DEMOGRAPHIC EFFECTS OF BIOLOGICAL INTERACTIONS BETWEEN DIFFERENT SPECIES

Species do not exist alone in the habitat

Different populations from different species do interact within the same habitat

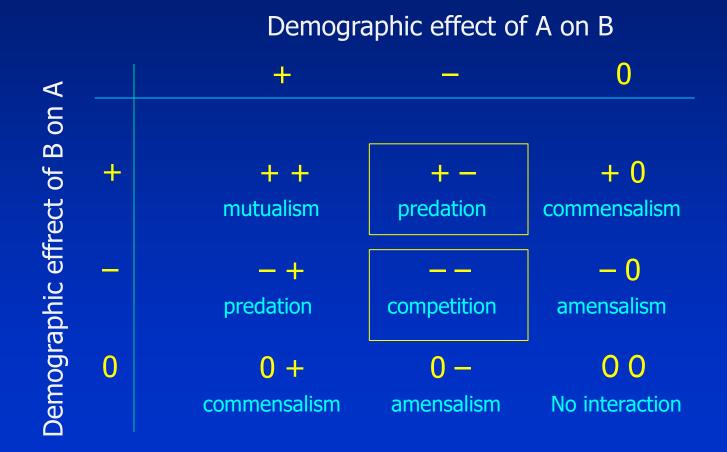
Interactions between members of different species do affect the demography of the natural populations

Demoecology of interactions start by considering simple binary systems (e.g. systems composed by just two populations of two different species)

Which kind of interactions?

Depending on the "sign" (positive, negative or null) of the effect of pop A on pop B and viceversa, we can distinguish 9 theoretical interactions

DEMOGRAPHIC EFFECTS OF BIOLOGICAL INTERACTIONS BETWEEN DIFFERENT SPECIES









Interspecific competition occurs when members of two different species having similar ecological niche and prophyle (trophic level) live together in the same area/environment they inevitably and share a given set of resources (trophyc, homeostatic and even reproductive)

Interspecific competition may be accomplished by different "methods":

A) By means of "exploitation" i.e. by better exploiting the resourcesB) By means of "interference" i.e. by impeding the other pop to access the resources:

Biochemical-physiological interaction (emission of chemical signals, poisoning etc.) Morphological interaction (e.g. overgrowth, overshadowing etc.) Behavioural interaction (emission of postural or vocal signals, fighting etc.)



Interspecific competion: why does it matter?

ECOLOGICAL INTEREST

Packaging of populations having similar conditional and trophic niche in the same community

Dynamic substitution of species in terrestrial biomes

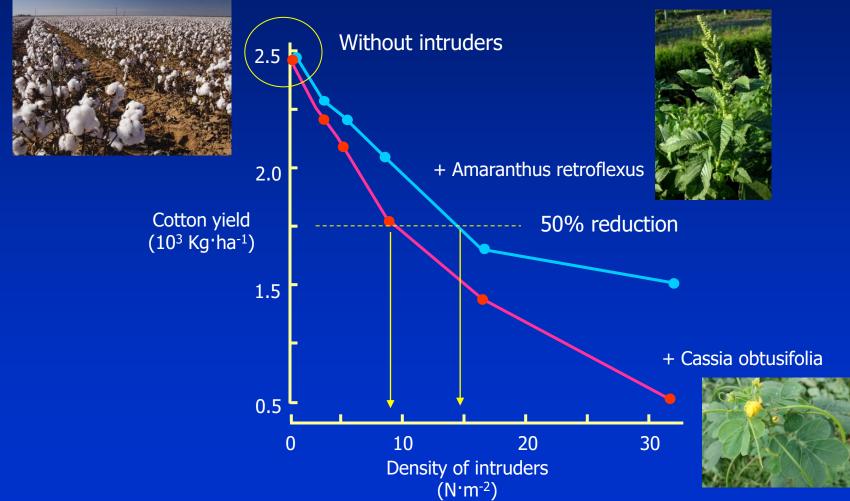
Perturbation of natural communities following biological invasions of alien species

ECONOMICAL INTEREST

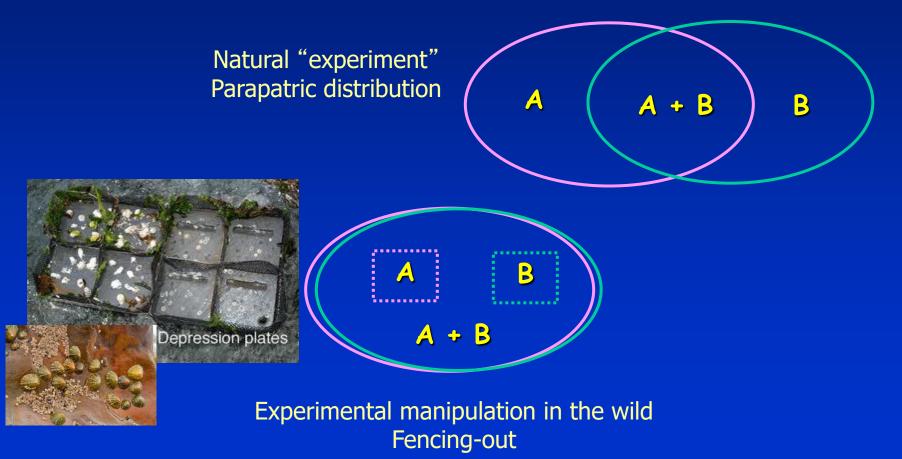
Economic cost of "invading" species in agroecosystems

Possibility to use competition in biological control of "pests"

Economic cost of competition. Example: reduction of cotton yield

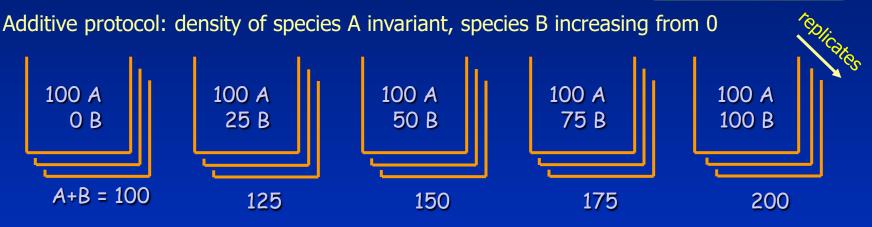


Demographic effects of interspecific competition can be assessed in the wild by studying parapatric populations or by introducing experimental manipulation of density in experimental plots (fencing)

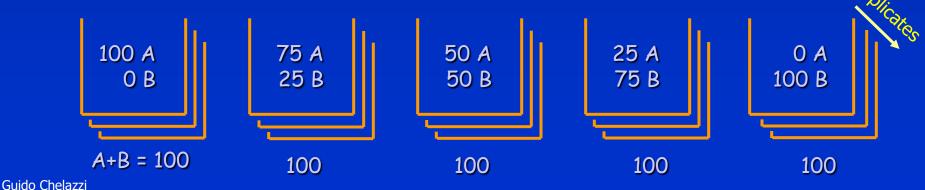


Experimental protocols: input combinations of densities





Substitutive (replacement) protocol: Species A decreasing, species B increasing

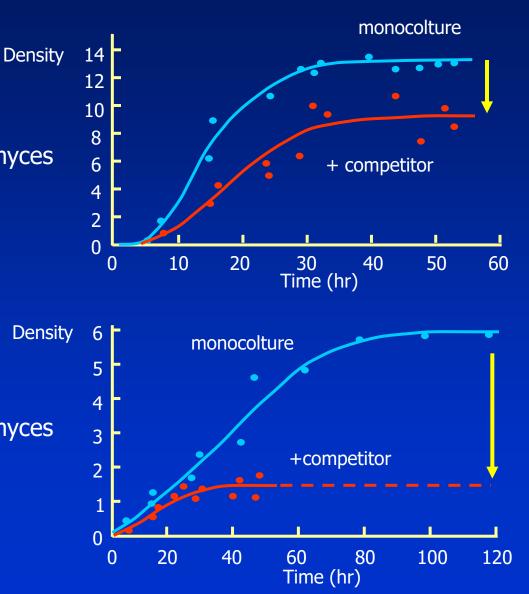


Gause's "free-running" experiments on yeast coltures



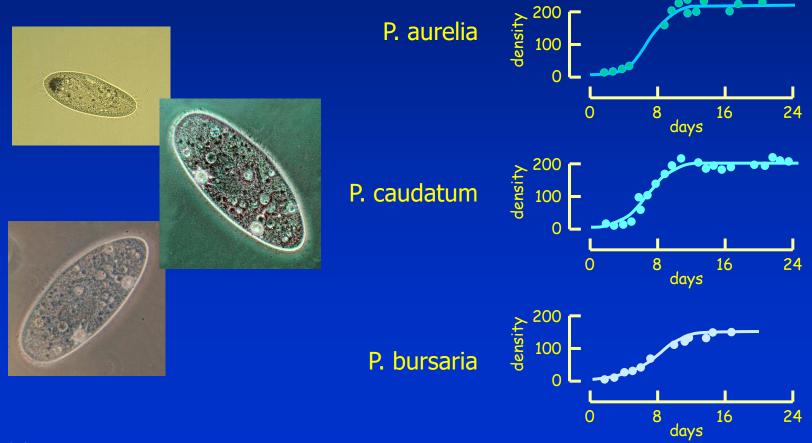
Schizzosaccharomyces

Asymmetric reduction of K



Gause's "free-running" experiments on protozoans (Paramecium spp.)

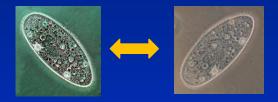
logistic growth in monocolture

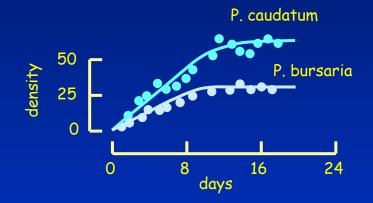


Gause's "free-running" experiments on protozoans (Paramecium spp.)

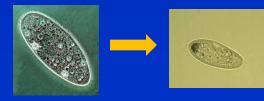
growth in mixed colture

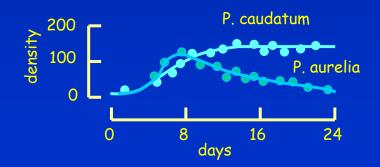
P. caudatum vs. P. bursaria Stable coexistence



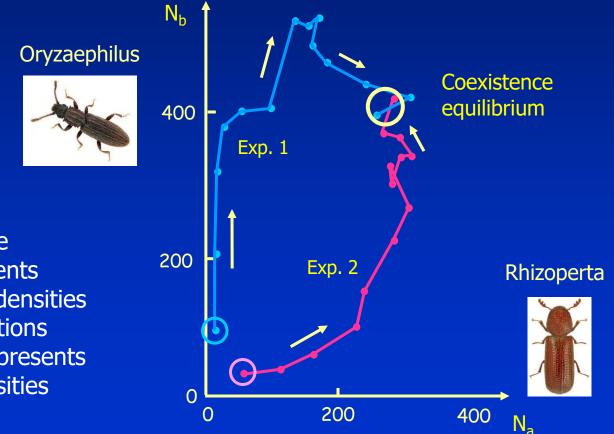


P. caudatum vs. P. aurelia Competitive exclusion





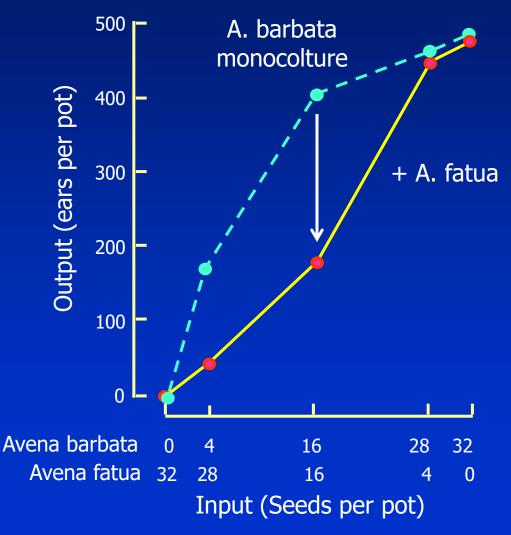
Free running experiments on a couple of competing species of Coleoptera plotted in the "phase plane" $N_a - N_b$



In the phase plane each point represents a combination of densities of the two populations and a segment represents a variation of densities

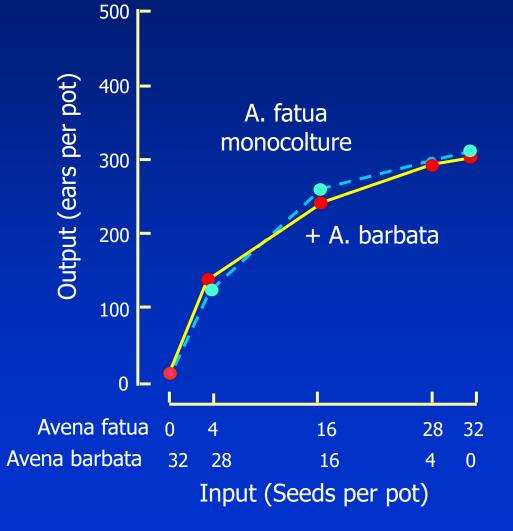
De Witt's substitutive (replacement) experiments on two Avena spp.





De Witt's substitutive (replacement) experiments on two Avena spp.





How to design a model for interspecific competition?

- 1) Parsimony: start from the model of intraspecific competition and add inter-specific interactions
- 2) Completeness: the model must be able to predict different outcomes (exclusion or coexistence) when changing values of the parameters. The model must be able to reproduce symmetric and asymmetric outcomes

Design two linked equations, one for each species, each including competion of species a on b and viceversa

dt

The model of Alfred Lotka and Vito Volterra

 $\alpha_{a,b}$ = competition "strength" of b on a

 $\alpha_{b,a}$ = competition "strenght" of a on b

$$\frac{dN_a}{dt} = N_a r_a \frac{\left(K_a - N_a - \alpha_{a,b} \cdot N_b\right)}{K_a}$$
$$\frac{dN_b}{dt} = N_b r_b \frac{\left(K_b - N_b - \alpha_{b,a} \cdot N_a\right)}{K_b}$$

Which are the basic predictions of L-V model ?

$$\frac{dN_a}{dt} = N_a r_a \frac{\left(K_a - N_a - \alpha_{a,b} \cdot N_b\right)}{K_a}$$

$$\frac{dN_a}{dt} = 0 \quad \text{when} \quad N_a r_a \frac{\left(K_a - N_a - \alpha_{a,b} \cdot N_b\right)}{K_a} = 0$$

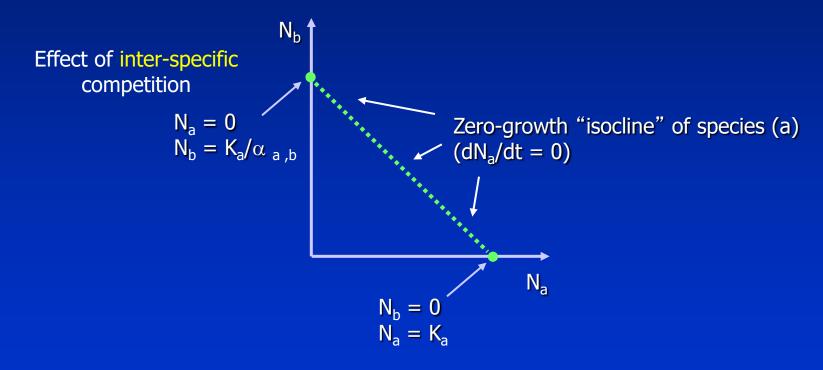
i.e when
$$r_a = 0$$
 or when $(K_a - N_a - \alpha_{a,b} \cdot N_b) = 0$

if $N_b = 0$, this happens when $N_a = K_a$ (pure intra-specific competition)

if $N_a \rightarrow 0$, this happens when $\alpha_{a,b} \cdot N_b = K_{a,j}$ i.e when $N_b = K_a/\alpha_{a,b}$ (pure inter-specific competition)

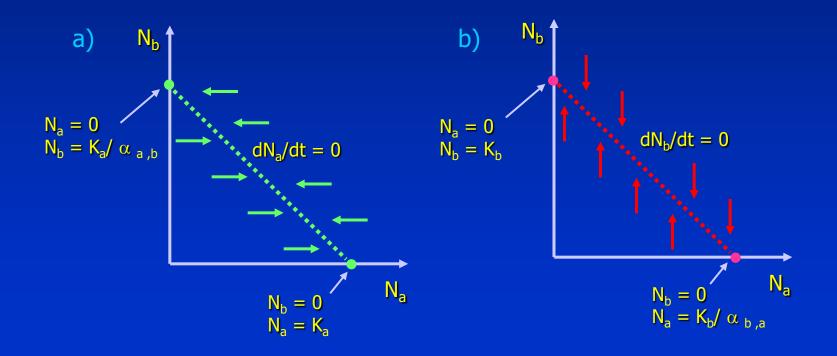
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Equilibrium densities in the L-V model. The phase plane of species (a)

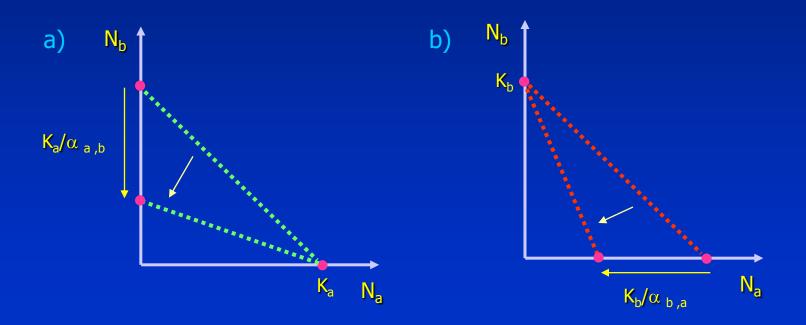


Effect of intra-specific competitiuon

Equilibrium densities of the two species in the L-V model



Equilibrium densities in the L-V model. The effect of increasing alfa values (strength of inter-specific competition



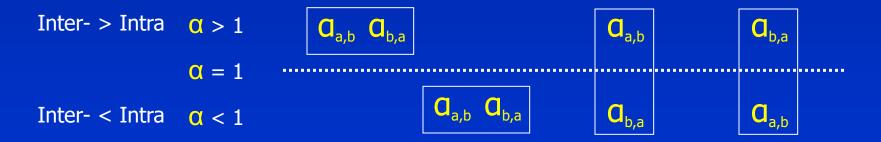
The predicted outcome of competition under L-V models depends on the relative position of the zero growth isoclines of the two species in the common phase-plane

This in turn depends on the values of the critical parameters for intraand interspecific competition:

Species (a): K_a , K_a / $\alpha_{a,b}$

Spedies (b): K_b, K_b / α _{b,a}

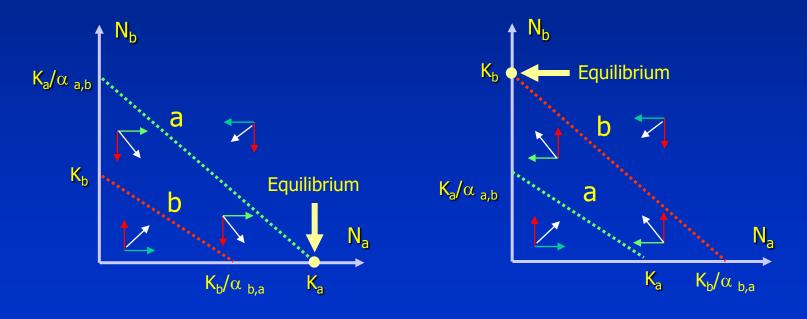
In particular, regarding the alfas the situation of the two species can be as follows



Predictions of the L-V model

Case I – Competitive exclusion independent from initial densities

This occurs when one species (overcompetitor) is less constrained than the other both with respect to intra- and inter-specific competion

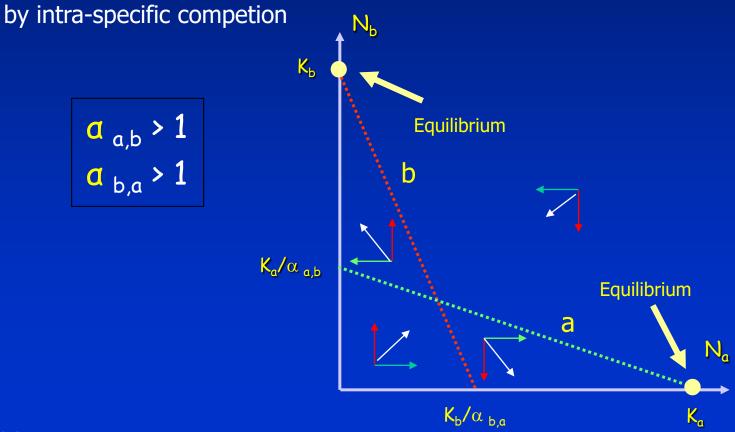


Species (b) overcompetitor on (a)

Species (a) overcompetitor on (b)

Predictions of the L-V model

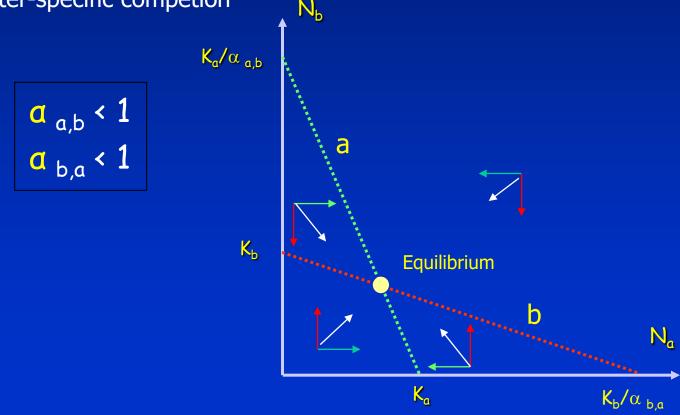
Case II – Competitive exclusion dependent from initial densities This occurs when both specxies are more constrained by inter- than by intra-specific competion



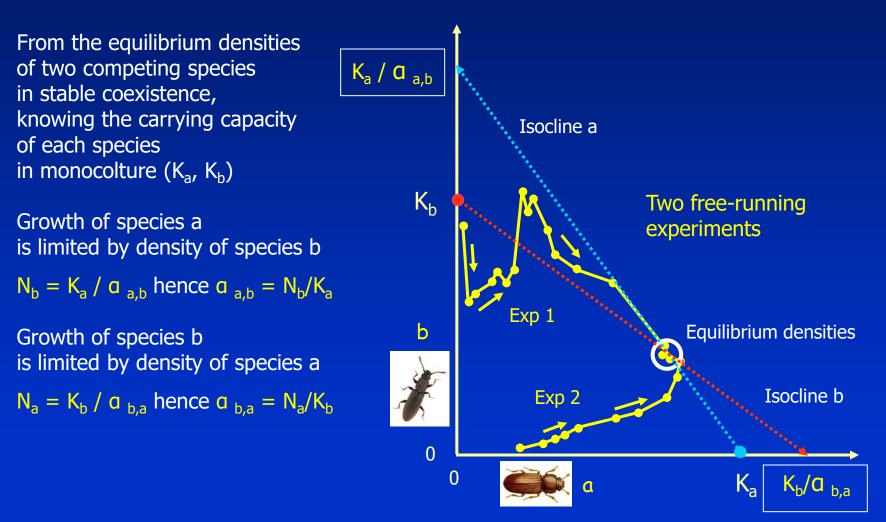
Predictions of the L-V model

Case III – Stable coexistence

This occurs when both specxies are more constrained by intra- than by inter-specific competion



How to asses the alfa coefficients according to the L-V models ?



Interspecific competition can be assessed by obtaining the "trophic niche" of two or more species having similar trophic habits

 Offer to individuals of two species (a and b, independently), a set of R different resources equally represented (e.g. seeds of different size "offered" to two species of ants)

2) Register the frequency of choices toward each class of items (i) made by each of the two species (how many individuals made their first choice on each class of seeds) Probability of use

3) Normalize the frequency distribution to the total of individuals tested to obtain probabilities of resource use (U)

Assessment of "trophic niche overlap" between two species



Species (a)





Species (b)



PLASTICITY OF RESOURCES USE IN COMPETING SPECIES

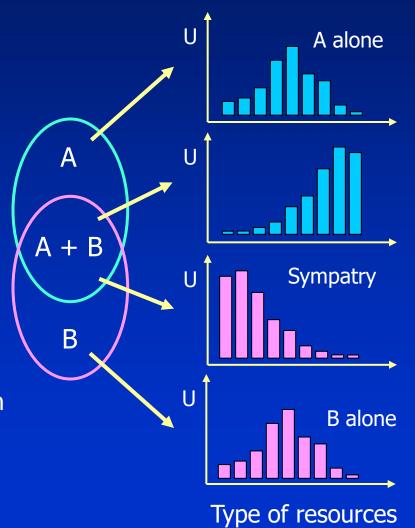
Trophic niche shift (niche apportionment)

Allopatric populations have similar trophic niches

Sympatric populations have different trophic niche

Niche shift can be based on:

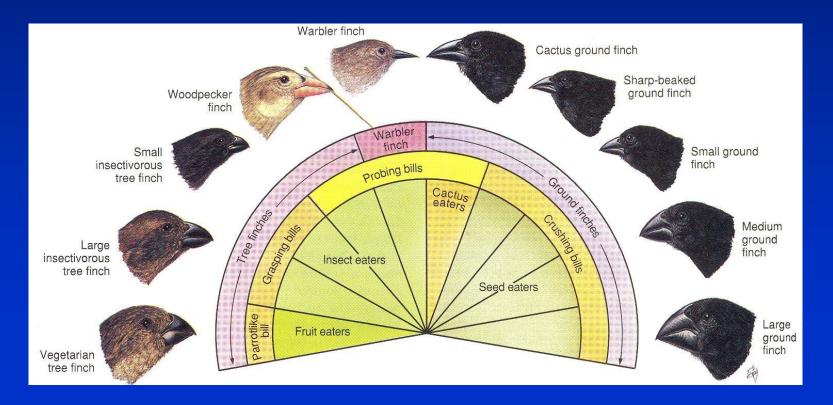
1) Individual behavioural plasticity
2) Selection at the level of population



PLASTICITY OF RESOURCES USE IN COMPETING SPECIES

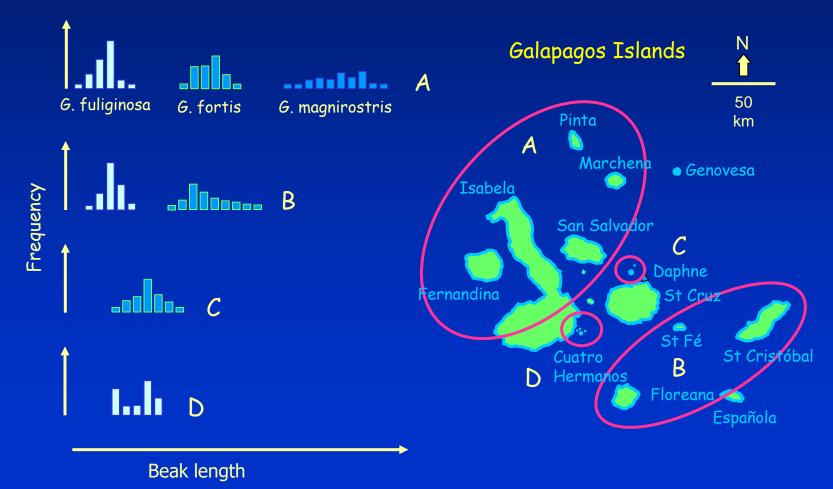
Trophic niche shift (niche apportionment)

The different species of Galapagos' finches have different trophic niches. The differences are both between species and between the populations of the same species. This can be assessed from the beak morphology.



PLASTICITY OF RESOURCES USE IN COMPETING SPECIES

Trophic niche shift (niche apportionment) occurs when two or more species are in sympatry



Guido Chelazzi









Guido Chelazzi

The meaning of the word "predation" is manyfold in ecology

Predation *sensu stricto* occurs when a population (the predator) gains materials/energy from killing and consuming individuals of a second populations (the prey). The process of predation *s.s.* thus involve subtraction of individual from one population (increasing the mortality rate of the prey) and demographic growth of the predator (increase of survival and fertility)

Other forms of "- +" interactions include:

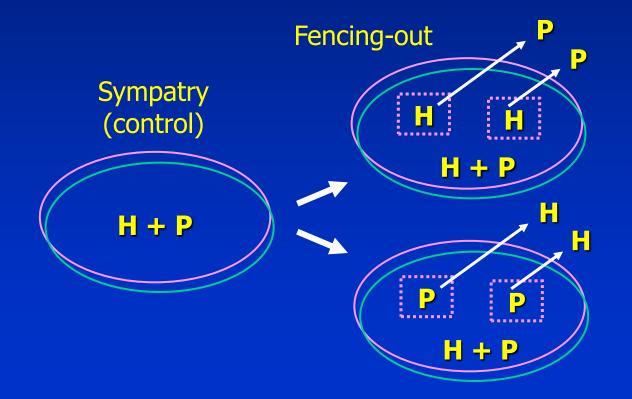
Parasitoidism, when a species (parasitoid) lays its eggs into the body of larvae or adults of a second species (host/prey) which are then consumed by larvae during their development, eventually leading to the their death

Destructive herbivory, when the consumption of parts or products of a species by a consumer (e.g. browsing, grazing, sucking) leads to death or decrease of fertility/survival of the consumed population

When a consumer utilizes parts (or products) of another species without impairing its survival/fertility the interaction can be classified as commensalism (+ 0)

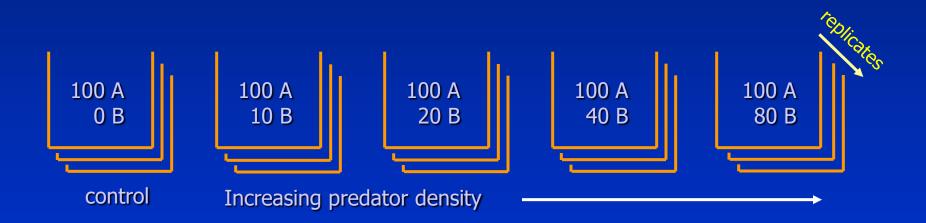
How to study empirically the demography of predation ?

Similat to competition, demographic effect of predation can be assessed by changing prey-predator densities in the field, by using fencing-out or fencing-in experiments



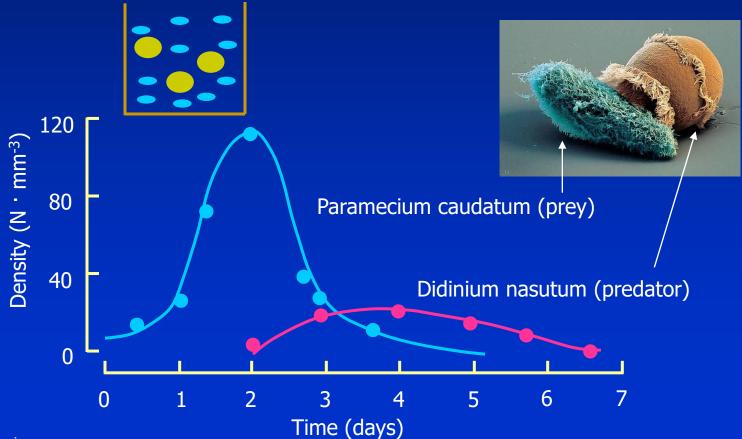
How to study empirically the demography of predation ?

Similar to competition, demographic effect of predation can be performed in the laboratory, by experiments where different densities of the prey and the predator are set as initial conditions



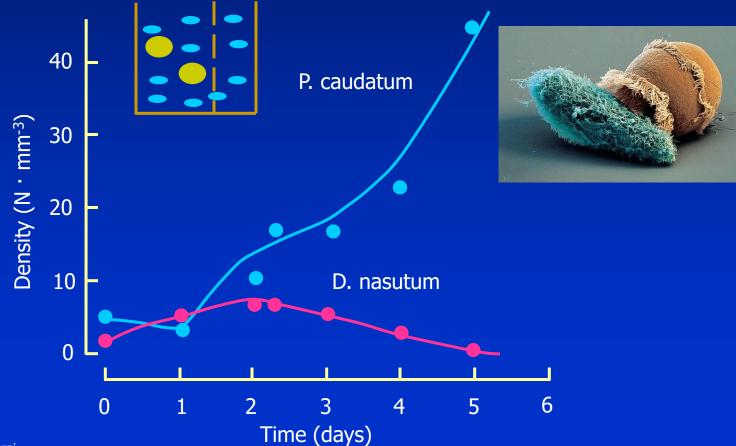
Gause's free-running experiments on a couple of protozoan species

1) Closed, homogeneous environment: one single demographic growth followed by extinction of both prey and its predator



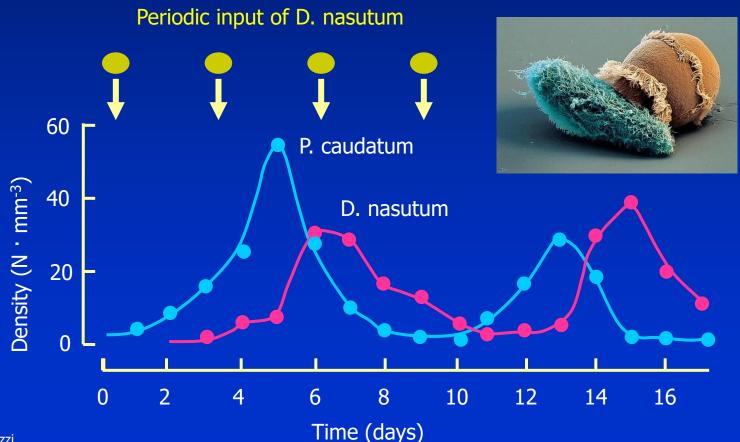
Gause's free-running experiments on a couple of protozoan species

2) Closed environment, but refugia accessible to the prey: extinction of the predator after one cycle, prey growing to its carrying capacity



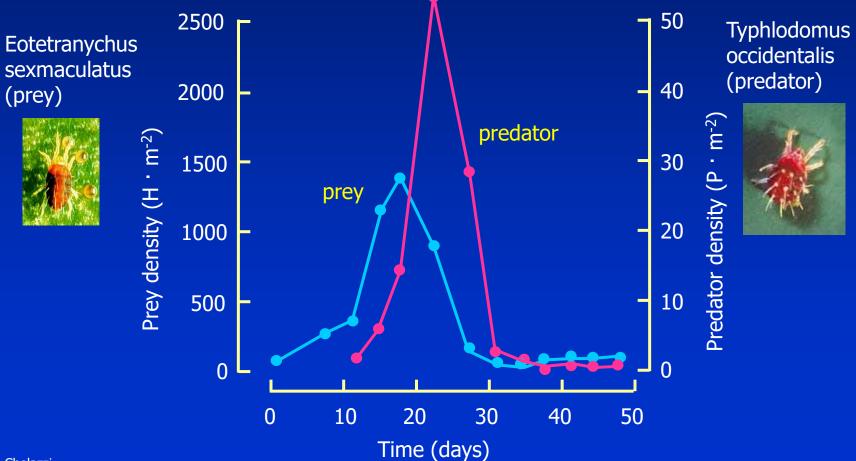
Gause's free-running experiments on a couple of protozoan species

3) Open environment, periodic immigration of the predator: demographic cycles with predator's increase-decrease shifted with respect to the prey



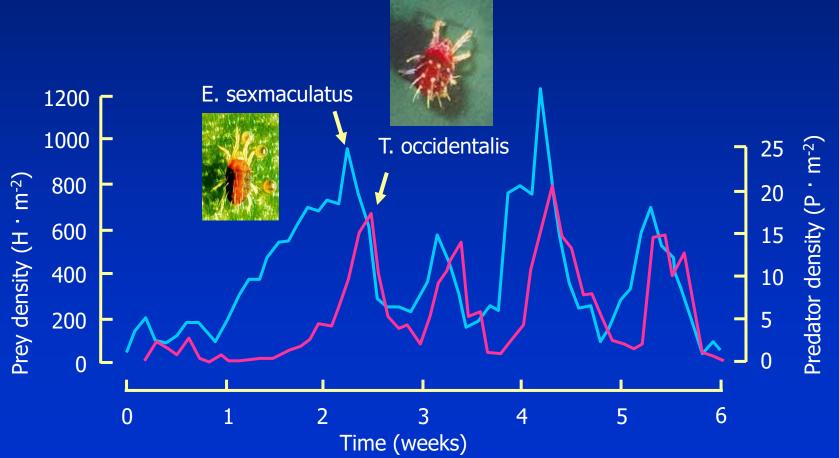
Huffaker's free-running experiments on a couple of mites (Acari)

1) Homogeneous environment: One demographic cycle, followed by extinction of the predator

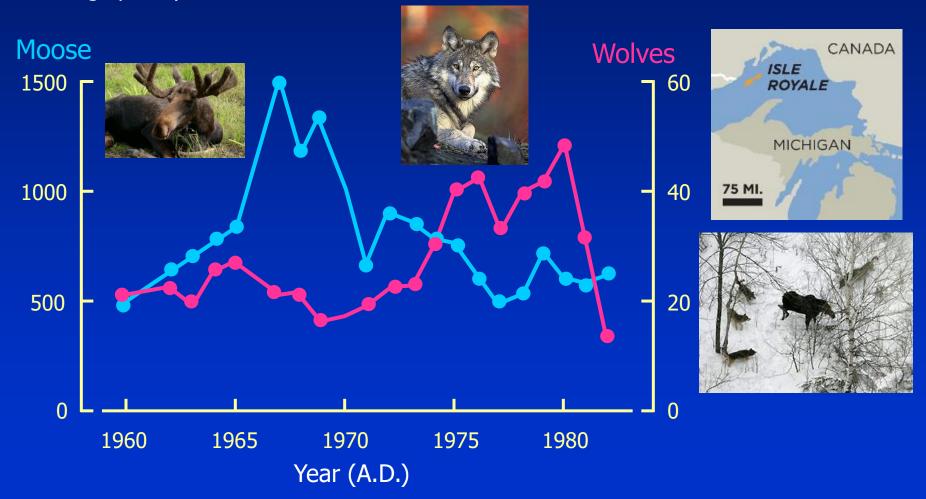


Huffaker's free-running experiments on a couple of mites (Acari)

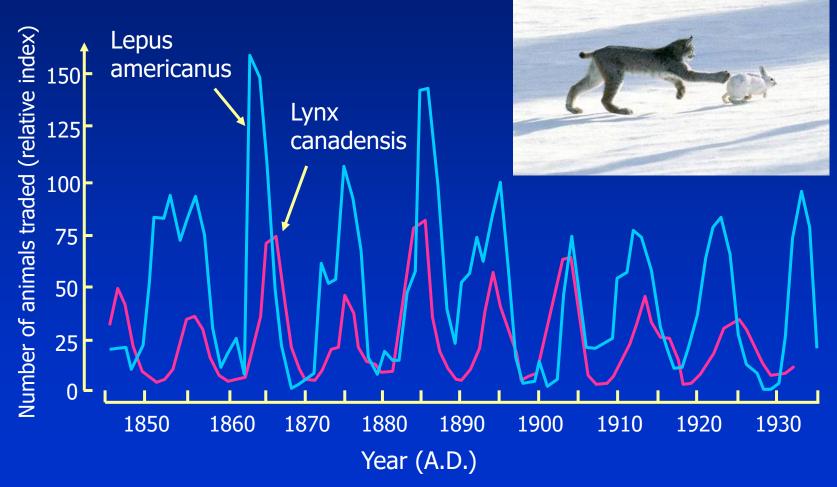
2) Partitioned environment: Sustained demographic cycles

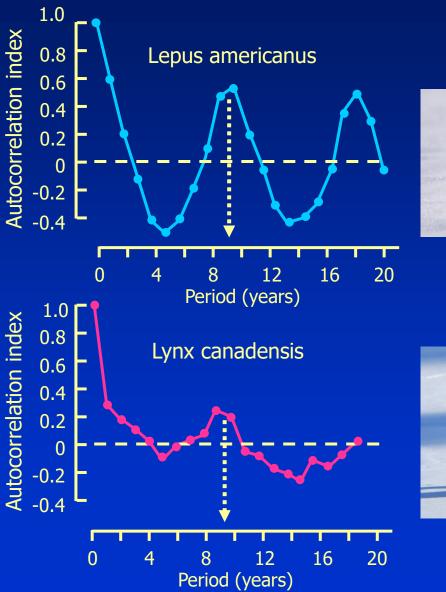


Isle Royale "free-running experiment" on wolves and moose Demographic cycles



Hudson Bay "free-running experiment" on lynx and hare in North America Demographic cycles









How to design a model for interspecific competition?

1) Parsimony: two species linked by an exclusive interaction not dependent from external constraints (homogeneous space, no climatic fluctuations, no other interactions)

2) Completeness: the model must be able to predict different outcomes (exclusion or coexistence) when changing values of the parameters. The model must be able to reproduce demographic cycles

Design two linked equations, one for each species, each including predation of species a on b

The model of Alfred Lotka and and Vito Volterra

Prey $\frac{dH}{dt} = f(H) - g(H, P)$ Predator $\frac{dP}{dt} = e \cdot g(H, P) - m(P)$

Generic equations

 $f(H) = r \cdot H$ $g(H, P) = a \cdot H \cdot P$ $m(P) = \mu \cdot P$

Specific assumptions

The original model of Alfred Lotka and and Vito Volterra

$$\frac{dH}{dt} = r \cdot H - a \cdot H \cdot P$$

$$\frac{dP}{dt} = b \cdot H \cdot P - \mu \cdot P$$

 μ = mortality rate of the predator by starvation

Prey stability

$$\frac{dH}{dt} = 0 \quad if \quad r \cdot H = a \cdot H \cdot P$$

Predator stability

$$\frac{dP}{dt} = 0 \quad if \quad b \cdot H \cdot P = \mu \cdot P$$

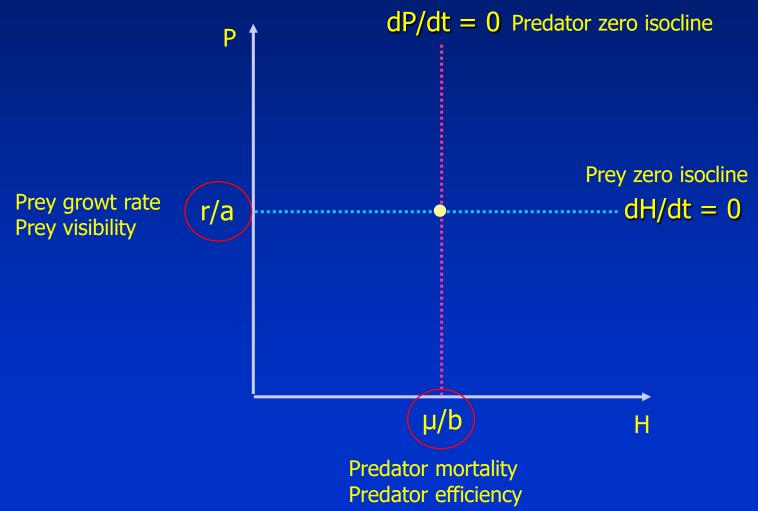
Prey zero growth isocline

$$P = \frac{r}{a}$$

Predator zero growth isocline

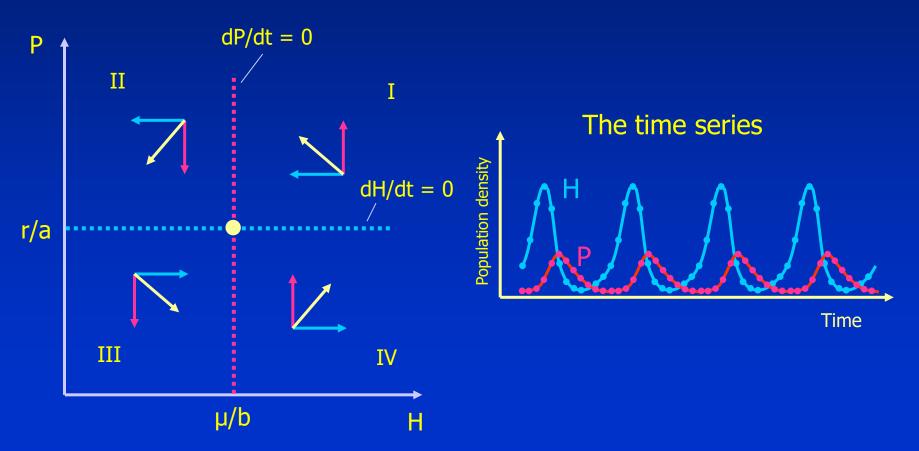
$$H = \frac{\mu}{b}$$

The original model of Alfred Lotka and and Vito Volterra

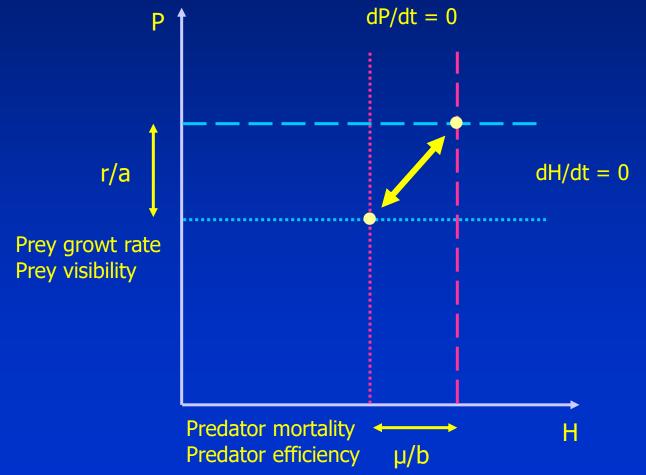


The predictions of the original model of Alfred Lotka and and Vito Volterra

The phase-plan

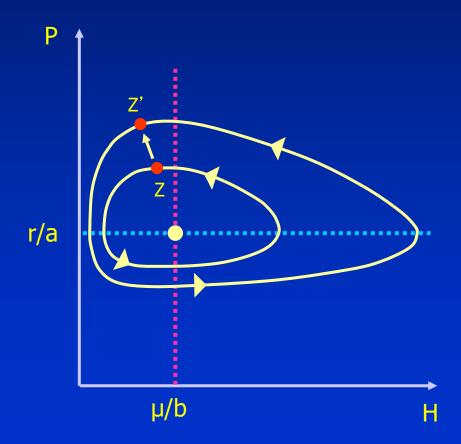


The effect of changing the biological characteristics of the predator and the prey in the original model of Alfred Lotka and and Vito Volterra



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The instability of the original model of Alfred Lotka and and Vito Volterra: a variation in the density of the two species (from Z to Z') is followed by a new cyclic pattern



The effrect of prey visibility (predation avoidance)

 \mathbf{J} $g(H,P) = a \cdot H \cdot P$

Extraction function

The whole population is exposed (standard model)

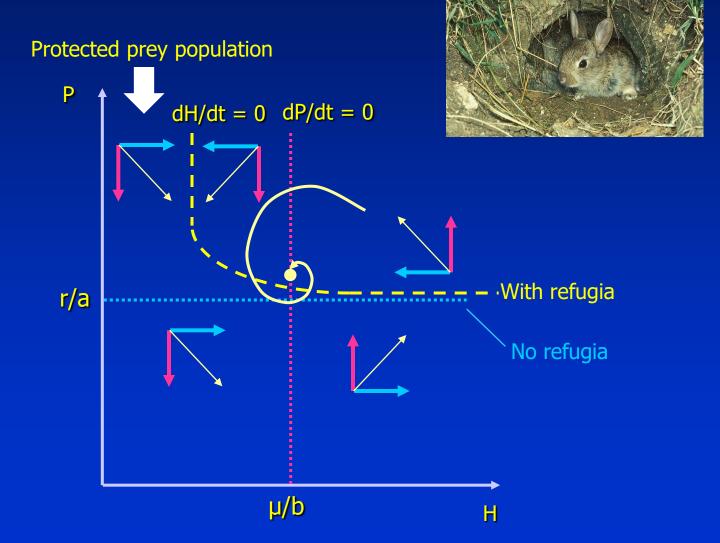
A)
$$g(H,P) = a \cdot (H - s \cdot H) \cdot P$$

A fraction (s) of the population avoid the predation (es. mimetismo criptico)

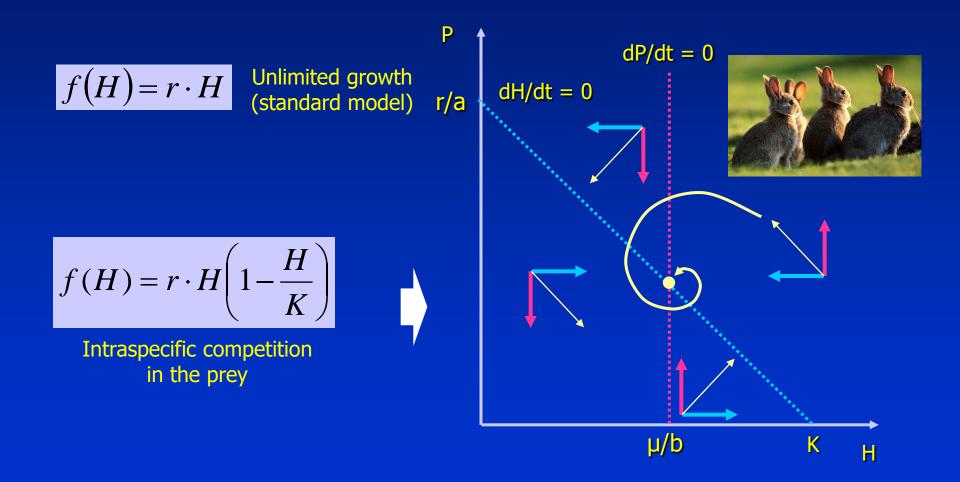
B)
$$g(H,P) = a \cdot (H-H^{-}) \cdot P$$

A constant number of preys (H⁻) avoid the predation (es. refugia)

The effrect of predation avoidance



The effrect of intraspecific competition in the prey



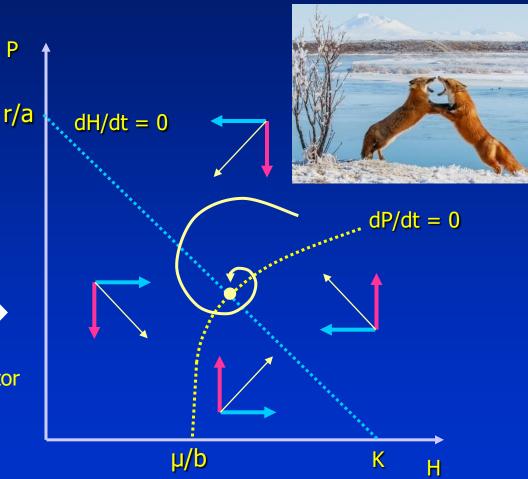
The effrect of intraspecific interference in the predator

$$g(H,P) = a \cdot H \cdot P$$

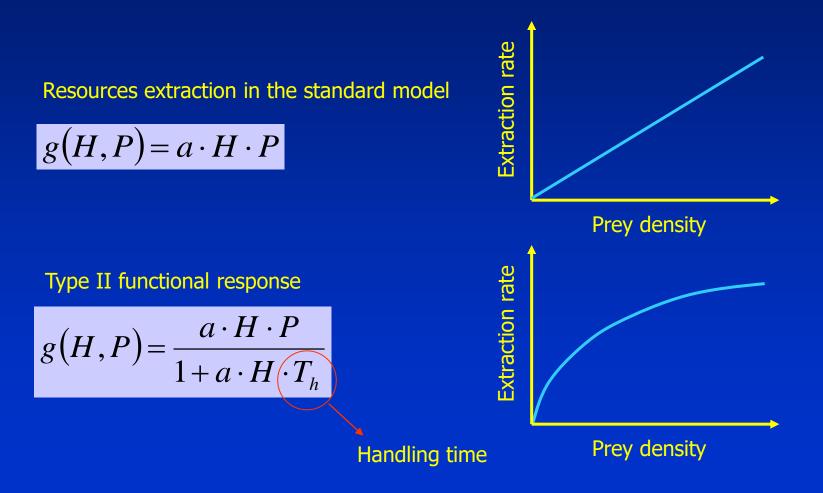
Extraction function in the standard model

$$g(H,P) = \frac{H}{P^i}$$

Mutual interference in the predator i = interference coefficient

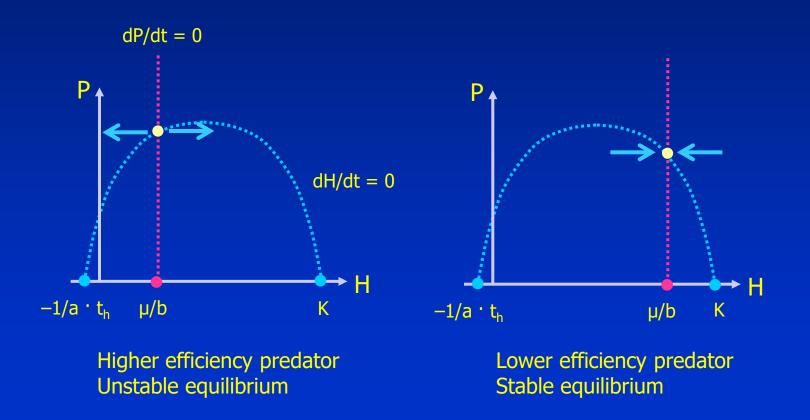


The effrect of type II functional response

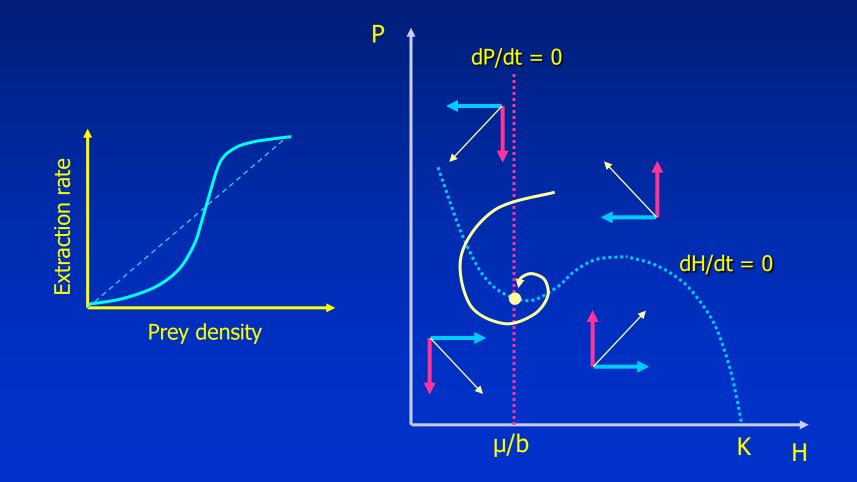


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The effrect of type II functional response depends on the characteristics of the predator



The effrect of type III (switching) functional response



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DEMOGRAPHIC EFFECTS OF HERBIVORY

Herbivory is a form of consumption in which an organism principally obtains energy/biomolecules by eating primary producers such as plants, algae, phytoplancton and photosynthesizing procariotes, or their parts or products

Such wide definition includes:

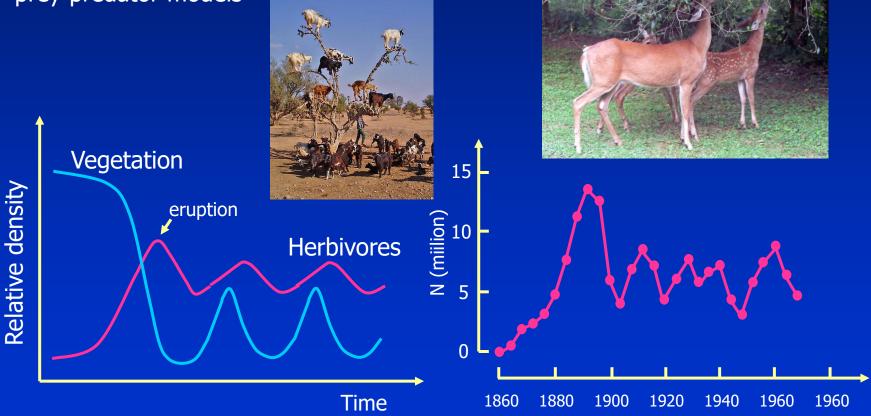
Grazers: eating parts of the stem and leaves of the vascular plants (e.g. herbs) Browsers: eating leaves or shots of vascular plants (e.g. shrubs, trees)

Frugivores Granivores Xylofages Root eaters Nectarivores Palinivores



DEMOGRAPHIC EFFECTS OF HERBIVORY

In the case of "destructive" grazing-browsing co-dinamics of herbivores and their resources Can be described and predicted by using prey-predator models



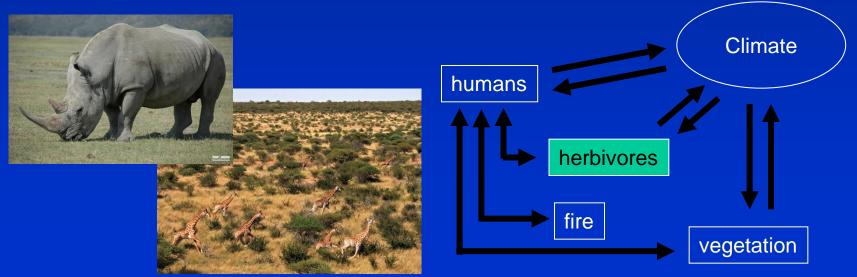
DEMOGRAPHIC EFFECTS OF NON-DESTRUCTIVE HERBIVORY

Grazing and browsing are generally conservative and regulative (i.e. individual plants are not eliminated, and their regrowth can be stimulated by g-b)

Frugivore may favour seed dispersal Nectarivores and palinivores facilitate fertilization by impollination

The demoecological role of herbivory cannot be understood without considering herbivore-plants coevolution

Herbivores have often a role of "Ecosystem Engineers", being at the core of ecological interactions and feedbacks

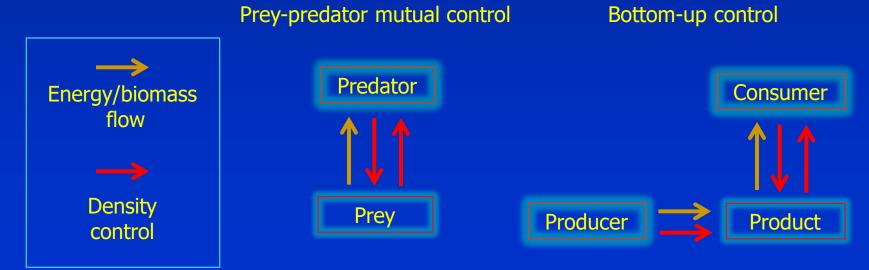


FROM HERBIVORY TO COMMENSALISM AND SAPROPHITISM

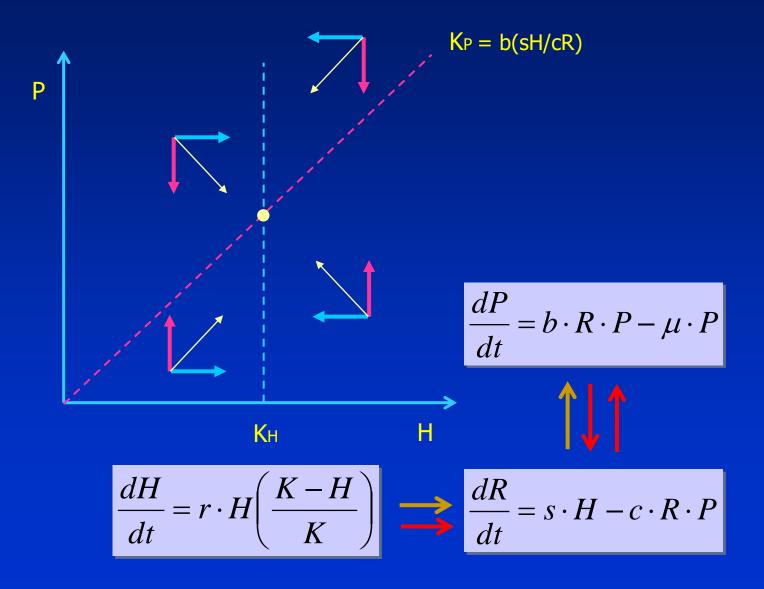
When the consumer takes food/energy from parts or products of individuals of a second species, this can lead to a +0 interaction called commensalism

The same occurs when the consumer utilizes dead individuals or part of individuals of a second species, which is currently called saprophitism

In both cases the density of exploited population can control the density of exploiter, but not the contrary. This leads to asymmetric, bottom-up demographic regulation along the food chain



BOTTOM-UP DEMOGRAPHIC CONTROL OF "+0" RELATIONSHIPS



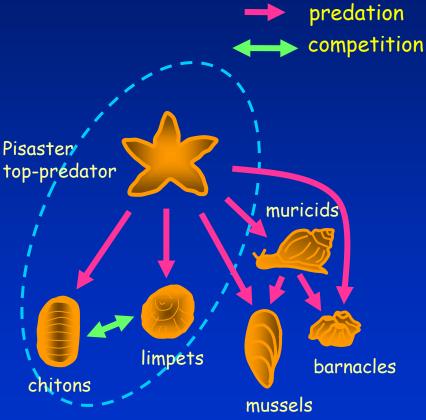
A simplified picture of the intertidal community along the rocky shores of Nort-Western America

The echinoderm Pisaster ochraceus is a top predator preying on different species of the community, including grazing molluscs (chitons and limpets) which compete for microalgae

Pisaster has a shifting behaviour, preferring more dense species

This has the benefit to regulate the competion between species (e.g. chitons and limpets)

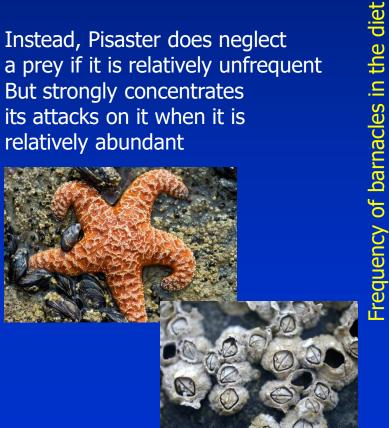
If Pisaster is removed from the shore competitive exclusion occurs between the other species and the demographic equilibria among them are brocken

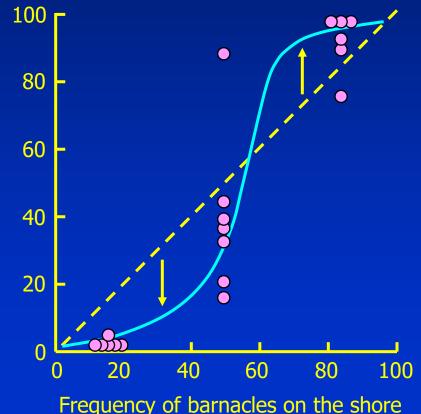


The density-dependent, shifting predation of Pisaster

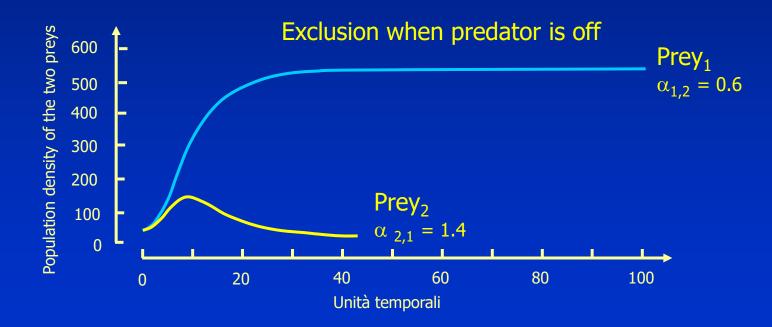
The frequency of attacks toward a given prey (e.g. barnacles) is not linearly related to their abundance on the shore.

Instead, Pisaster does neglect a prey if it is relatively unfrequent But strongly concentrates its attacks on it when it is relatively abundant



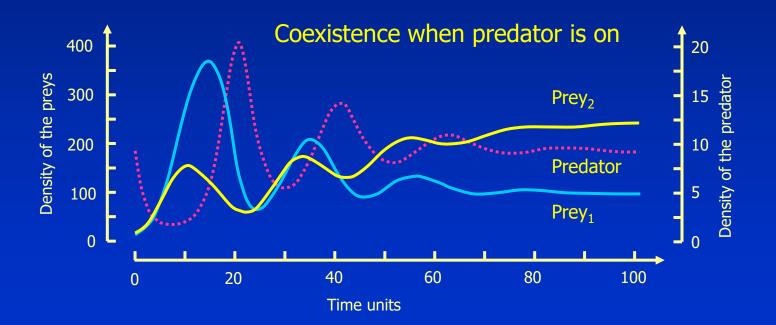


If the simulation is repeated including only the two competing species (without the predator) competitive exclusion is evident, displacing one of the two species (undercompetitor) from the habitat



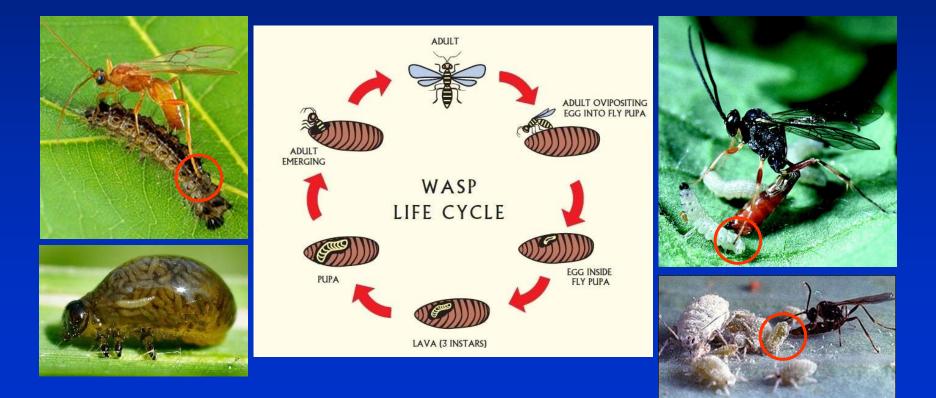
A simulation using L-V model of the dynamics of two competing species one of which (Prey2) is made overcompetitor on the other (Prey1) by assigning opportune values of alfa (a 2,1 = 1.4; a 1,2 = 0.6).

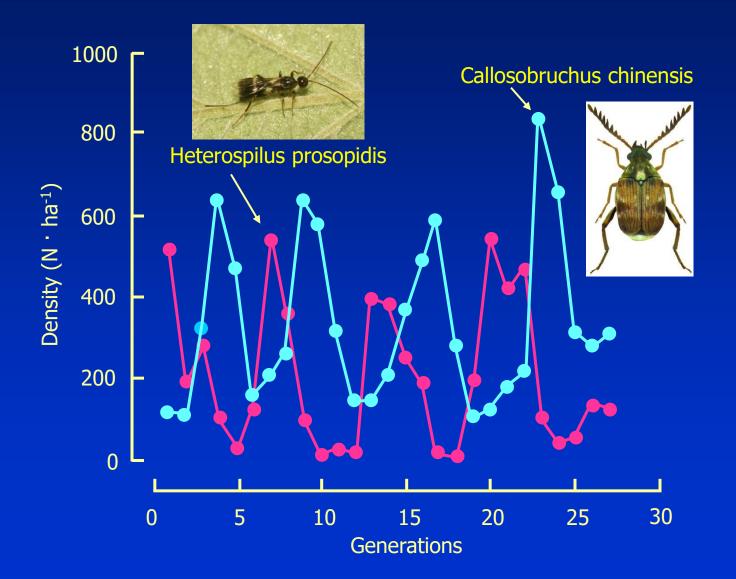
The model includes a third species (Predator) assumed to have a switching predation pattern, according to the density of each prey



The special case of the parasitoids

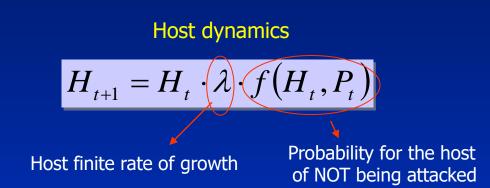
20% of Insects (particularly Mymenoptera Ichneumonidae) But also Diptera and Coleoptera Host-preys: other Hymenoptera, Coleoptera, Diptera, Araccnida, Gastropoda

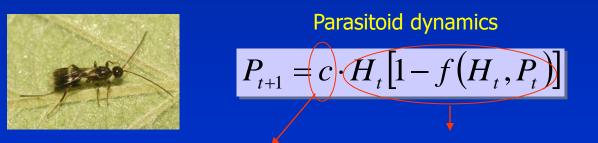




The standard Nicholson & Bailey discrete-time model



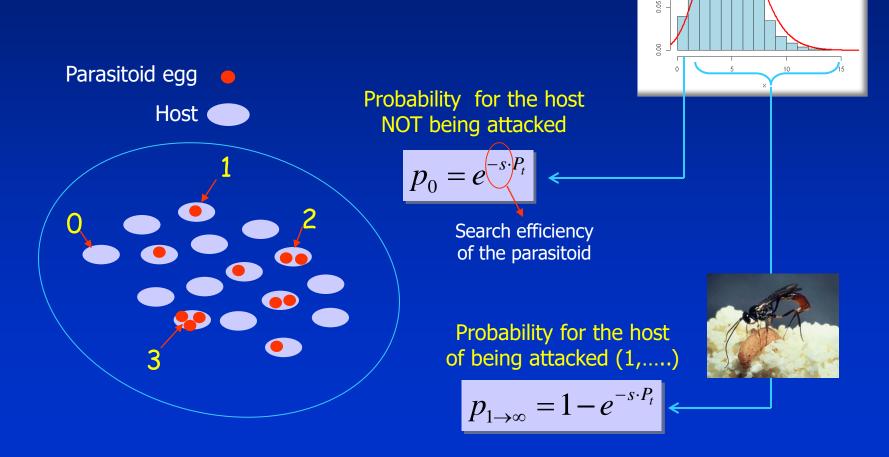




Hatching rate of parasitoid (n. newborns per host attacked)

Probability for the host of being attacked

The standard Nicholson & Bailey model Random attack: Poisson distribution



0.15

Density 0.10

The standard Nicholson & Bailey discrete-time model



Host dynamics

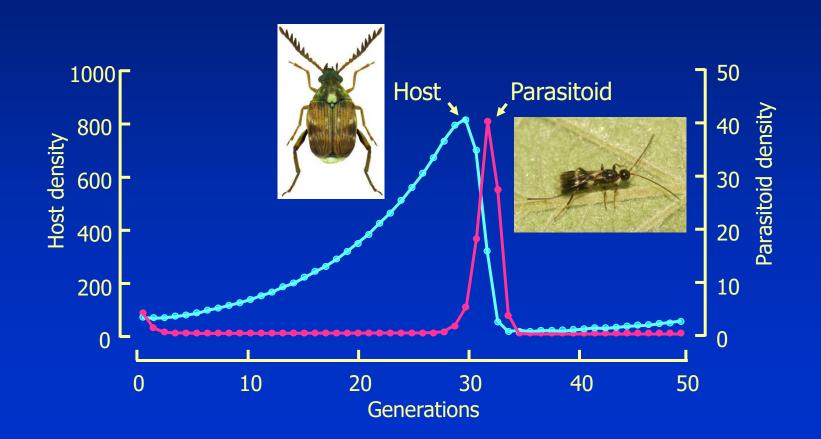
$$H_{t+1} = H_t \cdot \lambda \cdot e^{-s \cdot P_t}$$



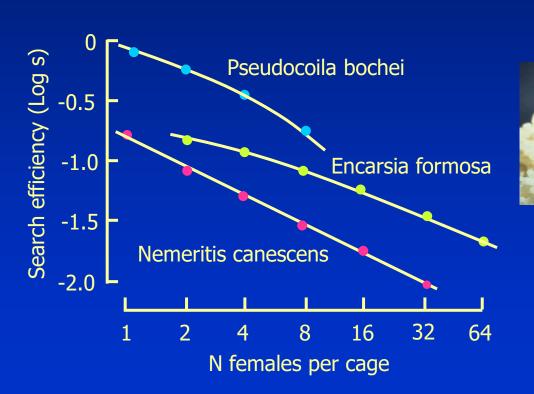
$$P_{t+1} = c \cdot H_t \cdot \left(1 - e^{-s \cdot P_t}\right)$$

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The standard Nicholson & Bailey discrete-time model

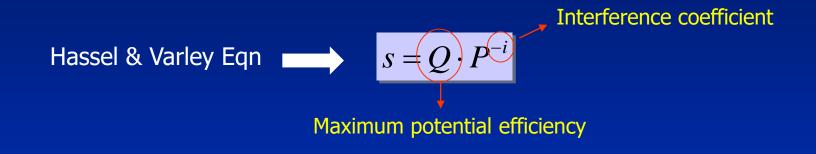


Interference within the parasitoid population





Interference within the parasitoid population



Nicholson & Bailey with mutual interference

$$H_{t+1} = H_t \cdot \lambda \cdot e^{-Q \cdot P_t^{1-i}}$$

$$P_{t+1} = c \cdot H_t \cdot \left(1 - e^{-Q \cdot P_t^{1-i}} \right)$$

Competition within the host population

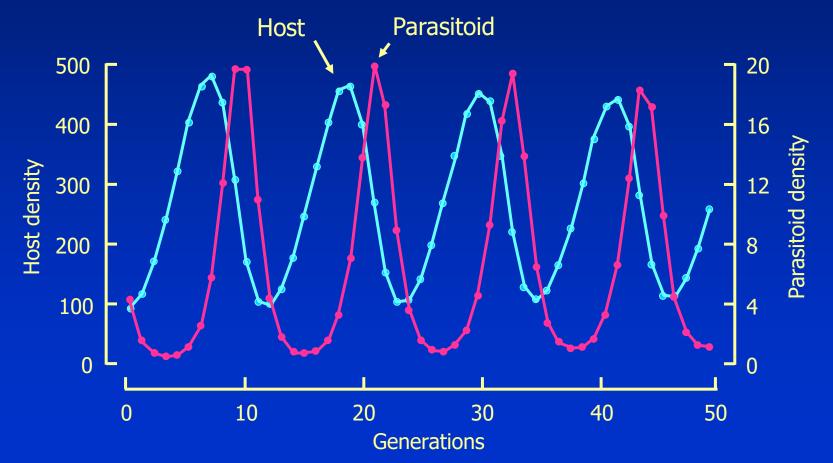
Host dynamics

$$H_{t+1} = H_t \cdot \lambda \cdot \left(\frac{1}{1 + a \cdot H_t} \right) e^{-sP_t}$$

(e.g. Maynard Smith & Slatkin)

HOST-PARASITOID CO-DYNAMICS

Simulation with implemented N & B model (interference+competition)



HOST-PARASITOID CO-DYNAMICS

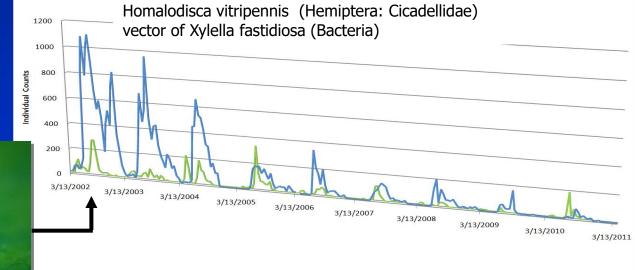
Use of parasitoids in the biological control of pests



Biological Control of Insect Pests Using Egg Parasitoids



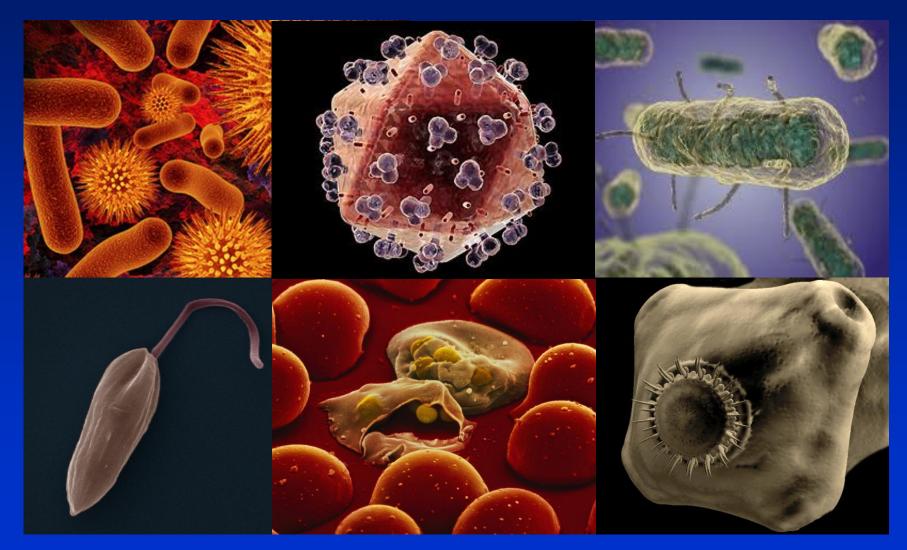
GWSS and Natural Enemy Phenology in Southern California

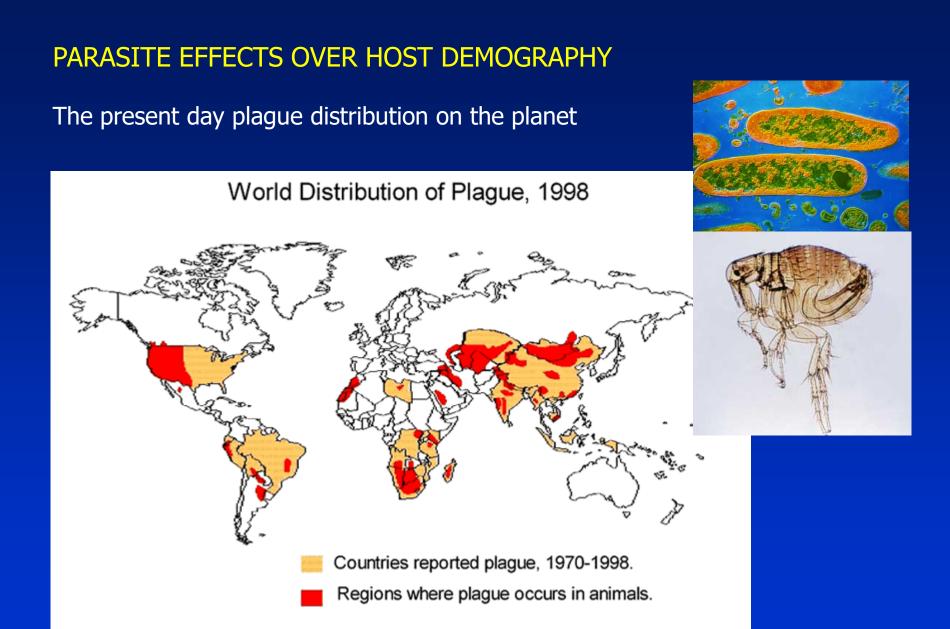


GWSS adults 🛛 🖾 Parasitoid fauna

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D Springer

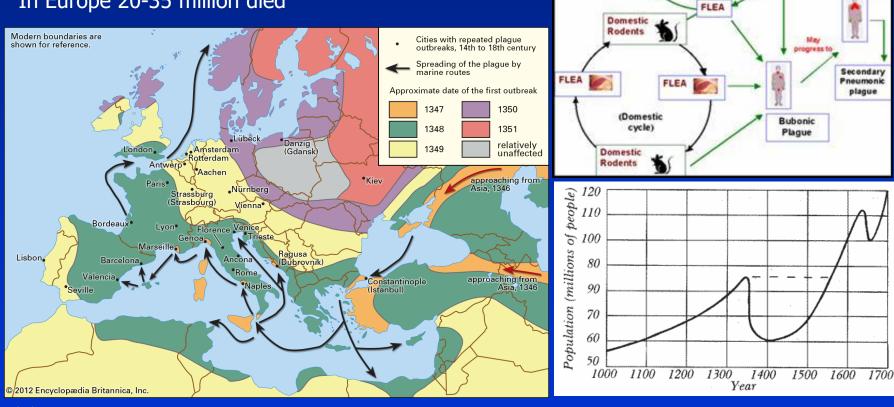




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The black plague in Europe (1347-1350)

The Black Death originated in or near China and spread into Western Eurasia. It may have reduced world population from an estimated 450 down to 350 million by the year 1400. In Europe 20-35 million died



FLEA

Wild Rodent cycle

Wild Rodents

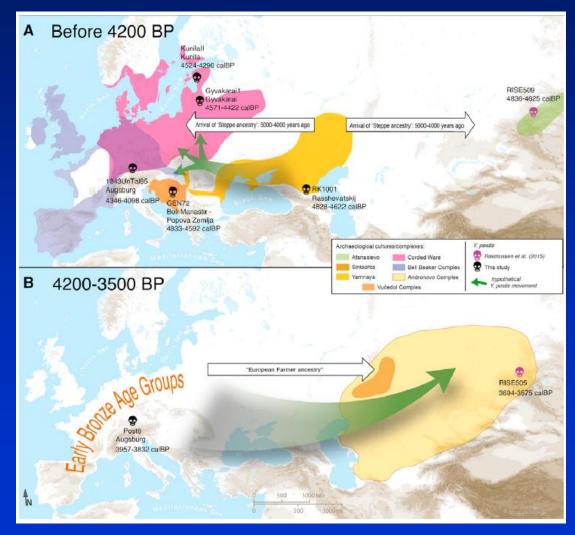
Primary

Plague

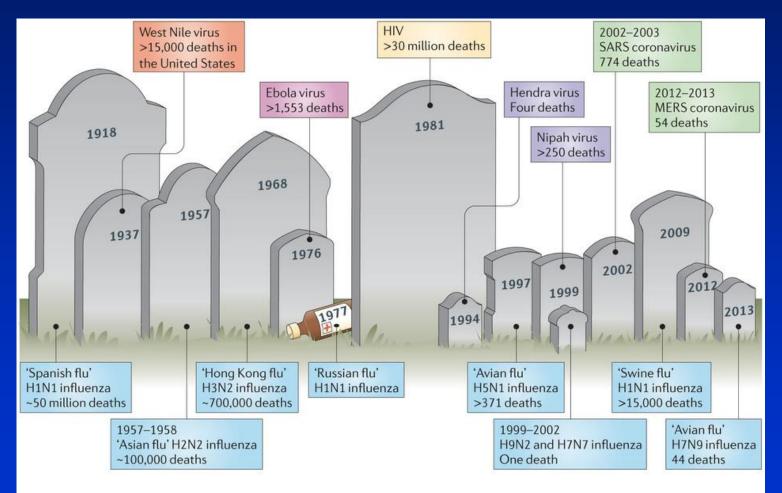
Wild

Rodents

Late Neolithic and Early Bronze Age spread of black plague in Eurasia



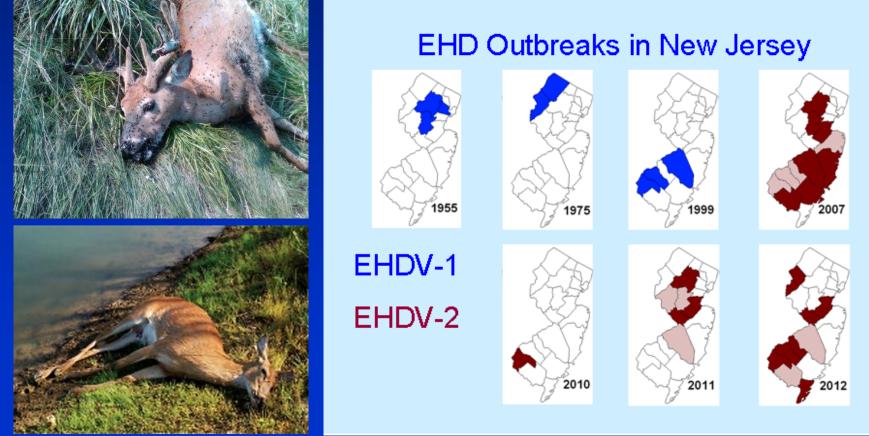
The impact of recent infection diseases on human populations



Nature Reviews | Immunology

Epizootic Hemorrhagic Disease (EHD)

A disease of white-tailed deer (*Odocoileus virginianus*) caused by an infection of a virus from the genus *Orbivirus*, transmitted by biological vectors (in N America the midge, *Culicoides variipennis*)



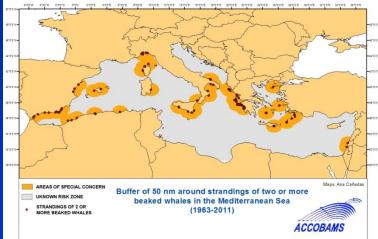
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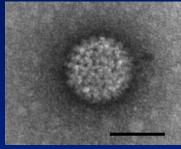
Epizootic diseases in marine mammals due to Paramyxoviruses

Phocine distemper virus Cetacean morbillivirus

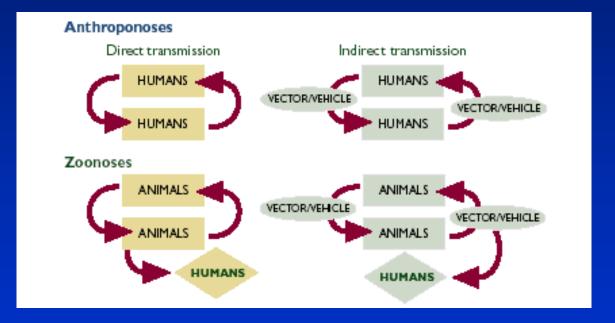




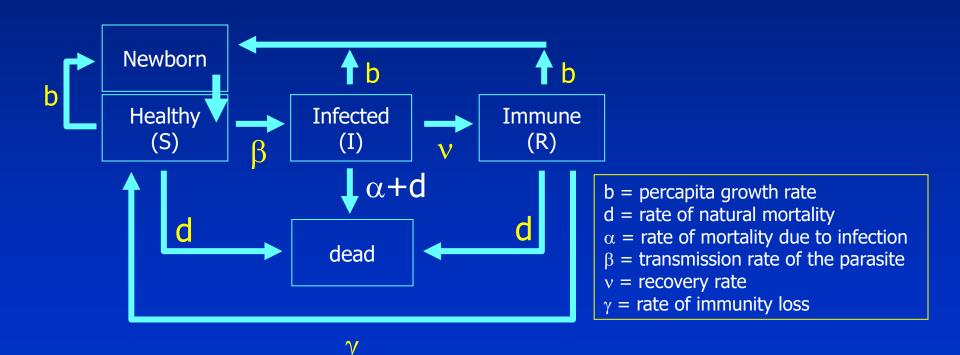




Anthroponoses and Zoonoses



The Anderson & May approach



The Anderson & May approach

$$\frac{dS}{dt} = b \cdot (S + I + R) - d \cdot S - \beta \cdot S \cdot I + \gamma \cdot R$$
 Healthy - Susceptible
$$\frac{dI}{dt} = \beta \cdot S \cdot I - (\alpha + d + \nu) \cdot I$$
 Infected
$$dR$$

$$\frac{dR}{dt} = v \cdot I - (d + \gamma) \cdot R$$
 Recovered - Immune

From the above eqns, by posing r = b-dwe obtain the predicted dinamics of the whole population

$$\frac{dN}{dt} = r \cdot (S + I + R) - \alpha \cdot I$$

- b = percapita growth rate
- d = rate of natural mortality
- α = rate of mortality due to infection
- β = transmission rate of the parasite
- v = recovery rate
- γ = rate of immunity loss

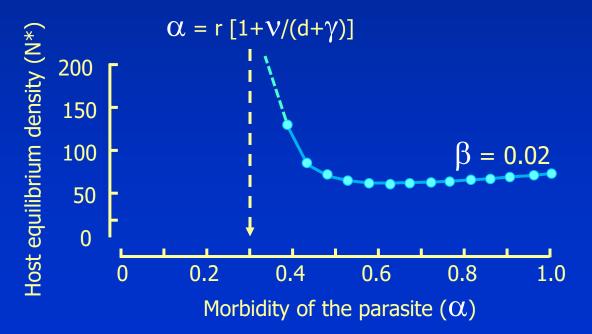
The Anderson & May approach

When the rate of mortality is so that:

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

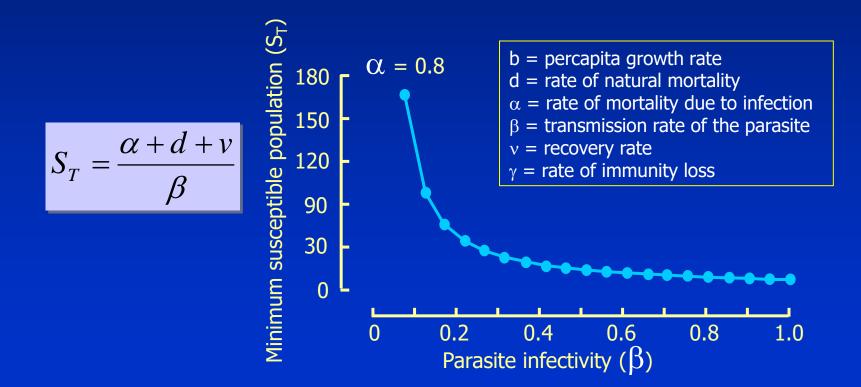
- b = percapita growth rate
- d = rate of natural mortality
- r = b-d
- α = rate of mortality due to infection
- β = transmission rate of the parasite
- v = recovery rate
- γ = rate of immunity loss

Then, the infection does <u>regulate</u> the host population at a stable density N^* which depends on the transmission rate



The Anderson & May approach

The A & M model predicts the minimum susceptible host population (S_T) necessary to keep the microparasite infeinfection in an endemic form, which depends on α , β , v and d



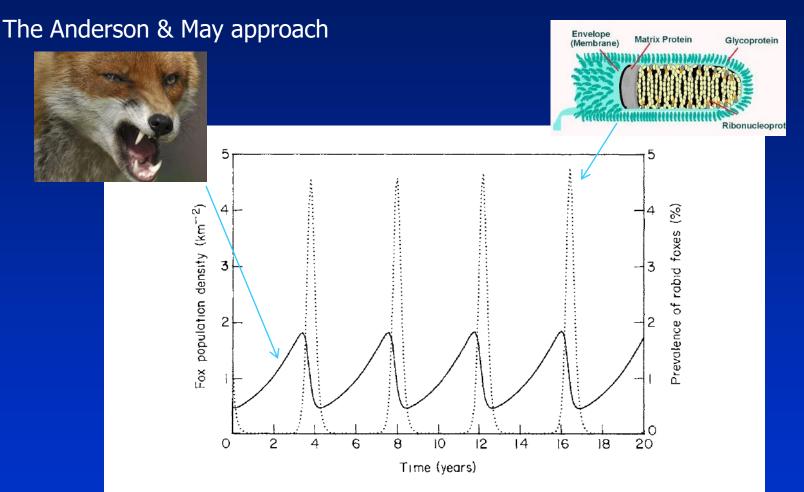


FIG. 3. Oscillatory fluctuations in total fox population density and the density of rabid foxes generated by a simple model of the interaction between the rabies virus and its host population (see Anderson *et al.* 1981). Parameter values; disease-free carrying capacity, 10 km², r=0.5 year⁻¹, $\beta=80$ year⁻¹, $\sigma=13$ year⁻¹, $\alpha=73$ year⁻¹.

The Hyman & Stanley (1988) model for HIV

$$\frac{dS(t)}{dt} = \mu(S_0 - S(t)) - \lambda(t)S(t),$$
$$\frac{dI(t)}{dt} = \lambda(t)S(t) - (\gamma + \mu)I(t),$$
$$dA(t)$$

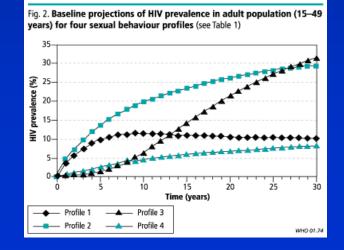
$$\frac{dA(t)}{dt} = \gamma I(t) - \delta A(t),$$

$$\frac{dA_T(t)}{dt} = \gamma I(t),$$

t = time

S(t) = number of susceptible individuals, I(t) = number of infected individuals without AIDS, A(t) = number of AIDS cases, $A_T(t) =$ accumulated number of AIDS cases, N(t) = number of susceptible and infected individuals without AIDS, $\mu =$ death rate of individuals without AIDS, $\delta =$ death rate of individuals with AIDS, $\gamma =$ rate of developing AIDS of infected individuals, i = probability of infection from a sexual contact with an infected, c = average number of contacts between sexual partners, r = average number of new sexual partners per year, and

 S_0 = population size before the AIDS virus was introduced.



Classical demoecology refers to populations evenly distributed in a structureless habitat, however:

Real populations have often complex spatial distributions consisting of different local populations connected in a network

Local populations occupy portions of ecologically suitable patches (conditions/resources) interspersed by a matrix of unsuitable habitats

Connection between local populations is based on mobility of individuals (and genes) from one population to other population

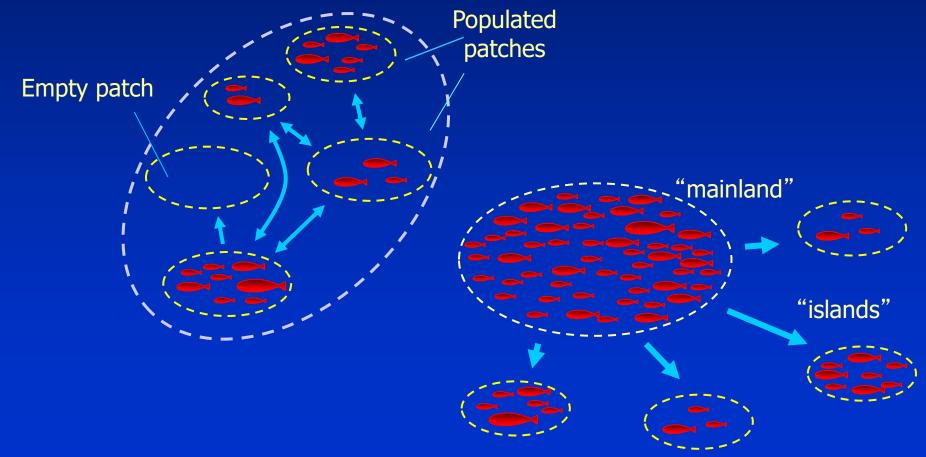
Ecological corridors maintain the connectivity of the system

Such network of interconnected local populations is called a metapopulation

Some good reasons to consider metapopulation approach in demoecology

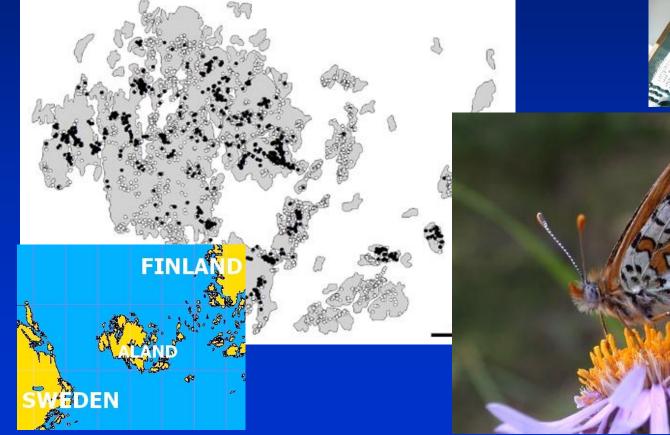
- Many (if not most) natural populations are in effect metapopulations
- Metapopulations reveal emerging properties with respect to the single populatons of the network
- Metapopulation approach opens new horizons to the conservation and restoration strategies, e.g. local vs global conservation
- Metapopulation approach is the basis for restoration strategies e.g. design of natural reserves (one large vs many small)
- Habitat fragmentation due to human activities (e.g. deforestation, building of infrastructures) create metapopulation structures: which consequences for the long term species/populations survival?

Different arrangements of metapopulations



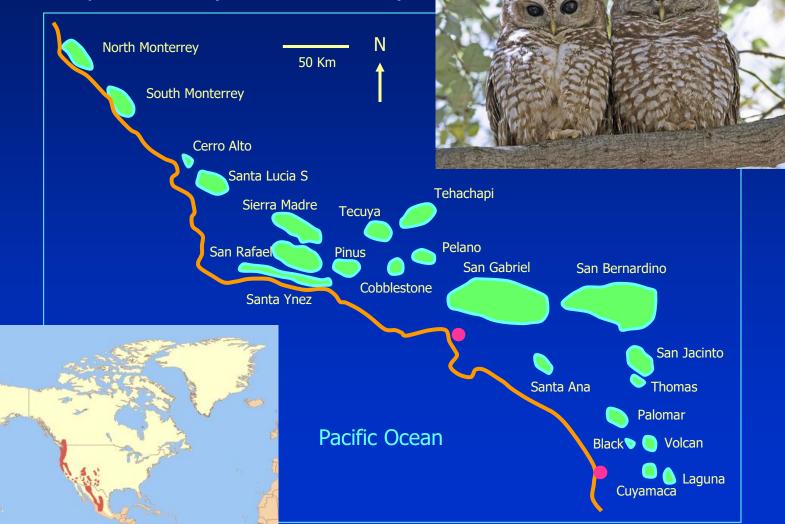
Glainville Fritillary butterfly (*Melitaea cinxia*) in the Aland Islands

Metapopulation structure – Ilkka Hanski



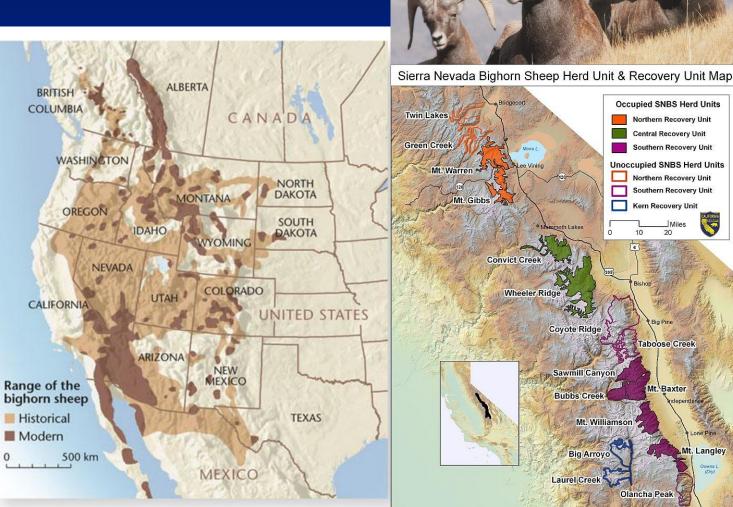


California Spotted Owl (Strix occidentalis)



Guido Chelazzi

Bighorn Sheeps (Ovis canadensis)



The spatially implicit Levins model (patch dynamics)

$$\frac{dP}{dt} = c \cdot P \cdot (1 - P) - e \cdot P$$

$$\frac{dP}{dt} = (c-e) \cdot P \cdot \begin{pmatrix} 1 - \frac{P}{1 - \frac{e}{c}} \end{pmatrix}$$

Local populations extinction rate

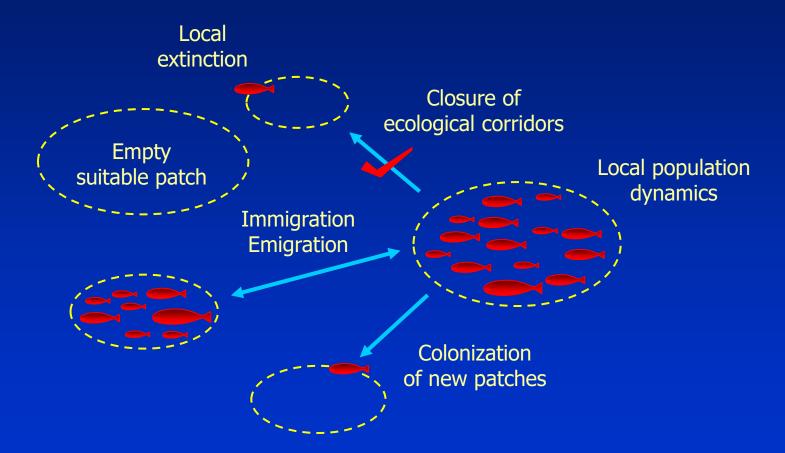
New patches colonization rate

"carrying capacity" of the metapopulation

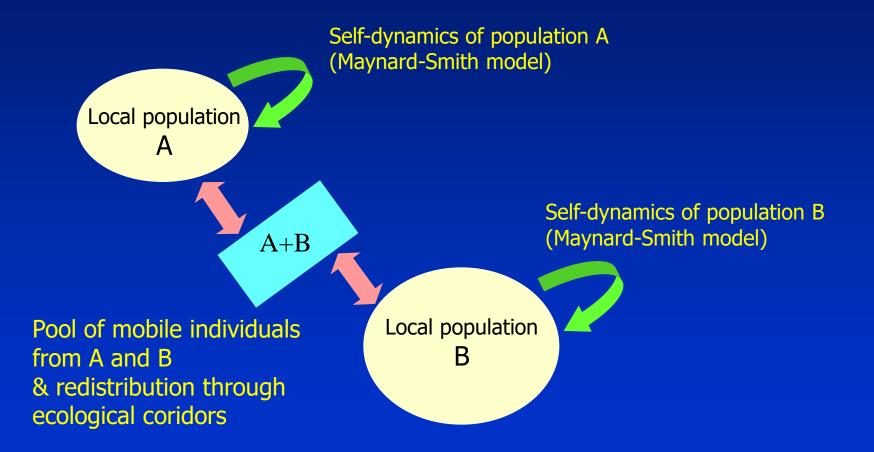
The metapopulation persists only if
$$c/e>1$$

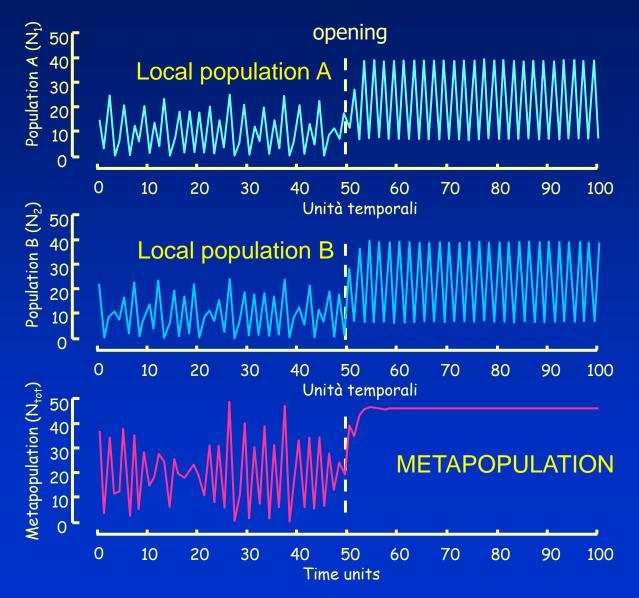
D* = 1 c/c

Spatially explicit models



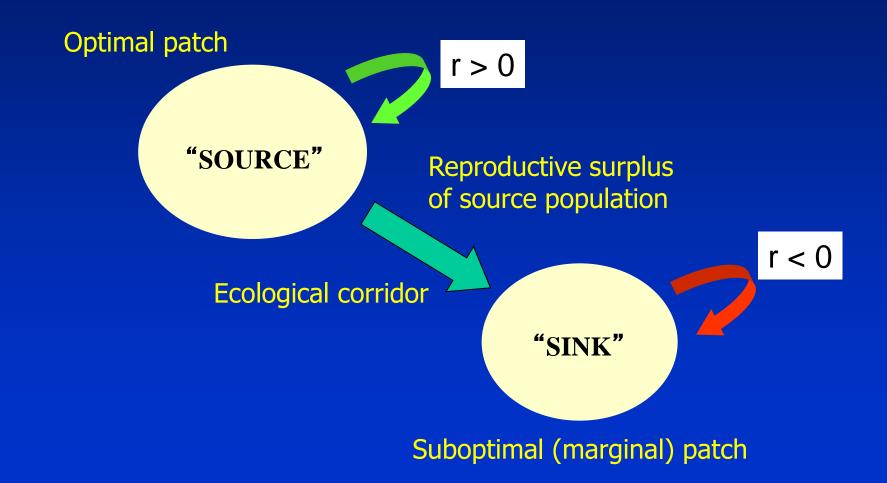
The spatially explicit Hanski model: source-sink populations





Guido Chelazzi

The spatially explicit Hanski model: source-sink populations



The rescue effect

Reduction of Local Extinction (stochasticity & inbreeding)