Cankers

FRST 307 Fall 2017

www.forestryimages.org

- Website maintained by the Warnell School of Forestry at the University of Georgia, USA
- Unlike google images, this website is curated and accurate
- “call number” in black box (usually bottom left) will take you to the image and subject page

Canker diseases of trees and other plants

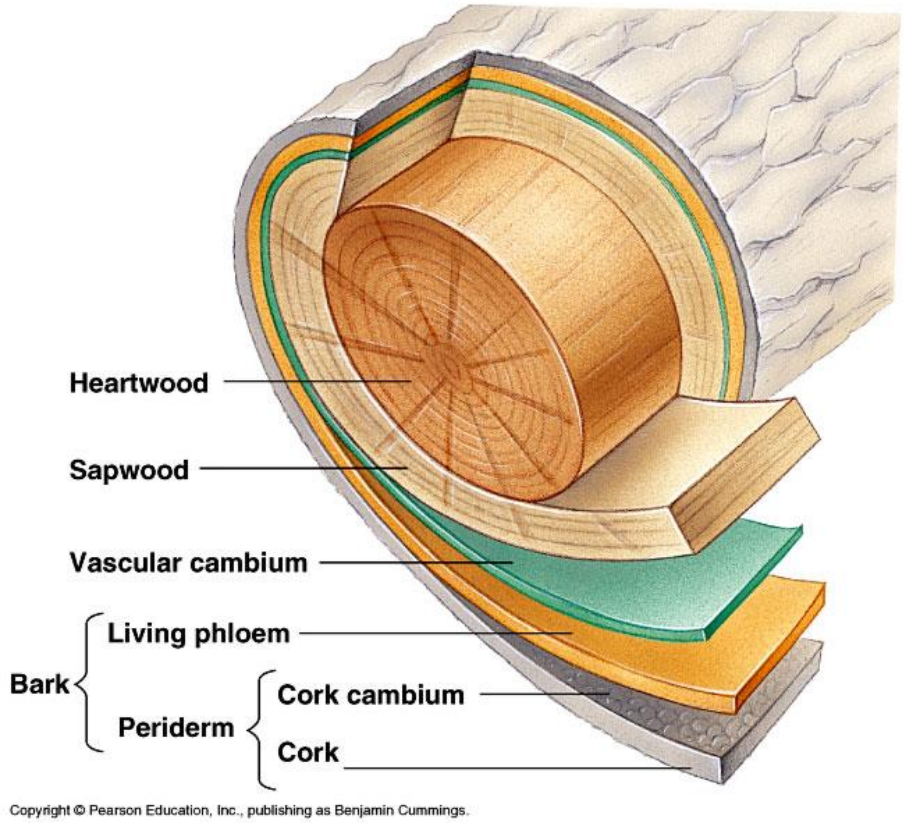
- Results from the death of definite and relatively localized areas of bark on branches and stems
- Most are caused by ascomycete fungi

Infection strategy

- Different strategy than root pathogens
- Have to overcome both physical barriers (bark) and active plant defence
- Most of the canker pathogens need a point of entry: the physical barrier of the tree is too much for the enzymatic arsenal of fungi
- Attacks living cells in phloem, cambium, xylem

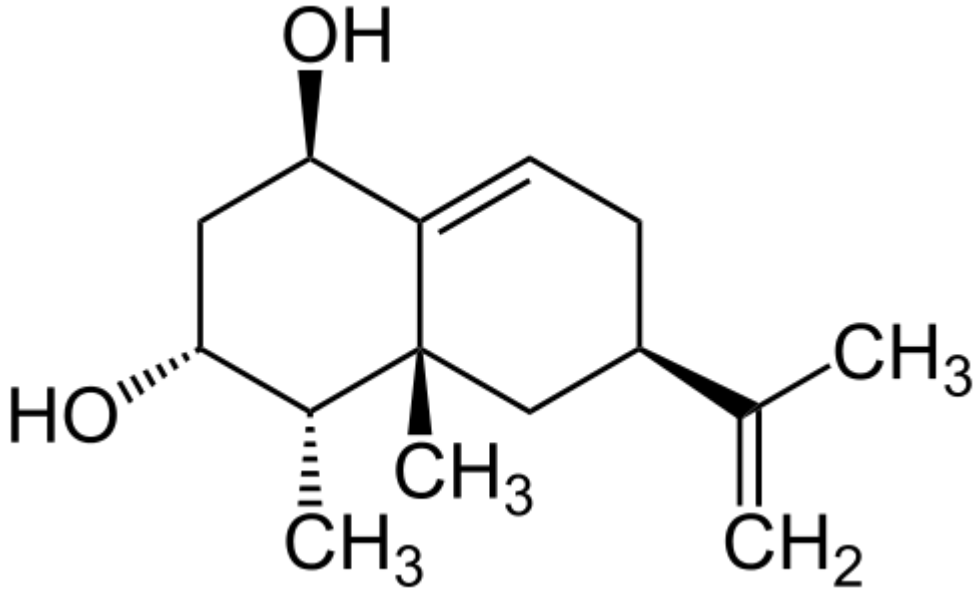
Natural host protection

- Outer surface of stems, branches and roots (but not the leaves!) are protected by periderm
- In response to wound or attack tree produces secondary periderm (called necrophylactic periderm)
- Efficacy of response depends on host vigor; complete necrophylactic periderm formation can take weeks to months



Phytoalexins – antibiotics for defense

- Defense compounds produced by plants
- Similar to antiobiotics: antimicrobial activity
- Mode of action: puncture fungal cell wall, disrupt metabolism, prevent reproduction
- Often specific to a pathogen
- Active response during growing season



https://en.wikipedia.org/wiki/Phytoalexin#/media/File:Capsidiol.svg

Dissemination of canker pathogen

- Often several spore types
- Ascospores
  - Wind disseminated
  - Result of sexual reproduction
- Conidia
  - Asexual reproduction
  - Often rain-splashed
  - Sometimes sticky, carried by insects
- These spores are generally not very resistant to UV light and drying
- Need periods of wet conditions for release and germination on new host
- Generally, no tree-to-tree spread by mycelium as with the root rots and decays

Pathways to infection

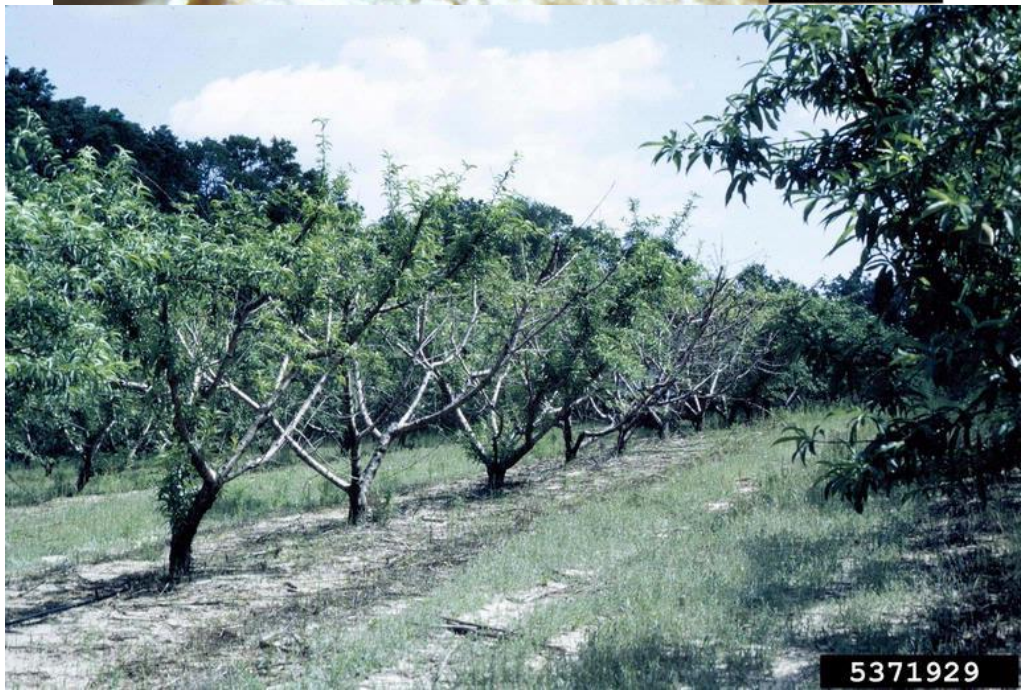
- Usually need wounding to enter host
- Insect wounds
  - Some insects create wounds and fungi use the wounds to enter
  - Other insects are vectors and create wounds to inoculate the trees
- Mechanical bark wounds, including pruning wounds
- Natural scars
  - Lenticels
  - Petiole scars
  - Dead branches or twigs
- Once inside the tree, pathogen still has to deal with active chemical and physical host response

Damage by canker diseases

- A single canker can be enough to kill a tree (if it is on the main stem)
- Bole deformation
  - Reduced lumber recovery in the sawmill
- Stain and resin soaking of wood and bark at the canker
  - Pulp chips contain resin-soaked bark
- Branch flagging
  - Leads to a general decline, dieback
- Point of entry for insects, other fungi, decay
- Breakage

Saprophytic cankers

- Associated with weakened trees
  - Can’t penetrate barrier of living cells in healthy tree
  - But in a tree that is otherwise stressed (abiotic, insects, other diseases such as root rots), tree cannot produce chemical and physical barrier to prevent the fungus from invasion
- Usually part of a complex that is often referred to as decline
- Example: *Cytospora* canker

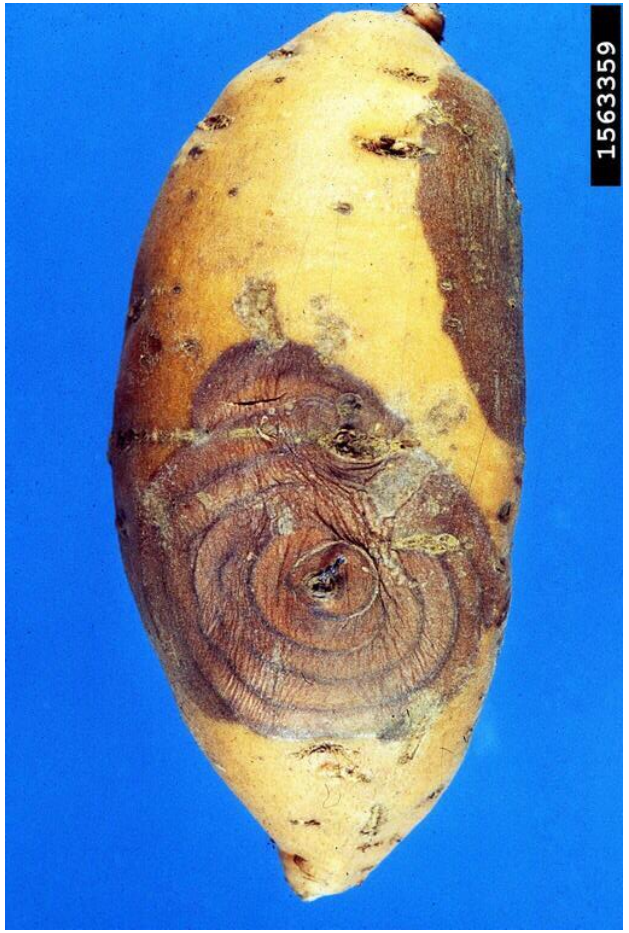


Cytospora canker of aspen

Annual cankers

- Opportunistic fungus that rapidly infects a tree through wounds before the host has a chance to respond
- Not particularly specialized;
  - few develop into large scale epidemics and are mostly related to some environmental stress
- Often overlooked and difficult to identify
- Example: *Fusarium* ‘pitch’ canker

Fusarium



Fusarium solani

Fusarium subglutinans

Fusarium subglutinans

Perennial cankers

- Invades the host when it is slow to respond
  - Invasion often in late summer, fall, and even winter when trees have formed terminal buds or are dormant
  - Often, fungi that are well adapted to growing in cold temperature, some can even grow at 2° C
- More aggressive than the annual cankers, more specialized
  - Must tolerate some host reaction
- Each year, when tree growth resumes, host responds by attempting to wall off the fungus by chemical and physical barriers
- When tree goes dormant again, fungus resumes its slow progression in the host
- Canker is the symptom of this annual battle between the tree and the fungus: causes typical « target shaped cankers
- Examples: Nectria canker, Ceratocystis canker

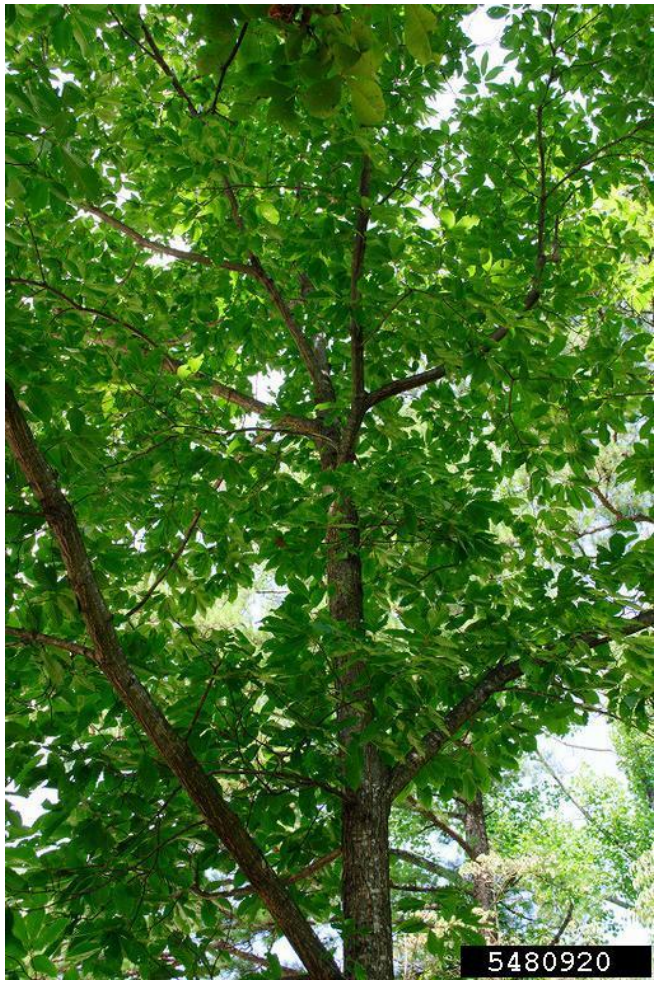
Nectria spp.

Diffuse cankers

- Pathogen dominates the host and the host response is not fast enough or the pathogen can defeat it
- Rapid invasion, production of toxins by the fungus
- Living host tissues often destroyed in a few years and tree is girdled causing breakdown in water and nutrient transport system
- Since tree does not have a chance to respond or produce callus, often a large canker not visible, instead have a diffuse canker
- Examples: Chestnut blight, Hypoxylon canker, Scleroderris canker

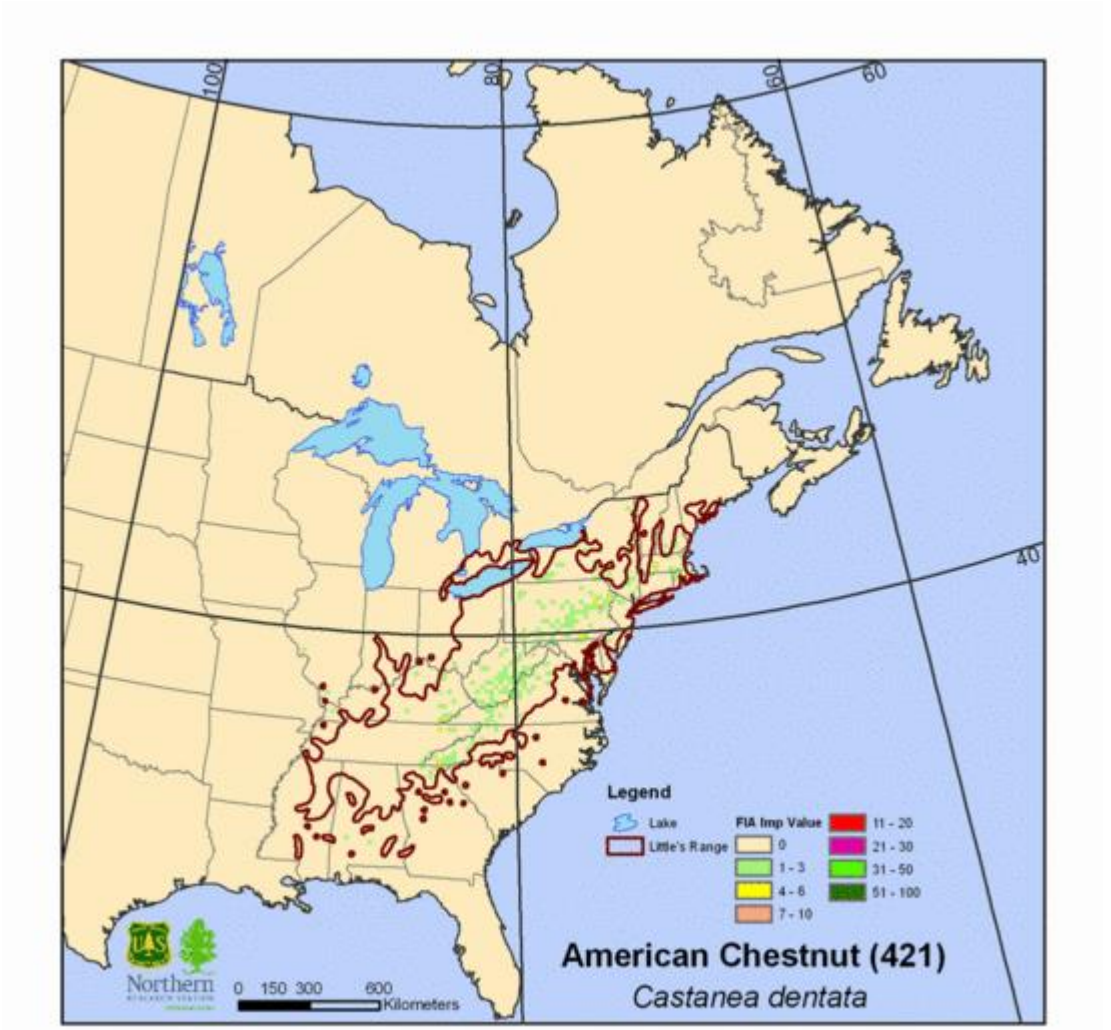
Chestnut blight - *Cryphonectria parasitica*

- American chestnut – *Castanea dentata*
- Extensive range in eastern NA, dominant angiosperm spp
- Timber, leather production, food (nuts)



Chestnut blight - *Cryphonectria parasitica*

- In late 1800's, Chinese chestnut introduced as ornamental
  - *Castanea mollissima*
- Chinese chestnut is resistant to endemic blight, *C. parasitica*
- *Cp* quickly spread though range of AC, destroying trees over ~ 2m tall
- Now, no AC bigger than 2m, after growing past that they die



https://www.fs.fed.us/nrs/atlas/tree/niche.php?spp=421&t=4

Chestnut blight - *Cryphonectria parasitica*

- *Cp* destroys phloem tissues (diffuse canker), girdling trees and cutting off photosynthate transfer

Chestnut blight – how do we fix this?

- Chinese chestnut is resistant, can we transfer that ability into American chestnut?
- Problems – AC very difficult to clone, clones needed for testing
- Dr. Scott Merkle – University of Georgia
- Somatic embryogenesis of backcrossed hybrids
  - Hybrids are created of AC x CC
  - These are backcrossed with AC to reduce CC morphology
  - SE of resistant individuals to propagate resistant AC phenotypes