Predation

“Populační ekologie živočichů“

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Plant-Herbivore

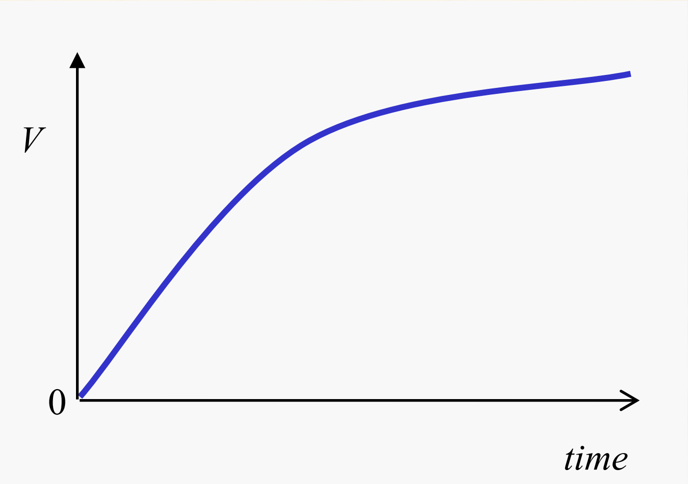
- consume small amount of many different plant species
- consume a lot during life to obtain sufficient amount of N
- grazers, granivores, frugivores, herbivores
- plants are not killed only reduced in biomass
- bottom-up control – herbivore abundance is regulated by quantity and quality of plants
-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

Herbivory-regrowth model

- Turchin (2003)
- assumptions

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

- hyperbolic biomass growth because only small part of aboveground tissues is consumed

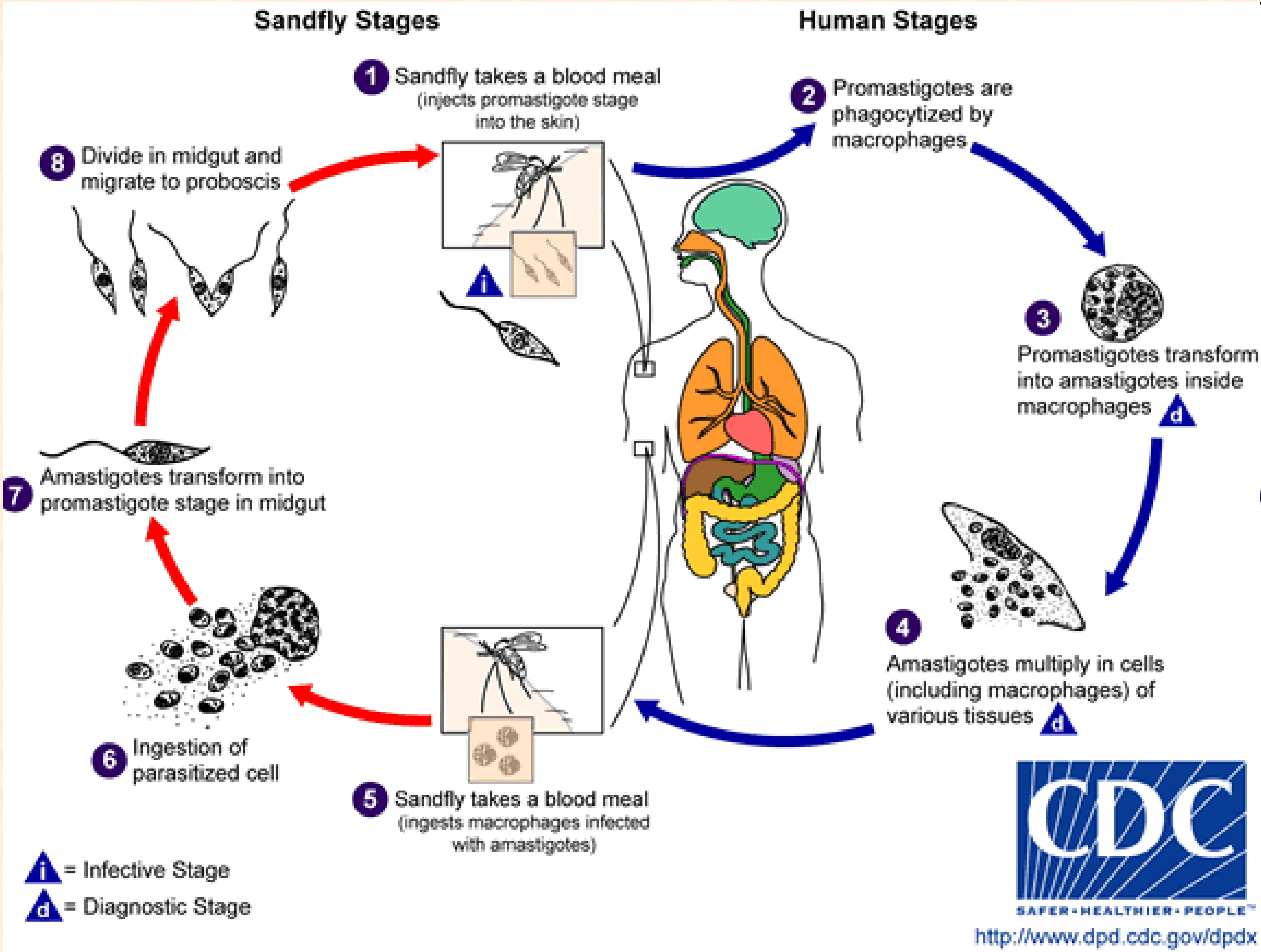


V .. plant biomass
H.. herbivore density
r.. intrinsic rate of regrowth
K.. carrying capacity
f .. efficiency of removal
T_h.. handling time

$$\boxed{\frac{\mathrm{d}V}{\mathrm{d}t} = r\left(1 - \frac{V}{K}\right) - \frac{fHV}{1 + fHT_h}}$$

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
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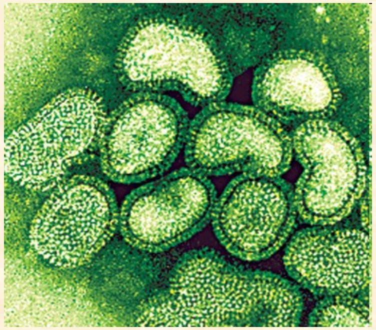
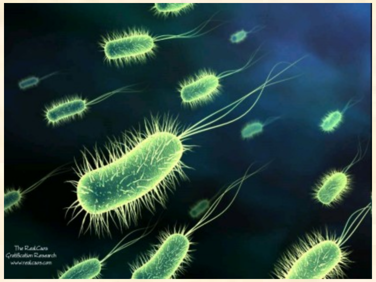
http://www.dpd.cdc.gov/dpdx

Agents

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



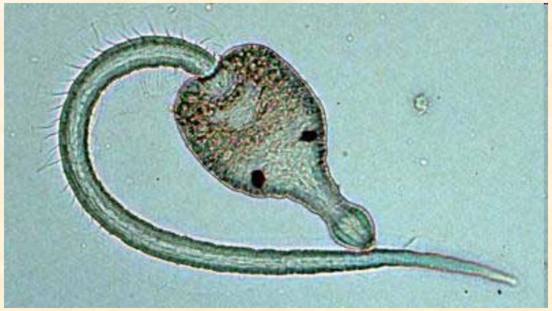
swine flu virus



E. coli (EHEC)

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

- incidence .. number of new infections per unit time
- prevalence .. proportion of population infected [%]



cercaria

nematode

Epidemiology

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

- number of human deaths caused by diseases exceeds that of all wars

- affects also animals

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

- biological control

- *Cydia pomonella* granulosis virus

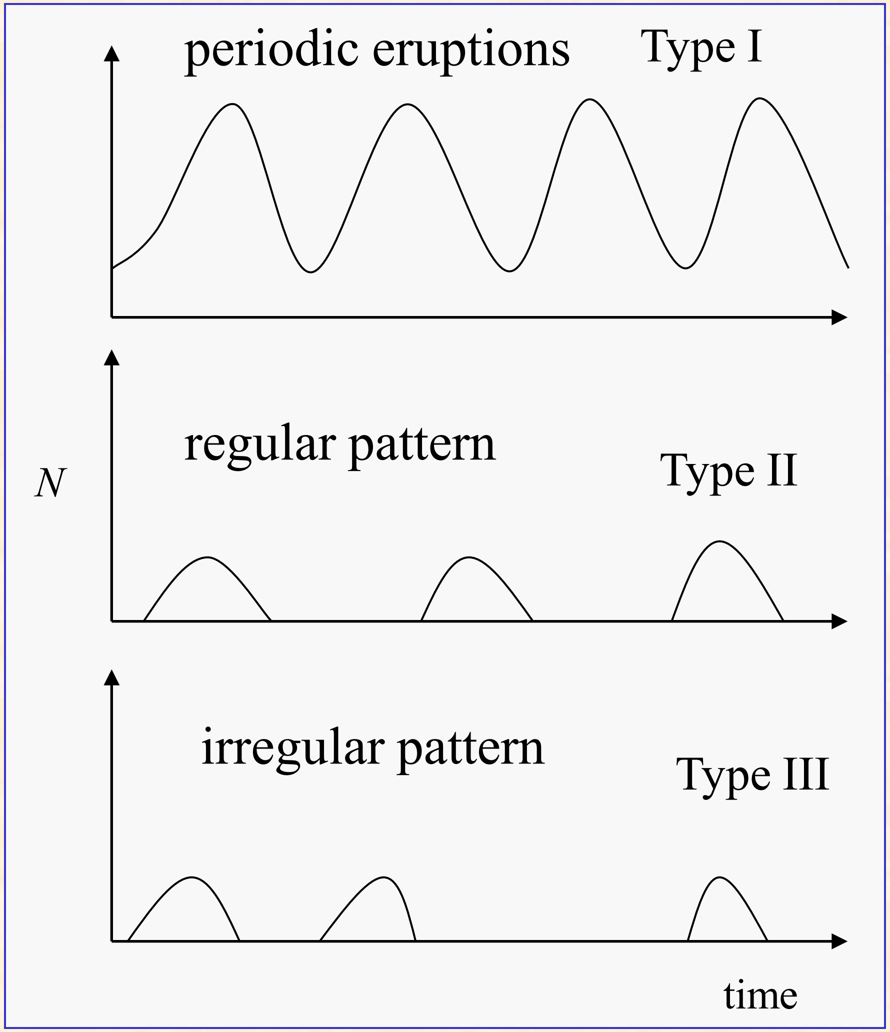
- epidemics occur in cycles
- follows 4 stages:

- establishment - pathogen increases after invasion

- persistence - pathogen persists within host population

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

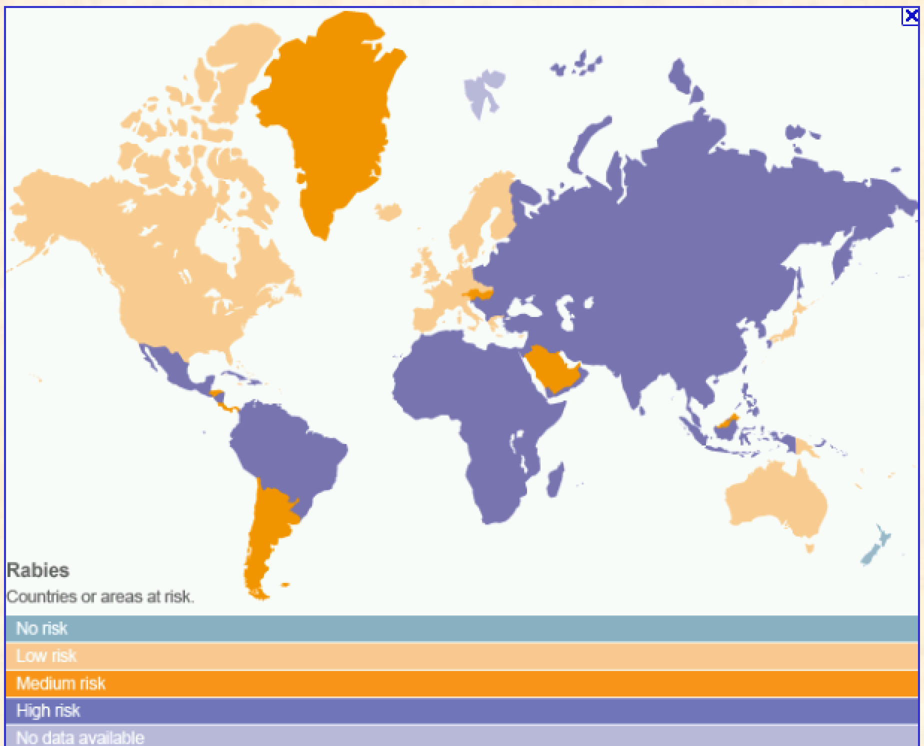
- epidemics terminates

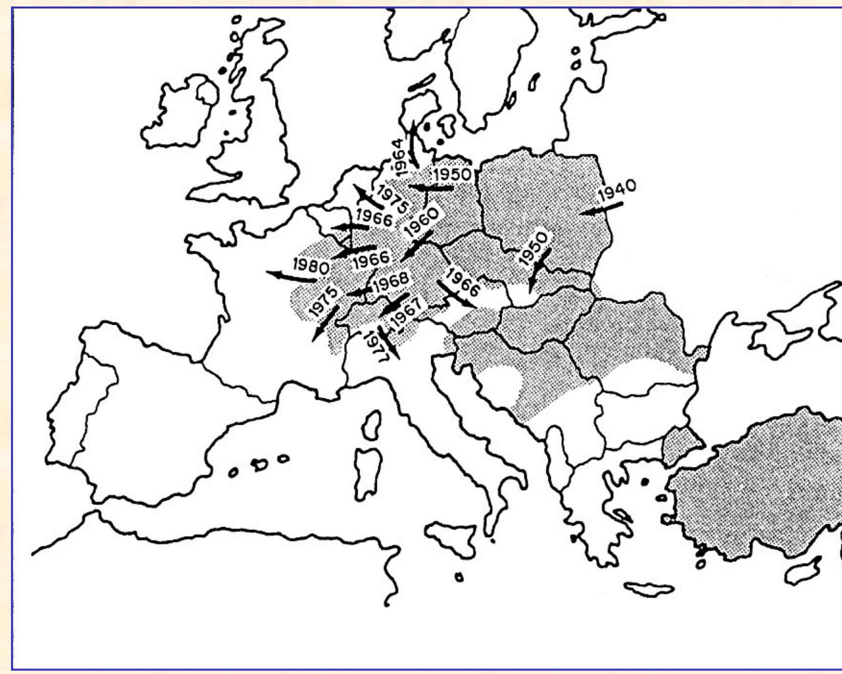


- rabies in Europe spread from Poland 1939
- hosts: foxes, badgers, roe-deer

- spread rate of 30-60 km/year

virus





Spread of rabies (Bacon 1985)

Host-pathogen/parasite system

- used to simulate spread of a disease
- pathogen is much smaller than host
- models:

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

- 3 components:

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

- latent stage - 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 unit 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)

$\gamma = 1/\text{duration of disease}$

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. transmission threshold, when density of *S* is high

- making the population size small will halt the spread: $S_0 < \frac{\gamma}{\beta}$

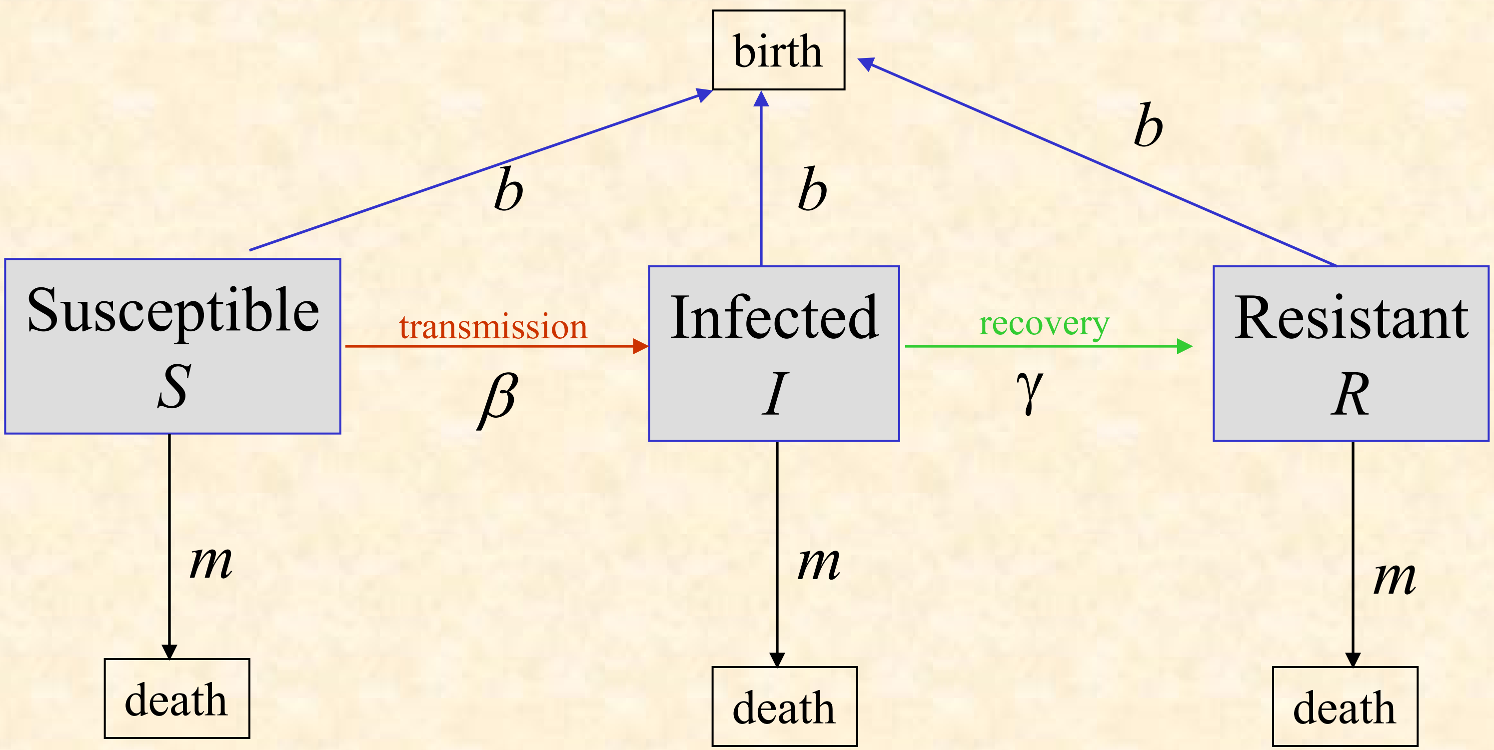
- e.g. by vaccination (not necessary to use for all)

- culling or isolation of *I* will stop disease spread

Anderson-May model

<u>Assumptions</u>:

- 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

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

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

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

$$N = S + I + R$$

N .. total population of hosts per area:

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

- R_0 .. basic reproductive rate of the disease

- number of secondary cases that primary infection produces
- if $R_0 > 1$ .. disease will persist, if $R_0 < 1$ .. disease will disappear
- is dependent on N – R_0 is larger in large populations
- after immunization the equilibrium of infection will decrease

Biological control

- fast biocontrol effect is achieved only with viruses with high β
- regulation is possible if pest r << mortality due to disease
- low host population is achieved with pathogens with lower β

Population dynamic of a moth and the associated granulosis virus