TRESS AND DEATH OF TREES

Aiex L. Shigo²

chief Scientist, USDA Forest Service, mortheastern Forest Experiment Station, 2.0. Box 640, Durham, NH 03824

. Abstract .-- Trees survive after injury and infection so long as they set effective boundaries to resist spread of pathogens. Boundary-setting is beneficial so long as the volume of walled-off infected wood is small and the intervals between infection are long enough to allow enough new cells to be generated in new spatial positions that can hold enough energy reserves to maintain the tree. A tree may be able to trap solar energy, but when space for energy storage has decreased, all systems of the tree begin to diminish. Many secondary agents then attack. Fighting the secondary agents will not solve the basic problem of energy depletion, or stress, that may progress to irreversible strain and death. Answer's to the basic problem of poor tree health will come only when tree management decisions are made on the basis of a much better understanding of the tree. Needed now is a new attitude about trees.

Introduction

Stress and death, and gloom and doom, are major current topics associated with forests worldwide: diebacks in Australia, exploitation of the tropical forests, large-scale dying of forests in Europe, dying of young planted forests in Korea, and, of course, all types of forest problems in the United States, and the list goes on and on. Is this recent attention to our world forests a media exaggeration, or do we really have some serious problems in our world forests? Are many forests of the world really in a state of stress and death?

During the last several years I have had opportunities to examine trees in several other countries. Some forests are very healthy; many were not.

17 Paper presented at SAF Region VI Technical Meeting: Forest Management and the Spruce Budworm. Burlington, Vermont, April 24-26, 1984.

 $\frac{27}{1000}$ Portions of the research discussed here were supported by CANUSA.

A common thread that runs through many of the global tree problems is poor tree health. Insects and microorganisms are always present and are blamed for the problems, but I doubt that they are the causes or starting points of many of the tree problems. The organisms are often secondary agents. Too often we fight the secondary agents or the symptoms rather than the causes. And, even if we are successful in eliminating one group of secondary agents, another group will be ready to start.

A great advancement for humankind came about when the benefits of health were realized. It is better to concentrate on what keeps you healthy rather than on what makes you sick. Medical science advanced when bodies were systematically dissected. Then immunization treatments were used to make the body's own defense system function to keep disease at low levels. We must keep these points in mind as we discuss tree health problems. We should also concentrate on what keeps trees healthy rather than on what makes them sick. We should have a clear understanding of how trees are constructed and how they function. And we must help trees help themselves to use their defense systems more effectively.

There are some basic reasons why poor health is a major tree problem. Ignorance of tree basics is the major worldwide problem. We desperately need an attitude change about trees. Trees cannot sustain repeated abuse without serious injury. Trees do die! Decay is not a natural process beyond our limits of regulation. We can reduce the amount of rot. Many of the inaccuracies about trees that are being taught and are deeply entrenched in textbooks must be adjusted (Shigo 1980, 1982a, 1982b). We can start by learning about trees. This is not to say that anyone was or is wrong. It does mean that science advances as adjustments are made on the basis of systematic reexamination. Indeed, it is time to reexamine a tree: how it is built -up, how it functions to stay built up, and how it eventually breaks down.

Some Tree Myths

Do you still believe that frost starts the long deep cracks that are called "frost cracks"? Do you still believe that wind starts the circumferential cracks called "wind shakes"? That trees heal wounds? That trees absorb minerals selectively from the soil to form mineral streaks? That heartwood is an unreactive tissue? That flush pruning is the best way to remove branches? The list goes on and on. My point is that if vou believe these myths, and the many others that are in textbooks, it is no wonder that proper management schemes cannot be developed to produce high-quality trees that will produce high-quality products. It is far beyond the scope of this paper to discuss the many misconceptions about trees, treatments, and factors affecting defects. I shall discuss the role of energy reserves in the tree defense process. Excellent information on starch reserves in trees has been given by Wargo (1971, 1975, 1976, 1979).

bone Definitions

First it is absolutely necessary to define some terms. Voltaire said to define your terms and arguments will be less than a few minutes. These are my definitions, and it is doubtful that you will agree with them totally. They are terms that are used commonly by many people, but seldom defined. This is why there are so many arguments.

HEALTH is the ability--dynamic state--to resist strain. STRAIN is an injurious, irreversible condition caused by excessive stress. STRESS is a gradation of events resulting in a drain, blockage, disruption, or shunt of energy. ENERGY is fuel or the force that "runs things" or maintains vitality. VITALITY is the dynamic-ability to grow and reproduce within the limits of vigor. VIGOR is the intrinsic genetically controlled capacity-potential--to survive after injury and infection. INFECTION is a process of energy transfer from host to pathogen. PATHOGEN is an agent that causes injury or strain to a host as a result of energy transfer. Organisms that decay trees are pathogens because pathogenesis is based on the entire organism and not on its parts. DISEASE is a process of energy transfer to pathogens from the host that results in a condition of strain to parts of the host or the entire host. HOST is the organism that has the stored energy and is interacting with a pathogen in energy transfer. ENERGY TRANSFER must consider the three laws of thermodynamics: (paraphrased here for emphasis) 1) you can never win, only break even, 2) you can only break even at absolute zero, and 3) you can never reach absolute zero.

Indeed, we cannot win them all, but we can surely win more than we are at this time.

How Do Trees Die

Trees, like other organisms, die three ways: dysfunction, infection, and mechanical disruption. There are many variations on these themes, and usually the variations overlap many times before death. Dysfunction occurs in many ways when tissues do not function properly. For example, this can occur when soil water is too high or too low for the tree, or when microelements or pH, or soil type, or any number of factors change or are changed. Trees can adapt to many conditions if they have the time. Any sudden change may make adaptation impossible or difficult. Tree problems associated with dysfunctions are least or poorest understood because they usually are not recognized until it is too late. We know so much about ends of processes, but so little about beginnings. Yet it seems that secondary agents can rapidly recognize the beginnings of the unhealthy condition when they attack. Tissue dysfunction affects not only growth and reproduction processes, but also the defense system. To be alive but defenseless is indeed the worst condition.

The real problem with dysfunction is that additional energy may not help the condition. Indeed, dysfunction is the malfunction of some vital tissue, organ, or process. Adding more energy or more fertilizers may not help a malfunctioning tissue or organ. In fact, some treatments, such as over fertilization, may actually make the malfunction worse. Disruption of sites by logging, mining, agriculture, and grazing will cause poor tree health: stress, strain, and death. Misunderstandings of tree problems also lead to planting the wrong tree in the wrong place (conifers on hardwood sites) --done repeatedly in Europe--and any number of disruptions that alter normal tree functions.

A major problem in understanding trees is that we have borrowed too many terms from other disciplines, especially human pathology. We then try to fit or force the terms onto the tree, and they often do not fit. A tree can have many large dead and dying branches, but also an equal number of very healthy branches. Is the tree half healthy or half dying and dead?

In a sense, trees are multiple plants, or communities of plants. The parts may die, and new parts are generated in new spatial positions. Or, the entire community may be in trouble. The dysfunction or infection may be affecting a part of the community, or the entire community of trees. We lack proper terminology to discuss clearly such an organism or "group of organisms."

Add to this the problems of mechanical disruption. The unique characteristic of a tree is its mechanical support system. Trees are still the biggest organisms ever to inhabit this planet. Trees have evolved unique ways to protect their mechanical support systems--they set boundaries to resist spread of pathogens. But, when a pathogen digests the support system, and the tree falls over, the tree is dead. This is why decay-causing organisms are pathogens.

In a sense, trees "attempt" to avoid some of the problems due to size by regulating generating with shedding. Trees by nature of the growth systems do get larger every growth period. But, trees regulate this somewhat by shedding twigs, branches, support roots, and absorbing roots that begin to age and die.

How a tree dies is not a simple matter. The more we know about how trees live, the more we can do to keep them from dying.

COST BY

Energy is the fuel or force that maintains biological machine. Trees get energy by c: spping solar energy in a molecule of carbon disride and water. Of the 0.1% of solar energy erapped by afl organisms on this planet, trees trap 50%. The energy is used as sugars and stored as arch, oils, and other materials. The energy maintains growth, reproduction, and a defense syptem. Insoluble energy reserves, mainly as starch and oils, are used to start the tree processes after dormancy. It takes a great amount of energy to maintain and operate a defense system. The other requirement is space for the needles end leaves to trap energy. Survival requires space and energy.

mergy and Boundary-Setting

To summarize to this point: Trees are highly compartmented, woody, perennial, shedding plants that are usually (but not always) large and single-stemmed. Trees generate new cells in new spatial positions and shed dying and dead parts. Trees do not restore injured and infected tissue. Trees do set boundaries to resist spread of pathogens--compartmentalization (Shigo and Marx 1977, Shigo 1979, Shortle 1979). A model of compartmentalization is called CODIT.

CODIT

To understand defects from the tree to the product it is absolutely essential to understand boundary-setting processes. To make this very complex subject clear, a model called CODIT was developed (Shigo and Marx 1977). CODIT is an erronym for Compartmentalization Of Decay In Trees. The model has two parts. Part I is in the tree at the time of injury and infection. Part I is sepresented in a model sense by three walls: walls I resist -- not stop -- vertical spread of the microorganisms, walls 2 resist inward spread, and walls 3 resist lateral spread. In a sense, the tree attempts to wall off the microorganisms to as small a volume as possible, while the microorganisms counter this attempt by spreading as far as possible, as rapidly as possible.

After injury and infection, the still-living combium about the injury begins to form a new tissue called the barrier zone. The barrier sone is a very protective tissue, but a very weak structural tissue. The barrier zone is a weak conducting tissue. In the CODIT model this separating boundary is called Part II, or wall 4. Wall 4 is where ring shakes and radial splits begin. Wall 4 may form after wounds or the death ef branches and roots.

Trees survive after injury and infection so long as they have the time, energy, and genetic espacity to recognize and compartmentalize injured and infected cells effectively, and then generate enough new cells in new positions to exatinue to maintain the tree. The new cells must be able to store sufficient energy to maintain growth, reproduction, and a defense system.

When energy is decreased, trees reduce growth and reproduction. When decreased energy reduces the defense budget, a high-risk situation begins. The risk is reduced when no further injuries are inflicted. But, when injuries are inflicted, the risk increases, especially when energy-drains caused by injuries and stresses repeat at shorter and shorter intervals.

The two important parts to energy reserves are: (1) the tree must have enough healthy needles or leaves in enough proper spaces to trap sufficient energy, and (2) the tree must have some place to store the energy reserves. This second requirement is often not considered. The second part also is counter to boundary-setting or compartmentalization.

Genetics of Boundary-Setting

The tree defense system is centered about boundary-setting. Trees that have the genetic program to resist spread of pathogens will limit the infected zones to small volumes (Garrett et al. 1979, Shigo et al. 1977, Lowert and Kellison 1981, Schmitt et al. 1978). Trees that have weak boundaries will have large volumes of infected wood. Compartmentalization is indeed an effective defense system when it functions to limit infected tissues to small volumes, and when the injuries and infections are not repeated within short periods. When more and more tissues that normally hold energy reserves are walled-off, the potential volume of wood for storage of reserves begins to decrease.

Starting Points for Infection

The three basic starting points for infections are: dying roots, dying branches, and mechanical injuries. It is impossible for a tree to grow in a forest without having some or all of the infection courts. Trees with strong genetic programs can wall off dying branches and roots effectively, and no volume of trunk wood is lost to the pathogens. When mechanical wounds are inflicted on the trunk, the tree responds to resist spread of the pathogen within the wood present at the time of injury, and then the tree forms a protective barrier zone that separates the infected wood from the new wood that continues to form.

As energy reserves decrease, the defense budget also decreases. The boundary-setting process is less effective. As new infections start, they spread faster and farther. When healthy wood is "trapped" between new infections and an older internal defect, the new infection will often spread rapidly into the "trapped" tissues.

The process of boundary-setting then begins to be a problem for the tree rather than the answer to survival.

Paradox of Boundary-Setting

One irony of the way natural systems operate is that a feature or characteristic of the system may be beneficial or destructive depending on the concentration of the feature, or characteristic--soo little compartmentalization, too much compartmentalization--and timing--compartmentalization when energy reserves are high is beneficial, compartmentalization when energy reserves are low may be destructive.

The tree must produce new cells in new positions during the growing period or it ceases to be a tree. Energy is required for these functions. To compensate for lower energy reserves, the tree walls off more of its parts. But, when more and more branches are walled off, so are leaves and needles that trap energy. At this stage there is little hope for survival of the tree. Even if treatments could retain needles and leaves, and even if needles and leaves could trap an abundance of energy, there would not be enough space within the tree to store energy.

Long before a tree exhausts its energy supply, many weakly parasitic or opportunistic organisms attack and drain the remaining energy reserves. So, it is unrealistic to think that a tree will die-because it exhausts its energy supply.

Aging and Boundary-Setting

When trees are young, 100% of their volume can be used for energy storage. The living cells in the wood store the energy reserves. As branches and roots die, and as injuries are inflicted, and as living cells in the wood die, the ratio of stem volume to volume of wood capable of storing energy reserves begins to decrease.

The tree regulates crown size and root mass by setting boundaries between aging, dying, and dead parts, and still living, healthy parts. The boundaries are usually effective in resisting spread of the microorganisms from the dying root or branch into the healthy wood. It seems that boundary-setting at the branch bases and root bases is under genetic control. When the microorganisms are aggressive, they may spread beyond the boundary and into the healthy wood. The tree usually responds by setting new boundaries deeper into the wood. The new boundaries may resist further spread and limit the microorganisms to small volumes of the joining stem or root. In other instances, the microorganisms may be even more aggressive and may breach the second boundary and spread into the trunk or the major root. The tree will still respond by resisting spread within the trunk or root, but also the tree does form a barrier zone that usually confines the infection to the trunk or root tissues present at the time the branch or root died.

How many boundaries are breached or broken may be a factor of aggressiveness of organisms, or it may be a factor of the tree. The tree may be a genetically weak tree that sets weak boundaries, or the tree may be an energy depleted tree that cannot set strong boundaries.

A young tree may be a genetically strong compartmentalizer, but as more and more branches and roots die, and as more and more injuries are inflicted, the boundary-setting capacity of the tree begins to decrease.

Once boundary-setting reduces the volume of wood that can hold sufficient energy to some yet unknown low point, the tree becomes strained. Once the tree is strained, the recovery is less and less likely.

Stress and Strain

A tree can have a great amount of stress without having strain. Stress includes all the dying branches, dying roots, and mechanical wounds. So long as the tree is able to collect and store sufficient energy, it will continue to grow and reproduce.

Stress also increases as dysfunction increases. Dysfunction of vital tree processes may be a genetic factor that becomes obvious very early in the life of the tree--usually leading to early death--or a factor associated with intrinsic genetically controlled aging processes. Dysfunction may also be associated with environmental factors such as discussed earlier, and also with some types of infections. Regardless, dysfunction results ultimately in an energy blockage, shunt, or drain, which is stress. And, when stress continues, strain may result.

Here are some important specifics about spruce and fir, based on my discussion on stress and strain.

Spruce, Fir, and CODIT

Fir has a very weak CODIT, Part I (Tippett and Shigo 1981, Tippett et al. 1982). What does this mean, especially for products? After branch death, root death, or mechanical wounds, the wood is rapidly invaded by microorganisms (Shortle and Ostrofsky 1983). But the tree produces very strong walls 4. In roots such a response permits the tree to remain alive with hollow roots, but the support function of the roots is greatly decreased. When such rot problems develop in large support roots that join at the tree base, a crack usually develops between the two joining roots. The vertical crack may extend far upward on the trunk. Cracks at the base of fir are reliable indicators of root rot, especially those caused by Armillaria mellea (Shigo and Tippett 1981).

devi bro upw 198

invi

FC

s

£1

c1

S

o

U

S

20

As roots die in fir, and as microorganisms invade the wood within Part I, the wood may develop into wetwood, or either white rot or brown rot. Large columns of wetwood often develop upward into the trunk from dying roots (Schütt 1981).

New clectrical methods have been developed co detect wetwood and decayed wood in fir and struce (Davis et al. 1980, Blanchard et al. 1983). These methods make it possible to determine the width of sound wood, and to determine acther wetwood is present.

Spruce has a stronger Part I CODIT than fir. For this reason the columns of defect are usually smaller and end more abruptly in spruce than in fir. Ring shakes are common in spruce, but basal cracking is not as common in spruce as in fir. Spruce does not have as much wetwood. But spruce often has more problems with fungi associated with branch death. Fomes pini is common in spruce Spruce seems to resist such infections very well until the number of infections becomes overwhelming, then the decay processes develop rapidly to form large central columns of defect.

What Can Be Done?

Excessive logging damage must be stopped now! Logging wounds--roots, trunks, branches-are major starting points for many types of defects including long cracks commonly called frost cracks. Frost does not start the cracks, wounds do (Butin and Shigo 1981).

Pruning practices must be adjusted now. Cuts behind the branch bark ridge and the injuries to the branch collar start many types of problems, including blue stains, ring shakes, vertical cracks, and cankers. Proper pruning (Shigo 1982a) does not mean leaving a stub, but the small branch collar must remain on the stem. If trees are pruned at an early age, this slight bulge will not reduce the width of clear wood for boards. But if the collars were removed, cracks and internal defects will form. Add to this the sawing of boards which contain the pith, and the splits will break outward, or the wet pockets associated with the branches will make drying difficult.

A logging wound reduction program must be started. Operators of machinery must be made aware of the damage that can result from wounds on the roots, trunks, and branches. Excessive logging damage must not be tolerated.

Decide whether the site will support highquality trees. Measurements of cambial electrical resistance may help in making this decision.

Highly defective trees should be removed. Trees with basal cracks, especially balsam fir, should be first. If many cracked trees are growing on one site, it may be a root rot site, and the likelihood of growing quality trees there would be remote. Know what you have on inventory plots. Use the Shigometer to determine internal condition of select trees. Use the Shigometer to help determine vitality of the trees.

Such a program involving removal of defective trees, thinning, proper pruning, and reduction of logging wounds on sites that will support high-quality trees, and the use of the Shigometer could result in a great increase in tree quality in a relatively short time.

Defective trees in our forests are primarily the result of long periods of high grading, taking the best and leaving the worst. In many of our forests, only the worst trees remain. It can be argued whether these trees are really genetically weak or tough trees, and that if given a reasonable change, whether they would grow into high-quality trees. I doubt this.

In an experiment involving deep drill bit wounds in sugar maples, paper birch, and yellow birch, selected as superior for growth rate and form, only a few of the trees were designated as strong compartmentalizers based on the volume of defect associated with the experimentally inflicted wounds.

At the same time we know from these studies and from other studies by our cooperators and other researchers that some individuals of a species are very strong compartmentalizers; they do limit defects to very small volumes. Studies on several species show that resistance to spread of decay is under moderate to strong genetic control (Shigo et al. 1977, Garrett et al. 1979, Schmitt et al. 1978, Lowerts and Kellison 1981). Genetic control is the key to future highquality forests. We must increase our programs to select strong-compartmentalizing trees, and also to find faster and more accurate ways of identifying tough trees.

Biochemical enzyme markers may be a way to do this. Wounding studies are being done now on many species of conifers and hardwoods. Studies are also being done on very young trees to determine whether tough trees can be selected very early.

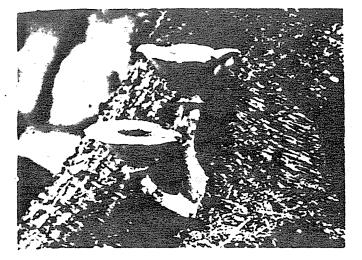
Conclusions and Warning

The worldwide pattern of poor tree health leading to many types of forest problems must be reversed before it is too late. Scientists must turn their attention to starting points of the problems rather than to the symptoms, secondary agents, and ends of the destructive processes. We cannot change decades, or even centuries, of mistreatment of our forests in a few years. We all share an education responsibility. We must make certain that decisions on our forests come from properly conducted experiments with controls. Learn what a tree is and learn how it functions to survive. Then let us help the tree help itself to survive in a healthy state. If we do not follow such a course, the fight against symptoms and secondary agents will continue, while more of our world forests die.

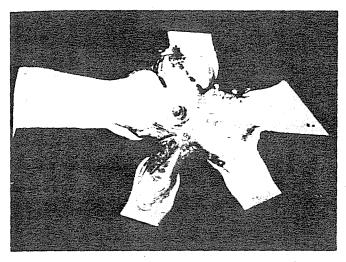
Literature Cited

- Blanchard, R.O., W.C. Shortle, and W. Davis. 1983. Mechanism relating cambial electrical resistance to periodic growth rate of balsam fir. Can. J. For. Res. 3:472-480.
- Butin, Heinz, and Alex L. Shigo. 1981. Radial shakes and "Frost cracks" in living oak trees. USDA Forest Service Research Paper NE-478. 21 p.
- Davis, Weston, Walter Shortle, and Alex L. Shigo. 1980. Potential hazard rating system for fir stands infested with budworm using cambial electrical resistance. Can. J. For. Res. 10:541-544.
- Garrett, P.W., W.K. Randall, A.L. Shigo, and W.C. Shortle. 1979. Inheritance of compartmentalization of wounds in sweetgum (Liquidambar styraciflua L.) and eastern cottonwood (Populus deltoides Bartr.) USDA Forest Service Research Paper NE-443. 4 p.
- Lowerts, G.A., and R.C. Kellison. 1981. Genetically controlled resistance to discoloration and decay in wounded trees of yellow poplar. Silvae Genet. 30:98-101.
- Schmitt, Dan, Peter Garrett, and Alex Shigo. 1978. Decay resistant hardwoods? You bet! North. Logger 27 (3):20-21, 30-31.
- Schütt, P. 1981. Die verteiling des Tannennasskerns in Stamm und Wurzel. Forstwiss. Centralbl. Heft 3-4:174-179.
- Shigo, Alex L. 1979. Tree decay: An expanded concept. USDA Forest Service Agriculture Information Bulletin 419. 73 p.
- Shigo, Alex L. 1980. Some myths and misconceptions. Forest Notes, Summer 1980. 2-7.
- Shigo, Alex L. 1982a. A pictorial primer for proper pruning. Forest Notes, Spring 1982. -4 p.
- Shigo, Alex L. 1982b. Tree decay in our urban forests: What can be done about it? Plant Disease 66:763-768.
- Shigo, Alex L. and Harold Marx. 1977. Compartmentalization of decay in trees. USDA Forest Service Agriculture Information Bulletin 405. 73 p.
- Shigo, Alex L., and Joanna Tippett. 1981. Compartmentalization of decayed wood associated with <u>Armillaria mellea</u> in several tree species. USDA Forest Service Research Paper NE-488. 20 p.
- Shigo, Alex L., Walter C. Shortle, and Peter W. Garrett. 1977. Genetic control suggested in compartmentalization of discolored wood associated with tree wounds. For. Sci. 23: 179-182.
- Shortle, Walter C. 1979. Compartmentalization of decay in red maple and hybrid poplar trees. Phytopathology 69:410-413.
- Shortle, W.C., and A. Ostrofsky. 1983. Decay susceptibility of wood in defoliated fir trees related to changing physical, chemical, and biological properties. Eur. J. For. Pathol. 13:1-11.
- Tippett, Joanna T., and Alex L. Shigo. 1981. Barriers to decay in conifer roots. Eur. J. For. Pathol. 11:51-59.

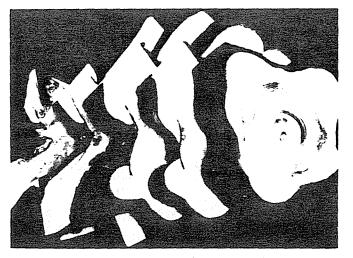
- Tippett, Joanna T., A.L. Bogle, and A.L. Shigo. 1981. Response of balsam fir and hemlock roots to injuries. Eur. J. For. Pathol. 12:357-364.
- Wargo, Philip M. 1971. Seasonal changes in carbohydrate levels in roots of sugar maple. USDA Forest Service Research Paper NE-213. 8 p.
- Wargo, Philip M. 1975. Estimating starch content in roots of deciduous trees--A visual technique. USDA Forest Service Research Paper NE-313. 9 p.
- Wargo, Philip M. 1976. Variation of starch content among and within roots of red and white oak trees. For. Sci. 22:468-471.
- Wargo, Philip M. 1979. Starch storage and radial growth in woody roots of sugar maple. Can. J. For. Res. 9:49-56.



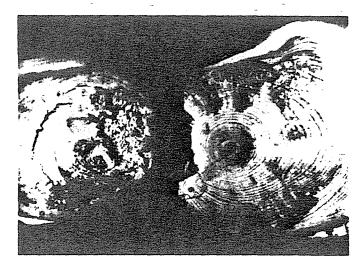
Armillaria mellea is a major cause of root rot in spruce and fir.



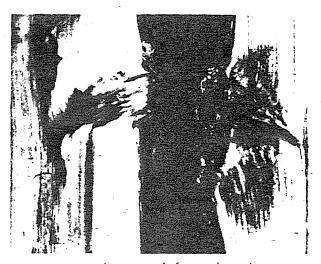
Base of fir showing advanced decay associated with A. mellea between the roots.



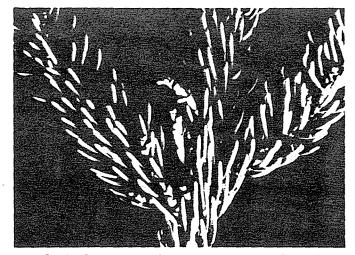
Basal sections from a fir show wetwood associated with root rot.



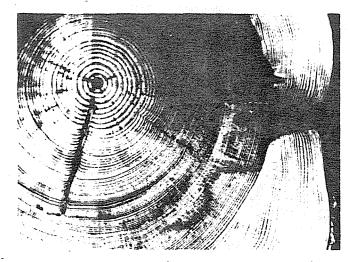
Two patterns of basal rot associated with A. mellea; left, center sound, right, decay advanced into the center.



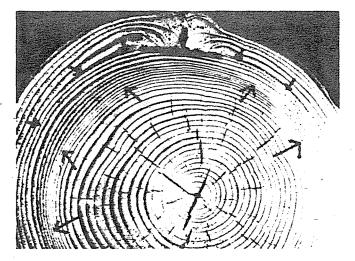
A proper pruning cut, left, and an improper flush cut, right, on a red pine after 2 years. Cuts should not be made behind the swollen collar at the branch base.



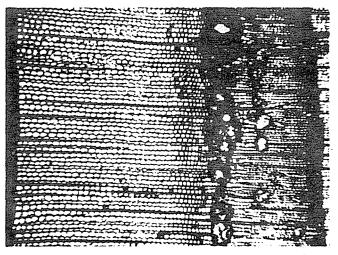
Dwarf mistletoe on red spruce can cause brooming, swollen trunks, and general decline of trees.



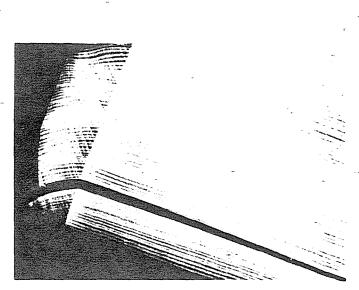
Wound on red spruce. Logging wounds are major causes of defects.



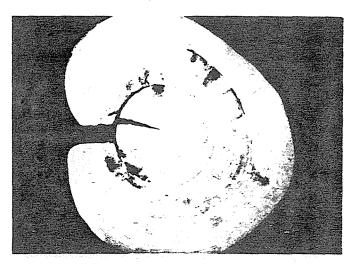
Small wound on Norway spruce. Small arrows show that barrier zone that formed after wounding. Large arrows show inward limit of resin ducts. Note lack of resin ducts behind wound.



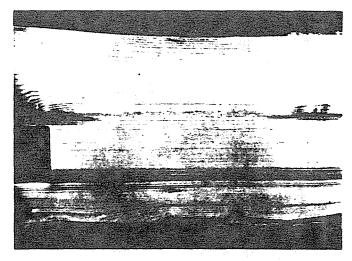
Microscopic view of the barrier zone 2 cm left of wound shown above. The barrier zone is the starting point for most of the shakes in trees.



Ring shake in red spruce. Barrier zones associated with wounds and dead branches often start the shakes.



Two major wounds in this red spruce limit its use for quality timber.



The 2 small wounds in the red spruce sample are the starting points for ring shake.