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Walter C. Shortle, CODIT, page 233-234

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wife and children to Germany, where he enrolled in the University of Jena and wrote a thesis on nematodes associated with whales; he received his Ph.D. in 10 months. His talent as an artist helped him procure a job at the Zoological Research Station in Naples, Italy, but a year later he moved to Sydney, Australia. Unable to find a suitable position, Cobb used his artistic skills to advertise oils and soaps, but within a year he secured a part-time job as consulting pathologist in the new Department of Agriculture in New South Wales. A few months later he had a permanent position as vegetable pathologist. In this, the first full-time plant pathologist position in Australia, he managed a Government Experimental Farm 300 miles from his office in Sydney and had broad responsibilities, including the study of nematodes, flukes, and tapeworms of farm animals as well as studies of various grains. He published a number of papers on plant diseases such as Australian rusts, stinking smut, and bacterial gumming of sugarcane.

About 1890 Cobb became interested in plant parasitic nematodes and published his first paper on "root gall" and discovered and identified the burrowing nematode, *Radopholus similis*, in diseased bananas. In 1905 Cobb moved to Hawaii to establish and direct the Division of Pathology and Physiology of the Hawaiian Sugar Planters Association Experiment Station in Honolulu. There he studied fungal diseases of sugar cane but also identified the first known parasite of nematodes. In 1907 he went to Washington, D.C., as agricultural technologist for the U.S. Department of Agriculture (USDA), working primarily on determining quality of cotton fibers. In 1910 he was called upon to inspect cherry trees sent to Washington, D.C., as a goodwill gift from the Japanese government. He found 72% of these trees to be infested with "root gall worm" (the root-knot nematode) and suspected that most were infested. This occurrence helped the passage of the Plant Quarantine Act of 1912, with Cobb serving as one of the authors.

Cobb published a paper on marine nematodes in 1914, in which he proposed that the study of nematodes be recognized as the separate science of nematology. Cobb's interest in the development and morphology of nematodes, which began with his Ph.D. thesis in 1888, continued throughout his career. In 1919 Cobb proposed a classification system that was published after his death as "The Key to the Genera of Free-living Nematodes." He died on June 4, 1932, at Baltimore, Maryland.

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CODIT

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CODIT is an acronym for compartmentalization of decay in trees (1). It is a simple model system originally designed to help forest managers understand the patterns of discoloration and decay in living trees. Decay is a process that begins with a wound and ends in the decomposition of wood exposed by the wound (2). Compartmentalization is a process that resists the spread of decay (3). When compartmentalization is fully successful, decay-causing infections do not spread into the wood formed after wounding.

Compartmentalization may be viewed either from the highly technical functional level of boundary-setting mechanisms or from the simple nontechnical view of the CODIT model system (4). The functional view considers the formation of reaction zones in wood present at time of wounding and barrier zones in wood formed immediately after wounding. Both zones are highly resistant to infection due to biochemical changes mediated by living parenchyma cells. Both zones become boundary layers to limit the spread of infection. The difference between the two types of defensive zones is the altered anatomy of the barrier zone, which makes the chemical protection more effective than in wood with normal anatomical structure.

The CODIT model does not focus on biochemical protective functions of reaction zones and barrier zones, but describes compartmentalization in terms of four structural "walls." According to the model, wall 1 resists vertical spread of infection in wood exposed by a wound; wall 2 resists inward (radial) spread; wall 3 resists lateral (tangential) spread; and wall 4 appears only after wounding and resists the outward spread into wood formed after wounding. Wall 4 in the model is considered the key wall because it allows trees to survive and function while wood exposed by a wound decomposes over time to produce a hollow tree. Wall 4 corresponds to the barrier zone formed in trees after wounding. However, walls 1, 2, and 3 do not correspond to reaction zones, but rather to a layer of "plugs" that appear in water-conducting elements (wall 1), to a layer of marginal parenchyma that exits between growth rings (wall 2), and to wood rays running from bark to pith (wall 3).

Wall 1 is the least effective wall in the model. It does not exist before the tree is wounded because the tree must maintain vertical transport of water and nutrients. After injury and infection, plugs form in the vertical elements of sapwood. Plugging of the vertical transport system decreases the rate of spread of decay-causing pathogens, but the layer of plugs (wall 1) is not continuous and pathogens get through. Observation of reaction zones indicate that tyloses and various polymeric substances do indeed block major avenues for the spread of pathogens. The tyloses and polymeric substances, which plug conducting elements, originate in live cells.

Wall 2 is more effective than wall 1 in the model. Wall 2 is the last wood to form in each growth ring at the end of the growing season. This wood is more dense than wood formed earlier in the season and often has several layers of living cells that store food like the ray cells of wall 3. Live cells of wall 2 take part in reaction zone formation as do the ray cells of wall 3.

Wall 3 is the most effective wall in the model, except for wall 4, which forms after wounding. Wall 3 is made up of

sheets of ray cells that react strongly to block the spread of pathogens. Rays are connected to the living phloem of trees and can bring additional food to form strong reaction zones to limit the horizontal spread of infection, especially in the outer sapwood near the bark (5).

In the CODIT model all the various patterns of discolored and decayed wood are explained in terms of the relative effectiveness of the four walls. Wall 1 allows relatively rapid spread of infection compared to walls 2 and 3, thus producing elongated columns of discolored and decayed wood. Horizontal spread of the columns is greater radially than tangentially, because wall 3 is more effective than wall 2. Columns generally do not extend horizontally into wood formed after wounding because wall 4 is highly effective because of its altered anatomy. Wall 4 keeps decay-causing pathogens contained within wood exposed at time of wounding with some exceptions such as canker-rot fungi. The major problem with the CODIT model is that walls 1, 2, and 3 do not correspond to the reaction zone as wall 4 corresponds to the barrier zone. This has created some confusion about the concept of compartmentalization in trees.

The concept of compartmentalization of decay in trees either understood in the functional view of reaction zones and barrier zones or the CODIT model view of walls 1, 2, 3, and 4, replaces the heart rot concept of decay in living trees. Heart rot has been defined as the decomposition of dead heart wood inside a living tree by saprobic organisms that gain entrance via wounds. According to the heart rot concept, the tree is passive; the decomposers are not pathogens that interact with living tissue. Compartmentalization involves an active tree in which live cells of the tree interact with complex infections involving a variety of pathogenic and saprobic microorganisms over long periods of time. Tree response to injury and infection results in the formation of a variety of protective layers that vary in their effectiveness, depending on genetic and environmental factors.

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COENOCYTIC

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Any structure (in fungi — any *spore*, *hypha*, or *mycelium*) that contains many nuclei and is not divided by cross walls (*septa*) is, by definition, coenocytic. A much broader definition is that any structure within which nuclei have contact with the same continuous cytoplasm is coenocytic. Thus even regularly septate hyphae, with their open-pored septa, are considered to be coenocytic by some authors.

COFFEE RUST

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The importance of coffee rust stems from the significance of the coffee crop in the social and economic stability of tropical countries worldwide. The genus *Coffea* includes nearly 100 species, but only *C. arabica* and *C. canephora* are agronomically important. *C. arabica* is widely planted throughout the world, and its varieties produce the kind of beverage known as "mild." *C. canephora* is cultivated mostly in Africa, and generates a beverage known as "bitter" (1).

The dispersal of *C. arabica* from Ethiopia, its center of origin, to become a major commodity, took place during the great explorations of the fifteenth and sixteenth centuries. Except in lowlands, *arabica* coffee adapted to tropical moist forest conditions. Coffee reached the American continent, where the current major exporters are located, around 1720 as plants that originated from seeds gathered from a single plant. Thereafter coffee estates in the Americas were planted mainly with two genetically related varieties, Typica and Bourbon. In the first half of the twentieth century, Caturra, a semidwarf natural mutant was found in Brazilian plantations. It has given rise to several varieties used because their smaller size facilitates an intensive cropping system (2). In spite of this change, the genetic basis of coffee plantations in the Americas has remained dramatically narrow.

Considering the devastating effect of the coffee rust in other parts of the world, the rust fungus, *Hemileia vastatrix* Berk. & Br., became a legitimate cause of concern for the coffee industry in Brazil, Colombia, and other major producing countries. Coffee rust was first reported from East Africa in 1861, and its first major outbreak took place in Sri Lanka (then Ceylon) in 1869. At the onset of that epidemic, Sri Lanka was the most important coffee exporter, but 10 years later coffee rust had taken a toll of over half the production and had ruined many coffee growers. In the end, Sri Lanka gave up coffee production and became instead a major tea exporter. In a few decades, the coffee rust pathogen spread to all coffee producing regions in the eastern hemisphere. In 1970 coffee rust made its way into Brazil, and by 1985 the disease was present in every coffee producing country in the Americas. In the absence of control measures, losses from coffee rust may run as high as 25 to 30% in Colombia and Brazil, respectively (3).

Hemileia vastatrix, the causal agent of coffee rust, is a uredial fungus with a partly expanded life cycle,