

Disease & Parasitism

BIOL/BOT 160 – Ecology

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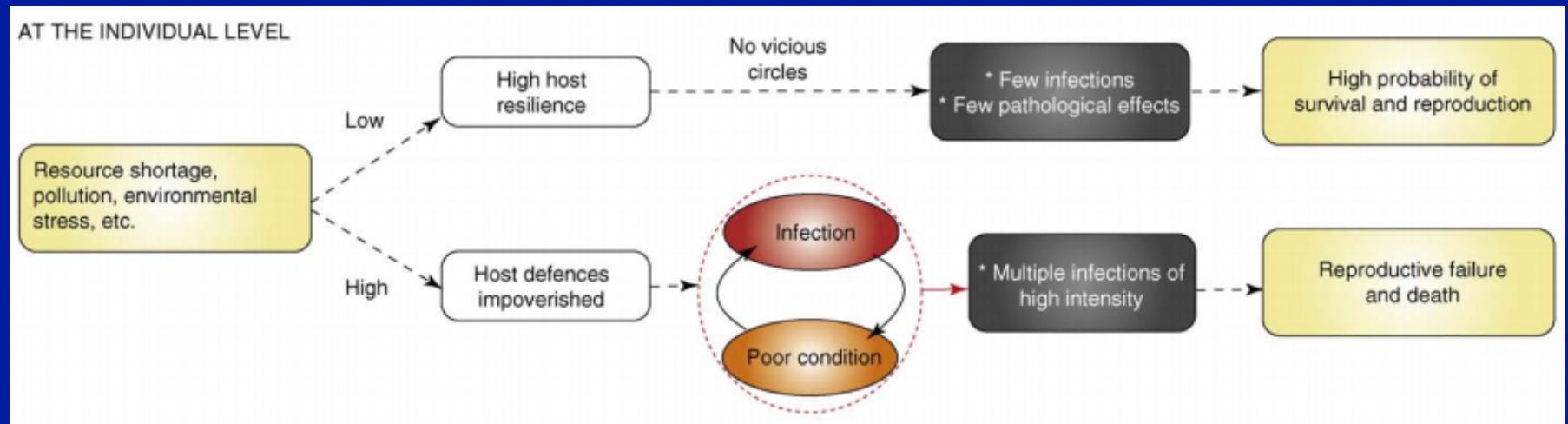
Learning objectives

- Students should be able to
 - Explain why infectious disease has an important role in population and community ecology
 - Describe how the pool of susceptible individuals, transmission rate, and infection duration impact the dynamics of an epidemic
 - Conceptually analyze the impact of virulence and transmission between species on coexistence & competitive exclusion
 - Explain how parasitic behaviors may evolve in social parasites

Infectious disease

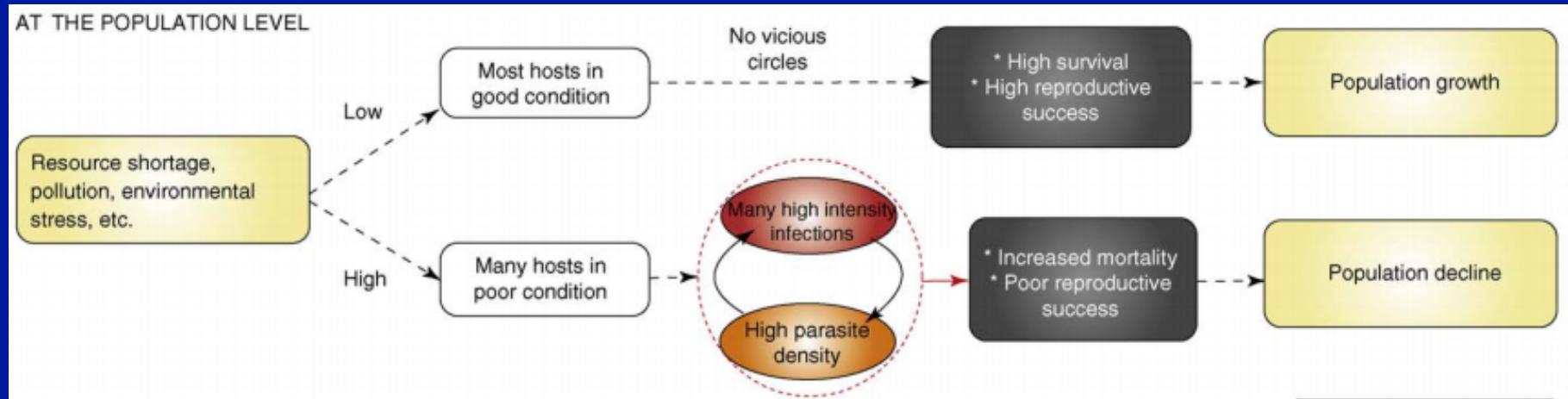
- Disease is the interaction between pathogenic organism and host organism
 - Usually viruses, bacteria, or fungi (but can be macroscopic as well)
- Lethal or sublethal effects
 - Death
 - Chronic ailments
 - Induce other illnesses in weakened state
- Virulence (i.e., harmfulness) is key

Why does disease matter ecologically?



Beldomenico & Begon 2009

Why does disease matter ecologically?



Beldomenico & Begon 2009

Types of parasites

- *Microparasites* are unicellular organisms
 - Viruses, bacteria, etc.
- *Macroparasites* are larger multicellular organisms
 - Worms, flies, and other insect parasites

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- *Microparasites* are unicellular organisms
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 - Worms, flies, and other insect parasites
- Both macro- and micro- parasites use the host as a habitat. You can think of disease transmission as analogous to metapopulation dynamics from the perspective of the pathogen

Classification: Sizes

- Microparasites
- Macroparasites



<http://www.biology.ccsu.edu/doan/ProjectHope/Malaria%20red.jpg>

Classification: Sizes

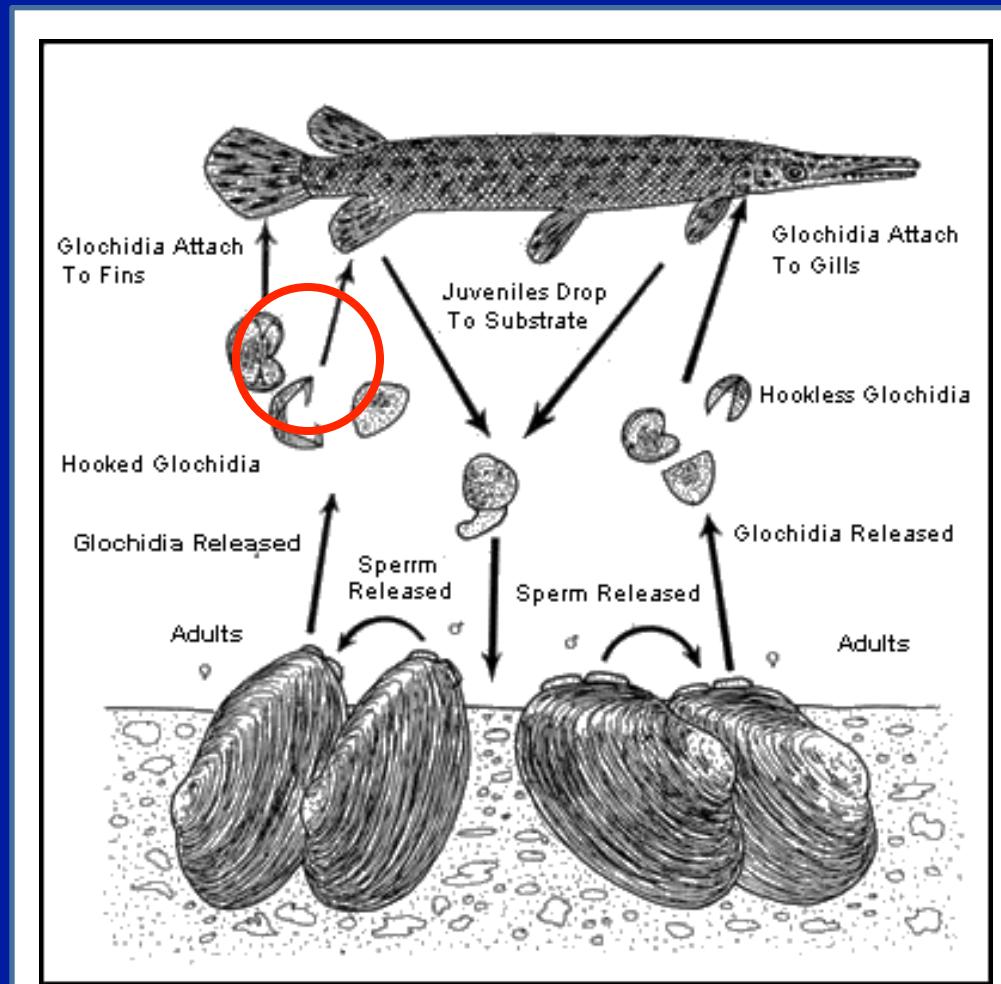
- Microparasites
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Classification:

Attach inside or outside

- Ectoparasites
- Endoparasites

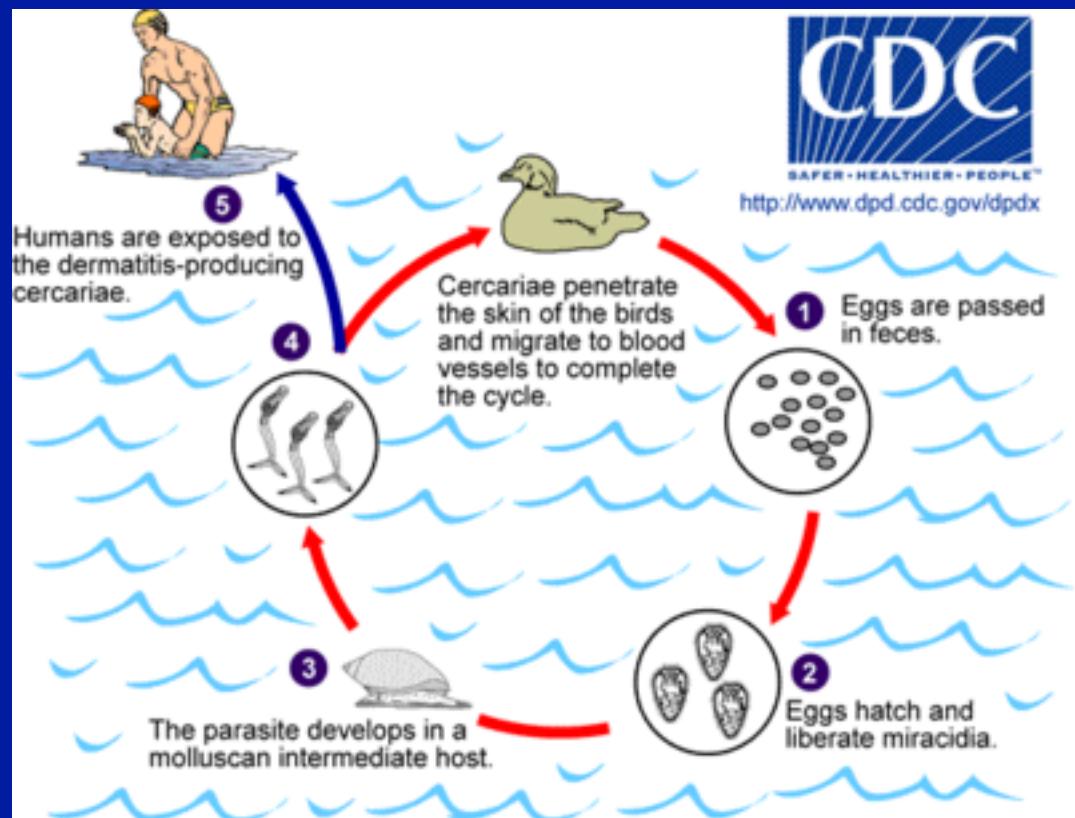


http://www.tpwd.state.tx.us/learning/texas_nature_trackers/mussel/images/02_mussel_reproduction.gif

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Attach inside or outside

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Parasitism – Ectoparasite Adaptations

- Method of attachment
 - SUCKERS
 - HOOKS



<http://successfulacademic.typepad.com/photos/uncategorized/denugemosquito.jpg>

Parasitism – Ectoparasite Adaptations

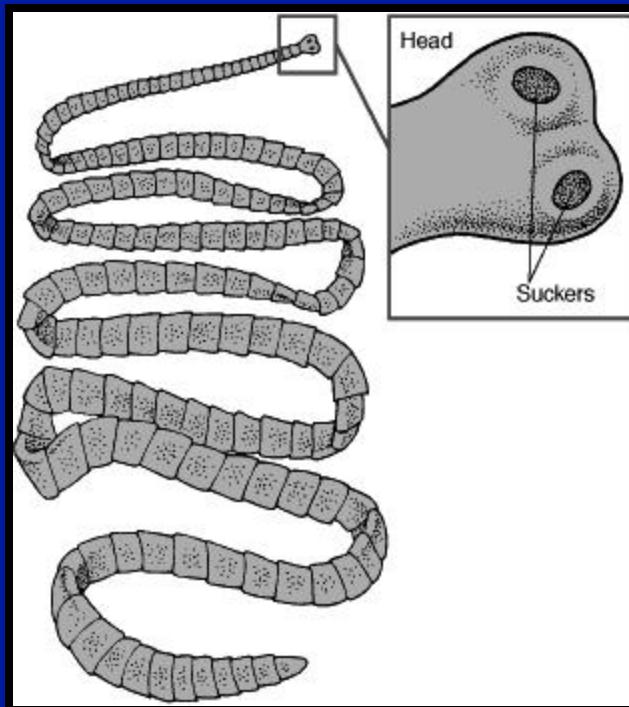
- Method of attachment
 - SUCKERS
 - HOOKS
- Penetrating skin
 - Produce anticoagulant



<http://successfulacademic.typepad.com/photos/uncategorized/denugemosquito.jpg>

Parasitism – Endoparasite Adaptations

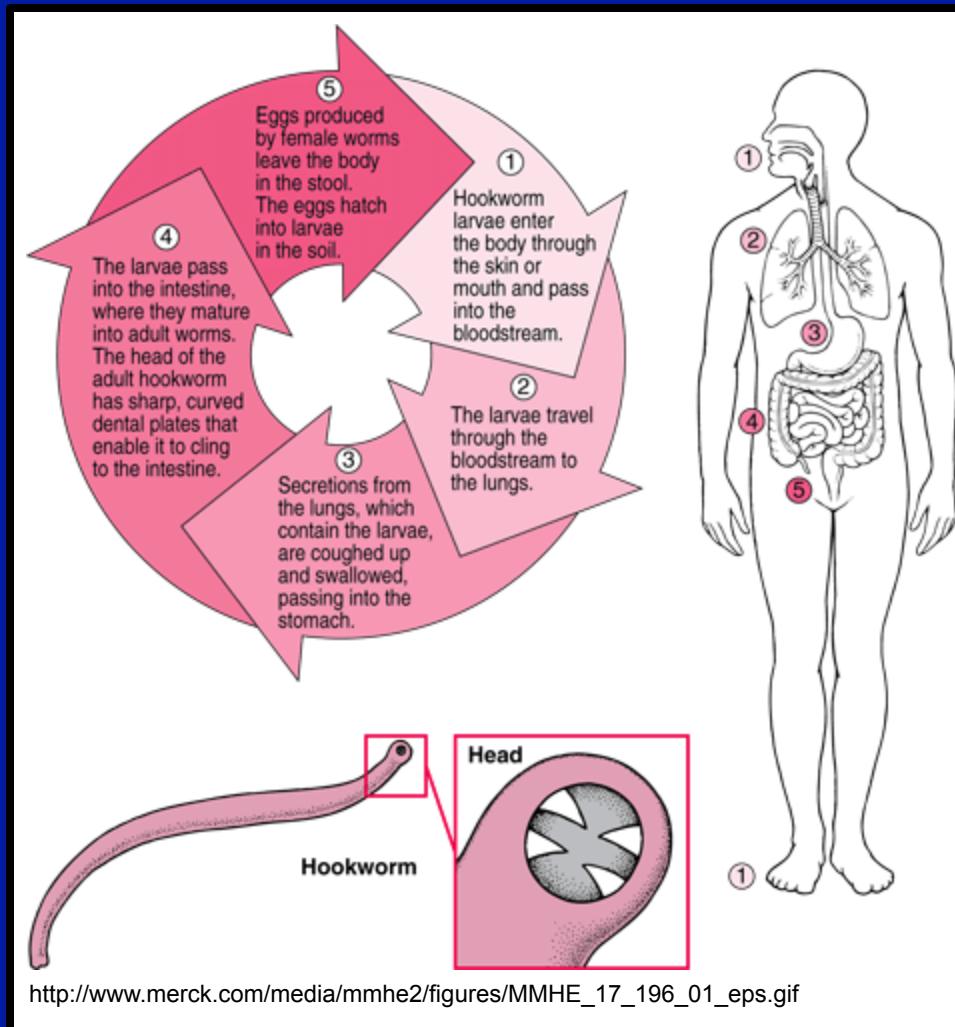
- Reduction in sense organs and digestive tracts (sometimes)



<http://www.uen.org/utahlink/tours/admin/tour/18753/18753cestoda.anat.jpg>

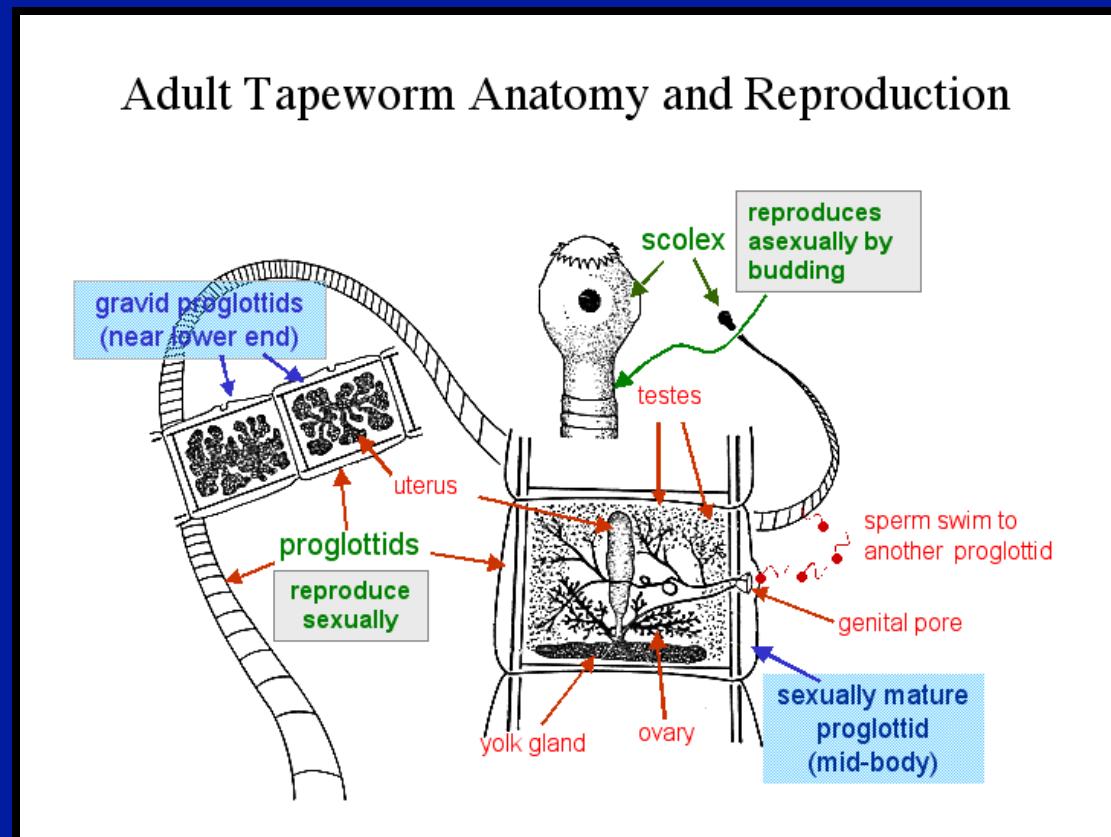
Parasitism – Endoparasite Adaptations

- Travel to intestines
 - Keep from being dissolved in the stomach



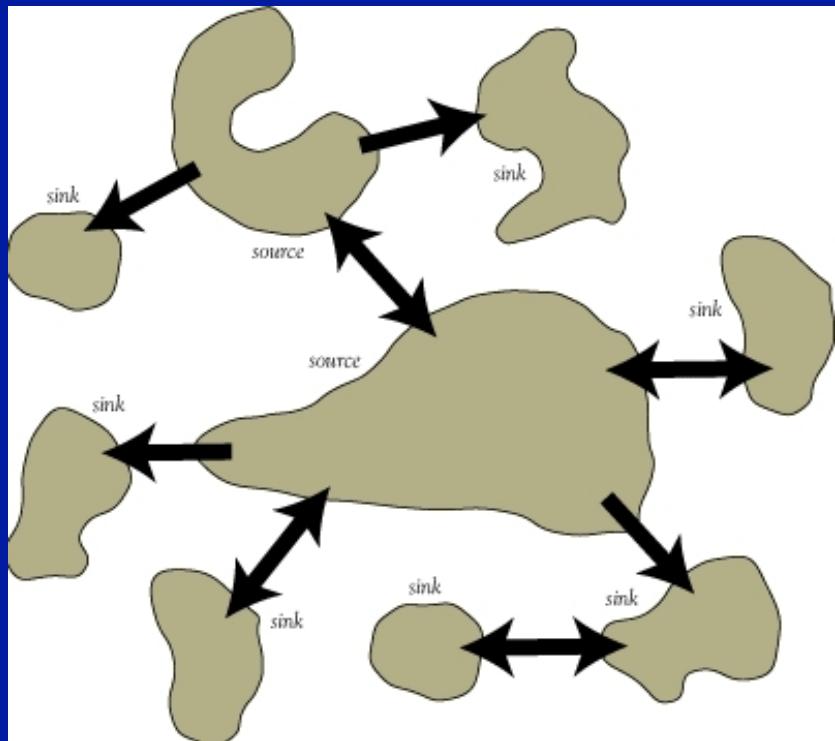
Parasitism – Endoparasite Adaptations

- Huge investment in offspring production

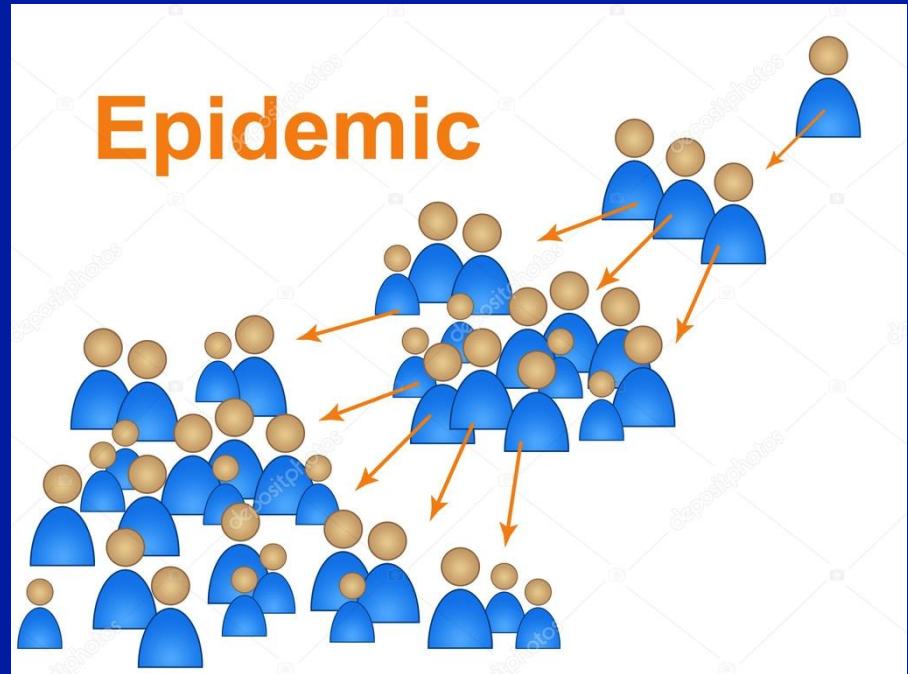
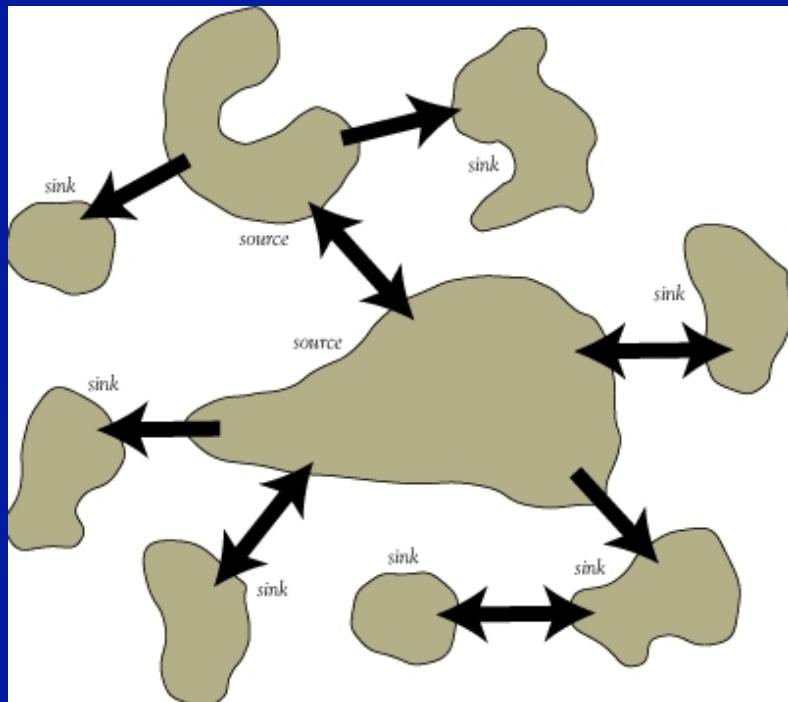


<http://www.cals.ncsu.edu/course/zo150/mozley/fall/Tapeworm1.gif>

Transmission dynamics



Transmission dynamics



What are the key epidemiological factors relevant to disease and parasites?

Virulence – influence on host health

Transmission – ability to jump hosts

H5N1 Influenza (aka Bird flu -> very high mortality but low transmission rate in humans)

H1N1 A influenza (aka Swine flu -> low mortality but high transmission rate in humans)

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Host symptoms – can be parasite's adaptations for reproduction or transmission

Example:

Rabies – symptoms enhance transmission (99% of humans who die from rabies are bitten by a rabid dog)

Disease and parasite dynamics

Why do measles cycle? (May and Anderson)

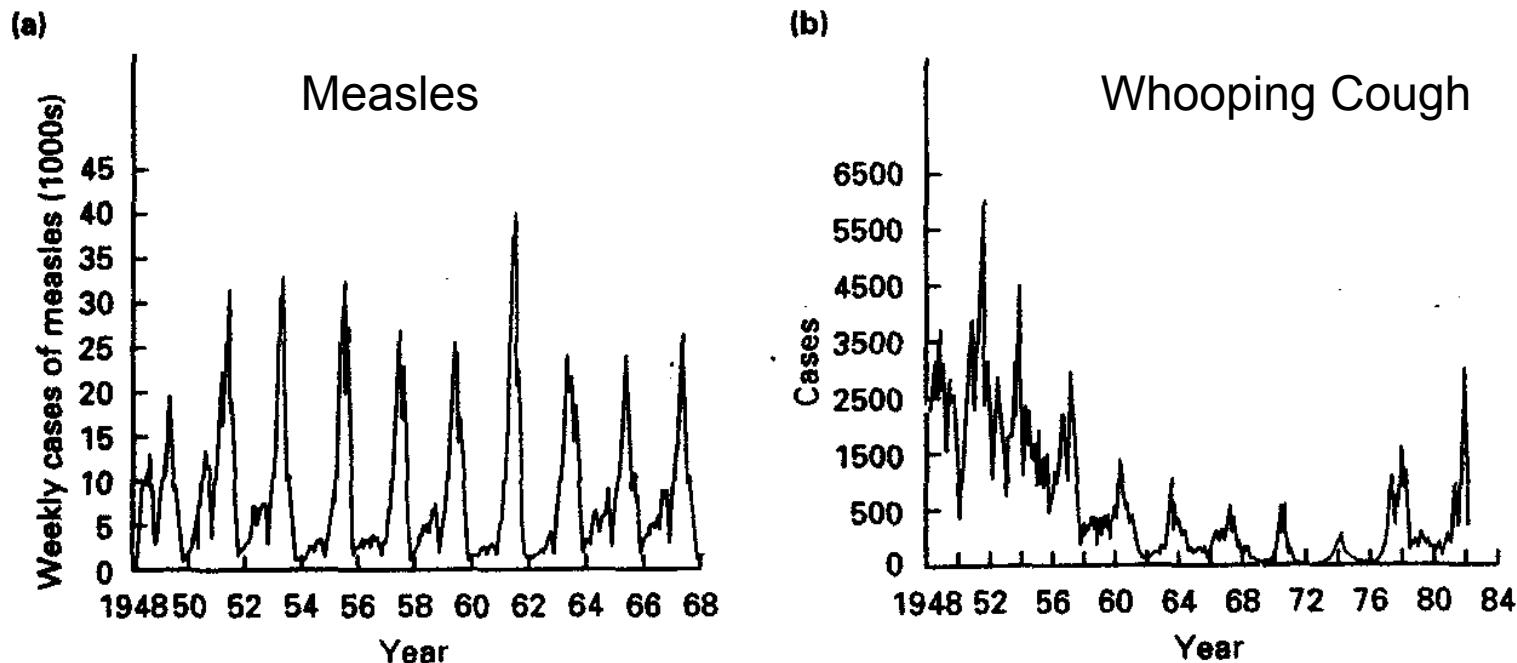


Figure 12.22 (a) Reported cases of measles in England and Wales from 1948 to 1968, prior to the introduction of mass vaccination. (b) Reported cases of pertussis (whooping cough) in England and Wales from 1948 to 1982. Mass vaccination was introduced in 1956. (After Anderson & May, 1991.)

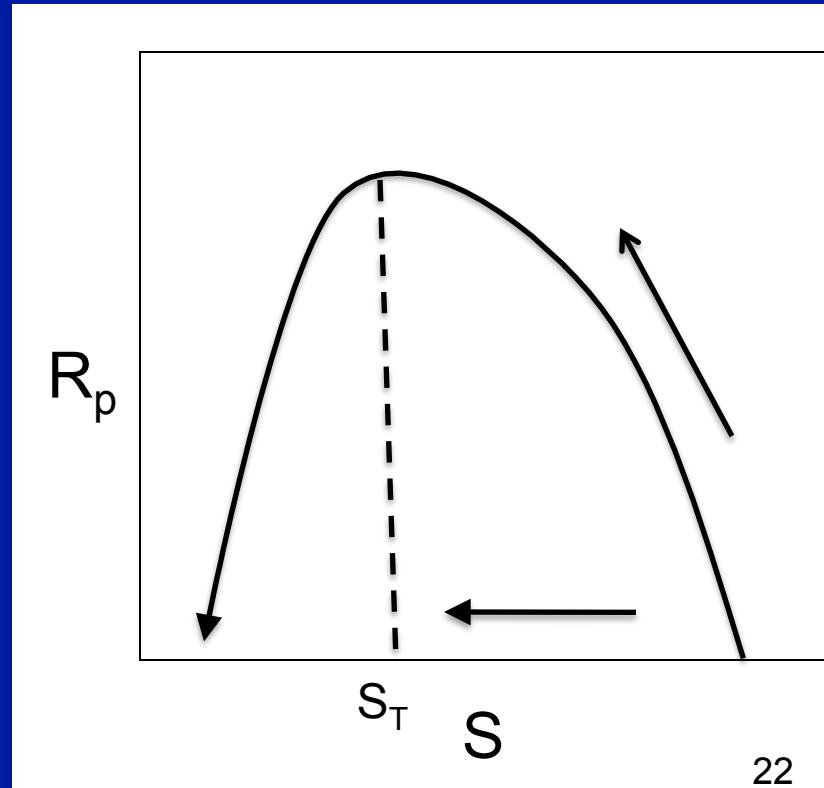
Disease and parasites

Why do measles cycle? (May and Anderson)

Focused on the spread of disease by looking at the rate of spread in terms of:

Reproductive rate of parasite (R_p)

- 1) Number susceptible (S)
- 2) Transmission Rate (β)
- 3) Infection period (L)



Disease and parasites

Implications of May/Anderson model:

1. Diseases with low transmission rates (β) or short infectious periods (L) can only persist in large populations

Measles example:

- High density in cities makes it possible for more diseases to persist
- S_T estimated at 300,000 under city conditions
- Unlikely to have been important to hunter gatherers

Disease and parasites

Implications of May/Anderson model:

2. The ability of organisms to acquire immunity can cause diseases/parasites to cycle
 - As disease spreads, $S \downarrow$ (fewer potential hosts available)
 - Eventually S falls below S_T and disease collapse

Disease and parasites

Implications of May/Anderson model:

2. The ability of organisms to acquire immunity can cause diseases/parasites to cycle
 - As disease spreads, $S \downarrow$ (fewer potential hosts available)
 - Eventually S falls below S_T and disease collapses
 - Get new pool of juicy susceptible hosts through births, S climbs back up again and cycle starts anew

Disease and parasites

Implications of May/Anderson model:

3. Point of immunization

- If we can bring S below S_T (i.e. $R_p < 1$), and keep it there
- Everybody benefits – herd immunity
- Don't need to immunize everybody in the population
- Example: whooping cough cycles before and after immunization

Effects of infection on community

- Suppose a pathogen can infect two species, each of which competes for some limiting resource.
 - Do you expect that the pathogen will
 - increase coexistence between the two species (i.e., make them weaker competitors),
 - decrease coexistence (i.e., make the competitive interaction stronger),
 - or it depends on the transmission & virulence of the pathogen?

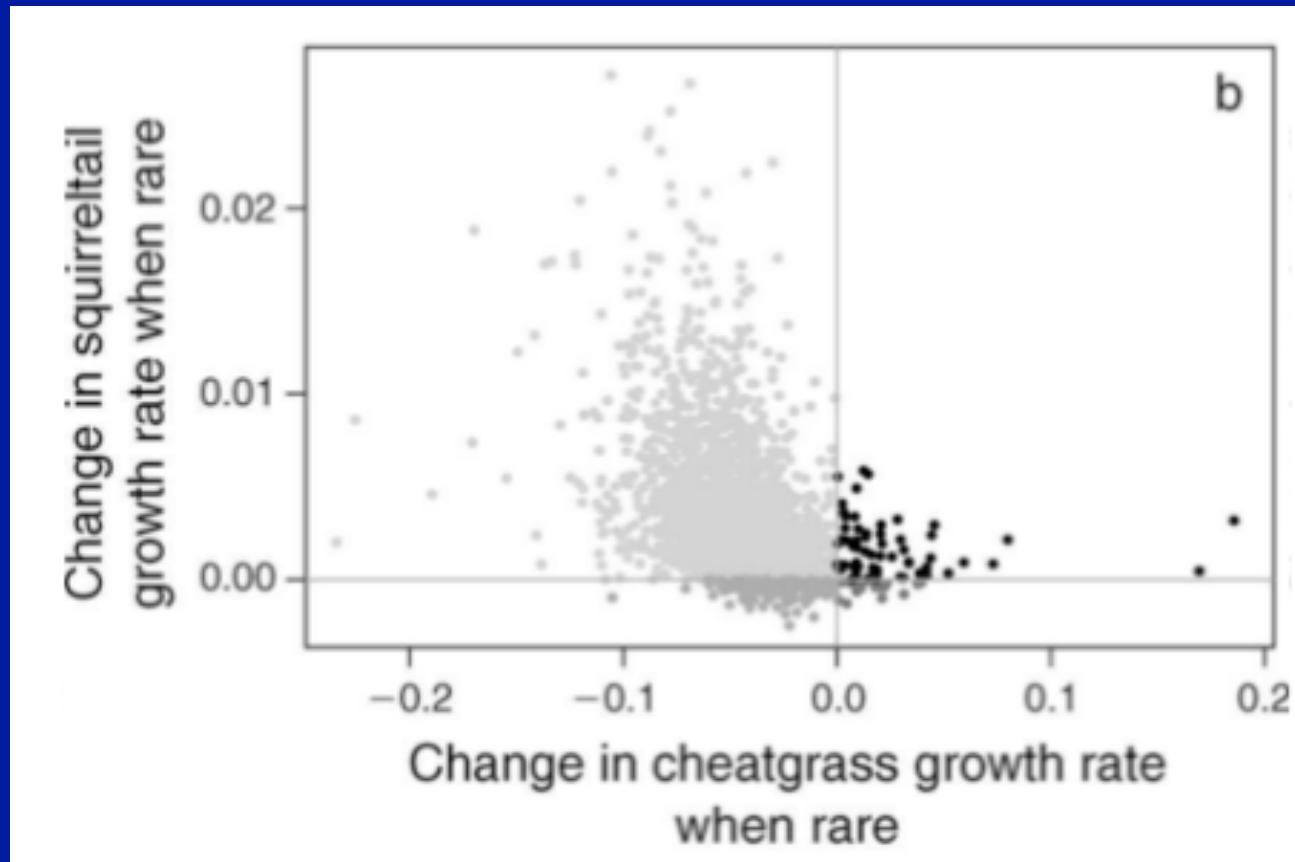
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Mordecai 2011

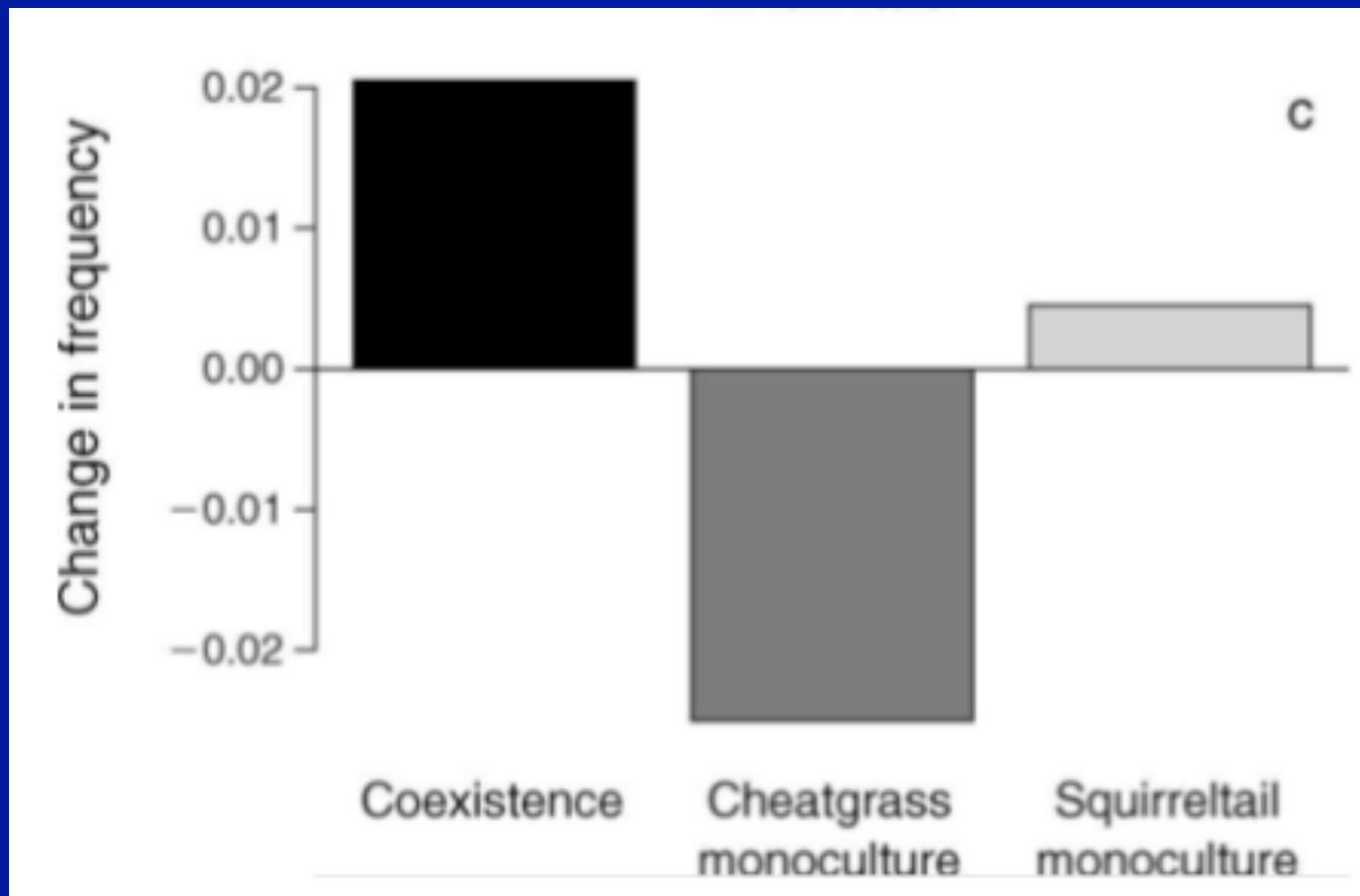
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BFOD



Mordecai 2011

BFOD



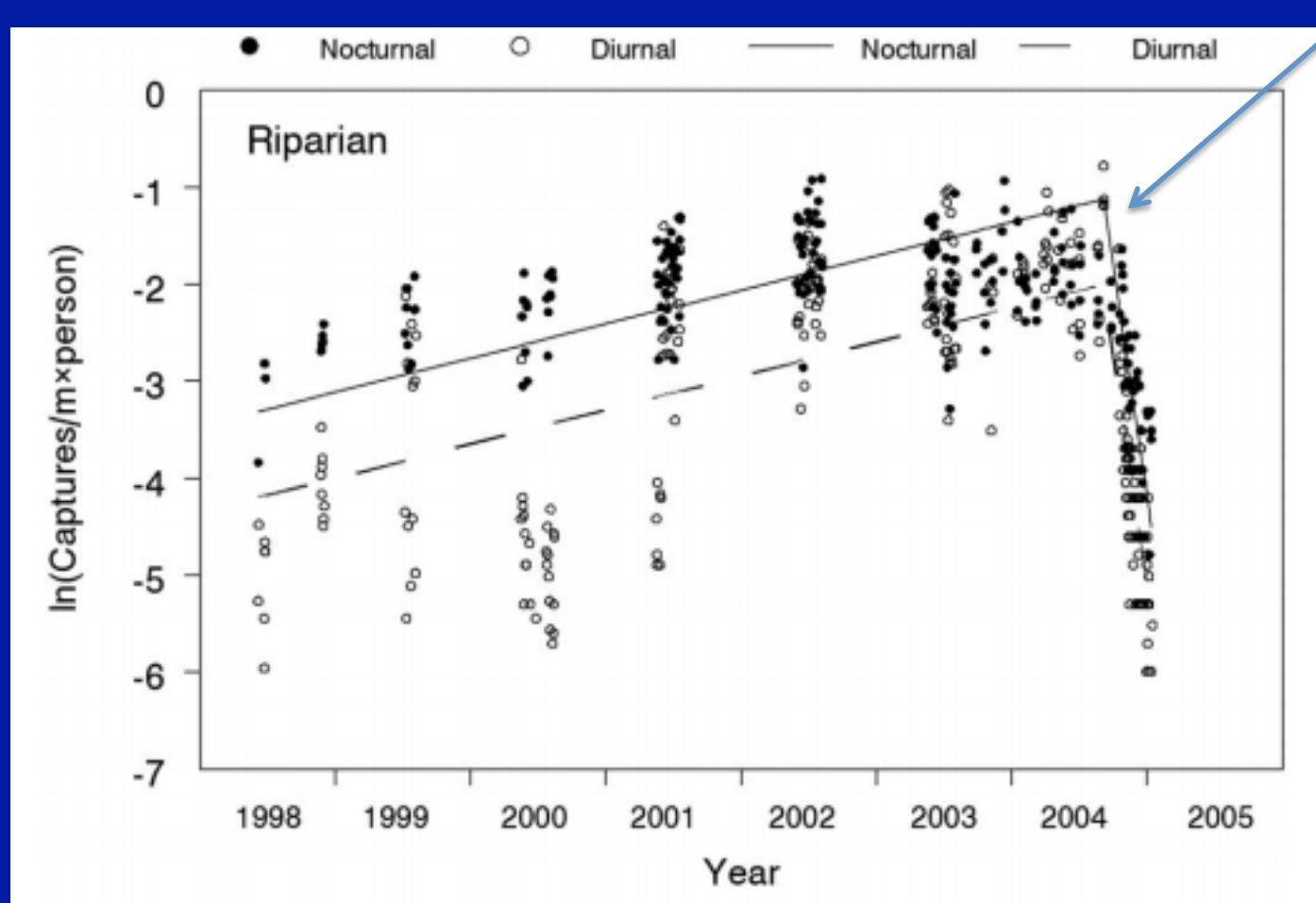
Mordecai 2011

Amphibian chytrid fungus

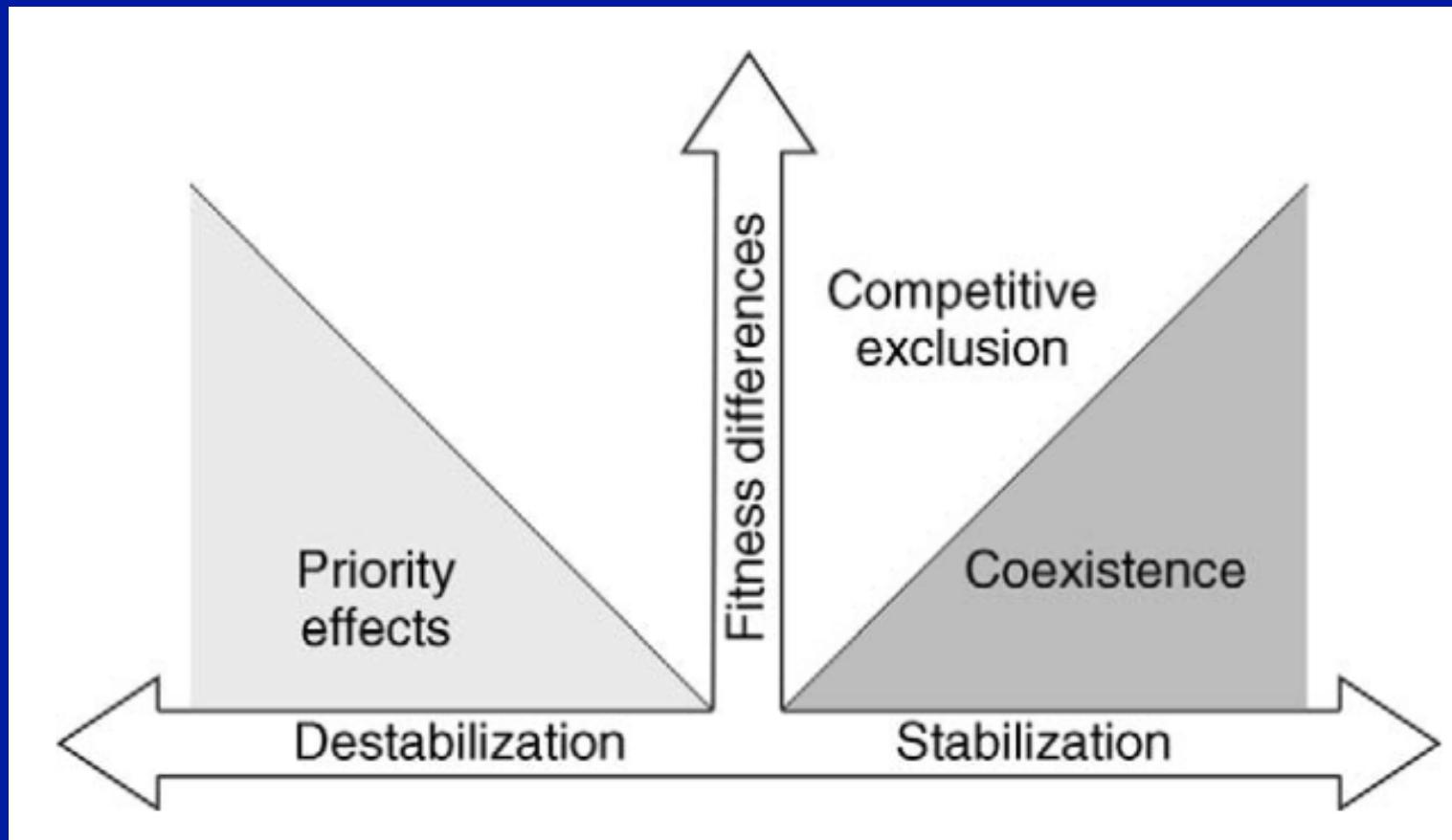


Chytrid fungus is driving amphibian declines

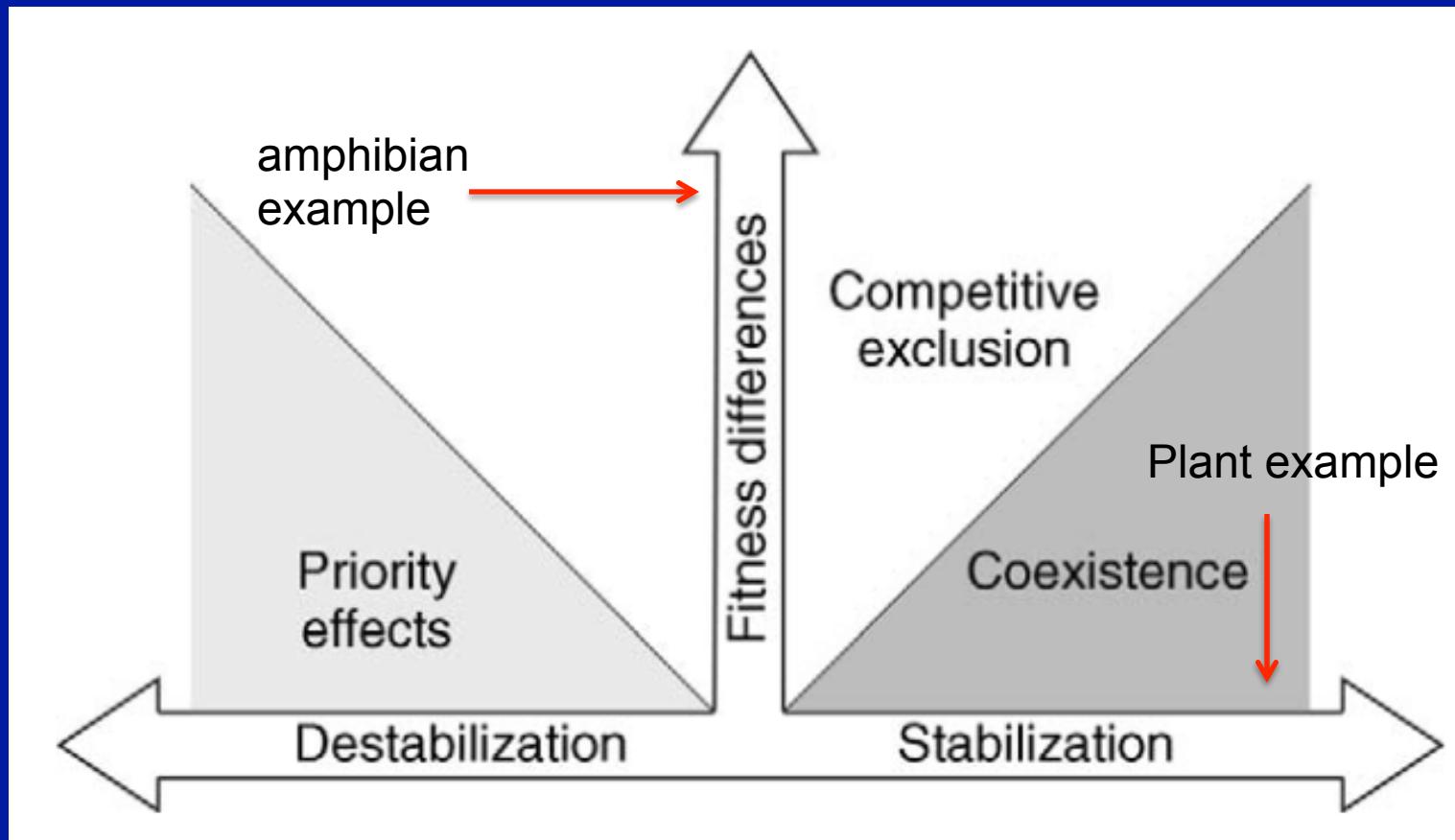
Chytrid fungus first detected



Why are different outcomes possible?



Why are different outcomes possible?



Evolution of parasites: red queen hypothesis

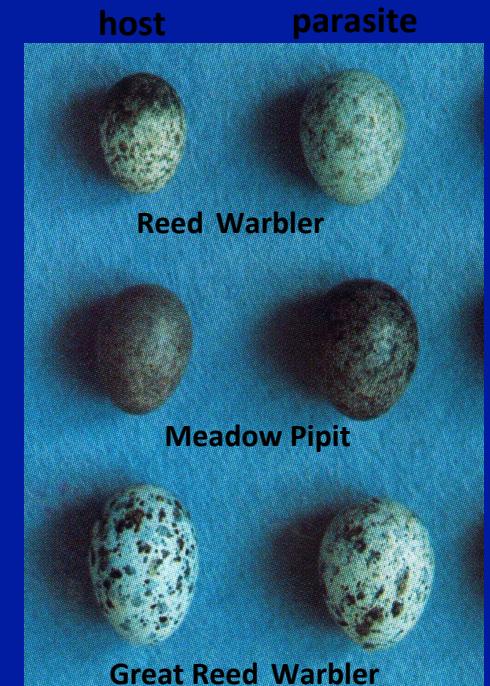
- Ecological arms race in coevolution
- The parasite is evolving virulence and host is evolving immunity
- Parasites evolve at a faster rate because generation times are short
- Host's genotypes change over time, present parasites with moving target
- Continual evolution and not stable equilibrium

Social/brood parasites

European cuckoo

- Has genetic races that specialize in different host species
- Lay mimetic eggs (resemble host's)

Common Cuckoo: example of antagonistic coevolution



(1) Parasitism is costly to hosts



(2) Hosts evolve egg recognition & rejection



(3) Parasites evolve egg mimicry & host specialization

How do social parasites come to exist?



- Suppose that an ancestral bird species evolves the ability to parasitize another bird species by leaving its eggs in the other species' nests
 - Why would this ability evolve? What does it suggest about the resources required to raise offspring?
 - Many years later, we observe that the ancestral bird species has diversified into many subspecies that parasitize the nests of specific types of birds and mimic their specific eggs. Can you propose an evolutionary mechanism for this diversification? (I.e., why has this mimicry evolved?)

Shiny Cowbird: parasitizes 200 host species



Brown-headed Cowbird egg in Chipping Sparrow nest



B. Lyon

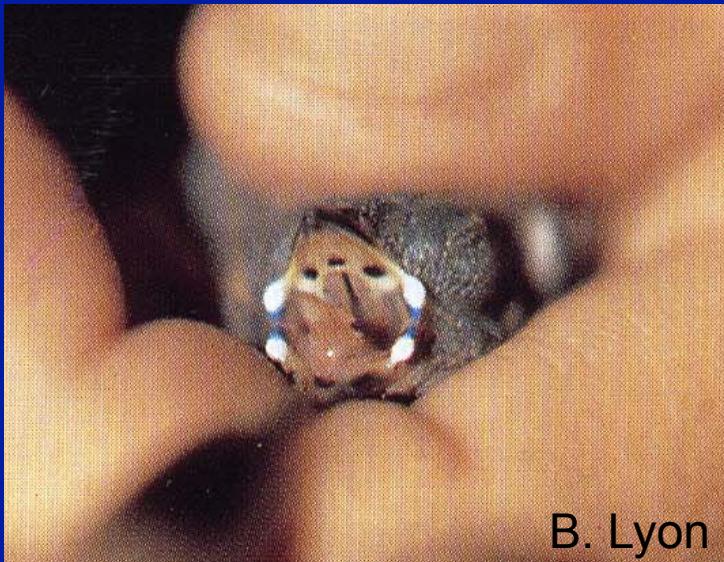
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Indigobirds

- parasitize finches
- specialist: 1 host species per parasite species

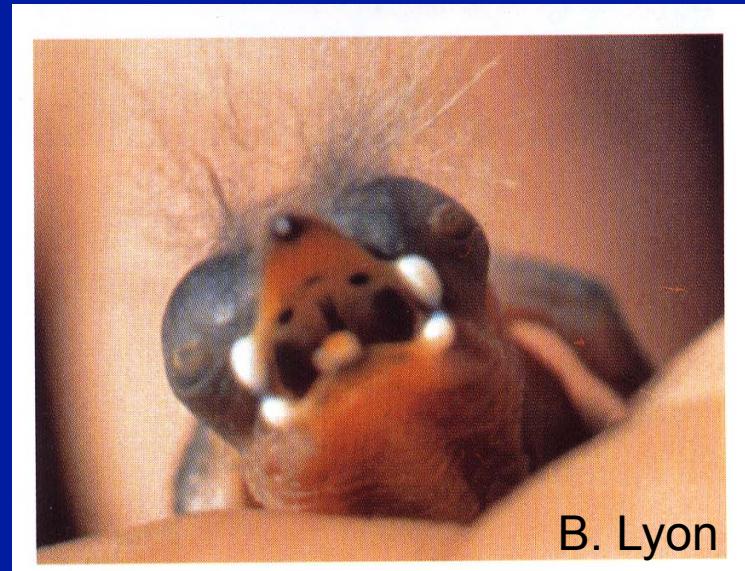


Parasitic chicks of indigobirds mimic mouths of host chicks



B. Lyon

Host
Red-billed Firefinch



Parasite
Village Indigobird

Checks and Balances

What keeps this strategy around

1. Reproductive Fitness of the Parasite
2. Becomes an Evolutionary Stable Strategy
3. Host tries to reduce it
4. Red Queen/arms race between species