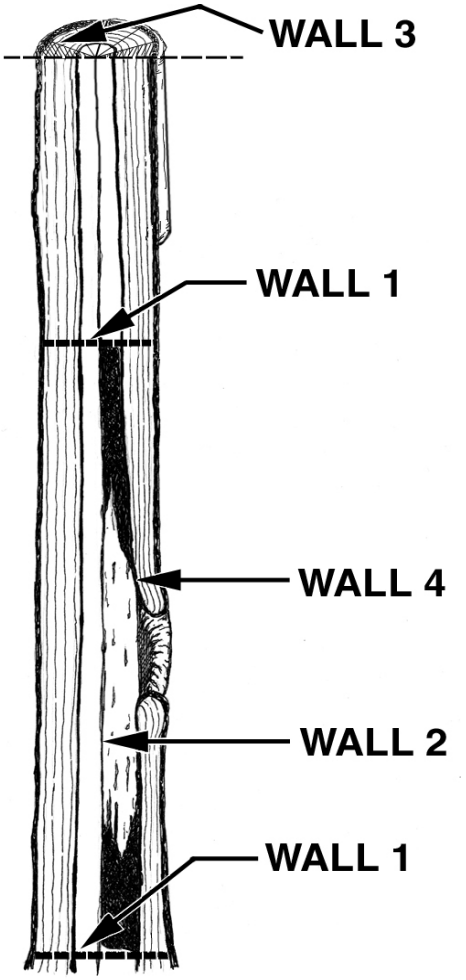
Sonja Kuster 08/01/2024
Woody Plant Physiology
Unit Code: A/602/3922
UNIT GUIDE 2023-24

LO 7 Understand the defence mechanisms used by woody plants

A plant's defensive mechanism kicks in when it comes under attack from a pathogen, which helps to stop the sickness from spreading. To stop the infection from spreading, this reaction may involve the synthesis of chemical defences, the activation of defence genes, and the construction of physical barriers. Compounds known as chemical plant defences are created by plants to fend off pathogens like bacteria and fungi. These substances may possess antibacterial qualities that aid in halting the development and dissemination of pathogenic microorganisms. Plants can defend themselves chemically using alkaloids, tannins, and secondary metabolites like salicylic acid and jasmonic acid. These substances can be harmful to pathogens and strengthen the plant's defences against infection. Plants have an internal defence mechanism called systemic acquired resistance (SAR) that enables them to fend off pathogen attacks in the future. A plant's defensive mechanism is triggered by pathogen exposure and aids in restricting the disease's progress. The plant's general immunity gradually gets stronger as a result of this reaction, increasing its defences against infections in the future.
Physical components and mechanisms known as "mechanical plant defences" aid in defending plants from illness. In addition to defence mechanisms that stop illness from spreading after it has entered the plant, these defences can include hard outer coverings like the cuticle and bark that keep pathogens out.
The waxy coating called cuticle, which coats the surface of leaves and stems, aids in retaining water. In addition, it acts as a physical barrier to help keep viruses out of the plant. Plant cell walls support structural integrity and aid in keeping diseases out of the plant. By creating and releasing antimicrobial chemicals that can aid in the destruction of disease-causing pathogens or stop their growth, they can contribute to defence.

7.1. Describe the formation of the walls/barriers formed as part of the CODIT model

Robert Hartig, a German forester who developed contemporary forest pathology, developed the conventional understanding of wood deterioration in living trees towards the end of the 19th century. According to conventional wisdom, fungi that compartmentalise deterioration in trees are the source of wood decay. Four barriers, or walls of defence, against deterioration in the plant's woody sections are depicted by the CODIT system. Bark wounds allow access to walls 1, 2, and 3. Wood is essentially dead, inert tissue; thus, once decaying fungi enter the tree, they will spread unrestrained throughout the stem. Up until recently, this idea was believed until Dr A. L. Sigo, a plant pathologist with the US Forest Service, and associates offered a more comprehensive understanding of decay. The enlarged idea still holds that wounds serve as a point of entry for organisms that cause wood decay, but it now acknowledges the involvement of a variety of microorganisms, including bacteria and fungus that do not cause decay. The enlarged idea also considers the way in which trees block off or compartmentalise unhealthy tissue in response to injury or infection. The broader definition of degradation has a significant impact on a variety of arboriculture techniques. The CODIT model is a natural defence mechanism that keeps decaying organisms from spreading throughout the wood and guarantees the tree's survival.
CODIT
A model known as CODIT was created to show how trees respond to damage and invasion by decay organisms. CODIT is either present in healthy plants or takes shape right after an injury. When new growth begins after injury, the cambium forms Wall 4.

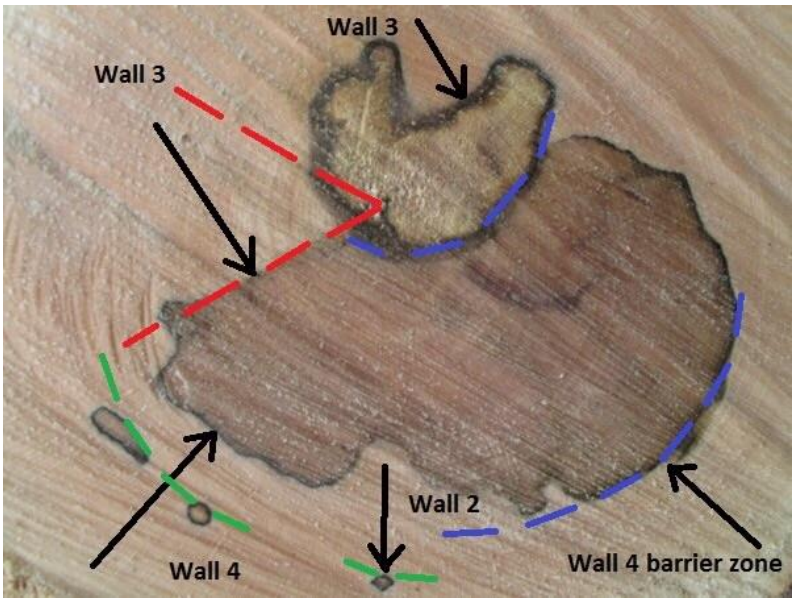


After the tree is injured, the cambium forms Wall 4, a new protective wall. This wall is an example of a barrier zone that prevents degradation from penetrating tissues that have been wounded. This barrier zone is made up of cells with strong walls that are poisonous to decaying organisms. The fourth wall is the strongest; provided that another lesion does not breach it, degradation can only spread to tissues that were developed before being wounded.

The ray cells that make up Wall 3 prevent decay from moving radially, or around the stem. The strongest wall in place now of wounding is wall 3.

The tangential wall, or wall 2, prevents decaying organisms from moving inward and towards the pith. The final cells in each development ring form this wall, which surrounds each growth ring continuously, except for the points where rays pass through. The second weakest wall is wall 2.

Decaying organisms are prevented from moving vertically by Wall 1. The vascular elements' production of gums, resins, tyloses, and other materials forms this wall in the vertical vascular system above and below the wound. Because Wall 1 is the weakest wall, decay organisms can travel vertically farthest.



A portion of the DNA makeup of the tree controls the compartmentalization of degradation. Studies show that some species and individuals within species differ significantly in how well they can compartmentalise degradation and damage. Breeding of urban trees that effectively compartmentalise degradation will be made possible as a result. To some extent, tree vigour also influences compartmentalization; robust trees are better at compartmentalising than weaker ones. To help trees compartmentalise degradation, it is crucial to maintain the vigour of urban trees through regular fertilisation, appropriate trimming of deadwood and competing and conflicting limbs, irrigation during dry months, and control of insects and diseases.

Polyporus squamosus, poorly confined within Sycamore, on which Polyporus squamosus is rather aggressive. Images progressing from top to bottom of decay column

https://arbtalk.co.uk/forums/topic/12987-the-beauty-of-codit/page/2/

7.2. Define the terms callus, wound wood and occlusion

Pathologists and botanists have focused a great deal of attention on trees' responses to wounding over the past 200 years, and for more than 4,000 years, people have tried to heal tree wounds. Arborists who frequently prune trees and are called upon to assess the response of trees to wounding in risk and plant health care assessments are also interested in the topic of tree response to wounding.

Pruning is how arborists wound trees, and assessing the growth of wound wood indicates whether or not the branch received the correct pruning cuts. This illustration shows a red maple (Acer rubrum) pruning wound with a full ring of wound wood surrounding it, signifying a correct pruning cut.

As you can see in the picture above, over time, this pruning wound has been almost completely sealed.

Here, an incorrect pruning cut has resulted in tissue dieback around the top and bottom of a pruning wound.

Callus

A collection of parenchyma cells, which are live, thin-walled cells found in leaves, wood, bark, and other tissues in plants, is called a callus, and it occurs soon after a wound. In sapwood or bark, callus can be produced by the cambium or parenchyma cells. For arborists, callus's key characteristic is that, if the tree is actively growing, it forms quickly after injury and is a transient response.

An amorphous mass of loosely grouped parenchymatous cells with thin walls that originate from the parent tissue's proliferating cells is called a callus. A callus frequently forms at the cut end of the stem or root after injury. It is a rather disorganised tissue made up of both differentiated (the precursor material for the induction of a callus, such as leaves, roots, shoots, etc.) and undifferentiated cells. Additionally, it is known that the gene expression profiles of the various callus types in Arabidopsis thaliana differ. As a result, cells with varying degrees of differentiation are included in the term callus. Unspecialized parenchyma cells make up the bulk of the callus's cells. This differentiation mainly occurs in the parenchymatous cells found in the explants. Therefore, a callus is essentially a less differentiated and disordered parenchymatous tissue. Usually, an unorganised proliferation of plant cells results in the formation of this amorphous tissue. As a result, callus cultures are essentially collections of cells grown as a result of the section of plant organs' uncontrollably proliferating cells. Using this method, plant cells multiply under appropriate conditions to create callus—clumps of undifferentiated cells. Before cell division may happen in a callus that has differentiated cells in isolated explants, the cells must dedifferentiate—that is, undergo modifications to become meristematic. Cell division occurs without dedifferentiation if explants are isolated and solely contain meristematic tissue. Mature adult cells can momentarily change back to their juvenile condition during the dedifferentiation process. The capacity for growth and division is higher in the revitalised cells. Under certain conditions, these cells can restore embryos or organs. As a result, when growing plants in vitro, dedifferentiation is an important stage to consider. Sufficient callus induction hormones are often supplemented at the time of callus commencement. Its growth and development require a growth-supporting medium and sterile conditions. The nutritional medium contains exogenously given growth regulators that affect the explants' ability to produce calluses. There are three types of hormone supplementation for callus development: cytokinin alone, auxin alone, or both cytokinin and auxin.

Callus has been widely used in basic research as well as industrial applications since the key findings. In addition to auxin and cytokines, other hormones that cause callus production include brassinosteroids and abscisic acid. In certain species, these hormones may even replace auxin or cytokinin. Fig. 2.5 highlights the factors that lead to the formation of calluses and the many ways in which they may do so. The main callus-inducing factors (auxin, cytokinin, wound, egg cell, and embryonic and meristematic fate) are indicated with dark grey circles, and the transcription factors that correspond to them are indicated with light boxes. In Arabidopsis, pericycle cells next to the xylem poles produce callus when shoot or root explants are cultured on callus-inducing media (CIM) containing auxin and cytokinin. This study has demonstrated that calluses are organised masses that take on varying morphologies according to environmental factors, both in nature and in vitro. There are various forms of callus, based on their ability for organ regeneration (Fig. 2.6). Furthermore, the morphology of wound-generated callus and callus induced by medium growth differs. In the natural world, calli are produced by hereditary tumours, wounds, and microorganisms that cause tumours (Ti gene) (Fig. 2.6).

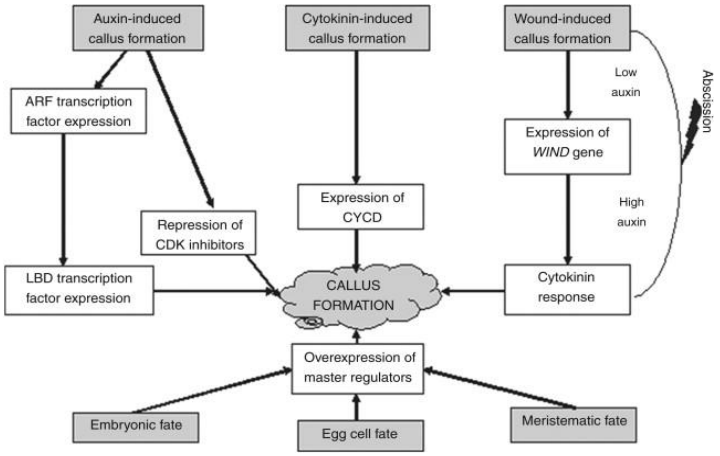


Figure 2.5. Mechanism involved in callus formation.

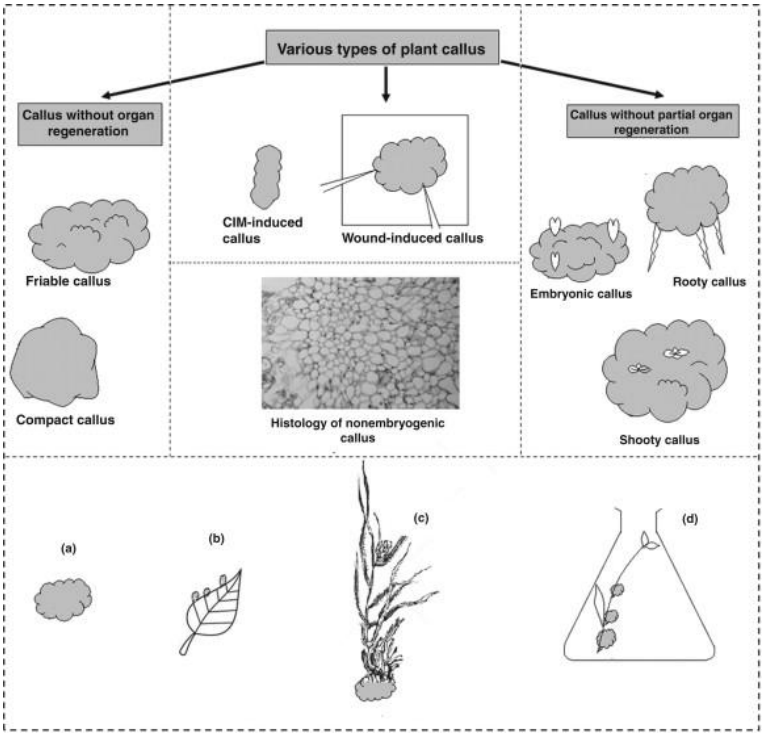


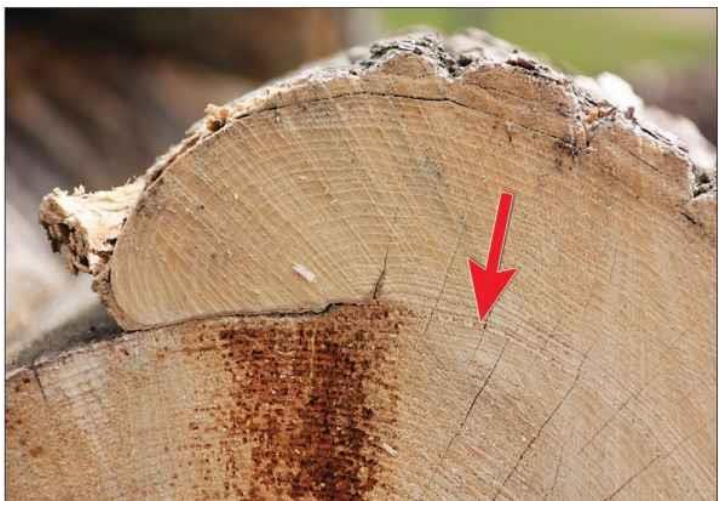
Figure 2.6. Diagram illustrating callus kinds, histology, and morphologies (adapted from in vitro and nature). (a) In vitro formation of a callus; (b) induction of a callus at the site of a wound; (c) bacterial infection-induced tumour; and (d) genetic tumour resulting from an interspecific cross between two Nicotiana sp.

Wound wood

Wound wood is a lignin-filled, extremely organised wood. De Vries came up with the phrase after he saw that the wood that had been wounded had cells that were shorter than usual and lacked medullary rays and arteries. The term "wound wood" was expanded by Küster (1913) to encompass tissues that resembled wood and were created following trauma. One is that, although callus production comes first, it develops into vascular cambium within months, which comes before wound wood. As a result, callus is rarely visible outside of the first few weeks or months following its creation and is swiftly covered up by this growing wound wood. Consequently, callus is one of the tissues from which wound wood is first created, while wound wood is the tissue that arborists can see years after damage. At the edge of injuries that have destroyed or exposed the phloem, vascular cambium, or sapwood, wound wood grows from callus or from unharmed vascular cambium. Wounds that are shallow and only affect the bark's outer layer do not encourage the growth of wound wood. Bark forms wound or necrophylatic periderm in response to wounding in a unique way. On the other hand, callus and wound wood can form due to cells found in the phloem or inner bark.

Callus forming along a root tear.
Callus (arrow) may have chlorophyll as seen in this image.

With normal cambium and bark, wound wood (arrow) eventually develops in the same way as regular sapwood. One way of figuring out when a tree was wounded is to count the number of rings each year in the wound wood. To use this ageing method, you must locate the barrier zone that developed in the oldest annual ring that was there at the time of the wound and count any subsequent annual rings that formed in the sapwood or wound wood. In general, this technique is more intricate than is mentioned here.



Wound on an ash surface gave rise to wound wood. Keep in mind that the age of the wound can be ascertained by counting the rings in the wound wood that follow the barrier zone (red arrow).

Occlusion

Pruning is an important part of tree management in both forestry and urban contexts.
Trees respond to pruning cuts in two ways: on the outside, callus wood forms over the cut, a process known as occlusion, and internally, chemical reactions in the wood encapsulate discolouration.

The process by which trees develop clear wood and callus over wounds is known as occlusion. Typically, callus formation over the branch stub is the first step in the process of branch wound occlusion. Eventually, a new cambium layer and fresh, transparent growth rings form. The occlusion zone, where the tree develops callus wood and new growth rings occlude the wound, is located between the branch stubs and the clear wood. The faulty core, an uneven cylinder whose size and form vary depending on the pruning regime, is the inner log that houses the occlusion zone and the unpruned core.

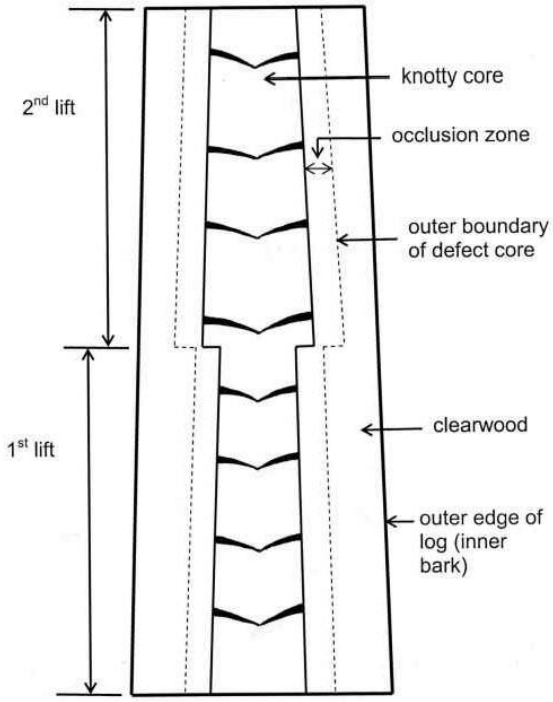


Diagram of a trimmed bole with the clear wood created outside the knotty or faulty core and the occlusion zone highlighted. A bole trimmed in two lifts is seen in this illustration. The occlusion zone and the knotty core are both parts of the defect core. The schematic does not scale well.

When the defect core size is as small as possible in relation to the log size, the goal of optimising clear wood output is accomplished. One significant way to achieve this goal is to use the pruning approach to reduce the size of the occlusion zone. Therefore, a trade-off between the time and intensity of pruning procedures and tree growth determines the size of the fault core. Early or severe pruning will increase the amount of clear wood produced by reducing the size of the defect core, but it may also have the unfavourable effect of reducing tree development. Forest trees are pruned to achieve goals including increasing fuelwood yield, improving aesthetics, lowering fuel ladders, and eliminating or lessening pathogen susceptibility. Urban trees are often not pruned to improve the quality of their wood but rather to change the shape of the tree, remove dangerous branches, or increase tree health. No matter the circumstance or the purpose of the pruning, branches must be removed, and the methods chosen to do so have an impact on the wood quality, beauty, and health of the tree.

Like the stem, the vascular cambium divides the xylem and phloem in a living tree branch. These are all linked to their corresponding elements on the stem. The sapwood, or working xylem, continues to carry water and nutrients out of the branches, while the carbon created during photosynthesis moves from the leaves to the stem phloem via the branch phloem. In certain species, especially on bigger branches, there is frequently a branch collar at the base of the branch where it joins the stem (Figure 2). The relationship between conducting tissues in the stem and branches is not broken by the branch collar, which is an enlargement of xylem and callus tissues. It appears that it serves as a barrier against fungal infection in the main stem and as a support structure. A dead branch may have a branch collar but otherwise have no living tissues or connections to the stem. For as long as the dead branch remains in situ, the stem cambium effectively builds a ring around it that expands outward with the formation of each annual ring. When wood is cut from this portion of the tree, it becomes loosely knotted due to the separation between the cambium and the stem xylem that forms following branch death. The wound occlusion following pruning is significantly different from that of dead branches because of the anatomical and functional distinctions between the two. Pruning a live branch severs the living xylem, phloem, and cambium tissues, whereas removing a dead branch

typically will affect no living tissues. The conundrum over pruning primarily involves live branch pruning. However, studies involving dead branch pruning provide some relevant insights into wound occlusion for both types of branches.

Walnut-wound open

Walnut-wound partial occluded

Walnut-wound-occluded

Resources

https://www.treerot.com/wp-content/uploads/2016/04/Arborist-News-Callus-and-woundwood_Luley.pdf
https://arbtalk.co.uk/forums/topic/12987-the-beauty-of-codit/page/2/
https://www.bartlett.com/resources/compartmentalization-of-decay-in-trees.pdf
https://studymind.co.uk/notes/chemical-and-mechanical-plant-defences/#:~:text=When%20a%20plant%20is%20attacked,the%20spread%20of%20the%20pathogen.
https://www.sciencedirect.com/topics/agricultural-and-biological-sciences/callus
https://chrisluleyphd.com/wp-content/uploads/2022/02/5.-tci-ww-2018.pdf
https://escholarship.org/content/qt2nh227fr/qt2nh227fr_noSplash_ff4f5a8caad5cdcc1bbe3eee976a4cab.pdf?t=lnq4cr