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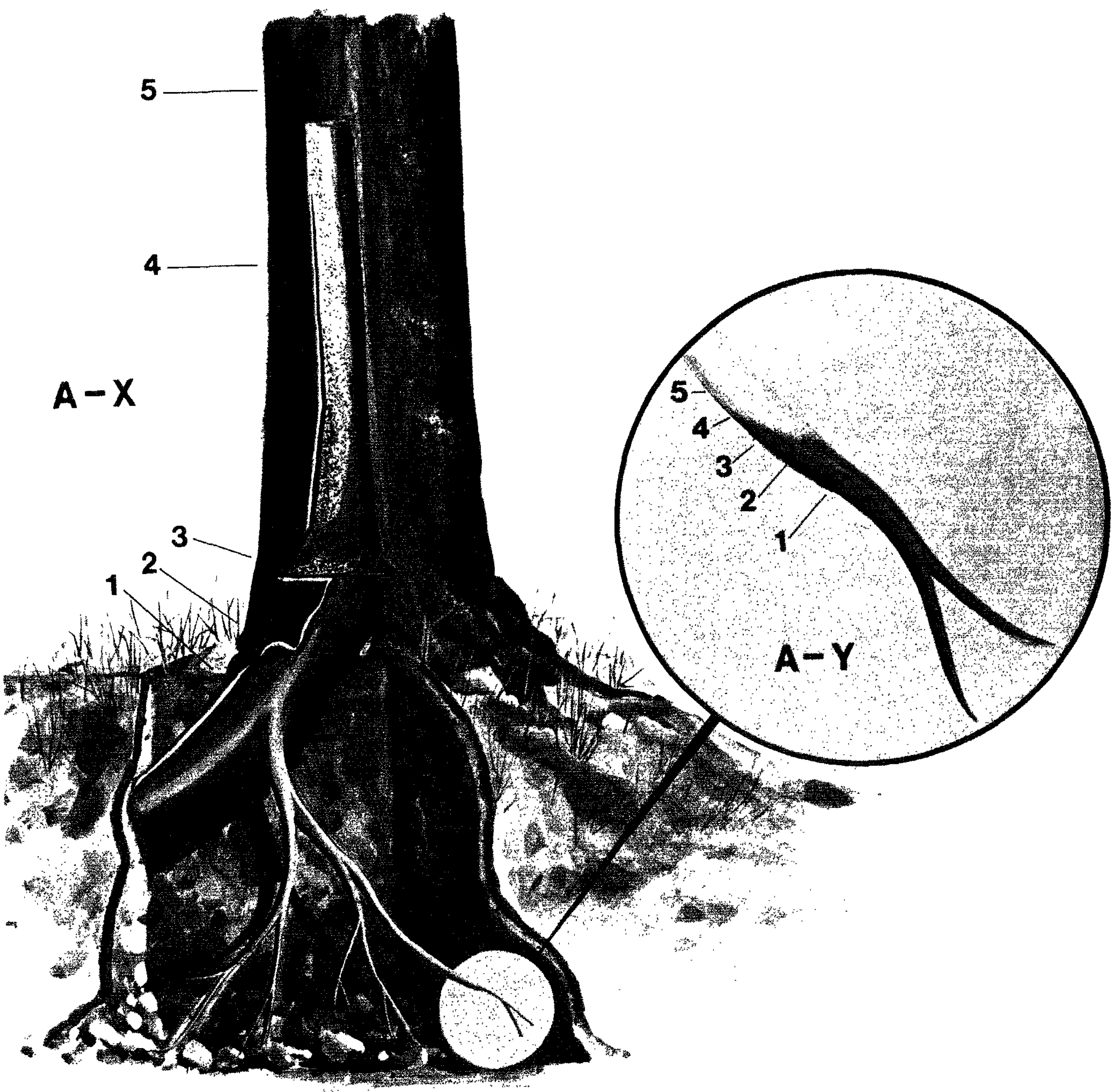
Compartmentalization of Decayed Wood

Associated with *Armillaria mellea* in Several Tree Species

by Alex L. Shigo and Joanna T. Tippett



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Abstract

Decayed wood associated with *Armillaria mellea* was compartmentalized according to the CODIT (Compartmentalization Of Decay In Trees) model. Compartmentalization in the sapwood began after the tree walled off the area of dead cambium associated with the inflection of the fungus. The fungus spread into dying sapwood beneath and beyond the area of killed cambium, but the fungus did not spread radially outward into new wood that formed.

Armillaria mellea (Vahl. ex Fr.) Kummer and other fungi cause root and butt rot of many species of woody plants. Some trees tolerate infections; others do not and they die quickly after infection (Rykowski 1975). Between these extremes are all gradations of injury caused by the fungus. There is an abundance of information and literature on *A. mellea* (Dimitri 1980; Shaw and Roth 1978).

The disease has four parts: (1) infection ; (2) spread into bark, cambium, and wood followed by walling off in those tissues by some trees; (3) compartmentalization in wood; and (4) decay of wood.

Armillaria mellea can infect and kill the bark, cambium, and wood when conditions for the fungus are favorable, or when conditions for tree health are unfavorable (Wargo and Houston 1974). Some trees of a species can stop the spread of the fungus; these trees continue to live after many infections. It is not fully known why other trees of the same species do not stop the spread of the fungus; these trees usually die after few infections.

From the walled-off dead bark, *A. mellea* can continue to grow deep into the sapwood beneath and beyond the limits of the dead area. It causes a white rot.

In this paper, bark killing refers to death of inner bark, cambium, and in some instances the most recently formed growth rings or rings of xylem. The tissues killed are called dead bark.

To clarify the points in this paper, it is also essential to understand the following terms: Sapwood has four major functions—storage, transport, protection, and support; heartwood has a protection and support function, but no storage or transport function; discolored wood does not have a storage and transport function, but has a support function, and a protective function that may be more or less than that of sapwood.

In CODIT, which is a model for compartmentalization of decay in trees, walls 1, 2, and 3 are movable, and wall 4 is stationary. Wall 1 resists vertical spread, wall 2 resists inward spread, and wall 3 resists lateral spread of microorganisms. Wall 4 separates the xylem present at the time of injury and infection from the xylem that forms later. Once formed, wall 4 remains in place, but walls 1, 2, and 3 may recede or give way to the pressure of the spreading microorganisms. Thus, discolored and decayed wood may increase in volume within the boundaries set by wall 4. A thorough understanding of CODIT is essential to understand the patterns of discolored and decayed wood associated with multiple infections of *A. mellea* over a period of many years.

The infection process, the factors affecting the spread of the fungus, and the walling off of the dead areas in the bark are beyond the scope of this paper. This paper focuses on the patterns of decayed wood associated with *A. mellea*.

The Study

Root systems of 30 trees were dug carefully by hand. The roots were washed, dissected, and studied. The trees ranged from 10 to 30 cm diameter at 1.4 m aboveground and were from 30 to 100 years old. In visual appearance, the trees ranged from suppressed dying to dominant healthy. The trees were in natural forests in southern Maine and central New Hampshire. The species were *Abies balsamea* (L.) Mill, *Picea rubens* Sarg., *Tsuga canadensis* (L.) Carr., *Populus tremuloides* Michx., *Fagus grandifolia* Ehrh., *Betula papyrifera* Marsh., *Betula alleghaniensis* Britt., *Acer rubrum* L., *Quercus rubra* L., and *Quercus alba* L.

Most of the trees had sporophores of *A. mellea* on roots and butts. Isolations from decayed wood confirmed the presence of *A. mellea* in roots that had no sporophores. More than 150 wood chips were taken from four *F. grandifolia* trees from dead basal trunk areas above large dead roots that had no sporophores. These wood chips were isolated. The small chips of wood were removed in an orderly pattern from base to top of the dead areas to determine the location of microorganisms. The isolation method and the malt-yeast medium were the same as those used in a previous study (Shigo 1977).

Dissections of roots and trunks were done by power and hand saws, knives, and razor blades. Selected wood samples with barrier zones were sectioned on a microtome. All large samples selected for examination were sanded smooth.

Results

The patterns of decayed wood associated with *A. mellea* in roots and butts can be explained by CODIT (Shigo and Marx 1977; Shigo 1979a).

When the bark killing associated with spread of *A. mellea* stopped before it circled a root or butt, the living cambium beyond the lateral and vertical limits of the dead bark area formed cells in the xylem that developed into a barrier zone (wall 4 of the CODIT model) (Figs. 1 and 2). The barrier zones formed in the early portion of the growth ring (Fig. 3). The position of the barrier zones indicated that the area of killed cambium associated with spread of *A. mellea* occurred during the dormant period, or soon after the onset of growth. The position of the barrier zones between two growth rings indicated that the area of dead bark was set in a short time, at least within one growing season.

In most instances, barrier zones form in response to injury and infection. The barrier zone was anatomically distinct from normal wood (Fig. 3). Anatomical details on barrier zones associated with *A. mellea* and other root-infecting fungi have been described (Tippett and Shigo 1980). After the barrier zone formed, normal xylem began to form again (Figs. 3 and 4). *Armillaria mellea* and other microorganisms spread deeper into the xylem beneath and beyond the area of dead bark, but they did not spread from the xylem present at the time of infection radially outward into the new xylem that formed after completion of the barrier zone (Fig. 5).

This boundary of barrier-zone tissue walled off decay even after many years (Fig. 2). In some roots, it was difficult to see an anatomically distinct barrier zone, but the new wood that formed after the bark stopped dying was free of *A. mellea* and other microorganisms.

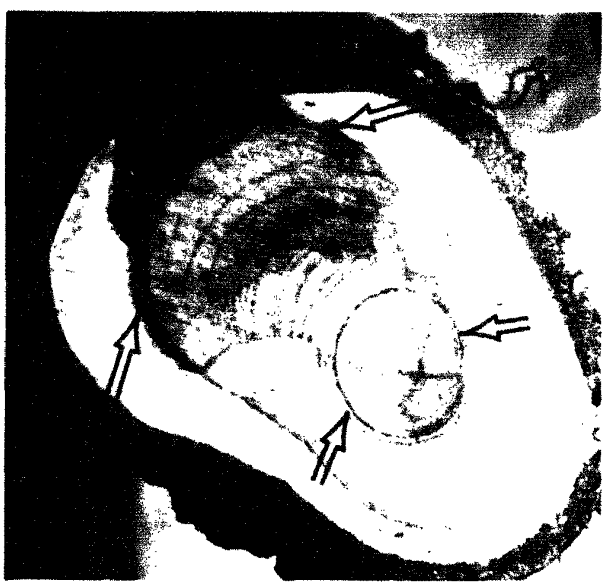

Figure 1.—Discolored and decayed wood associated with two infections in roots of *A. balsamea*. The root was infected when it was 8 years old (small arrows) and when it was 23 years old (large arrows). The arrows also point to the barrier zones. The distance between the two small arrows is 1 cm.

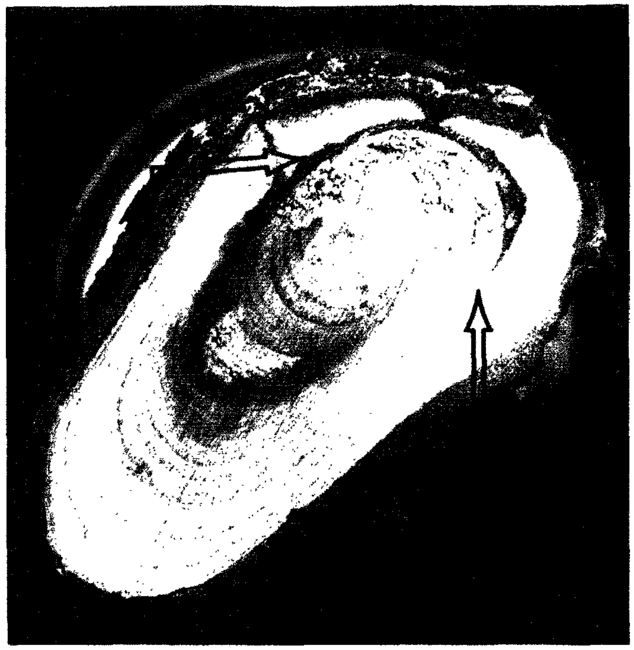

Figure 2.—Decayed wood associated with *A. mellea* was walled off by a barrier zone. Distance between the arrow points is 1.5 cm. The wood decayed to the center of the root.

When the wood in the center of roots and butts was healthy sapwood at the time of infection, *A. mellea* spread to the center (Figs. 6 and 7). When the wood in the center of butts was heartwood or discolored wood at the time of infection, *A. mellea* spread inward only to the outer margins of the heartwood or discolored wood (Figs. 8, 9, and 10). Heartwood extended downward from the butt into the transition zone between butt and root in the trees that have heartwood. Roots below this transition zone did not contain heartwood. Discolored wood associated with wounds and dead root stubs in roots looked similar to heartwood. Many wounds were found on the roots. When *A. mellea* spread upward from the roots into the root-butt transition zone that contained discolored wood or heartwood, the decayed wood was restricted to sapwood (Figs. 8, 9, 10, and 11). In species of *Fagus*, *Betula*, and *Acer* that had no heartwood, *A. mellea* spread to the center of the butt except when the butt contained discolored wood (Figs. 12 and 13).

The areas of dead bark were from very small, where a few small roots were killed, to very large, where many large roots and a portion of the trunk was killed (Figs. 12 and 13). Areas of dead bark associated with many small roots often

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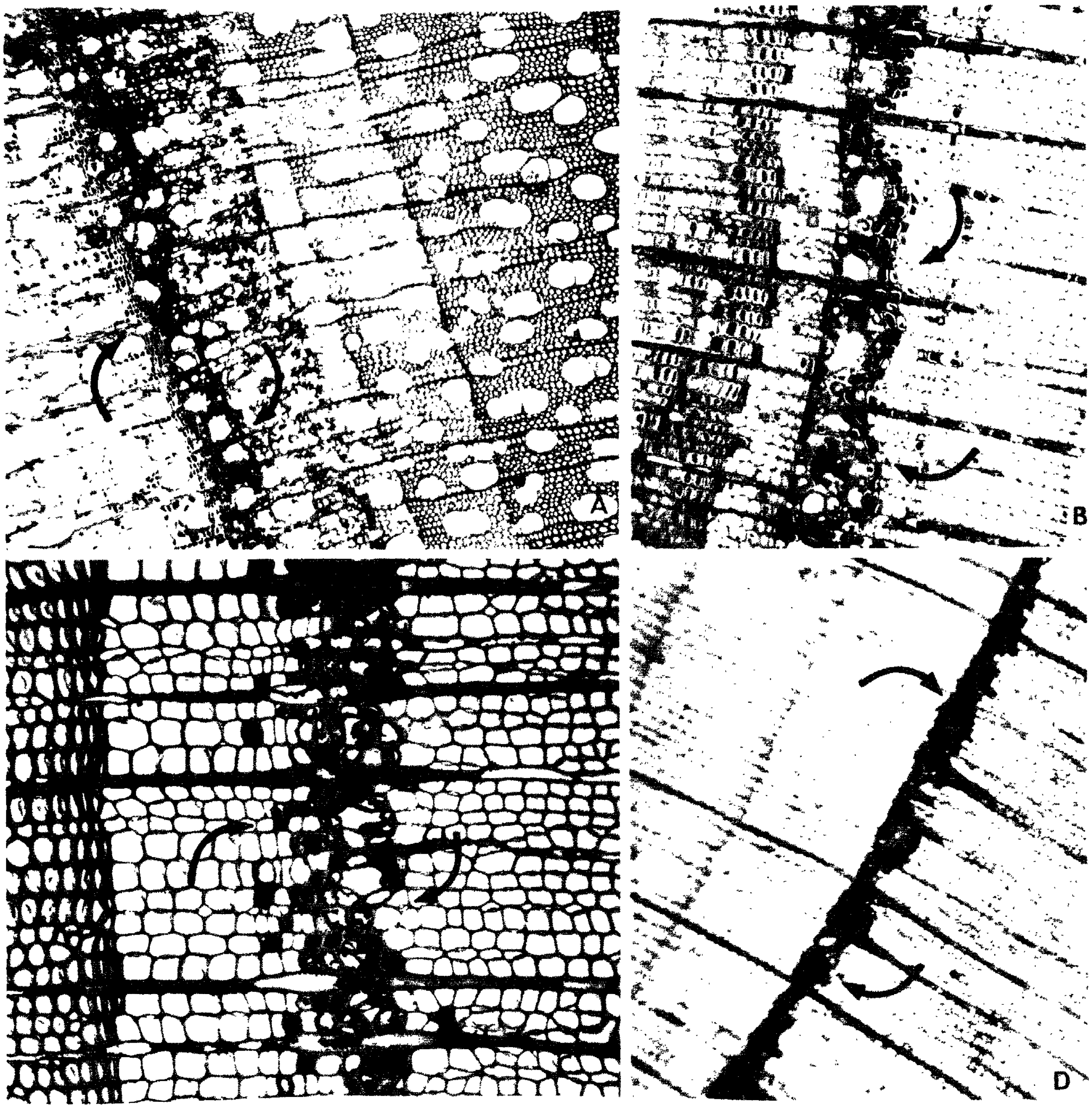

Figure 3.—Barrier zones (arrows) in *P. tremuloides* (A), *T. canadensis* (B), *A. balsamea* (C), and *P. rubens* (D).

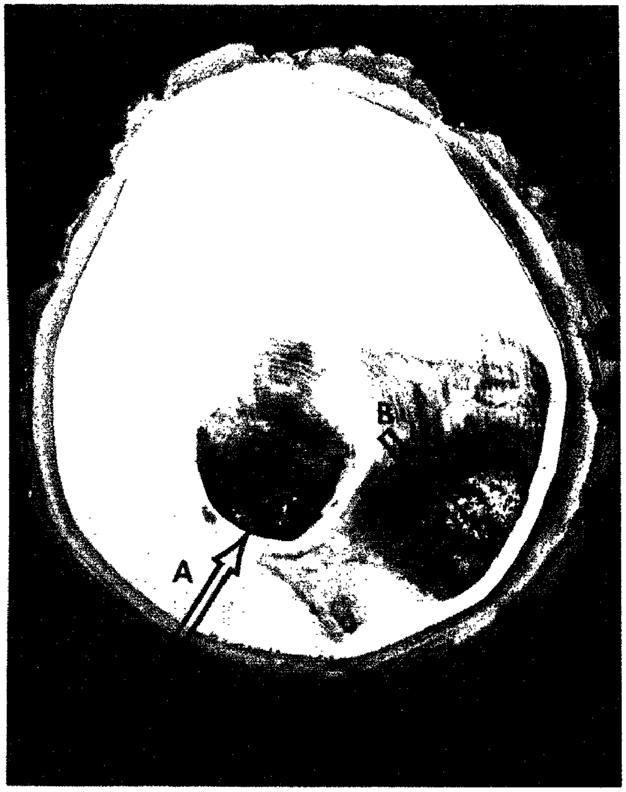

Figure 4.—Two columns of compartmentalized decayed wood in a butt of *P. tremuloides*. The section was cut 5 cm above the top of an approximately 10-year-old dead bark area, similar to that shown in figure 9. Arrow A shows the walled-off decayed wood associated with an early infection, and arrow B shows the same pattern for a later infection.

Figure 5.—Multiple columns of decayed wood associated with several infections in an *A. balsamea* tree. The butt section shown here was 15 cm in diameter. The arrows point to the barrier zones. The central hollow was associated with early infections. This tree had a green, suppressed crown.

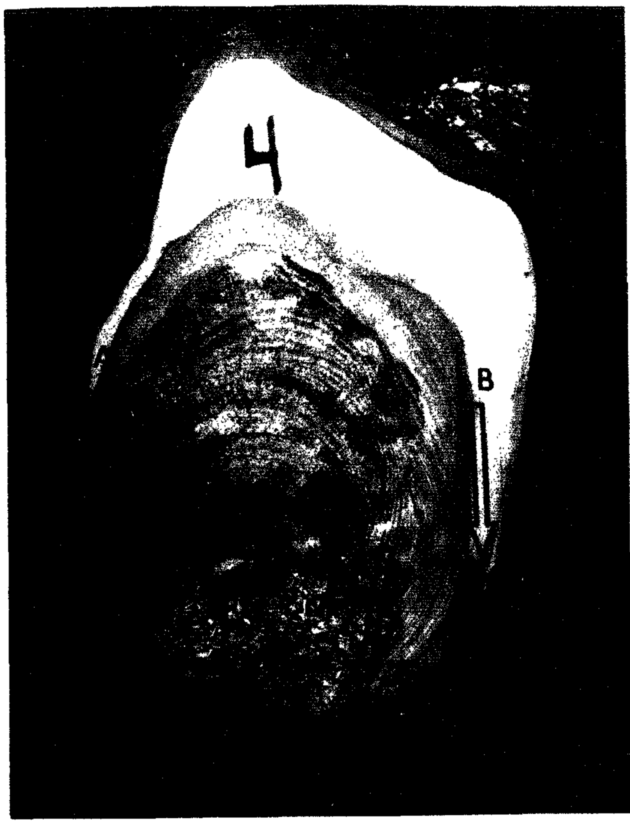

Figure 6.—Wood decayed in the center of this *P. rubens* butt which indicates that the central wood was healthy sapwood, not heartwood or discolored wood, at the time of infection. Arrows A show the lateral limits, or widths, of the early infection. Arrow B shows the extended limits of the second infection that occurred approximately 12 years later. The second infection was approximately 7 years before the tree was cut. Note the wide growth rings associated with the roots that remained alive after the first infection, and the very narrow growth rings that formed after the last infection.

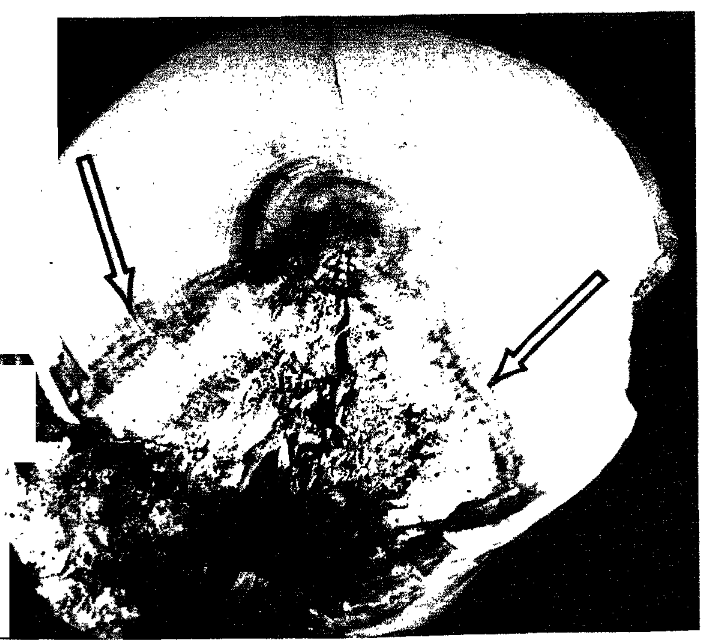

Figure 7.—Wood decayed to the center of this *P. tremuloides* butt which indicates that the central wood was healthy sapwood at the time of infection approximately 12 years ago. The arrows show the discolored wood that borders the decayed wood. The arrows indicate wall 3 of CODIT. The pressure of the developing decayed wood over the 12-year period pushed the lateral boundaries—wall 3—slightly beyond (arrows) the original width of the killed bark area.

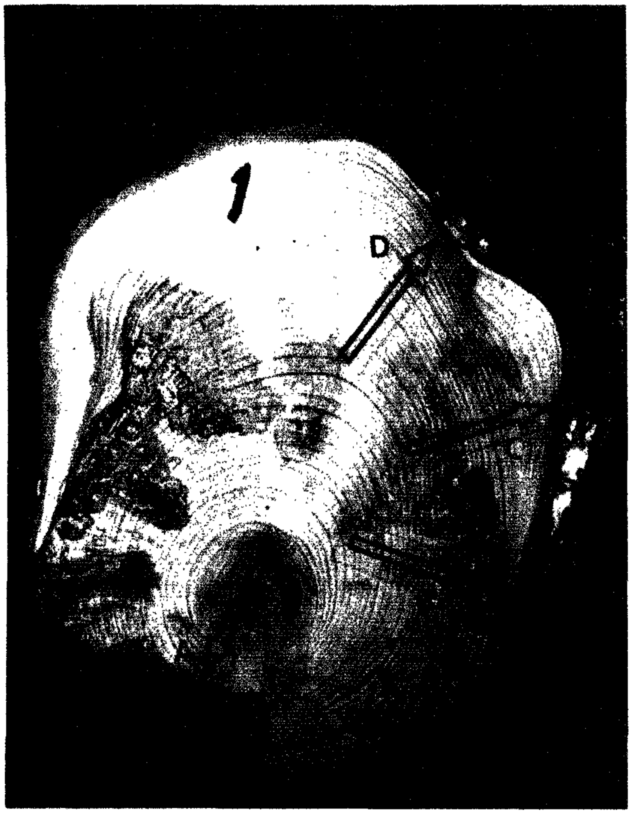

Figure 8.—Wood did not decay to the center of this *P. rubens* butt which indicates that the central wood was discolored wood or heartwood at the time of infection. Note the abrupt limit of decayed wood at arrow A. Arrow B shows the point where the lateral spread of the first infection stopped. Arrow C shows where an infection 15 years later extended the circumferential killing. The infection at arrow C was 1 year old. Also, note the heavy resin deposits in the bark at arrow C. Arrow D shows a cut through the top of a 1-year-old dead bark area. Again, there is heavy white resin deposit in the bark. This is typical for recent infections in bark.

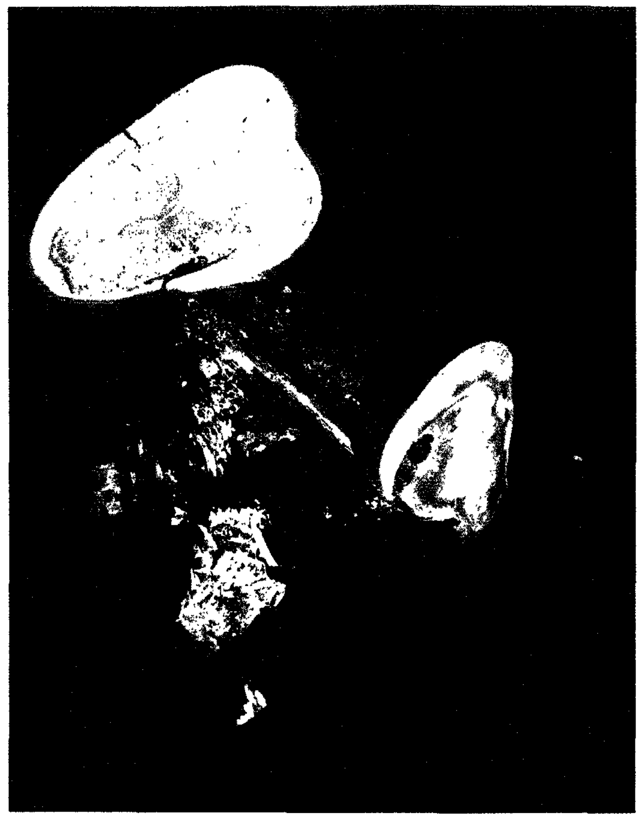

Figure 9.—The infection in this *P. tremuloides* was 21 years old. Note the triangular shape of the dead area as it developed up the butt from the large dead root. The advanced decay associated with *A. mellea* was primarily in sapwood at the time of infection. The fungi have slowly spread into the center of the butt, but it is difficult to see in this photograph. Over time, walls 1, 2, and 3 will slowly recede, but wall 4 will remain in position. This was occurring in the large dissected root, where walls 2 and 3 were moving after 21 years of infection, but wall 4 remained in position.

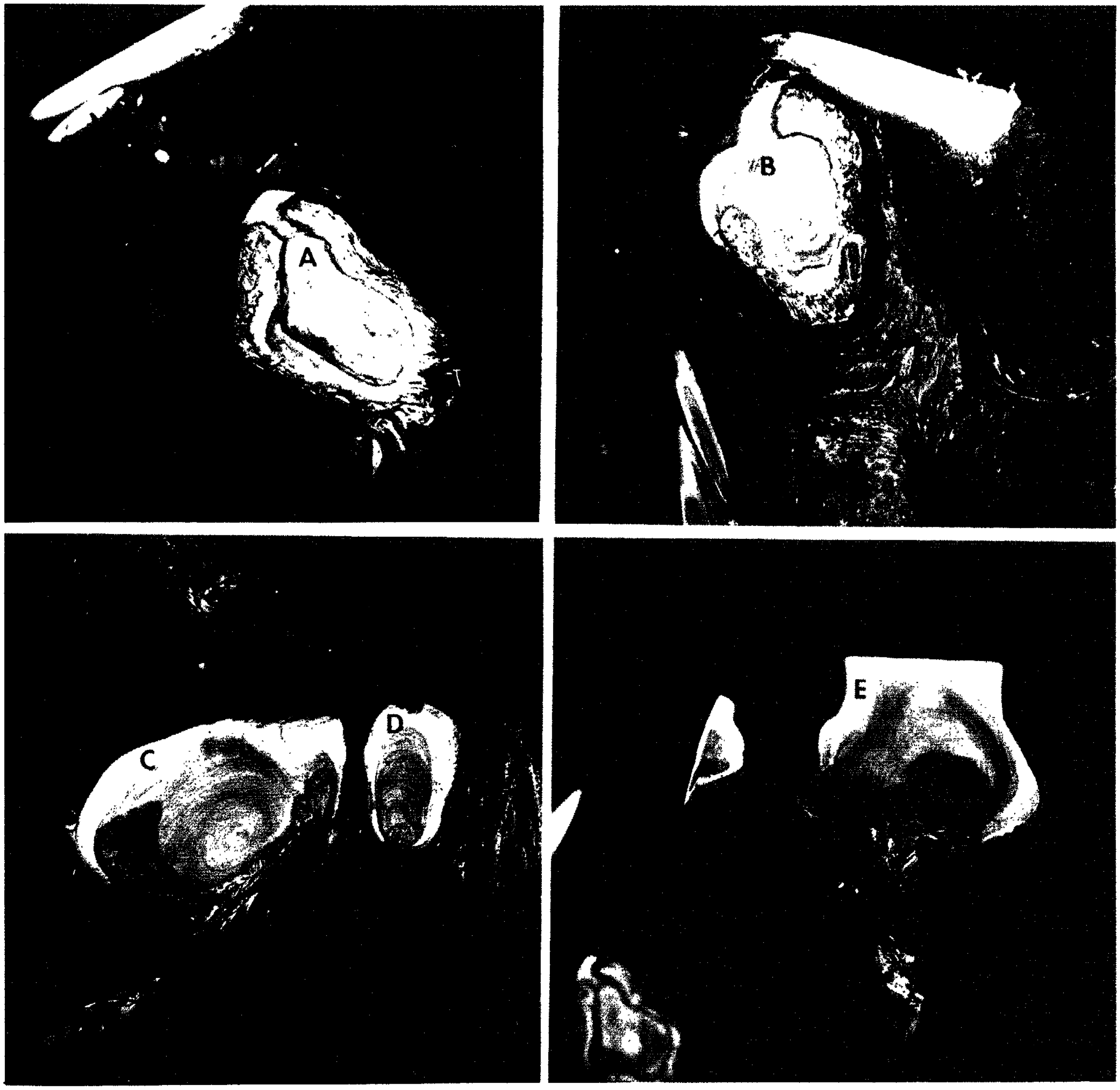

Figure 10.—Decayed wood associated with five roots of *A. balsamea* on the same stump. Roots A and B were almost completely girdled, roots C and E were about 50 percent girdled, and root D had only a small portion killed. The wood did not decay into the central column of discolored wood of roots A, B, and C, or outward into the wood that formed after infection in all roots. Wood did decay into the centers of roots D and E. Roots D and E apparently did not have central discolored wood at the time of infection. If the central wood in all roots was heartwood, and if *A. mellea* was able to infect heartwood, then all roots should have been decayed to the center.

Figure 11.—Wood in the butt of *A. balsamea* did not decay into the central discolored wood or outward into the wood that formed after the infection. A ring shake (arrow A) was associated with a barrier zone from an old wound. Arrow B shows the radial shake associated with the old wound. Note that the decay associated with *A. mellea* did not develop in the discolored wood around the radial shake. The faint discolored areas in this sample were associated with the tops of other columns of decayed wood associated with root infections.

Figure 12.—Half of the butt of this *F. grandifolia* was decayed. The infection force of *A. mellea* was as strong as the compartmentalizing force of the tree. When the infection force is stronger than the compartmentalizing force, the tree declines and may die. The wood decayed to the center of the butt which indicates that the central wood was not discolored wood at the time of infection. The infection into the butt was at least 15 years old. The large roots on this tree were in contact with large roots on an old dead tree.

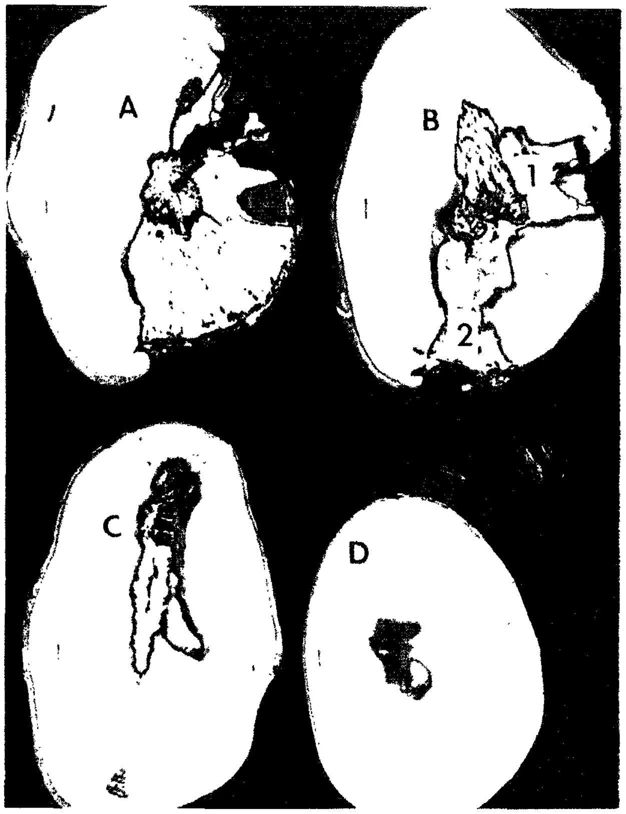

Figure 13.—Trunk sections, each 10 cm long, from above the *F. grandifolia* butt shown in figure 12. Section A, 10 cm above the butt, shows wood decayed to the center. Section B shows that two infection sites, 1 and 2, had coalesced in the butt. Section C, 30 cm above the butt, and section D, 40 cm above the butt, showed that the discolored and decayed wood was only in the center of the trunk. At the time of the infection, 50 percent of the butt was killed.

Figure 14.—The fungus-infected outer bark was walled off in this *P. tremuloides*. This sample is 10 cm above that shown in figure 7. The cambial killing attenuated as the dead bark area moved upward. The arrow shows where the cambial killing stopped.

coalesced to form one large dead area. The upward vertical extension of this dead area into the trunk was greatest when a large root or several roots were girdled.

The width of the areas of the dead bark was larger than the width of the decayed wood beneath the dead area (Fig. 14). It appeared that the fungus was also walled-off in the bark by necrophylactic periderm as described by Mullick (1977). The fungus in the bark appeared as wedges (Fig. 14). In the conifers, the wedges also contained great amounts of resin (Fig. 8).

Isolations from the dying and dead wood beneath the dead bark of the upper portions of the trunks in *F. grandifolia* yielded many bacteria, yeasts, and a variety of fungi, but not *A. mellea*. Yet, *A. mellea* was isolated with a high frequency from the wood beneath the bark of the lower portions of the trunk (Fig. 15). After several years, the limits of the dead bark area of the butt were bordered by callus (Fig. 16).

Bark killing was most extensive along the under sides of roots. When such dead areas extended upward to the trunk, it was the bark between the roots that was killed first (Fig. 17). On some species, long vertical basal cracks formed at this position. Figures 18 to 23 are diagrams of the points discussed and shown in the photographs.

Figure 15.—Current year dead bark area on a *F. grandifolia*. The sunken dead area on the butt was between two large infected roots. The arrows show the limits of the dead area. Isolations were made from wood chips taken from the base to the top of this dead area. *Armillaria mellea* was only isolated from the base of the dead area. The limits of the dead area are set in a very short time—a few weeks to a month—but the development of *A. mellea* into the dead area may take a much longer time.

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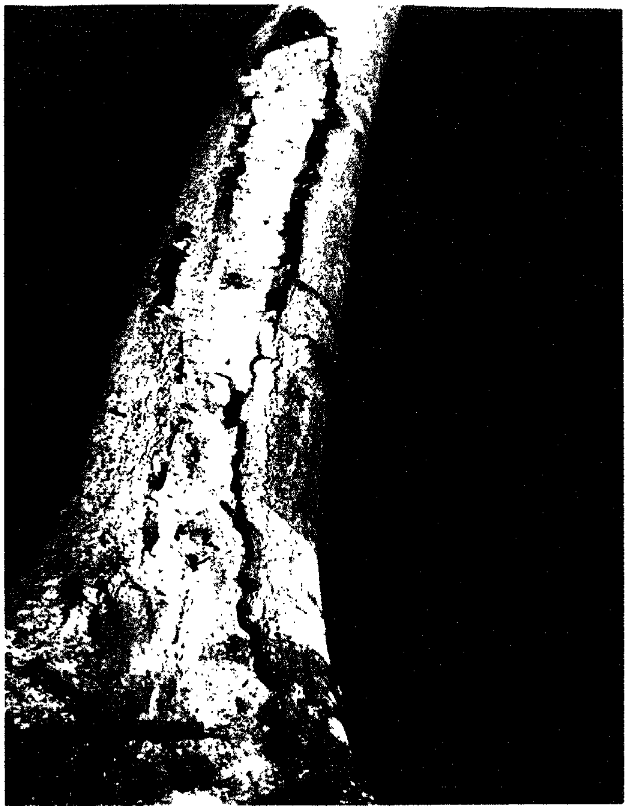


Figure 16.—Large ridges of callus mark the boundary of this old dead bark area on a *F. grandifolia.*

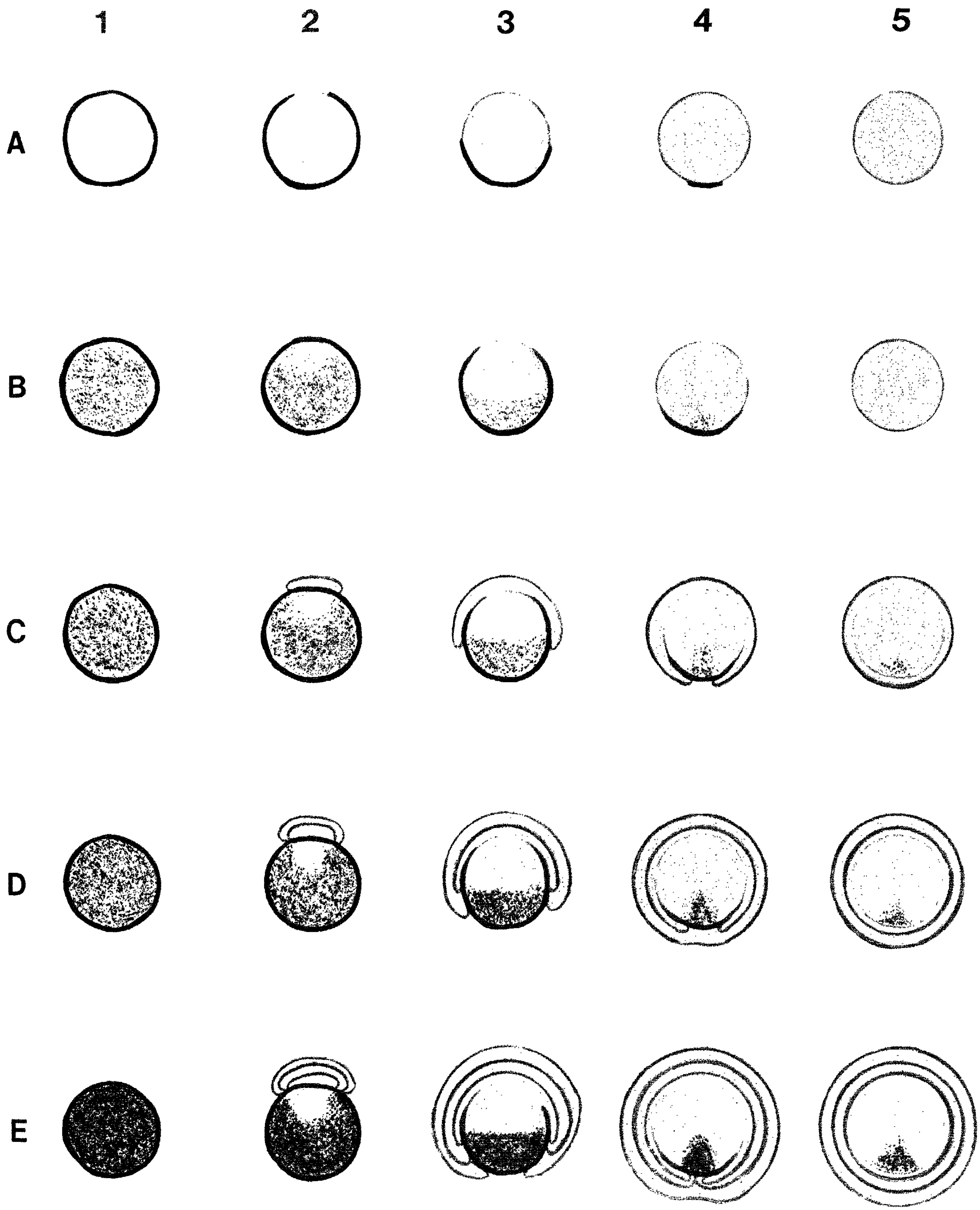
Figure 17.—Dissection of this *A. Balsamea* root base and butt shows the limits of the 7-year-old dead area. The wood decayed from the bottom of the roots upward on the butt between the infected roots. Note the fully-formed growth rings before and after the limits of the dead area (arrows). This indicates that the limits of the dead area were within a single growth period. The top section shows discolored wood associated with other infections.

Diagramatic Representations of Patterns of Compartmentalization Associated with *Armillaria mellea* in Roots and Butts.

Color Key

Green:	killed cambium and decaying wood
Red:	barrier zone
Blue:	growth rings

Figure 18.—Summary of general patterns associated with a single infection. Horizontal row A shows cross sections of one dead bark area from complete circumferential killing of a root tip, A-1, to healthy wood, A-5, directly above the vertical limit of the dead area. Row B shows the response of the living cambium after the bark killing has stopped. Rows C, D, and E show events after 1, 2 and 3 years, respectively. The vertical rows 1 to 5, show changes occurring over 3 years as viewed at the same cross-sectional plane of the root. (Letters, A-E, and numbers, 1-5, in the following figures refer to those given in this figure).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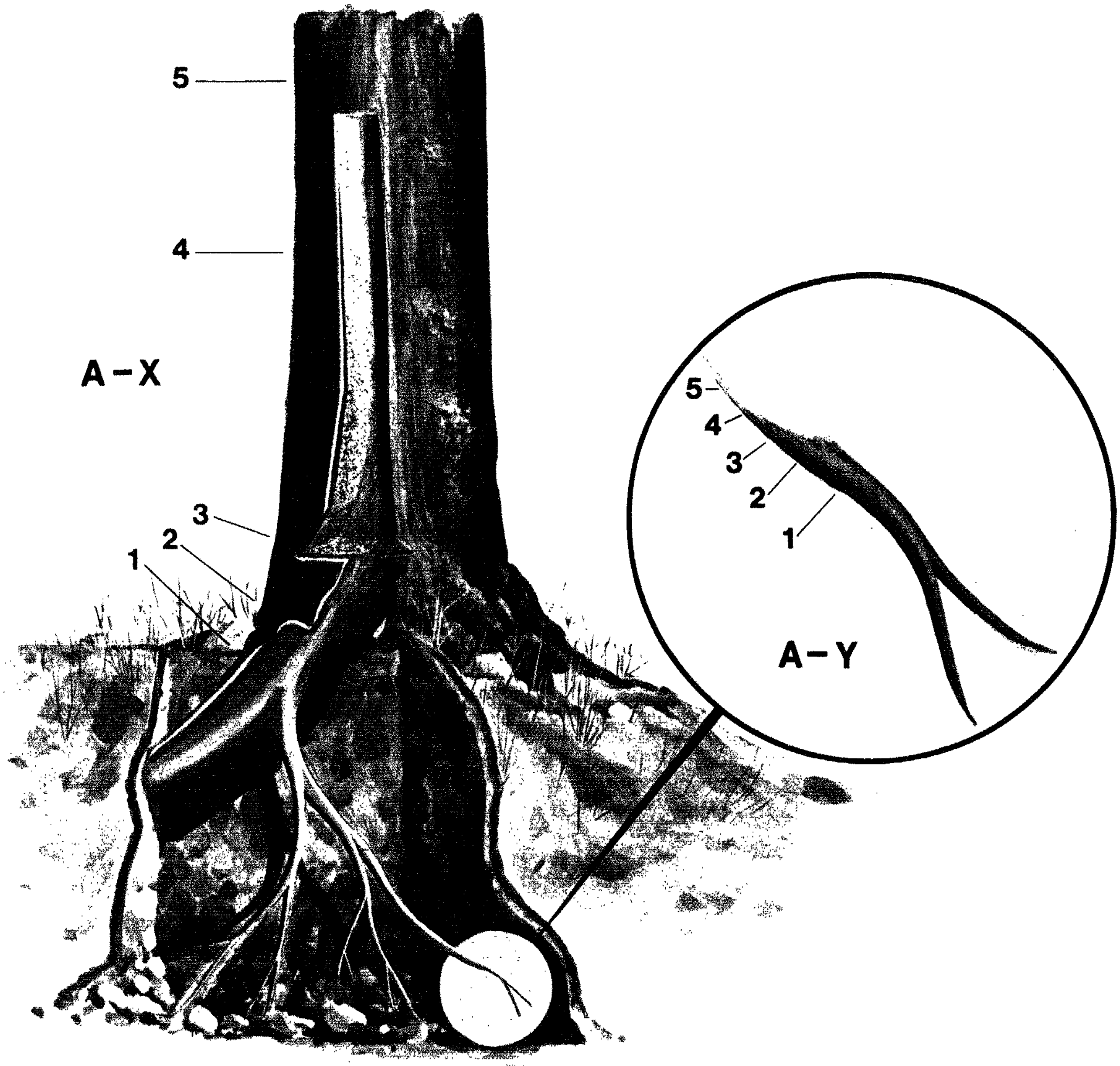

Figure 19.—The area of dead bark depicted as cross sections in row A. Dead area may be many meters long, A-X, or only a few centimeters long, A-Y. But, regardless of size there will always be an A-1 and an A-5. When A-1 is present on many roots, or at the butt, the tree dies. Many dying bark areas may coalesce to form one large dead area.

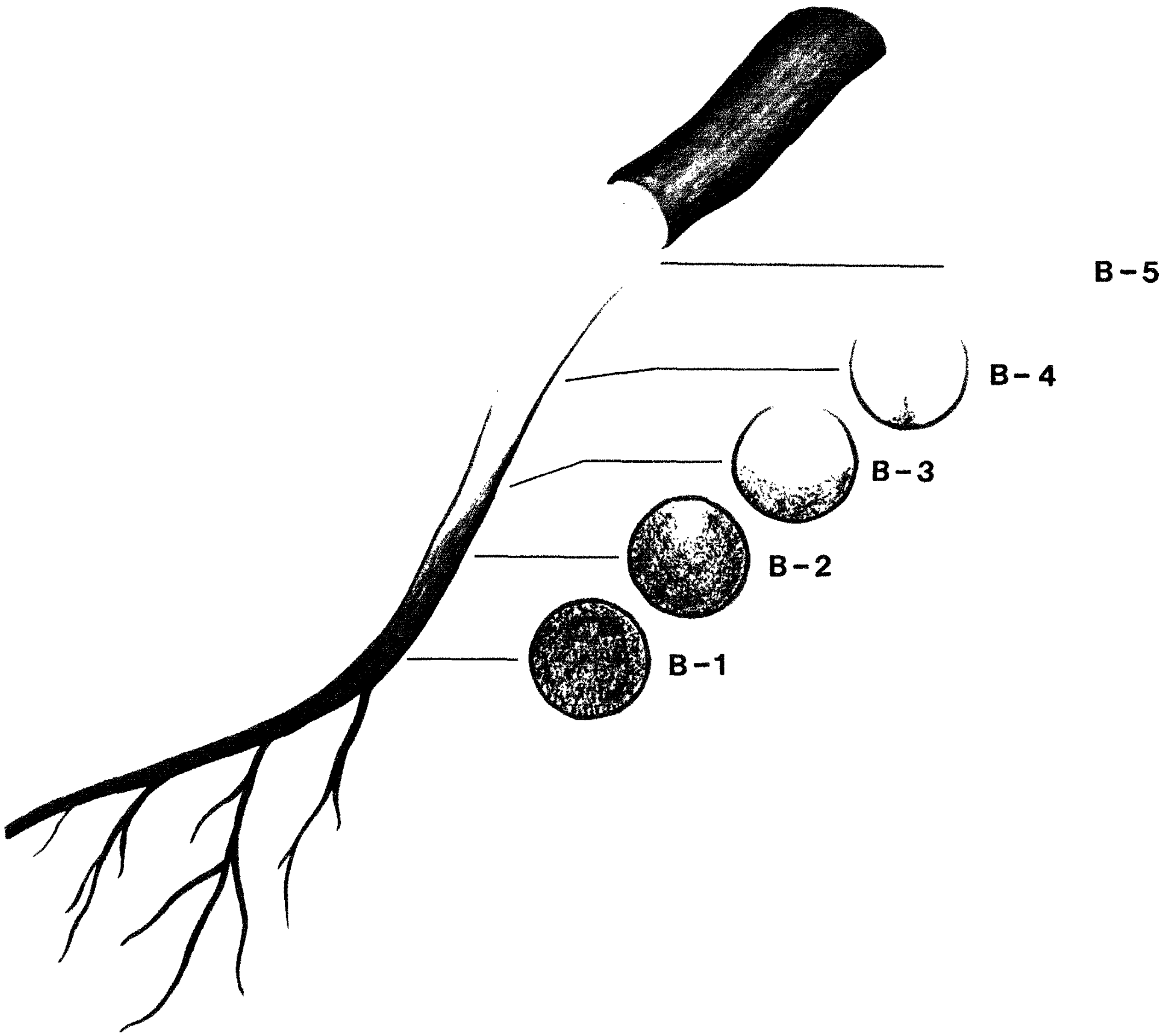

Figure 20.—Compartmentalization in wood present at the time of infection starts after bark killing stops. It is the living cambium beyond the limits of the dead bark area that produces xylem cells that develop into the barrier zone.

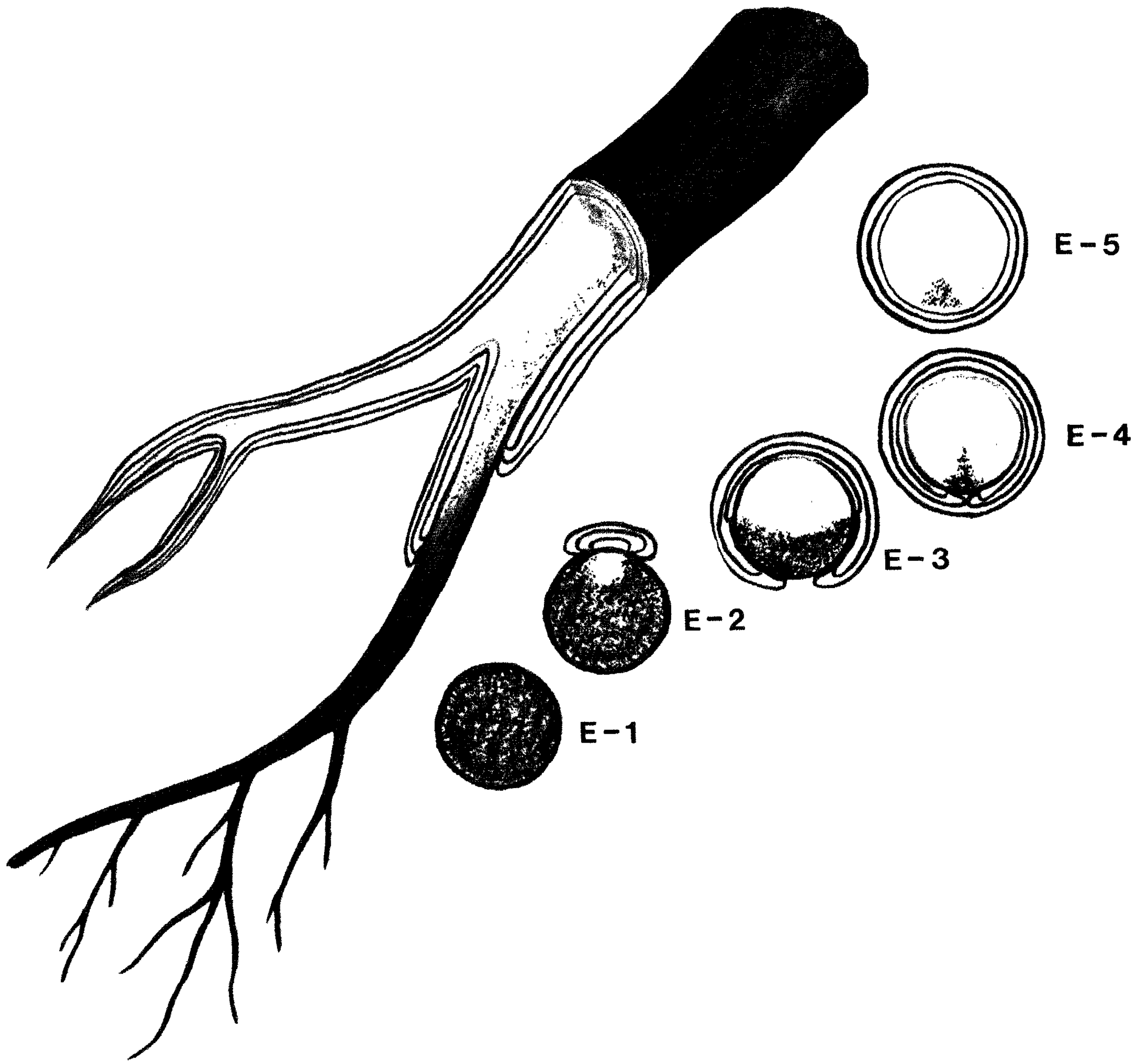

Figure 21.—*Armillaria mellea* may continue to spread within the wood present at the time the bark killing stopped. The fungus does not spread radially outward into xylem that forms after the bark killing stops.

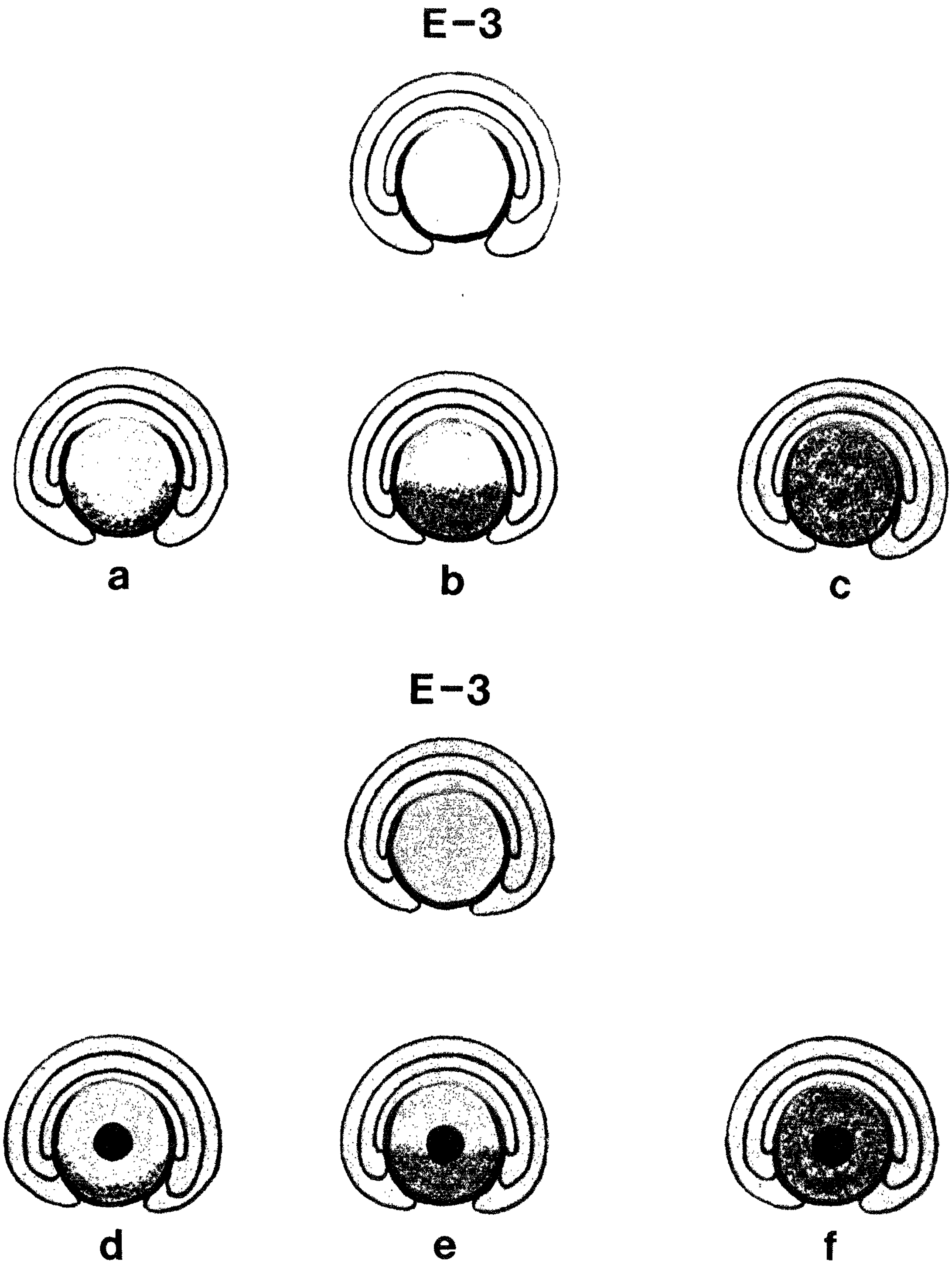

Figure 22.—How rapidly *A. mellea* spreads within xylem present at the time bark killing stopped depends on many factors. Some trees in a species and some different tree species can limit the spread of the fungus to small volumes, E-3-a; others have a moderate response, E-3-b; and others have a weak response, E-3-c. Some *A. rubrum* and *Quercus* spp. trees had patterns similar to E-3-a and E-3-b, and *A. balsamea* trees had patterns similar to E-3-c. The patterns, E-3-a, b, and c, occur in wood that has no heartwood or injured wood. When heartwood or injured wood is present, the patterns are similar to those shown in E-3-d, e, and f.

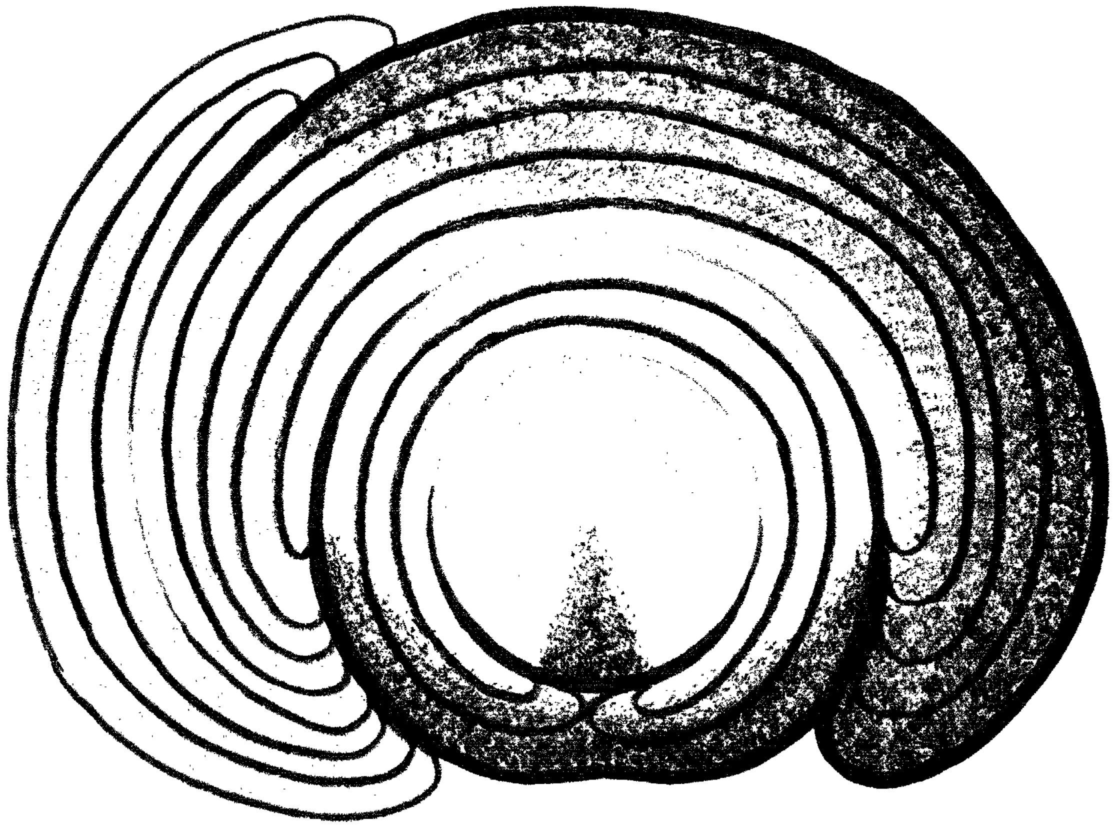

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Figure 23.—Multiple infections over time result in an endless array of patterns. As long as some cambium remains alive, the root or butt responds to wall off the spread of the fungus. As the living circumference of a root or butt decreases, additional infection leads to death. The ability to compartmentalize xylem to small volumes decreased as total volume of living wood decreased.

Discussion

Trees have many roots, but only one butt; when the butt is girdled, the tree dies. *Pinus resinosa* Sol. infected with *Heterobasidion annosum* (Fr.) Bref. is such an example (Shigo 1975; 1979b). Compartmentalization of decayed wood associated with *H. annosum* was similar to that reported here for *A. mellea.*

The long, narrow, triangular shape of the dead basal areas suggests that the walling-off force is more from the sides than downward from the top. Many microorganisms other than *A. mellea* were isolated from wood in the upper portions of the dead basal areas on *F. grandifolia.* After the tree sets the limits for the dead areas in the bark, cambium, and outer xylem. *A. mellea* may continue to spread deeper into the wood beneath and beyond the dead area.

After the dead bark is walled off, compartmentalization in the wood begins. The living cambium beyond the margin of the dead bark area forms cells that develop into a barrier zone (Tippett and Shigo 1981).

Armillaria mellea and many other wood-inhabiting microorganisms have an opportunity to spread into the wood beneath the dead bark area. This wood has limited biochemical mechanisms for defense. It is dying wood; no longer covered by living bark and a cambium. Such dying wood is quickly invaded by *A. mellea.* The wood beyond the limits of the dead bark area contains an abundance of living cells that can respond chemically to stop the spread of the fungus. The microorganisms may spread within the entire cylinder of wood present at the time of infection, or they may make only slight penetration into the wood. Once the parenchyma cells in the wood die, further deterioration of the wood depends on the types of microorganisms in the succession. The wood may discolor slightly or decay.

Two ways that the area of walled-off dead bark may increase in size are (1) new infections on roots can cause new areas of dead bark, and (2) the fungus in the dead bark at the margins of the dead area may break out of its wedge-shaped confinement and spread again into the new layer of living bark. This reinfection from wedges of fungus material at the margin of dead bark areas is the way that canker rot fungi, such as *Poria obliqua* and *Polyporus glomeratus,* continue to spread (Shigo 1969).

There is a walling-off process in bark which is similar in some ways to the walling-off process in wood as described in CODIT. The walling off is not similar in anatomy and biochemistry because of the many differences between bark and wood.

The information given here also appears to be similar for species of *Eucalyptus* (Kile 1980, Fig. 2).

Our studies of *A. mellea* and many other tree diseases indicate a basic design for survival of host and parasite. To survive, a parasite must infect when the defense systems of the host are at the lowest point and spread as far as possible before recognition and walling-off by the host. The parasite must then reproduce. For the host to survive, it must recognize the parasite as soon as possible, and wall it off to a small volume of bark and wood as rapidly as possible. The host must then generate new tissues, bark, and wood.

Acknowledgments

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Decayed wood associated with *Armillaria mellea* was compartmentalized according to the CODIT (Compartmentalization Of Decay In Trees) model. Compartmentalization in the sapwood began after the tree walled off the area of dead cambium associated with the infection of the fungus. The fungus spread into dying sapwood beneath and beyond the area of killed cambium, but the fungus did not spread radially outward into new wood that formed later.

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Keywords: Root and butt rot

Headquarters of the Northeastern Forest Experiment Station are in Broomall, Pa. Field laboratories and research units are maintained at:

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- **Beltsville, Maryland.**
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