# Dynamic Responses of Differentiated Sapwood to Injury and Infection

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Heartwood of many tree species contains phenolic compounds which inhibit wood decay fungi and are largely responsible for the durability of heartwood in service. Sapwood of most species lacks appreciable amounts of inhibitory compounds and is considerably less durable in service than is heartwood when exposed to conditions favoring decay (43).

The relative susceptibility of heartwood and sapwood to decay in living trees, however, is reversed. Decay fungi are largely confined to the hostile environment of the heartwood to the extent that heartrot is the most destructive tree disease (22). Sapwood, on the other hand, may remain relatively free of infection for many years even when neighboring heartwood is extensively decayed. Our understanding of this phenomenon is, at best, fragmentary.

Two other papers of this symposium (20,57) critically evaluate the state of knowledge of certain aspects of the trees' defenses against injury and decay. I will limit my comments to an induced mechanism of resistance by differentiated sapwood. The role of preformed inhibitors (24) and the formation of barrier zones (58), therefore, will not be included in this paper.

Observed responses of differentiated sapwood to injury and subsequent infection, or to the encroachment of decay fungi from a central core of heartrot, include the production of two types of tissue: the transition zone and the reaction zone. For the purpose of orientation, these contiguous tissues separate infected wood from moist, functional sapwood. The pale-colored transition zone is contiguous with functional sapwood and the phenol-enriched reaction zone is contiguous with infected wood. This description takes into account the histological and cultural evidence that the transition zone and the reaction zone are produced in advance of infection by decay fungi (28,45,46,65).

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## TRANSITION ZONES

**Moisture content.** Transition zones are drier than surrounding sapwood, hence their pale color. The moisture contents of transition zone and sound sapwood of Norway spruce (*Picea abies*) attacked by *Fomes annosus,* for example, were about 40 and 120% (dry wt), respectively (1). Some authors (10,16) have chosen the name "dry zone" to describe this tissue.

A plausible explanation has been provided for the rapid formation of dry zones adjacent to wounds in sapwood containing water columns under hydrostatic tension. In conifers, gas emboli are restricted to injured tracheids, particularly in earlywood, due to the valve-like action of tori causing aspiration of bordered pit pairs. This occurs because the pressure required to deflect tori into a closed position is sufficiently less than that required to move air-sap menisci through pit pairs (17). Drying in latewood, under similar conditions, is not particularly noticeable probably because it is nonconducting due to a low degree of water saturation (17,19). Embolism also is restricted to the severed vascular components in angiosperms due to insufficient pressure to draw menisci through pits that connect vessel segments (44).

The mechanism for replacement of water with gas in internal tissues not in direct contact with the atmosphere (ie, transition zones surrounding heartwood or reaction zones encircling central columns of decay), however, is more difficult to explain. Harris (18) found that over 80% of the bordered pits were aspirated in the dry-wood zone encircling heartwood of *Pinus radiata.* This compared to less than 50% pit aspiration in sapwood and about 50% at the

sapwood-dry zone boundary. He reasoned that a tissue containing tracheids with more than 50% of their pits aspirated would be impermeable to water movement. Direct measurement subsequently showed that dry zones surrounding cankers caused by *Peridermium pini* on Scots pine (*Pinus sylvestris*) were quite impermeable to water (16). Harris (18) further speculated that utilization by metabolizing parenchyma of water made available by low hydrostatic tensions in inner sapwood and subsequent pit aspiration were the causes of drying.

Coutts (11) presented evidence for the involvement of living parenchyma in the formation of dry zones in the sapwood of Corsican pine (*Pinus nigra* var. *maritima*) and Scots pine. Logs injected with dilute poison and subjected to autoclaving, an anaerobic atmosphere, or cold temperature (2–5 C) produced dry zones considerably smaller than their injected counterparts that were incubated under conditions favorable for metabolism. Sizable dry zones, however, were produced when autoclaved logs were injected with an aqueous extract from a log inoculated with *Fomes annosus*. The explanation given was that lysis of tori by the fungal extract provided an avenue for withdrawal of water and entry of gas. This explanation, however, seem inappropriate for living systems in which tori in transition zones are not lysed and the zone itself is quite impermeable to the movement of water (10,16). Coutts (11), contrary to Harris (18), concluded that dry zones were produced under conditions of high hydrostatic tension. Fresh logs with hydrostatic tension presumably relaxed by placing their ends in water prior to injection produced smaller dry zones than similar ones whose ends were not placed in water. From these results it was suggested that cavitation in individual tracheids resulting from the entry of gas from adjoining parenchyma under conditions of high hydrostatic tension could explain the formation of dry zones.

To add to this conjecture, I suggest that gradients in water potential from transition zone to surrounding sapwood could account for the movement of water out of this zone, particularly after the hydrostatic tension in its tracheids was released. Water would be expected to move from such tracheids into transition-zone parenchyma whose water potential may be expected to be higher than that of sapwood parenchyma because: water in sapwood tracheids would be under greater hydrostatic tension and, therefore, less available to its parenchyma; soluble metabolites in the transition-zone parenchyma are being converted to less soluble extractives; and the possible increase in membrane permeability of transition-zone parenchyma. This early response to pathogenesis (63) could be induced by ethylene (36), which was shown to accumulate in transition zones (48,50).

It seems less likely that the metabolic processes of transition-zone parenchyma could account for the dramatic decrease in water in this tissue. For example, a sample of sapwood containing 10 g of wood substance and 10 g of water (moisture content = 100% dry wt) with a starch content of 5% would not lose more than 0.05 g of water if all of the starch was degraded by hydrolysis.

Additional experimental evidence is necessary to formulate a comprehensive explanation for the drying of this tissue.

**Metabolic activity.** The wide range of biochemical events observed in transition zones lends convincing evidence that this tissue contains parenchyma that is living and quite active metabolically. The obvious effects of this metabolism in the systems studied are the disappearance of starch, the accumulation of phenols, and, probably, the death of transition-zone parenchyma.

Ethylene, a gas with hormone-like properties, may play a fundamental role in this process. This gas is produced by a wide variety of plants during flowering, fruit ripening, senescence, and in response to mechanical injury and infection (2,5). It has been implicated in increased respiration and the synthesis of enzymes required in the synthesis of phenols (41,52,60). A possible involvement of ethylene in transition-zone drying by increasing permeability of cell membranes also was cited earlier.

Enhanced production of ethylene was obtained from lesions caused by the wood wasp *Sirex noctillio* and its associated decay fungus *Amylosterium areolatum* in sapwood of *P. radiata*. Because some fungi also produce ethylene (25), it was necessary to determine if this ethylene was the product of host or pathogen. Lesions with transition zones removed and cultures of *A. areolatum*, with or without *P. radiata* sapwood produced no more than negligible amounts of ethylene (48). This demonstrated that the ethylene was of host origin and that the seat of enhanced ethylene production was the transition zone.

Lesions 1–4 wk old, caused by *S. noctillio* in two multi-stemmed trees, produced about 17 times more ethylene than control tissue obtained from adjacent, sound sapwood. Comparable lesions in a suppressed tree produced only about twice as much ethylene as did controls and one-tenth that produced by lesions in the dominant trees. Three weeks after inoculation, phenols were present in considerable quantities in lesions in the dominant trees but still were not detectable in lesions in the suppressed tree (48). The major phenolic compound detected in *S. noctillio* lesions was pinosylvin (23) which was quite inhibitory ($ED_{50} < 25$ ppm) to *A. aerolatum* in an agar medium (9). In subsequent studies, it was found that increases in ethylene production were detectable 1 day after inoculation (more than a 10-fold increase over controls) and that pinosylvin was detectable by gas-liquid chromatography (49) within two days of inoculation (50). Internal concentrations of ethylene, furthermore, were substantially higher than in controls 3 days after inoculation and a lesion 4 wk old had an internal concentration as high as 5 ppm (L. Shain, *unpublished*). Ethylene also has been implicated directly in the synthesis of pinosylvin (49) and other phenols (7,42).

Phenols have been implicated in the resistance of *P. radiata* to attack by *S. noctillio* (12). Greater resistance, therefore would be expected in individuals that produce larger quantities of ethylene, and pinosylvin, at a faster rate. It would be very desirable, from the tree-breeders' standpoint, if this capacity to respond were highly heritable. In the three-tree experiment mentioned above, however, it was not possible to distinguish whether the observed idfferences in response were due to the trees' crown class or genotype.

A modest attempt was made to determine whether genotype or growing conditions most affected the hosts' capacity to respond. Two clones of *P. radiata* each growing on a good and a poor site (measured by significant differences in apical growth) were subjected to a controlled *S. noctillio* attack by insects with clipped wings. Ethylene production by *S. noctillio* lesions measured 32 days after attack did not significantly differ, indicating that genotype may be playing a greater role than environment in the hosts' response. These results certainly need to be substantiated.

We are now measuring the amount of ethylene produced in response to standardized wounds in cottonwood (*Populus deltoides*). If this can be correlated with observed differences in resistance to decay and discoloration (56) it could be a rapid and convenient means for identifying resistant individuals.

Ethylene also was produced in response to wounds in all other species we have tested; ie, several species of *Ecalyptus,* black locust (*Robinia pseudoacacia*), and white pine (*Pinus strobus*) (L. Shain, *unpublished*). There is little doubt that ethylene production is a common, if not universal, early response of trees to wounding and infection.

Slight increases (about 1.4 times) were reported in oxygen uptake by *S. noctillio* lesions with their transition zones 1–6 days after attack as compared to adjacent sapwood (50). Approximately five fold differences, however, were obtained between lesions several months old and their controls. Manometric determination of oxygen uptake by *S. noctillio* lesions with and without transition zones in the presence or absence of a phenolic substrate (1-napthol at $3.5 \times 10^{-4}$ M) indicated that transition zones contribute more to the apparent respiratory rise than does the fungus in the necrotic portion of the lesion. Phenol oxidase activity, while present in transition zones, was 2–3 times greater in the necrotic tissue. With this technique, phenol oxidase was not detectable in adjacent sapwood (L. Shain, *unpublished*). By enzyme histochemistry, it was possible to demonstrate increased activity of both malic and glucose-6-phosphate dehydrogenases in the parenchyma of transition zones surrounding heartwood of *P. radiata* (51) and transition zones surrounding lesions caused by *S. noctillio*.

These modest increases in oxygen uptake by transition zones, however, are overshadowed by the rapid and greater increases that

sometimes were obtained in ethylene production.

The histochemical demonstration of starch in sapwood and its paucity or absence in adjacent transition zones (11) is good evidence that starch degradation occurs in this zone. Isolation of starch-degrading enzymes from transition zones apparently has not been reported.

Much remains to be learned about the physiology and metabolism of transition zones. Such information could greatly increase our understanding of reaction-zone formation.

## REACTION ZONES

Reaction zones are necrotic tissues that are enriched with inhibitory extractives and are produced in advance of infection. Their formation has been related to a dynamic mechanism of host resistance (45,46). The term reaction zone probably is analogous to several other terms which have been used; eg, pathological heartwood (6), protection wood (28), discolored wood (8,53), wound-initiated discoloration (54), and walls 1, 2, and 3 of the CODIT model proposed by Shigo and Marx (55).

**Stimulus for reaction zone formation.** As host parenchyma in the transition zone die, reaction zones are formed. The occurrence of some reaction zones apparently devoid of microbial colonization (46,65) and the intraspecific, qualitative consistency of its chemical components (23,45,47) provide strong circumstantial evidence that this tissue is the end product of transition zone, but not microbial, metabolism.

It was suggested that the stimulus for necrosis of reaction zones may be provided by toxins produced by invading pathogens (11,45). The nonspecificity of this response and the production of reaction zones around presumably sterile wounds (45) argue against the necessity for such toxins. It seems more likely that the physiological and metabolic processes of the transition zone are programmed to terminate in reaction-zone formation and cell death in what could be considered a hypersensitive response. Heartwood formation, which in some ways may be similar to reaction-zone formation (45,62) also seems to occur in the absence of microbial stimuli.

The initial stimulus for the formation of transition zones and then reaction zones appears to be injury of nearby cells caused by wounding or infection. The response is measured; ie, it keeps pace with the margin of wounded or infected tissue rather than perpetuating itself throughout the entire sapwood. Investigation of the biochemical mechanisms for triggering and controlling this response should be a challenging, and perhaps rewarding, area of research.

**Antifungal compounds.** The accumulation of antifungal compounds in a reaction zone has provided evidence that this tissue constitutes a defense mechanism. Some of the major points of evidence are presented in the examples below. A more comprehensive coverage of this topic, including some of the anomalous results obtained by different bioassay techniques is available elsewhere (29) and in Hart and Shrimpton (20), the preceding symposium contribution.

The term reaction zone first was applied to a necrotic tissue enriched with oleoresin and phenols produced in advance of *F. annosus* infection in sapwood of loblolly pine (*Pinus taeda*) (45). The formation of similar tissues were described in other pine species in response to *F. annosus* or other injurious stimuli (12,28,38).

Oleoresin (about 30–40% dry wt) probably flowed into this zone passively upon the death of the thin-walled epithelial parenchyma which maintain oleoresin under pressure in a comprehensive resin duct system. Oleoresin is largely composed of resin acids dissolved in volatile terpenes (35). Phenols (up to 2%, dry wt), largely pinosylvin in early infections (45), probably were produced during the necrobiotic metabolism of the transition zone described above.

**Evidence for reaction zone formation.** The following points serve as evidence that reaction-zone formation is a function of the hosts' capacity to respond and that the accumulated compounds in this tissue are responsible for a dynamic mechanism of host resistance:

*In vivo observations.* One year after inoculation, *F. annosus* was isolated an average of 38.6 cm from inoculum dowels in two trees that died 4–6 mo prior to harvest and an average of 4.5 cm in comparable trees that remained alive. Reaction zones were present around all inoculations in living trees but absent in the dead trees except in control positions inoculated with sterile dowels (45). This last point, as well as others (30–32,39,45,59) were taken as evidence that decay fungi can slowly degrade reaction-zone constituents, but that living trees are capable of a continued response.

A significant negative correlation was obtained between the extent of infection of Corsican pines inoculated with *F. annosus* and their pinosylvin content (38).

The characteristic shape of the reaction zone; ie, greater penetration of decay fungi with increasing distance from the cambium (45,64), indicates that the rate of physiological activity is related directly to resistance. This was further substantiated by greater infection and a longer lag period in reaction-zone formation during the dormant season than during the growing season (33,45). Resin flow also was greatest in trees of increasing dominance and during the growing season (14,15). Finally, roots of dominant trees were invaded by *F. annosus* at a slower rate than roots of suppressed trees, but only when these roots remained attached to the tree (14,34).

*Decay tests.* Acetone-extracted reaction zone and incipiently decayed wood (which was resin soaked, but contained substantially less pinosylvin than did the reaction zone) were decayed significantly more by *F. annosus* than were their nonextracted counterparts (45). Wood samples naturally or artificially impregnated with resin acids were decayed significantly less by two decay fungi, particularly by the white rot fungus, *Coriolus versicolor,* than were their nonimpregnated counterparts (21).

*In vitro bioassays.* Inhibition of *F. annosus* and other decay fungi by pinosylvin and oleoresin or some of their components was generally obtained by a variety of bioassay techniques. Even though the effective dosages of these compounds varied considerably in some of these tests (29), the bulk of evidence supports the view that pinosylvin and some constituents of oleoresin are inhibitory to decay fungi at concentrations that occur in vivo.

A reaction zone also was described in Norway spruce as a nonspecific response to several decay fungi and to mechanical injury. This reaction zone contained a disproportionate amount of phenols, particularly the lignan, hydroxmatairesinol (up to 6% dry wt as compared to less than 0.5% in uninfected heartwood). The reaction zone in interior sapwood of spruce, unlike that in pine, was not resin soaked. This could be due to the less extensive resin duct system and thicker-walled epithelial cells in spruce than in pine (46,47).

The most convincing evidence for the presence of inhibitors in this reaction zone was the fungistatic effect of its filter-sterilized, expressed sap on *F. annosus.* Fungal growth, furthermore, was progressively greater on similar extracts from decayed wood, sound heartwood, and finally sound sapwood which supported luxuriant growth (46). The identity of compound(s) responsible for this inhibition is unclear. Results of an in vitro bioassay indicated that hydroxymatairesinol was the most inhibitory of three reaction-zone lignans that were tested (inhibition was 25, 30, and 40% of controls for concentration of 0.1, 0.2, and 0.4%, respectively) (47). A highly significant correlation also was found between the lignan content of wood samples and their inhibitory effect on *F. annosus* (1). In another bioassay, however, inhibition by hydroxmatairesinol was not detected in concentrations up to 2%, but the lignan, liovil, and another phenol found in the reaction zone, 4-methylcatechol, completely inhibited *F. annosus* at concentrations of ~0.1% and ~0.005%, respectively. It was suggested that synergism may occur in reaction zones among the numerous phenolic compounds that separately are relatively weak inhibitors (37). Inhibition of several extracellular enzymes of *F. annosus* by reaction-zone extracts was demonstrated in vitro (26).

The reaction zone in spruce was more alkaline than were neighboring tissues; eg, about pH 8.0 as compared to pH 5.5 for expressed sap from sound sapwood and incipiently decayed wood (46). This probably was due to the accumulation of inorganic

carbonates (27,46). The significance of this elevated pH is that *F. annosus* is inhibited substantially at levels above pH 7.0 (40).

**Mineral content.** Analyses of spruce xylem demonstrated that the mineral content of the reaction zone and decayed tissues was substantially higher than that of sound sapwood. Particular increases were noted in potassium, calcium, magnesium, and manganese (1,27,46). Elevated mineral contents which were related to higher pH values also were reported in discolored and decayed tissues of several other tree species (61).

In an earlier report (13), mineral accumulation was observed in decayed wood and it was suggested that fungi may selectively accumulate certain elements. The more recent studies cited above, however, show that mineral accumulation can occur well in advance of fungal penetration; ie, in the reaction zone and even in the transition zone (1). Furthermore, initial increases in potassium concentration in wounded tissue were not related to uptake of that element from the soil (4). Mineral accumulation in uninfected tissue of some tree species, therefore, could be considered as part of the hosts' general response to injury and infection

## CONCLUDING REMARKS

A nonspecific response to injury and infection by differentiated sapwood in several tree species that were studied seems to follow the sequence: sapwood → transition zone → reaction zone → infected wood. Parenchyma in the reaction zone dies in advance of fungal penetration, probably as a result of the altered metabolism in the transition zone. During necrobiosis, compounds are produced, or accumulate, which impede but may not necessarily stop invading pathogens. Reaction-zone formation, therefore, could be considered within the hosts' arsenal of defense as a dynamic mechanism of host resistance to a wide variety of injurious agents, including insects (3). The inhibitory compounds that accumulate in the reaction zone, accordingly, could be considered to be phytoalexins.

The extractives-enriched reaction zone has been studied more intensively than the less conspicuous transition zone. If, as proposed, reaction zones are the product of transition zone metabolism, then additional studies of the latter will be required to further develop understanding of reaction-zone formation. The transition zones of many lesions are large, achlorophyllous, and woody—particularly well suited for such studies.

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