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COMPARTMENTALIZATION: A CONCEPTUAL FRAMEWORK FOR UNDERSTANDING HOW TREES GROW AND DEFEND THEMSELVES[1]

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INTRODUCTION

The purpose of this chapter is to describe a conceptual framework for understanding how trees grow and how they and other perennial plants defend themselves. The concept of compartmentalization has developed over many years, a synthesis of ideas from a number of investigators. It is derived from detailed studies of the gross morphology and cellular anatomy of the wood and bark of roots and stems in healthy angiosperms and gymnosperms. It is based on research in tree physiology and the chemistry of wood and bark. It is founded on observations of trees injured in the field by wind, snow, ice, fire, animals, and insects, as well as during pruning, coppicing, sugaring, and other forest and orchard management practices. It is based on experimental studies of natural and artificial wounds with and without controlled inoculations with selected pathogenic and saprophytic microorganisms. These microbes have included wood-decaying Basidiomycetes and Ascomycetes, wood-staining Ascomycetes and Fungi Imperfecti, canker fungi, and a myriad of wood-inhabiting bacteria. The end result of all these studies is an integrating concept that involves defenses laid down by trees prior to injury and defenses laid down by trees after injury. These defenses explain many of the visible patterns of

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lecay and discoloration in the roots and stems of living trees. They are mportant because they explain the very longevity and capacity of perennial lants to survive countless wounds and subsequent development of even more ountless pathogenic and parasitic microorganisms.

Let us begin our discussion with a brief introduction about how the cells and ssues of healthy trees are organized and compartmented.

IOW HEALTHY TREES GROW BY COMPARTMENTATION

rees are highly compartmented, woody, perennial, shedding plants. They are ften long-lived and massive. In a sense trees are multiple plants, because the ew set of cells produced in every growth period envelops the cells laid down in arlier growth periods. The repeated development of long cones of cells over ach other results in the concentric pattern of the trunk, branches, and roots. he vascular cambium is the cell generator. The cells formed on the outer side f the cambium differentiate into phloem, which transports photosynthate ownward from the leaves. The cells formed on the inner side of the cambium e xylem, which transports water and other materials upward. Two basic types f cells are formed by the cambium: those that have their long axis oriented ong the stem or root, and those that have their long axis at right angles to the em or root. In wood, some of the longitudinal cells differentiate into vessels, acheids, fibers, and axial parenchyma. Aging begins as soon as the cells are rmed. The vessels and tracheids become functional in water transport after eir living contents die, a few days or weeks after they are formed. Fiber cells ve thick walls. Parenchyma cells are thin-walled and contain living cyto- asm for a few to many years. Storage of energy reserves is in the parenchyma.

The highly ordered arrangement of cells in a tree defines its compartments. he cells with their strong walls of cellulose and lignin make up the basic mpartment. The group of wood cells within longitudinal sheets of ray renchyma make up the next order of compartmentation. The growth rings are e next order, and the nonwoody parts that are normally shed are also mpartments.

Materials stored in the living parenchyma cells provide the substrates for nthesis of antimicrobial phytoalexins. These substances are synthesized by e parenchyma cells in advance of and in response to wood- and bark-invading icroorganisms and provide a chemical boundary that retards the spread of ese microorganisms.

When trees are wounded, the cambium responds by laying down a thin layer unique cells called a barrier zone (see Figure 1B and 2). This zone separates e normal tissue formed prior to wounding from the normal tissue formed after unding. The cells of the barrier zone are impervious to most wood- and

bark-inhabiting fungi and bacteria. Thus, the barrier zone restricts the development of decay and discoloring fungi in tissue formed prior to wounding, as shown in Figure 2.

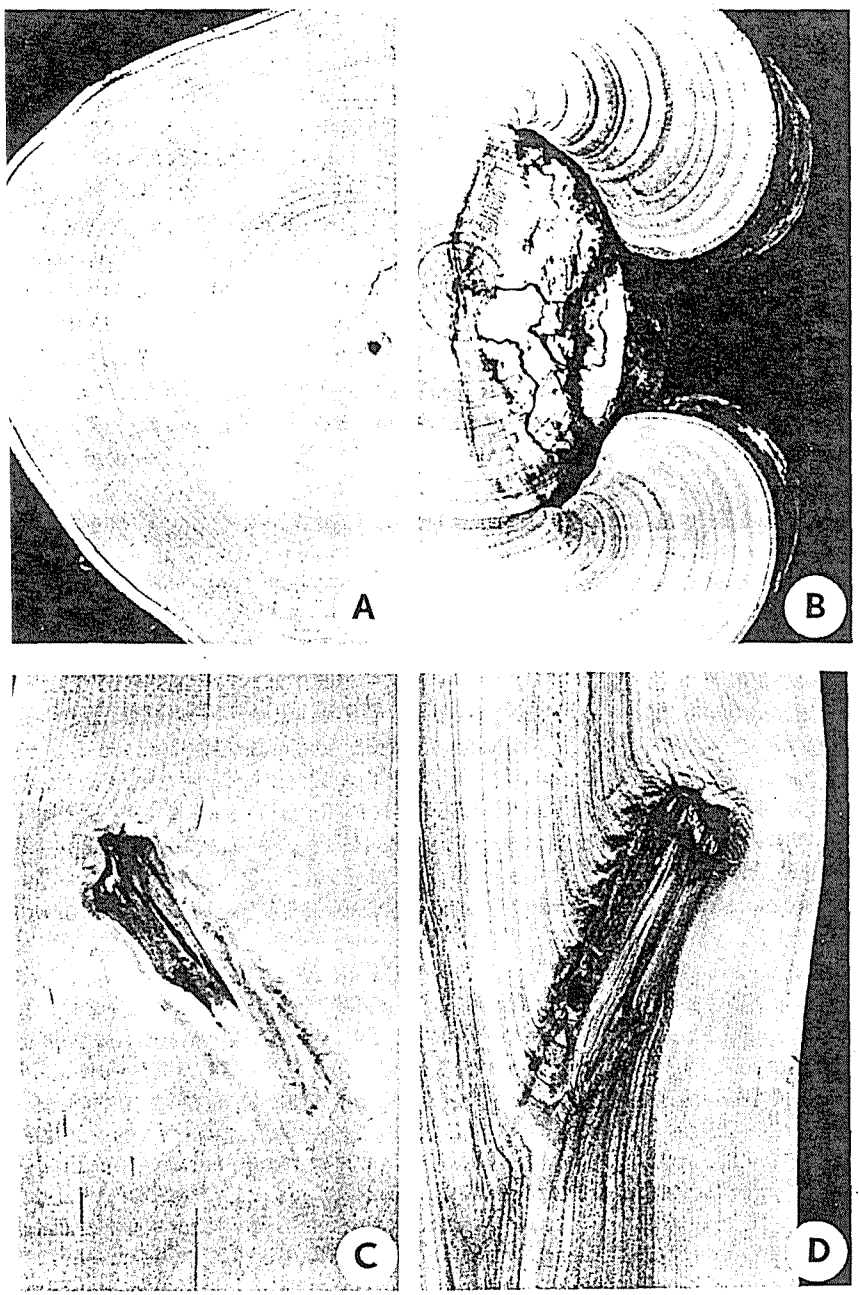
Biological aging is the intrinsically controlled changes of an organism or its parts over time. Cell contents change and are removed, cell walls thicken, and transport and storage functions decrease until they no longer are active. Wood vessel cells die as soon as they become functional in water transport. When wood has aged to the point where the parenchyma cells also no longer contain living contents, and no transport or storage takes place, we call it heartwood. Heartwood continues to provide mechanical support and retains some residual enzyme activity that becomes evident after the injury or infection of this "dead" tissue. The nature of heartwood defense reactions is poorly understood.

Aging of wood takes place in both axial and radial directions. As new cones of wood envelop the older cones, aging occurs along the radius of tree stems. Cross sections of trunks are often used to show the aging process from cambium inward to the pith. But aging also occurs in an axial plane as trunk tissues connected with the dying branches age. This type of aging is poorly understood. All trees have branches, and branches age and die on all trees. The axial aging processes then are superimposed over the radial aging processes (Figures 1 and 3).

A unique feature of trees is their highly ordered, woody, compartmented structure (33, 117, 160). Wood is a highly ordered arrangement of cells in all gradations of aging (33, 42, 116, 160). The wood and bark of trees store and transport food materials, support the tree, and maintain a defense system. Sapwood performs all four of these functions. Heartwood is wood that has been altered as a result of a genetically controlled aging process.

In wood there are connections among cells that contain protoplasm, called the symplast (33, 160). Cells with protoplasm that border the vessels are contact cells (23, 33). They appear to regulate the plugging of vessels in woody and nonwoody plants (33, 152). There are many variations on the theme of vessel plugging and pit closure (11, 101, 160). Many changes take place within the cells that contain protoplasm, as storage materials are changed to materials that inhibit the spread of pathogens (42, 54, 89). The tree must use its energy reserves for growth, reproduction, and defense (66, 154). Very few attempts have been made to add energy-releasing materials to trees (151).

The tree has many built-in boundaries, a maze of cellular walls that make it difficult for pathogens to spread. The woody structure and its defense system may persist for hundreds or even thousands of years. Every growth period the tree generates new cells in new spatial positions (117). Under some conditions, some trees regenerate bark where injury has occurred (24). The new bark cells may be in the same spatial position as those that were injured. In every growth period, the tree also sheds some of its parts (50, 145). The regulation of these



generating and shedding processes is very poorly understood. Trees usually shed aging or injured nonwoody parts (145) and some woody parts after aging, injury, and death (10, 118).

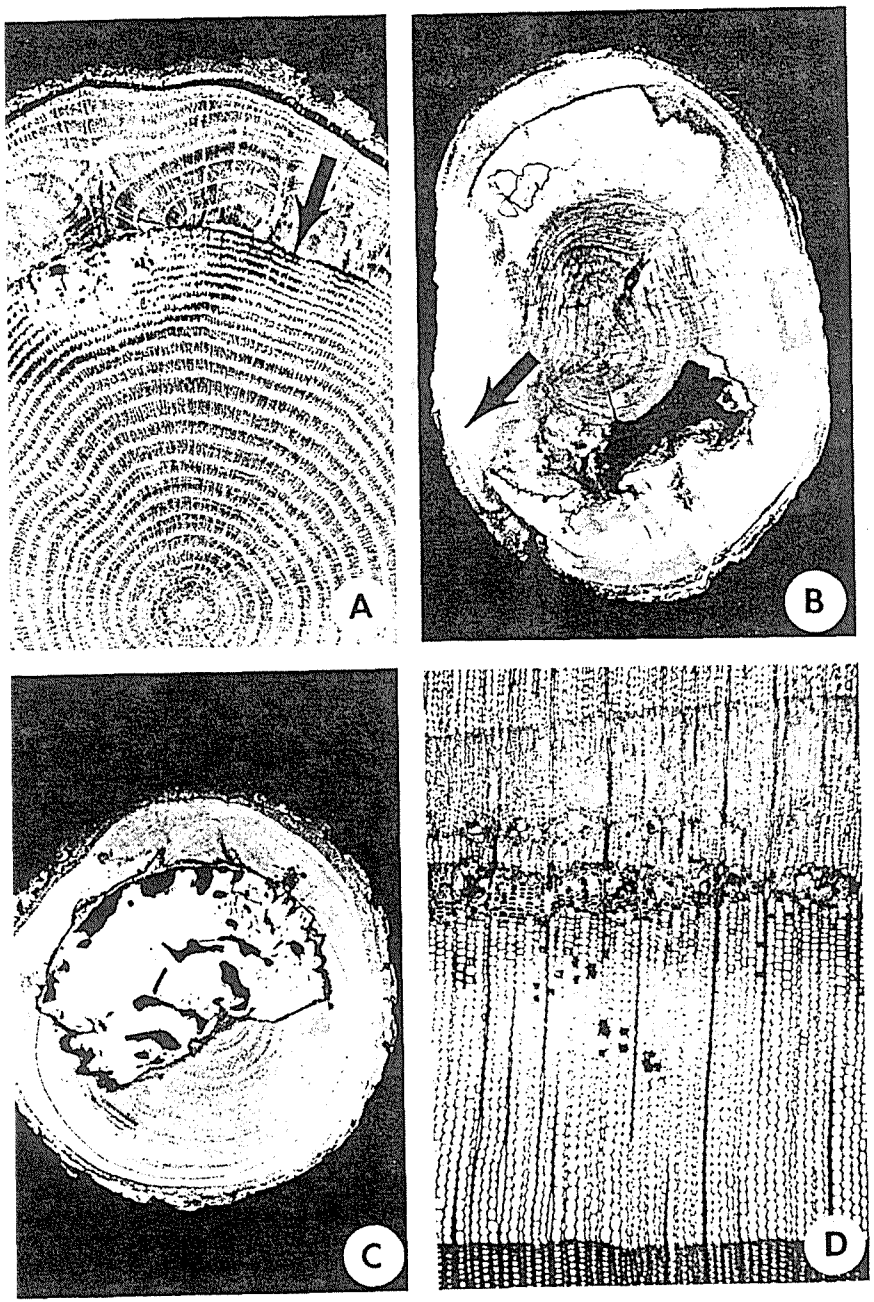
Trees do not cast off dead and dying parts. Trees form boundaries between dying parts and the healthy frame of the wood and the inner bark. Trees, like other organisms, die in three basic ways: mechanical disruption, dysfunction, and infection. Many organisms that attack wood interfere with both normal functioning and mechanical support; thus, many wood-inhabiting microorganisms are pathogens. They are also parasites because they derive energy and substance for their development from the tissues of the host tree. Tree decay is a disease caused by microorganisms that are both pathogenic and parasitic (65).

HOW INJURED TREES PROTECT THEMSELVES BY FORMING BOUNDARIES

Compartmentalization in trees is a defense process by which boundaries are formed to isolate the injured tissues and thus resist the spread of pathogens. In wood, the process has two parts: (*a*) chemical boundaries formed within tissues present at the time of injury and infection as tree and pathogens interact; (*b*) chemical and anatomical boundaries formed after infection as the living cambium responds to form a barrier zone between the tissues present at the time of infection and the new tissues (111, 117). In bark there are also two types of processes: (*a*) chemical and anatomical boundaries that are formed once and may or may not resist the spread of pathogens; (*b*) chemical and anatomical boundaries that are formed several to many times in response to repeated breakouts of pathogens (15, 72, 142, 146).

Boundary setting after injury and infection can be very beneficial to the tree as long as the volume of cells remaining or newly generated outside the boundaries is sufficient for normal functions (11, 117). The process is det-

Figure 1 Healthy and injury-altered wood in yellow birch, *Betula alleghaniensis* Britt. A: Cross section of a birch shows 50 growth rings of nondiscolored wood. Many species of trees do not have a central core of colored heartwood. B: A 13-year-old wound on a birch. The discolored and decayed wood is confined to the wood present at the time of injury. Note the dark boundaries that separate the discolored wood from the healthy wood within the core present at the time of injury. The decayed wood developed within the column of discolored wood. C: Longitudinal view of a well-closed branch stub. Boundary setting was very effective and the discolored wood associated with the stub was confined to the inner core of the stub. A tree with this type of branch stub would have a cross sectional view similar to that shown in A. D: Longitudinal view showing a branch stub and a central core of discolored wood. The trunk wood was connected to several branches that died at the same time on the tree, aged and discolored. Birch trees with columns of discolored wood associated with such branches will have a discolored central core. Columns of discolored wood associated with branches and wounds may also develop within heartwood of trees that do form a colored central core of heartwood.



rimental when too much of the tree is walled off and normal processes do not function properly (125, 127).

The concept of compartmentalization attempts to unify facts about boundary setting in trees. It helps not only to clarify and strengthen many notions about tree diseases but also to clarify how a tree is constructed and how it responds after injury and infection.

The best way to understand any organism is to apply a controlled stimulus and then observe and measure the response. Many dissection studies and wounding experiments on trees have shown that they respond in an orderly way to injury and infection (9–11, 39, 52, 102, 103, 107, 109, 119, 120).

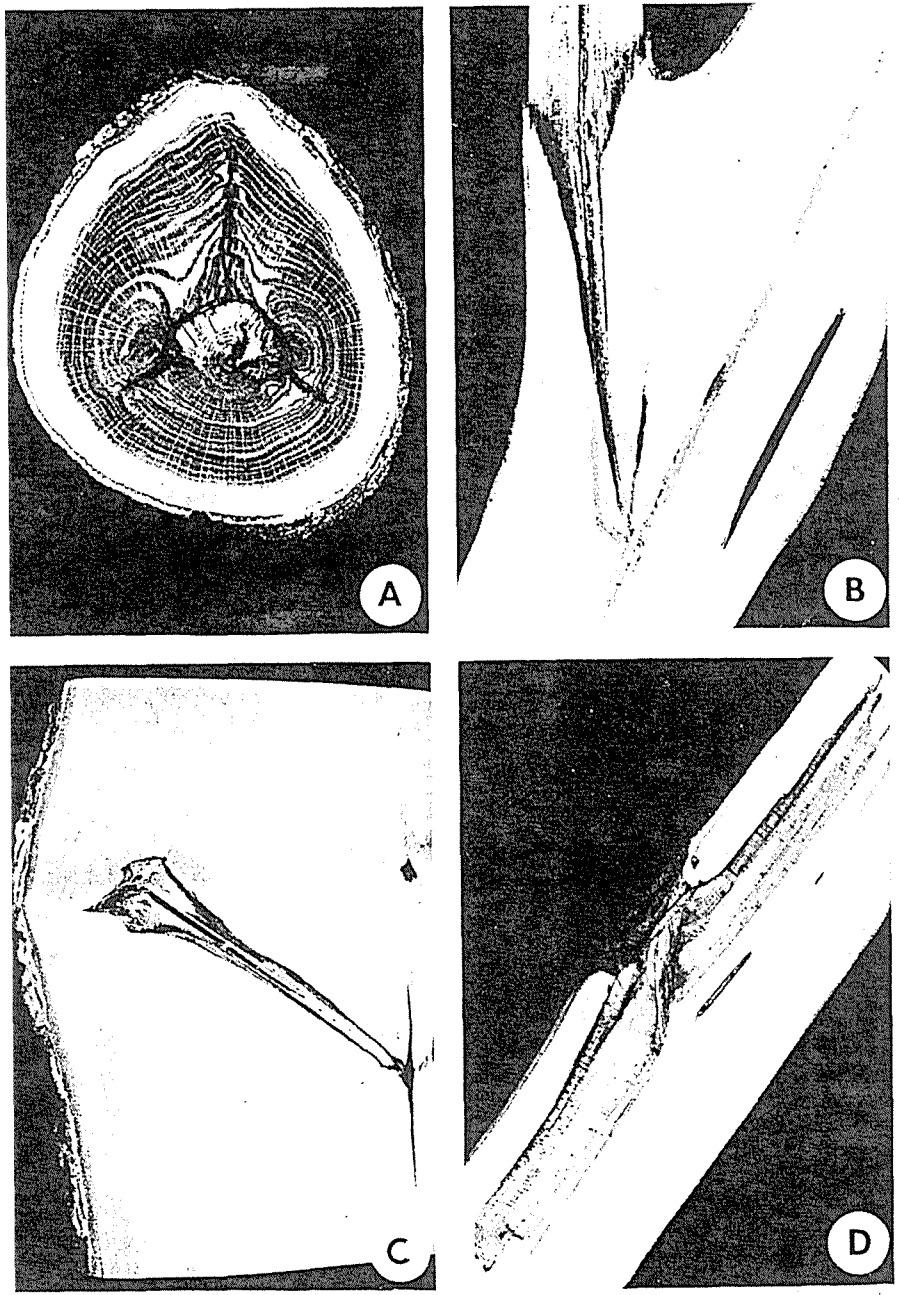
Survival of a tree after injury or infection depends greatly on its ability to compartmentalize pathogens. The survival and virulence of pathogens depend on their ability to occupy as much tissue as possible before they are compartmentalized. Both interact in an ever-changing environment. Because trees cannot move, many types of wounds accumulate on or in them during their long lives. Compartmentalization makes long-term survival possible, even after hundreds or even thousands of infections. Compartmentalization is a defense process that has the potential to be effective for millennia. Otherwise, trees would not be thousands of years old, full of rot, and still growing.

The aim of this paper is to clarify compartmentalization by focusing on boundary setting.

EARLY STUDIES OF COMPARTMENTALIZATION

Boundaries separating decayed wood from sound wood were first shown in figures and diagrams by Hartig in 1887 (37) and Frank in 1895 (27). In 1913, Küster (51) gave a detailed account of tissue alterations after wounding in trees. In addition, he discussed work on the subject by DeVries that preceded even Hartig's. In 1935, Hepting (39) further strengthened the concept of boundaries during his dissection studies of fire-wounded trees in the Mississippi Delta:

Figure 2 Compartmentalization in trees is a boundary-setting process. A: Decayed wood in red oak, *Quercus rubra*, associated with a 9-year-old wound. A barrier zone (arrow) separates decayed wood from sound heartwood. A boundary of discolored heartwood surrounds the decayed wood. B: Compartmentalization of hollows and decayed wood in a tropical hardwood, Maria, *Calophyllum brasiliense*. Note the barrier zone that formed far beyond the wound (arrow). Boundaries of discolored heartwood surround the columns of decayed wood. The decayed wood did not spread into the center of the tree. The inward spread stopped where other older boundaries had formed. The inward spread of the two large columns stopped at different positions. C: A strong barrier zone separates decayed wood from sound wood in this sweetgum, *Liquidambar styraciflua*. The barrier zone formed completely around the trunk. Usually this does not happen. D: A thin section of a spruce, *Picea abies*, showing a portion of a barrier zone within a growth ring. The zone may be strong in a protective way, but cracks usually begin at such zones when other pressures are applied.



A characteristic of decay in young Delta hardwoods that is particularly striking in red gum, is that decay which sets in subsequent to fire-scarring practically confines itself to the cylinder of wood extant at the time of scarring. For example, if a gum tree is five inches in diameter at the time it is scarred, and composed entirely of sapwood, the decay which follows will remain confined to that five-inch cylinder, not spreading appreciably into the sapwood layers laid down after scarring.

A year later, Hepting & Blaisdell (40) described the protective zone on the surface of fire scars on red gum, *Liquidambar styraciflua,* and persimmon, *Diospyros virginiana*. The zones appeared to protect the underlying wood from decay.

While studying the anatomy of elms infected with *Ceratocystis ulmi,* Buisman (20) described a barrier zone (149). Shain published accounts of the chemical nature of boundaries that resist the spread of pathogens (97–99), and more recently Shortle (128, 129) has clarified some of the biochemical mechanisms involved in boundary setting.

Hepting established that boundary-setting is a response to wounding. Buisman and Shain established that boundary-setting can be a response to infection. Shain also demonstrated the chemical changes that take place during boundary setting. These papers set the stage for the development of compartmentalization as a general concept of defense.

Many other scientists have added valuable information to the on-going research on compartmentalization: Banfield (6), Sinclair & Larsen (136), Sinclair et al (138), and MacHardy (57) on boundary setting in elms infected with *C. ulmi;* Schoeneweiss (92) and Marchetti (60) on boundary setting in *Quercus* spp. infected with *C. fagarcearum;* Gerry (30), Moore (67), Sharon (100), Shortle (128), Shortle & Cowling (131), Tippett & Shigo (147–149), and Pearce & Rutherford (78) on barrier zone formation; Jewell et al (44, 46), Jewell & Spiers (45), and Jewell & Walker (47) on boundaries in rust-infected pines; Beckman (12), Beckman et al (13), Moreau & Catesson (68), and

Figure 3 The concept of compartmentalization helps to clarify many tree problems. A: Fire wounded this white oak, *Quercus alba,* when it was small and young. As the wound closed, decay developed in the wood present at the time of injury. A primary crack formed as the callus lips closed the wound. Secondary cracks formed where the first boundary stopped. When other pressures occur, such as sudden cold, the internal secondary cracks may split outward to the cambium. B: As branches die, a boundary of protective chemicals forms at the branch base. Most decay-causing fungi are not able to spread through the boundary. The dead branch is usually decayed into the protective socket, and pressures of wind and other forces cause it to fall away. C: When branches fall away, the small pocket of decayed wood within the basal socket of the branch is usually compartmentalized and does not spread into the surrounding tissues. D: When the protective chemical zone is removed by harsh, close pruning cuts, the pathogens spread rapidly into the trunk above and below the wound. This section shows the results of a 2-year-old close branch cut on a small red maple, *Acer rubrum*. The tree still sets boundaries to resist the spread of pathogens, but the pathogens are now deep within the tree.

Moreau et al (69) on boundary setting in nonwoody plants; and Mullick (72) on oundaries in bark.

THE EXTENT AND LIMITATIONS OF THE CONCEPT

Boundary setting is applicable not only to discoloration and decay in the trunks f trees (111, 117), but also to cankers (142), canker rots (105), wilt diseases 125, 149), root rots (124, 126), wetwood in roots (96), branch shedding (4, 2, 50, 118), root shedding (38, 145), bark (15, 72, 142), and rusts (45–47).

Trees cannot prevent infections after injuries or the death of tissues and rgans. Trees can only form boundaries to resist the spread of pathogens. Wounds and the normal shedding of roots (38, 145) and branches trigger the rocesses of boundary setting (111, 157, 158). Although the concept of ompartmentalization was developed mainly for xylem, it appears to be appliable also to bark (15, 72, 142). Wounds must penetrate the many layers of ark (35, 141) before they can mechanically injure the cambium or xylem. The ambium may respond to nearby injury to phloem (72). Many pathogens infect ark and stay there (21). Some pathogens become established in the bark and ıen infect wood (43, 79, 139, 150), while others infect wood first and then pread to the bark (105).

Defense by compartmentalization is not always successful. Many things can o wrong. When a tree stays alive for years after injury and infection, compartıentalization is functioning well. When compartmentalization does not funcon, parts of the tree or the entire tree will die. Given enough time or pressure :om virulent pathogens, all boundaries will fail. Compartmentalization is the *·amework* for a tree defense system. The system is unique because the ıteraction between tree and pathogens usually takes place within the tissues resent at the time of injury and infection, and the tree sets a boundary between ıese tissues and newly forming tissues. The newly forming tissues act as new trees" growing over the older "trees." This system has long-term survival enefits.

SOME PROBLEMS IN UNDERSTANDING COMPARTMENTALIZATION

Understanding compartmentalization is difficult because of some basic similarıes and differences in the responses of animals, nonwoody plants, and woody lants to injuries. Research on nonwoody plants is applicable to the nonwoody arts of trees (23, 38, 68, 69, 145), but the information on nonwoody parts is ot completely applicable to the very long-lived wood and bark of perennial lants. Much of the confusion about compartmentalization in trees centers round the terms *healing* (61) and, to a much lesser extent, *localization*.

Healing is a restoration or regenerative process by which injured and infected cells are repaired or replaced in the same spatial position. Localization is defined as the restriction of a lesion to a limited area. Sinclair et al (137) stated, "The concept of compartmentalization encompasses that of localization and in addition identifies a significant role of the vascular cambium in producing a physico-chemical barrier which surrounds all xylem present at the time of wounding." Beckman (12) gives an excellent discussion on localization as a general mechanism by which nonwoody plants, once infected, protect themselves from systemic invasion. The speed of the host reaction is an essential part of effective localization. Gäumann (29) summarized the situation succinctly: "Man is able to destroy the pathogen in many infectious diseases, whereas the plant can only localize it."

A compartment is a three-dimensional space. Compartmentation is the result of subdividing space in healthy trees. Compartmentalization is the active phase of subdividing and also *breaking* connections, interactions, or cooperation among the subdivisions in response to wounding and infection. Problems start when compartmentalization is equated with healing as a repair process, or with localization as an absolute restricting process.

Compartmentalization must be understood as a two-part process. Part 1 is the formation of a chemical reaction zone by the living parenchyma cells of sapwood and as a result of enzymatic reactions in heartwood. This reaction zone serves to retard the advance of pathogenic microorganisms in tissues that were already present at the time of the injury. Part 2 is the formation of a barrier zone by the cambium of a tree. In wood, this barrier serves to isolate or separate the tissue that was formed *inside* and therefore prior to the injury from the tissue that is *outside* and therefore formed after the injury. Each of these two parts is described in detail in the sections below.

PART 1: REACTION ZONES

A reaction zone in sapwood is a region of normal host tissue in which antimicrobial substances produced by living wood storage cells accumulate at the margin of infection. Reaction zones serve to retard the longitudinal and inward spread of pathogenic organisms that develop after wounding in the stems or roots of trees (97, 99, 129). The size of reaction zones varies from very thin to very broad depending on the virulence of the pathogen and the vigor of the host response. Usually there is a gradation of changes in the wood from the position where pathogens are present to the places where the tissues are responding (100, 120, 129). Reaction zones often can be identified by color, but the important biochemical changes may be taking place far beyond the visibly changed zone within healthy-appearing wood. In conifers, many investigators have described reaction zones that show up as resin-soaked boundaries

31, 85) or, more specifically, as monoterpene boundaries (53, 93–95). Long before wood begins to decay in a decay chamber, it shows a distinct change in electrolytic state that can be detected with a pulsed current meter (81, 130, 144). Similar changes take place in living trees.

The study of boundary setting at the biochemical level is very complex. Results depend on the stage or position of the boundary when chemicals are extracted or added for studies, and boundaries are not stationary. As the tree forms boundaries at one position, pathogens may break them down at another position. Some chemicals from boundaries may seem very effective for resistance in laboratory studies, but in the living tree the same chemicals may not be so effective (54). Chemistry interpreted within the framework of boundary setting will help to clarify many of these processes (128, 129, 134).

PART 2: BARRIER ZONES

A barrier zone is a protective tissue consisting of unique cells and formed by the living cambium of a tree in response to mechanical wounding (3, 63, 67, 70, 140) or infection (20, 58, 149) or both. In wood, barrier zones serve to isolate or separate the infected wood on the inside from the healthy wood that continues to form on the outside of the cambium after the zone is completed.

> Buisman (20) was the first to describe a barrier zone: When we study through the microscope, the cross section of a branch that a few months previous has been inflicted with *Graphium ulmi*, we can invariably observe a number of layers of flat parenchyma cells in the last annual ring that are filled with *starch*. Sometimes no more wood is formed after these starch-containing layers have developed so that the annual ring is shut off with those layers. This need not be so, however; it may also occur that after the starch-containing layers new wood is again formed. If this new wood is attacked by *Graphium ulmi*, then the flat parenchyma cells may form again.

Banfield (6) stated that "the band effectively walled off the vessels of the new sheath from the invaded vessels of the old sheath."

The barrier zone is usually nonconducting (70). In oaks, Pearce & Rutherford (78) showed suberin in the cell walls of such tissue. In conifers, the barrier zone often includes traumatic resin ducts (94, 147, 148). These observations help to explain why so few pathogens or parasitic microorganisms can penetrate a barrier zone. The zone may form in response to infection as in wilt disease (113, 125, 149). The zone may be very large and encircle the stem (70), or it may extend only a limited distance around the circumference of the tree at the time of injury (67). Factors affecting the spatial extension of barrier zone formation are poorly understood.

Jewell et al (46) used the term *reaction parenchyma* in their histological studies of *Cronartium fusiforme* in the phloem tissue of slash pine, *Pinus*

eliottii, seedlings. Later, Jewell & Spiers (45) described a resistance zone that limited the growth of *C. fusiforme*. This zone may be a type of barrier zone formed in the bark of trees.

CLOSURE OF WOUNDS BY CALLUS

Closure of wounds by callus (7, 155) takes place after wounding as new cells are generated in new spatial positions. If callus formation is considered healing, then the definition of healing must be expanded to include the generation of new cells in new spatial positions. The confusion over callus formation as a closure-healing process stems from a century of misunderstanding about the proper pruning and treatment of wounds (51, 114, 122). Trees close wounds on the outside and compartmentalize pathogens on the inside.

CODIT: A CONCEPTUAL MODEL OF COMPARTMENTALIZATION

Compartmentalization may be viewed from the functional level of boundary setting (59, 99) or from a conceptual model that describes the expanding configurations of infected tissues. The model is called CODIT, an acronym for compartmentalization of decay in trees (117). Like compartmentalization, the model has two parts. Part 1 is represented by three walls: wall 1 resists vertical spread, wall 2 resists inward (radial) spread, and wall 3 resists lateral (tangential) spread. Walls 1, 2, and 3 are built into the tree prior to injury and infection. Part 2 starts after infection, when the tree forms wall 4, which separates wood present at the time of injury or infection from new wood that forms subsequently. CODIT is a model by which arborists and forest-industry personnel can increase their understanding of compartmentalization and researchers can reexamine tree diseases (113, 115).

Wall 1 is the weakest, since the growing tree must maintain vertical transport. After injury and infection, plugs form in the vertical elements. Chemicals such as paraformaldehyde inhibit vessel plugging and increase the spread of decay-causing pathogens (152).

Wall 2 is moderately strong. When shallow wounds kill cambium, wall 2 resists the inward spread of pathogens behind the wound. If wounds are inflicted more deeply and the injuries approach older sheaths of xylem, the resisting power of wall 2 diminishes (122). When wall 2 fails to the pith, the inward spread ends.

Hollows form as wall 3, the strongest wall in part 1, fails over time and the boundaries open like a fan. If the walls fail in part 1, the tree generates new cells in new positions, and these cells are protected as the result of the second part of

he process of compartmentalization. In the CODIT model, only part 1 is resent at the time of wounding. Part 2 may not begin to form for nearly a year, epending upon the time of wounding in relation to the next surge of cambial ctivity.

In a sense, a tree is a multiple plant. It has many arenas for defense against athogens. As long as the tree generates "new trees" over the older infected nes, and keeps strong boundaries between the infected arenas, it will live for a ery long time. How well a tree does all those things depends greatly on its enetic program and its ability to generate and allocate energy. Defense onsumes much energy.

CHANGES WITHIN BOUNDARIES OF THE SAPWOOD

fter a plant is wounded, the living cells of the sapwood respond first in an lectrical way (71). Long before the microorganisms develop within the tree, ie living wood storage cells of the stem or root have responded. In many istances the region of response extends far beyond the injury. Wood within iese boundaries of response is then altered chemically and may discolor (97, 29).

Many microorganisms grow in succession within the wood surrounded by oundaries (9, 10, 16, 87, 88, 104, 157). Small, sometimes dormant popula-ons of microorganisms exist in trees with no visible wounds (86, 102, 133). fter a few days to many months (107, 109, 119), isolations from the tissues in id near wounds show much larger and active populations developing in iccessional patterns in both time and space (120, 152). Hymenomycetes are ometimes the pioneers (first in the sequence) but often they follow other iicroorganisms (132). The major point in this succession is that *many* microor-anisms are involved in an orderly sequence from wounding to wood decom-osition (9, 10, 74, 86–88, 111). The successional pattern may be the key eterminant of the rate of spread (41). When the normal patterns are disrupted, evelopment of decay may be retarded or accelerated (82, 152).

In some ways, discolored wood, especially discolored wood in early stages development, may be similar to heartwood (134), but there are many fferences (36, 116). Some distinctive features of discolored wood are its creasing concentrations of phenols, its low moisture content, and its high sistance to a pulsed electric current (134). The wood is often bleached or olored slightly. Later it may be darker than the contiguous wood.

Discolored wood may be more protective than the contiguous healthy wood 9), or it may be near the beginning of decomposition. It is wood in transi-on—a gradation. Changes in wood color after injury and infection may or may

discolored wood ↑phenols ↓ H_2O Hi resistance to pulsed current

not reflect the gradation of chemical changes (120, 132). The concept of discolored wood is straightforward, but its implications are diverse.

BOUNDARIES IN HEARTWOOD AND DISCOLORED WOOD

When wounds penetrate discolored wood, new boundaries do not form (117). When wounds penetrate heartwood, however, boundaries of a chemical nature begin to form (76, 121). Discolored wood may develop within heartwood (116, 117). When the discolored heartwood is wounded again, no new boundaries form (117).

HEARTROT AND COMPARTMENTALIZATION

Heartrot is defined as the decay of heartwood in living trees (5, 18). Shortle (129) gave an excellent summary of the heartrot concept. He defined heartrot as the decomposition of dead heartwood inside a living tree by saprobic organisms that gain entrance via wounds. Many plant pathologists do not consider heartrot a disease because the decay-causing agent is not interacting with living host tissue (18). The traditional heartrot concept states that, when all the heartwood is digested, a hollow results (18). Compartmentalization argues against this concept because it states that the spread of pathogens within living trees is confined to the barrier zone laid down by the cambium (Figure 2) and is retarded by the chemical reaction zones formed in living wood storage cells at the advancing margin of infection (Figure 3).

The heartrot concept was developed initially by researchers viewing the ends of logs, a two-dimensional view. A multidimensional view of both longitudinal and radial dissections is essential for an understanding of the processes of compartmentalization (39, 103). Hepting's work in the 1930s (39) is very important because he concentrated on longitudinal dissections. It is very difficult to understand compartmentalization by studying cross sections of trunks, especially trunks infected with canker rot fungi or perennial cankers. Yet canker rots were some of the major problems first studied by looking at cross sections (18). The ring-rot patterns caused by *Phellinus pini* are difficult to understand from cross sections because it appears that the fungus has spread outward to the cambium (16, 18), when in fact the fungus spreads from bark inward to the wood. Cross sections of wetwood areas also make it difficult to understand this phenomenon (96, 153) because the wetwood column expands as a cone. The tissues in a tree present at the time of wounding appear to form a cylinder. In fact, the pathogens spread from point of entry through the stem as

expanding cones. When a cross cut is made through a stem that has concentric cones within it, confusion results.

HOLLOWS AND HEARTWOOD

What is heartwood (17, 36, 42, 49, 56, 116)? Is heartwood a dead, nonresponsive tissue? Why does it discolor after wounding in living trees (117, 121)? Hollows are common in heartwood. Why don't fungi decay the heartwood that surrounds advanced decayed wood?

Heartwood maintains a boundary-setting defense system that is based in part on the effectiveness of barrier zones formed by the cambium in response to injury sustained when the heartwood was sapwood. Heartwood also has defenses based on residual enzymatic activity (76, 117, 121). In addition, heartwood contains extractives that impart protection (42, 158). But if it were the extractives alone that provided protection, heartwood decay patterns would be random, and they are not. The patterns of decay in the heartwood of trees are highly ordered (111, 117) and the boundary-setting models developed for compartmentalization (117) follow these patterns. Wounds usually start the processes that result in discolored and decayed heartwood. Yet severe wounds may inhibit the normal senescence processes by which sapwood is transformed into heartwood (64). How heartwood sets boundaries is still not well understood.

CANKER ROTS

Fungi that cause canker rots usually infect wood via wounds or dying branches, and then force a wedge of hard fungal tissue into the bark (105, 111, 117). The wedge material of some fungi has been termed a sterile conk (18). The tip of the wedge develops farthest into the phellem, and then the base expands until some cambium is involved and killed at the wound site. The tree responds during the growing season by setting new boundaries. This "seesaw" pattern is obvious in many canker rots (11, 117).

The canker rot fungi circumvent compartmentalization for short periods (16), but not completely or the tree would die. The wedges associated with many branch infections may coalesce in time; large portions of the trunk may die or the entire tree may die (111). The important point here is that compartmentalization is temporary. But the seesaw interaction may proceed so slowly that the fungi and the tree will live together for many years. Trees may break at the canker site and die a mechanical death.

In Douglas fir, *Pseudotsuga menziessii*, dead spots or cankers caused by *Phellinus pini* may be so large on old trees that compartmentalization seems ineffective. Indeed, it is not effective in those areas where large patches of

cambium are killed by the fungus wedges, but it is effective where boundaries separate the dead spots from living tissues.

Phellinus pini seems to infect the slightly resin-soaked wood at the heartwood-sapwood boundary in *Pinus strobus* (16, 111). The heartwood on the inner side of the infection and the sapwood on the outer side usually are not infected, and a ring pattern of decayed wood results.

PERENNIAL CANKERS

Microorganisms that induce both perennial cankers and root rots usually infect bark first and then grow into the wood (14, 43, 79, 142). Trees have no defense in the phellem beyond the phellogen (21). When an injury penetrates the phellogen, the inner bark responds (72, 83, 84). When the wound injures the vascular cambium, compartmentalization starts, and an annual canker may result as wood and bark set boundaries. Some miroorganisms may grow circumferentially within the outer bark first, without injuring the vascular cambium, and the tree apparently does not recognize the microorganisms (19, 150). When the microorganisms grow from the bark inward to the cambium, they are recognized and compartmentalization starts. Some trees produce wedges of wood outward into the bark and temporarily inhibit the growth of canker fungi (7, 19, 90, 115, 150).

In beech bark disease, the fungus *Nectria coccinea* var. *faginata* is often walled off or localized within the bark (26). In some instances, the localized dead bark patches are shed (26). But when many bark infections coalesce during a growth period and kill large portions of the trunk, the tree is overwhelmed and it may die (26).

In chestnut blight, the causal fungus, *Cryphonectria parasitica,* usually infects large portions of the bark and overwhelms the tree. The hypovirulent strain of the fungus is walled off by the tree (1, 2, 34, 150). Chestnut trees compartmentalize the normal strain of *C. parasitica,* especially when tree energy reserves are high (75). If a microorganism or its metabolic products are recognized quickly by a tree, or if the microorganisms penetrate directly to the cambium, the tree will compartmentalize it. But if a microorganism grows in the outer bark for great distances first, and then penetrates to the cambium, the results will overwhelm the tree.

A great amount of work was done by Rumbold (90) and later by Taylor (143) and Bramble (19) on the formation of wedges of xylem ray tissue in the bark of chestnut trees. The xylem ray wedges act as boundaries that inhibit the spread of *C. parasitica*. They also form a new cambium in the bark that generates new cells beyond the infected bark tissues (115). In this instance, the wedge-type resisting boundaries develop into "new trees."

The variations on the theme of compartmentalization seem almost endless.

very time the tree sets a new type of boundary, the pathogen finds a new way
break it down. This may not give trees the perfect answer, but it does give
iem the advantage of *time*. Indeed, there are very few absolutes.

UTCH ELM DISEASE

lany investigators have shown that elms set boundaries that resist the spread of
ifection by *Ceratocystis ulmi* (20, 48, 57, 136, 138). Barrier zones are
ommon in infected elms (6, 20, 113, 125). Ouellette (77) gave a detailed
ccount of ultrastructural modifications in the cell walls of xylem in elms that
irvived after infection by *C. ulmi*.

As the twigs are walled off, or the branches die and are walled off, or as
ying branches open the trunk to infection and these infections are compart-
ientalized, the tree loses more and more space for energy storage (113, 125).
/hen energy is insufficient for continued effective compartmentalization, the
ingus spreads rapidly into more trunk tissues and into the cambium and inner
ark. When large patches of cambium are killed in the trunk, the tree dies. Most
merican elms die from many infections (113). In a sense, the fungus and
isects maneuver the tree into a still-living but defenseless state.

A similar sequence of events has been observed in wounded peach and maple
ees (127, 155). As more and more wounds are inflicted, less and less sapwood
available for storage of food reserves. As energy levels drop, the wound
sponse weakens (127, 155).

RANCHES

/hen branches die, trees form a boundary within the branch base (4, 32, 61,
18). This boundary contains antimicrobial substances that usually inhibit the
owth of decay-causing fungi (4, 32). The boundary usually functions effec-
vely—the branch falls out of the decayed socket (118) and a new cambium
ay form over the branch opening (Figure 3C). In this way, the tree compart-
entalizes the decayed wood in the socket. Sometimes the microorganisms
read beyond the basal boundary of the branch and infect the inner core of the
anch wood (10, 61, 118). This is shown in Figure 3B. At other times, the
icroorganisms spread into the tree trunk within trunk tissues that connect with
e branch (117, 118). When many branches die at about the same time, the
dividual columns of infected wood from the many branches may coalesce to
rm a solid cylinder of infected wood (117). This infected wood is often
correctly called heartwood in species of *Acer* and *Betula*.

BARK

Mullick (72) has provided an excellent review of boundary setting in bark. Biggs et al (15) and Tippett et al (146) expanded on some of the details. Callose has been reported a few days after injuries in red maple bark (84). Boundary setting in bark is different from that in wood (35, 79, 141, 142). Some bark cells change position as the tree grows and the circumference expands, whereas xylem cells remain in position once formed. Some restoring of injured and infected bark cells may take place, and for short periods the new cells may be in the same spatial position as the injured cells (72, 83).

COMPARTMENTALIZATION IN ROOTS

Compartmentalization also occurs in response to wounds in roots (111, 148) and to root diseases (108, 110, 124, 126, 147). In some instances, compartmentalization of pathogens is temporary, e.g. *Phytophthora cinnamomi* in the secondary phloem of *Eucalyptus* spp. (146). Tippett et al (146) indicated that the lesions were contained by necrophylactic periderms (72) formed in the bark. Although the necrophylactic periderm restricted fungal activity during winter and spring, the fungus did break out in summer and invade new areas of phloem in 50% of the inoculated roots and stems (146). This type of break-out pattern is common in perennial cankers and also in root infections, such as those by *Armillaria mellea* (126, 148). Alternating breakout and compartmentalization results in a concentric pattern of cankers on stems, but in roots the breakout results in more and more root death until a threshold is reached and the tree dies. Whether the tree has time to regain its capacity to resist further invasion depends on when the pathogen is compartmentalized. If environmental conditions do not favor the tree, the advantage may go to the pathogen.

GENETIC CONTROL

The ability of an individual tree to set firm boundaries seems to be under moderate to strong genetic control in some species (28, 55, 91, 112, 123). Some anatomical features, such as vessel size (57) and vessel grouping (25), may be the reason, or there may be a combination of features (159). This knowledge gives us an opportunity to begin selecting and possibly breeding trees for resistance to the spread of decay (112).

In studies of wound dressings, some individual trees in a species closed and compartmentalized all experimentally inflicted wounds, regardless of treatment; others did not close wounds and decay developed rapidly in all wounds (122).

ELECTROPHYSIOLOGY

Why does the same microorganism cause a hypersensitive reaction on one host but not on another selection of the same host species? What role does electrolyte loss or membrane leakage play (73)? These questions might be answered through additional research in electrophysiology.

PRACTICAL SIGNIFICANCE OF BOUNDARIES

Boundary setting gives trees the opportunity to survive for long periods under the stress of periodic injuries and infections. However, although the boundaries are very strong protective zones (11, 63, 80), they are also very weak structural zones, especially the barrier zone, or wall 4 of CODIT (62, 80). The barrier zone is a separating zone. Under the mechanical stress imposed by wind, ice, snow, sudden temperature extremes, or felling impact, the zone may split or pull apart. When the split is along the circumferential plane, it is called a ring shake (8, 106); when radial separations occur they are called ray shakes or radial shakes (22, 80). Great variety in patterns of circumferential and radial separations is possible, and the patterns may have many common names (8, 22, 62, 80).

The separating boundaries are common starting points for cracks that cause trees to break during storms. Many times cracks do not develop until the stem is cut into wood products. Too many times the obvious defect remains on the culled portion of the log, and the not-so-obvious crack may find its way into a valuable product.

Often radial cracks develop outward from the circumferential barrier zones. When sudden temperature extremes occur, the cracks may split out to the cambium and a so-called frost crack or a drought crack results (22).

The wound-altered wood is usually the first place infested by termites and ants and the first place infected by other types of microorganisms. Indeed, many problems in wood products have their origin in the living tree. A better understanding of boundaries will help reduce problems in wood products.

The tree sets the configuration of the boundaries, and the microorganisms that infect determine the condition of the wood.

An understanding of boundaries has helped to develop more effective methods for pruning trees. Mayer-Wegelin (61) cites 269 papers on pruning in Europe that started in the seventeenth century. The final recommendation from these studies and studies by others (61, 156) was to cut branches close to the trunk because such cuts stimulate healing. Yet when such cuts were made, the timber was highly defective because of decay associated with the cuts (61). The cuts removed the protective boundary in the base of the branch. The pathogens then infected the trunk above and below the cut. New pruning recommendations state that the collar at the base of branches should not be injured or removed (114). The protective boundary resists infection (Figure 3). Another important adjustment related to boundaries is cavity filling in trees. The boundary that surrounds the cavity should not be broken when one is preparing to fill a cavity.

The proper use of electrical methods for detection of decayed wood in trees and utility poles depends on an understanding of compartmentalization (58, 134, 135). Confusion over the method exists, not because of the lack of understanding of electronics, but because of the lack of understanding of how to interpret the electronic signals.

In summary, it is impossible to separate an understanding of compartmentalization from an understanding of how trees grow and how they respond to wounding and subsequent development of microorganisms. It is just as impossible to separate the role of the pathogen completely from the role of the tree after infection. And the almost endless gradations of interactions and processes over time add to the challenge of understanding. Yet there is hope. There is high order in compartmentalization. How well that order is perceived will depend greatly on how the many parts of this natural system are connected.

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THE RELEVANCE OF NON-HOST-SPECIFIC TOXINS IN THE EXPRESSION OF VIRULENCE BY PATHOGENS

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INTRODUCTION

The interaction between the pathogen and the plant is complex, and the present state of our knowledge about this interplay is fragmentary. Our ultimate goal should be to define plant pathogenesis at the molecular level, not only in terms of altered plant biochemistry but also in terms of the pathogen's biochemistry, its alteration in the plant environment, and its function within the plant environment as time progresses. Studies using chemically defined toxins provide a powerful tool and a useful starting point for these objectives.

An understanding of the relevance of toxins to the expression of virulence by plant pathogenic microorganisms has been slow to develop. This has been largely because many of the earlier investigations pertaining to the biological activity of toxins proceeded without an understanding of the chemistry involved, or of the necessity for chemical purity. Chemical studies of toxins have in fact only gained impetus in recent years, so that now several precisely defined chemical compounds of microbial origin are known to be associated with the diseases caused by a number of bacteria and fungi. It is only since this advance in our chemical knowledge that we have been able to move on to meaningful examinations of the biological significance and function of toxins in plant-pathogen interactions.

Scope and Terminology

Toxins, pathogen-produced chemicals that produce part or all of the symptoms of the plant disease, may constitute only one small part of the disease process

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