Nutrition and Diseases of Forest Trees

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Most important diseases of forest trees are caused by fungi or parasitic plants (dwarf mistletoes)

Pathogenic fungi obtain nutrients by secreting extra-cellular enzymes that digest various parts of plant cells, often sequentially. The fungus then absorbs the products of the degradation across its membranes.

Two general categories of fungal pathogens, based on mode of obtaining nutrients are Biotrophs and Necrotrophs, aka “Sneaks and Thugs”
Biotrophs (Sneaks, obligate parasites)

- Enter intact living parts of tree directly or through natural openings such as stomates
- Most penetrate living cells and absorb nutrients with specialized hyphae
- They can exchange signals across membranes, creating a metabolic sink (swellings)
- Biotrophs often are favored by increased tree vigor, i.e., feeding the tree feeds the pathogen, too
- Examples: Rust fungi, powdery mildews, Swiss needle cast fungus

Western gall rust
Necrotrophs (Thugs, facultative parasites)

- Enter living or dead plant parts directly, through wounds or natural openings
- Kill host tissues (sometimes with secreted toxins) and degrade with enzymes, then absorb nutrients.
- Some are uniquely adapted to break down precisely those compounds produced by the plant for defense.
- Necrotrophs often are favored by *decreasing* tree vigor or increasing stress (injury, drought, etc.)
- Examples: Many common and important root pathogens, decay fungi, canker fungi, foliage diseases.
DISEASE TRIANGLE

PATHOGEN

ENVIRONMENT

HOST

DISEASE

Nutrient supply / balance
Disease Resistance in Trees

Disease resistance in trees is the result of several defense mechanisms that act in complimentary ways.

First are the pre-existing chemical and physical boundaries that make up passive or constitutive resistance.

Next is active or induced resistance - chemical and physical mechanisms triggered by infection or injury. Critical to their effectiveness is the timing of their arrival at the infection site (this has been shown to be affected by nutrient imbalance or water stress).
Constitutive or Passive Resistance is the first line of defense

- **Structural barriers**
  - Outer tissues (bark, leaf cuticle) contain suberized and heavily lignified cells – barriers to most pathogens.
  - Few pathogens can penetrate these in the absence of wounds, but some important ones are adapted to do just that (*Phellinus, Armillaria, Heterobasidion*)

- **Biochemical barriers**
  - Antifungal phenolic compounds, tannins, etc. (example: *P. weirii* and red alder)
  - Resins and related volatiles (some are fungitoxic)
  - Expensive for the plant to produce, therefore potentially affected by nutrition
**Induced Resistance is an active response**

- **Localized Induced Resistance** – most active defense is of this type
  - Necrosis of cells adjacent to wound or infection point (programmed cell death)
  - Hypersensitive reaction (rusts) – from direct contact with pathogen
  - Deposition of lignins and phenolic compounds in a boundary zone
  - Formation of a wound (or necrophylactic) periderm that walls off the pathogen
  - Nutrient status of the plant can affect the speed of this response, which is critical in the case of aggressive pathogens which can grow through the boundaries before they are complete.

- **Systemic Induced Resistance** – In some plants, prior infection induces resistance in previously uninfected parts of the plant. Not well researched in forest trees but there is some evidence of it.
The special case of mutualism: Mycorrhizae

- Increase nutrient uptake
- Reduce carbohydrate levels in roots
- Provide physical barriers to infection
- Produce antibiotics
- Favor rhizosphere microorganisms inhibitory to pathogens

*Very high N or P can suppress formation of mycorrhizae, but in practical forest situations these levels probably are not reached.*
Nutrition and Diseases of Forest Trees

• When certain nutrient elements become deficient or excessive, the balance may tip in favor of certain pathogens (or other agent)

• The mechanism for this can be:
  – Diminished capacity of the tree for active or passive defense.
  – Increased capacity for the pathogen to infect and cause disease.

• In pristine native forests, native trees / pathogens usually are in reasonable balance. However, specific nutrient deficiencies or excesses in the plant can result from fertilization, climate change, silvicultural practices, atmospheric deposition, or other stressors)

• In exotic plantations and afforestation, deficiencies often appear because of a different environment and the lack of co-evolved processes of nutrient cycling; often exacerbated by nutrient additions to increase fiber production (often N, because it is limiting in most instances)
NITROGEN is the nutrient most frequently associated with increased disease

TOO MUCH NITROGEN MAY:
• Increase and prolong succulence
• Alter phenology of host plant
• Reduce production / concentration of some compounds inhibitory to pathogens (lignin, phenolics)
• Provide greater energy source for pathogen growth and survival
• Alter pH
• Create imbalance of other nutrients import to defense
• Favors obligate parasites (stem and leaf rusts)

TOO LITTLE NITROGEN MAY:
• Slow induced response to pathogen attack
• May favor pathogens that attack weak or senescing tissue
• May not offer enough nutrition for pathogen
POTASSIUM fertilization often is associated with improved disease resistance.

- Poplar leaf rusts - slight deficiency in K resulted in severe disease; easily remedied with balanced fertilization.
- Pine needle cast diseases often (but not always) mitigated by K fertilization.
- Armillaria root disease (inland PNW) apparently more severe on sites with deficient K.
**CALCIUM**

- Affects composition of cell walls and their ability to resist pathogens.
- Also affects membrane permeability / leakage.
- Involved in solution movement across membranes (important for tolerance to low temperatures, water stress).
- Messenger element for induced response to pest attack.
SULFUR

• Balanced with nitrogen with the plant
• High N additions without S can create condition of deficiency
• Deficiency correlated with increased disease from *Dothistroma* on radiata pine in Australia
Examples of nutrient / disease interactions

- *Armillaria* root disease
- Laminated Root rot
- *Dothistroma* needle blight of radiata pine
- Swiss Needle cast
Armillaria Root Disease

Armillaria spp., Armillaria ostoyae

- **Hosts:** Very broad range of hardwoods and conifers
- Some species require pre-stressed host, others are primary pathogens.
- On hardwoods, Armillaria can be opportunistic, becoming aggressive when trees are stressed.
- *A. ostoyae* is an aggressive killer of conifers in the inland PNW
Armillaria Root Disease

- Susceptibility varies among tree species, by biotype of the pathogen, and by tree age.
- One mechanism of resistance involves rapid formation of a necrophylactic periderm that isolates or compartmentalizes the pathogen (Robinson and Morrison).
- Ability to form this quickly may explain part of the variation in resistance to Armillaria among different tree species and tree ages (Robinson, Morrison, Vanderkamp).
Armillaria Root Disease and Nutrition in the Inland Northwest

Observations: Sites with low K (metasedimentary and granite rock types), particularly when fertilized with N, appeared to have more root disease than sites with adequate K.

Entry and others (1991):
- Ratio of phenolics to sugars in roots was related to susceptibility of roots to infection and colonization by Armillaria (high phenol is good for the tree, hi sugar is good for the pathogen).
- High N plus low K, or just low K resulted in low Phenol : sugar ratio, and increased disease.

Shaw and others (1998):
- N levels affect starch concentration.
- K levels affect phenolic and tannin concentrations.

Schwandt (2002), fertilization trials in 30-35 year-old Douglas-fir plantations:
Manipulated the phenol : sugar ratios but found no relationship to amount of disease or mortality rates after 10 years.

Moore and others (2003, 2000):
Increased probability of tree mortality with increasing N fertilization across the inland northwest due to 1) increased competition, 2) decreased pathogen resistance, 3) increased probability of windthrow and snow damage.
Laminated Root Rot

*Phellinus weirii*

- **Hosts** – all conifers to varying degrees. Douglas-fir, mountain hemlock and true fir highly susceptible, hardwoods immune.
- Decays structural roots
- Failure of live green trees common
Laminated Root Rot
*Phellinus weirii*

- Spreads across rot contacts, does not require wound
- Can survive saprophytically for 50+ years in buried roots
- Disease patches expand radially 1-2 feet per year
LAMINATED ROOT ROT
Nutrition interaction #1

• *P. weirii* cannot assimilate nitrate, but antagonistic microorganisms can (Nelson, Li, others)

• **Hypothesis**: High N fertilization reduces *P. weirii* inoculum by increasing the activity of antagonistic soil microorganisms.

• **Fertilizer trials** to test effect of fertilization on fungal growth, disease intensification, or tree mortality:
  – Urea: No effect (Thies and Nelson 1988)
  – Urea, ammonium Nitrate, Ca nitrate, sodium nitrate: No effect (Wallis and Reynolds 1962)
  – Ammonium nitrate: No effect (Thies and Westlind 2005)
Hypothesis: In the Oregon Cascades, trees growing in the improved light and nutrient environment behind the wave front of an expanding disease patch are more resistant to *P. weirii* infection because of increased tree vigor (Waring and others 1987, Matson and Boone 1984, Matson and Waring 1984).

Experimental results: Inoculation of wounded seedling roots of mountain hemlock in controlled laboratory environments showed that trees without nutrient additions (low vigor) developed disease symptoms more rapidly than seedlings with nutrients (high vigor) under the same light conditions.

Field test: Douglas-fir and mountain hemlock trees were selected from different vigor classes (thinned vs. unthinned, dominant vs. suppressed) and roots were inoculated with infested wood dowels. No differences in infection rate or rate of fungal growth between treatments (Goheen and Hansen 2000).

Conclusion: There are better ways to manage Laminated root rot.
Dothistroma on Radiata pine in Australia

1. Low foliar sulfur concentrations appeared correlated with high disease severity.
2. Sulfur deficient seedlings in controlled environment produced high levels of arginine in foliage.
3. Arginine can be utilized by Dothistroma as a nitrogen source, causing rapid colonization.
4. In field studies, foliar arginine levels correlated positively with disease severity rating.
5. High N decreased disease resistance because of induced P and S deficiency and improved pathogen nutrition.
6. Allows risk rating and treatment options based on soil nutrient status (parent material) or plant nutrient status.

(Lambert, 1986)
1. **Hypothesis:** excess N contributes to the recent outbreak.

2. **Evidence:** 1) High foliar N in coast range Douglas-fir, and; 2) Disease severity correlates positively with foliar N (Waring et al. 2000)

3. **Experiment 1:** In 10 year old Douglas-fir, high N fertilization increased amount of disease caused by the pathogen as measured by percentage of stomates blocked, linking N levels inside needles to increased disease severity (El-Hajj 2004).
SWISS NEEDLE CAST OF DOUGLAS-FIR
(caused by *Phaeocryptopus gauemanni*)

4. **Experiment 2**: Reduced foliage retention was associated with high N fertilization at two of four Coast range study sites; treatment has not been linked to fungal abundance (Rosner and Rose 2004).

5. Many other related studies in progress

6. Much variation in disease severity can be explained by climate based models (Manter et al 2005) and tree genetics.

7. A nutrition solution is attractive because we can fertilize existing stands but we can’t change climate or their genetics.
Summary

• No generalization should be made other than balanced nutrition is best (or at least good enough) for the functioning of inherent pathogen defense mechanisms.

• The effects of nutrient balance on tree resistance to pathogens can be extremely complex, and always is linked to environmental factors and the specific host-pathogen relationship.