Plant-Herbivore

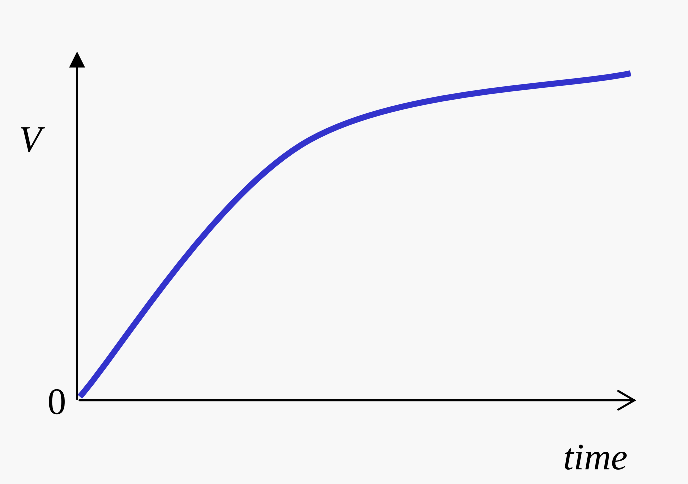
- consume small amount of many different plant species
- consume a lot during life to obtain sufficient amount of N
- grazers, granivores, frugivores, herbivores
- plants are not killed only reduced in biomass
- top-down control – herbivore abundance is regulated by enemies
- bottom-up – herbivore abundance is regulated by quantity and quality of plants

Herbivory-regrowth model

- Turchin (2003)
- assumptions

- continuous herbivory
- herbivore is polyphagous
- plant biomass is homogenous
- functional response Type II
- herbivore density is constant
- only small quantity of biomass is removed

- hyperbolic biomass growth

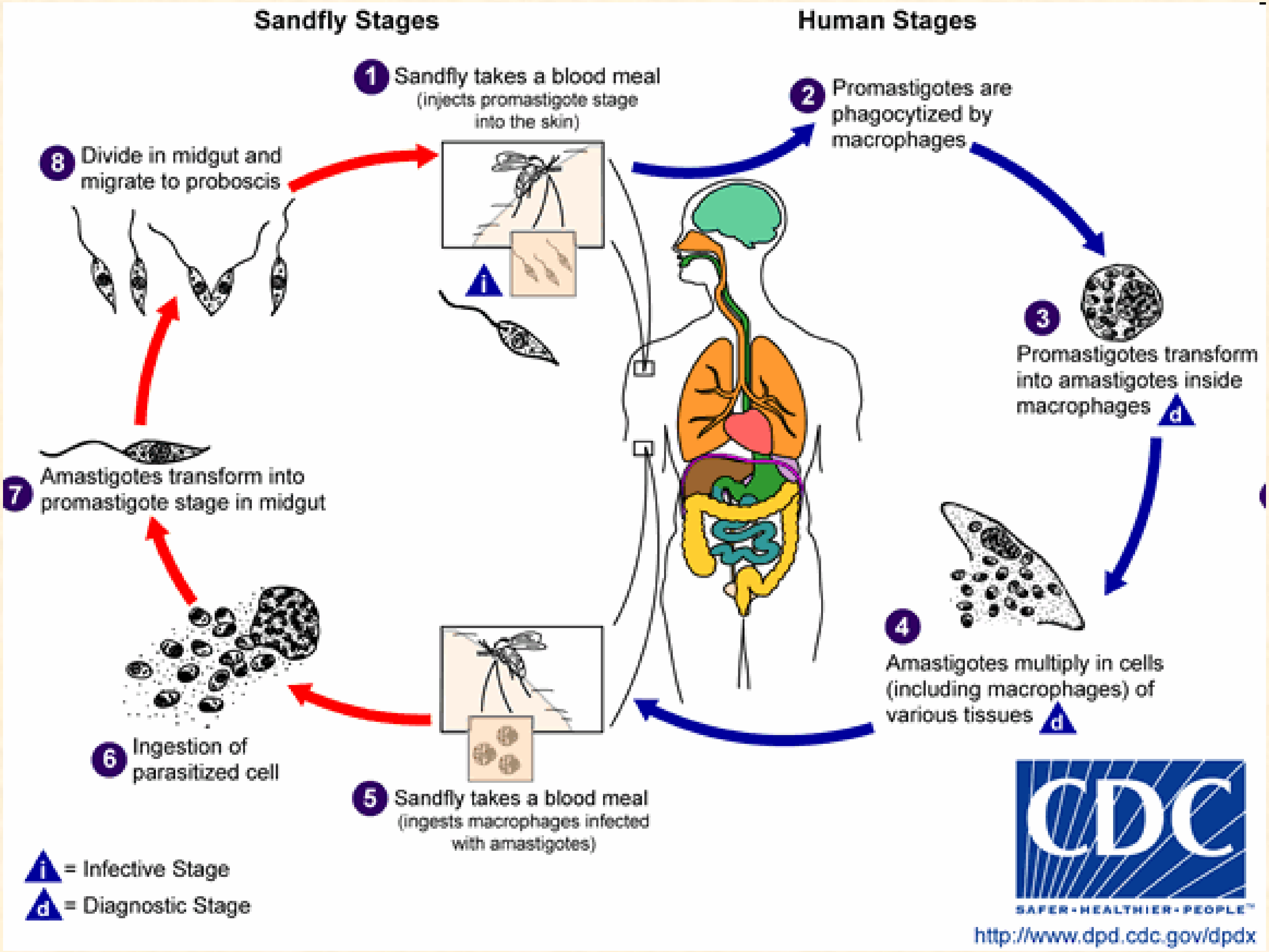


V .. plant biomass
N.. herbivore density
r.. intrinsic rate of increase
K.. carrying capacity
f .. efficiency of removal
T_h.. handling time

$$\frac{\mathrm{d}V}{\mathrm{d}t} = r\left(1 - \frac{V}{K}\right) - \frac{fNV}{1 + fNT_h}$$

Host-Pathogen

Leishmania

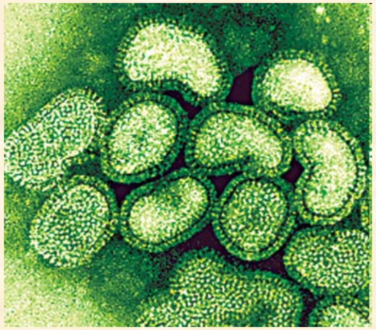


Sandfly Stages
Human Stages
1 Sandfly takes a blood meal (injects promastigote stage into the skin)
2 Promastigotes are phagocytized by macrophages
3 Promastigotes transform into amastigotes inside macrophages
4 Amastigotes multiply in cells (including macrophages) of various tissues
5 Sandfly takes a blood meal (ingests macrophages infected with amastigotes)
6 Ingestion of parasitized cell
7 Amastigotes transform into promastigote stage in midgut
8 Divide in midgut and migrate to proboscis
i = Infective Stage
d = Diagnostic Stage
CDC
SAFER·HEALTHIER·PEOPLE™
http://www.dpd.cdc.gov/dpdx

Agents

- microparasites: viruses, bacteria, protozoans

- reproduce rapidly in host
- level of infection depends not on the number of agents but on the host response



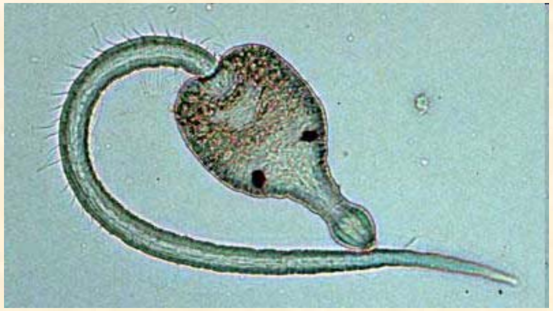
swine flu virus

- macroparasites - helminths

- reproduce in a vector
- level of infection depends on the number

E. coli (EHEC)

- incidence .. number of new infections per until time
- prevalence .. proportion of population infected =1/N



cercaria

nematode

Epidemiology

- predicts rates of disease spread
- predicts occurrence of epidemics
- predicts expected level of infection

- number of deaths caused by disease exceeds that of all wars

- affect also animals

- rinderpest introduced by Zebu cattle to South Africa in 1890
- 90% buffalo population was wiped out

- biological control

- *Cydia pomonella* granulosis virus

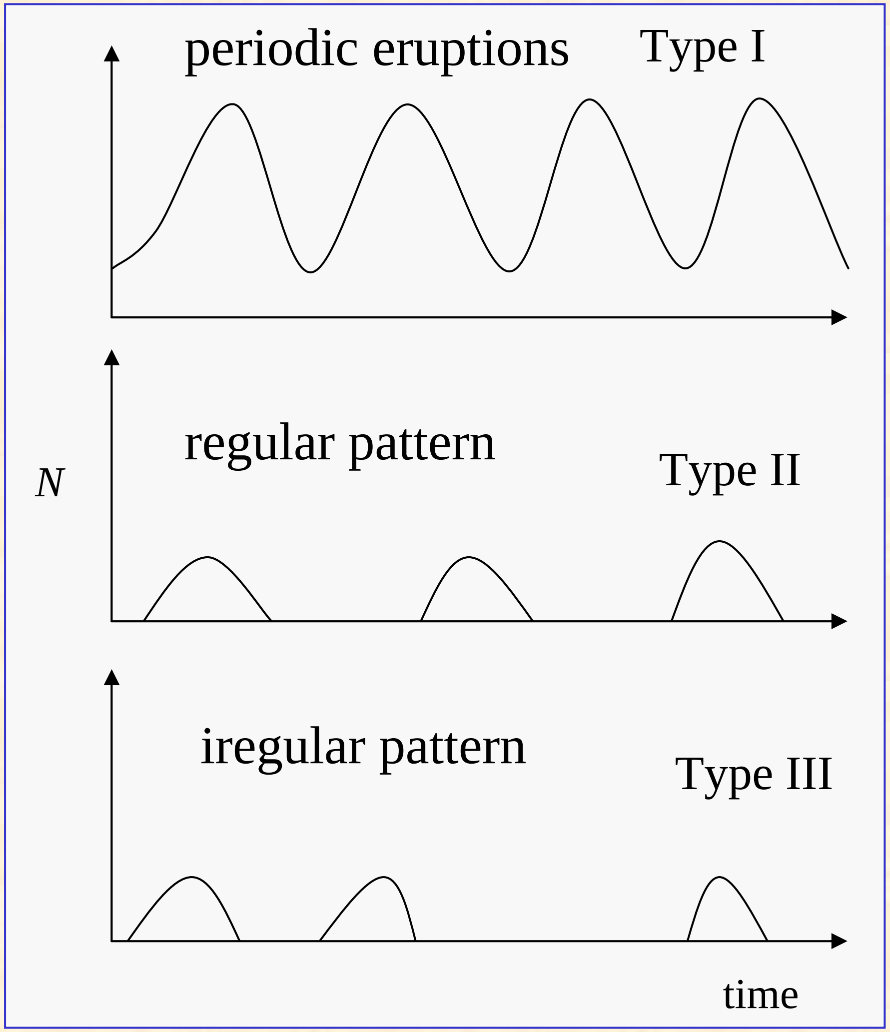
- epidemics occur in cycles
- follows 4 stages:

- establishment - pathogen increases after invasion

- persistence - pathogen persists within host population

- spread - spreads to other non-infected regions, reaches peak

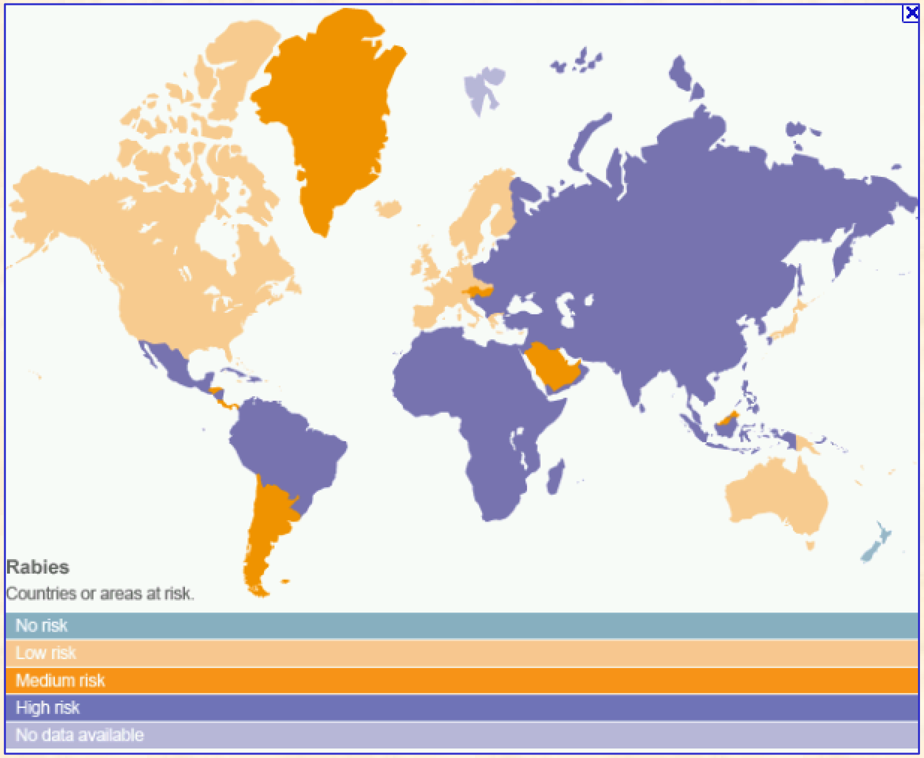
- epidemics terminates

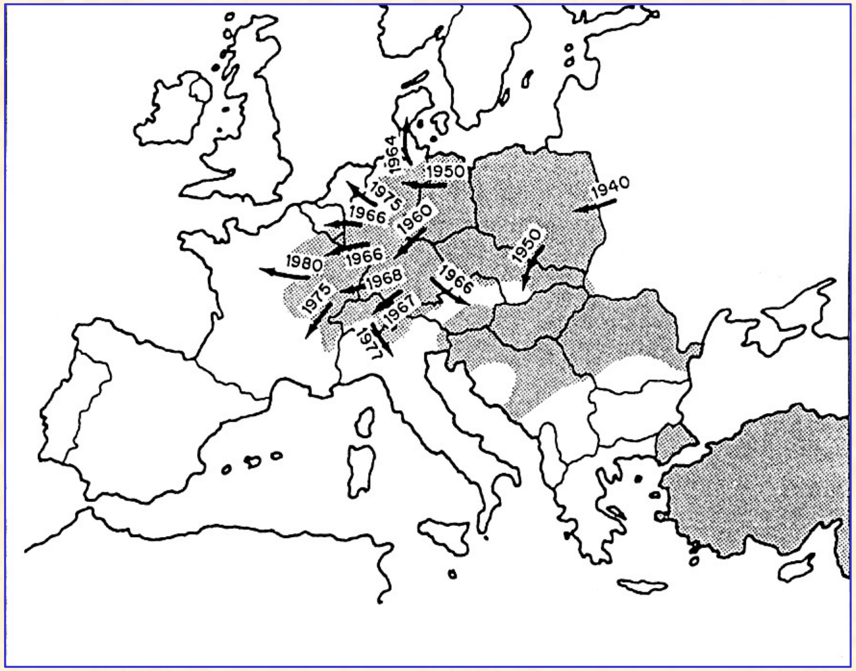


- rabies in Europe spread from Poland 1939
- hosts: foxes, badgers, roe-deer

- spread rate of 30-60 km/year

virus





Spread of rabies (Bacon 1985)

Host-pathogen/parasite system

- used to simulate spread of a disease in the human population or in the biological control
- models:

- Kermack & McKendrick (1927)
- later developed by Anderson & May (1980, 1981)

- 3 components:

- *S* .. susceptible
- *I* .. infected
- *R* .. resistant/recovered and immune + dead individuals - can not transmit disease

- latent population - infected but not infectious
- vectors (*V*) and pathogens (*P*)
- malaria is transmitted by mosquitoes, hosts become infected only when they have contact with the vector
- the number of vectors carrying the pathogens is important
- such system is further composed of uninfected and infected vectors

Kermack-McKendrick model

- β .. transmission rate - number of new infections per untit time

βSI.. density-dependent transmission function (proportional to the number of contacts)

- mass action
- analogous to search efficiency in predator-prey model

$1/\beta$ .. average time for encountering infected individual

- γ.. recovery rate of infected hosts

(either die or become immune)

γ = 1/duration of disease

Assumptions:

- $S_0 >> I_0$
- ignores population change (increase of S)
- incubation period is negligible

SI model

$$\frac{\mathrm{d}S}{\mathrm{d}t} = -\beta SI$$

$$\frac{\mathrm{d}I}{\mathrm{d}t} = \beta SI - \gamma I$$

Outbreaks

- outbreak (epidemics) will occur if $S_0 > \frac{\gamma}{\beta}$

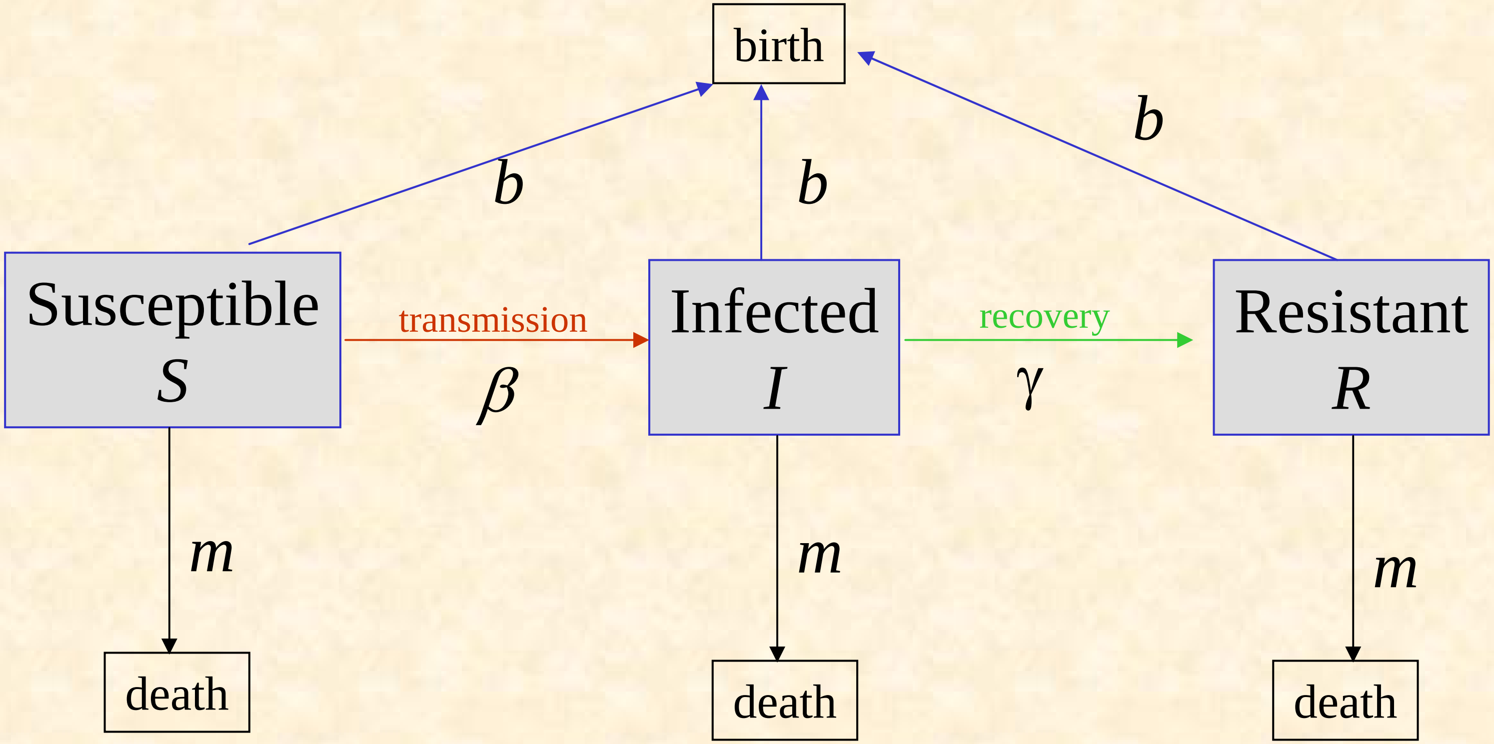
- i.e. when density of *S* is high

- making the population size small will halt the spread: $S_0 < \frac{\gamma}{\beta}$
- vaccination of *S*, culling or isolation of *I* will stop disease spread

Anderson-May model

Assumptions:

- host population is dynamic
- newborns are susceptible
- *b* .. host birth rate
  =1/host life-span, given exponential growth and constant population size
- *m* .. host mortality due to other causes



SIR model

$$\frac{\mathrm{d}S}{\mathrm{d}t} = b(S + I + R) - \beta SI - mS$$

$$\frac{\mathrm{d}I}{\mathrm{d}t} = \beta SI - \gamma I - mI$$

$$\frac{\mathrm{d}R}{\mathrm{d}t} = \gamma I - mR$$

N .. total population of hosts per area: $N = S + I + R$

- R_0 .. basic reproductive rate of the disease

- number of secondary cases that primary infection produces
- if $R_0 > 1$ .. outbreak is plausible

$$R_0 = \frac{\beta N}{b + \gamma + m}$$

Biological control

- fast biocontrol effect is achieved only with viruses with high β
- low host population is achieved with pathogens with lower β

Population dynamic of a moth and the associated granulosis virus