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Plant Defenses - How Trees Defend Themselves and Why it Matters

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Dept. of Plant Pathology

THE OHIO STATE UNIVERSITY DEPARTMENT OF PLANT PATHOLOGY

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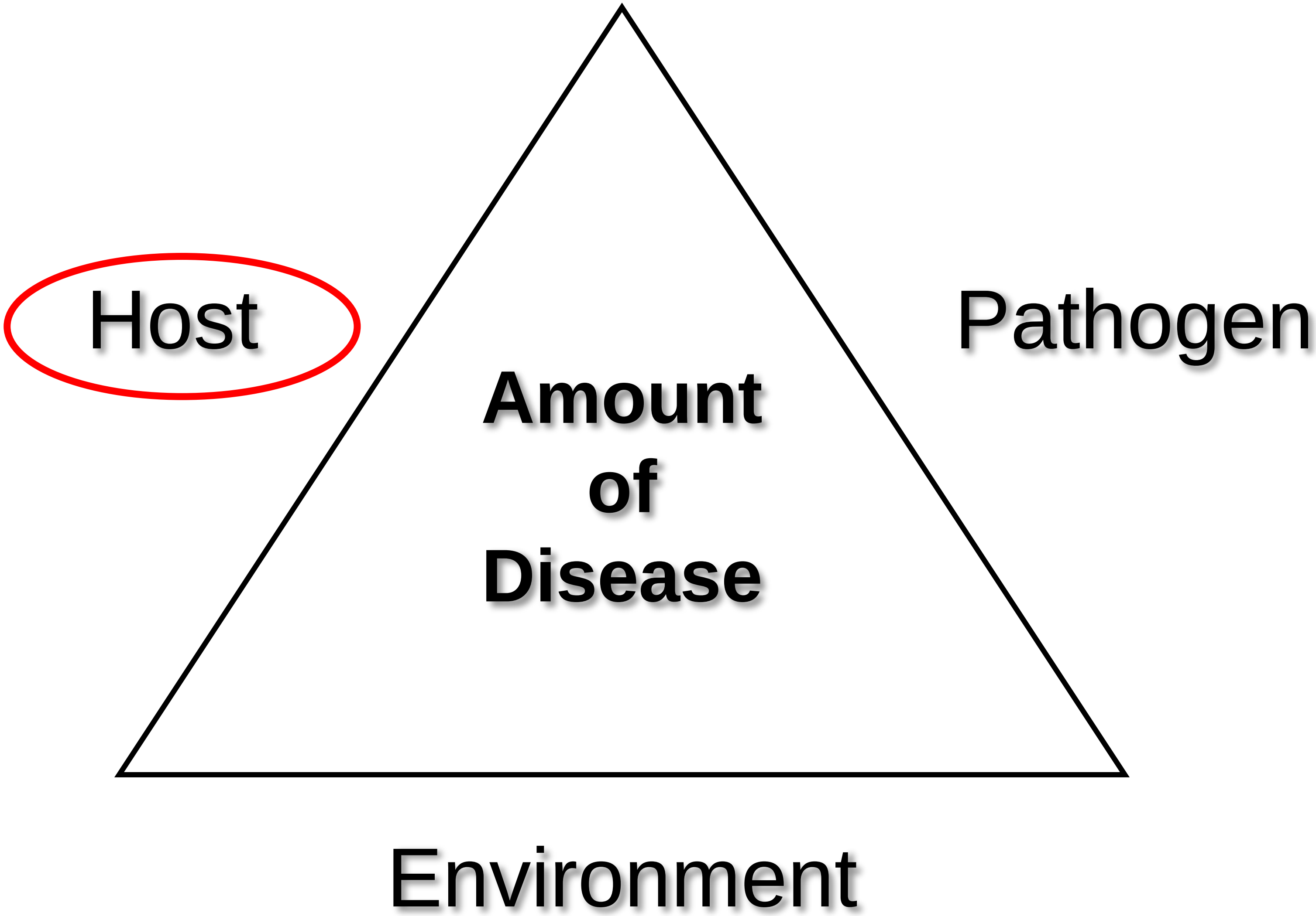
Why are we interested in this subject?

- Plant defenses are the foundation of host plant resistance
- Resistance is a cost-effective and ecologically-sound approach to disease and insect management
- Resistance is compatible with other IPM techniques and sometimes it is singularly sufficient to suppress pest damage to tolerable levels
  - In IPM, defense traits associated with resistance can be used as biomarkers for selection of resistant plant germplasm

Disease Triangle



Host

Pathogen

Amount of Disease

Environment

Why are we interested in this subject?

- To apply resistance as an IPM tool we need to understand it
- Only once we understand it will we be able to harness it to our advantage, i.e.
    - lower costs of pest control
    - lower human and environmental impacts of pesticide use

Layout of today’s talk

è Major concepts in tree defense against pathogens

è Current research on tree disease resistance

Mechanisms of Pest Resistance

Plants do not have an immune system as we understand it in humans, they defend themselves using:

è Mechanical (physical) barriers: preformed (passive), induced

è Chemical barriers : preformed (passive), induced

But: mechanical barriers are always the result of chemical processes!

Preformed Mechanical Barriers

è Leaves: e.g. cuticles, leaf hairs (trichomes)

è Stem and roots: outer bark

LEAVES

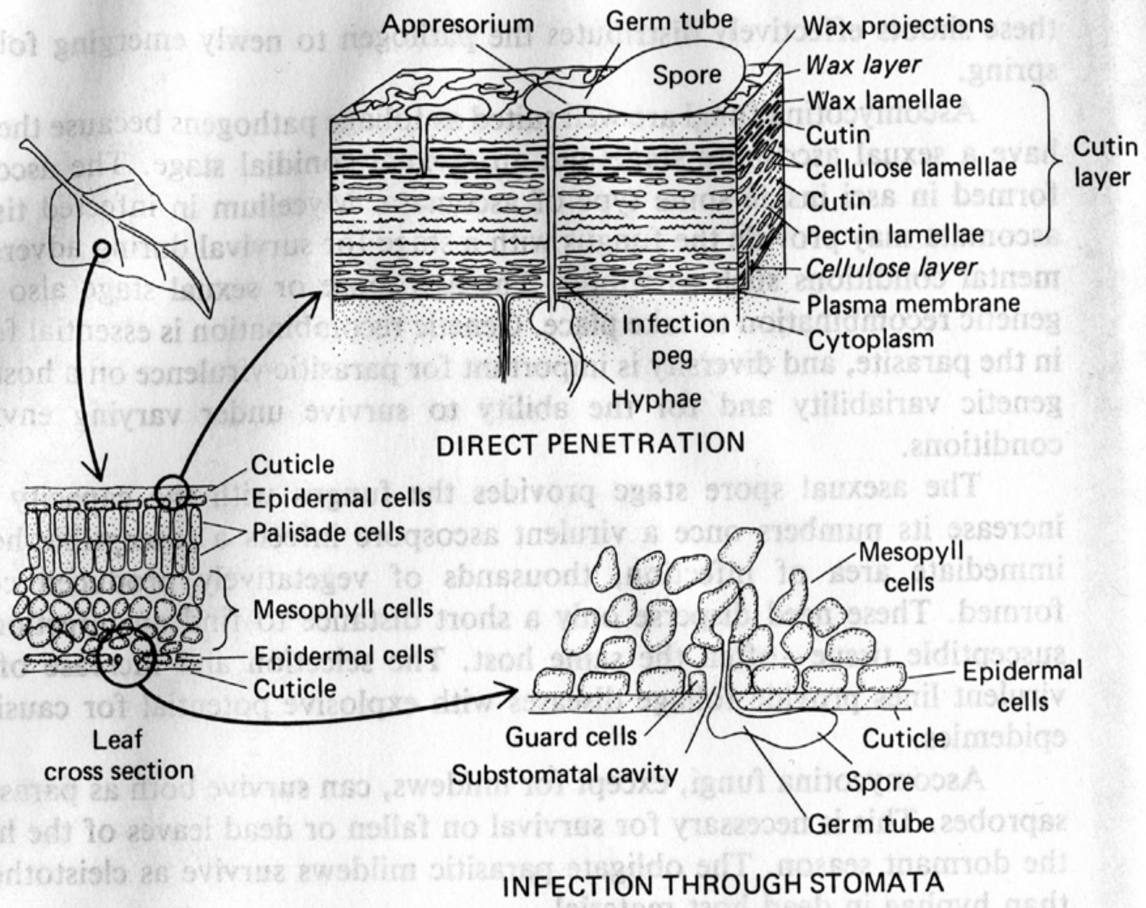


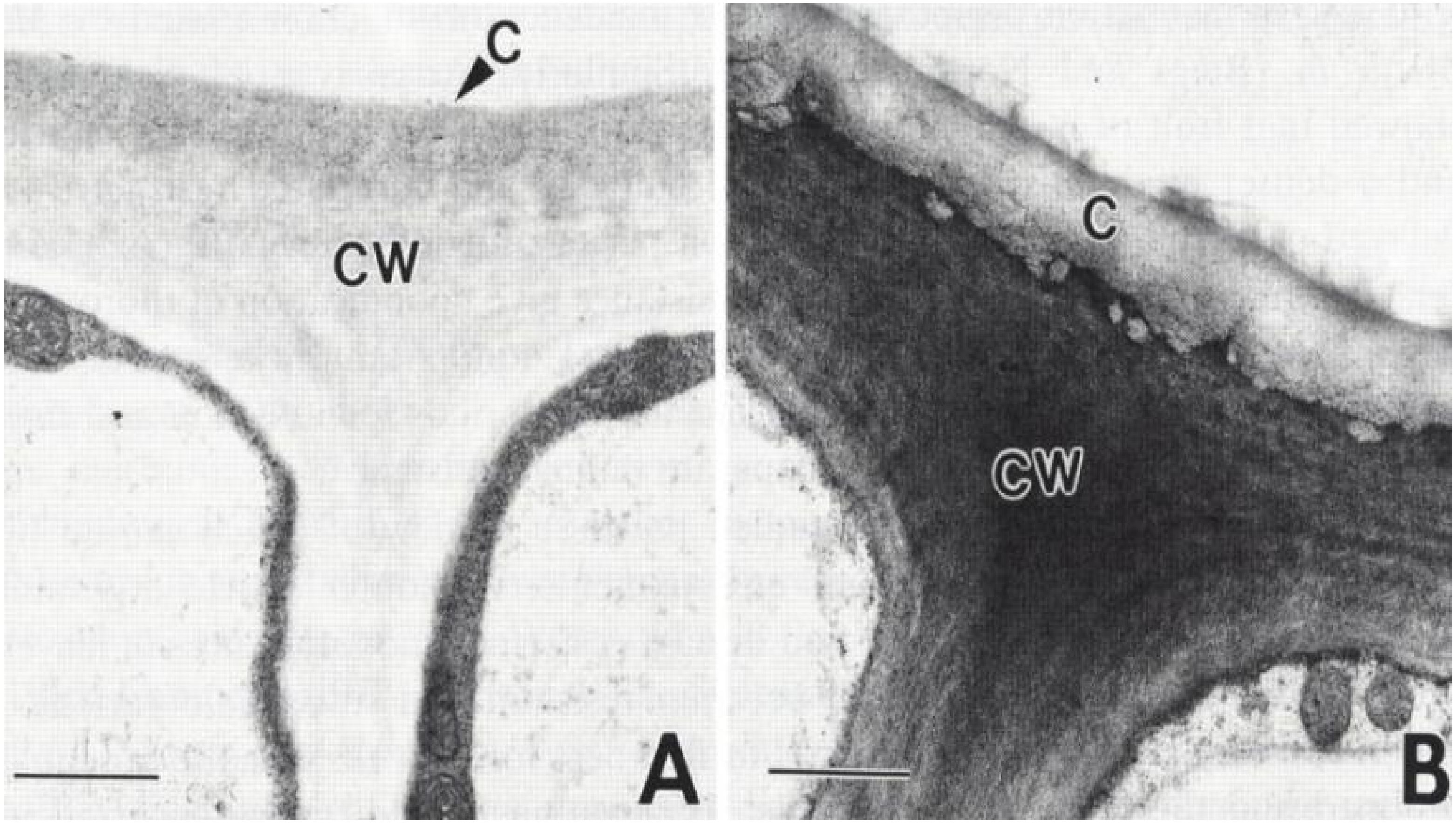
Figure 10–3 Infection by foliage fungi is by direct penetration or through stomates.

(Manion, P.D. (1991) *Tree Disease Concepts.* Prentice Hall, Inc., Englewood Cliffs, N.J.)

LEAVES

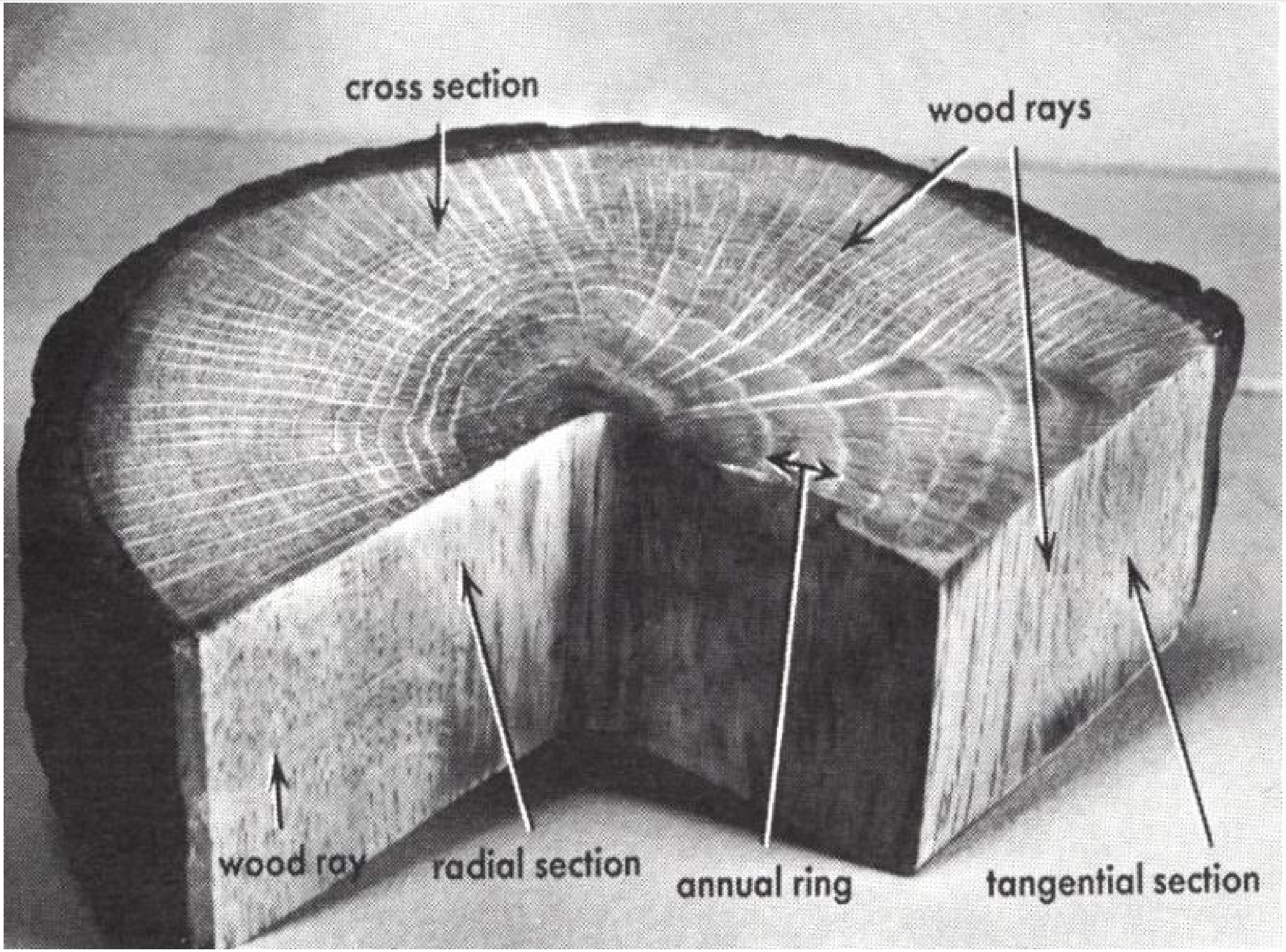
Young poplar leaf
(susceptible to
Marssonina leaf spot)

Older poplar leaf
(resistant to
Marssonina leaf spot)

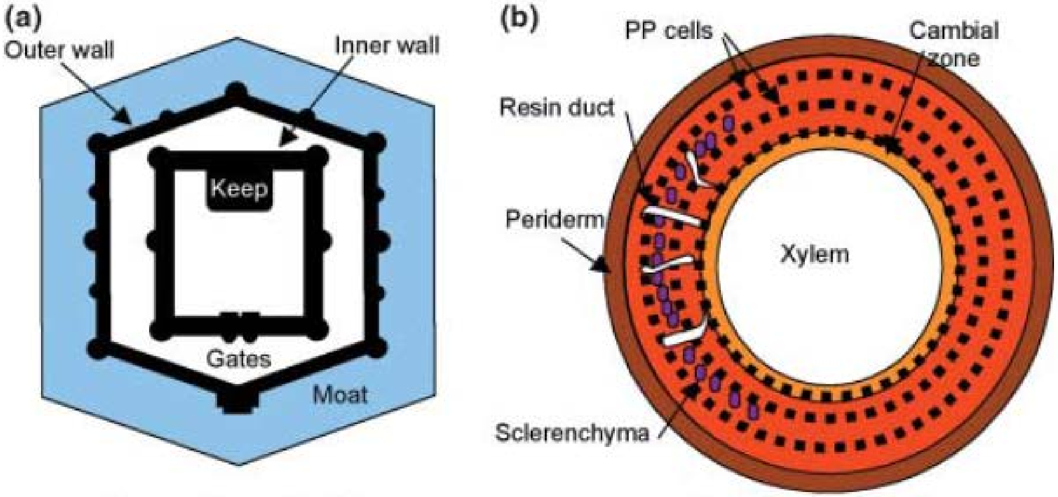


(Blanchette, R.A. & Biggs, A.R. (Eds.) (1992) *Defense Mechanisms of Woody Plant Against Fungi.* Springer-Verlag, Berlin)

STEMS

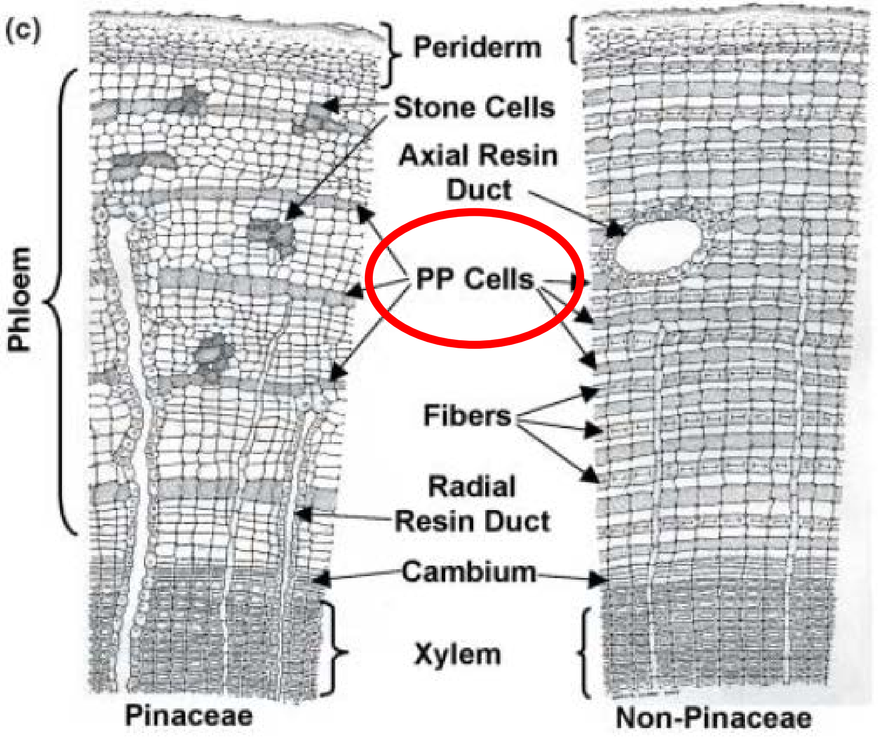


(Rost et al. (1984) *Botany - A Brief Introduction to Plant Biology*. John Wiley & Sons, New York)



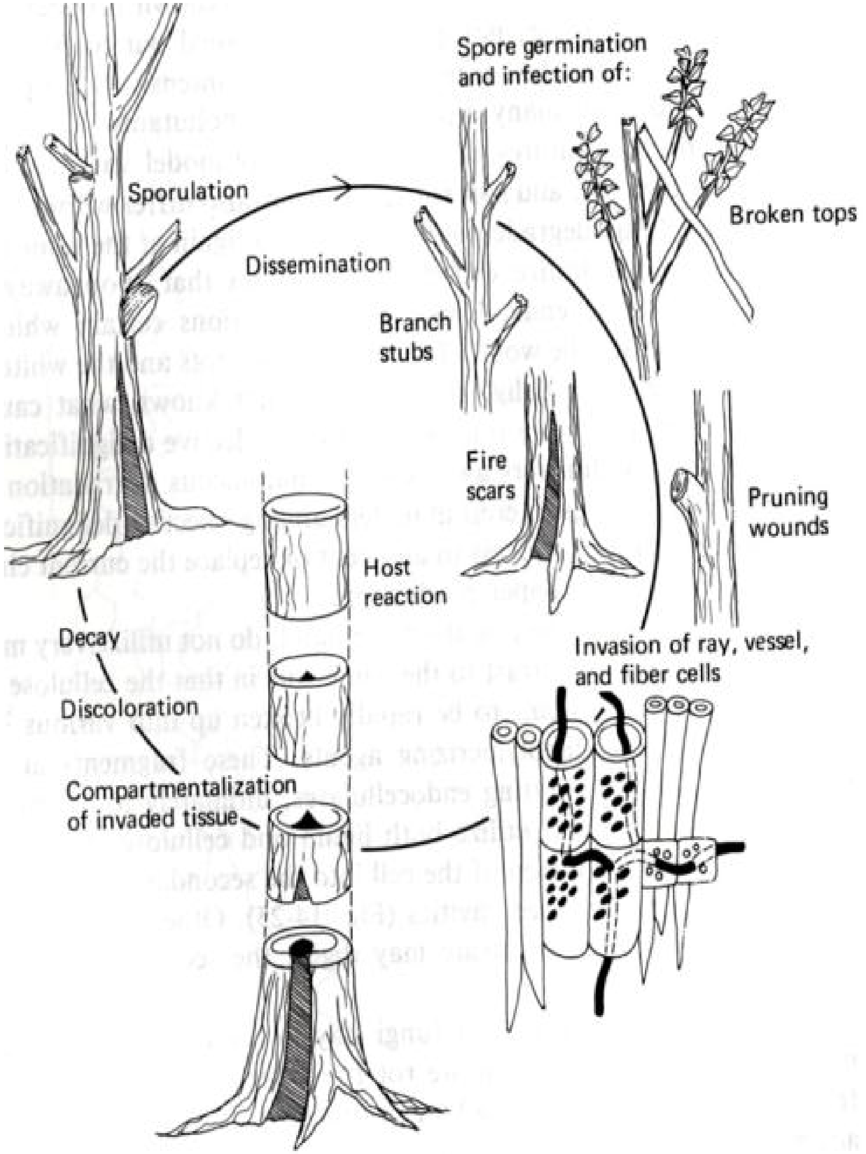
Concentric castle defense

Concentric bark defense



Franceschi VR, Krokene P, Christiansen E, Krekling T (2005) Anatomical and chemical defenses of conifer bark against bark beetles and other pests. New Phytologist 167 (2):353-375

STEMS



(Manion, P.D. (1991) *Tree Disease Concepts.* Prentice Hall, Inc., Englewood Cliffs, N.J.)

Preformed Chemical Barriers

è Leaves:

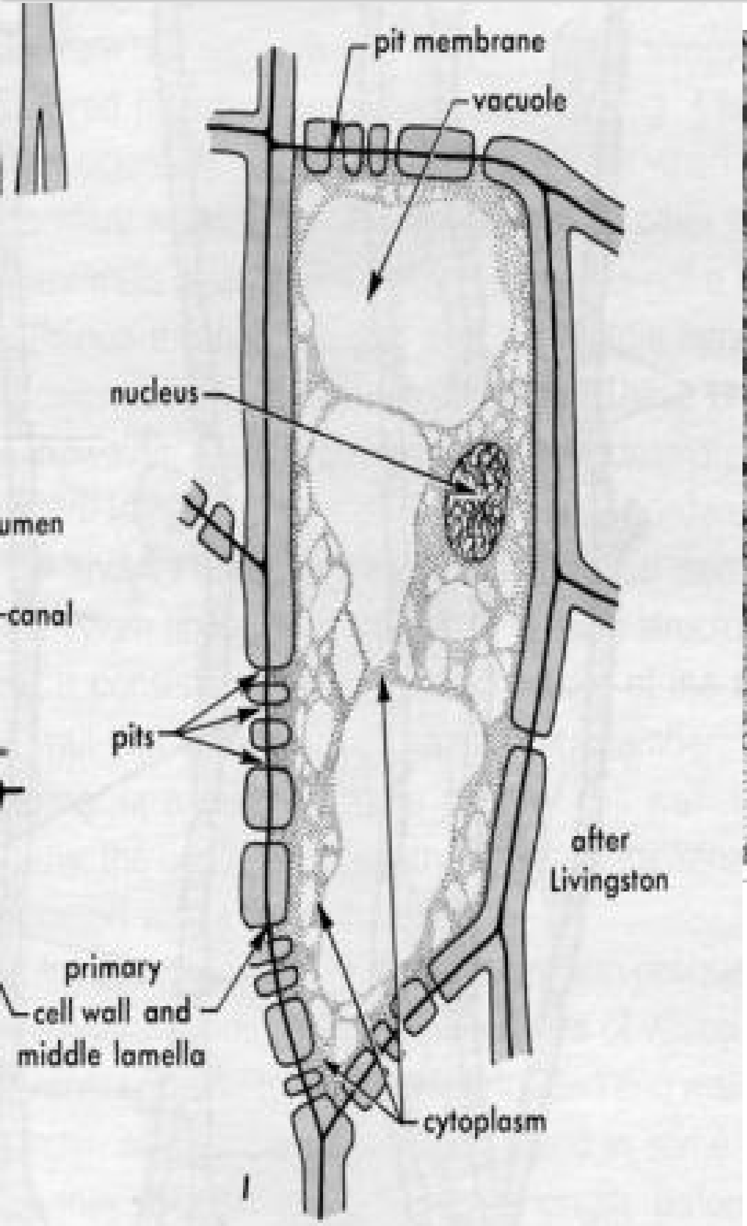
  è exuded antibiotic chemicals, e.g. gallic acid from Norway maple leaves

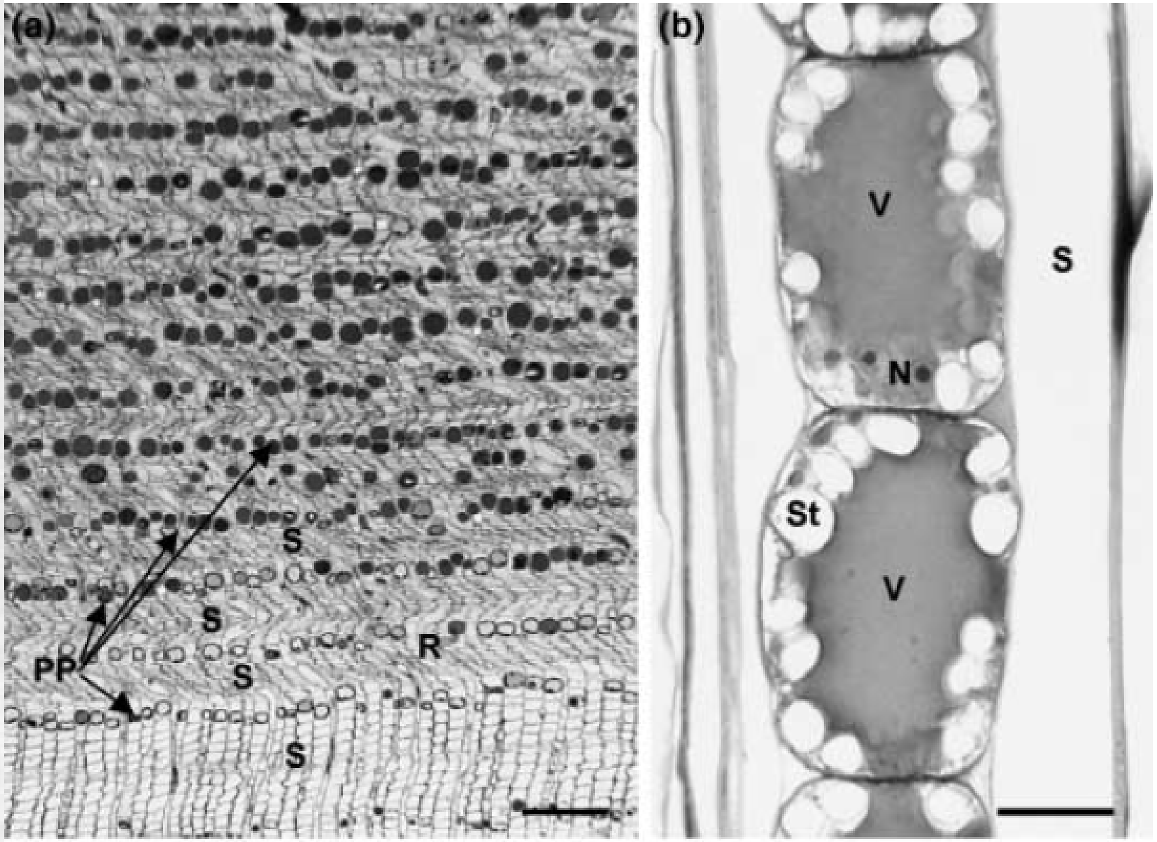
  è antibiotics inside leaves (a plethora), e.g. resin in pine needles.

è Stem and roots:

  è phloem and xylem usually contain all sorts of nasty chemicals

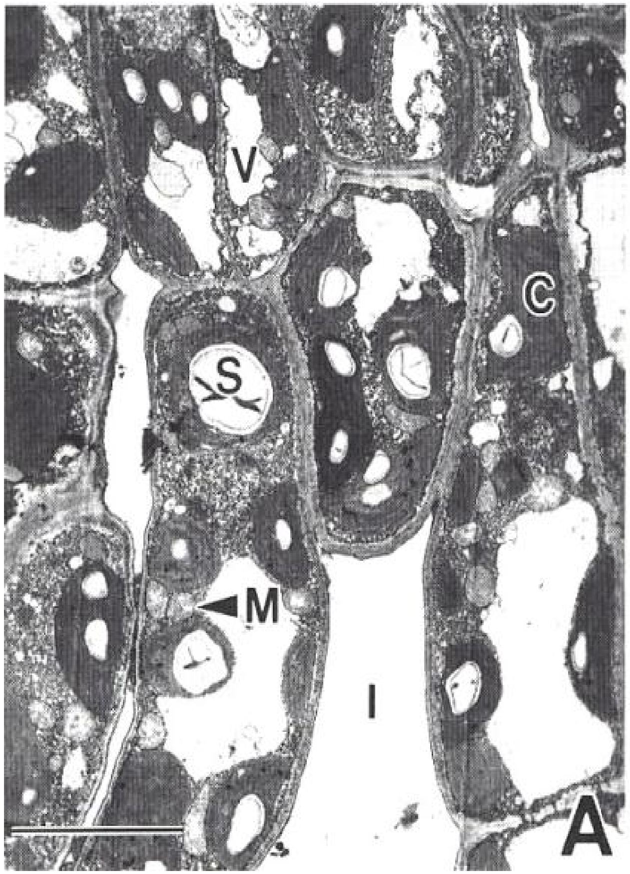
Soluble chemicals



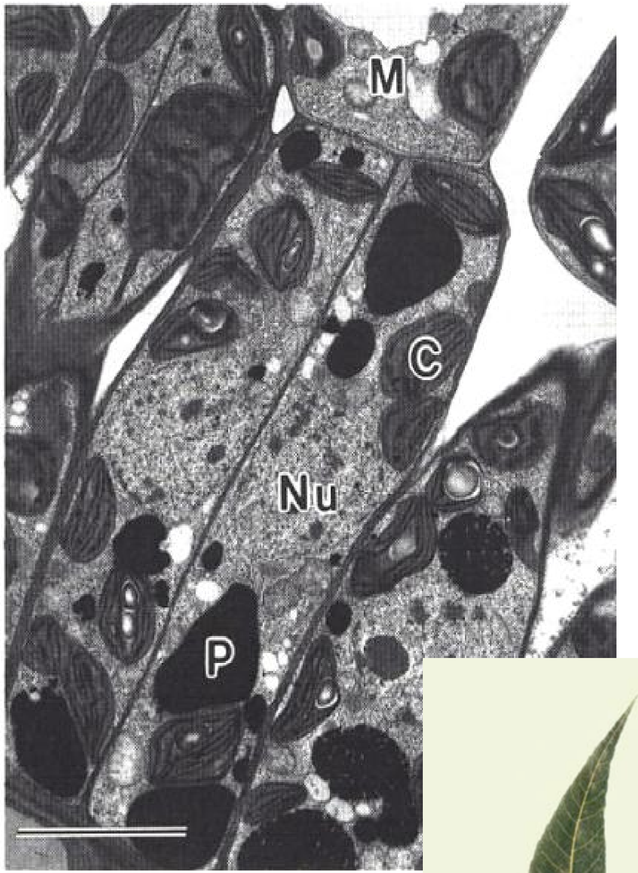


Franceschi VR, Krokene P, Christiansen E, Krekling T (2005) Anatomical and chemical defenses of conifer bark against bark beetles and other pests. New Phytologist 167 (2):353-375

Hickory cultivar susceptible
to *Cladosporium caryigenum*



Hickory cultivar resistant
to *Cladosporium caryigenum*



(Blanchette, R.A. & Biggs, A.R. (Eds.) (1992) *Defense Mechanisms of Woody Plant Against Fungi.* Springer-Verlag, Berlin)

Induced Mechanical Barriers (Wound Healing)

è Leaves: e.g. abscission zones, corky layers

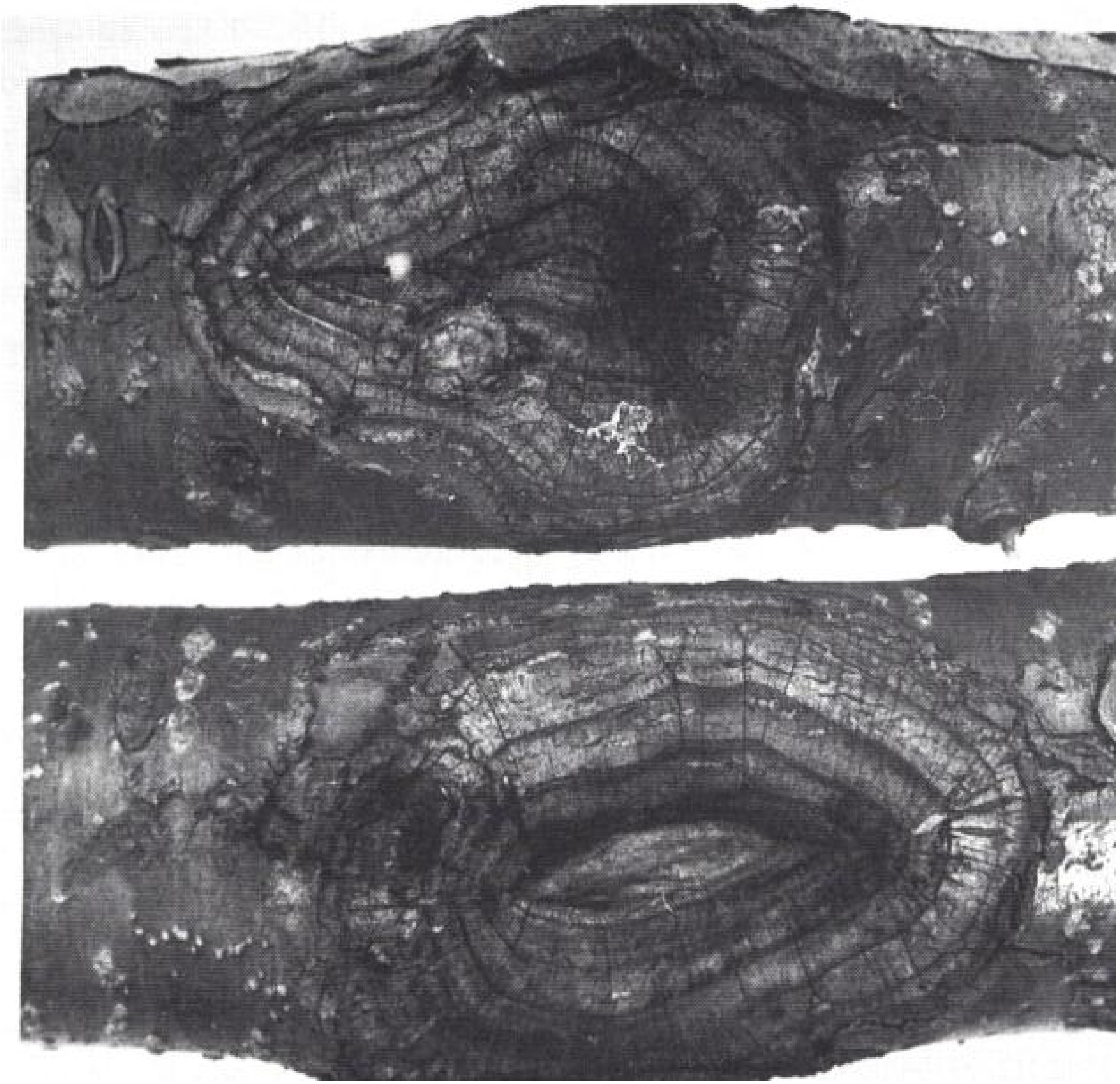
è Stem and roots: corky layers, new periderms

LEAVES

Phyllosticta leaf spot of red maple

Alternaria leaf blight of Zinnia

STEMS



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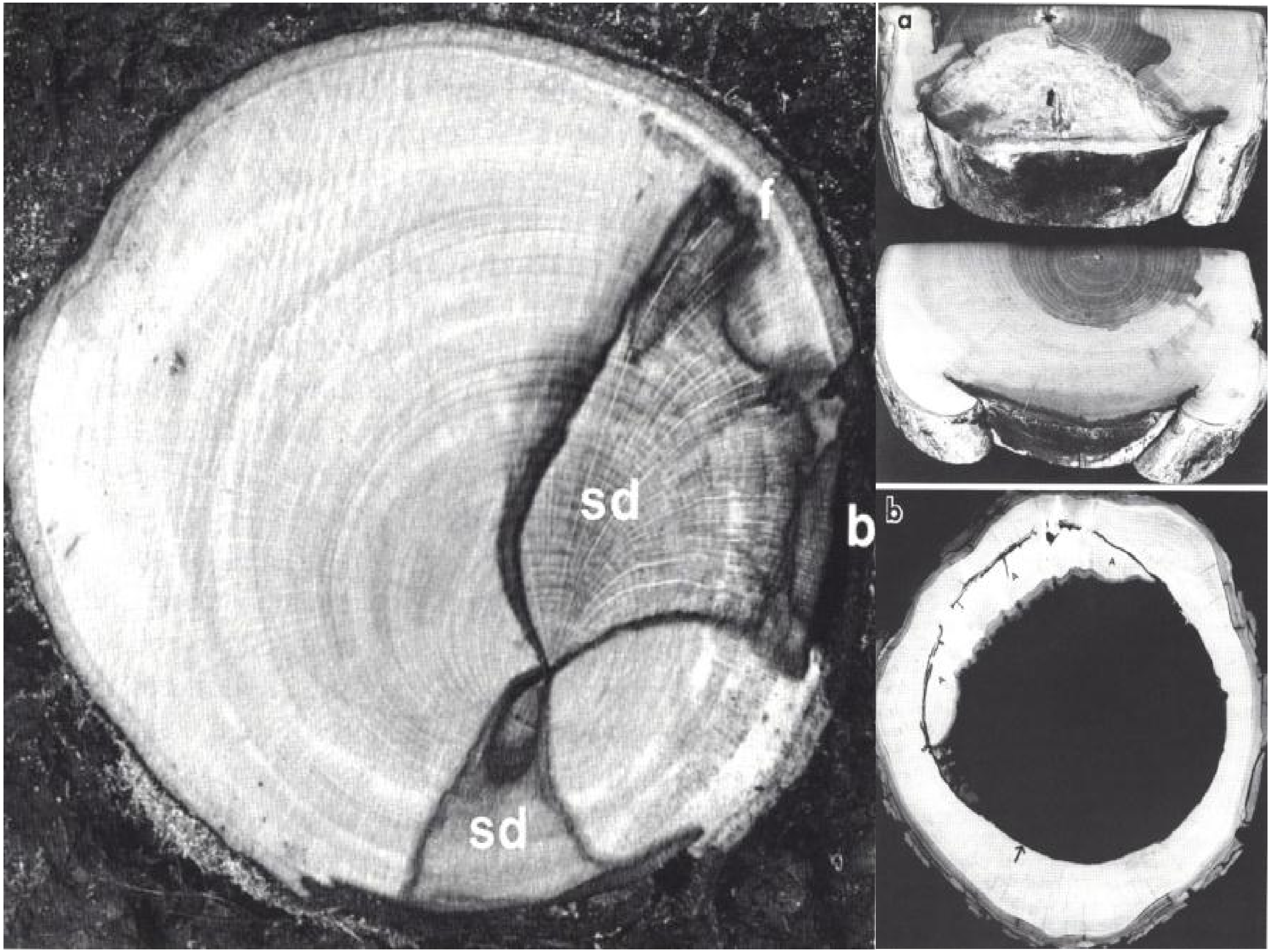
STEMS



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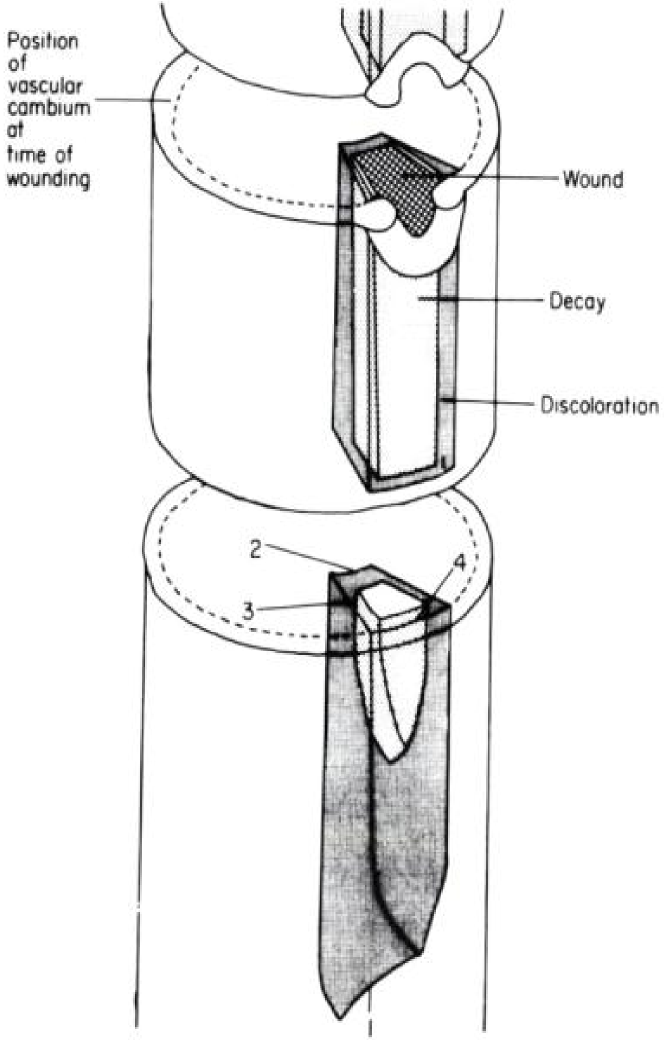
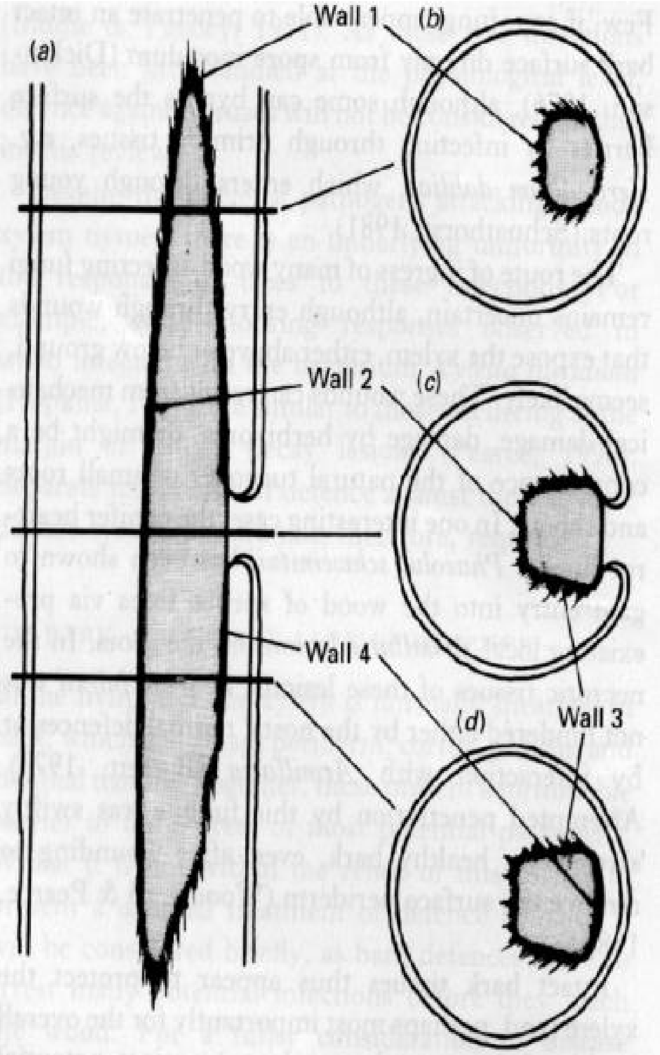
STEMS





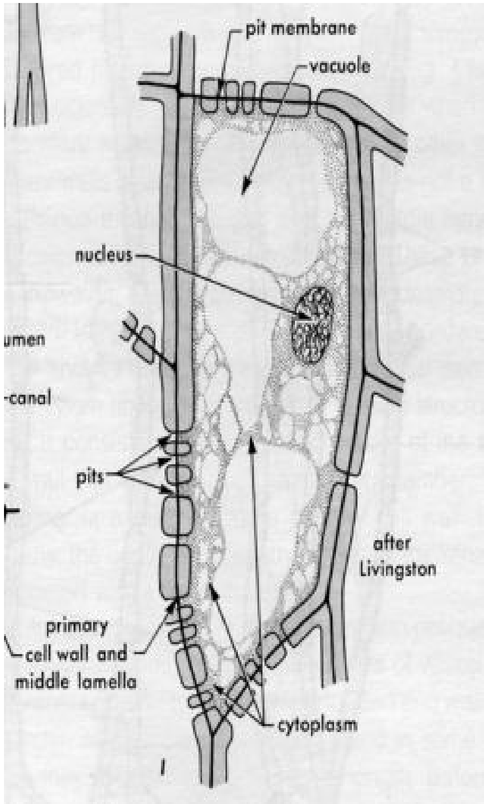
(Blanchette, R.A. & Biggs, A.R. (Eds.) (1992) *Defense Mechanisms of Woody Plant Against Fungi.* Springer-Verlag, Berlin)

(Pearce, R.B. (1996) Tansley Review No. 87. Antimicrobial defences in the wood of living trees. *New Phytologist* 132, 203-233)

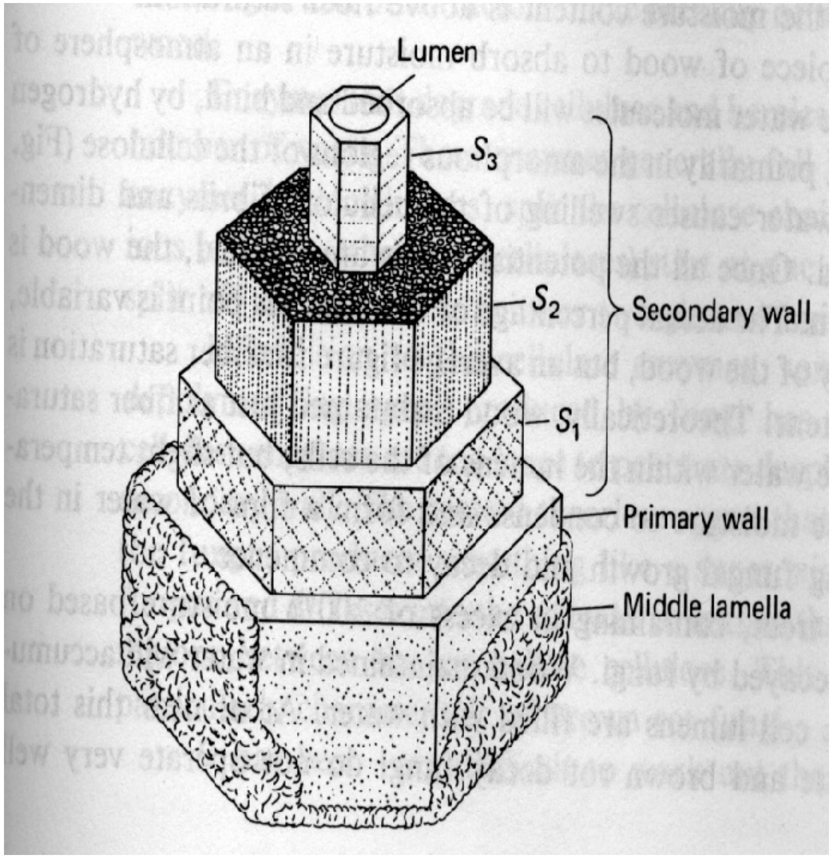
Induced Chemical Barriers

- All tissues: increased amounts of preformed antibiotic chemicals, new antibiotic chemicals, chemical changes of the cell walls, etc.
- One of the main induced defense responses is increased lignification

Soluble chemicals



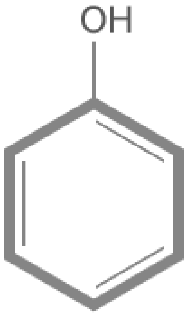
Cell wall-bound chemicals, e.g. lignin



Chemical Plant Defense

- Components of defense
  - Constitutive (phytoanticipins) and induced (phytoalexins)
- Against herbivory (e.g. insects)
  - Typically relies on constitutive phenolics
- Against infection (e.g. fungal pathogens)
  - Typically relies on induced phenolics
  - Intensity and, more importantly, speed of accumulation often correlated to resistance

Phenolics



Specialized (secondary) metabolites

- Low molecular weight
- Derivatives of phenol

Chemical properties

- Organic compounds with structural heterogeneity
- Benzene ring with one or more hydroxyl group
- Absorb strongly in UV and / or visible light
- Hydroxyl group allows for different reactions

“Raison d’être” of phenolics

Synthesis selected for throughout evolution

- Plant defense
- Protection from environmental exposure, e.g. UV radiation
- Physical support
- Insect/animal attractants
- Signaling
- Allelopathy

Case Study: Sudden Oak Death

New Approaches to Assess Coast Live Oak Resistance Before Infection by the Invasive Pathogen *Phytophthora ramorum*

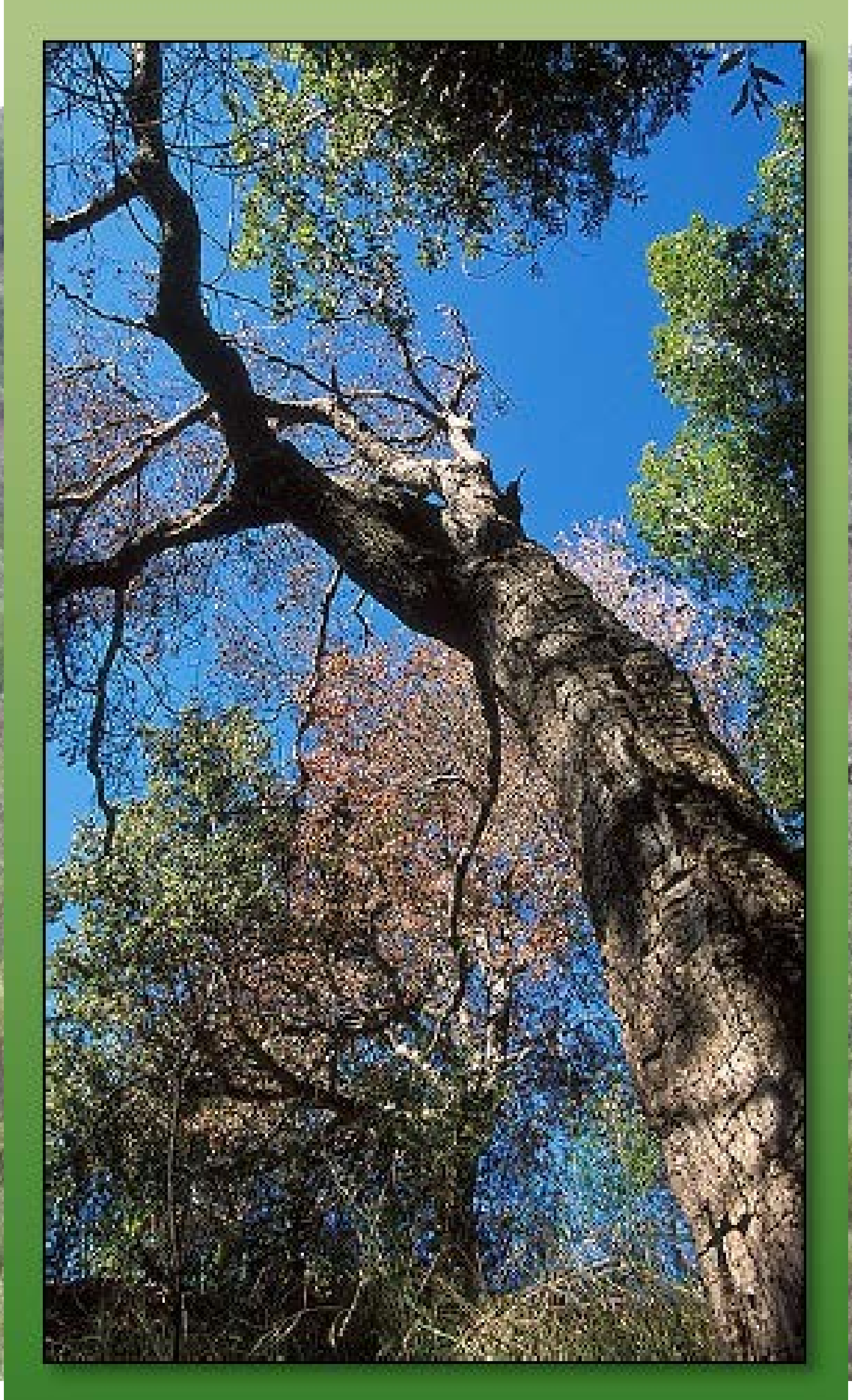
- Collaborators
  - Anna Conrad, L o – Ohio State
  - Brice McPherso ornia, Berkeley
  - Sylvia Mori – Fo

- Statistical guidance
  - Larry Madden –

Anna O. Conrad

Sudden Oak Death

- First documented in Marin County, CA in the mid-90’s.
- Associated with die-off of tanoak and coast live oak (CLO).
- Attacks by ambrosia and bark beetles and presence of fungi (e.g. *Annulohypoxylon thouarsianum* and *Trametes versicolor*) were associated with dying trees.
- *Phytophthora ramorum* identified as the causal agent in 2002.

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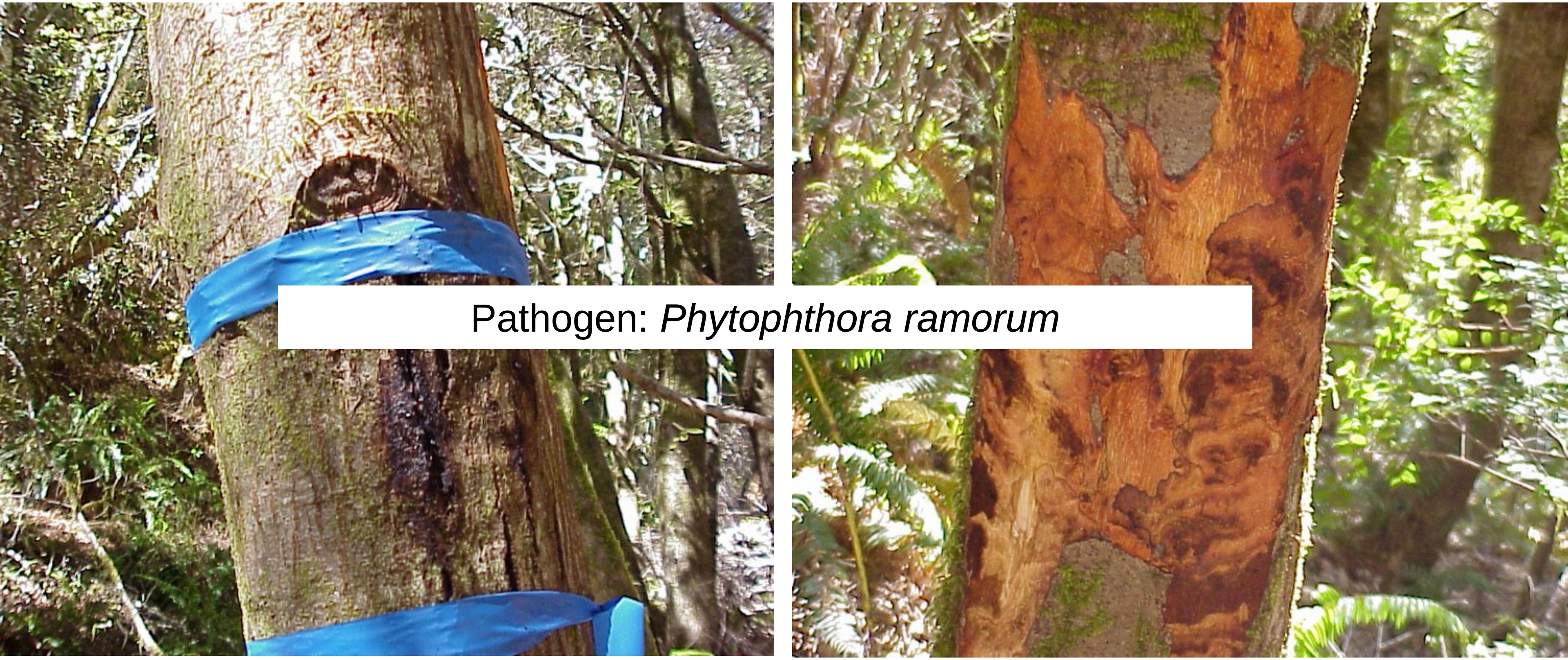
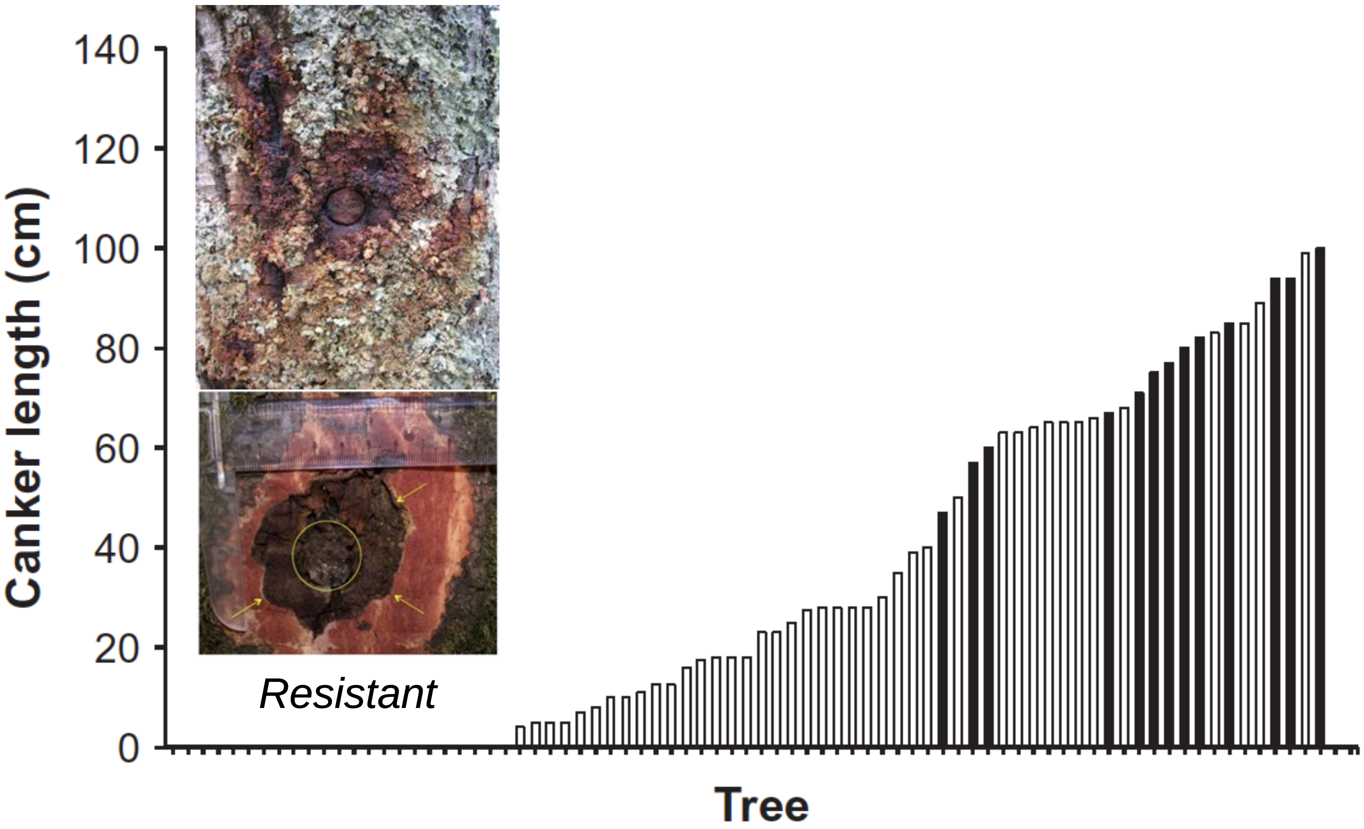


Pathogen: *Phytophthora ramorum*

COAST LIVE OAK SUSCEPTIBILITY VARIES



Resistant

Susceptible

Fig. 1. Distribution of external canker lengths (cm) of inoculated coast live oaks at both the Nike and Novato sites measured 9 months after inoculation, in April 2003. Trees that did not exhibit observed cankers at this date were recorded as having canker lengths of 0 cm. Solid bars denote the trees that died by July 2009.

CANKER LENGTH PREDICTS RESISTANCE

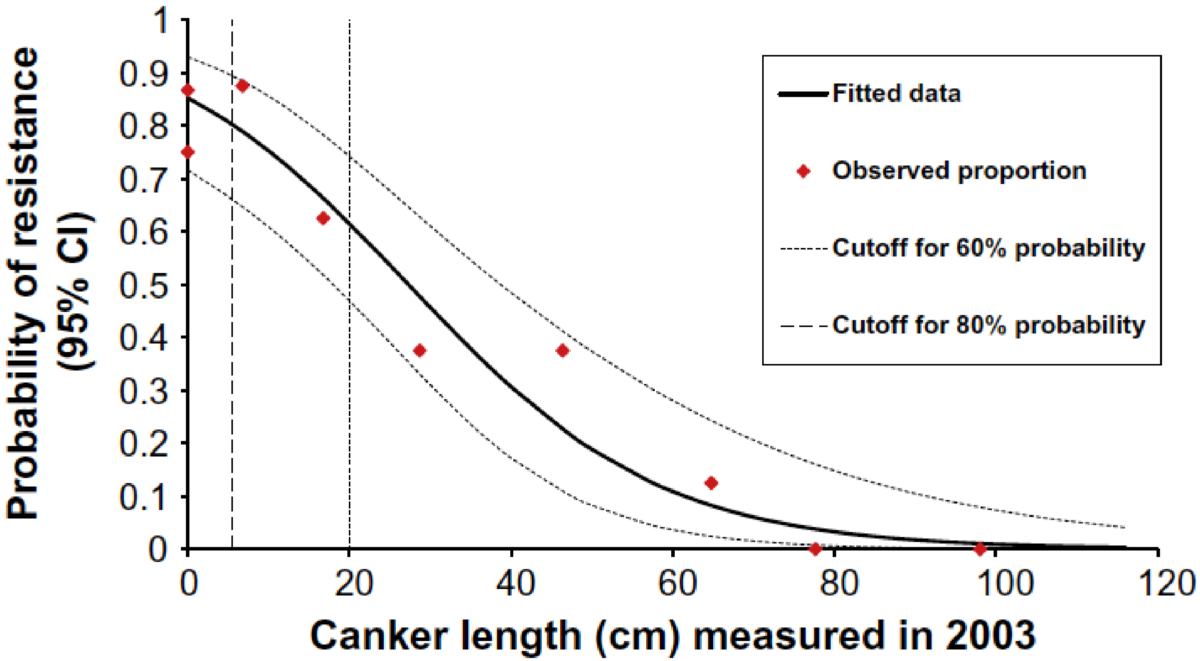
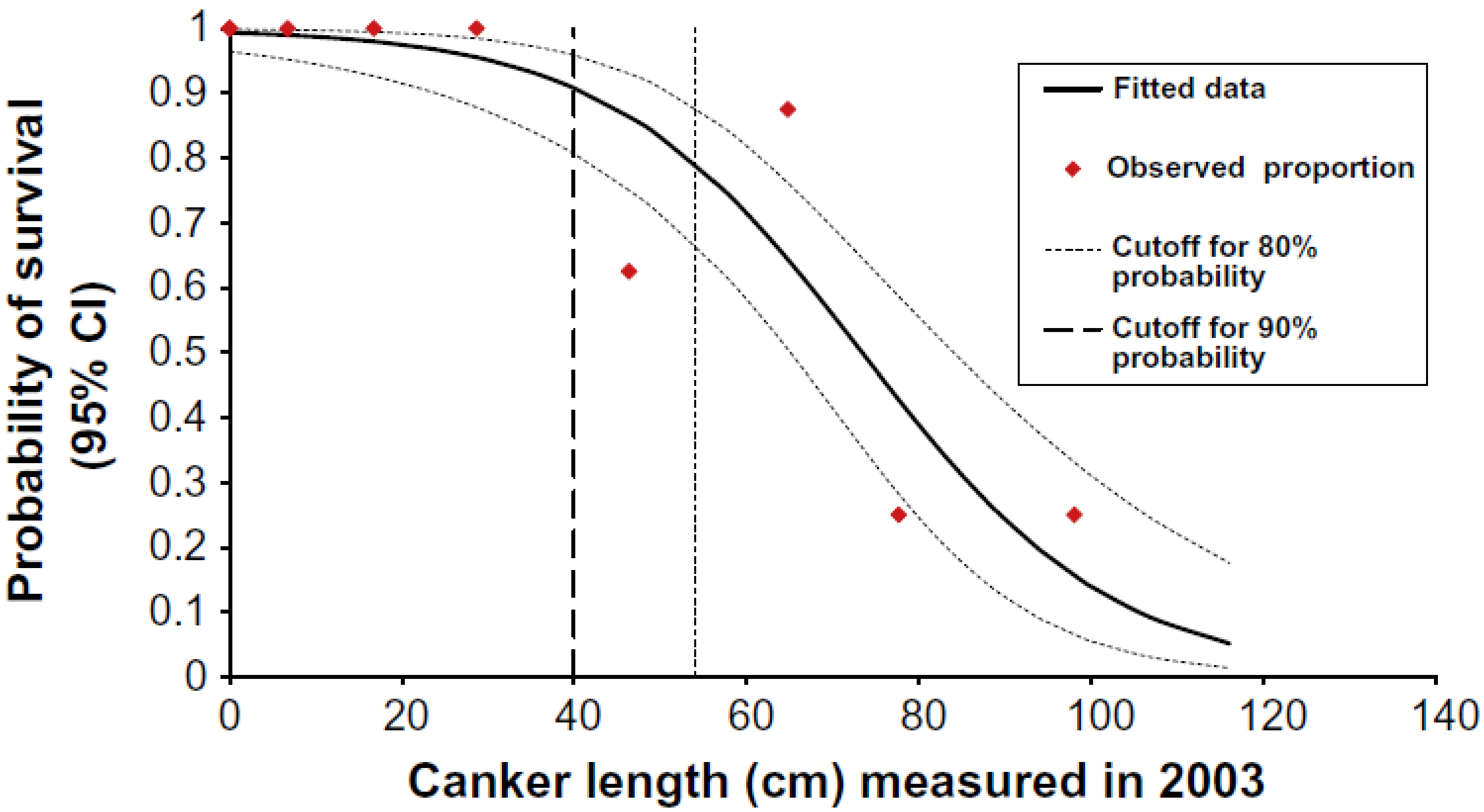


Fig. 4. Estimated probability that an infected coast live oak will (a) survive infection or (b) be resistant (either PR or IR) 7 year after inoculation, as a function of external canker length measured 9 months after inoculation. Observed proportions represent mean canker lengths, binned into discrete intervals for graphical representation. The dashed vertical lines in (a) show threshold canker lengths for probabilities of survival of 80% and 90%, whereas dashed vertical lines in (b) show threshold canker lengths for probabilities of resistance of 60% and 80%.

CANKER LENGTH PREDICTS SURVIVAL

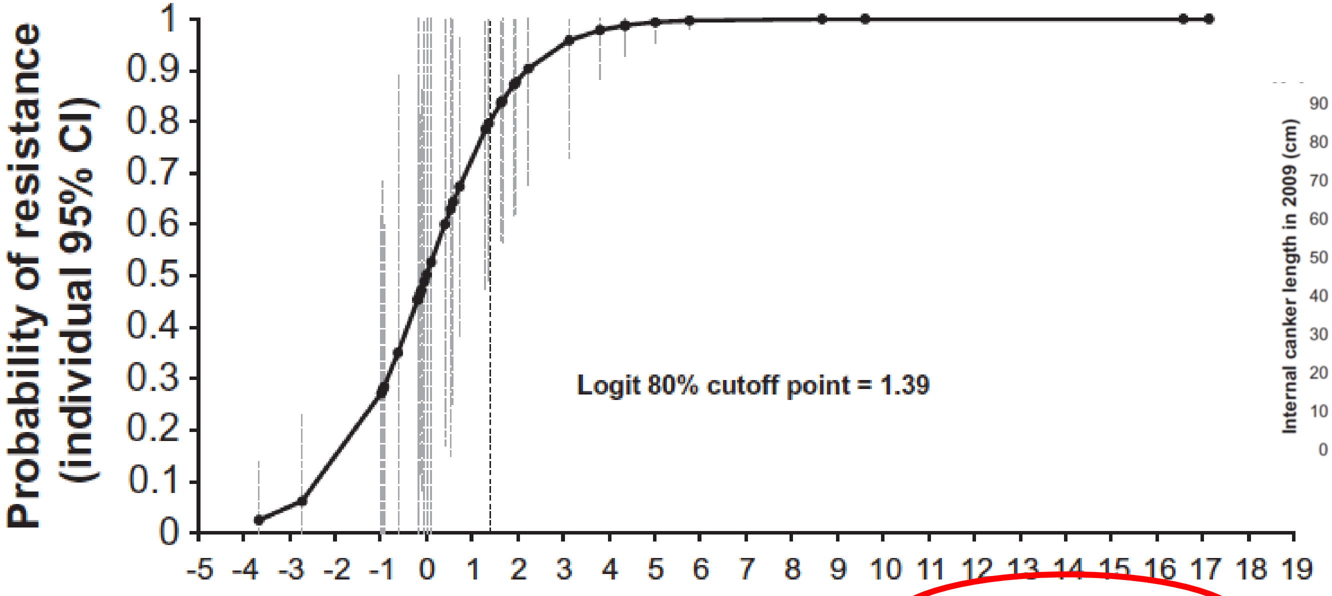


External canker length measured 9 months following inoculation can be used to predict coast live oak survival 7 years following inoculation (McPherson et al., 2014).

PHYTOCHEMICALS AND DEFENSE

- Ellagic acid and a tyrosol derivative are associated with resistant CLO (Nagle et al., 2011)
- Concentrations of 4 putative phenolic biomarkers of resistance were identified from asymptomatic tissue of already infected CLO (McPherson et al., 2014)
- Ellagic acid and crude methanol extract from CLO phloem tissue both inhibit the growth of *P. ramorum in vitro* (McPherson et al., 2014)

PHENOLIC BIOMARKERS IDENTIFY RESISTANT CLO BEFORE INFECTION



Logit = -12.69 - 29.41[P10] + 18.23[ellagic acid] + 76.61[P16] + 10.14[EA3]

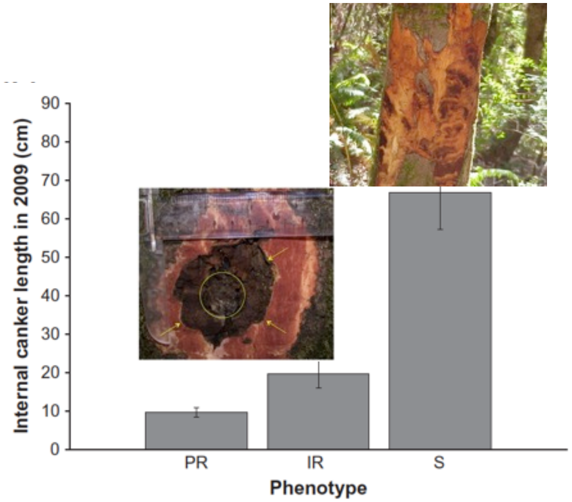
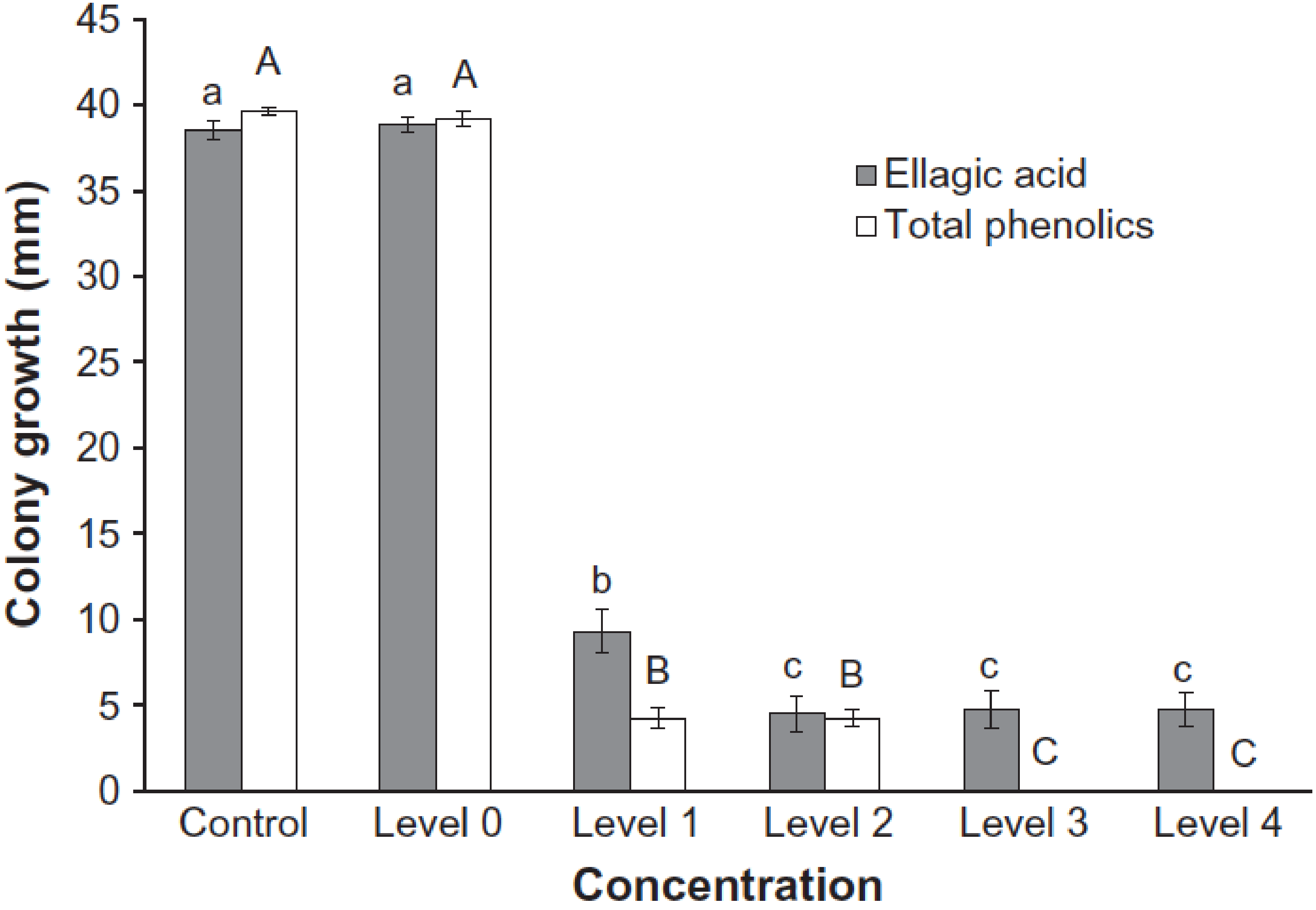


Fig. 5. Relationship between resistance and selected putative phenolic biomarkers of resistance. The plot shows the estimated probability of resistance and logit values. The probability of resistance is greater than 80% when logit values are greater than 1.39 (dashed line).

ELLAGIC ACID HAS STRONG ANTI-*PHYTOPHTHORA RAMORUM* EFFECTS *IN VITRO*



CONCLUSIONS

- Phytochemical biomarkers can predict CLO resistance and, more importantly, survival
- First example of biomarkers of resistance developed for naturally regenerated trees growing under field conditions

Why are we interested in this subject?

è To apply resistance as an IPM tool we need to understand it

è Only once we understand it will we be able to harness it to our advantage, i.e.

- lower costs of pest control
- lower human and environmental impacts of pesticide use

MROSD sprays to battle sick trees

Scientists work to wipe out Sudden Oak Death

By Lily Bixler [lily@hmbreview.com]
Published/Last Modified on Wednesday, Nov 10, 2010 - 11:18:22 am PST

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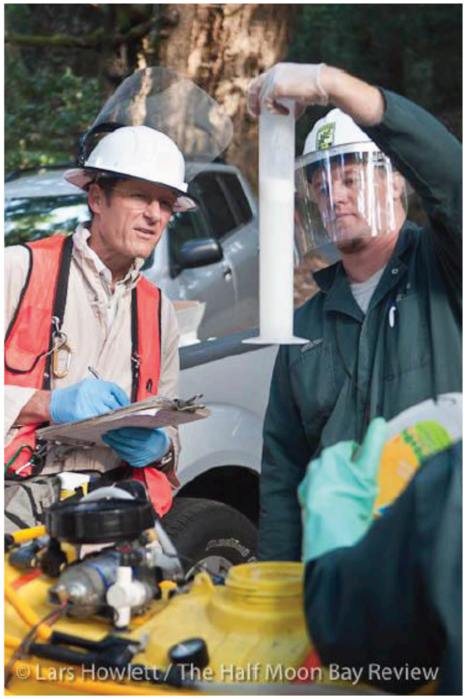
With an orange vest covering a tan one-piece protective suit, scientist Ted Swiecki measures carefully a concoction of chemicals on the bed of his pickup truck. He and a group of tree workers are preparing to spray the fungicide mixture on a grove of at-risk tan oaks in El Corte de Madera Creek Open Space Preserve.

The endeavor is part of an effort to save California trees from a deadly disease.


© Lars Howlett / The Half Moon Bay Review

Dr. Ted Swiecki of Phytosphere Research checks the pressure levels of Agri-Fos fungicide that will be sprayed on 150 tanoaks off Bear Gulch road in La Honda. The Midpeninsula Regional Open Space District is studying methods of prevention against sudden oak death at El Corte de Madera Creek Open Space Preserve.

Sudden oak death has affected more than one million trees in California. It threatens five tree species in 14 of California's counties. The disease gets its name because infected trees quickly deteriorate in mere weeks. The relatively new plant disease is known to leave infected trees brown with bushy tops and with droopy overhanging leaves before ultimately dying. Concentrations of dead trees provide fuel for fires, and a thinned canopy perpetuates the spread of invasive species. The sheer quantity of dead oaks and tan oaks has proven overwhelming for scientists, property managers and park goers.

"Oaks do die, but when so many of them die, we know it's not natural," said Midpeninsula Regional Open Space District resource management specialist Cindy Roessler.

To figure out what causes the disease, Midpeninsula Regional Open Space District chose three healthy sites within its preserves with virtually no Sudden oak death infestation to study prevention, Roessler explained.

The district, in conjunction with California Oak Mortality Task Force and several West Coast universities, is working on three different studies to examine the tree disease. One is geared toward finding genetic resistance to the disease. Another determines how the pathogen actually spreads inside trees and whether some oak species are more susceptible than others.

Swiecki and his research institute, Phytosphere, head up the third study. They spray the trees with a fungicide mixture called Agri-Fos. Also they remove California bay trees, which are the primary transmitters of the disease because they host the pathogen on their leaves.

On Thursday morning Swiecki sprayed about 130 tan oaks, in one of the three study areas. This was the fourth spraying in an experiment that started in 2008 and is expected to run for roughly 10 years.

Right off Skyline Boulevard, the stand in El Corte de Madera Open Space Preserve has no bay trees, but Swiecki and men from a tree services group venture out into the forest on a four-wheeler. One of the men, dressed in full protective gear, uses a long spraying rod to reach far up on the trunk of the tan oaks. He sprays the knotty oak bark as another worker times the exposure to the chemical as part of the experiment. Though not a "miracle juice or anything," Swiecki explained the natural fungicide builds up the tree's resistance to sudden oak death.

Last week's spraying focuses only on tan oaks. Other tree species with the disease include California black oak, coastal live oak, Shreve's oaks and canyon live oaks. Blue oak and valley oak trees don't get the disease.

"Tan oak is widely spread but generally under recognized," said Swiecki, who added that tan oaks generally grew in to replace logging clearings.

San Mateo County is one of the places sudden oak death started. Scientists are therefore using local preserves as a starting point to find a remedy. "We're looking to see if this is a useful management tool," Swiecki said.

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Like

Bay trees being removed to save oak trees

Crew to fell roughly 250 bay trees in Santa Clara, San Mateo counties

SHARE

A crew started removing roughly 250 bay trees in several open space lands in San Mateo and Santa Clara counties Monday, July 11, to stop the spread of a plant pathogen that kills oak tree species.

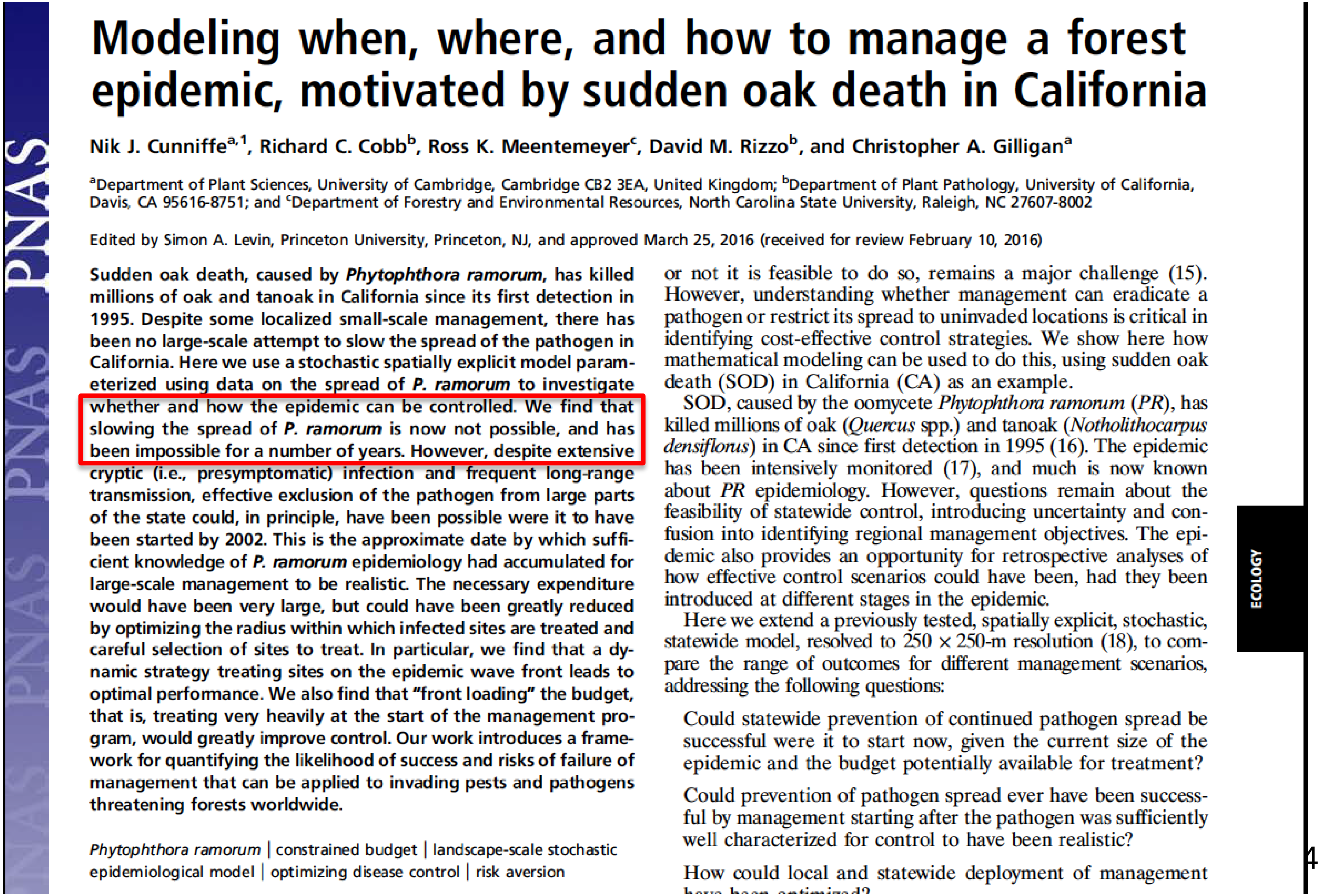
The pathogen, Phytophthora Ramorum, causes certain species of oak trees to die from sudden oak death. In an effort to understand and eradicate the disease, the Midpeninsula Regional Open Space District has enlisted the help of a work crew with the California Conservation Corps to remove nearly 250 bay trees within 15 feet of 49 healthy oak trees.

According to the open space district, research has shown that removing bay trees within 15 feet of oak trees significantly lowers their chances of becoming infected and dying from sudden oak death. Bay trees are a host species that can transmit the pathogen.

The tree removal started July 11 and will continue through July 28. The crew will work this week at Monte Bello and Los Trancos open space preserves in Santa Clara County and Long Ridge Open Space Preserve in San Mateo County. Next week, the crew will tackle the trees at the Long Ridge and Russian Ridge preserves in San Mateo County. The week of July 25, they will work at Saratoga Gap in Santa Clara County and Skyline Ridge in San Mateo County.

The project, which was funded by a Proposition 84 grant, is meant to help prevent the buildup of dead trees and protect open space lands.

— Bay City News Service



Modeling when, where, and how to manage a forest epidemic, motivated by sudden oak death in California

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Edited by Simon A. Levin, Princeton University, Princeton, NJ, and approved March 25, 2016 (received for review February 10, 2016)

Sudden oak death, caused by *Phytophthora ramorum*, has killed millions of oak and tanoak in California since its first detection in 1995. Despite some localized small-scale management, there has been no large-scale attempt to slow the spread of the pathogen in California. Here we use a stochastic spatially explicit model parameterized using data on the spread of *P. ramorum* to investigate whether and how the epidemic can be controlled. We find that slowing the spread of *P. ramorum* is now not possible, and has been impossible for a number of years. However, despite extensive cryptic (i.e., presymptomatic) infection and frequent long-range transmission, effective exclusion of the pathogen from large parts of the state could, in principle, have been possible were it to have been started by 2002. This is the approximate date by which sufficient knowledge of *P. ramorum* epidemiology had accumulated for large-scale management to be realistic. The necessary expenditure would have been very large, but could have been greatly reduced by optimizing the radius within which infected sites are treated and careful selection of sites to treat. In particular, we find that a dynamic strategy treating sites on the epidemic wave front leads to optimal performance. We also find that "front loading" the budget, that is, treating very heavily at the start of the management program, would greatly improve control. Our work introduces a framework for quantifying the likelihood of success and risks of failure of management that can be applied to invading pests and pathogens threatening forests worldwide.

Phytophthora ramorum | constrained budget | landscape-scale stochastic epidemiological model | optimizing disease control | risk aversion

or not it is feasible to do so, remains a major challenge (15). However, understanding whether management can eradicate a pathogen or restrict its spread to uninvaded locations is critical in identifying cost-effective control strategies. We show here how mathematical modeling can be used to do this, using sudden oak death (SOD) in California (CA) as an example.

SOD, caused by the oomycete *Phytophthora ramorum* (*PR*), has killed millions of oak (*Quercus* spp.) and tanoak (*Notholithocarpus densiflorus*) in CA since first detection in 1995 (16). The epidemic has been intensively monitored (17), and much is now known about *PR* epidemiology. However, questions remain about the feasibility of statewide control, introducing uncertainty and confusion into identifying regional management objectives. The epidemic also provides an opportunity for retrospective analyses of how effective control scenarios could have been, had they been introduced at different stages in the epidemic.

Here we extend a previously tested, spatially explicit, stochastic, statewide model, resolved to 250 × 250-m resolution (18), to compare the range of outcomes for different management scenarios, addressing the following questions:

> Could statewide prevention of continued pathogen spread be successful were it to start now, given the current size of the epidemic and the budget potentially available for treatment?
>
> Could prevention of pathogen spread ever have been successful by management starting after the pathogen was sufficiently well characterized for control to have been realistic?
>
> How could local and statewide deployment of management

CONCLUSIONS

- Studying plant chemistry associated with resistance can inform management
- Such methods can be used on many other systems
    - We are in the process of applying these approaches to Austrian pine resistance to Diplodia tip blight, as well as other key diseases like white pine blister rust in whitebark pine, and ash dieback in Europe

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plantpath.osu.edu/bonello