* was the prime cause of jarrah dieback in good faith (Harris 1965). Foresters also believed that infested sites were unproductive because Podger presented measurements of six trees that showed they grew at less than 15% of trees of similar size in nearby healthy forest (Podger 1972). By the time the explanation that *P. cinnamomi* caused jarrah dieback had become common knowledge (Forests Department 1967), the Forests Department had upgraded or constructed new research facilities in Perth, Dwellingup and Manjimup (Wallace 1969). The equivalent of six full time research offices were working on different aspects of the disease: mapping extent, rate of spread, screening alternative timber species, evaluating environmental factors and investigating control measures (Batini 1973; Christensen 1975; Shea 1975). In 1968 the Forests Department invited Zentmyer and Frank Newhook (Professor of Plant Pathology, University of Auckland) to visit Western Australia to provide feedback on the research programmes that it had initiated (Newhook 1968; Zentmyer 1968). The Forests Department also sponsored research programmes on *P. cinnamomi* at the University of Western Australia and the Australian National University in Canberra (Wallace 1969).

Concerns about *P. cinnamomi* as the cause of eucalypt decline and death spread from Western Australia to other states. There was an increase in the number of researchers working on it in native vegetation from one in 1966 to the equivalent of 15 in 1971 (Newhook and Podger 1972). The Lakes Entrance Seminar in 1973 was the next step (Marks and Idczak 1973).

### Failure by Podger to adequately address Koch's postulates, and problems with methodology

Koch's postulates are fundamental to demonstrating the cause of a disease. They state that:

- 1. The suspected causal organism must be constantly associated with the disease.
- 2. It must be isolated and grown in pure culture.
- 3. When a healthy plant is inoculated with it the original disease must be reproduced.
- 4. The same organism must be reisolated from the experimentally infected plant.

Although Podger was confident that he had shown that *P. cinnamomi* caused jarrah dieback (Podger 1966b) he had not demonstrated all steps of Koch's postulates. He had used soil baiting to show that jarrah deaths were associated with infested sites with impeded drainage (Podger et al. 1965). He had used *Phytophthora*-selective agar to show that he could isolate *P. cinnamomi* from many forest plants including jarrah (Podger 1965; 1968; 1972). He had cultured *P. cinnamomi* and had shown that it could infect and kill jarrah and banksia seedlings in pot experiments (Podger et al. 1965). He had deliberately infested 26 field plots, with symptoms developing in 14 (Podger 1966b). What he had not done, however, was satisfy the first of Koch's postulates by showing a constant association between infection of jarrah trees in the forest and jarrah deaths. Also he had not satisfied the fourth postulate by showing that the plants that died in the field were infected.

On order to address the first of Koch's postulates, Podger conducted a large field sampling programme between 1965 and 1968. He plated root pieces from a large number of forest plants directly onto *Phytophthora*-selective agar, rather than looking for pathogenic lesions and attempting to isolate from those, as he had done with Doepel (Doepel and Podger n.d.). He sampled 100 jarrah trees, but was only able to isolate *P. cinnamomi* from five (Table 1); this low rate does not satisfy the first of Koch's postulates. Isolation frequencies were much higher for *B. grandis* (29%) and for other forest plants (19%). Zentmyer (1968) suggested that the low recovery of *P. cinnamomi* from jarrah was because it caused a fine root necrosis, whilst the higher recovery from banksias was because it invaded much larger roots. This explanation was accepted without question by the Forests Department (Batini and Hopkins 1972); it was based on soil baiting. Unfortunately Podger failed to mention the low recovery of *P. cinnamomi* from jarrah or the isolation frequencies from other plants (Table 1) in any of his reports to the Forestry and Timber Bureau, the Forests Department, or publications (Podger 1968; 1972).

Table 1. Frequency of the isolation of *Phytophthora cinnamomi* from various hosts in the jarrah forest of Western Australia, from the isolation books May 1965 to December 1968. Isolation methods: direct plating onto 3P or  $P_{10}VP$  agar (Erwin and Ribeiro 1996). Viewed with permission by CSIRO in February 1980, and published with permission from CSIRO in 2011. Data from Davison (2011) with permission from the New Zealand Journal of Forestry Science.

Host	Sample size	Isolation frequency (%)
Eucalyptus marginata (jarrah)	100	5
Banksia grandis	121	29
Other plants	546	19
Soil samples	1,163	23

Podger's contention that he had satisfied the fourth of Koch's postulates is also unsatisfactory. He conducted field inoculations with either cultures or infested soil in 26 plots at three field sites in 1966 (Podger 1972) and had reproduced symptoms of disease in 14 of these plots but not in 26 control plots (Podger 1966b). The symptoms that had developed after 18 months were deaths in mid- and under-storey plants, members of the Proteaceae and Dilleniaceae, not jarrah (Podger 1972). Between 1968 and 1971 two of these three sites were destroyed by salvage logging and pole cutting, but at the remaining site, which had impeded drainage, one jarrah tree died. There is no mention of any attempt to directly isolate *P. cinnamomi* from roots of symptomatic plants; the re-

isolations were done by lupin baiting soil samples. This procedure does not satisfy the fourth of Koch's postulates because it does not demonstrate that the plants were infected.

There are also problems of interpretation that result from his extensive use of soil baiting and *Phytophthora*-selective agar. Soil baiting is a very useful method for showing that the soil is infested; however it does not show whether roots of any or all species within the soil are infected. In order to demonstrate that roots are infected, they would need to be separated from the soil, sorted into the different species, washed thoroughly, surface sterilised and then plated onto selective and non-selective agar. Similarly selective agar is very useful to separate the organism(s) of interest from a mixture of other microorganisms, however it is essential to also use non-selective agar as well to determine whether other potential pathogens are also present which would need to be considered in interpreting the results. Podger does not appear to have recognised the limitations of the methods that he used so extensively.

No one who had the appropriate skills in diagnostic plant pathology appears to have checked Podger's interpretation at that time. There was a failure by plant pathologists to recognise the limitations of his methods, and to request information about sample sizes and isolation frequencies. During the 1970s his methods of soil baiting and plating onto *Phytophthora*-selective agar were used widely in eastern Australia, raising concerns that the widespread occurrence of *P. cinnamomi* would eventually lead to eucalypt decline and death (Weste and Taylor 1971; Marks et al. 1972; Pratt and Heather 1973). Even the observations that there was no extensive fine root necrosis in jarrah trees did not raise questions about severity of infection, and how the trees died (Shea et al. 1980; Shea and Dell 1981).

## Two problems, not one

### Jarrah deaths

Podger argued that jarrah dieback was a single problem affecting many of the indigenous plants in the south west of Western Australia, and that it was caused solely by *P. cinnamomi* (Podger 1972). He extended the name jarrah dieback to include the many mid- and under-storey species that died on infested sites, so that it was no longer a forestry problem, but one of whole ecosystems. This change in the definition has resulted in great confusion in interpreting Podger's work because it ascribes a common cause to all deaths, even though his work did not support this. In his references to jarrah dieback it is not clear whether he was referring to the death of jarrah trees, to the death of mid- and/or under-storey vegetation, or both. For example when he reported that he had reproduced the symptoms of jarrah dieback in 14 of 26 infested plots (Podger 1966b), he was referring to the death of Proteaceae and Dilleniaceae (Podger 1972), it was only later that one jarrah tree died and no evidence is presented that it was infected.

From the weather records, which show that the 1940s to 1960s were exceptionally wet (Davison 2014), the site characteristics and site histories where jarrah trees died (Waring 1960; Hamilton 1951; Loneragan 1961), the lack of pathogenic lesions (Harding 1949; Stahl and Greaves 1959; Doepel and Podger n.d.; Podger et al. 1965), the known sensitivity of jarrah to waterlogging (Podger 1967b; Davison and Tay 1985), and the symptoms of tylosed sapwood (Harding 1949; Stahl and Greaves 1959) which is caused by waterlogging (Davison and Tay 1985), the most credible explanation is that the jarrah trees died from waterlogging damage. Podger rejected this explanation because he noted deaths in essentially undisturbed forest and on well-drained soil on 28° slopes (Podger 1967b), however it is not clear whether he was referring to jarrah deaths, deaths of mid-and under-storey vegetation, or both.

Further confusion is apparent from Figure 4 (Podger 1959b). Although this shows the major development of jarrah dieback on the west of the northern jarrah forest, it also shows a number of

isolated occurrences of jarrah and wandoo (*E. wandoo*) deaths. As wandoo is field tolerant of *P. cinnamomi* (Hopkins 1973) it is more likely that these deaths had resulted from infection by the native pathogen *Armillaria luteobubalina* (Shearer et al. 1997). The isolated occurrences of jarrah dieback in the central and southern forest areas (see Figure 4) may have also included sites infested with *A. luteobubalina* because its wide host range includes jarrah, banksia, and many other mid- and under-storey species (Shearer and Tippet 1988). At the time the map was prepared there was no knowledge of the impact of *A. luteobubalina* on tree health in Western Australia.

Since the 1970s the rainfall in the south west of Western Australia has decreased significantly (Figure 5), and the mortality rate of jarrah has dropped (Davison 2014). The last occasion when there were widespread deaths resembling the early occurrences of jarrah dieback, followed the highest ever rainfall (237.2 mm at Dwellingup, mean 15.5 mm) in January 1982 (Shearer and Tippett 1989). Jarrah trees died on sites with impeded drainage, and *P. cinnamomi* was recovered from the bark and wood of large roots of most of the trees (Shea et al. 1982). There is no record of tylosed sapwood being seen or being looked for, although it was mentioned that the sites had water ponding above the impeding layer. It was concluded that *P. cinnamomi* had killed the jarrah trees by reducing conduction through the root sapwood (Shea et al. 1982). This is unlikely because *P. cinnamomi* primarily causes lesions in the bark (Tippett et al. 1983), and invasion of sapwood is rapidly contained (Davison et al. 1994); sapwood invasion would only occur if the tree was already dying from another cause. The only known way that conduction can be reduced in jarrah is by tylosed sapwood, resulting from waterlogging (Davison and Tay 1985).

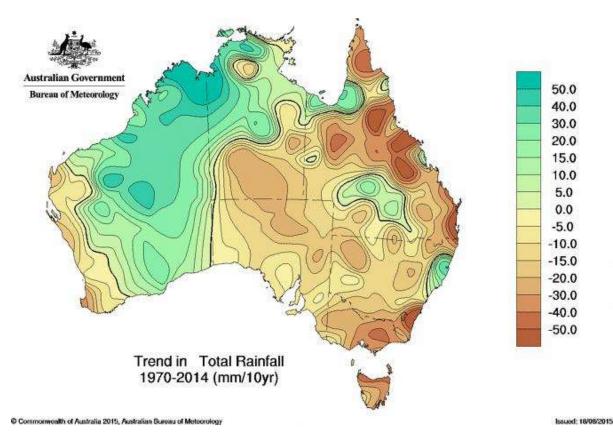


Figure 5: Rainfall trends 1970-2014. *Source:* <u>Bureau of Meteorology</u>

## Mid- and under-storey deaths

Podger's data for *P. cinnamomi* causing the death of mid- and under-storey species are far more convincing than his evidence for it killing jarrah trees and has been supported by numerous other studies (e.g. Shea 1979; Shearer and Dillon 1995; Shearer et al. 2010; Crone et al. 2013). As he

observed, infested areas occur in banksia woodland and sandplains. However in the mid-1960s the main concern was the economic impact on the supply of jarrah timber; there would have been less interest in the role of *P. cinnamomi* in killing other native plants in an area of great botanical diversity (Cahill et al. 2008). The current concern about *P. cinnamomi* relates to its effect on the conservation of the many susceptible species in this biological hotspot rather than timber production, and to better reflect this the name has been changed from jarrah dieback to Phytophthora dieback (Department of Conservation and Land Management 2003).

## Two problems, two names

Many of the sites where jarrah died in the past would undoubtedly also been infested with *P. cinnamomi* because the earliest reports mention that *B. grandis* died before jarrah (Waring 1950; Hamilton 1951; Wallace and Hatch 1953). Banksias are used as an indicator of site infestation because of their extreme susceptibility (Brandis 1983). It is likely however, that there would have been some uninfested sites where jarrah died, and Podger (1968) mentions one such site. Also Waring (1950) and Wallace and Hatch (1953) mention that *Allocasuarina fraseriana* died before jarrah if it was present on the site, which is surprising because present experience is that it is not very susceptible, and is an unreliable indicator species (E. Brown, pers. comm. 8 May 2014). Its sensitivity to waterlogging is unknown, but if it is as sensitive as jarrah, this would indicate that some sites were waterlogged, but uninfested.

Confusion has arisen because the old name jarrah dieback and the new name Phytophthora dieback have been applied to at least two problems. I suggest that the name jarrah dieback reverts to its original meaning of groups of jarrah trees that die and decline on sites with a tendency to become waterlogged following exceptionally heavy rainfall, with deaths resulting from waterlogging damage. The name Phytophthora dieback should be used for the death of mid-and under-storey species that die as the result of infection by *Phytophthora* spp. Making this distinction is important because an accurate diagnosis is the first step in designing appropriate management options.

# The legacies

Podger made an enormous contribution to the pathology of plants in native ecosystems through conceiving that a *Phytophthora* might be involved in the deaths of jarrah trees and in spite of many failures, persisting in his attempts to demonstrate this. However, by overstating his case, his work has resulted in considerable confusion that still persists within forestry, forest pathology and the pathology of woody plants. Foresters in the 1960s and 1970s responded to this perceived emergency by developing a suite of novel, innovative management practices that aimed at identifying infested areas, identifying risk and minimising spread (Batini and Cameron 1971; Batini 1976; Bradshaw and Chandler 1978; Underwood and Murch 1984). In Western Australia these practices have been applied to the management of forested areas, national parks, nature reserves and minimg operations, which has minimised spread of *P. cinnamomi* in this botanically rich area. They have been adopted in other parts of Australasia and throughout the world. Foresters can be proud of their achievements.

## Acknowledgements

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