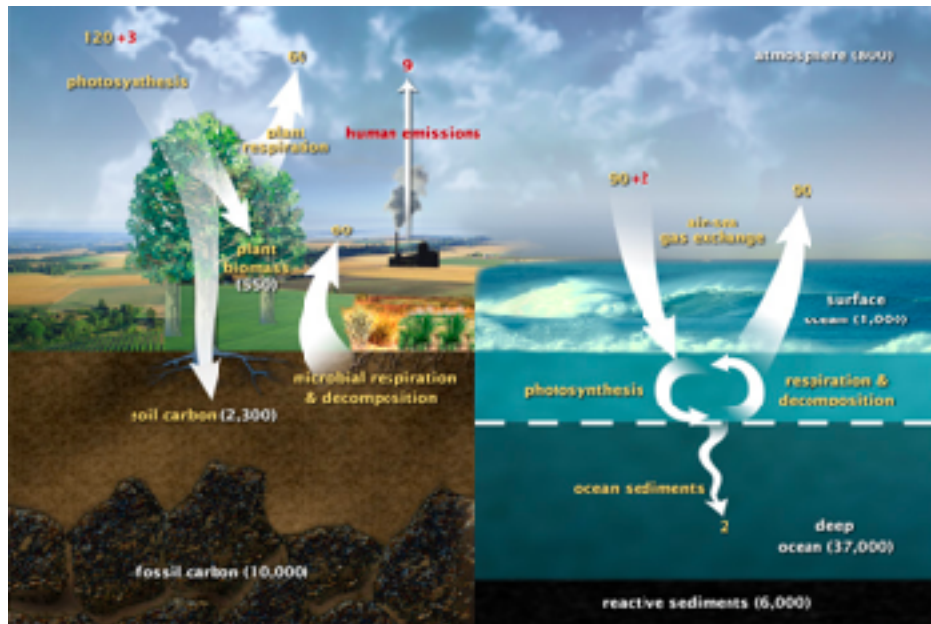
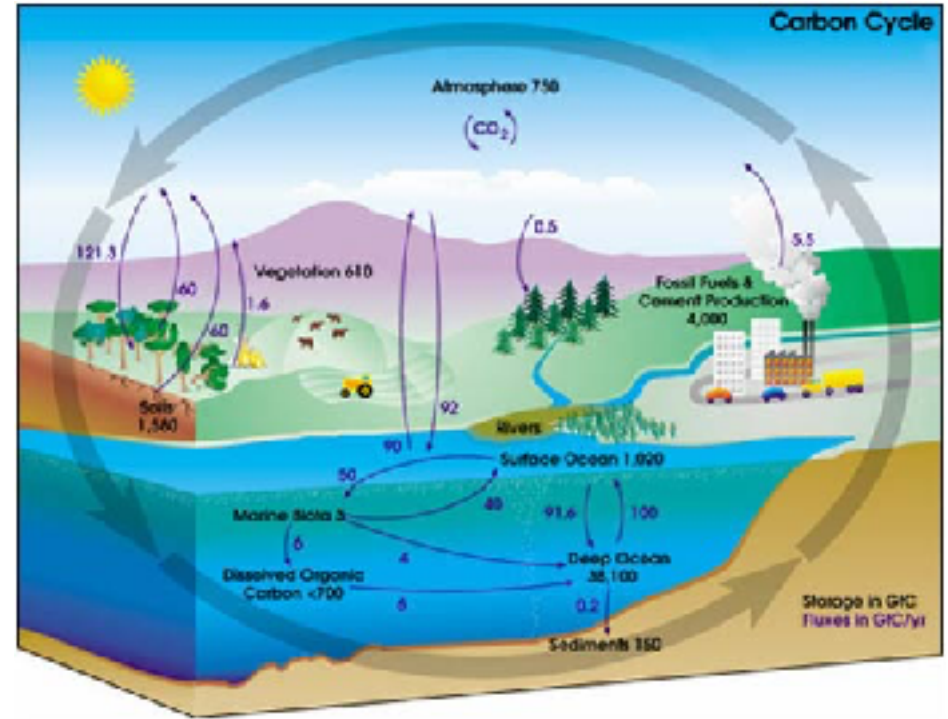


The Carbon cycle



earthobservatory.nasa.gov/features/CarbonCycle



skepticalscience.com/co2-residence-time.htm

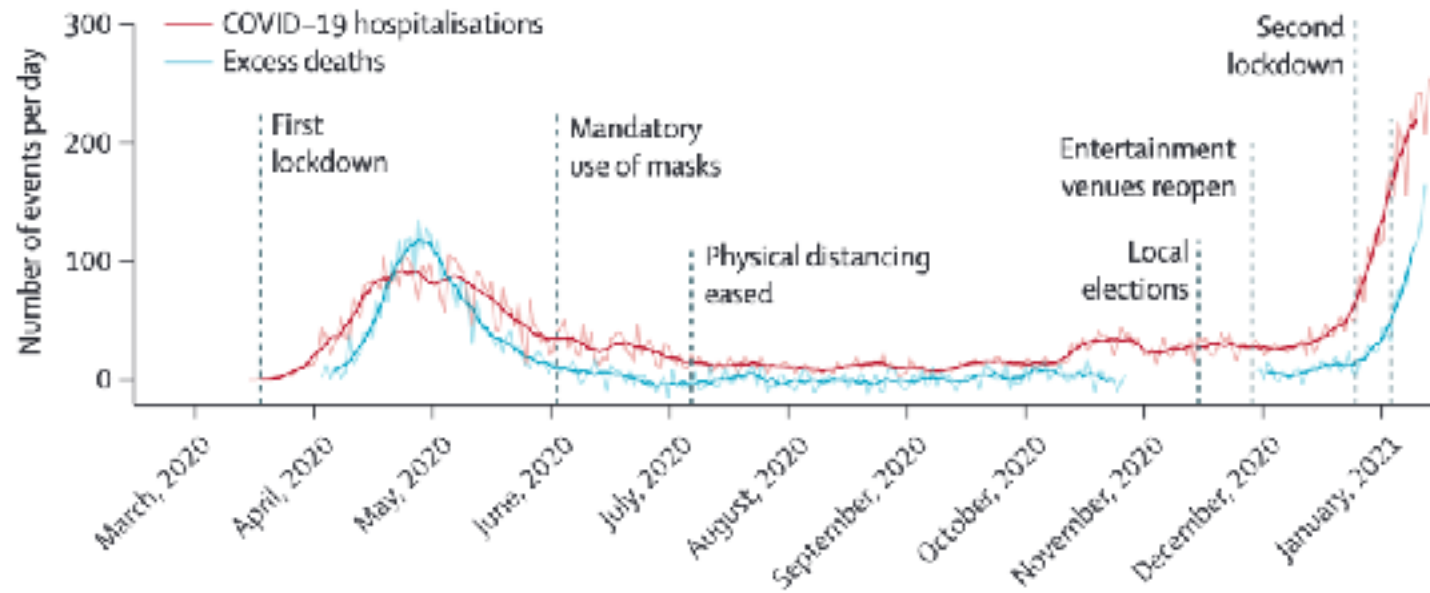
The human CO₂ production (9 giga ton/y) is small compared to the natural emissions (60+60+90), and the natural buffers. Why then do we observe such a large increase in atmospheric CO₂ levels?

$$\frac{dA}{dt} = f_{OA}O - f_{AO}A + f_{VA}V - f_{AV}A + h$$

Resurgence of COVID-19 in Manaus, Brazil, despite high seroprevalence

www.thelancet.com Vol 397 February 6, 2021

*Ester C Sabino, Lewis F Buss, Maria P S Carvalho, Carlos A Prete Jr, Myuki A E Crispim, Nelson A Fraiji, Rafael H M Pereira, Kris V Parag, Pedro da Silva Peixoto, Moritz U G Kraemer, Marcia K Oikawa, Tassila Saloman, Zulma M Cucunuba, Márcia C Castro, Andreza Aruska de Souza Santos, Vitor H Nascimento, Henrique S Pereira, Neil M Ferguson, Oliver G Pybus, Adam Kucharski, Michael P Busch, Christopher Dye, Nuno R Faria



Lecture Jan Paul Question 6.4

$$\frac{dS}{dt} = wR - \beta S \frac{I}{N}, \quad \frac{dI}{dt} = \beta S \frac{I}{N} - (d + r)I \quad \text{and} \quad \frac{dR}{dt} = rI - wR,$$

You fitted the first wave to a SIR model.
 Data of the first and second wave are publicly available
 Can both waves also be explained by this model?
 If not, what can we learn from this about long-term immunity?

Co-existence by trade-offs? Q9.9

PRL 118, 028103 (2017)

PHYSICAL REVIEW LETTERS

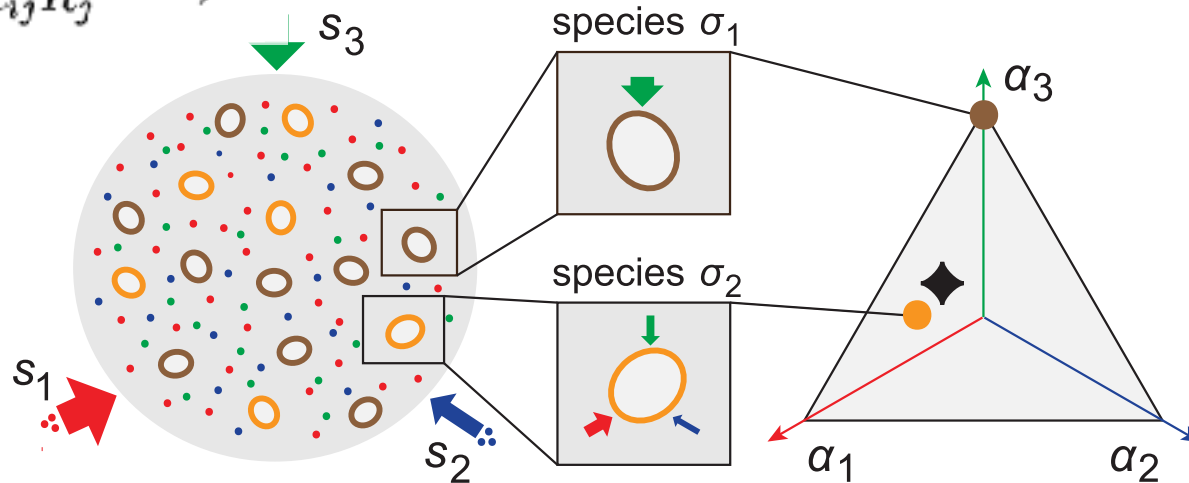
week ending
13 JANUARY 2017



Metabolic Trade-Offs Promote Diversity in a Model Ecosystem

Anna Posfai,¹ Thibaud Taillefumier,² and Ned S. Wingreen^{1,3}

$$\frac{dN_i}{dt} = \left(\sum_j \frac{\beta_{ij} c_{ij} R_j}{h_{ij} + c_{ij} R_j} - \delta_i \right) N_i$$

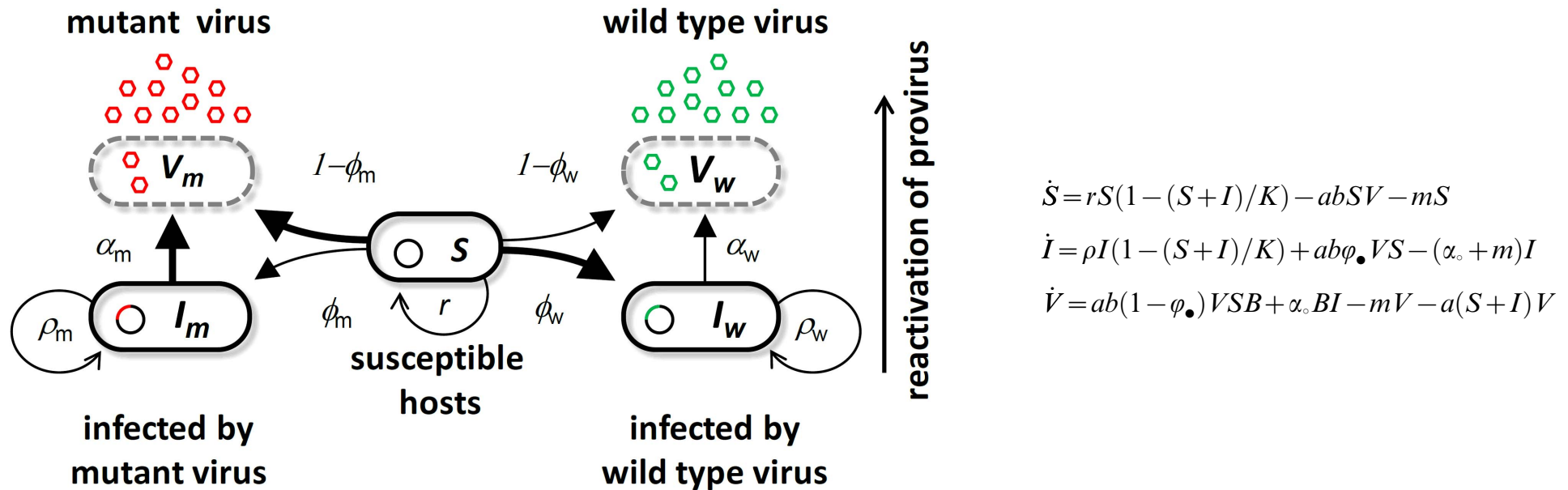


Motivated by recent studies of phytoplankton, we introduce trade-offs into a resource-competition model and find that an unlimited number of species can coexist. Our model spontaneously reproduces several notable features of natural ecosystems, including keystone species and population dynamics and abundances characteristic of neutral theory, despite an underlying non-neutral competition for resources.

Temperate phages

Evolution of Virulence in Emerging Epidemics

Thomas W. Berngruber^{1*}, Rémy Froissart^{2,3}, Marc Choisy^{3,4}, Sylvain Gandon¹



$$\dot{S} = rS(1 - (S+I)/K) - abSV - mS$$

$$\dot{I} = \rho I(1 - (S+I)/K) + ab\phi_{\bullet}VS - (\alpha_{\bullet} + m)I$$

$$\dot{V} = ab(1 - \phi_{\bullet})VSB + \alpha_{\bullet}BI - mV - a(S+I)V$$

Why are some pathogens more virulent than others? Theory predicts that pathogens that ‘keep their host alive’ can sometimes outcompete virulent pathogens in times when transmission to new susceptible hosts is unlikely. Yet, this prospect of finding a new susceptible host changes itself throughout an epidemic. In the early stage of an epidemic susceptible hosts are abundant and virulent pathogens that invest more into horizontal transmission should win the competition. Later on, the spread of the infection reduces the pool of susceptible hosts and may reverse the selection on virulence. This may favor benign pathogens after the acute phase of the epidemic. To put these predictions to the test we monitor the competition of the temperate bacterial virus λ and its virulent mutant λ cl857 in experimental epidemics. Our experimental results agree remarkably well with all our theoretical predictions.

Competitive exclusion and parasitism (Q10.6)

We studied the effect of a pathogen on winning species:

$$\frac{dS_j}{dt} = bN_j(1 - \sum N_i/k) - d_jS_j - \beta_jS_jI_j \quad \text{and} \quad \frac{dI_j}{dt} = \beta_jS_jI_j - (d_j + \delta_j)I_j ,$$

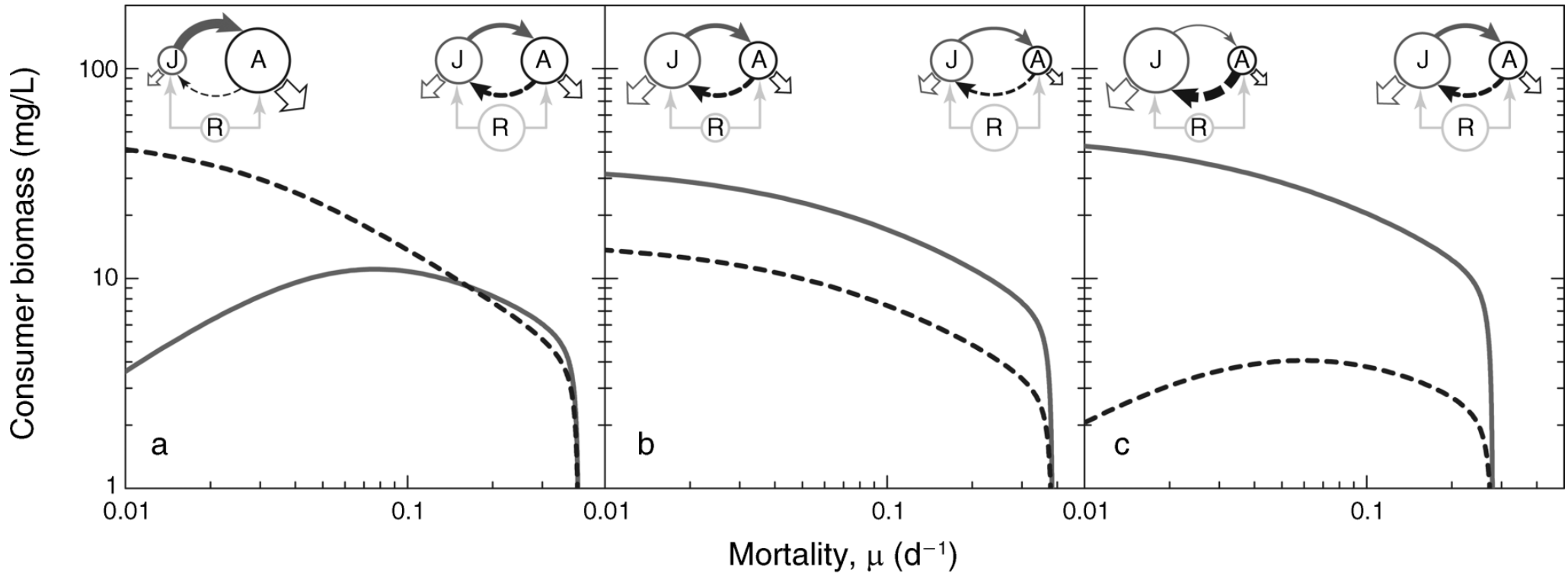
$$N_j = S_j + I_j$$

What is the effect of pathogens on co-existence?

How does this depend on their virulence?

Janzen-Connell hypothesis: parasites evolve towards most dominant species (negative density dependence)
[Bagchi *et al.*, Nature, 2014]

Symmetry breaking in ecological systems through different energy efficiencies of juveniles and adults

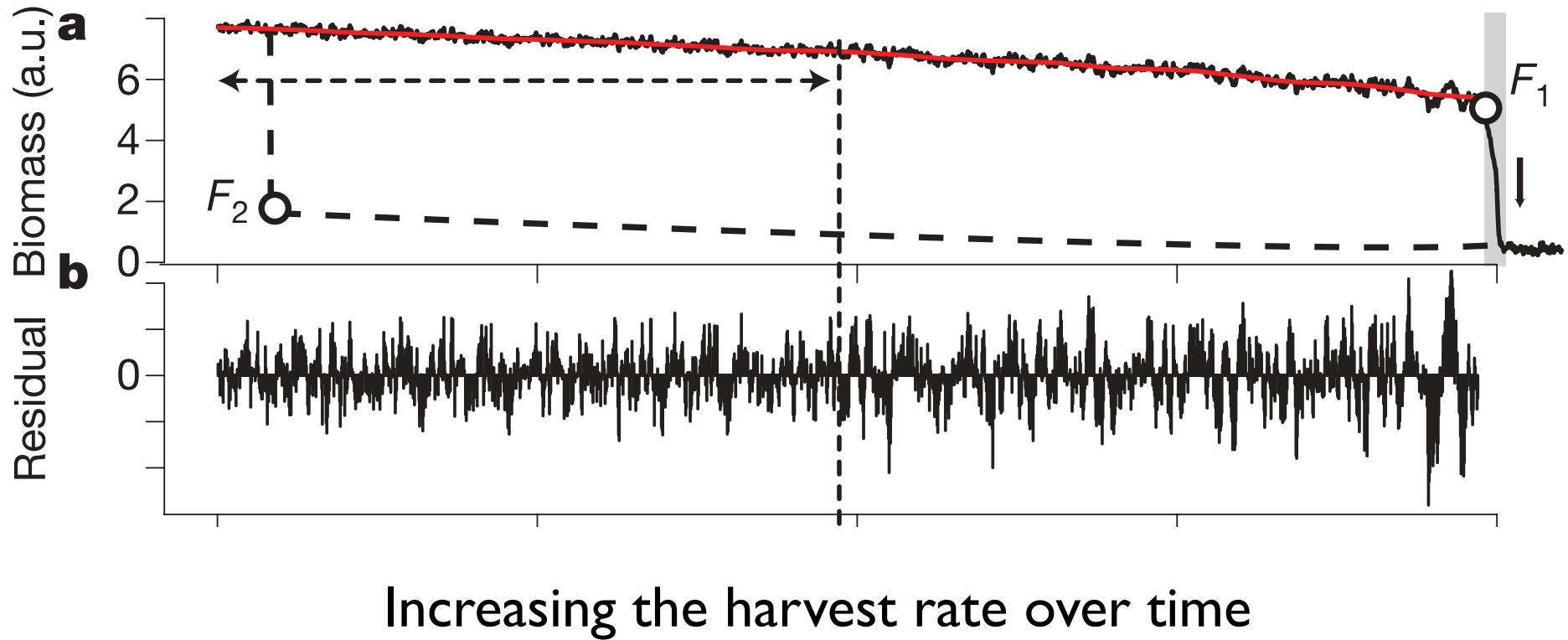


Persson & De Roos Ecology 2013; De Roos & Persson, Princeton UP, 2013

Ontogenetic development for dummies, try to repeat these results with:

$$R = K - c_1 J - c_2 A, \quad \frac{dJ}{dt} = \frac{eAR}{h_2 + R} - \frac{mJR}{h_1 + R} - \mu d_1 J \quad \text{and} \quad \frac{dA}{dt} = \frac{mJR}{h_1 + R} - \mu d_2 A$$

Early-warning signals for critical transitions (Q1 I.2)

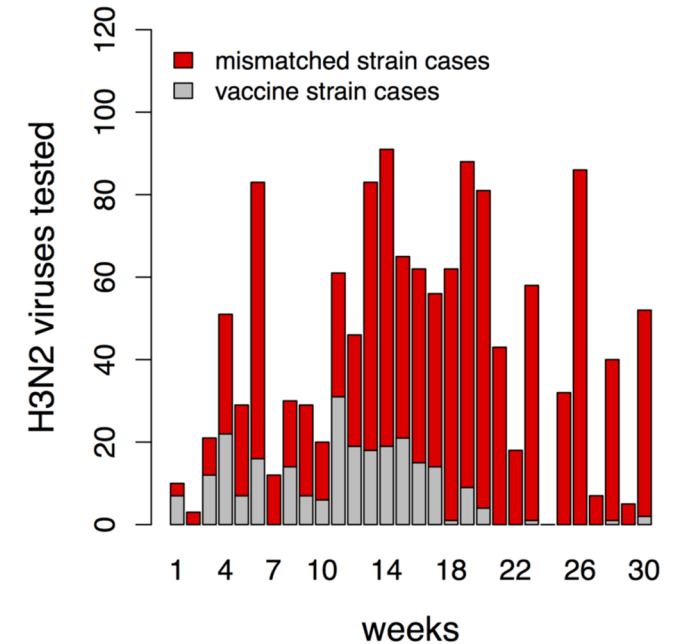
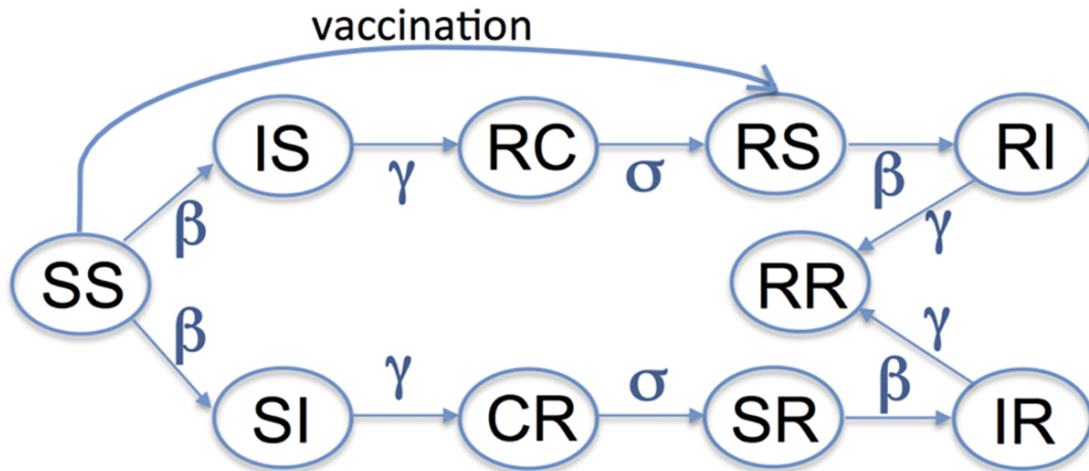


Test whether or not this works in a “real-world” example of models for grazing in the Sahel zone.

Influenza strain replacement

Intermediate levels of vaccination coverage may minimize seasonal influenza outbreaks

Veronika I. Zarnitsyna^{1*}, Irina Bulusheva², Andreas Handel³, Ira M. Longini⁴, M. Elizabeth Halloran^{5,6}, Rustom Antia^{7*}



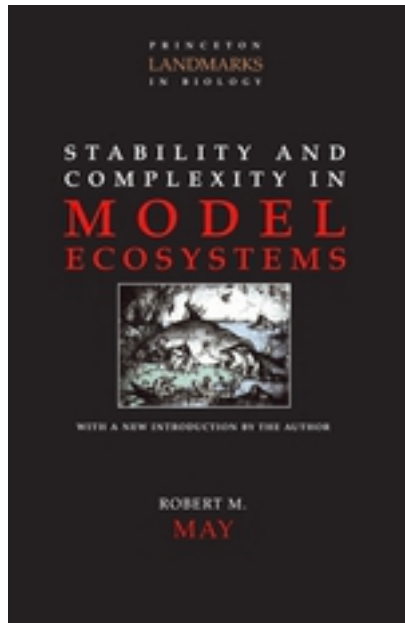
The total number of seasonal influenza infections is minimized at an intermediate (rather than maximal) level of vaccination.

Increasing the level of the vaccination coverage may be detrimental.

Recent bioRxiv paper: why is immunity to respiratory viruses short-lived?

<https://www.biorxiv.org/content/10.1101/2024.07.23.604867v2>

How can large systems be stable?



This book is a classic studying the relationship between the complexity of a system (number of species and their number of interactions), and the likelihood that the system would be stable.

$$\sigma\sqrt{nP} < 1$$

In chapter 10 we provide several exercises, each with R-scripts, to repeat and extend this classic work. Central questions are the structure of the interaction matrix, and the “scaling” by setting all diagonal elements to $A_{ii} = -1$.

Long term effects of vaccination

Fire

Elephants

Wildebeest

Rainfall



Getz, PLoS Biol 2009

The effect of elephants is through regular browsing and coppicing of trees, fire through episodic burns linked to fuel load, wildebeest after being released from the suppressing effects of endemic rinderpest (a morbillivirus of artiodactyls), and rain through its connections to all system components.

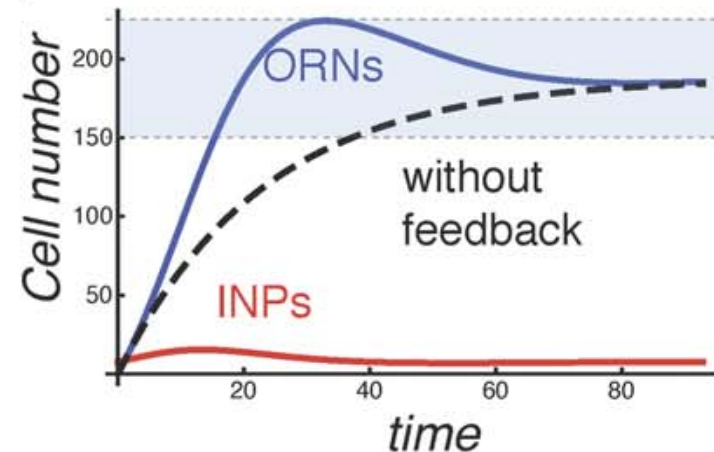
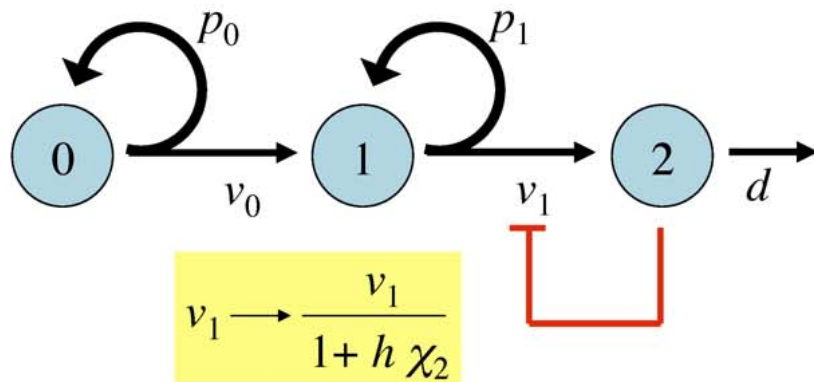
Holdo et al. [2009] demonstrate that eradication of rinderpest is responsible for the Serengeti switch from a net source to net accumulator of carbon.

Wolves and bears in Yellowstone

Stem cell renewal (Q12.5).

Cell Lineages and the Logic of Proliferative Control

Arthur D. Lander^{1,2,3}*, Kimberly K. Gokoffski^{1,4,5}*, Frederic Y. M. Wan^{3,5}, Qing Nie^{2,3,5}, Anne L. Calof^{1,3,4}*



Introduce a novel feedback mechanism where stem cells tune the fraction of asymmetric divisions that they make. Does this lead to faster recovery after tissue damage?