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ORIGINS OF DECAY IN LIVING DECIDUOUS TREES: THE ROLE OF MOISTURE CONTENT AND A RE-APPRAISAL OF THE EXPANDED CONCEPT OF TREE DECAY

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SUMMARY

It is argued that the development of decay in living hardwoods can best be explained in terms of the unsuitability of functional sapwood for mycelial establishment owing to its high moisture content and lack of easily assimilable nutrients other than within living cells. Decay occurs when these limitations are removed by any mechanisms which prevent or interfere with the normal functioning of sapwood. Recent concepts of decay in living trees have implied an active host defence against infection. This view is discussed against the alternative that non-specific mechanisms which maintain sapwood function will, by their very nature, prevent establishment of mycelium of decay and stain fungi. The significance of mixed microbial communities in the development of decay is discussed, particularly in relation to the supposed requirement for specific sequences to overcome host defences.

INTRODUCTION

The foundations for tree decay studies were laid over a century ago by Robert Hartig (Merrill, Lambert and Liese, 1975) and gave rise to the classical concept that most decay in standing trees originates by saptrophic growth in dead heartwood. According to this concept, provision of wounds large enough to expose the heartwood allows entry of heartrot fungi which directly results in decay (Boyce, 1961; Merrill and Shigo, 1979; Shigo, 1979; Shortle, 1979).

Over the last 20 years numerous studies of discoloration and decay associated with wounds, particularly of living hardwood trees, have been made in North America, and these have frequently been reviewed (Shigo, 1965, 1966, 1967, 1979; Shigo and Hillis, 1973; Shigo and Marx, 1977; Shortle, 1979; Mercer, 1982). These studies have shown that colonization can occur other than via heartwood by microorganisms including bacteria, non-hymenomycetes and hymenomycetes. As a result, the classical heartrot concept has been challenged and an expanded concept of tree decay proposed in its place (Shigo, 1970, 1976, 1979; Shigo and Hillis, 1973; Shigo and Marx, 1977; Merrill and Shigo, 1979). This expanded concept is based on four major tenets: (i) the decay process is initiated via wounds exposing sapwood and/or heartwood; (ii) activity of fungi causing discoloration and decay is localized within compartments; (iii) localization of decay and discoloration is due to an active response by the tree to wounding and invasion (the compartmentalization of decay in trees, or CODIT concept); (iv) many

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different organisms contribute, in an ordered sequence or succession culminating in hymenomycetes, to the development of decay.

These ideas are becoming widely accepted as fact and there is urgent need for their careful re-appraisal (Cooke and Rayner, 1984). The approach adopted here will be first to consider factors affecting the ability of fungi to grow in wood in relation to conditions within standing trees. Within this context, and using our own observations with hardwoods for illustration, we will then evaluate each of the four major tenets above. We hope to show that alternative, but seemingly neglected, explanations of decay processes in standing trees are available. These are based on long established principles and concepts, and in some respects account more readily for observed patterns than does the expanded concept.

FACTORS AFFECTING GROWTH OF FUNGI IN WOOD

Of the many factors which affect fungal growth in wood, those considered below are regarded as salient within the context of conditions in the standing tree.

Availability of organic nutrients and their distribution

As heterotrophs, all fungi require a supply of organic nutrients to supply their growth needs. Readily accessible, assimilable substrates such as soluble sugars, lipids, peptides and other primary metabolites, may well be important in early establishment of mycelia from spores or similar propagules as well as being the only available carbon source for non-decay microorganisms. These substrates occur in relatively small amounts ($< 10\%$ by dry weight) and are located almost exclusively within parenchyma, which, in functional sapwood, will be living. Otherwise, the dominant available carbon sources are the relatively refractory major structural polymeric components of woody cell walls, that is cellulose, lignin and hemicelluloses. Within the standing tree much will therefore depend on the accessibility of these substrates to fungi and the extracellular enzymes necessary to break down the structural polymers. Partly this will reflect the influence of those other environmental conditions discussed below, but it will also be dictated by anatomical considerations. Access to nutrients is provided via an interconnected system of portals (lumina) surrounded by solid, relatively impenetrable cell walls (Levy, 1975, 1982). In purely physical terms, the orientation, maintenance of access to, and size of these portals will critically affect microbial and fungal distribution (see Cooke and Rayner, 1984).

Extractives

Besides the organic substrates mentioned above, wood contains a wide variety of extractives, many of which are phenolic, and some of which have fungitoxic or fungistatic properties and are believed to confer resistance to decay (Hillis, 1962; Scheffer and Cowling, 1966). These are particularly characteristic of heartwood, but in sapwood they accumulate in discoloured tissues associated with wounding, or around decay columns in living trees, whence in many instances they render the affected wood less susceptible to degradation (Hart, 1964; Hart and Johnson, 1970; Hillis, 1977).

Moisture content and aeration

High moisture content and associated restriction of aeration are well-known factors limiting the activity of mycelial fungi in felled or fallen timber (Boddy,

1980, 1983). That an excessive water content can effectively prevent decay is evident from the long-term survival of submerged timbers and from procedures such as the ponding of Douglas fir baulks to prevent deterioration during storage (Cartwright and Findlay, 1958). In living stems and branches water content is highest where living cells and actively conducting tissues are present.

Aeration is inevitably greatly restricted under these conditions, and even where a substantial gaseous phase exists, as in heartwood, this is normally enriched in carbon dioxide and impoverished in oxygen. Carbon dioxide levels of 10 to 20 % have commonly been recorded (Chase, 1934; Hintikka, 1982) and more sophisticated techniques suggest that they may by higher still – approaching 100 % in living stems of *Acacia* (Carrodus and Triffett, 1975). Even under experimental conditions where cultures are grown in contact with a continuous gaseous phase unbroken by liquid water, 10 to 20 % carbon dioxide greatly restricts or prevents mycelial growth of the majority of fungi, although wood-rotting Basidiomycotina are generally tolerant of such conditions (Hintikka and Korhonen, 1970; Schánĕl, 1976), and others may show yeast-like growth (Romano, 1966). Growth at carbon dioxide levels approaching 100 % is possible for certain wood-decay species such as *Peniophora quercina* (Fr.) Cooke, *Phlebia rufa* (Fr.) M.P. Christ., *Stereum gausapatum* Fr. and *Vuilleminia comedens* (Nees ex Fr.) Maire which invade attached oak branches (Boddy and Rayner, 1983a; Rawles, Boddy and Rayner, unpublished data), but is much reduced and the hyphae have an unusual morphology, often with bud-like branching patterns.

Evaluation of Tenets of the Expanded Concept

Initiation of decay via wounds

It is probably true that initial access of fungi which will ultimately bring about decay or discoloration is dependent on entry via discontinuities in the outer covering of the tree. Both the heartrot and expanded concepts are centred on the role of major stem wounds, not only as entry points, but as locations around which establishment and spread of decay and discoloration is focused. However, this does not explain patterns of decay in, for example, attached branches, or in trunks predisposed by abiotic or biotic environmental stresses to disease and decay. Characteristic of such decay is the development, within a short time, of extensive single individuals of Basidiomycotina or xylariaceous Ascomycotina occupying considerable volumes of wood and identified by somatic incompatibility tests in culture. These individuals are often more extensive than can readily be accounted for in terms of mycelial spread from an infection court – evidence of which, in the form of a major wound, is also normally lacking (Adams, 1982; Boddy and Rayner, 1982, 1983a, b; Cooke and Rayner, 1984).

The origins of these extensive individuals therefore require consideration, especially in connection with the fact that if mycelial spread from a substantial infection court does not occur, alternative modes of establishment may be operating. Besides major wounds, minor discontinuities through which entry may be effected include leaf and twig scars, sapsucker and woodpecker wounds, lenticels and minor fissures, dead or abscising twigs. These have received little attention as potential infection courts, although *Echinodontium tinctorium* E. & E. has been reported to establish heartrot in Western Hemlock *Tsuga heterophylla* (Raf.) Sarg. via living branch stubs only a few millimetres in diameter (Etheridge and Craig, 1976). Although decay-causing Basidiomycotina and Ascomycotina can

be isolated from attached twigs (Boddy and Rayner, unpublished data), the establishment must be a rare event judging from the limited number of individuals present and entry sites remain obscure.

Evidently, many of the fungi forming such extensive individuals are ecologically specialized, either showing marked host specificity, as with *Piptoporus betulinus* (Bull. ex Fr.) Karst. on birch (*Betula* spp.), *Hypoxylon nummularium* Bull. ex Fr. on beech and *H. fuscum* L. on hazel (*Corylus avellana* L.), or only normally being found in association with living trunks or attached branches, as with *V. comedens*. One implication is that such fungi might already be present extensively, but not overtly, under conditions imposed in functional sapwood. Such latent invasion could result from the development of separate mycelial units (modules) such as buds, cells, mycelial fragments, oidia, etc. (Cooke and Rayner, 1984) whose production might well be associated with high carbon dioxide concentrations. These could be disseminated widely within the sap stream, reverting later to mycelial development and consequent decay associated with loss of sapwood function. Such a possibility represents a significant departure from established views of decay development in trees and obviously requires substantiation. However, it serves to emphasize the potentially distorted view which may result from only considering major wounds as colonization courts.

Localization of decay and discoloration

There is no doubt that decay and discoloration originating from major, mechanically inflicted, sapwood wounds in living trunks are markedly localized within the vicinity of the damage (Fig. 1). A column of decay or discoloration commonly develops, the size of which is directly related to the severity of the wound and which is particularly abruptly truncated at its outer margin adjacent to wood newly laid down by the cambium subsequent to wounding. Where decay develops, as it normally does eventually, it is usual for this to occupy an approximately central position, with the periphery and distal parts of the column being discoloured. Decay-causing Basidiomycotina can usually be isolated only from the decayed region, bacteria and non-basidiomycetes being abundant in the discoloured parts. Similar patterns occur in cut hardwood stumps exhibiting regrowth (Rayner, 1979), where spread of decay or discoloration from the cut surface, either from natural sources or deliberate inoculations, do not proceed beyond a critical depth which is greatest in the central wood or, where heartwood is present, in the innermost sapwood (Fig. 2). Furthermore, when lateral inoculations are made, upward spread of decay fungi occurs readily, while downward spread typically tapers abruptly. In some instances, perhaps associated with mycelial growth down an empty vessel, very narrow decay columns can penetrate for considerable distances below the cut surface as though escaping the normal restrictions. When several such columns occur in proximity in oak, apparent premature heartwood formation, which also occurs around decay columns developing from wood plug inocula inserted in the cut surface, may be induced around them (Fig. 3).

Recent experiments involving inoculation of decay fungi into living attached oak branches have similarly demonstrated the marked localization of decay columns arising from mechanical wounds (Fig. 4; Boddy and Rayner, unpublished data). Whilst *S. gausapatum*, *Peniophora quercina* and *V. comedens* all readily became established, their decay columns were markedly restricted, in a manner similar to that illustrated in Figure 1, but without discoloured wood containing non-decay

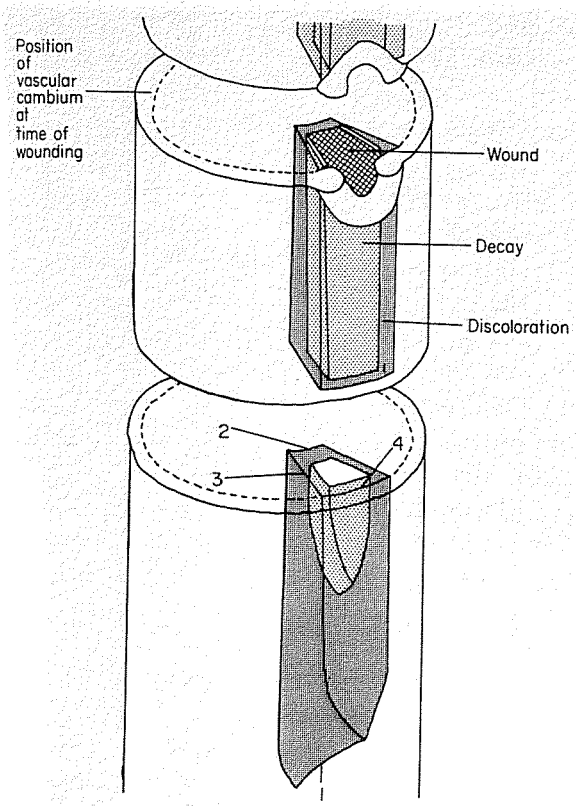

Fig. 1.

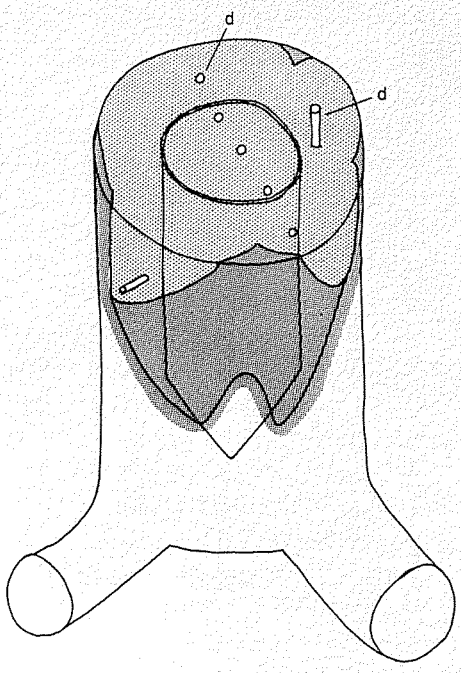

Fig. 2.

Fig. 1. Diagram illustrating localization of decay and discoloration in wounded sapwood of a living tree. The positions of the supposed compartmentalizing 'walls' 1, 2, 3 and 4 are shown.

Fig. 2. Idealized diagram illustrating the patterns of spread of *Coriolus versicolor* 26 months after inoculation of colonized dowel plugs (d) into oak stumps. Light stippling indicates zones of colonization spreading from inocula and dark stippling indicates zones of discoloration surrounding colonization zones. The heartwood is shown as a tapering cylinder, free from colonization. (After Rayner, 1979).

fungi, and they failed to continue to extend after 6 months from inoculation. Other decay fungi such as *Exidia glandulosa* Fr. and *Schizopora paradoxa* (Fr.) Donk. failed to become established significantly in the wood, and in these cases the sapwood either remained unaffected or stained to a very limited degree in the vicinity of the inocula. In all cases wood formed by cambial activity after damage remained uncolonized. Parts of the branches were ring-barked at the time of inoculation, and longitudinal spread in the outermost sapwood was facilitated towards a girdle from inocula placed either distal or proximal to it (Fig. 4). Decay and discoloration arising by natural colonization also frequently developed in peripheral wood via girdles, long narrow 'escape columns' sometimes being formed as described above for cut stumps. Also, extensive mycelial individuals, delimited by heartwood wings (see below), of *S. gausapatum* (other than the isolate inoculated) sometimes developed during the course of a 2-year experiment.

As has already been implied, different patterns occur when decay is established other than via major mechanical wounds. In the case of decay occupied by single extensive mycelial individuals referred to earlier, the decay column may occupy virtually the entire wood cylinder in dead trunks or branches, with little or no evidence for the occurrence of non-decay fungi in peripheral discoloured regions.

Fig. 3. Transverse section through part of a regrowing oak stump approximately 2 years after felling. Several small decay columns are present surrounded by living sapwood. Notes sessil

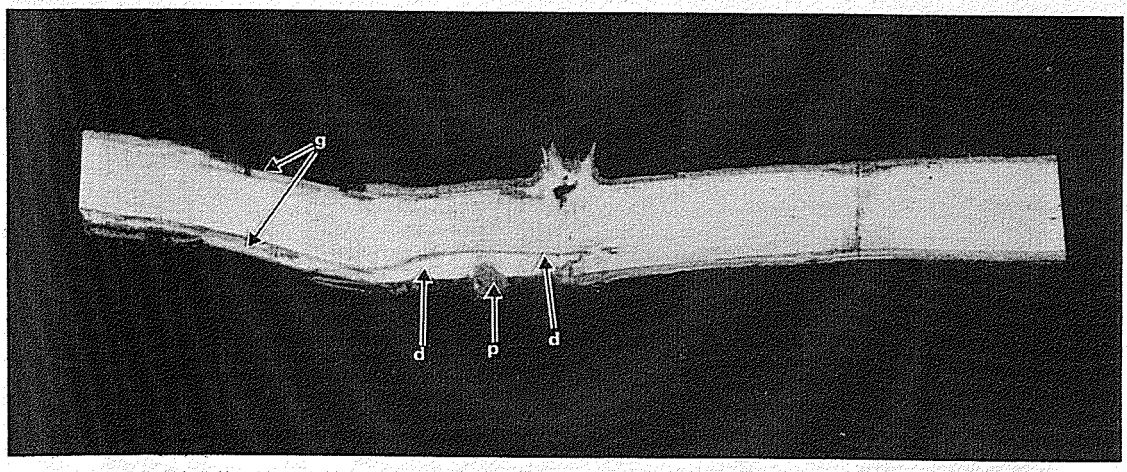

Fig. 4. Longitudinal section through an oak branch approximately 6 months after inoculation with *Vuilleminia comedens*. A decay column (d) has developed from the inoculum plug (p). Spread into the proximal portion of the branch is markedly restricted whilst distal spread towards the point of girdling (g) appears to have been facilitated in the outermost wood.

Furthermore, the longitudinal extent of the decay columns is often correlated with branching pattern such that they are abruptly truncated at the point of intersection of a subordinate branch with a main stem (Fig. 5). Where decay develops in partially living stems, it is common for this to be demarcated from living sapwood by narrow discoloured zones filled with abundant tyloses which characteristically develop in a radial orientation outwards from the centre of the wood cylinder to the junction between living and dead cambium. In oak, these discoloured zones are morphologically similar to heartwood and in stems of sufficient size are continuous with the heartwood. They persist as ridges, often internodal in length, of undecayed wood after the sapwood has disintegrated in long-dead attached branches and have been termed 'heartwood wings' (Boddy and Rayner, 1981). The wings effectively delimit a wedge-shaped column, separated from functional sapwood, in which decay can become established. The process of wing formation can precede establishment of decay – where wings have recently formed, this can often be detected by the presence of an undecayed zone between the wing and the decay (Fig. 6).

Besides wounds made by mechanical means, decay and discoloration can develop in wood adjacent to bark affected by canker diseases, as in sycamore (*Acer pseudoplatanus* L.) trees affected by diamond-bark canker, a disease characteristically associated with drought stress (Bevercombe and Rayner, 1980; Fig. 7). Colonization of the sapwood behind diseased bark proceeds as far as a dark-stained region which either follows the orientation of outer annual rings, or develops along radii, delimiting a wedge-shaped region. The latter situation is characteristic in upper parts of diseased trees, and the former in the lower regions of infected trunks.

Our discussion thus far has related to the development of decay in sapwood by fungi which gain access other than by direct, active pathogenesis. However, access via direct pathogenesis may occur with *Chondrostereum purpureum* (Pers. ex Fr.) Pouz. which colonizes aerially, and the root pathogens *Heterobasidion annosum* (Fr.) Bref. and *Armillaria* species. *Chondrostereum purpureum* invades wounds of *Prunus* species bringing about silverleaf disease, the symptoms of which are normally attributed to toxin production by the fungus. The fungus appears very rapidly in cut stems and stumps of many deciduous trees where its capacity for

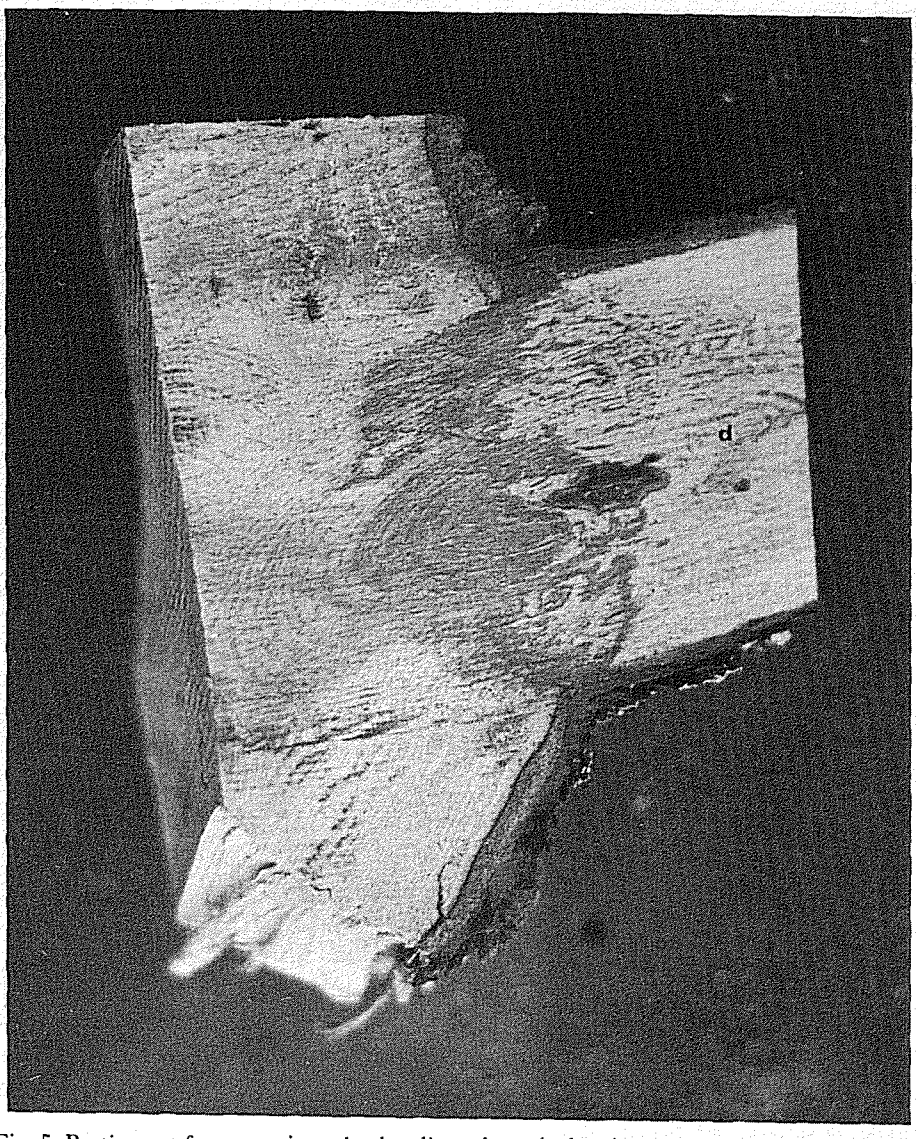

Fig. 5. Portion cut from a main and subordinate branch showing restriction of decay to the latter (d).

longitudinal and radial spread appears far greater than that of other decay fungi (Rayner, 1979). Its early occurrence has been attributed to its invasion only of wood in which parenchyma containing reserve materials is still alive (Guinier, 1933) and pathogenesis, involving unlocking of nutrients sequestered in living parenchyma, might therefore be important in its establishment. In the case of *Heterobasidion annosum* in pine (*Pinus* spp.) and *Armillaria*, colonization of the sapwood is characteristically preceded by ectotrophic mycelial development in the bark which leads to death of the cambium.

Early stages in the development of decay in heartwood have not been studied as effectively as those in sapwood, perhaps relating to the much longer time-scales which may be involved. Certainly patterns of decay in heartwood are extremely varied and difficult to generalize about (Boyce, 1961). However, decay or discoloration resulting from drill-bit wounds or dowel inocula in heartwood

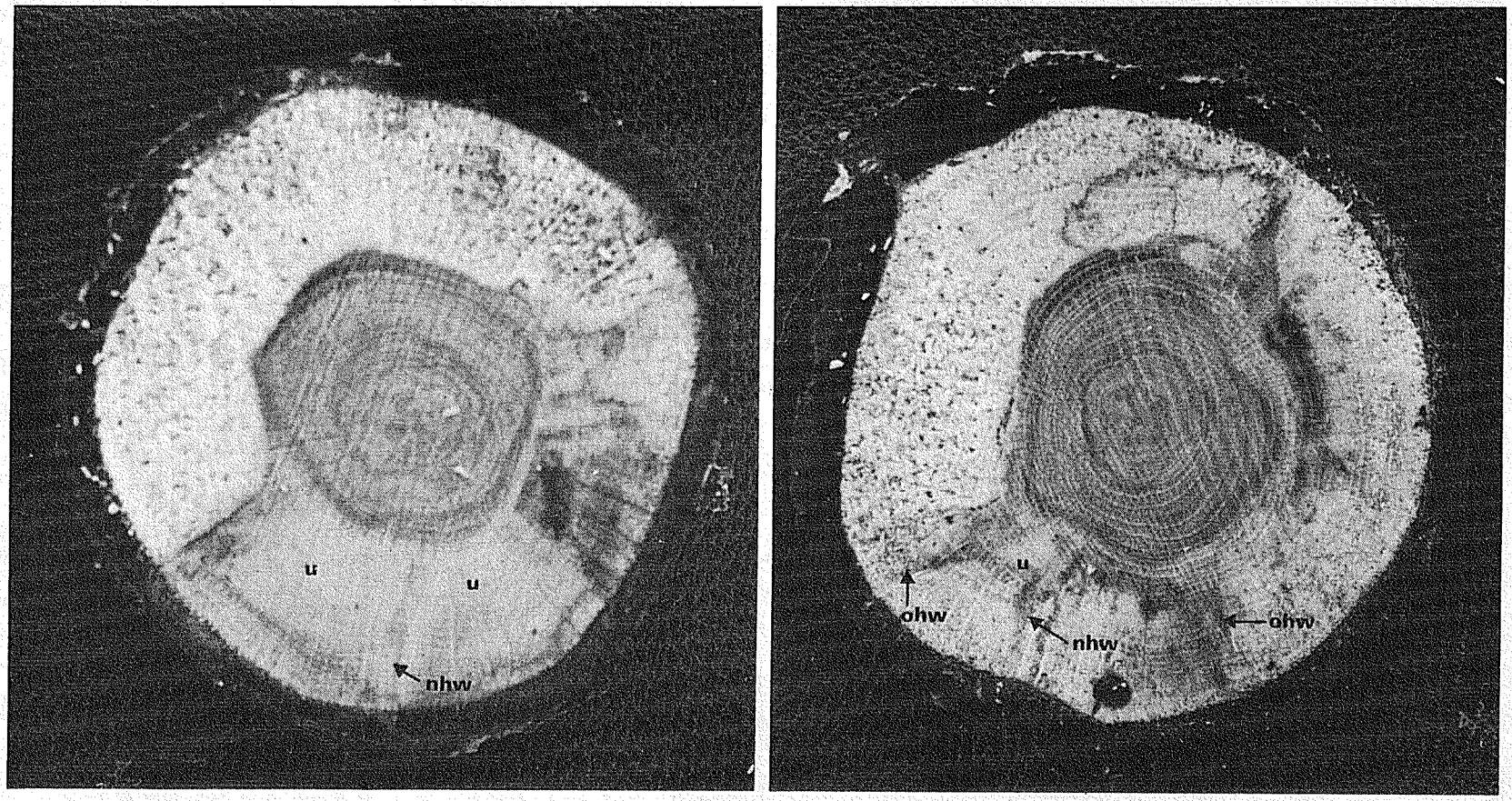

Fig. 6. Sections from a well decayed but until recently partly living branch of oak. Recently living wood is in the process of becoming colonized; a heartwood wing (nhw) has developed dividing this into two columns which still contain undecayed wood (u) this being more substantial in the section from nearest the base of the branch (left). Older heartwood wings (ohw) are associated with the more well-decayed wood.

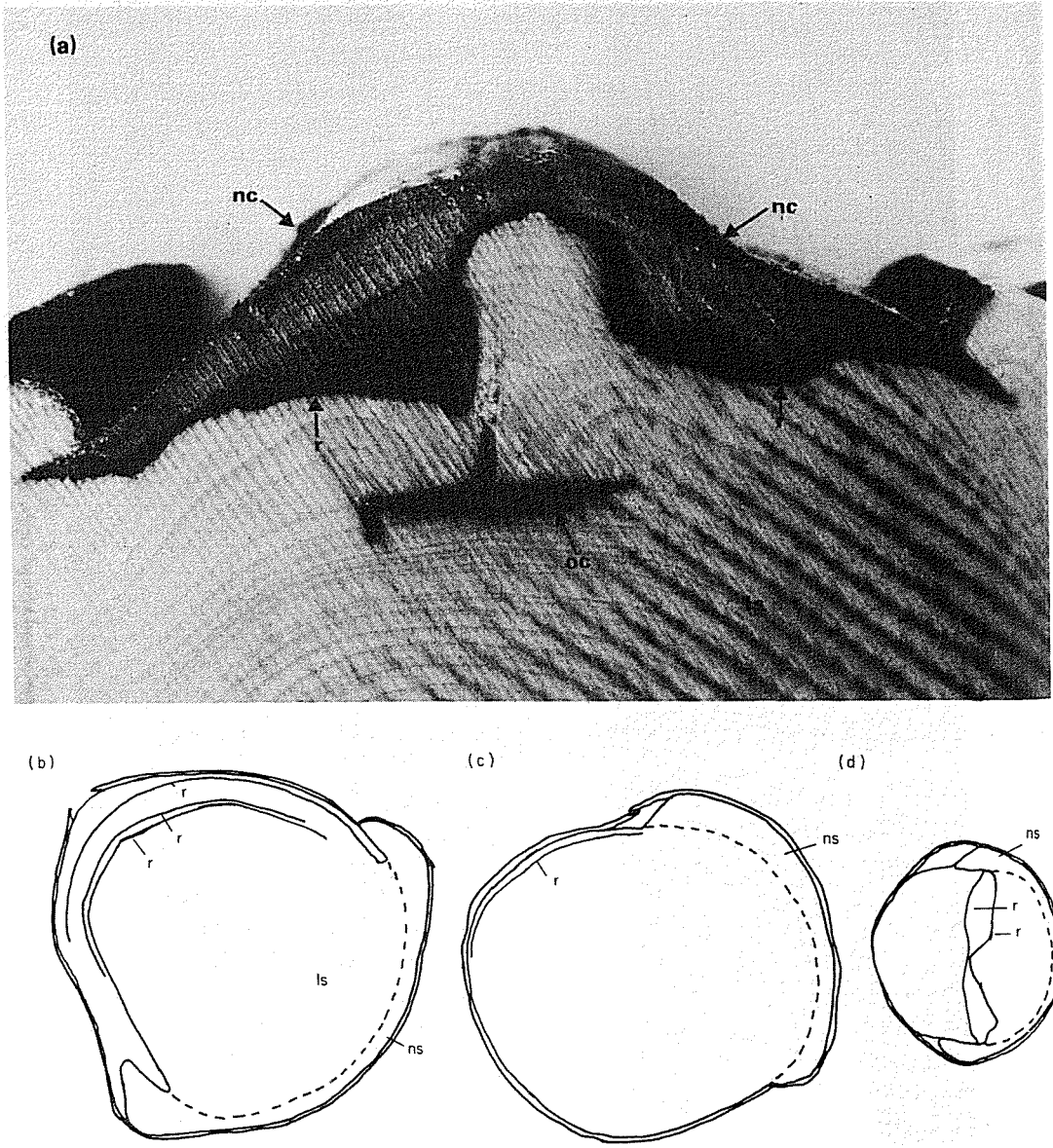

Fig. 7. Decay and discoloration of sapwood behind diamond cankers in sycamore. (a) Part of a transverse section through a living tree where two new cankers (nc) had developed over the site of an old canker (oc) the junction with living sapwood (ls) being marked by a dark 'reaction zone' (r) whose position closely follows the conformation of successive zones of autumn wood produced as the old canker 'healed over' (from Bevercombe and Rayner, 1980); (b) to (d) transverse sections through cankers formed successively higher up a diseased tree, encroachment of discoloration is very restricted in the lowermost section, despite extensive cambial death. Nonetheless three successive 'reaction zones' have been formed. In (c) one reaction zone has formed, but then the entire wood cylinder present at the time of canker formation has become accessible to decay and stain, only newly-formed sapwood (ns) being inaccessible. In (d) the 'reaction zone' has formed along radii, the outermost points of which are found at the limit of cambial death. Two 'reaction zones' are evident, the older of which was evidently undergoing degradation (drawn from Bevercombe, 1980).

characteristically form narrow-sided columns, at least initially (Shigo and Shortle, 1979; Rayner, unpublished).

Aetiology of localization of decay and discoloration

Implicit in the expanded concept is that the localization of decay and discoloration described above is due to an active defence response (compartmentalization) by living tissues which on average constitute 10% by volume even of functional sapwood (Hillis, 1977) to wounding and infection. According to CODIT this is mediated via a series of physicochemical barriers described as 'walls' (Shigo and Marx, 1977; Shortle, 1979). In order of increasing 'holding strength', wall 1 is formed by 'plugging' of axial elements by gums or tyloses, which impede vertical spread; wall 2 is due to the presence of dense latewood in each annual ring and impedes inward radial spread; wall 3 is due to living cells in medullary rays (which incidentally constitute a spatially discontinuous tissue!) which impede tangential spread; wall 4 occurs at the junction of damaged wood and wood newly formed by the vascular cambium, supposedly due to a change in activity of the latter which produces a so-called 'barrier' zone. Fungi and other microorganisms are envisaged as growing between the 'compartments' delimited by these walls. The relative differences in holding strength of the walls is used to explain the resulting characteristic shape of the wood columns occupied; that is, the model is based on transgression of barriers, which are actively produced and / or maintained. However, it is also possible to explain the shape of the columns, with the exception of the presence of a barrier zone, and their greater longitudinal extent in inner sapwood, in terms of the relative *accessibility* in different directions of woody tissue to permeation by fluids and fungal hyphae. Longitudinal access is facilitated by the predominantly elongated lumina of xylem elements such as vessels, tracheids, fibres and axial parenchyma. Radial access will be enhanced by the radially elongated, nutrient-enriched parenchyma of medullary rays (regarded as barriers in the CODIT concept), providing these are dead. However, it will be restricted in non-ray tissue by the close-packed xylem elements in the late (autumn) wood of each annual ring. Except where a dead cambial layer facilitates subcortical mycelial spread, tangential access is greatly restricted by the lack of any system, apart from trauma-induced resin or gum-filled canals in a few cases, of tangentially elongated lacunae in wood. Any spread in this direction must be effected via numerous woody cell walls, either by direct penetration, formation of bore holes, or through pits. Contiguous large vessels or other elements in spring wood, particularly that which is ring porous, may slightly enhance tangential access in this tissue. On the basis that the effects of these anatomical features operate without interference from other factors, the expected shape of a mycelium arising eccentrically in intact timber is a longitudinally tapering, wedge-shaped column, with slightly corrugated radial margins due to alternation between early and late wood in annual rings (Cooke and Rayner, 1984). Certainly such anatomical considerations adequately explain decay distribution in dead and even processed timber (Corbett, 1963, 1965; Rayner and Todd, 1979). Localization of decay or discoloration in heartwood can therefore readily be explained, but Shigo and Shortle (1979) have insisted that this is due to active compartmentalization by living tissue, a conclusion based on an inappropriate comparison of living with extensively decayed trees in which the heartwood was open to colonization from decaying sapwood on all sides.

Whilst these anatomical considerations may therefore underly the shape of decay

columns in living trees, they do not in themselves explain the prevention or slowing of continued extension, nor the greater penetration in inner sapwood. CODIT implies that these are due to the cumulative effect of walls 1, 2, and 3 which successively impede colonization, and the immediate effect of wall 4 which is regarded as an unbreachable barrier. Greater or more effective production of wall 1 in the outer sapwood explains the greater limitation of longitudinal penetration in this tissue.

A rather different explanation, but one still based on the idea of an active defence response, has been developed by Shain (1967, 1971, 1979). This rests on the observation that discoloured or decayed wood resulting from sapwood injury or arising by encroachment of decay fungi from a central core of heartwood is marked at its boundary by production of two tissue types; a discoloured 'reaction zone' which forms a dark edge to the colonized tissues, and a pale 'transition zone' which is contiguous with functional sapwood. These zones form in trees both with and without true heartwood, and in hardwoods and softwoods, and the dark zones in sycamore and heartwood wings which we have observed and described above probably constitute equivalent or related features. The reaction zone is interpreted as being due to a defensive response of the tree to wounding or invasion, preventing spread of microorganisms into the functional sapwood which it delimits. Unlike CODIT walls 1, 2 and 3, with which it is sometimes equated (Manion and Zabel, 1979), it constitutes a one-step defensive barrier. However, it can be breached, resulting in production of successive reaction zones, often at decreasing intervals, with the older zones becoming progressively decolourized (Shain, 1979; Mercer, 1982; cf. Fig. 7). The effectiveness of the reaction zone as a barrier to invasion is accredited to its high content of polyphenols (including, in *Pinus*, the stilbene pinosylvin). However, this provides problems in that although such chemicals may well be fungitoxic, it is difficult to conceive that they could entirely prevent access of at least a few hyphae across such narrow barriers – this is borne out by the fact that they can be breached and even degraded. Shain (1979) has suggested therefore that the reaction zone polyphenols are maintained at high levels by continuous release as part of a dynamic host response. If the polyphenols are released by dying cells in the reaction zone this may not be possible. Furthermore, the whole argument depends on the notion that reaction zone polyphenols are sufficiently fungitoxic, and present at sufficient levels, to arrest fungal growth entirely. This seems unlikely to be true, especially when it is realized that extractives from the heartwood of certain trees, such as elm (*Ulmus* spp.) are not particularly fungitoxic, and may even selectively stimulate growth of certain fungi (Rayner and Hedges, 1982).

Given that conditions in functional sapwood, associated with its high moisture content and lack of freely available nutrients, are inimical to mycelial development, there is no requirement for an active host response to *infection*. There is merely a requirement for a non-specific mechanism which maintains sapwood function adjacent to damaged or non-functional tissue. Such a mechanism may be caused or enhanced by factors such as blocking by gums and tyloses or production of suberized layers which seal-in the damaged tissue. This would inevitably confine colonization to the damaged or non-functional tissue. Certain vascular wilt fungi, including species of *Verticillium* Nees es Link (e.g. in *Acer* L.) and *Ceratocystis* Ellis & Halst. (e.g. in oak and elm) are, however, sometimes regarded as growing in functional sapwood. Whilst they may be capable of some mycelial development, spread within the xylem is probably largely affected via abundant production of

conidia. They are not, of course, decay fungi and may represent an extreme and very specific example of latent invasion associated with rapid pathogenesis.

Viewed in these terms, the composite represented by the reaction and transition zones (henceforth termed 'transition region') marks the dividing line between functional and non-functional tissues; that a region of non-functional sapwood is associated with injuries extending below the cambium has been demonstrated histologically by Mullick (1977). In the transition region a marked gradient in moisture conditions can be expected between fully functional sapwood and damaged, non-functional tissue which will allow access of air and hence be subject to fluctuating moisture regimes, some of which may be conducive to mycelial growth. The formation of a relatively dry zone in which there is apparently increased metabolic activity has been implicated in heartwood formation associated with relatively high ethylene levels and subsequent formation of polyphenolic extractives (Hillis, 1977). There is evidence that these are indeed features of the transition zone (Shain, 1979), and they would certainly explain the development of heartwood wings adjacent to damaged oak sapwood.

If maintenance of water-saturated sapwood explains the limitation of decay or discoloration development in this way, the greater colonization of inner sapwood, which contains less water and is therefore better aerated than the outer tissues, is readily understood, without the need to invoke differences in the response of living cells to invasion (cf. Coutts, 1976). By the same token, functional sapwood newly formed after damage, such as occurs outside the 'barrier' zone, would be inaccessible to mycelial development. The production of a barrier zone of morphologically abnormal tissue would then be expected to follow exposure of cambial tissue to desiccating conditions and production of suberin (Pearce and Rutherford, 1981) would not be unexpected. Rather than being a barrier to outward spread of infection as such, the barrier zone would constitute a mechanism preventing ingress of air or loss of water from the newly formed conducting elements. In these terms the production of a suberized barrier is likely to be of importance only in the immediate vicinity of damage, and evidence that such a barrier is formed only adjacent to a wound, or fungally invaded tissue, has been provided by Moore (1978) and Pearce and Rutherford (1981). This is in contrast with the suggestion (Shigo and Marx, 1977) that the barrier zone is formed completely around the stem.

These comments do not rule out the possibility of an interaction between an invading fungus and the sealing off of functional sapwood. If a fungus is capable of establishment at a wound, as after deliberate inoculation, then there is the chance of its invading the tissues, and even extending access of air to them (Coutts, 1976), before sealing mechanisms become fully effective. In these terms, inoculations made in spring when sap flow rates are high are likely to be less effective in introducing decay organisms than those in autumn, and we have obtained some evidence for this in attached oak branches. The capacity of fungi to establish in this way is likely to vary, for example in relation to their tolerance of high carbon dioxide and low oxygen levels. This would explain, for example, the differing abilities of *Stereum gausapatum* and *Schizopora paradoxa* to establish from inocula in oak branches (see above). Furthermore, fungi capable of rapid extension under these selective conditions could form considerable colonized columns; for example, this could help explain the behaviour of *Chondrostereum purpureum*.

Decay community development

Strongly associated with the CODIT concept is the idea that decay in living trees is brought about by a wide variety of interacting organisms which colonize in succession. It has even been suggested that this succession is a necessary device by which microorganisms overcome the compartmentalizing response of the tree (Shigo, 1979). These ideas are based on the observation that decay columns originating from major wounds in standing trees are surrounded by discoloured wood from which non-Basidiomycotina and bacteria are consistently isolated, wood-decaying Basidiomycotina only being isolated from proximal regions visibly undergoing decay. This is taken to show that the wounds are colonized first by pioneer non-Basidiomycotina, which discolour the wood and necessarily precede the decay species, perhaps ameliorating conditions for the latter by detoxifying phenolic inhibitors released by the active response of invaded sapwood (Shigo, 1979). A more complicated view is that initial discoloration may be caused by phenoloxidases of Basidiomycotina acting on phenols released by living cells as an active response. The Basidiomycotina are then unable to grow in the discoloured wood until it is detoxified by bacteria and non-Basidiomycotina that are themselves incapable of causing discoloration (Shortle and Cowling, 1978). This latter view is based on comparison of the growth of a limited number of non-Basidiomycotina and decay Basidiomycotina on excised living and steam-sterilized sapwood blocks; the Basidiomycotina only grew poorly if at all on the unsterilized sapwood, where their growth was correlated with considerable discoloration of the tissue, but developed luxuriantly on steam-sterilized blocks. However, certain pioneer non-Basidiomycotina (*Ceratocystis coerulescens* (Munch) Bak., *Fusarium oxysporum* Schlecht and *F. moniliforme* Sheldon) grew on the unsterilized sapwood without causing discoloration, and were also capable of growth at concentrations of gallic acid inhibitory to the few decay fungi tested which attack standing trees (but not to a variety of species which attack wood products). The explanations given for these observations are based on assumptions that the discoloration induced by the Basidiomycotina is the same as that occurring in the standing tree, and that the poor growth of these fungi is directly attributable to the discoloration mechanism. However, the better growth on sterilized blocks could also be explained in terms of release of assimilable nutrients and possible improvement of aeration.

The whole question of the applicability of successional principles to the community dynamics of fungi is open to doubt and confusion (Rayner and Todd, 1979; Cooke and Rayner, 1984). This partly relates to lack of spatiotemporal considerations (as opposed to purely temporal ones) in succession studies, and to the fact that extension of indeterminate mycelial thalli is an integral part of community development. It is therefore always difficult to be certain of the time at which fungi first colonize a substratum, as opposed to the time at which they become dominant. The interpretation that Basidiomycotina necessarily *colonize* late, and therefore that some form of pre-conditioning of the wood is necessary, is thus immediately open to question. This doubt is enhanced by the successful establishment of appropriate species from deliberate inoculations.

The apparent predominance of non-decay species at early stages of community development is more probably a reflection of the particular circumstance of entry via major wounds, which expose considerable volumes of wood to immediate colonization. Parallel patterns of community development are seen whenever a large supply of a previously unoccupied resource containing readily assimilable substrates is suddenly made available to decomposer organisms; this includes

felled and processed timber (Käärik, 1974; Levy, 1982). An initially species-diverse community, including organisms only capable of growth on easily assimilable substrates, but with associated powers of effective dispersal and rapid commitment to sporulation first develops. This then changes to a more specific community containing combative organisms which replace many of the pioneers and deplete remaining nutrient supplies, including lignin and cellulose in wood.

It will be apparent that the succession concept omits consideration of patterns of decay development other than in association with major wounds acting as entry sites. As has already been indicated, these may often involve single individuals of decay-causing Basidiomycotina and Ascomycotina acting entirely independently.

Concluding Discussion

Two salient points arise from the above considerations. The first is that moisture content may be a principal determinant of decay distribution. Following from this, non-specific mechanisms which preserve the moisture content of living sapwood can limit its accessibility to decay organisms, and there is no general need to invoke active mechanisms in host defence to invasion.

Even if the fundamental premises of the 'expanded' concept of decay in trees are correct, it fails to encapsulate all known decay processes and patterns, in particular, it over-emphasizes major wounds as colonization foci. By contrast, variations of moisture content account well for decay patterns developing under a wide range of circumstances. The implication is that this simpler concept is equally, if not more widely, applicable as a possible explanation of decay distribution in standing trees. For example, it is widely accepted that external stresses and infections which adversely affect physiological functioning can result in predisposition of trees to decay and disease (Schoeneweiss, 1978). These stresses range from water shortage, light suppression, extensive wounding, defoliation and root infection to any of a variety of biotic and abiotic factors which can lead to bark death. Their operation appears to be particularly associated with development of extensive decay individuals (see above) and, whilst diverse in themselves, they could well be expressed via a simple underlying mechanism. One such mechanism would be the lowering of moisture content to a level allowing mycelial development. This could occur either generally, or be localized to particular sides of, or patches around, a trunk or branch. Mycelial development would then occur in the affected regions. In association with the concept of latent invasion, this could well explain, for example, the internodal distribution of heartwood wings and the decay columns they delimit. Whilst it might be argued that such effects of direct or indirect water stress could operate through a decrease in host resistance, water stress is the primary factor, and would have the same effects regardless of the presence or absence of defence mechanisms.

Some of the best evidence for the role of water stress in predisposition of trees to infection is provided by canker diseases. For example, a close correlation exists between development of cankers caused by *Cryptodiaporthe salicella* (Fr.) Petrak on *Salix* sp. and by *F. lateritium* Nees. on *Populus trichocarpa* Torrey & Gray, and the relative turgidity of the bark (Bier, 1959, 1961). Diamond-bark disease of sycamore is one such canker disease whose occurrence is closely correlated with summer drought (Bevercombe and Rayner, 1980). The pattern of colonization of the wood behind diamond-cankers (see above, Fig. 7) may therefore be particularly instructive. The occurrence of wedge-shaped colonization zones in the upper

portions of trunks, but only peripheral zones in the lower parts, is consistent with expectation from the moisture distribution pattern in water-stressed trunks. Presumably, as the moisture content decreases with height to a critical level, the limitation of air and fungi to peripheral tissue adjacent to the dead bark changes because medullary rays provide radial access resulting in a wedge-shaped colonization zone. Similarly, wedge-shaped columns of stain caused by *Ceratocystis* spp. in stressed or damaged trees have been attributed to facilitated access of air to the damaged wood (Nelson, 1934).

Also demonstrated in Figure 7 is the production of successive reaction zones as access is afforded to tissues deeper within the wood. As has been mentioned, the breaching of reaction zones in this way demonstrates that they are not permanently impenetrable barriers to fungal invasion, and the possibility of maintaining them as such by an active mechanism is difficult to countenance. Much simpler is the idea that these barriers are breached as soon as moisture levels behind them drop below a critical level. The decreasing distance between successive reaction zones which is sometimes observed (Mercer, 1982) is also readily understood in these terms.

In more general terms, variations in decay development both across the diameter of an individual tree, and between different trees can all be readily interpreted in terms of moisture content. Within a tree, moisture content tends to decline progressively towards the centre of the trunk, and the effects of this in promoting colonization of more central tissues has already been mentioned. Heartrot is then readily understood: in the standing tree central tissues are the most susceptible to decay, even when containing fungistatic or fungitoxic chemicals, the latter principally having a *selective* effect rather than preventing colonization altogether. The extent of moisture gradients in individual trees will also vary considerably, both within and between species. Thus it is well known that in certain tree species, only one or a few annual rings of the sapwood are conductive, whilst in others the entire wood column may be so (Greenidge, 1958). Hearts of the former would be expected to be more subject to colonization than the latter. However, differences in the susceptibility of the hearts of trees to colonization are attributed by CODIT to 'weak' and 'strong' compartmentalization responses (Shigo, 1979). The compartmentalization response varies even between trees of the same species (Copony and Barnes, 1974; French and Manion, 1975). Differences in the response by different clones of *Populus deltoides* Marsh × *Populus trichocarpa* Hook were attributed to genetic differences between the clones (Shigo, Shortle and Garrett, 1977). Such differences may be attributed equally well to variation in the water relationships of the trees based on genetic or environmental factors.

To some extent the CODIT concept may be an example of the ease with which apparent host defence mechanisms can be confused with non-specific maintenance of function. This is epitomized by the idea that higher plants have means for preventing the growth of almost all pathogens, resistance being the rule (Wood, 1974). This introduces the danger of treating mechanisms which *confer* resistance as being *for* resistance. Given that the nutrients within functional tissue are not readily accessible, three options are available to a heterotroph: biotrophy in which nutrients are absorbed with minimal interference from living cells (by means of haustoria or equivalent structures), necrotrophy in which the living tissue is first killed and its nutrients released for consumption, and latent invasion where restricted saprotrophic existence awaits a change in circumstances. True active defence mechanisms can then be seen as evolving in relation to specific cases of necrotrophy or biotrophy. Viewed in these terms the idea of provoked non-specific

resistance mechanisms (as opposed to maintenance functions which confer resistance), which is at the root of the CODIT concept, may not be tenable. The fact that reaction zones, heartwood wings and equivalent features are non-specific and sometimes produced in advance of hyphal penetration (Shain, 1979; Fig. 6) has obvious bearing in this context.

A useful illustration of these principles has recently been provided by Mullick (1977) in relation to the production of wound periderms in tree bark, which has often been interpreted as a defence mechanism. However, such periderms are produced whenever, and for whatever reason, phellogen becomes inactive. The need for constant phellogen activity, to maintain normal tree functioning, is met by the formation of a new 'necrophylactic periderm' via de-differentiation of cells to form a non-suberized impermeable tissue under which new phellogen can form. Pathogenesis, such as can be effected by certain aphids, occurs whenever this normal process of phellogen restoration is interfered with.

Finally, we wish to emphasize that this is not just a semantic argument; the issues affect the whole direction and concentration of effort which underlies tree decay studies.

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