Housekeeping

- Attendance is expected, if you cannot attend a lecture please let me know
- Powerpoints, Zoom recordings and all readings are posted on the class website
- Suggested readings are indicated when appropriate in the lower right corner of a slide in a white box
- If the title is not visible in the list, it is in the Forest Pathology Book by Garbelotto and Gonthier

Summary of first lecture

- Definition of disease: decrease in productivity, life span, reproduction and biological associations but there is also an economy-centric definition: loss of economic value. How can this happen?
- Symptoms vs. signs: importance in today's Dixie and Fly Fires.
- Disease vs syndrome.
- Type of pathogens (primary vs secondary vs. latent etc….)
- Causation vs. association: Koch's postulate

Fallen tree caused Dixie Fire, PG&E worker tells 911 operator

PG&E said in a court filing that it doesn't know why a Douglas fir fell on its power line in the area where the Dixie Fire started, but an arborist reported seeing signs of rot on one of the tree's roots.

Reduce Ignitions: Vegetation management along power lines

- Utility companies need to mitigate risk of tree failures on lines and infrastructure (4 feet and 12 feet clearance areas)

  - Problems

  – Sheer scale of task; you have to accept **you can only reduce risk**, not zero it

  – Surveys designed based on statistical information, but **key indicators** of tree failure may be missing

  – **Surveys very long,** affected by operator bias, **pencil and paper operation**, data transcription manual

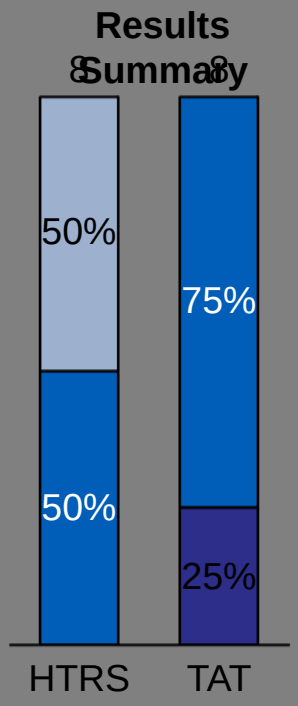
  – Lack of clear prioritization and **many trees are borderline** with actions decided by operator

Update on progress made by PG&E

- New Tree Assessment Tool (TAT):
  - went from 50 to 15 questions, half of which are “yes/no” and result in “abatement vs. no abatement” based on answer. More powerful and faster survey
  - Includes important indicators of hazard trees (SIGNS AND SYMPTOMS)
  - Being digitalized: data collected on a smartphone resulting in real time analysis capability, and providing evidence that survey was performed properly
  - Increases abatement and eliminated borderline trees, now all trees are ABATE or DON’T ABATE
  - In Fall 2020 working on a further prioritization effort based on statistical relevance

TAT Sample field testing

Field Testing Tree Scores, n = 8



HTRS Assessment

Likelihood of failure	0 (Very Low)	1 (Low)	2 (Mod)	3 (High)	4 (Very High)
7 (Very High)					
6 (High)					
5 (High)					
4 (Mod)					
3 (Mod)			E		
1, 2 (Low)		B2, C	A2, A, D		
0 (Very Low)			C2, B		

Severity of impact

TAT Assessment

Tree Health Score	1 (Low)	2 (Mod)	3 (High)	4 (Very High)
5	A			
4				
3				E
2			A2	
1 (Very low)	C2, C, B	B2		D

Tree Environment Score

Auto-abate from major wounds on trunk

Poor canopy health, codominance, highest tendency to cause ignitions, creek

- **HTRS tool has multiple borderline trees** which will rely heavily on individual, subjective decisions
- **HTRS does not directly recommend abatement** of hazard tress based on this sample

- **TAT tool has no borderline trees** as it provides a clear abatement recommendation
- **All abatement decisions are clear;** TAT more likely to abate hazard trees based on this sample

DISEASE

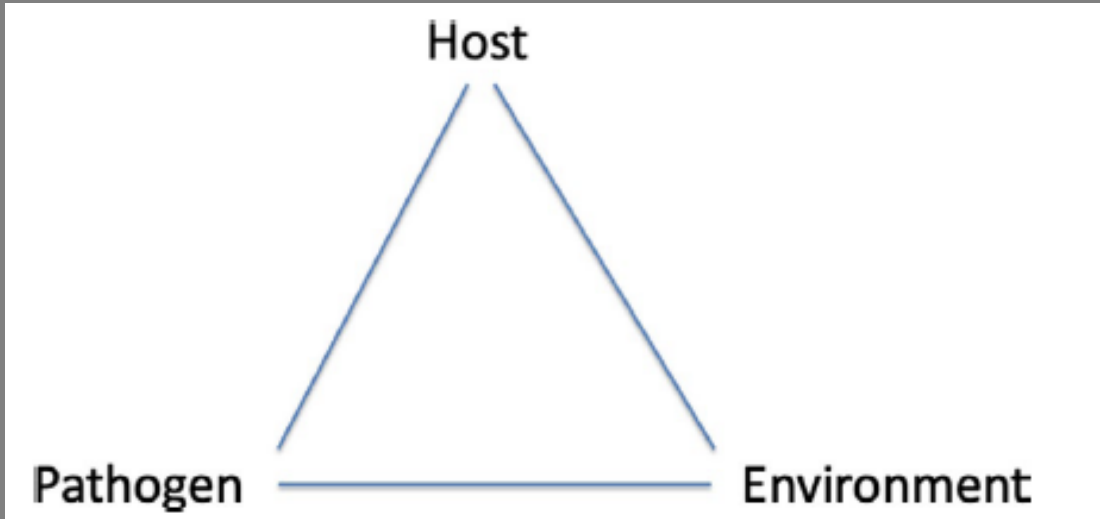
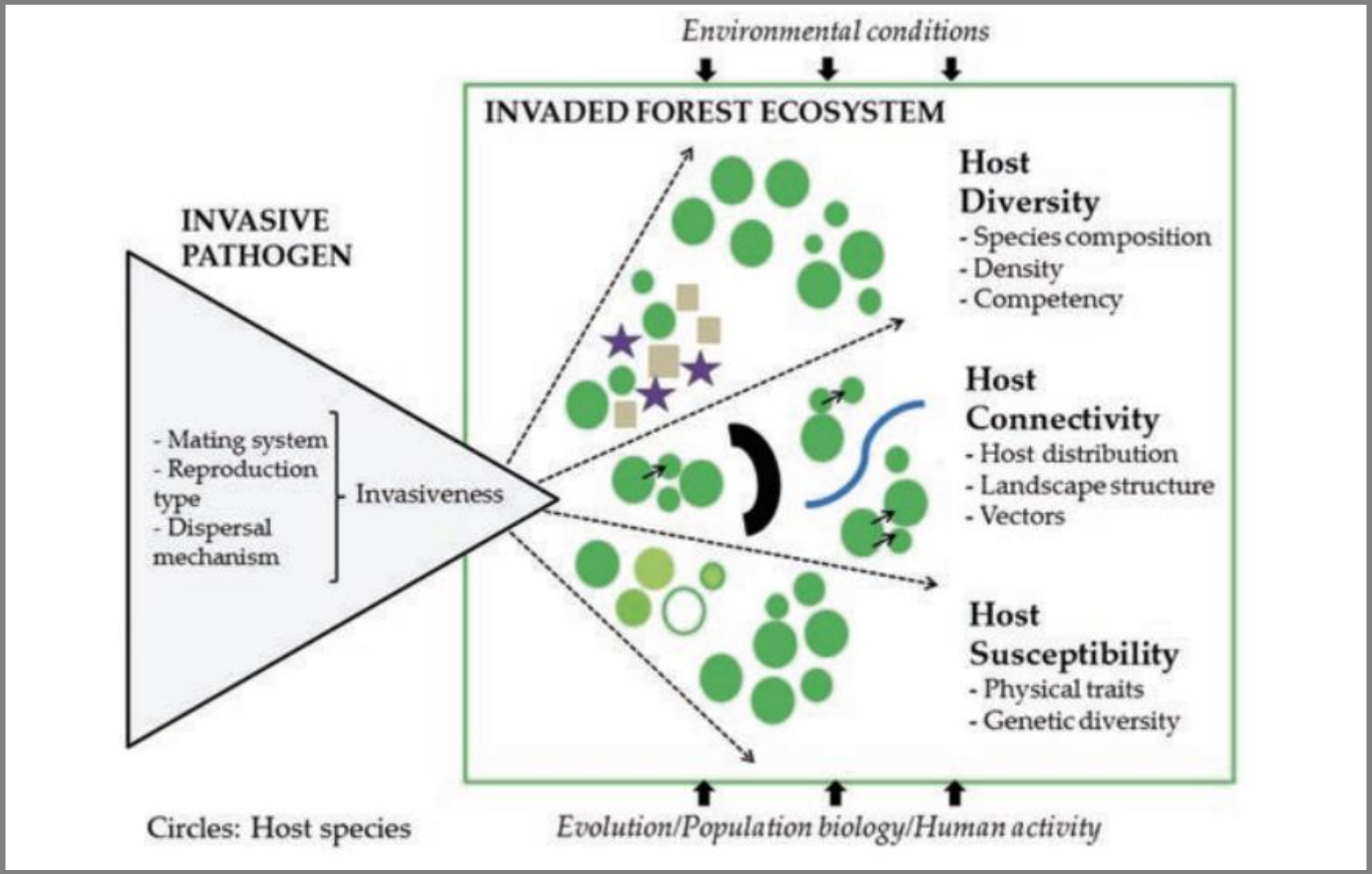


FIGURE 1 The plant disease triangle

Additions to the triangle:

- Time
- Humans
- Disturbances



Review

Effects of Host Variability on the Spread of Invasive Forest Diseases

Figure 1. Local, regional, and continental-scale factors governing the spread and impact of invasive forest pathogens *post-introduction and establishment* are affected by three main factors: host diversity, host connectivity, and host susceptibility. Following arrival and establishment, invasiveness is inherently affected by organismal traits (mating system, reproduction type, and dispersal mechanisms). ***Host diversity*** is mainly affected by plant species richness (density/composition) creating a dilution effect of pathogen impacts on the ecosystem. Non-hosts, competent hosts, and less competent hosts will have variable effects on their ability to intercept inoculum and subsequently reduce pathogen spread. ***Host connectivity*** is largely influenced by the distribution of available host species; more or less aggregated. Spatial heterogeneity of hosts becomes important for vector-induced pathogens. Variations in landscape structure (topography, natural geographic/environmental barriers, forest fragmentation) will largely influence spread dynamics on the landscape level. ***Host susceptibility*** to invasive pathogens is influenced by physical traits (size, age, morphology), the random presence of other (potentially antagonistic) organisms, environmental and site factors, and host genetic background. Intraspecific genetic diversity (mixtures of host genotypes) offers the best insurance against invasive pathogens through a dilution effect on inoculum production/deposition and the likelihood that some hosts will possess effective mechanisms to resist or minimize damage caused by invasives. Evolutionary and environmental factors, as well as continuous pressures caused by human activity will influence spread dynamics over time. Understanding how host variability is affected by ***host diversity, connectivity***, and ***susceptibility*** will improve our ability to predict disease spread on the landscape and potential consequences to ecosystem services.

Review

Effects of Host Variability on the Spread of Invasive Forest Diseases

Hosts

- Non hosts: lack of host-pathogen compatibility
- Non-competent vs. competent: not supporting pathogen sporulation vs. supporting sporulation
- Transmissive: supporting high levels of sporulation but usually not overly symptomatic
- Dead-end: usually highly symptomatic and not supporting pathogen sporulation
- Source: high quality host supporting high levels of a pathogen
- Sink: low quality hosts mostly receiving pathogens because of spillover from source hosts
- Reservoir: a secondary host for a pathogen, usually asymptomatic that serves as a source of infection for a primary host
- Superspreader: responsible for a a high level of infections

Our main goal is to be able to predict 1 & 2 D: how much mortality and where

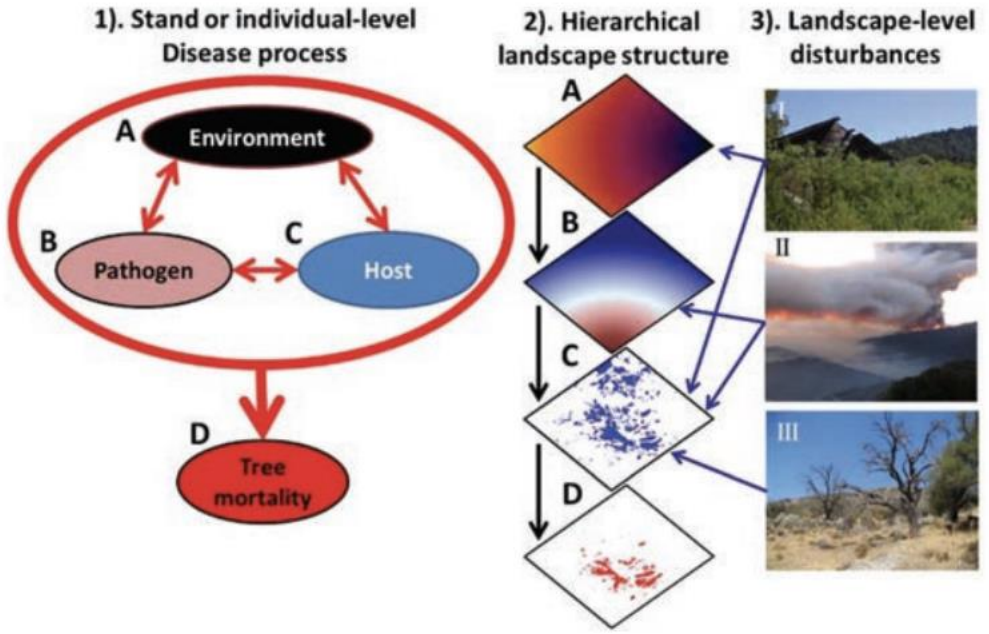


Figure 1. Linkages between forest pathology and landscape ecology with example landscape-level disturbances which can influence disease emergence and impacts. The traditional disease triangle (**1**) factors (A–C) are shown simultaneously in the spatial context of landscape ecology (**2**) along with landscape-level disturbances that are likely to interact with disease (**3**). Environment (1-A and 2-A) is shown on a gradient along with a waveform pathogen invasion process (1-B and 2-B) and realistic spatial heterogeneity of host distribution (1-C and 2-C) and mortality (1-D and 2-D). Examples of interactive disturbances are shown with arrows indicating the components of landscape structure and the disease triangle that are impacted: Invasive plants competitively inhibit forest host reestablishment following land abandonment with impacts to environmental conditions and host distribution (I—*Genista monspessulana* invasion of an old vineyard), fire can alter host and pathogen distribution (II—Soberanes Fire 2016, Big Sur—Photo credit K. Frangioso), and emergent insect outbreak-caused mortality of host populations (III—*Agrilus coxalis* mortality of coast live oak).

Review

Tree Diseases as a Cause and Consequence of Interacting Forest Disturbances

FACTORING TIME

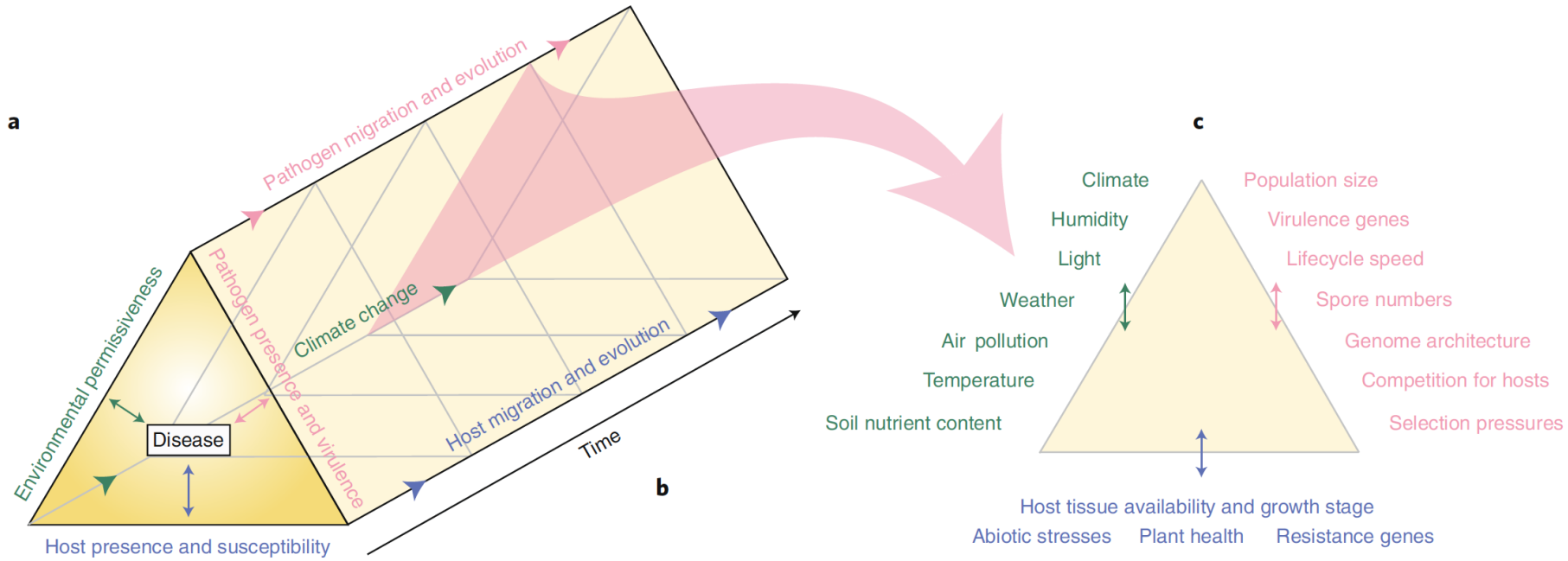


Fig. 4 | Parameterizing the disease triangle in evolutionary time. a, Disease occurrence requires the interaction of a virulent pathogen with a susceptible host under permissive conditions, forming the 'disease triangle'. **b**, As the parameters that underlie susceptibility, virulence and permissiveness change over time, it is helpful to reframe the triangle as a triangular prism, with time on the fourth axis. **c**, At any given moment in time, a triangular slice can be recovered and examined to determine the parameters on each face of the disease triangle. Parameters important for host virulence include genome and lifecycle traits that contribute to a pathogen's evolutionary potential over time, and thus to virulence in a given moment. On the host side, host tissue availability, architecture and developmental stage, overall plant health and nutrition, and R-gene complements are all important. Finally, environmental parameters determining disease outcomes include soil and air quality, weather factors, light, temperature and water availability. All these parameters interact with the features of agroecosystems that promote disease, as well as any control measures taken (see Fig. 1).

Threats to global food security from emerging fungal and oomycete crop pathogens

HOW PATHOGEN VIRULENCE CHANGES THROUGH TIME: IT ACTUALLY INCREASES, BUT RATE OF INCREASE WILL DEPEND ON PATHOGEN ATTRIBUTES SUCH AS EFFECTIVE MATING FREQUENCY IF SEXUAL AND POPULATION SIZE IF ASEXUAL

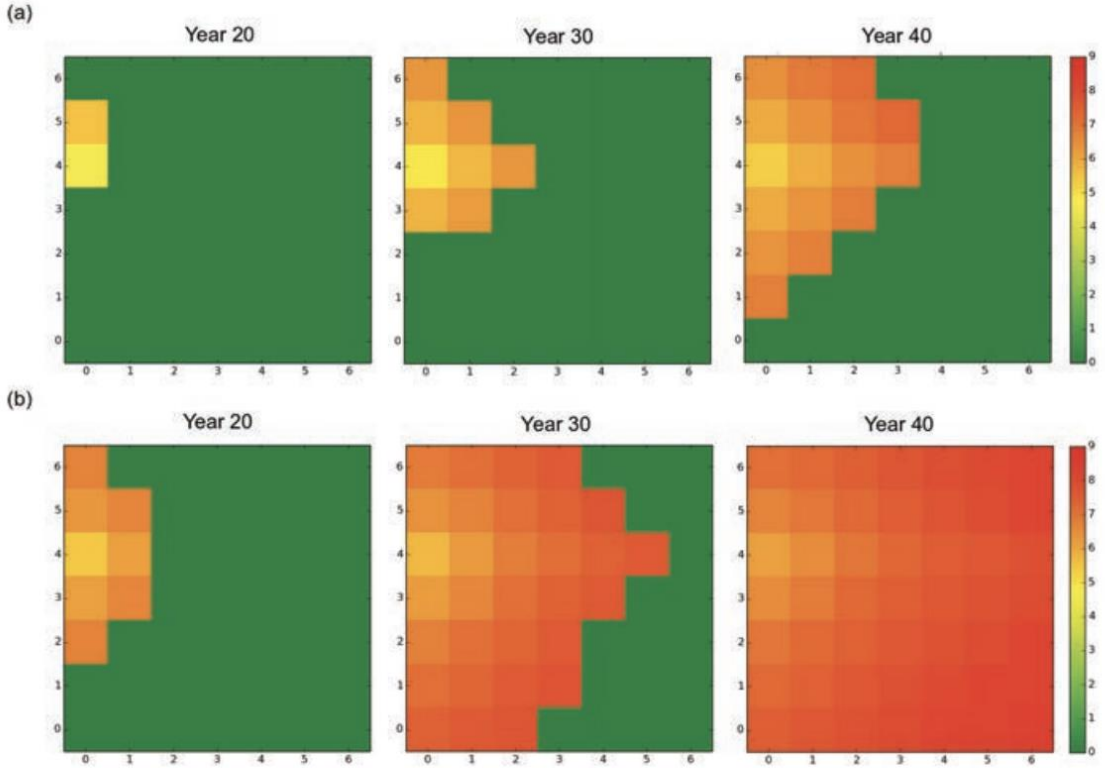


Figure 2. Spatial distribution of the mean virulence level over the forest landscape, at years 20, 30 and 40, for a rotation length of 15 years. Dark green corresponds to healthy stands. The other colors inform about the mean genetic value of the local fungal sub-population in each infected stand. (**a**) mostly clonal fungal pathogen profile *fp1* (clonality rate $c = 0.8$); (**b**) mostly sexual fungal pathogen profile *fp2* ($c = 0.2$). In both cases, the starting genetic variance G_0 was 4.06 (default scenario). The mean fungal genetic values were computed in each stand from the 50 independent replicates simulated.

NO SEX

SEX

Article
Short Rotations in Forest Plantations Accelerate Virulence Evolution in Root-Rot Pathogenic Fungi

DISEASE and HUMANS

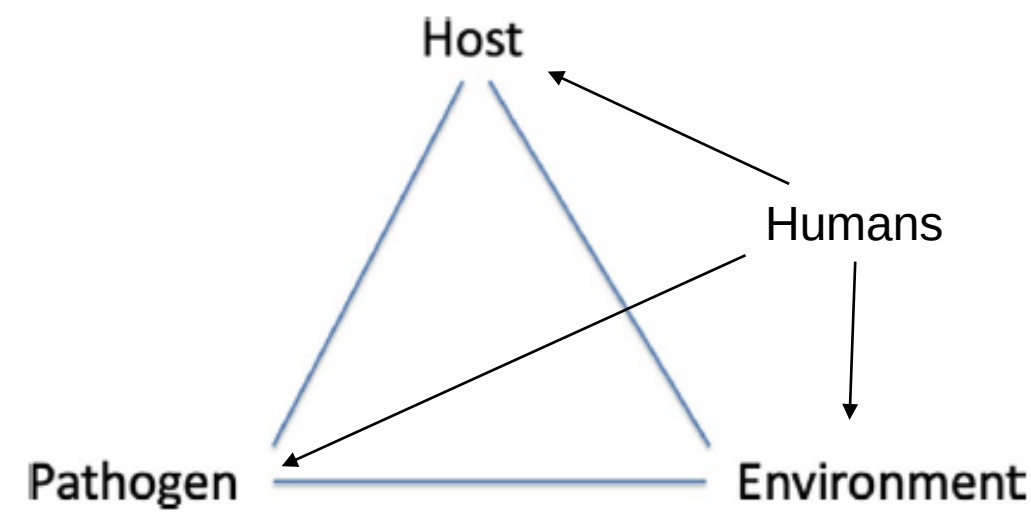


FIGURE 1 The plant disease triangle

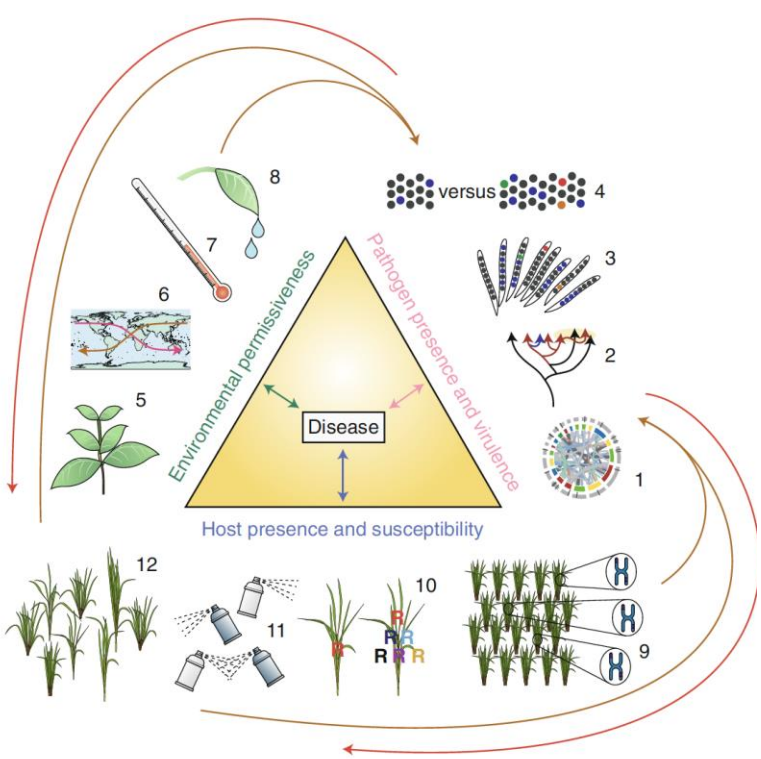


Fig. 1 | Anthropogenic effects that impact the disease triangle. Many factors affect the three facets of the disease triangle, and many of these are themselves influenced by agricultural practices and systems. In particular, pathogen presence and virulence are affected by factors endogenous to the pathogen and variables associated with environmental permissiveness and host susceptibility (see Fig. 4). Endogenous pathogen features include genome structure (1), reproductive systems and capacity for horizontal gene transfer (2), genetic variation within the population (3) and effective population size (4). These combine to determine the pathogen's evolutionary potential. Of the features, population size and variation are most obviously influenced by factors on the other sides of the triangle (yellow arrows). The availability of alternative hosts (5) and the ability to travel (6) by either natural or anthropogenic means are important in determining whether a pathogen is present and how large its population is. Climate factors, such as temperature (7) or humidity (8), can also affect population size, reproduction speed and mutation rates. Meanwhile, large genetically uniform crop monocultures (9) also affect the pathogen's population size and genetic variability. Agricultural practices such as choosing single or pyramids of R genes (10) in crop cultivars, use of single-target-site fungicides or fungicide mixtures (11) or growing cultivar mixes (12) also feed into pathogen evolution. In turn, these factors must be adapted by growers to respond to changing pathogen virulence or resistance to control measures (red arrows). Thus, we see an arms race between man and pathogen.

Threats to global food security from emerging fungal and oomycete crop pathogens

Endogenous factors

1. Genome structure: simpler, leaner, gene duplication, repetitive elements
2. Reproductive system and ability to acquire genes horizontally
3. Genetic variation and effective population size

Environmental or exogenous

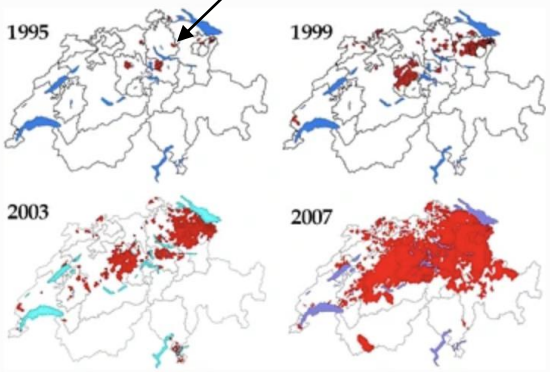
- 1 Alternative hosts
- 2 Ability to travel
- 3 Climate (rainfall, temperature and relative humidity)

Human

- 1 Monocultures or varieties
- 2 Use of single Resistance genes
- 3 Use of single target chemicals

Effect of site-specific human introduction of a pathogen creates a pattern that is otherwise counterintuitive

Pathogen introduced in this region



Development of the fire blight epidemic (due to the bacterium *Erwinia amylovora*) in Switzerland, 1995–2007. The pathogen was introduced into Switzerland from South-West Germany in the 1980s (this explains why the climatically more suitable Ticino has been less affected by fire blight than northern Swiss Cantons). It affects tree and shrub species of the family Rosaceae (e.g. *Malus*, *Pyrus*, *Crataegus*) and is favoured by humid and mild springs, as was the case in 2007, when the epidemic reached unprecedented levels (from Holdenrieder et al. 2008)

With time, effect of introduction location may wane

Impacts of climate change on plant diseases—opinions and trends

Pathogens of natural ecosystems that transition through anthropic system change their phenotype

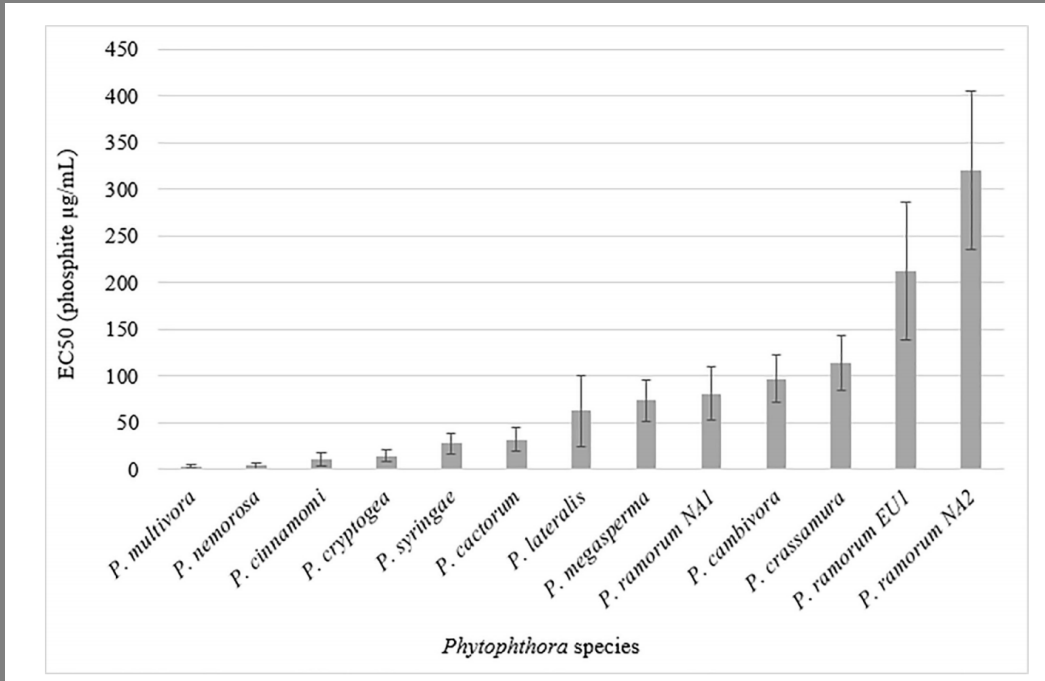


Fig 2. Average EC50 values of the *Phytophthora* species. The *P. ramorum* lineages NA1, NA2 and EU1 are shown separately. Error bars are standard error.

Different species have different tolerance to chemicals but this could be a species-specific trait developed through long evolutionary periods

Evidence for rapid adaptive evolution of tolerance to chemical treatments in *Phytophthora* species and its practical implications

Pathogens of natural ecosystems that transition through anthropic system change their phenotype

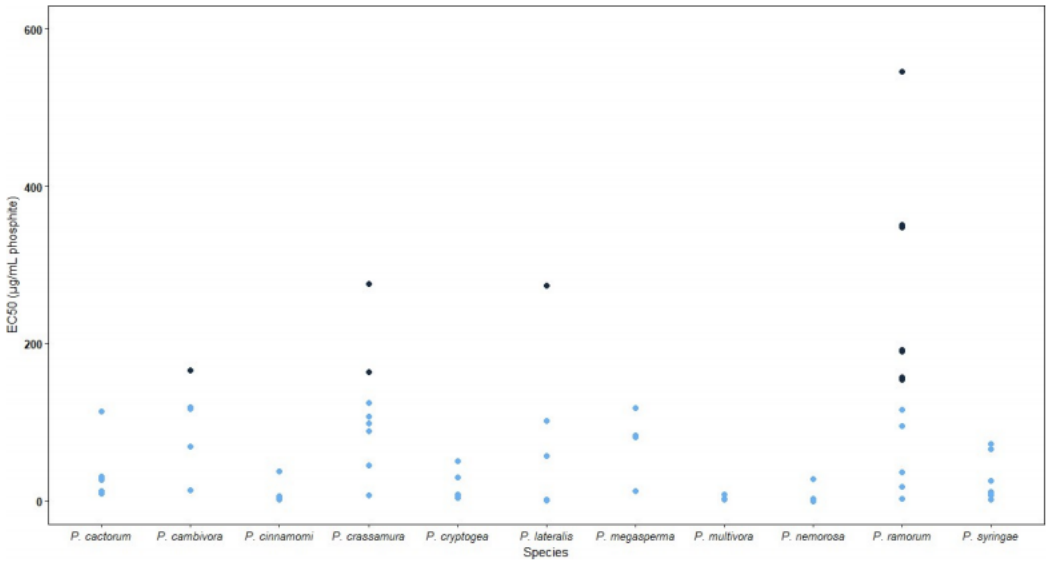


Fig 3. K means analysis showing phosphite sensitive (blue dots) and more phosphite tolerant isolates (black dots). The sensitive isolates (blue dots) had a mean EC50 of 33.8 µg/mL phosphite and the more tolerant isolates (black dots) had a mean EC50 of 251.0 µg/mL phosphite. Isolates from *P. cambivora*, *P. crassamura*, *P. lateralis* and *P. ramorum* clustered in both groups, the isolates of the remaining species all clustered in the more sensitive group 1.

Some species have individuals that are susceptible to a chemical (light blue dots) and individuals that are resistant (dark blue dots). These are differences that must have happened in a shorter time frame. Resistant individuals came from agricultural settings providing strong evidence the emergence of resistance is human- related