HOW TREES SURVIVE AFTER INJURY AND INFECTION

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Abstract.--Trees survive after injury and infection so long as they have the time, energy reserves, and genetic capacity to compartmentalize injured and infected tissues rapidly and effectively to small volumes, and to generate enough new tissues in new spatial positions to store enough energy to maintain the tree. Trees have many protection features and a defense system. The defense system is centered about compartmentalization, which is a boundary-setting process to resist spread of pathogens. Tree pathogens survive so long as they can spread fast and far enough to gain enough space and energy to reproduce. Trees and pathogens interact under the constant pressure of the ever-changing environment. A tree is reexamined from the view of its boundarysetting defense system. When branch development is clarified, proper pruning methods become obvious. When boundary-setting is understood to be under strong genetic control, trees resistant to spread of decay can be selected for our orchards. When tree decay is clarified, the major cause of damage to trees worldwide can be reduced.

Additional key words: Compartmentalization, discoloration and decay, tree defense.

SOME PROBLEMS AND SOLUTIONS

Tree decay is a major worldwide cause of injury to trees in forests, parks, cities, and orchards. The major problem is that decay is considered a natural process beyond our regulation. Orchard managers accept it and try to work around it.

Some textbooks still in use (Boyce 1961) state that decay is not a disease because it affects only the dead, nonresponsive heartwood. Most orchard managers learned about decay by being taught the heartrot concept. The heartrot concept is an excellent beginning concept, but it does not include the response of the living tree to injury and infection. It is a concept dealing primarily with types of wood decomposition, and the fungi that cause decay. Decay is considered a problem mainly on old, over-mature trees.

Improper pruning is a major worldwide problem. Pruning is one of the most common tree treatments, and the oldest agricultural treatment. For over 200 years, the recommendation has been to cut living and dead branches as close as possible to the joining stem--a flush cut--and then coat the wound with some wound dressing.

The flush cut branch is the most injurious treatment man has ever inflicted on trees worldwide. At least 14 serious tree problems, including rapid development of decay and cracks, can be started by flush cuts. Flush cuts were thought to stimulate the wounded trunk to form large callus ribs that were considered a sign of rapid healing. Because it was well documented over a century ago by Mayer-Wegelin (1936) that trees that had flush cut

branches had large pockets of decay, a wound dressing was applied. The thought was that the flush cut solved the branch-healing problem--large callus ribs--but some materials were needed to solve the internal decay problem. Today flush cuts are still the rule, and the search for the perfect wound dressing that will stop decay continues. No data show that any material prevents or stops decay (Shigo and Shortle 1983). The real problem behind the improper pruning techniques is that branch development has not been described clearly. Diagrams of branches in books before 1900 and still today show the trunk xylem tissues above a branch connecting with the xylem in the branch. Branch and trunk xylem tissues meet only below the branch. The natural shedding line at the branch base was known a century ago (Mayer-Wegelin 1936), but more emphasis then was on healing than on natural shedding. Once branch development and natural shedding are clarified, proper pruning becomes obvious. Pruning cuts should never injure or remove the branch collar that is at the branch base (Shigo 1983).

The problem with wound dressings and with many other tree treatments center about the desire to treat trees like people. People who work with trees have borrowed too much from the people who work with animals. Indeed, trees are different. The wound dressings and healing rationale fit for animals, but not for trees.

Another problem is that trees are big organisms and their size makes it very difficult for intensive internal studies. It was not until the powerful chainsaws were made after 1940 that it was possible to systematically dissect large trees. Until then our concepts of trees and defects were primarily from the end of logs.

The major advancement with all of humankind came about when the benefits of health were realized. The same must be done now with trees. It is better to focus on what keeps you healthy, than on what makes you sick. We need to know more about what keeps trees healthy and concentrate on this, rather than continue to fight the many very obvious secondary agents that always follow poor health. We must examine the tree and its survival systems.

A very old problem centers about the belief that wood is dead, nonreactive material. Most of the decay story was about heartwood. Heartwood was and still is considered a dead, nonreactive tissue. It may be dead according to our definitions, but it is surely a tissue that will react to injury and infection. Heartwood in most trees has a built-in protection feature--extractives--but it also has a system that reacts to resist spread of pathogens. Discolored wood has often been confused with heartwood, and the types of discolored wood have been given names that imply a type of heartwood--wound heartwood, false heartwood, pathological heartwood.

Too often color has been the major factor used to determine a type of wood. All wood darker than the sapwood was called heartwood or a type of heartwood. The term discolored wood is an extremely poor term (and I feel badly that I have helped to set it so firmly in the literature). A better general term would be "injury- altered wood." A long gradation of changes starts in the wood--sapwood, heartwood-- after injury and infection. Some injury-altered wood is much less protective than contiguous wood. A great amount of confusion about wood could be clarified if wood anatomists and

physiologists were presented with a much clearer explanation of wood changes that occur after injury and infection.

When a branch or root dies, there is an opening into the tree that can be infected by organisms. Dying roots and branches are the most common infection courts for tree-inhabiting organisms. Yet these courts are often not recognized as starting points for defects. Because of the many microorganisms that infect trees, it has been assumed that it is impossible to select trees resistant to decay. In part this is correct. Trees cannot prevent infections in wood killed by wounds or in dying branches and roots. But, trees can resist spread of microorganisms. It is possible to select individuals within a species that set boundaries very rapidly and effectively to resist spread of microorganisms. This feature seems to be under moderate to strong genetic control.

Compartmentalization has been misunderstood as a process that always stops microorganisms, and succession has been misunderstood as a process where bacteria always infect first, then followed by non-hymenomycetous fungi, and finally the infection of Hymenomycetes. First, compartmentalization is definitely not an absolute process. It does not function perfectly all the time. When it does not function perfectly, some tree part, or the entire tree may die. Some microorganisms have developed effective ways to grow around boundaries. Second, succession is a highly ordered sequence of many microorganisms that assures survival of the groups. Hymenomycetes may be the first to interact with the tree--pioneers--or other types may be pioneers. The point is that many microorganisms are involved in the processes that may or may not result in total decomposition of wood.

Trees have many protection features and a defense system made up of many parts. Time is the name of the game. The tree and the tree-inhabiting organisms must stay alive long enough to reproduce. The time game is played against the environment. Trees must have superior protection features and a strong defense system to live and maintain reproduction.

I believe that the basic worldwide problem in the tree industries is the lack of understanding of what a tree is and how it functions to survive after injury and infection. The more tree managers know about trees, the better the decisions will be in favor of healthy trees.

The purpose of this paper is to reexamine a tree from the view of its defense system, which is compartmentalization (Shigo and Marx 1977, Shigo 1984). Compartmentalization is a boundary-setting process to resist spread of pathogens.

IN THE BEGINNING

Plants that were to become trees started to grow tall and massive on land over 400 million years ago. Our extensive coal deposits were formed by masses of fallen trees over 300 million years ago. Trees as we know them today began to develop 200 million years ago. Trees as we see them today have changed little over the last 50 million years.

Tree survival

To survive is to stay alive. After injury and infection, some individuals of a species die and some stay alive. The species exist only so long as some individuals stay alive long enough to reproduce. It is possible that some individuals of a species stay alive long enough to reproduce because they have not been seriously injured or infected. This is highly unlikely with trees. Unlike animals, trees cannot move away from a destructive agent. Animal survival strongly depends on the ability to move away from destructive agents. Trees have evolved under the constant pressures of all types of wounding agents--fire, storms, animals, insects, volcanoes, man--and all types of organisms that could digest the tissues exposed by wounds. The forms and functions of trees are adaptations to the survival threatening pressures of injuries and infections, and to the ever-changing environment over very long periods. If this were not so, trees would not be here today. Not only are trees still on earth, but they continue to be the tallest, most massive, and longest lived organisms. To be so tall and massive for such a long time must require unique protection features--static state--and superior defense systems--dynamic state.

HYPOTHESIS

If trees do have unique protection features and superior systems for defense that makes long survival possible, and if trees' form and functions are successful adaptations for survival after many types of injuries and infections, then the best way to understand a tree is to study its response or reaction to the many types of naturally caused and experimentally caused injuries and infections.

Indeed, long-term environmental pressures also played a role in shaping the forms and functions of trees. But, if trees did not stay alive long enough to reproduce after injury and infection--relatively short time--the long term effects of the environment would not take place.

RESEARCH BACKGROUND

I have tried to test the hypothesis over a 25-year period. The research background or data base for this discussion comes from studies on the systematic dissections of thousands of trees that had naturally caused internal defects and changes in wood started by a great variety of experimentally inflicted wounds, in which many were inoculated by organisms. Also, systematic isolations of microorganisms were made to show temporal and spatial relationships after injury. Then these data and data from the studies of many other investigators were connected to expand the concept of tree decay. The living tree and its survival features were brought into the concept. The expanded concept included compartmentalization--the orderly response of the compartmented living tree to injury and infection--the microbial succession--the orderly sequence of many microorganisms in the process of decay. Then a model of compartmentalization of decay in trees--CODIT--was developed (Shigo and Marx 1977). New electrical tools and

methods were developed with the help of many people (Shigo and Shortle, in press). Now the expanded concept, the CODIT model, and the electrical methods are being used to reexamine trees, many tree problems, and tree treatments.

In the beginning of our research program, emphasis was on the development of internal defects. As this sphere or circle of understanding increased, it was apparent that more had to be learned about the tree. As the basics of tree biology were studied, another sphere of understanding began to develop. Now the two spheres have combined, and they reinforce each other.

BASIC TREE BIOLOGY REVISITED

Trees are perennial, woody, compartmented, shedding plants that are usually, (but not always) tall, massive, long-lived, and single-stemmed. The first four characteristics are consistent and the most important. The unique feature of a tree that separates it from all other organisms is its superior mechanical support. The combination of cellulose and lignin in the wood cell walls, and the cell arrangements, give trees their unique feature. In a teleological sense, the first priority of the evolving tree was to protect and defend its unique feature. If the evolving tree were not successful at this, then the tree plant would be similar to other nonwoody plants. This is an oversimplified statement to clarify some misconceptions about trees.

Decay-causing microorganisms are pathogens, even if they are attacking only the heartwood (whatever definition you have for heartwood). Pathogenesis is defined on the basis of the entire organism. If mechanical support is disrupted, the unique feature of a tree is disrupted, and the tree is back to a small nonwoody plant. Trees, like all organisms, die three ways: mechanical disruption (accidents, wounds, etc.), dysfunction, and infection (an agent blocks or takes energy). Any agent that causes the tree to fail and die is indeed a pathogen.

The tree has many built-in protection features--for example, bark with suberin, heartwood extractives, low nitrogen to high carbon content of wood, arrangements of cells, arrangements of microfibrils--but these protective features are only the first line for survival. A second line starts after injury and infection. The tree cannot restore--repair, replace--an injured xylem cell. But it can set boundaries to resist the spread of pathogens from injured to sound cells. If trees had only one line of protection features, it would not be long before the microorganisms would adapt and break them down. The evolving tree developed a dynamic defense system to resist or stall the microorganisms that were able to spread within the wood that had the protection features. The microorganisms survived by attacking in succession. If the tree did not have a second line of defense, the mechanical support system would be disrupted rapidly and the tree plant would be back to low plant again.

It is important to keep in mind that no matter how strong the protection features and the defense systems become, they cannot stop all organisms all the time. The same for organisms, no matter how aggressive they become as individuals or in succession, they cannot digest all tree tissues all the time. If this did happen, it would only happen for one period and the host

and pathogens would no longer exist. Compartmentalization makes it possible for host and pathogens to survive. The pathogens do digest the tree, but digestion of tissues is regulated with generation of new tissues in new spatial positions. This interaction has the capacity for long-term survival.

Trees and pathogens have a common enemy and friend in the environment. In a sense, trees and pathogens "need" each other as a common orderly life force to survive against the short-termed disordered environment. The view of host, pathogen, and environment is different from the view that shows the three equally interacting with each other as a triangle. In the view I present, the host and pathogen represent the life force against the nonlife force of the environment. This is not to say that the environment does not affect the host and pathogen each in a positive or negative way, or both positive or both negative. A force is measured at the plane where two opposing pressures meet. If environmental pressures eliminated tree-inhabiting organisms, I doubt that trees would survive alone against the environment. The same can be said for tree pathogens. Time is the important factor. So long as the tree and pathogens have time to survive long enough to reproduce, the complex but orderly natural system will persist. The built-in static protection features and the dynamic defense system are the keys to long-term survival for trees after injury and infection. As stated, if the protection features were the entire survival system, it would not be long before the microorganisms would adapt to it and digest it. Any feature that "stands still" is an easy target. The long-term survival advantage of the defense system is that it does not occur until after injury and infection. An organism cannot adapt to something that is not there at the time it arrives. The defense system of each tree will always be slightly different because of genetic programming. The organism that is able to survive the competition on the wound surface may not have the genetic capacity to interact with the internal defense system of a tree.

The wood-inhabiting microorganisms have a double problem. They must compete effectively with many other microorganisms on the fresh wound surface and then grow into the tree against the protection features and the defense system. Many of the microorganisms that can survive on the wound surface are poorly adapted to survival inside the tree. Once in the tree, the pathogens must interact with the tree and also defend itself and its occupied space from other microorganisms.

When the protection features, defense systems, competition, and the ever-changing environment outside and inside the tree are considered, it is a miracle that some microorganisms do survive inside the tree.

If the microorganisms can be resisted long enough for the tree to generate enough new cells in new spatial positions, then the tree wins that battle.

Some defense systems had to evolve that were ready to react after injury and infection to resist rapid spread of microorganisms. The defense system had to function where there were still some cells with living contents and aged cells without living contents.

Every tree has some microorganisms that will attack its injured or dying sapwood or injured heartwood, regardless of amounts of extractives.

Extractives do stall the growth of microorganisms and some trees have an extremely effective extractive-based protection system. After injury and infection, moisture content near wounds and in dying cells begin to change. Changes also occur in pH and concentration of microelements. In some trees, wood is infected by bacteria and wetwood results. Wetwood seems to inhibit infection by decay-causing fungi, and the mechanical support system is protected. In summary to this point, many different types of protection features make wood difficult to digest by microorganisms, and boundary setting limits the spread of the digestion when it does occur. This double-powered approach buys time for the tree to continue generating new cells in new spatial positions. Yet, wood-inhabiting microorganisms do digest trees when time is extended.

In the section on problems, a point is made that heartwood is considered a dead, nonreactive tissue. And yet heartwood does discolor when injured and infected. If discolored wood is wounded, it does not discolor further. Discolored wood has reached the lowest limit of energy reaction; heartwood has not. Heartwood will not only discolor after wounding, but also it will discolor in orderly predictable patterns depending on the wound. Heartwood may not contain cells with living contents, but it may contain some materials that are not at the lowest energy state. Because heartwood was always considered a dead, nonreactive tissue, no research was done to investigate its ability to react to resist spread of microorganisms. If the heartrot concept and heartwood were not able to resist spread of microorganisms by setting boundaries, disordered columns of decayed wood would be in heartwood. Yet the patterns of decayed wood in heartwood follow the CODIT patterns for trees that do not have a heartwood. I believe we have a situation with heartwood where our definition or concept of dead do not apply. It may be that the cells are chemically cocked, as a mousetrap, when they reach a point of genetically controlled aging. When the "trigger" is hit, the trap reacts in a predictable way, and usually kills the mouse. The mousetrap is not alive. But once cocked, it can react once. It takes energy to cock the trap. There may be some energy in "cocked" chemicals in sound heartwood.

Discolored wood is even more confusing than heartwood. There is no one type of discolored wood. Discolored wood is like a color spectrum or rainbow. It is the condition of a tissue in transition. It is a gradation of changes. Too much emphasis has been placed on color and color changes. Discolored wood is wood altered as a result of injury and infection. After a tree is wounded, or after a branch or support root begins to die, contents of living cells begin to change, usually to a protective state. The changes are the result of tree-microorganism interactions. Discolored wood is usually more protective soon after it is formed, and then it may or may not begin to become less protective as different microorganisms infect. It is very important to separate cells that die and discolor, from cells that discolor and die. The latter is more in tune with hypersensitive reactions when the organism kills portions of itself to save the whole. In other instances, healthy wood tissues are "trapped" between an older inner defect and a new wound. The new wound isolates the healthy wood from the cambium, the symplast is broken, and transport to the "trapped" wood is disrupted. Cells with living contents may die when branches or roots die.

Recent experiments show that the symplast may have an effect on the cambium. Holes were drilled through sugar maples, red maples, and paper

birch. The holes ended approximately 0.5 cm from the cambium on the opposite side of the trees. Although the cambium on the opposite side of the hole was not touched, nor were there any cracks from the wound tip to the cambium, the growth rings distal to the end of the drill hole increased in width. The width of the growth rings attenuated tangentially away from the distal ends of the drill holes. It may be that the symplast plays a more important role than suspected in the regulation of the cambium. The cambium may be like a queen bee, it only produces cells. The differentiation of the cell may be determined by other conditions. When a hive is in trouble, more soldiers develop. After tree injury or infection, a barrier zone develops.

The barrier zone is a key boundary in trees (Tippett and Shigo 1980, Mulhern et al. 1979). The zone is a nonconducting tissue that has a few vessels in deciduous hardwoods and an abundance of axial and ray parenchyma. The cell walls have low amounts of lignin. In oaks, suberin was in the cell walls (Pearce and Rutherford 1981). The barrier zone separates the tissues present at the time of injury and infection from the few cells that continue to form after the zone is completed. In conifers, more resin ducts are formed in the barrier zone, and in eucalyptus species, more kino forms in the zone.

Within tissues present at the time of injury and infection, chemical boundaries are formed to resist spread of pathogens. Vessels begin to plug, and pits in tracheids close. Gums and other materials plug the vessels. Oxidation processes start. The contents in cells change to inhibitory materials. The changes serve to set chemical boundaries to resist spread of pathogens. The boundaries are the result of nonspecific reactions. The tree will react the same way for any number of microorganisms or types of wounds. It is impossible to separate the effects of the wound from the microorganisms, and the wound and microorganisms from the tree on the formation of boundaries. The boundaries are the result of interactions between the tree and the pathogen after the trigger has been released by the wound.

The wound and microorganisms can kill cambium. When large areas of cambium are killed, that part of the tree dies. When more cambium is killed, the entire tree may die. Usually the killing does stop at some point. Then the first cells formed by the cambium differentiate to form a barrier zone. Under some conditions, aggressive microorganisms may grow through a barrier zone. If they do, it only occurs once because the cambium will be killed. A barrier zone will also form as a response to infection. For example, as Ceratocystis ulmi grows through a vessel, it does not contact the cambium, but a barrier zone may form. Little is known about the factors that affect formation and size of the barrier zone. Small wounds may elicit large barrier zones, and large wounds may have small zones. Sometimes the zones develop completely around the trunk, while most of the time they do not. Barrier zones form in roots also.

ENERGY NEEDS

Energy is required to run the biological machinery. Trees store energy as starch or oils in living cells. It is one thing to trap energy, and another to have some place to store it. When compartmentalization walls off too much tissue, the process can be counterproductive. Trees have evolved in such a way that the volume of storage space is a basic feature of different

species of trees. For example, American elms store starch in 15 to 18 growth rings. When elms infected by C. ulmi begin compartmentalizing the pathogen, portions of the tree are reduced to a one-growth-ring tree. The fungus is well adapted to killing one-growth-ring branches or trees.

When a tree is two years old, all the living cells in the wood can store energy. As the tree gets older, and as living cells die, the ratio of the volume of wood in the stem to the volume of wood that can store energy reserves decreases. This is a part of the aging process.

Biological aging is the orderly, genetically controlled change in a part, or the entire organism, as highly ordered processes deteriorate. Aging is an intrinsically controlled process. The changes are set in the genetic program, but environmental factors affect the expression of the changes. The different types of wood cells age at different rates. All cells start as cambium and then begin to differentiate. Vessels lose their contents and become functional for transport. Vessels age rapidly to the point of function. Fibers may contain living contents for a few months or for several years. Ray and axial parenchyma may maintain living contents for over a hundred years. The entire rhythm of changes built into one growth ring repeats as a new growth ring is formed.

Biological decaying is the interaction of intrinsic and extrinsic factors that affect changes in parts or the entire organism as high order goes to low order, and energy is transferred from the host to the pathogen. Decaying involves intrinsic and extrinsic factors, and there is an energy transfer from tree to pathogen that is detrimental to the tree. As cells age and as new growth rings envelop older rings, the aging cells are buried deeper. The young growth rings with the high percentage of living cells can respond rapidly and effectively to injuries and infections. But, as wounds go deeper into the wood, the older, aged cells are exposed to the pathogens. If trees did not have some way to keep the microorganisms from quickly digesting the aging cells, trees would not exist today.

Some aged cells contain extractives that stall the growth of microorganisms. But in spite of all of the tree's protection and defense adaptations, the microorganisms do win at times, and wood is digested. Hollows are common in old trees.

CODIT

Compartmentalization is a boundary-setting process and CODIT is a model of the process. CODIT is an acronym for Compartmentalization Of Decay In Trees. CODIT has two parts: Part I is represented by 3 model walls and Part II by one wall. In Part I, wall 1 resists--not stops--vertical spread of pathogens, wall 2 resists inward spread, and wall 3 resists lateral or tangential spread. After injury and infection, Part II or wall 4 is formed and separates Part I from the healthy cells that continue to form in a new spatial position. Wall 4 is a model representative of the barrier zone. The CODIT model should not be identified too closely with anatomical or biochemical features. It is a model to help orient the mind to the three-dimensional spread of the pathogen and to show the separation between infected cells and the cells that continue to form.

When CODIT is used to reexamine tree problems, the orderly patterns of spread and resistance to spread emerge. CODIT shows that the spread of microorganisms is orderly, and so is the response of the tree.

CANKER AND CANKER ROTS

Cankers are localized lesions or dead spots. Cankers show the intimate interaction of tree and pathogen.

Pathogens infect trees four ways: 1) infect in bark and stay in bark; 2) infect in bark, grow into wood; 3) infect in wood, stay in wood; 4) infect in wood, grow into bark. The pathogens that stay in the wood are typical "wound rots." Those that stay in bark and may grow slightly into the wood, are those that cause annual cankers. The pathogens that grow from wood to bark, or bark to wood, start a seesaw interaction with the tree. In such interactions, it seems that compartmentalization is not functioning, and that is correct for short periods. Compartmentalization is intermittent. If compartmentalization were not effective at all, the tree would die.

Some microorganisms infect bark and continue to grow until the tree either forms a boundary in the bark, or the tree forms a wood wedge from xylem into the bark. The ray sheets that extend from xylem to phloem may form wedges of wood in front of the advancing pathogen in the bark. The wood wedges form only up to the phellem. When the pathogen begins to grow again, it usually expands into the phellem around the wood wedge and then back down into the bark. The tree then forms another wood wedge, and the process is repeated. Wood wedges are formed by oaks attacked by Strumella coryneoidea, and aspens attacked by Hypoxylon mammatum. Fungi that cause canker rots get established in wood and then produce wedges of hard fungus material into the bark. The fungus wedge expands in the bark and kills cambium from the bark side. When the cambium is killed, the still-living cambium beyond the dead spot responds to begin compartmentalization again--another seesaw interaction.

In some American chestnut trees, pillars of xylem ray parenchyma burst into the " dying bark and a new cambium is generated, and wood and bark continue to form.

Trees have marvelous ways of staying alive.

LIMITS OF COMPARTMENTALIZATION

Compartmentalization, like healing, has its limits. Here are some common examples where boundaries are broken and the pathogens spread rapidly.

1. Cracks that split outward from wounds--so called "frost cracks".
2. Insects and animals that rupture the barrier zones from the inside.
3. Barrier zone broken during cleaning prior to cavity filling.
4. Holes drilled through barrier zones.
5. Perennial cankers and canker rots--temporary seesaw interaction.
6. Aging. Given enough time, all natural materials will break down.
7. Energy depletion. When energy reserves are so reduced, inhibitory materials may not form.

TREES RESISTANT TO SPREAD OF DECAY

The tree protection features and defense systems when operating properly, make it possible for trees to survive for hundreds or even thousands of years. Some individuals in a species have much more effective protection features and defense systems than others. Recent research shows that compartmentalization is under moderate to strong genetic control (Lowerts and Kellison 1981). This means that the strong individuals can be selected for seeds or cuttings. Indeed, it is possible to select trees resistant to spread of decay.

GROUP SURVIVAL

What has been discussed to this point deals with protection and defense of the individual trees against many microorganisms and the environment. Trees must also have group protection and defense against pathogens and environmental pressures.

Some of the group protection features may be gregarious growth habits, great diversity in gene pool, and asynchronous phenology--factors that prevent selfing. By this type of reasoning, trees must also have a group defense system. It is difficult to go beyond conjecture here, but some of the group dynamic defense systems may include volatile chemicals that signal possible injury to the group and changes in some electrical type signals that may alert the group to react to change their physiology in favor of defense. Enough said here. There has been too much said already on this subject and not enough sound work done to support the statements.

SUMMARY

We do not have all the answers about trees. We must go back and reexamine many parts of tree biology. I am sure there are still many surprises.

Trees and tree inhabiting organisms do play a time game with the ever-changing environment. Trees survive so long as they have time to generate enough new cells to maintain the tree after injury and infection. The new cells must not only store energy reserves and carry out the normal tree physiological functions, but also they must maintain mechanical support. While the new cells are being faced with these responsibilities, the older cells that were present at the time of injury and infection must resist, stall, or limit the spread of the pathogens. If the pathogens spread faster than the newly generated cells can take over, the tree part or the entire tree may die.

Trees have protection features and a defense system. The defense system is centered about forming new boundaries or reinforcing existing boundaries. After injury and infection, the tree can respond or react only within its genetically set anatomy and physiology. It can only form so many inhibitory compounds, and it can alter its anatomy only so much. Some of the changes that are made are similar to the built-in protection features. But, a tree cannot overplug its transport system or use too much of its stored reserves for boundary materials without putting a strain on its health.

What keeps a tree alive eventually kills it. It is a time game against environmental factors and the many other organisms that want its space and energy. But so long as some trees can continue to play the game as they have for hundreds of millions of years, trees will survive.

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