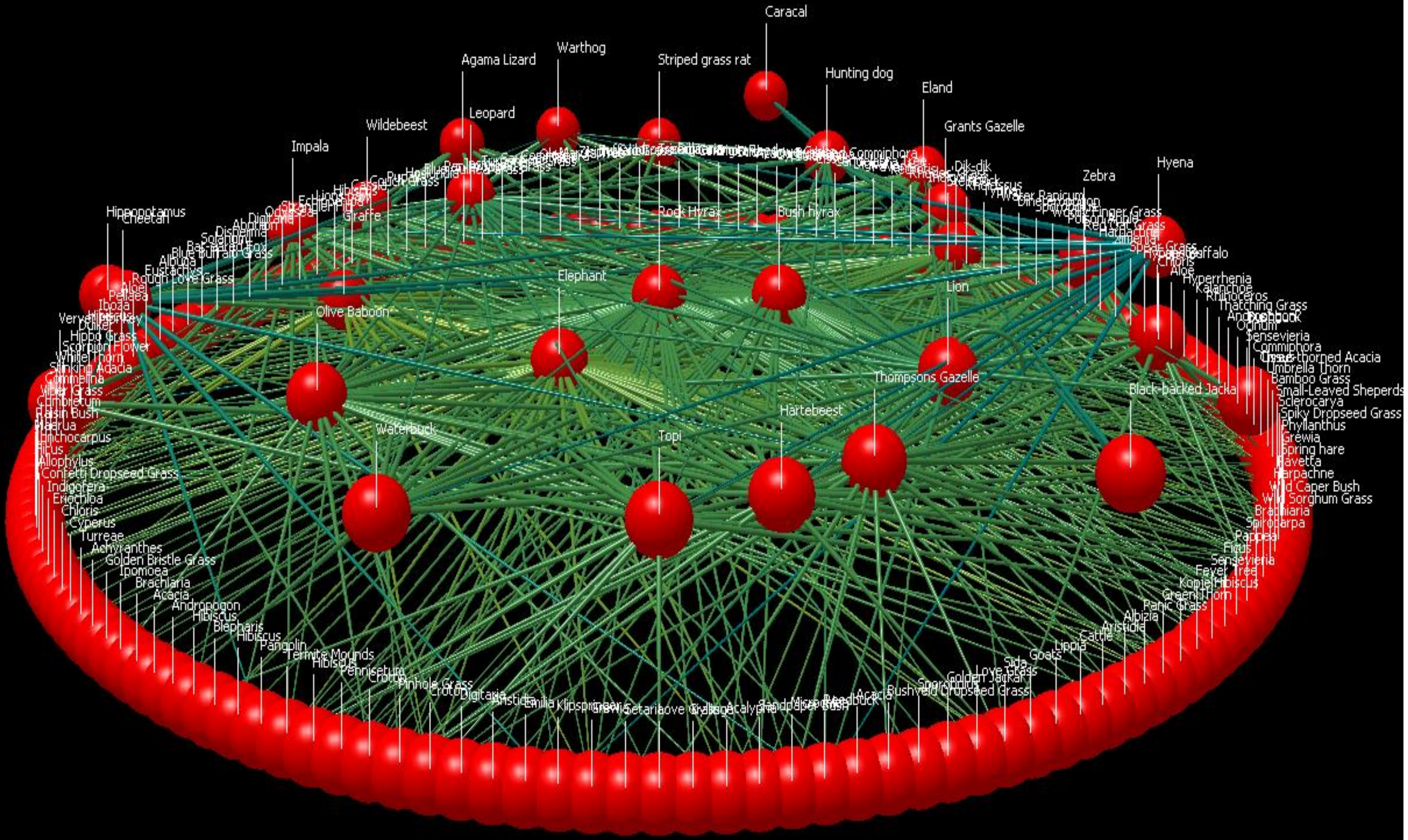
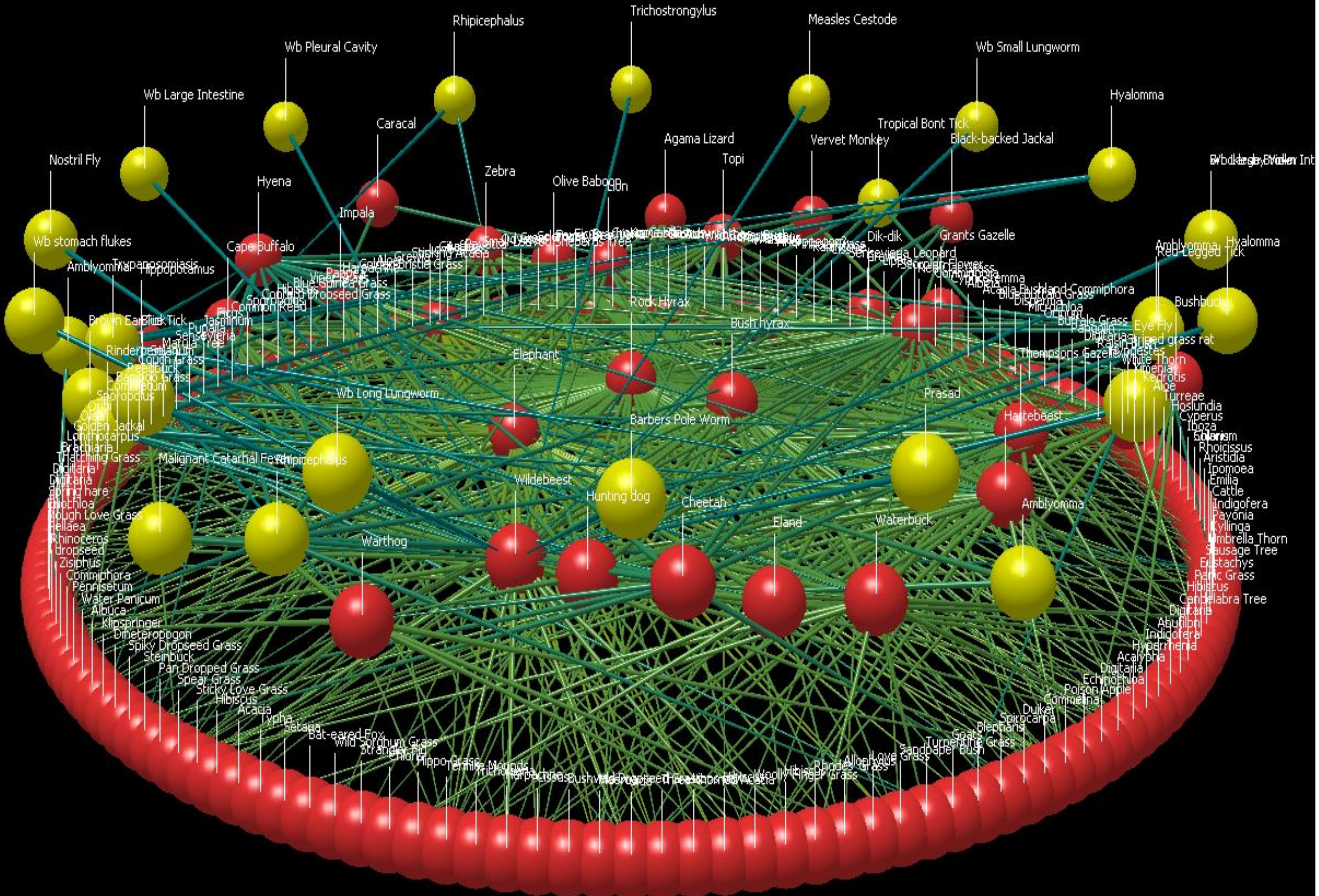
Pathogen with multiple hosts

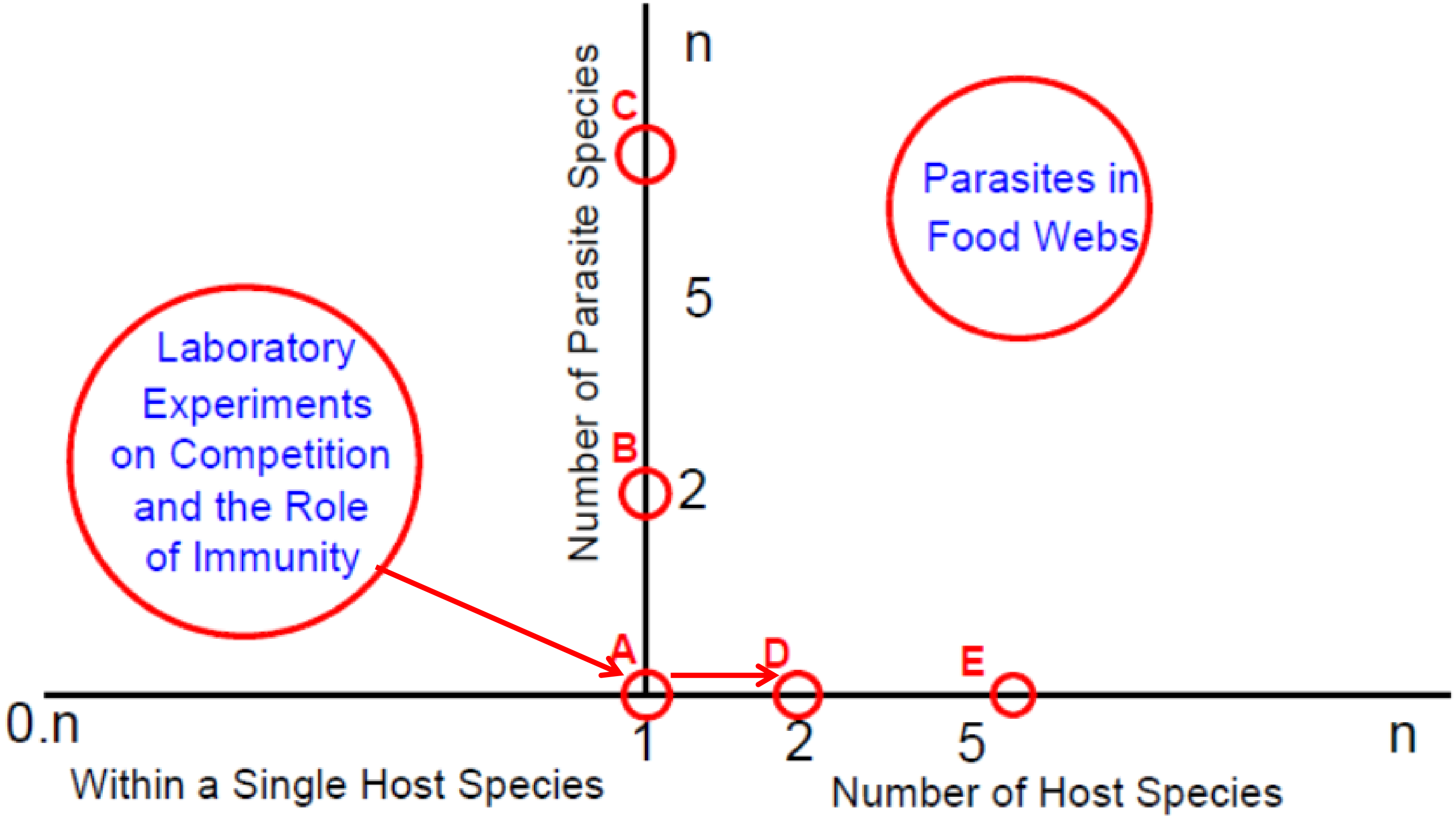
Andy Dobson

Deconstructing the Serengeti Foodweb:


Caracal
Hunting dog
Eland
Grants Gazelle
Striped grass rat
Warthog
Agama Lizard
Leopard
Wildebeest
Impala
Dik-dik
Zebra
Hyena
Hippopotamus
Cheetah
Rock Hyrax
Bush hyrax
Giraffe
Buffalo
Elephant
Olive Baboon
Lion
Thompsons Gazelle
Black-backed Jackal
Hartebeest
Topi
Waterbuck
Rough Love Grass
Blue Buffalo Grass
Couch Grass
Finger Grass
Red Oat Grass
Spear Grass
Aloe
Hyperrhenia
Kalanchoe
Rhinoceros
Thatching Grass
Ocimum
Sensevieria
Commiphora
Umbrella Thorn
Bamboo Grass
Small-Leaved Sheperds
Sclerocarya
Spiky Dropseed Grass
Phyllanthus
Grewia
Spring hare
Pavetta
Harpachne
Wild Caper Bush
Wild Sorghum Grass
Brachiaria
Spirocarpa
Pappea
Ficus
Fever Tree
Kopje Hibiscus
Green Thorn
Panic Grass
Albizia
Aristidia
Cattle
Lippia
Goats
Sida
Love Grass
Golden Jackal
Bushveld Dropseed Grass
Acacia
Reedbuck
Acalypha
Klipspringer
Emilia
Aristida
Digitaria
Croton
Pinhole Grass
Pennisetum
Termite Mounds
Hibiscus
Pangolin
Blepharis
Andropogon
Acacia
Brachiaria
Ipomoea
Golden Bristle Grass
Achyranthes
Turreae
Cyperus
Chloris
Eriochloa
Indigofera
Confetti Dropseed Grass
Allophylus
Ficus
Lannea
Erythrocarpus
Maerua
Raisin Bush
Combretum
Vilet Grass
Commelina
Stinking Acacia
Whilet Thorn
Scorpion Flower
Hippo Grass
Vervet Monkey
Duiker
Iboza
Pellaea
Aloe
Eustachys
Albuca
Solanum
Digitaria
Abutilon
Hibiscus
Echinochloa

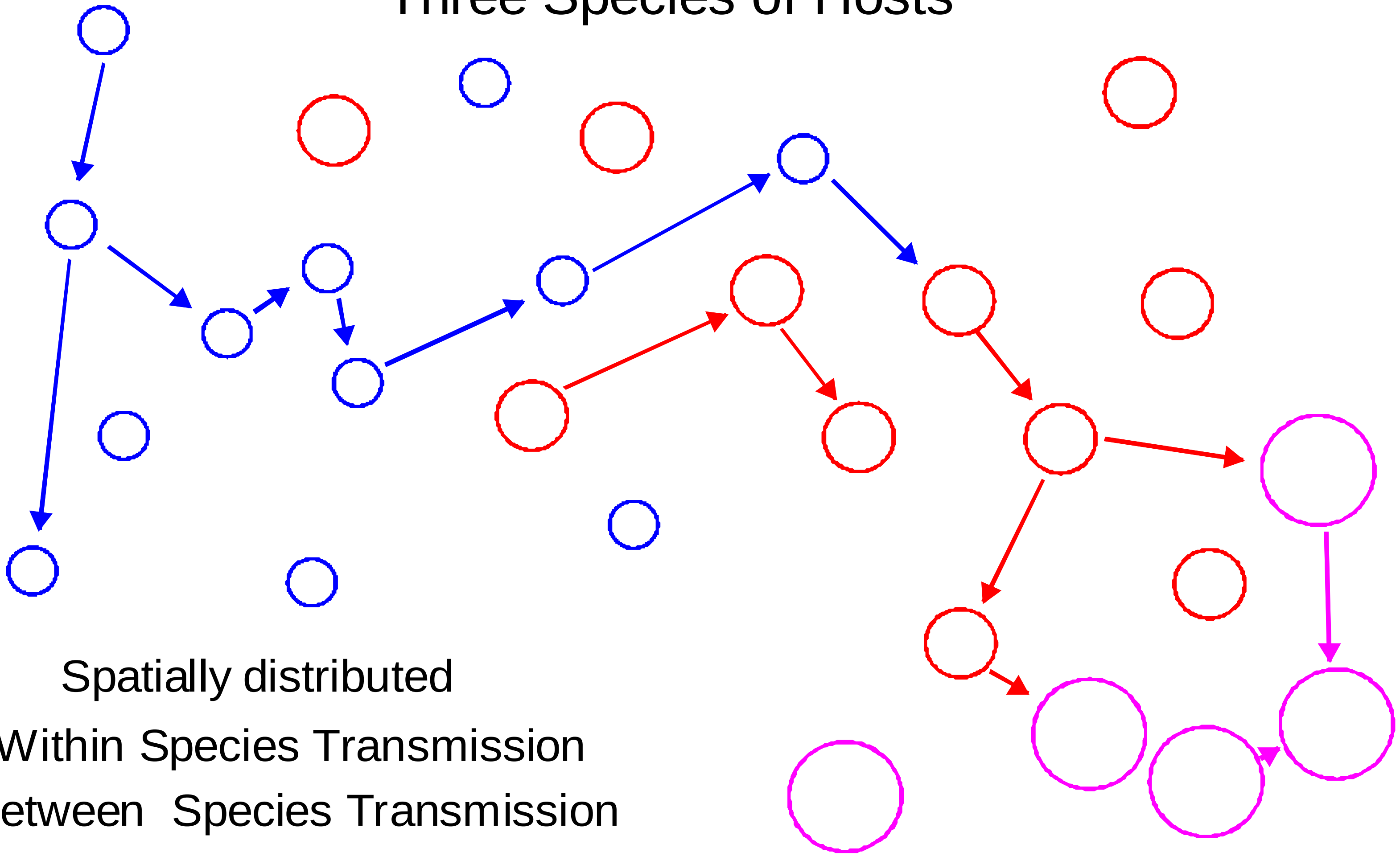

Trichostrongylus
Measles Cestode
Rhipicephalus
Wb Pleural Cavity
Wb Small Lungworm
Wb Large Intestine
Hyalomma
Caracal
Tropical Bont Tick
Agama Lizard
Vervet Monkey
Black-backed Jackal
Nostril Fly
Topi
Hyena
Zebra
Olive Baboon
Lion
Impala
Dik-dik
Grants Gazelle
Wb stomach flukes
Cape Buffalo
Amblyomma
Hyalomma
Red-Legged Tick
Trypanosomiasis
Hippopotamus
Leopard
Blue Buffalo Grass
Bushbuck
Rock Hyrax
Bush hyrax
Brown Ear Tick
Blue Tick
Eye Fly
Striped grass rat
Common Reed
Elephant
Thompsons Gazelle
White Thorn
Rinderpest
Wb Long Lungworm
Prasad
Hartebeest
Aloe
Turreae
Hoslundia
Cyperus
Barbers Pole Worm
Golden Jackal
Lonchocarpus
Brachiaria
Thatching Grass
Malignant Catarrhal Fever
Rhipicephalus
Rhoicissus
Aristida
Ipomoea
Emilia
Cattle
Indigofera
Pavonia
Kyllinga
Umbrella Thorn
Sausage Tree
Wildebeest
Hunting dog
Cheetah
Amblyomma
Spring hare
Rough Love Grass
Rhinoceros
Dropseed
Zisiphus
Commiphora
Pennisetum
Water Panicum
Albuca
Klipspringer
Dineteropogon
Spiky Dropseed Grass
Steinbuck
Pan Dropped Grass
Spear Grass
Sticky Love Grass
Hibiscus
Acacia
Typha
Setaria
Bat-eared Fox
Wild Sorghum Grass
Warthog
Eland
Waterbuck
Eustachys
Panic Grass
Hibiscus
Candelabra Tree
Digitaria
Abutilon
Indigofera
Hyparrhenia
Acalypha
Digitaria
Echinochloa
Poison Apple
Commelina
Duiker
Spirocarpa
Blepharis
Goat
Turpentine Grass
Sandpaper Bush
Love Grass
Finger Grass
Hippo Grass
Termite Mounds

Mathematical epidemiology has too limited a perspective……!!



A cartoon of the talk.....

Three Species of Hosts



Spatially distributed

Within Species Transmission

Between Species Transmission

Rinderpest – Serengeti

Basic model structure..

Susceptibles

Allometric scaling of all birth and death rates

$$dS_i \big/ dt = (b_i - d_i - \Delta_i (S_i + I_i))S_i - (\underset{\substack{\uparrow \\ \text{Within}}}{\beta_{ii}} I_i + \sum \underset{\substack{\nwarrow \\ \text{Between}}}{\beta_{ij}} I_i) S / (\sum_{j=1,n} N_n)^c$$

Infecteds

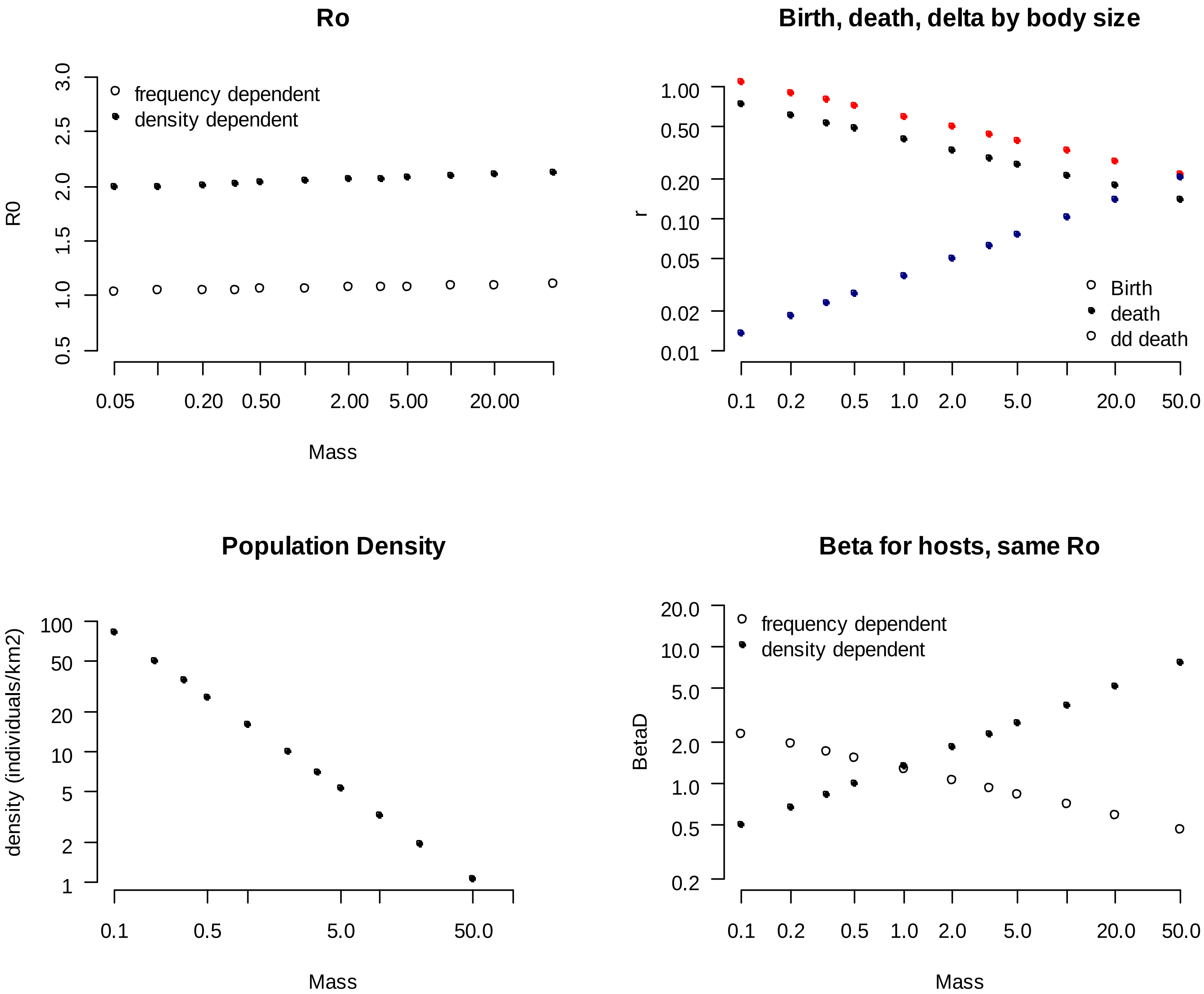
$$dI_i / dt = (\beta_{ii} I_i + \sum \beta_{ij} I_i) S / (\sum_{j=1,n} N_n)^c - d_i (1 + \alpha_i) I_i$$

Scale virulence
as a proportion
of life expectancy

Between species transmission

$$\beta_{ij} = c\sqrt{\beta_{ii}\beta_{jj}}$$

Underlying demography based on allometric scaling with body size (DeLeo and Dobson, Nature 1997)



Basic model structure..

Susceptibles

Allometric scaling of all birth and death rates

$$dS_i / dt = (b_i - d_i - \Delta_i (S_i + I_i))S_i - (\underset{\underset{\text{Within}}{\uparrow}}{\beta_{ii}} I_i + \sum \underset{\underset{\text{Between}}{\nwarrow}}{\beta_{ij}} I_i) S / (\sum_{j=1,n} N_n)^c$$

Infecteds

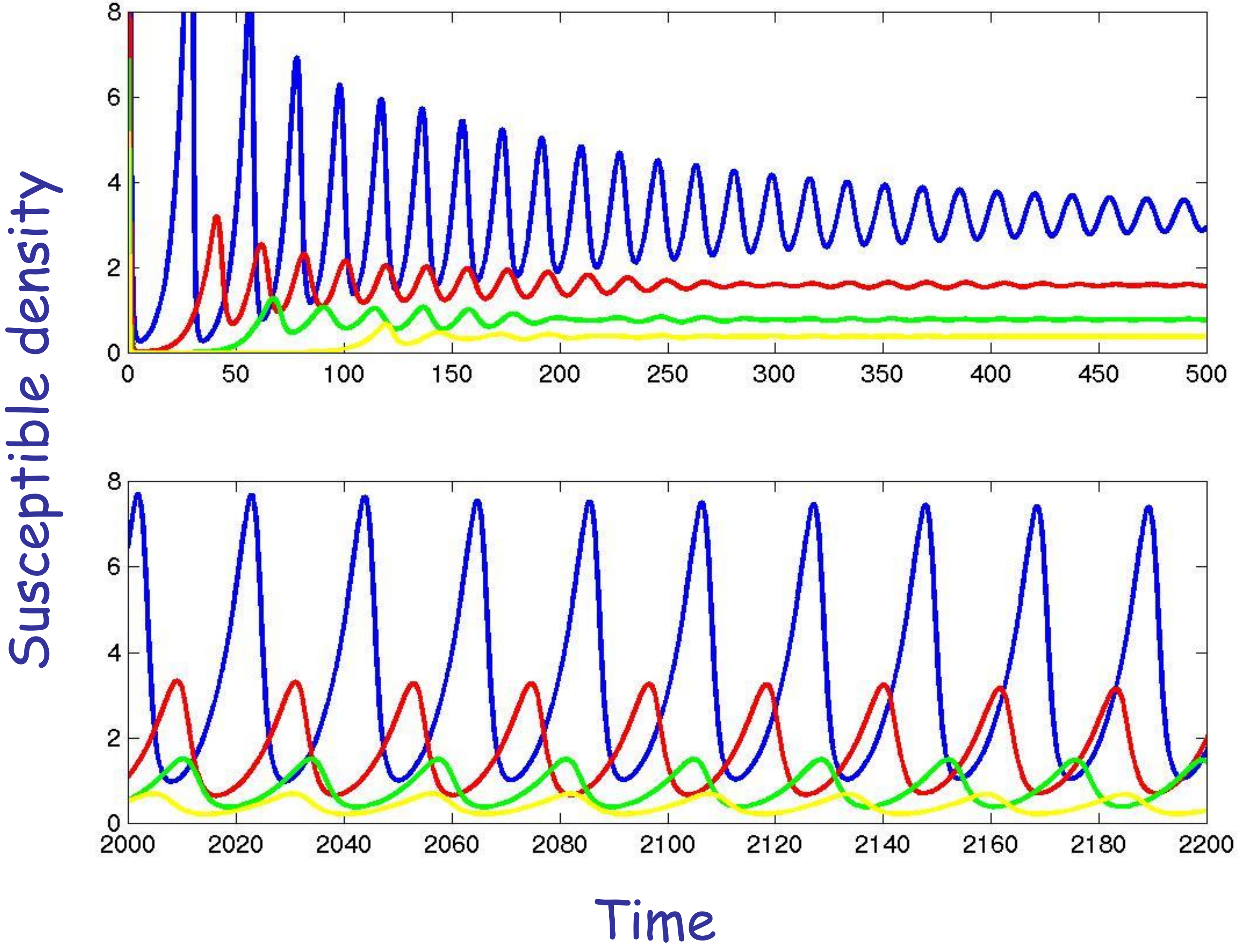
$$dI_i / dt = (\beta_{ii} I_i + \sum \beta_{ij} I_i) S / (\sum_{j=1,n} N_n)^c - d_i (1 + \alpha_i) I_i$$

Scale virulence
as a proportion
of life expectancy

Between species transmission

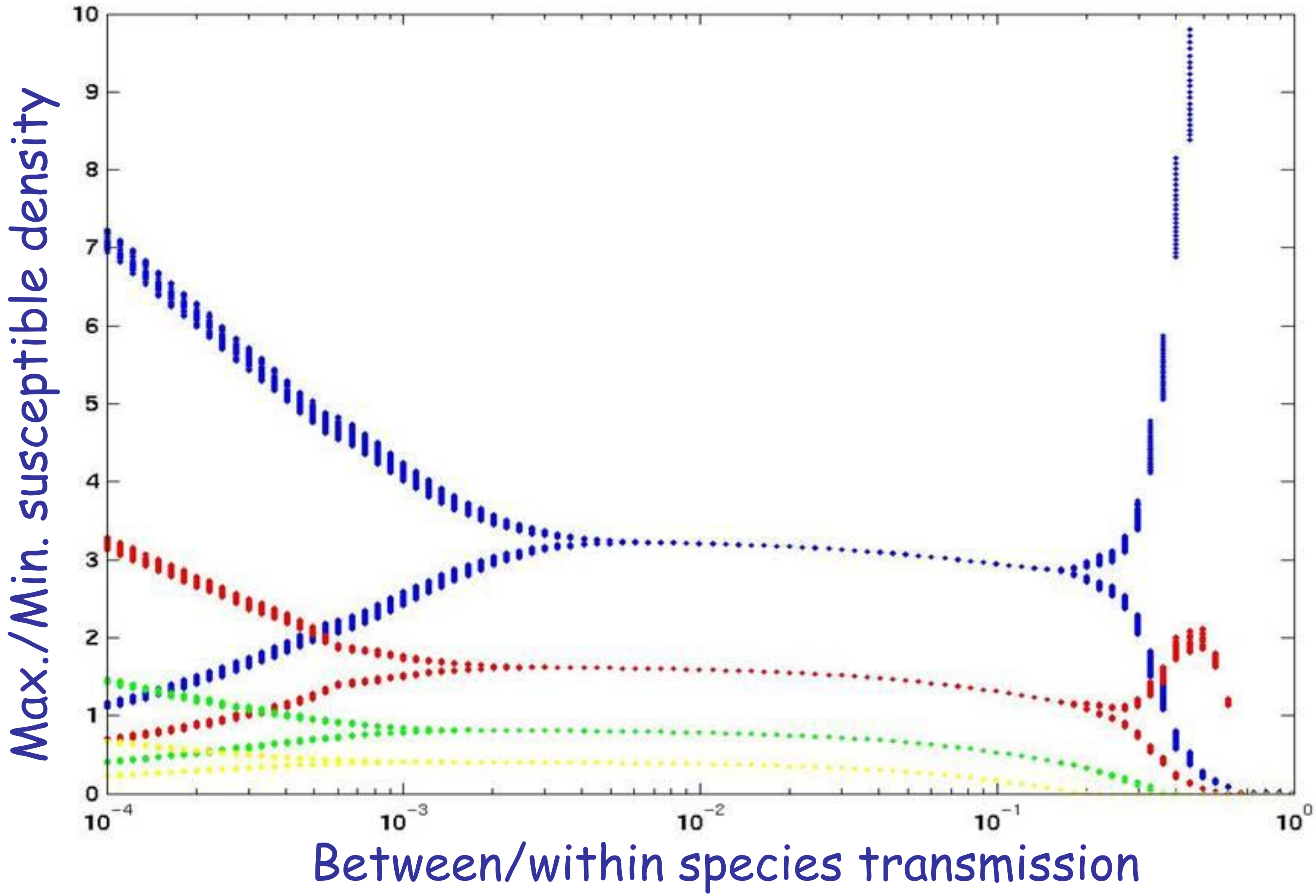
$$\beta_{ij} = c \sqrt{\beta_{ii} \beta_{jj}}$$

Buffering: dynamics in DD case

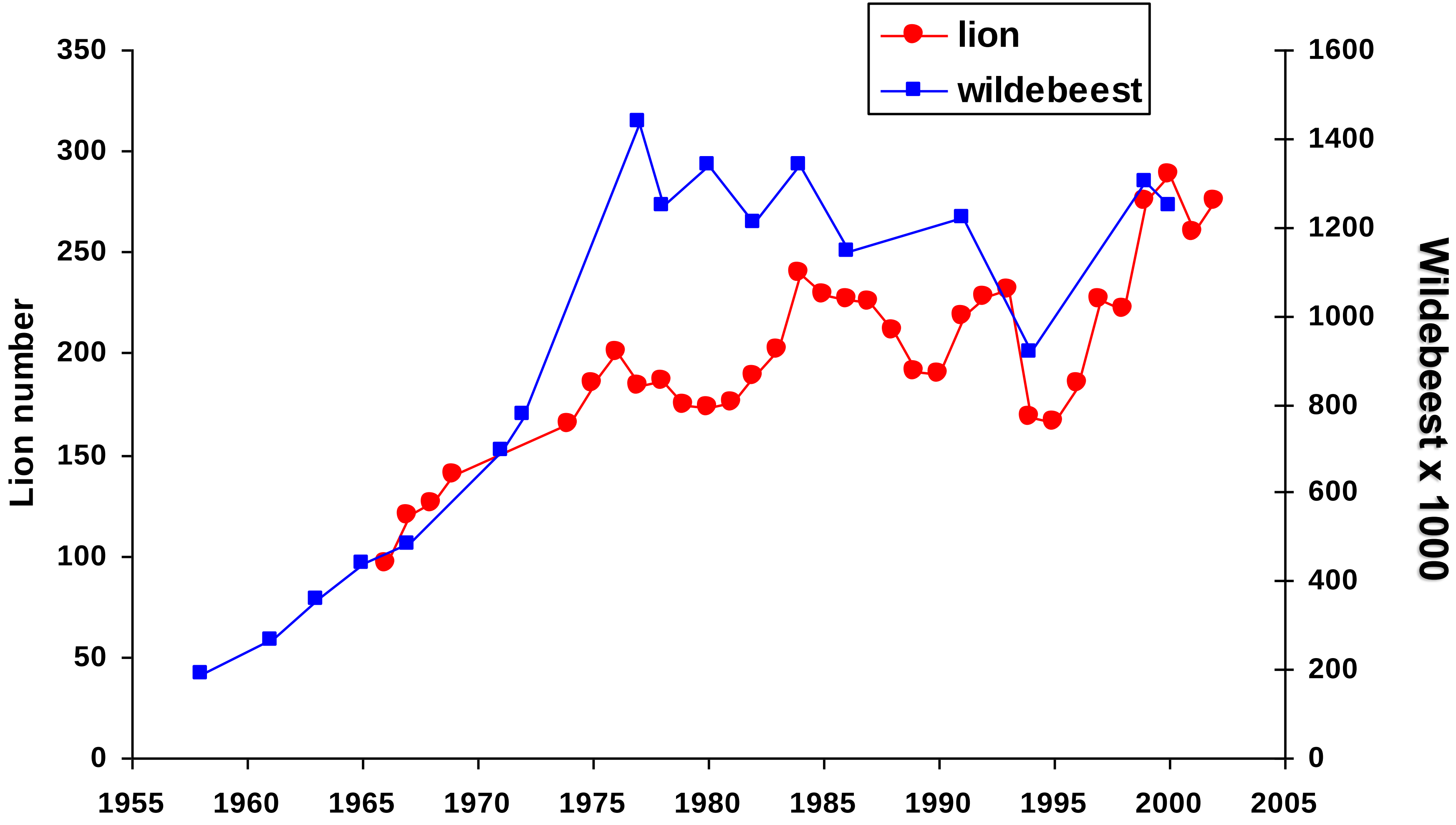


Between sps.
transmission

Buffering: dynamics in DD case



Serengeti woodland lion numbers



Lion data from C. Packer

Serengeti secrets : Vaccinating to Control Multiple Pathogens

Dynamics of viral pathogens in Serengeti

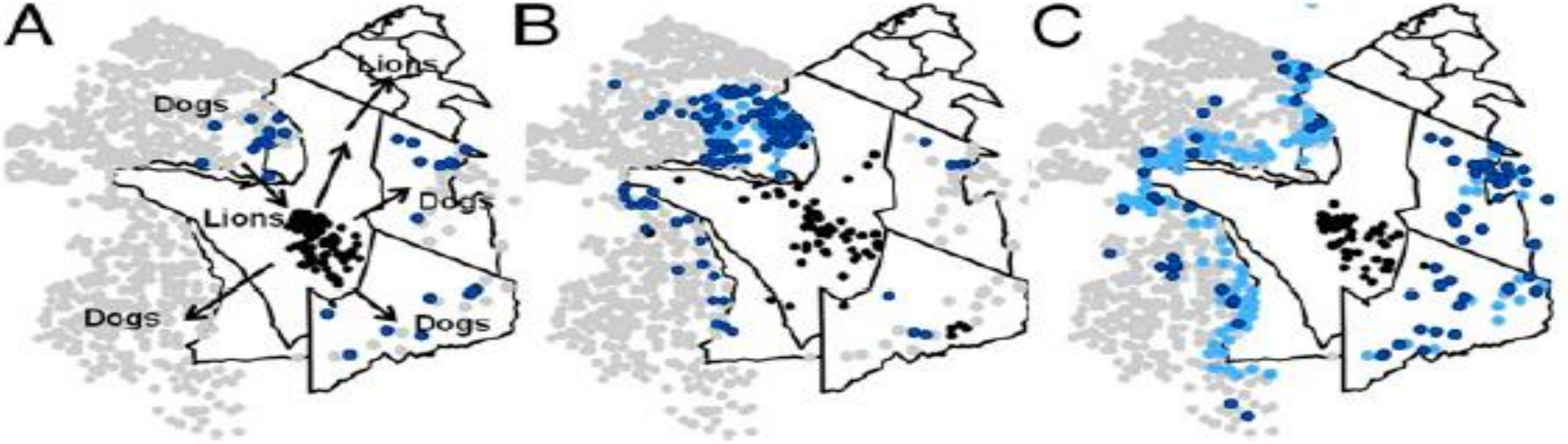
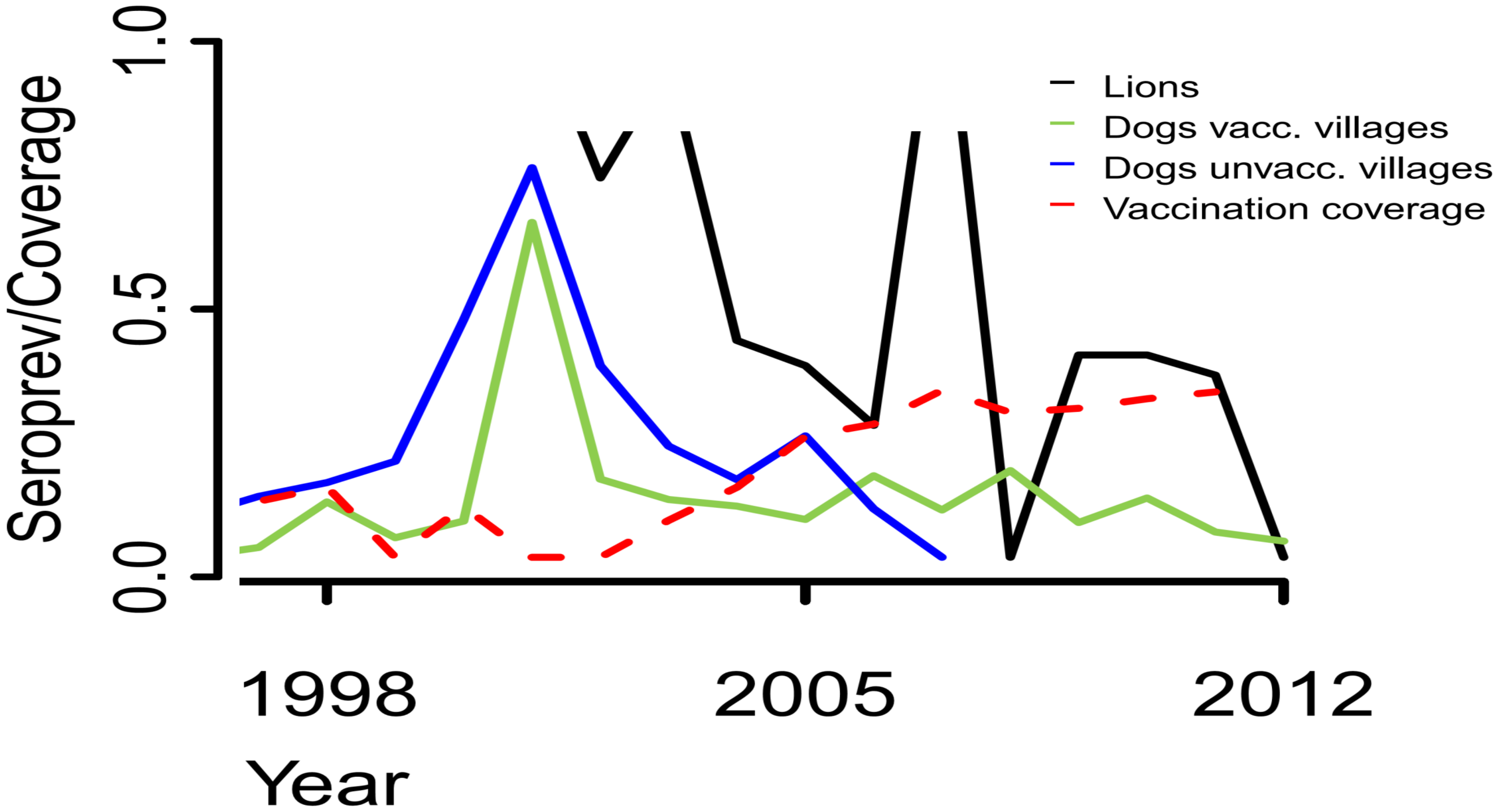
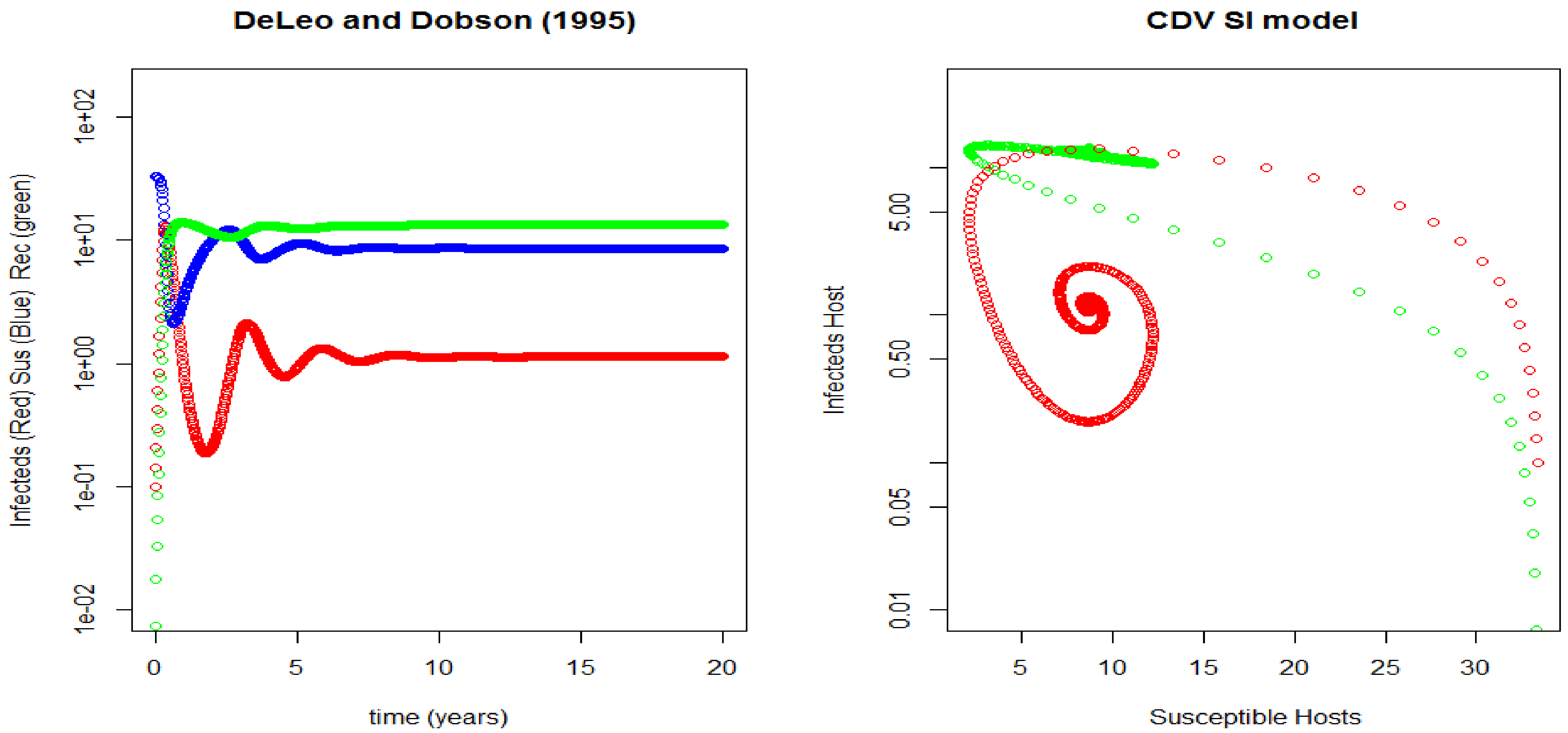


Fig. 1. Map of the Serengeti ecosystem (Tanzania). Circles represent human settlements (gray) surrounding the Serengeti National Park, villages/households from which domestic dogs were sampled (dark blue), locations where lions were sampled (black), and villages included in domestic dog vaccination campaigns that were not sampled (pale blue). (*A*) Arrows indicate the direction of the spread of CDV during the 1994 epidemic as reconstructed by Cleaveland et al. (16). (*B*) Small-scale domestic dog vaccination campaigns conducted during 1996–2002. (*C*) Expanded domestic dog vaccination program implemented during 2003–2012.

Joint work with Sarah Cleaveland, Katie Hampson, Craig Packer, Tiziana Lembo, Mafalda Viana and many others…

www.pnas.org/cgi/doi/10.1073/pnas.1411623112

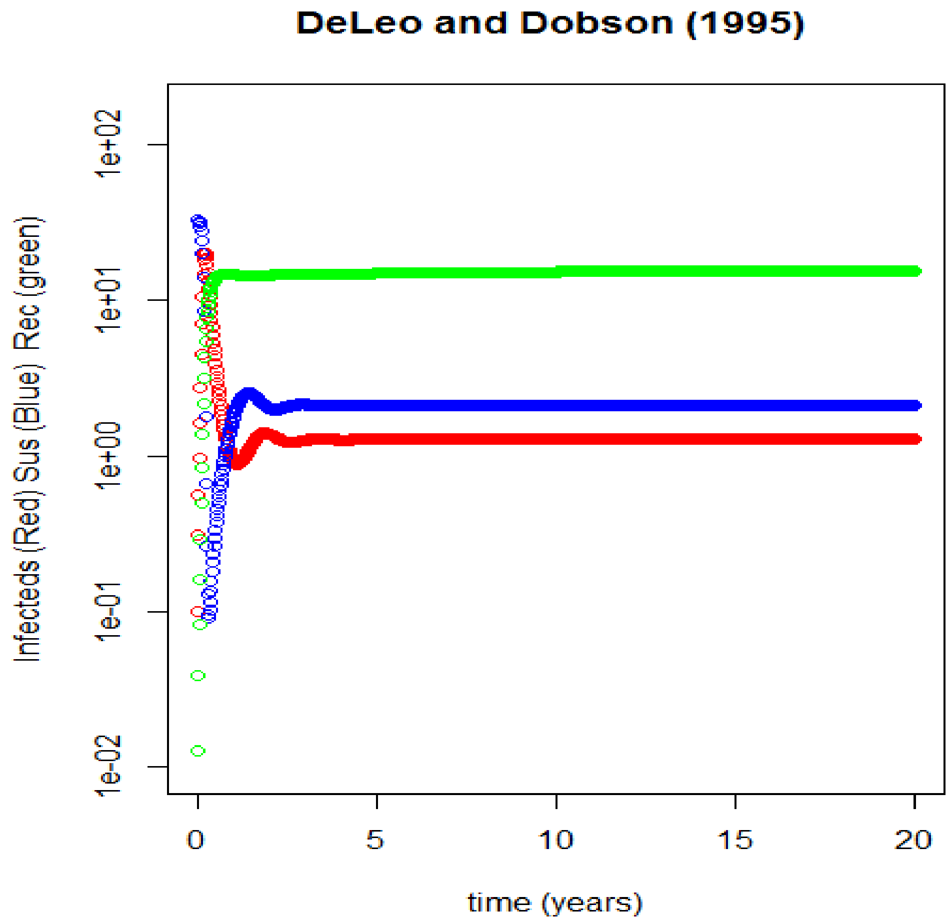

Seroprev/Coverage
1.0
0.5
0.0
Lions
Dogs vacc. villages
Dogs unvacc. villages
Vaccination coverage
1998
2005
2012
Year



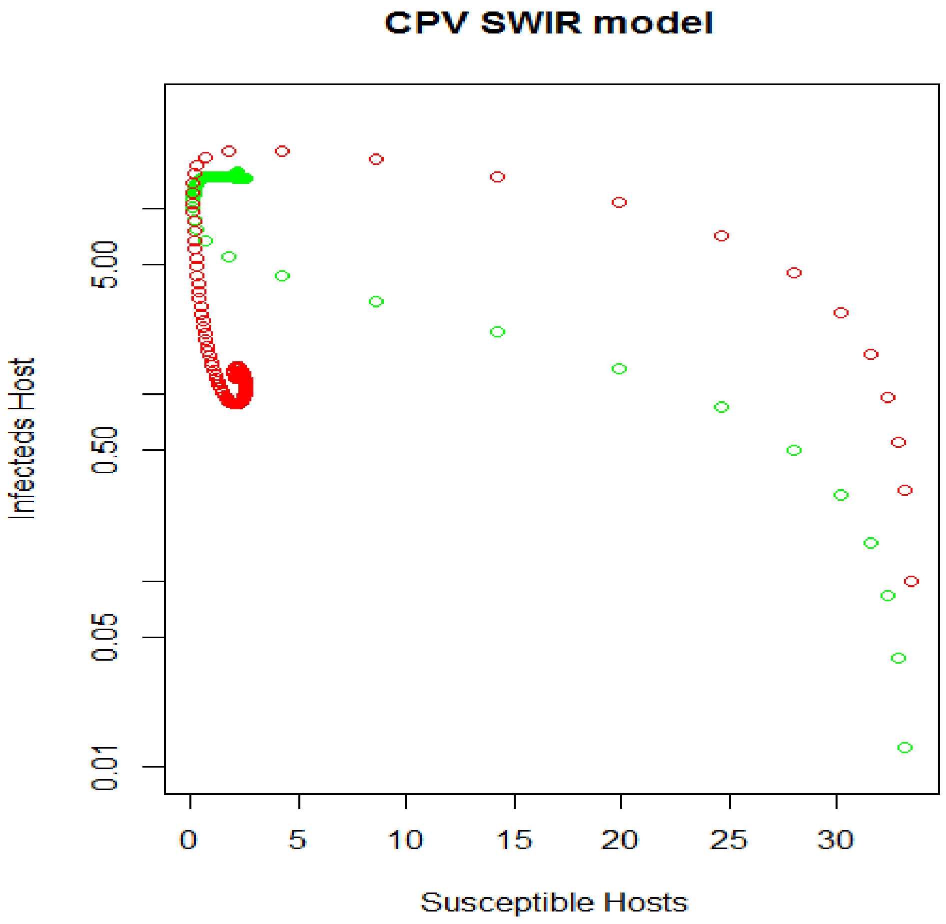
DeLeo and Dobson (1995)

CDV SI model

Canine distemper as only pathogen in population

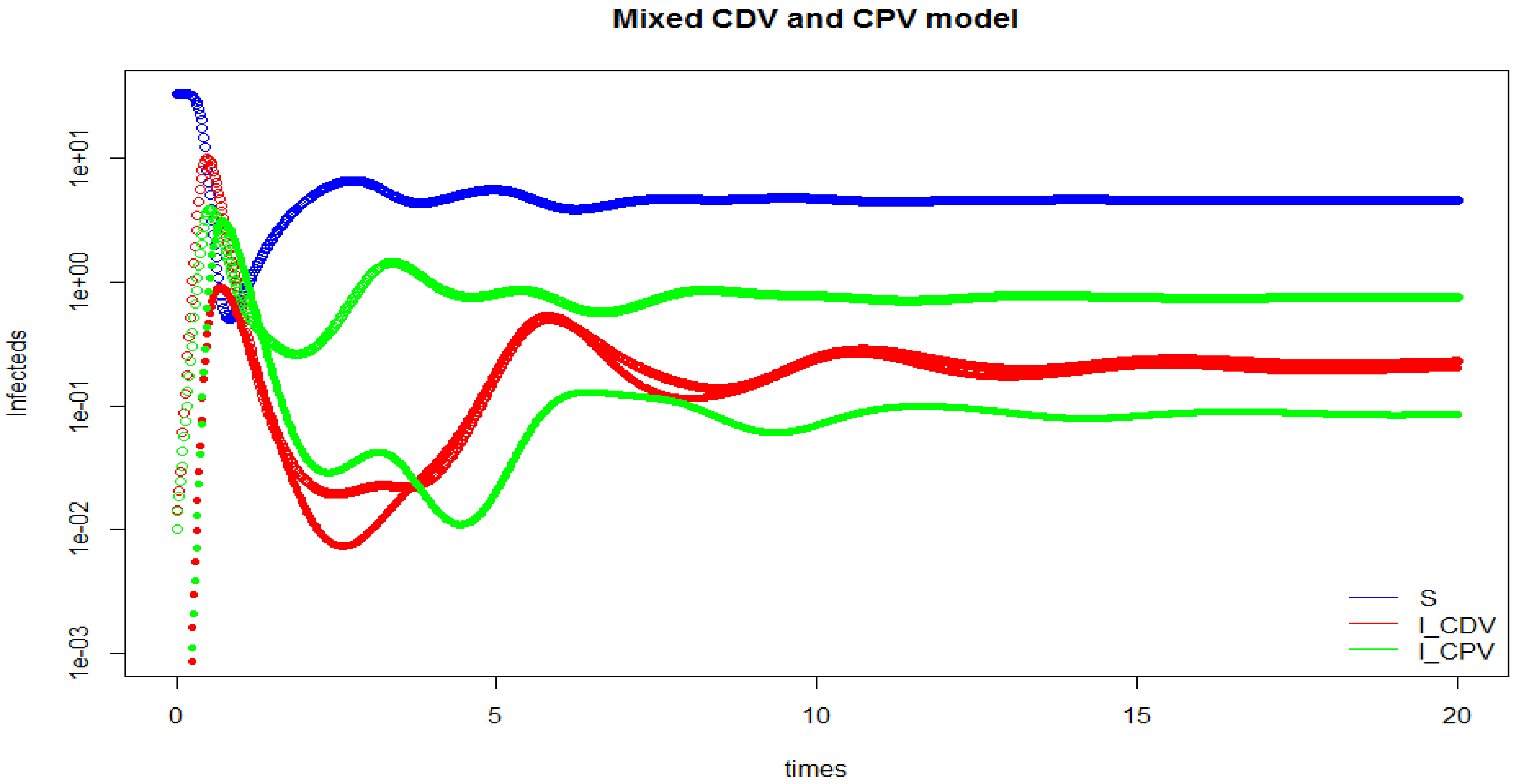


DeLeo and Dobson (1995)

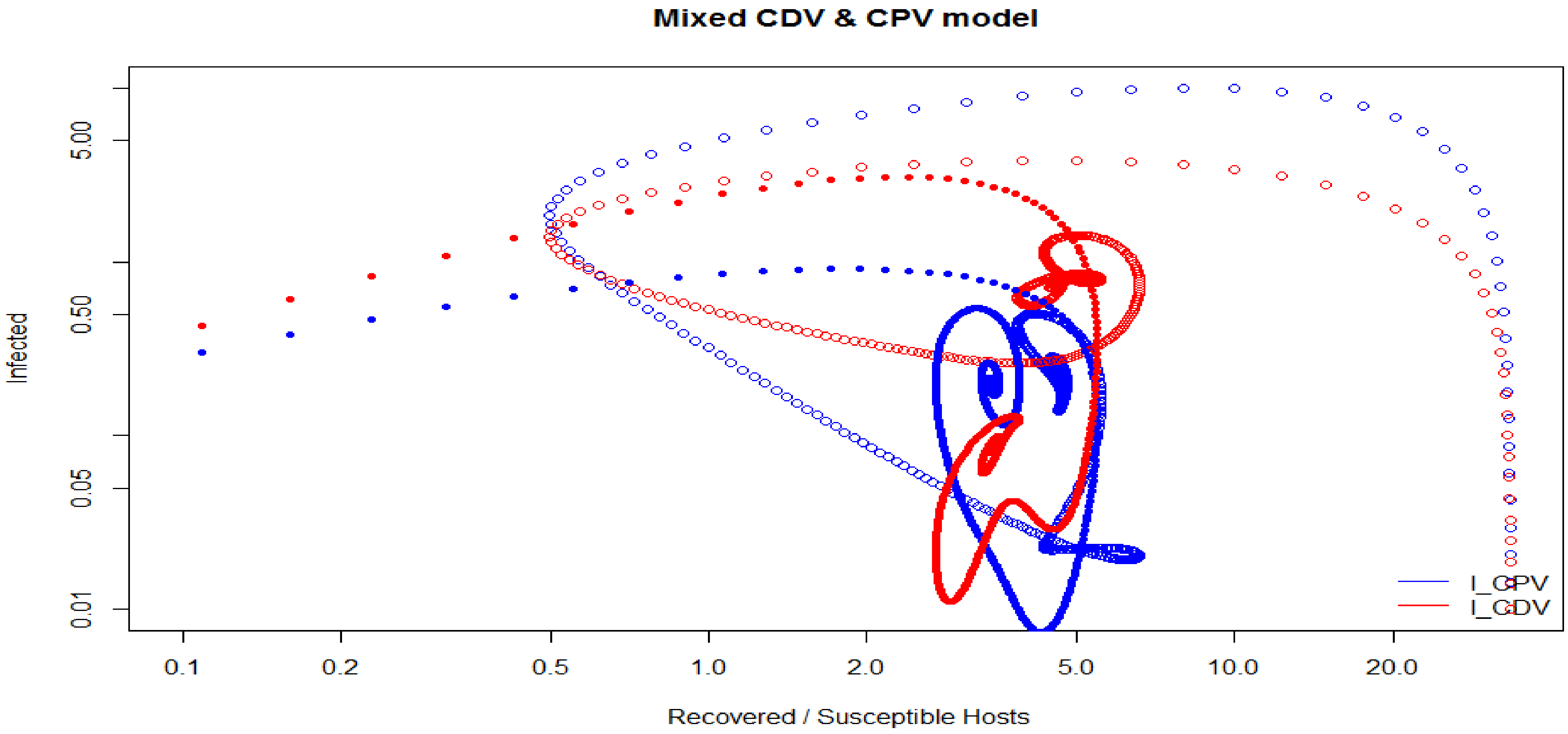


CPV SWIR model

Canine parvovirus as only pathogen present….equally boring..!

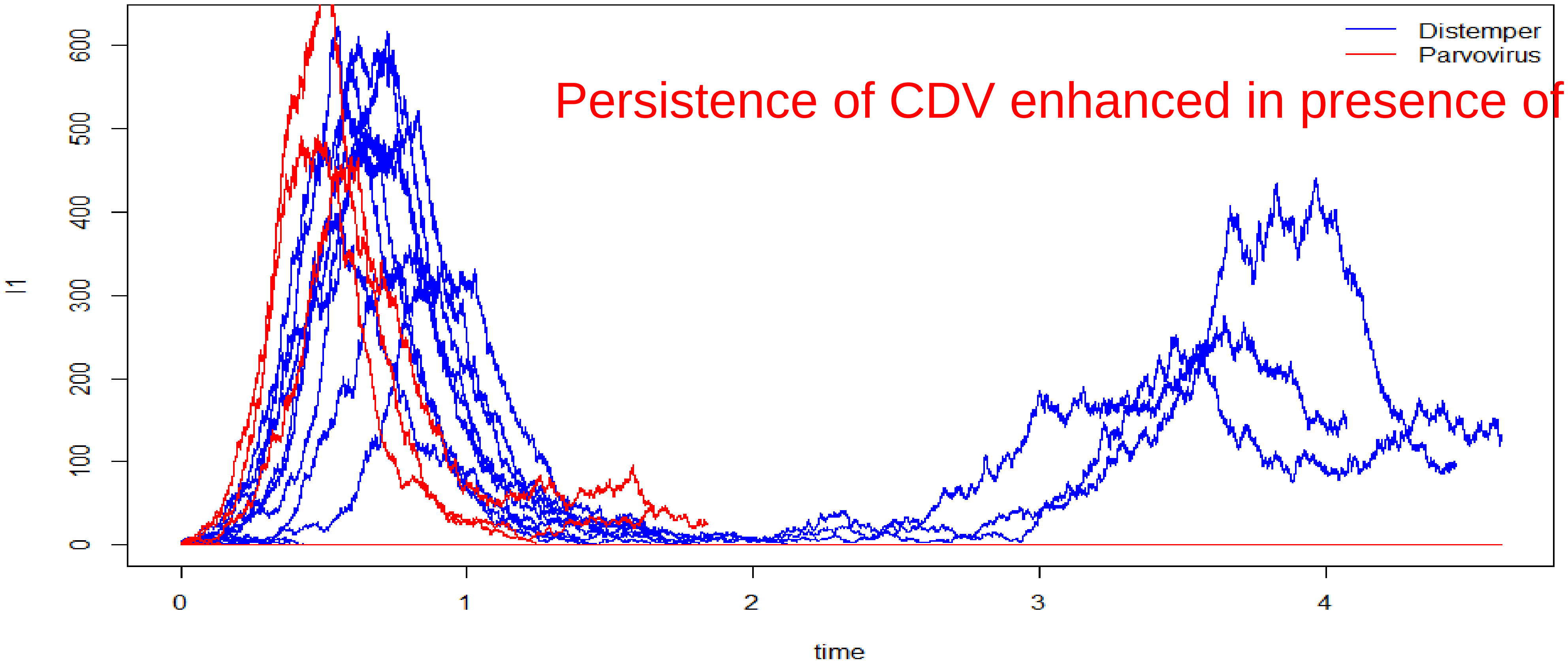


Both CPV and CDV circulating in the host population….!!



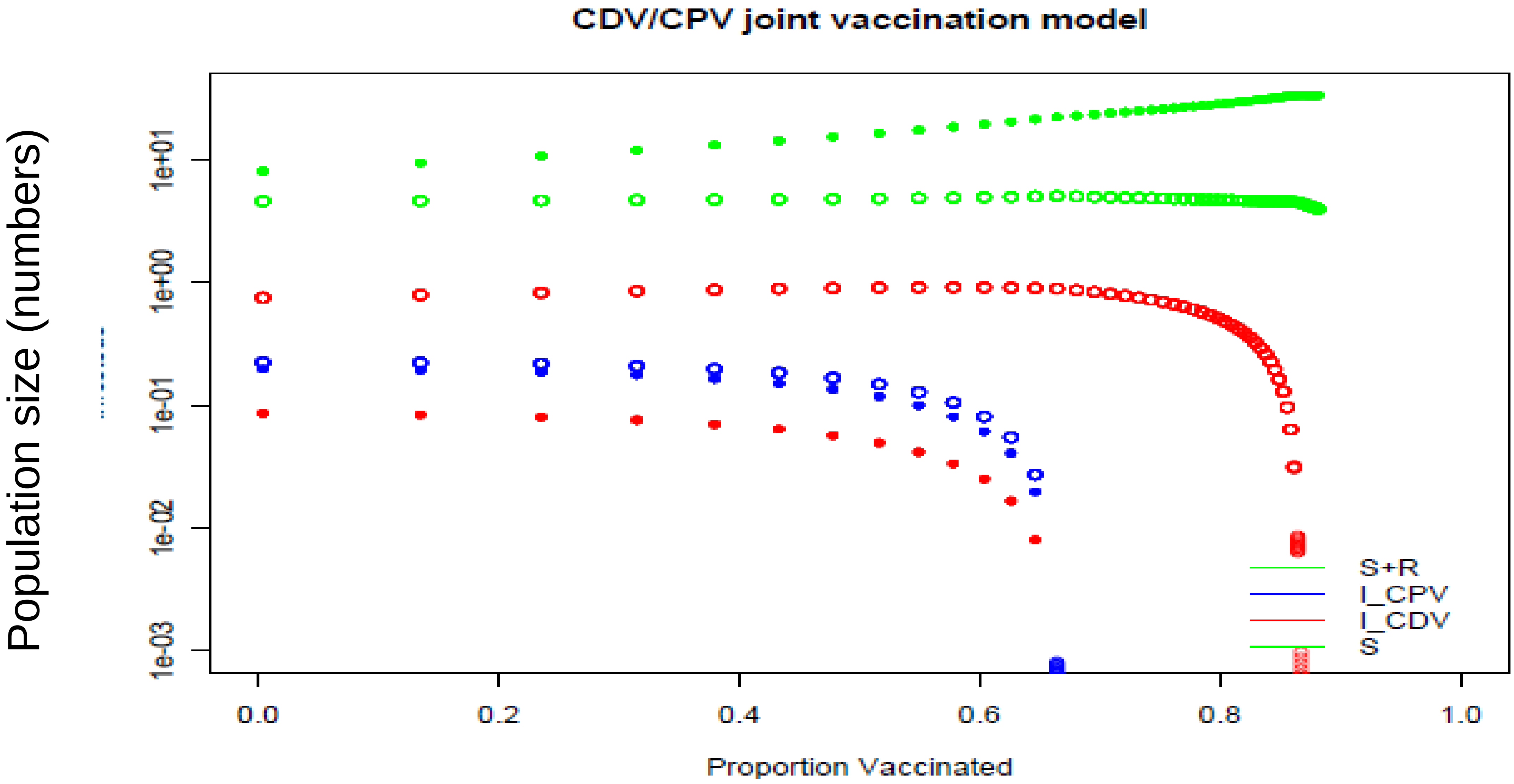
Dynamics are much more interesting although each host is only
Ever infected with one virus at any one time…

Stochastic CDV and CPV in population of 10,000 dogs



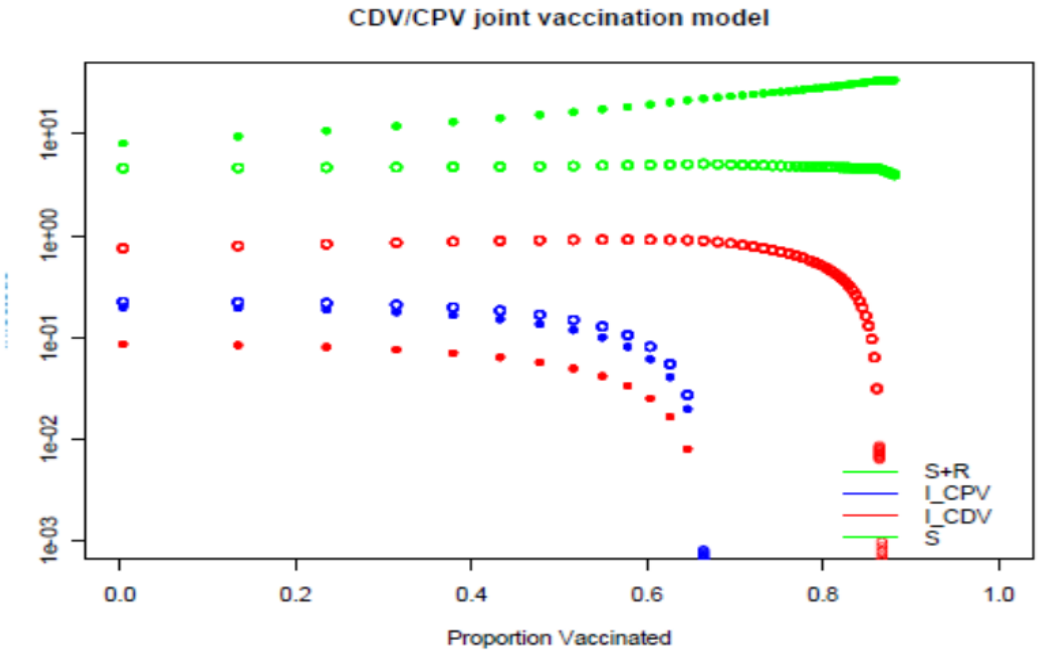
Persistence of CDV enhanced in presence of CPV

Vaccination with two pathogens and a single host (CPV, CDV and dogs)

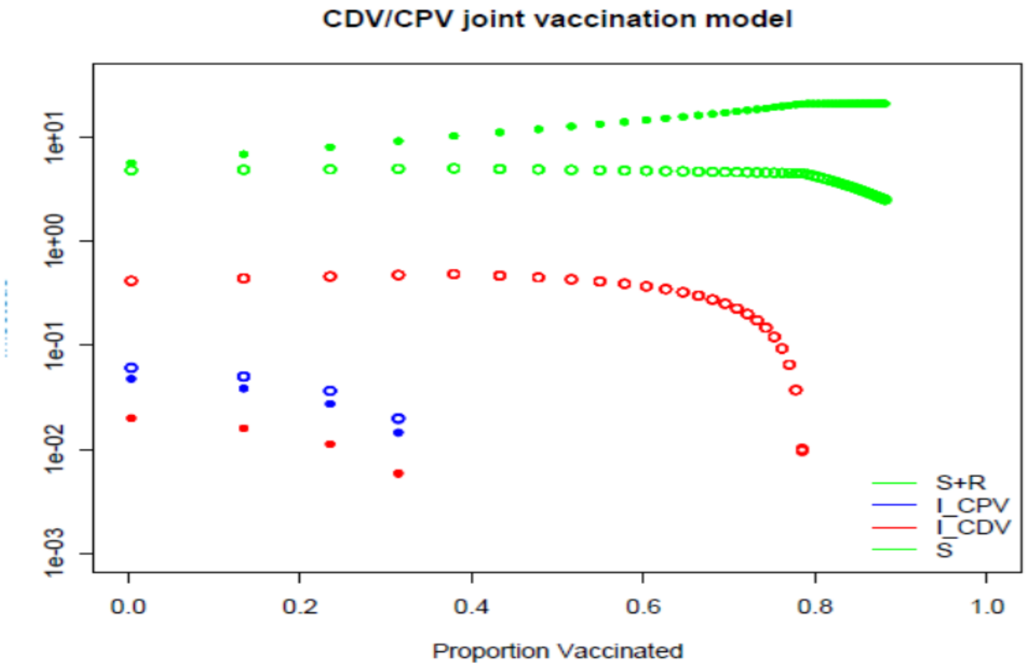


Vaccination interacts with sterilization

No Sterilization

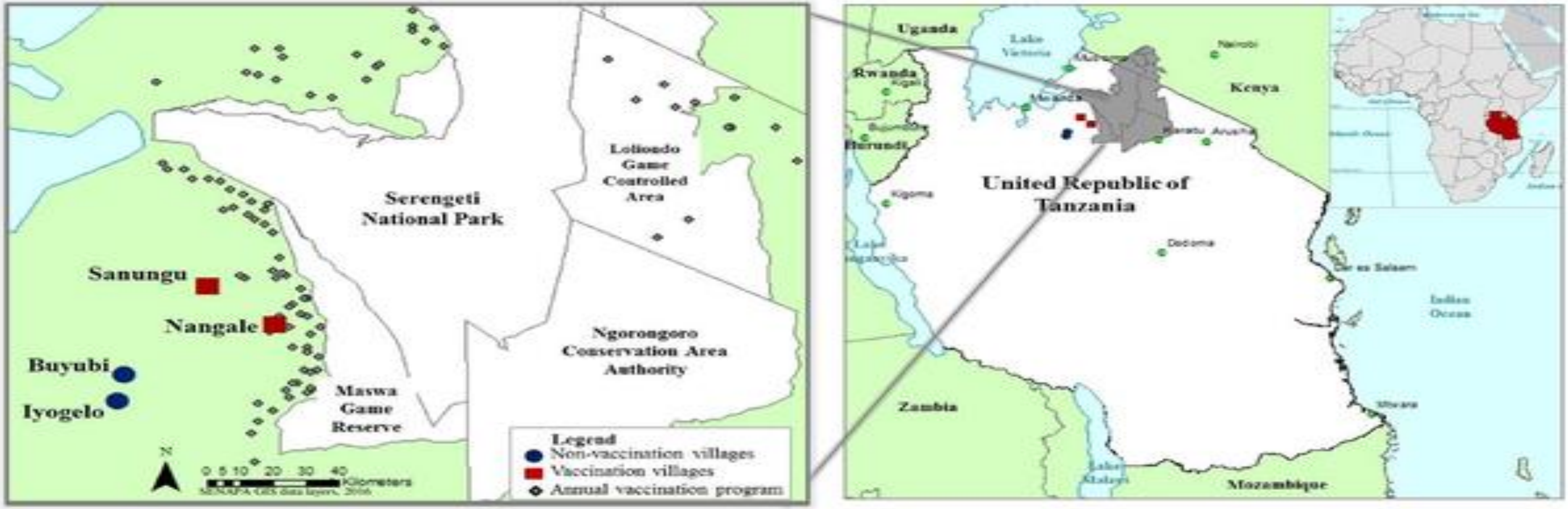


25% Sterilization



All of this will also apply to attempts to eradicate PPRV by vaccination; much, nu

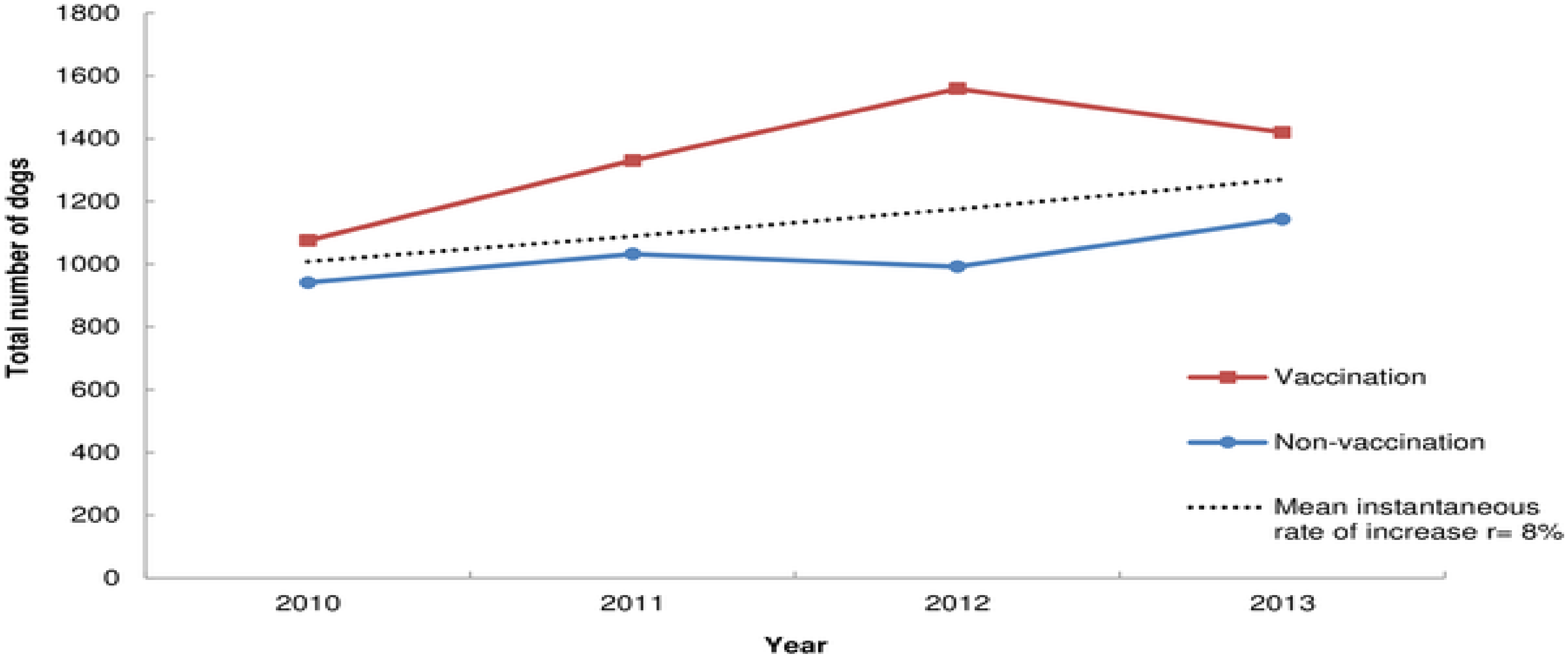
Fig 1. Location of study villages in relation to Serengeti National Park and existing dog rabies vaccination campaign.



Czupryna AM, Brown JS, Bigambo MA, Whelan CJ, Mehta SD, et al. (2016) Ecology and Demography of Free-Roaming Domestic Dogs in Rural Villages near Serengeti National Park in Tanzania. PLOS ONE 11(11): e0167092. doi:10.1371/journal.pone.0167092
http://journals.plos.org/plosone/article?id=10.1371/journal.pone.0167092

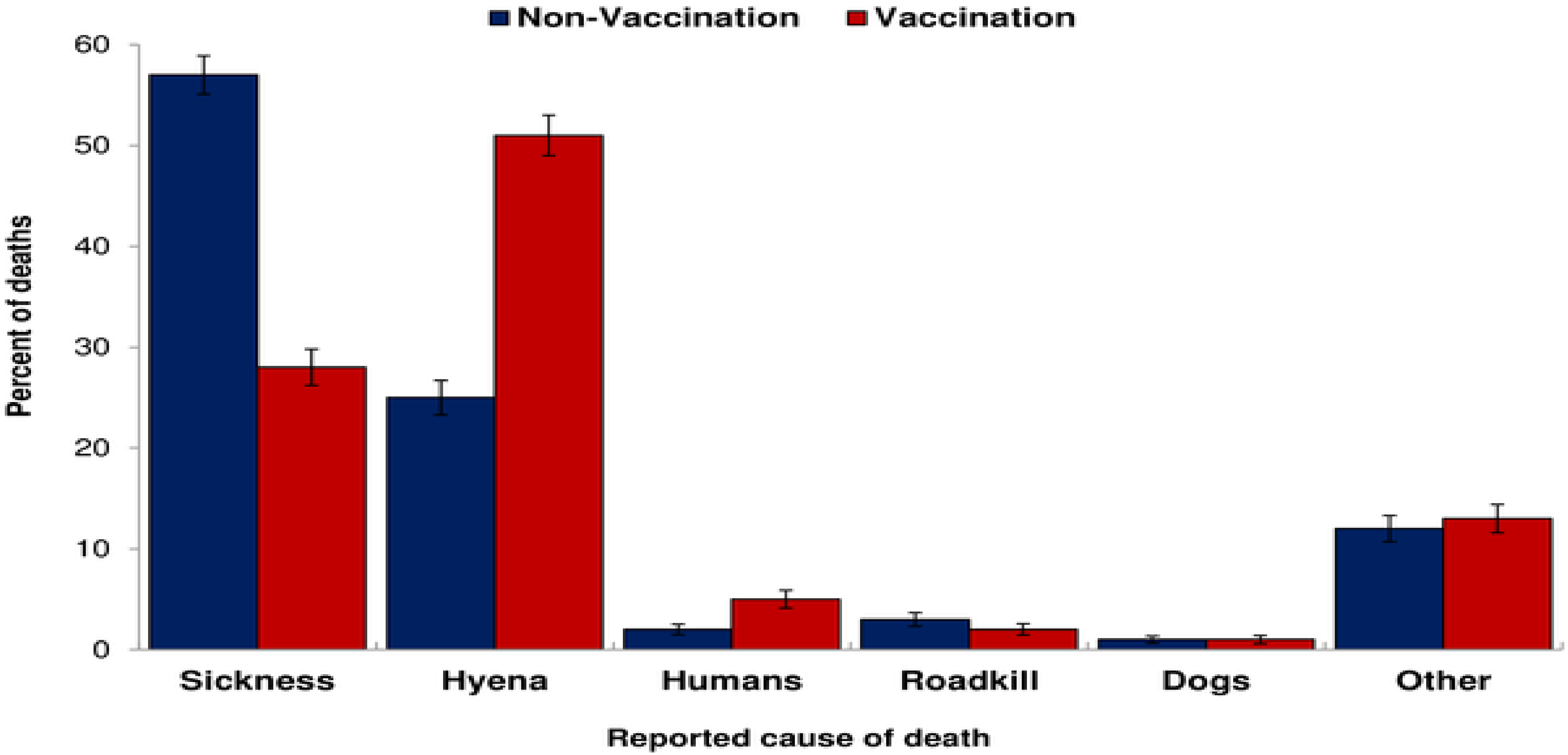
PLOS | ONE
TENTH ANNIVERSARY

Fig 8. Total number of dogs recorded each year of the study in each village census.



Czupryna AM, Brown JS, Bigambo MA, Whelan CJ, Mehta SD, et al. (2016) Ecology and Demography of Free-Roaming Domestic Dogs in Rural Villages near Serengeti National Park in Tanzania. PLOS ONE 11(11): e0167092. doi:10.1371/journal.pone.0167092
http://journals.plos.org/plosone/article?id=10.1371/journal.pone.0167092

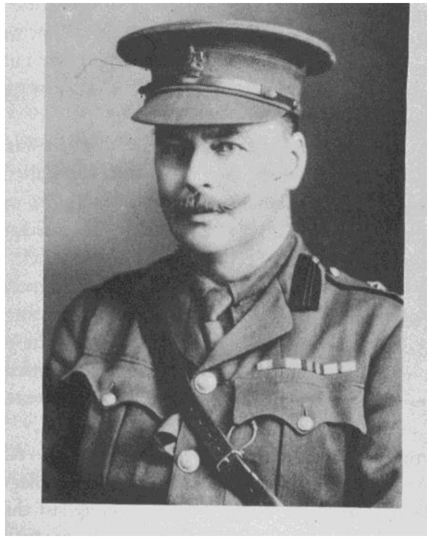
Fig 7. Percentage of owner-reported causes of death of dogs enrolled in the study.

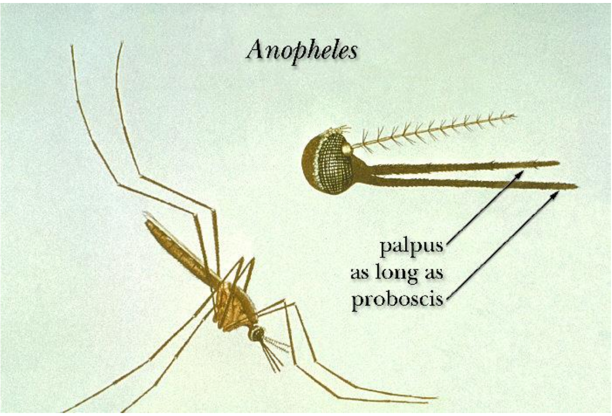


Czupryna AM, Brown JS, Bigambo MA, Whelan CJ, Mehta SD, et al. (2016) Ecology and Demography of Free-Roaming Domestic Dogs in Rural Villages near Serengeti National Park in Tanzania. PLOS ONE 11(11): e0167092. doi:10.1371/journal.pone.0167092
http://journals.plos.org/plosone/article?id=10.1371/journal.pone.0167092

Thank you!

Scarier and scarier, Next we have malaria!

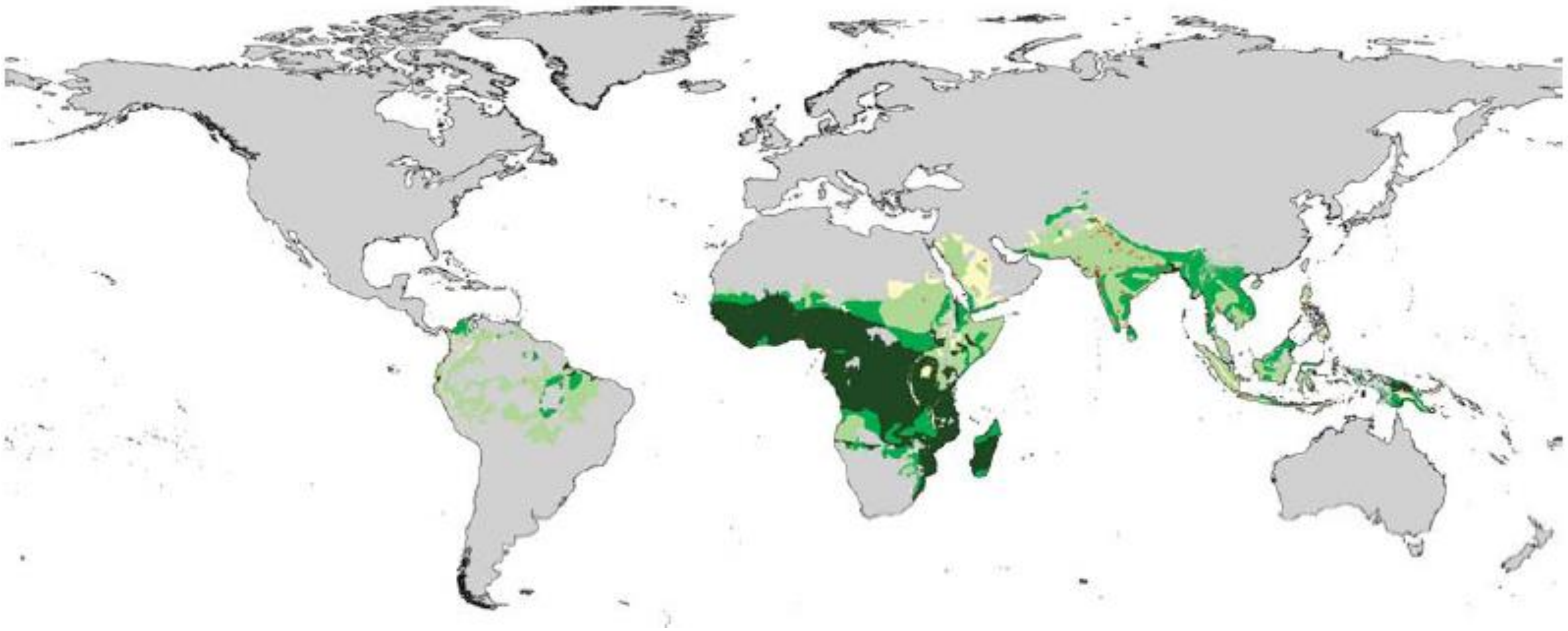




Useful Websites: http://www.rph.wa.gov.au/labs/haem/malaria/index.html
http://www.pitt.edu/~super1/lecture/lec0172/001.htm

Global burden of malaria

estimated 515 million episodes of clinical *Plasmodium falciparum* malaria in 2002 (Snow *et al* 2005).



Anopheles gambiae - major vector of malaria in Africa,

The burden of malaria in Africa

- Africa accounts for approximately 85% of malaria cases in the world
- estimated that African children have between 1.6 and 5.4 episodes of malarial fever each year.
- 3,000 deaths each day
- accounts for 1 in 5 of all childhood deaths in Africa.
- causes low birth weight, anaemia, epilepsy, and learning difficulties.
- imposes huge losses in economic productivity

Ronald Ross

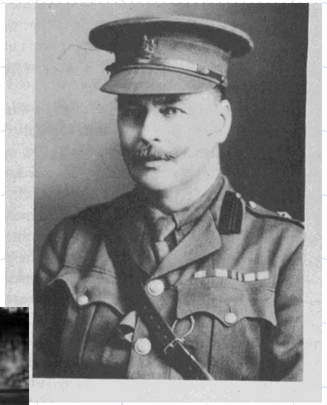
IN THE SMALL
LABORATORY
70 YARDS TO THE
SOUTH EAST OF THIS GATE
SURGEON MAJOR
RONALD ROSS I.M.S.
IN 1898 DISCOVERED
THE MANNER IN WHICH
MALARIA IS CONVEYED
BY MOSQUITOES.

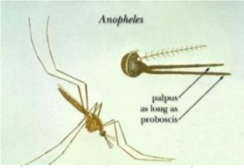
THIS DAY RELENTING GOD
HATH PLACED WITHIN MY HAND
A WONDROUS THING; AND GOD
BE PRAISED. AT HIS COMMAND,

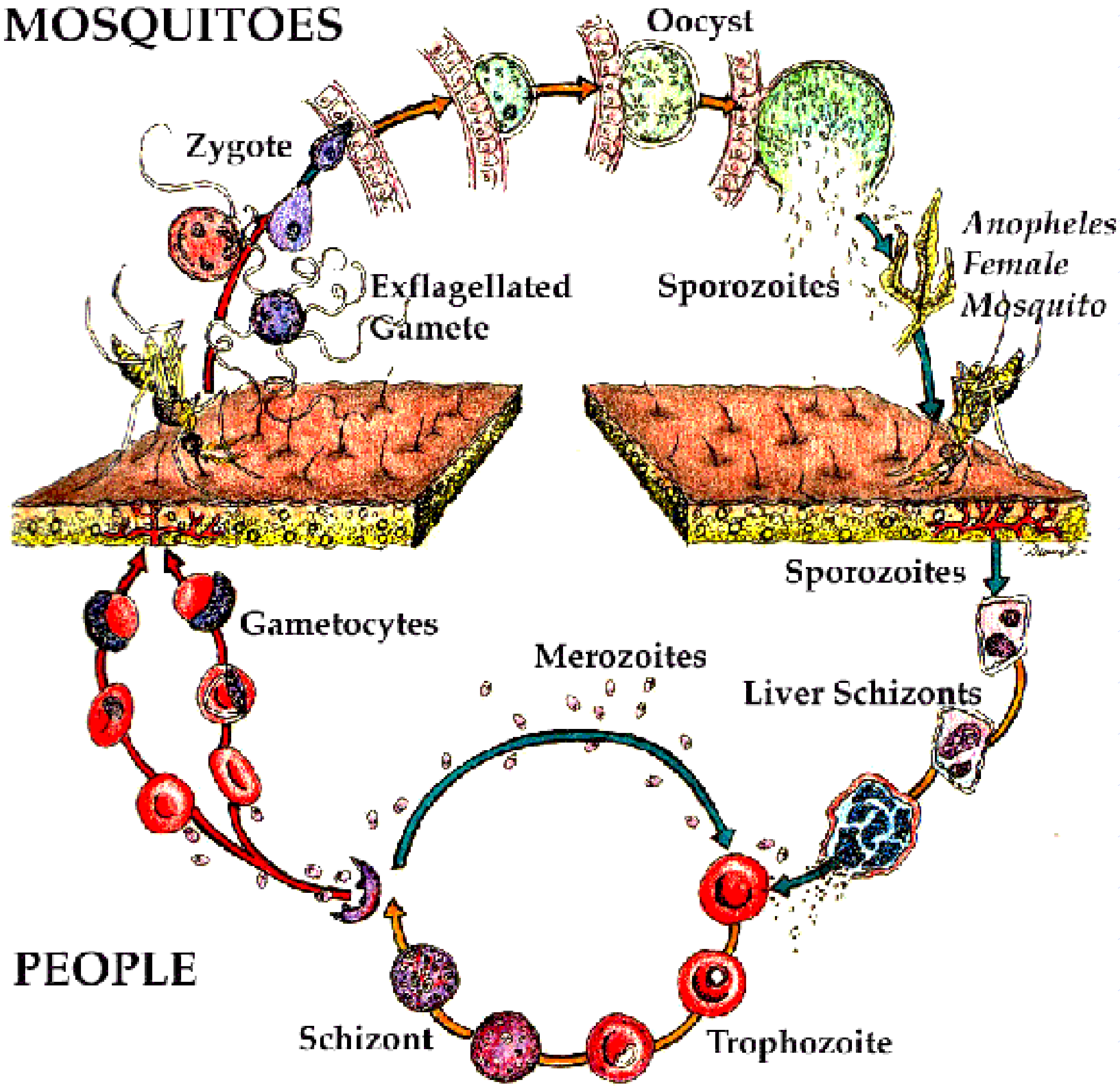
SEEKING HIS SECRET DEEDS
WITH TEARS AND TOILING BREATH,
I FIND THY CUNNING SEEDS,
O MILLION-MURDERING DEATH.

I KNOW THIS LITTLE THING
A MYRIAD MEN WILL SAVE.
O DEATH WHERE IS THY STING?
THY VICTORY, O GRAVE?

Ronald Ross







Sexual stages

Protozoan that uses mosquitoes as vectors.

Or is it the other way around?

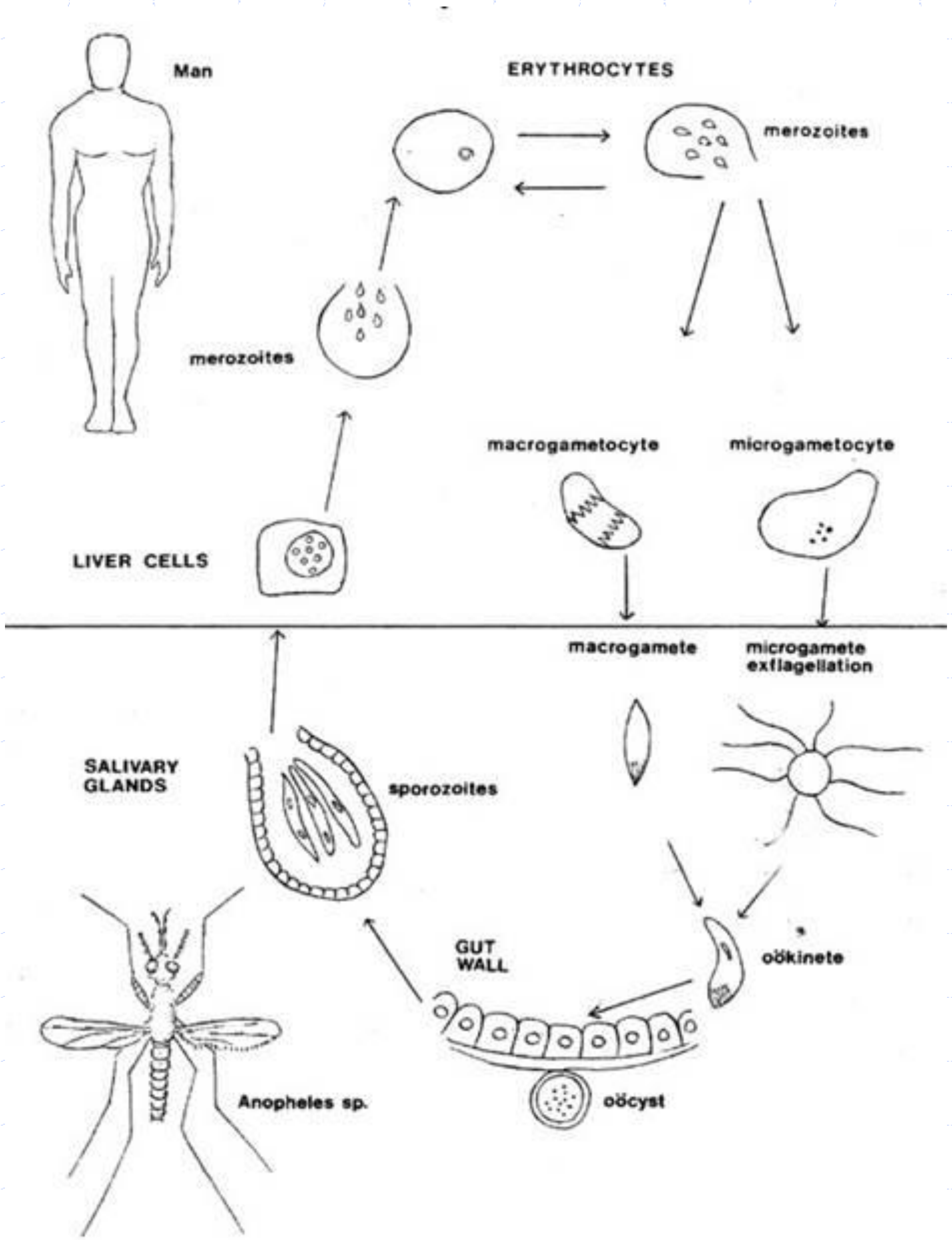
Asexual stages

Back to malaria

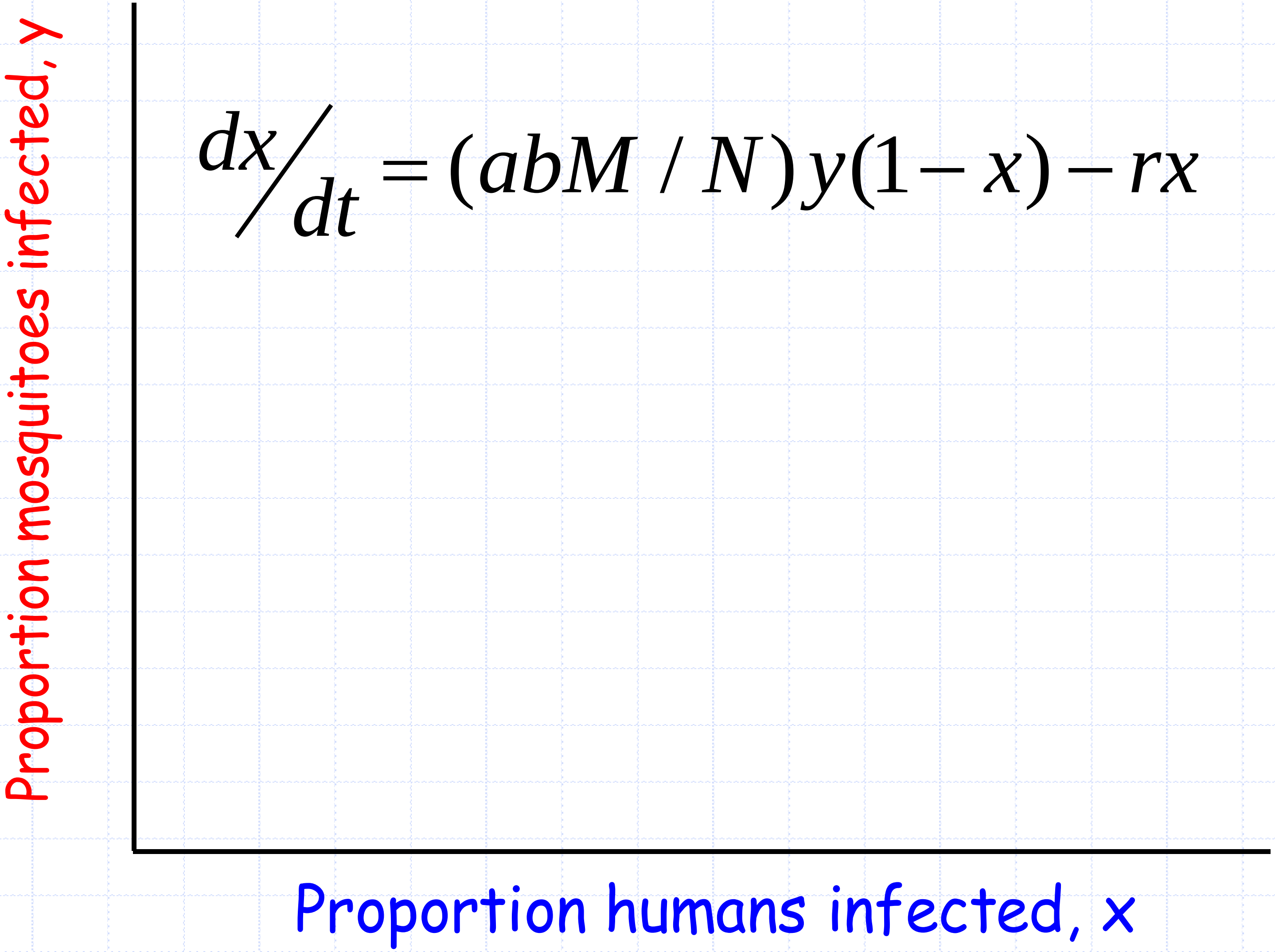
Life cycle..

- proportion of the human population infected
- proportion of the female mosquito population infected

Model by Ross and McDonald (1916-1957)

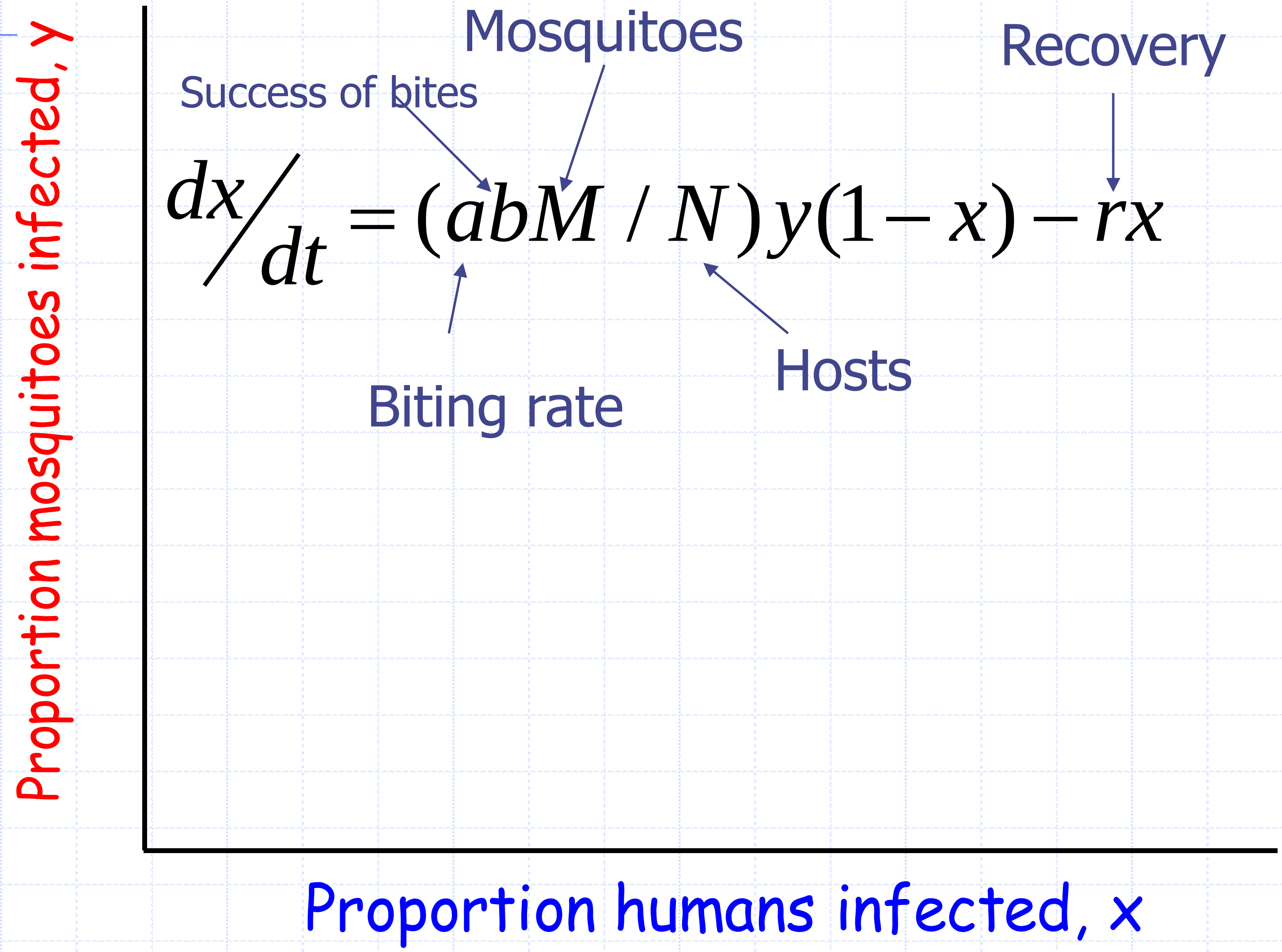


Population dynamics of malaria (Aron & May, 1982).



$$dx/dt = (abM / N)y(1 - x) - rx$$

Population dynamics of malaria (Aron & May, 1982).



Mosquitoes

Recovery

Success of bites

$$\frac{dx}{dt} = (abM / N)\,y(1 - x) - rx$$

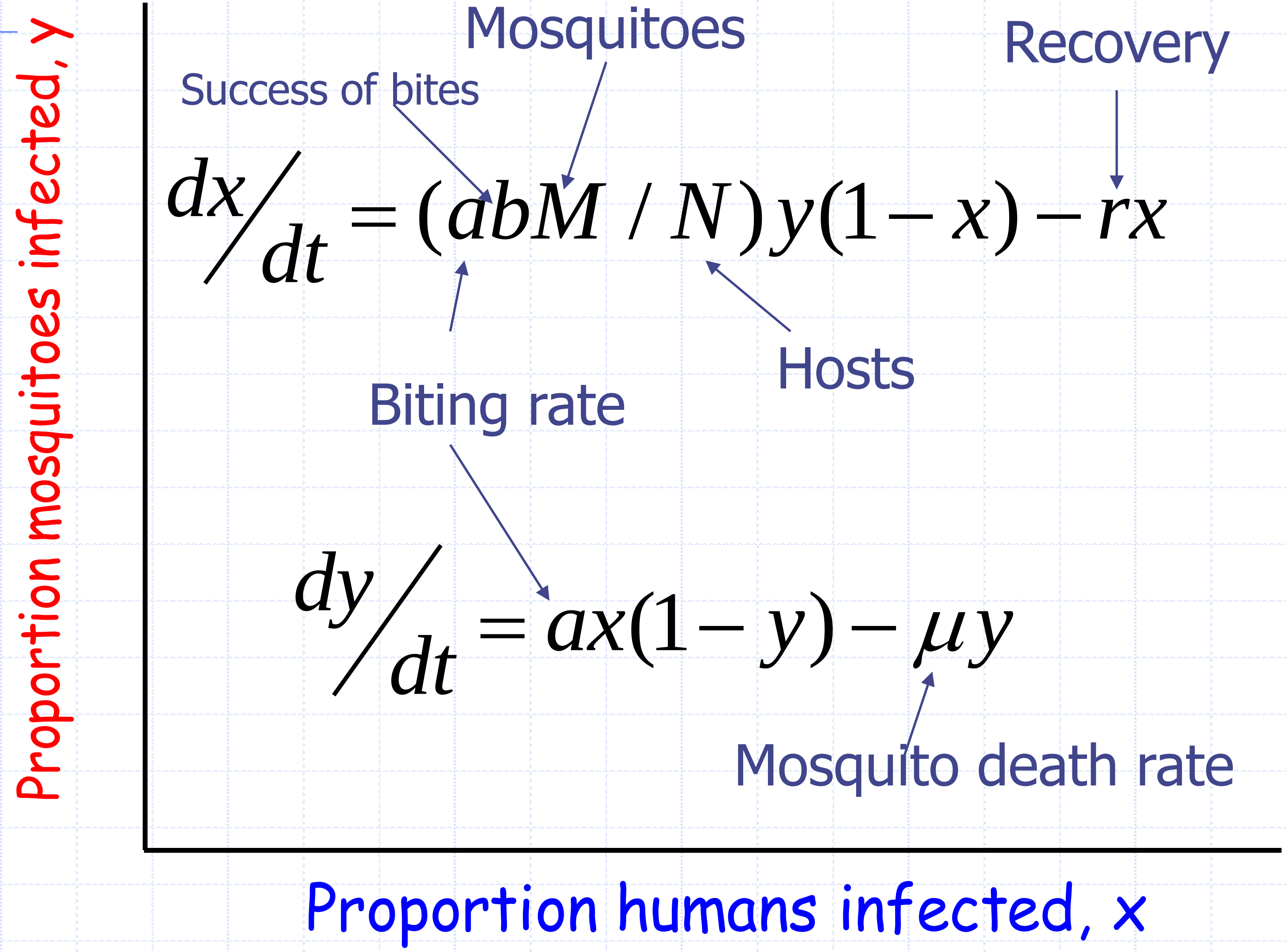
Biting rate

Hosts

Proportion mosquitoes infected, y

Proportion humans infected, x

Population dynamics of malaria (Aron & May, 1982).



Success of bites

Mosquitoes

Recovery

$$dx/dt = (abM/N)\,y(1-x) - rx$$

Biting rate

Hosts

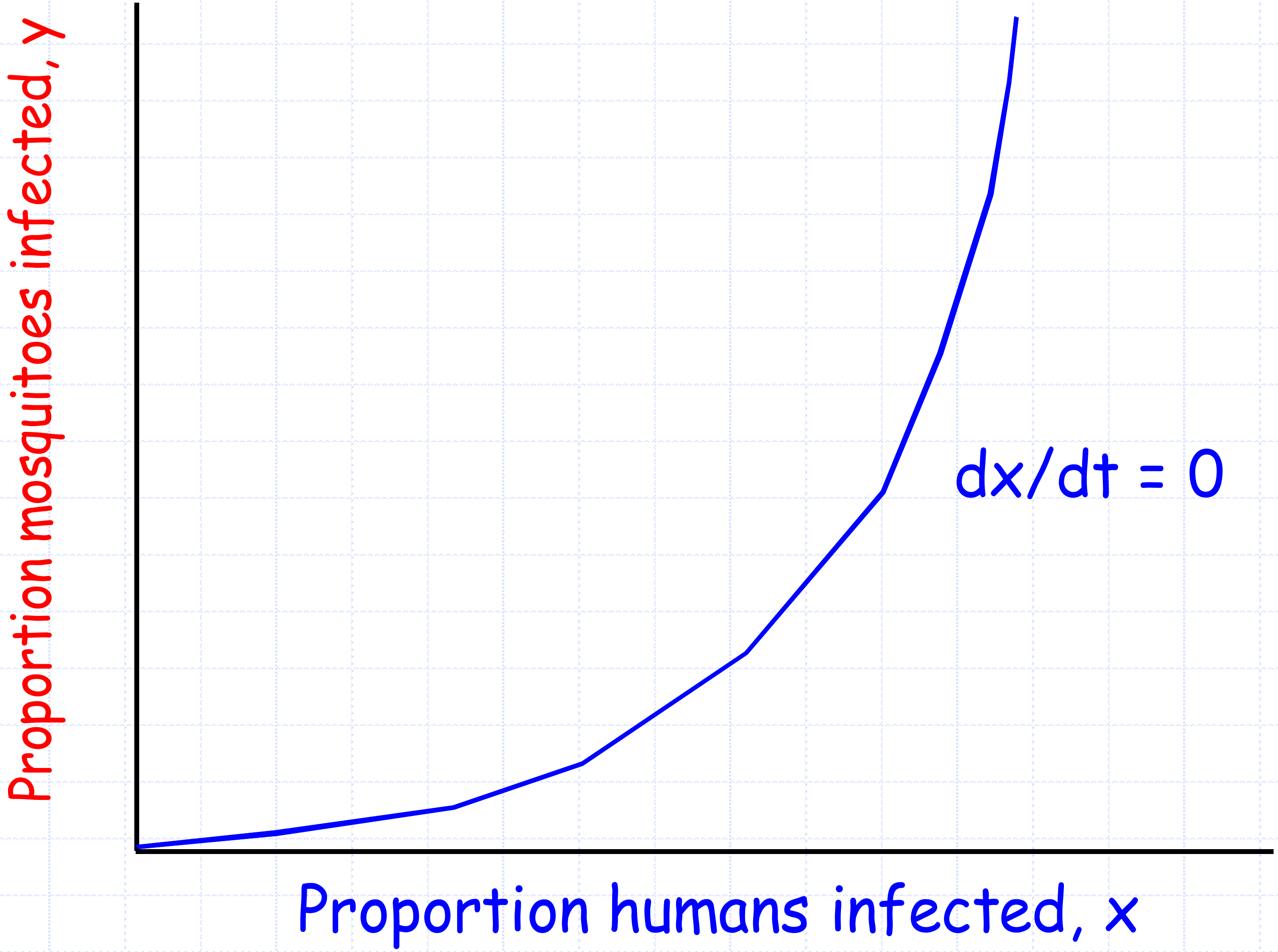
$$dy/dt = ax(1-y) - \mu y$$

Mosquito death rate

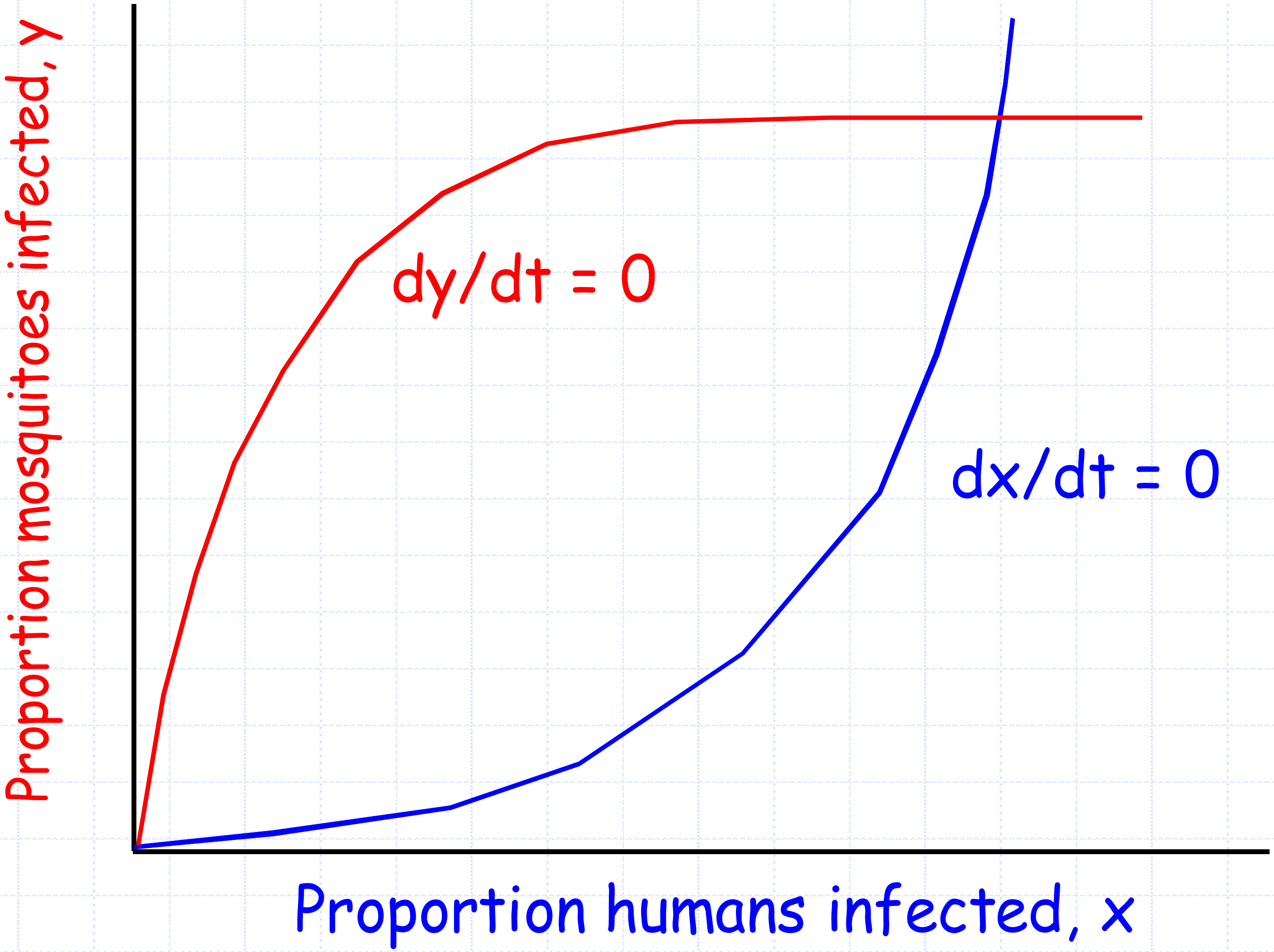
Proportion mosquitoes infected, y

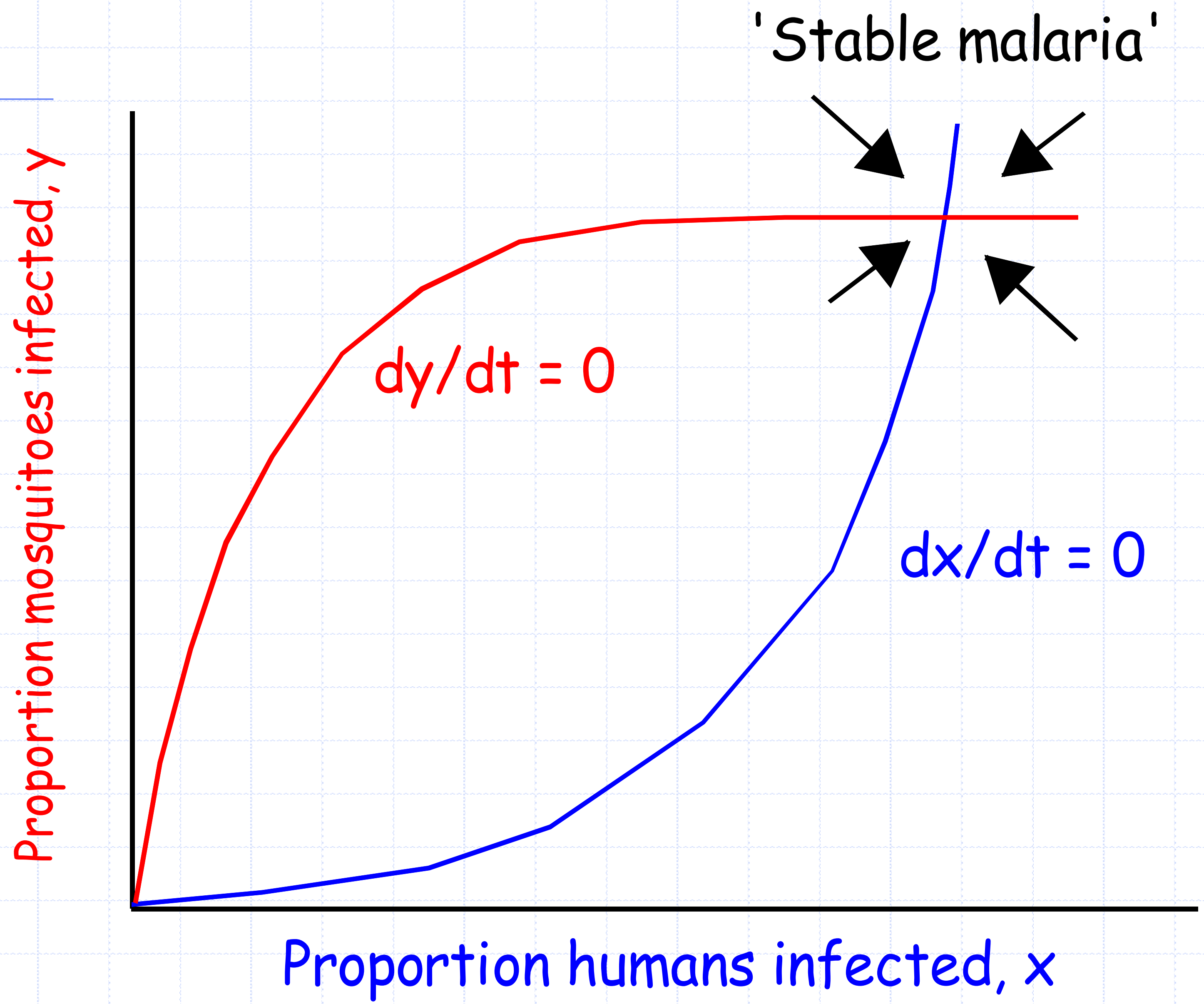
Proportion humans infected, x

Plot ‘zero-growth’ isoclines



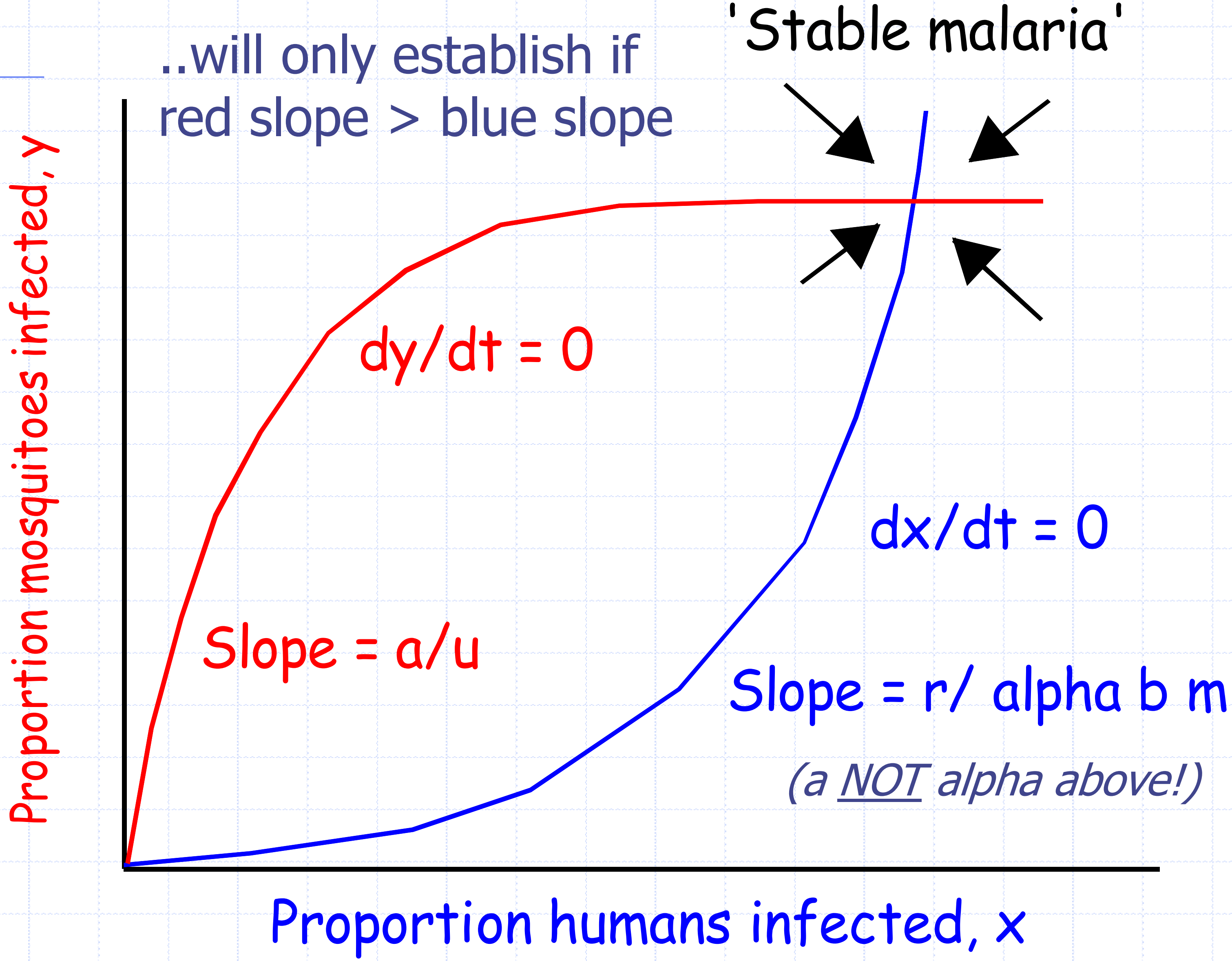
….add the second zero-growth isocline….




'Stable malaria'
dy/dt = 0
dx/dt = 0
Proportion mosquitoes infected, y
Proportion humans infected, x

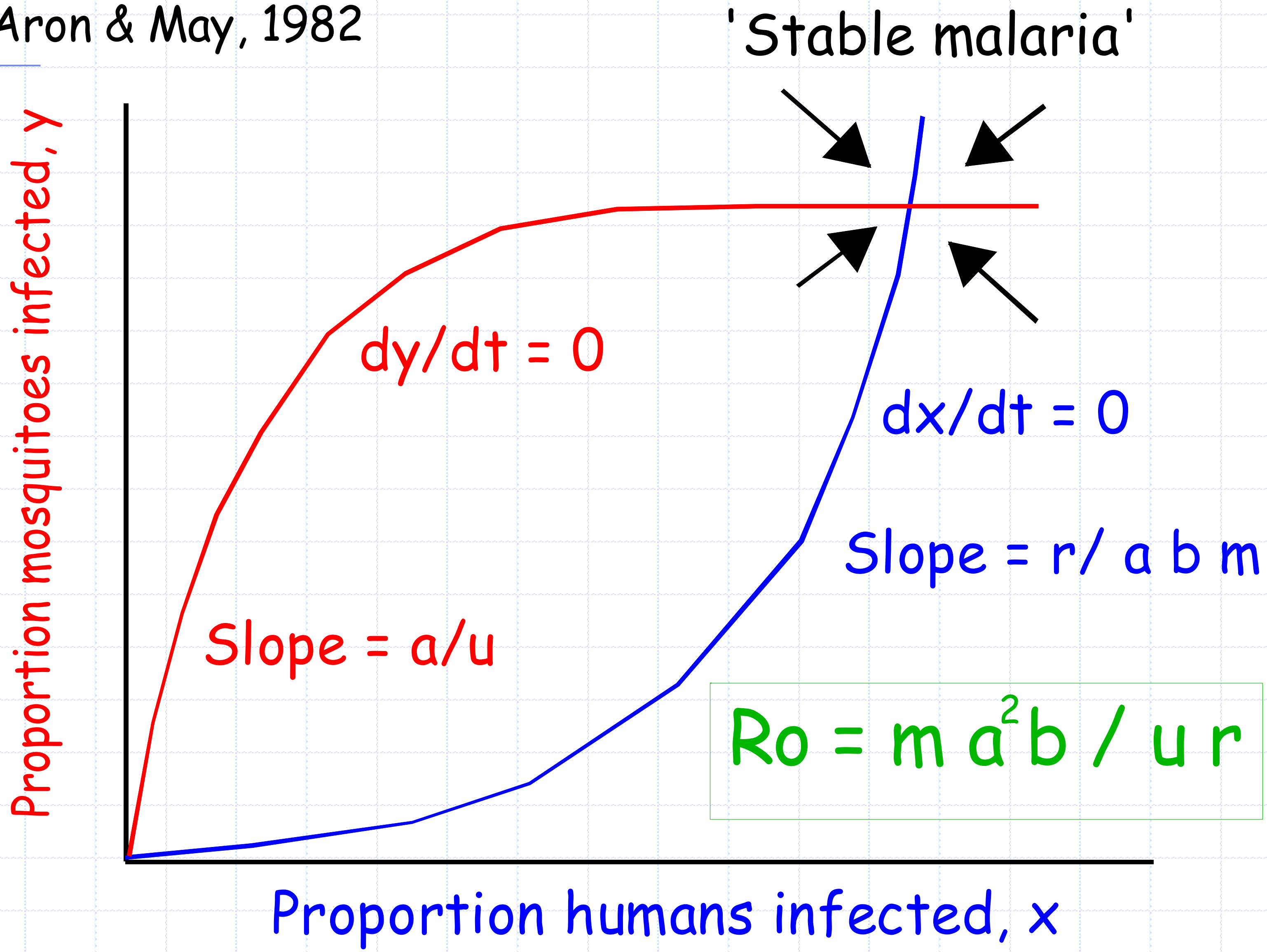
Almost ‘Ro’……..!

..will only establish if
red slope > blue slope



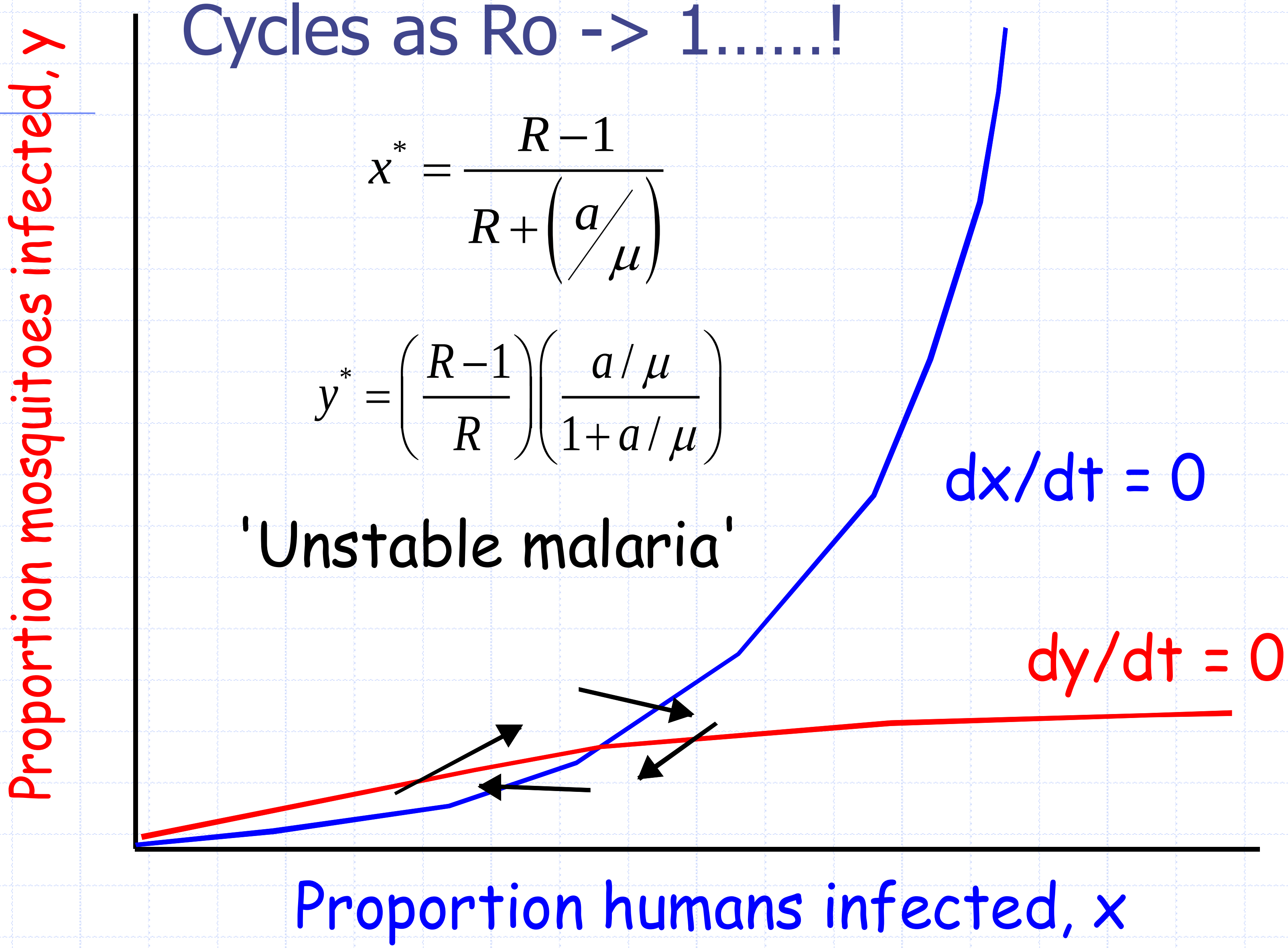
Wow! Another Ro!

After Aron & May, 1982



$$Ro = m\,a^2 b\,/\,u\,r$$

Will tend to give epidemic Cycles as Ro -> 1……!



$$x^* = \frac{R-1}{R+\left(a/\mu\right)}$$

$$y^* = \left(\frac{R-1}{R}\right)\left(\frac{a/\mu}{1+a/\mu}\right)$$

'Unstable malaria'

Could also obtain this by WAIFW - next generation matrix…………

$$\begin{array}{ccc} & \text{Human} & \text{Mosquito} \\ \text{Human} & 0 & \dfrac{a}{\mu} \\ \text{Mosquito} & \dfrac{abm}{r} & 0 \end{array}$$

Dominant eigenvalue
Gives an expression
for Ro….

$$R_0 = \sqrt{{a^2bm}\big/{\mu r}}$$

Could also obtain this by WAIFW – next generation matrix…………

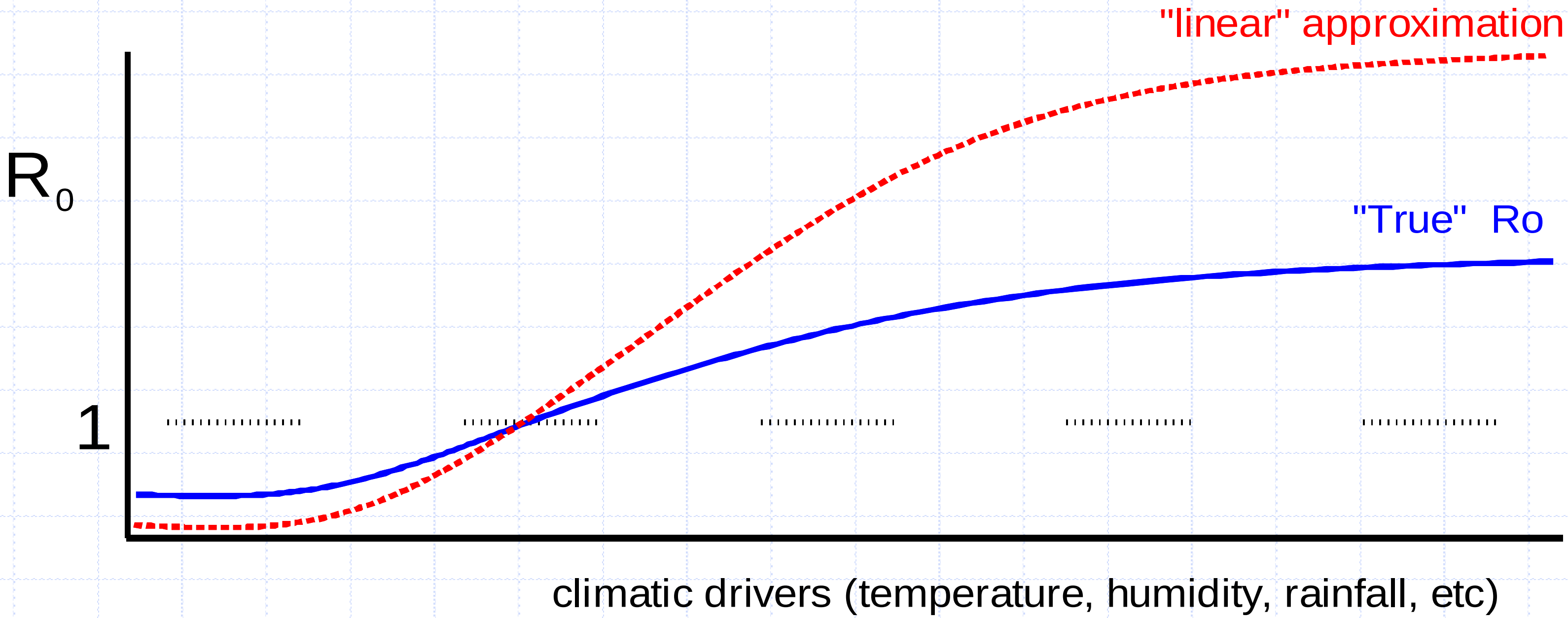
$$\begin{array}{ccc} & \text{Human} & \text{Mosquito} \\ \text{Human} & 0 & \dfrac{a}{\mu} \\ \text{Mosquito} & \dfrac{abm}{r} & 0 \end{array}$$

Dominant eigenvalue
Gives an expression
for Ro….

$$R_0 = \sqrt{a^2bm \big/ \mu r}$$

The square root term makes a difference – particularly when Ro >>1 !

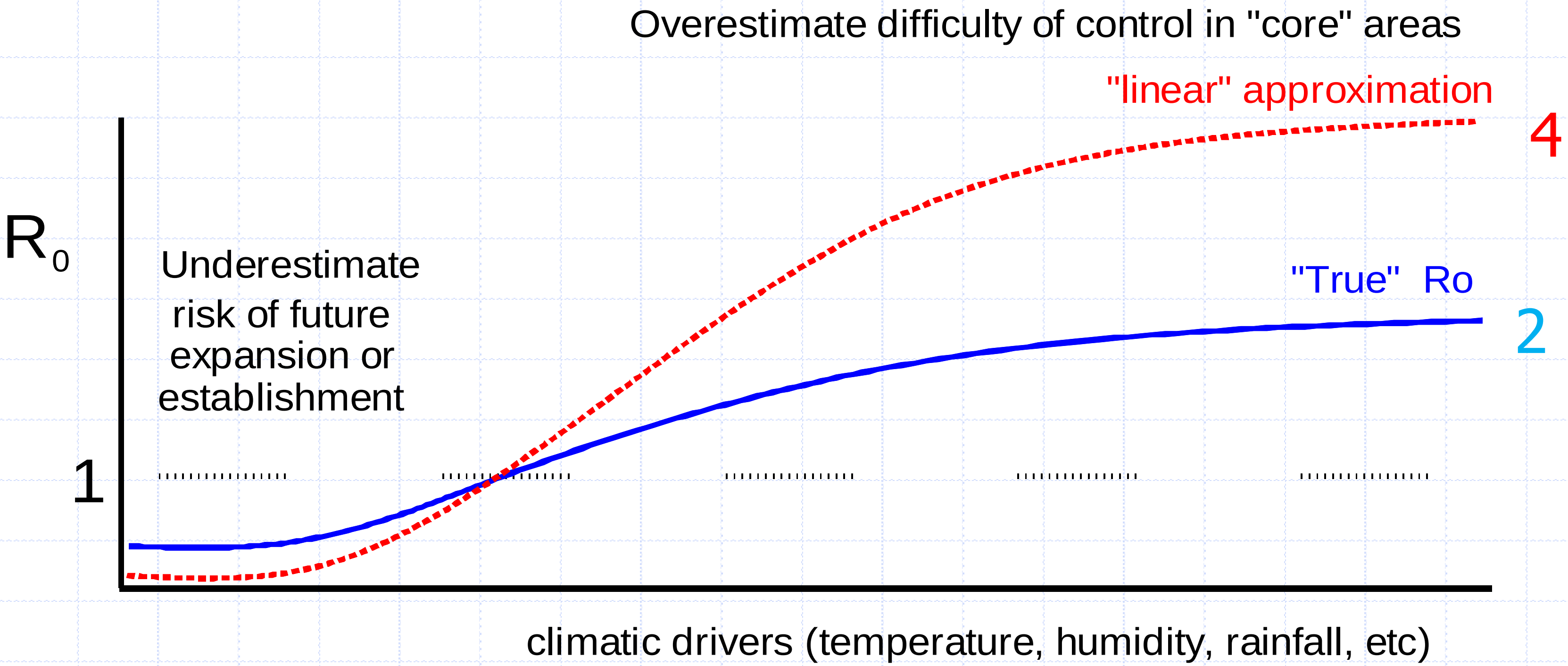
Consider maps of malaria risk…..



Maps of malaria risk will look great because of fancy computer GIS graphics

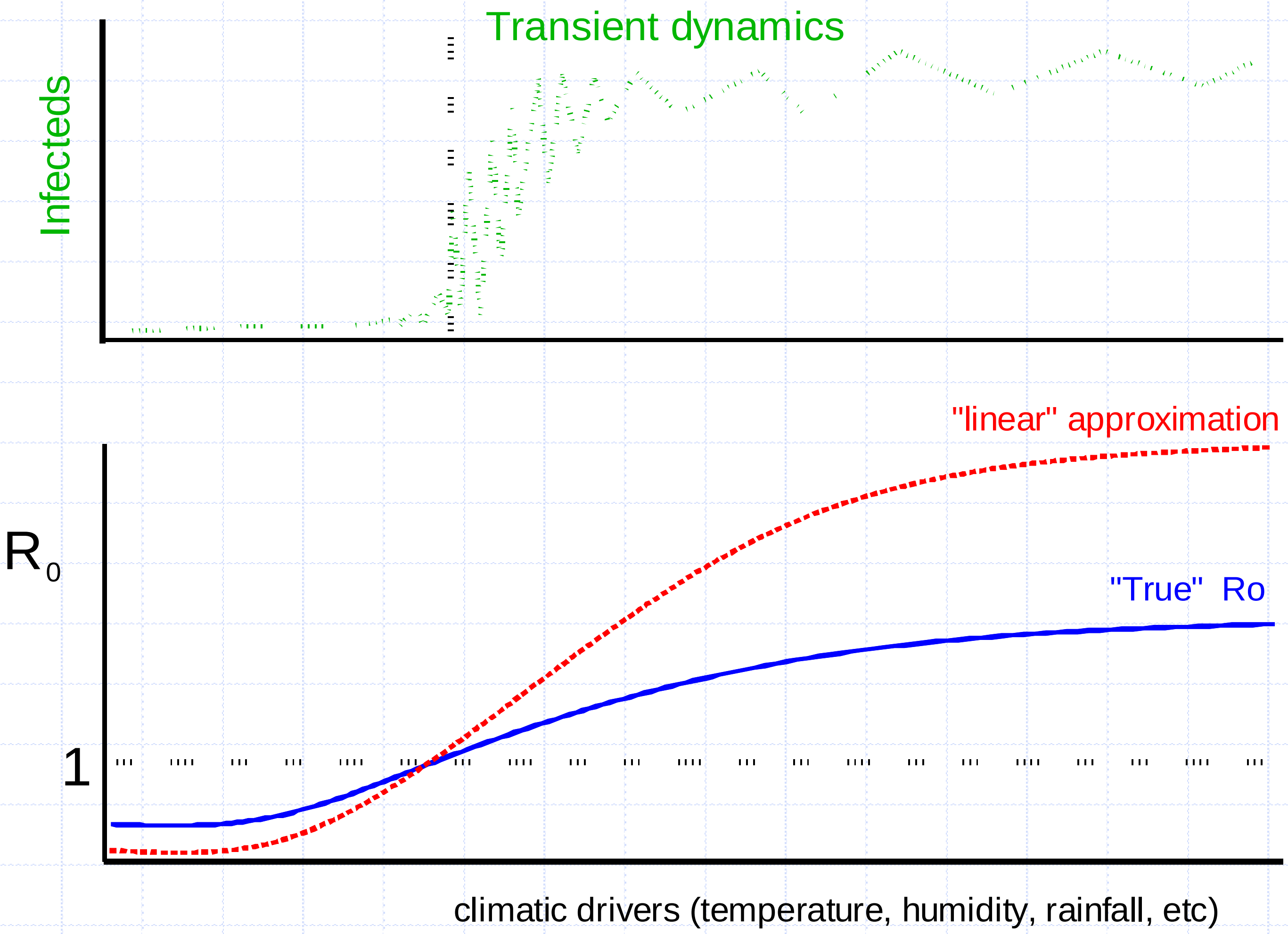
BUT, the linear approximation for Ro will cause them to distort risk.

Risk maps for vector borne disease



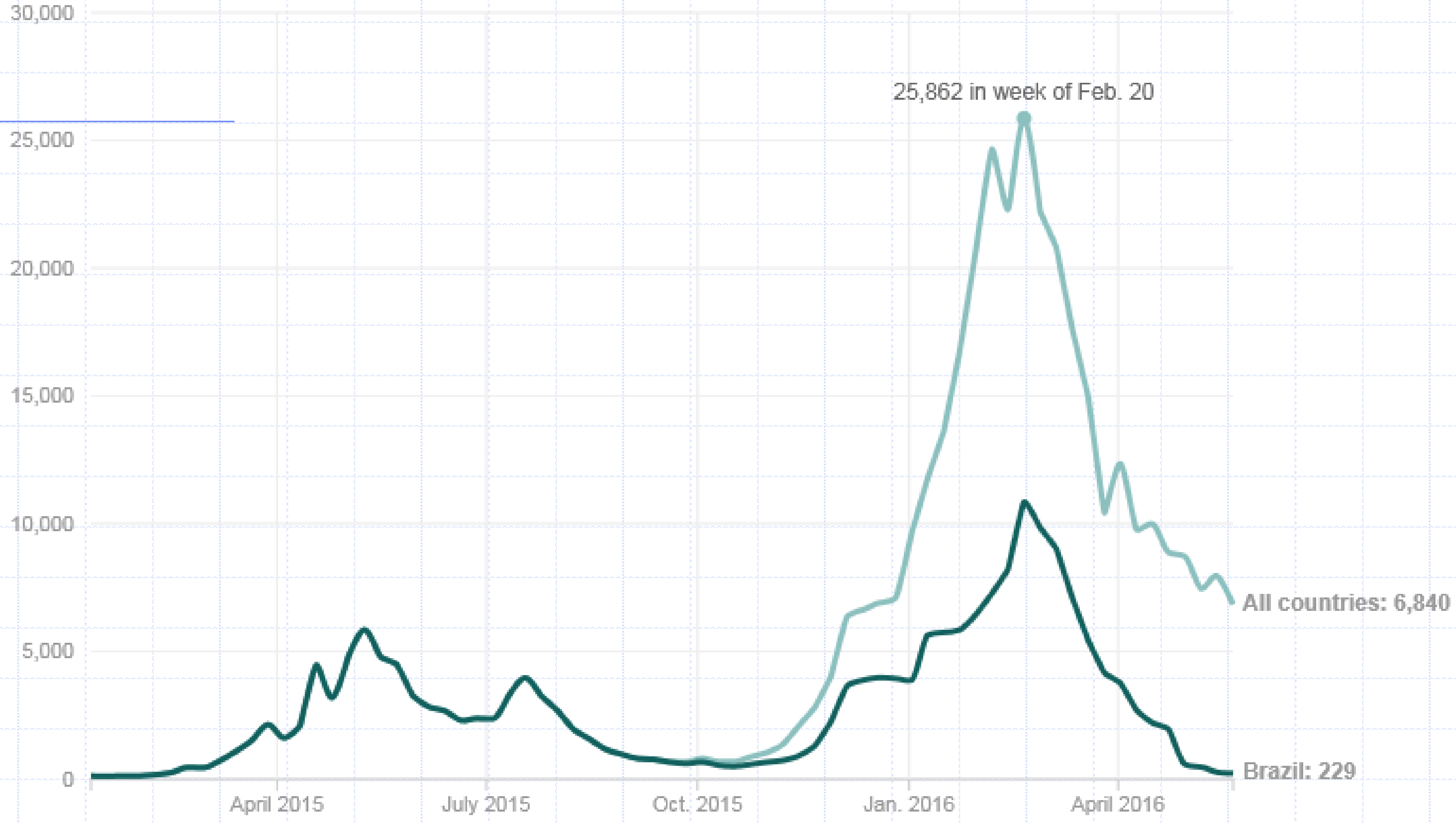
One (of many) problems that underlie these ‘risk’ maps (many thanks to Jeremy Farrar for discussions about this).

System is most unstable when Ro close to unity…range boundaries will be fuzzy

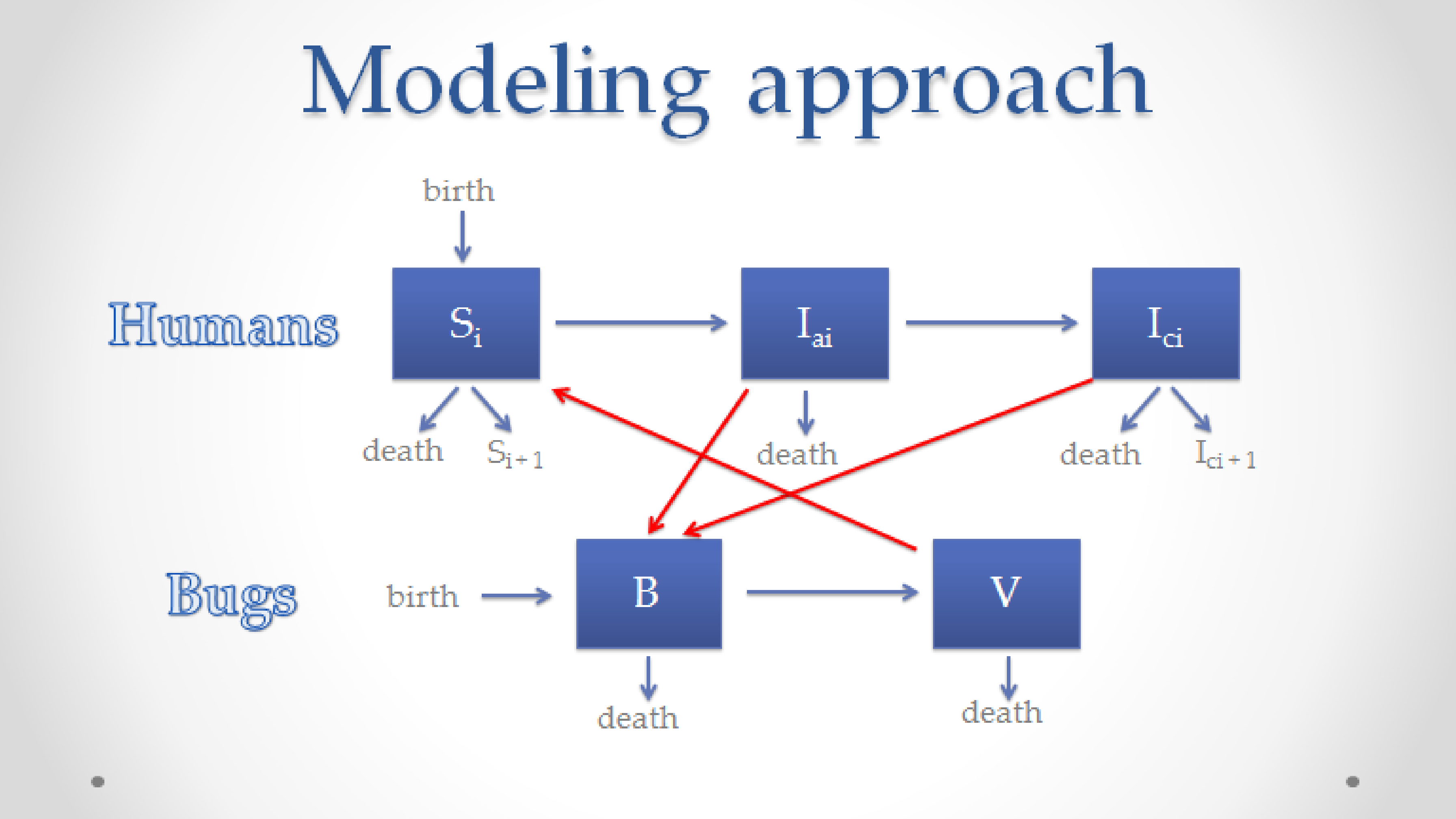


Zika virus in Brazil

Cases of Zika : Global and in Brazil



Age-structured vector model

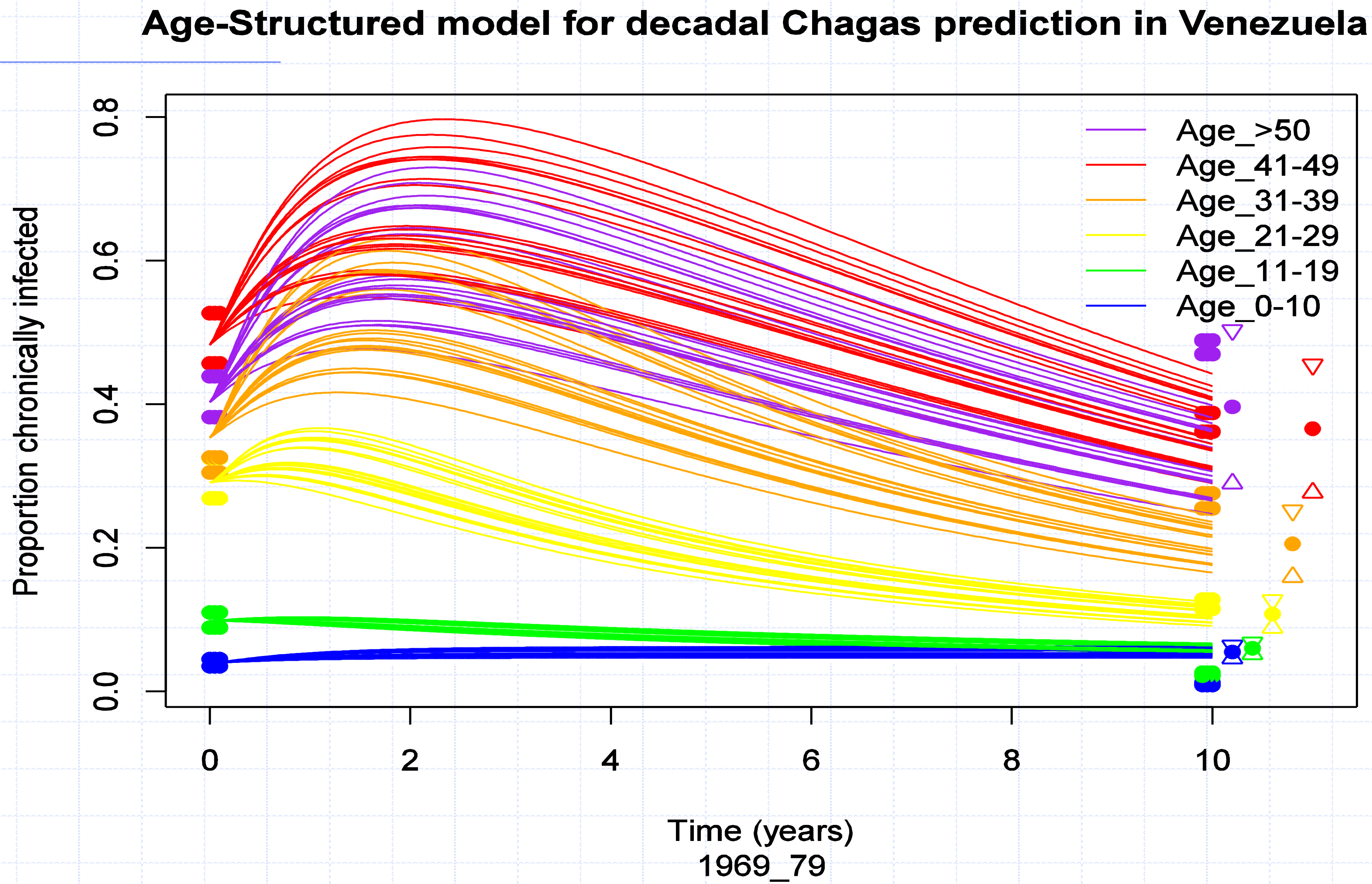


Modeling approach

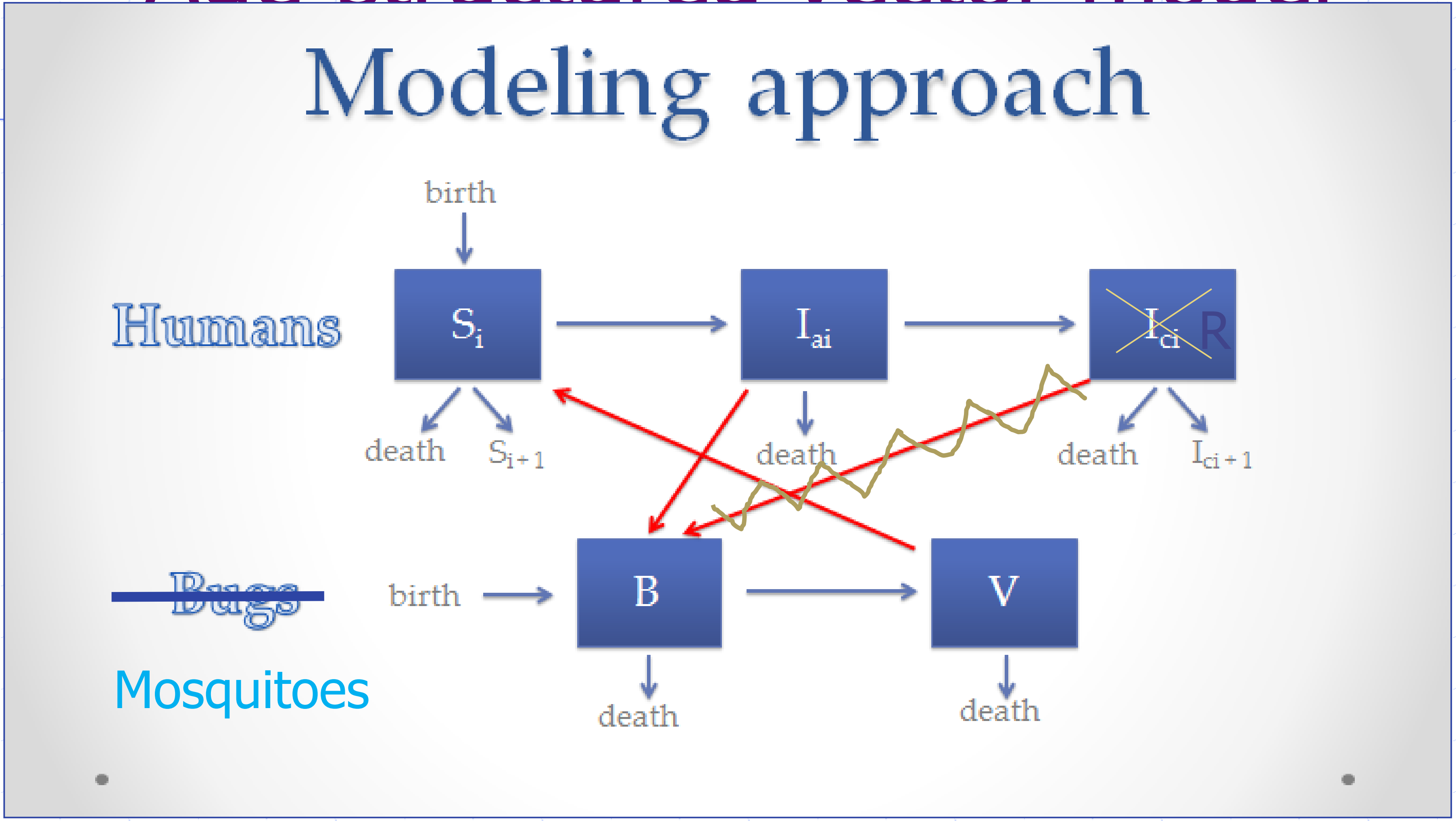
Six age classes and focus on decline in vectors and houses infected over 10

Peterson and Dobson, Vectors and Health; GATES NTD Modelling cons

Forward projection 1969 to 1979



Age-structured vector model



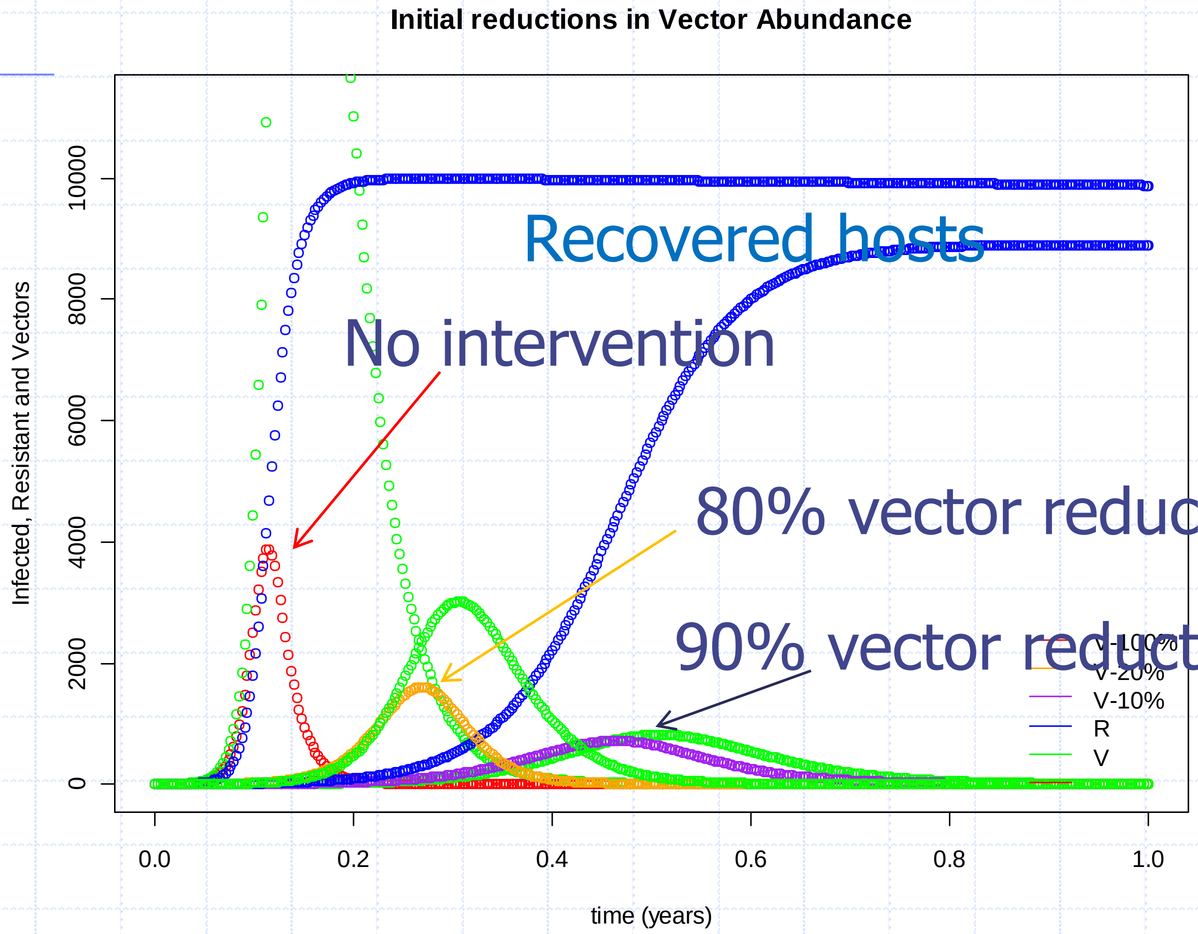
Modeling approach

Six age classes and switch bugs to mosquitoes

Change chronics to recovered/resistants and speed up dynamics by around

Dynamics are driven by build up of herd immunity in human population

Infected and recovered and resistant hosts



Recovered hosts

No intervention

80% vector reduction

90% vector reduction

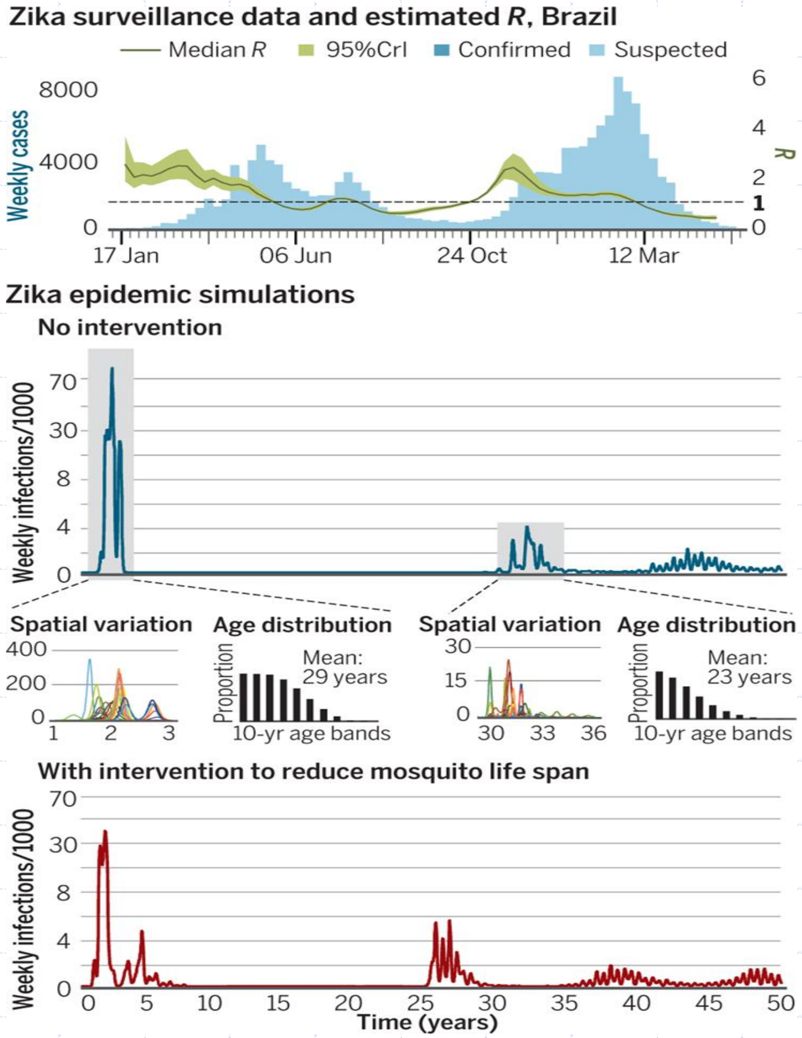
Infected mosquitoes in green

Ooopss!!
Reduction in vector population has almost no impact on the total number of infected hosts!!

Zika transmission dynamics.

Dam!!

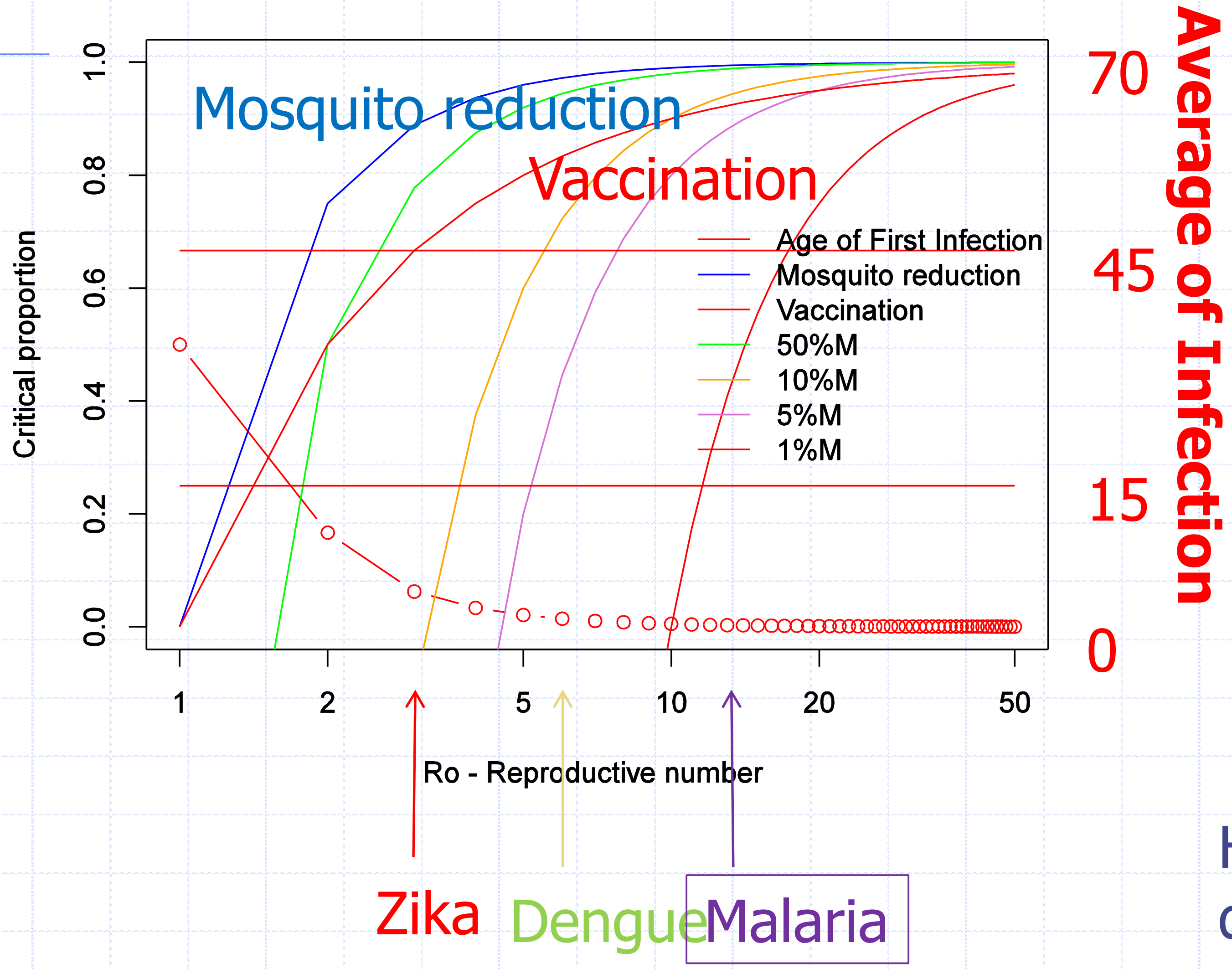
Pipped at the post!!



Neil M. Ferguson et al. Science 2016;353:353-354

Published by AAAS

Generalized control strategy for any vector borne disease



This is key for Zika!!

Herd immunity is very different for VBDs!!!

Dobson, Alonso, Peterson and Pascual (in need of a home..)

WAIFW matrices and R_0

Who Acquires Infection From Whom

(Schenzle, 1984; Anderson and May, 1985)

$$W = \begin{bmatrix} \beta_{ii} & \beta_{ij} & \beta_{ik} \\ \beta_{ji} & \beta_{jj} & \beta_{jk} \\ \beta_{ki} & \beta_{kj} & \beta_{kk} \end{bmatrix}$$

→

R_0 = basic reproductive number of the pathogen

(Diekmann *et al.*, 1990)

Four special cases

1) All2All

$$\begin{bmatrix} \beta_{AA} & \beta_{AB} & \beta_{AC} & \beta_{AD} \\ \beta_{BA} & \beta_{BB} & \beta_{BC} & \beta_{BD} \\ \beta_{CA} & \beta_{CB} & \beta_{CC} & \beta_{CD} \\ \beta_{DA} & \beta_{DB} & \beta_{DC} & \beta_{DD} \end{bmatrix}$$

2) Asym2All

$$\begin{bmatrix} \beta_{AA} & 0 & 0 & 0 \\ \beta_{BA} & \beta_{BB} & 0 & 0 \\ \beta_{CA} & \beta_{CB} & \beta_{CC} & \beta_{CD} \\ \beta_{DA} & \beta_{DB} & \beta_{DC} & \beta_{DD} \end{bmatrix}$$

$$\begin{bmatrix} \beta_{AA} & 0 & 0 & 0 \\ \beta_{BA} & \beta_{BB} & 0 & 0 \\ 0 & \beta_{CB} & \beta_{CC} & 0 \\ 0 & 0 & \beta_{DC} & \beta_{DD} \end{bmatrix}$$

3) SpecChain

$$\begin{bmatrix} 0 & 0 & 0 & \beta_{AV} \\ 0 & 0 & 0 & \beta_{BV} \\ 0 & 0 & 0 & \beta_{C\nu} \\ \beta_{VA} & \beta_{VB} & \beta_{VC} & \nu(?) \end{bmatrix}$$

4) All2Vector

Ticks versus mosquitoes I

Basic matrix expression for ‘next generation’ of infections
whenever an infected host is introduced into the population

$$\begin{array}{cc} & \text{Transmission host to vector} \\ \begin{array}{c}\text{Transmission vector} \\ \text{To host}\end{array} & M = \begin{pmatrix} 0 & \dfrac{M\beta B}{\mu(B+nC)} & \dfrac{M\beta nC}{\mu(B+nC)} \\ \dfrac{bB}{(B+nC)} & 0 & 0 \\ \dfrac{cnC}{(B+nC)} & 0 & 0 \end{pmatrix} \end{array}$$

The basic reproductive number for the pathogen, Ro, is then given by the dominant eigenvalue of this matrix

Key assumption – M is vector abundance – independent of hosts for mosquitoes, dependent for ticks.

Ro for insect vectored

This can readily be shown to generalize for n-species of hosts to

$$R_0 = \frac{1}{\sum_{i=1}^{n} a_i A_i} \sqrt{\frac{M\beta}{\mu} \left(\sum_{i=1}^{n} v_i (a_i A_i)^2\right)}$$

Here a_i is the relative attraction of species i to mosquitoes,
A_i is the abundance of species i and v_i is it's 'viability as a host ($1/(\alpha_i+d_i+\delta_i)$).

Ro for tick vectored

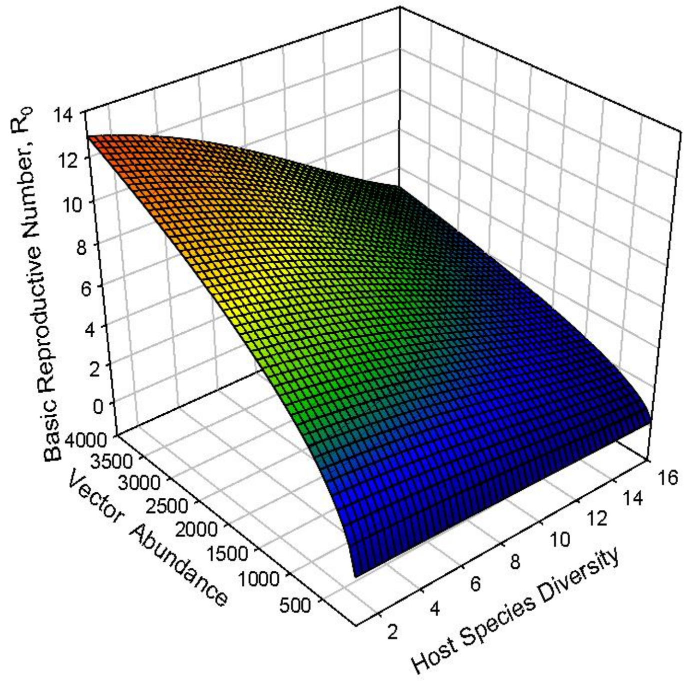
This needs to now vary with host abundance…

This can readily be shown to generalize for n-species of hosts to

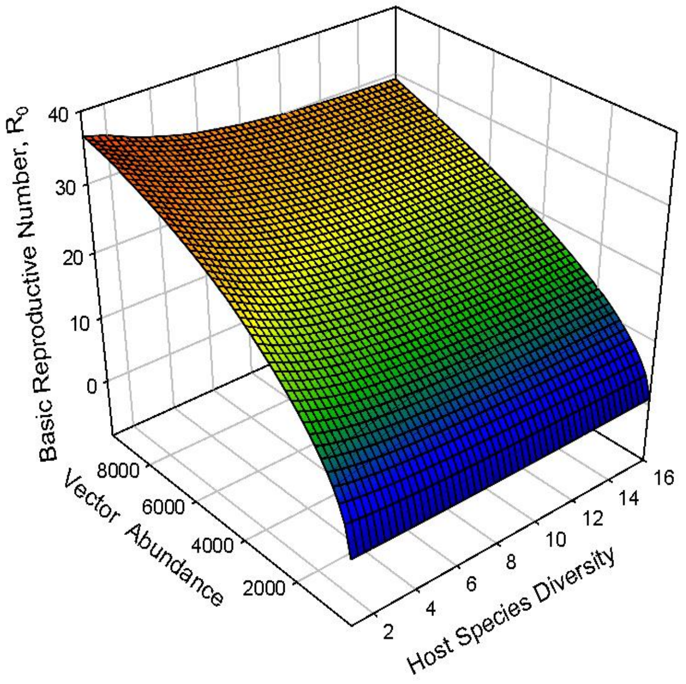
$$R_0 = \frac{1}{\sum_{i=1}^{n} a_i A_i} \sqrt{\frac{\enclose{circle}{M}\beta}{\mu} \left(\sum_{i=1}^{n} v_i (a_i A_i)^2 \right)}$$

Here a_i is the relative attraction of species i to mosquitoes,
A_i is the abundance of species i and v_i is it's 'viability as a host $(1/(\alpha_i+d_i+\delta_i)$).

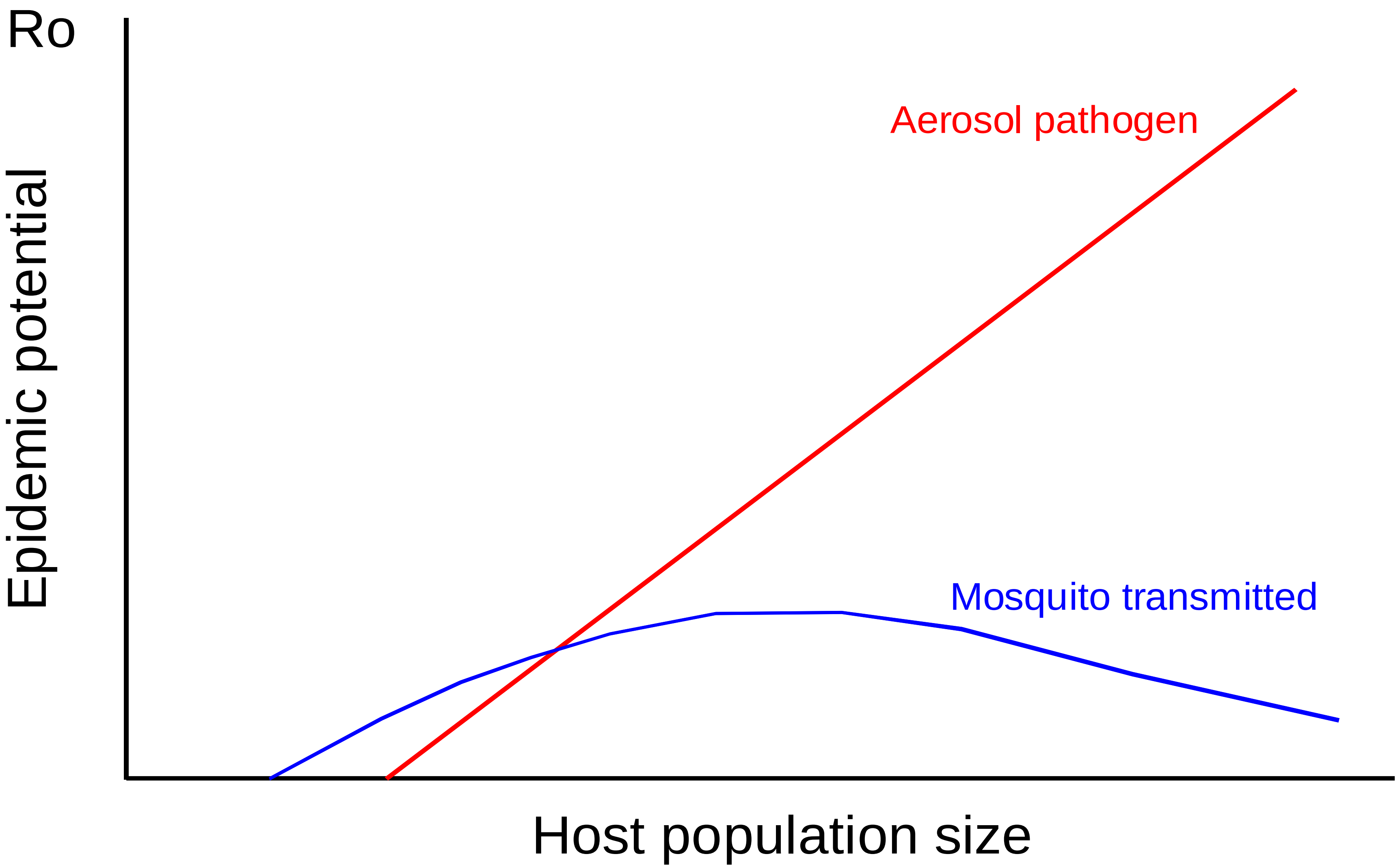
Mosquitoes versus ticks

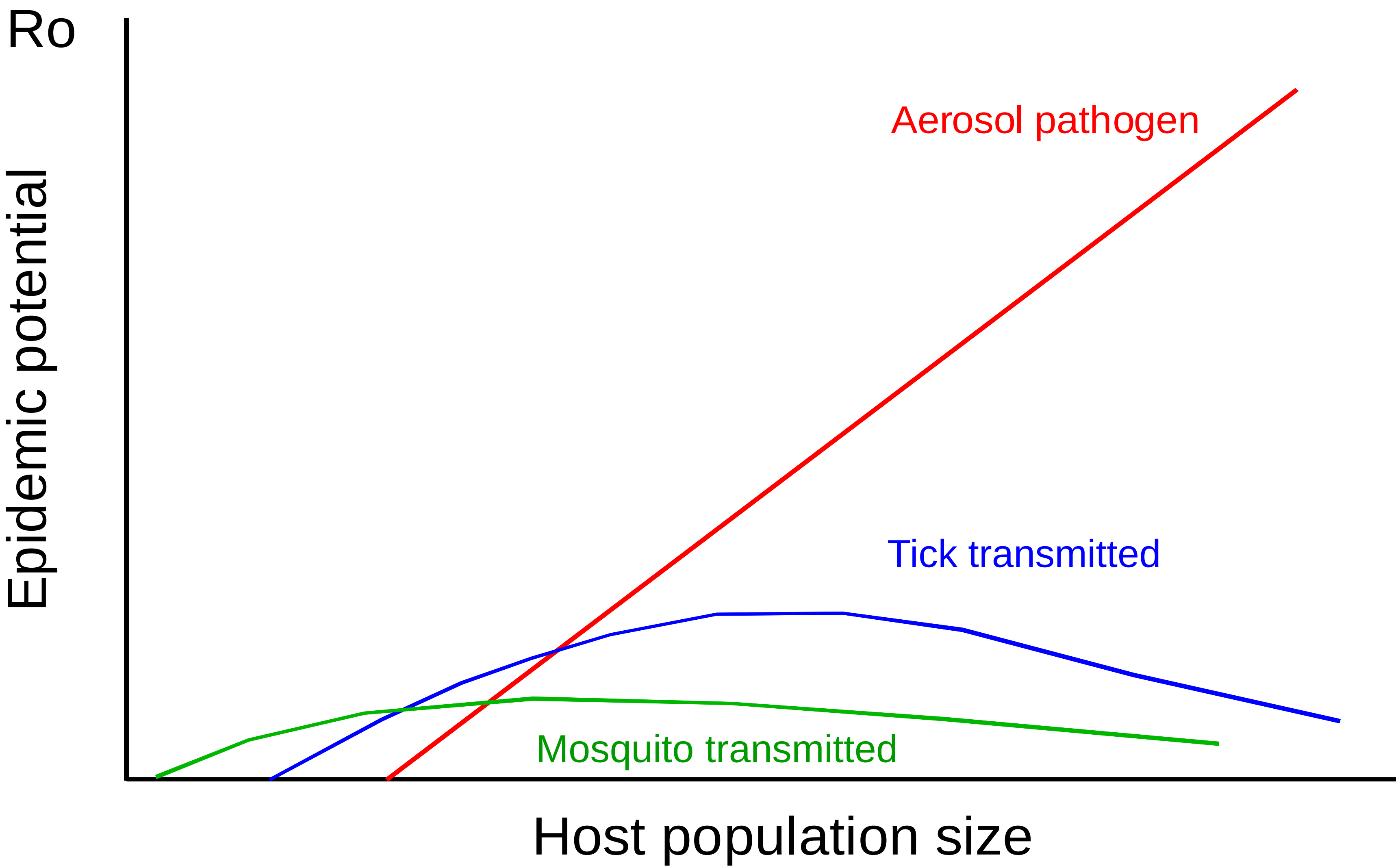


Mosquito Vector Abundance
and Dilution Effects

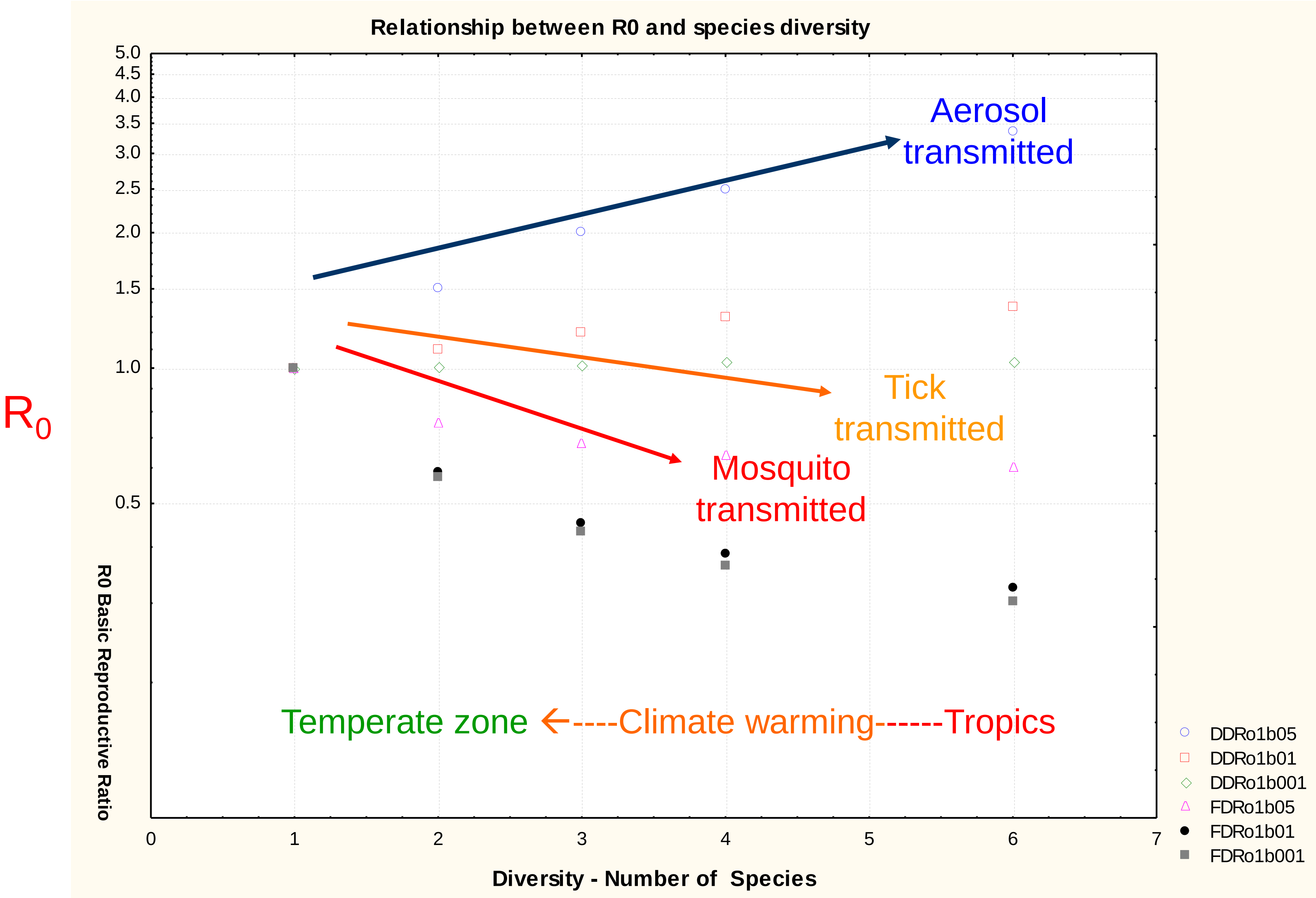


Tick Vector Abundance
and Dilution Effects


Ro
Epidemic potential
Aerosol pathogen
Mosquito transmitted
Host population size


Ro
Epidemic potential
Aerosol pathogen
Tick transmitted
Mosquito transmitted
Host population size

The relationship between biodiversity and potential for disease outbreaks



Lyme disease: the `dilution effect'

R. Ostfeld *et al.*

Lyme disease

Reservoir host –
white-footed mouse

Vector - ticks

In my back yard in Princeton……
………also on campus, in Institute woods

Ecology, 82(3), 2001, pp. 609–619

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

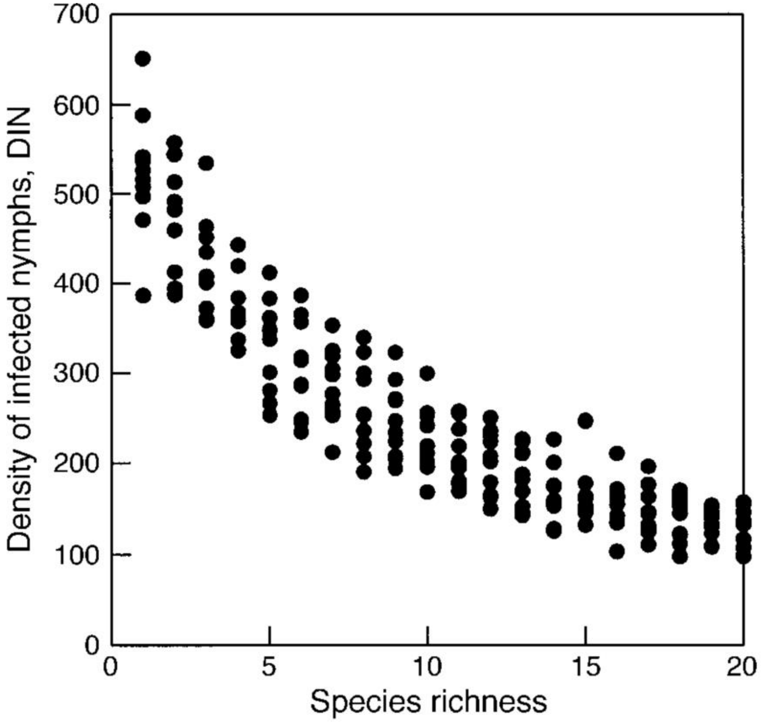
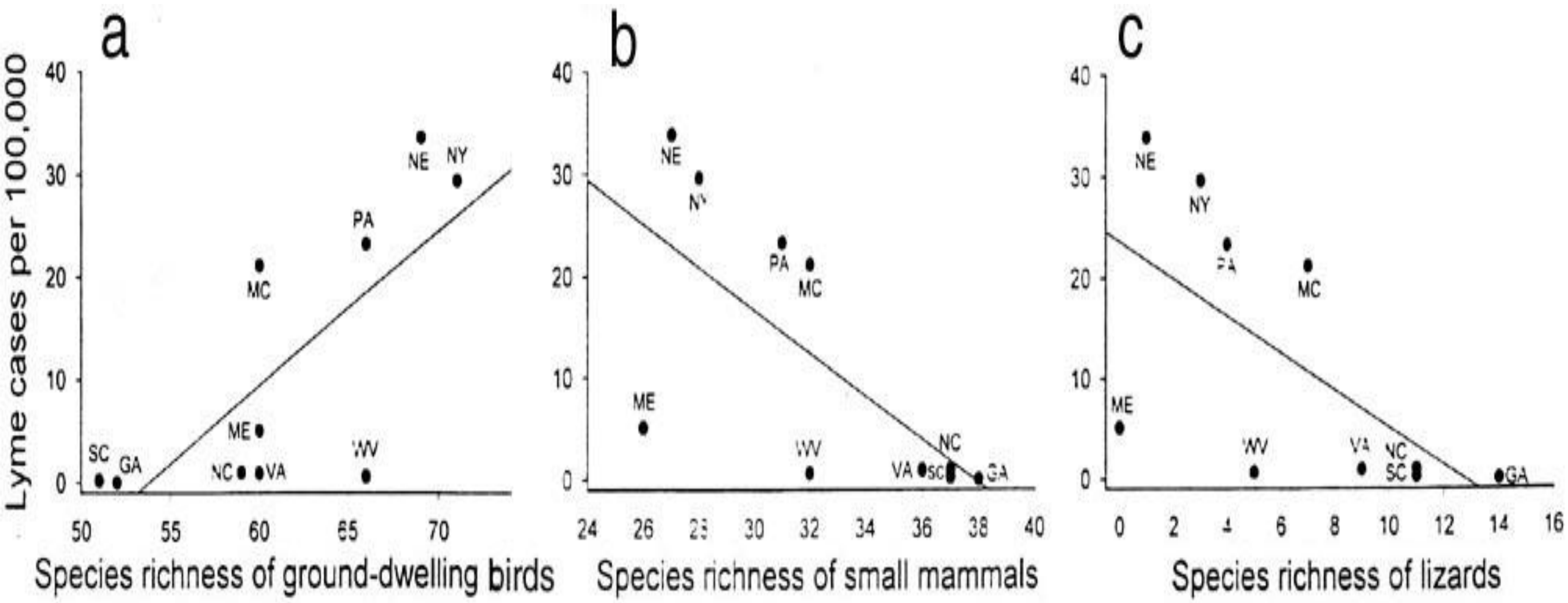


Fig. 2. The density of infected nymphs (DIN) declines with species richness (number of non-mouse, non-chipmunk hosts). Data were generated from 10 simulated host communities at each level of species richness (plus mice and chipmunks). DIN as used in Figs. 2–6 is based on the relative dominance and reservoir competence values assigned to host species in the model simulations and is for comparative purposes.



R. Ostfeld and F. Keesing,
Conservation Biology 2000.

Host viability for Lyme disease and the ticks that transmit it

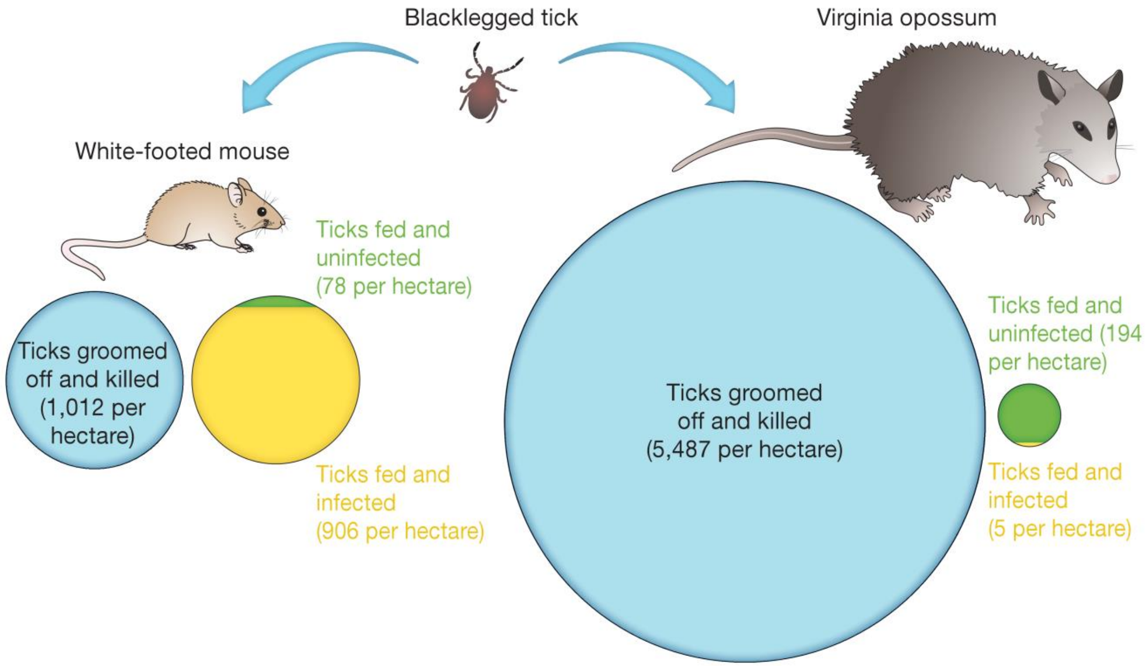
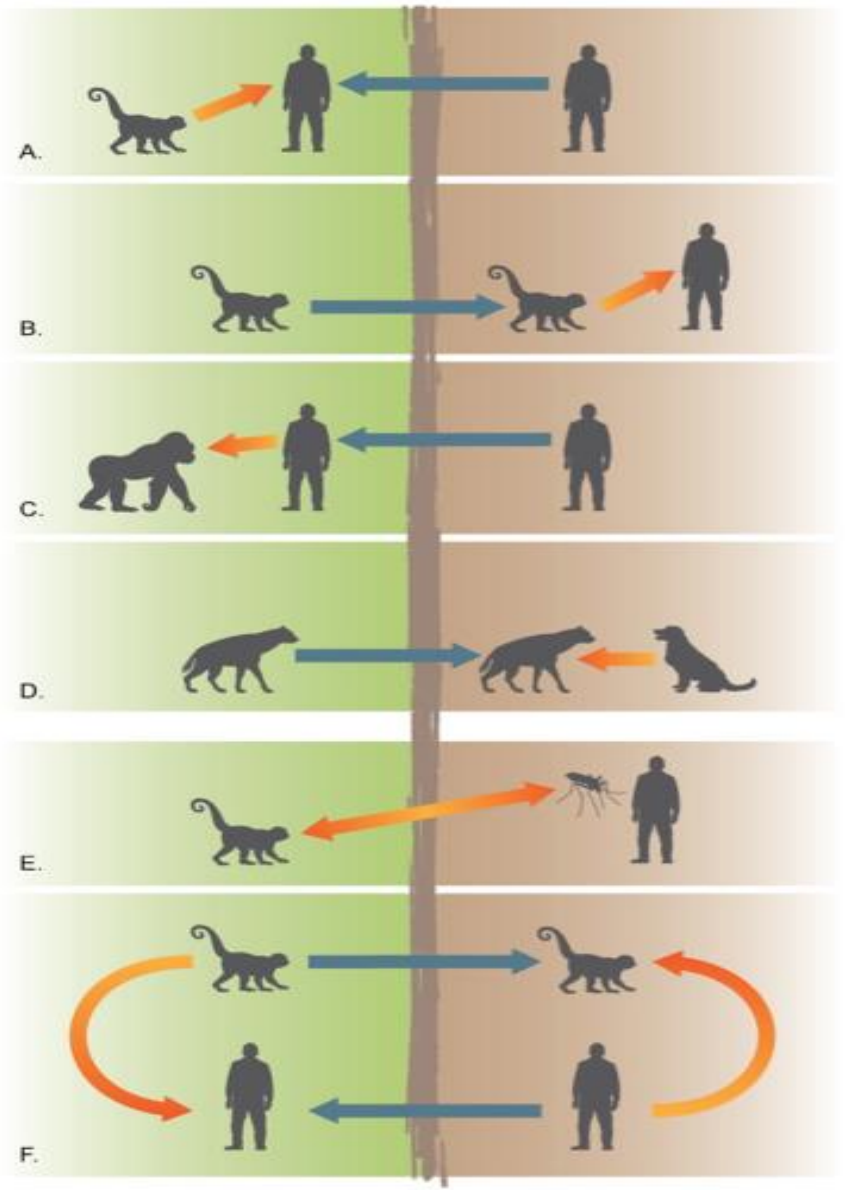


Figure 1 | Roles of host species in the transmission of Lyme disease in the northeastern USA. Lyme disease is transmitted to humans by the bite of an infected blacklegged tick (*Ixodes scapularis*). Immature ticks can acquire the infection if they feed on an infected host and can become infectious to humans if they subsequently survive to the next life stage. White-footed mice are abundant in northeastern forests and feed many ticks[18]. Ticks that attempt to feed on Virginia opossums are likely to be groomed off and killed. Green-and-yellow circles show the mean number of ticks per hectare fed by mice or opossums; yellow shading shows the proportion of ticks infected after feeding. Blue circles show the mean number of ticks per hectare groomed off and killed. Ticks that feed on mice are highly likely to become infected with the bacterium that causes Lyme disease, whereas those that feed on opossums are not.

Keesing et al Nature (2010)

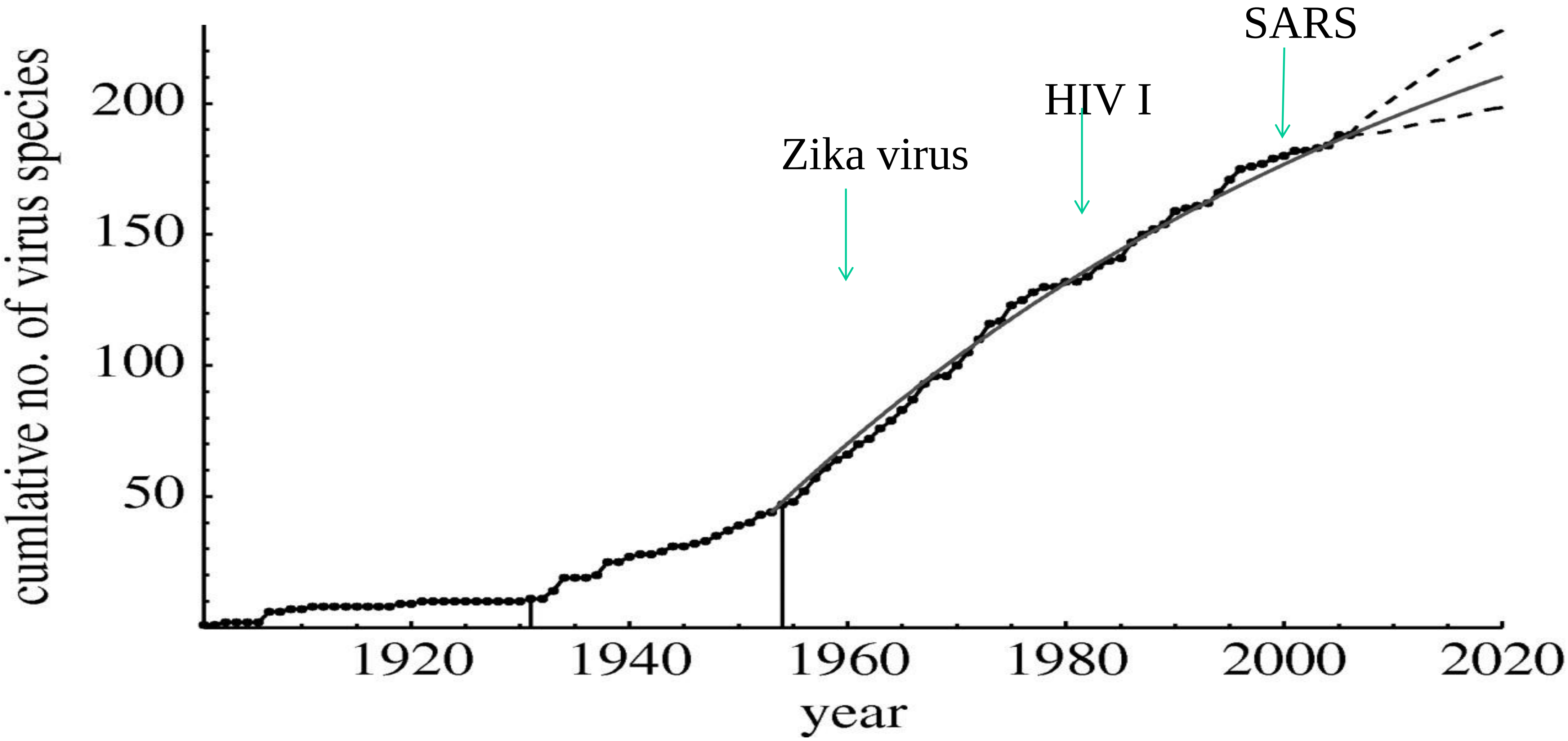
Habitat fragmentation in the Brazilian Amazon over the last 40 years.



Ways in which pathogens 'jump' the species barrier to establish in new hosts

Core : Matrix - Habitats

The discovery curve for human virus species.



Mark E.J Woolhouse et al. Proc. R. Soc. B 2008;275:2111-2115

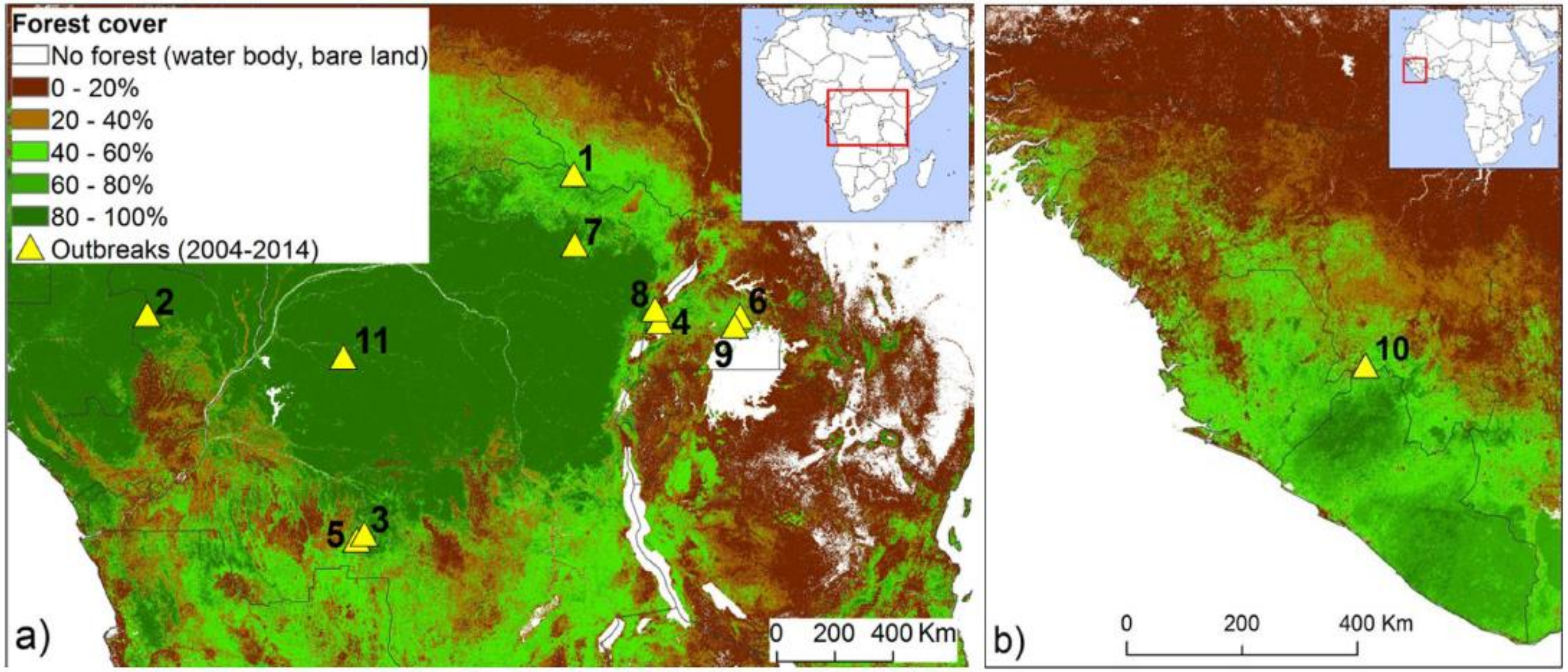


Figure 1. Forest cover maps and locations of first infection events in humans. Forest cover maps and locations of independent first infection events in humans (triangles, see Table 1) in Central (**a**) and West (**b**) Africa. The insets indicate the two African regions considered in this study. Legend in (**b**) is the same than in (**a**). Maps generated by the authors using ARCGIS 10.2-Version 10.2.0.338, licensed to Politecnico di Milano. The license term can be found on the following link: http://www.esri.com/legal/software-license.

SCIENTIFIC REPORTS

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The nexus between forest fragmentation in Africa and Ebola virus disease outbreaks

Received: 17 August 2016
Accepted: 22 December 2016

Maria Cristina Rulli[1], Monia Santini[2], David T S Hayman[3] & Paolo D'Odorico[4,5,6]

OPEN

The nexus between forest fragmentation in Africa and Ebola virus disease outbreaks

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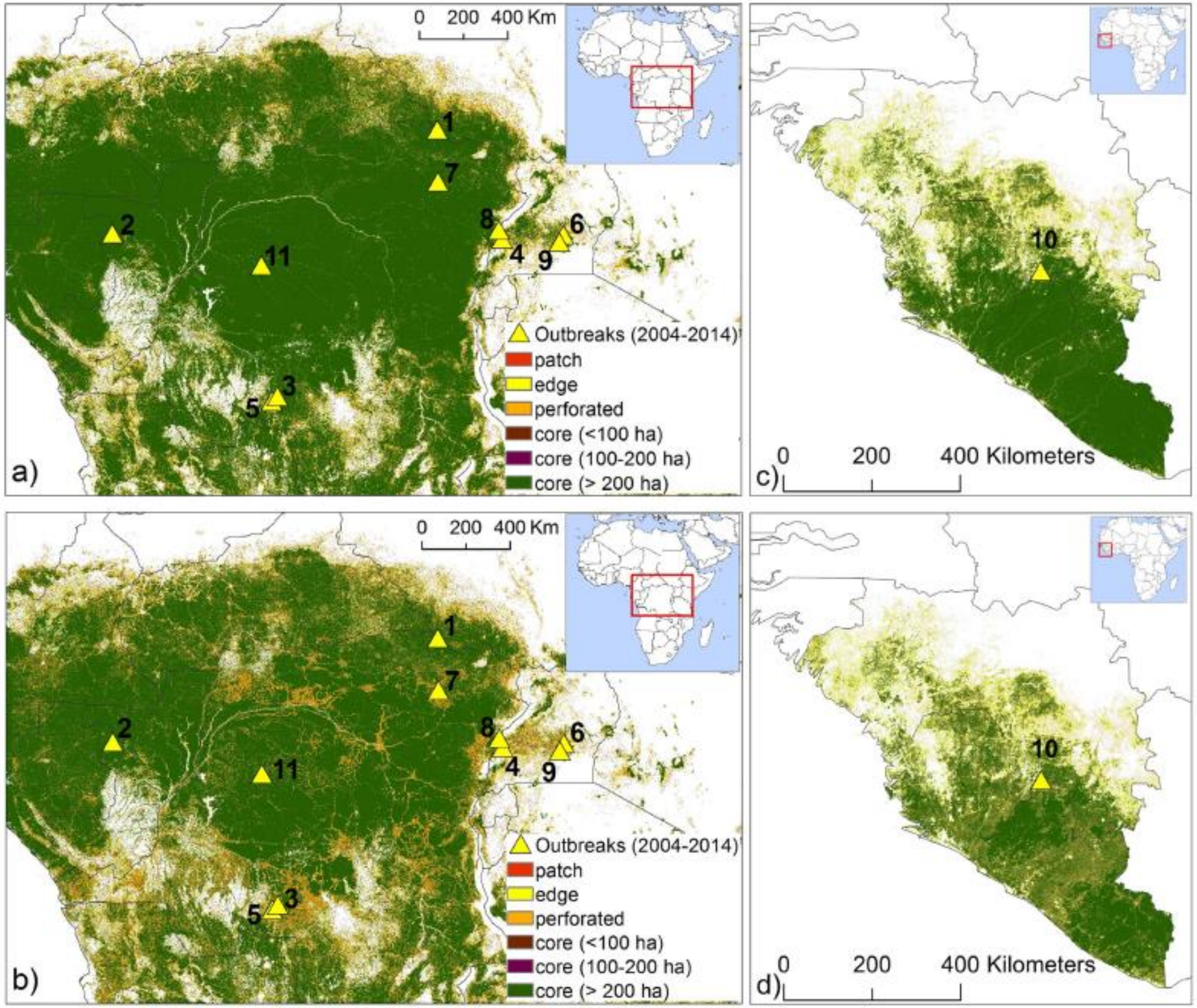
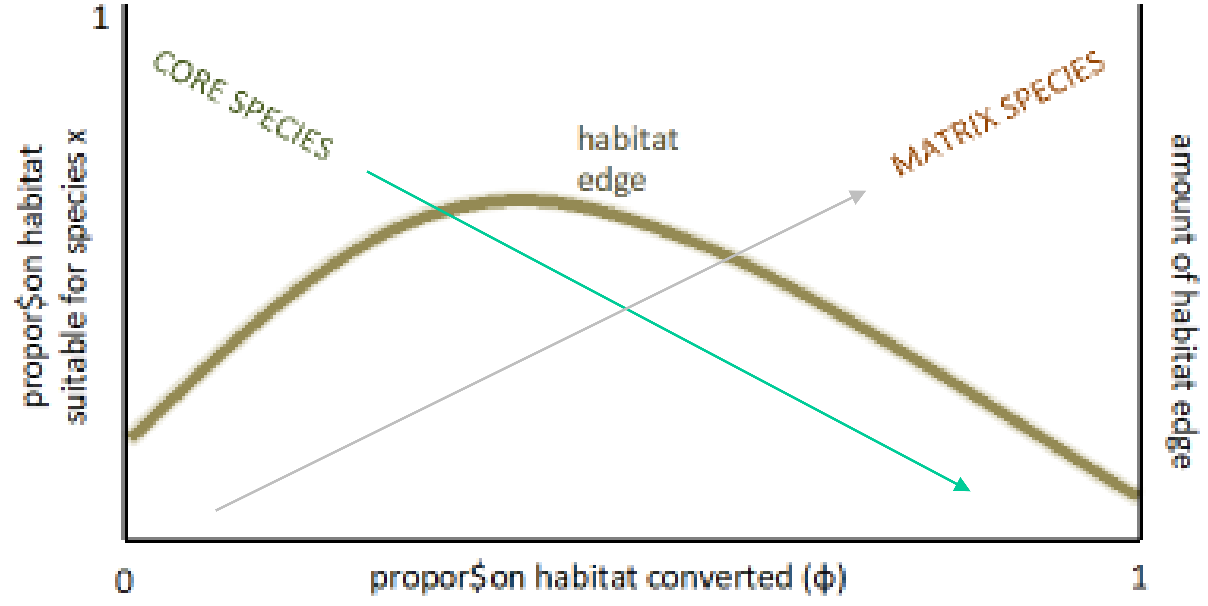
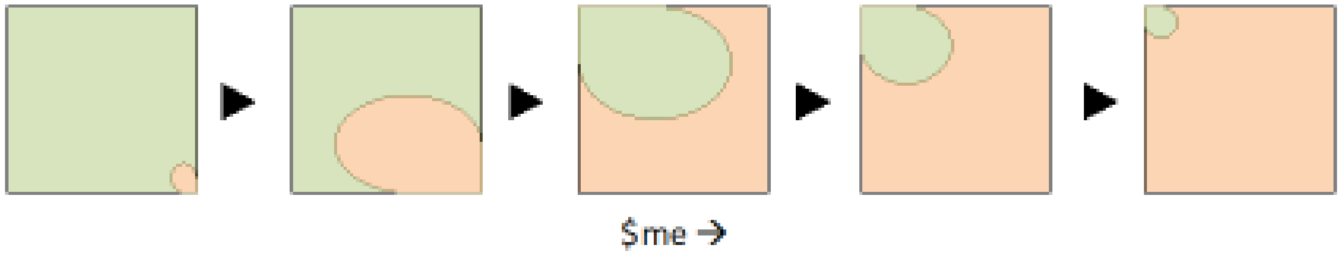


Figure 2. Forest fragmentation in Central and West Africa. Forest fragmentation in Central (panels a, and b)

Faust, McCallum, Bloomfield, Dobson , Plowright et al (in press)





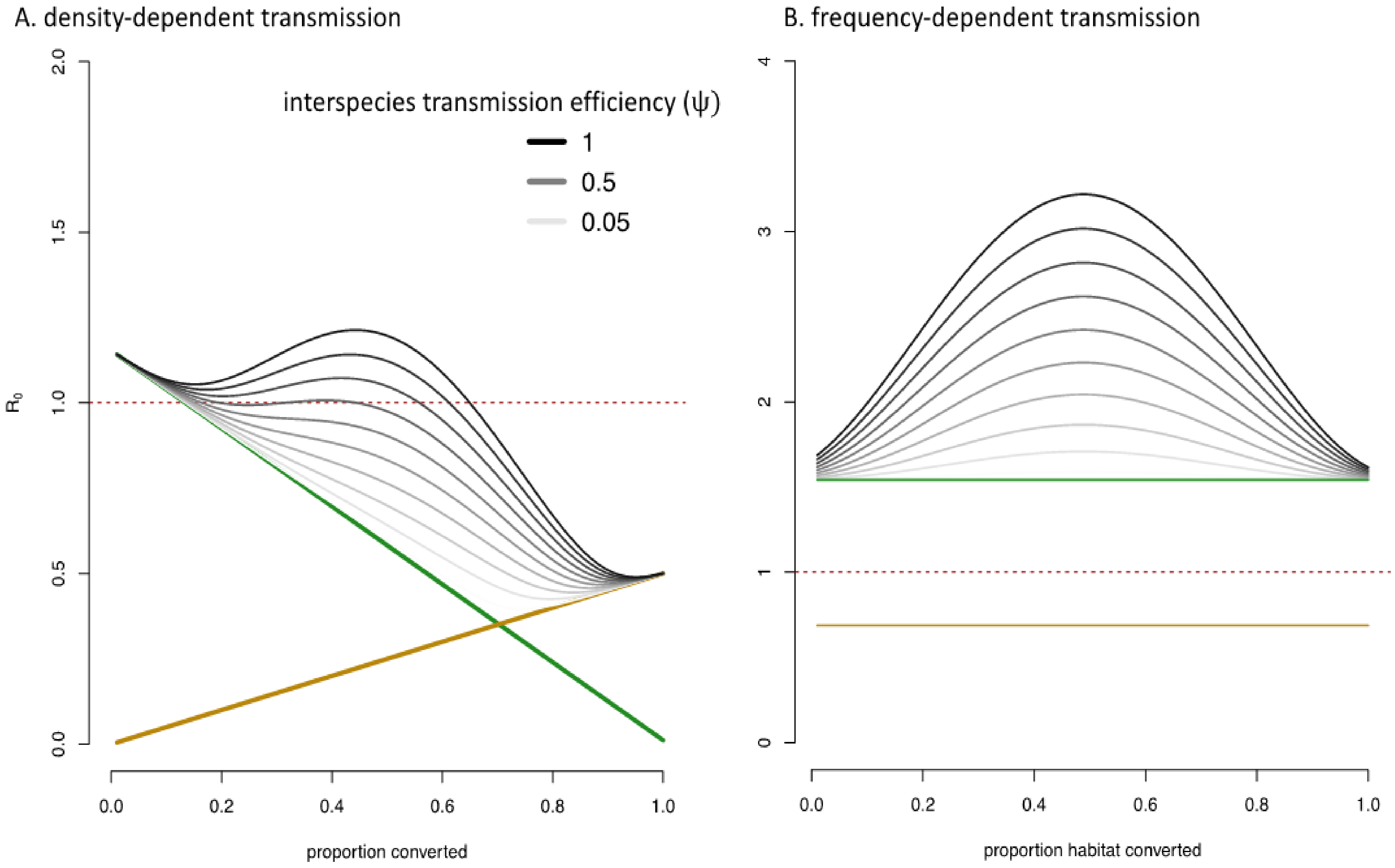
Faust, McCallum, Bloomfield, Dobson , Plowright et al (in press)

$$\frac{\mathrm{d}S_i}{\mathrm{d}t} = b_i\, N_i \left(1 - \frac{N_i}{K_{ia}}\right) - \frac{S_i(\beta_{ii}I_i + \ \psi \sum_j^n \beta_{ij}I_j)}{a} - \ d_iS_i, \tag{2.1}$$

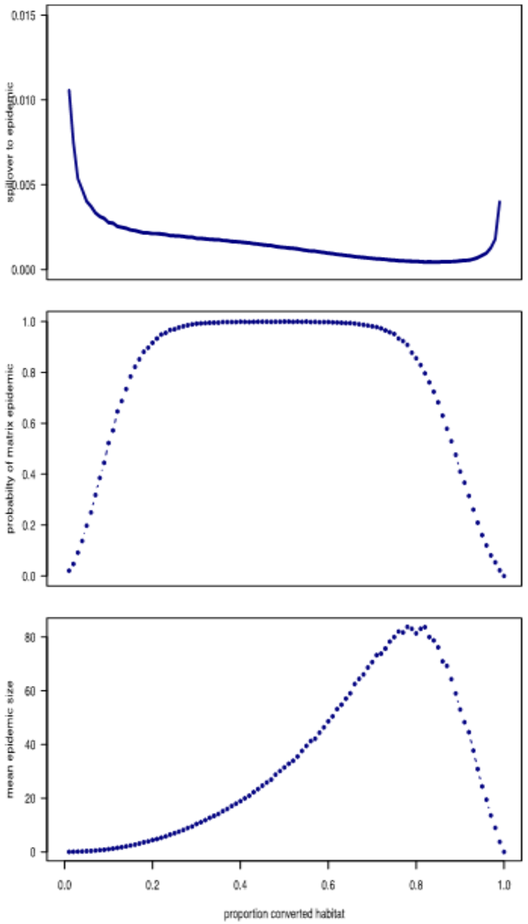
$$\frac{\mathrm{d}I_i}{\mathrm{d}t} = \frac{S_i(\beta_{ii}I_i + \ \psi \sum_j^n \beta_{ij}I_j)}{a} - \ I_i(\alpha_i + \gamma_i + d_i) \tag{2.2}$$

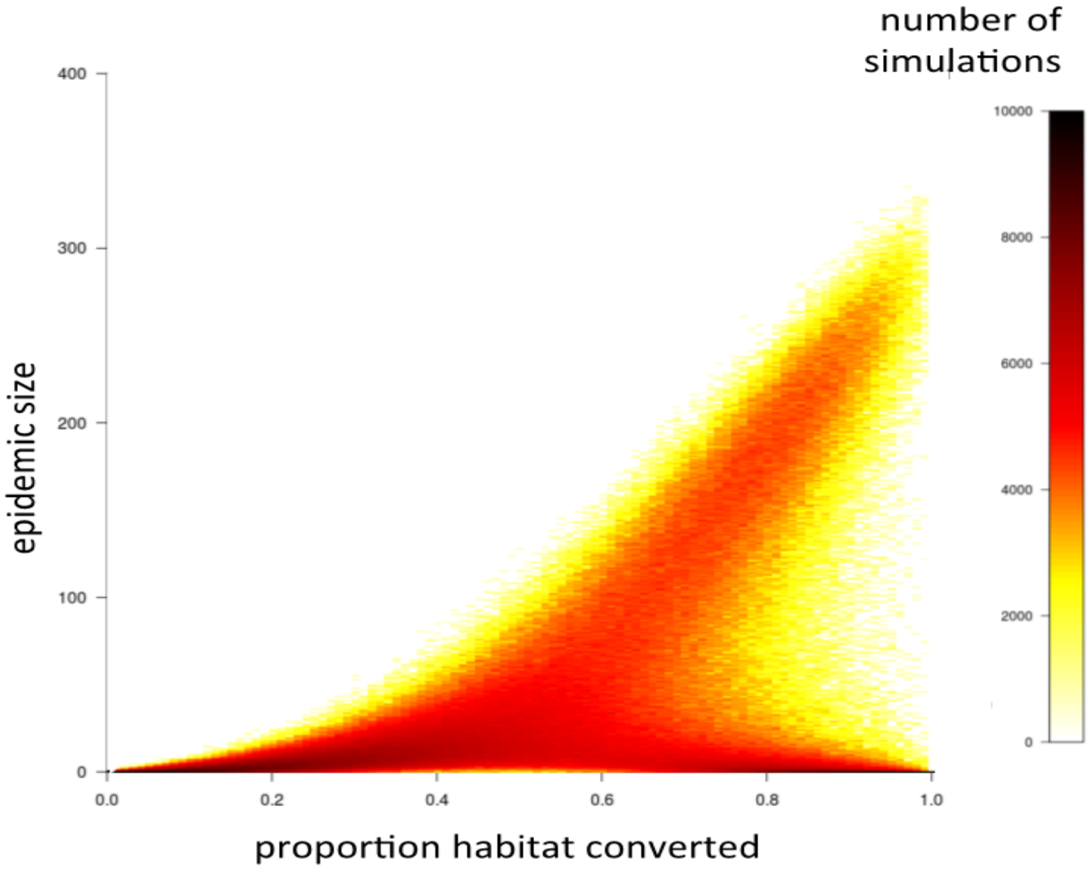
and $$\frac{\mathrm{d}R_i}{\mathrm{d}t} = \gamma_i I_i - d_i R_i. \tag{2.3}$$

Faust, McCallum, Bloomfield, Dobson , Plowright et al (in press)

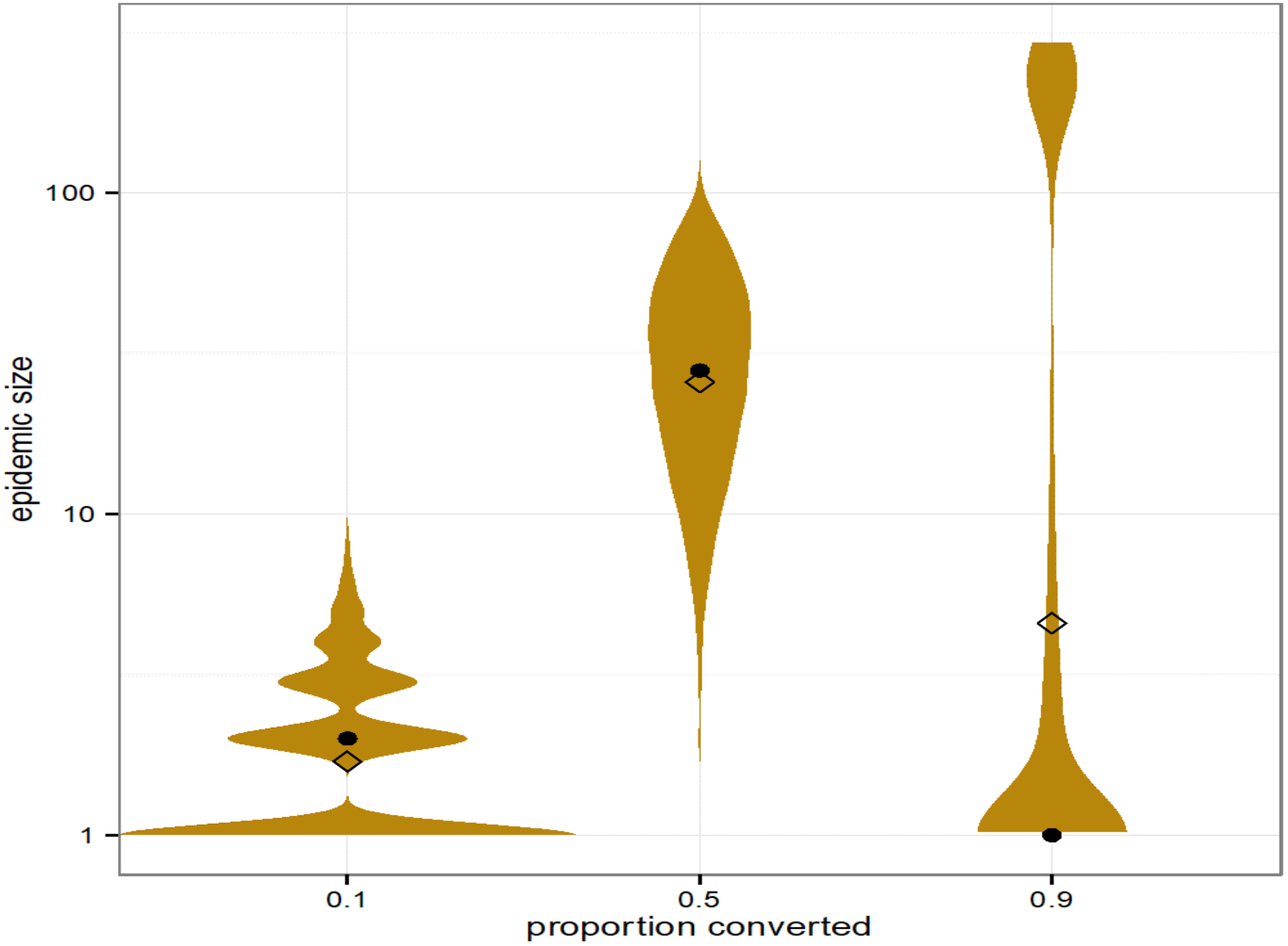


Faust, McCallum, Bloomfield, Dobson , Plowright et al (in press)

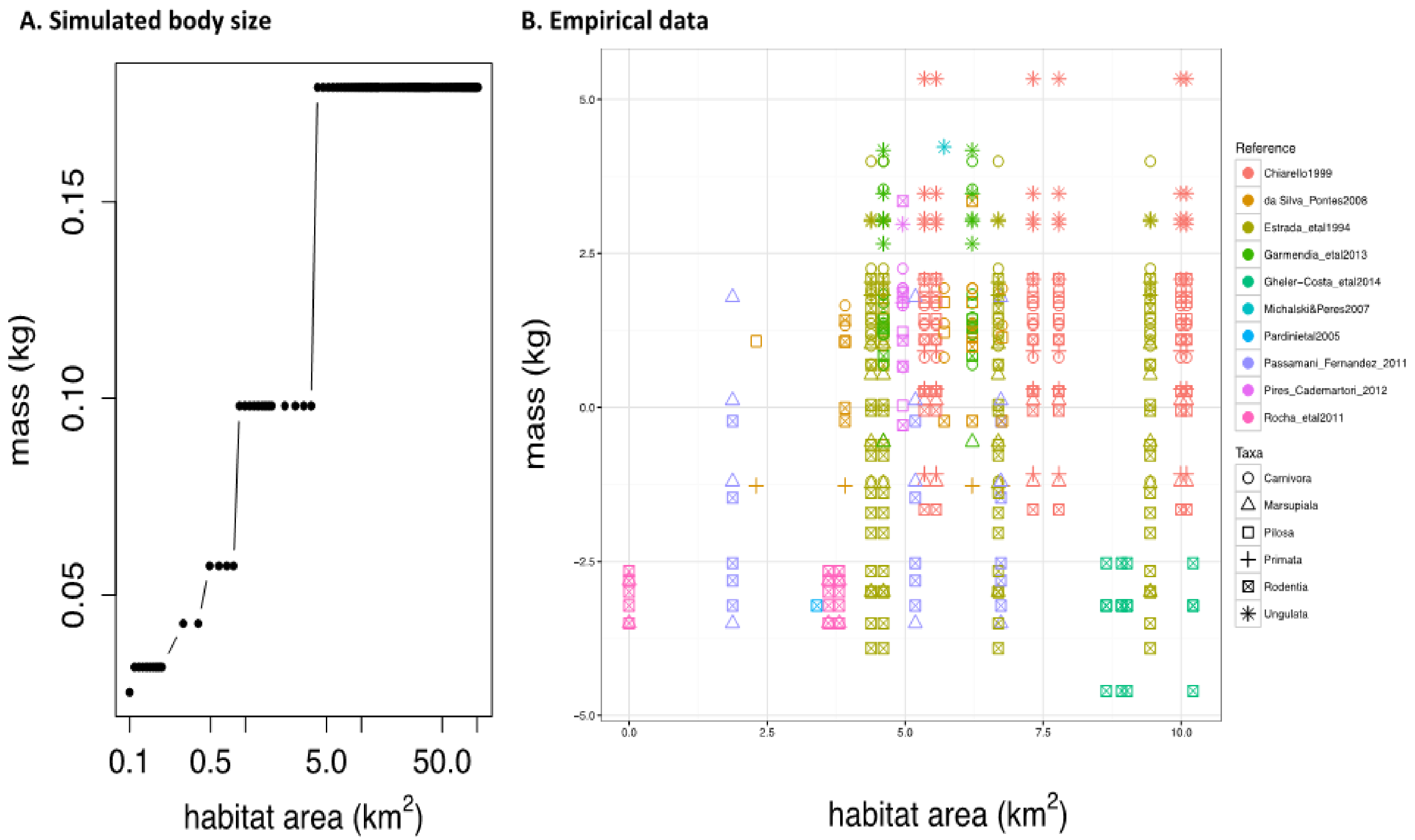




Faust, McCallum, Bloomfield, Dobson , Plowright et al (in press)



What happens in any individual patch as it erodes?

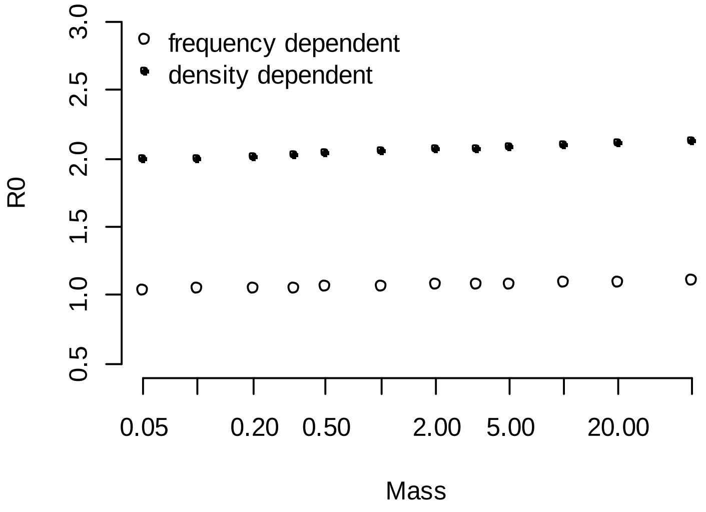

A. Simulated body size

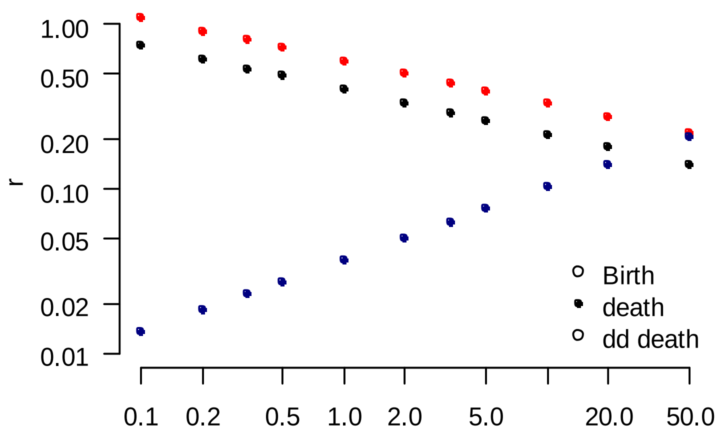
B. Empirical data

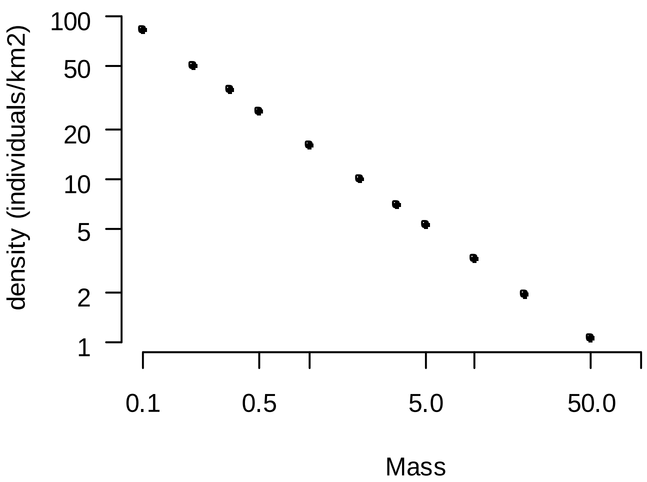
Habitat patch occupancy by body size. A) The simulated average body size in a patch at disease free equilibrium. B) Empirical data from all studies that we could find showing that smaller bodied species are present in smaller patch sizes and persist in the larger patches when larger species are added (data from [10-20], listed in supplementary text information).

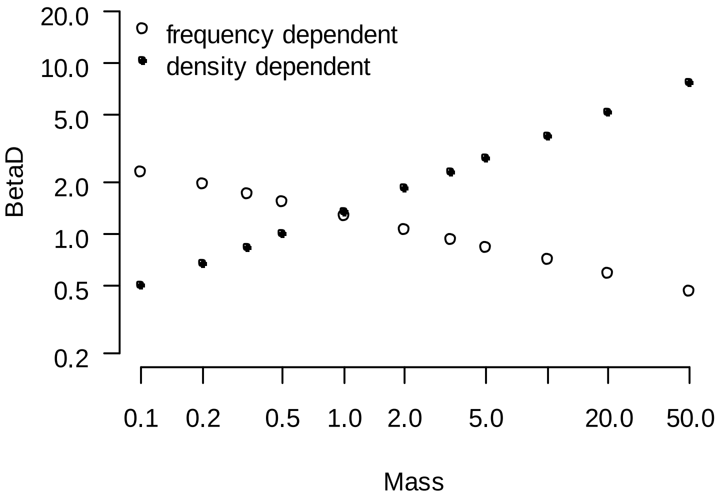
Faust, Dobson, Bloomfield, Gottdecker, McCallum, Gillespie, Plowright (2017)

Underlying demography based on allometric scaling with body size (DeLeo and Dobson, Nature 1997)

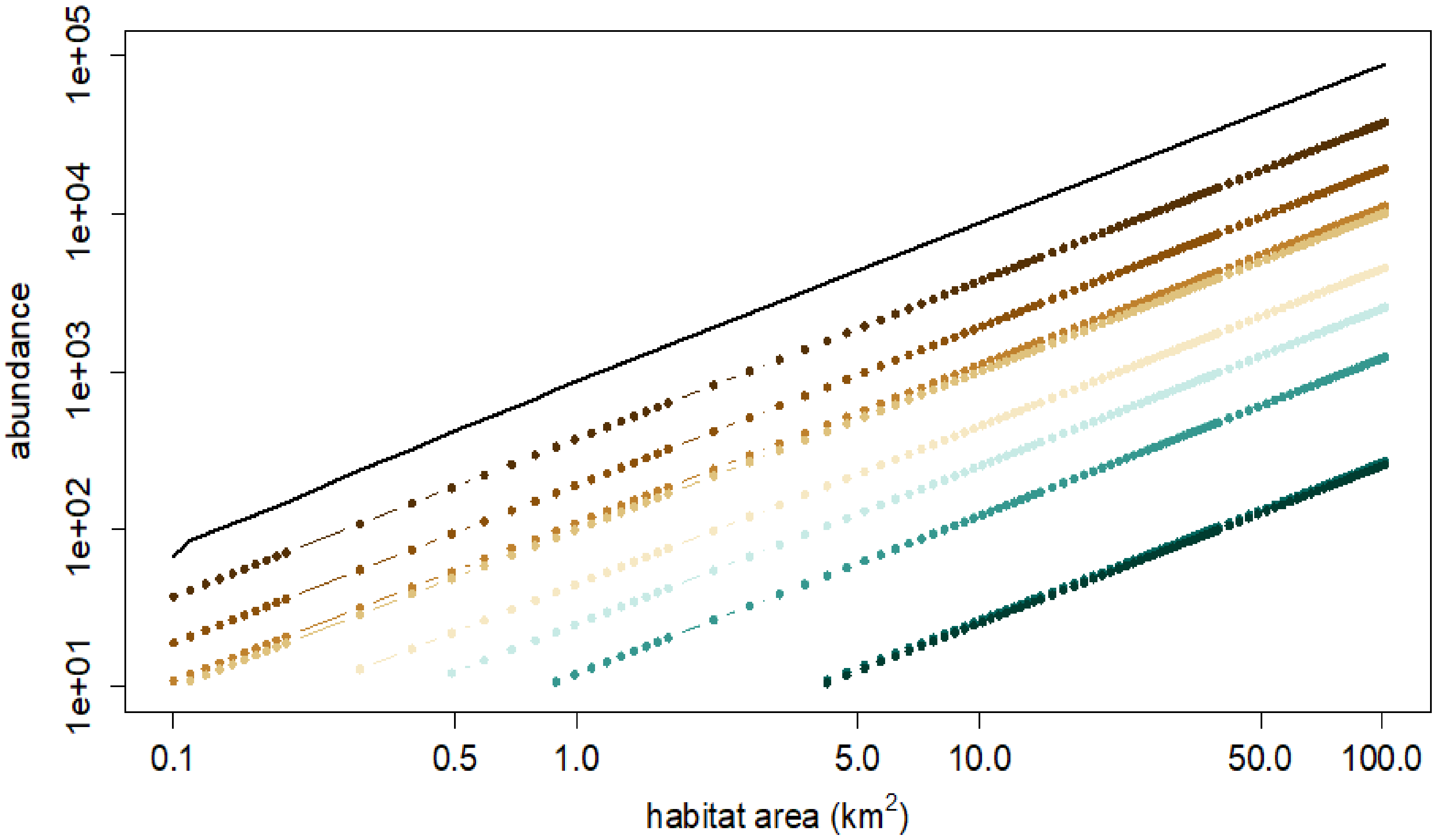

Ro


Birth, death, delta by body size


Population Density

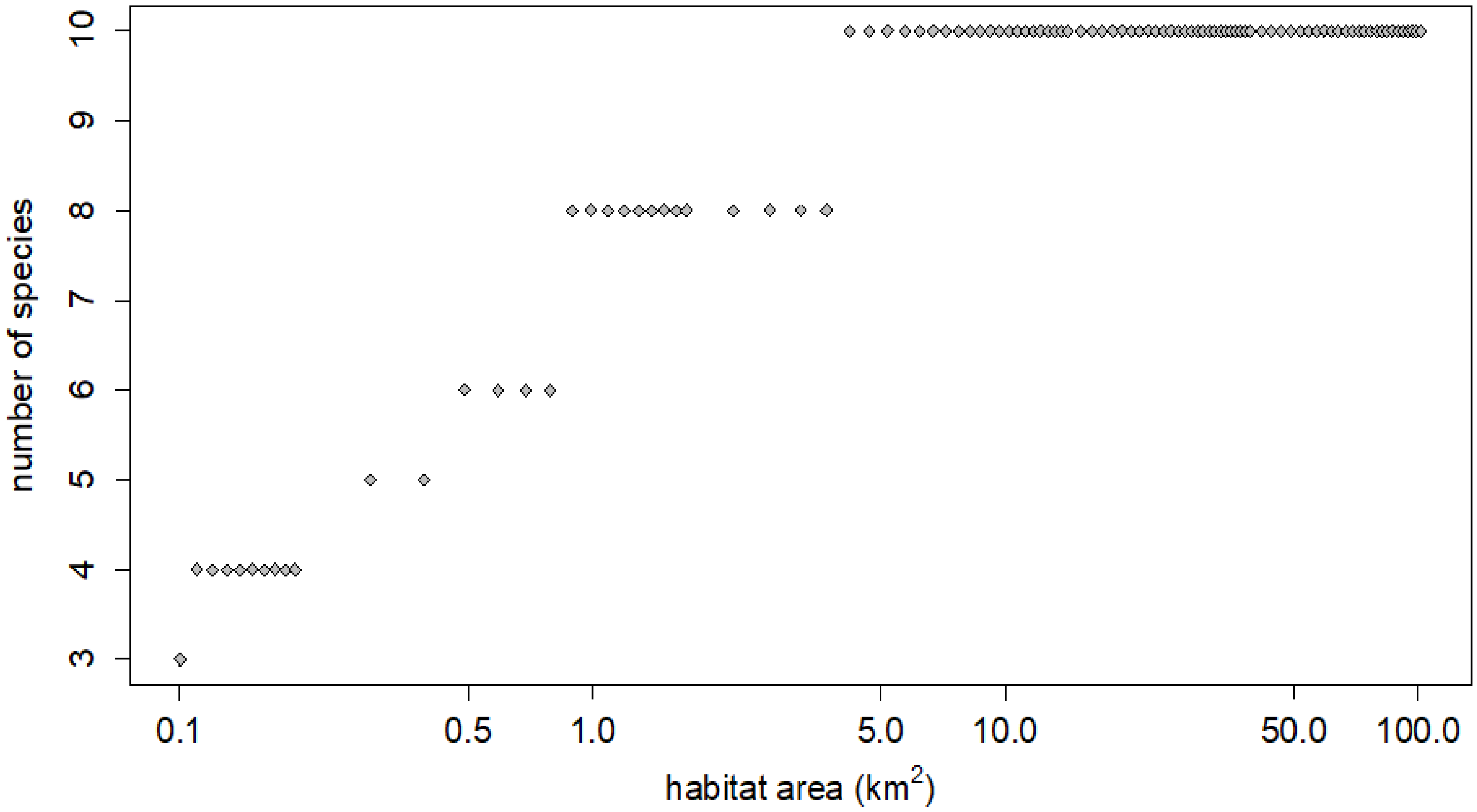

Beta for hosts, same Ro

Abundance of species in a disease free equilibrium. The abundance of each species and total abundance in each habitat for example community (mean body size of 10 species = 0.011, 0.030, 0.065, 0.075, 0.23, 0.537, 1.505, 1.515, 13.333, and 14.201 kg).



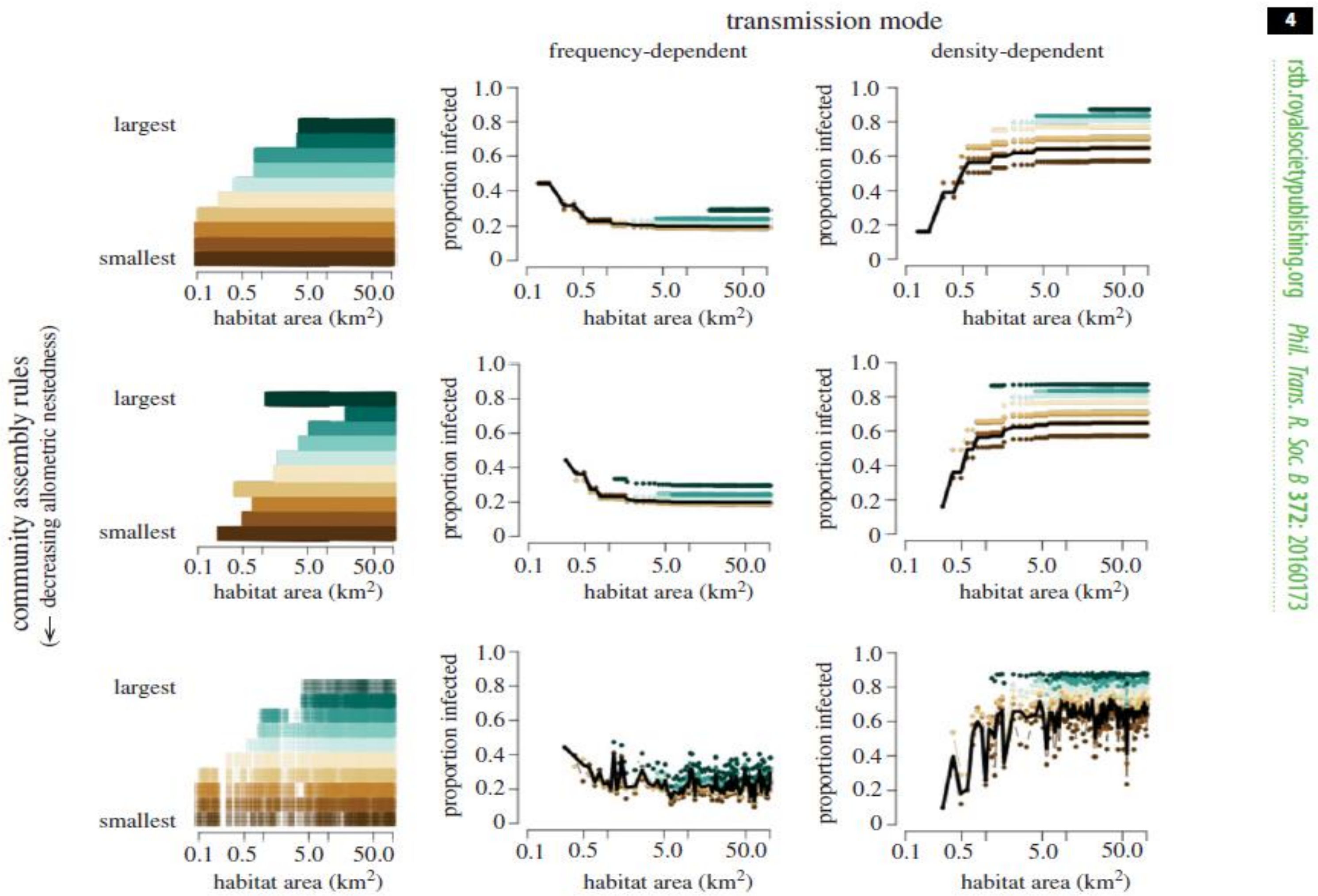
Faust, Dobson, Bloomfield, Gottdecker, McCallum, Gillespie, Plowright (2017)

Species accumulation curve for disease free equilibrium. The number of species in each habitat patch for a community with a ten host species pool with an average body size of 0.011, 0.030, 0.065, 0.075, 0.23, 0.537, 1.505, 1.515, 13.333, and 14.201 kg.



Faust, Dobson, Bloomfield, Gottdecker, McCallum, Gillespie, Plowright (2017)

Risk of exposure to different pathogens will vary between larger and smaller patches



Faust, Dobson, Bloomfield, Gottdecker, McCallum, Gillespie, Plowright (2017)

Relatively robust result even when hosts of different competency are added in

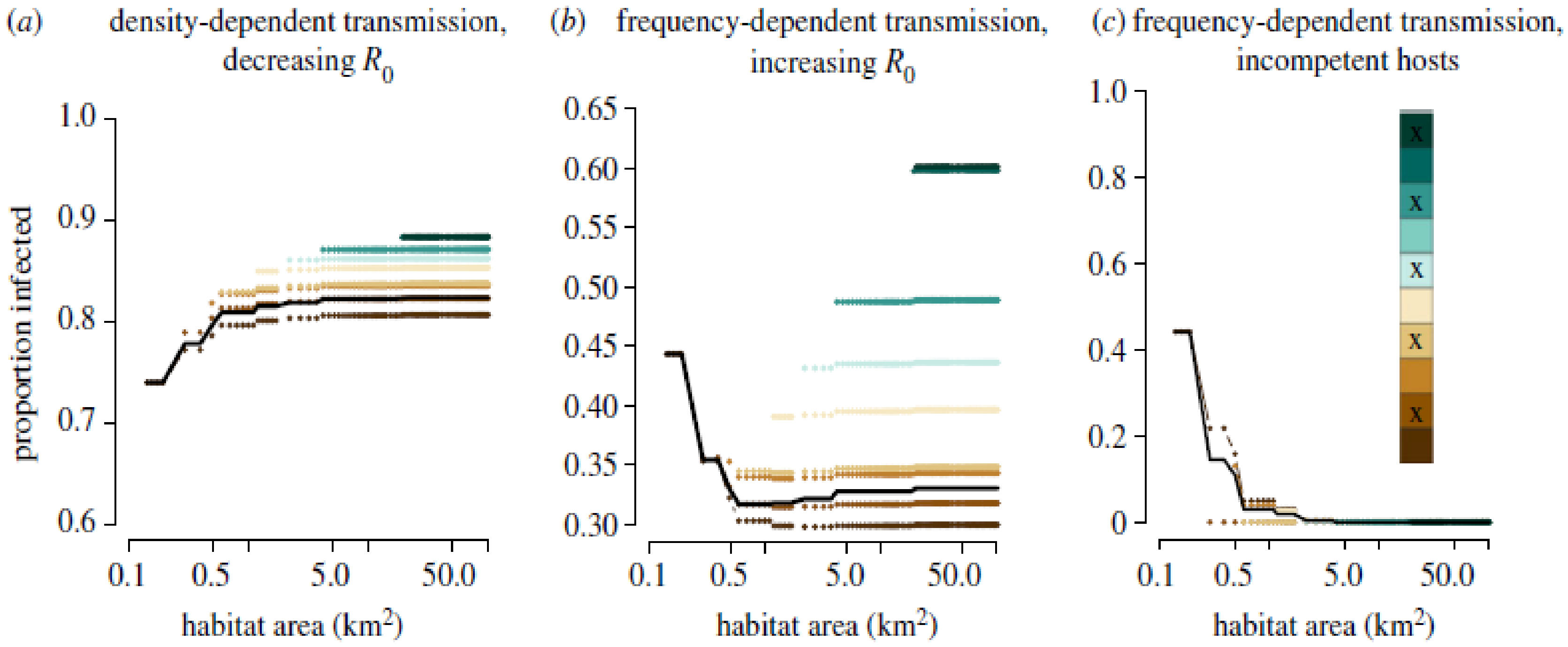


Figure 2. Variation in host competence and underlying assumptions impact diversity–disease relationships. (*a*) Even if the smallest hosts are the most competent [61] and there are extreme differences in R_0 between body sizes (electronic supplementary material, figure S5), only the amplification effect is observed for density-dependent pathogens. (*b*) If behavioural allometry leads to an increase in R_0 across body size, this can lead to an amplification effect for frequency-dependent pathogens at larger patch sizes, but a dilution effect for small to intermediate patches. (*c*) When species that can become infected but are in turn not infectious (incompetent hosts, denoted by x) are randomly assembled along the distribution of body sizes, then dilution effects can be exacerbated for frequency-dependent pathogens, but this depends on the order of community introduction of these incompetent hosts.

rstb.royalsocietypublishing.org *Phil. Trans. R. Soc. B* 372: 20160173

Faust, Dobson, Bloomfield, Gottdecker, McCallum, Gillespie, Plowright (2017)

Subtle effects of host territoriality – within-species between territories, etc

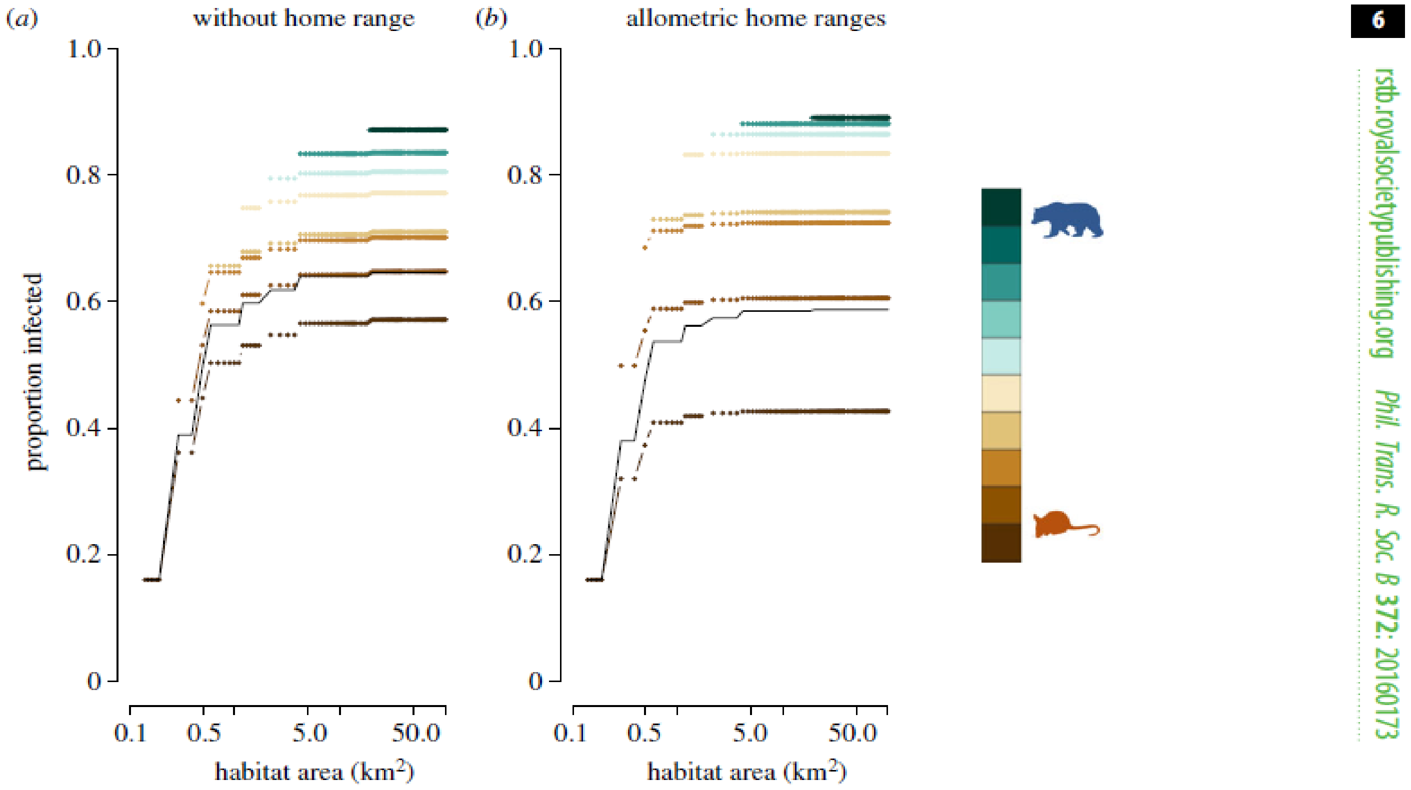


Figure 3. Impact of heterogeneous contacts on density-dependent pathogen transmission. Using identical disease parameters, endemic prevalence was observed for a host system that assumed contacts were determined by density of hosts (*a*) and compared with a system in which home range determined the average contacts of an individual from a given species (*b*). When home range is not taken into account, overall prevalence is higher, but when home range is considered, larger-bodied species that have larger home ranges have higher within-species infection prevalence.

Faust, Dobson, Bloomfield, Gottdecker, McCallum, Gillespie, Plowright (2017)

The dynamics of multi-host, multi parasite systems are more subtle than those of single pathogen, single hosts.

- Basic models can be scaled up – key parameter is relative rate of transmission between versus within species.

- Allometric scaling of demographic and epidemiological parameters allows generalization to n-species of hosts
    - This can also be done for macroparasites – DeLei, Dobson and Gatto, 2016.

- Frequency-dependent transmission can give rise to a dilution effect, density dependent usually gives an amplification of epidemic size.