

A REVIEW OF SOME OF THE EARLY WORK ON JARRAH DIEBACK

(5)

In 1948, the State-Commonwealth Forest Research Laboratory was set up at Dwellingup for investigations of the anatomy, pathology and ecology of jarrah (1). Crown deterioration in jarrah had been recognised as a problem for many years; the regeneration programme of the 1930's was devised to improve crown development, and to give fire protection to the regenerated forest (2). The initial investigations of the Dwellingup Research Station were centred around crown deterioration and death of jarrah that occurred with death of the understorey in patches in the forest; the early results were summarised by Wallace and Hatch (3). Harding carried out the initial pathological investigations. His work was very broadly based and included grafting and inoculation experiments to look for viruses, attempts to isolate pathogens such as fungi and bacteria from affected foliage, sap and heartwood, and anatomical examination of roots, twigs, branch and bole for fungal invasion. None of this work showed evidence for a pathological disorder, and in 1949 Harding concluded that "there was no evidence of any pathogenic disorder either from the anatomical or induced infection aspects" (3). The only indication that a pathogen might be involved was that there were frequent tyloses even in the small roots of dieback affected saplings. There was no evidence of consistent insect attack, although the possibility of an insect vector was considered.

Forest soils from healthy and dieback areas were compared, as were nutrient analyses of leaves from healthy and dying trees, but in neither case were there any marked differences (3). In both healthy and unthrifty forest, fertilization trials consisting of nitrogen, phosphate and mixed fertilizer (Ca, P, K, ^SMg, Cu, Zn, B and ^{Mn}M) applications gave no response in jarrah, although in the case of the nitrogenous fertilizers there was an increase in understorey vigour (4).

→ *Al and Cl toxicity investigated for jarrah*

As far as determining the cause of dieback was concerned, the work was not very productive, however, considerable time was spent in describing

and mapping the disorder. The Teesdale Regeneration Transect was set up in 1949 and was monitored regularly until destroyed by fire in 1961 (5). The spread of the disorder and recruitment to the dieback site could be followed in such transects. It was soon established that Banksia grandis was being eliminated, Eucalyptus marginata was not developing beyond the advanced growth stage, and that jarrah which made dynamic shoot growth died before making more than a few feet in height. Marri, on the other hand, suffered relatively little mortality, replacing jarrah as the dominant plant, even though its growth rate was only 1.4 ft. per year, and the rate of increase of the marri sapling class was slow (5).

In 1959 the Forestry and Timber Bureau appointed Frank Podger as a Research Officer to work on jarrah dieback and crown deterioration at the Dwellingup Research Station (6). He was familiar with the problems, having been appointed an assistant divisional forestry officer in Western Australia two years earlier (7). Also in 1959, Stahl and Greaves from the Forestry and Timber Bureau, visited Dwellingup in September to look again for a pathogenic cause of the disorder. They examined a number of different stands in the Gleneagle, Dwellingup, Nanga Brook and Tallanalla areas examining jarrah, Banksia grandis, Macrozamia reidlii and Persoonia longifolia for pathogenic fungi and insect pests. In each case several trees were felled and the crowns examined; the bark was removed from the boles, branches and twigs, and the phloem and wood inspected. In most cases a portion of the root system was examined. Although many pathogens and pests were observed, there was nothing that could be regarded as a primary pathogen. In Persoonia the terminal portions of roots up to an inch in diameter were dead in unthrifty trees, in Macrozamia the roots in both healthy and yellowing plants appeared identical, whilst in jarrah most of the small roots were lost during excavation. The possibility of a virus disease was discussed, owing to the yellow mottling on some leaves of all the jarrah trees felled, but the overall conclusion drawn from the

investigation was that "with the possible exception of a virus, there is no evidence that a pathogen is the sole cause of the disorder" (8).

It was against this background that Podger took up his position at Dwellingup in 1959. He started by reviewing the work of the previous ten years (4) and concluded that it was a serious problem of unknown cause occurring over a wide range of sites and over most of the range of jarrah, affecting many of the understorey as well as the overstorey species. As it was probably of recent origin, Podger considered the major changes which had occurred in the forest in the previous hundred years, i.e. cutting, complete exclusion of fire from many areas, and the increase in the incidence of severe fires. The continual removal of leaf litter by fire might result in a decline in soil fertility, but as dieback occurred in both burned and protected forests, as well as being absent from severely burnt areas, fire seemed unlikely to be a causative factor. In addition, the earlier work of Wallace and Hatch (3) discounted declining soil fertility as a cause of dieback (4).

Podger considered that the most promising area for investigation lay in the field of changing site water relations due to cutting (9) so he started by conducting transpiration studies of jarrah and marri combined with various watering experiments. By 1961 he had shown that under water stress E. calophylla does restrict its use of water whereas E. marginata does not (10), and by 1962 he had shown that jarrah dieback is probably not due to drought (11). He rejected drought as a cause for three reasons : firstly the severe summers of 1960-61 and 1961-62 produced no decline in the condition of weakened trees although there were new extensions of dieback into adjacent healthy forest, secondly species resistance to dieback and relative tolerance to drought were not the same, and thirdly because despite severe summers, there was no increase in dieback in the low rainfall Eastern jarrah forest (11).

Having dismissed drought as a possible cause he considered water-

logging, and showed that E. marginata had a lower tolerance to waterlogging than any of the other of jarrah forest eucalypts (12). However, he rejected waterlogging as a possible cause of dieback because of its then recent occurrence in the relatively undisturbed low plateau west of Nannup, because of its restricted occurrence in the wetter southern forests around Manjimup, Pemberton and Walpole, and because waterlogging would not explain the death of such understorey species as Banksia littoralis, which are adapted to poorly drained sites (13). In addition, the development of dieback on steeply sloping sites at Serpentine Pipehead Dam Wall and near Churchman's Brook Dam Wall argued against waterlogging as a cause (14).

The 1961 fire which burnt down the Dwellingup Research Station (15) resulted in the Forestry and Timber Bureau establishing a separate Research Station at Kelmscott, subsequently opened in 1964, with Podger as the officer in charge (16). Consequently, much of the work done between 1961 and 1964 was performed under difficult circumstances.

In 1962 there were several cases of shelter belt mortality of Pinus radiata on the Swan coastal plain. From the results of a pot trial which he carried out, Podger thought that these deaths were probably due to a root rot. Publications by Newhook (17) on shelter belt mortality of P. radiata in New Zealand caused by Phytophthora spp., and by Campbell (18) on little-leaf disease of Pinus echinata in the United States caused by Phytophthora cinnamomi directed Podger's attention towards pathogenic fungi. Both the New Zealand and American reports were of tree diseases where the pathogen had a wide host range, where it was difficult to isolate by normal plating methods, and where it primarily attacked the fine feeder roots. Podger considered that a similar organism might be important in Western Australia, and in December 1962 he started to look for a root pathogen, such as a Phytophthora species as a cause of jarrah dieback (11).

He started with pot experiments, growing jarrah seedlings in sterile

and unsterile soil from healthy and dieback forest sites, and he found that while seedlings in sterilized soil remained healthy, those in unsterilized soil from under dying jarrah trees showed extreme root rot and died (12). As Podger realised that attempts to isolate fungi such as Phytophthora spp. would be difficult and would require special techniques, arrangements were made for Ralph Doepel, a plant pathologist with the W.A. Department of Agriculture, to look for these fungi (19). Field samples were initially used; small roots were plated directly onto non-selective and 3P agar, and in addition Campbell's apple trap method was used for soil and roots. The sampling was continued during the winter and spring of 1963, but nothing considered to be pathogenic was isolated.

However during this time a number of pot trials indicated that a root pathogen was probably involved. Seedling mortalities in unsteamed soil from around dying trees could be reduced by thiram drenching, and prevented by formalin treatment (20). In addition, seedling trials with Banksia grandis in dieback and healthy forest sites, with and without formalin sterilization, showed that seedling survival was lowest at the margin of the dieback patches.

As a result of the negative results from the field isolations in 1963, further work was concentrated on attempting to isolate a pathogen from the pot trials using Newhook's modification of Campbell's apple trap technique (17). In addition, a soil sample was tested for nematodes, but nothing pathogenic was found (19). Then, in October 1964, Zentmyer, who had been visiting Western Australia, recovered Phytophthora cinnamomi from jarrah roots from a pot experiment using dieback soil, and Doepel subsequently recovered it from soil in a typical dieback area using Zentmyer's avocado technique (20). Pathogenicity testing on jarrah and Banksia seedlings established that P. cinnamomi could infect and kill both hosts, and could be reisolated from the host roots (20).

TABLE 1

Frequency of isolation of Phytophthora cinnamomi from various hosts in the jarrah forest. (Isolation methods : direct plating onto 3P or P₁₀ VP agar, or by lupin baiting). Data from Kelmscott isolation books, May 1965-Dec 1968.

Year	<u>E. marginata</u>			<u>E. calophylla</u>			<u>B. grandis</u>			other plants			soil samples		
	+ve	-ve	Total	+ve	-ve	Total	+ve	-ve	Total	+ve	-ve	Total	+ve	-ve	Total
1965	5*	42	47	0	5	5	16	34	50	60	115	175	175	281	456
1966	0	9	9	0	7	7	7	10	17	35	195	230	56	398	454
1967	0	42	42	0	3	3	10	35	45	7	103	110	30	209	239
1968	0	2	2	0	0	0	2	7	9	2	29	31	7	7	14
Totals	5*	95	100	0	15	15	35	86	121	104	442	546	268	985	1163
% recovery	5%			0%			29%			18.5%			23%		

* +ve isolation 30.8.65 Karnet, D.B.Z., direct plating of roots onto 3P agar.
 8.10.65 Willowdale " " " " " " "
 4.12.65 E. Kirup; 3 advance growth seedlings, direct plating onto 3P agar,
P. cinnamomi recovered from roots, lignotubers and up to 4"
 up the stems.

The next two years were spent testing the hypothesis that P. cinnamomi was the cause of jarrah dieback. P. cinnamomi was isolated consistently from dieback patches but not from healthy forest, and was recovered from 55 species of indigenous plants, including jarrah. The pathogenicity of these isolates was tested against different provenances of jarrah seedlings and against seedlings of other indigenous species (14,21).

In addition in October 1965 Podger inoculated three healthy stands of forest with cultures of Phytophthora cinnamomi and with soil from both a dieback patch and from healthy forest. By February 1967 typical dieback symptoms including some deaths had appeared in the understorey of the dieback and P. cinnamomi inoculated plots, and P. cinnamomi was recovered from soil and root samples from these, but not from comparable samples from plots inoculated with soil from healthy forest. Symptoms in jarrah appeared much more slowly, but by February 1971 one tree had died and many others showed typical dieback symptoms (21). P. cinnamomi was not however isolated from soil samples taken at this time from the plots, although it was isolated from the margin of the dieback patches which had developed (22). Podger concluded that there is "little doubt that P. cinnamomi is the cause of..... jarrah dieback." (21).

There are however difficulties with this interpretation which were apparent at this time, and which have not been resolved since then. The most important is the infrequency with which P. cinnamomi was isolated from jarrah; Podger's isolation figures are shown in Table 1.

It may be that the methods being used at this time, i.e. lupin baiting and direct plating onto 3P agar, were not very sensitive. However the only positive isolations of P. cinnamomi from jarrah were made onto 3P agar with frequencies shown in Table 2.

TABLE 2.

Positive isolations of *P. cinnamomi* from jarrah by direct plating onto 3P agar (data from Kelmscott isolation books).

Date	Locality	No. root pieces plated	No. positive	Comments
30.8.65	Karnet (dying banksia zone)	8	3	
5.10.65	Willowdale	10	1	
4.12.65	E. Kirup	20	8	lignotuber
"	"	26	3	roots
"	"	14	3	stem

3
advance
growth
seedling

Palzer (23) estimated that from his experience the probability of recovering P. cinnamomi from a sample in which it was known to exist was $\frac{1}{4}$, and the frequency with which it was recovered from soil and plant samples (other than E. marginata and E. calophylla) as shown in Table 1, agree with this figure.

More recent sampling has failed to isolate P. cinnamomi from jarrah except in a consistently wet site (24) or from trees which were irrigated during the summer (25). It may therefore be valuable to consider that jarrah dieback refers to two distinct but related things; firstly to the patch death of the understorey in the jarrah forest which is caused by P. cinnamomi and secondly to the slow decline and death of jarrah which occurs in these patches, but from which P. cinnamomi has only rarely been isolated. Perhaps the assumption was made that because P. cinnamomi was killing the understorey it must also be killing the jarrah.

Podger's evidence from his Koch's postulates inoculations do not conclusively show that P. cinnamomi can kill jarrah trees. The results of the preliminary work (20) which showed that P. cinnamomi is pathogenic to jarrah seedlings are not unexpected as Phytophthora spp. are serious nursery pathogens. The field inoculations were similarly inconclusive. Although one jarrah tree died and several others showed dieback symptoms in plots which had been inoculated with P. cinnamomi and diseased soil, no attempt was made to isolate from jarrah roots, so that it was not established whether or not the death of jarrah was directly due to root invasion by P. cinnamomi (21).

This therefore raises a number of possibilities which should be considered :

1. That P. cinnamomi is present in the root system of jarrah but it is impossible to isolate it readily. Isolations from jarrah by Podger, shown in Table 2, Gardner (24) in a wet site, Shea and Dell (25) on irrigated trees, and by Malajczuk, McComb and Parker (26) and more recent work by Dell (27) on roots inoculated in the field show that P. cinnamomi can be recovered from jarrah roots. At the moment there is no reason to propose that if the fungus is present it cannot be isolated.
2. That P. cinnamomi is present in the root system of jarrah at a very low level, and that the small amount of root damage suffered by jarrah is sufficient to cause the trees to decline and eventually die.

Although jarrah must sustain some root damage during selective logging there is no evidence that this causes a decline in the trees. In addition, Loneragan (28) states that there is no damage and no loss of increment resulting from prescribed burning if crown scorch does not occur, ~~and~~ ^{although this} must cause some root damage. However burning effects will be complicated by the alteration of soil nutrients.

The effect of root damage at different times in the jarrah flowering cycle can conveniently be considered here. Dell (29) has shown that young roots may be produced at any time of the year when soil moisture and temperature are not limiting, but it is not known whether the roots are produced irrespective of the flowering cycle and state of the crown.

At the moment it seems unlikely that the jarrah root system is particularly sensitive to damage, but the evidence is only based on casual observation.

3. That P. cinnamomi is present in the root system of jarrah at a very low level, but it affects the host physiology in some way that causes the decline and death of jarrah.

Hart (30) has suggested that P. cinnamomi can produce HCN from cyanogenic glycosides, and that the cyanide so formed acts as a toxin. Malajczuk et al. (26) in their infection study of P. cinnamomi in jarrah

and marri roots do not mention toxic effects on the host at a distance from the fungus, but they may not have considered this possibility.

An increase in the resistance to the movement of water through diseased as compared with healthy plants, such as those described by Duniway (31) for Phytophthora cryptogea in safflower, may also occur.

The effects of P. cinnamomi on jarrah physiology have not been considered, and may well indicate why jarrah trees die.

4. That P. cinnamomi is present in the root system of jarrah at a very low level, and that jarrah responds to this by excessive root shedding.

Palzer (23) and Malajczuk et al. (26) both showed that in artificially inoculated unuberised jarrah roots of mature trees, lesion development was limited, and was followed by subsequent regeneration. Excessive root shedding as a result of infection therefore seems unlikely.

5. That P. cinnamomi is present in the root systems of jarrah at a very low level and this, together with other pests and diseases brings about the decline and death of jarrah.

Stahl and Greaves (8) mentioned a number of pests and diseases on jarrah from dieback sites. Although they did not consider any sufficiently important to kill jarrah by themselves, in conjunction with a root pathogen, their effects might be compounded.

This is perhaps worth further investigation.

6. That although P. cinnamomi may be present in the jarrah root system at a very low level, the most important effect of the fungus on jarrah is by the alteration of the site by the removal of the understorey.

Removal of the understorey would affect the nutrient status of the site; but Wallace and Hatch (3) discounted differences in nutrient status as a cause of death of jarrah and even if jarrah is suffering from a mild root-rot there is no reason to doubt their conclusion.

Another effect of the removal of the understorey is to raise the water table. In a limited experiment Shea (32) showed that soil

moisture levels showed greater fluctuation in response to rainfall, and were higher during August and September 1968 in plots where the understorey and litter had been removed, than in untreated plots. On a grander scale, data from the Wellington Catchment (33) indicates that surface water tables rise gradually over a number of years when all the vegetation is cleared.

Data on when jarrah trees start to decline might indicate whether this occurs shortly after a tree becomes infected with P. cinnamomi, as deduced from understorey symptoms, or whether the decline occurs several years later, and might therefore be attributed to an alteration of the site. The only measurements available (34) are of trees 2 and 3, shown in Fig. 1. which became dieback affected in October 1964. There is no immediate check on growth; unfortunately measurements were not continued after 1966.

Finally one must ask whether it is important to know why jarrah dies.

It only becomes possible to evaluate the possibilities for control or management of a disease when that disease is fully understood. At the moment the management of jarrah dieback has been by minimising the spread of soil infected with Phytophthora cinnamomi, by replanting dieback affected areas with tolerant eucalypts and by manipulating the understorey by fire. If the reasons why jarrah dies are understood, the search for naturally occurring selections which will tolerate the disease can be undertaken. Eventually management options may also include replanting dieback sites with tolerant jarrah.

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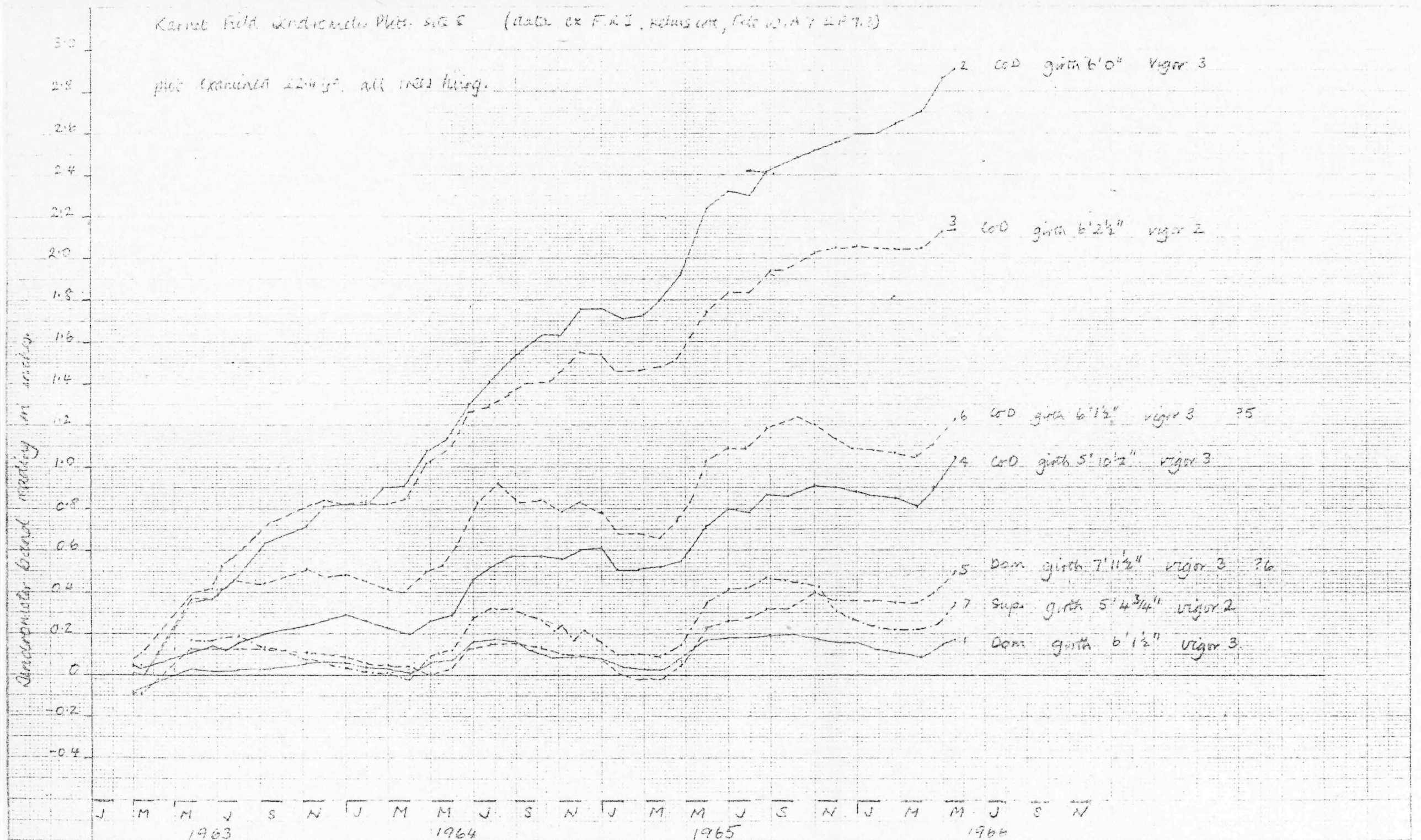
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Karnat Field, Windward Side, Plot 5 (data ex F.H.I., Nelson, Feb 12, 1967, 2 P. 7, 8)

plot examined 224 y², all trees living.



Karnet Field Windward Plot, site 8 (data ex F.W.I. Nelson et al, Feb 10, 1967, p. 7, 8)

plot examined 22-4-67, all trees living.

