

## Lecture 24: Parasitism, Disease and Epidemiology

Criteria defining parasitism

Relationship to other interspecific interactions

Classification

Endo and ectoparasites

Micro and macroparasites

Direct and indirect transmission

Coevolution

Immune responses and molecular mimicry

Reduced virulence

Epidemiology and population dynamics: SIR (Susceptible-Infected-Recovered) model

Vaccination

Disease and Conservation

The organisms considered "parasites" or "agents of disease" come from several kingdoms, including viruses, bacteria, fungi, protozoans, helminths (worms) and arthropods. So what exactly defines a parasite?

### ***Criteria defining parasitism:***

1. Parasite is physically dependent on host for food.
2. Parasite has higher reproductive potential ( $r_{max}$ ) and shorter generation time than host.
3. Parasite does not necessarily kill host. If host is killed, death is slow relative to predation.

### ***Relationships to other ecological interactions:***

As an ecological interaction between a pair of species, parasitism is similar to predation:

- (+/-) interaction
- parasites and predators both have the potential to regulate prey or hosts.

The effects of parasitism on population dynamics are complicated by two major differences from predation:

1. Infected hosts might recover, while prey never recover.
2. Recovered hosts may have immunity. Escaping a predator doesn't confer immunity - though *learning* by prey could have a similar effect.

An ecological interaction intermediate between parasitism and predation is ***parasitoidism***. Parasitoids have a stage that lives in or on the host for an extended period (like parasites), but they invariably kill the host (like predators). Parasitoid wasps like *Nasonia* are good

example. *Nasonia* injects its eggs into the bodies of blowfly larvae using a syringe-like ovipositor. As the wasps develop, they eat the fly larva and kill it before hatching out.

Other extreme: Virulence of parasite may ↓ through evolutionary time. Longer the host lives, longer parasite can exploit host's resources. Consequently, parasite might become commensal (+/0).

**Classification of parasites:**

1. **Endoparasites** - internal. Some internal but in lumen of gut, others in tissue or bloodstream,  
**Ectoparasites** - external.
2. **Microparasites** (viruses, bacteria, protozoans).
  - Small
  - Short generations
  - High rate of direct reproduction within infected host
  - Induce long-term acquired immunity (sometimes lifelong) in host that survives
  - Duration of infection usually short.

**Macroparasites** (helminths and arthropods).

- Larger
  - Longer generations (but still short relative to host)
  - Little or no direct reproduction in host
  - Acquired immunity depends on number of parasites, usually short term
  - Infection usually long-term
3. **Direct Transmission.** Single stage of parasite passes directly from definitive host to definitive host, with no intermediate host or vector
    - Physical contact of hosts - e.g. gonorrhea
    - Inhalation - e.g. common cold virus
    - Ingestion - e.g. pinworm
    - Penetration of skin - e.g. hookworm

The 4 routes of transmission above are '**horizontal**' - from one independent host to another. Some pathogens are also transmitted **vertically**, so that offspring of infected mother is born with infection (rubella, syphilis, AIDS).

**Indirect Transmission.**

- **Vector transmission:** Host passes one stage of parasite to a **vector** (usually a biting insect - ticks, fleas, flies, mosquitoes). Parasite produces an infectious stage while in the vector. Vector then passes on infection to new hosts.
- Example is malaria, once a major killer in the eastern U.S., as far north as Washington DC, and still a major killer in the tropics. *Plasmodium* (protozoan) is vectored by *Anopheles* mosquito, affecting most vertebrates.

(Overhead - Fig 12.1 Begon et al: Plasmodium life-cycle)

- **Indirect host transmission:** Parasite reproduces sexually in **definitive host**. Eggs shed by host (usually in urine or feces). Eggs develop into stage that infects **intermediate host**. Asexual reproduction in intermediate host produces free living stage that infects definitive host (often by penetrating skin).
- Example is *Schistosoma mansoni* Schistosomiasis (bilharzia) is a major tropical disease of humans, causes organ damage by migrating from skin through heart and lungs to liver. Intermediate host is a snail, *Biomphalaria*.

(Overhead - Fig. 13.1 Cohen chapter in May 1976: Schistosome life-cycle)

### **Parasite-Host Coevolution.**

**Coevolution** is the joint evolution of 2 or more species, due to strong ecological interaction, so that evolutionary change in one species drives evolutionary change in the other.

Many species of parasite are specialized to infect a single species of host. Once a parasite is sufficiently **host-specific**, the strength of the ecological interaction may lead to increased specificity, via coevolution. The intrxn is strong for the parasite because it has already 'invested' in beating the defences of a given host species. The intrxn is strong for the host b/c the entire burden of that parasite now falls on that host species and no other.

Produces coevolved pairs of hosts and parasites - phylogeny of parasite is same as phylogeny of host.

(Overhead: Fig 15-14 Pianka - primates and their pinworms)

Types of coevolution:

1. **Molecular mimicry.** Proteins on surface of pathogen = antigens. Specific antibodies are produced by the host's immune system to recognize these antigens. Antibodies then mediate many cellular processes that either kill pathogen cells, kill pathogen within infected host cells, or kill infected cells.

(Overhead: Figs. 2.16 & 2.17 Roitt - actions of antibodies in specific immunity).

In a coevolved pathogen, cell-surface proteins evolve to resemble the host's proteins - so they don't provoke specific immunity.

2. **Reduced virulence.** **Virulence** refers to the severity of symptoms a parasite induces. It is not in the interests of the parasite to kill its host.
  - When the host dies, so does the parasite, in some cases.
  - At a minimum, the parasite can no longer exploit the host's resources.

- Might kill host before transmission to new host.

Some evidence that virulence decreases over evolutionary time, for a given host-parasite combination. Best evidence is from pathogens introduced to naïve host populations.

Example is European rabbit (*Oryctolagus*) and *Myxoma* virus (from S. America) in Australia. *Myxoma* only causes mild illness in S. American (*Sylvilagus*) rabbit host, with which it evolved, but killed > 99% of naïve *Oryctolagus* when introduced.

### ***Population dynamics of infectious diseases***

Predation and parasitism are both (+/-) ecological interactions, so it might seem that you could apply the models of population dynamics developed for predator/prey interactions (see lectures 21-23). But parasitism is more complex because:

1. Infected hosts might recover, while prey never recover.
2. Recovered hosts may have acquired specific immunity.

The effects of infectious diseases on host populations are usually modelled with an SIR model, which describes changes in the number of hosts that ***are susceptible, infected, and recovered/resistant***.

(Overhead: Fig 3 Anderson & May 1979. Flow diagram for SIR model).

### ***SIR model assumes that:***

1. Microparasitic life-cycle, i.e. short-term infection with high reproductive rate in host, induces long-term immunity in hosts that don't die.
2. No vertical transmission (hosts are uninfected at birth).
3. No latency or carrier state (host develops symptoms immediately, and can transmit disease immediately)
4. Host population grows exponentially in absence of parasite.
5. Infection occurs randomly, in proportion to density of infectious and susceptible individuals.

### ***Model parameters:***

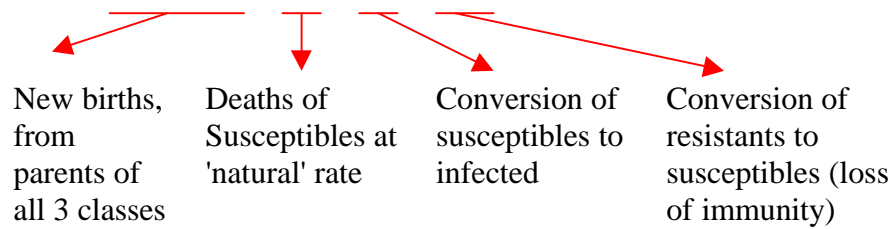
N = density of hosts = S + I + R  
S = density of susceptible hosts  
I = density of infected hosts  
R = density of recovered and resistant hosts

b = per-capita birth rate of hosts  
d = per-capita 'natural' death rate of hosts (all causes other than disease)

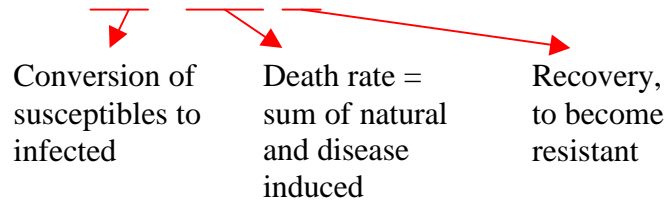
- $\alpha$  = per-capita disease-induced death rate of infected hosts
- $\beta$  = transmission rate between hosts
- $\nu$  = per-capita recovery rate of infected hosts
- $\gamma$  = rate of immunity loss

From the flow-diagram, the equations describing the population dynamics of infection are:

$$dS/dt = b(S + I + R) - dS - \beta SI + \gamma R$$



$$dI/dt = \beta SI - (\alpha + d + \nu)I$$



$$dR/dt = \nu I - (d + \gamma)R$$

