

Discussion: Response of bark tissues to injury and infection¹

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Received March 9, 1983²

Accepted January 4, 1984

BIGGS, A. R., W. MERRILL, and D. D. DAVIS. 1984. Discussion: Response of bark tissues to injury and infection. *Can. J. For. Res.* **14**: 351–356.

Research of nonspecific defense processes in woody plants has focused on xylem. From these studies the concept of compartmentalization *sensu* Shigo was developed. Responses of bark to injury and infection, however, are understood poorly. This discussion summarizes evidence for the occurrence of processes similar in function to xylem compartmentalization in bark. We suggest that a developmental approach to research on woody plant responses to injury and infection could result in conceptual unity regarding structural and functional changes in both xylem and bark.

BIGGS, A. R., W. MERRILL et D. D. DAVIS. 1984. Discussion: Response of bark tissues to injury and infection. *Can. J. For. Res.* **14**: 351–356.

L'étude des mécanismes de défense des plantes ligneuses a surtout porté sur les réactions du xylème. C'est ainsi qu'a été développé le concept du compartimentage *sensu* Shigo. Par contre, les réactions de l'écorce aux blessures et aux infections sont peu connues. Nous présentons ici une synthèse des connaissances tendant à démontrer qu'il existe dans l'écorce des processus semblables au compartimentage du xylème. Nous sommes d'avis qu'une nouvelle approche dans l'étude des réactions des plantes ligneuses aux blessures et aux infections permettrait de lier les changements structuraux et fonctionnels du xylème et de l'écorce par un seul concept.

[Traduit par le journal]

Introduction

Studies of the defense systems in trees have focused on xylem and not bark (bark being all tissues external to vascular cambium). Because bark tissues shield the xylem from the environment, containment of mechanical injuries and infectious microorganisms by bark tissues is of primary importance. The integrity of normal periderm and the ability of plants to form new periderms at wounds or injuries are essential characteristics for normal plant growth and development. However, responses of periderm and other bark tissues to injury and infection are understood poorly. This discussion focuses on aspects of periderm initiation at injuries and infections and attempts to integrate observations from both bark and wood into a unified approach to understanding tree responses to injuries and infections.

Periderm and wound periderm

Periderm is a protective tissue of secondary origin which replaces the epidermis in stems and roots that have continual secondary growth. Detailed descriptions of periderm formation are available (Srivastava 1964; Esau 1965; Fahn 1967). Briefly, the periderm consists of the following: phellogen (cork cambium), the meristem that produces the periderm; the phellem (cork), the suberized protective tissue formed outwardly by the phellogen; and the phelloderm, a living parenchyma formed inwardly by the phellogen.

In most woody plants, a periderm replaces the epidermis as the protective layer within the 1st year of growth. As trees age, sequent periderms arise at successively greater depths and

thus cause an accumulation of dead tissues on the surface of stem or root. This dead part of the bark, composed of layers of tissues isolated by the periderms and of layers of inactive periderms, is called rhytidome. Rhytidome thus constitutes the outer bark and is especially well developed in older stems and roots (Esau 1965).

According to Esau (1965), natural (including first and sequent periderms) and wound periderms are basically alike in method of origin and growth. The difference between them is mainly in timing of origin and restriction of wound periderm to the place of injury. Also, wound periderm is believed to differ from natural periderms in that it is induced by a stimulus or injury, or by factors other than those responsible for the origin of natural periderms (Srivastava 1964; Akai 1959; Bloch 1941, 1952, 1953). Microbial toxins and insect saliva (Akai 1959; Oechssler 1962; Carter 1962), and hormones and auxins (Bloch 1941; Davies 1949) are believed to induce formation of wound or pathological periderms. Bloch (1953) first suggested that induction of specific responses could result from chemical substances released from dead or dying host cells alone, rather than pathogen-introduced substances in the host pathogen interaction zone.

Traditionally, wound periderm formation has been considered to be a passive defense mechanism. After the growth of an invading organism was slowed by host-mediated biochemical defenses, the final act of preventing further spread appeared to be wound periderm. The observation that wound periderm was formed at some distance from the host pathogen interface was additional support for its passive role in plant defense. Interpreted in this manner, wound periderm formation does not fall within the scope of active nonspecific defense because it is formed "after the fact" and because pathogen spread has been already delimited by other more active, less clearly defined means.

¹Contribution No. 1423 from the Department of Plant Pathology, Journal Paper No. 6788 of the Pennsylvania Agricultural Experiment Station.

²Revised manuscript received December 15, 1983.

New concepts in periderm and wound periderm

Traditional concepts of first, sequent, and wound periderms distinguish their differences only on the basis of time of origin and site of occurrence (Esau 1965; Fahn 1967; Srivastava 1964) rather than on any fundamental microanatomical or chemical differences. Research by Mullick (1971) and Mullick and Jensen (1973a) show that, by using cryofixation and chemical techniques, first and sequent periderms may be biochemically distinct. Mullick and Jensen (1973a, 1973b) were able to distinguish three periderm types in *Abies amabilis* (Dougl.) Forbes, *A. grandis* (Dougl.) Lindl., and *Tsuga heterophylla* (Raf.) Sarg.: brown first periderm (BFP), reddish-purple sequent periderm (RSP), and brown sequent periderm (BSP). The distinctions are based on 15 characteristics revealed by cryofixation and various modes of illumination including fluorescence microscopy. Although BFP and BSP were found to be similar in all 15 characteristics, RSP was distinctly different and unique. The term first periderm became functionally incomplete with these findings.

Further field observations on wound and pathological periderms, periderms formed at abscission zones, old resin blisters, and rhytidomes of *A. amabilis*, *A. grandis*, *T. heterophylla*, and *Thuja plicata* Donn established that all these periderms are of the reddish-purple sequent type rather than the brown first or brown sequent types. The reddish-purple periderms at the sites mentioned above, like the usual reddish-purple sequent periderm (RSP) in the preceding paragraph, were found abutting necrotic tissues and were identical with RSP in cryofixation and chemical characteristics. Because of the biochemical equivalence between the reddish-purple periderms at rhytidome and wounds, Mullick and Jensen (1973b) proposed that these constitute one category, the necrophyllactic periderms (NP). These periderms arise whenever a periderm forms after death of cells and their main function seems to be protection of living tissues from the adverse effects associated with cell death. Brown first periderm and brown sequent periderm constitute another category, the exophylactic periderms. This second category of periderms has a common function of protecting living tissues against the external environment (Mullick and Jensen 1973b). Further support for this terminology comes from earlier studies where similar observations were made on 40 species representing 13 genera of conifers (Mullick 1971).

A newly recognized tissue essential for necrophyllactic periderm formation

Most studies on wound periderm formation have been carried out on agricultural crop plants such as potato, sweet potato, or sugar beet, where injured surfaces become suberized and periderm forms below the suberized surface 1–2 days after injury. Studies by Mullick (1975) and Soo (1977) have shown that necrophyllactic (wound) periderm develops internal to a tissue that is nonsuberized and impervious to water. The tissue is delimited histochemically with the F–F test developed by Mullick (1975). The test is based on penetration of 2% FeCl₃ followed by 4% K₃Fe(CN)₆ through the bark over a 6-day period. Formation of this newly recognized tissue layer, nonsuberized impervious tissue (NIT), precedes formation of necrophyllactic periderms and may provide the environment necessary for NP formation in tissues internally abutting NIT. The presence of NIT is also a marker for distinguishing NP from exophylactic periderms. The formation of NIT in the absence of injuries, e.g., at rhytidome, as well as in the presence of injuries, e.g., insects, diseases, and mechanical

wounds, suggests that NIT production is a nonspecific inherent process. Nonsuberized impervious tissue formation may also be the physiological basis of host response to bark diseases (Mullick 1975). Development of NIT and its intimate association with NP suggests a more active role for NP in nonspecific defense.

Initiation of NP in vigorous plants probably occurs within hours after mechanical injury (Mullick 1977). With biotic pathogens, however, NP may or may not be initiated based on the ability of the pathogen to interfere with or delay initiation. Host responses exhibit a broad range of temporal variability in NP formation rates depending on pathogen virulence and aggressiveness, host susceptibility as influenced by age and growing conditions, and the influence of environment on all the above factors.

Histology of necrophyllactic periderm initiation

Our work with *Populus* hybrid NE-388 and the stem canker organism, *Cytospora chrysosperma* (Pers.) Fr., serve well to illustrate the sequence of events and chemical characteristics of tissues formed after inoculation or wounding. Part of this description has been given in Biggs *et al.* (1983); although, more sophisticated histochemistry and fluorescence microscopy has resulted in the present reevaluation of their original results. Investigations in progress by the senior author on wound responses of cultivated peaches, cherries, plums, and several southern Ontario hardwoods appear to support the general nature of nonspecific defense processes in both angiosperms and gymnosperms; although some fundamental differences may exist regarding the nature of impervious tissues (Biggs 1984).

One-year-old stems of the *Populus* hybrid were divided into cuttings approximately 15 mm in diameter. Five weeks after planting, a 7-mm (diameter) bark disk was removed with a cork borer to the depth of the cambium. The wounds were inoculated with mycelium of *C. chrysosperma* or treated as checks by substituting plain agar.

During the first weeks after wounding or inoculation, purple-pigmented zones formed on the inner surface of the bark surrounding the point of stimulation. In inoculated plants the zones appeared as a series of longitudinally oriented concentric ellipses. In wounded plants, there was usually a single circular pigmented zone 2 to 3 mm beyond the margins of the wound.

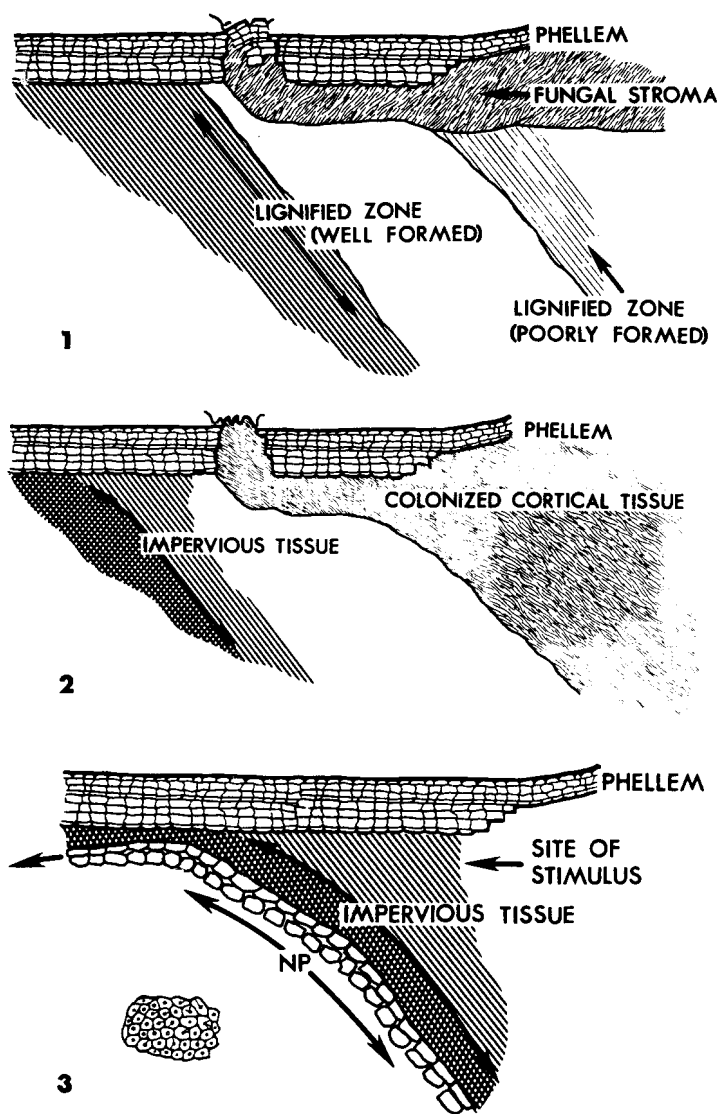
These pigmented zones can be characterized using various histochemical tests. The walls of cells comprising the tissue are characterized by the following: (i) pale blue or yellow–green autofluorescence with Leitz filter blocks A or H2, respectively; (ii) positive reactions with phloroglucinol + HCl, orcinol, Schiff's reagent before periodic acid oxidation, and 2% aniline; (iii) negative reactions with the Maule and chlorine–sulfite tests, Sudan IV, 1% Nile blue, Hoephner–Vorsatz reagents, aniline blue, and lacmoid; (iv) perviousness to the F–F test solutions; and (v) insolubility of cell wall material in cold and boiling water, 95% ethanol, sulfuric acid, cold nitric acid, cold aqua regia, and 0.5 M sodium hydroxide.

Although Bloomberg and Farris (1962) observed lignin formation after wounding in *Populus* (phloroglucinol test only), our additional histochemistry provides evidence that walls of reacting cells do not contain typical angiosperm lignin (i.e., lignin with guaiacyl and syringyl units (Srivastava 1964)). Joseleau *et al.* (1977) have shown that the lignin first laid down in angiospermous wood fiber cells (in the cell corner – middle lamella regions) has a lower syringyl content than

that laid down subsequently. As a result, younger tissues contain higher concentration of guaiacyl lignin and a correspondingly lower methoxyl content. Similar patterns of lignification (using the phloroglucinol test, Maule reaction, and extraction of wallbound phenolic acids with 0.5 M NaOH) have been observed in stone fruit bark tissues wounded or inoculated with *Leucostoma personii* and in several species of mechanically wounded hardwoods (Biggs (1984). Patterns of lignification are particularly important in light of recent evidence which indicates that, in xylem, antifungal extractives may be bound to lignin (Hart and Shrimpton 1979).

The formation of the zone of lignified cells was associated with a decline in the growth rate of the pathogen in host tissues. During the 1st week after inoculation, the pathogen easily penetrated and colonized sequent lignified zones (Fig. 1). Lignified zones did not strongly autofluoresce and reacted weakly with phloroglucinol + HCl prior to colonization. However, in subsequent weeks, pathogen growth slowed and the fungus appeared to grow out through the outer bark in the areas between poorly formed and well-formed lignified zones (Fig. 1). Prior to initiation of NP, the last-formed lignified zone developed impervious qualities detectable with the F-F test (Fig. 2). Pervious and impervious lignified zones were morphologically inseparable. Both were derived from the hypertrophic dedifferentiation of preexisting cells. The F-F test was used to distinguish between the two tissue types; however, more sophisticated tests for suberin than those used by Mullick have demonstrated a suberinlike inner lining, approximately 0.5 μm thick, in a layer of lignified cells immediately abutting regenerating phellogen. The lining, although often difficult to detect, is characterized by the following: (i) demonstrable sudanophilia based on repeated destaining and restaining; (ii) red fluorescence with Leitz filter combination H2 after Sudan IV treatment; (iii) silvery-white fluorescence with phosphine and Leitz filter combination A; (iv) elimination of sudanophilia by concentrated sulfuric acid; (v) negative reaction with fuchsin sulphurous acid; (vi) violet autofluorescence with Leitz filter combination A; and (vii) violet autofluorescence after treatment with phloroglucinol + HCl. The last test quenches autofluorescence in all lignified tissues but does not affect autofluorescence of the suberized inner lining. Results are identical with frozen or chemically fixed tissues. There is some question regarding the importance of intracellular suberization as described by Scott (1950). Thin suberinlike linings on the inner surface of cells in the impervious zone were described and dismissed as artifact by Mullick (1975). In addition, suberinlike linings were described in histopathological studies of leaf scars of olive (Hewitt 1938). This aspect requires a battery of more refined histochemical tests than those used by Mullick and more detailed biochemical analyses such as those described by Kolattukudy (1980). New findings on the structure of suberin have shown the importance of phenolic compounds and it has been suggested that microbial degradation of suberin may release fungitoxic phenolics from suberin layers (Kolattukudy 1980).

Necrophyllactic periderm initiation was detectable 2 or 3 days after the onset of imperviousness in poplar and occurred in tissues immediately internal to and abutting impervious tissue (Fig. 3). Impervious tissue was unquestionably a prerequisite to NP formation. Because imperviousness developed in lignified zones, and lignified zones were initiated soon after wounding or inoculation with *C. chrysosperma*, it follows that processes leading up to and including NP formation were



FIGS. 1-3. Schematic diagrams of the tissues formed during phellogen regeneration in *Populus* bark following inoculation with the weakly parasitic fungus *Cytospora chrysosperma*. Fig. 1. Movement of fungal stroma through poorly formed lignified zone and growing through the phellem away from well-formed lignified zone. Fig. 2. Impervious tissue forms in inner region of lignified zone. Fig. 3. Necrophyllactic periderm (NP) forming internal to and abutting the previously formed zone of impervious tissues.

actively involved in nonspecific defense processes. Development of impervious tissues appears to be of primary importance in the process of phellogen regeneration.

The general occurrence of NP and impervious tissue in woody plants

Observations have been made which suggest that NIT and NP are common features of many woody plant genera (Table 1). Soo (1977) described the general occurrence of NIT and NP in 10 species from the four families in the Coniferales and from five angiosperms in the Floriferae. The senior author has demonstrated impervious tissues in 15 angiospermous genera from nine woody plant families, although their nonsuberized nature appears questionable (Biggs 1984).

A review of the literature suggests that other researchers have observed tissues similar to impervious tissue and NP (Table 2), although none of the earlier work included the F-F

TABLE 1. Woody plant genera shown to possess nonsuperficialized in pervious tissue and necrophylactic periderm (Soo 1977)

Gymnospermae	
Coniferales	
Pinaceae	
	<i>Picea glauca</i> (Moench) Voss
	<i>Picea sitchensis</i> (Bong.) Carr.
	<i>Picea engelmannii</i> Parry
	<i>Pinus contorta</i> Dougl.
	<i>Pinus monticola</i> Dougl.
	<i>Pseudotsuga menziesii</i> (Mirb.) Franco
	<i>Larix occidentalis</i> Nutt.
	<i>Abies grandis</i> (Dougl.) Lindl.
	<i>Abies amabilis</i> (Dougl.) Forbes
	<i>Tsuga heterophylla</i> (Raf.) Sarg.
Taxodiaceae	
	<i>Sequoia sempervirens</i> (D. Don) Endl.
Cupressaceae	
	<i>Thuja plicata</i> Donn
	<i>Cupressus macrocarpa</i> (Hartw.) Gord.
Taxaceae	
	<i>Taxus brevifolia</i> Nutt.
Angiospermae	
Floriferae	
	<i>Pyrus</i> spp. L.
Leguminosae	
	<i>Robinia pseudoacacia</i> L.
	<i>Gleditsia triacanthos</i> Inermis L.
Aceraceae	
	<i>Acer macrophyllum</i> Pursh
Malvaceae	
	<i>Hibiscus syriacus</i> Hamabo.

test specific for impervious tissue. The list of host-pathogen combinations in Table 2 was developed from our interpretation of journal photographs and the degree of care given by the original authors to the description of morphological and (or) histochemical changes in the host-pathogen transition zone. Definitive assessment of impervious tissue and NP in these systems awaits application of tests designed to locate specific tissues.

Integration of nonspecific responses in bark and wood

It would benefit tree pathologists and physiologists to develop a model of host-pathogen interaction that provides the basis for a unified approach to studying tree diseases. Few comparisons of nonspecific responses in tissues as structurally and functionally diverse as xylem, phloem, and periderm have been attempted (Mullick 1977). Information on structural modifications of woody plant tissues is plentiful at the gross morphology level; less information is available at the cellular and subcellular levels (Shain 1979). Research on structural and functional responses of individual tissues, particularly xylem, has provided the basis for the concept of compartmentalization *sensu* Shigo (Shigo 1979). Investigation of biochemical mechanisms for triggering and controlling nonspecific responses in bark and wood should be a productive area of research in the years to come.

Development of a unified concept of nonspecific processes in trees would be greatly enhanced if it could be shown that cellular transformations and tissue morphogenesis at injuries, infections, and in natural growth patterns (leaf scars, rhytidome) were basically similar in functional purpose. One such functional similarity could be the restriction of passive diffusion of

either fungal toxins or phytotoxic metabolites produced by the host's necrotic reaction towards meristematic tissues (Biggs *et al.* 1983). The role of suberin in the restriction of passive diffusion is generally accepted (Esau 1965) and its presence in impervious tissues in bark and barrier zones in xylem has been demonstrated (Pearce and Rutherford 1981; Biggs 1984).

It may be generally accepted that a woody plant stem has three major points of weakness for potential pathogens. The first two are phellogen and vascular cambium. Interference with the regeneration of either lateral meristem could result in the eventual death of the plant. The third potential point of weakness is the apoplastic tissue of the functional xylem. Without wound-initiated modification of sapwood, any wound deeper than the bark tissues would be rapidly colonized by potentially lethal microorganisms.

In this regard, Mullick (1977) outlined three nonspecific autonomous processes, initiated at wounding or during pathogenesis, which probably occur in all woody plants possessing exophylactic and necrophylactic periderm habits. The first process is that of phellogen regeneration, which appears to be triggered whenever phellogen becomes nonfunctional, regardless of cause. Superficial injuries to the bark, deeper than one cell but not too deep, trigger only the process of NIT formation and phellogen regeneration.

Deeper injuries to the bark trigger the second process, that of vascular cambium regeneration. The second process can be triggered without direct physical injury to the vascular cambium. The question of why a certain depth of injury triggers cambial changes while an injury slightly less deep triggers only phellogen changes is not understood. However, Mullick (1977) has shown that NIT formation (phellogen regeneration) must be completed before the second process (vascular cambium regeneration) is completed.

Injuries of still greater depth are responsible for initiating the third process, blocking sapwood conduction. Studies of compartmentalization have shown that wound-initiated events in xylem occur over an extensive area and involve vessel plugging and chemical changes that act as barriers to decay (Shortle 1979). Three published articles (Dessureault and Tattar 1975; Mullick 1977; Mulhern *et al.* 1979) have demonstrated the nonconductive nature of discolored wood and a thin zone of normal-appearing wood abutting the discolored wood. They suggested that this normal-appearing nonconducting wood corresponded to the "reaction" zone of Shain (1979), the "bleached" zone of Shigo and Sharon (1970), and the "protection" zone of Shigo and Hillis (1973). However, none of these were developmental studies and direct demonstration of nonconduction in the "reaction," "bleached," and "protection" zone has not been reported. Demonstration of nonconduction, in conjunction with developmental studies for the three nonspecific processes described by Mullick, is necessary to interrelate these processes and to develop a unified approach to the study of woody plant responses to pathogens.

Conclusions

The purpose of this discussion has been to promote awareness of some new concepts in bark responses to injury, infection, and rhytidome formation and to encourage a more unified "whole plant" approach to related investigations. Much work is needed to understand the basis of cambial regenerations and the formation of impervious or nonconductive tissues. It is quite possible that research in this area could alter traditional concepts of plant defense mechanisms. For example, whenever the

TABLE 2. Host-pathogen combinations which exhibit similarity to the nonspecific host responses described for phellogen regeneration

Host	Pathogen	Reference
<i>Juglans nigra</i> L.	<i>Nectria galligena</i> Bres.	Ashcroft 1934
<i>Castanea dentata</i> (Marsh.) Borkh.	<i>Endothia parasitica</i> (Murr.) Anderson	Bramble 1934
<i>Populus tremuloides</i> Michx.	<i>Macrophoma tumefaciens</i> Shear Lichens Mechanical injury (wind)	Kaufert 1936
<i>Oleo europa</i> L.	<i>Pseudomonas savastanoi</i> Smith	Hewitt 1938
<i>Malus pumila</i> Mill.	<i>Nectria galligena</i> Bres.	Crowdy 1949
<i>Populus</i> hybrids	<i>Cytospora chrysosperma</i> (Pres) Fr. <i>Dothichiza populea</i> Sacc. & Briard	Butin 1955
<i>Pinu strobus</i> L.	<i>Cronartium ribicola</i> Fisch.	Struckmeyer and Riker 1951
<i>Populus</i> hybrids	Mechanical injury (heat)	Bloomberg and Farris 1962
<i>Pinus echinata</i> Mill. (roots)	<i>Phytophthora cinnamomi</i> Rands	Jackson and Hepting 1964
<i>Populus trichocarpa</i> Torr & Gray	<i>Macrophoma tumefaciens</i> Shear	Zalasky 1964
<i>Populus tremuloides</i> Michx.	<i>Ceratocystis fimbriata</i> Ell. & Halst.	Zalasky 1965
<i>Vaccinium</i> spp.	<i>Botryosphaeria corticis</i> (Tode) Fr.	Milholland 1970
<i>Malus pumila</i> Mill.	<i>Valsa ceratosperma</i> (Tode) Fr.	Tamura and Saito 1982

phellogen (or vascular cambium) is rendered nonfunctional, the process of its regeneration is triggered. The ability of the pathogen to interfere with this process, or the effects of environment on the process, could determine resistance or susceptibility (Mullick 1977). Hence, the regeneration process is also the defense process; otherwise, if there is a specific defense, it would coincide with phellogen regeneration and the two would be empirically indistinguishable. Quoting Mullick (1977) "... it should be possible to delineate how various environmental and edaphic factors affect these processes in the predisposition of hosts to attack, and how each pathogen specifically affects these nonspecific responses." It is time for a unified approach.

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