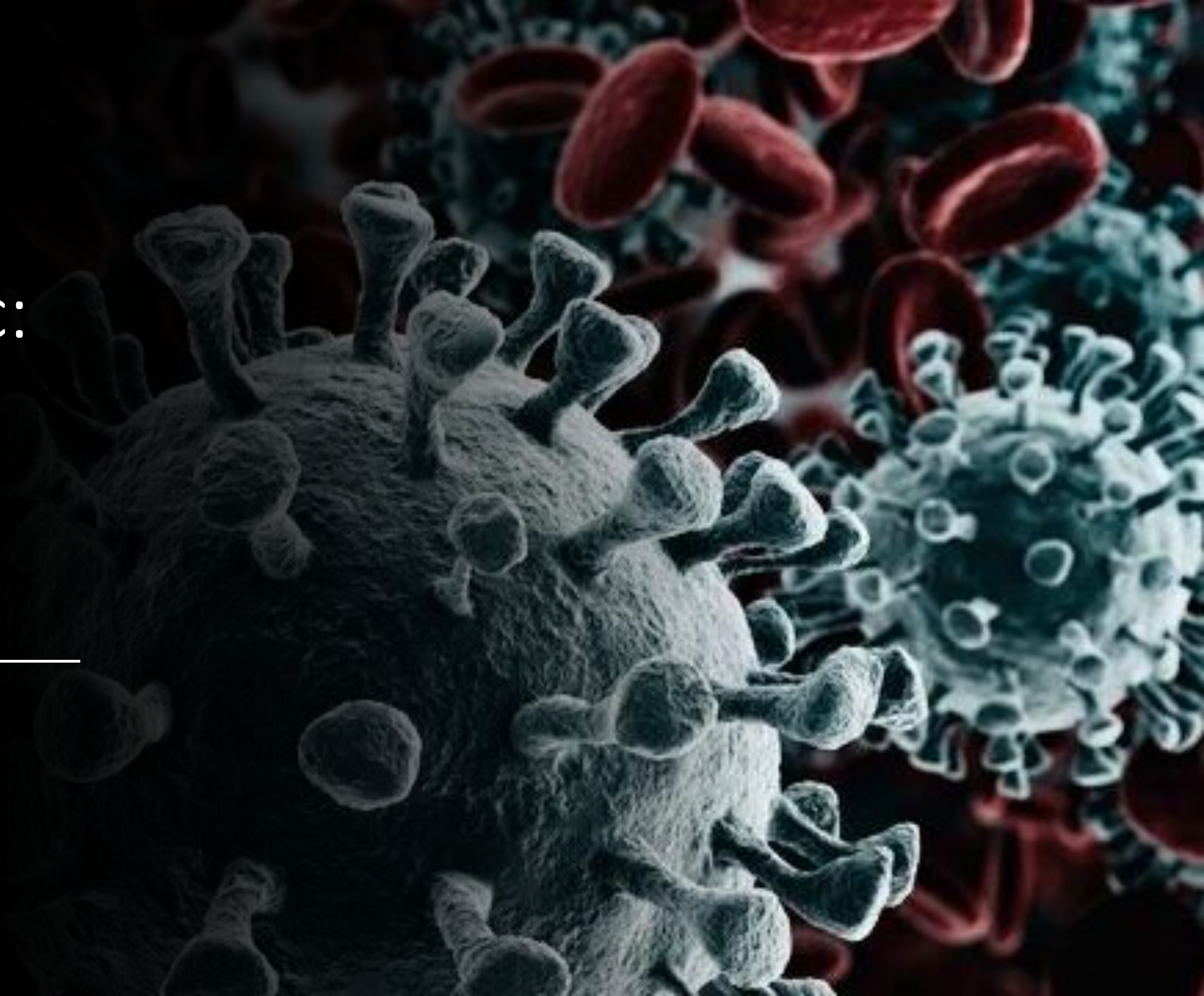
Preventing the Next Pandemic: Ecology, Economics, Evolution

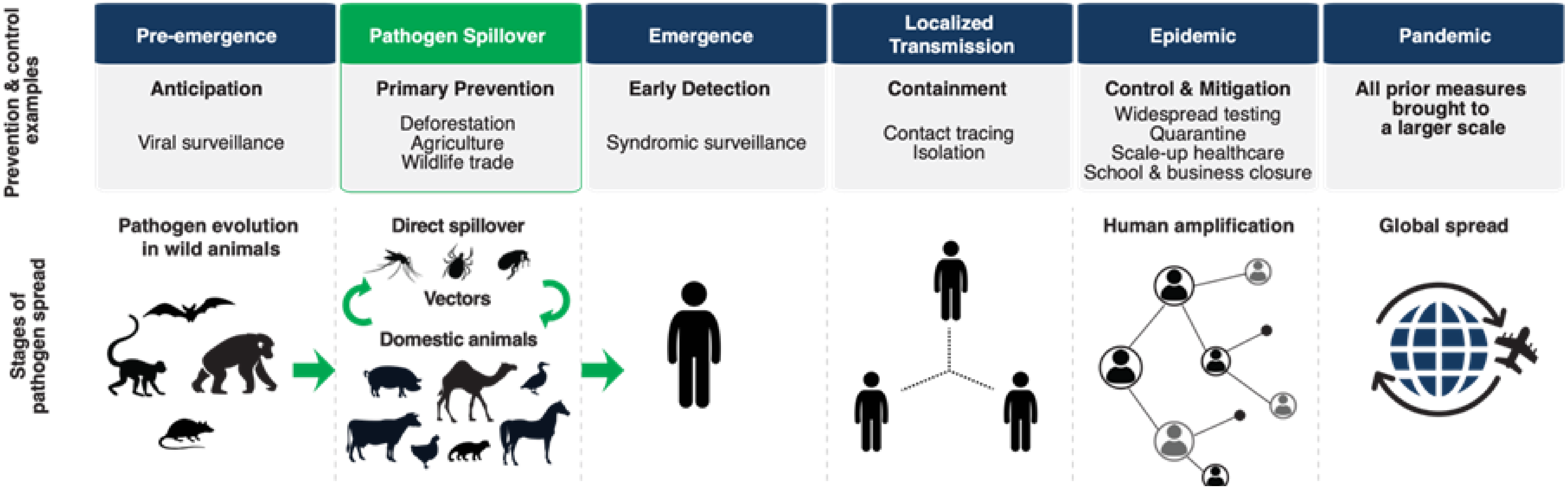


Andy Dobson, EEB, Princeton University,

Santa Fe Institute, New Mexico

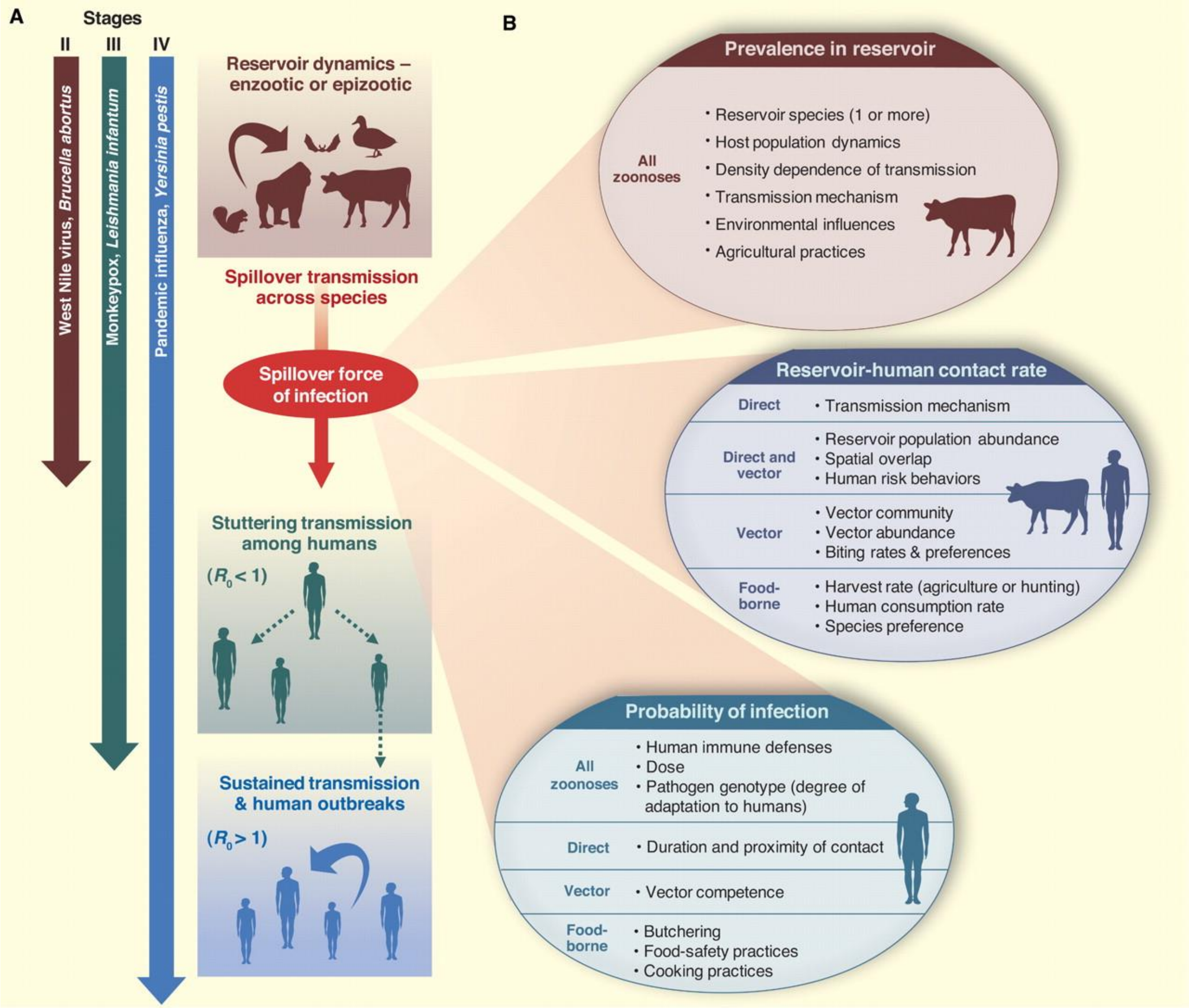
Structure of Talk

- **Ecology of Emerging Pathogens**
  - Agricultural intensification
  - Habitat fragmentation and emergence
  - The role of the wildlife trade
  - Rate of exposure and cross immunity
- **Economics of Emerging Diseases**
  - Cost-benefit at each stage of intervention
  - Overall cost-benefit
  - Missing costs and additional benefits
- **Evolution of Emerging Pathogens**
  - Will Covid-19 become more or less virulent?
  - Insights from an avian emerging pathogen



	Pre-emergence	Pathogen Spillover	Emergence	Localized Transmission	Epidemic	Pandemic
Prevention & control examples	**Anticipation** Viral surveillance	**Primary Prevention** Deforestation Agriculture Wildlife trade	**Early Detection** Syndromic surveillance	**Containment** Contact tracing Isolation	**Control & Mitigation** Widespread testing Quarantine Scale-up healthcare School & business closure	**All prior measures brought to a larger scale**
Stages of pathogen spread	**Pathogen evolution in wild animals**	**Direct spillover** **Vectors** **Domestic animals**			**Human amplification**	**Global spread**

Six stages of viral emergence (Bernstein, Dobson et al, in review)



Prevalence in reservoir

All zoonoses
- Reservoir species (1 or more)
- Host population dynamics
- Density dependence of transmission
- Transmission mechanism
- Environmental influences
- Agricultural practices

Reservoir-human contact rate

Direct
- Transmission mechanism

Direct and vector
- Reservoir population abundance
- Spatial overlap
- Human risk behaviors

Vector
- Vector community
- Vector abundance
- Biting rates & preferences

Food-borne
- Harvest rate (agriculture or hunting)
- Human consumption rate
- Species preference

Probability of infection

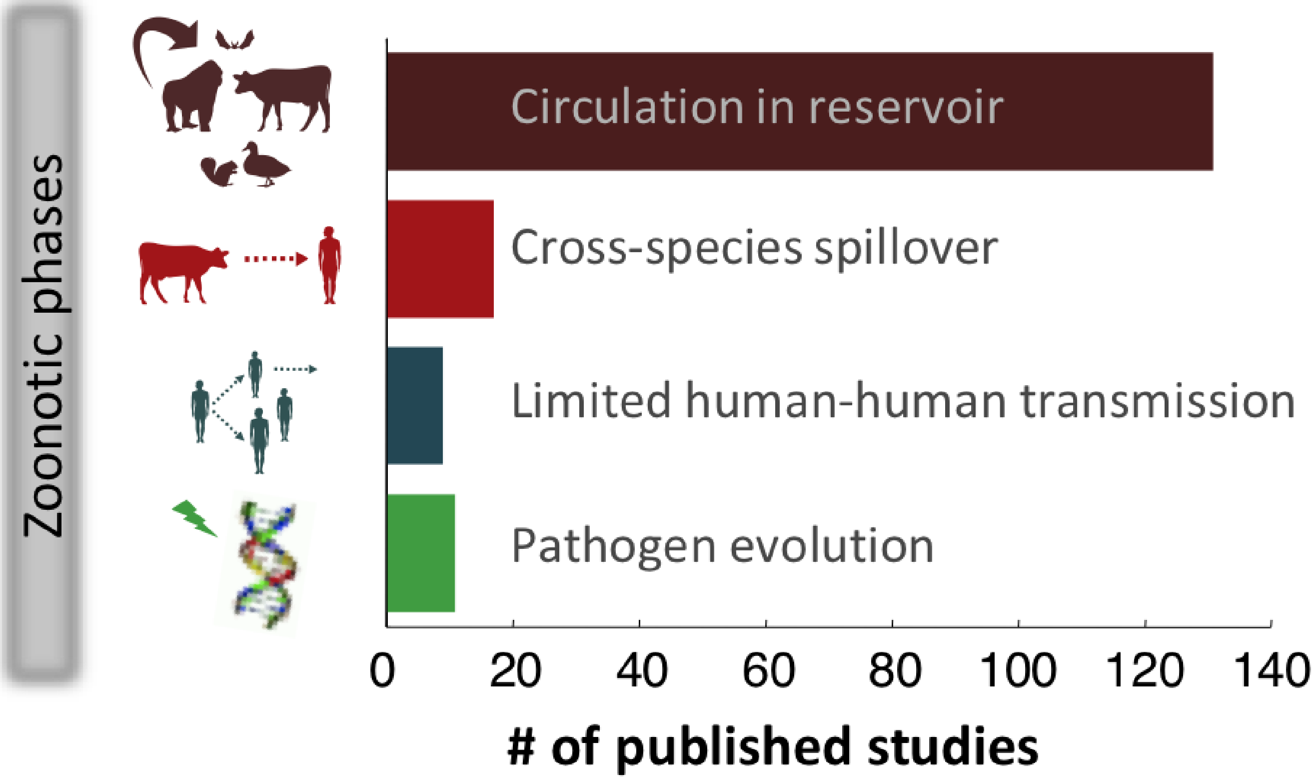
All zoonoses
- Human immune defenses
- Dose
- Pathogen genotype (degree of adaptation to humans)

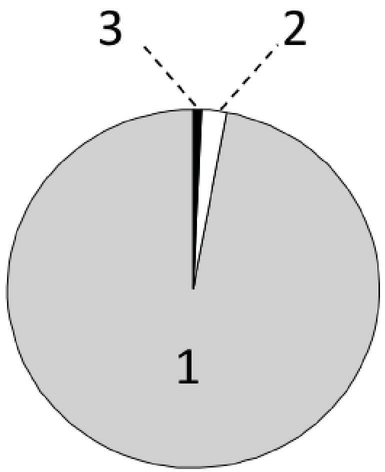
Direct
- Duration and proximity of contact

Vector
- Vector competence

Food-borne
- Butchering
- Food-safety practices
- Cooking practices

Lloyd-Smith et al
Science, 2009.



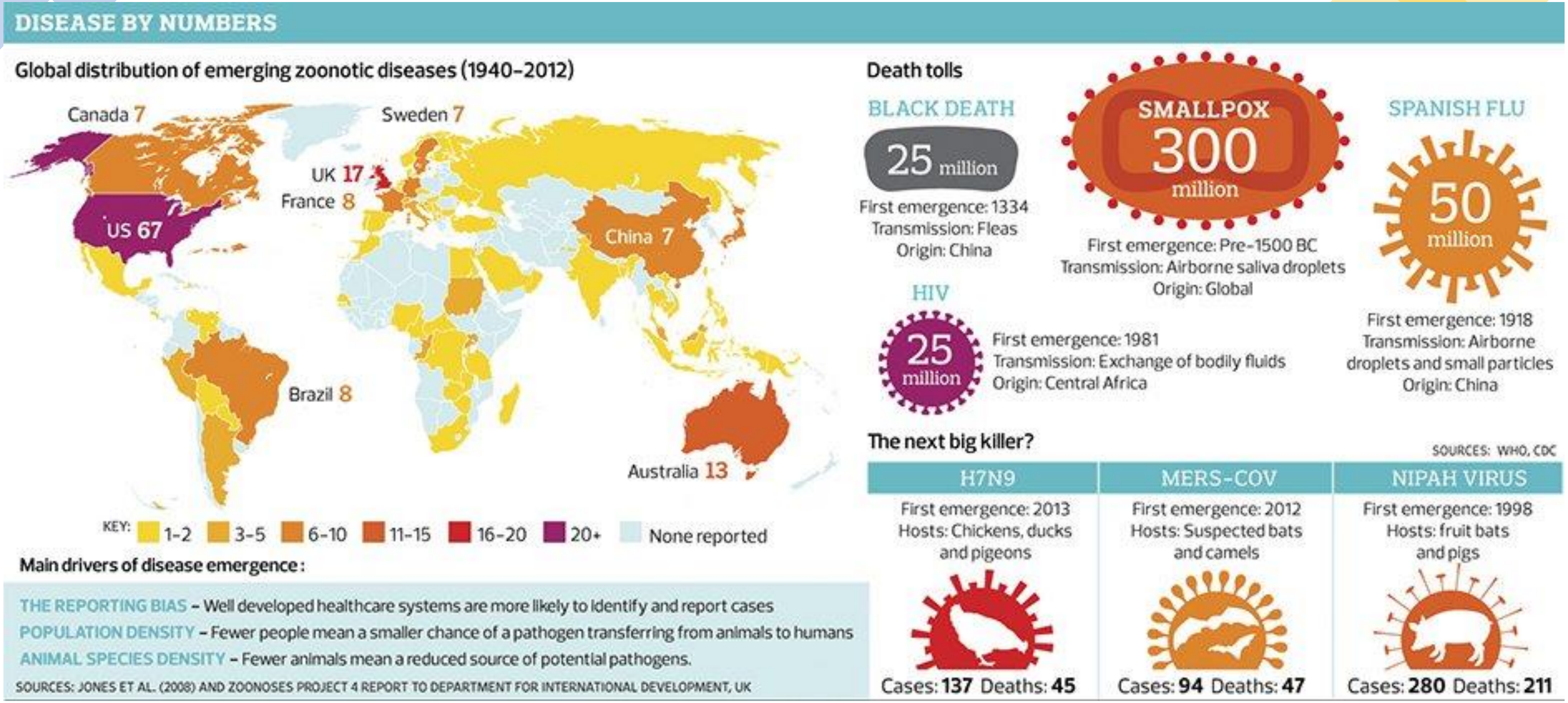


# of zoonotic phases included in published studies

Lloyd-Smith et al (2009)

1 .Ecology of Emerging Pathogens

- Agricultural intensification
- Habitat fragmentation and emergence
- The role of the wildlife trade
- Rates of exposure and cross immunity



DISEASE BY NUMBERS

Global distribution of emerging zoonotic diseases (1940-2012)

KEY: 1-2 | 3-5 | 6-10 | 11-15 | 16-20 | 20+ | None reported

Main drivers of disease emergence :

THE REPORTING BIAS – Well developed healthcare systems are more likely to identify and report cases

POPULATION DENSITY – Fewer people mean a smaller chance of a pathogen transferring from animals to humans

ANIMAL SPECIES DENSITY – Fewer animals mean a reduced source of potential pathogens.

SOURCES: JONES ET AL. (2008) AND ZOONOSES PROJECT 4 REPORT TO DEPARTMENT FOR INTERNATIONAL DEVELOPMENT, UK

Death tolls

BLACK DEATH
25 million
First emergence: 1334
Transmission: Fleas
Origin: China

SMALLPOX
300 million
First emergence: Pre-1500 BC
Transmission: Airborne saliva droplets
Origin: Global

SPANISH FLU
50 million
First emergence: 1918
Transmission: Airborne droplets and small particles
Origin: China

HIV
25 million
First emergence: 1981
Transmission: Exchange of bodily fluids
Origin: Central Africa

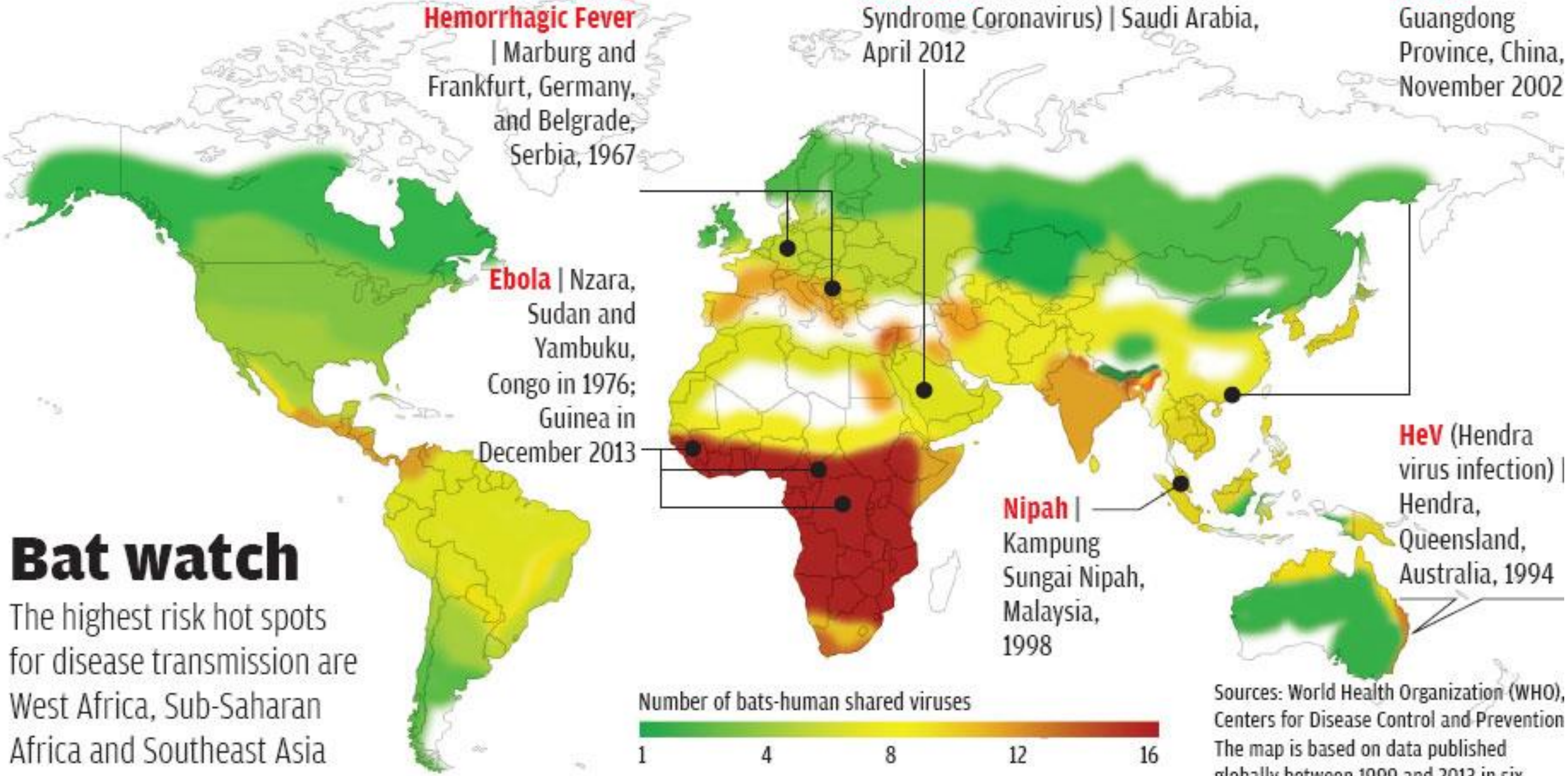
The next big killer?

SOURCES: WHO, CDC

H7N9	MERS-COV	NIPAH VIRUS
First emergence: 2013 Hosts: Chickens, ducks and pigeons	First emergence: 2012 Hosts: Suspected bats and camels	First emergence: 1998 Hosts: fruit bats and pigs
Cases: **137** Deaths: **45**	Cases: **94** Deaths: **47**	Cases: **280** Deaths: **211**

Bat watch

The highest risk hot spots for disease transmission are West Africa, Sub-Saharan Africa and Southeast Asia



Marburg Hemorrhagic Fever | Marburg and Frankfurt, Germany, and Belgrade, Serbia, 1967

MERS-CoV (Middle East Respiratory Syndrome Coronavirus) | Saudi Arabia, April 2012

SARS | Guangdong Province, China, November 2002

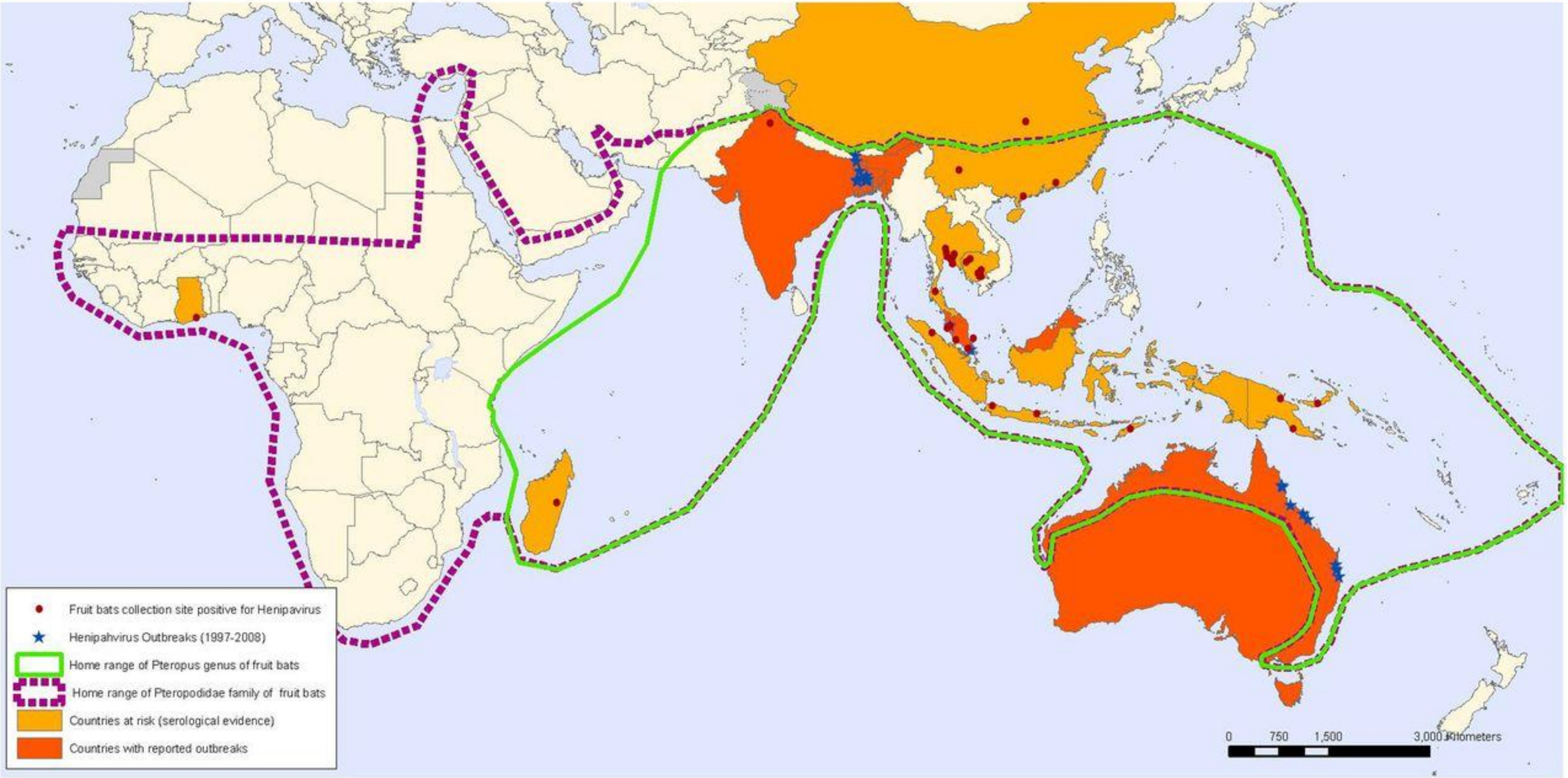
Ebola | Nzara, Sudan and Yambuku, Congo in 1976; Guinea in December 2013

HeV (Hendra virus infection) | Hendra, Queensland, Australia, 1994

Nipah | Kampung Sungai Nipah, Malaysia, 1998

Sources: World Health Organization (WHO), Centers for Disease Control and Prevention
The map is based on data published globally between 1999 and 2013 in six world regions, as grouped by WHO

Geographic distribution of Henipavirus outbreaks and fruit bats of Pteropodidae Family



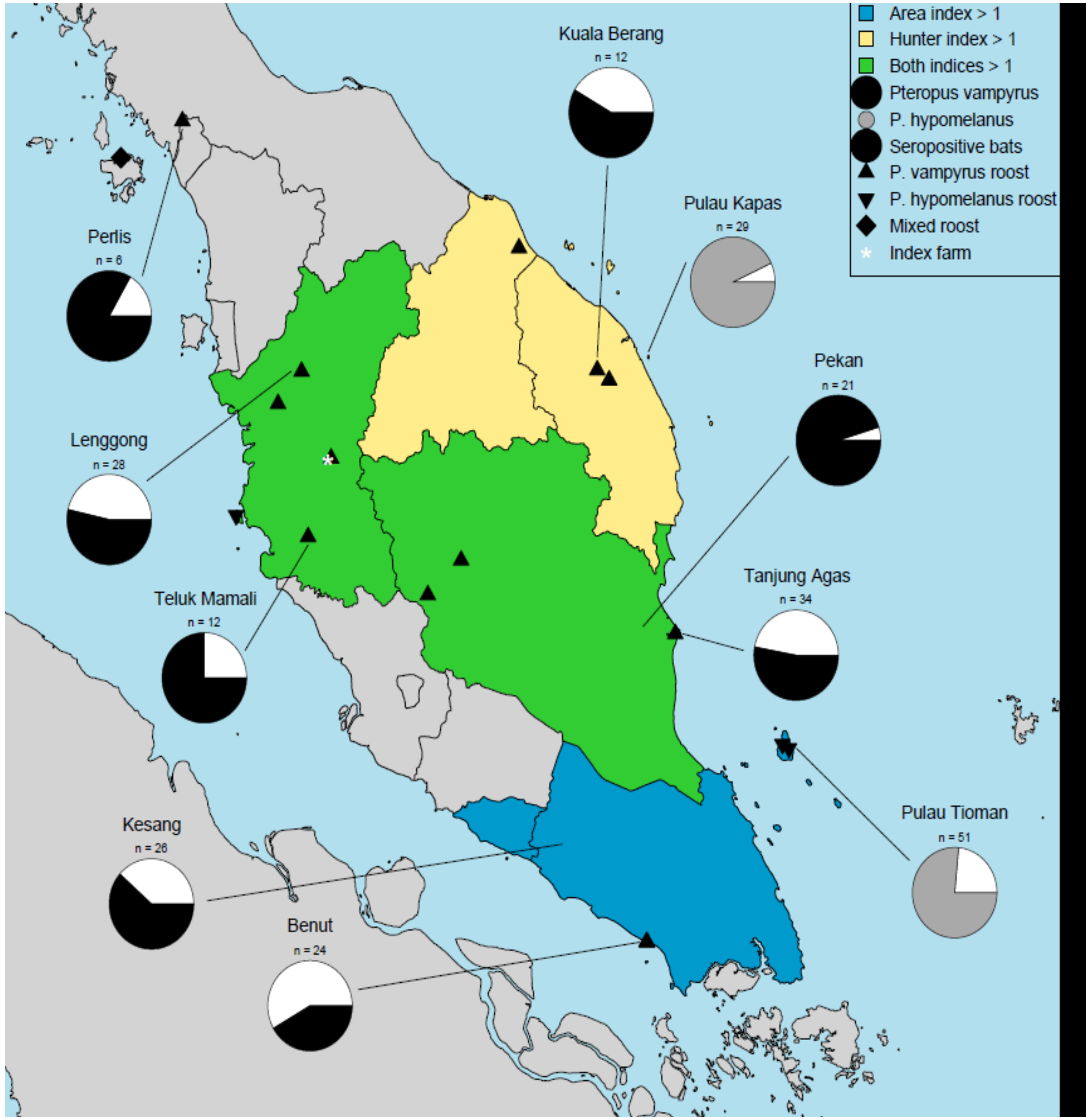
Data Source: Global Alert and Response Department
World Health Organization
Map Production: Public Health Information
and Geographic Information Systems (GIS)
World Health Organization

World Health Organization

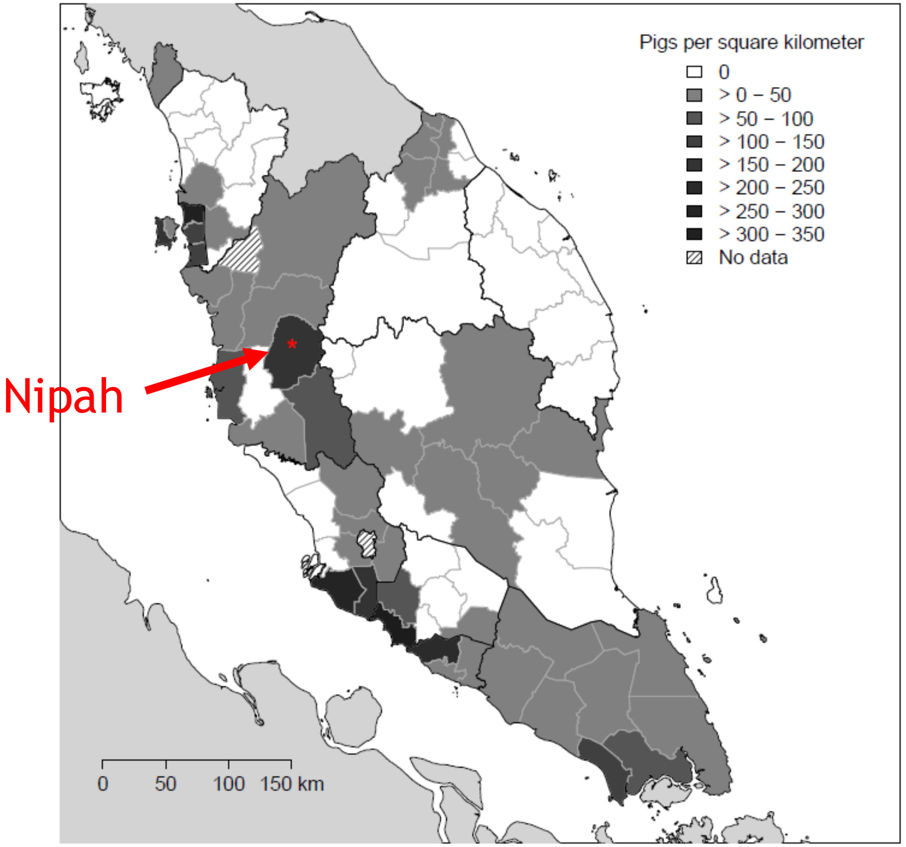
Zoom in on the farm at the center of the Nipah outbreak

The Malaysian epidemiologist, Dr K.W.Chang, who reported and investigated the outbreak

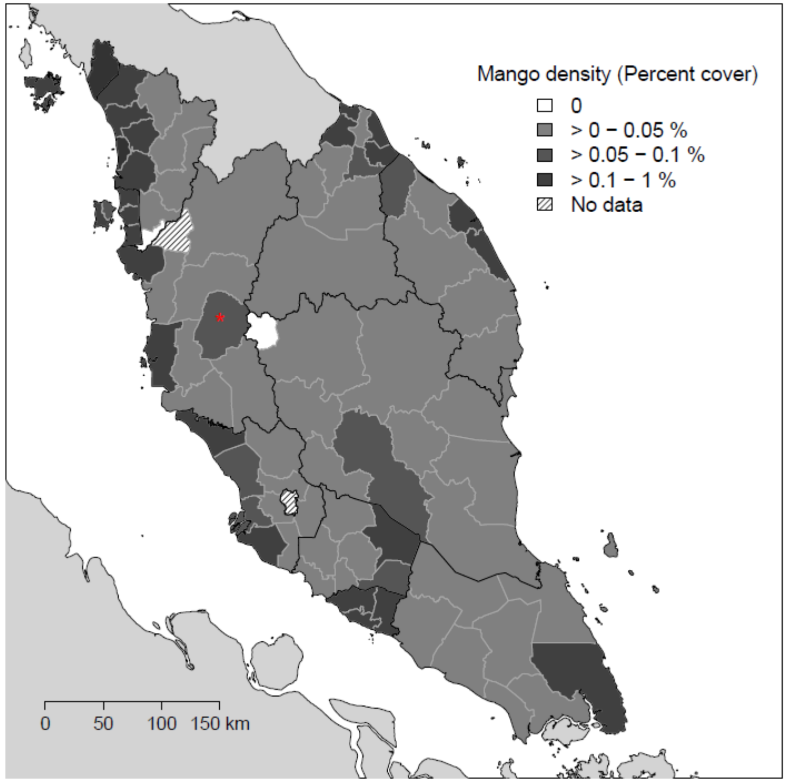
Nipah virus prevalence in bats



Pulliam et al 2008.

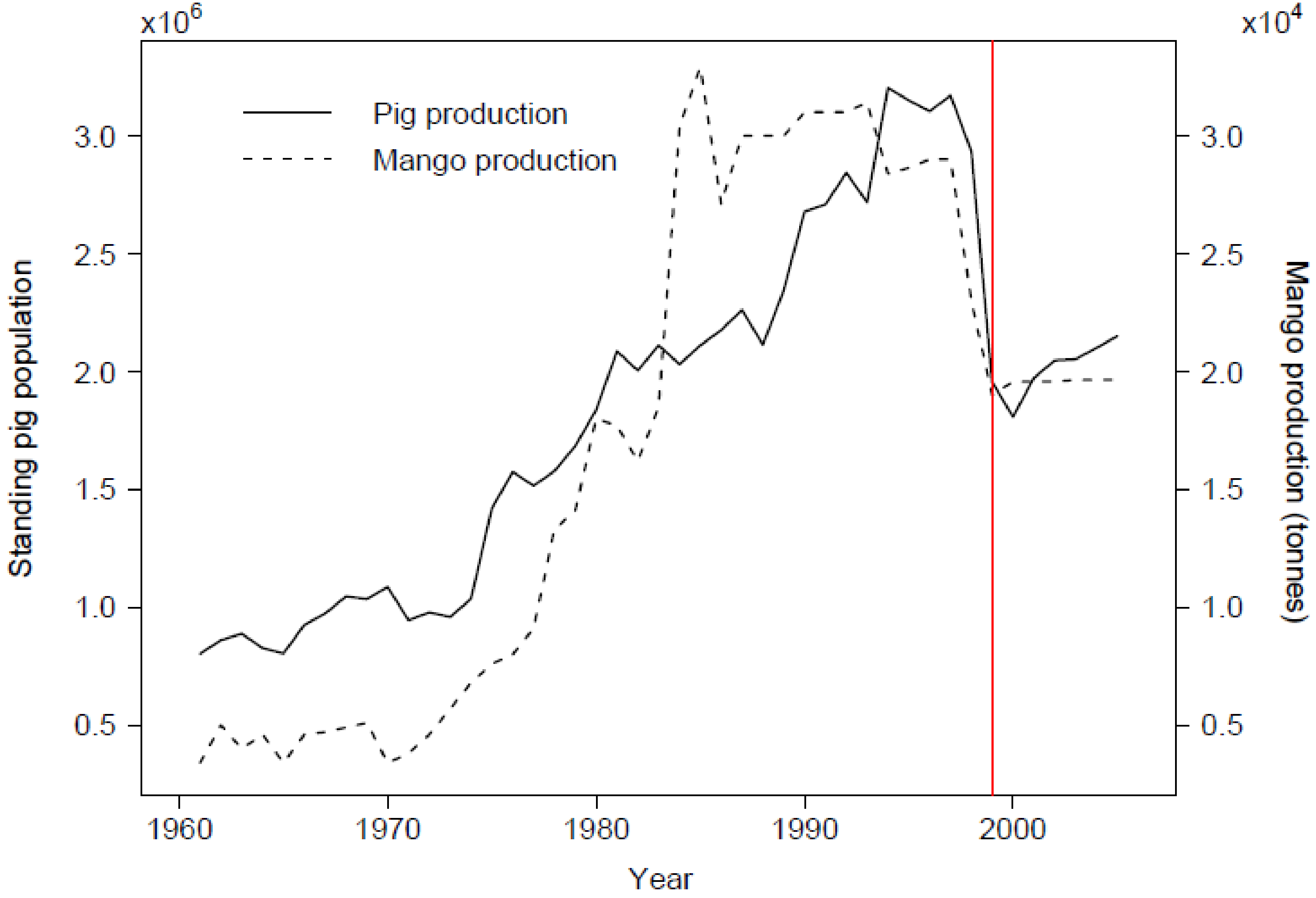


Pig density at the district level

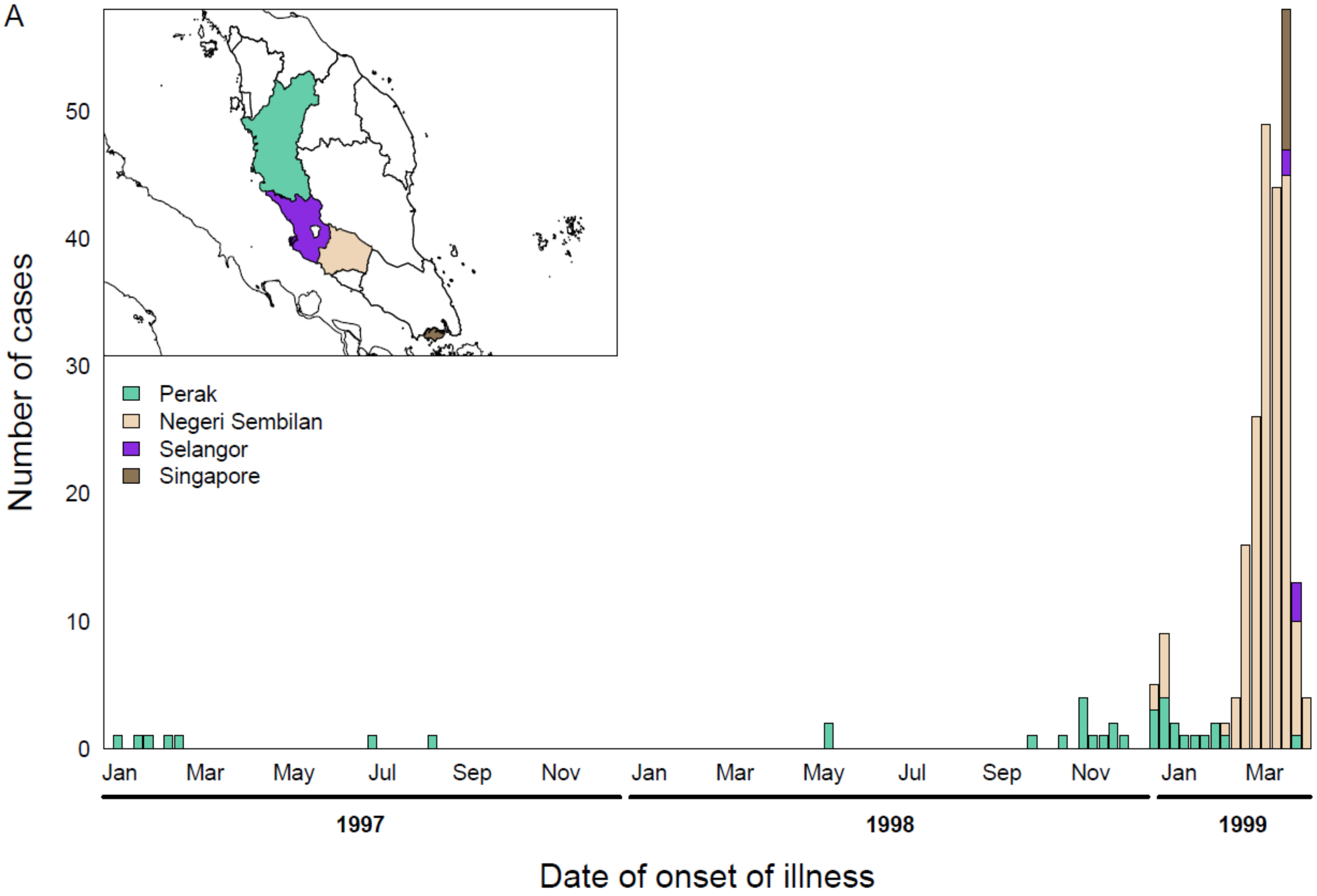


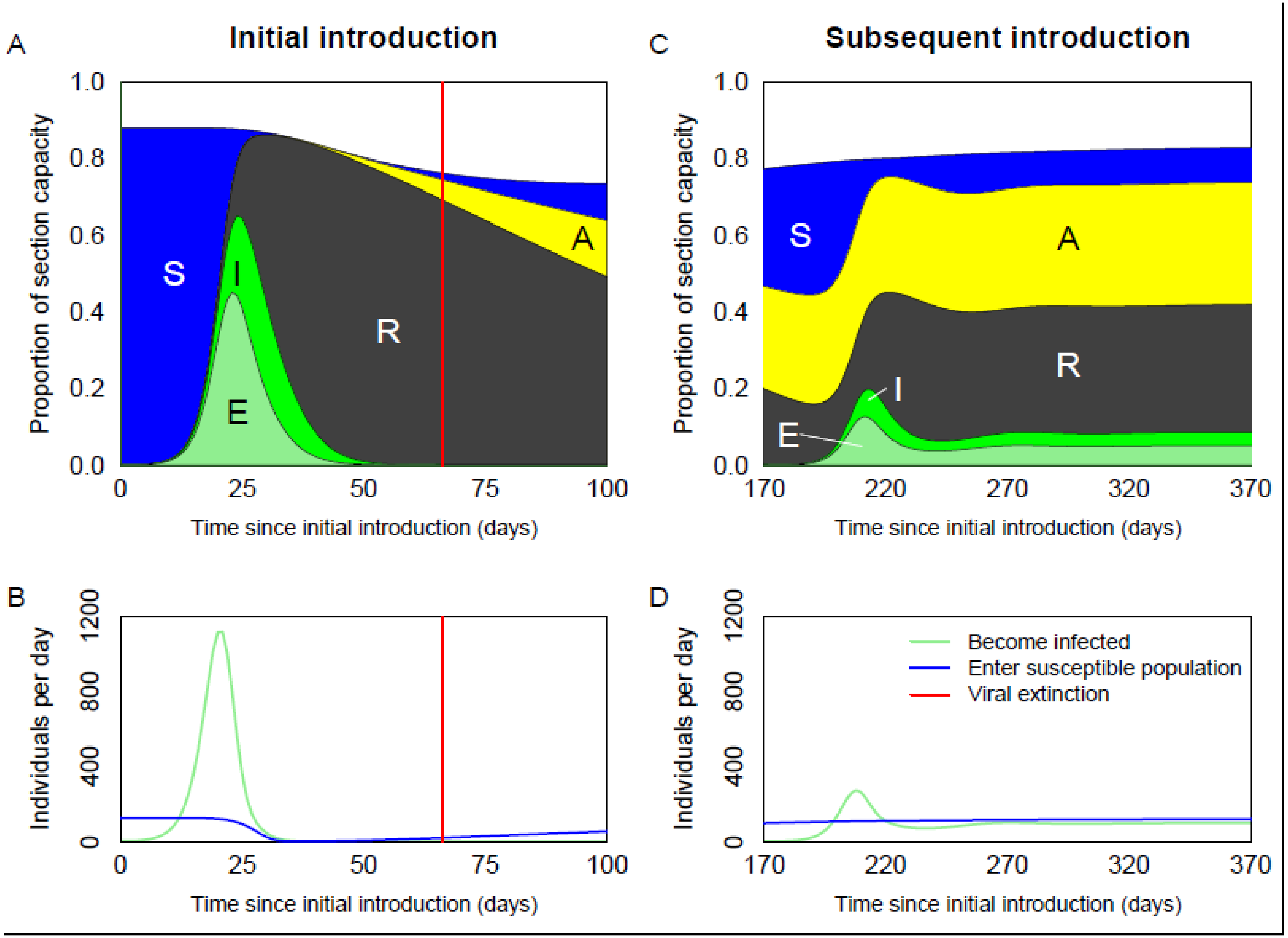
Mango density at the district level

Mango trees used to shade pig barns and provide a second crop to the farmer ……………….and additional food for the pigs

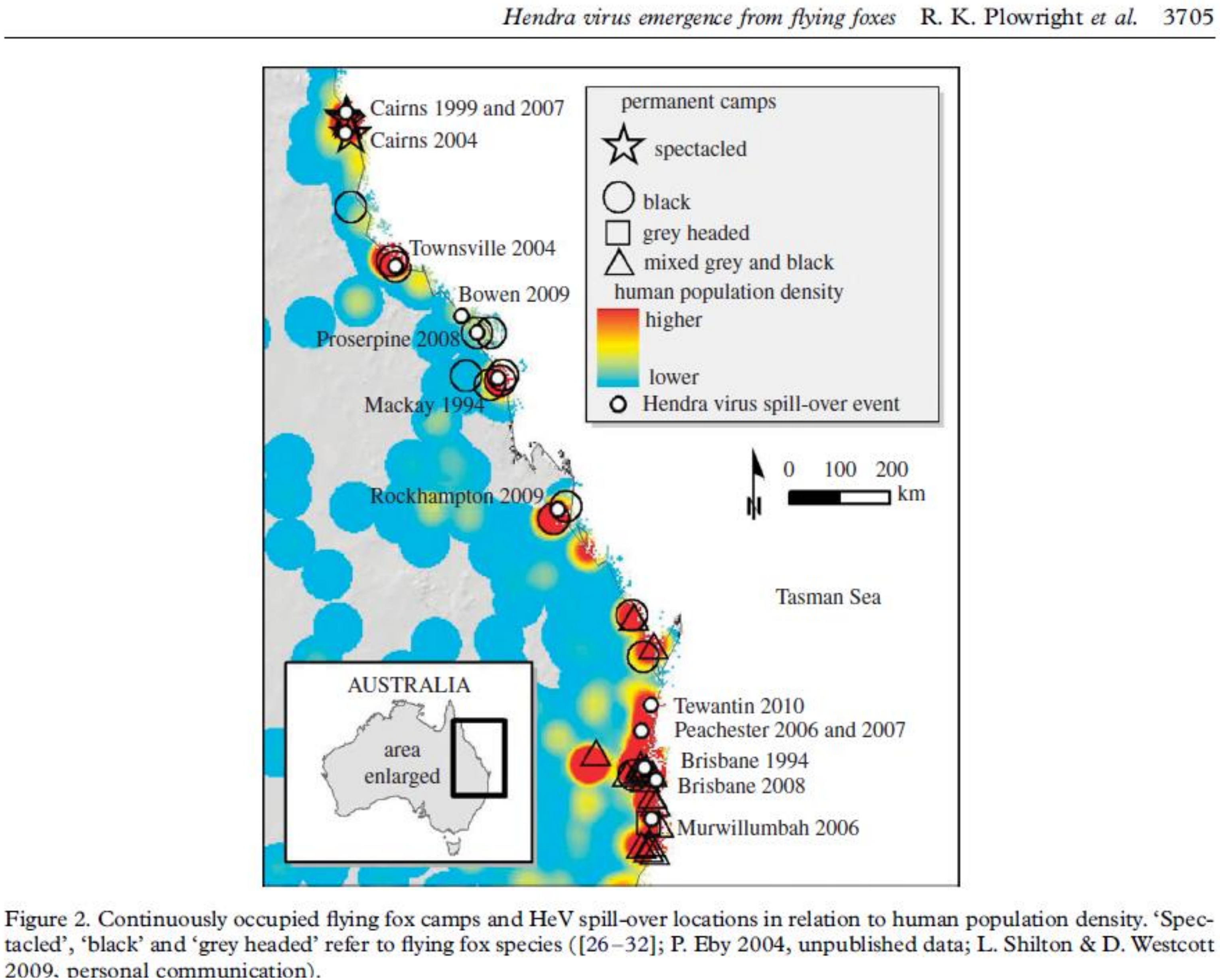


Nipah virus in Malaysia, Pulliam et al 2008





Dr Juliet Pulliam
Director, SACEMA
Stellenbosch
University
South Africa



Hendra virus emergence from flying foxes R. K. Plowright *et al.* 3705

Figure 2. Continuously occupied flying fox camps and HeV spill-over locations in relation to human population density. 'Spectacled', 'black' and 'grey headed' refer to flying fox species ([26–32]; P. Eby 2004, unpublished data; L. Shilton & D. Westcott 2009, personal communication).

Dr Raina Plowright,
Montana State,
Bozeman.

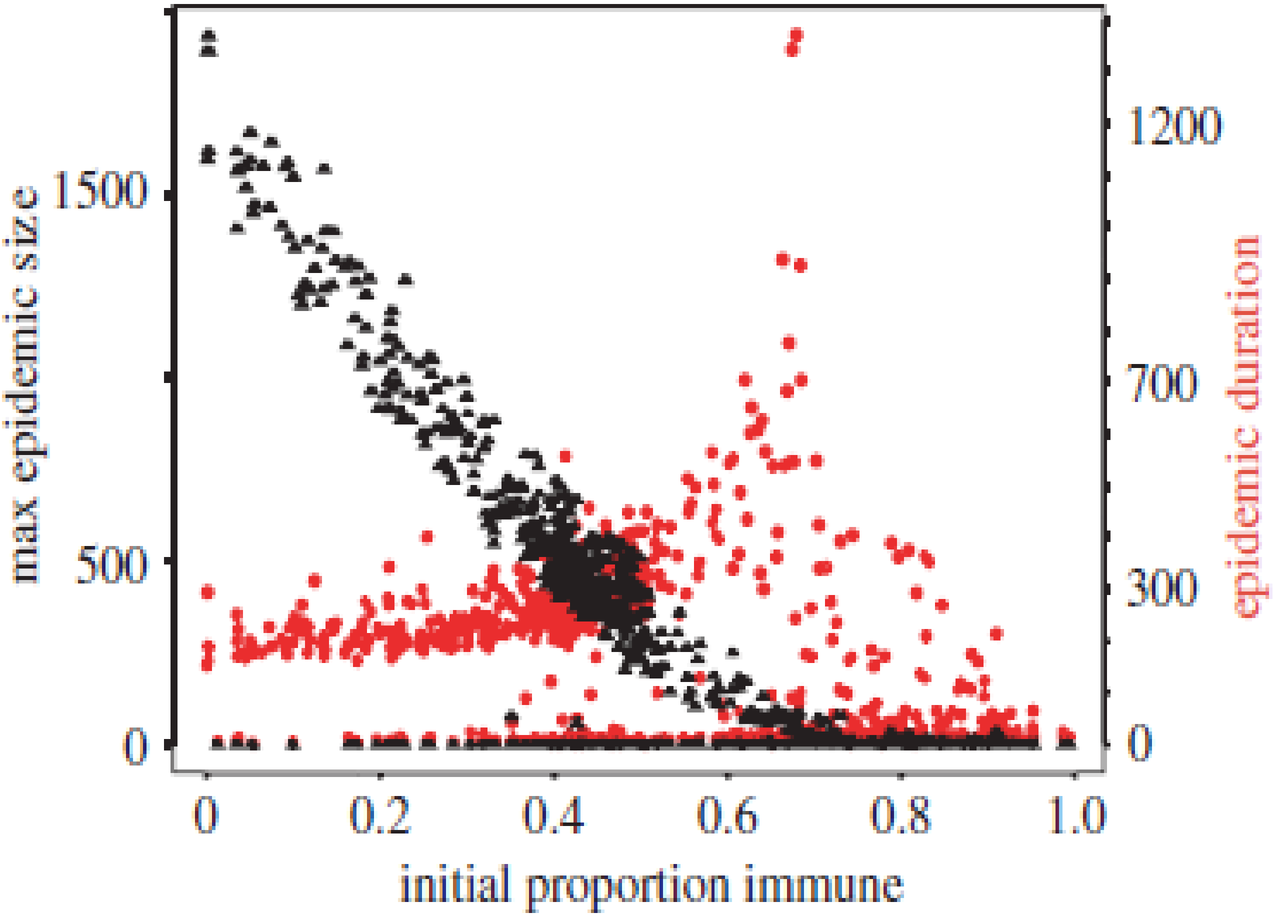
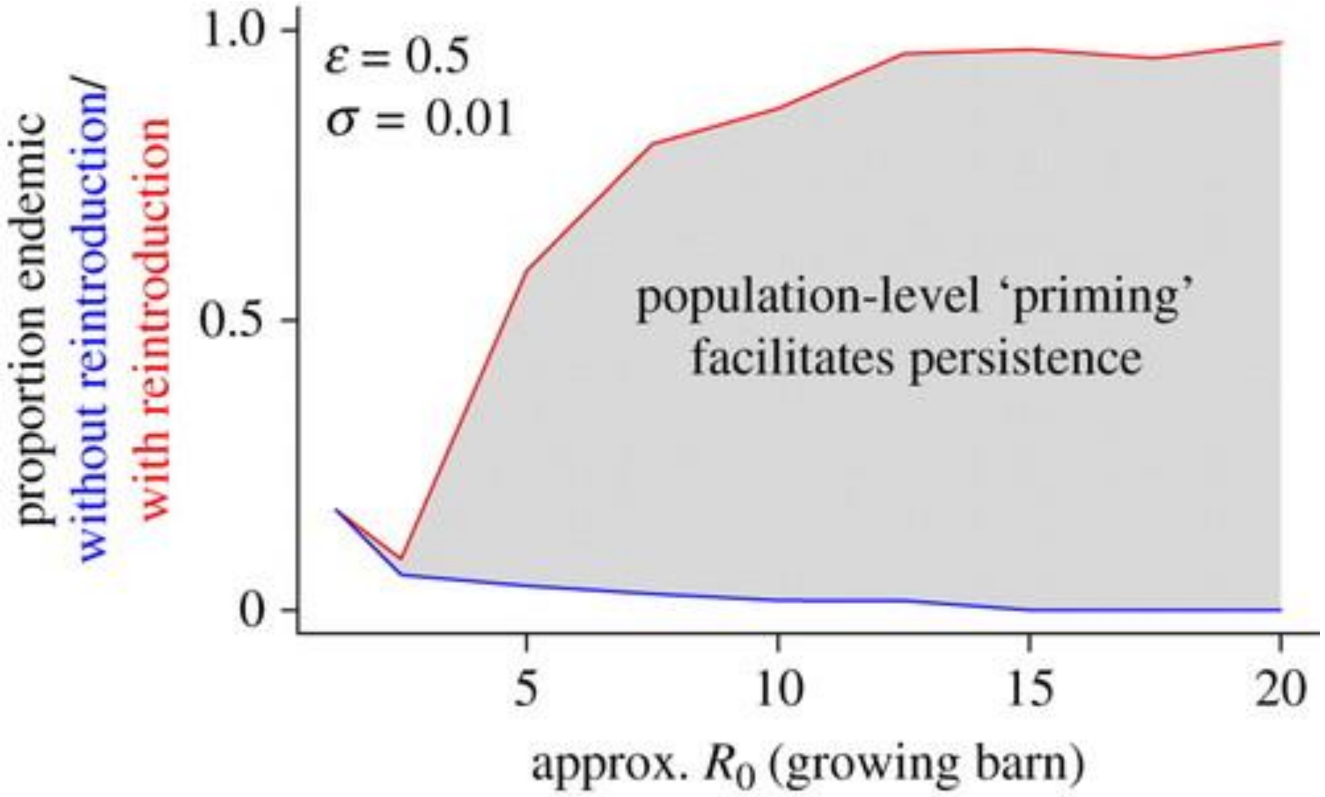


Figure 3. Herd immunity and epidemic size. Relationship between initial herd immunity, epidemic amplitude (black triangles) and epidemic duration (red circles) in a stochastic metapopulation simulation ($N_0 = 10\,000$, $\beta = 4.76\mathrm{E}-05$, $\gamma = 0.143$, $h = 200$, $c = 0.16$). The deterministic threshold number of susceptibles required for disease invasion in this model system is approximately 3000 (initial proportion immune = 0.7). When virus is introduced into a population with initial herd immunity approaching the threshold for invasion, low amplitude, persistent smouldering epidemics may result. When virus is introduced into a more susceptible population, high amplitude, shorter epidemics may result.



Agricultural intensification, priming for persistence and the emergence of Nipah virus: a lethal bat-borne zoonosis, Volume: 9, Issue: 66, Pages: 89-101, DOI: (10.1098/rsif.2011.0223)

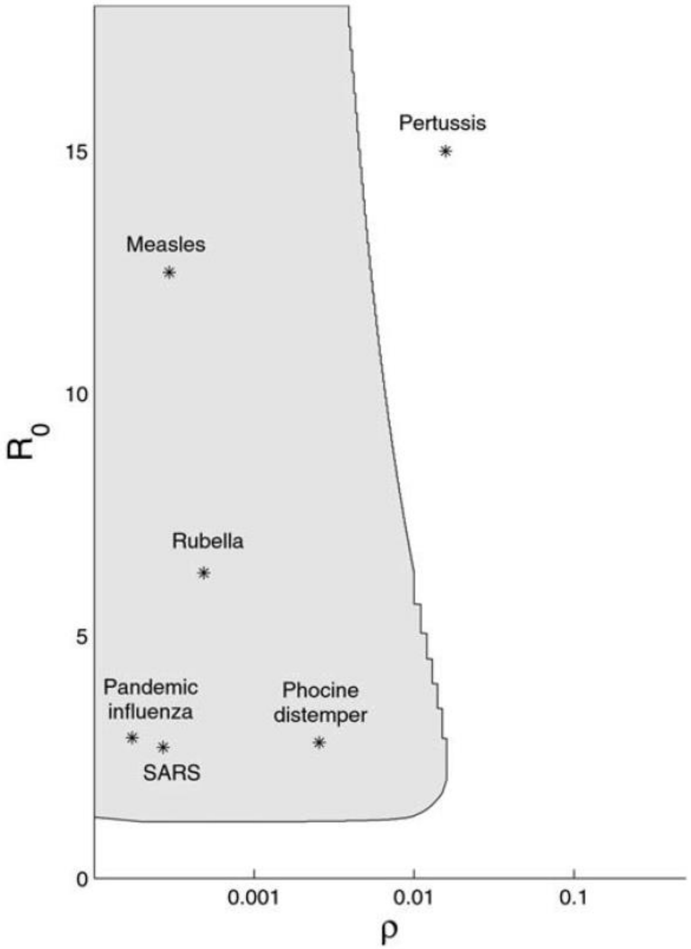
Pulliam et al (2011) Royal Society Interface.

OPEN ACCESS Freely available online PLoS one

Epidemic Enhancement in Partially Immune Populations

Juliet R. C. Pulliam[1*], Jonathan G. Dushoff[1,2], Simon A. Levin[1], Andrew P. Dobson[1]

1 Department of Ecology and Evolutionary Biology, Princeton University, Princeton, New Jersey, United States of America, 2 Fogarty International Center, National Institutes of Health, Bethesda, Maryland, United States of America



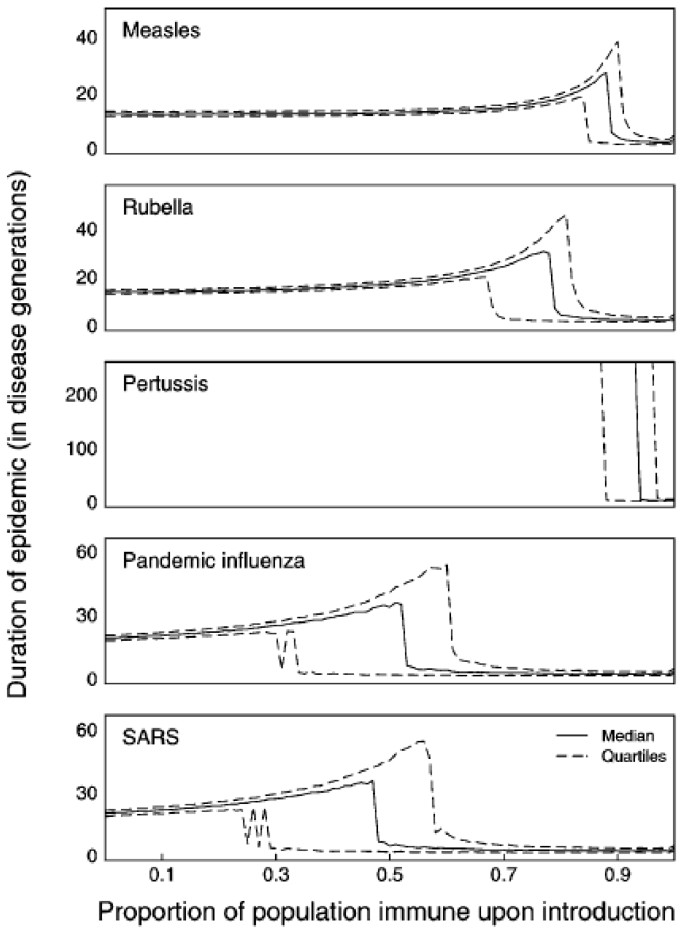


Figure 3. Enhancement of epidemic duration for diseases in human populations. Epidemic duration in a population of $N = 50{,}000$ individuals for a variety of human pathogens as a function of population immunity at introduction. Solid lines show the median duration in disease generations for 1,000 simulation runs at each level of initial population immunity; dashed lines show quartiles. Each pathogen shows some level of enhancement of epidemic duration with increased immunity except pertussis. Enhancement of epidemic size is not observed for these pathogens for $N = 50{,}000$.
doi:10.1371/journal.pone.0000165.g003

p, is the ratio of duration of infectiousness to duration of immunity

Rondonia, Brazil, 1975 – 2010 (NASA, Visible Earth)

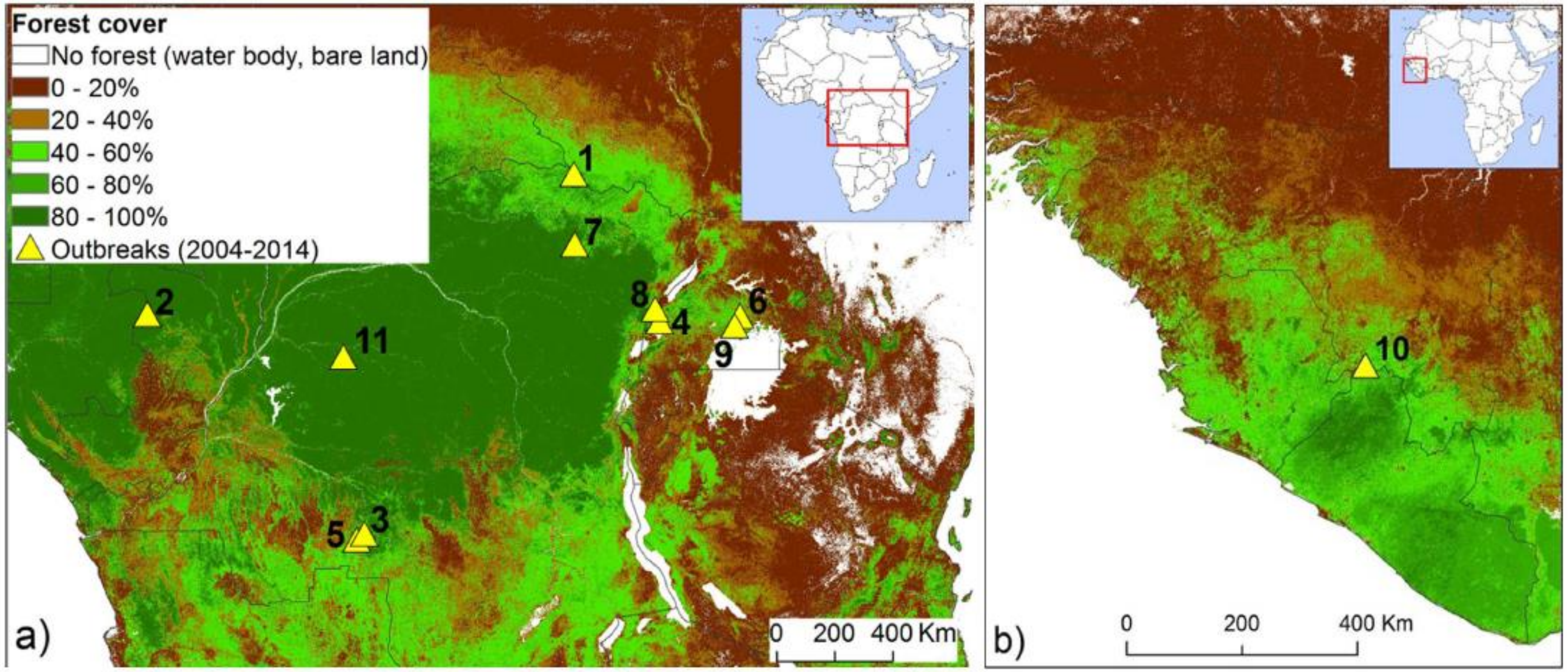


Figure 1. Forest cover maps and locations of first infection events in humans. Forest cover maps and locations of independent first infection events in humans (triangles, see Table 1) in Central (**a**) and West (**b**) Africa. The insets indicate the two African regions considered in this study. Legend 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

OPEN

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]

Sites of Ebola outbreaks in Central and West Africa

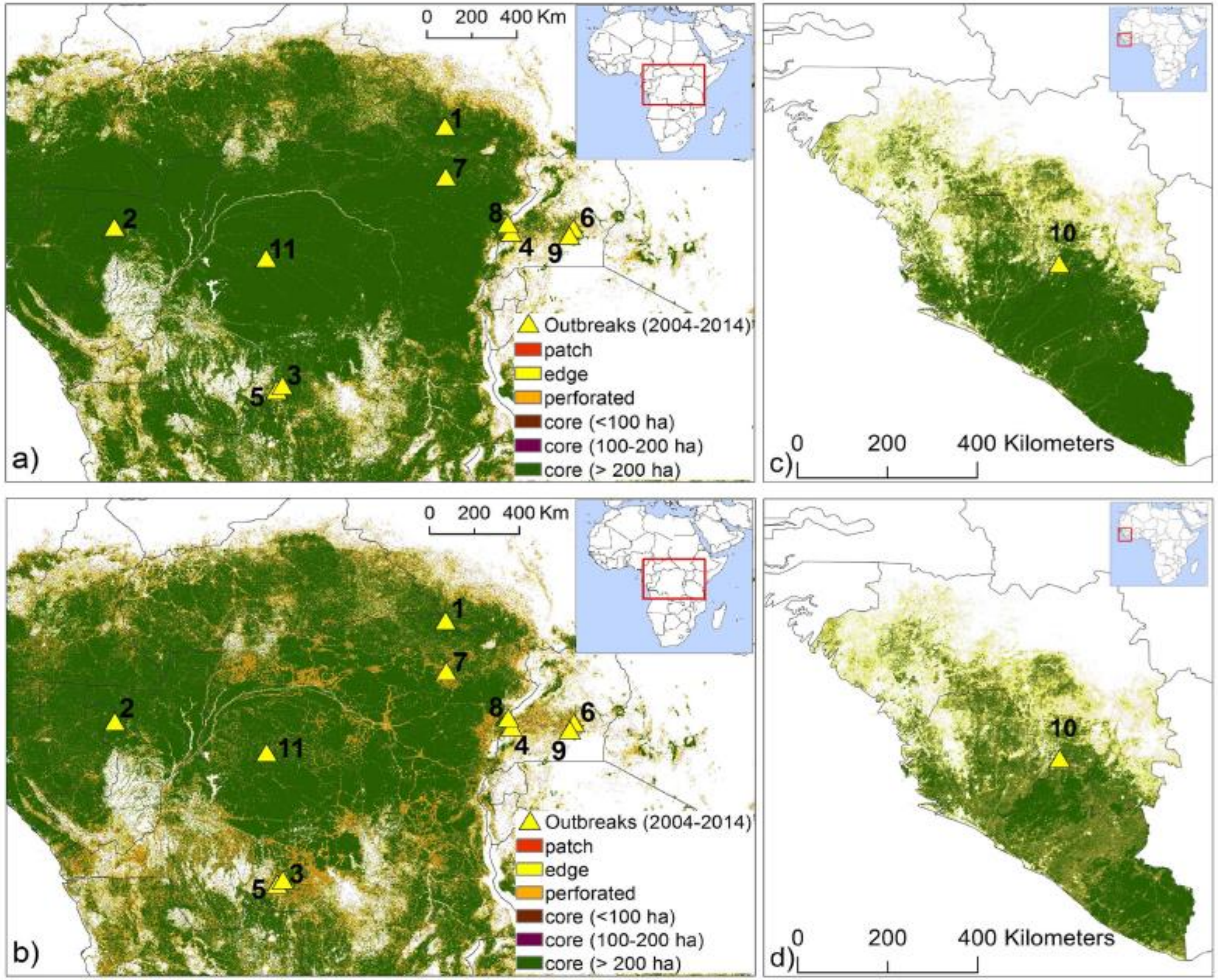
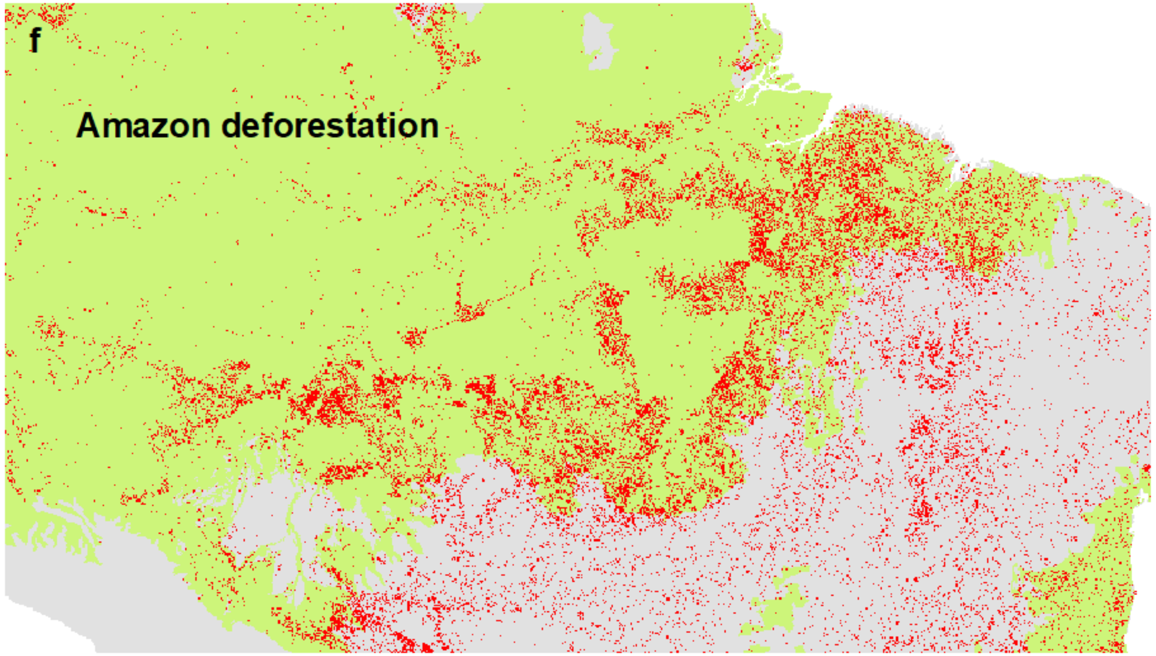
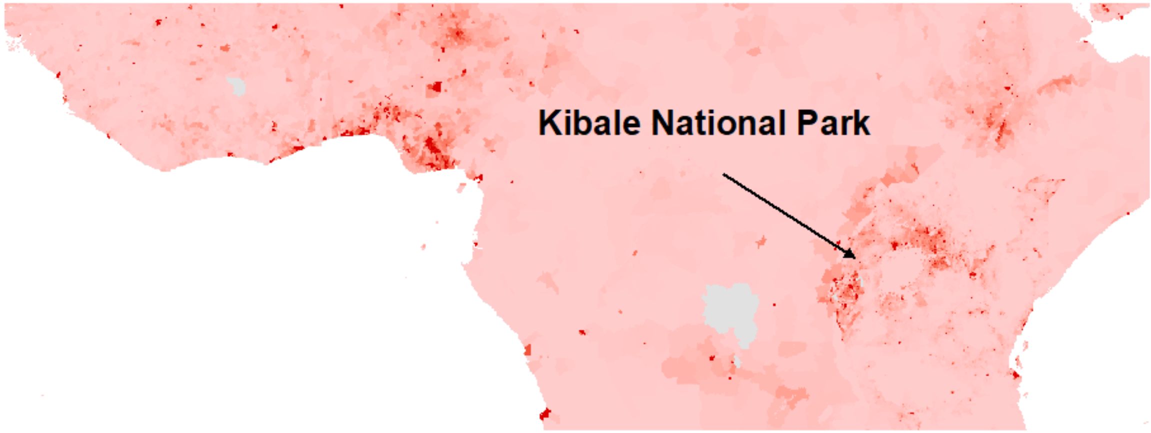


Figure 2. Forest fragmentation in Central and West Africa. Forest fragmentation in Central (panels a, and b)

Rulli et al. Sci Reports 2017.


f
Amazon deforestation


Kibale National Park

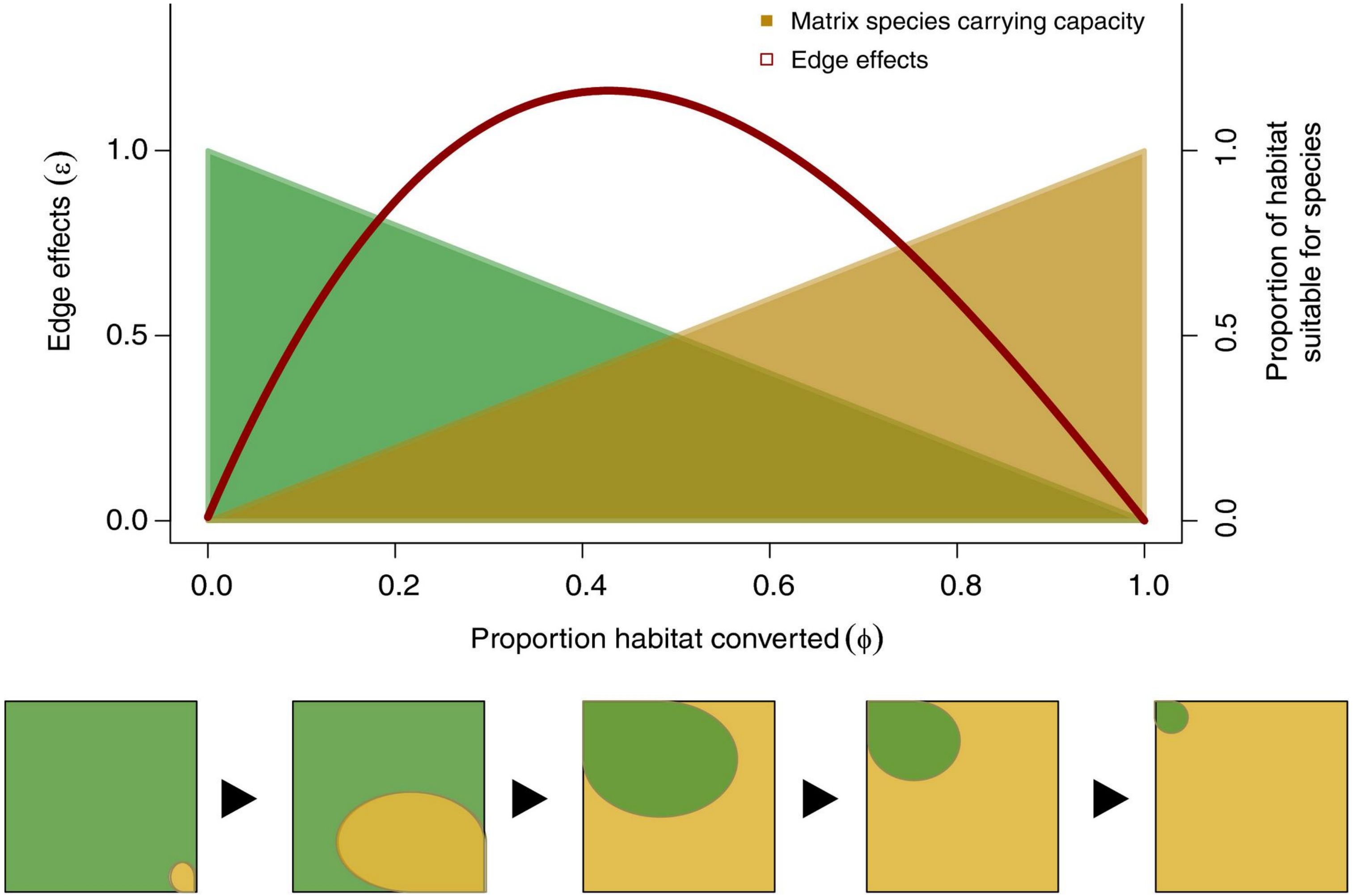
What happens to pathogens in the remaining patches of forest?

Null expectations for disease dynamics in shrinking habitat: dilution or amplification?

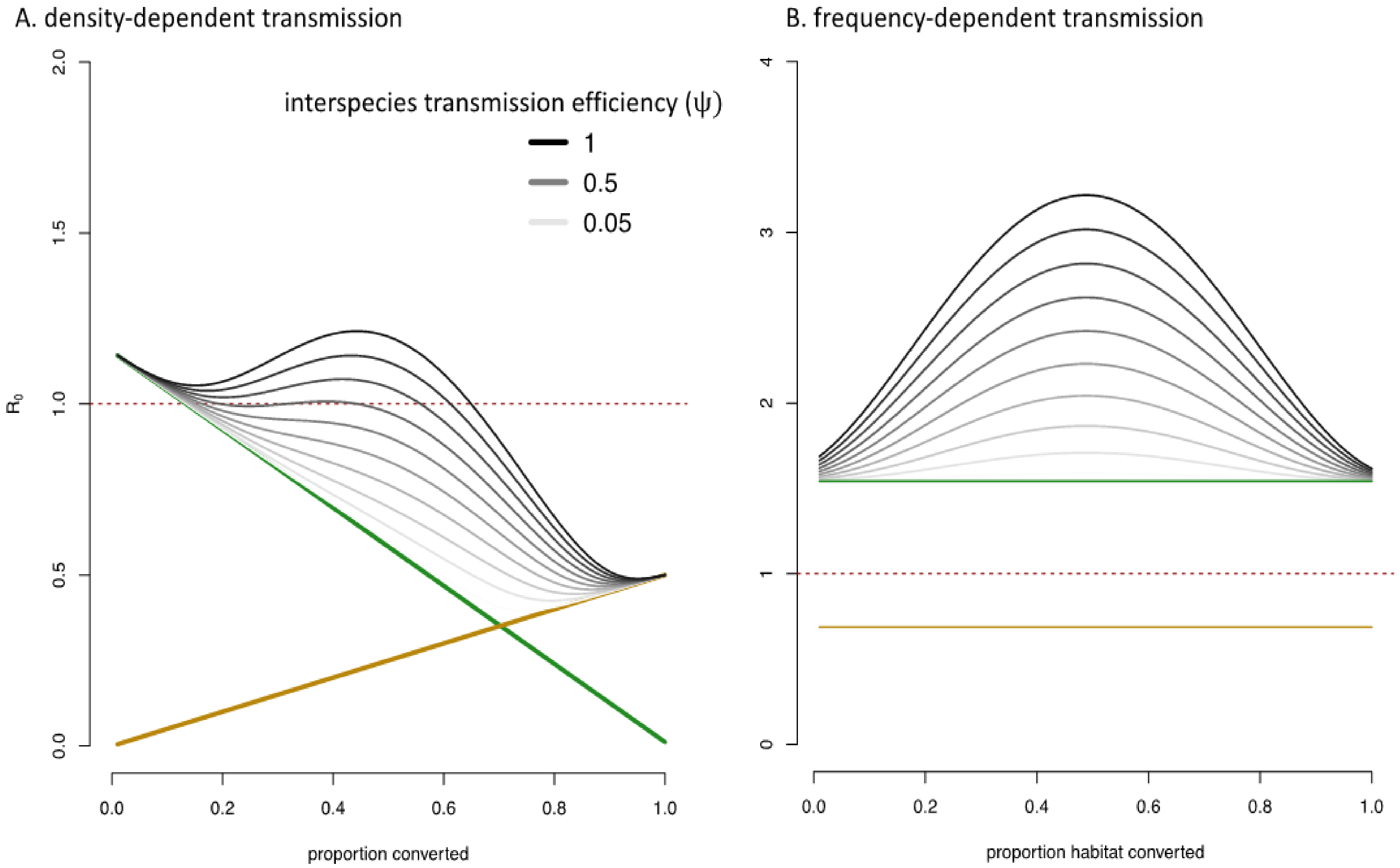
Christina L. Faust[1,2], Andrew P. Dobson[1], Nicole Gottdenker[3], Laura S.P. Bloomfield[4], Hamish I. McCallum[5], Thomas R. Gillespie[6], Maria Diuk-Wasser[7], and Raina K. Plowright[2]

Phil Trans Roy Soc (2017)

Faust,
McCallum,
Bloomfield,
Dobson ,
Plowright et al
(Ecology Letters,
2017)



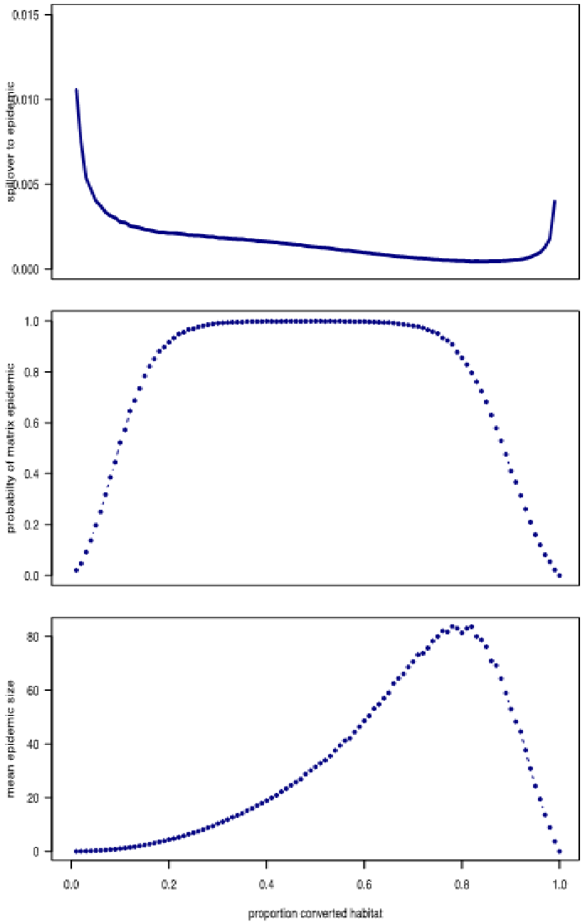
Transmission across the core-matrix boundary
Faust, McCallum, Dobson et al, Plowright, Ecology Letters (2017)

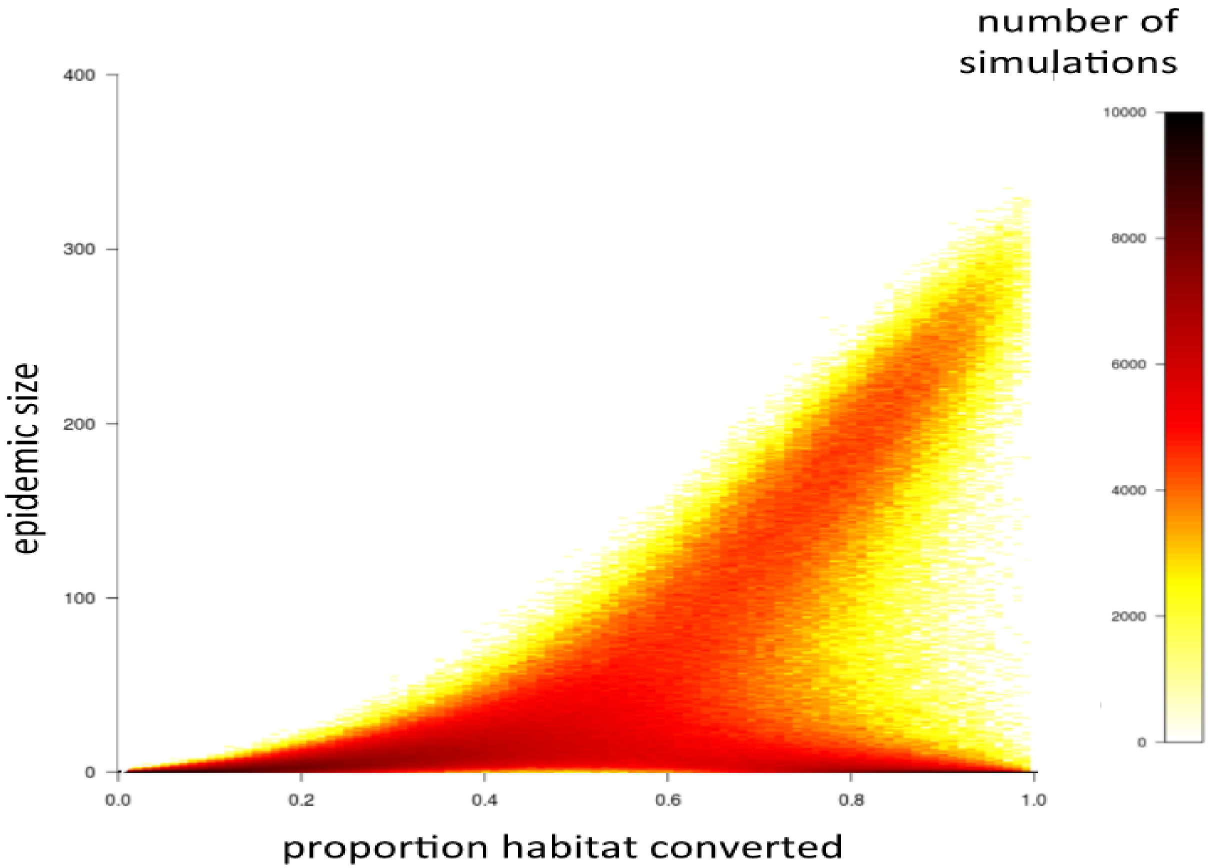


Transmission across the core-matrix boundary Gillespie stochastic case

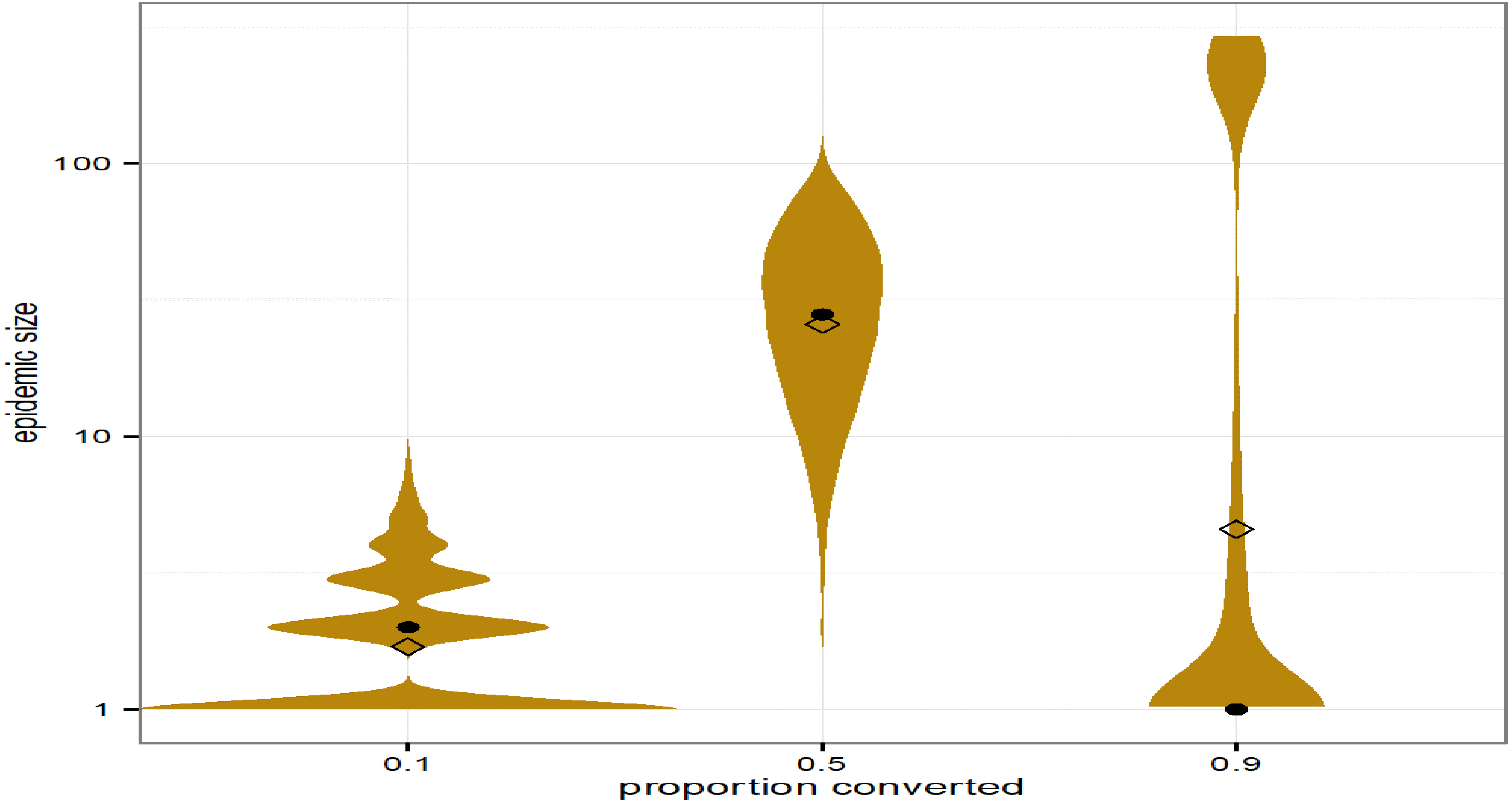
Faust, McCallum, Dobson et al, Plowright,

Ecology Letters 2017





Transmission across the core-matrix boundary
Faust, McCallum, Dobson et al, Plowright.
Ecology Letters, 2016.



LETTER
doi:10.1038/nature22975

Host and viral traits predict zoonotic spillover from mammals

Kevin J. Olival[1], Parviez R. Hosseini[1], Carlos Zambrana-Torrelio[1], Noam Ross[1], Tiffany L. Bogich[1] & Peter Daszak[1]

LETTER RESEARCH

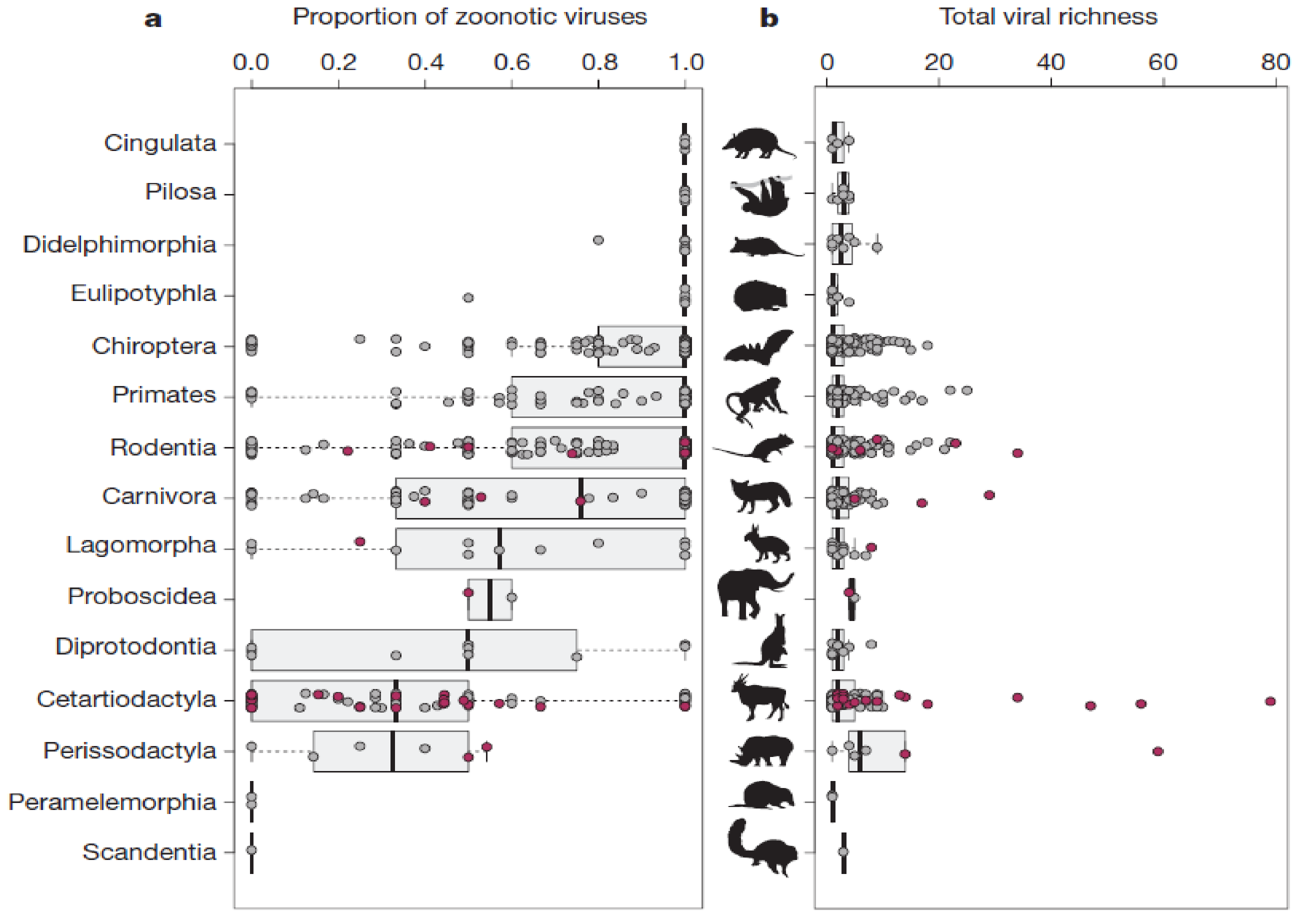
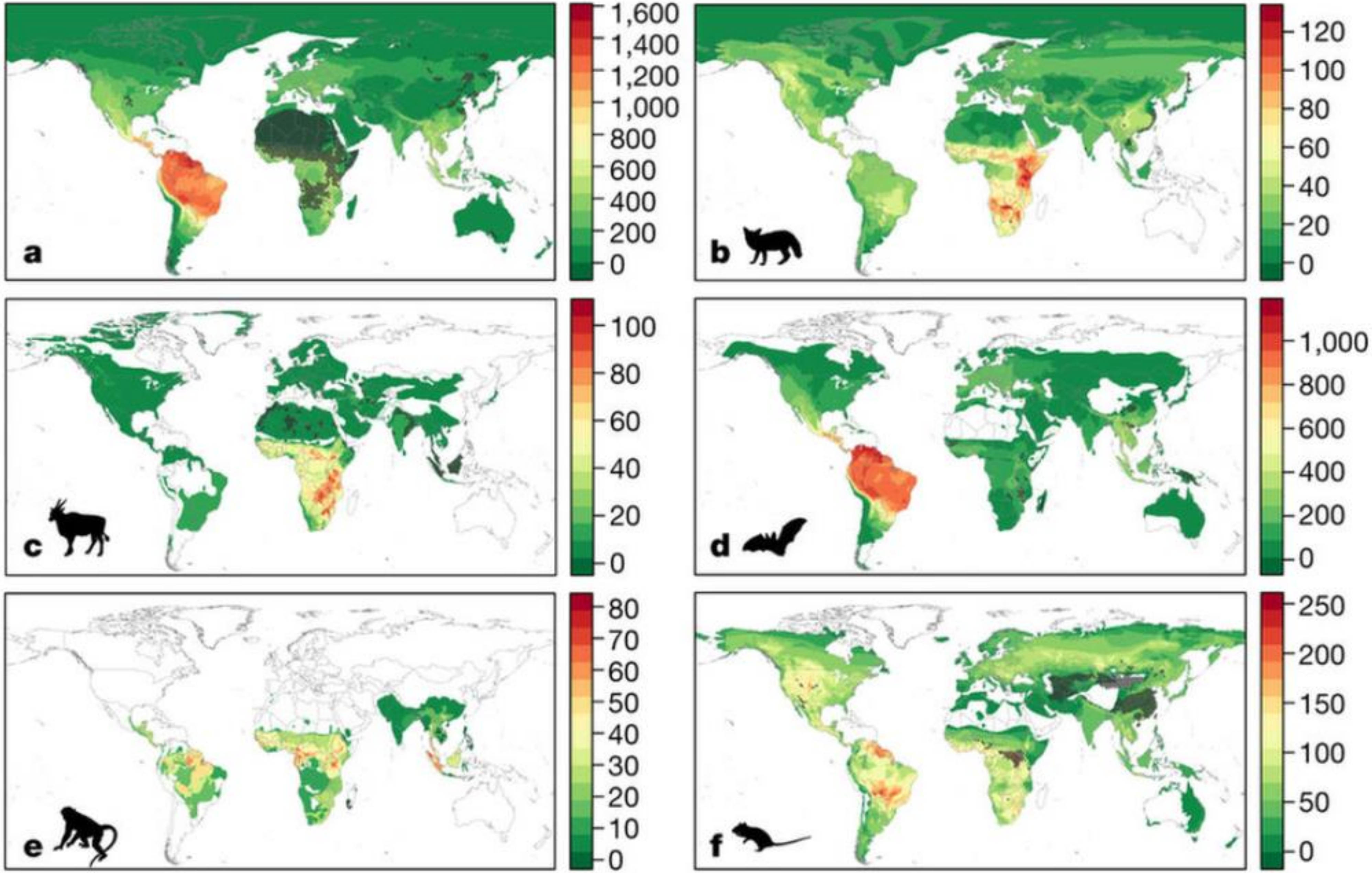


Figure 1 | Observed viral richness in mammals. a, b, Box plots of proportion of zoonotic viruses (**a**) and total viral richness per species (**b**), aggregated by order. Data points represent wild (light grey, $n = 721$) and domestic (dark red, $n = 32$) mammal species; lines represent median, boxes, interquartile range. Animal silhouettes from PhyloPic. Data based on 2,805 host–virus associations. See Methods for image credits and licensing.


a
1,600
1,400
1,200
1,000
800
600
400
200
0
b
120
100
80
60
40
20
0
c
100
80
60
40
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d
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800
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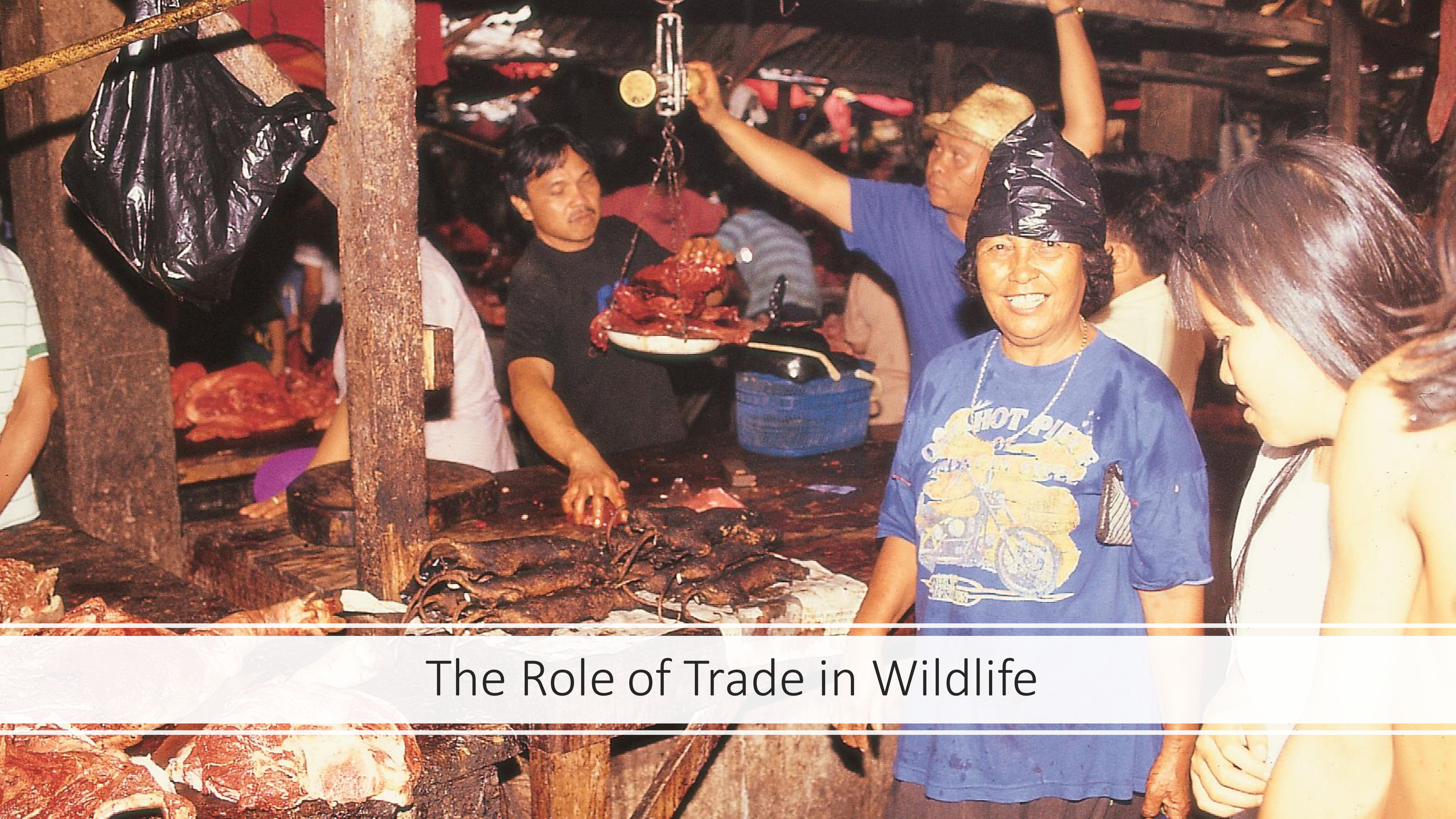


The Role of Trade in Wildlife

Legal registered wildlife trade through Singapore, 1976 – 2015.

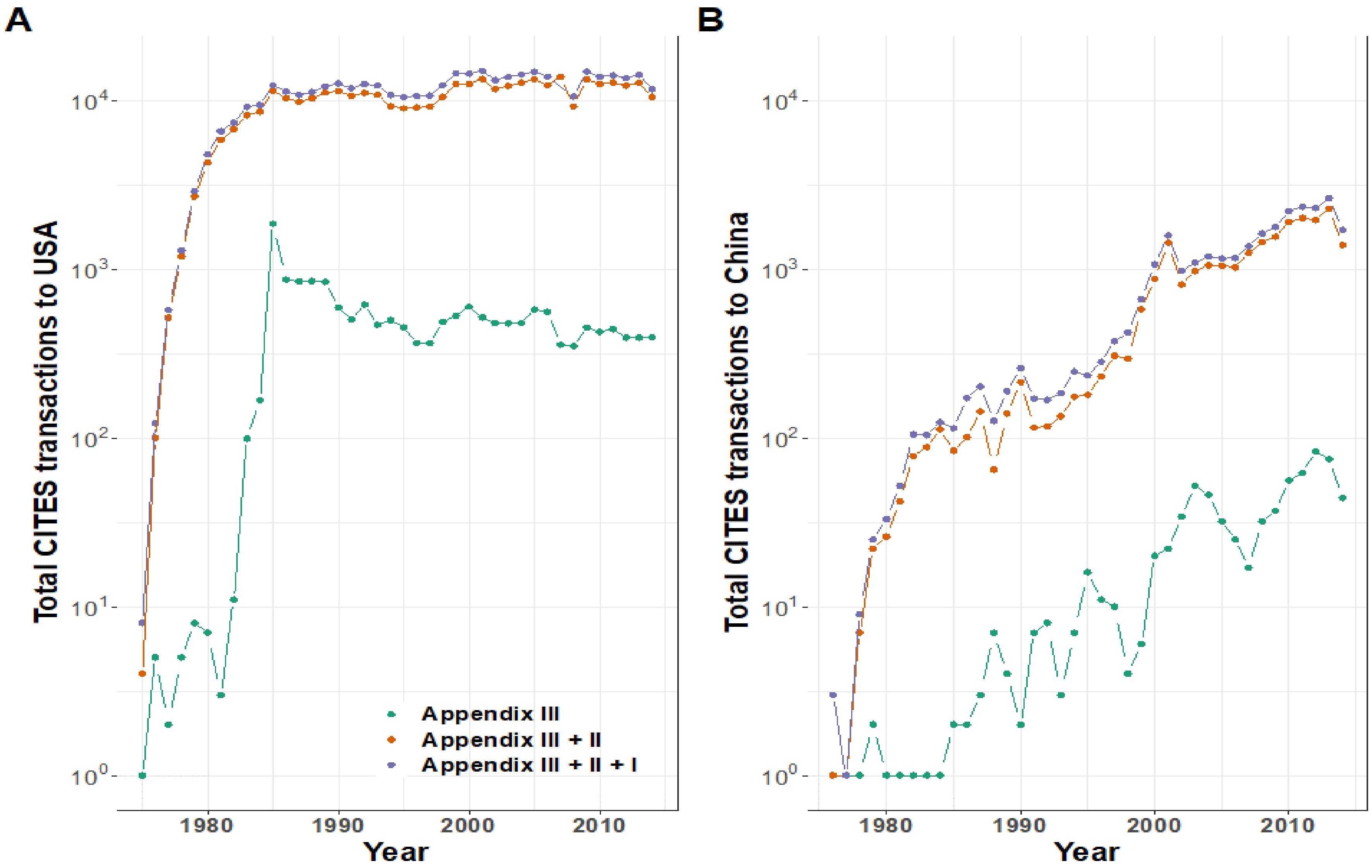


Figure 2 Annual animal imports from Singapore to the United States (A) and China (B) as recorded by CITES (Convention on International Trade in Endangered Species) The data start when CITES was first set up 1975. Data are the cumulative number of transactions (within each year), for Species listed under Appendix I, II and III. The records are predominantly for mammals, birds, fish and reptiles. They do not include fish harvested for food.

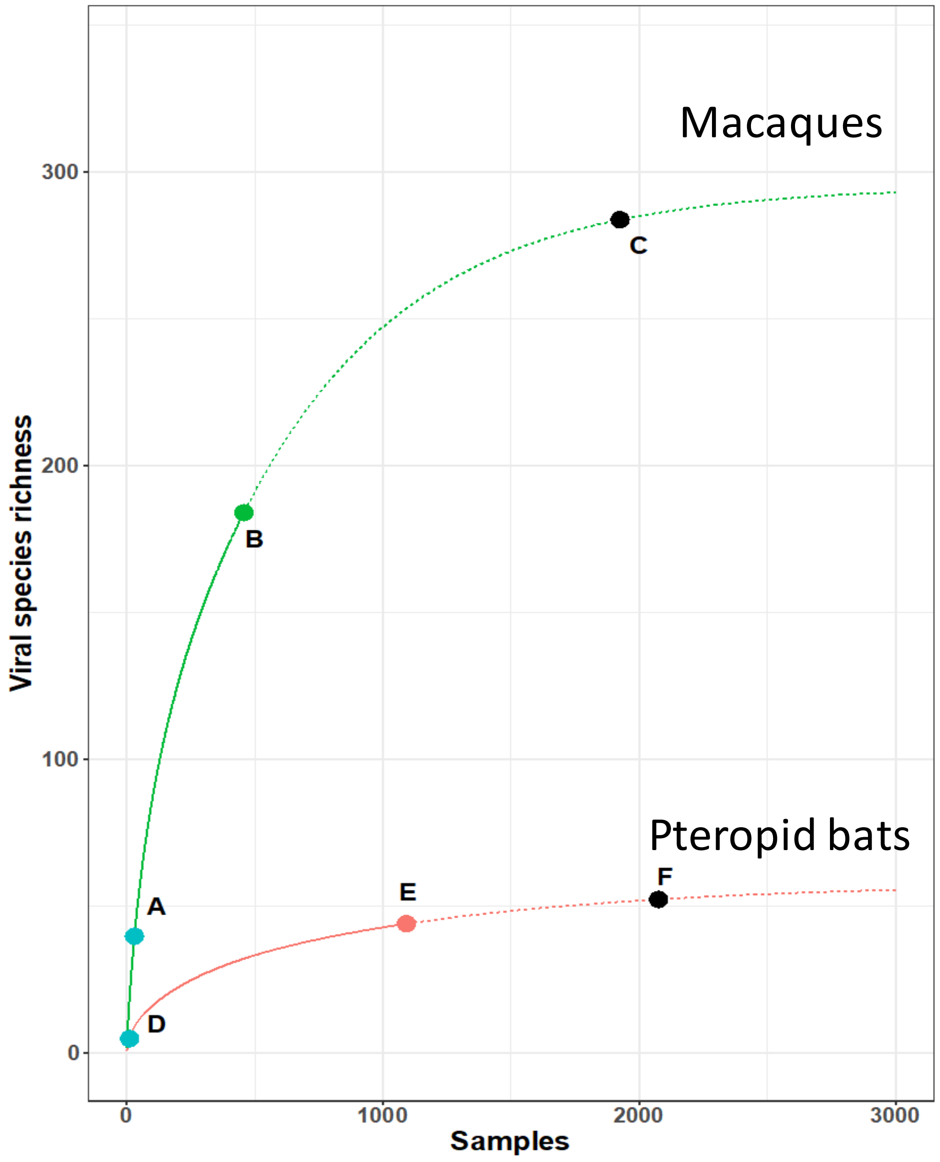
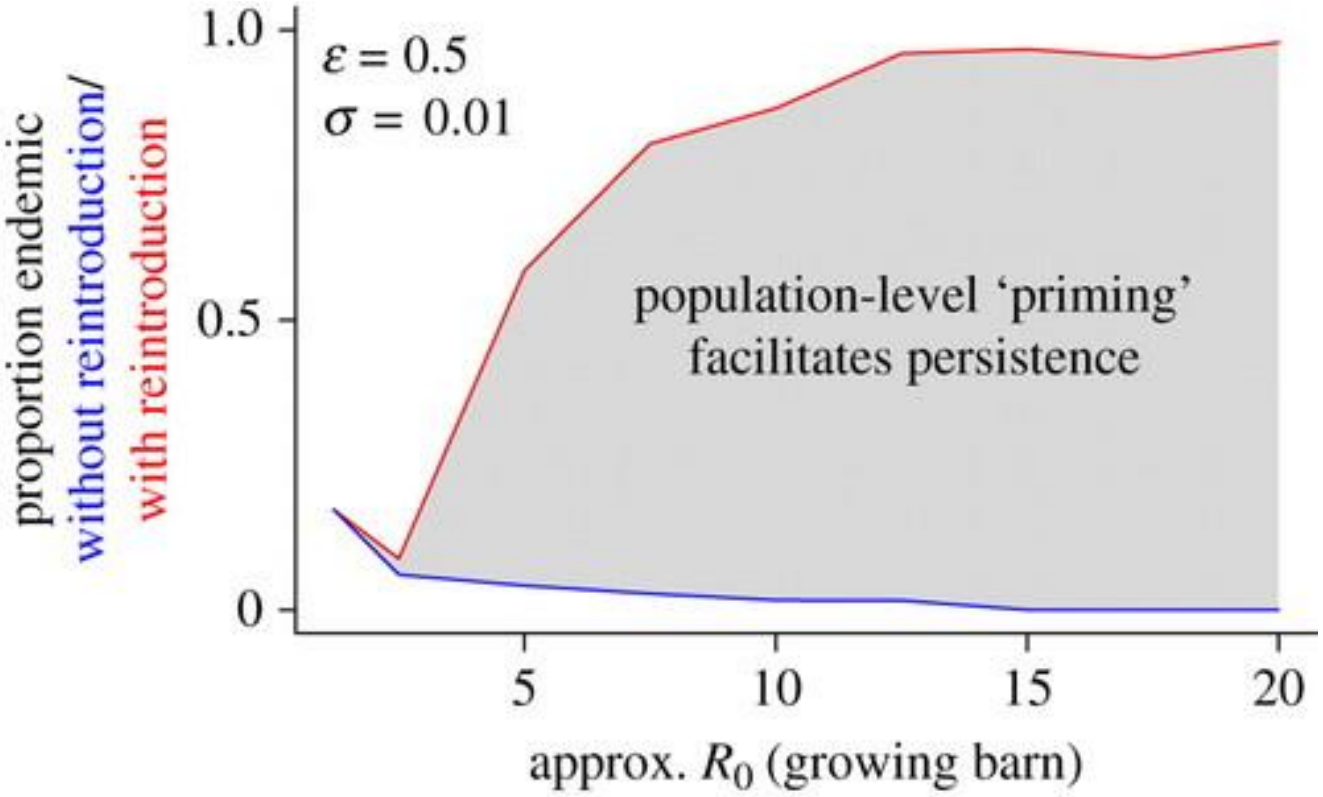


Figure 3 The amount of viral species richness discovered as the number of animals sampled increases for macaque monkeys (green) and Pteropid bats (red). Solid lines are based on rarefaction; dotted lines are extrapolations (using double sample size). Dots A (samples 31, richness 39) and D (samples 13, richness 5) represent 50% sample of sample coverage and dots C (samples 1925, richness 284) and F (samples 2075 and richness52) represent 99% of sample coverage. Dots B and E are the observed viral species richness. Shaded areas represent 95% confidence intervals. Data used for plots obtained from (20, 21).

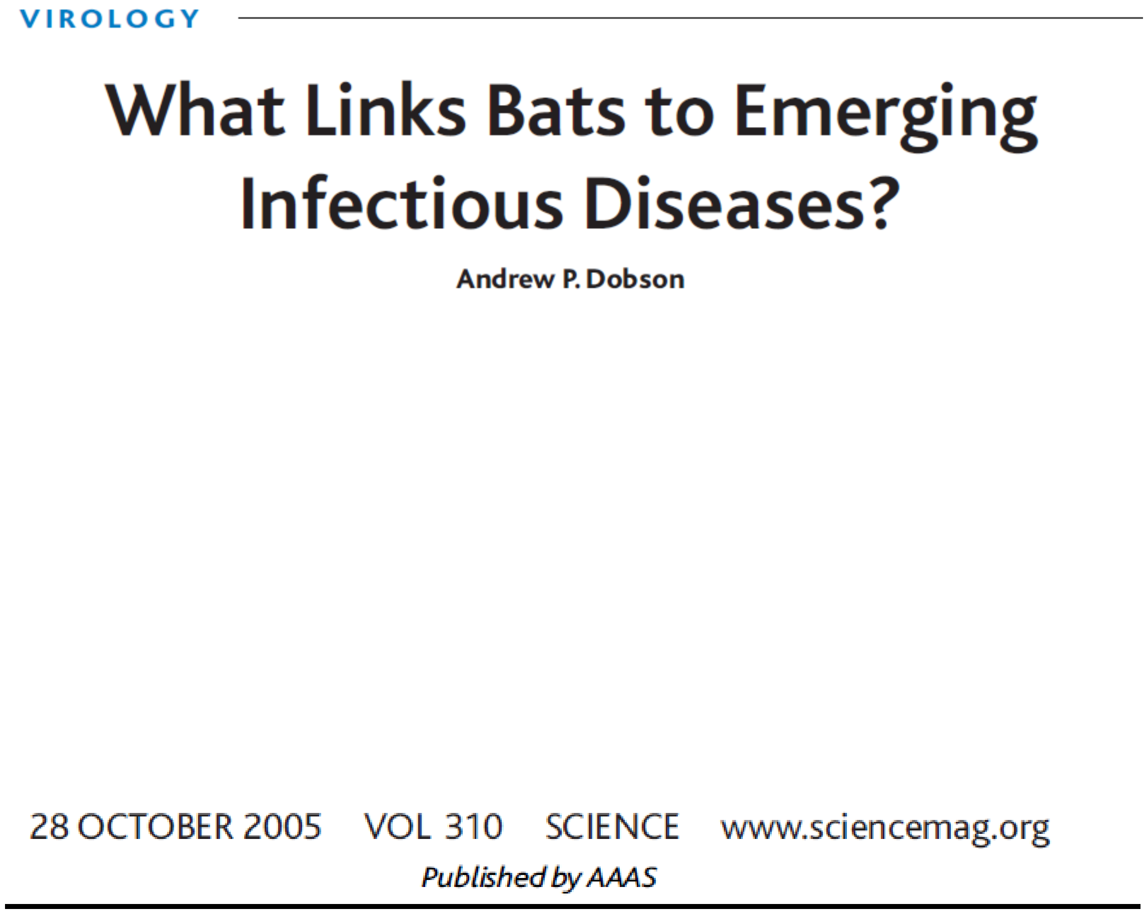
OPEN QUESTION: What are levels of cross-immunity Between these viruses? How many crossover and fade-out? What levels of immunity does this build-up in people working in the wildlife trade?
Does this form a barrier or facilitate persistence/emergence?
(sensu Pulliam et al, above and PLOS-One.)

Pulliam et al (2011) Royal Society Interface.



Agricultural intensification, priming for persistence and the emergence of Nipah virus: a lethal bat-borne zoonosis, Volume: 9, Issue: 66, Pages: 89-101, DOI: (10.1098/rsif.2011.0223)

So is this also happening with people working in the wildlife trade..? Enhanced by cross immunity?



VIROLOGY

What Links Bats to Emerging Infectious Diseases?

Andrew P. Dobson

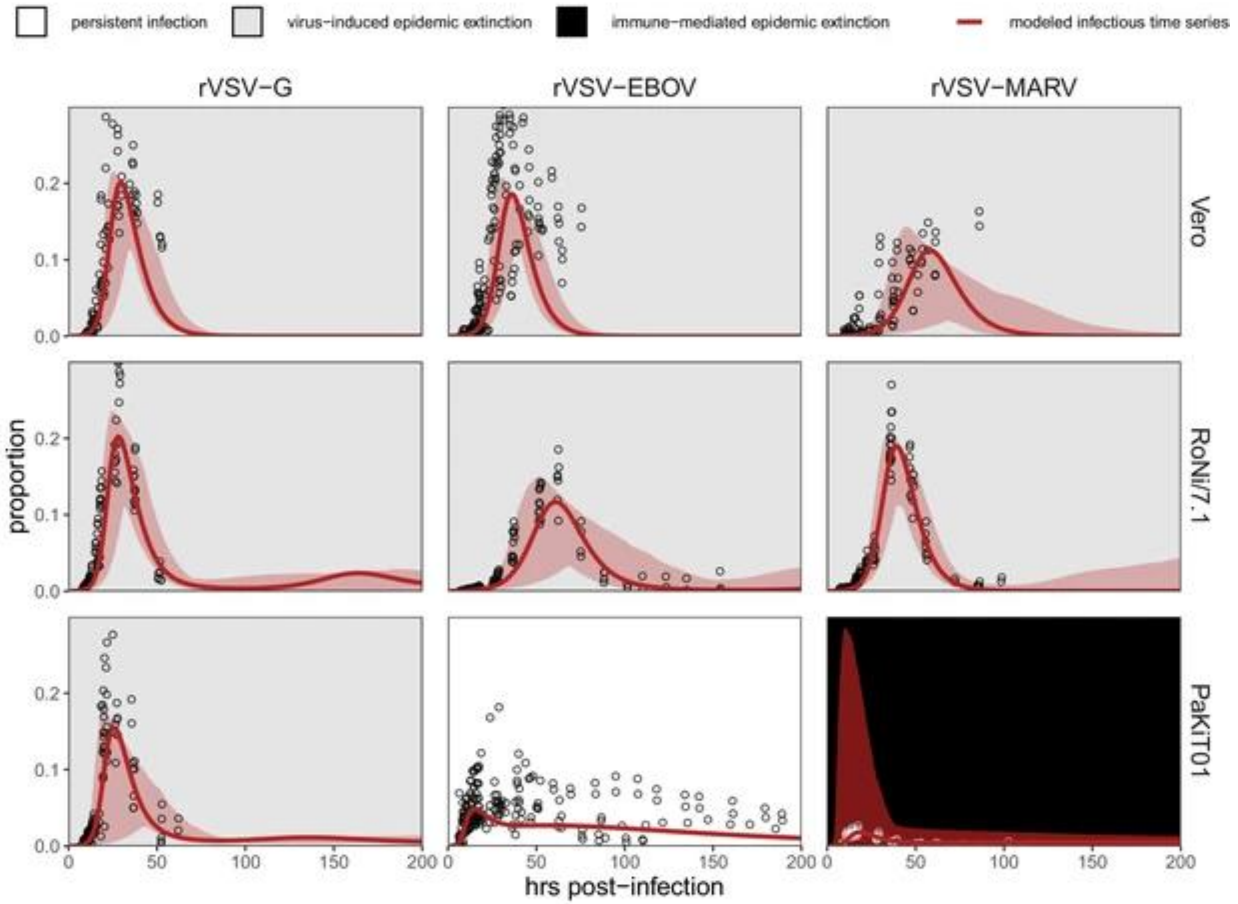
28 OCTOBER 2005 VOL 310 SCIENCE www.sciencemag.org
Published by AAAS

Bats, the great natural reservoir for viruses. Knowing more about bat ecology and immunology is crucial to controlling spillover of viruses and related diseases to humans.

Brook, C. And Dobson, A. (2015) Trends in Microbiology.

Accelerated viral dynamics in bat cell lines, with implications for zoonotic emergence

rVSV-eGFP-virus rVSV-Ebola virus rVSV-Marburg virus



African green monkey cells
(IFN-defective)

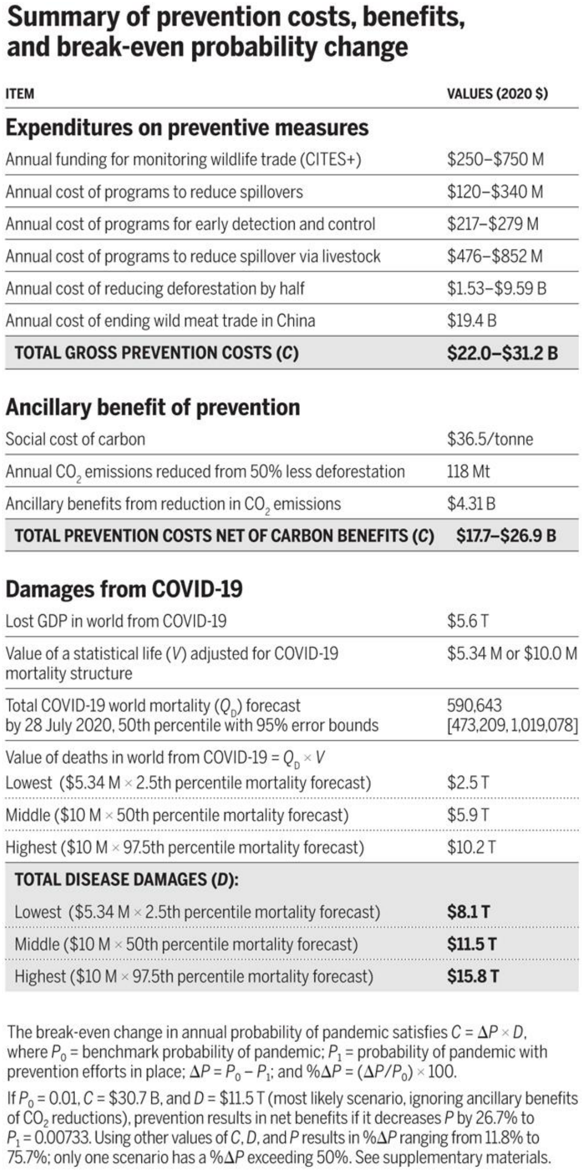
Rousettus aegypticus,
African fruit bat
(idiosyncratic interferon)

Pteropus alecto
S.E.Asian fruit bat
(expresses IFN-α)

Cara E Brook Is a corresponding author, Mike Boots, Kartik Chandran, Andrew P Dobson, Christian Drosten, Andrea L Graham, Bryan T Grenfell, Marcel A Müller, Melinda Ng. eLife/48401. 2020

2. Economics of Emerging Diseases

- Cost-benefit at each stage of intervention
- Overall cost-benefit
- Missing costs and additional benefits



Summary of prevention costs, benefits, and break-even probability change

ITEM	VALUES (2020 $)
Expenditures on preventive measures	
Annual funding for monitoring wildlife trade (CITES+)	\$250–\$750 M
Annual cost of programs to reduce spillovers	\$120–\$340 M
Annual cost of programs for early detection and control	\$217–\$279 M
Annual cost of programs to reduce spillover via livestock	\$476–\$852 M
Annual cost of reducing deforestation by half	\$1.53–\$9.59 B
Annual cost of ending wild meat trade in China	\$19.4 B
TOTAL GROSS PREVENTION COSTS (*C*)	**\$22.0–\$31.2 B**
Ancillary benefit of prevention	
Social cost of carbon	\$36.5/tonne
Annual CO_2 emissions reduced from 50% less deforestation	118 Mt
Ancillary benefits from reduction in CO_2 emissions	\$4.31 B
TOTAL PREVENTION COSTS NET OF CARBON BENEFITS (*C*)	**\$17.7–\$26.9 B**
Damages from COVID-19	
Lost GDP in world from COVID-19	\$5.6 T
Value of a statistical life (V) adjusted for COVID-19 mortality structure	\$5.34 M or \$10.0 M
Total COVID-19 world mortality (Q_D) forecast by 28 July 2020, 50th percentile with 95% error bounds	590,643 [473,209, 1,019,078]
Value of deaths in world from COVID-19 = $Q_D \times V$	
Lowest (\$5.34 M × 2.5th percentile mortality forecast)	\$2.5 T
Middle (\$10 M × 50th percentile mortality forecast)	\$5.9 T
Highest (\$10 M × 97.5th percentile mortality forecast)	\$10.2 T
TOTAL DISEASE DAMAGES (*D*):	
Lowest (\$5.34 M × 2.5th percentile mortality forecast)	**\$8.1 T**
Middle (\$10 M × 50th percentile mortality forecast)	**\$11.5 T**
Highest (\$10 M × 97.5th percentile mortality forecast)	**\$15.8 T**

The break-even change in annual probability of pandemic satisfies $C = \Delta P \times D$, where P_0 = benchmark probability of pandemic; P_1 = probability of pandemic with prevention efforts in place; $\Delta P = P_0 - P_1$; and $\%\Delta P = (\Delta P / P_0) \times 100$.

If $P_0 = 0.01$, C = \$30.7 B, and D = \$11.5 T (most likely scenario, ignoring ancillary benefits of CO_2 reductions), prevention results in net benefits if it decreases P by 26.7% to $P_1 = 0.00733$. Using other values of C, D, and P results in $\%\Delta P$ ranging from 11.8% to 75.7%; only one scenario has a $\%\Delta P$ exceeding 50%. See supplementary materials.

Figure 1: Costs and Ancillary Benefits of Prevention (million $)

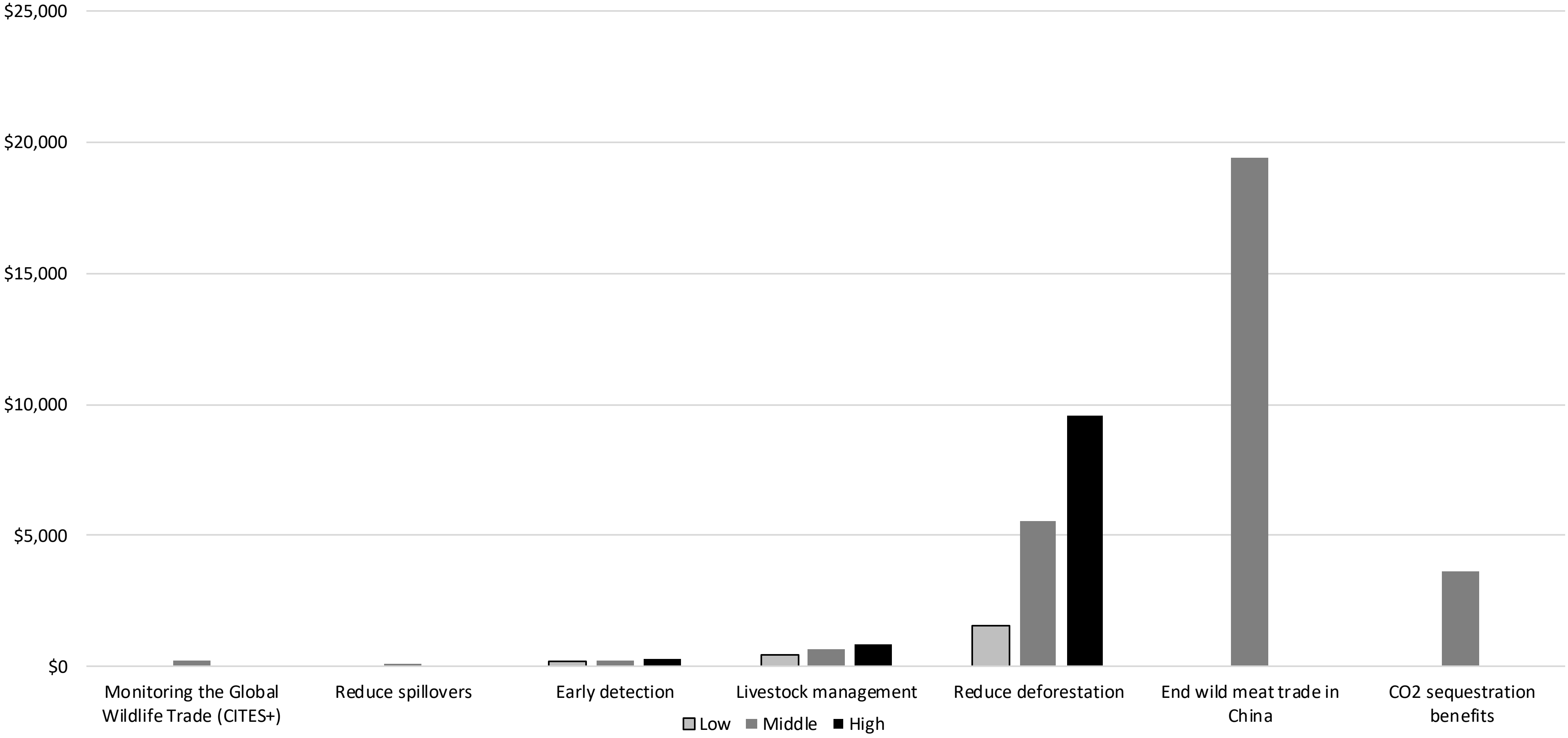
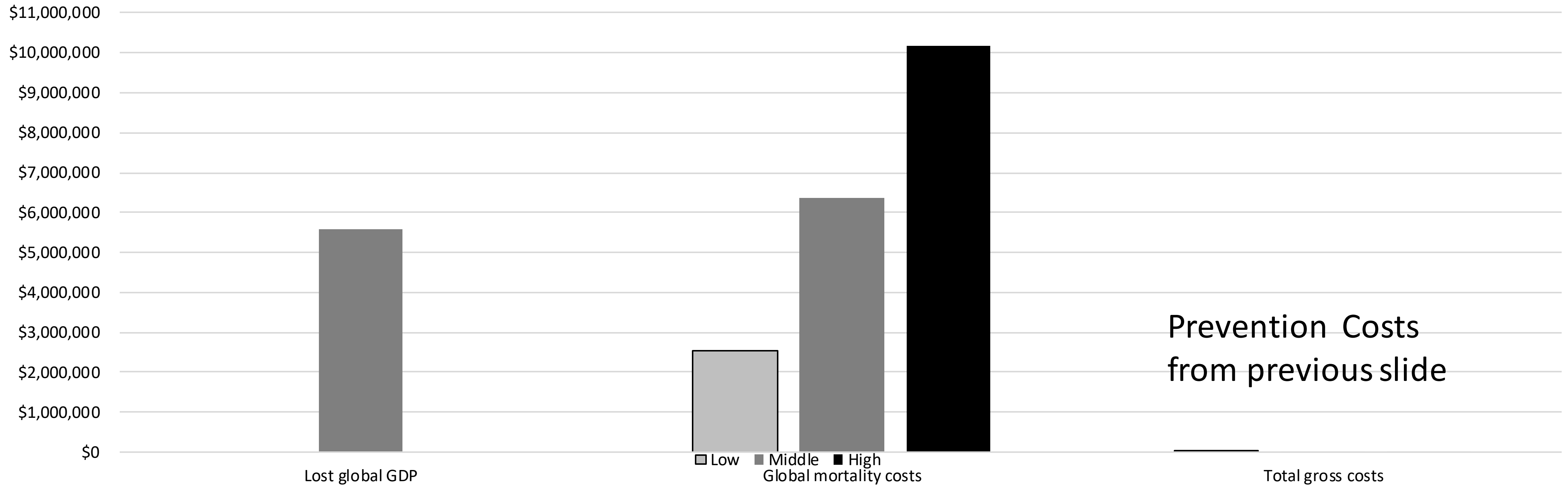
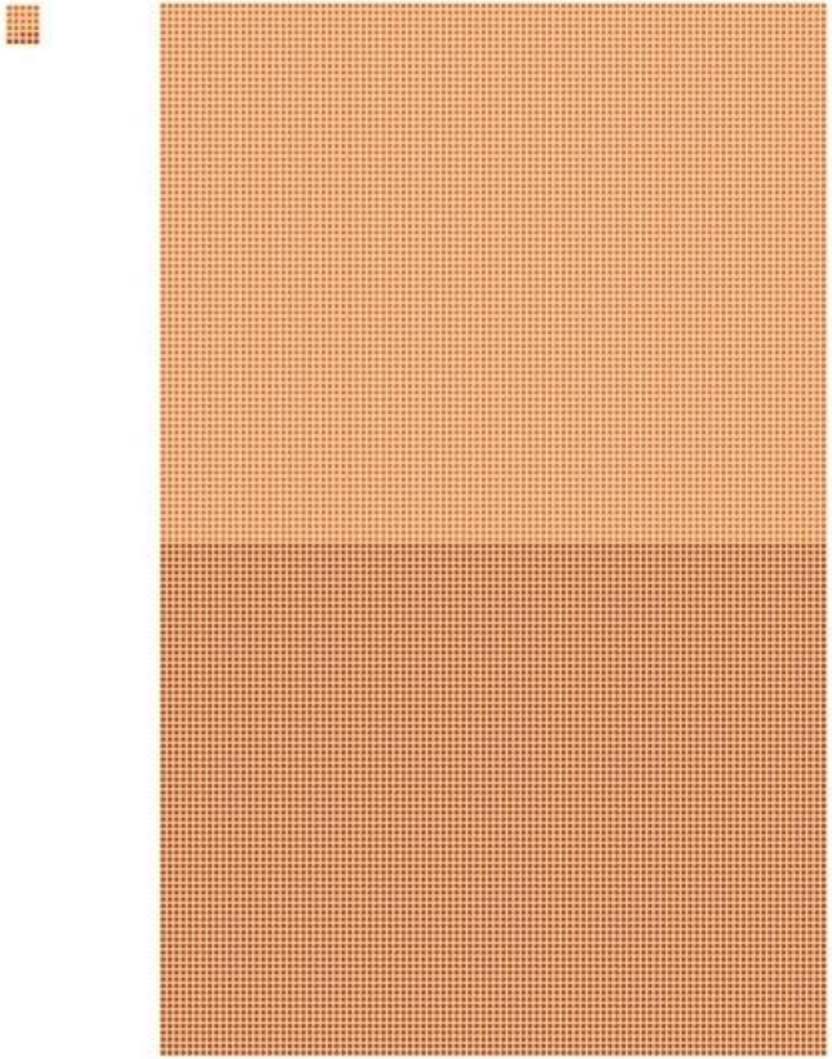


Figure 2: COVID-19 Damages and Total Gross Prevention Costs (million $)



Prevention Costs from previous slide

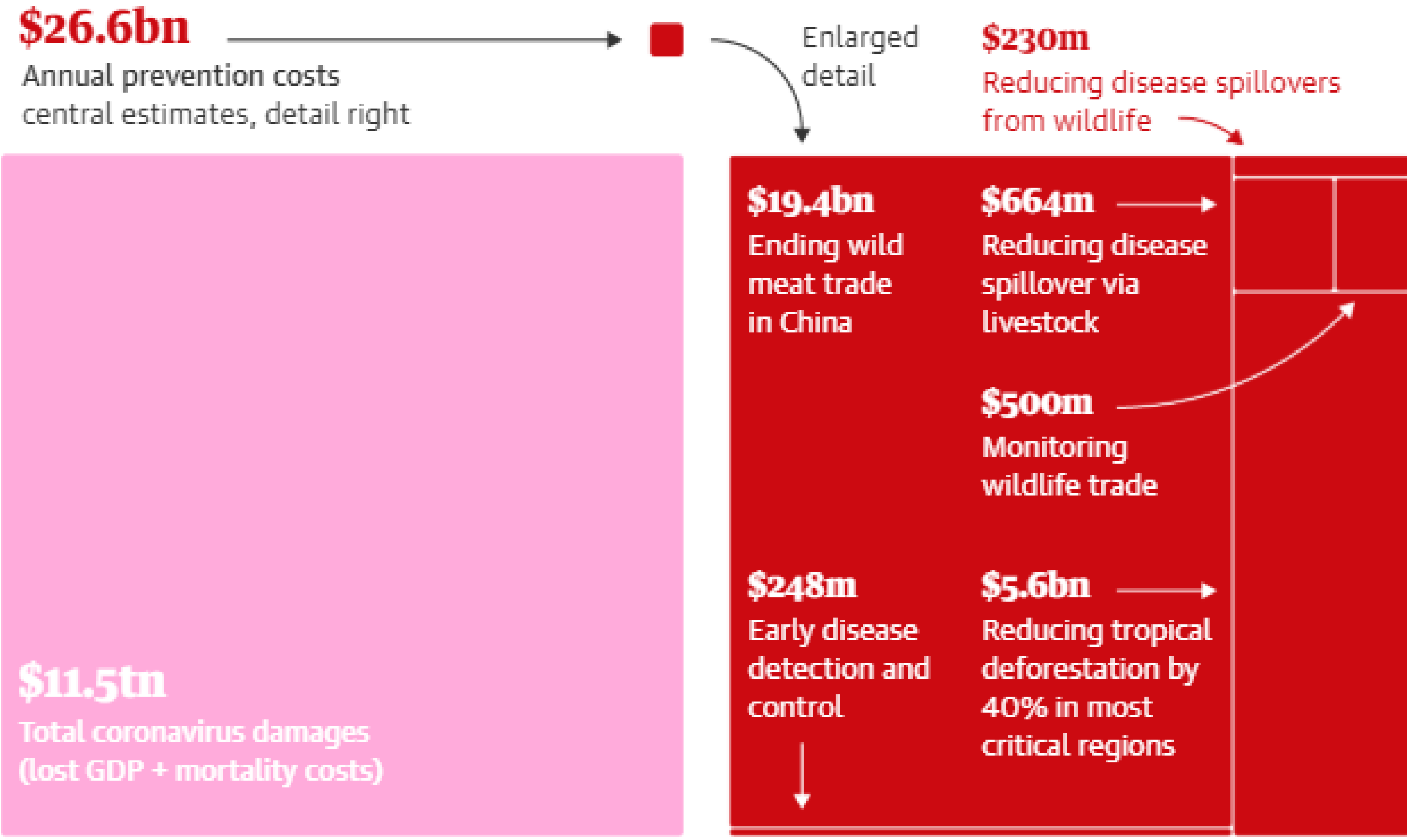


Annual cost of preventing zoonotic diseases →
(low: $22.2 billion)
(high: $30.7 billion)

← **Damage from COVID-19**
(low: $8.1 trillion)
(high: $15.8 trillion)

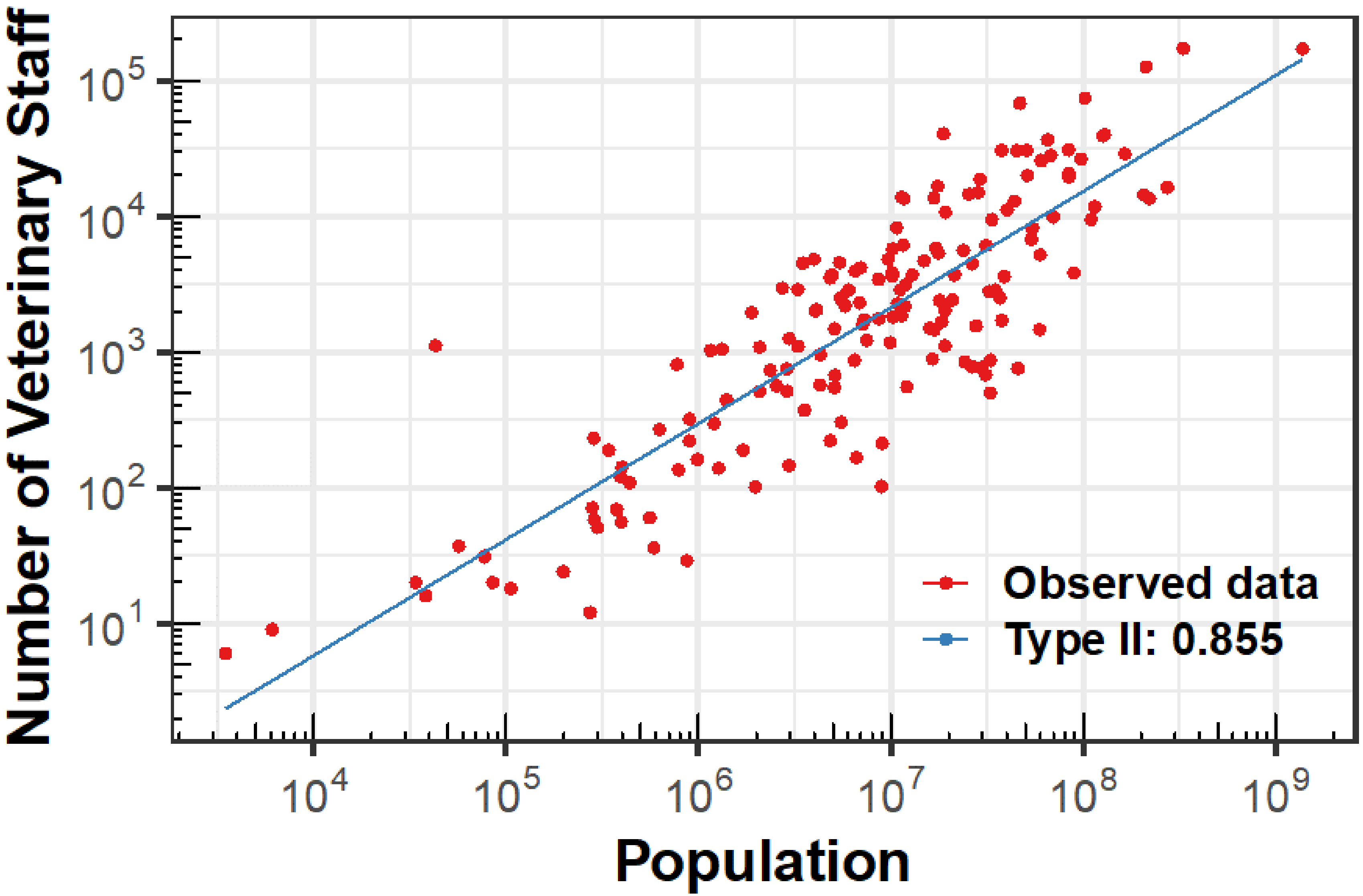
Each cell represents $1 billion in 2020 USD. Annual cost of preventing zoonotic diseases includes forest protection, control of wildlife trade, farmed animal biosecurity, and early detection. Damage from COVID-19 includes lost lives through July 28, 2020 and lost GDP in 2020.
Source: Dobson et al., *Science*, 2020. "Ecology and Economics for Pandemic Prevention." sciencemag.org/linktobepopulatedhere
Graphic: @jonahbusch

Annual costs of preventing future pandemics from wildlife are tiny compared with costs of the Covid-19 crisis

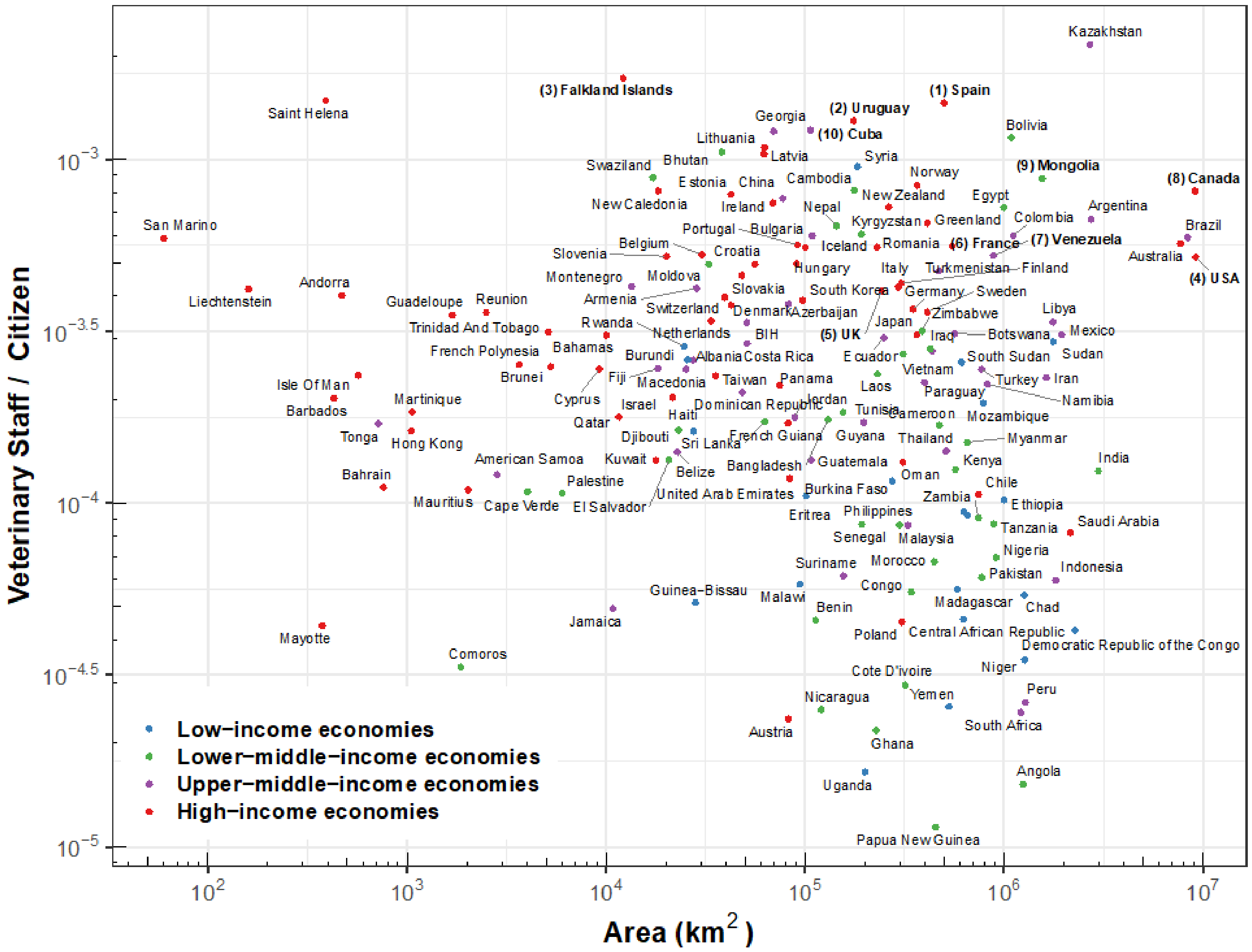


Guardian graphic. Source: Dobson et al, Science 2020

First line of defense – veterinarians!



Relative abundance of front-line defenders



Data were absent from the OIE database for several nations (including China and Russia). The ratio of veterinarians to civilians plotted against the nation’s area. Countries are color- coded based on World Bank income categories.

3. Evolution of Emerging Pathogens

- Will Covid-19 become more or less virulent?
- Insights from an avian emerging pathogen

Mutation has given rise to multiple strains that characterize cluster in different locations

When will we see selection for changes in virulence?

Natural habitats of house finches……

House Finches were introduced on Long Island in 1940

1960-61

male House Finch
by Larry McQueen

A Tractable Disease System: MG in House Finches

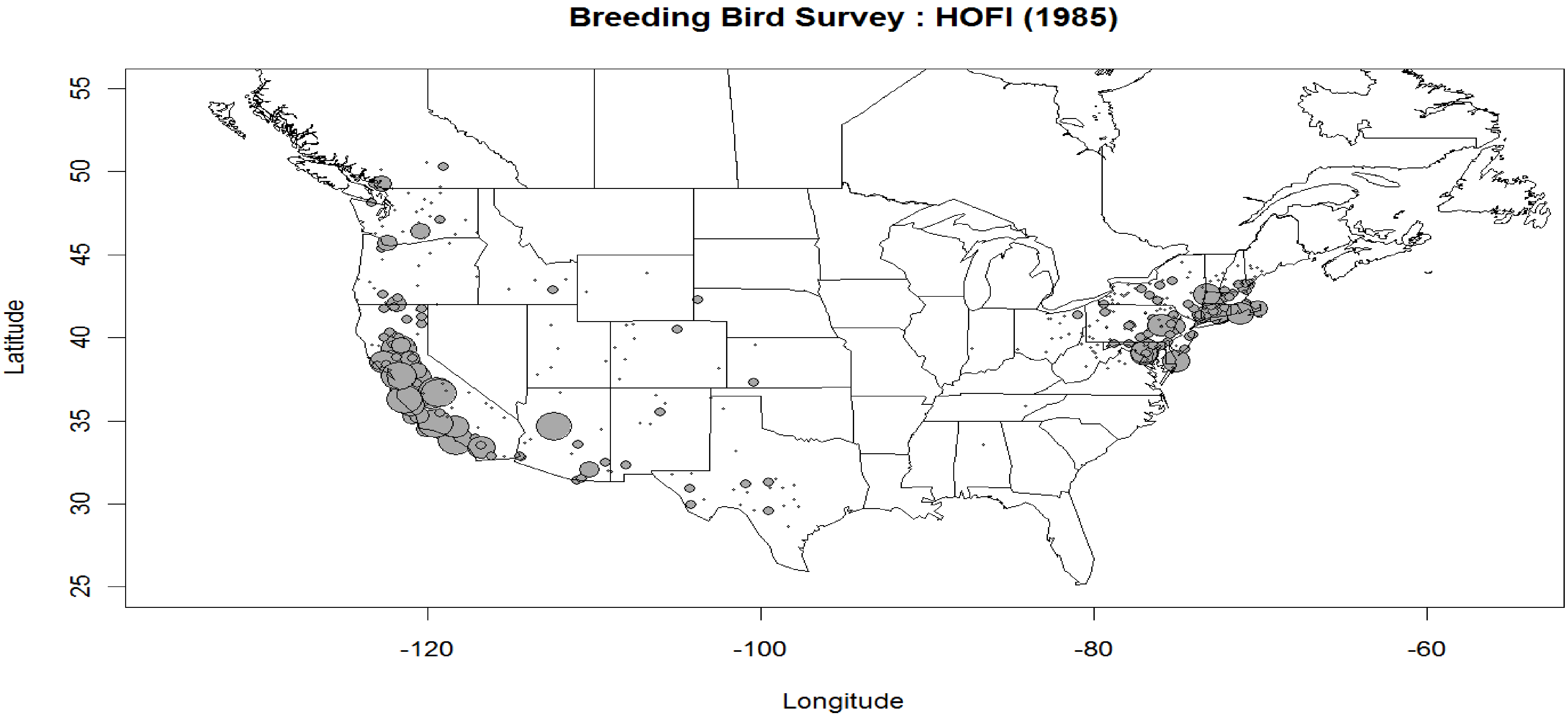
Mycoplasma gallisepticum
= MG

→

1993

House Finch
Carpodacus mexicanus

House finch distribution in the US



Mycoplasmal conjunctivitis

- caused by the bacterium *Mycoplasma gallisepticum*
- found in domestic poultry worldwide
- novel strain infects House Finches
- clear external clinical signs
- clinical signs are closely related

  to presence of pathogen

Humans aren't only species at risk from emerging pathogens
– monitoring focuses on humans and livestock….intervene as soon as possible
model systems can provide important insights not otherwise available

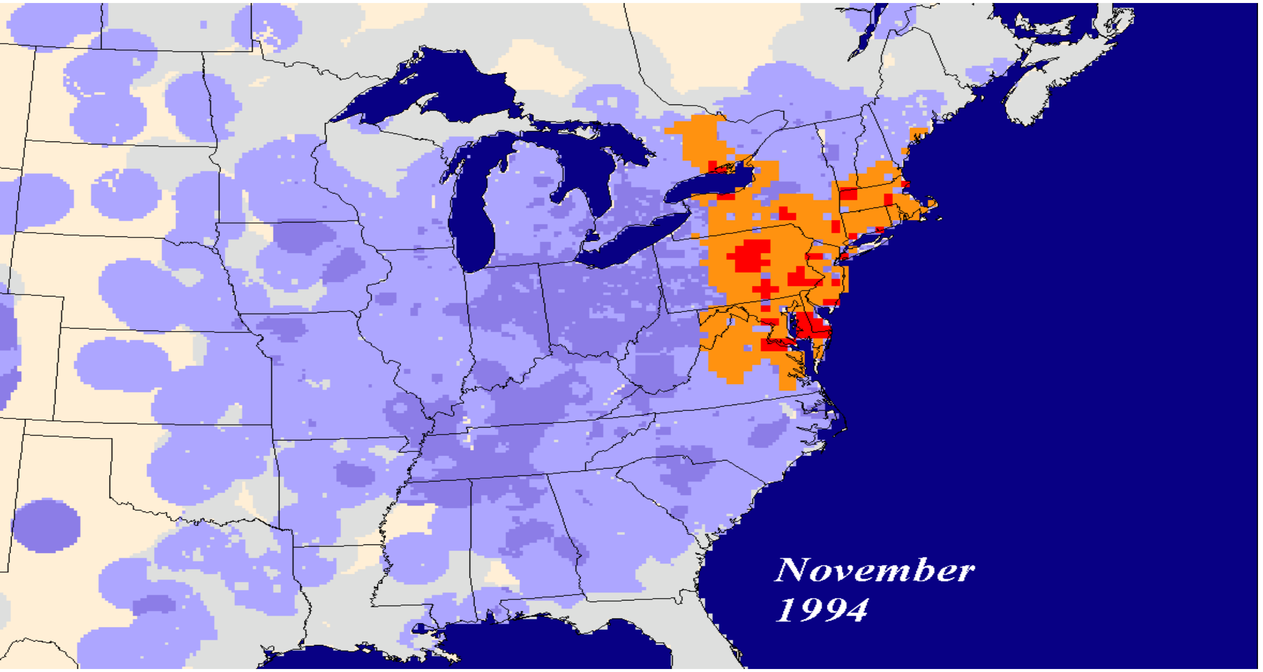
Wide Geographical Distribution of Participants

- most data from the north-eastern U.S.

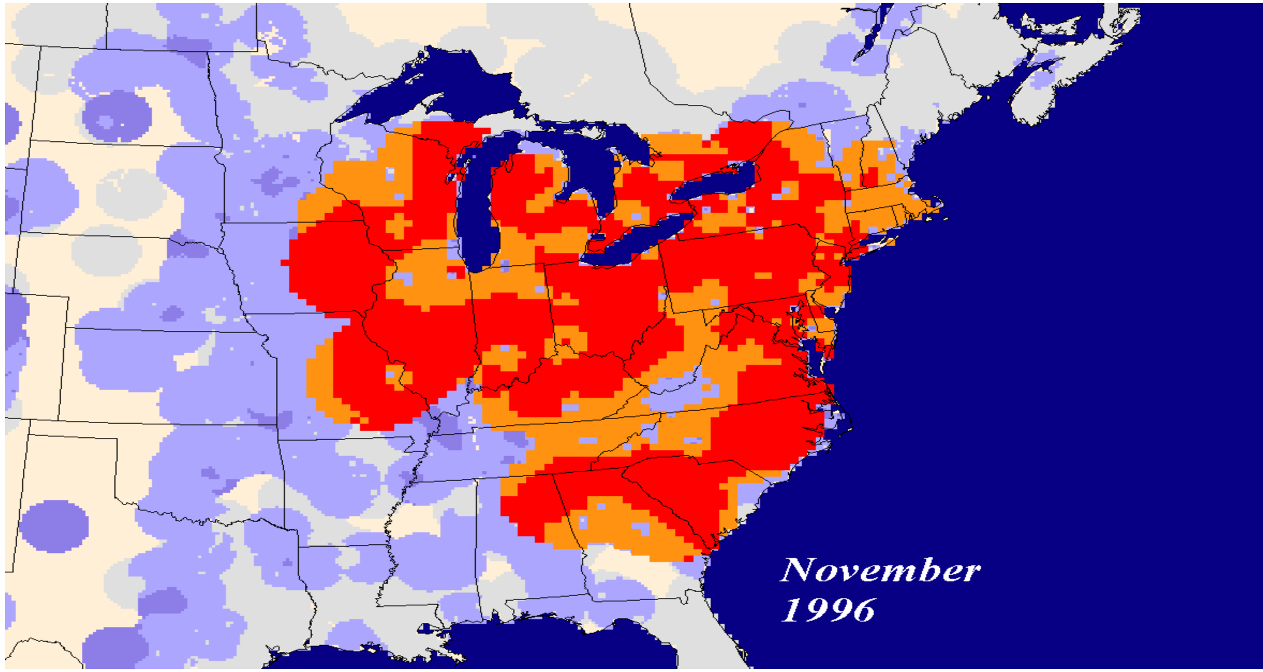
- high-density regions would swamp patterns in other areas if averaged

- disease arrived at different times in different regions

Disease Spread Rapidly Through Eastern North America



After 10 month



After 2.5 Years

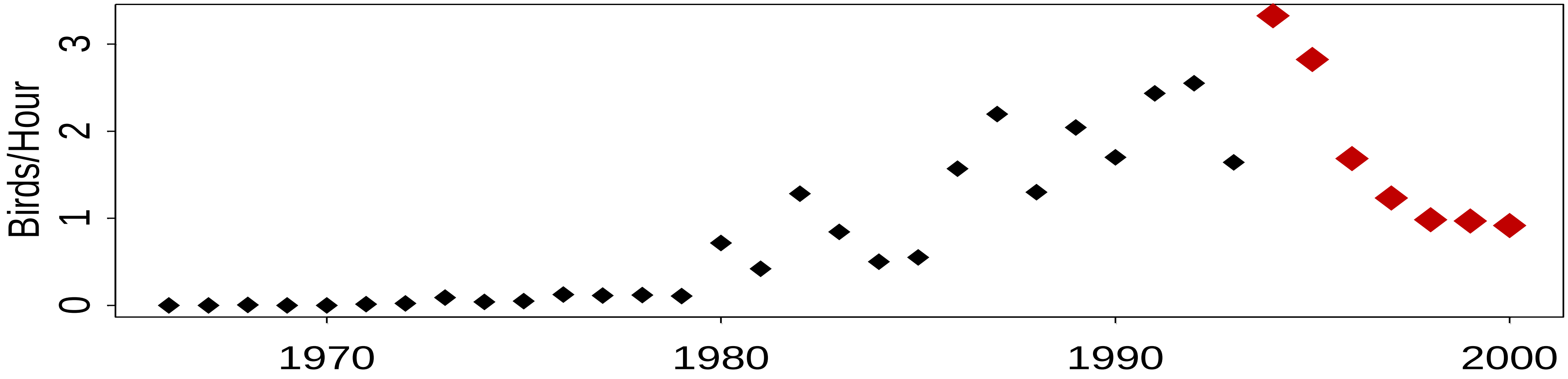
House finches are pest in wineries…very handy for sponsorship!

Upstate new York Wine….

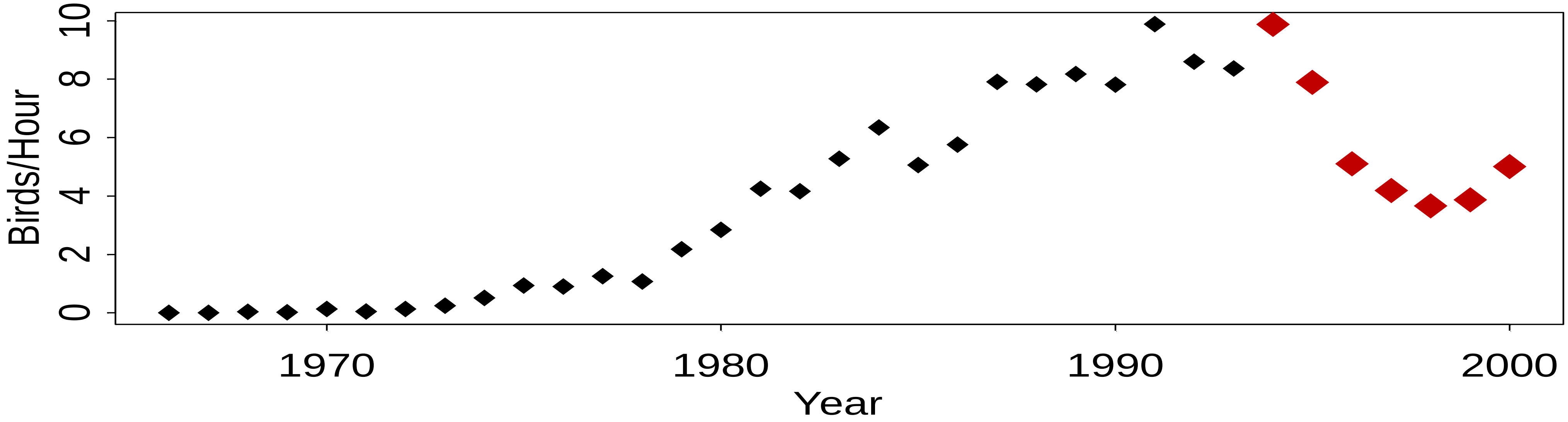
Delicious California wine!!

House Finch abundance decreased dramatically because of the new disease

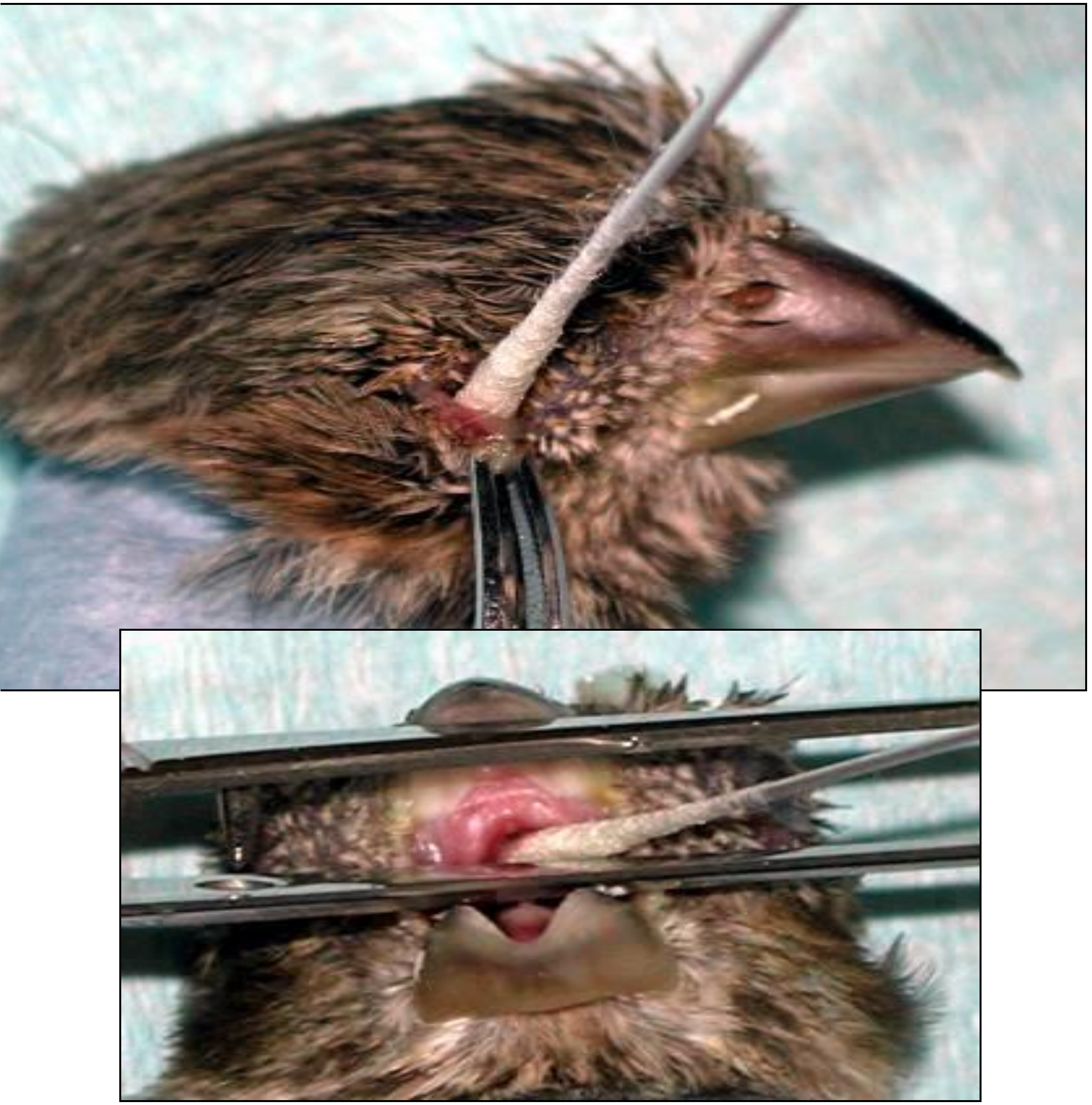
Eastern VA, Eastern MD, DE



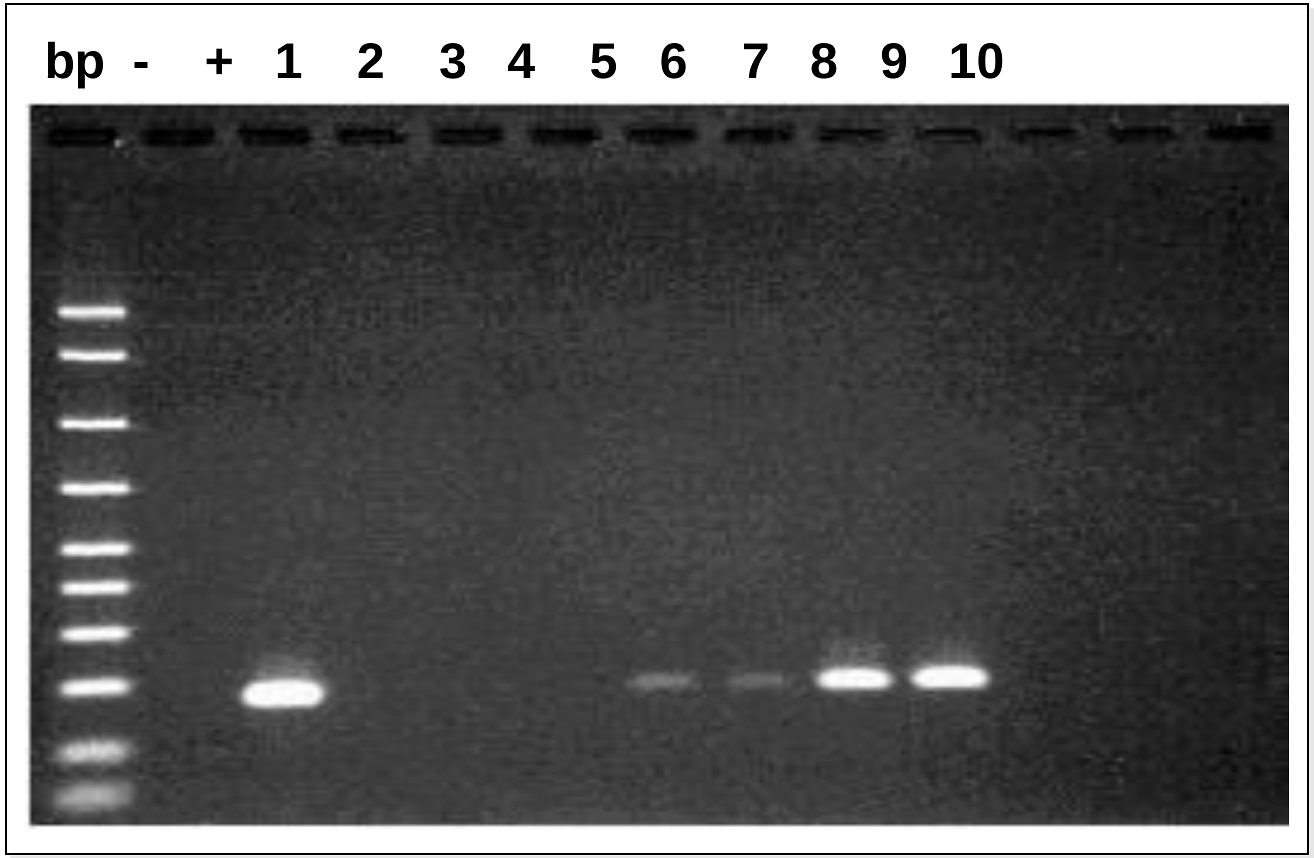
Central PA, Eastern NY



PCR to determine infection status using MG-specific polymerase chain reaction (PCR)



David Ley, NCSU



A Tractable Disease System

-- disease response can be measured non-invasively.

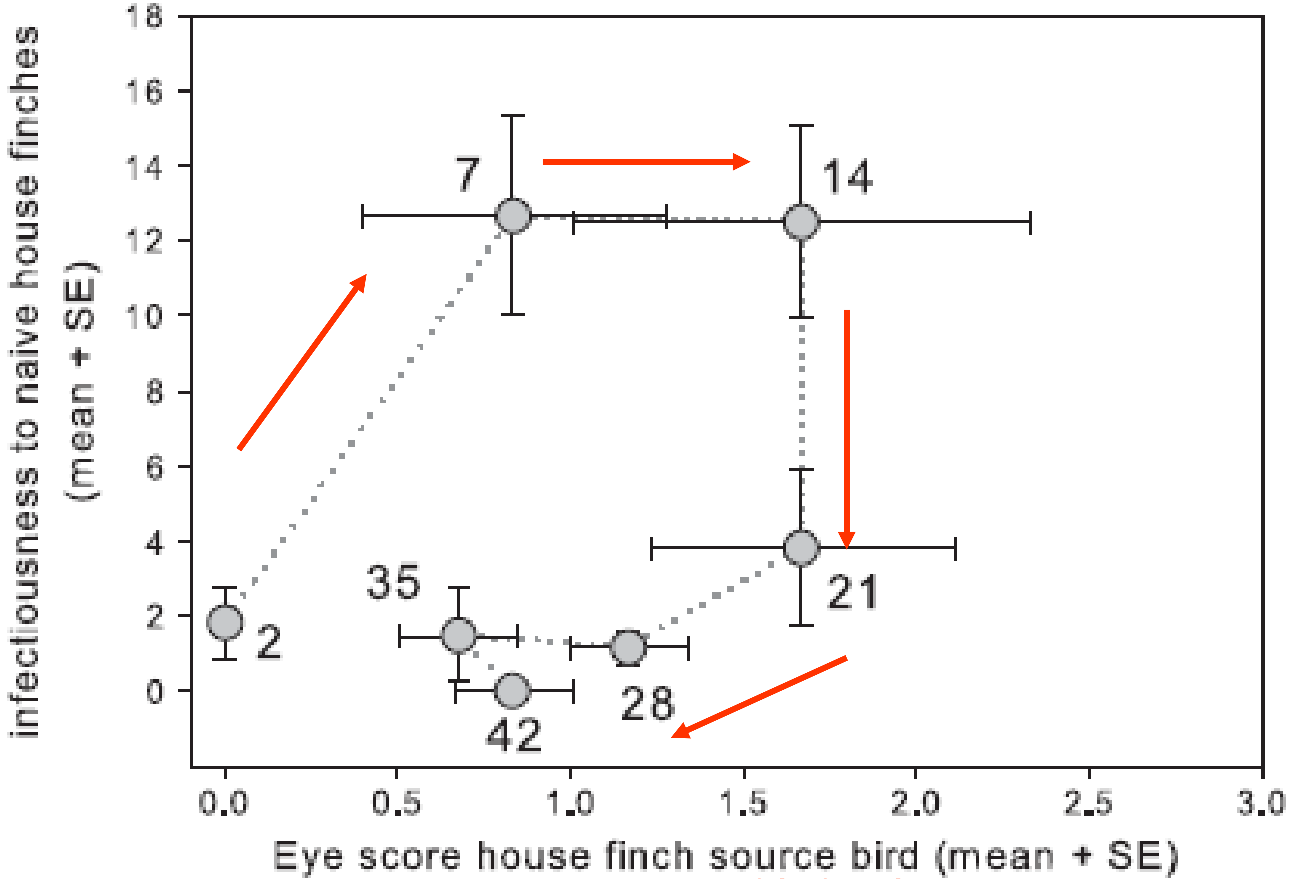
Score
0

Score
2

Score
1

Score
3

Time course of infection in individual infected birds (days)



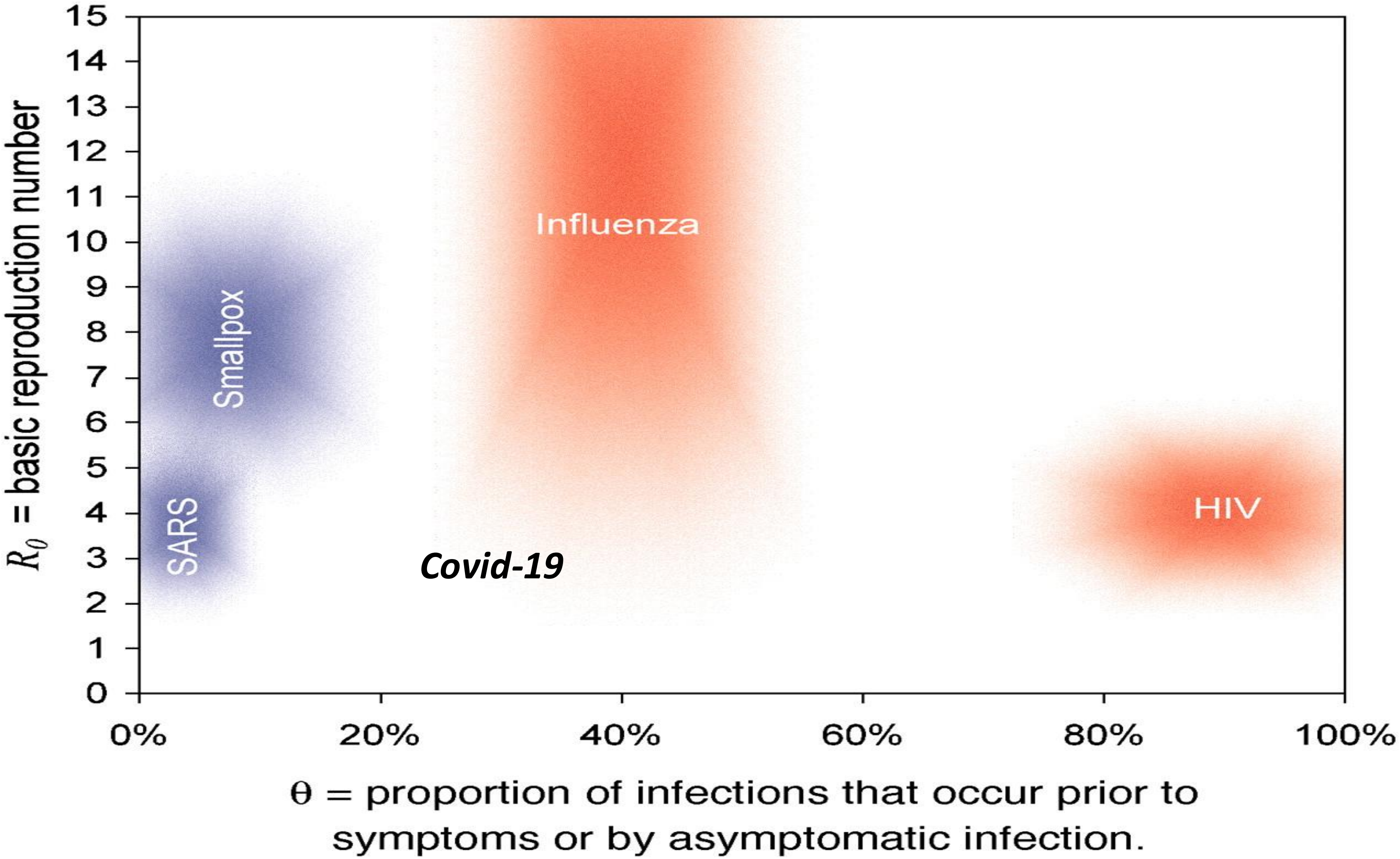
Transmission potential

Symptoms of infection

Figure 2. *Relationship between clinical signs of house finches at intervals after conjunctival inoculation with the house finch strain of* M. gallisepticum *and the degree to which they were infectious to naïve house finches. The digit by each point represents the number of days post inoculation.*

Dhondt, et al (2008) Avian Pathology, 37, 635-640

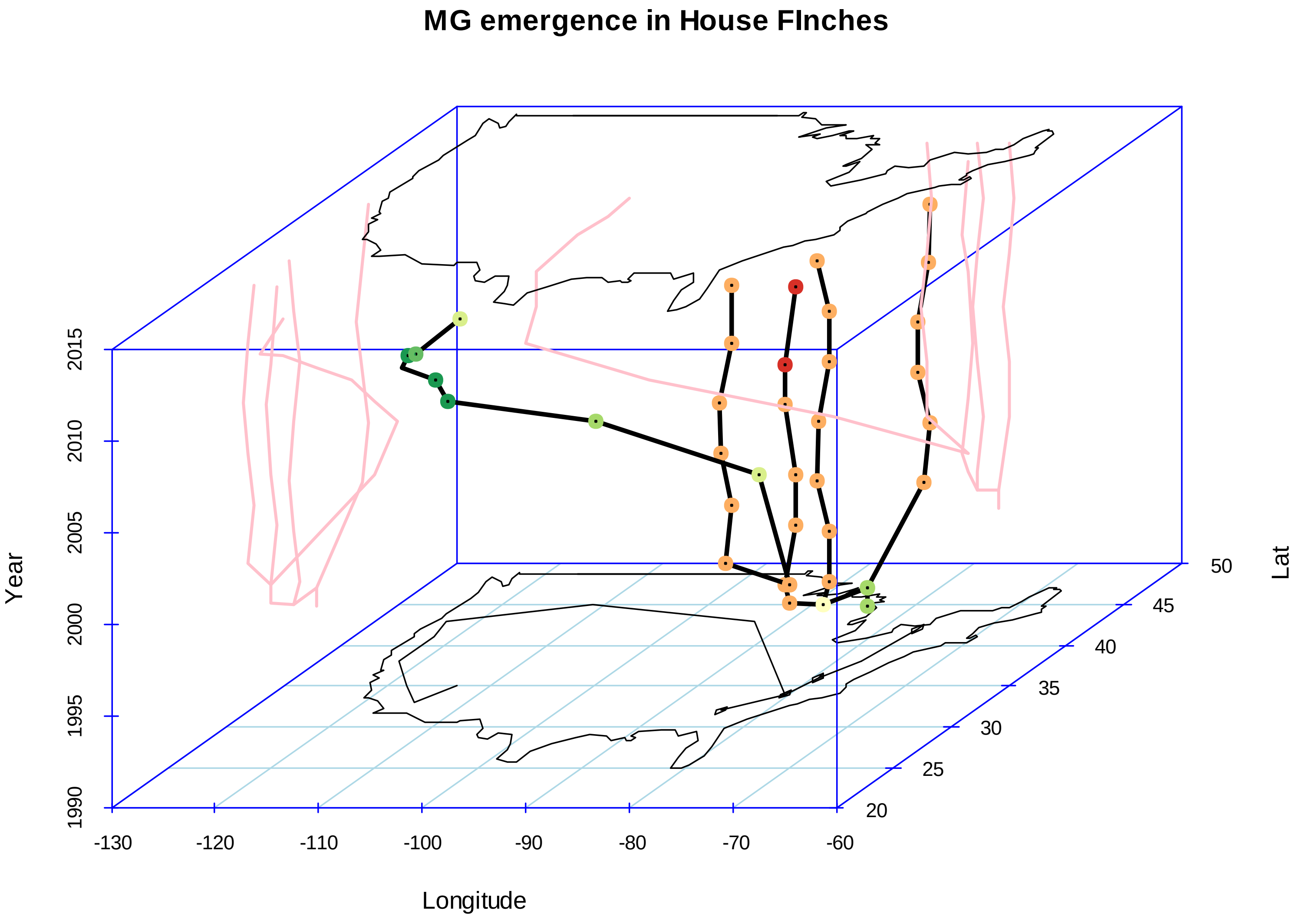
How do Ro and appearance of symptoms effect ability to control outbreaks?



Christophe Fraser et al. PNAS 2004;101:6146-6151

PNAS

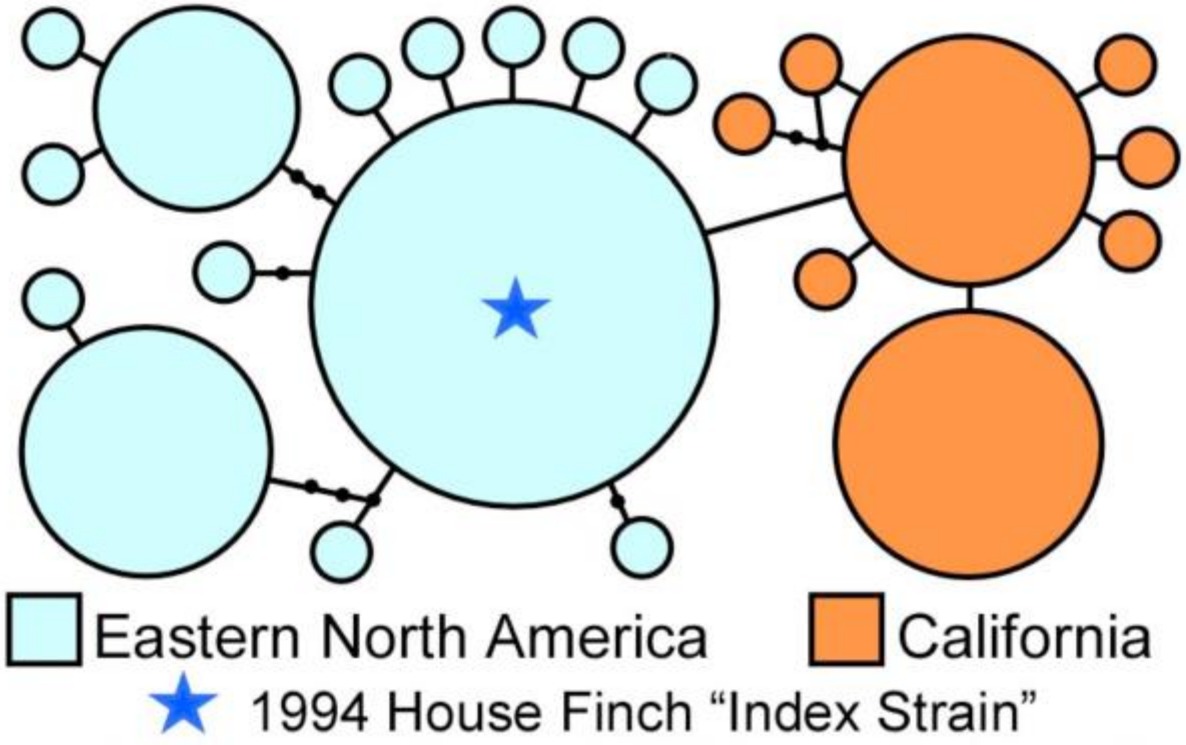
Virulence in Time and Space



Virulence levels shade green (low) to red (high)

Eastern & Western *M. gallisepticum* lineages independent

- Evolution of spatial variation in virulence is possible because genetic variation in *M. gallisepticum* is spatially structured



in prep.

Why does virulence evolve in two directions?

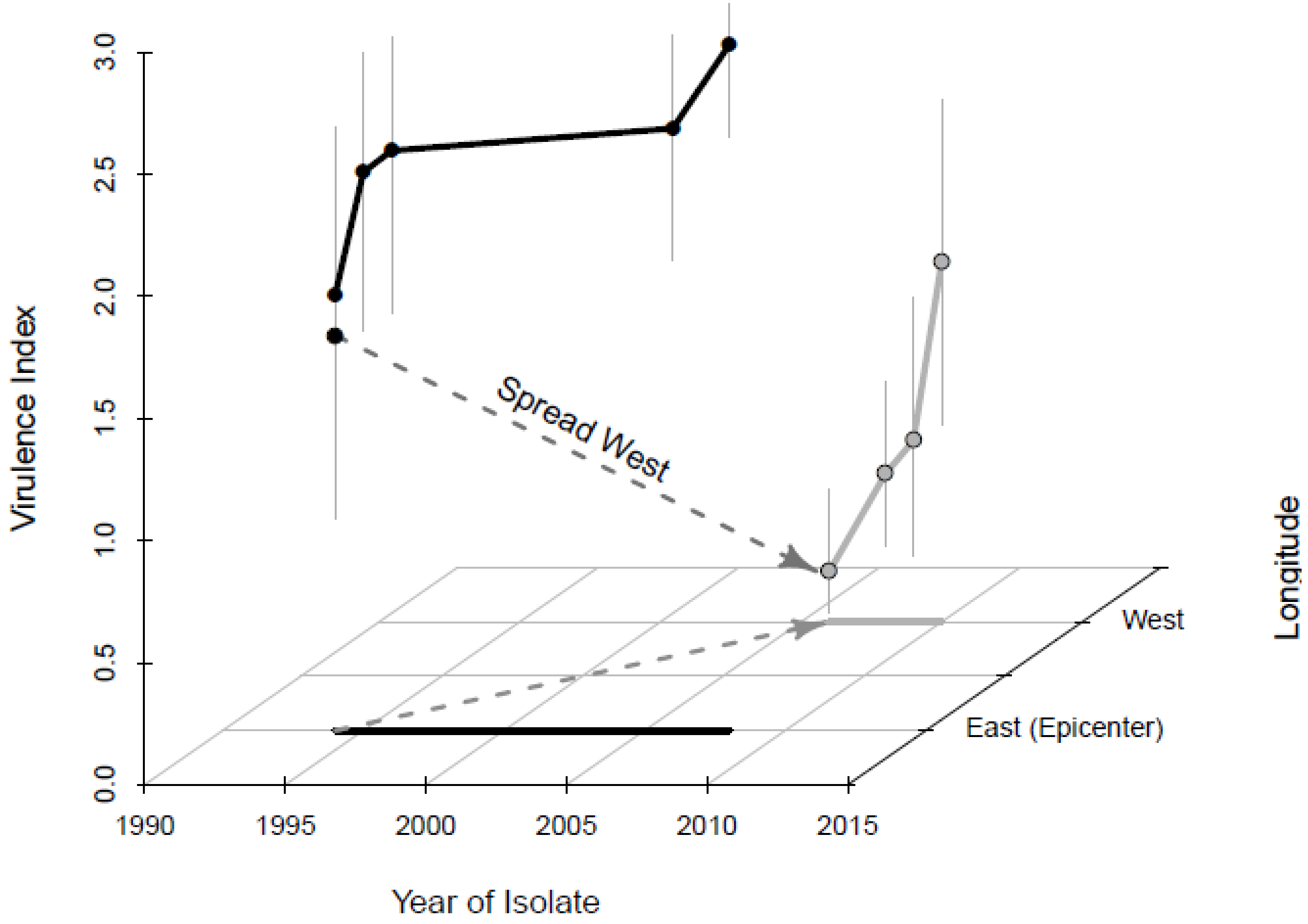


Figure 1: Virulence dynamics over time in two regions -- near the epicenter in eastern North America (black line), and the west coast (gray line) -- for the bacterium *Mycoplasma gallisepticum* after its emergence in the House Finch (*Haemorhous mexicanus*). For perspective, the projection of

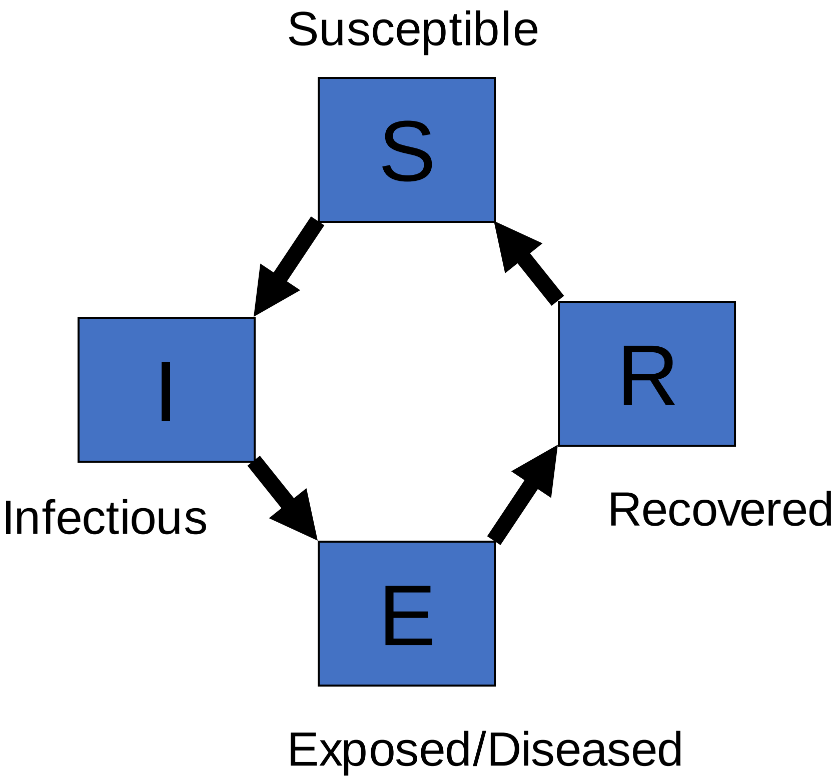
The model

$$\frac{dS}{dt} = b(1 - N/K)N - (\beta I + \phi \beta E)\frac{S}{N} - dS + \delta R$$

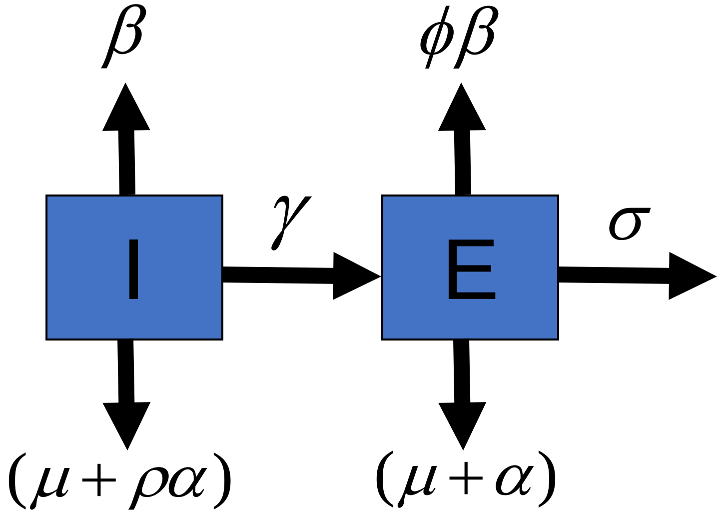
$$\frac{dI}{dt} = (\beta I + \phi \beta E)\frac{S}{N} - (d + \alpha_I + \gamma)I$$

$$\frac{dE}{dt} = \gamma I - (d + \alpha_E + \sigma)E$$

$$\frac{dR}{dt} = \sigma E - (d + \delta)R$$

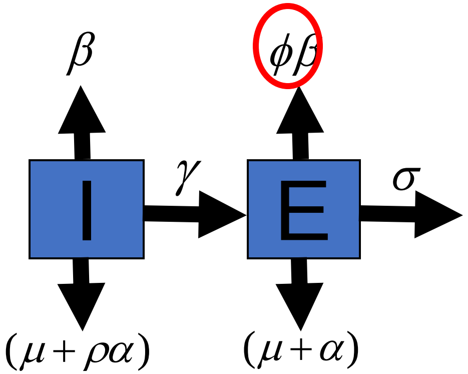


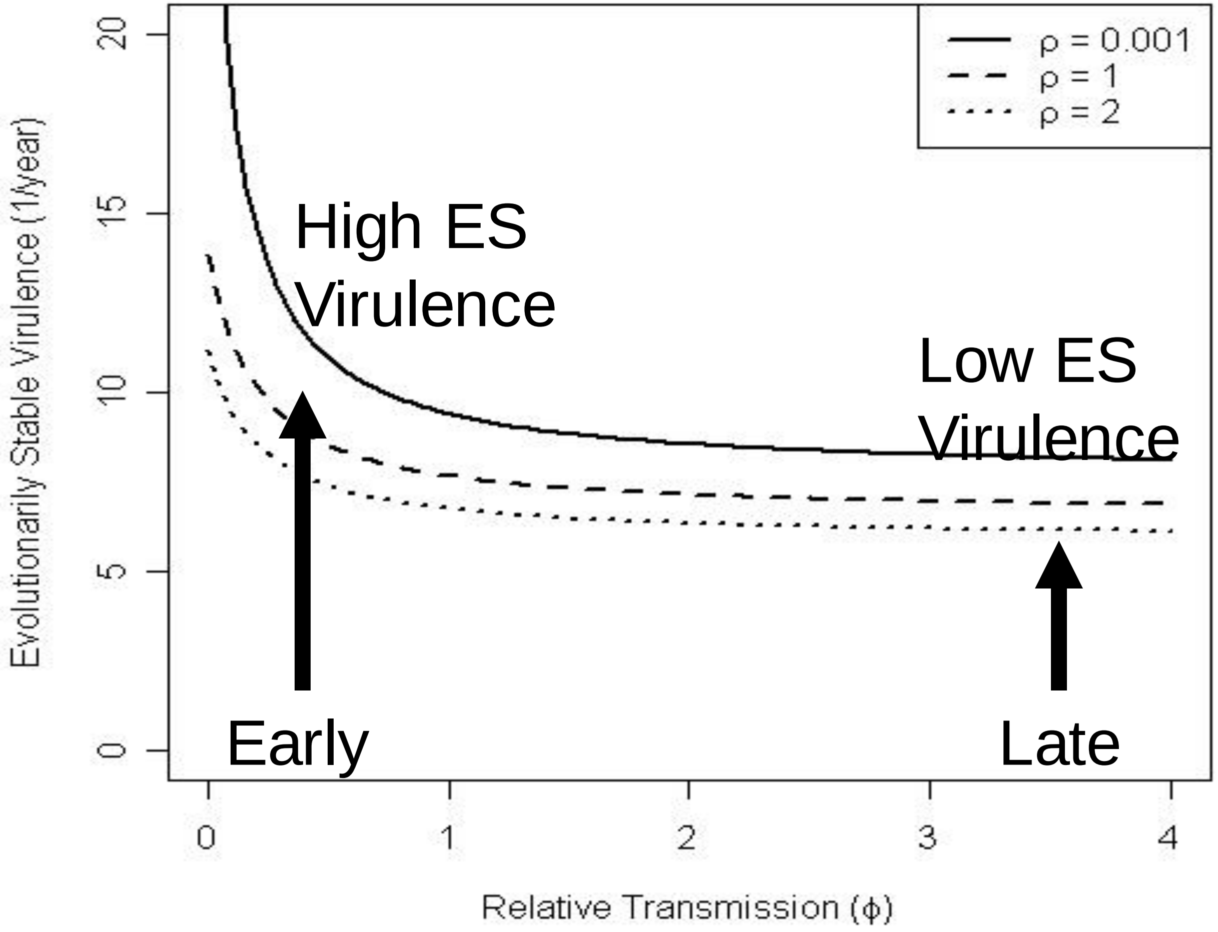
Virulence Evolution: the model



$$R_0=\frac{\beta}{(\mu+\rho\alpha+\gamma)}+\frac{\phi\beta}{(\mu+\alpha+\sigma)}\frac{\gamma}{(\mu+\rho\alpha+\gamma)}$$

ES Virulence and Relative Timing of Transmission (ϕ)





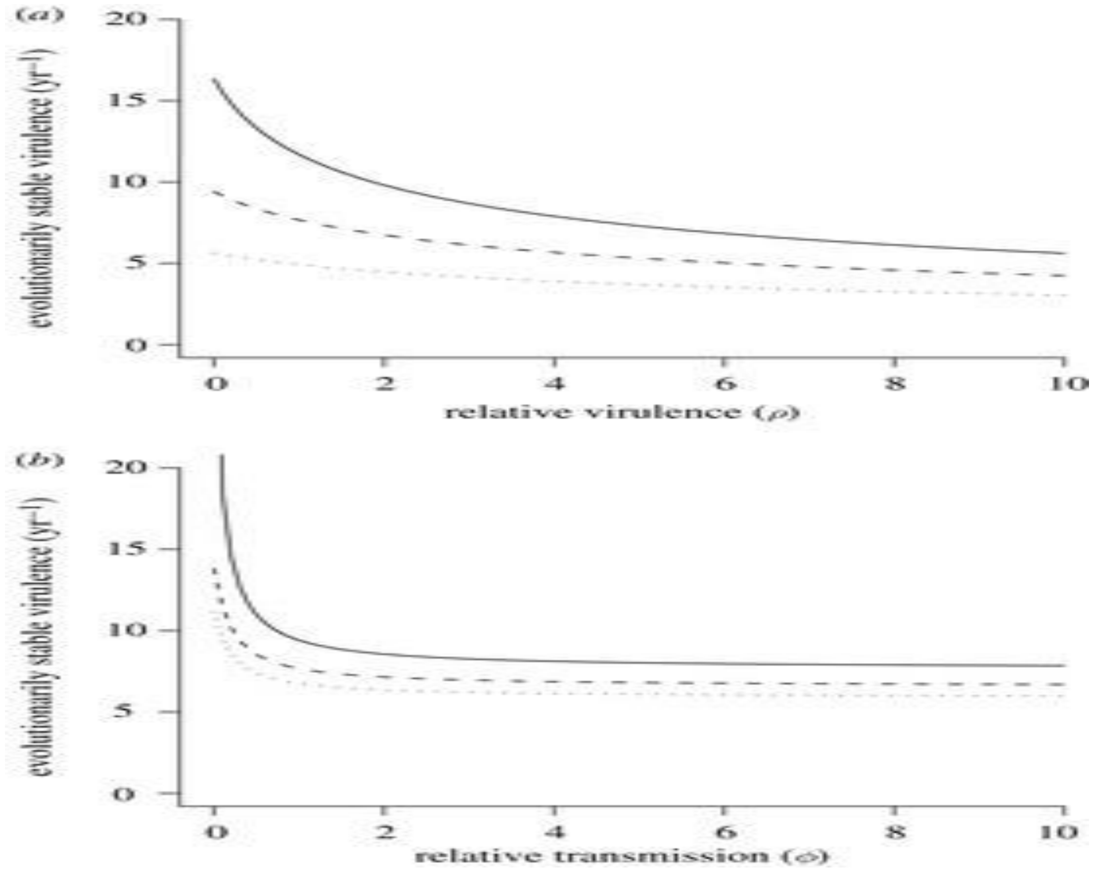
Osnas and Dobson, 2010, *Biology Letters*

Evolution of virulence when transmission occurs before disease

Erik E. Osnas and Andrew P. Dobson, Biology Letters

Published:**10 February 2010****https://doi.org/10.1098/rsbl.2009.1019**

Evolutionary stable level of virulence when symptoms appear



Relative virulence and relative transmission in Initial infection period before symptoms appear.

Evolution of virulence when transmission occurs before disease, Volume: 6, Issue: 4, Pages: 505-508, DOI: (10.1098/rsbl.2009.1019)

Why does virulence evolve in two directions?

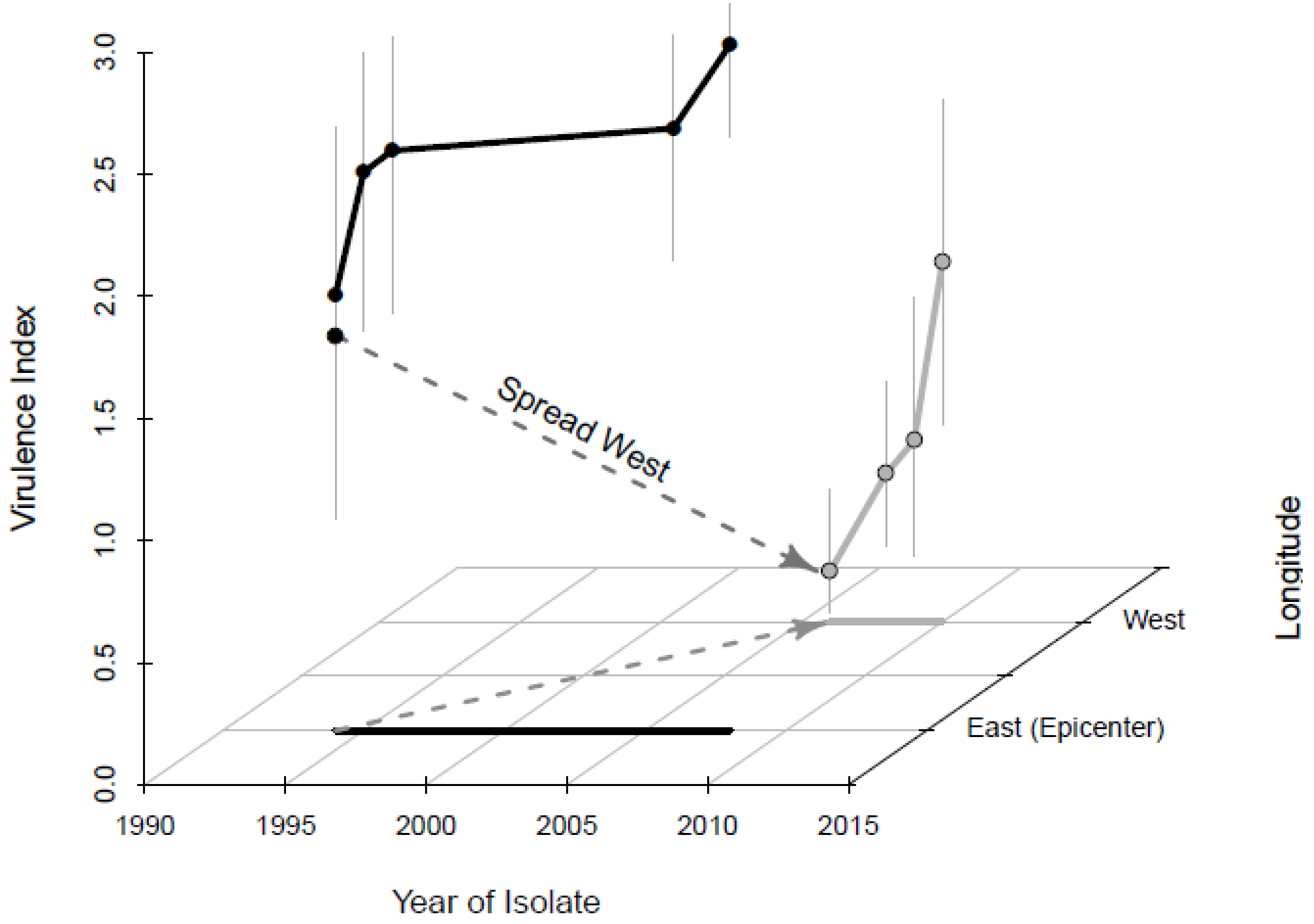
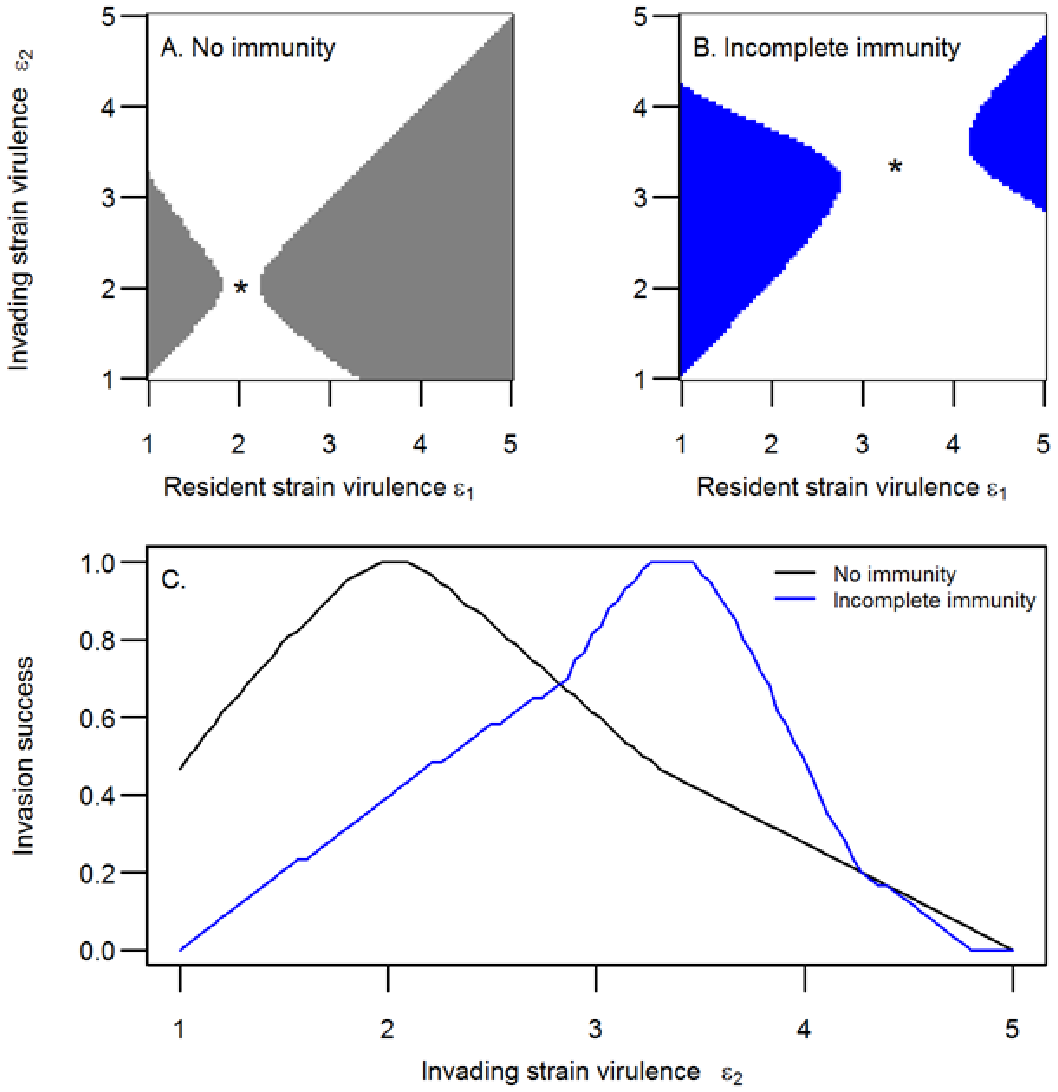


Figure 1: Virulence dynamics over time in two regions -- near the epicenter in eastern North America (black line), and the west coast (gray line) -- for the bacterium *Mycoplasma gallisepticum* after its emergence in the House Finch (*Haemorhous mexicanus*). For perspective, the projection of



Pairwise invasibility plots (PIPs) show that adding the empirically observed effects of incomplete immunity to a baseline model with no immunity effects (A) results in an almost twofold increase in optimal virulence (B). Shaded areas show parameter space for which a new mutant introduced at very low densities was able to invade a population with the resident pathogen present at equilibrium densities, and asterisks mark the Evolutionary Stable Strategy (ESS) for each model.

Fleming-Davies, Williams, Dobson, Dhondt, Hawley, (2018) Science.

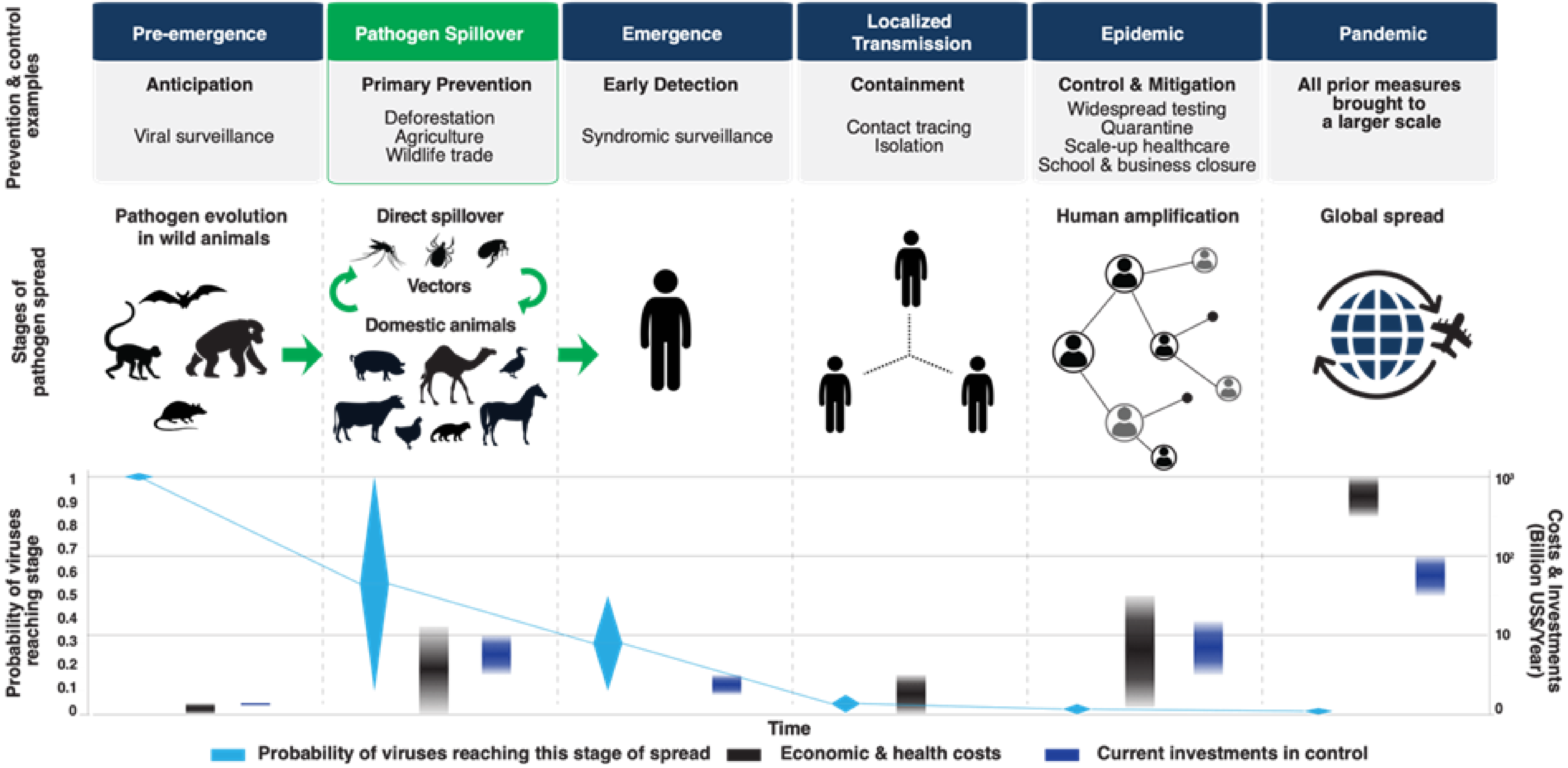
A message from the birds…

- Once herd immunity begins to make its presence known…
    - Covid will start competing within the immune population it has created
    - This will place selection pressure on the virus to change

    - Transmission before virulence is expressed selects for increased virulence

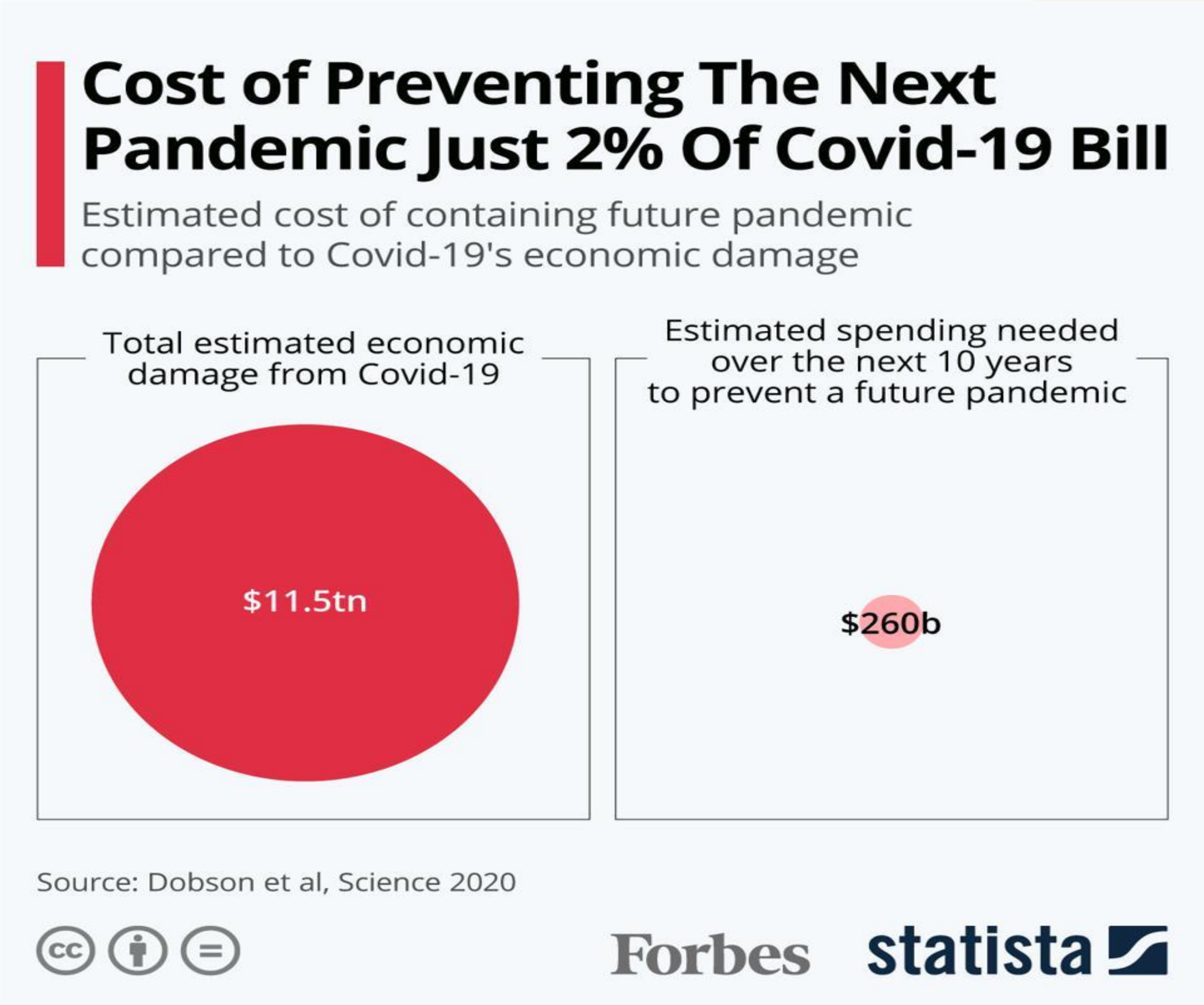
    - Immunity selects for asymmetrical immunity

    - Implications for vaccine development..?


Prevention & control examples
Pre-emergence
Anticipation
Viral surveillance
Pathogen Spillover
Primary Prevention
Deforestation
Agriculture
Wildlife trade
Emergence
Early Detection
Syndromic surveillance
Localized Transmission
Containment
Contact tracing
Isolation
Epidemic
Control & Mitigation
Widespread testing
Quarantine
Scale-up healthcare
School & business closure
Pandemic
All prior measures brought to a larger scale
Stages of pathogen spread
Pathogen evolution in wild animals
Direct spillover
Vectors
Domestic animals
Human amplification
Global spread
Probability of viruses reaching stage
1
0.9
0.8
0.7
0.6
0.5
0.4
0.3
0.2
0.1
0
Costs & Investments (Billion US$/Year)
10³
10²
10
0
Time
Probability of viruses reaching this stage of spread
Economic & health costs
Current investments in control

Conclusions

1) We will see future emerging pathogens
2) Cost of prevention is well worth the investment.
3) Viral evolution will speed up as herd immunity rises, it could go either way..

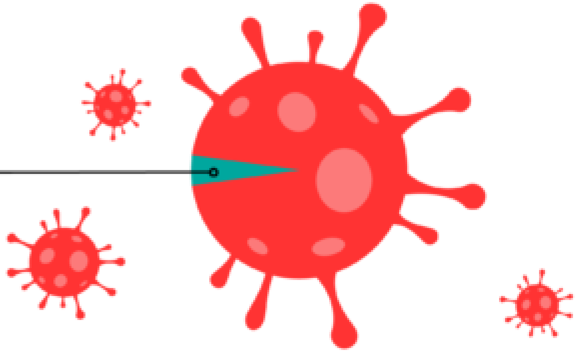


Cost of Preventing The Next Pandemic Just 2% Of Covid-19 Bill

Estimated cost of containing future pandemic compared to Covid-19's economic damage

Source: Dobson et al, Science 2020

Pandemic Prevention – a Climate Solution



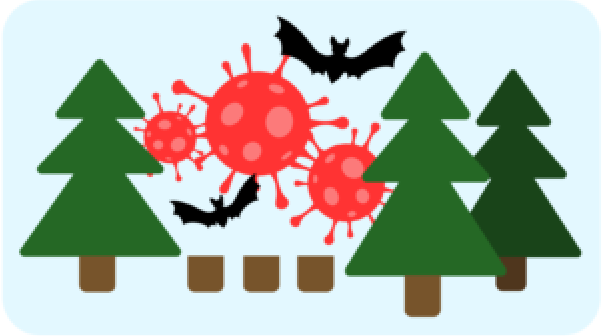
it will cost the US only **$30 billion**

(less than 1% of what it spent controlling COVID-19 this year)

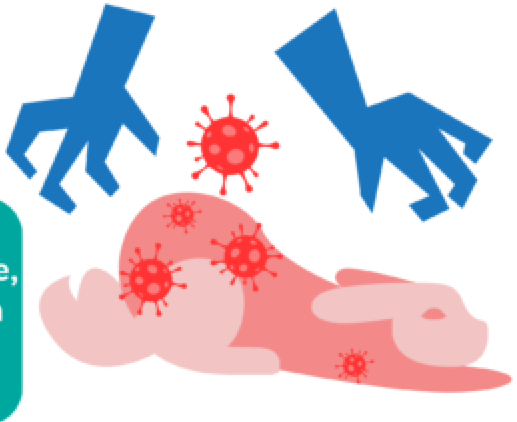
to prevent another animal-borne pandemic.

That's great! **But HOW?**

Prevention starts at the source. **Forests** provide a **natural shield** that prevents people from contracting animal viruses, like **COVID-19**, MERS, and SARS. When trees are cut down, **viruses escape** with the wildlife, who come into contact with livestock and people.



Similarly, unregulated **wildlife trade** brings people into contact with animal viruses.



Investing $30 billion in a 10-year plan to reduce deforestation, regulate wildlife trade, and contain viruses through early detection could prevent another global tragedy and save trillions of dollars in disease control.

Source: https://www.conservation.org/blog/3-ways-to-prevent-the-next-pandemic-with-nature-according-to-science

OUR DAILY PLANET


GET THEE AWAY, CORONAVIRUS!
THIS IS GONNA BE SO EASY...
P
LALO ALCARAZ ©2020 DISTRIBUTED BY ANDREWS McMEEL SYNDICATION


WHO NEEDS W.H.O. WHEN YOU'VE GOT ME?!
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Many thanks to
Amy Ando and Ted Loch-Temzenides,
Stuart Pimm and Peter Daszak,
Margaret Kinnaird, Katrzyna Nowak,
Jorge Ahumado, Marcia Castro,
Mariana M. Vale, Binbin Li, Hongying Li,
Jonah Busch, Colin Chapman,
Carlos Zambrana-Torrelio, Lingyun Xiao,
Les Kaufman, Lee Hannah

SYSENC/NSERC Emerging pathogens:
Christian Faust, Mercedes Pascual,
Raina Plowright, Hamish McCallum,
Nicole Gottdenker, Laura Bloomfield,
Tom Gillespie & Maria Diuk-Wasser

House Finches and Mycoplasma:
Andre Dhondt, Dana Hawley,
Wes Hochachka, Parviez Hosseini,
Ariana Fleming Davies, Paul Williams
Eric Osnas and Paul Hurtado.