EMERGENT DISEASES

- Caused by exotic pathogens introduced from a different region of the world. Either pathogen or pathogen+vectors may be introduced

- Caused by climatic or ecological changes increasing pathogenicity of native microbes
  - Global warming, strongest effects are at the margin of ranges
  - Anthropogenic alteration of the ecosystem
  - Exotic ecosystem and native pathogens: planting of exotics or planting off site

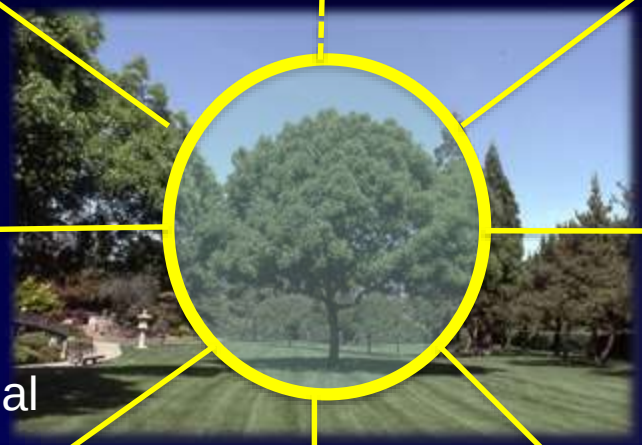
Emergent diseases

- They are normally infectious, not to be confused with syndromes caused by abiotic factors
- They are long lasting or permanent, as opposed to secondary diseases that emerge periodically, for instance during prolonged drought

Stunted growth

Fungal twig dieback
Cryptocline

Fungal branch dieback
Diplodia



Stem cankers & terminal
secondary decay fungi

Twig girdlers *Agrilicus*
Foliar insects, oak pit scale

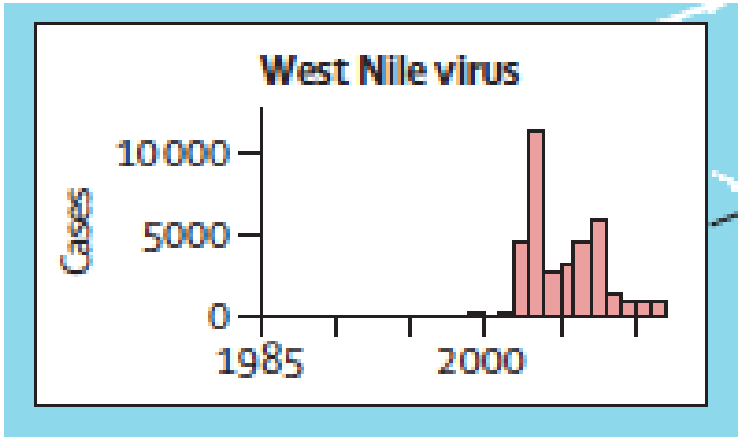
Trunk insects &
associated fungi

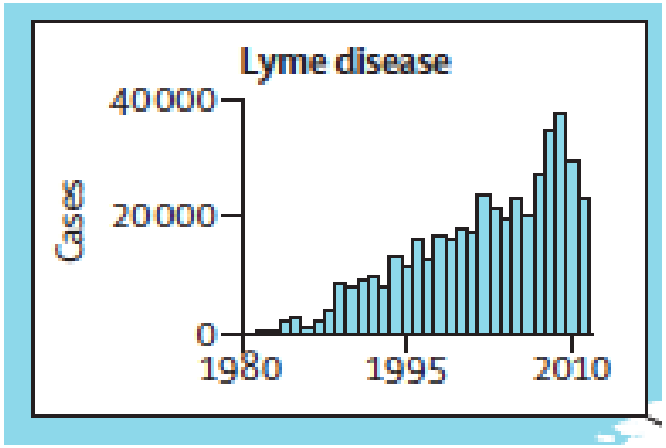
Root rots, *Armillaria*
Soilborne Phytophthoras

Canker rots, *Hymenochaetale*

Emergent Diseases: temporal patterns are generally different between

- EXOTIC AGENTS
  - Rapid outbreaks
  - May cycle down after outbreak
- NATIVE AGENTS
  - Progressive, gradual even if dramatic increase
  - Less likely to cycle down





Bases of Invasion Biology of Pathogens

- **Source**: it can be either a pathogen in its native area, possibly with a cryptic phase, or not (weak pathogen or endophyte). In general, the larger the populations of the pathogen, the easier they will become a source

- **Transport**, survival: related to resting structures, distance, season, substrate (wood, soil, live plants, animal vector)

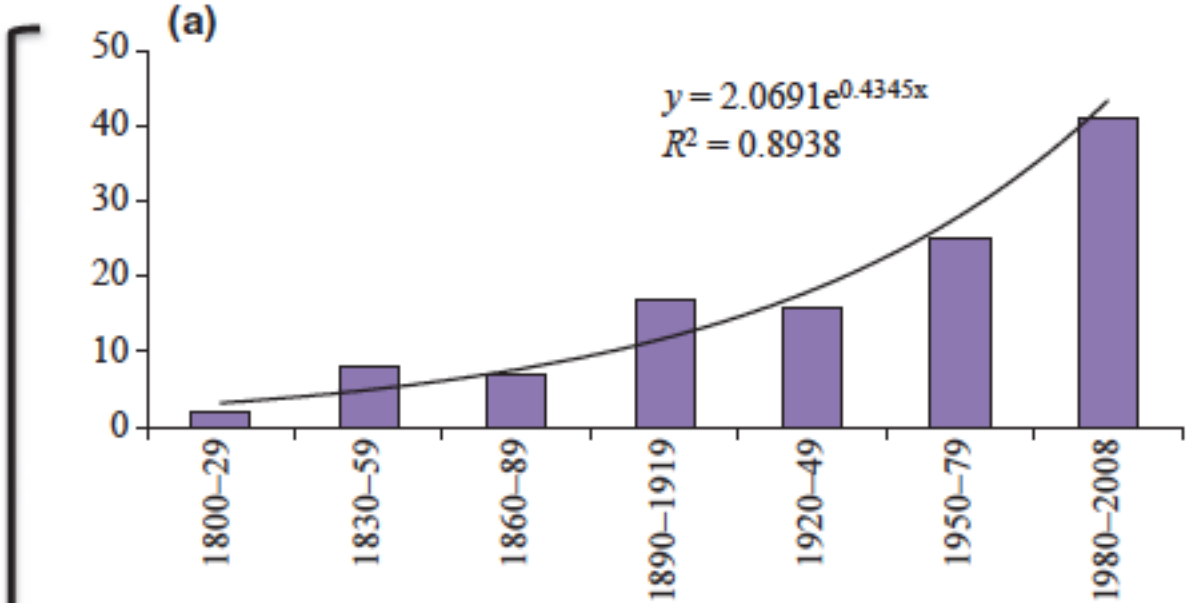
New Phytologist

Biogeographical patterns and determinants of invasion by forest pathogens in Europe

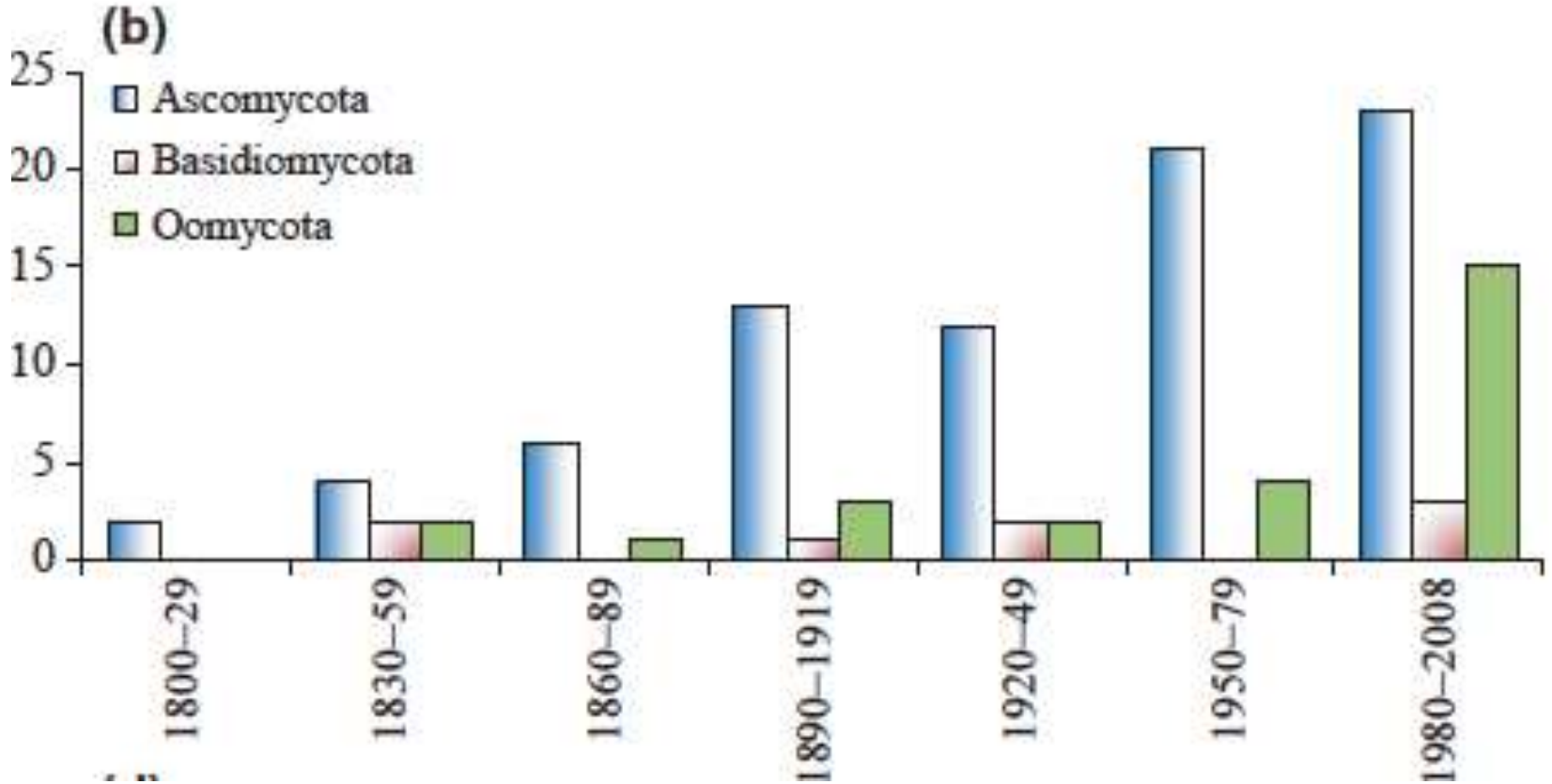
2013

A. Santini[1], L. Ghelardini[1], C.De Pace[2], M. L. Desprez-Loustau[3], P. Capretti[4], A. Chandelier[5], T. Cech[6], D. Chira[7], S. Diamandis[8], T. Gaitniekis[9], J. Hantula[10], O. Holdenrieder[11], L. Jankovsky[12], T. Jung[13], D. Jurc[14], T. Kirisits[15], A. Kunca[16], V. Lygis[17], M. Malecka[18], B. Marcais[19], S. Schmitz[5], J. Schumacher[20], H. Solheim[21], A. Solla[22], I. Szabò[23], P. Tsopelas[24], A. Vannini[25], A. M. Vettraino[25], J. Webber[26], S. Woodward[27] and J. Stenlid[28]

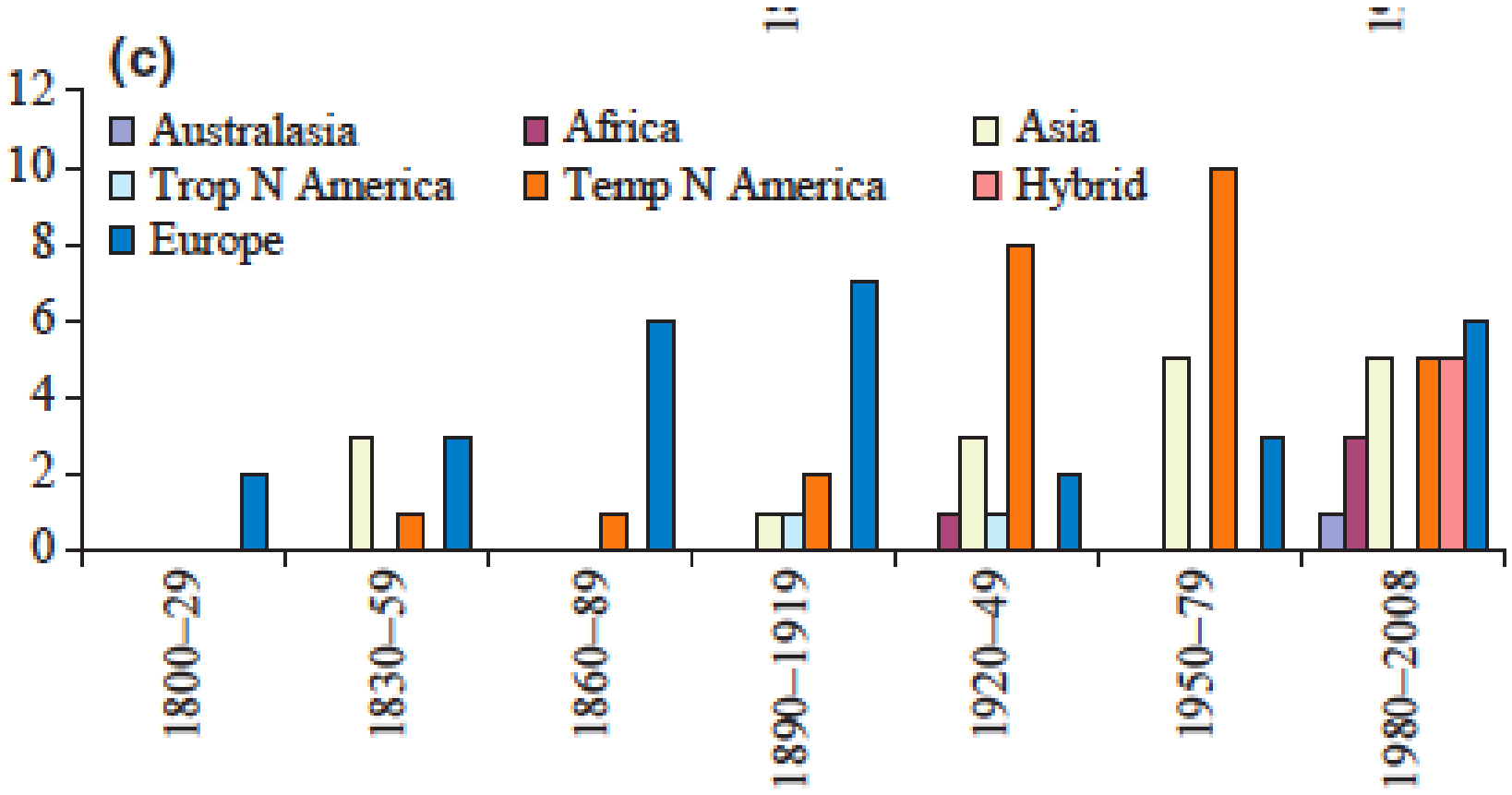
Invasive Forest Pathogens



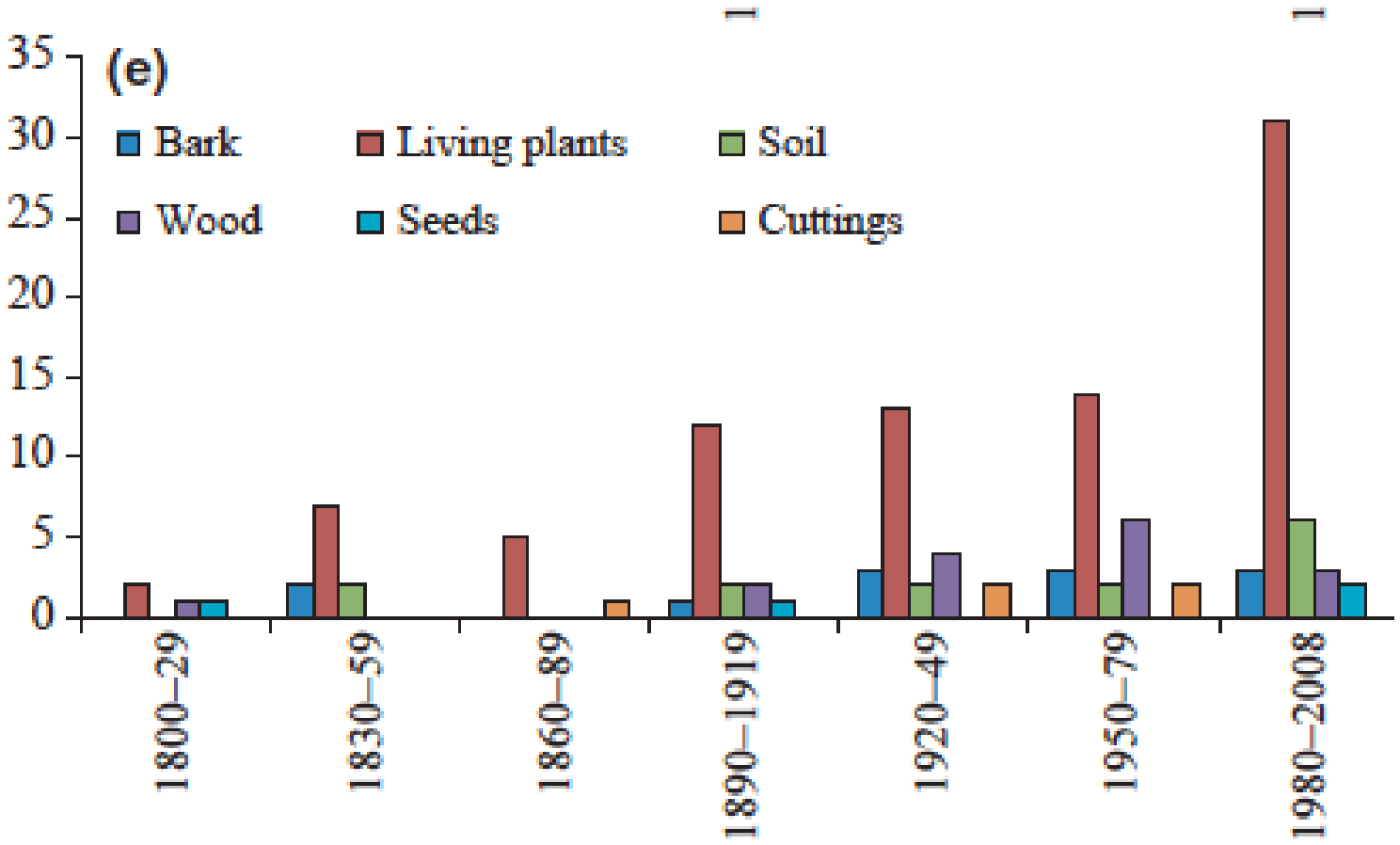
What is being moved



Where from


(c)
Australasia
Africa
Asia
Trop N America
Temp N America
Hybrid
Europe
12
10
8
6
4
2
0
1800–29
1830–59
1860–89
1890–1919
1920–49
1950–79
1980–2008

SUBSTRATE/PATHWAY of introduction



Globalization

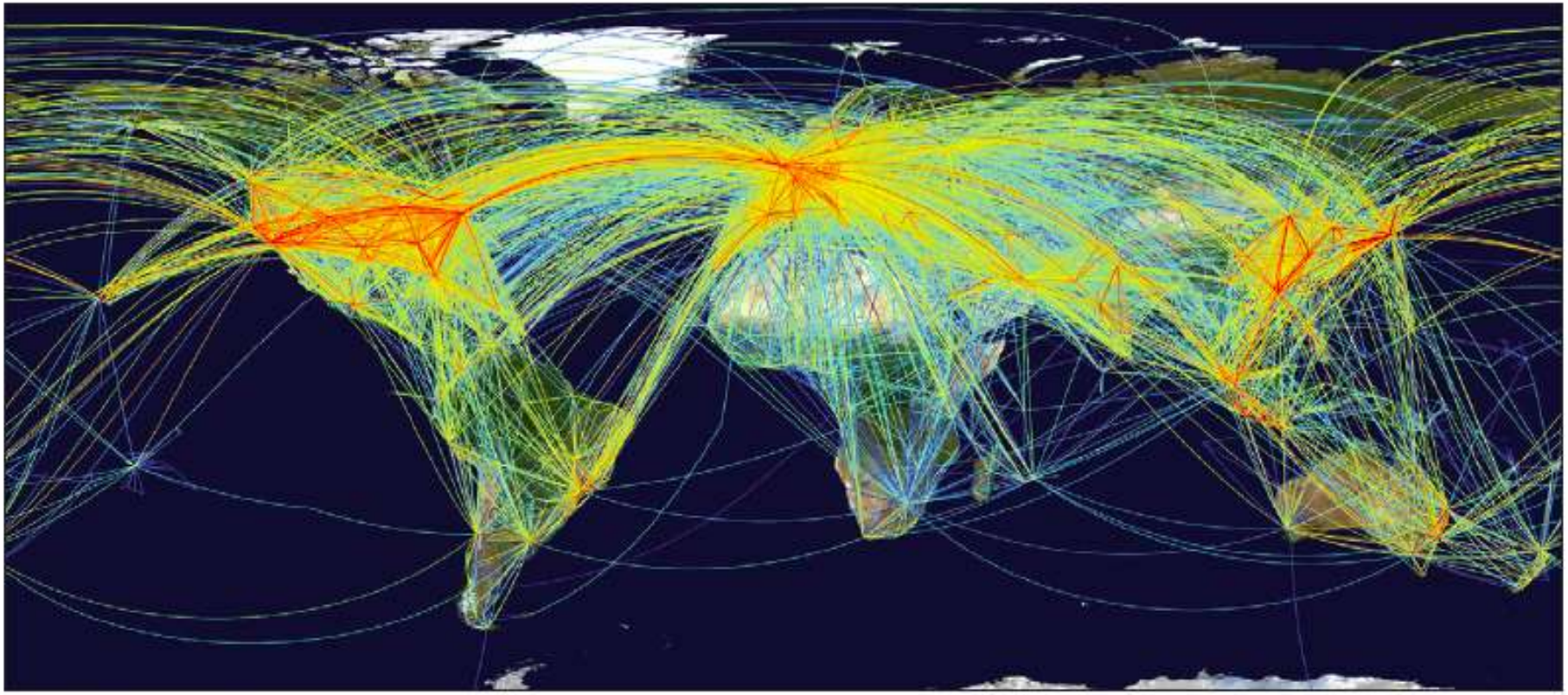


Figure 2: The global aviation network

Bases of Invasion Biology of Pathogens

- Establishment success is related to
  - presence of host (if host specific) or of similar host
  - survival as saprobe
  - similarity in climate between home and new region
  - lack of competitors/predators.
- If transmission > mortality then organism becomes invasive

Transmission

- If naïve hosts do not have any resistance then

Transmission=Reproductive potential

If exotic pathogen has a one or a few hosts, spread is usually faster. Gaps in host presence may be less important than dilution of inoculum (some hosts will not be infectious)

Bases of Invasion Biology of Pathogens

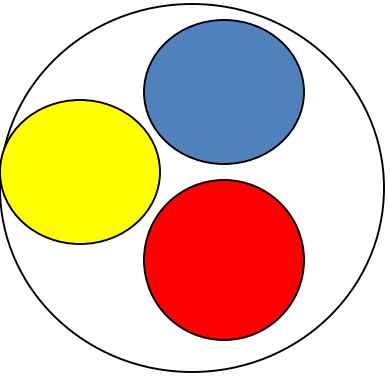
- Exotic organisms are all characterized by a strong genetic bottleneck, because only a few individuals make it!! How can a few individuals conquer a new continent
  - Drop traits they do not need
  - De novo mutation
  - Hybridization with native relative: btw most of the genes go from native to exotic species
  - Mate with other introduced individuals maybe originally from a different area

With a few exceptions

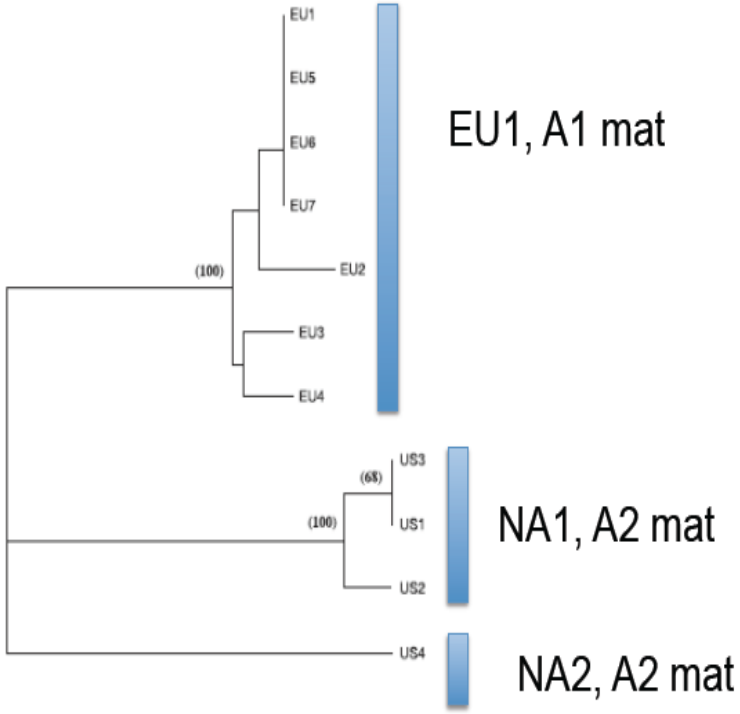
- Most successful exotic invasive species were introduced multiple times. That is why we need not to be complacent about re-introducing what apparently we think is the same pathogen, or even moving introduced pathogens from one site to another

- Eg. Only one sex of exotic forest pathogens is often introduced, that limits the ability of organism to adapt; need to avoid further introductions

Species has ancient lineages evolved in isolation (e.g. *P. ramorum*)



- Genetic divergence
- Phenotypic diversity:
  - Mating type
  - Growth rate
  - Pathogenicity
  - Host-specificity



Mating type

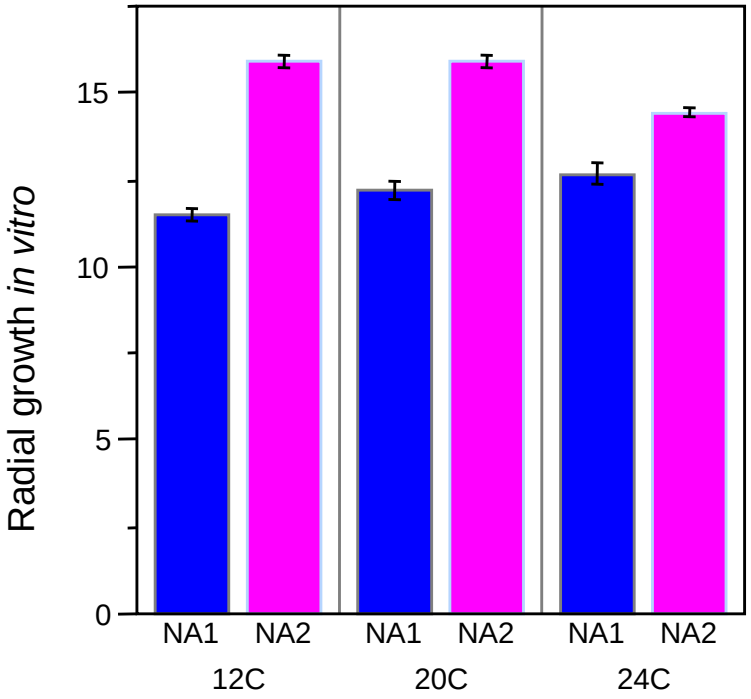
- Loci under selection may be linked to mating type
- Recombination will accelerate evolutionary rate
- Sexual structures may confer advantage

Sexual structures may confer advantage

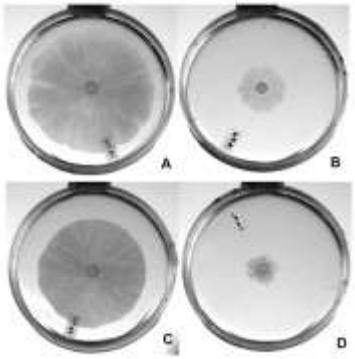
- Sexually produced **oospores** allow for survival in harsher climatic conditions

- Homothallic species such as *P. nemorosa* and *P. pseudosyringae* are less virulent than *P. ramorum* but are also introduced in California (Linzer et al. 2008) and have a **much broader distribution**, why?
  - History (introduced earlier ?)
  - Because homothallic they produce oospores

Growth rate…

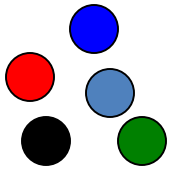


NA2 NA1



NA2 > NA1

Specific genotypes associated with specific phenotypes



- Some specific genotypes carry specific phenotypes
- *Phytopthora cinnamomi*: different genotypes associated with different commodities and transported around the world: are genotypes equivalent?

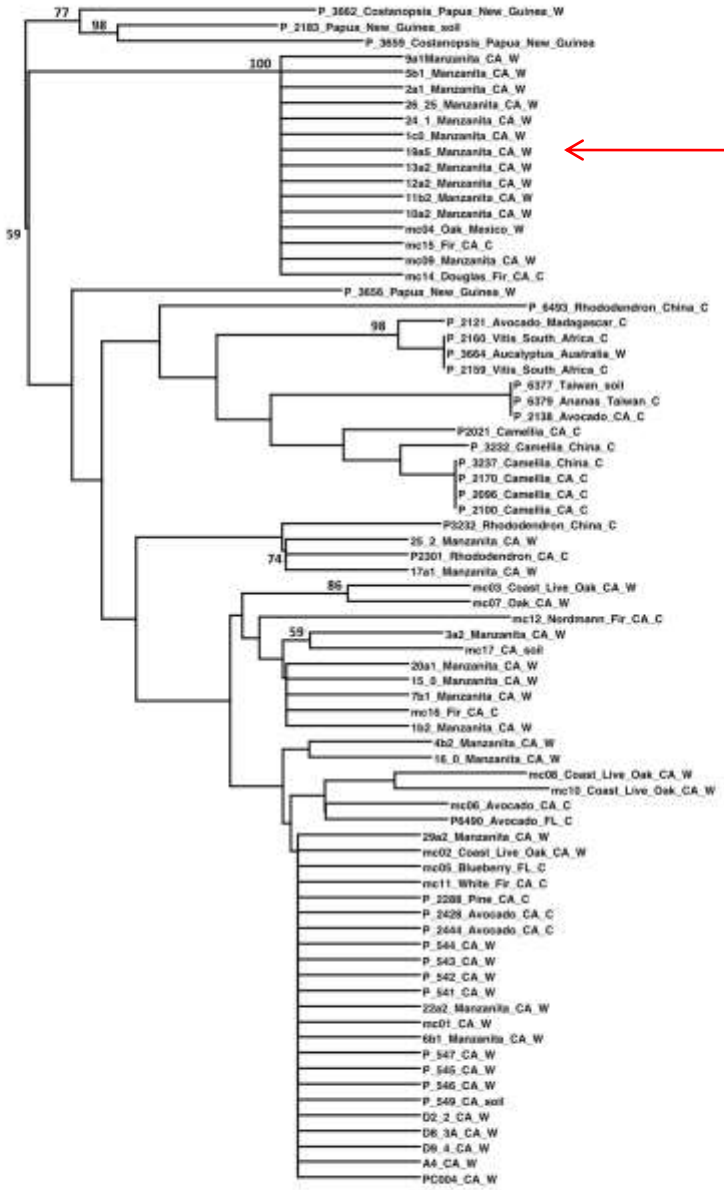
1- Huge oak mortality in Colima, Mexico (Tainter *et al*. 2000)

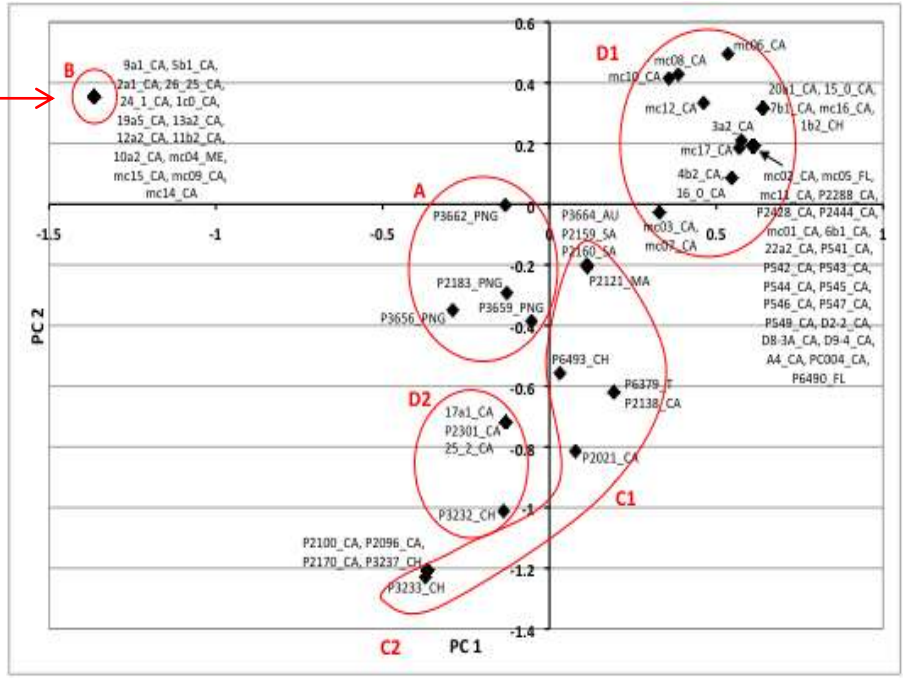
2- New problems in CA Christmas tree nurseries (2002).

3- Quasi-extinction of rare manzanita in CA (Swiecki *et al*. 2004)

- All three recently reported (10 years)
- All three associated with *P. cinnamomi*
- All three associated with the same novel genotype of the pathogen

Widespread mortality of lone manzanita caused by *P. cinnamomi*





Newly discovered single genotype now found in different locations, on multiple hosts, and represents a new threat

Predicting impacts of invasions (I)

$$I = k * i * t_{(hcpsv)}$$

- k = constant depending on the pathosystem
- i = number and source of introduction events
- t = transmission rate

h = density of sporulating hosts

c = permeability of landscape
p = favorability of climatic conditions
s = synchronicity between host susceptibility and pathogen life cycle
v = variation in susceptiility of hosts and virulence pathogen

Garbelotto & Pautasso, 2012

<u>Transmission</u> is an overlooked aspect, which includes, but is much more, than just pathogenicity