# BUILDING WALLS AND BREACHING WALLS: TRUTH AND CONSEQUENCES IN WOUND COMPARTMENTALIZATION.

**by Frank S. Santamour, Jr.**

**Abstract.** Many trees have the genetic potential to wall off wound-induced wood discoloration from moving into their interiors. These trees actually build walls, mainly of phenolic compounds, that retard the spread of microorganisms. To build these walls, they use carbohydrate reserves from their sapwood parenchyma cells interior to the wound. New wounds that breach old walls of strong-compartmentalizing trees, or wounds that intrude into discolored areas of weak-compartmentalizing trees expose areas depleted of carbohydrate content. Here strong new walls cannot be built. The amount of decay resulting from breaching old walls is greater in weak-compartmentalizing trees. Wood-discoloring microorganisms can penetrate Wall 4 when weak-compartmentalizing trees are re-wounded outside this wall. Thus, while any accidental or intentional wounding of tree trunks may result in potential decay problems, any intentional *re*-wounding, especially when these wounds penetrate through established walls, definitely will lead to decay. Such practices should be discouraged.

**Résumé.** Plusieurs arbres ont le pontentiel génétique de compartimenter, et ainsi de limiter la progression de la décoloration du bois suite à une blessure. En fait, ces arbres érigent des murs constitués principalement de composés phénoliques qui retardent la progression des microorganismes. Pour ériger ces murs, ils utilisent les réserves en hydrates de carbone des cellules du parenchyme de l'aubier à l'intérieur de la blessure. Les nouvelles blessures qui ouvrent un brèche dans les vieux murs des arbres forts compartimenteurs, ou les blessures qui sont localisées dans les zones de décoloration des arbres faibles compartimenteurs, exposent des zones très pauvres en hydrate de carbone. A ces endroits, de nouveaux murs résistants ne peuvent être érigés. La quantité de carie résultant des brèches dans les vieux murs est plus grande dans les arbres faibles compartimenteurs. Les microogranismes qui décolorent le bois peuvent pénétrer le Mur 4 quand les arbres faibles compartimenteurs sont blessés à nouveau à l'extérieur du mur. Ainsi, bien qu'une blessure accidentelle ou délibérée sur le tronc d'un arbre peut résulter en des problèmes potentiels de carie, toute blessure délibérée répétée au même endroit, spécialement quand ces blessures pénètrent à travers des murs établis, conduiront d'finitivement à la carie. De telles pratiques devraient être évitées.

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The original exposition of the concept of Compartmentalization of Decay in Trees (CODIT,10), implies that of the four compartment "Walls", Walls 1,2, and 3 are already *present* in the tree at the time of wounding. Only Wall 4, the "barrier zone" formed by cambial activity after wounding, was considered a *response* to wounding. On the other hand, terms like "reacts", "exerts", and "responds" used in explaining the CODIT model implied that the tree is *doing* something. The simplification of complex phenomena, especially in the early stages of testing an hypothesis, can cause problems. Yet such hypotheses, are the starting point for the successive approximations to truth that result from scientific research.

## Building Walls

Let us consider the individual cell as the smallest "compartment". Groups of cells along the tangential plane (terminal parenchyma) and the radial plane (ray parenchyma) are convenient illustrations of even larger compartments, and in the CODIT model they are Walls 2 (annual ring) and Walls 3 (rays). There are no distinct cell groups in the vertical plane, so Walls 1 (tops and bottoms of cells) are harder to visualize. Shigo (9) has introduced the term "compartmentation" to describe the normal steady state in wood. This term can be interpreted as the result of "boundaries" that exist because of the anatomical features described above. My interpretation, which differs semantically from Shigo's, is that boundaries already *exist;* walls need to be *built.* Regardless of the words used, "Compartmentalization" is real and its recognition helps us to understand many problems.

Shortle (12) presented and his concept of the walls, in relation to the spread of microorganisms: *Wall 1 is a plugging component that limits the vertical spread. Wall 2 is an anatomical component that limits the spread parallel to the rays. Wall 3 is a vital component that limits spread perpendicular to rays. Wall 4 is a differentiation component that limits spread into wood formed after wounding. Walls 1,2, and 3 act in wood extant at the time of wounding; Wall 4 forms only after wounding.* In fact, it is almost certain that cell plugging (with insoluble materials) is a factor in Walls 1,2, and 3. Shortle (11) has himself shown a significant (2 - to 4 -fold) increase in phenolic compounds in Wall 3

as compared to normal wood. Of all the Walls, I consider Wall 2 as the most amenable to experimentation and differentiate between "strong" and "weak" compartmentalizing trees on the basis of the tree's ability to resist the inward spread of wood discoloration from the wound site to the pith (Fig. 1). Illustrations provided in an earlier paper (7) showed that Wall 2 is *not* coincidental with the terminal parenchyma laid down at the border of an annual ring. Rather, Wall 2 is composed of an irregular, but connected, patchwork of cells plugged (in many genera) with water-insoluble phenolic compounds (Fig. 2).

A quantitative example of the "cell plugging" aspect of compartment Walls is taken from the work of Rowe *et al.* (4), who identified the major insoluble phenolic compounds in the walls of wounded maples as two insoluble coumarinolignan compounds. Only two of my wounded red maples *(Acer rubrum)* were analyzed and data are given below for clear sapwood and Walls produced after chisel-wounding in the trunks of weak-and strong-compartmentalizing trees. The numbers represent mg of coumarinolignans per gram of oven-dry wood. The positions of the Walls are indicated by the numbers "2" and "3" on the trunk cross-sections in Figure 1.

| | *Clear sapwood* | *Wall 2* | *Wall 3* |
|---|---|---|---|
| Weak | 0.58 | 3.8 | 4.1 |
| Strong | 0.56 | 13.1 | - |

The unwounded (normal) wood of the two trees did not differ in coumarinolignan content.

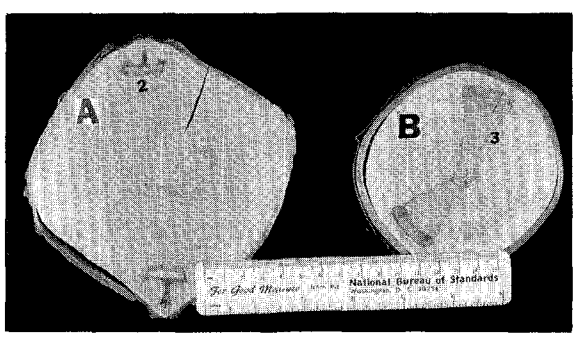


**Fig. 1. Cross-sections through strong-(A) and weak-compartmentalizing (B) red maples; numerals indicate strong Wall 2 and strong Wall 3.**

However, the strong compartmentalizing tree "built" a real Wall 2 that effectively blocked the inward spread of discoloration and contained 22.4 times as much coumarinolignan as normal sapwood. The weak-compartmentalizing tree also exhibited increased levels of coumarinolignan in the area that should have been Wall 2, but the Wall was not dense enough (or it formed too slowly) to prevent the inward spread of discoloration. However, the same relative coumarinolignan content in the radial boundary cells (Wall 3) did constitute a strong wall, perhaps because of synergism with the living ray cells.

Thus the "plugging component" may be a major part of Walls 1,2, and 3. Also, anatomical and vital components may enhance the effectiveness of these Walls. It is also possible that an antimicrobial component could be operative—more in some species than in others.

Thus, effective Walls are build **after** wounding. The wall materials, insoluble or soluble, are likely to be the products of an increased and abnormal (wound-induced) metabolism of living cells rather than products of dying cells injured in the wounding process.

### Wall 2 Building Blocks

Let us presume that the Wall 2 constituents, both soluble and insoluble, are phenolic compounds formed via the shikimic acid pathway. Then there must be a source of carbohydrates sufficient to account for the tremendous increase in wall chemicals (coumarinolignans, etc.) beyond the level in normal tissue. Carbohydrates in sapwood are generally stored in parenchyma cells, especially ray cells, in the form of starch (insolu-

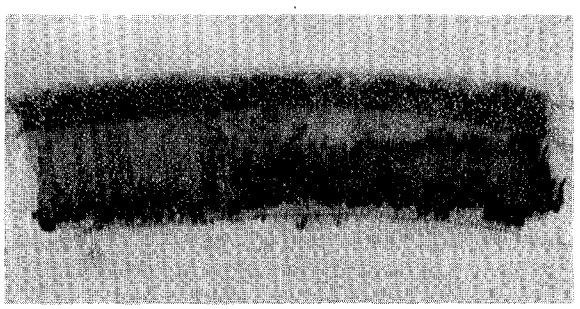

**Fig. 2. Cross-section of strongly compartmentalized chisel wound in red maple, 3 cm above wound site. Plugged cells interior to the wound (lower uneven border) constitute a strong Wall 2.**

ble). Hydrolysis of starch into sugars (soluble) allows carbohydrates to move from cell to cell.

The cross-sections in Figs. 3 and 4 were stained with a potassium iodide-iodine solution that turns starch dark. The depletion of starch (indicated by light-colored zones) interior to Wall 2 in strong-compartmentalizing trees suggests that the wall-building process drew carbohydrates from this area. Weak-compartmentalizing trees may have too little carbohydrate reserve in sapwood parenchyma cells to build a strong Wall 2. Restricted zones of starch depletion that appear along the sides of the discolored "wedge" may indicate Wall 3 construction (Fig. 4).

Do weak-compartmentalizing trees differ from strong-compartmentalizing trees merely because they do not store starch in sapwood parenchyma? Do genetic differences between weak and strong trees affect production of the enzymes needed to make phenolics out of sugars? What factors speed up or slow down phenolic production? These are questions for tomorrow.

### Constancy of Compartmentalization

Fig. 5C shows that strong-compartmentalizing trees can build another strong Wall 2 even when new wounds are made close to old wounds. Figs. 5A and 5B show that weak-compartmentalizing trees usually remain unable to build a strong Wall 2. We never observed a weak compartmentalization response to repeat wounds (made outside previous wounds) in strong-compartmentalizing trees, but occasionally one or more of the repeat wounds on weak-compartmentalizing trees did appear to produce a strong Wall 2. This may result from a local increase in the carbohydrate content of the tissues.

### Breaching Walls

The best way to examine the results of wounding and to know how well a tree can compartmentalize is to wound it and then later to cut it down and dissect it in various ways. Such drastic treatment is rarely possible, and it is not necessary. In an earlier study (6), we could find strong Walls 2 with an increment borer. The area of discolored wood in strong-compartmentalizing trees is virtually limited to the cells killed at the time of wounding whereas in weak compartmentalizers the discolored zone is extensive. During 1983 and 1984, we took increment cores from borings made through wound zones in more than

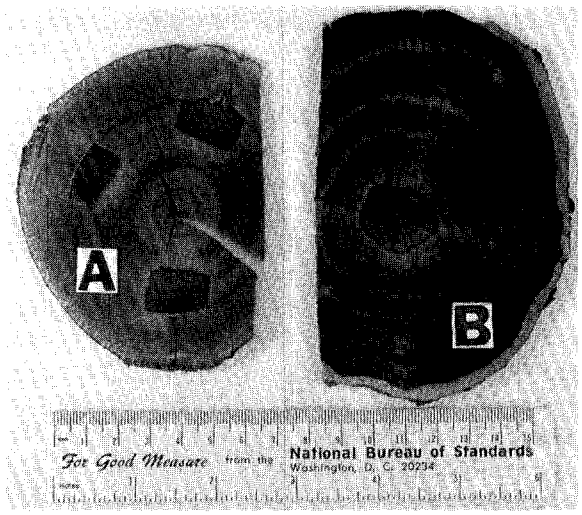


**Fig. 3. Cross-sections of strong-compartmentalizing trees of *Tilia americana* (A) and *Quercus Palustris* (B), stained with iodine to illustrate starch depletion (reduced staining) interior to Wall 2.**

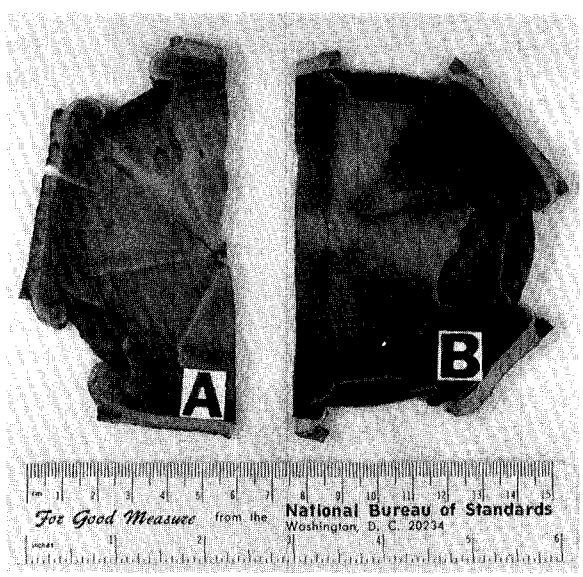


**Fig. 4. Cross-sections through re-wounded red maples, stained with iodine. Reduced staining interior to Wall 2 in strong-compartmentalizing tree (B), but not in weak-compartmentalizing tree (A). Some starch depletion (reduced staining) along Walls 3 in (A). Note that wound-induced wood discoloration from second wounding of weak-compartmentalizing tree (A-lower left) has moved *through* Wall 4 (see also Fig. 5).**

150 red maples that had been chisel-wounded in 1974. This was done to select trees of known compartmentalization potential to study new chemical wound "dressings". When those trees were felled for analyses in 1984 and 1985, it was obvious that the additional wounding caused by the increment boring had dramatically increased wood discoloration and decay (especially decay) beyond that caused by the chisel wounds or increment borer wounds alone. The amount of decay in weak-compartmentalizing trees (Fig. 6) appeared greater than that in strong-compartmentalizing trees (Fig. 7).

Two lessons may be learned from this work. One is a re-affirmation of the "succession of organisms" concept of decay (8). None of the trees wounded in 1974 and harvested in 1978 exhibited any decay (5), nor did trees wounded in 1974 and harvested, without increment boring, in 1984. Decay organisms began to operate when new wounds were made into the discolored wood (of weak compartmentalizers) or through Wall 2 and into the starch-depleted zone interior to that Wall in strong compartmentalizers.

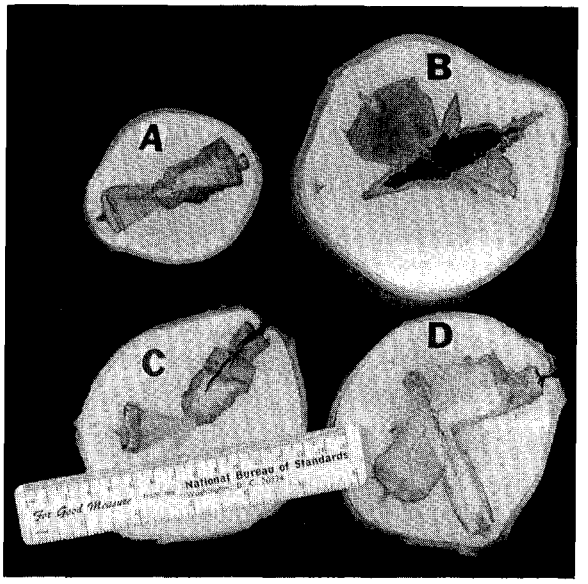


**Fig. 6. Wood discoloration and decay in weak-compartmentalizing trees of red maple following chisel wounds in June, 1974 and increment boring through wound in March, 1983 or 1984. Trees cut December, 1985. Note that width of discoloration in (B) and (D) is more extensive than original wound width. Entire center of section B fell out during sanding. Tree D was bored twice, once through a wound and once through clear wood.**

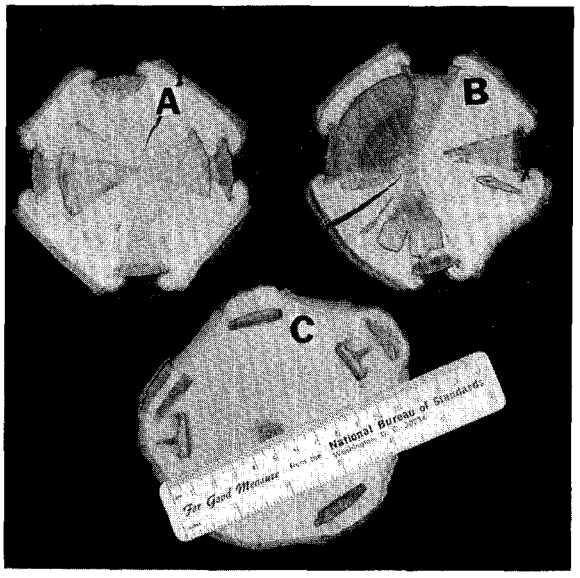


**Fig. 5. Wood discoloration in red maple following chisel wounding in 1974 (2 wounds) and 1981 (4 wounds). Tree C was a strong compartmentalizer and formed a strong Wall 2 following both woundings. Trees A and B were weak compartmentalizers and discoloration caused by 1981 wounding penetrated through Wall 4 of 1974 wound (A) and completely obliterated the discoloration of one 1974 wound (B).**

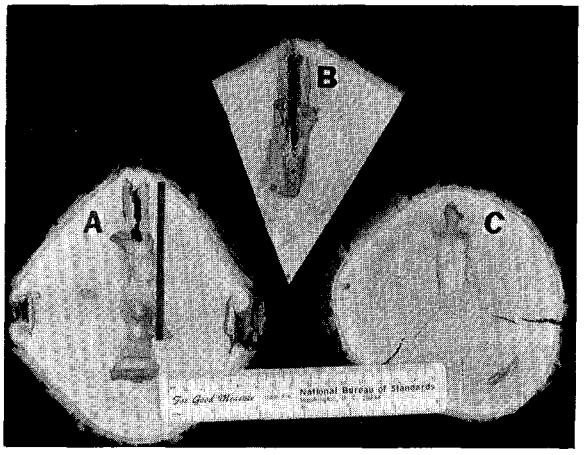


**Fig. 7. Wood discoloration and decay in strong-compartmentalizing trees of red maple following chisel wounds in June, 1974 and increment boring through wound in March 1983 or 1984. Trees cut December, 1985. Depth of increment core in (A) indicated by black band. Note that width of discoloration caused by increment boring is limited to width of original chisel wounds (1.8 cm). Depth of discoloration in (B) and (C) is limited to depth of increment boring. In (A), where increment boring extended through the pith, discoloration continued out to original (1974) strong Wall 2 on the other side, even though a new, but weak, Wall 2 was built interior to the original Wall 2.**

The second lesson is that purposeful breaching of any potential Wall or barrier invites disaster. Both older arboricultural literature (1) and more recent works (3) suggest that cavities in trees brought about by decay be thoroughly cleaned out and scraped down to sound wood before filling the cavity. There is always a band of sound, but discolored, wood interior to the decayed wood. Any incursion into this zone will result in more rapid and more extensive decay than if the cavities had been left alone. Scribing, shaping, or tracing around wounds in trees of unknown compartmentalization potential should also be discouraged. No matter how esthetically pleasing the resultant ellipse or other shape may be, this kind of treatment can only increase the potential for wood decay and is of no advantage in hastening callus growth to "cover up" the wound (2). It is evident that treatments that require drilling into or completely through tree trunks (cabling, bracing, injection) should not be applied to trees that are known to have internal decay.

**Strength of Wall 4**

In the CODIT model (10), Wall 4 is described as the "strongest wall". This is "barrier zone" (10) or the "differentiation component" (12) formed exterior to the wound by cambial activity. Wall 4 usually is strong enough to limit the spread of discoloration and decay to wood that was already present at the time of wounding. Anatomical studies of this zone have been reported in various tree species (12). Wall 4 is formed to both strong- and weak-compartmentalizing trees.

Examination of re-wounded weak-compartmentalizing trees (Figs 4A, 5B) suggests that although Wall 4 may be strong enough to resist the spread of discoloration "from the inside out", it may not be capable of resisting such spread "from the outside in". In strong-compartmentalizing trees, new wounds made outside old wounds are compartmentalized by a new Wall 2 before there is any spread of discoloration inward to the previous Wall 4 (Fig. 5C). This is just one more illustration of the wisdom of selecting and using only strong-compartmentalizing trees for landscape planting.

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