Resistance to diseases and pests in forest trees

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Trees resistant to spread of decay associated with wounds

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ABSTRACT

Trees are higly compartmented perennial plants. Normal growth is dependent on shedding, walling off, and regenerating new tissues. Trees cannot move. They have evolved under the constant stress of wounds and a wide variety of organisms that infect wounds. Trees cannot prevent wounds and infection. Trees survive after injury and infection by also shedding, or walling off the infected tissues and then by regenerating similar tissues in a new position. Trees that have the ability to shed or wall off rapidly and effectively, and to regenerate new tissues rapidly, will survive for a long time under the stress of many infections. Walling off processes in the bark and xylem are non-specific to type of injury or organism. Therefore, controlled wounding techniques where the response of the tree can be measured accurately, may be an effective way to determine a tree's genetic capacity to survive after injury and infection by a wide variety of organisms. Preliminary results from wounding experiments on several tree species indicate that the walling off processes are under strong genetic control. Some individuals in a species walled off decayed wood to very small volumes while other individuals still walled off the decayed wood, but to much larger volumes. A large scale pilot project using this information is now in progress to select trees with strong resistance to the spread of decay associated with wounds.

INTRODUCTION

Decay is a major cause of low quality in trees throughout the world. The economic loss is extremely high. Decay is also a major cause of hazard trees. When such trees fall they cause a great amount of damage to power lines, property, and people. With all this damage caused by tree decay, why

has so little been done to produce trees resistant to decay?

The literature provides very little information on the subject. The textbook by Wright (1976) on forest genetics does not discuss it. Boyce (1961), in his fine forest pathology text, gives only a few comments on resistance to tree decay. Proceedings from past conferences on forest genetics show that tree decay was not discussed. Several investigators have mentioned decay resistance, but in wood products. It must be kept in mind that root diseases are different in many ways from those initiated by mechanical wounds. For a current review and excellent background information on resistance to root and butt rot in conifers, see von Weissenberg (1980).

The question remains, why so little progress? The answer is that geneticists have not been given an accurate account of the decay process and reliable simple methods to work with tree decay. Geneticists need to know how a tree is set anatomically and biochemically to resist decay, and how the decay process operates. Geneticists need accurate methods for detecting, measuring, and working with tree decay. Given these basics they can progress rapidly with the many fine techniques and basic information already well developed in the field of genetics.

Advances in science are made not on the presentation of new observations, hypotheses, and methods alone, but on the combination or connection of these, whether they are old or new, in such a way that a new perspective emerges. It is with this rationale that this paper is presented: it is hoped that a new perspective will emerge.

BACKGROUND

Trees are perennial plants with well developed systems for shedding parts after they have been used or injured. Trees shed fruits, leaves, twigs, branches, bark, and roots. The openings left by the parts shed are often where microorganisms attack. To defend itself, the tree must have systems to close or to wall off the opening. Timing is extremely important. If the shedding and walling off are not well synchronized, the pathogen will gain the advantage to grow into the tree. This process is seen best in the shedding of branches. Trees also wall off injured and infected tissues in bark (Mullick, 1977; see also the preceding chapter), trunk wood (Shigo, 1979b), and root wood (Tippett & Shigo, 1980). It is conjectured that the same processes that became well developed through evolution for shedding parts, were used for walling off injured and infected tissues that by nature of their position in the tree could not be shed easily. Just as the normal shedding processes are timed to best benefit the tree, there must be times when the walling off processes induced by injury and infection function most rapidly and effectively, and times when they do not.

Trees are constructed as highly compartmented plants. After wounding they wall off the injured and infected wood (Shigo, 1979b). After wounding,

many types of microorganisms infect the wound surface and exert a force to grow into the tree, and to digest the wood. The microorganisms may spread and determine the rate of decay, but the tree defines the limits of the decayed wood. To understand the compartmentalization process more easily, we developed a simple model, CODIT, an acronym for Compartmentalization Of Decay In Trees (Shigo & Marx, 1977).

The CODIT model makes it possible to understand the 3-dimensional configuration of the developing column of discolored and decayed wood. The terms in the model - Walls 1, 2, 3 and 4 - must not be confused with the real process of compartmentalization.

After wounding, the cambium begins to form cells that differentiate into a barrier zone, wall 4 of CODIT (Sharon, 1973; Moore, 1978; Mulhern et al., 1979). Once the barrier zone forms, the continued spread of the microorganisms is confined to tissues extant at wounding.

Summaries of work on decay resistance and a description of a nondestructive method using the Shigometer for selection of strongly compartmentalizing trees in a species have been published (Shigo, 1979c; Schmitt et al., 1978).

RESEARCH RESULTS

Anatomical features

Geneticists have shown that many anatomical features are under genetic control (for a review see Hattemer, 1963).

Vessel features were strongly related to the degree of resistance to Dutch elm disease (Elgersma, 1970, and page 143 in this book; McNabb et al., 1970; Sinclair et al., 1979). Vessel features were also strongly associated with wounds in American elm (Shigo et al., 1980). Vessel grouping was the important feature. Trees that had rapid lateral spread of discolored wood had vessels closely grouped. The strongly compartmentalizing trees had large single vessels surrounded by many fibers. A similar situation was reported by Eckstein et al. (1979) for hybrid poplar.

Vessel grouping may be the main feature limiting the lateral spread of infection in wood, but vessels may also be important in limiting vertical spread (Bauch et al., 1980). In red maple (*Acer rubrum* L.) the ability to plug vessels rapidly after wounding appeared to be the main feature limiting vertical spread. For elm, see page 143 in this book.

In the wound studies, the column sizes of discolored wood associated with all the wounds were fairly uniform for each tree, suggesting that it was the tree that mainly regulated the dimensions of the discolored wood. A similar result was reported by Ekman & von Weissenberg (1979) for *Picea abies* (L.) Karst. Little is known about the plugging features in conifers.

In birch (*Betula papyrifera* Marsh., and *B. alleghaniensis* Britt.) lengths of discolored columns associated with drill wounds were correlated

with lengths of vessels (Bauch et al., 1980).

Biochemical features

Shortle (1979) gives an excellent discussion of the biochemical mechanisms of compartmentalization. He shows how biochemistry relates to the CODIT model. In other studies not yet published, he has shown that wood from strongly compartmentalizing red maples and hybrid poplars does not support rapid growth of selected wood decay fungi in laboratory experiments. Wood from weakly compartmentalizing trees supported more growth of the fungi.

Compartmentalization in hybrids

The original studies that suggested genetic control of compartmentalization of decayed wood associated with wounds were done on hybrid poplars (Garrett et al., 1976; 1979; Shigo et al., 1977). Santamour (1979) did studies on compartmentalization in red maple (*Acer rubrum* L.), and silver maple (*A. saccharinum* L.) and hybrids of these. His conclusion was that compartmentalization of discolored wood was under genetic control.

Roots

Decayed wood associated with *Heterobasidion annosum* (Fr.) Bref. was compartmentalized in roots of red pine (*Pinus resinosa* Ait.) (Shigo, 1979a). Compartmentalization may help to explain why trees that appear healthy and green can still have so many decayed roots. Trees may remain alive after many infections because they constantly compartmentalize infected tissues to small volumes. Recent results suggest the same pattern of compartmentalization in roots of several tree species infected with *Armillaria mellea* (Vahl. ex Fr.) Quel.

Great care must be taken here with the root infecting fungi that are also associated with decayed wood. They have the ability to act as pathogens first: they kill cambial tissues and a lesion forms. The tree responds by limiting the size of the lesion. In some cases the fungi spread so rapidly in the bark that the root or trunk is killed. But after the spread of the lesion is stopped by the tree, the fungi have the first opportunity to grow rapidly into the wood beneath the lesion.

Bark

It may be that some of the information gained from studies of walling off in wood will be applicable to bark. The work of Mullick (1977) does show that bark forms a protective new periderm after injury. It is a type of walling off. His work will have great application to many bark diseases, and may well fit with *H. annosum* and *A. mellea*. Again, how fast and how effectively the tree walls off infections in the bark determines the size of the lesion.

Wood decay

To this point 2 types of decay-causing fungi have been discussed. First, the fungi that enter wood through wounds. They stay in the wood. This is the type that this paper addresses mainly. Second, the type that start in the bark and then move into the wood. Here the tree must first limit their spread in the bark, and then in the wood. When they grow into the wood, then the information given here is applicable. A third type, not discussed in this paper should be mentioned. These fungi start in the wood after wounding and later grow into the bark. They are the canker rot fungi (Shigo, 1969).

Spread

It may not be the number of infections that injure or kill a tree, but the extent or spread of each infection. A tree that cannot respond rapidly to the spread of infection and hold the infection in place will die. It is extremely important to note that infections per se do not kill a tree. If they did, no tree would live more than a few years, because every tree has small and large wounds and branch stubs that are infected. There is hardly a tree that does not have hundreds of infections at any one time. Trees survive as long as they are able to wall off the spread of infections to small volumes, and then regenerate new healthy tissues.

Heartwood compartmentalization

The heartrot concept states that decay-causing fungi infect dead, non-responsive heartwood exposed by wounds, and that fungi grow through the heartwood until all is digested, leaving a hollow (Boyce, 1961). It was also believed that because the decay fungi infect dead, non-responsive heartwood, that decay was not a disease. And because heartwood was dead, it would be impossible to select or breed for features in dead, non-responsive tissue. These beliefs have held back progress on selecting and breeding trees resistant to decay.

Heartwood may be dead according to our definition, but it is surely not non-responsive. When holes were drilled into healthy heartwood, a column of discolored heartwood formed and was compartmentalized (Shigo & Shortle, 1979). The CODIT model is applicable to heartwood!

This point is very important: investigators must not think it impossible to select and breed for strong compartmentalization of decay in heartwood. The methods described for selection of strongly compartmentalizing trees are also applicable for heartwood.

The heartwood confusion is at the very core of the reason there is no progress in the development of a decay-resistant tree. Rennerfelt (1946) stated that because decay goes on in dead wood of spruce it would be impossible to have physiologically controlled resistance.

Pilot study for selecting decay-resistant trees

The United States Forest Service is now conducting a large pilot study in natural forests in the north-eastern United States to select trees resistant to the spread of decay. The trees in the study, over 200, were those already selected as superior on the basis of form and growth rate. The species are *Acer saccharum* Marsh., *Betula alleghaniensis* Britt., and *B. papyrifera* Marsh. Each tree received 4 large, deep drill wounds at 1.4 m aboveground. After 2 growing seasons the Shigometer method will be used to determine the length of the columns associated with the wounds. The trees with the smallest columns will be selected as superior, not only for growth rate and form, but for their ability to compartmentalize defects associated with wounds. Details on methodology have been published (Shigo et al., 1977).

REFERENCES

Bauch, J., A.L. Shigo & M. Starck, 1980. Wound effects in the xylem of Acer and Betula species. Holzforschung 34: 153-160.

Boyce, J.A., 1961. Forest pathology. McGraw-Hill Book Company, New York/London. 572 p.

Eckstein, D., W. Liese & A.L. Shigo, 1979. Relationship of wood structure to compartmentalization of discolored wood in hybrid poplar. Canadian Journal of Forest Research 9: 205-210.

Ekman, R. & K. von Weissenberg, 1979. Sapwood extractives in Norway spruce inoculated with Fomes annosus. Acta Academiae Aboensis, B. 39, No 7, 8 p.

Elgersma, D.M., 1970. Length and diameter of xylem vessels as factors in resistance of elms to Ceratocystis ulmi. Netherlands Journal of Plant Pathology 76: 179-182.

Garrett, P.W., A.L. Shigo & J. Carter, 1976. Variation in diameter of central columns of discoloration in six hybrid poplar clones. Canadian Journal of Forest Research 6: 475-477.

Garrett, P.W., W.K. Randall, A.L. Shigo & W.C. Shortle, 1979. Inheritance of wound healing in sweetgum (Liquidambar styraciflua L.) and Eastern cottonwood (Populus deltoides Bartr.). United States Forest Service Research Paper NE-443. 4 p.

Hattemer, H.H., 1963. Estimates of heritability published in forest tree breeding research. Proceedings of the World Consultation on Forest Genetics and Tree Improvement. Stockholm, Sweden. Vol 1, session 2a, paper 3. FAO/FORGEN-63. 14 p.

McNabb, H.S., H.M. Heybroek & W.L. MacDonald, 1970. Anatomical factors in resistance to Dutch elm disease. Netherlands Journal of Plant Pathology 69: 1158-1159.

Moore, K.E., 1978. Barrier zone formation in wounded stems of sweetgum. Canadian Journal of Forest Research 8: 389-397.

Mulhern, J., W.C. Shortle & A.L. Shigo, 1979. Barrier zones in red maple: an optical and scanning electron microscope examination. Forest Science 25: 311-316.

Mullick, D.B., 1977. The non-specific nature of defense in bark and wood during wounding, insect and pathogen attack. Recent Advances in Phytochemistry 11: 395-411.

Rennerfelt, E., 1946. Om rotrotan (Polyporus annosus Fr.) i Sverige. Dess utbredning och satt att upptrada. Meddelanden fran Statens Skogsforskningsinstitut 35(8): 1-88.

Santamour, F.S. Jr., 1979. Inheritance of wound compartmentalization in soft maples. Journal of Arboriculture 5: 220-225.

Schmitt, D., P.W. Garrett & A.L. Shigo, 1978. Decay resistant hardwoods? You bet! Northern Logger and Timber Processor, September. p. 220-225.

Sharon, E.M., 1973. Some histological features of Acer saccharum wood formed after wounding. Canadian Journal of Forest Research 3: 83-89.

Shigo, A.L., 1969. How Poria obliqua and Polyporus glomeratus incite cankers. Phytopathology 59: 1164-1165.

Shigo, A.L., 1979a. Compartmentalization of decay associated with Heterobasidion annosum in roots of Pinus resinosa. European Journal of Forest Pathology 9: 341-347.

Shigo, A.L., 1979b. Tree decay: an expanded concept. United States Department of Agriculture Information Bulletin 419. 73 p.

Shigo, A.L., 1979c. Decay resistant trees. North-eastern Forest Tree Improvement Conference Proceedings. School of Forest Resources. Pennsylvania State University. p. 64-69.
Shigo, A.L. & H.G. Marx, 1977. Compartmentalization of decay in trees (CODIT). United States Department of Agriculture Information Bulletin 405. 73 p.
Shigo, A.L. & W.C. Shortle, 1979. Compartmentalization of discolored wood in heartwood of red oak. Phytopathology 69: 710-711.
Shigo, A.L., W.C. Shortle & P.W. Garrett, 1977. Genetic control suggested in compartmentalization of discolored wood associated with tree wounds. Forest Science 23: 179-182.
Shigo, A.L., R. Campana, F. Hyland & J. Anderson, 1980. Anatomy of elms injected to control Dutch elm disease. Journal of Arboriculture 6: 96-100.
Shortle, W.C., 1979. Mechanisms of compartmentalization of decay in living trees. Phytopathology 69: 1147-1151.
Sinclair, W.A., J.P. Zahland & J.B. Melching, 1975. Anatomical marker for resistance of Ulmus americana to Ceratocystis ulmi. Phytopathology 65: 349-352.
Tippett, J.T. & A.L. Shigo, 1980. Barrier zone anatomy in red pine roots invaded by Heterobasidion annosum. Canadian Journal of Forest Research 10: 224-232.
Weissenberg, K. von, 1980. Resistance of Picea abies to Heterobasidion annosum. Proceedings of the Fifth International Conference on Problems of Root and Butt Rot in Conifers. Ed. L. Dimitri. Hessische Forstliche Versuchsanstalt, Hann. Münden. p. 67-74.
Wright, J.W., 1976. Introduction to forest genetics. Academic Press, New York/London. 463 p.