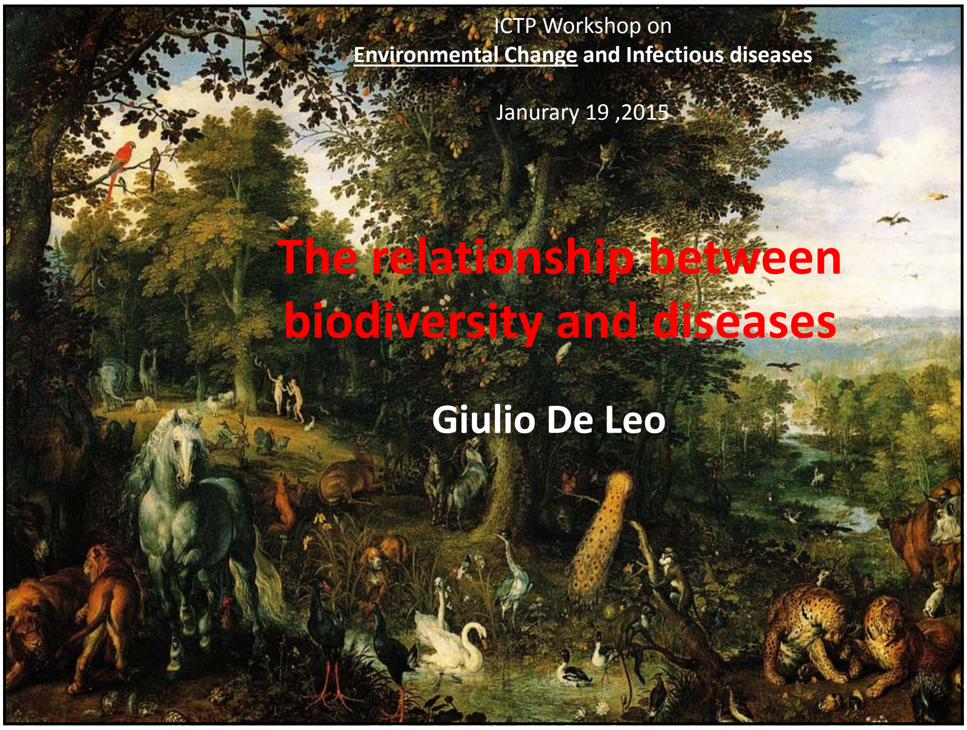


Presentation structure

- Why should we care?
- What is the problem?
- What models show:
  - A simple SI model with predatory release
  - A simple model of dilution effect
  - A model with dilution and vector amplification
  - Tick borne disease models with multiple hosts
  - Multi-parasite, multi-host model
- What data tell us: Bio-geography of infectious diseases and biodiversity

Why should we care?

Woolhouse and Gowtage-Sequeria 2005

Of 1,407 recognized species of human pathogen, 58% are zoonotic, i.e. an animal disease that can be transmitted to human.



PATHOGEN	*ORIGINAL HOST*	*YEAR REPORTED*
Ebola virus	**Bats**	**1977**
***Escherichia coli* O157:H7**	**Cattle**	**1982**
Borrelia burgdorferi	**Rodents**	**1982**
SIV/HIV-1	**Primates**	**1983**
SIV/HIV-2	**Primates**	**1986**
Hendra virus	**Bats**	**1994**
BSE/vCJD	**Cattle**	**1996**
Australian bat lyssavirus	**Bats**	**1996**
H5N1 influenza A	**Chickens**	**1997**
Nipah virus	**Bats**	**1999**
SARS coronavirus	**Palm civets**	**2003**



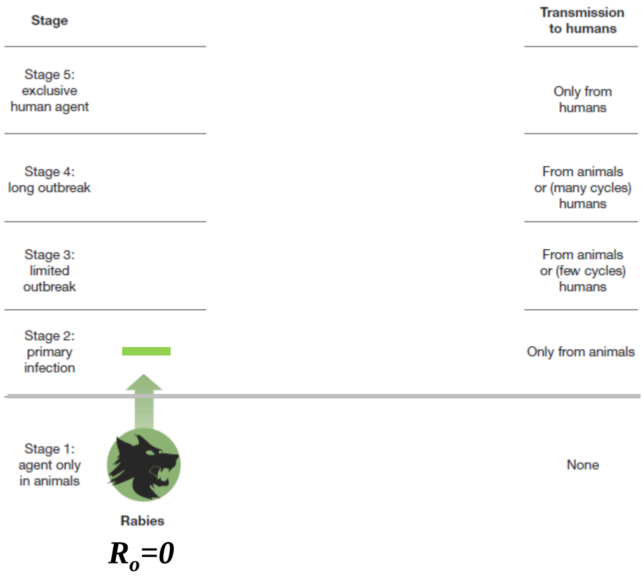
Why should we care?

- Zoonotic pathogens are twice as likely to be regarded as emerging or reemerging out of the total of 177 pathogens in this category
  (Woolhouse and Gowtage-Sequeria 2005)
- Nearly all of the 25 most important humans pathogens are either zoonotic or originated as zoonoses before adapting to human *(Wolf et al. 2007)*
- Emerging and reemerging zoonoses are associated with a wide range of drivers, but changes in land use and agriculture and demographic and societal changes are most commonly cited
  (Woolhouse and Gowtage-Sequeria 2005).

Origins of major human infectious diseases

Nathan D. Wolfe[1], Claire Panosian Dunavan[2] & Jared Diamond[3]

2007 nature



- 25 diseases of major concern

Origins of major human infectious diseases

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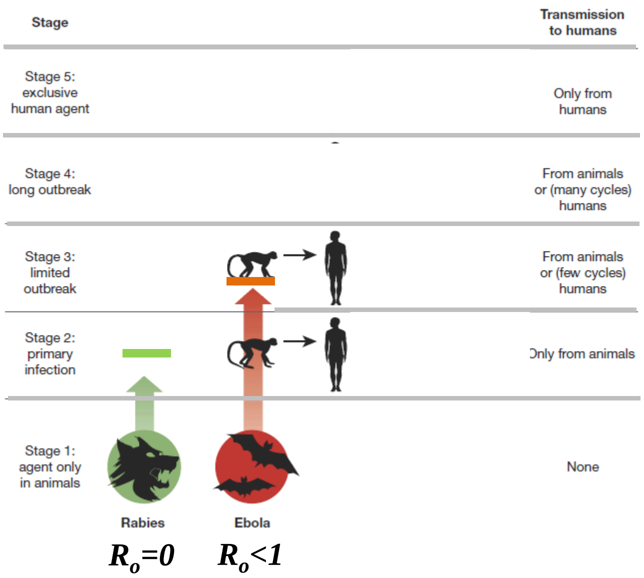
2007 nature



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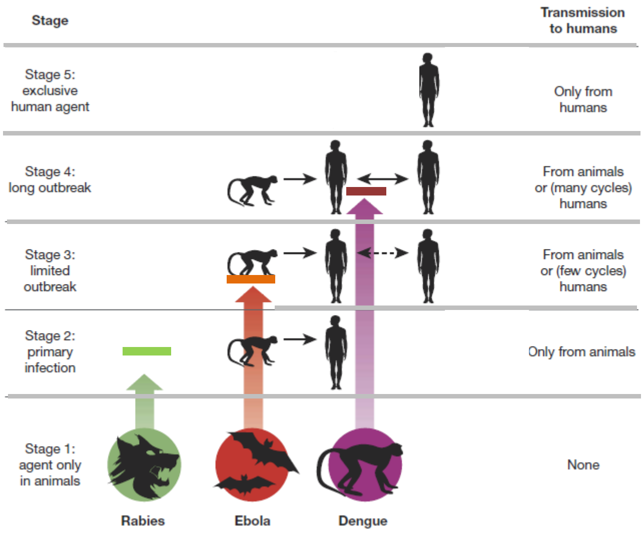

R_o=0 R_o<1 R_o>1

- 25 diseases of major concern

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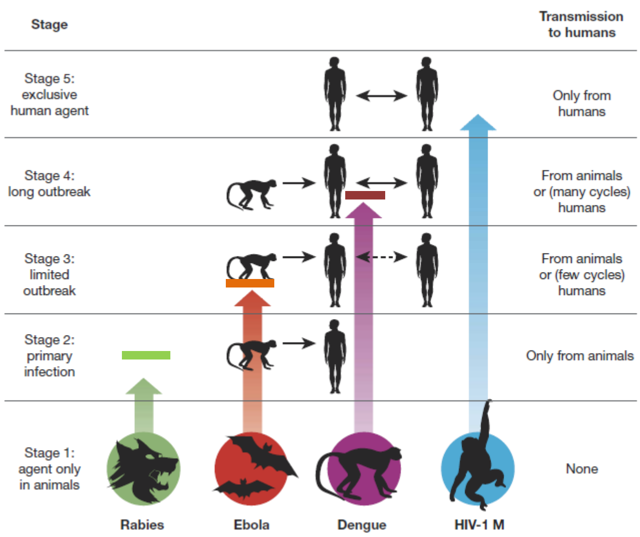
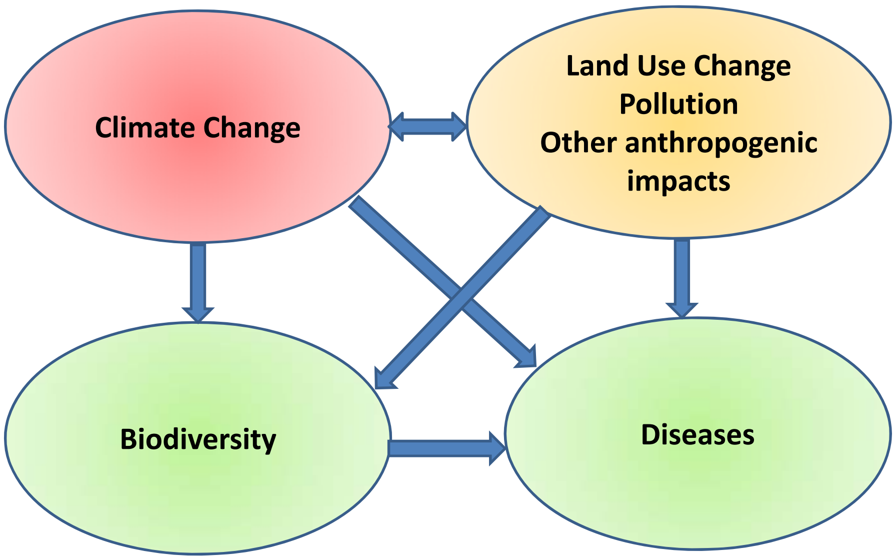


R_o=0 R_o<1 R_o>1

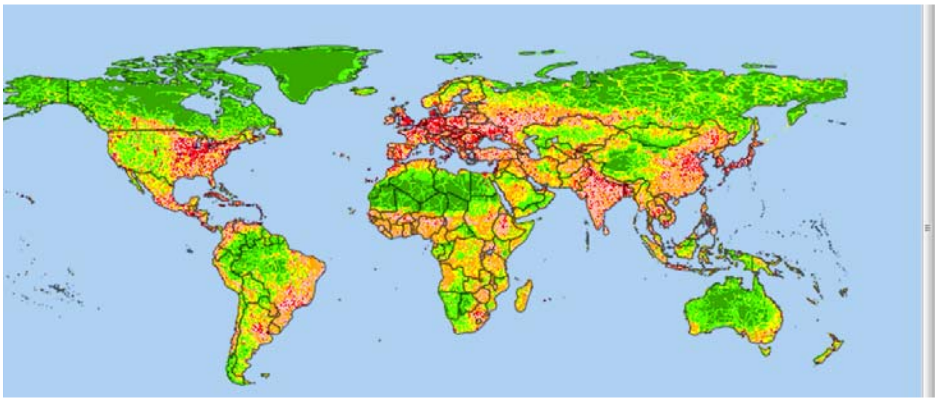
- 25 diseases of major concern

Why should we care?



Biodiversity hotspot

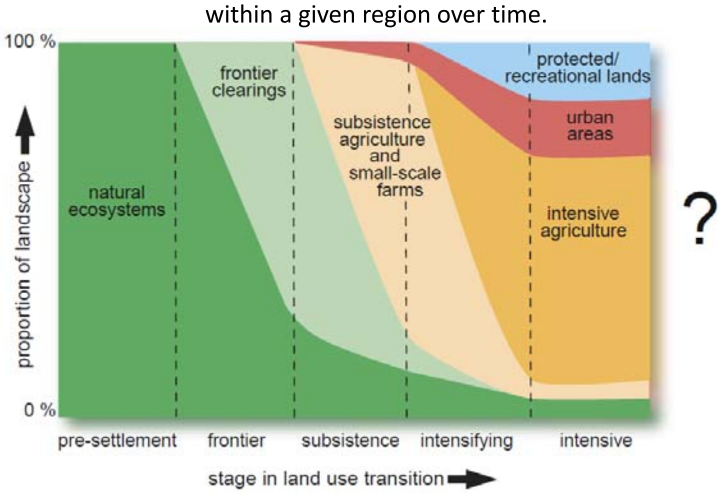
Global map of human footprint



Source: http://sedac.ciesin.columbia.edu/data/set/wildareas-v2-human-footprint-geographic/maps

Global Consequences of Land Use
Jonathan A. Foley *et al.*
Science **309**, 570 (2005);
DOI: 10.1126/science.1111772

Transitions in land-use activities that may be experienced within a given region over time.



Perception of the potential effects of anthropogenic global change on biodiversity loss

- <u>Negative burden</u> for "good bugs" - such as pollinators and natural pest controllers - that are doomed to extinction (Pimentel, Costanza, Millenium assessment…)

Recent evidence for the climate change threat to Lepidoptera and other insects

Author(s): Wilson, RJ (Wilson, Robert J.)[1]; Maclean, IMD (Maclean, Ilya M. D.)[1]

Source: JOURNAL OF INSECT CONSERVATION Volume: 15 Issue: 1-2 Special Issue: SI Pages: 259-268 DOI: 10.1007/s10841-010-9342-y Published: APR 2011

biology letters

rsbl.royalsocietypublishing.org

Research

Cite this article: Jevanandam N, Goh AGR, Corlett RT. 2013 Climate warming and the

Global change biology

Climate warming and the potential extinction of fig wasps, the obligate pollinators of figs

Nanthinee Jevanandam[1], Alexander G. R. Goh[1] and Richard T. Corlett[2]

[1]Department of Biological Sciences, National University of Singapore, 14 Science Drive 4, Singapore 117543, Republic of Singapore
[2]Xishuangbanna Tropical Botanical Garden, Center for Integrative Conservation, Chinese Academy of Sciences, Menglun, Mengla, Yunnan 666303, People's Republic of China

Is Pollination at Risk? Current Threats to and Conservation of Bees

Author(s): Grunewald, B (Gruenewald, Bernd)

Source: GAIA-ECOLOGICAL PERSPECTIVES FOR SCIENCE AND SOCIETY Volume: 19 Issue: 1 Pages: 61-67 Published: 2010

Perception of the potential effects of anthropogenic global change on biodiversity loss

- Yet, <u>positive effects</u> for bad bugs – typically mosquitoes and ticks acting as agents of vector-borne diseases - that will benefit from anthropogenic changes and will increase their geographical range and abundance ➔ <u>the dilution effect</u>

Ecology, 82(3), 2001, pp. 609–619
© 2001 by the Ecological Society of America

BIODIVERSITY AND THE DILUTION EFFECT IN DISEASE ECOLOGY

KENNETH A. SCHMIDT[1] AND RICHARD S. OSTFELD

Institute of Ecosystem Studies, Box AB, Millbrook, New York 12545 USA

Hosts as ecological traps for the vector of Lyme disease

F. Keesing, J. Brunner, S. Duerr, M. Killilea, K. LoGiudice, K. Schmidt, H. Vuong and R. S. Ostfeld

Proc. R. Soc. B published online 19 August 2009
doi: 10.1098/rspb.2009.1159

Ecology Letters, (2006) 9: 485–498 doi: 10.1111/j.1461-0248.2006.00885.x

REVIEWS AND SYNTHESIS

Effects of species diversity on disease risk

F. Keesing,[1,3]* R. D. Holt[2] and R. S. Ostfeld[3]

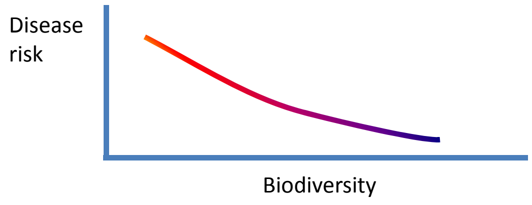
Abstract

The transmission of infectious diseases is an inherently ecological process involving interactions among at least two, and often many, species. Not surprisingly, then, the

REVIEW

Impacts of biodiversity on the emergence and transmission of infectious diseases

- This has lead to the hypothesis of a negative relationship between biodiversity and disease risk



- Possibly true for a number of pathogens, as Lyme disease and malaria (zooprophylaxis)

Two arguments

- Susceptible host regulation
- Transmission interference

Dirzo et al. 2014
Science,
Defaunation
in the Anthropocene

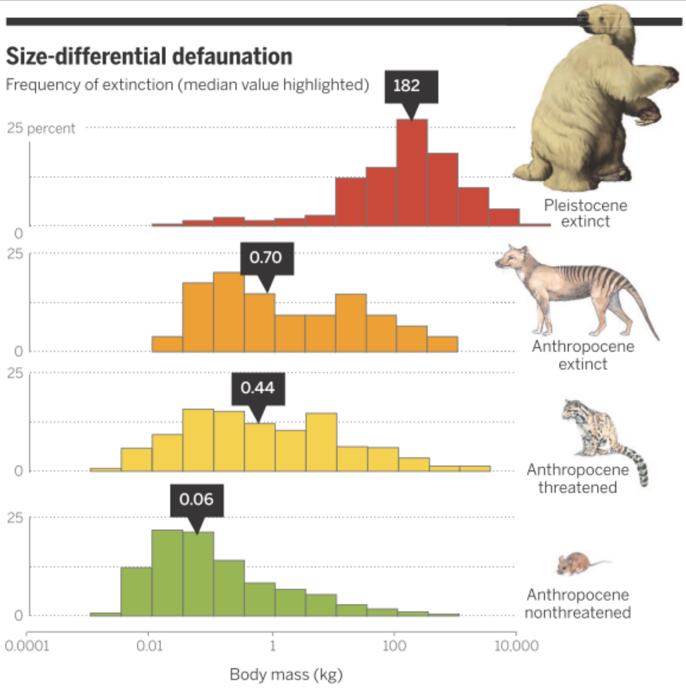
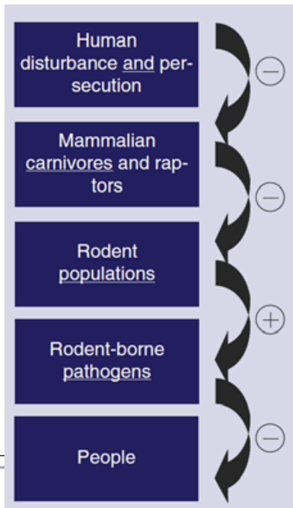
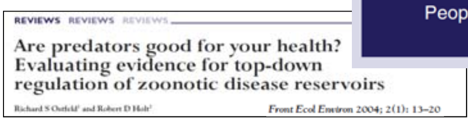


Fig. 3. Extinction and endangerment vary with body size. Comparing data on body size of all animals

A density-dependent cascading effect

- A rodent population is the natural reservoir of a zoonotic pathogen (such as Lyme disease, plague, hantavirus pulmonary syndrome,…)
- Their predators keep the rodent population below the density for disease invasion
- The removal of the top predators relaxes the rodent population that increases above the threshold for disease invasion
- An outbreak occurs with possible transmission of the disease to the human host



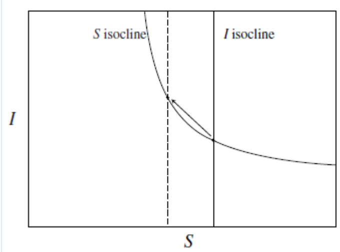

REVIEWS REVIEWS REVIEWS

Are predators good for your health? Evaluating evidence for top-down regulation of zoonotic disease reservoirs

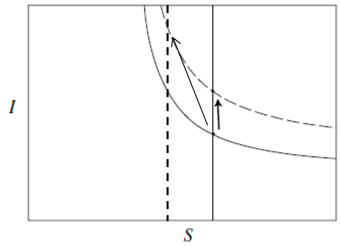
Richard S Ostfeld and Robert D Holt — Front Ecol Environ 2004; 2(1): 13–20

$$\frac{dS}{dt} = (b - m(C))S - \beta SI + (\gamma + b^1)I,$$
$$\frac{dI}{dt} = \beta SI - (\gamma + m^1(C))I$$

Predators increase mortality $m^1(C)$ of only infected individuals

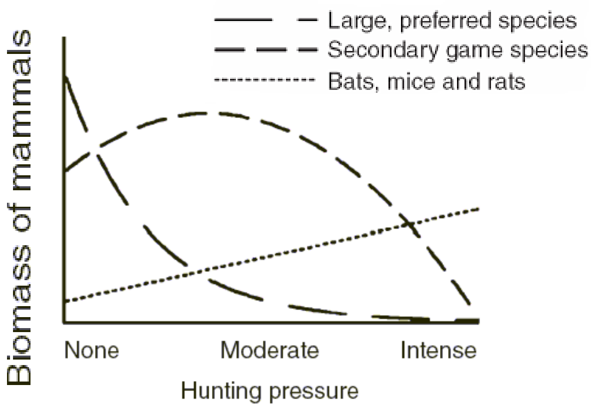


Predators increase mortality of both susceptible and infected individuals

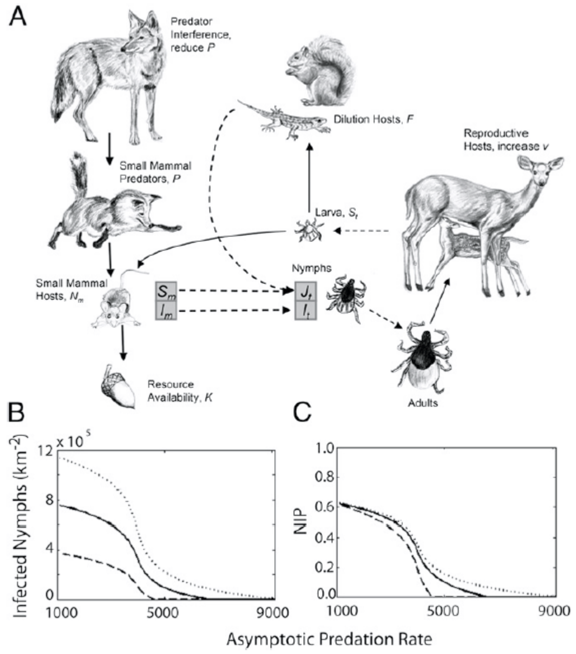


Ostfeld & Hold 2004, FEE

FA and BROWN. 2009.
Impacts of hunting on mammals in African tropical moist forests: a review and synthesis. ***Mammal Review 39: 231–264*.**



Levi et al. PNAS 2012



Transmission interference and the dilution effect

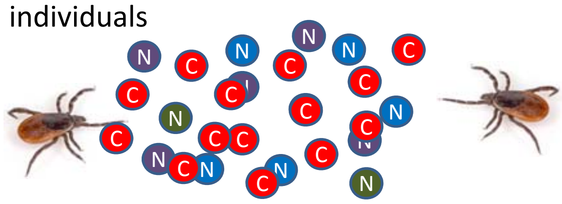
Ostfel and Keeing (2000)

- Assumptions
  1. Generalist vector
  2. Horizontal (frequency-dependent) transmission
  3. Differences in competence among host species:
     - Competent primary host
     - Non-competent secondary host
  4. the most competent host is not affected by, or even benefits, from anthropogenic disturbance leading to biodiversity loss. The least competent hosts are the first to go

Ostfel and Keeing 's syllogism

- As the female vector (e.g. a tick) requires a limited number of blood meals to complete its life cycle…

  in a highly biodiverse ecosystem, a fraction of potentially infective bites will be wasted over the non-competent host, thus "diluting" the pathogen in the primary competent host.
- By removing the non-competent hosts, the vector can target only the competent one.
- Because of competitive/predatory release, the density of competent host could also increase and so the that of the infected individuals



On dilution effect

- # infected vectors/host (competent + non-competent)

infected vectors

$$\frac{Y_i}{X_{tot} + A}$$

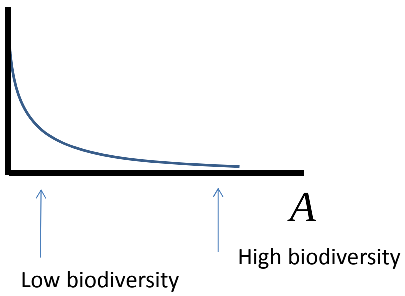
competent host

Alternative, non-competent host

Underline assumption

- The second non-competent host is a sink for the pathogen and **does not exert any effect on vector abundance…**

$$\frac{Y_i}{X_{tot} + A}$$



Alternative assumption:

- the non-competent host may increase vector density
  - either directly supporting more hosts (through more or larger blood meals, as for ticks)
  - or simply by attracting vectors from the nearby (as could occur in the case of mosquitoes)

if this happens…

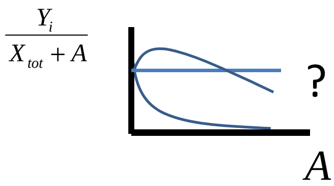
→there may be a tradeoff between:

pathogen dilution

&

amplification of vector abundance

→ the result of the loss of the non-competent host might not be unidirectional



A general model

$$\frac{dX_i}{dt} = -rX_i + ab\frac{Y_i}{X_{tot}+A}X_s$$

$$\frac{dY_i}{dt} = -dY_i + ac\frac{Y_s}{X_{tot}+A}X_i$$

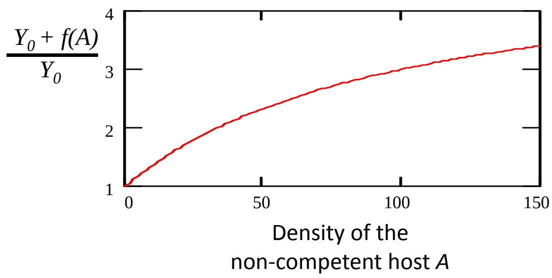
- X_i, X_s : density of infected/ susceptible primary hosts
- $X_{tot} = X_i + X_s$
- Y_i, Y_s : density of infected/ susceptible vectors

- r : recovery/mortality rate of infected hosts
- a : biting rate
- b : transmission prob. from infected vectors to susceptible host
- d : recovery/mortality rate of infected vectors
- c : transmission prob. from infected hosts to susceptible vectors
- A : density of the secondary, non competent host

Further assumptions…

- Constant populations
  - $X_{tot} = X_i + X_s = const.$
  - $Y_{tot} = Y_i + Y_s = const.$
  - $A = const.$
- $Y_{tot} = Y_0 + f(A)$

Amplification of the vector population



goal

- To use the model to assess how prevalence and density at equilibrium of infected hosts and of infected vectors change as a function of the density of the secondary, non-competent host A

A little bit of algebra…

$$\frac{1}{X_{tot}}\frac{dX_i}{dt} = -r\frac{X_i}{X_{tot}} + ab\frac{Y_i}{X_{tot}+A}\frac{X_s}{X_{tot}}$$
$$\frac{1}{Y_{tot}}\frac{dY_i}{dt} = -d\frac{Y_i}{Y_{tot}} + ac\frac{Y_s}{X_{tot}+A}\frac{X_i}{Y_{tot}}$$

$x_i = X_i / X_{tot}$ $\quad$ $y_i = Y_i / Y_{tot}$

$x_s = 1 - x_i$ $\quad$ $y_s = 1 - y_i$

The final model…

$$\frac{dx_i}{dt} = -rx_i + abm\delta\gamma(1 - x_i)y_i$$
$$\frac{dy_i}{dt} = -dy_i + ac\gamma(1 - y_i)x_i$$

x_i , y_i : prevalence of infected hosts/vectors

$1\text{-}x_i$: prev. of susceptible hosts

$1\text{-}y_i$: prev. of susceptible vectors

$$m = \frac{Y_0}{X_{tot}}$$

Number of vectors/host in the absence of the non competent host ($A=0$)

$$\gamma = \frac{X_{tot}}{X_{tot} + A} \leq 1$$

the fraction of competent hosts among all hosts on which vectors feed.
→ 1-γ = fraction of wasted bites

$$\delta = \frac{Y_{tot}}{Y_0} = \frac{Y_0 + f(A)}{Y_0} \geq 1$$

Relative increase in vector density driven by the introduction of non-competent alternative hosts

Note that when γ=δ= 1…

$$\frac{dx_i}{dt} = -rx_i + abm(1 - x_i)y_i$$
$$\frac{dy_i}{dt} = -dy_i + ac(1 - y_i)x_i$$

Ross-McDonald model for malaria (1911, 1957)

The basic reproductive number of the extended model

$$R_0(\gamma, \delta) = \frac{a^2bcm\gamma^2\delta}{d \cdot r}$$

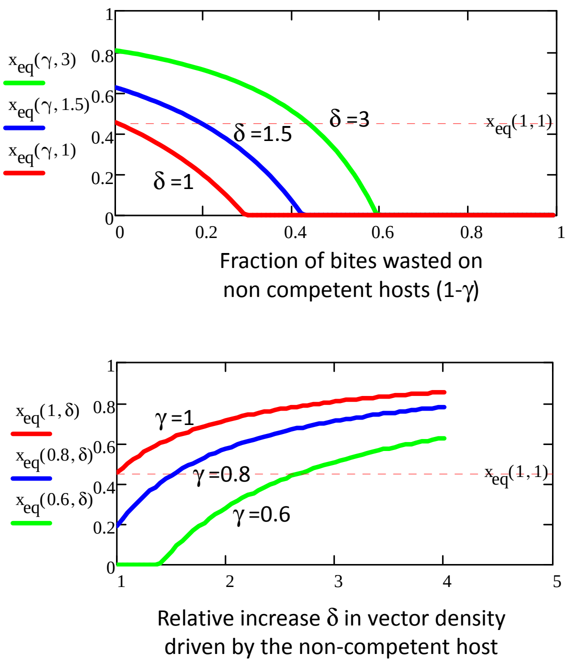
and the equilibrium prevalence:

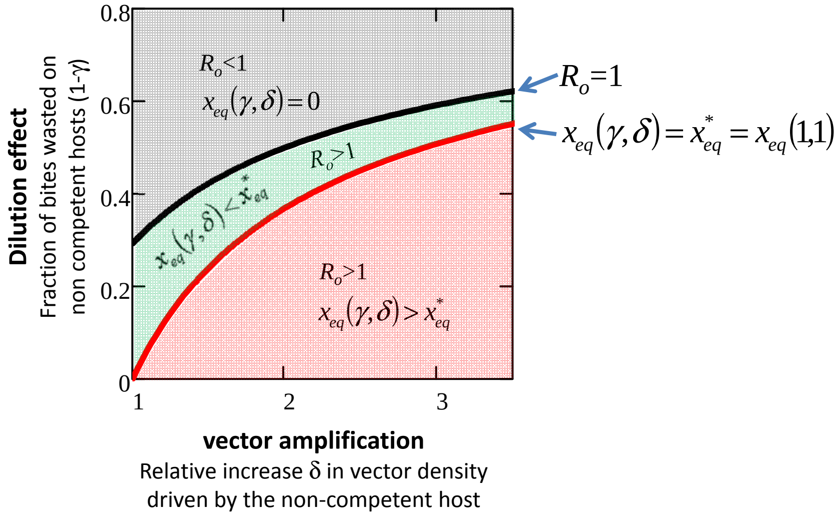
$$x_{eq}(\gamma, \delta) = \frac{a^2bcm\gamma^2\delta - dr}{a^2bcm\gamma^2\delta + rac\gamma}$$

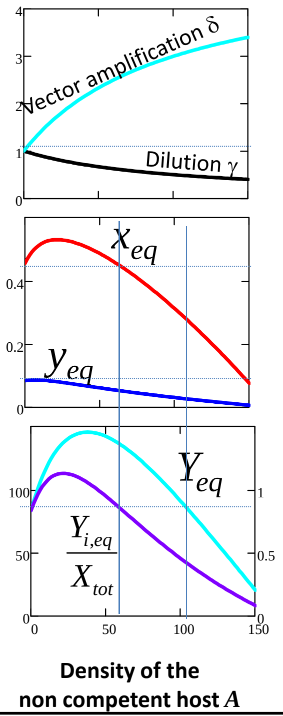
$$y_{eq}(\gamma, \delta) = \frac{a^2bcm\gamma^2\delta - dr}{a^2bcm\gamma^2\delta + abdm\gamma\delta}$$

A numerical example

a = 10	biting rate
b = 1 c=1	probability of transmission for infective bite
r =10	recovery/death rate of infected host
d=50	recovery/death rate of infected vectors
X_{tot} = 100	host abundance
Y_0= 1000	vector abundance
m=Y_0/X_{tot}	vector/host







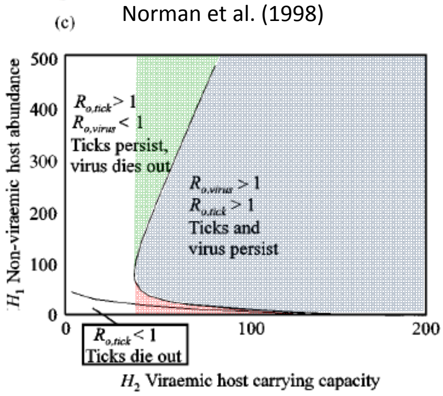
NB: what is relevant is the density of infected ticks, not their prevalence

Norman et al. (1998), Rosà Pugliese (2007, MB)

- Population dynamics of ticks (larvae/nymph and adults)
- infection with a pathogen (*Borrelia burgdorferi*, TBEv, Louping-ill virus)
- Two host classes:
  - small viremic host (mice and voles, grouse)
    - sustain tick larvae and lymph feed
  - medium-large, non-viremic (i.e. non-competent) mammals (hare, deer)
    - sustain adult ticks and complete their life cycle.

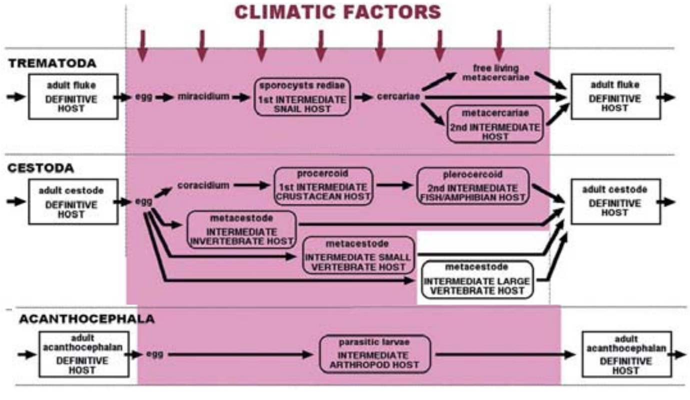
$$\dot{L}_Q^i = \varepsilon\sigma^A a_T(T)A_F^i - d^L L_Q^i - (\beta_1^L H_1 + \beta_2^L H_2)L_Q^i$$
$$\dot{L}_Q^s = (1-\varepsilon)\sigma^A a_T(T)A_F^i + \sigma^A a_T(T)A_F^s - d^L L_Q^s - (\beta_1^L H_1 + \beta_2^L H_2)L_Q^s$$
$$\dot{L}_F^i = (\beta_1^L H_1 + \beta_2^L H_2)L_Q^i + p_1^L \beta_1^L H_1^i L_Q^s - \sigma^L L_F^i$$
$$\dot{L}_F^s = (\beta_1^L (H_1 - p_1^L H_1^i) + \beta_2^L H_2)L_Q^s - \sigma^L L_F^s$$
$$\dot{N}_Q^i = m^L \sigma^L L_F^i - d^N N_Q^i - (\beta_1^N H_1 + \beta_2^N H_2)N_Q^i$$
$$\dot{N}_Q^s = m^L \sigma^L L_F^s - d^N N_Q^s - (\beta_1^N H_1 + \beta_2^N H_2)N_Q^s$$
$$\dot{N}_F^i = (\beta_1^N H_1 + \beta_2^N H_2)N_Q^i + p_1^N \beta_1^N H_1^i N_Q^s - \sigma^N N_F^i$$
$$\dot{N}_F^s = (\beta_1^N (H_1 - p_1^N H_1^i) + \beta_2^N H_2)N_Q^s - \sigma^N N_F^s$$
$$\dot{A}_Q^i = m^N \sigma^N N_F^i - d^A A_Q^i - (\beta_1^A H_1 + \beta_2^A H_2)A_Q^i$$
$$\dot{A}_Q^s = m^N \sigma^N N_F^s - d^A A_Q^s - (\beta_1^A H_1 + \beta_2^A H_2)A_Q^s$$
$$\dot{A}_F^i = (\beta_1^A H_1 + \beta_2^A H_2)A_Q^i + p_1^A \beta_1^A H_1^i A_Q^s - \sigma^A A_F^i$$
$$\dot{A}_F^s = (\beta_1^A (H_1 - p_1^A H_1^i) + \beta_2^A H_2)A_Q^s - \sigma^A A_F^s$$
$$\dot{H}_1^s = a_1(H_1)H_1 - d_1 H_1^s - (q_1^L \beta_1^L L_Q^i + q_1^N \beta_1^N N_Q^i + q_1^A \beta_1^A A$$
$$\dot{H}_1^i = (q_1^L \beta_1^L L_Q^i + q_1^N \beta_1^N N_Q^i + q_1^A \beta_1^A A_Q^i)H_1^s - (d_1 + \gamma_1 + \alpha_1$$
$$\dot{H}_1^r = \gamma_1 H_1^i - d_1 H_1^r.$$

Norman et al. (1998)



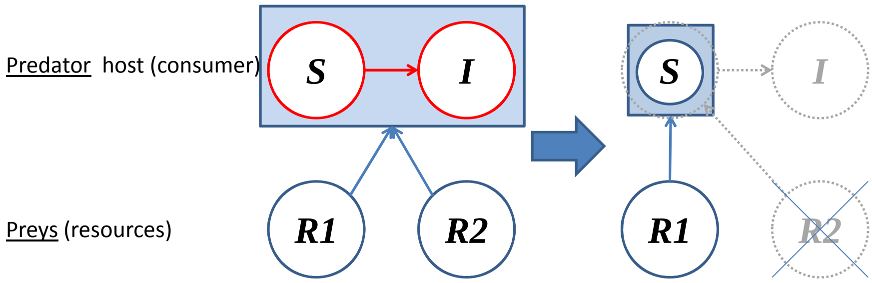
Other conceptual models in which a loss of free-living biodiversity might entail the loss of parasite diversity

1) Loss of an intermediate host for an indirectly transmitted macroparasite

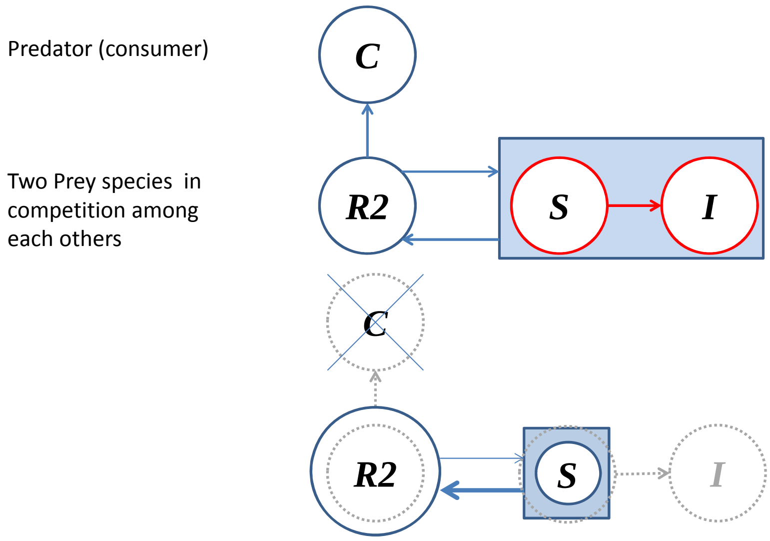


Source: Mas-Comas et al. *Rev. sci. tech. Off. int. Epiz.*, 2008, 27 (2), 443-452

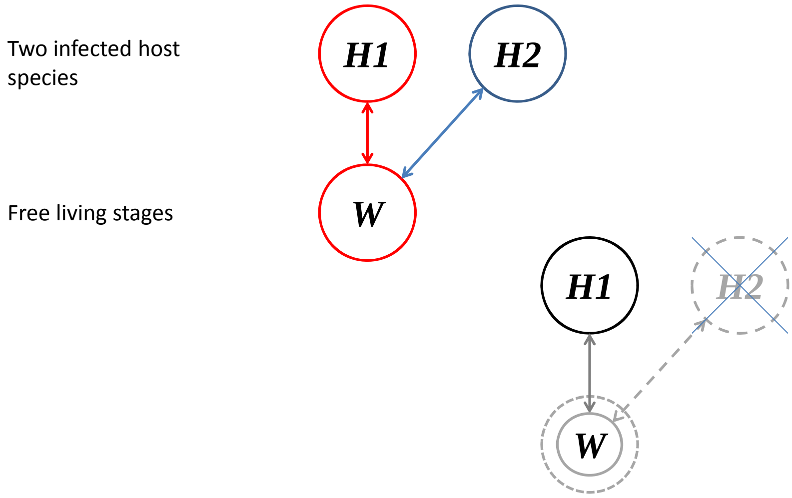
2) Loss of a prey species causes a predator host to drop down below the minimum threshold density for disease eradication: its parasite population is doomed to extinction



3) Removal of a top predator that allowed a less efficient prey host to coexist with a more efficient competitor, again causes the prey host to drop down the threshold density for disease eradication: its parasite population is doomed to extinction



4) Loss of a reservoir species causes a reduction of free living stages: the infective parasite or pathogen is not any longer able to sustain itself in the remaining (less efficient) host species and it is thus doomed to extinction



Further considerations

- So far only two/three-host species systems
- from the point of view of a parasite, the host is just a resource, a patch of suitable habitat
  - → as much as in the theory of island biogeography, a general loss of free living biodiversity at a wider geographical scale could imply a loss of parasite biodiversity

- Questions:
- **Are generalist/opportunistic/weedy species more competent than non generalist/endangered ones?**
- **What are the patterns of parasite biodiversity at a larger community level / bio-geographical scale?**

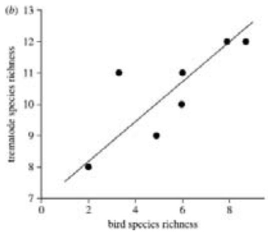
- Substantial literature on parasite ecology shows that richer, more connected, undisturbed communities of free living species harbor a richer parasite diversity than exploited, species poor community

PROCEEDINGS OF THE ROYAL SOCIETY B BIOLOGICAL SCIENCES

Host diversity begets parasite diversity: bird final hosts and trematodes in snail intermediate hosts

Ryan F Hechinger and Kevin D Lafferty

Proc. R. Soc. B 2005 **272**, 1059-1066



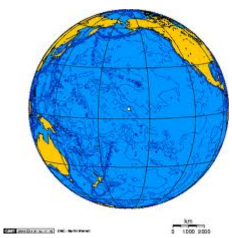
EcoHealth 5, 338–345, 2008
DOI: 10.1007/s10393-008-0196-7

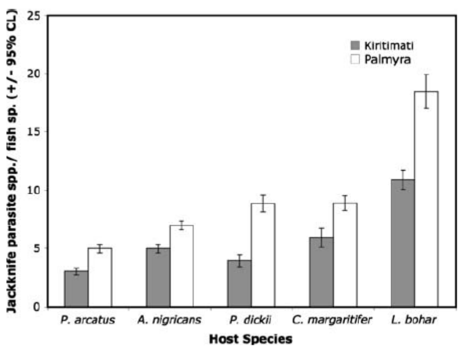
ECOHEALTH

Original Contribution

Reef Fishes Have Higher Parasite Richness at Unfished Palmyra Atoll Compared to Fished Kiritimati Island

Kevin D. Lafferty,[1] Jenny C. Shaw,[2] and Armand M. Kuris[2]





Effects of biodiversity loss on parasite biodiversity

ECOLOGY LETTERS
Ecology Letters, (2010) 13: 761–775 doi: 10.1111/j.1461-0248.2010.01467.x

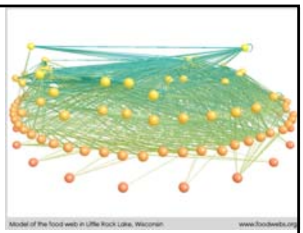
REVIEW AND SYNTHESIS

Fishing out marine parasites? Impacts of fishing on rates of parasitism in the ocean

Chelsea L. Wood,[1]* Kevin D. Lafferty[2] and Fiorenza Micheli[1]

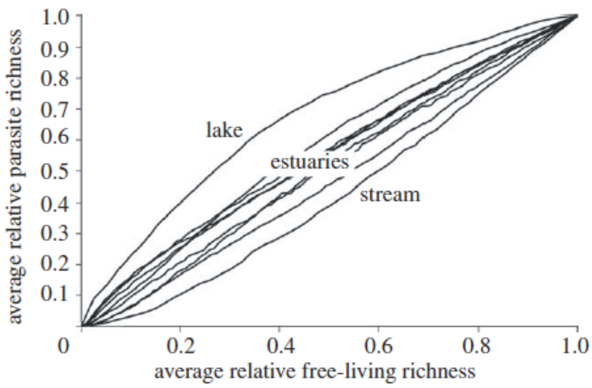
Abstract
Among anthropogenic effects on the ocean, fishing is one of the most pervasive and extends deepest into the past. Because fishing reduces the density of fish (reducing

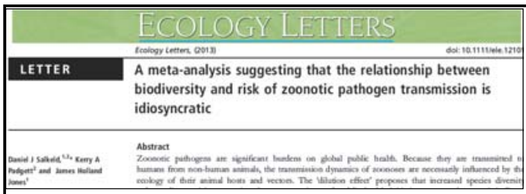
PHILOSOPHICAL TRANSACTIONS OF THE ROYAL SOCIETY B BIOLOGICAL SCIENCES



Biodiversity loss decreases parasite diversity: theory and patterns

Kevin D. Lafferty





ECOLOGY LETTERS

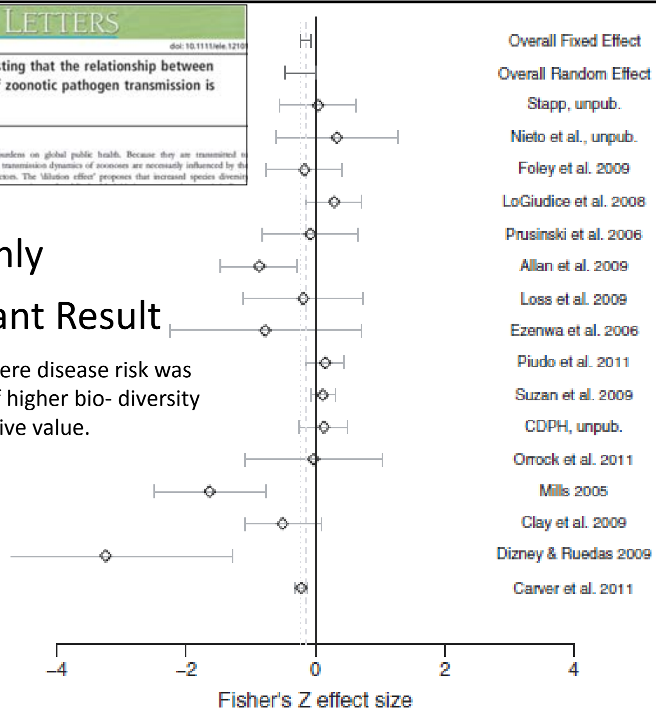
Ecology Letters, (2013) doi: 10.1111/ele.1210

LETTER

A meta-analysis suggesting that the relationship between biodiversity and risk of zoonotic pathogen transmission is idiosyncratic

Daniel J Salkeld,[1,2]* Kerry A Padgett[3] and James Holland Jones[1]

Abstract
Zoonotic pathogens are significant burdens on global public health. Because they are transmitted to humans from non-human animals, the transmission dynamics of zoonoses are necessarily influenced by the ecology of their animal hosts and vectors. The 'dilution effect' proposes that increased species diversity



- 13 studies only
- Non Significant Result

Effect size: Studies where disease risk was lower in treatments of higher bio- diversity were assigned a negative value.

Effects of biodiversity loss on human disease risk

CONCEPTS & SYNTHESIS

EMPHASIZING NEW IDEAS TO STIMULATE RESEARCH IN ECOLOGY

Ecology, 95(4), 2014, pp. 817–832
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Does biodiversity protect humans against infectious disease?

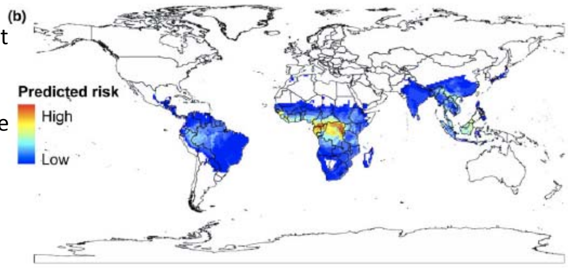
CHELSEA L. WOOD,[1,2,8] KEVIN D. LAFFERTY,[3] GIULIO DELEO,[1,4] HILLARY S. YOUNG,[5,6] PETER J. HUDSON,[7] AND ARMAND M. KURIS[5]

Cross-Species Pathogen Transmission and Disease Emergence in Primates

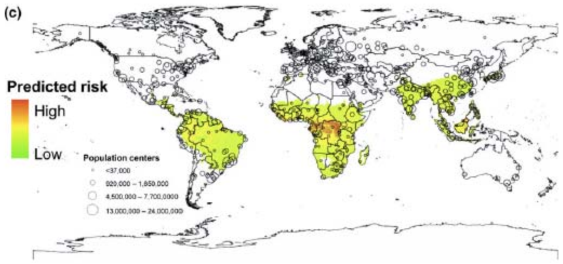
Amy B. Pedersen[1] and T. Jonathan Davies[2,3]

ECOHEALTH

Phylogenetic risk of pathogens host shifting to humans from wild primates. West central Africa is a hotspot of high risk to humans, due to the overlapping ranges of many of our closest relatives.



The intersection between high phylogenetic risk and an index of human population growth (increase in density from 1990–2000),

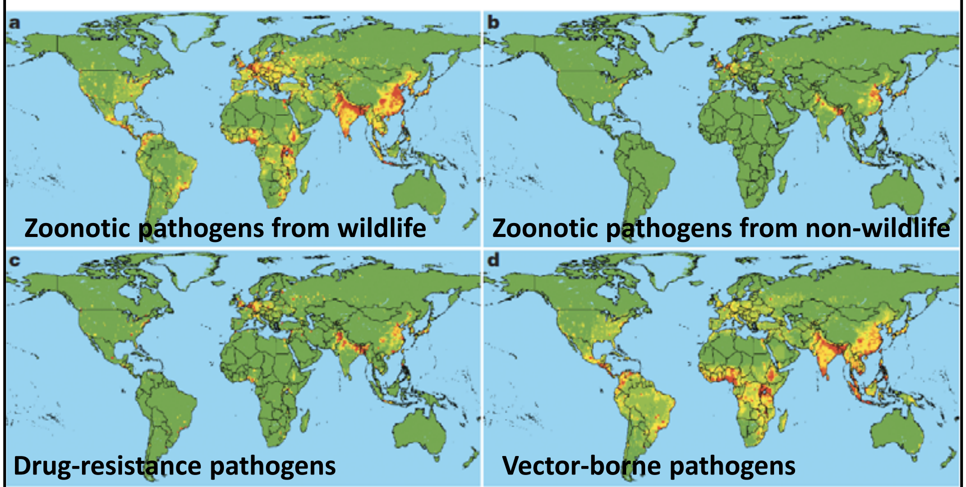


Global trends in emerging infectious diseases

Kate E. Jones[1], Nikkita G. Patel[2], Marc A. Levy[3], Adam Storeygard[3]†, Deborah Balk[3]†, John L. Gittleman[4] & Peter Daszak[2]

Nature 2008

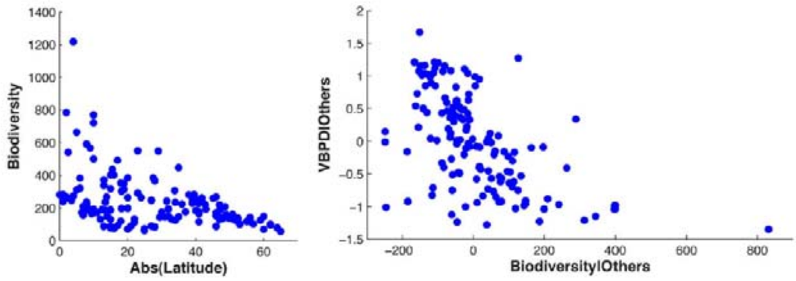
Risk of emerging infectious diseases



OPEN ACCESS Freely available online — PLOS | BIOLOGY

Disease Ecology, Biodiversity, and the Latitudinal Gradient in Income

Matthew H. Bonds[1]*, Andrew P. Dobson[2], Donald C. Keenan[3]



- **biodiversity gradient from low biodiversity at high latitude to high biodiversity at low latitudes.**
- **Yet, at low latitudes, countries with more than average biodiversity have lower incidence of disease**

summary

- There are certainly documented cases in which the loss of biodiversity increases disease risk for humans
  - Cascading effect benefitting competent reservoirs
  - Dilution effects driven by non-competent hosts
- Disease risk is highest at the interface between natural and human changed environment
- Loss of a non-competent host does not always lead to an increase in disease prevalence in the primary host or in the vector
- At a larger community or bio-geographical scale:
  - Loss of free living biodiversity might entail loss of parasite biodiversity
  - Habitats with more free-living biodiversity might well be areas with also more parasite biodiversity

Some questions open for discussion

- Does the relationship between biodiversity and disease depend upon the geographical scale?
- Is the answer depending upon what we are looking for?
  - Is the relationship between biodiversity & diseases different from that of loss of biodiversity & diseases?
  - Is the relationship between parasite diversity and the diversity of free living species fundamentally different if the focus is on assessing the risk of infectious diseases for humans instead of the analysis of geographical patterns of parasite biodiversity?
  - Pathogen vs. macroparasite?
  - Vector-borne diseases vs waterborne/soil borne?
- Is it possible that the the process of land use change, habitat fragmentation, habitat loss and human encroachment at the boundary between pristine and urbanized environment generate a confounding effect at the ecotone of pristine, high diversity habitat?
- Is it possible that the areas of highest biodiversity present low disease incidence also because they are by definition pristine and so there are few susceptible humans to be infected?