Fungal Invasions: beyond the Lack-of Co-evolution Hypothesis

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Invasion Biology: Hypotheses

- Enemy Release
- Better utilization of resources than native spp.
- Optimal Ecological Adaptation

Invasive Fungal Pathogens: Hypotheses

- Lack of Co-evolution: naïve host populations have never been exposed to exotic pathogens, hence they have not been selected for resistance. High host susceptibility leading to outbreaks

Different approaches have rarely been employed

- An organism becomes invasive when:

  transmission rate > mortality rate

- We are interested in studying those traits that will affect transmission rate of fungi

Gonthier and Garbelotto 2013, CABI Mini Review

Two scenarios

- I Invasive species occupies an empty niche, i.e. there is no inter-specific competition

- II Invasive species occupies a niche already occupied by a native species with similar ecological parameters
  - **COMPETITION**
  - **HYBRIDIZATION AND GENE INTROGRESSION**

Experimental Approaches

- Population genetics to understand origin(s) of invasive species and their range expansion history
- Common garden studies to compare various phenotypic traits including virulence, sporulation, temperature adaptation, phenotypic plasticity
- Population genetics to determine if trait variation is random or adaptive (Qst-Fst)
- Comparative genomics to understand the genomic basis that gives the invasive species an advantage over native taxa

Cypress Canker

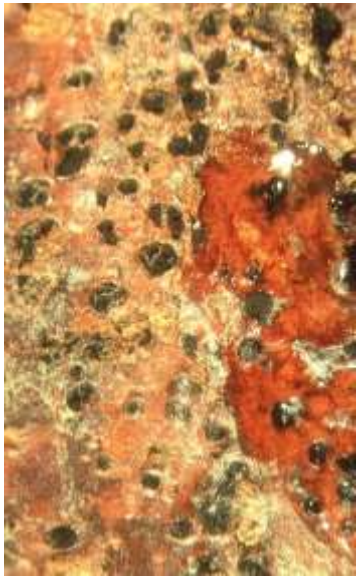
- Pathogen: *Seiridium cardinale*
- Native Range: Pacific Southwest, where it reproduces sexually and asexually
- Limited host range: one family, *Cupressaceae*
- Invasive outbreaks in the Mediterranean caused by the introduction through nursery stock of a single genotype reproducing clonally
- No competitors, as related taxa (*S.*

Lesion and resinosis

Mitospores (conidia)

wounds

Cypress dieback
and mortality



Asexual fruiting produced
ONLY ON LIVE TISSUE

Population Biology

Sequence and Simple-Sequence Repeat Analyses of the Fungal Pathogen *Seiridium cardinale* Indicate California Is the Most Likely Source of the Cypress Canker Epidemic for the Mediterranean Region

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Accepted for publication 3 August 2011.

Forest Pathology WILEY-BLACKWELL

For. Path. doi: 10.1111/efp.12019

AFLP analyses of California and Mediterranean populations of *Seiridium cardinale* provide insights on its origin, biology and spread pathways

By G. Della Rocca[1], T. Osmundson[2], R. Danti[1], A. Doulis[3], A. Pecchioli[1], F. Donnarumma[4], E. Casalone[4] and M. Garbelotto[2,5]

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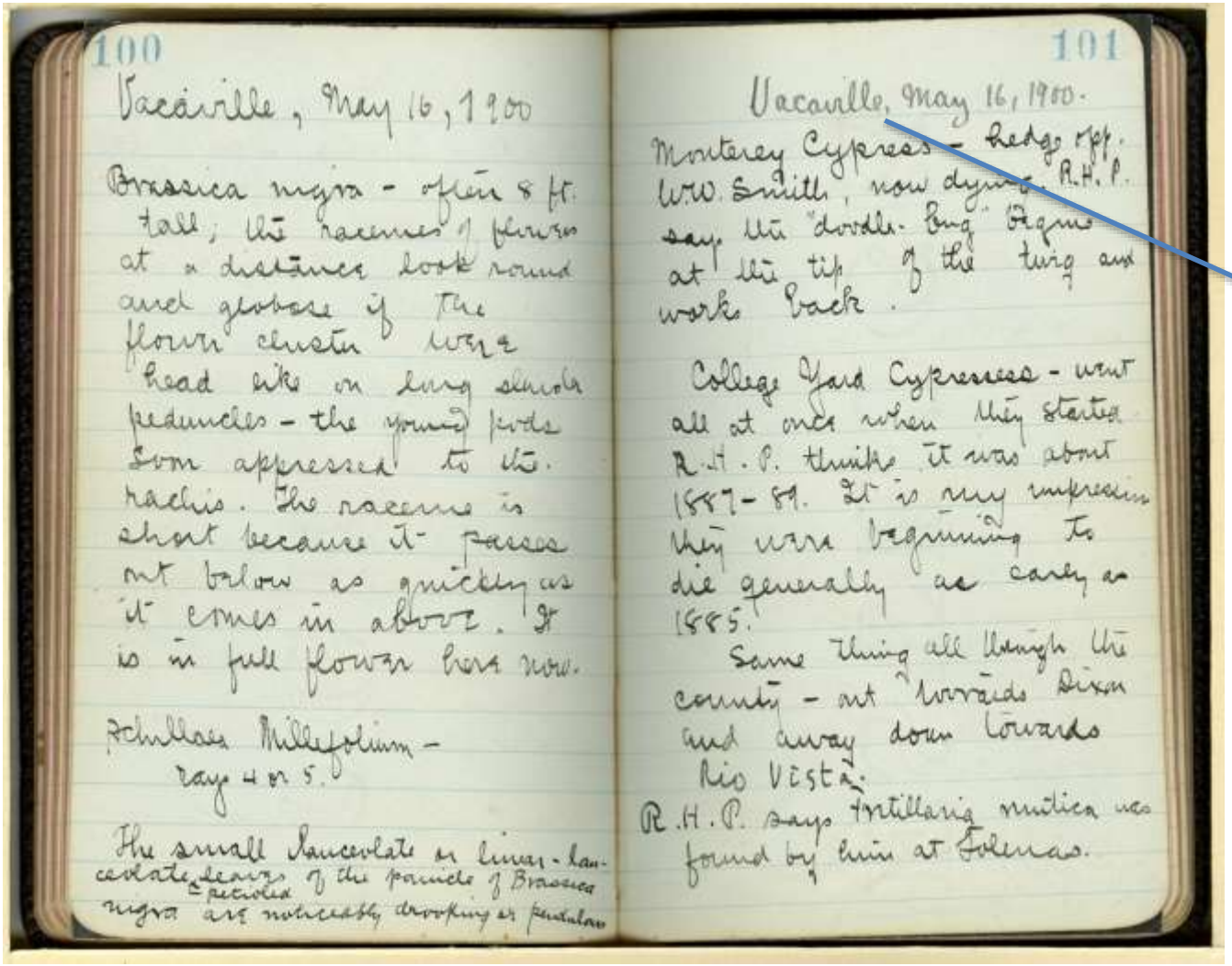
Cypress canker described as early as 1885, in the field book of Jepson



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Vacaville, May 16, 1900

Brassica nigra - often 8 ft. tall; the racemes of flowers at a distance look round and globose if the flower clusters were head like on long slender peduncles - the young pods soon appressed to the rachis. The raceme is short because it passes out below as quickly as it comes in above. It is in full flower here now.

Schkuhria Millefolium - rays 4 or 5.

The small lanceolate or linear-lanceolate leaves of the panicle of Brassica nigra are noticeably drooping or pendulous - petiolate

101

Vacaville, May 16, 1900.

Monterey Cypress - Hedge ref. W.W. Smith, now dying. R.H.P. says the "dieback" begins at the tip of the twig and works back.

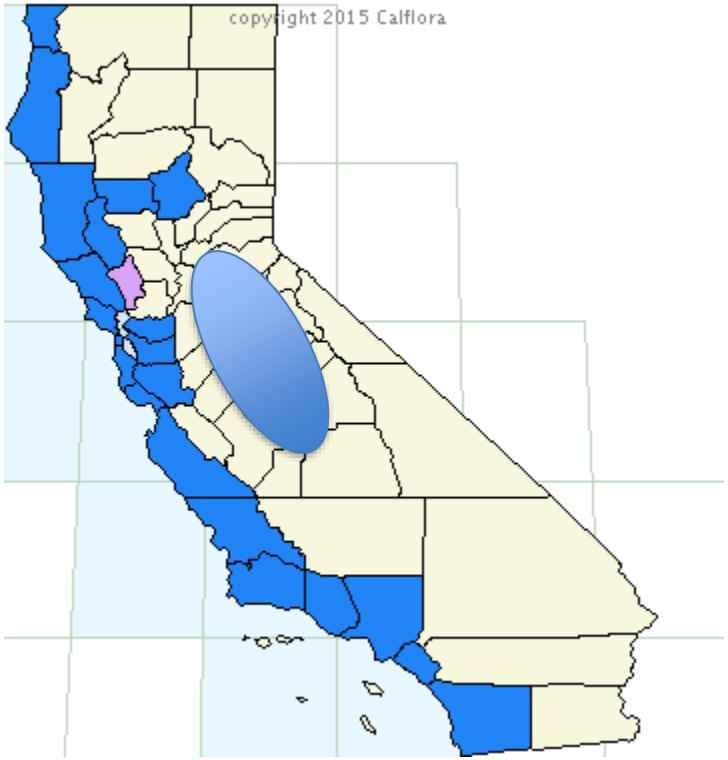
College Yard Cypresses - went all at once when they started. R.H.P. thinks it was about 1887-89. It is my impression they were beginning to die generally as early as 1885.

Same thing all through the county - out towards Dixon and away down towards Rio Vista.

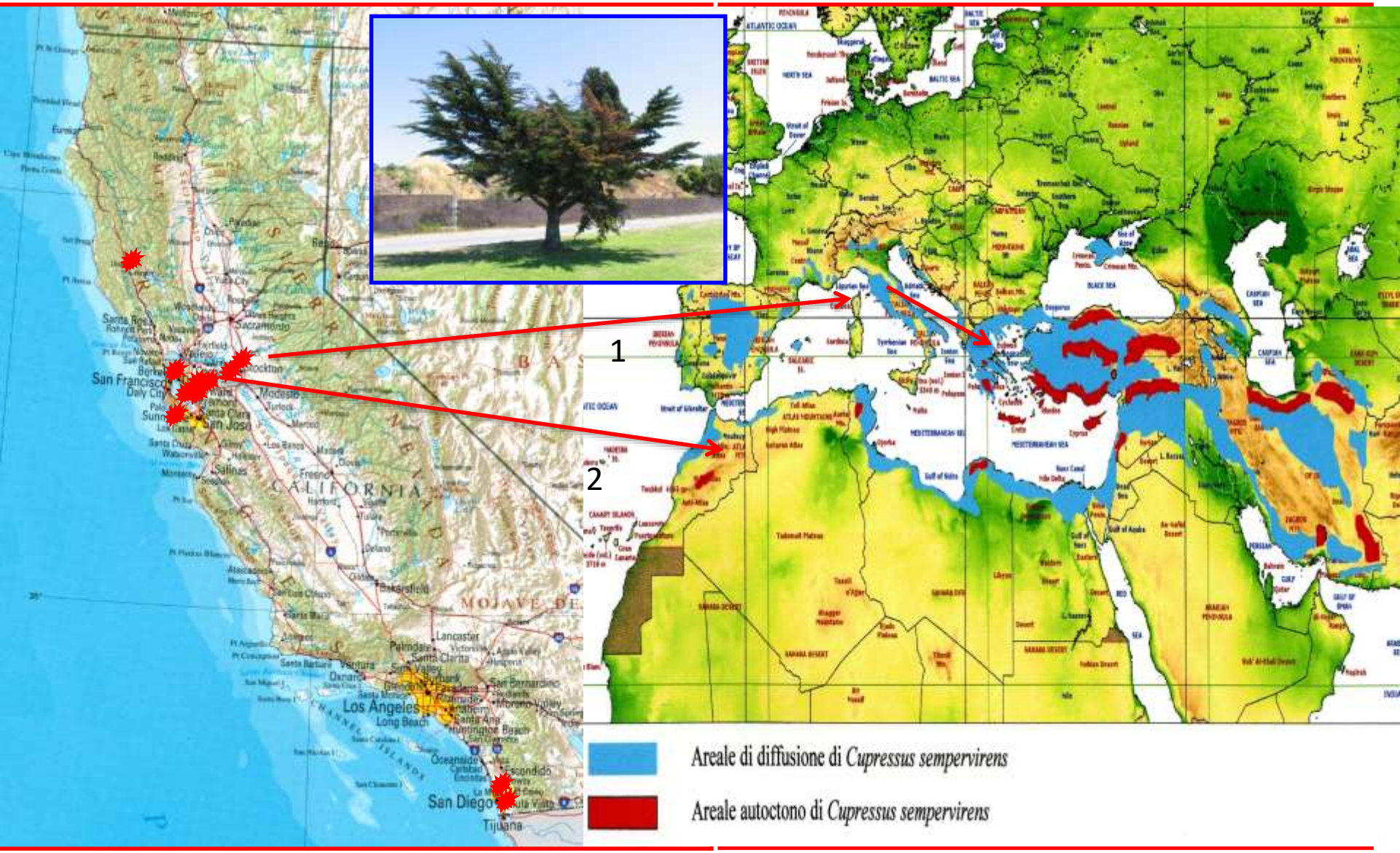
R.H.P. says Antillaria mutica was found by him at Folsom.

In central valley where Monterey cypress Is not native

Natural Distribution of Monterey Cypress is coastal. Planted in inland valleys for trade and for agricultural purposes



Two separate introductions, both from California. The first to Europe matches the high importation of Monterey cypresses from CA Valley nurseries in between the two world wars, and is documented in notes by the owner of the largest nursery in Europe



Global cypress epidemic traced to CA

Robin Meadows

Massive die-offs of Italian cypresses from Portugal to Greece have been linked to California's Monterey cypresses (*Cupressus macrocarpa*), solving a long-standing puzzle in the world of plant pathology and surprising researchers. "When I was a student, this was one of the big mysteries", says study

A Potov/Wikimedia Commons

International trade in Monterey cypresses caused a canker disease pandemic.

1- In CA population explosion of the pathogen due to off site planting of Monterey Cypress in the Valleys

2-Large CA population of pathogen increased likelihood of long distance Transfer

3- EU outbreaks due to fact pathogen is exotic there, but how did it succeed?

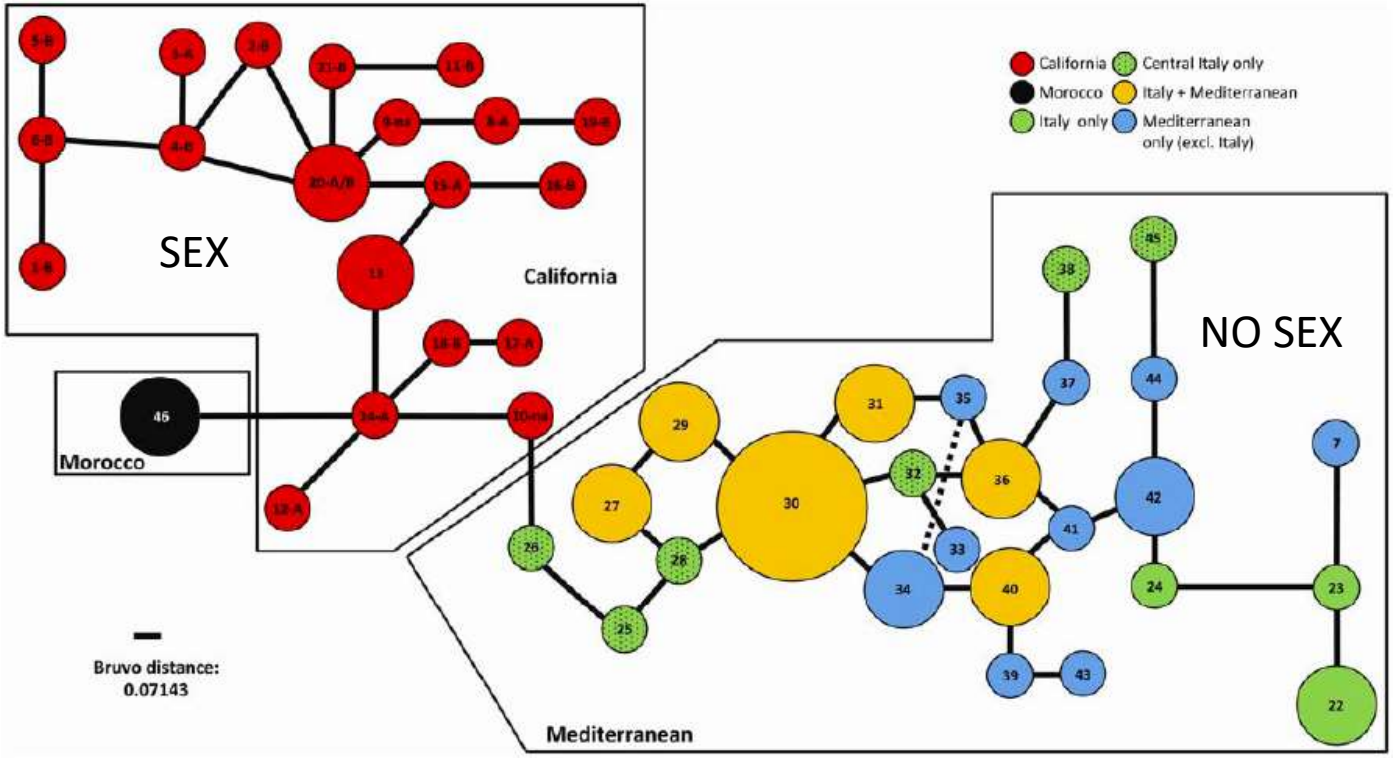


Fig. 2. Minimum spanning network (MSN) showing relationships between multilocus genotypes (MGs) in Californian and Mediterranean populations. Each node represents a different MG and nodes are scaled to reflect the numbers of individuals sharing the same MGs: small = singleton MG, medium = 2 to 5 individuals with same MG, large = 30 individuals with same MG. Labeling with A, B, or na within nodes relates to whether individuals with that MG were found within clade A or B or not included in the analysis, respectively, in the *Seiridium cardinale* neighbor-joining tree (Fig. 1). Dotted line between nodes 34 and 35 indicates where the branch length was unable to be scaled properly without overlap. Distance is the equivalent of the shortest distances in the network; for example, equal length to those between 30 and 32 or 32 and 36.

1- CA and EU networks are connected by a single link between two genotypes (red to green): introduction of a single genotype likely source of EU outbreak

2- Introduced population becomes invasive with time (green to yellow; increased size of circles)

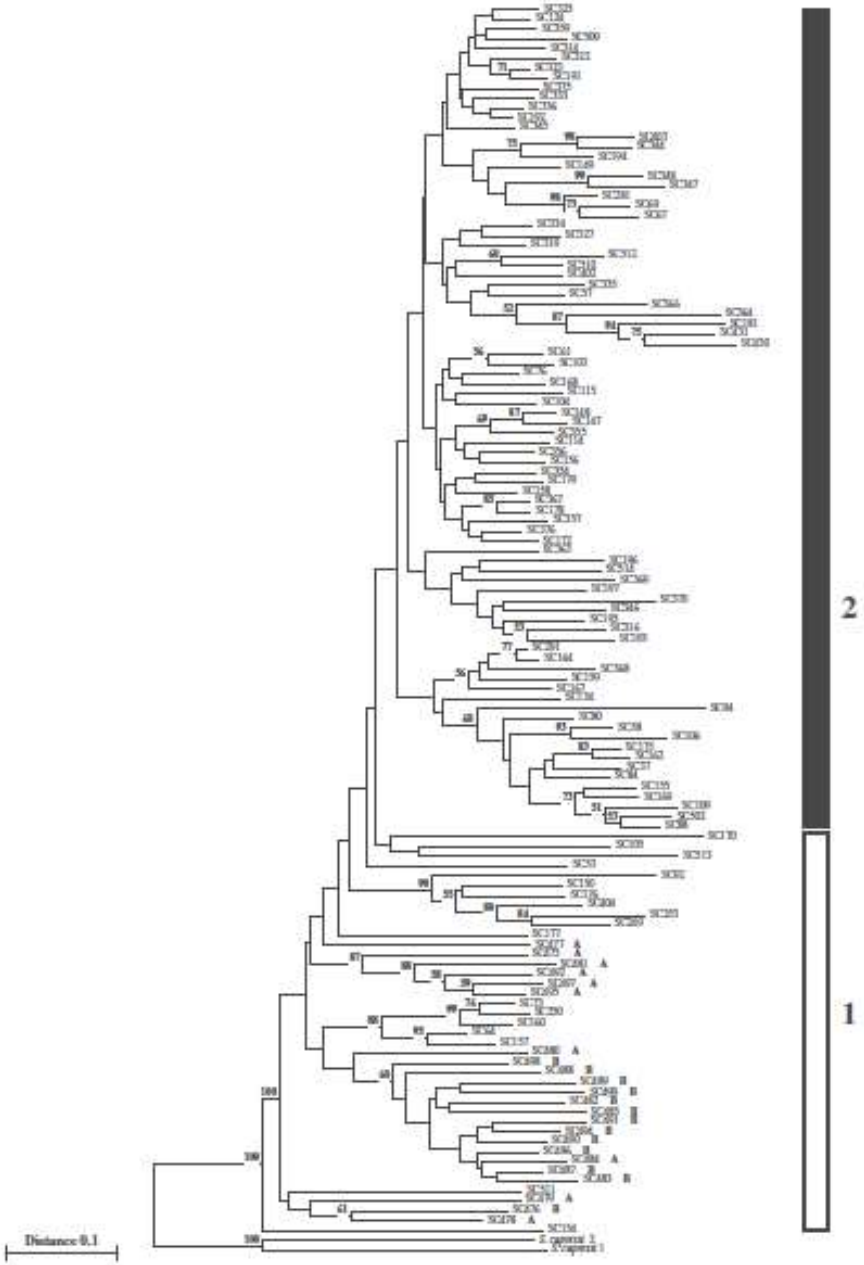
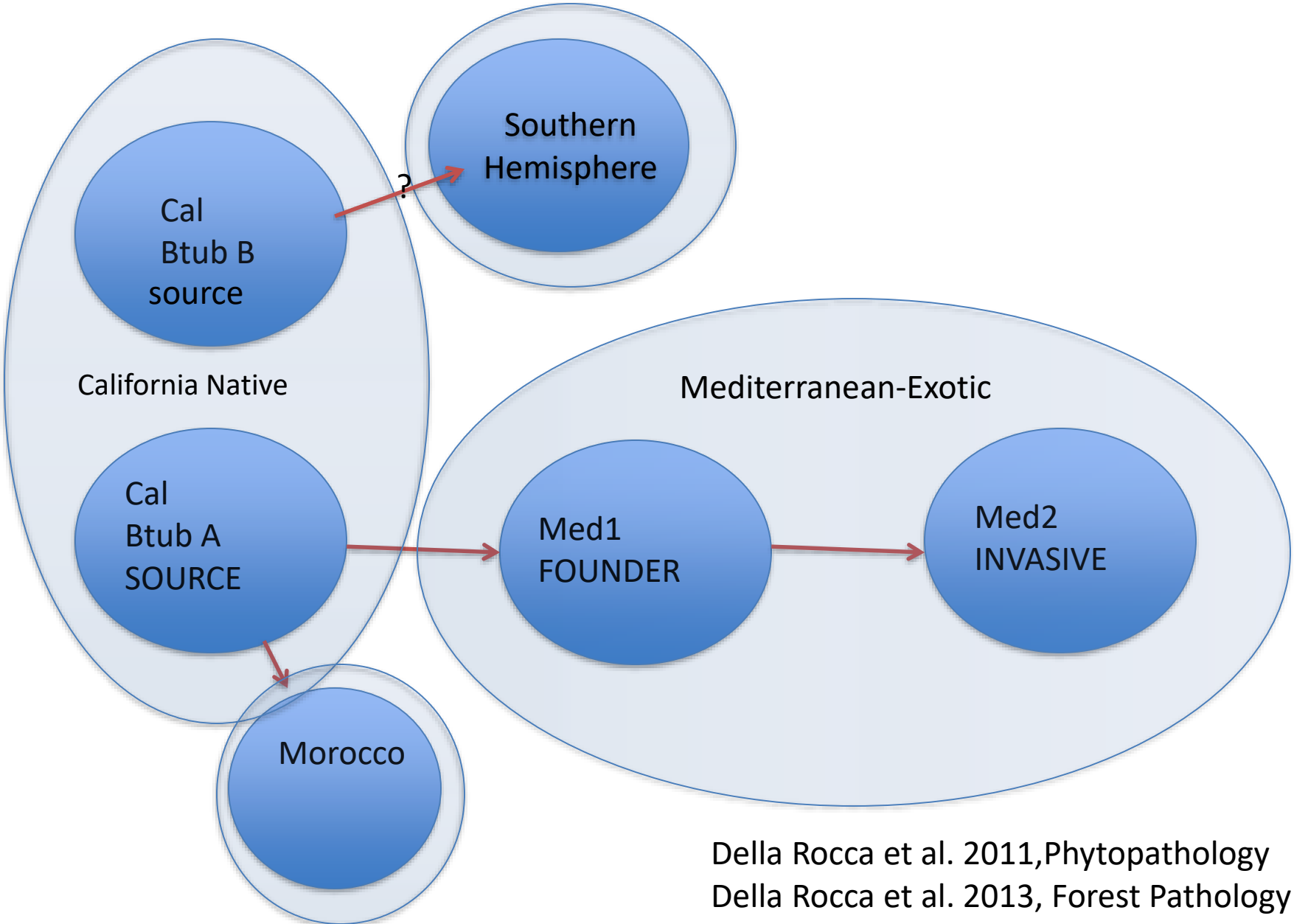


Fig. 2. NJ trees calculated using pairwise Jaccard genetic similarity coefficients (S_J). Bootstrap values >50% appear above branches. Lateral bars (1 and 2) indicate clusters inferred in the structure analysis. Letters A and B indicate the β-tubulin haplotype of each Californian isolate. All Mediterranean isolates belong to the haplotype A.

Invasive EU population derived from founder pop

Source (CA) and Founder (EU from CA) populations

Invasion history (Sequencing, SSRs, and AFLPs)



Della Rocca et al. 2011,Phytopathology
Della Rocca et al. 2013, Forest Pathology

esa

ECOSPHERE

An increase in transmission-related traits and in phenotypic plasticity is documented during a fungal invasion

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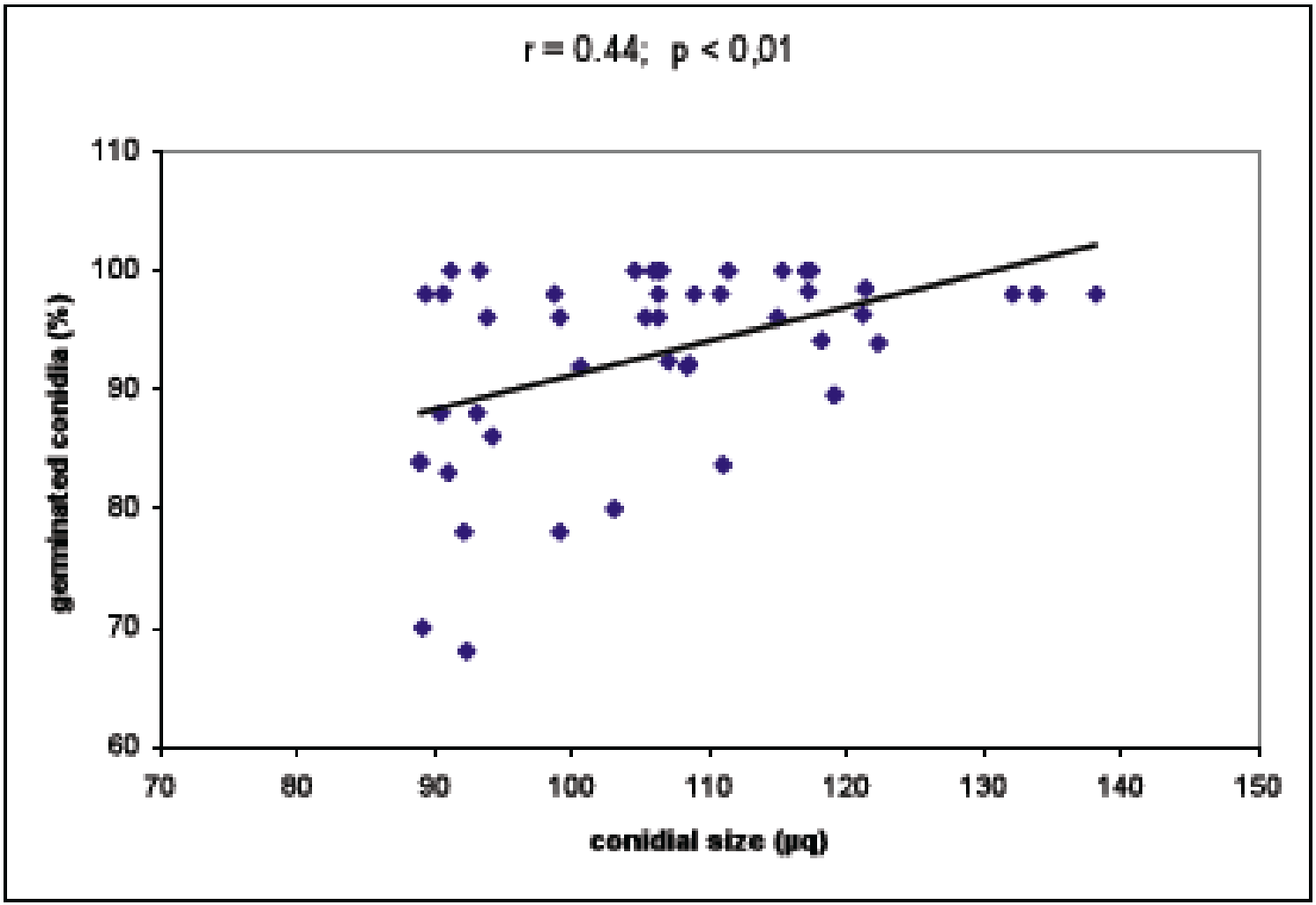
Citation: Garbelotto, M., G. Della Rocca, T. Osmundson, V. di Lonardo, and R. Danti. 2015. An increase in transmission-related traits and in phenotypic plasticity is documented during a fungal invasion. Ecosphere 6(9):XXXX. http://dx.doi.org/10.1890/ES14-00426.1

Variation in transmission traits in three successive populations

Trait	Source CA Pop	Founder Med Pop	Invasive Med Pop	Adaptive Qst-Fst
In vitro growth rate	High	Low	Low	Yes
Canker size	High	Low	Low	No
Fruiting 30 days	High	Low	Low	No
Fruiting 90 days	High	Medium	High	n/a
Spore size	High	Medium	Low	Yes
Spore germination	High	Medium	Medium	Yes

Garbelotto et al. 2015, Ecosphere, In press

Trade off between spore size and germination



Variation in phenotypic plasticity index in three successive populations

Trait	Source CA Pop	Founder Med Pop	Invasive Med Pop
In vitro growth rate	Base	Increased	Decreased
Canker size	Base	Increased	Decreased
Fruiting 30 days	Base	Same	Decreased
Fruiting 90 days	Base	Increased	Decreased
Spore size	Base	Increased	Decreased
Spore germination	Base	Increased	Decraesed

Garbelotto et al. 2015, Ecosphere, In press

CA and EU populations are clearly different genetically and phenotypically

T (°C)	Geographic population: MED	Geographic population: CAL
15°	3.18 a	4.39 b
20°	5.10 a	5.94 b
25°	5.61 a	6.92 b
30°	2.04 a	3.39 b

Med populations are not suited to grow at low and especially at high temperatures, those traits have been selected in native populations, but probably have been lost due to the bottleneck of the introduction. “Master of some” scenario

In less than 100 years, in spite of large population expansions both in Ca (1920-1940s) and in Europe (1950-1970s), CA and EU pops are phenotypically distinct

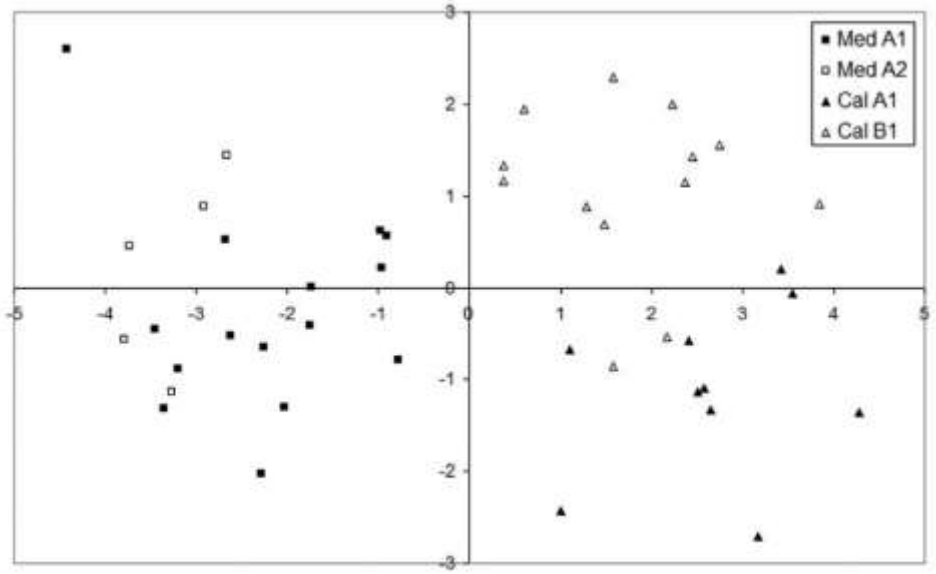


Fig. 3. A discriminant analysis performed on a matrix containing the mean values calculated for each *S. cardinale* isolates for all the assayed traits. Isolates were subdivided in four groups according to their geographical origin; their β-tubulin haplotype group (A or B, see text) and Structure AFLP group (1 or 2, see text).

USA-EU trade

- Populations of the pathogen are clearly distinct both genetically and phenotypically with USA populations displaying sexual reproduction, higher virulence, and better adaptation at low and high temperatures when compared to EU isolates
- Further USA to EU introductions could be catastrophic

Seiridium conclusions

- No selection towards increased virulence. Excessively virulent genotypes may produce less spores due to host death, but within moderate virulence the best performing isolates produce more spores
- No need to produce spores fast (e.g. in 30 days), because no interspecific competition
- As invasion progresses, selection for higher overall sporulation (e.g. in 90 days)
- Continued adaptive selection towards smaller spores (further dispersal) in spite of trade-off of reduced germination
- Plasticity increases in first phase and then decreases

Heterobasidion root rot

- Invasion of Central Italy by Eastern North American species *H. irregulare*, brought by US Army in 1944
- Generalist with preference for *Pinaceae*
- Presence of native sister taxon *H. annosum*
  - *Exotic species is much more widespread and damaging in Italy than native species*
  - *Hybrid swarms and rapid evolution: up to 50% of isolates have admixed genomes and 20% of alleles are chimeric (recombinant)*

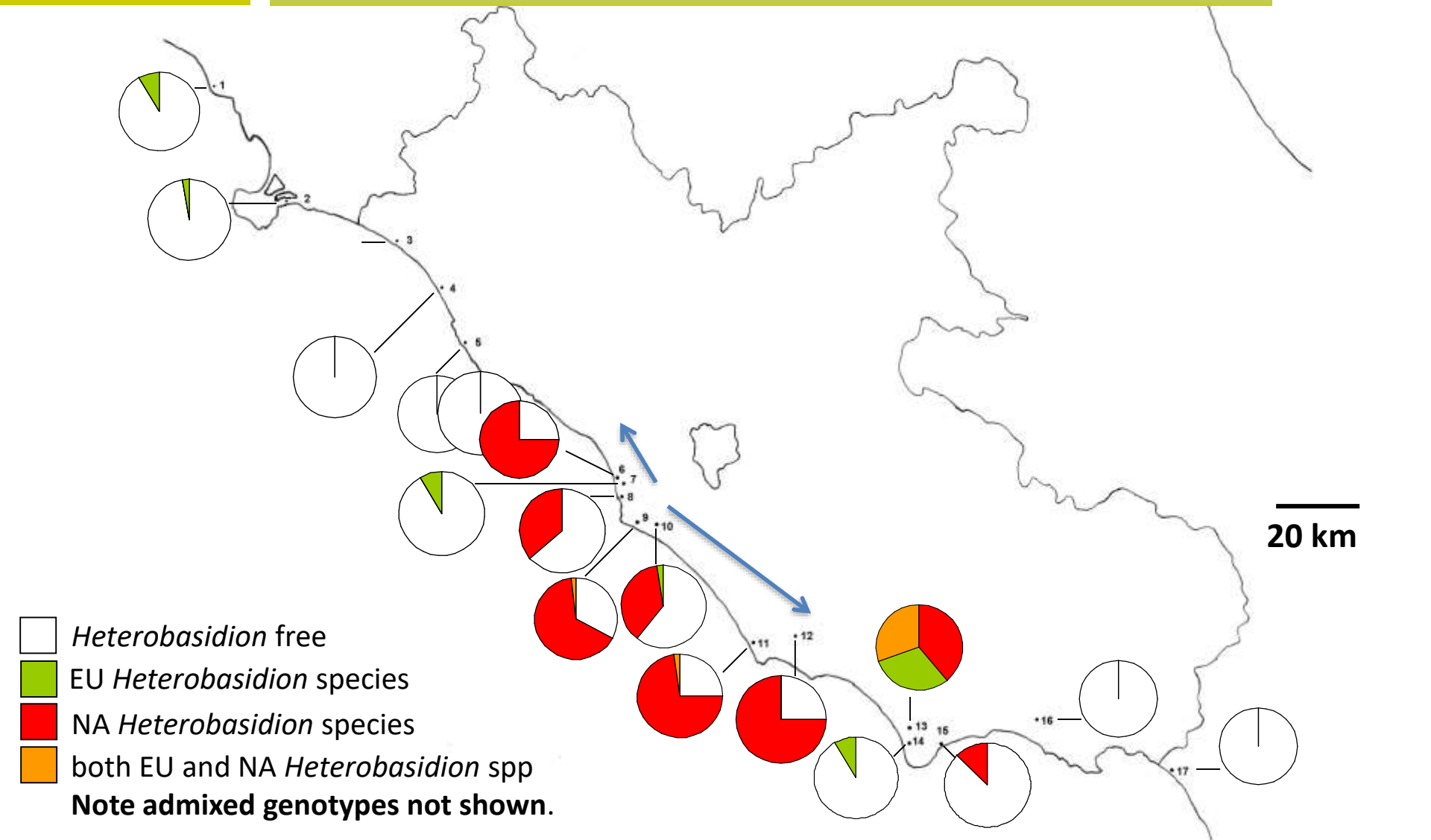
Gonthier et al. 2008, Molecular Ecology
Gonthier and Garbelotto, 2011, Molecular Ecology

The WAR on Nature:
best known proven example of pathogen carried by military, during world war

Trees become casualties of war

Gonthier et al. 2008, Molecular Ecology

Distribution of the exotic pathogen



Yosemite Lodge complex
1972

cabin crushed by tree with
rotted roots

since 1973
7 fatalities
19 serious injuries
Over $1M property damage

Yosemite Lodge 1975 Root disease centers outlined

Yosemite Lodge 1997 Root disease centers outlined

Many gaps with very little regeneration and have not closed in

Change in gap area 1972-1999

Year	Area in gaps (m^2)	Percent in gaps
1972	6125	3.5
1999	53,981	31