$$\frac{dN}{dt} = Nr$$

Predation

"Populační ekologie živočichů"

Stano Pekár

Predator categories

True predators - consume several animals and gain sustenance for their own fitness (spiders, lions)

Parasitoids - free adults but larvae developing on or within a host, consuming it prior to pupation, consume about single host (Hymenoptera, Diptera)

Parasites - live in close association with a host, gain sustenance from the host, but often do not cause mortality (Acari, Trematodes)

Herbivores - feed on plants, may totally consume plants (seed-eaters) or partially (aphids, cows)

Dietary specialisation

▸ monophagous (single prey type)
▸ oligophagous (few prey types)
▸ polyphagous/euryphagous (many prey types)
- not capable of consuming all prey types

▸ predators choose most profitable prey
- select prey items for which the gain is greatest (energy intake per time spent handling)

Krebs (1978)
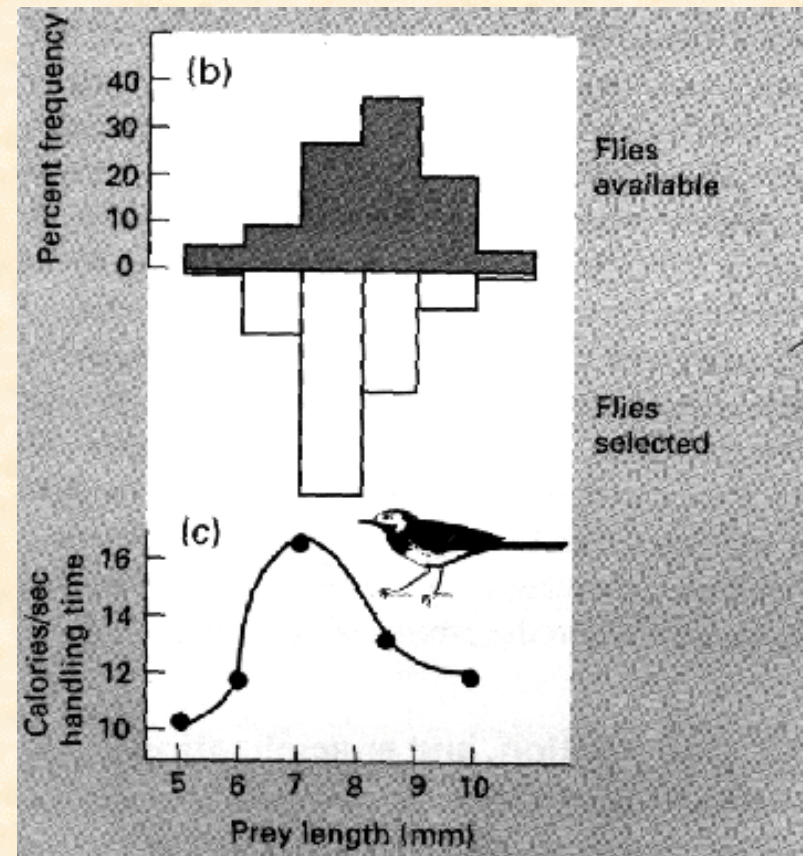
▶ predators tend to specialise to a greater or lesser extent during evolution
- monophagy evolved where prey exerts pressures which demand
morphological adaptations
- polyphagy evolved where prey was unpredictable

▶ true predators - majority are polyphagous
▶ parasites - commonly monophagous due to intimate association with
hosts, their life-cycle is tuned to that of their host
▶ parasitoids - often monophagous but some are polyphagous presumably
because adults are free living
▶ herbivores - rather polyphagous, many insect herbivores
 are specialised as a result of adaptation to plant
secondary metabolites (*Drosophila pachea* consumes
rotten tissues of *Senita* cactus which contain poisonous
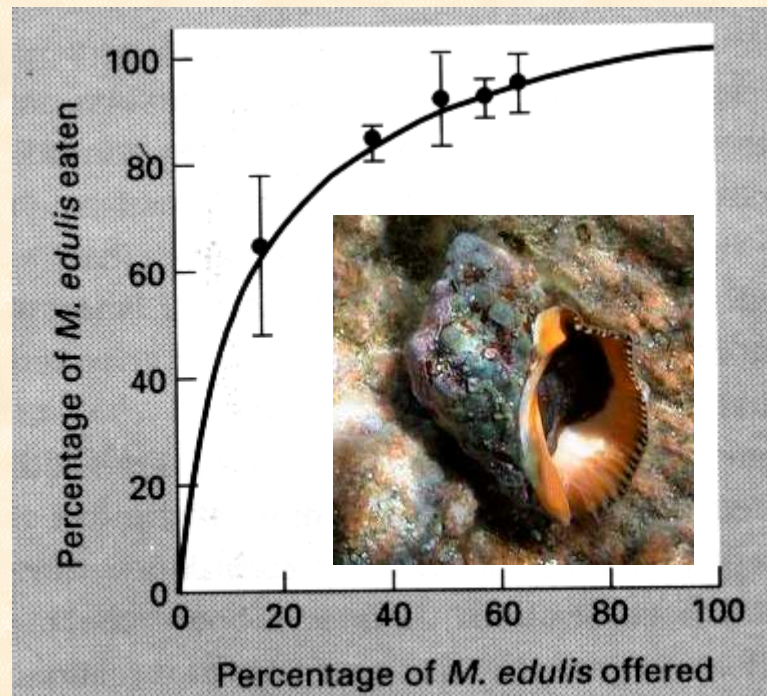alkaloids)
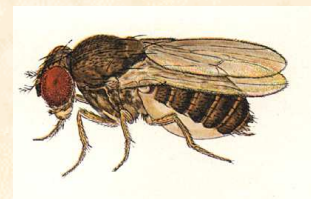
Preference & switching

▶ even polyphagous predators prefer certain prey
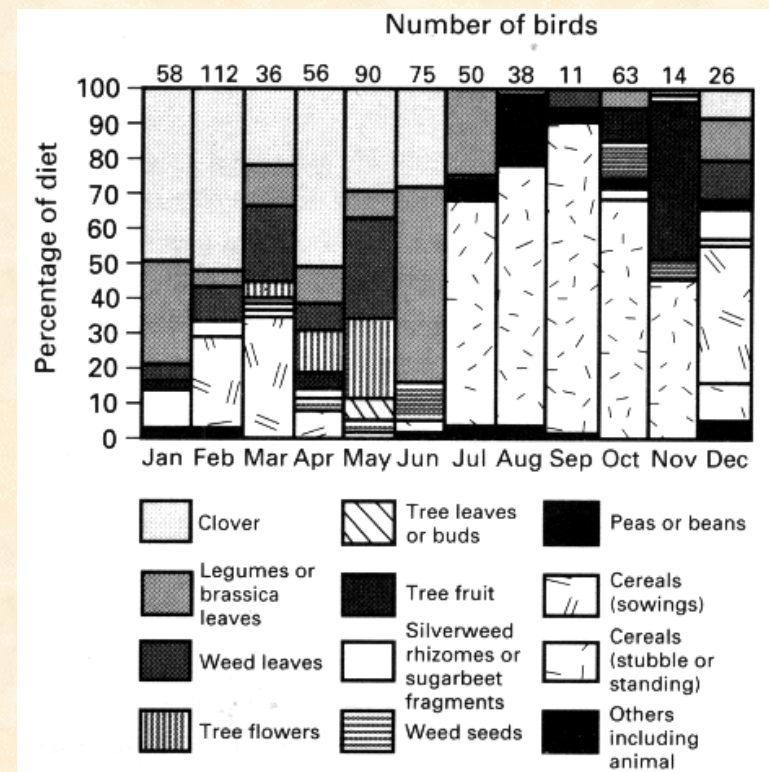- constant preference irrespective of prey density
- switching to more common prey

Seasonal shift in *Columba*

Thais preferred *Mytilus edulis* over *M. californianus*
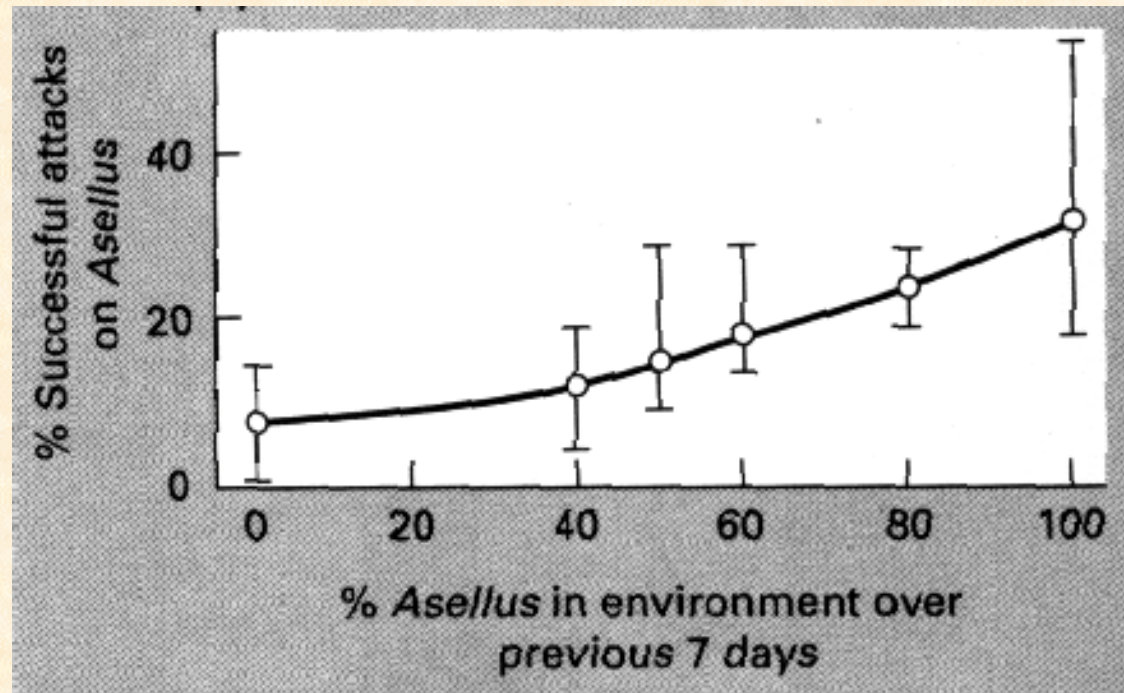

Murdoch & Oaten (1975)

Murton et al. (1964)

▸ switching

- on individual level: certain individuals develop „searching image" that facilitate the search for prey
- on population level: proportion of specialists is changing their preference

Effect of experience on the foraging success of *Notonecta* in the capture of *Asellus*
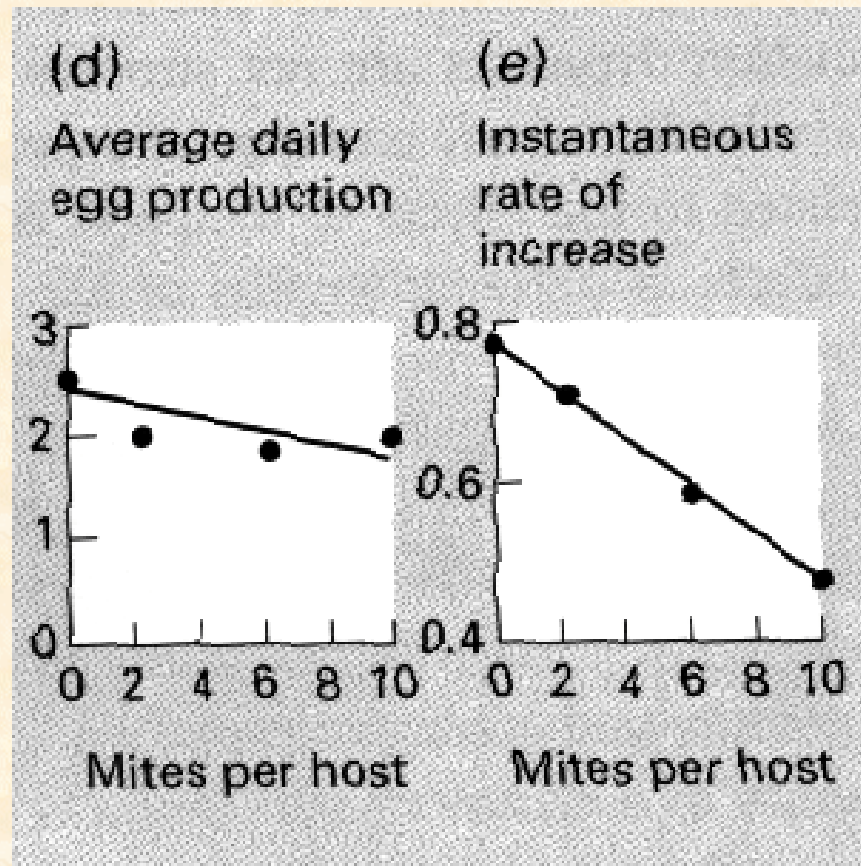

Lawton et al. (1974)

Effect on fitness of prey

▸ predation has positive effect on population of prey because reduce intraspecific competition - stabilise prey population dynamic

▸ true predators and parasitoids reduce fitness of prey to „0"
- *Mustela* consumed mainly solitary and injured individuals, so it has little effect on the *Ondatra* population growth

▸ caterpillars defoliate partially so that re-growth can occur, but cause reduction in fertility

▸ parasites - reduce fitness partially, effect is correlated with the burden

Negative effect of mite parasites on *Hydrometra*

(d) Average daily egg production

(e) Instantaneous rate of increase

Mites per host

Mites per host

Lanciani (1975)

Plant-Herbivore

Model

▸ consume small amount of many different plant species
▸ consume a lot during life
▸ functional response Type II and III
▸ plants are not killed only reduced in biomass
▸ similar to predator-prey models

V .. plant biomass
a .. assimilation rate
F .. efficiency of removal

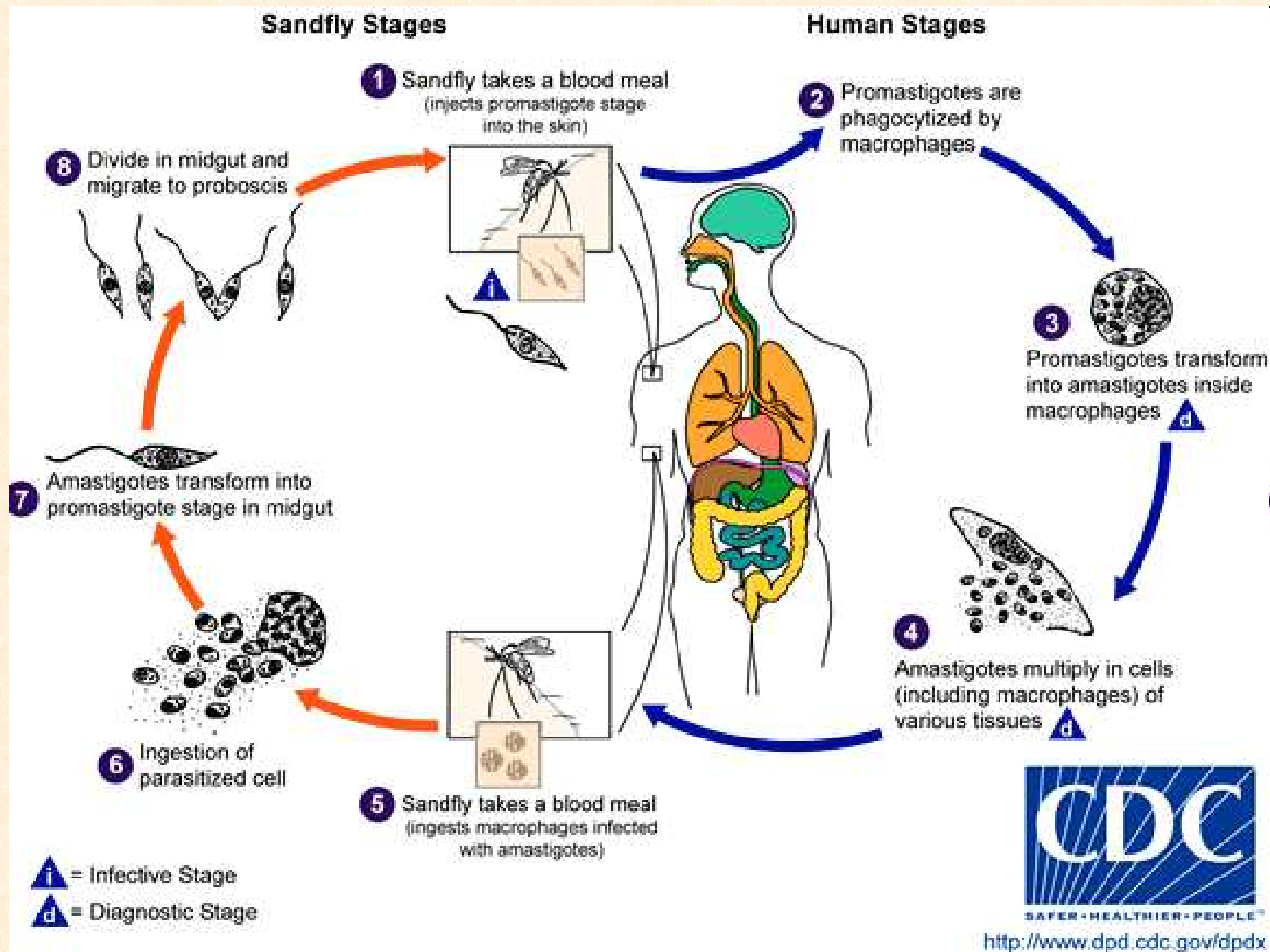$$\frac{\mathrm{d}V}{\mathrm{d}t} = rV\left(\frac{K-V}{K}\right) - \frac{FNV}{1+FNT_h}$$

$$\frac{\mathrm{d}N}{\mathrm{d}t} = \frac{aFNV}{1+FNT_h} - mN$$

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

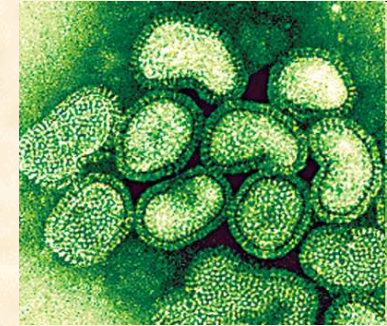△d = Diagnostic Stage
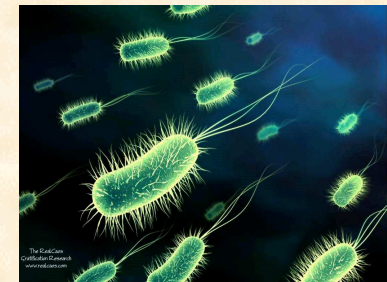
http://www.dpd.cdc.gov/dpdx

CDC
SAFER·HEALTHIER·PEOPLE™

Agents

▶ microparasites: viruses, bacteria, protozoans
- reproduce rapidly in host
- level of infection depends not on the number
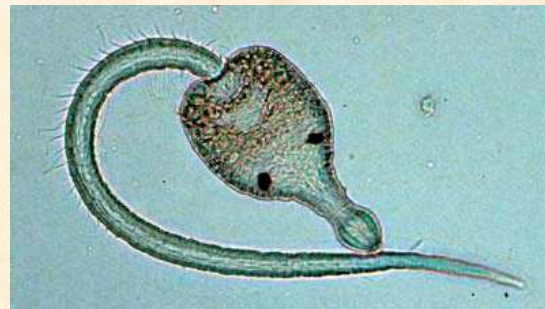of agents but on the host response

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

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

swine flu virus

E. coli (EHEC)

cercaria

nematode

Epidemiology

▸ predicts rates of disease spread
▸ predicts expected level of infection

▸ number of deaths caused by disease exceeds 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
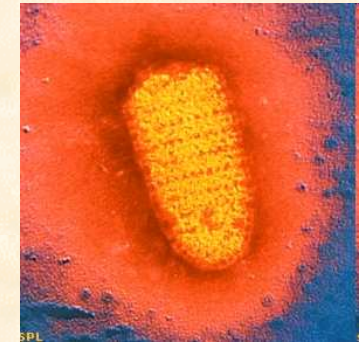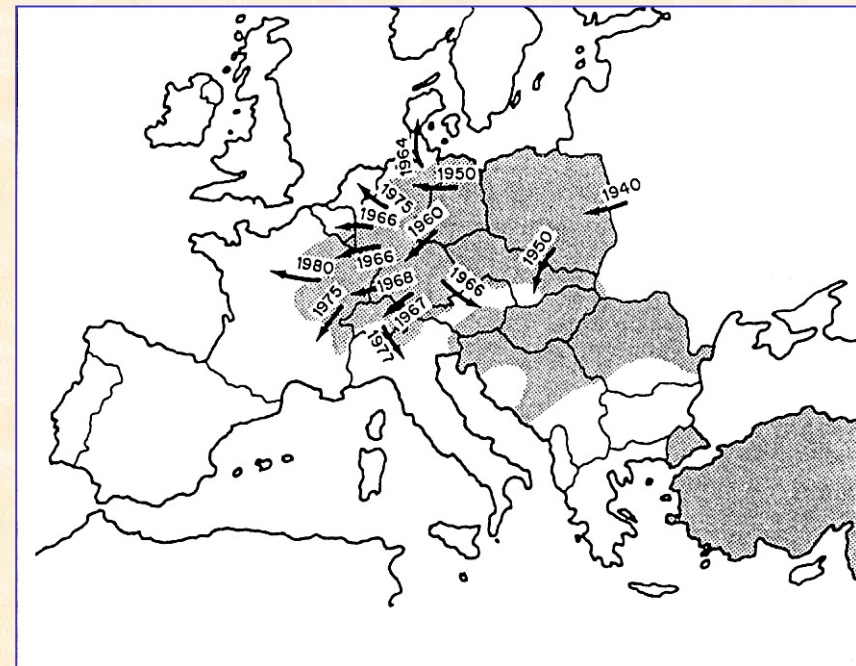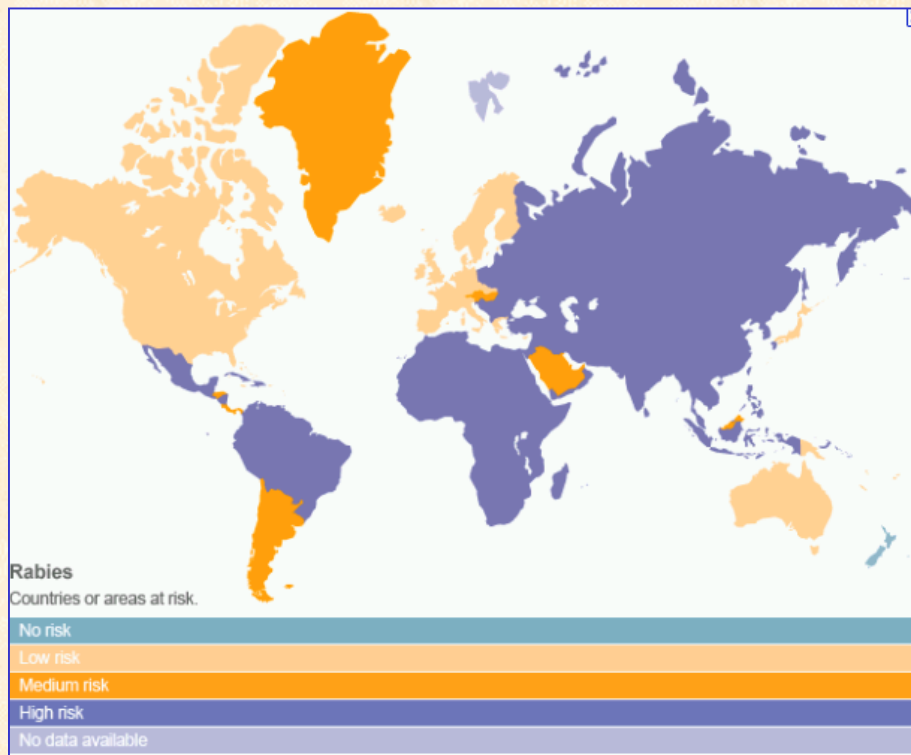
- epidemics terminates
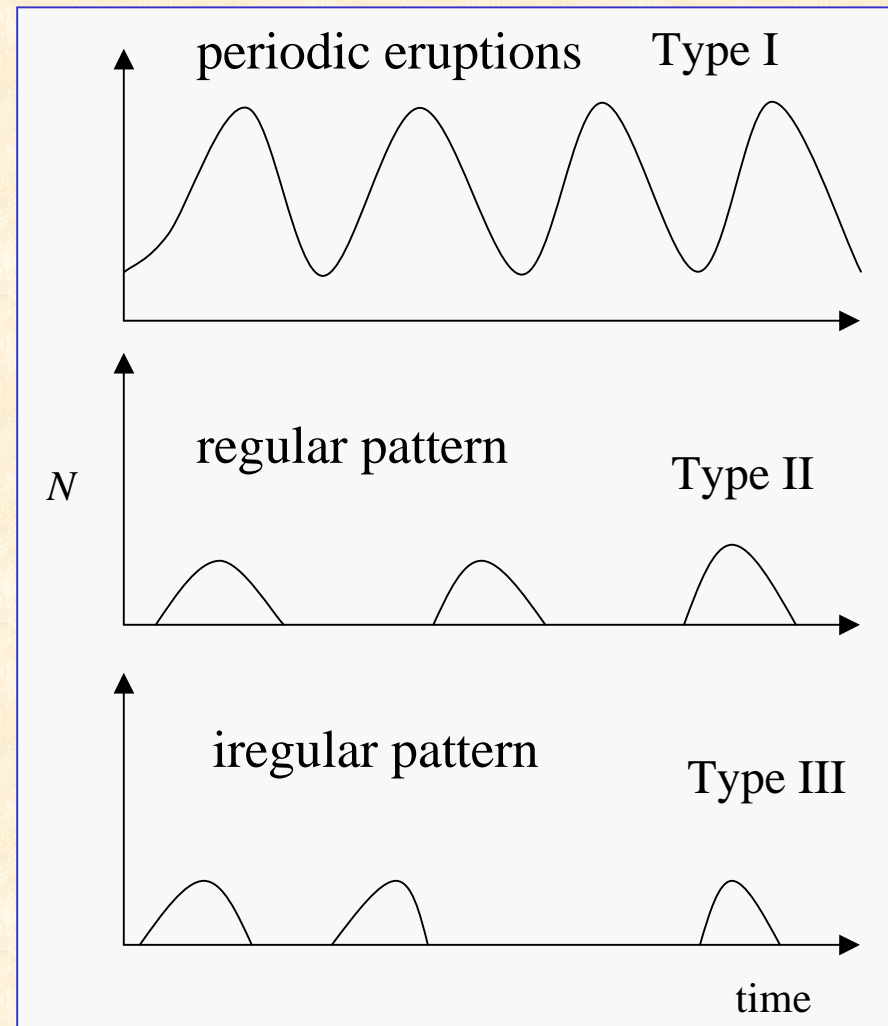
▸ rabies in Europe spread from Poland 1939
- hosts: foxes, badgers, roe-deer

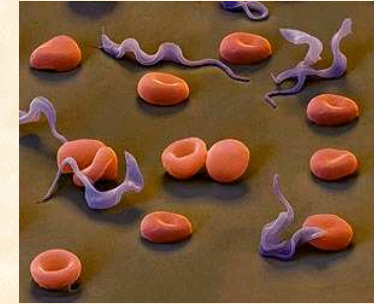▸ spread rate of 30-60 km/year

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)

$\gamma = 1/$duration of disease

$$S_0 >> I_0$$

- ignores population change (increase of S)

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 $\quad S > \dfrac{\gamma}{\beta}$
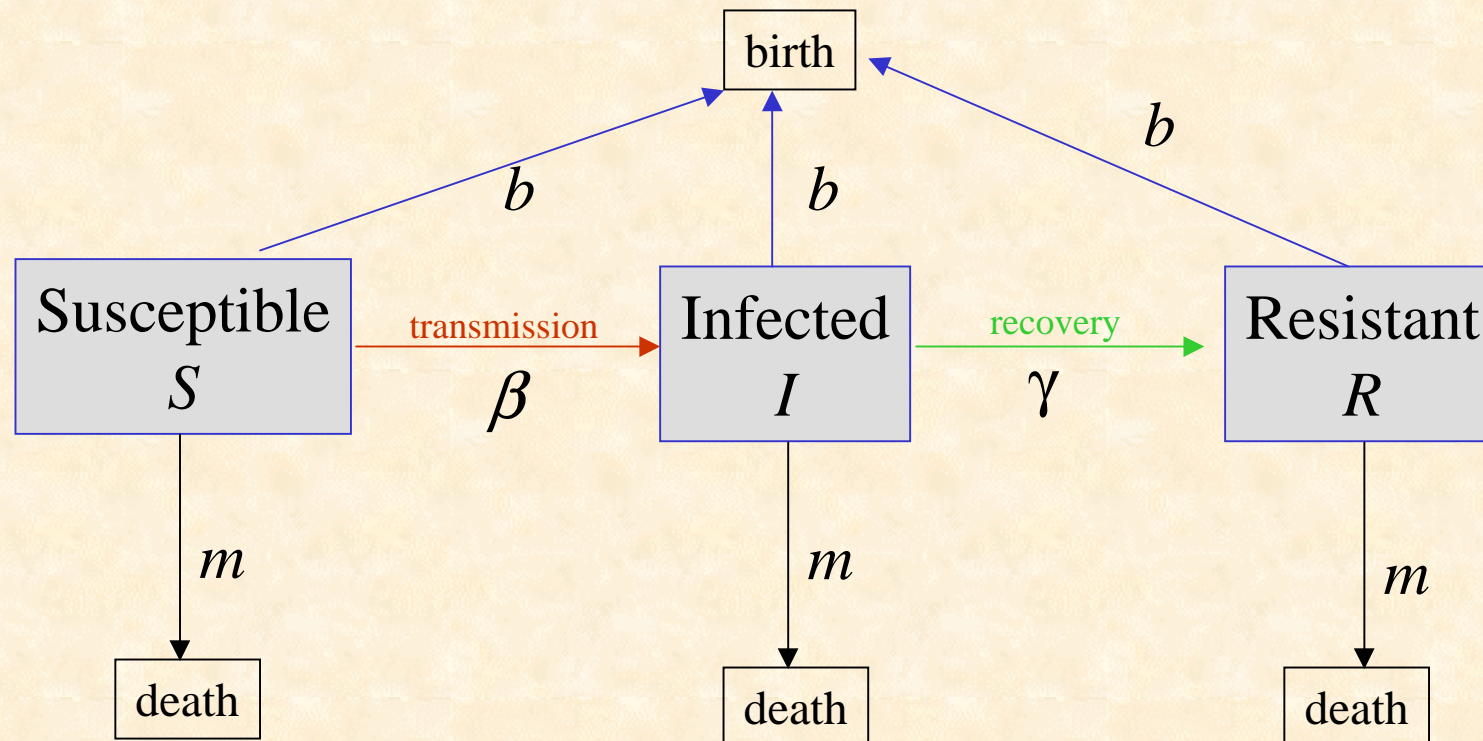
- i.e. when density of S is high

▶ making the population size small will halt the spread: $\quad S < \dfrac{\gamma}{\beta}$

▶ vaccination of S, culling or isolation of I will stop disease spread

Anderson-May model

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

SIR model

$$\frac{dS}{dt} = b(S + I + R) - \beta SI - mS$$

$$\frac{dI}{dt} = \beta SI - \gamma I - mI$$

$$\frac{dR}{dt} = \gamma I - mR$$

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
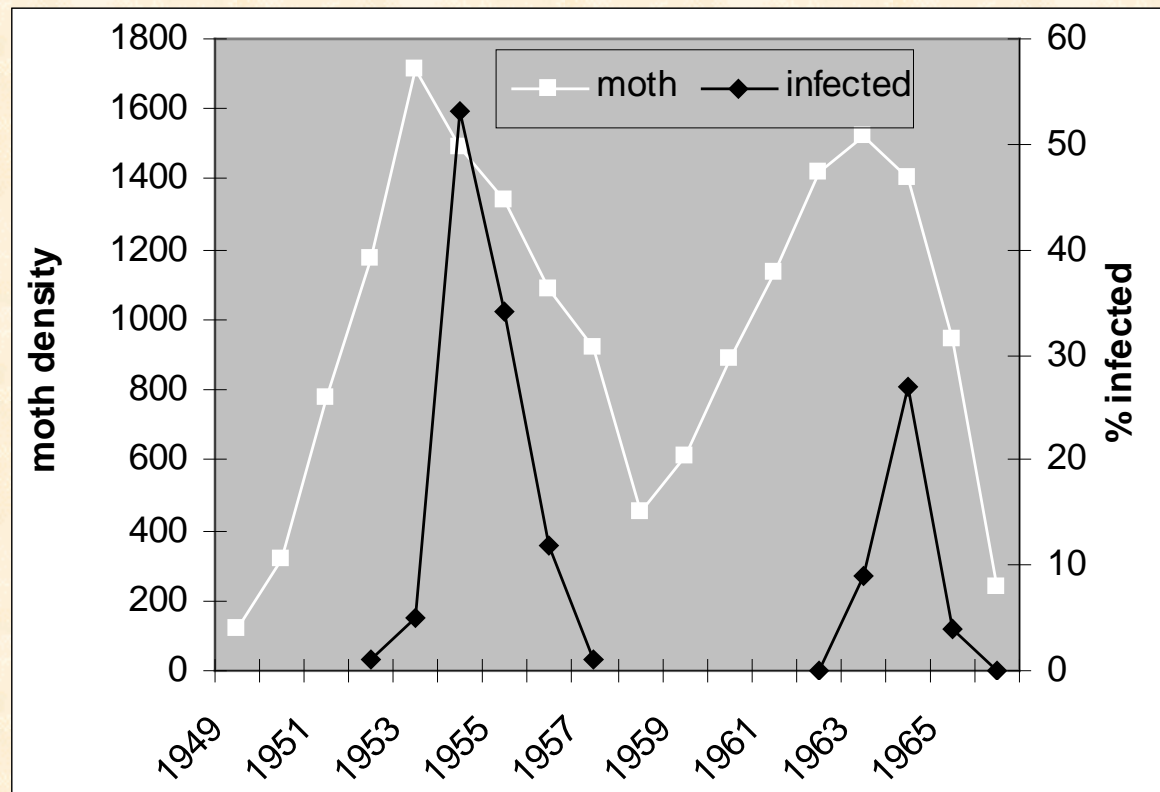- if $R_0 > 1$ .. outbreak is plausible

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

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

Example 23

Rabies has occurred in two cities. In one city 75% of dogs were vaccinated, in the other only 5%. In both cities there are 20 dogs/km². It is known that rabies lasts for 5 days (*d*). The dog lifespan (*l*) is 10 years. One dog per 10 days (*T*) becomes infected. Density of dogs is constant, thus mortality (*m*) is equal to natality (*b*).

1. Estimate parameter (*b*, *m*, *β*, *γ*) values from given data.
2. Use SIR model for each city for next 60 days.
3. Will there be epidemics (more than 50% infected)?
4. How the disease dynamic will be affected by dog isolation?

$$m = b = 0 \qquad \beta = \frac{1}{T} \qquad \gamma = \frac{1}{d}$$

```
sir<-function(t,y,param){
S<-y[1]
I<-y[2]
R<-y[3]
with(as.list(param),{
dS.dt<-b*(S+I+R)-B*I*S-m*S
dI.dt<-B*I*S-g*I-m*I
dR.dt<-g*I-m*R
return(list(c(dS.dt,dI.dt,dR.dt)))})}


g<-1/5
b<-0
B<-1/10
m<-b


N<-20;I<-1;R<-1;S<-N-I-R
time<-seq(0,60,0.1)
pa<-c(b=b,B=B,m=m,g=g)
library(deSolve)
out<-data.frame(ode(c(S,I,R),time,sir,pa,hmax=0.01))
matplot(time,out[,-1],type="l",lty=1:3,col=1)
legend("right",c("S","I","R"),lty=1:3)
```

```
N<-20;I<-1;R<-15;S<-N-I-R
time<-seq(0,60,0.1)
pa<-c(b=b,B=B,m=m,g=g)
library(deSolve)
out<-data.frame(ode(c(S,I,R),time,sir,pa,hmax=0.01))
matplot(time,out[,-1],type="l",lty=1:3,col=1)
legend("right",c("S","I","R"),lty=1:3)

N<-20;I<-1;R<-1;S<-N-I-R
time<-seq(0,60,0.1)
pa<-c(b=b,B=1/365,m=m,g=g)
library(deSolve)
out<-data.frame(ode(c(S,I,R),time,sir,pa,hmax=0.01))
matplot(time,out[,-1],type="l",lty=1:3,col=1)
legend("right",c("S","I","R"),lty=1:3)
```
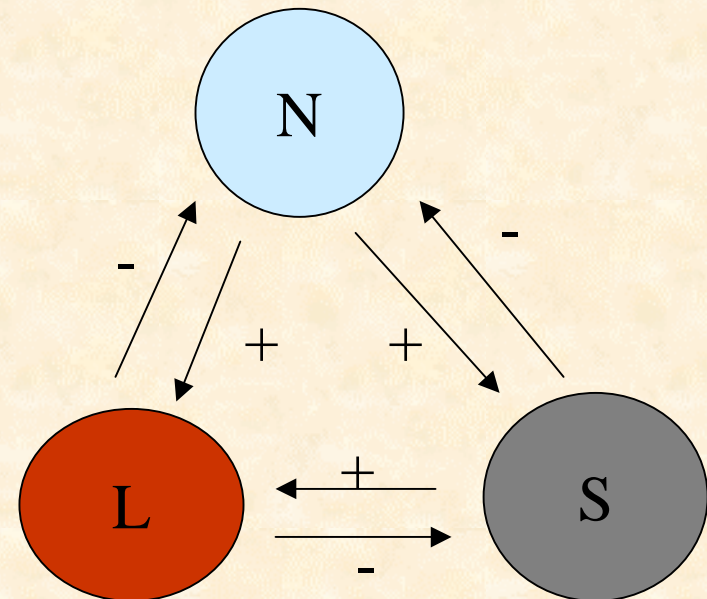
Example 24

Construct an intraguild model composed of two predators (L, S).
Both feed on prey (N), the larger predator (L) also feeds on the
smaller one (S). Use Lotka-Volterra predation model with
functional response of Type I with capture efficiency (a),
conversion rate (b), $r = 1.5$ and $K = 1000$.

1. Find parameter estimates producing stable dynamic.

$$\frac{\mathrm{d}L}{\mathrm{d}t} = a_{N1}b_{N1}LN + a_S b_S LS - m_L L$$

$$\frac{\mathrm{d}S}{\mathrm{d}t} = a_{N2}b_{N2}SN - a_S LS - m_S S$$

$$\frac{\mathrm{d}N}{\mathrm{d}t} = Nr\left(1 - \frac{N}{K}\right) - a_{N1}LN - a_{N2}SN$$

Parameter estimates should meet the following conditions:
- L is by half less effective in prey capture of N than of S ($a_{N1} = 0.02$)
- S is twice more effective in prey capture of N than L
- N is by half less nutritious for L than for S ($b_{N1} = 0.03$)
- S is twice more nutritious for L than N is
- mortality of L and S is identical ($m_S = m_L = 0.1$)
- density of N is twice higher than that of S and that is twice higher than of L ($L_0 = 20$)

```
igp<-function(t,y,param){
L<-y[1]
S<-y[2]
N<-y[3]
with(as.list(param),{
dL.dt<-an1*bn1*L*N+as*bs*L*S-ml*L
dS.dt<-an2*bn2*S*N-as*L*S-ms*S
dN.dt<-r*N*(1-N/K)-an1*L*N-an2*S*N
return(list(c(dL.dt,dS.dt,dN.dt)))})}

L<-20;S<-40;N<-80
param<-c(an1=0.02,bn1=0.03,as=0.01,bs=0.06,ml=0.1,
an2=0.04,bn2=0.06,ms=0.1,r=1.5,K=1000)
time<-seq(0,30,0.1)
library(deSolve)
out<-data.frame(ode(c(L,S,N),time,igp,param))
matplot(time,out[,-1],type="l",lty=1:3,col=1)
legend("right",c("L","S","N"),lty=1:3)
```