

Lecture 24: Parasitism and Disease

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

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.

} extreme is virus - not even alive outside of host

**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

generally from predators but parasitism

OVERVIEW

1. calculate  
2. regulate

1. recovery  
2. immunity

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).

FIN.

**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  
indirect host (+ vector sometimes)

- **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.

Endo/ecto

Micro/Macro

A's. int  
for  
modelling  
of effect  
on host  
pop

Direct/indirect

vert  
horizontal

Malaria  
overhead

O-HEAD  
→

(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*.

O-HEAD  
end of classification  
→

(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.

Copy  
Range  
Tree  
↑  
1  
2

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 defenses 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.

} parasite view  
} host view

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

O-HEAD

(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).

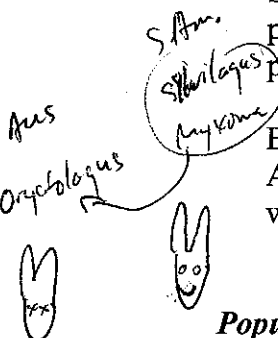
In a coevolved pathogen, cell-surface proteins evolve to resemble the host's proteins - so they don't provoke specific immunity.

O-HEAD

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.

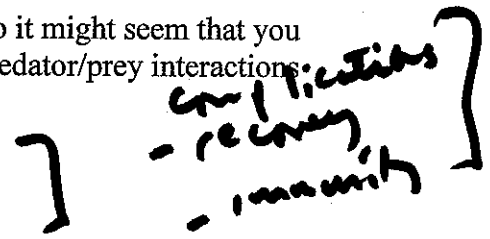
Example is European rabbit (*Oryctolagus*) and *Myxoma* virus (from S. America) in Australia. *Myxoma* only causes mild illness in S. American (*Sylvilagus*) rabbit host, with which it evolved, but killed > 99% of naïve *Oryctolagus* when introduced.



### 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 modeled 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)

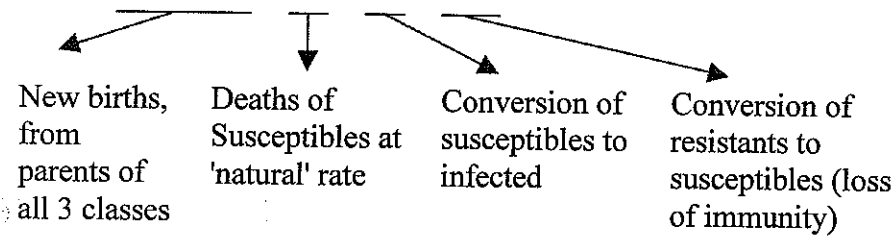
OVERHEAD

- $\alpha$  = 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:

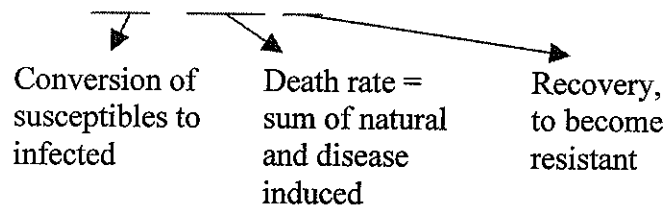
***Susceptible:***

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



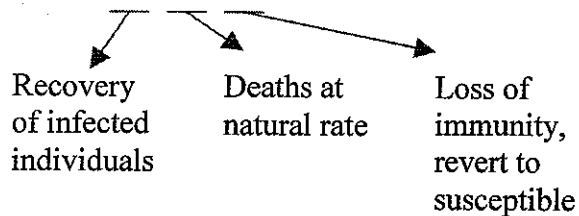
***Infected:***

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



***Recovered and Resistant:***

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



① Derivation of  $R_0$  for infection persisting in a population

$$\frac{dI}{dt} = \beta SI - (\alpha + d + \nu)I$$

set  $\frac{dI}{dt} = 0$  } This implies  $R_0 = 1$  for parasite.

$$0 = \beta SI - (\alpha + d + \nu)I$$

$$(\alpha + d + \nu)I = \beta SI$$

$$1 = \frac{\beta SI}{(\alpha + d + \nu)I}$$

$$1 = \frac{\beta S}{(\alpha + d + \nu)}$$

$$R_0 = 1 = \frac{\beta S}{(\alpha + d + \nu)}$$

— creation of I's
— persistence (loss) of I's

② What host pop size will allow endemic infection to persist?

Set  $R_0 = 1$ , solve for S, threshold # susceptibles.

$$S_T = \frac{\alpha + d + \nu}{\beta}$$

} what are  $\alpha, d, \nu, \beta$

These three equations give the number of host individuals entering and leaving each of the three classes - susceptible, infected and recovered. The equations can answer:

1. Will a microparasitic disease ~~will~~ persist in a host population *endemically* (without reinfection from another host species)?
2. What host population size is needed to sustain endemic infection? Culling to hold host population below this threshold will eliminate the microparasite.
3. Will a microparasite regulate host numbers?
4. What intensity of vaccination is needed to eliminate a microparasite from the host population?

1. *Will microparasite persist as endemic infection?*

Endemic disease persists if its  $R_0 > 1$ . In this case,  $R_0$  measures secondary infections per primary infection - if each infection produces  $>1$  secondary infection in the host population, the disease will persist endemically.

$$R_0 = \frac{\beta S}{\alpha + d + v}$$

$R_0$  increases as transmission rate & # of susceptible hosts increase

$R_0$  increases as persistence of infectious carriers increases. Persistence of carriers is inverse of disappearance rate, due to natural death (d), disease-induced death ( $\alpha$ ), and recovery (v).

So, diseases are more persistent if:

- Transmission rate ( $\beta$ ) is high
- Virulence ( $\alpha$ ) is low
- Hosts are long-lived (d is low)
- Single infections are persistent (v is low).

*implications for conservation*  
*recovery rate*

3 of 4 at least  
BISON ;  
BRUCELLA  
ABORTUS

2. *What host population size will sustain endemic infection?*

If the density of hosts drops below a threshold, then it becomes unlikely that an infected host will encounter a susceptible host while still infectious. What is this threshold?

Set  $R_0 = 1$  and solve for S. This is the threshold number of susceptible hosts, below which endemic infection cannot persist.

$$S_T = (\alpha + d + v) / \beta$$

Example: rinderpest in Ngorongoro crater. Rinderpest virus is in genus *Morbillivirus*, an ancestor of measles virus, which kills ungulates by causing severe diarrhea. Evolved in Asia, and was kept out of sub-Saharan Africa because ungulates do not naturally cross the Sahara. Introduced in 1889 by cattle brought by Italian army. Swept through Africa over next 10 years, killed ~5 million cattle and tens of millions of wild ungulates. Killed as much as 90% of African buffalo.

OHOM

(Overhead: Fig 16.1 Dobson & May in Soule 1986: spread of RPV)

Remained endemic in Serengeti and Ngoro<sup>2</sup> wildlife until controlled by vaccination of cattle in early 1960's. With control, the wildebeest population then increased 6x, buffalo 5x over next 2 decades.

(Overhead: Fig 1.1 Sinclair - map of Sgeti/Ngo)

OHOM

(Overhead: Fig. 1.2 Sinclair in Sinclair & Arcese 1995: increase of WB)

Lion and spotted hyena populations tracked increases in WB and buffalo.

(Overhead: Fig 15.7 Hanby et al in Sinclair & Arcese 1995: lion pop size).

Rinderpest had huge impact on large mammal community!

A

RPV persists in Ngorongoro cattle, threat of epidemic b/c most wildlife are now *naïve*, meaning never exposed to the disease and therefore not immune.

Dobson (1995) estimated  $S_T \approx 20,000$  to 50,000 for Ngorongoro cattle, using above equation. In 1982,  $S_T > 50,000$ . Outbreak occurred. Controlled by 99% vaccination of the cattle population (small due to die-off) in next year.

OHOM

(Overhead: Fig. 23.8 Dobson in Sinclair & Arcese 1995: 1982 outbreak)

### 3. Will a microparasite regulate host numbers?

Host population growth rate is:

$$dN/dt = (b - d)N - \alpha I$$

Exponential growth

Extra mortality due to disease

conceptually

Substitute:

$$r = b - d$$
$$y = I/N$$

$r$  = intrinsic rate of increase for host population  
 $y$  = prevalence of infection in host population

So that host population growth is:

$$dN/dt = (r - \alpha y)N$$

Microparasite regulates host **if virulence ( $\alpha$ ) is high enough.**

Quantitatively,  $dN/dt$  will go to zero (parasite regulates growth), when

$$\alpha > r \left( 1 + \frac{d}{\gamma} \right)$$

Regulation by disease is more likely when:

- Host intrinsic rate of increase is low
- Recovery rate is low
- Natural rate of mortality of hosts is high
- Loss of immunity is fast, or there is no immunity

Example: Larch budmoth and granulosis virus. Using equation above and the 3 basic SIR equations, can solve for population size of host. The algebra is complicated, but shows that actual population dynamics of host match the model predictions. Suggests that virus regulates moth numbers.

(Overhead: Figure 5 May 1983 Am Sci 71:40).

4. **What intensity of vaccination is needed to eliminate a microparasite from the host population?**

Vaccination programs can eradicate a disease if they reduce the number of susceptible hosts below the threshold that allows an endemic infection.

$$R_0' = (1-p)R_0$$

Where  $p$  = proportion of hosts vaccinated.

Goal is to get  $p$  large enough that  $R_0' < 1$

$$R_0' = 1 = (1-p)R_0$$

Solve for  $p$ :

$$p = 1 - 1/R_0$$

big  $R_0 \rightarrow \frac{1}{R_0}$  is small  $\rightarrow$  must vaccinate more hosts (bigger  $R_0$ )

substituting in for SIR eqn's.

Bison - Brucella eqn's - regulation of B. on NOT necessary

RED GROUSE, MOUNTAIN GOAT

OVERHEAD

If  $R_0$  for parasite is high, then a high proportion of hosts must be vaccinated to eradicate disease.

Can measure  $R_0$  directly (equation in section about conditions for endemic infection).

Rough estimate is:

$$R_0 = 1 + L/A$$

L = life expectancy of host  
A = average age of infection

recall exact def:  $R_0 = \frac{\beta S}{(\alpha + d + \nu)}$

need very good data for given pathogen + best estimate

(Logic of this - parasite with high  $R_0$  will infect most host individuals while young)

Example of eradication by vaccination is smallpox in humans:

OVERHEAD

(Overhead: Table 1 May 1983 Am Sci -  $R_0$  and p for human pathogens)

Same logic used in wildlife conservation and management to eradicate diseases:

CDV vaccination in Serengeti

(Overhead: Munson et al Nature.)

Rabies vaccination in European red foxes.  
Aerial baits

**Cordon Sanitaire** ('clean band') or firebreak approach - remove or vaccinate all susceptible individuals from a band in front of epidemic front:

OVERHEAD

(Overhead: Fig 16.1 Dobson & May in Soule 1986: spread of RPV, mange, *Diadema*)

(Overhead Fig. 2 Dobson & May in Soule 1986)

40-100 km in fox rabies

50-70 km in rinderpest (this prevented Rinderpest from entering Rhodesia in 1937)

Note that these bands are very wide → to halt an epidemic wave:

- Need a massive vaccination campaign, or
- Have to remove animals from area larger than most protected areas.