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THE IMPACT OF ECOLOGICAL, CULTURAL AND BIOLOGICAL FACTORS ON THE STRATEGY AND COSTS OF CONTROLLING ROOT DISEASES IN TROPICAL PLANTATION CROPS AS EXEMPLIFIED BY *HEVEA BRASILIENSIS*

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SUMMARY

Profitability and the availability of financial, material and manpower resources are studied before starting a commercial enterprise ; the longer its term, the more complex the studies. The costs of controlling plantation diseases (in the tropics or elsewhere) are part of a long-term investment programme be the diseases sporadic (some leaf diseases), predictably annual (seasonally induced, juvenile leaf), or perennial (root diseases). Especially in the case of root diseases, the pattern of investment, and hence profitability, can be affected by the choice of control measures and their timing, factors which must be evaluated by plant pathologists. These factors are examined in general terms and with particular reference to effects on them of some aspects of the ecological, cultural and biological control of major root disease pathogens of some tropical plantation crops, Armillariella (Armillaria) mellea, Ganoderma philippii (pseudoferreum), Phellinus (Fomes) noxius, *and* Rigidoporus (Fomes) lignosus.

INTRODUCTION

The introduction 100 years ago of *Hevea brasiliensis* to the East from the New World probably had a greater long term impact on the overall economic development of the region than any other single factor, certainly in agriculture. The natural rubber industry emerged when systems of tapping based on excision of the outer bark were developed by H. N. Ridley, then Director of the Singapore Botanic Gardens, in the last decade of the last century and in the first decade of this century when he was joined by R. Derry. Thereafter, the industry increased rapidly and had an ever-increasing impact on the economics of the region ; the numbers of recognized pests and diseases also increased rapidly and, in turn, had their impact on the economics of the industry. There is no doubt that root diseases have caused more losses than all other pests and diseases combined and of them by far the worst has been White Root disease, first ascribed to *Fomes semitostus* Berk by Ridley (1904) and later identified as *Fomes lignosus* Klotzsch by Lloyd (1912), a name validated by him 3 years later (Lloyd, 1915), although it is now referred to *Rigidoporus lignosus* (Klotzsch) Imazeki (Imazeki, 1952). Nomenclatural problems and those of misidentification were reviewed in detail by Fox (1961a) when he also adduced evidence to fulfil Koch's postulates for the first time for this pathogen —over half a century after the disease it causes had first been described. This paper is biased towards this pathogen because it can be so severe in its effects, it is so widely distributed throughout the world in the lowland tropical rain-forest zone and it has such an extensive host range among plantation and forest tree crops.

Despite the severity of the losses they cause, root diseases — probably because they are unseen and insidious — have always attracted less attention than the other more spectacular but less dangerous maladies that occur above ground. Moreover, expenditure on root disease control always seems first to attract cuts in times of financial stringency or recession — again, probably because the effects of such cuts are not immediately evident. It seems, therefore, appropriate on this occasion to stress again the economic importance of root diseases, the costs of their control, and how these have been influenced by research.

It is axiomatic that profitability and availability of financial, material and manpower resources are studied before starting a commercial enterprise ; in general, the longer the term of the enterprise the more complex the studies. The costs of controlling plantation diseases in the tropics or elsewhere are part of a long-term investment programme be they sporadic (some leaf diseases), predictably annual (seasonal or monsoon induced, juvenile leaf) or perennial (root diseases). Especially in the case of root diseases the pattern of investment, and hence profitability, can be affected substantially by the choice of control measures and their timing ; it is the responsibility of plant pathologists, if necessary in consultation with economists, to evaluate these factors before making recommendations. As O. S. Peries (1966), now Director of the Rubber Research Institute of Sri Lanka (RRISL), pointed out at a Conference 11 years ago, "... if we spend one rupee on controlling a disease and the return we get from it, in extra crop and so on, is only worth 99 cents, well we are merely strangling ourselves".

The Extent of the problem

When I joined the Rubber Research Institute of Malaya (RRIM), research staff were then, and for some years after, expected to fulfil both a research role for the industry and an advisory function for estates. Over 20 years ago I was faced with the problem of trying to devise a scheme to control root disease neglected during the war in plantings of young mature rubber (Fox, 1961b). Costs had to be established in terms of materials, and of labour requirement and its capabilities ; this was relatively straightforward but setting these costs against projected profit proved more difficult ; to questions of putting a potential value on an individual tree in terms of its future production and profit I received nearly as many answers as persons asked. Many were, however, variations on the same theme, *i.e.* that it was not feasible to expect a direct answer. At that time, economics *per se* were not being studied at the RRIM, an Agricultural Economics Section within the Statistics Division not being formed until 1963. It was therefore necessary to attempt to estimate the impact of disease on yields over the life of a stand which itself required an investigation to estimate "life yields" for an individual tree. The late Ir. P. de Jonge, in co-operation with his then colleagues of the Botanical Division of the RRIM, calculated a table of individual tree yields based on 8 tapping cycles over a period of 38 years from first opening. The potential effects of yield stimulation were deliberately omitted and I used the data to calculate life yields in a theoretical disease-free area — but allowing for some natural wastage *e.g.* wind damage, lightning, Brown Bast *etc.* —and then superimposing on this wastage "light", "moderate" and "heavy" disease losses of the order known to occur. The results of this simple model were surprising to many when presented in summary or graphically (Fox & Newsam, 1958). The important point in relation to the summary in Table 1 is that the rates of losses used were not high when considered as events in a passing year. With changes in planting material, tapping procedures, and stimulation techniques the actual figures — hypothetical then — are not valid today ; but what is valid today, as then, are the relationships between them indicating production losses over the life of a stand of *ca.* 20%, 30% and 45% for the three disease levels.

TABLE 1 : THE EFFECTS OF 3 LEVELS OF ROOT DISEASE OR NONE ON THE FINAL STANDS AND CUMULATIVE YIELDS PER ACRE
FOR A 38 YEAR TAPPING LIFE
(PROJECTED YIELDS IN 1957, INITIAL STAND 120/ac)

Disease	Cumulative yield (lb)		Final stand	
None	43,320	(100%)	108	(90%)
Light	34,991	(81%)	79	(66%)
Moderate	30,587	(71%)	64	(53%)
Severe	24,469	(56%)	42	(35%)

Production losses are, however, only part of the total loss and I pointed out that : " the largest single item in the cost of production is tapping. Re-organising tapping tasks is a major undertaking that is done only when the stand has fallen low enough to make it practicable and worth while. Up to that time the output per tapper will have been progressively falling, and the cost of production correspondingly rising. In addition, the estate's general charges, remaining at much the same level, will be spread over a smaller output, further increasing the production cost ".

In contrast to theoretical calculations, the figures shown in Table 2, previously cited (Fox, 1964b), are still pertinent ; these are of yields averaged over several fields for four successive years in two areas where root diseases had not been properly controlled ; in the first mainly white root disease and in the second mainly red root disease, *Ganoderma philippii (pseudoferreum)*.

TABLE 2 : YEARLY DECREASES IN YIELD (LB/AC) DUE TO A, *Rigidoporus (Fomes) lignosus* AND B, *Ganoderma philippii (pseudoferreum)*

A	953	897	861	659
B	1155	985	939	885

One other example, taken from West Africa, not only illustrates the problem elsewhere but is also of interest to the pathologist ; besides the classic trio of white, red* and brown (*Phellinus (Fomes) noxius*) root diseases there is also — and locally devastating — root disease caused by *Armillariella mellea*, better known as a temperate climate pathogen but with curious distribution in the lowland tropical rain forest zone in Central and West Africa (Fox, 1971). The figures shown in Table 3, as at 1963, are important because management of this group of estates (planted mostly from jungle) had not been expecting root disease problems and had not realised that the situation was becoming severe until the year before, thus lending emphasis to the comments above about " unseen and insidious " and " events in a passing year ". A major drive to control disease in this area was made after 1963 but it was halted by civil strife and the position by 1970 is all too evident from the comparisons in Table 3 and the details in Table 4.

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* Doubts that "red" root disease in West Africa is caused by *G. philippii*, raised by Fox (1964a), have not yet been resolved.

TABLE 3 : EFFECT OF 4 ROOT PATHOGENS IN WEST AFRICA COMPARISONS 1963 — 1970 ; TREES LOST AS % OF TREES PLANTED AND TREES DISEASED AS % OF THOSE STILL STANDING

Year of planting		1957s	1957	1958	1959	1960	1961
Lost %	1963	32	26	36	40	28	31
	1970	49	46	45	42	26	26
Diseased %	1963	20	23	10	7	2	1
	1970	26	63	24	23	22	25

S = seedlings

TABLE 4 : EFFECT OF 4 ROOT PATHOGENS ON STANDS /AC IN 1970 IN WEST AFRICA

Year of planting	1957s	1957	1958	1959	1960	1961
Initial stand	247	160	180	180	180	180
Found healthy	88	38	68	73	81	85
Infected/treatable	14	20	13	12	16	18
Potential stand	102	58	81	85	97	103
Potential % initial	41%	36%	45%	47%	54%	57%

S = seedlings

Patterns of expenditure

The greater the proportion of the initial stand that is available for selection, the better the final stand should be — but not at any price. Management must decide when to replant a given area (or to initiate new plantings) on the basis of current and forecast yields and income, of available capital (ready or borrowed), and of forward assessments including such processes as risk analysis that endeavour to take into account fluctuations of commodity demand, costs of labour and raw materials *etc.* Clearly, the pattern of investment is important whether planting rice or building a factory ; the delay between capital expenditure and income may be shortened in the one case by planting a short-growth-duration cultivar and in the other by pre-fabricated building techniques. The longer the delay in capital expenditure the greater the benefit-either by interest earned on capital not spent or by reduced borrowing requirements.

Consider the case, over a six year period of immaturity, where six hundred units of currency may be spent directly or indirectly on root disease control. Some very simplified patterns of nominal expenditure are shown in Fig. 1. Pattern 1, a straight line, is a simple outlay of 100 units/annum which may be used as a base line. Pattern 2 might illustrate the outlay for full mechanical clearing, initially high in year 1 at 350 units and then, with lower post-planting disease costs, rising by 50 units/annum to year 6. Pattern 3 might be poisoning and hand clearing at 150 units in year 1, then 25 units each in years 2 and 3 for foliage inspection with little other expenditure, rising to 100 units for the third and fourth year and, say, 200 units in the last year as a sequel to precautionary collar inspection. Pattern 4 represents, say, our best current procedures with a lower notional total of 400 units.

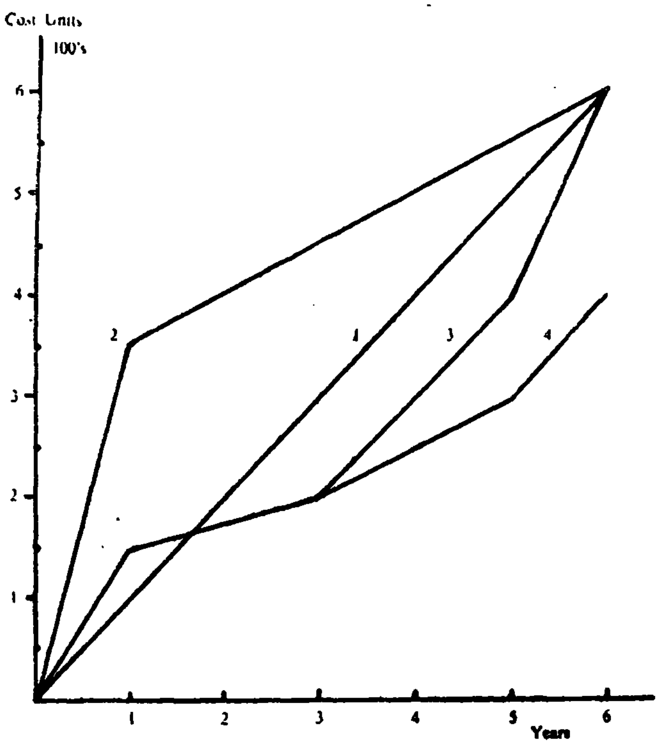

Fig. 1 Hypothetical patterns of cumulative expenditure on root diseases during immaturity (for details see text)

Superficially, the first three patterns represent the same outlay of 600 units but this is an over-simplification ; their shape determines the real value of the expenditure ; the more the slope is curved to the right the more efficient the pattern. A simple method of comparison (assuming we have achieved the same level of disease control) is to examine the patterns by discounted cash flow at, say, a modest 7·5% compound interest rate. This may be done in two ways, either as an "internal account" for root disease control within the overall budget for replanting (or new planting) or as part of the whole budget. In the latter case, discounting would be carried forward until the time, usually well after the fifteenth year, when the net cash flow after discounting becomes positive. In the former case, the value of the currency units expended in each year may be discounted forward over the six years of planned root disease control. This shorter period of discounting gives greater prominence to the numerical differences between patterns and is therefore used here for illustration.

The value now of 100 units of capital discounted at 7·5% compound would be 93·0233, 86·5333, 80·4961, 74·8801, 69·6559 and 64·7962 for six successive years and the discounted value of pattern 1 will be the sum of the foregoing figures as shown in column 1 of Table 5. For different patterns, the lower the discounted total, the poorer the investment. The difference figures at the foot of Table 5 show that for nominal equal expenditures of 600 units, pattern 2 is poorer than 1 by 40·3 cash units, but that pattern 3 with its bulge to the right is 17·0 units better than 1 and 57·3 units better than 2. Pattern 4a in Table 5 (omitted from Fig. 1 for clarity) represents a total of 400 units spent equally (66·6 units) in each year and its discounted 'loss' is of course, two thirds that of pattern 1. Pattern 4 (Fig. 1) — a 'sigmoidal' variation of 4a and with a 'loss' nearly the same — highlights an important point: apart from costing a nominal total of 200 units less than patterns 1, 2 and 3 its 'loss' is less than *HALF* that of pattern 2 thus emphasising the importance of the expenditure pattern as well as its total. Clearly, this more precise approach will reveal differences either not shown or distorted by the simple arithmetical additions that have been used previously (Newsam, 1967).

TABLE 5 : INVESTMENT PATTERNS FROM FIG. 1 DISCOUNTED FORWARD FOR 6 YEARS AT 7·5% compound interest

Pattern Year	1	2	3	4a	4
1	†64·7962*	226·7867	97·1943	43·1974	97·1943
2	69·6559**	34·8280	17·4140	46·4372	17·4140
3	74·8801	37·4401	18·7200	49·9200	18·7200
4	80·4961	40·2481	80·4961	53·6640	40·2481
5	86·5333	43·2667	86·5333	57·6888	43·2667
6	93·0233***	46·5117	186·0466	62·0155	93·0233
Totals	469·3849	429·0813	486·4043	312·9229	309·8664
" Loss " from nominal total	130·6151	170·9187	113·5957	87·0771	90·1336

Between :—	1 and 2	1 and 3	2 and 3	2 and 4
Difference =	40·30	17·02	57·32	119·21

† Equals discount factor X100 for sixth*, fifth** . . . first *** year from date of expenditure

In the decade and a half commencing about 1955, the results of research have not only altered the shape of the curve in the desired manner but they have also lowered the final total. Later, I will examine some of the details of and understanding behind the alterations. It is, however, salutary to examine, at least briefly and in principle, what may happen if the shape of the curve goes so far to the right that disease is not brought under control during immaturity and there is a residual or even a major disease problem in a mature stand after it comes into tapping.

Fig. 2, again a much simplified example, represents a falling yield due to disease which, at time A, is predicted as continuing to fall to F. Control measures are initiated which, at first, require the removal of some diseased but still yielding trees thereby dropping the yield at A. Thereafter, suppose yield remains at a constant level shown by the horizontal line A-E. It is not until time B that $Y_1 = Y_2$ and the lost yield is recovered ; however, further time is required to recover y — the lost interest from income on Y_1. Moreover, the position of E is determined by the need also to recover C the capital expenditure at A and then c the interest on that capital. Substituting estimated costs and values and using customary accounting procedures to determine net cash flow after applying appropriate discount rates, readily shows how heavy the penalties may be for failing to control root diseases at an early date whether it be because of bad techniques or ill-judged economies in expenditure. It is unfortunate that E can approach F with disquieting rapidity and in a period of unstable prices C becomes risk capital which should not be borrowed or, if available, is best put to earn interest elsewhere. This is what happened in the example cited above from Fox (1961b) and in the case of some thousands of the acres in the example from West Africa. Such fields must be relegated to the role of a wasting asset, to remain an eyesore and a reminder of ill-judgement, incompetence or enforced error until they can be replanted.

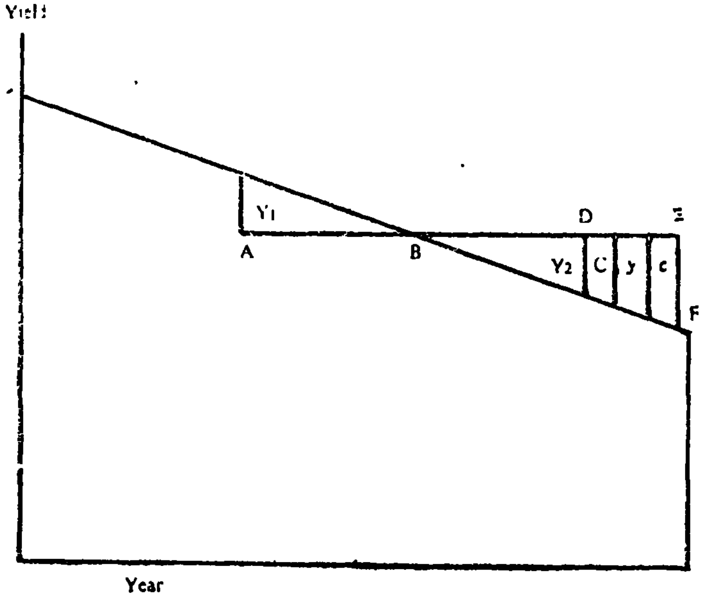

Fig. 2 Hypothetical yield patterns as affected by expenditure and losses from root diseases during maturity; for details see text.

The type of model described above, alone or suitably elaborated to accord more with reality, has its uses in the determining the future of a *field* and conveniently illustrates the principles involved. A more precise approach, which can also be used for fields, is based on potential profit and should always be used *to set economic limits on what may be spent on an individual tree at any time during its life*. The slope in Fig. 3 represents the potential profit value of an individual tree from the time of initial yield at A to the time B when the field is scheduled for replanting when, of course, its potential profit is zero. A very simple arithmetical model may be derived using figures approximating to some cited during this Conference. Suppose that over the economic life of a field there is an average (constant) yield of 1,500 pounds per annum from 120 trees per acre for an economic tapping life of 25 years, and further assume a constant profit factor of one currency unit per pound of dry rubber. The simple calculations in Table 6 show a potential profit per tree of 312·5 units at the beginning of year 1 falling to 12·5 units at the beginning of the final year, 25. The potential profit at the beginning of year 14 is 150 units and suppose at that time that an individual tree has been found to have root disease and the treatment necessary to save it amounts to 90 units, made up from 70 units for root treatment plus 20 units for corrective pruning. Its potential profitability is immediately reduced to a nominal 60 units, but the 90 units expended now are part of the future profit from that tree and, therefore, must be discounted at what ever interest rate is relevant. Table 7 shows the individual values of 12·5 units for the remaining years of the tree's life and their cumulative totals discounted at 7·5% and 10% compound. At the lower rate the 90 units will not be recovered until the penultimate year of tapping and at the higher discount level they represent an excess of expenditure over income *i.e.* a loss of 5 units. But that is not all, for examination of figures published by the RRIM on the affects of corrective pruning to minimise wind damage, show that such treatment may quite drastically reduce yield. Thus one may incur a double penalty by combining excess expenditure with yield reduction.

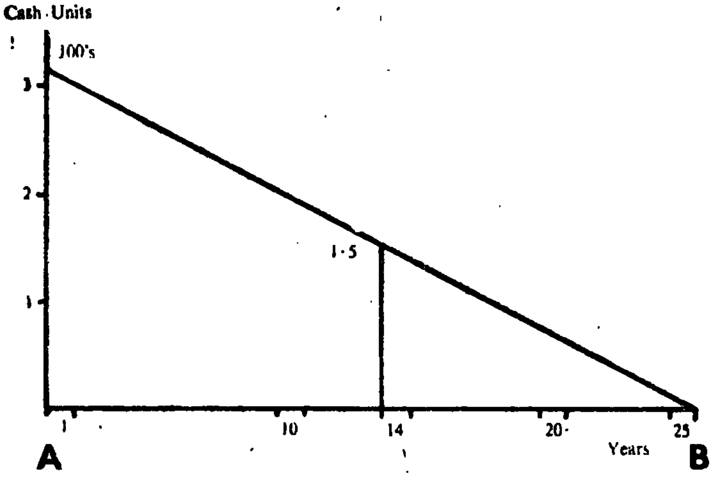

Fig. 3 Potential profit value of an individual tree from first opening (A) to replanting (B); for details see text.

This type of assessment may equally be applied to small groups of trees affected by root disease and simple age-related models show that tree-treatment combined with isolation drains may sometimes prove no more than an elaborate system for losing money. Forward profitability estimates are, of course, of wider relevance and may be applied also to the treatment of trees affected by Brown Bast, Black Stripe, or to damage consequent upon storms or lightning in all of which the treatment may also adversely affect yield.

TABLE 6 : POTENTIAL PROFIT FOR A SINGLE TREE WITH A THEORETICAL CONSTANT FIELD YIELD (1500 LB/YR ; 120 TREES/AC)

Yield year	1	2	3	4	5
Potential profit	312·5	300	287·5	275	262·5
Yield year	6	7	8	9	10
Potential profit	250	237·5	225	212·5	200
Yield year	11	12	13	14	15
Potential profit	187·5	175	162·5	150	137·5
Yield year	16	17	18	19	20
Potential profit	125	112·5	100	87·5	75
Yield year	21	22	23	24	25
Potential profit	62·5	50	37·5	25	12·5

TABLE 7 : POTENTIAL PROFIT FROM TABLE 6 DISCOUNTED FORWARD FROM YEAR 14 AT 10% AND 7·5% COMPOUND INTEREST ; ANNUAL AND CUMULATIVE FIGURES

	annual to final year at		cumulative to year 25		24	23
Yield year	10%	7·5%	10%	7·5%	7·5%	7·5%
14	3·983	5·248	3·983	5·248	5·642	6·065
15	4·381	5·642	8·364	10·980	11·707	12·585
16	4·819	6·065	13·183	17·495	18·227	19·594
17	5·301	6·520	18·484	23·997	25·236	27·128
18	5·831	7·009	24·315	31·006	32·770	35·138
19	6·414	7·534	30·729	38·540	40·780	43·845
20	7·056	8·010	37·785	46·550	49·487	53·205
21	7·762	8·707	45·547	55·257	58·847	63·267
22	8·538	9·360	54·085	64·887	68·909	74·084
23	9·391	10·062	63·476	74·949	79·726	(85·712)
24	10·331	10·817	73·807	85·766	91·354	
25	11·364	11·628	85·171	(97·394)		

Inoculum potential

The preceding section reviewed some aspects of factors which may affect the economic decisions of when to treat root diseases or even if to treat them at all. However, the economic decisions require prior biological decisions on what treatments — if any — to apply. It is appropriate to make three challenging generalisations ; the first and second proceed from the many to the few, the third from the few to the many and each may be prefaced by: " in, say, the first six years from planting ".

First : the number of trees that have some of their roots infected greatly exceeds the number on which infection could be detected by collar inspection — no matter how frequent or how done, by digging, baiting or mulching.

Second : The number of trees on which infection could be detected by any form or frequency of collar inspection greatly exceeds the number which would ever develop symptoms above ground.

Third : the number of trees lost from root disease will be far fewer if nothing is done than if the most elaborate methods of detection and treatment are used.

All three generalisations are concerned with the notion of inoculum potential specifically as defined by Garrett (1956) and a full understanding of this concept and its applicability is essential to the subterranean plant pathologist concerned with tree or forest crops. It is a curious fact of history that although Garrett (1959) paid handsome tribute to the early work of plantation pathologists in the East for their observations leading to the development of the concept, as propounded by him, it is nevertheless precisely in the East and in plantation pathology that it has been periodically misunderstood and its applicability too often ignored.

Garrett's 1956 definition is as follows : "Inoculum potential may be defined as the energy of growth of a parasite available for infection of a host at the surface of the host organ to be infected". He noted that the potential might be increased by increasing the number of infective units per unit area of the host and/or by increasing their nutritional status. Contact between host and pathogen is obviously necessary before infection can occur, but such contact does not mean that infection will follow — the inoculum potential may be too low. Moreover, even if infection does occur, disease — a subjective phenomenon — does not necessarily ensue nor death in turn ensue from disease. However, an individual part or parts of plant, a root or roots, might be found to be infected and judged as diseased yet the plant as a whole may still be considered healthy in that neither growth not yield is detectably affected ; it is not recognisably "sick" (Fox, 1964b). This situation, certainly in the early years of a new planting or replanting, is probably the rule rather than the exception with root diseases of rubber and of many other tree crops and many such infections are never detected.

To restate such apparently self-evident points may seem to verge on the ludicrous, but some pathologists have been peculiarly blind to them and to the truisms that infection can be stopped and the progress of disease halted not necessarily by manual or chemical interference but by host resistance alone.

Among the earliest, if not the earliest demonstration of the effect of inoculum potential were the experiments described by Bancroft (1912), elegant in the simplicity of their conception and execution. They also foreshadowed by quarter of a century Garrett's (1938) ecological classification of fungi into soil inhabitants and soil invaders based on the earlier work, cited by Garrett, of O. A. Reinking and M. M. Manns on soil Fusaria. The experiments justify a repeat quotation from an earlier paper (Fox, 1965a) because they so clearly demonstrated that *R. lignosus* is both a weak pathogen and a soil invader. Bancroft had observed that under very damp conditions the mycelium of *R. lignosus* could spread freely into heavy soils for nearly a foot from infected roots which, whilst not uncommon in pot experiments, is unusual in the field although I have seen extensive spread from old infected stumps in wet, heavy, coastal alluvial clays. He buried infected roots in soil which was kept damp and observed that "the mycelium will frequently spread through it, permeating it with white silky strands," and, "if the infected root be removed, the hyphae soon die, there being no evidence of mycelium in the soil at the end of 4 or 5 days after the root has been removed. Similarly, if the mycelium does not come into contact with any material from which it can derive nutriment it soon dies out. The growing mycelium does not appear to have any appreciable capacity for retaining its vitality when it is separated from its source of nutriment." Following these observations he placed infected roots in boxes of soil into which he allowed the mycelium to grow, removed the infected roots and then transplanted pairs of young seedlings into boxes. He commented, "I have made many attempts to infect young plants... but have never obtained any positive results." Subsequently, he used pieces of infected roots to inoculate 3-month-old seedlings and killed 13 of 18 plants so treated.

Petch (1921, 1928) confirmed from field observations that a substantial food base in the form of infected timber was necessary to establish infections. In spite of this, he still deemed it relevant to attempt to disinfect soil with lime after clearing infected patches. Napper (1932) also perpetuated the notion that mycelium in the soil was dangerous and he recommended drenching treated areas with a 2% solution of copper sulphate and exposing the soil to the "sterilising" action of the sun.

He also advised washing roots with 2% copper sulphate after removing superficial mycelium and persisted with this advice despite the experiments and observations of de Jong (1933) who showed that mycelial inocula from agar cultures were ineffective and experimentally demonstrated the need for a food base. More evidence on inoculum size was provided by Altson (1953) from simple experiments on inoculated seedlings following which he suggested that for *R. lignosus* diseased material only of more than 2 or 3 cubic inches need be eradicated. In 1954 (Fox, 1961c) I treated a number of infected trees in a manner suggested by these results ; penetrated portions of lateral roots were roughly severed (in some cases, leaving bits of infected tissue), but superficial mycelium was not removed and no fungicidal treatment was given : the roots healed. However, the "fear factor" (For, 1961b) continued to operate for some years with copper sulphate — which, in any case, was ineffective, (Fox, 1961c) — being replaced by common tar acid fungicides, the relevant authorities in the RRIM not being persuaded that the equation, fungus present = fungicide application, was irrelevant. The same factor also operated when Riggenbach (1957), most regretably, advocated the use of a liquid organo-mercurial compound and argued about the hazards of "minute pieces of mycelium" left in the soil. It is most regrettable that the same compound has recently been advocated (Momoh, 1976) for the control of *R. lignosus* on teak in Nigeria despite, in addition, the warnings given over a decade and a half ago (Fox, 1961d) on the hazards of using such chemicals in the tropics.

The other major aspect of inoculum potential has been termed the " despair factor" (Fox, 1961b) that is, the failure to appreciate the ability of even severely infected trees to recover with or even without treatment. Spontaneous recovery had long been recorded by many workers such as Reydon (1931) as well as by de Jong (1933), and I have previously summarised results from large scale field experiments where this occurred (Fox, 1965a). Table 8 summarises the interim results of RRIM experiment CI — 51 in which there were three post-planting control procedures : green, — traditional collar inspection, removal or treatment of infected trees and the tracing and removal of sources of infection ; red — as green except that detection was by foliar symptoms ; yellow — a no treatment control. When the responsibility of terminating this experiment 11 years after its inception devolved on me I found that the interim results were misleading; there were many unrecorded vacancies (especially in the yellow plots), associated with problems of supervision in an area of difficult access made more so by insurgence activity. The collars of all trees in the experiment were excavated for a final inspection to estimate the true position. A most striking feature was the very large number of trees in the yellow and red plots in which spontaneous recovery had occurred or was occurring even from very extensive lesions. Estimates of the relationships of the probable true losses at 5½ and 11 years after planting following the three post-planting treatments are expressed as percentages in Table 9. Routine recording and treatment had ceased in the seventh year and whereas the position remained virtually unchanged in the green and red plots, losses in the yellow plots continued to rise from, by then, well established centres of disease on the maturing trees.

TABLE 8 : THE EFFECTS OF 3 METHODS OF POST-PLANTING CONTROL ON NUMBER OF CASUALTIES PER AC FROM ROOT DISEASE AFTER 5½ YEARS (CI — 51)

Control system	Number of trees Lost	Treated
Red*	24	10
Green	21	1
Yellow	11	0

* for details see text

TABLE 9 : ESTIMATES OF THE PROBABLE RELATIONS BETWEEN 3 METHODS OF POST-PLANTING CONTROL AFTER 5½ AND 11 YEARS (CI — 51) EXPRESSED AS PERCENTAGE OF THE STANDARD TREATMENT

	Red*	Green	Yellow	Year
recorded	100	87	46	5½
estimate*	100	94	67	5½
estimate	100	105	90	11

* for details see text

The interim results had, however, encouraged the initiation of experiment CI — 56 of which the main results are summarised in Table 10. The green treatment was as in CI — 51 except that disease sources were eradicated only if they were within the clean weeded planting row, if in the inter-row they were merely isolated. The orange treatment was based on foliage inspection with eradication limited to the planting row alone, no digging being done among the cover plants in the inter-rows.

TABLE 10 : THE EFFECTS OF 2 METHODS OF POST-PLANTING CONTROL ON NUMBER OF CASUALTIES PER AC FROM ROOT DISEASE AFTER 6 YEARS (CI — 56)

Control system	Number of trees Lost	Number of trees Treated	Cost (man-days)
Green*	9·5	9·3	16·1
Orange	8·0	3·6	4·6

* for details see text

It was mainly these results that lead to the formulation of the far cheaper recommendations summarised by Fox (1965a) and recently restated (Rubber Research Institute of Malaya, 1974a, b). However, but for the untimely death of R. P. N. Napper in 1942, less costly recommendations might have come earlier. Altson (1950) commented that, "As a result of Napper's work, we now have a fair idea of the cost of root disease control ; but we know little of the influence of these measures on yield and, through yield, on revenue". He also noted that shortage of labour and increased wage rates made Napper's pre-planting eradication and post-planting control schemes either impracticable or economically unattractive. However, even before the last war Napper was aware that his recommendations were costly and of the need for more field experiments and he initiated twelve between 1937 and 1941. One of this series of experiments (HB1 — 41) was designed to compare his standard collar inspection with foliage inspection, both followed by tree treatment, tracing sources and eradicating them and with two further systems based on foliage detection with treatment and eradication limited to a circle of radius 10 ft or 5 ft (Treatments 1, 2, 3 and 4 respectively in Table 11).

TABLE 11 : THE EFFECTS OF 4 POST-PLANTING TREATMENTS ON THE INCIDENCE OF ROOT DISEASE, MOSTLY *Rigidoporus* (*Fomes*) *lignosus* (90%) AND *Phellinus* (*Fomes*) *noxius* (90%), 5 YEARS AFTER PLANTING

Treatment*	Trees removed	Trees treated	Unit Costs
1	114	66	292
2	81	—	82
3	86	—	57
4	80	—	37

* for details, see text

Although Altson salvaged a lot from the pre-war and war-time records (Fox, 1961b) he disregarded those of HB 1 — 41 and they came to light again only by chance in 1964 among old discarded files. The records were incomplete in that not all vacancies could be accounted for (as in CI — 51), moreover, there had been inter-row cropping for food. According to A. Newsam (personal communication) Altson found it difficult to accept the implication of these results as, in turn, did Newsam of similar indications from experiment P — 48 (Fox, 1965a) and those of CI — 51. Nevertheless, the results summarised in Table 11 permit comparisons between treatments, as do their costs which indicate potential reductions at least similar to those I estimated much later (Fox, 1964b) of 2,000,000 man-days per annum on 4,000,000 ac based on a 40 year cycle giving a mean replanting rate of 100,000 ac per year.

APPLICATION OF CONTROL MEASURES

From the foregoing one may now briefly review the basis of current RRIM recommendations together with other supporting evidence.

Pre-planting treatments

Eradication : The need for pre-planting eradication of diseased trees as potential sources of infection — part of the " fear factor " (Fox, 1961b) — has been examined in a number of experiments. Of these, the most important in Malaya were SR — 57 and J — 57, where there was manual eradication of infected trees, and ES 17 — 58 and L — 60 where complete (both experiments) and partial (L — 60) mechanical clearing were compared also with tree and stump poisoning. The results of three of these experiments, summarised in Tables 12 and 13, show that the benefits in terms of number of trees saved is small and uneconomic (Fox 1965a; Newsam, 1967). It has, however, been argued by Peries (1970) that under different topographical conditions and with rocky soils pre-planting eradication may be justified, but not if the incidence in the old stand is low (Peries, 1974). The great disadvantage of any scheme of total clearing or pre-planting eradication is that it tends towards pattern 2 in Fig. 1, the least desirable from an economic viewpoint. In general, the cheap operation of poisoning alone — tree or stump — is enough.

TABLE 12 : THE EFFECT OF PRE-PLANTING ERADICATION OF DISEASED TREES ON SUBSEQUENT LOSSES PER AC IN THE YOUNG STAND AFTER 6 YEARS

Experiment Code	Old diseased trees eradication	not eradicated
SR - 57[a]	7·5	9·4
J - 57[b]	2·5	4·1

[a]91 % *R. lignosus* [b]83 % *G. philippii*

(After A. Newsam, 1967)

TABLE 13 : THE EFFECTS OF 4 METHODS OF PRE-PLANTING CLEARING ON LOSSES PER AC FROM ROOT DISEASE AFTER 6 YEARS (L - 60)

	Losses	Unit Costs
Complete mechanical	2·4	194
Mechanical, no rooting	5·0	137
Tree poisoning	6·3	139
Stump poisoning	9·3	142

(After Newsam, 1967)

The results of experiment ES17 — 58 are summarised in Table 14 and may appear to run contrary to part of the comments above. However, they must be considered separately because they were confounded with a complex cover plant experiment in which some species of cover plant were not kept constant. The important comparison lies in the interaction between pathogen and method of clearing. The ratio of losses for felled and stump-poisoning to mechanical clean clearing for *R. lignosus* is *ca* 3 : 1 whereas that for *G. philippii* is *ca* 28 : 1. The explanation is that *R. lignosus* is not readily displaced by secondary saprophytes from timber it has infected — its fructifications may still be produced after several years on old stumps reduced almost to a friable mass. In contrast, *G. philippii* usually persists much longer, for up to two decades and occasionally more, but once its pseudosclerotial skin has been broken, it is readily displaced by a number of fungi including *Botryodiplodia theobromae*, *Trichoderma viride* and a *Penicillium* sp. Simple root ripping or deep disc ploughing prior to replanting have yet to be evaluated as treatments to aid in the control of this pathogen ; their potential as an aid to controlling *Armillariella mellea* is considered later in this paper.

TABLE 14 : NUMBER OF LOSSES AFTER 7 YEARS FOLLOWING TWO METHODS OF CLEARING THE OLD STAND

	Number of losses from *R. lignosus*	*G. philippii*	Others
Stump poisoning	115	57	4
Mechanical clearing	37	2	0

Stump treatment : Although the recommendations of the RRIM still include the application of creosote to the cut surfaces of stumps when replanting (Rubber Research Institute of Malaysia, 1974b), this advice must be treated as non-proven. Fox (1971) pointed out that the dangers from spore colonisation of stumps *at the time of replanting or in a new planting* are probably much less serious than was once thought. The conclusions from early experiments were misleading because exaggerated results were obtained on savings, (whether expressed in terms of money, or the numbers of trees treated or lost), as a result of detection by collar inspection followed by tracing and eradication supposed dangerous sources of infection many of which would have been of no consequence. Moreover, the very few spore colonised stumps which do develop into effective inocula will fall into the same category as stumps of trees infected before felling and, with a foliage inspection system, will be dealt with — or just allowed to rot — in the same way as such stumps : the cost of protection the many is not justified by the requirements and consequences of the few.

Estimates of the results following foliage inspection in CI — 56 (Fox, 1971) indicate that the costs of prophylactic stump surface treatment at the time of clearing are not recovered ; moreover, such costs fall into pattern 2 of Fig. 1. This emphasises an important principle when comparing ANY conclusions about a particular factor from different experiments : *it is essential to remember that there may be marked interactions between different steps in a control programme so that (unsuspectingly) one may not always be comparing like with like.*

Post-planting treatments

Disease detection : The practicability and efficacy of detection by foliage symptoms was clearly evident from the experiments cited above and has been further demonstrated in Sri Lanka (Peries, 1970). Detection by baiting (Declert, 1961) or by systems of mulching (Martin, 1964 ; Martin & du Plessix, 1969) advocated in West Africa are retrograde steps ; apart from obviating wounding they have all the disadvantages of collar inspection in that they detect the presence of the fungus regardless of its inoculum potential. Moreover, as previously reviewed in relation to problems of nomenclature (Fox, 1961a) they raise problems of mis-indentification with *Rigidoporus zonalis* and, in particular in West Africa, with at least one other saprophytic fungus (Fox, 1964a) which has not yet been identified (neither the perfect nor imperfect stage has been found).

Tree treatment : The current RRIM recommendations for tree treatment were foreshadowed in 1960 by Fox (1961b, c) and included in the latter paper the development of collar protective dressings. The development of the first collar protective dressings for use against *R. lignosus* were described by Fox (1965b, 1966) and the evidence for minimal treatment confirmed by John (1966) in Malaya and by Peries (1970) in Sri Lanka. Dressings against both *P. noxius* and *G. philippii* are now available and their use advocated (Rubber Research Institute of Malaya, 1974b) though evidence for their economic value has yet to be established.

Disposal of sources of infection : Hutchison (1961) summarised the results of many early experiments on methods of disposing of sources of infection which showed that isolating diseased old stumps was cheaper and, in terms of losses in the young stand, as effective as eradicating them. The results of the orange treatment of CI — 56 later showed that, in general, eradication outside the clean wooded planting rows was unnecessary. Current advice is to take action in the inter-rows only if a stump persistently causes new infections.

Disease control during maturity: If current advice is correctly implemented, modified as necessary in Africa for *A. mellea* (Fox, 1964a), then few centres of disease should remain during early maturity and these can be dealt with by spot treatment as necessary. However, it is especially important to avoid the introduction of new centres by spore colonisation of stumps of trees cut out for any reason, or of branch stubs and wounds from careless pruning or storm damage. Evidence to date suggests that although all the principle root pathogens may initiate new centres in this fashion the two most important are *G. philippii* and *P. noxius*, examples of the latter arising from branch and other wounds having been well documented by Lim (1970). Often there is no indication that such centres have arisen until adjacent yielding trees die and revenue is lost ; to avoid this, stumps of trees cut out should be poisoned if possible and their cut surfaces *must* be treated with creosote or some other suitable protectant. Pruning wounds should be properly treated and other damage wounds also, although it is usually not economic to treat mature trees damaged extensively in storms as noted previously.

General estate sanitation during maturity is also important since timber left on the ground after felling dry trees or pruning or felling those damaged in storms may both help spread existing disease centres (Fox, 1957) and initiate new ones (Fox, 1957 ; 1971), but at all times the economic limits on returns from expenditure must be borne in mind.

General

Cover plants: The effects and importance of cover plants, the adverse effects of woody plants and the beneficial effects of creepers have long been known and previously reviewed (Hutchison, 1961 ; Fox, 1961b, 1965a, 1971). Table 15 summarises the results of four field experiments (to which could be added many more) and the approximate ratio of 2 : 1 for losses in favour of creeping legumes is remarkably consistent.

TABLE 15 : NUMBER OF ROOT DISEASE CASUALTIES PER AC AVERAGED FROM 4 FIELD EXPERIMENTS

Cover plant	Number of trees Lost	 Treated
Creeping legumes	15	29
Grasses	32	78

Disease control summary: The present recommendations of Anon (1974, a,b) are based on an integrated system of control. Potential sources of infection among the old stand are initially ignored, all trees — or stumps — being poisoned to accelerate death thereby permitting rapid entry of saprophytes which minimise the extension of existing centres of infection as would normally occur as root systems otherwise slowly become moribund. Thereafter, the presence of the pathogen is ignored unless its inoculum potential is so high as to produce evident disease on the young stand. Maximum use is made of what has long been known of the beneficial effects of creeping leguminous cover plants in promoting various aspects of biological control — antagonism, inoculum wastage and dispersal — and of maximising use of the natural host resistance enhanced — only where necessary — by precise placement of fungicides in special durable formulations.

EPIDEMIOLOGY AND THE DYNAMICS OF CONTROL PROCEDURES

The three major root disease pathogens of the East have in common the ectotrophic habit, growth on the outside of the root (either in strands or as an investment) preceding that of penetration within the root. In the case of *R. lignosus* internal penetration of young roots is entirely dependent on its extensive external growth which may ramify several metres ahead of penetration ; that of *P. noxius* and *G. philippii* is not as dependent on their much more limited external growth — to be measured in centimetres — but is sufficiently so as to affect the rate of internal pathogenesis. (The last two fungi may also spread upwards from the roots to cause butt rots above ground without the aid of external growth and *P. noxius* by spore colonisation of wounds may cause branch rots also as noted earlier). Within the soil, the ectotrophic growth is open to attack by the soil microflora and fauna and damage to it must constantly be repaired for translocation to continue to support pathogenesis which is wholly or partly dependent on a continuing supply of food from further back, either from part of the root currently invaded and/or from the initial inoculum.

The implications of this dependency are interesting. Consider a root of radius R surrounded by a fungal investment of thickness T ; the potential for growth, pathogenesis, or survival of the fungus, denoted by S, will be related to the ratio of the food (wood) volume to fungus volume and elementary arithmetic gives the expression shown in Table 16. Clearly, as the root gets smaller so the situation militates against pathogenesis because the surface area of the fungus open to attack by soil micro-organisms is proportionately greater in relation to the immediate root volume and pathogenesis must become more dependent on translocation from the original food base, a process proportionately more susceptible to disruption.

TABLE 16

$$\frac{\text{FOOD (WOOD) VOLUME}}{\text{FUNGUS VOLUME}} \simeq \frac{R^2}{T\,(2R + T)} = S$$

When T	=	R	$\frac{R}{2}$	$\frac{R}{4}$	$\frac{R}{10}$		$\frac{R}{20}$		
then S	=	$\frac{1}{3}$	$\frac{4}{5}$	$\frac{16}{9}$	$\frac{100}{21}$	$(\frac{5}{1})$	$\frac{400}{41}$	$(\frac{10}{1})$	

As R increases relative to T then S $\simeq \frac{R}{2}$

Theoretical considerations apart, the concept of interactions between food base and whole soil antagonism (*i.e.* not related to any specific organism or group of organisms) affecting growth needs to be demonstrated and the nature of ' growth ' more clearly defined. Two aspects of the importance of the food base in enabling the fungus to advance in the face of whole soil antagonism are shown for *R. lignosus* in Tables 17 and 18. In the first, the volume of the food base not only affects the initial growth rate into the soil but also the number of strands produced. In the second, although the varying concentrations in the food base affect only the initiation and not the later growth rate, the number of strands is markedly affected. The effects of varying volume and concentration are highlighted by Figs. 4 and 5.

TABLE 17 : EFFECT OF VARYING THE VOLUME (ML) OF THE FOOD BASE (2% MALT EXTRACT AGAR) ON THE GROWTH (CM) OF *Rigidoporus* (*Fomes*) *lignosus* INTO FIELD SOIL *In Vitro*

Volume of food base	5	10	15	20	40
Growth at day 4	$2{\cdot}4^{*}_{a}$	$3{\cdot}35_{ab}$	$4{\cdot}10_{b}$	$5{\cdot}25_{c}$	$5{\cdot}75_{c}$
Number of strands	19_{a}	27_{ab}	32_{bc}	32_{bc}	39_{c}

* values with the same subscript do not differ significantly (5%)

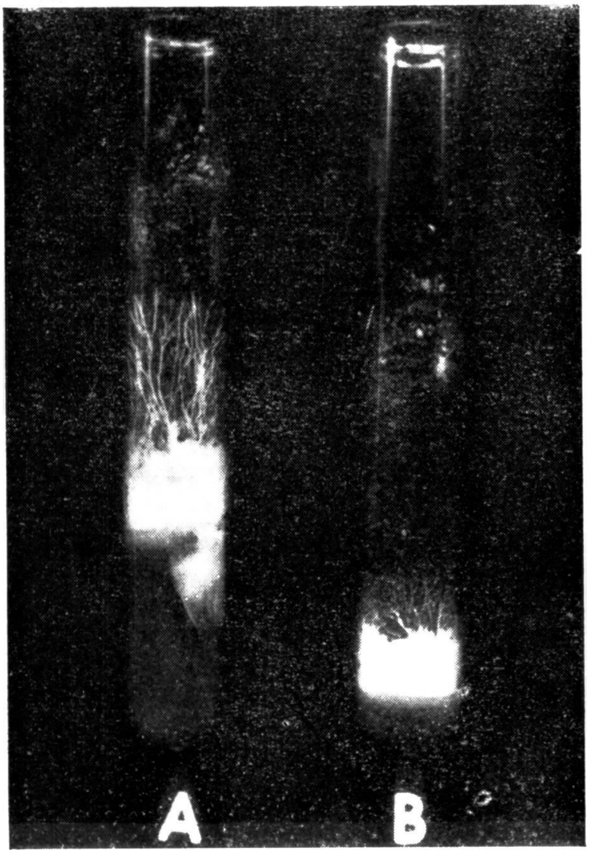

Fig. 4 The effect of varying the volume (A 40 ml, B 10 ml) of the food base (2% malt extract agar) on the growth of *R. lignosus* through field soil (see also Table 17).

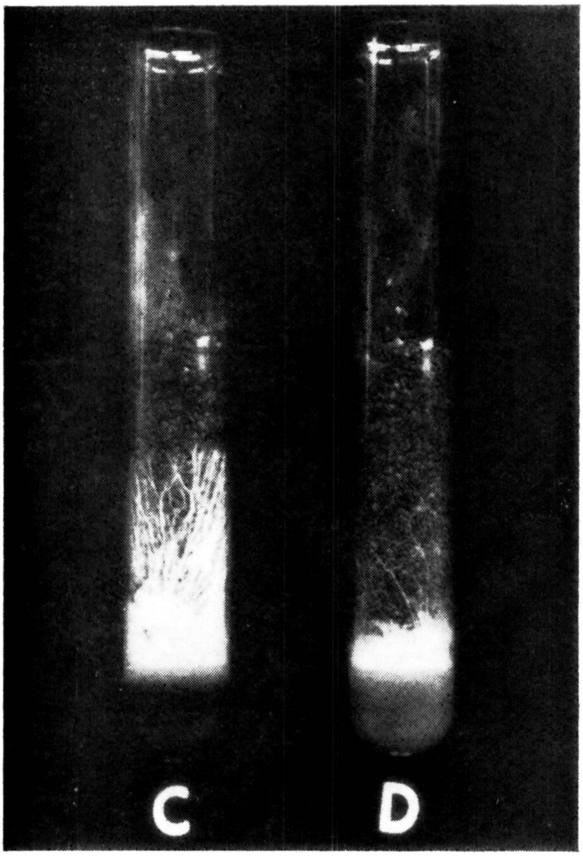

Fig. 5 As fig. 4 but varying the concentration of malt extract (C 4%, 1%) in a fixed volume (15 ml) of food base (see also Table 18).

TABLE 18 : EFFECT OF VARYING THE CONCENTRATION (% MALT EXTRACT) OF THE FOOD BASE (15 ML) ON THE GROWTH OF *Rigidoporus* (*Fomes*) *lignosus* INTO FIELD SOIL *In Vitro*

Concentration in food base	0	0·5	1·0	2·0	4·0	8·0
Growth at day 4	·6*$_{a}$	2·0$_{b}$	4·6$_{cd}$	4·1$_{c}$	5·1$_{d}$	4·7$_{d}$
Growth 4th-7th day	·5$_{a}$	2·5$_{b}$	2·3$_{b}$	3·7$_{b}$	3·6$_{b}$	3·8$_{b}$
Number of strands	0	4·8$_{a}$	21$_{b}$	32$_{c}$	39$_{c}$	50$_{d}$

* values with the same subscript do not differ significantly (5%)

In previously drew analogies (Fox, 1965a) between the root-rhizosphere phenomenon on the one hand and rhizomorph (or investment or strand) — hyphosphere on the other. Both the plant root and fungus structures influence the microflora and fauna in the soil near them and in this manner both eventually tend to bring about their own destruction. The basic dynamics of the process are illustrated in Figs. 6a, 6b, and 6c, which are self explanatory. To extend a military analogy used before (Fox, 1965a), the lines of communication become over extended and so weakened by attack that there is a logistics failure ; the front line advance outruns its supplies, fails and may be cut off and destroyed. These illustrations give pictorial emphasis to the earlier statement that many infections arise that will never be detected. Moreover, a small extension of growth in 6c would give a positive result either at a collar inspection or by using a mulching or baiting technique (with subsequent treatment costs) when not only might the inoculum potential be inconsequentially low but secondary invaders may well, or have already severed the link between the original source of infection by rotting away the old or new roots somewhere between the young tree and the old stump.

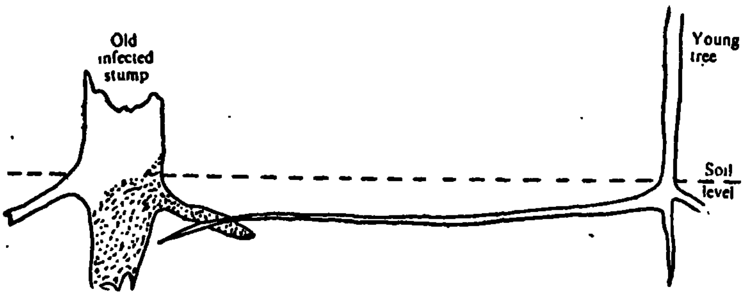

Fig. 6 (a) contact between new root and source of infection

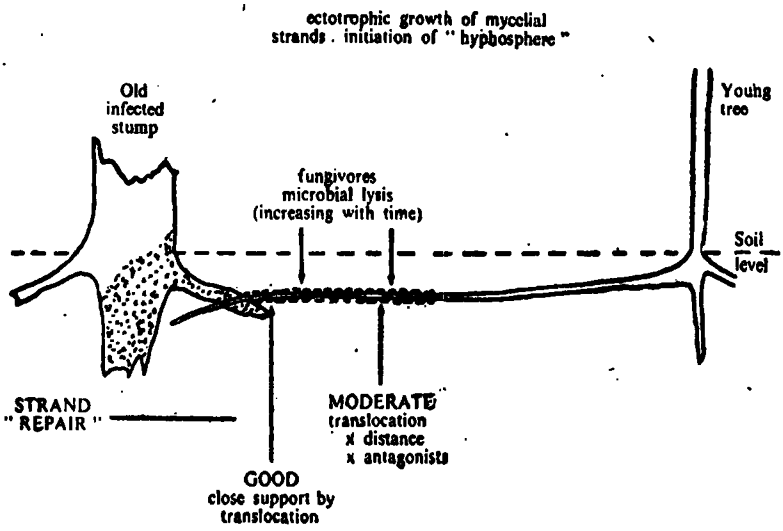

Fig. 6 (b) early stages of interaction

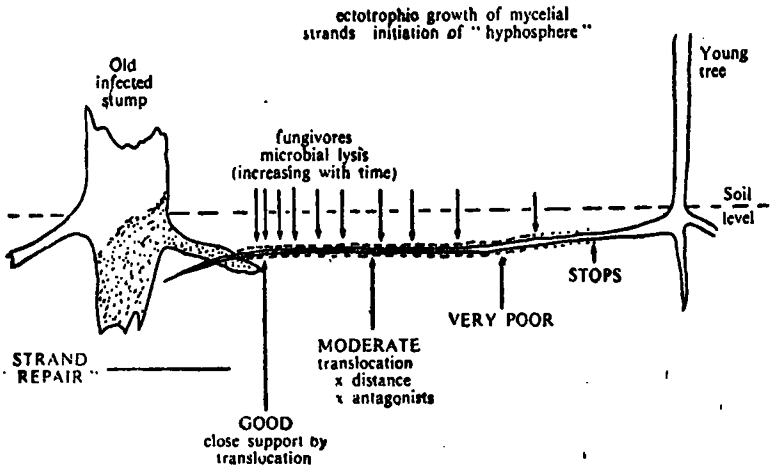

Fig. 6 (c) growth halts, pathogenesis fails

The effect of creeping cover plants in reducing the incidence of disease has already been alluded to and the mechanisms discussed in detail by Fox (1965a, 1971) and an account of the succession of insects and fungi in stumps and of the enhancement of their decay given by Newsam, John & Sripathi Rao (1967). The effects of cover plants may be summarised by the three d's — decoy, decay and deter. The first of these is simply explained and is a forerunner of the second. In brief, cover plants provide physical conditions that encourage, particularly, *R. lignosus* to grow before there are many rubber roots to infect and at a time when those present are too small to successfully propagate infection. Some roots of cover plants may themselves be infected but they, in turn, dissipate inoculum potential in that single, large and potentially dangerous inocula become harmless by depleting their nutrient status when they infect the small cover plant roots whose individual inoculum potential in turn is too low to be of future consequence.

The results presented in Table 19 show that moderate moisture levels in a model system in soil promote faster linear growth than higher levels, but the mass growth at higher saturation capacities is greater because of the density of growth. In the litter layer temperatures are higher than in the soil and some effects of temperature are given in Table 20. The combination of temperature and moisture in the litter may combine to promote high density and rapid growth enhancing the decoy effect because not only are there no roots to infect but the activity of antagonists is notably high. However, this beneficial effect of promoting futile growth may be seriously reversed by its becoming purposeful if strip or circle weeding is neglected so that the cover plants grow across the planting row to envelop the base of young trees creating a moist chamber effect. The pathogens, especially *R. lignosus*, may then quickly encircle the bole of the tree, unhindered mechanically or biotically by the soil, and effectively ring-bark it causing rapid death.

TABLE 19 : EFFECT OF VARYING THE MOISTURE CONTENT OF SOIL ON THE GROWTH (CM, 14 DAYS) OF *Rigidoporus (Fomes) lignosus*

	Percent soil saturation capacity							
	91	86	81	66	52	38	24	12
Growth, cm	$11{\cdot}4^{*}_{a}$	$11{\cdot}4_{a}$	$11{\cdot}4_{a}$	$12{\cdot}0_{a}$	$11{\cdot}6_{a}$	$13{\cdot}8_{b}$	$13{\cdot}3_{b}$	$13{\cdot}1_{b}$
Number of strands	$33{\cdot}5_{a}$	$34{\cdot}5_{a}$	$34{\cdot}5_{a}$	$24{\cdot}0_{b}$	$12{\cdot}5_{c}$	$12{\cdot}5_{c}$	$6{\cdot}0_{c}$	$5{\cdot}5_{c}$
Strands >1 mm	$14{\cdot}0_{a}$	$12{\cdot}0_{ab}$	$10{\cdot}0_{abc}$	$7{\cdot}5_{abcd}$	$5{\cdot}0_{bcd}$	$4{\cdot}5_{cd}$	$2{\cdot}0_{d}$	$1{\cdot}5_{d}$

* values with the same subscript do not differ significantly (5%)

(After Fox, 1971)

TABLE 20 : THE EFFECTS OF TEMPERATURE ON THE GROWTH OF *Rigidoporus (Fomes) lignosus In Vitro*

Temperature °C	Growth per day mm	mg	density mg/mm
32	11	16	20
27	10	8	10
21	7	5	8
16	not measurable		

(After Fox, 1971)

Leguminous covers also affect the nitrogen status of the soil (Anon, 1972) and it is reasonable to suppose that the additional nitrogen enhances both decay and decoy effects. The nitrogen content of most timber, with C : N ratios of 300 : 1 to 1000 : 1 or more, is a limiting factor in its rate of decay ; most investigations have shown that the optimum C : N ratios for fungal growth lie in the range 8 : 1 to 15 : 1 and the marked affects of different ratios on the growth of *R. lignosus* are shown in Table 21.

TABLE 21 : EFFECT OF C : N RATIO OF FOOD BASE ON GROWTH (CM) OF *Rigidoporus* (*Fomes*) *lignosus* INTO ACID WASHED QUARTZ SAND

C : N ratio	5 : 1	10 : 1	20 : 1	40 : 1	80 : 1	160 : 1	320 : 1
Growth at day 4	$5{\cdot}0^{*}_{a}$	$5{\cdot}0_{a}$	$5{\cdot}1_{a}$	$4{\cdot}6_{b}$	$3{\cdot}4_{c}$	$3{\cdot}1_{c}$	$1{\cdot}6_{d}$
Growth 4th — 7th day	$3{\cdot}3_{a}$	$3{\cdot}3_{a}$	$3{\cdot}0_{b}$	$2{\cdot}7_{c}$	$2{\cdot}7_{d}$	$1{\cdot}9_{e}$	$0{\cdot}9_{f}$
Number of strands	55_{a}	52_{a}	44_{b}	36_{c}	23_{d}	11_{e}	$0{\cdot}3_{f}$

*values with the same subscript do not differ significantly (5%)

(After Fox, 1971)

Perhaps the most striking decoy effect to the casual observer, and which is symptomatic of decay, is the abundant proliferation of fructifications on stumps beneath the cover plants. In this situation their propagative function is virtually zero and they serve only to provide both table and food for many mycophagaous organisms — both microfloral and faunal.

Although cover plants encourage fungal growth in the litter layer they also are a cause of increased inhibition within soil — the ' deter ' effect. Fox (1965a) illustrated enhanced growth inhibition, mycelial lysis and inoculum destruction by soil taken from beneath two (*Pueraria phaseoloides* and *Centrosema pubescens*) of the three (with *Calopogonium mucunoides*) creeping legumes, commonly planted as a mixed cover, compared to ' bare ' clean-weeded soil. The technique used detected differences in growth inhibition between ' bare ' and ' cover ' soil five months in one field and ten months in another after first planting the mixed cover. Typical effects are illustrated in Figs. 7 and 8. Growth of *R. lignosus* on soil extract agars showed the cover soil supported better growth suggesting that the effects observed were not associated with soil nutrient levels. Lysis of mycelium usually commenced four weeks after placing inocula on either type of soil but it was noticeably faster on the cover soil. Sieving cover soil to remove plant debris noticeably diminished its inhibitory effect suggesting that the debris served as a substratum for continuous production of fungitoxic compounds.

The observations on mycelial growth in relation to nutrient levels and plant debris contrasts with the general phenomenon of mycostasis of spores where inhibition is annulled by added nutrients. This contrast was further examined in experiments using the two soil types in which unautoclaved soil was amended with 1% glucose in the water agar or not and autoclaved soil either similarly amended or not or re-inoculated with one tenth of its volume of non-sterile soil. There was no growth inhibition on either type of soil following autoclaving alone and with the added glucose growth was enhanced ; small differences in growth rate on the two soils probably reflected the higher nutrient status of the cover soil. With no autoclaving, adding glucose enhanced inhibition on both soils increasing that on the bare soil to a level approximately equivalent to that of the unamended cover soil. Both

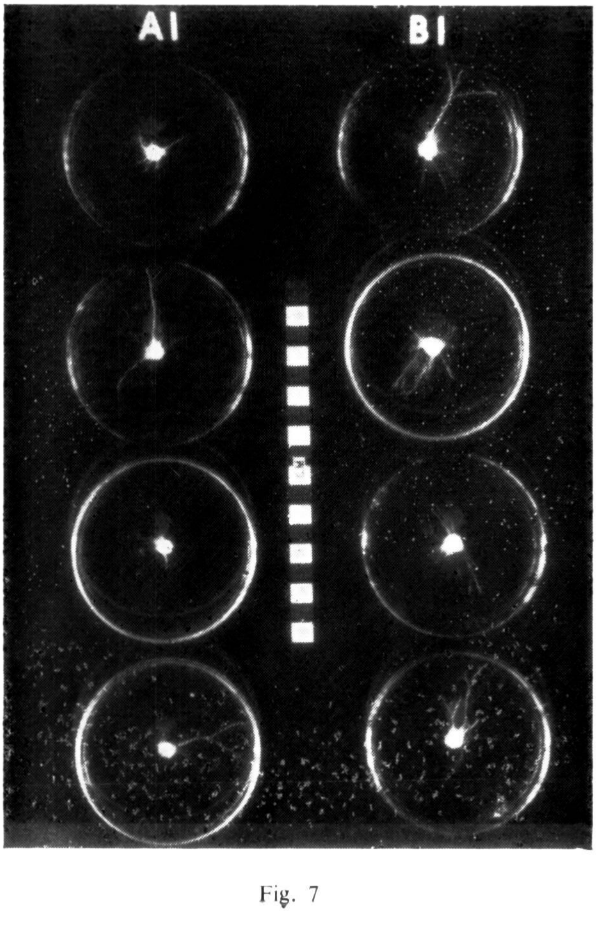

Fig. 7

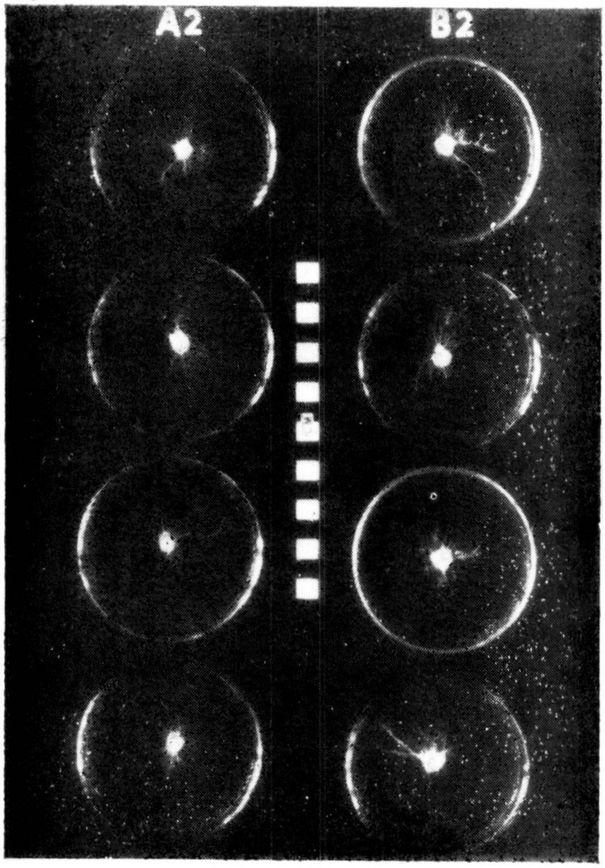

Fig. 8

Figs. 7 and 8 Inocula of *R. lignosus* (2% malt extract agar) on soil surfaced with water agar from a clean weeded inter-row (Fig. 7 B1; Fig. B2) and on adjacent soil from beneath cover plants (Fig. 7 A1; Fig. 8 A2); scale marks = 1 cm.

types of soil when autoclaved and reinoculated showed more inhibition than the unautoclaved glucose amended soils possibly indicating the extent to which nutrients were released by autoclaving. Additional experiments involving various combinations of autoclaving, propylene oxide sterilisation, amending, and alcohol extraction all supported the concept of active growth inhibition affected by soil nutrient status. When plant debris was sieved from cover soil, mixed and then incubated for seven days with an appropriate amount of bare soil, subsequent growth inhibition was similar to that of the original cover soil.

The experimental techniques used in these investigations were open to the criticism that the water agar used to produce a smooth surface on the soil or the disc of agar inoculum in contact with soil might provide sufficient nutrients to generate antagonism thus giving misleading results. Some experiments were therefore repeated with or without water agar and with or without a cover slip beneath the inoculum disc to prevent diffusion of nutrients into the soil. Essentially similar results were obtained. A similar experiment was conducted during this Conference using cover and bare soils sampled at 2 and 22 cm depths. Inocula of *R. lignosus* growing on 2% malt extract agar were placed on cover slips on the smoothed surfaces of soil samples in petri dishes and scored after 9 days growth. Scoring was done comparatively on a scale of 1 = 0 — 1 (no growth to a trace of growth) up to 5 = 4 — 5 (5 was the score for the maximum amount of growth from any inoculum of all samples and replicates). The results shown in Table 22 may be interpreted by postulating that growth was greatest at 2 cm in bare soil where antagonism might be low because of high temperature effects in reducing soil microbial populations and at 22 cm beneath covers where nutrients might be high and soil populations intermediate. The low growth at 2 cm beneath covers reflects the expected interaction with high nutrients and high microbiol populations and that at 22 cm beneath bare soil a combination of low nutrients and intermediate populations. The speculative nature of these explanations clearly favour more experimentation rather than any particular hypothesis.

TABLE 22 : MEAN SCORES (MAX. 5) FOR *R. lignosus* GROWING FROM MALT AGAR INOCULA ON TWO TYPES OF SOIL

Soil	Beneath cover		Clean weeded	
Depth (cm).	2	22	2	22
Score	$1{\cdot}40_a$	$2{\cdot}65_b$	$2{\cdot}55_a$	$1{\cdot}80_b$

Scores with the same subscript do not differ significantly (5%)

More detail has been obtained, but not yet quantitatively, of enhanced antagonism beneath cover plants using a simple buried slide technique. If an infected root is uncovered and the end of a microscope slide pressed at right angles into a strand to sever it, partly or wholly, the mycelium subsequently will grow over the surface of the slide from its natural food base after the root is again covered. Details of the hyphosphere phenomenon may then readily be followed and observations made on wall erosion, lysis and nematode attack.

The effects of antagonism on growth and subsequently on lysis have also been examined using the growth tube technique (Fox, 1965a) by growing *R. lignosus* into acid washed quartz sand which had been wetted with sterilised soil extract inoculated or not with 1 g/100 ml of untreated soil. Cell wall erosion was not visually detected in this system although the strands and individual hyphae became dotted and encrusted with colonies of bacteria. Growth of strands was evidently sparser in the inoculated than in the uninoculated tubes. Qualitative gravimetric tests, to indicate the binding effect of the mycelium on the coherence of the sand columns, suggested that strands in the inoculated tubes were also more fragile.

The phenomenon of leachates from a plant affecting the growth of other plants (allelopathy) has been known for well over two centuries. Recently, study of the details involved, and of plant-plant-insect-microorganism interactions has attracted much attention and relevant review papers include those of Lohdi (1976) and the conference compilation by Anon (1971), that of the Oregon State University School of Forestry edited by Berg (1972) and that of the Western Regional Project W – 38 on the nature of the influence of crops residues on fungus-induced root diseases by Cooke & Watson (1969). Wong (1964) detected another effect of cover plants when he showed that extracts of *Mikania cordata*, a tropical ' weed ' which sometimes is allowed to develop as a natural cover, inhibited the growth of rubber plants in pot experiments as it did in field experiments. Records from the field had also shown that it was associated with decreased levels of white root disease. We showed that extracts from it inhibited the growth *in vitro* of *R. lignosus* (Wong, 1964). Recently, Wu *et al.* (1975) have shown that *C. pubescens* also produces phenolics that inhibit plant growth.

I have already remarked on the potential for limited mechanical clearing to decrease the incidence of *G. philippii* and there appears to be similar potential in Africa for use against *A. mellea*. Observations from inoculation experiments indicated that infected roots broken and buried in the dry season quickly developed pseudo-sclerotial protection conferring a high survival value. In a simple experiment roots were buried at intervals throughout the year in either a well or poorly drained site. At each date of burial at each site half of the roots were buried in unprotected soil and the other half buried in a plot sheltered by a miniature roof of corrugated iron. The results are summarised in Table 23 and show a striking difference in survival resulting from the effects of high moisture inhibiting pseudo-sclerotial formation. The detailed results at the dry site were of especial interest. The roots buried earlier, towards the end of the dry season had well developed pseudo-sclerotial protection and had survived better than those buried as much as 6 months later, near the end of the wet season, whose pseudo-sclerotial development was relatively poor. Thus the survival slope went the "wrong" way. The conclusion is that root ripping might well be beneficial in the control of *A. mellea* in this region but it will be counter-productive if it is done during the dry season.

TABLE 23 : EFFECT OF SOIL MOISTURE ON DECAY SCORE OF BURIED ROOT SEGMENTS INFECTED WITH *Armillariella mellea*

Dry (grass slope)		Wet (swamp edge)	
Covered	Bare	Covered	Bare
$3{\cdot}76_a$	$2{\cdot}13_b$	$\cdot 37_c$	$\cdot 45_c$

values with the same subscript do not differ significantly (5%) ; 0 = disintegrated, .6 (maximum) = little changed since buried.

CONCLUSIONS

There is little doubt that the greatest advances in reducing the costs of root disease control whilst keeping losses at an acceptable level have already been made. These changes came about in part from an empirical programme of root disease experiments advancing stepwise and in part from accompanying advances in our understanding of the complex interactions of host, pathogens, and ground cover plants.

The current advice on pre-planting eradication and treatment, and on post-planting disease detection and disposal of sources of infection, is based on what has been a traditional duration of immaturity of some 5 to 7 years. These years, in some ways, have in them an element of crop rotation (Fox, 1965a) commencing with the establishment of the sown cover and progressively decreasing as the host root system and canopy extend and the cover is gradually shaded out over some 4 — 6 years. These years are the planter's and the pathologist's period of grace' ; the sources of infection waste away, host resistance increases and because the number of trees planted exceeds the number finally required some losses are acceptable. The system of detection and control is, in effect, based on roguing over a period when individual trees have no real, only hypothetical values for it is only after thinning to select the final stand that individual trees represent a precise fraction of capital expended. Moreover, they also then represent a precise proportion of future income : after thinning, all losses in the economic sense are real losses.

I would suggest that there are two areas which must affect the future direction of research on root diseases.

First, despite all that has been written and done in the last twenty years we still lack sufficient depth in understanding the biological processes involved in our recommendations nor have we fully exploited the knowledge that we have by adequately testing it in the field. Thus we do not have adequate evidence that the system of integrated control which we recommend, and which so successfully deals with *R. lignosus*, will also cope with the much slower moving *G. philippii* and *A. mellea*. The last named pathogen has not yet been the subject of adequate field experiments in the lowland tropical rain forest zone. *G. philippii* was the dominant pathogen in experiment J — 57 but it may be argued that recording stopped too soon to detect late spread from residual sources of infection. We are still only guessing in our explanations of why the incidence of root disease, when mainly caused by *R. lignosus*, is reduced so much more by tree than by stump poisoning. Empirically we have not done enough to examine the potential advantages of delayed felling, first tried in CI — 56, which may give some of the advantages of tree poisoning whilst lessening the danger from falling trees and branches of injury to labour and damage to young trees. We need to know more about spore colonisation of the surface of the stump and of the corresponding cut surface of the felled trunk, whether these are the only sites of spore infection and how and under what circumstances felled timber can contribute to the above-ground spread of disease. Whilst it is obvious that the rate of decay of sources of infection is of prime importance we know little about fungal succession *per se* or of the effects on it of tree or stump poisoning or of different cover plants. Such information as we have relates to the succession of fructifications rather than to information on actual succession that could be gained with an increment borer followed by plating and then using a system of identification based on mycelial characters as pioneered by Mildred K. Nobles in Canada.

Our understanding of the effects of cover plants is crude and based largely on unquantified observations which, however persuasive, tend more to conceal than reveal the extent to which our knowledge is based on assumptions about the effects of moisture and temperature. The enhancement of antagonism in the soil by cover plants, noted in this paper, is readily demonstrated, but ' whole soil antagonism ' is a blunt weapon with which to investigate differences between cover plants or soil/site/cover interactions. The sand-growth tube technique might well be refined by inoculating not with whole soil but with groups of antagonists selected by, perhaps, variations of the nutritional groupings so elegantly demonstrated and investigated by A. G. Lochhead and his co-workers in Canada over some two decades up to 1960. A group selective medium, possibly based on chitin or fungal cell walls as the main source of carbon, would appear to be a promising starting point.

Another aspect of the action of cover plants, which surely must be considered as having received only the most superficial attention, is that of leaf, stem and root leachates as exemplified by those of *M. cordata* and *C. pubescens.*

The importance of the high proportion of the total rubber production contributed by smallholders or small farmers has been stressed time and again at this and other Conferences. However, we can do little more than make statements of the apparently obvious, and hope that there is some truth in them, about the effects on root disease incidence of different food and catch crops, and their associated agronomic practices, when these are grown in the inter-rows.

We know that the feeding roots of *Hevea* affect the physical and possibly the biological destruction of infected timber not only below ground but even above it in stumps, surface roots and beneath the bark of trees with butt rots. However, we know little of the mechanisms of this action nor why these roots are apparently immune to infection by any of the major pathogens. Here may lie one possible key to understanding a fascinating story the beginning of which has been revealed at this Conference by G. W. Liyanage and his colleagues of families of selfed clonal seedlings differing in resistance to *R. lignosus.*

At the beginning of this Section I referred to ' a traditional duration of immaturity of some 5 to 7 years ' ; this brings me to the second area to which work must be directed. Many research workers and plantation managers have sought to shorten the period of immaturity and correspondingly shorten the period of negative cash flow. Some benefits have been obtained in the past from better budding techniques, improvements in planting material, agronomic practices, and clone vigour and by earlier opening. During this Conference we have heard an able and well illustrated exposition by E. Pushparajah of his and his colleagues work at the RRIM on advanced planting material the use of which may well halve the period of immaturity. On the one hand it will greatly improve the cash flow position, on the other hand the pathologist and plantation manager alike will lose the best of their " period of grace ". Such material will proportionately far more rapidly exploit the soil than conventional planting material and hence more rapidly contact potential disease sources. Moreover, its high cost must progressively bring about ' planting to a stand ', thus lessening the potential for detection based on roguing trees that have only an hypothetical value.

The use of advanced planting material clearly requires a complete reassessment of the value of pre-planting disease detection in the old stand (for example, by mulching) and of its eradication and of methods of disposal of the old stand, and of post-planting disease detection and disposal of sources of infection. Tree examination, treatment and prophylactic application of collar protectants may well need to be extended to trees in adjacent rows as well as to adjacent trees within a row.

Not withstanding the title of this paper, I have made no specific references to other perennial plantation crops. To do so would have greatly extended an already lengthy paper. However, from my knowledge of other perennial crops — and not only in the tropics — I hope that some of the principles which I have emphasised have emerged sufficiently clearly to shed some light on old practices and to suggest new ones.

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