5th Quizzes

• When talking about dispersal we contrasted a direct dispersal with a stepping stone mode. What are the differences between the two?

• Why specific alleles and genetic similarity can be used as way to determine spread potential of a microbe?

• Explain why and how different generation time between host and pathogen affect their reciprocal adaptation

• Explain how metapopulation structure affects host-pathogen adaptation
Local Adaptation

• Process strongly dependent on generational rates, that is why microbes increase virulence more rapidly than hosts, however a generational turn over of the host may increase host resistance

• Importance of metapopulations: locally co-evolving hosts and pathogens are more likely to undergo selective processes. If long distance effective dispersal occurs, resistance will be slower to show up in hosts, and virulence will increase more slowly in pathogens

• Red queen hypothesis: relationship between hosts and pathogens is always dynamic: pathogen increases virulence, plants will be selected for increased resistance. Often virulence and resistance are determined by individual genes, but these genes cannot be accumulated indefinitely due to their cost
Environment

Climatic

Climate patterns match pathogen biology (high RH, rainfall when needed, temp range for growth: thermophilic vs. psychrophilic organisms, Max-min temperatures)

Host phenology: synchrony between pathogen and host
Wetness > 12 h
Temp > 19 C
Optimal conditions for transmission of SOD
Previous slide shows that optimal conditions for spread of SOD were not met in 2012:

1- When rainfall was present (necessary to have 12 hours of wetness), temperatures were too cold.

2- When temperatures reached the optimum, there was no rainfall, so not enough wetness for optimal infection.
Synchrony pathogen-host: pathogen sporulation matches highest host susceptibility

High sporulation by pathogen

Susceptibility of oaks (lesion size)
The classic disease triangle can be used to understand dynamics of native diseases, but it can also be used to understand the dynamics of emergent diseases, see following slides.
Host

Disease

Pathogen

Environment

- Monocultures
- Off site
- Exotic
- Artificial cross
- Enemy Release Hypothesis
Hevea brasiliensis: severely affected by foliar pathogen in South America, but does extremely well in Asia where the pathogen has not been introduced.
Swiss needle cast on Douglas-fir: excessive homogeneity in plantations

Caused by *Phaeocryptopus gaeumannii*
Host — Pathogen — Environment

Pathogen

• Exotic pathogens
• Pesticide resistance

Disease

Host — Environment

Environment
American chestnut - *Castanea dentata*

Chestnut blight caused by *Cryphonectria parasitica*

Stump sprouts
Port-Orford Cedar root disease
Phytophthora lateralis

Pitch canker
Fusarium circinatum

White Pine Blister Rust
Cronartium ribicola

Phytophthora root rot
Phytophthora cinnamomi

Introduced Forest Pathogens in CA
Marin County, CA
June 2000

Sudden Oak Death
*Phytophthora ramorum*
Gypsy moth
Lymantria dispar
Golden spotted oak borer
*Agrilus coxalis*
Laurel Wilt

Raffaelea lauricola sp. nov. consistently isolated from affected tissue...& from exotic boring beetle: Xyleborus glabratus (an ambrosia beetle)
Pesticide resistance

- Late blight of potato, caused by *Phytophthora infestans*

- 1845-1849 caused the Irish famine, potato was like the rubber tree. Moved from its native range, but without the pathogen (ERH). Then pathogen was introduced in Europe with dire consequences on crop production.

- More recently, introduction in Europe and North America of different mating type allowed pathogen to reproduce sexually and overcome effect of pesticide methalaxyl with 50% drop in potato production.
Environment

- Logging
- Fire suppression
- Pollution
- Climate change

Disease

Pathogen

Host
Armillaria spp

Change in land use (stress).

Heterobasidion spp.

Logging as pathogen establishes itself through stumps.
Dothiostroma needle blight: fire exclusion
New host pathogen combinations possibly leading to an emergent disease or biological control

- Pathogen stays/Plant moves: invasive plant
- Pathogen native/plant introduced
- Pathogen moves/Plant stays: exotic epidemic
- Pathogen moves/Plant moves: biological control
Success. The “1:10” rule

- This rule applies to introduced organisms and has been drafted for plant and animal invasions.

- The rule states that only 1 in ten organisms that are transported will successfully make it, introduction of 9 out of ten organisms that are moved will fail.

- In the case of microbes the rule may actually be 1:100.

- Notwithstanding that only a small fraction of organisms that are moved will become successfully invasive, the number of successful invasives is proportional to the scale of movement of goods and people.
So, which one will be the successful invasive?

• Can exotic be transported (where will it survive: resting structures, soil, insect, wood, live plants)

• What pathways are in place: single event not likely to be successful but repeated events increase chances

• Can exotic withstand new environment (obviously the more similar the environment in the native and invaded area, the more likely its success)

• Can it withstand attacks of predators
So, which one will be the successful invasive?

- Can it outcompete similar native organisms by accessing resources
  - Can a pathogen be pathogenic
  - Can a pathogen be sufficiently virulent
  - Can a pathogen use a saprobic stage to enhance its success

- How will it survive when conditions are unfavorable?

- How effectively can it reproduce: two strategies
  - r selection (reproduce constantly because spectrum of conditions favorable to reproduction are broad)
  - K selection: large reproductive potential in specific condition

- If there is a competitor for same resource, can it reproduce rapidly to “arrive first”
• Invasion driven by ecological conditions: conditions for growth, reproduction and transmission are ideal for invasive species

• **Enemy release hypothesis**: the invasive has been moved but not its enemies

• **Resource availability** (pathogenicity/virulence): lack of coevolution; see next slide
Pathogenicity

• Qualitative: ability to cause disease

• Often regulated by a single gene

• Avr genes in pathogen and resistance genes in host
Gene for gene

• Resistance in host is dominant
• Virulence is recessive

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Gene for gene

- Resistance in host is dominant
- Virulence is recessive

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Resistance: no disease
Functions of avr/R genes (other than regulating defense response)

- Avr genes may help detoxify plant enzymes, secure necessary aminoacids or proteins, plant toxins, promoting pathogen growth. Normally they are mobile, wall-bound products.
- R genes normally recognize multiple avr genes and start hypersensitive response (programmed cell death).
Avr/R genes matches are specific

- Race of the pathogen (avr1) matched by variety of the crop (R1).

- At the base of crop breeding science

- If R genes target avr genes linked to important housekeeping functions, they are more durable
Can be R genes accumulated?

• There is a cost associated with R genes

• Mostly R genes initiate costly defense processed, often even when challenged by innocuous microbes

• Some evidence that in absence of specific avr, R are lost
Plants immune response

- Plants do not possess an immune system such as that of animals

- They do recognize pathogens

- Recognition initiates secondary metabolic processes that produce chemicals that will stop or slow microbial infections: thickening of cell wall, premature cell death (HR response), systemic acquired resistance

- There are also constitutive defences that will make plants less susceptible without a recognition system
Virulence: quantitative response

• Multiple genes controlling:
  – Phenotypic traits conferring virulence
  – Production of plant detoxifying enzymes
  – Production of plant toxins
• Qualitative vs. quantitative resistance (I)

– Qualitative, single gene, resistance may be present in a species attacked by an exotic pathogen because R receptor may have evolved and have been selected to deal with a similar pathogen. Or it may be absent

– Issue is: can the population of the host increase the frequency of R to effectively maintain a healthy population? That depends on the baseline frequency of R, on generation size of the pathogen (the shorter the better) and on how much gene flow comes in from areas without the pathogen (the more the worse)
• Qualitative vs. quantitative resistance (II)

– Qualitative, single gene, resistance is easier for humans to manipulate. A heterozygous Rr mother will generate 50% R progeny

– Issue is: single gene resistance is less durable because it can be circumvented by a single mutation of the pathogen
Lag phase: the time that incurs between the introduction and the expansion of a microbe. That is when Transmission > Mortality.

Normally, invasions are identifiable only after the Lag Phase

Invasions by pathogens can be Visible but other microbial Invasions may not be
Fungus-mediated invasions

- Fungal species that form mutualistic associations with plants can help them become invasive
  - Grass endophytes (asomycetes) produce alkaloids that reduce herbivory and also make plants drought tolerant
  - Mycorrhization makes pines invasive in Southern Hemisphere (w/o them they fail to become established)
The weblike structure of fungi, usually immersed in the soil or in plant matter is involved in an essential symbiosis that greatly enhances the ability of plants to grow.
The visible part of root tips of most trees is actually a mantle of fungal hyphae fused with the plant tissue.
What is the deal of this mutualism?

- Fungus absorbs nutrients for plants
- Plant gives fungus carbohydrates it produces via photosynthesis
Enemy Release and Biological Control

- Rubber trees without pathogens in Asia
- Blackberry in Australia

- If we introduce pathogen in new range occupied by invasive plant then we may bring down invasive potential of plant. It is essential introduced fungus (Biological Control) is host specific to avoid starting an exotic disease
Examples of Biol. Cont. involving fungi

- *Miconia calvescens* in Pacific islands and fungus *Colletotrichum gleosporoides*. This is an interesting example because endophytic fungal community further mediates success of biological control
Miconia calvescens is a highly invasive shrub to tropical island ecosystems and is regarded as the most extreme case of a weed threatening biodiversity in tropical oceanic island ecosystems. A moderately successful fungal biocontrol agent, Colletotrichum gloeosporioides f. sp. miconia (Cgm), is most effective at higher elevations at controlling M. calvescens. Previous
• Endophytic fungi (fungi that reside inside plant without apparent symptoms) from lower altitudes were more competitive with biological control Colletotrichum and limited its effect.

• Conditions were favorable to Colletotrichum infection independent of altitude.
Analyzed fungal communities (endo and epiphytic).

- *Colletotrichum* present at all altitudes

**Figure 2**: Presence and absence chart of the OTUs by number in the three elevation groups. A black cell indicates presence and a white cell indicates absence.
• However, fungal communities substantially different at different altitudes. Lower altitude communities prevent efficacy of biological control.
CAN WE PREDICT SUCCESS?

- Success of an exotic microbe depends on:
  - Survival structures such as cysts, thick walled spores, etc
  - Saprotrophic ability (ability to feed on dead matter)
  - Degree of host specialization, the more specialized the harder it may be to establish
  - Phylogenetic distance of hosts (the closer the native and new hosts are, the easier the establishment)
  - Similar ecology: sometimes host jumps are facilitated by similar habitat/ecology
CAN WE PREDICT:

• Levels of the epidemic?

  – Density dependence: abundance of susceptible hosts

  – Genetic variation in host. In general it is assumed that genetic variation in host populations slows down epidemics, however backing data from natural ecosystems is missing. It could be that low genetic diversity associated with widespread presence of resistance may be more beneficial than genetic variability

- Selection of R genes (discussed previously)
Prediction of invasive ability of fungi (i)

- Ability to survive through different stages of introduction and frequency of transport (is pathway of introduction common and repeated).

- Overall reproductive potential is important because Transmission needs to be > mortality

- Adaptation through sexual reproduction, magnitude of asexual reproduction

- In case of competitor: ability to sporulate quickly (short latent period) is favorable
Prediction of invasive ability of fungi (ii)

- Adaptation through ability to generate diversity by reproduction between populations from different sources
- Ability to gain adaptive genes through hybridization with native relative species followed by introgression of genes
- Ability to lose costly but useless genes through purifying selection
- Epigenetic regulation or differential gene regulation
- Demographic phenotypic plasticity: a species may be a very successful invasive if it can shift from low plasticity (presumably in its native range) to high plasticity and then back to low plasticity during different stages of invasion
Prediction of invasive ability of fungi (iii)

- Spore size:
  - Smaller spore size will be selected for even if it negatively affects success of germination

- Ability to kill host:
  - If no alternate hosts, intermediate mortality may be selected rather than high levels of mortality. Also this is a trait not dependant on pathogen only