WOUND HEALING IN BARK OF WOODY PLANTS [1]

by George W. Hudler

Abstract. Bark is a complex sheath that has two primary functions, it conducts photosynthate from leaves to other parts of plants, and it provides protection from insects, diseases, and mechanical abrasions. When live bark is injured, it responds by generating a new phellogen near the site of injury. However, environmental factors that reduce plant vigor may retard the restoration process and, thus, increase susceptibility to pathogenic microbes.

What is bark? Native Americans collected it from birch trees and made canoes with it. In Portugal, it is stripped from oaks, cut into small cylinders, and used to seal bottles of wine. And bark that once was waste from sawmills now commands a high price as mulch. Twigs, branches, stems and roots of all woody plants are covered with it, but most people know little of its structure and function.

From a botanical perspective, bark is a complex sheath that protects trees from insults of nature and transports carbohydrates and growth regulators within a plant. Without it, a woody perennial plant simply would not suvive. The objective of this paper is to present an overview of the nature of bark with emphasis on how it protects trees and responds to wounding.

Plant anatomists define bark as all cells and tissues beyond (to the outside of) the vascular cambium, and in a first-year-shoot these include the epidermis, cortex, and primary phloem (Fig. 1). Epidermis is usually just a single layer of cells coated with wax to impede outward diffusion of water and gaseous materials, but in some cases it is modified to contain stomata, hairs, and glandular appendages. Cells of the cortex, the largest and most numerous cells in primary bark, usually contain chlorophyll and may supply some photosyn-

thate to the plant, but their main function apparently is to give mechanical support. Phloem is, of course, the tissue responsible for carbohydrate transport.

As bark ages, it undergoes a number of morphological and physiological changes, and in most plants mature bark bears little resemblance to that of the young shoot. There is little or no evidence of original tissues as the epidermis dies and is sloughed, and the cortex and primary phloem are simply crushed beyond recognition. They are replaced by a layered array of groups of cells including (from the outside in) dead bark, phellem, phellogen, phelloderm, and secondary phloem (Fig. 2). The phellogen is a single layer of meristematic cells that originates through modification of some cortical cells late in the first season of growth. It produces all other bark constituents except phloem, and the nature of its development has much to do with the ultimate structure and function of bark.

Perhaps the most important product of phellogen is phellem, a tissue composed of short-lived cells whose walls are impregnated with suberin as the cells mature and die. Dead, suberized phellem (often called "cork") is impervious to water and many other substances, and it protects live bark from leakage and desiccation. As it accumulates on the outsides of most stems, cork becomes the major component of dead bark.

In addition to preventing drying, dead bark protects trees in several other ways. First, its structure includes many tiny air pockets that serve to insulate the tree from rapid fluctuations in temperature. Before dead bark is sufficiently thick

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to be a good insulator, a young tree is more likely to suffer winter splitting or sunscald, and wrapping with a reflective tape supposedly helps to prevent such injuries by maintaining lower bark temperatures on cold, sunny days.

Second, dead bark provides physical protection from minor abrasions and some insects. For instance, one seldom finds scale insects or aphids feeding on thick-barked portions of trees because most insects simply do not have mouthparts long enough to penetrate dead bark and reach living cells below.

Third, the chemical constituents of dead bark (suberin, lignin, tannins, etc.) apparently are not metabolized easily by microorganisms. In fact, they may be toxic to some. Thus, most diseases of bark are limited to sites on trees where pathogens gain access to live cells via wounds or infection of foliage and subsequent growth to live bark through petioles.

Phellogen also produces phelloderm. In comparison to phellem, production of phelloderm is markedly less, and the cells are often difficult to discern microscopically. The function of phelloderm is unclear, and it may be little more than a superfluous product of evolution.
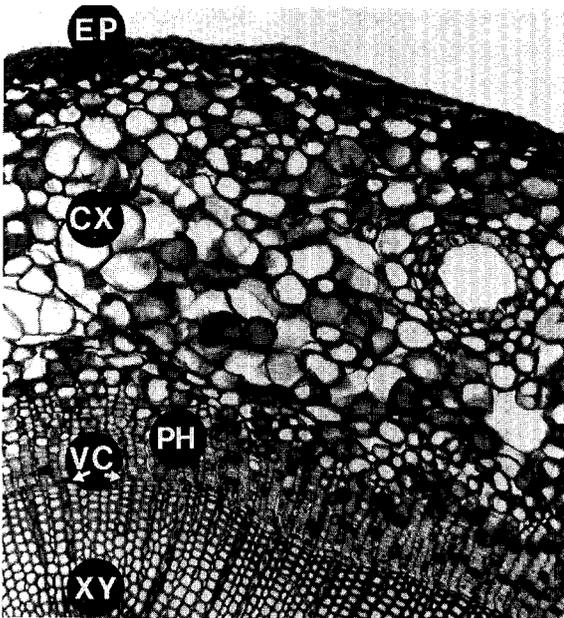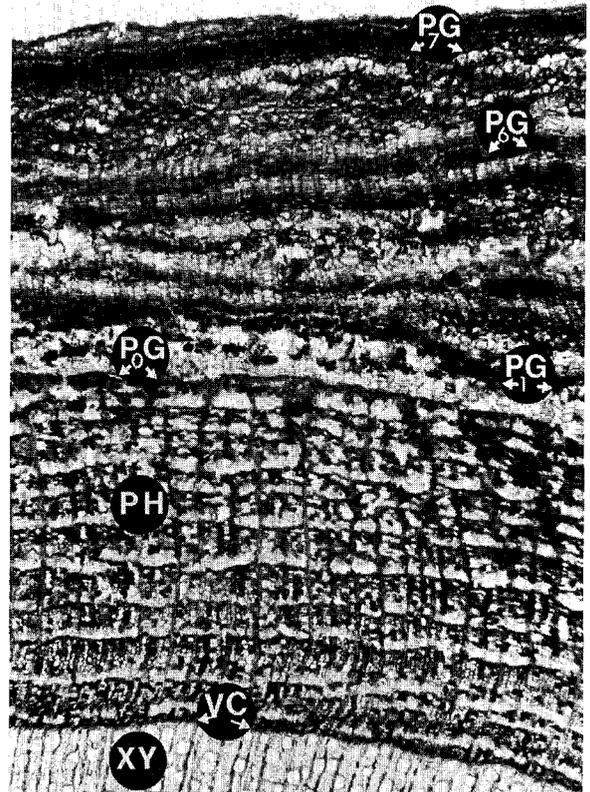

Fig. 2. Transverse (cross) section of a 9-year-old black willow branch showing xylem (XY), phloem (PH), approximate location of current phellogen (PG 0), bands of previously active periderm (PG 1 - PG 7). Cells from PG 0 to the outside of the branch (top of picture) are dead.

Trees with smooth bark (eg. American beech, blue beech, striped maple) have a continuous phellogen that persists, barring injury, for the life of the tree. Cork is produced at a very slow rate and is sloughed almost imperceptibly as single cells.

In species with rough bark, however, the phellogen does not persist indefinitely as a continuous tissue; segments of it periodically cease to function and eventually die. This is a part of normal development that could, with increased radial growth, lead to cracks in the dead bark and exposure of live cells within. Repair of these natural gaps is accomplished through a remarkable sequence of events known as "phellogen restoration" (Fig. 3).

The first visible step in phellogen restoration is that phelloderm and/or phloem cells near a senes-

Fig. 1. Transverse (cross) section through a new Douglas-fir twig showing xylem (XY), phloem (PH), cortex (CX), and epidermis (EP).

cent phellogen segment swell, and their walls become darker and thicker than surrounding cells. Physiological changes result in deposition of fungitoxic chemicals in these cells, and at least one layer of cells on the inner margin becomes impervious to dyes and, presumably, other liquids. Chemical tests have shown that this first impervious layer does not contain suberin. Thus, it is different from normal cork and is referred to as non-suberized, impervious tissue (NIT)(4).

Following NIT formation, phelloderm and/or phloem parenchyma subjacent to the NIT are transformed to phellogen through a process of "dedifferentiation." They form a sheet beneath the senescent region and beyond its edges and join with previously existing phellogen to restore continuity. The new phellogen then functions as the original did to produce phellem (eventually cork) and to protect the tree. The new phellogen and cells it produces are referred to collectively as necrophyllactic (*Necrus* = dead; *phyllaca* = guard) periderm (NP)(6).

If segments of dead bark delimited by NP remain attached to other dead bark through intertwined fibers, the bark becomes furrowed as in ash, boxelder, and walnut. If the segments are not interconnected by fibers, dead bark is shed in scales or plates. Sycamore, hickory, and most conifers are typical examples.

The process of phellogen restoration is not restricted to normal senescence of phellogen segments. Instead, *it is a non-specific response* *that begins whenever phellogen dies for any reason,* including mechanical injury, extreme temperatures, insect attack, or colonization by microbes (5)(Fig. 4-[4],5). In some cases, initial injury may include death of more than just the phellogen. Sapsuckers, for instance, usually make a hole that extends to the xylem. Likewise, scale insects or aphids pass their stylets right through the phellogen and probe deep into the phloem in search of food.

The reaction of bark to deeper wounds is similar to that already described near the phellogen, but additional changes may also take place. If there is not enough room for phellogen restoration to occur between the innermost point of injury and the vascular cambium, the vascular cambium may also become inactive, and adjacent xylem vessels may plug with gums, tyloses, and other materials. In this way, xylem dysfunction can occur even though xylem is not directly wounded or invaded by a pathogen (Fig. 4-[5]).

Fig. 4. [4] When bark is wounded, the repair response is similar to that occurring during normal growth (see Fig. 3). However, if a wound is close to the vascular cambium, it and subjacent xylem may be killed or plugged.
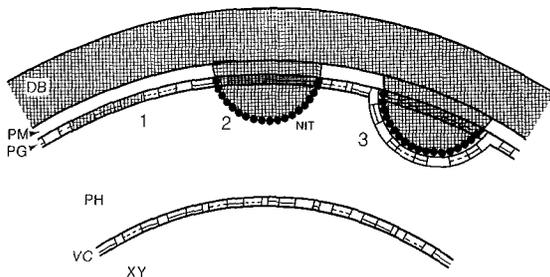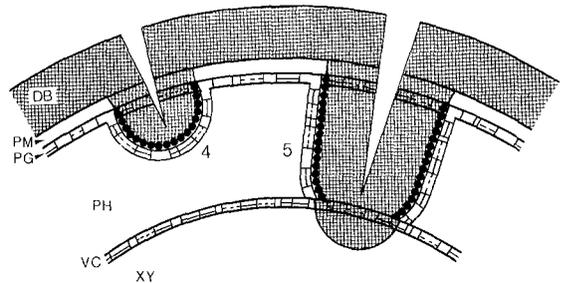

Fig. 3. Schematic diagram of a transverse (cross) section of a tree with rough bark. [1] Segment of phellogen becomes inactive; [2] cells in the phloem near the inactive segment accumulate toxic metabolites and a band of non-suberized impervious tissue forms around the boundary of the reaction zone; [3] new phellogen arises from phloem parenchyma to bridge the defective segment and maintain continuity of the original phellogen. DB = dead bark, PM = phellem, PH = phloem, VC = vascular cambium, XY = xylem, PG = phellogen.
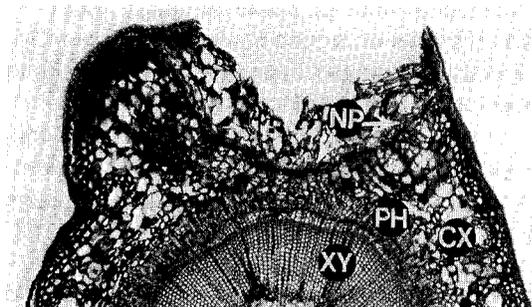

Fig. 5. Necrophylactic periderm (NP) formed around a wound in a young Douglas-fir twig. XY = xylem, PH = phloem, CX = cortex.

Phellogen restoration:
Its role in resistance to pathogens

Theoretically, the phellogen restoration process should help in several ways to minimize injury sustained by trees following wounding. Fungitoxic chemicals accumulate near the wound, flow of nutrients to potential invaders is cut off by NIT, and new phellogen produces protective cork. Obviously, however, these mechanisms are not always successful because some trees or parts thereof are still killed by bark pathogens. Now, the challenge is to understand why phellogen restoration fails in those cases and to determine what, if anything, can be done to prevent such failures.

In this regard, there is good evidence to indicate that trees suffering from water stress are slower to begin phellogen restoration following wounding than are non-stressed trees (9). Trees suffering from water stress are also more susceptible to canker diseases, especially those caused by facultative parasites (1,2,3). It is entirely possible that one major consequence of water stress in trees is to slow down phellogen restoration and allow pathogens to continue to advance unimpeded into healthy tissue.

Inasmuch as lethal cankers caused by native pathogens on native trees are almost always associated with lowered host vigor, one might speculate with some confidence that interactions between other environmental stresses, phellogen restoration, and disease also occur. If so, then it seems to me that we ought to know as much as we can about such things so as to be able to make the best possible judgments about shade tree care and management. For this reason, activities in my research program are directed toward determining effects of some of these other inducers of stress on wound healing in tree bark. Infertile soil and chronic exposure to air pollutants are the topics of highest priority at present.

Knowledge about phellogen restoration not only allows us to speculate about effects of environment on disease susceptibility, but it also provides some insight into the reasons why we see different kinds of cankers on trees.

Some canker diseases, especially those caused by endemic pathogens on vigorous hosts, are characterized by host production of successive layers of callus (Fig. 6). Since phellogen restora-

tion occurs most rapidly in late spring and early summer and is negligible when the host is dormant (7), we assume that the process is sufficient to contain these pathogens during the growing season, but they advance while the tree is dormant. Successive years of alternate rest and growth by the host and pathogen, respectively, result in the target-like shapes of cankers caused by *Nectria galligena, Strumella coryneoides, Eutypella parasitica,* and others.

Pathogens causing cankers that lack prominent callus ("diffuse" cankers) apparently have some means to circumvent or suppress host defense mechanisms throughout the year. The chestnut blight pathogen *(Endothia parasitica)* and the white pine blister rust pathogen *(Cronartium ribicola),* (Fig. 7) are good examples.

Phellogen restoration and resistance to insects

Major contributions to our current knowledge of phellogen restoration in bark were made by D.B. Mullick and his colleagues while studying resistance of conifers to the balsam wooly aphid. Apparently, resistant trees seal off aphid feeding

Fig. 6. Nectria canker, caused by *Nectria galigena,* is characterized by concentric ridges of host callus. The pathogen apparently advances during the dormant season and is walled off when the tree is actively growing.
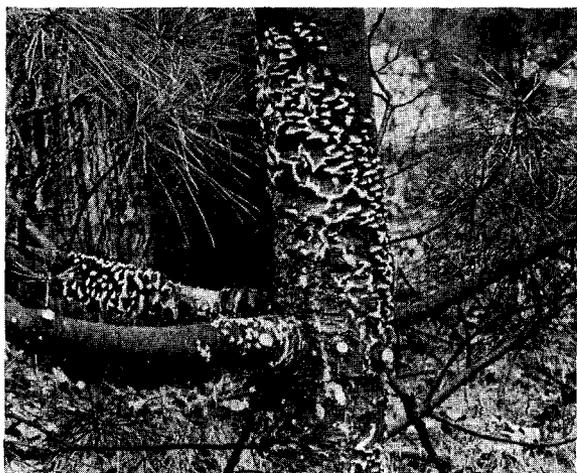
Fig. 7. *Cronartium ribicola,* cause of white pine blister rust, negates host defense mechanisms and quickly kills a branch or stem.

sites via production of an impermeable layer of host tissue, but the process is impeded in susceptible trees by one or more components of aphid saliva (5). Resistance of other trees to other bark feeding insects is presumed to be similar, and this character may prove to be a valuable tool in screening trees for insect resistance.

How does knowledge about phellogen restoration help the professional arborist?

To be sure, there is much more to be learned about phellogen restoration and its role in insect and disease resistance. However, in view of what is known, one might wish to consider the following:

• The evidence to date is that trees in good health and optimum vigor are less likely to suffer from insect and disease problems. This is not new information, but with added knowledge about effects of stress on host defense mechanisms, perhaps the professional arborist will be better able to present the case for good care to his clients.

• Every time a branch is pruned, bark is wounded and phellogen restoration must occur to protect remaining bark from disease. It is logical to suspect, therefore, that the best time to prune is when phellogen restoration will occur most quickly. For most trees, this is just after they break bud in the spring, but pruning at that time is contrary to current practices in many locales where it is customary to prune any time during the dormant season. Perhaps the whole question of the best time to prune should be re-examined.

• Shigo and Shortle (10), Neely (8), and others have demonstrated that wound dressings do little or nothing to prevent discoloration and decay of wood, but there is no specific information about effects of wound dressings on phellogen restoration. If one had a non-toxic dressing to apply to wounds to serve strictly as a physical barrier to pathogen ingress until NIT formed, it could be a valuable tool for the tree care industry.

Undoubtedly, many other practical applications of knowledge about phellogen restoration will be forthcoming, and arborists should expect to hear more about them as research on this topic continues.

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