Teórica 8:
Interacciones interespecíficas:

Parasitismo y
enfermedades infecciosas

Repaso Teórica 7: Depredación

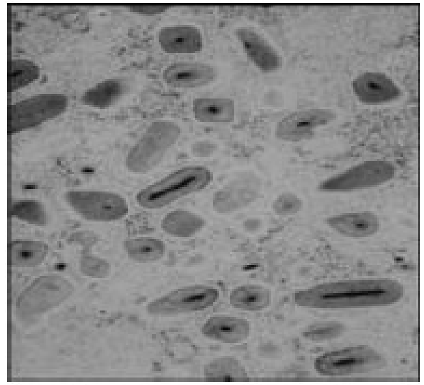
- ¿Cómo podemos modificar el modelo exponencial para incorporar interacciones predador-presa? ¿Es estable este modelo?
- ¿Qué factores contribuyen a la estabilidad de los sistemas predador-presa?
- ¿Qué son las respuestas numérica y funcional?

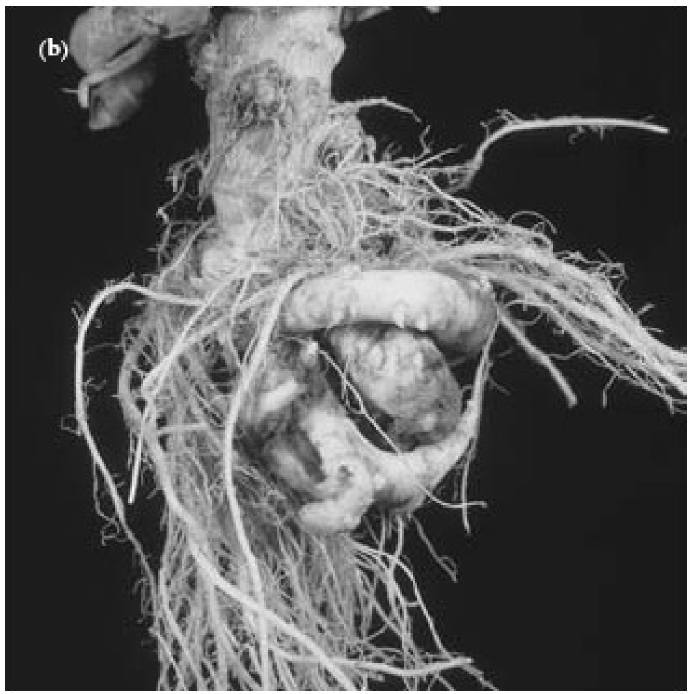
Teórica 8: Esquema conceptual

- Tipos de párasitos: micro y macroparásitos
- Ejemplos de parasitismo
- Modelos de compartimientos de dinámica hospedador-parásito
- Ejemplos de dinámica hospedador-parásito
- Efectos de los parásitos sobre sus hospedadores a nivel individual y poblacional
- Evolución de interacciones hospedador-parásito

Tipos de parásitos

- Microparásitos: Pequeños y frecuentemente intracelulares, se reproducen dentro o sobre sus hospedadores (virus, bacterias y protozoos).
- Macroparásitos: Crecen, pero no se reproducen, sobre sus hospedadores, producen estadios infectivos especializados, intercelulares (helmintos, artrópodos, etc.).





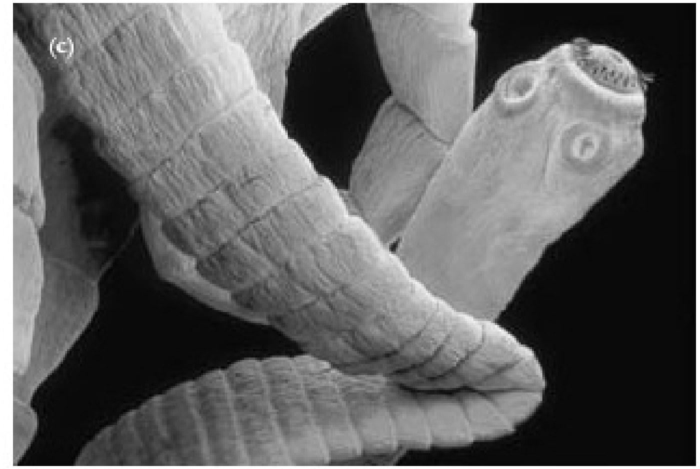


Figure 12.1 Plant and animal micro- and macroparasites. (a) An animal microparasite: particles of *the Plodia interpunctella* granulovirus (each within its protein coat) within a cell of their insect host. (b) A plant microparasite: 'club-root disease' of crucifers caused by multiplication of *Plasmodiophora brassicae*. (c) An animal macroparasite: a tapeworm. (d) A plant macroparasite: powdery mildew lesions. Reproduced by permission of: (a) Dr Caroline Griffiths; (b) Holt Studios/Nigel Cattlin; (c) Andrew Syred/Science Photo Library; and (d) Geoff Kidd/Science Photo Library.

Figure 12.2 A cuckoo in the nest. Reproduced by permission of FLPA/Martin B. Withers.

Video de abeja cleptoparásita *Coelioxys* cf *conoidea* visitando nido de abeja cortadora de hojas *Megachile* sp. Fuente: https://en.wikipedia.org/wiki/Coelioxys.

10 causas principales de muerte en la población humana mundial

World	Deaths in millions	% of deaths
Coronary heart disease	7.20	12.2
Stroke and other cerebrovascular diseases	5.71	9.7
Lower respiratory infections	4.18	7.1
Chronic obstructive pulmonary disease	3.02	5.1
Diarrhoeal diseases	2.16	3.7
HIV/AIDS	2.04	3.5
Tuberculosis	1.46	2.5
Trachea, bronchus, lung cancers	1.32	2.3
Road traffic accidents	1.27	2.2
Prematurity and low birth weight	1.18	2.0

Total enfermedades infecciosas 16.8%

Fuente: Organización Mundial de la Salud, 2004

Los parásitos en los ecosistemas

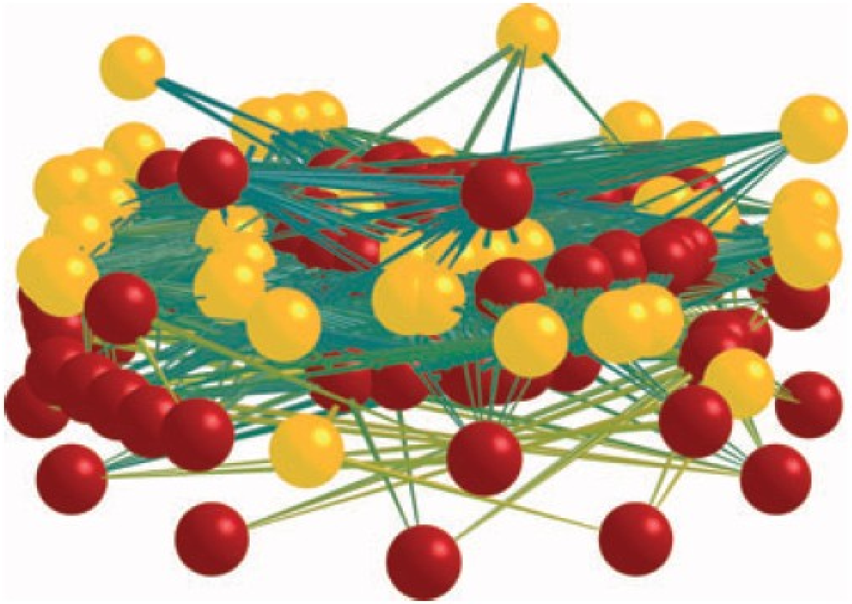


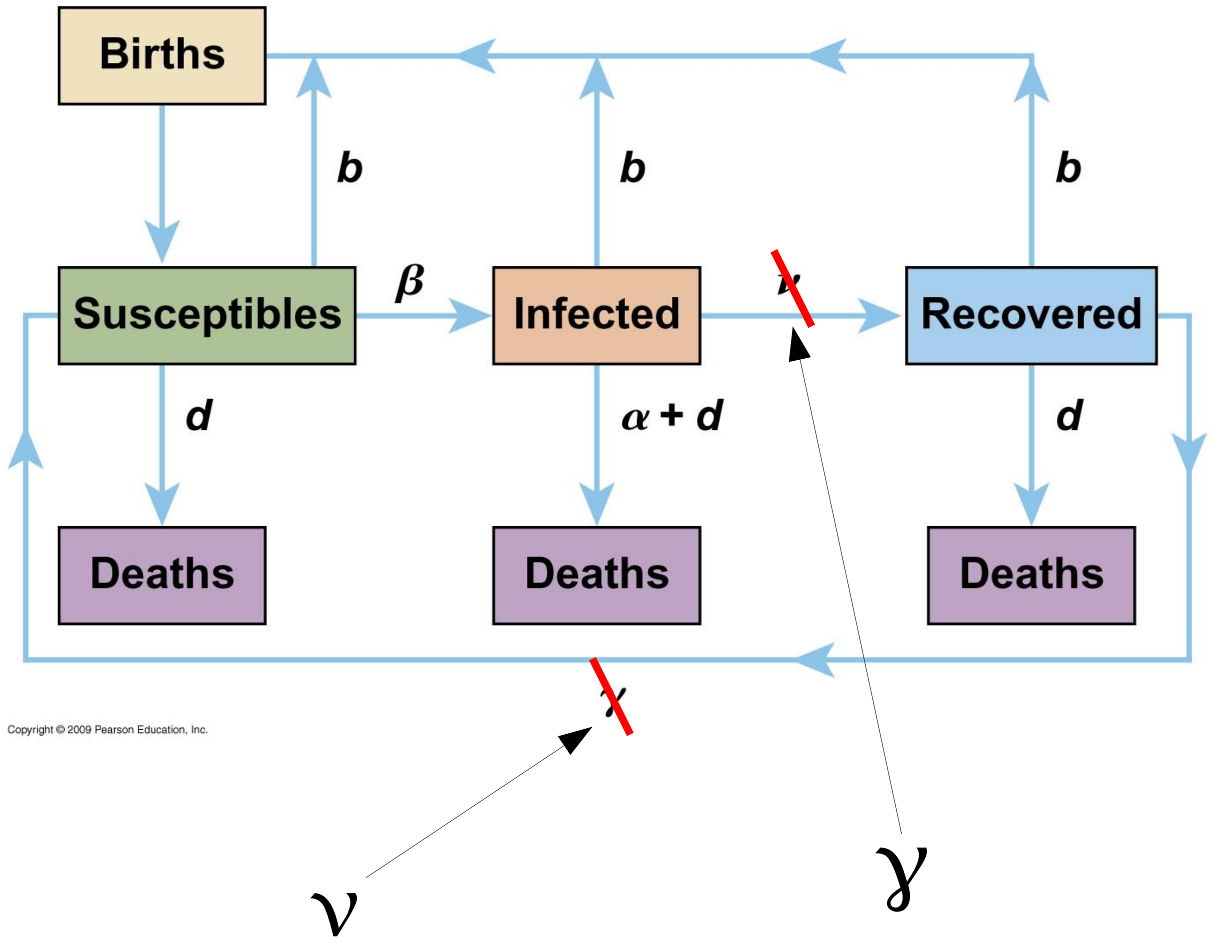
Figure 2 Three-dimensional visualization of the complexity of real food webs with parasites using data from the Carpinteria Salt Marsh Web (Lafferty *et al.* 2006b). Image produced with software available from the Pacific Ecoinformatics and Computational Ecology Lab, http://www.foodwebs.org. Balls are nodes that represent species. Parasites are the light-shaded balls and free-living species are the dark-shaded balls. Sticks are the links that connect balls through consumption. Basal trophic levels are on the bottom; upper trophic levels are on the top.

Fuente: Lafferty et al. (2008) Ecology Letters 11: 533-546

Dinámica de la transmisión: Modelos de compartimientos S-I-R

Roy Anderson

Robert May



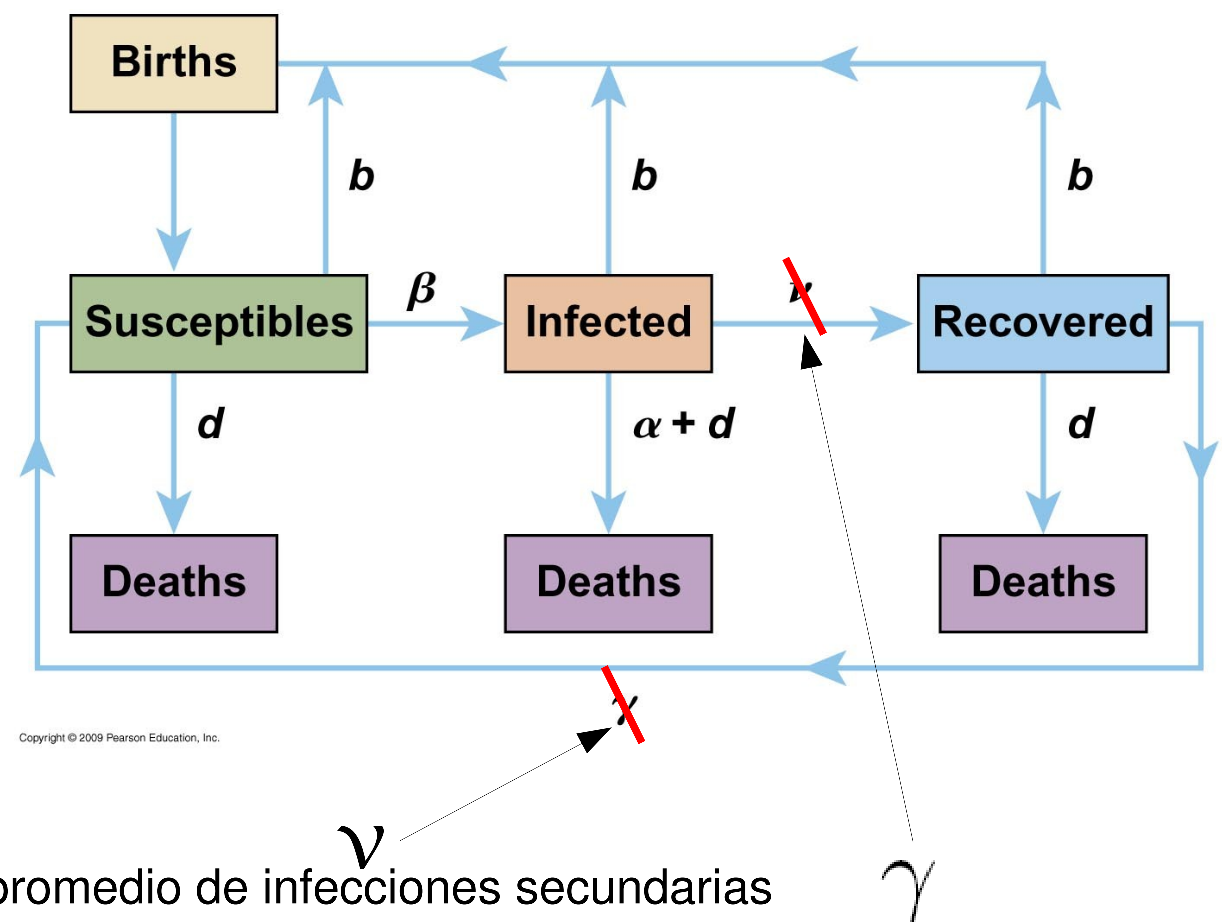
Dinámica de la transmisión: Densidad de hospedador constante

$$\frac{dX}{dt} = -\beta XY$$

$$\frac{dY}{dt} = \beta XY - \gamma Y$$

$$\frac{dZ}{dt} = \gamma Y$$

$$R_0 = \frac{\beta X}{\gamma}$$



Número promedio de infecciones secundarias producidas por un individuo infectado. Para que haya epidemia, $R_0 > 1$.

Cálculo de R_0

Para que haya transmisión, $dY/dt > 0$, o

$$\beta XY - \gamma Y > 0$$

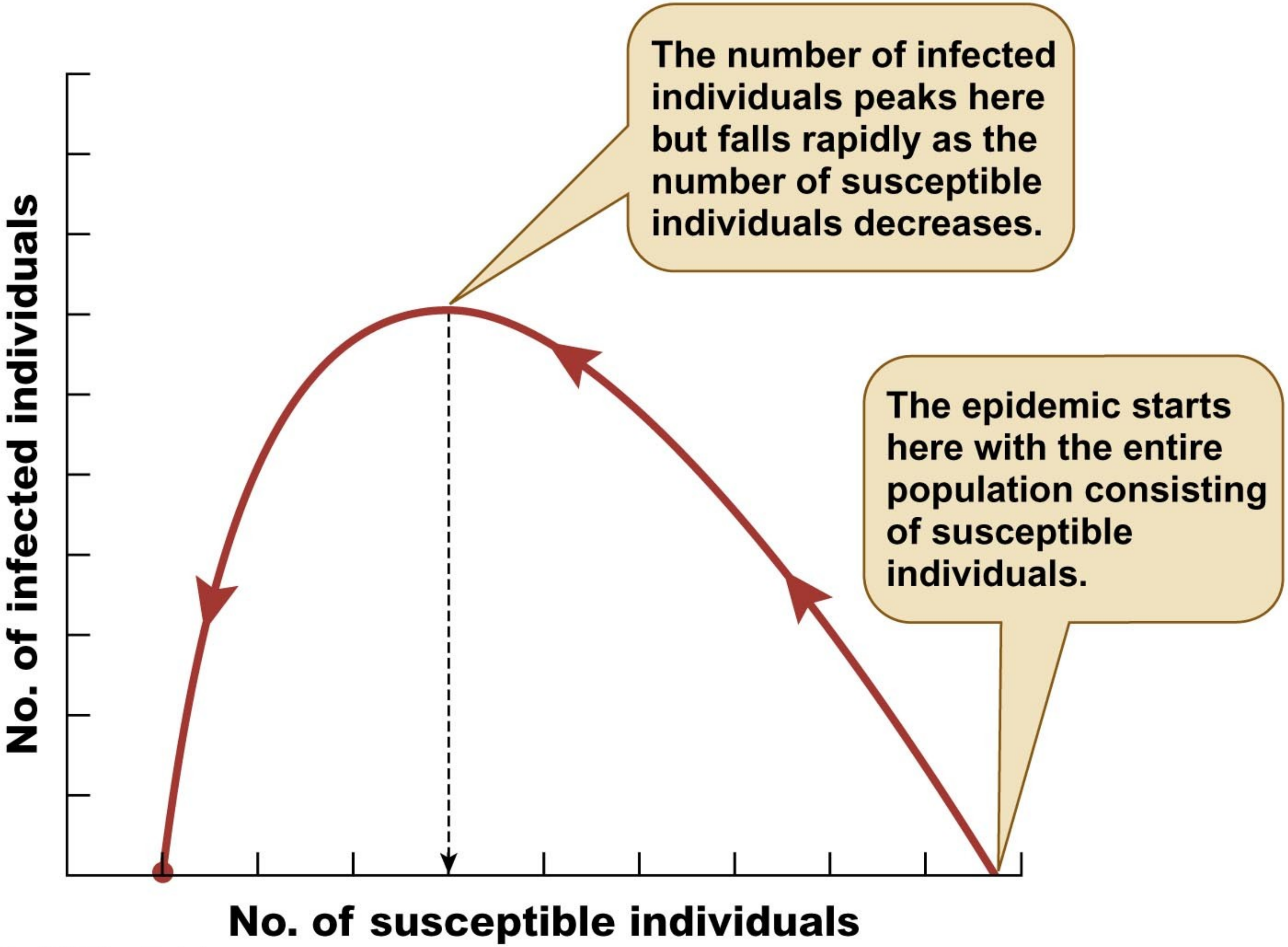
Simplificando,

$$\beta X - \gamma > 0$$

$$\beta X > \gamma$$

$$\frac{\beta X}{\gamma} > 1 \quad \xrightarrow{\text{Definimos}} \quad \boxed{R_0 = \frac{\beta X}{\gamma}}$$

Dinámica de la transmisión: Densidad de hospedador constante



Control de enfemedades infecciosas por vacunación

Si vacunamos a una proporción *c* de la población, los individuos susceptibles serán $(1 - c)X$, y entonces

$$R_0 = \frac{(1-c)\beta X}{\gamma}$$

Para evitar una epidemia, $R_0 < 1$. Entonces

$$\frac{(1-c)\beta X}{\gamma} < 1$$

y

$$c > 1 - \frac{\gamma}{\beta X} = 1 - \frac{1}{R_0}$$

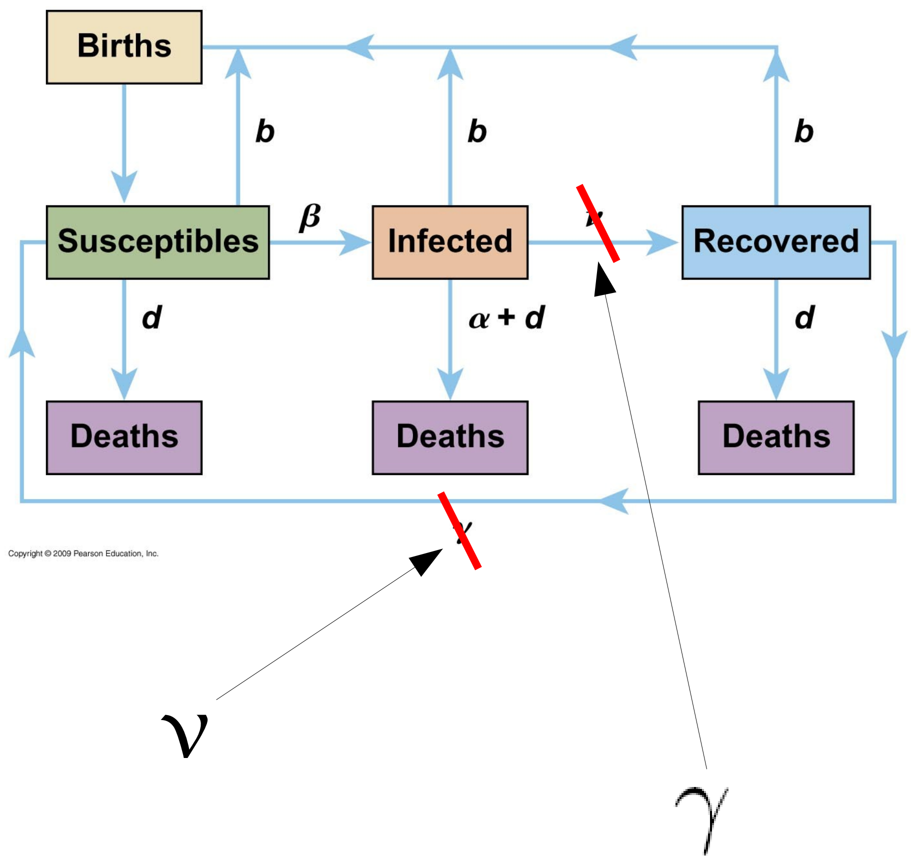
Dinámica de la transmisión: Densidad de hospedador variable

$$\frac{dX}{dt} = bN - dX - \beta XY + \nu Z$$

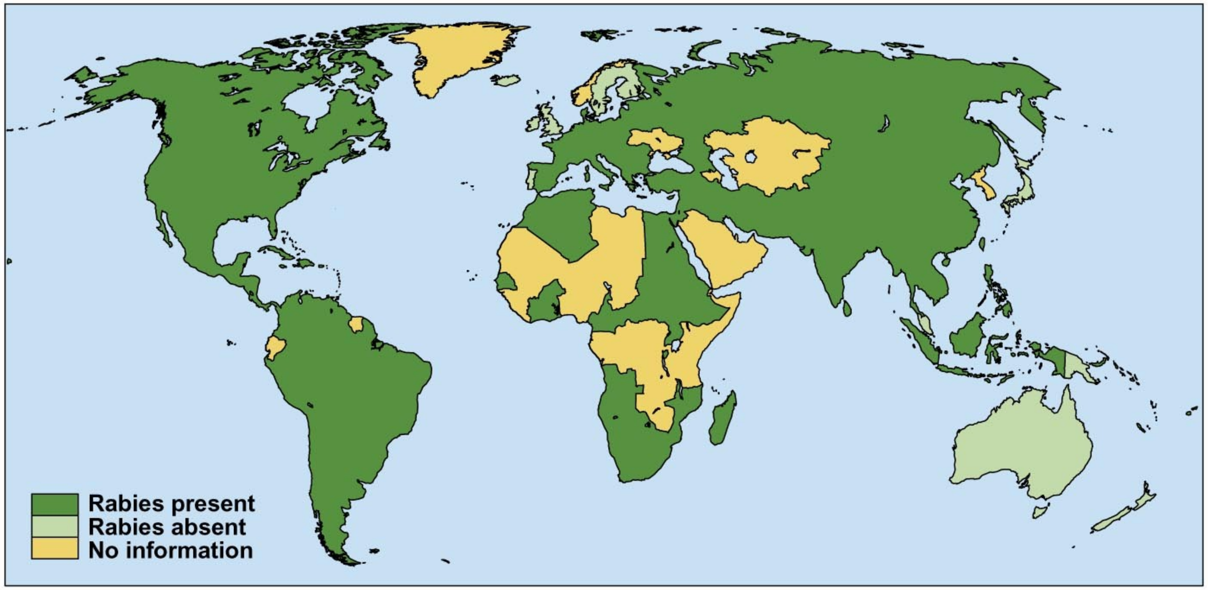
$$\frac{dY}{dt} = \beta XY - (\alpha + d + \gamma)Y$$

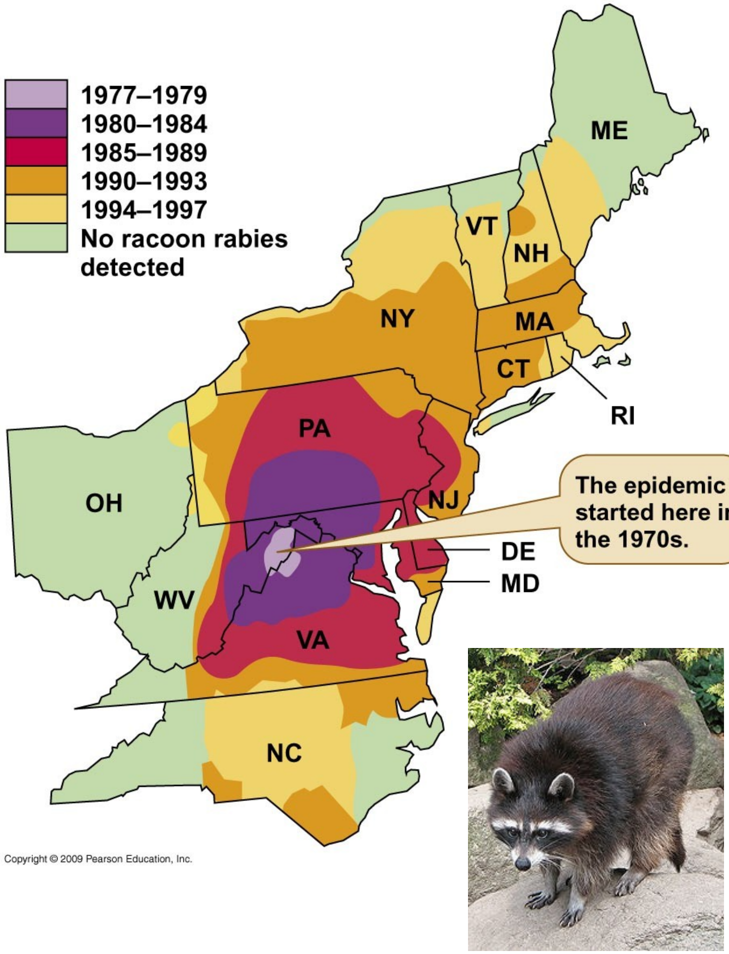
$$\frac{dZ}{dt} = \gamma Y - (d + \nu)Z$$

$$R_0 = \frac{\beta X}{\alpha + d + \gamma}$$



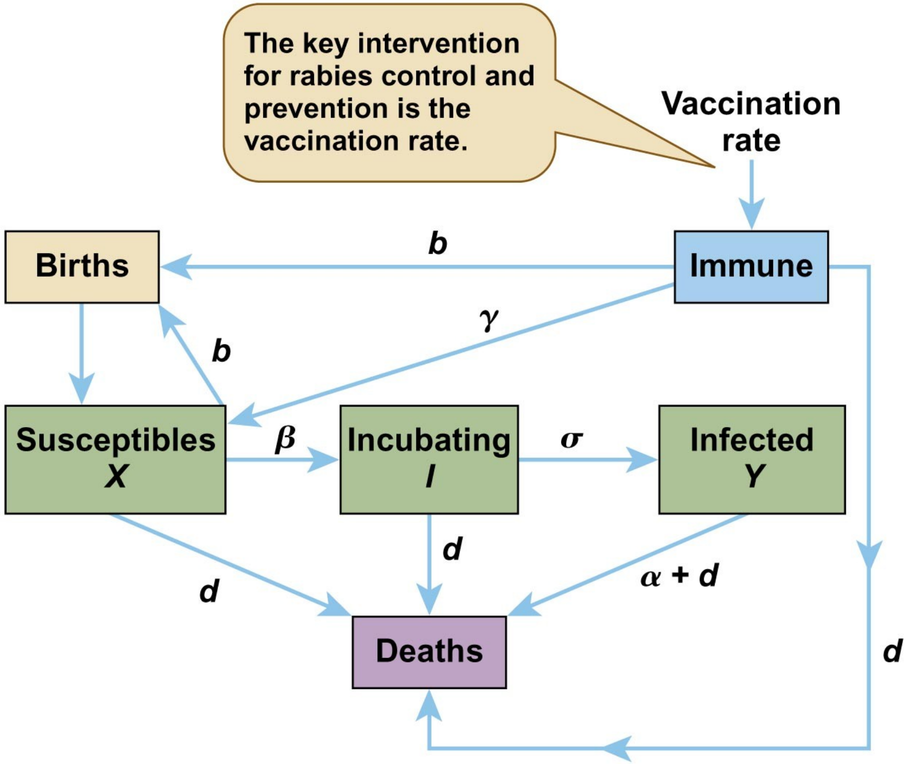
Ejemplos: Rabia en mamíferos silvestres

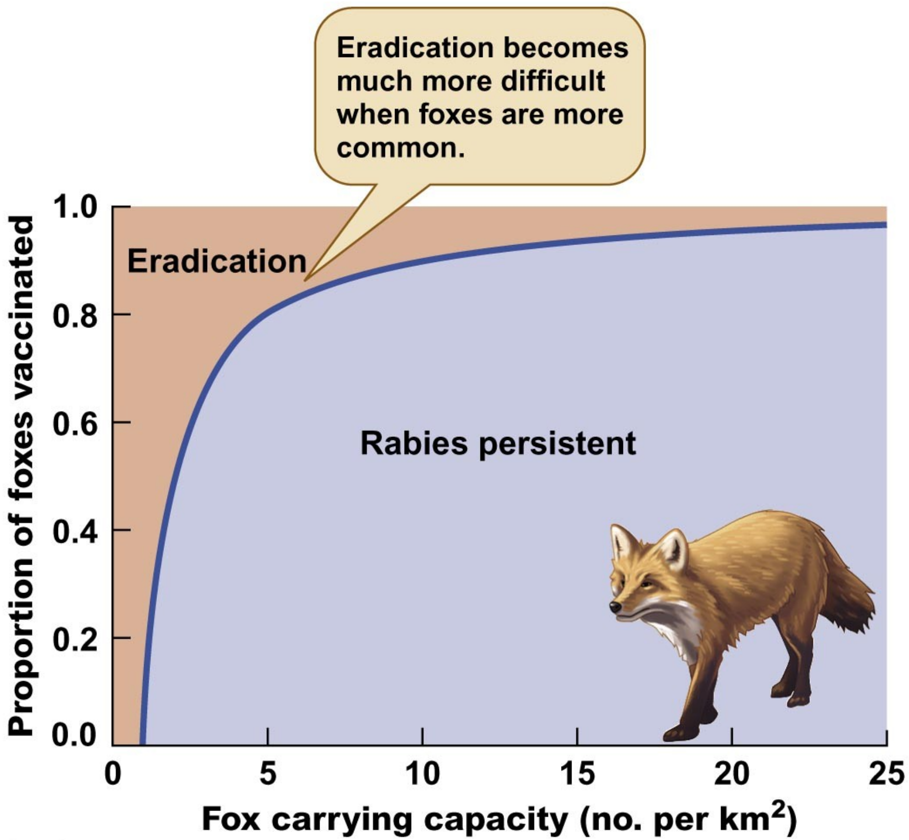




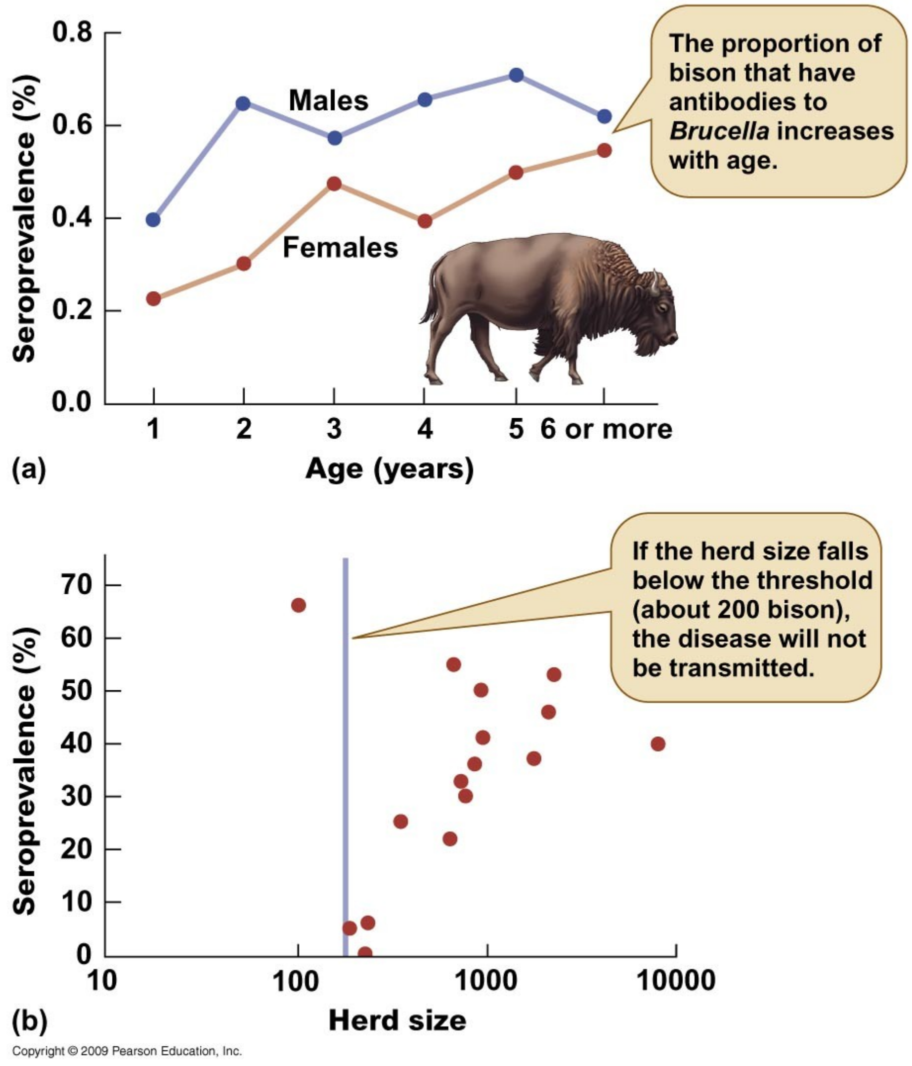
Procyon lotor

Ejemplos: Rabia en mamíferos silvestres

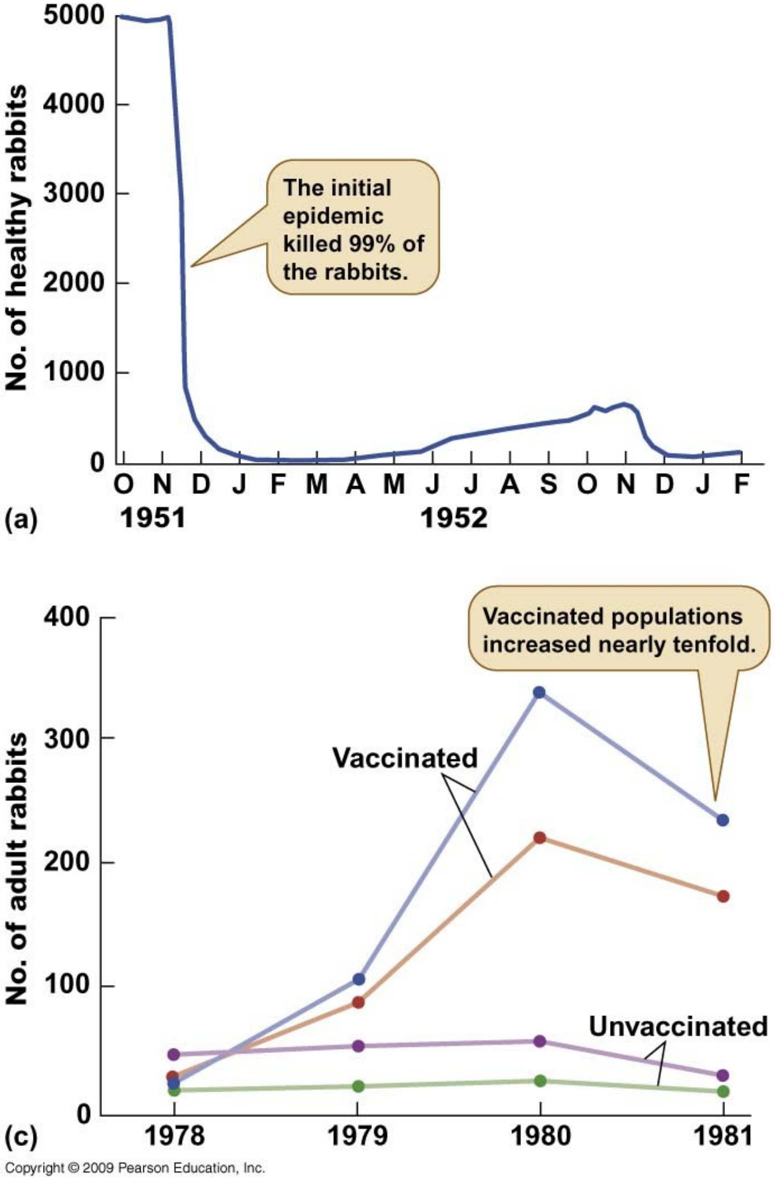




Ejemplos: Brucelosis en el bisón de Yellowstone

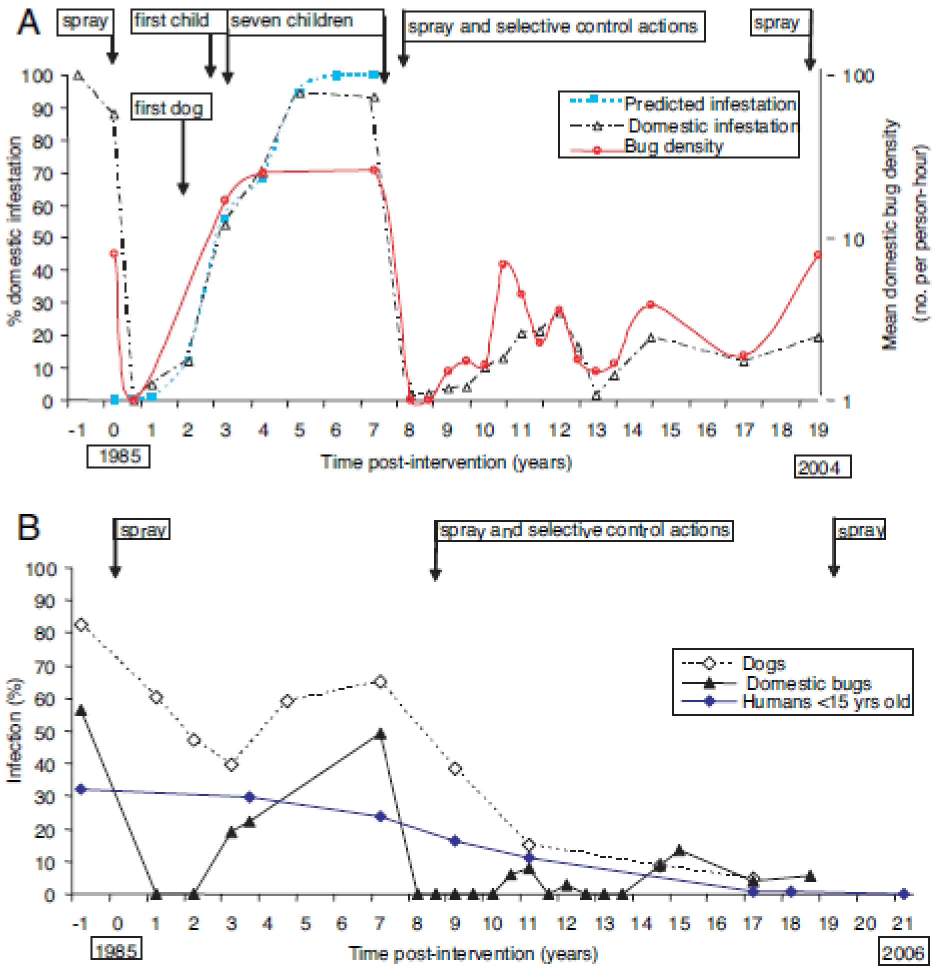


Ejemplos: Mixomatosis en conejos



Oryctolagus cuniculus

Ejemplos: Control del mal de Chagas



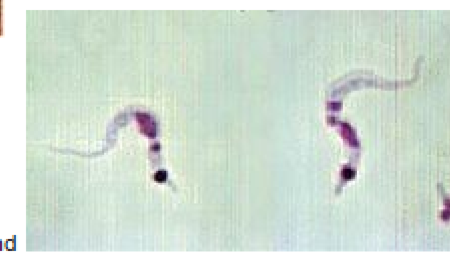


Fig. 1. Prevalence of domestic infestation (observed and predicted by a logistic model), mean domestic density of *T. infestans*, and the timing of appearance of new cases of *T. cruzi* infection after two community-wide campaigns including residual insecticide spraying, Amamá and neighboring villages, 1984–2006. (*A*) Domestic infestation and bug density and timing of appearance of new cases and insecticide sprays. (*B*) Infection with *T. cruzi* in domestic *T. infestans*, dogs, and children <15 years of age.

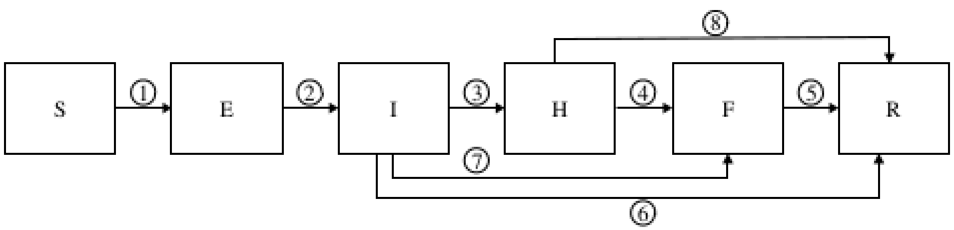
Fuente: Gürtler et al. (2007) PNAS 104: 16194-16199

Ejemplos: Transmisión del ébola

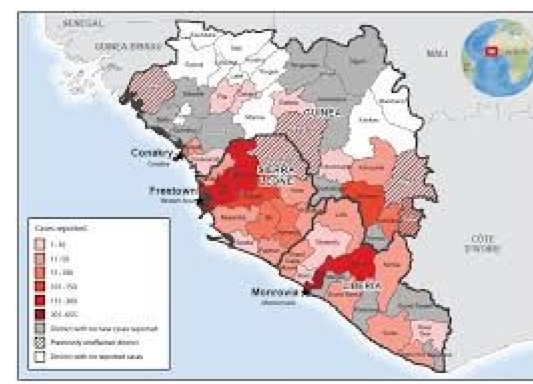
fever epidemics taking into account transmission in different epidemiological settings. We estimated the basic reproduction number (R_0) to be 2·7 (95% CI 1·9–2·8) for the 1995 epidemic in DRC, and 2·7 (95% CI 2·5–4·1) for the 2000 epidemic in Uganda. For each epidemic, we

Table 2. *The stochastic compartmental model*

Transition		Transition rate (λ_i)
1	(S, E)→(S − 1, E + 1)	$(\beta_I SI + \beta_H SH + \beta_F SF)/N$
2	(E, I)→(E − 1, I + 1)	αE
3	(I, H)→(I − 1, H + 1)	$\gamma_h \theta_1 I$
4	(H, F)→(H − 1, F + 1)	$\gamma_{dh} \delta_2 H$
5	(F, R)→(F − 1, R + 1)	$\gamma_f F$
6	(I, R)→(I − 1, R + 1)	$\gamma_i (1 - \theta_1)(1 - \delta_1) I$
7	(I, F)→(I − 1, F + 1)	$\delta_1 (1 - \theta_1) \gamma_d I$
8	(H, R)→(H − 1, R + 1)	$\gamma_{ih} (1 - \delta_2) H$



S, Number of susceptible individuals; E, number of exposed individuals; I, number of infectious cases in the community; H, number of hospitalized cases; F, number of cases who are dead but not yet buried; R, number of individuals removed from the chain of transmission; β_I, transmission coefficient in the community; β_H, transmission coefficient at the hospital; β_F, transmission coefficient during funerals. θ_1 is computed in order that θ% of infectious cases are hospitalized. δ_1, δ_2 are computed in order that the overall case-fatality ratio is δ. The inverse of the mean duration of the incubation period is α. The mean duration from symptom onset to hospitalization is γ_h^{-1}, γ_{dh}^{-1} is the mean duration from hospitalization to death, and γ_i^{-1} denotes the mean duration of the infectious period for survivors. The mean duration from hospitalization to end of infectiousness for survivors is γ_{ih}^{-1} and γ_f^{-1} is the mean duration from death to burial. Values presented in days in Tables 3 and 5 were converted to weeks for computation. Transmission coefficients are expressed in weeks^{-1}.



Fuente: Legrand et al. (2007) Epidemiol. Infect. 135: 610-621

Ejemplos: Efectos poblacionales del control de la fiebre

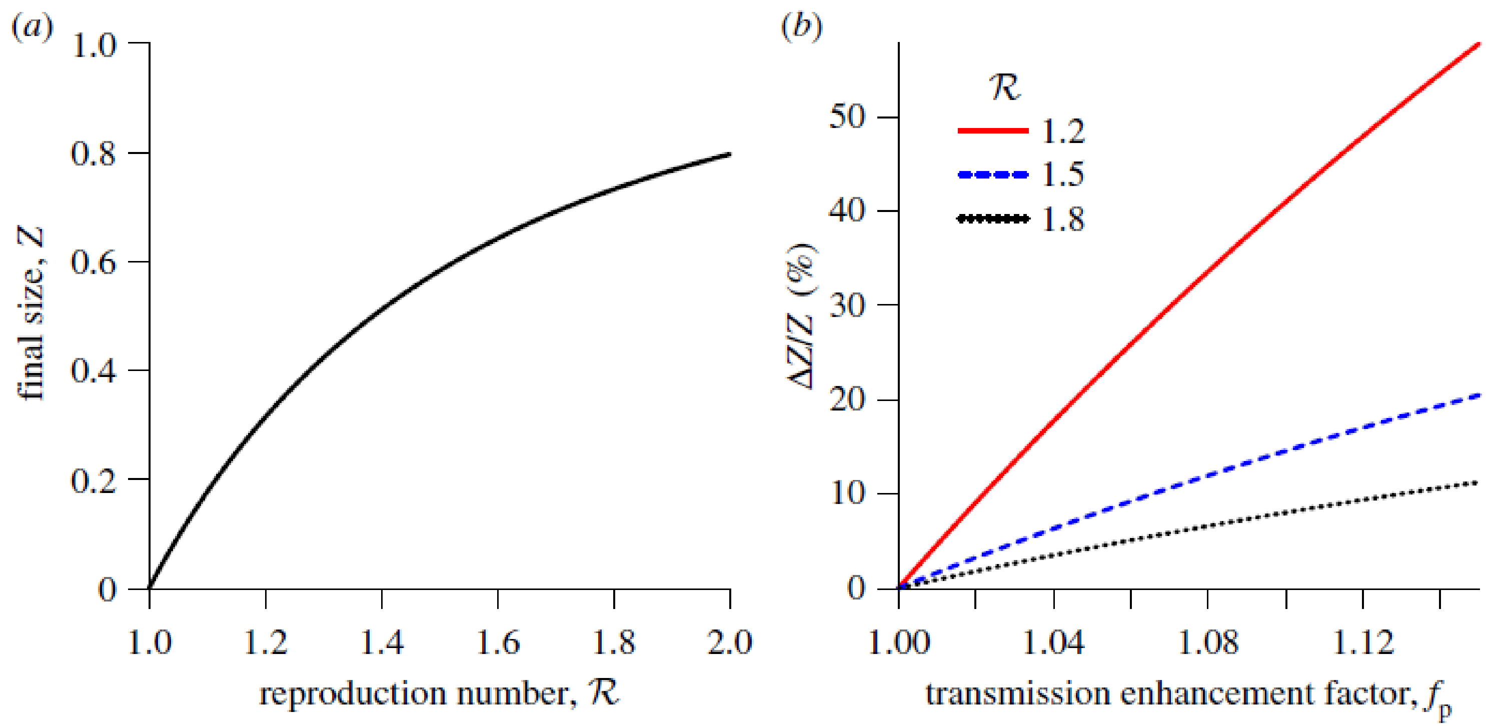
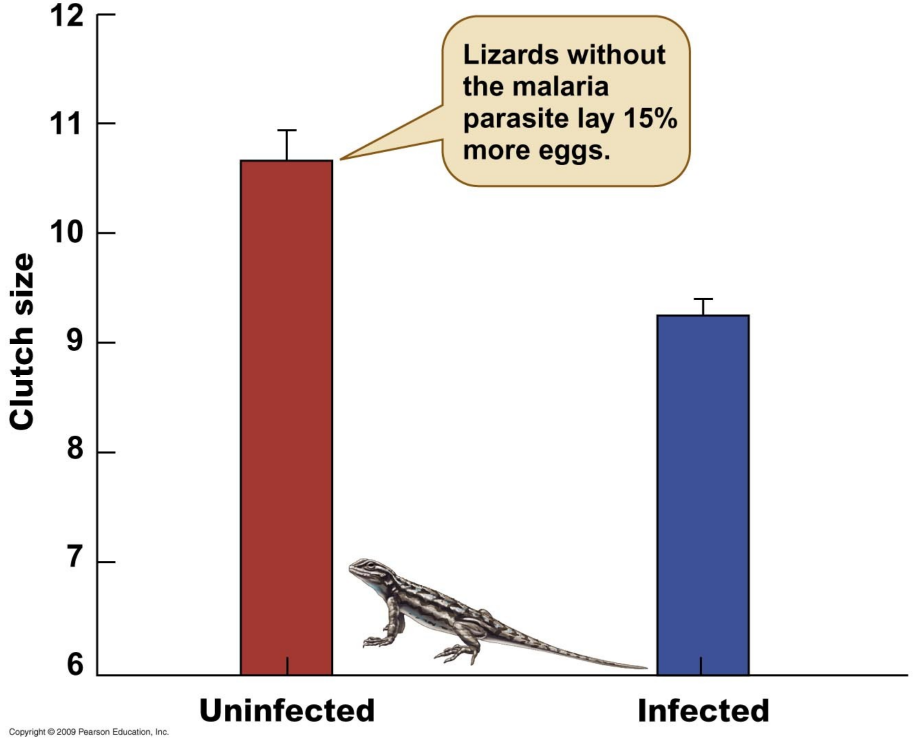
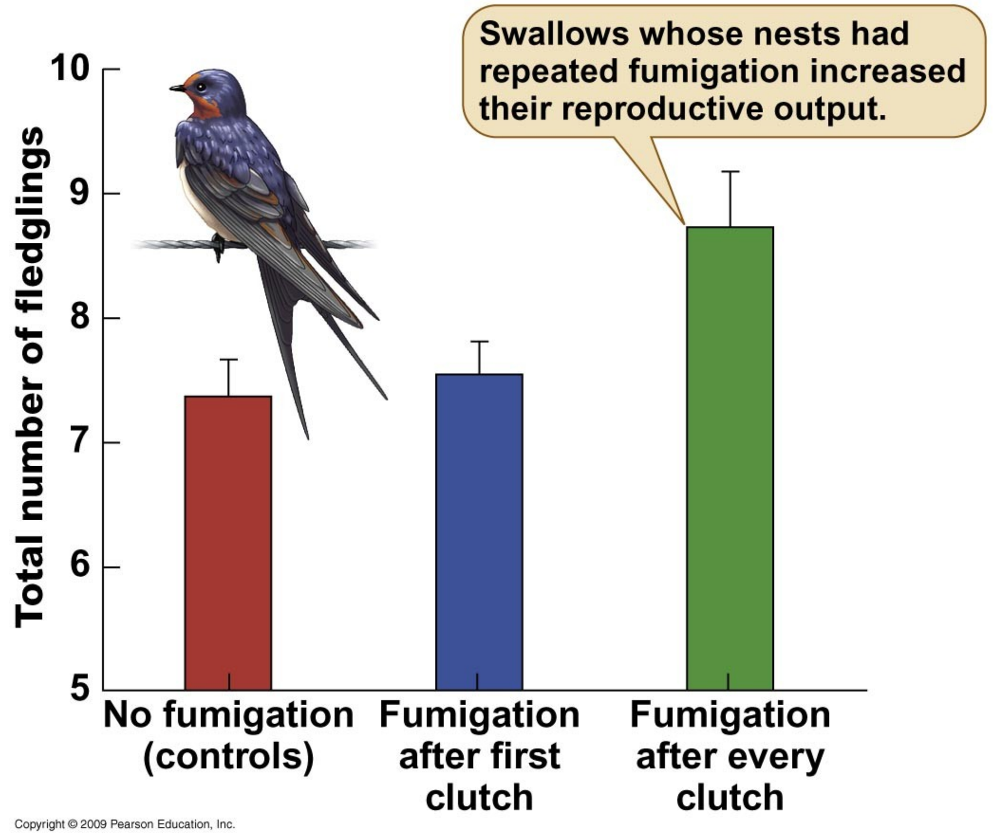


Figure 1. The effects of increases in transmission rate (by the factor f_p) on the expected proportion of the initially susceptible population that will be infected in a single influenza epidemic (the final size Z). (*a*) The standard final size relation (2.3), for the plausible range of (effective) reproduction number for influenza. (*b*) The relative increase in final size resulting from increasing the transmission rate by the factor f_p. For example, a 10% increase in the proportion of individuals infected during an epidemic will arise from a 2% transmission enhancement if $\mathcal{R} = 1.2$, a 6% enhancement if $\mathcal{R} = 1.5$ or a 12% enhancement if $\mathcal{R} = 1.8$. (Online version in colour.)

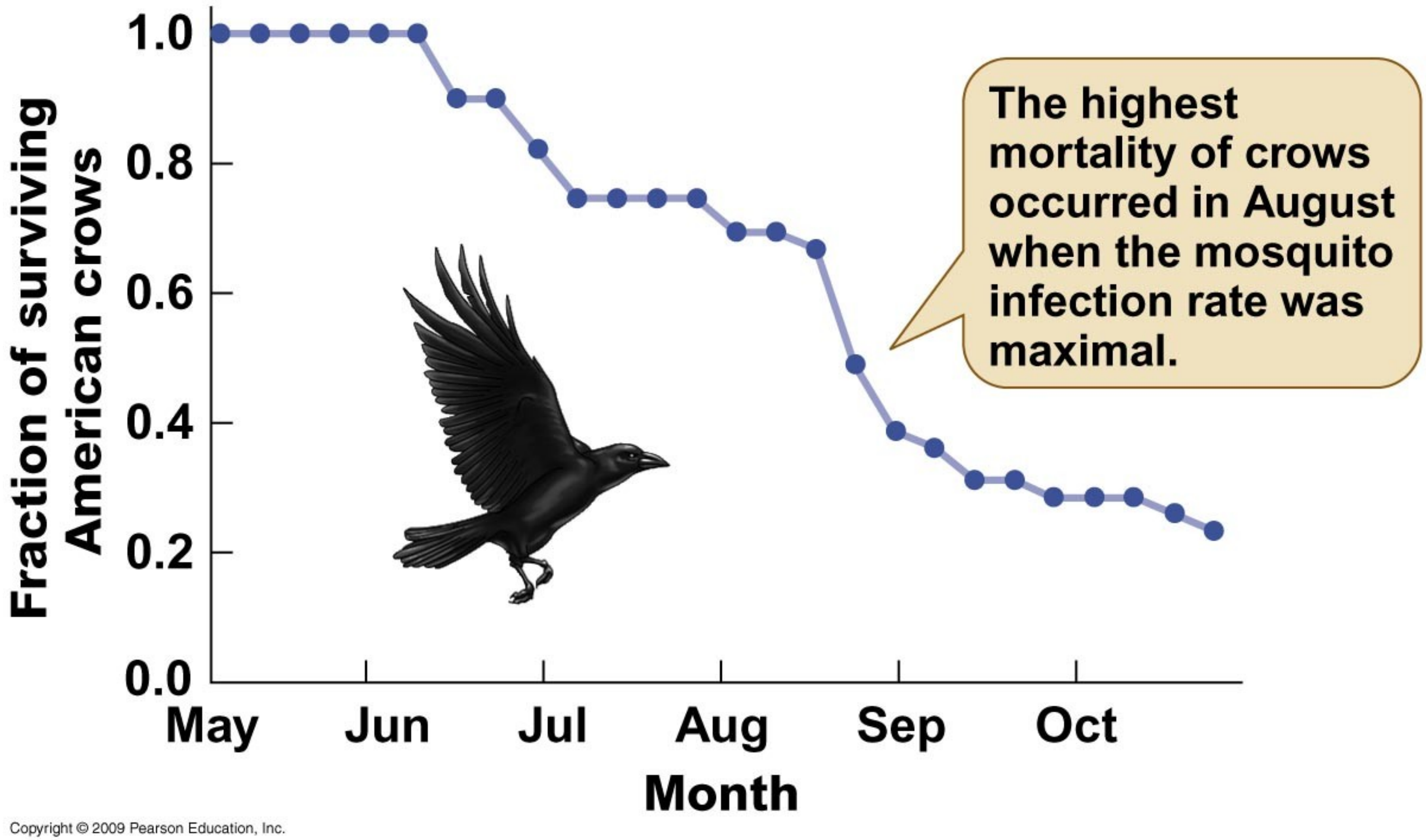
Fuente: Earn et al. (2014) Proc. R. Soc. B 281: 20132570

Efectos de los parásitos sobre la reproducción de sus hospedadores



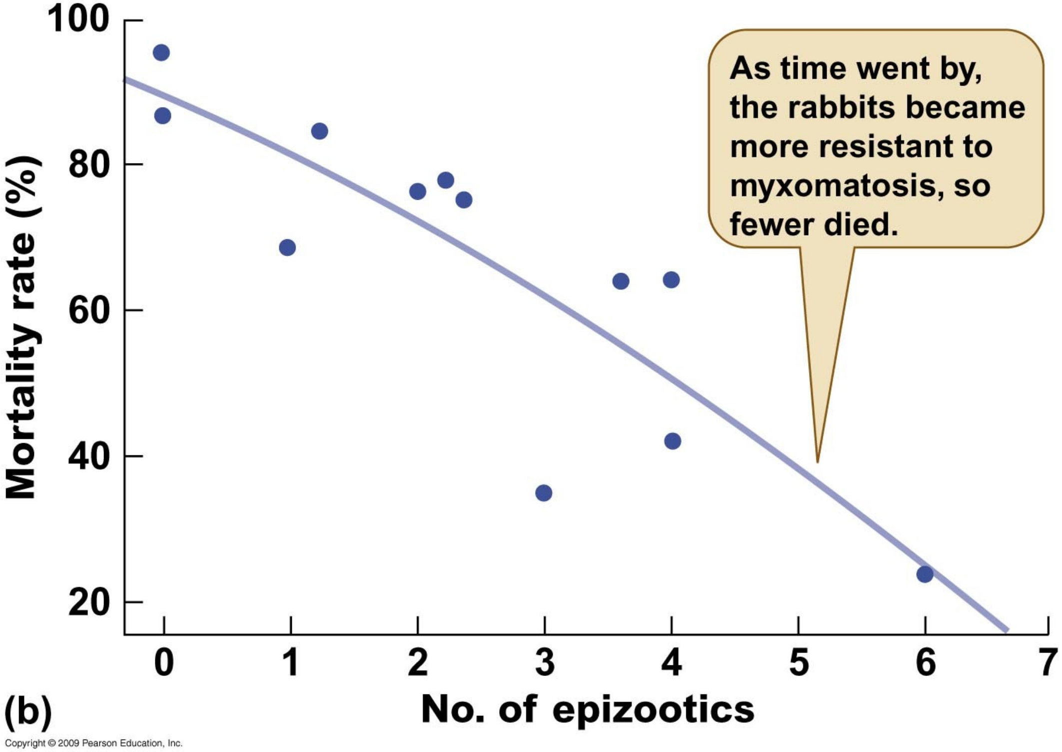


Efectos de los parásitos sobre la mortalidad de sus hospedadores

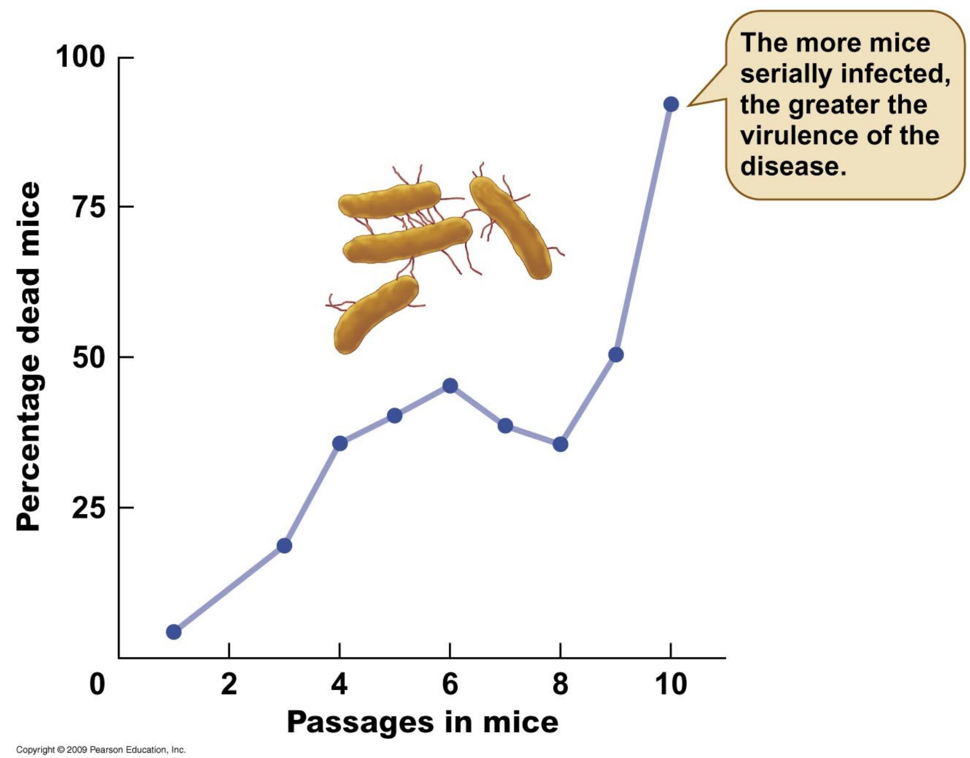


Evolución de la virulencia

Virus myxoma en conejo europeo



Salmonella typhimurium en ratones



Evolución de la virulencia: Hipótesis de la reina roja

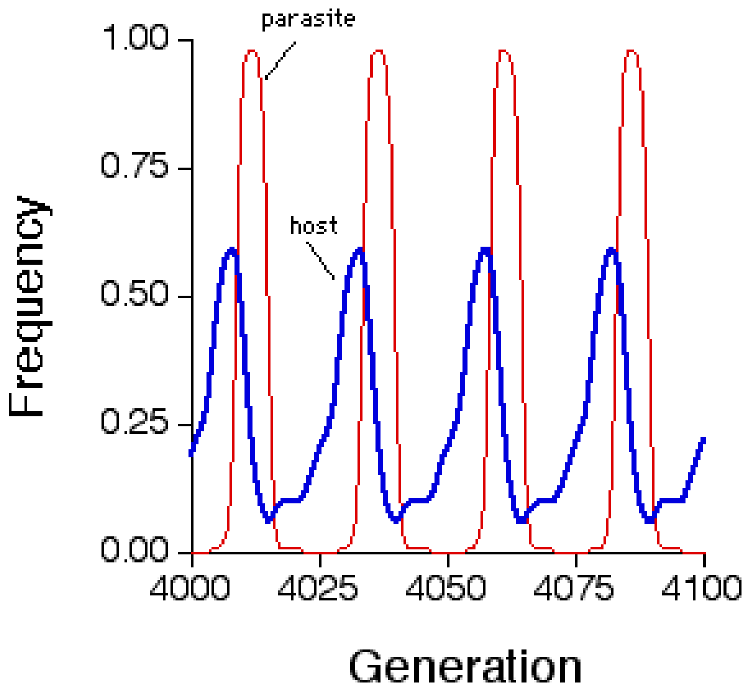


Figure 1. Red Queen dynamics: results from a computer simulation for host-parasite coevolution. The blue line gives the frequency of one host genotype; the red line gives the frequency of the parasite genotype that can infect it. Note that both genotypes oscillate over time, as if they were "running" in circles. The model assumes that hosts have self-nonself recognition systems, which can detect foreign organisms. The model also assumes that hosts and parasites both reproduce sexually.

Teórica 8: Recapitulación

- Pueden utilizarse modelos matemáticos simples para estudiar la dinámica de los sistemas hospedador-parásito
- Los parásitos pueden afectar a sus hospedadores tanto a nivel individual (fecundidad y mortalidad) como poblacional
- Los sistemas h-p pueden coevolucionar para volverse más benignos, o mantenerse altamente perjudiciales mediante una escalada armamentista