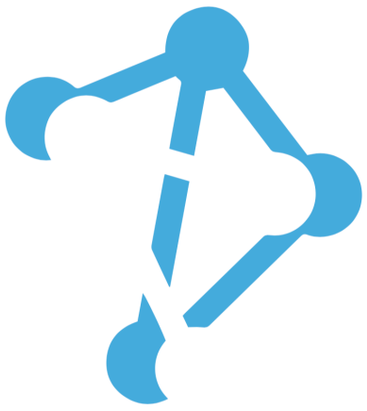
Network Science Theory and COVID-19

Samuel V. Scarpino, PhD
Assistant Professor
Network Science Institute
Northeastern University

External Faculty
Santa Fe Institute
Vermont Complex Systems Center

Co-Founder, Global.health

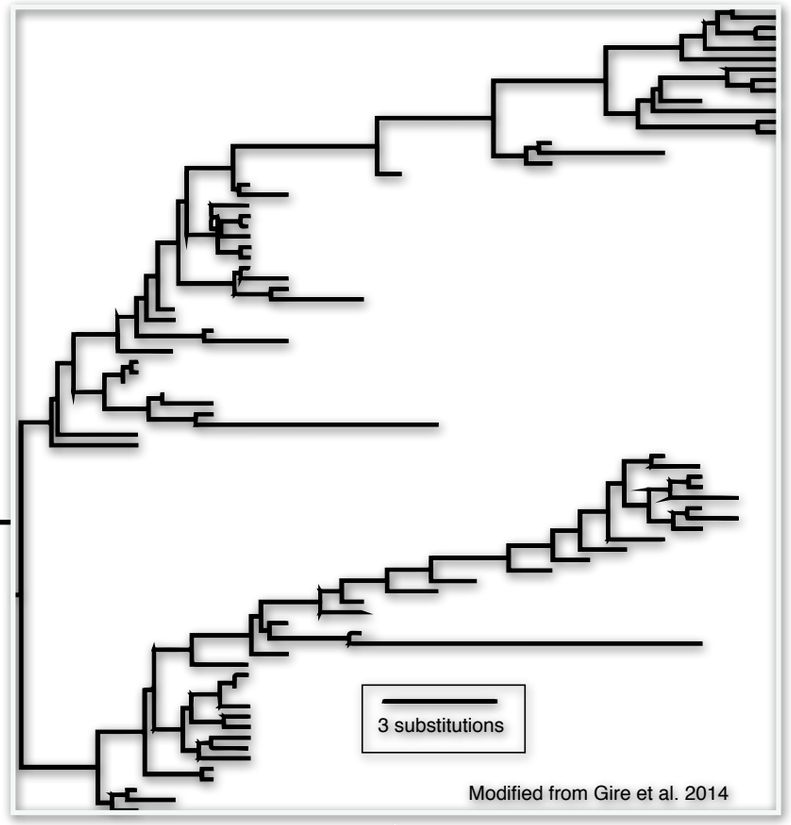
@svscarpino
scarpino.github.io
s.scarpino@northeastern.edu



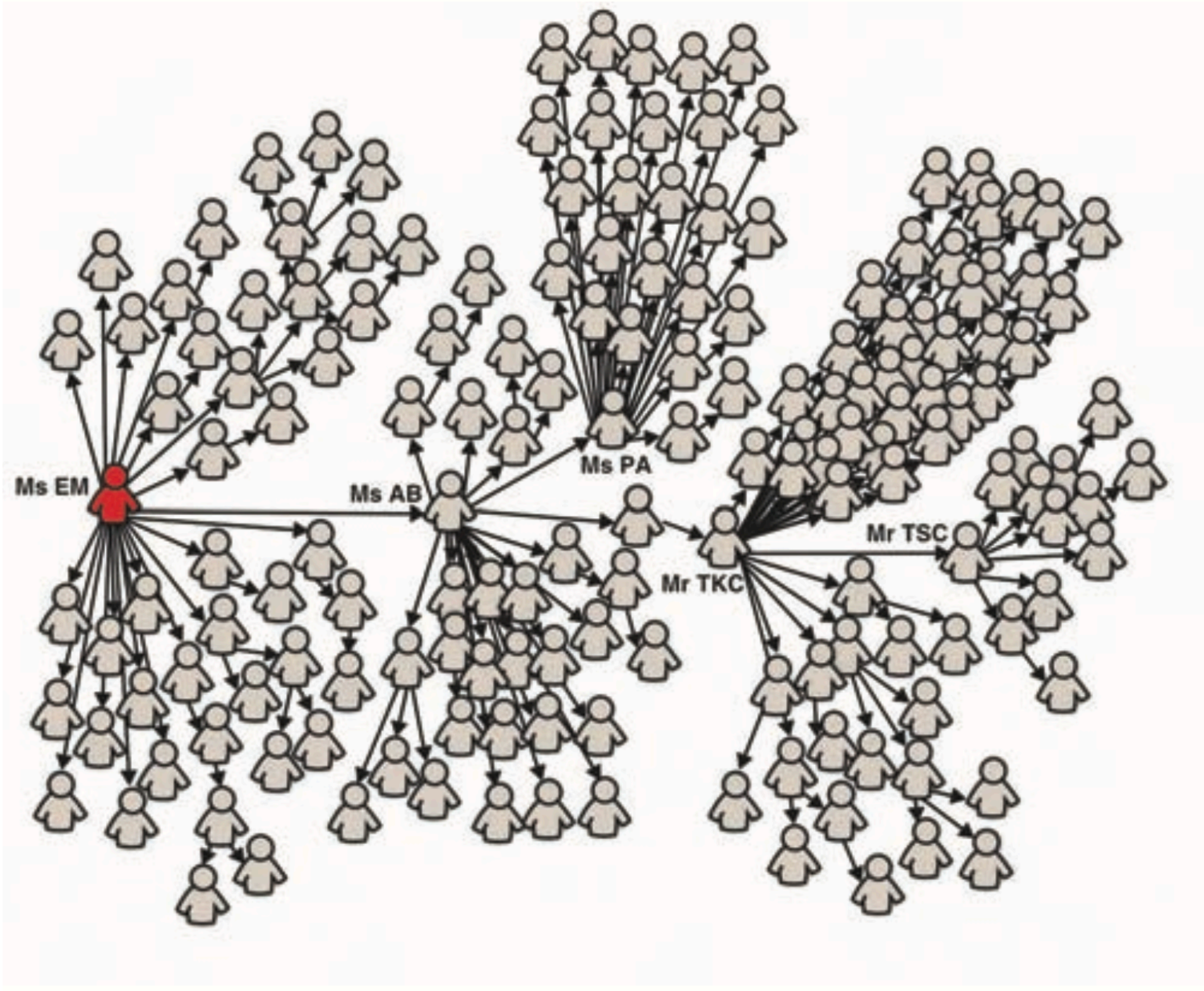
Emergent Epidemics Lab

COVID-19 became a pandemic because the world doesn’t understand complex systems (networks)

Complex systems and disease



Modified from Gire et al. 2014



This diagram detailing SARS transmission in Singapore shows the important role of "super-spreaders" in transmitting the disease. Five people caused more than half of the 205 cases there.

The SARS Scare

// A cautionary tale of emerging disease caught in the act

by Jonathan Shaw

At the height of the epidemic, one Canadian infectious-disease expert who had come down with SARS herself predicted that the virus would spread around the globe: "If we don't have a vaccine—yes, we are all going to get it," she told Canadian television. Her opinion was shared by many that spring.

Network theory and SARS: predicting outbreak diversity

Lauren Ancel Meyers[a,b,*,1], Babak Pourbohloul[c,1,2], M.E.J. Newman[b,d], Danuta M. Skowronski[c,2], Robert C. Brunham[c,2]

[a]*Section of Integrative Biology and Institute for Cellular and Molecular Biology, University of Texas at Austin, 1 University Station C0930, Austin, TX 78712, USA*

[b]*Santa Fe Institute, 1399 Hyde Park Road, Santa Fe, NM 87501, USA*

[c]*University of British Columbia Centre for Disease Control, 655 West 12th Avenue, Vancouver, British Columbia, Canada V5Z 4R4*

[d]*Center for the Study of Complex Systems, University of Michigan, Randall Laboratory, 500 E. University Ave., Ann Arbor, MI 48109-1120, USA*

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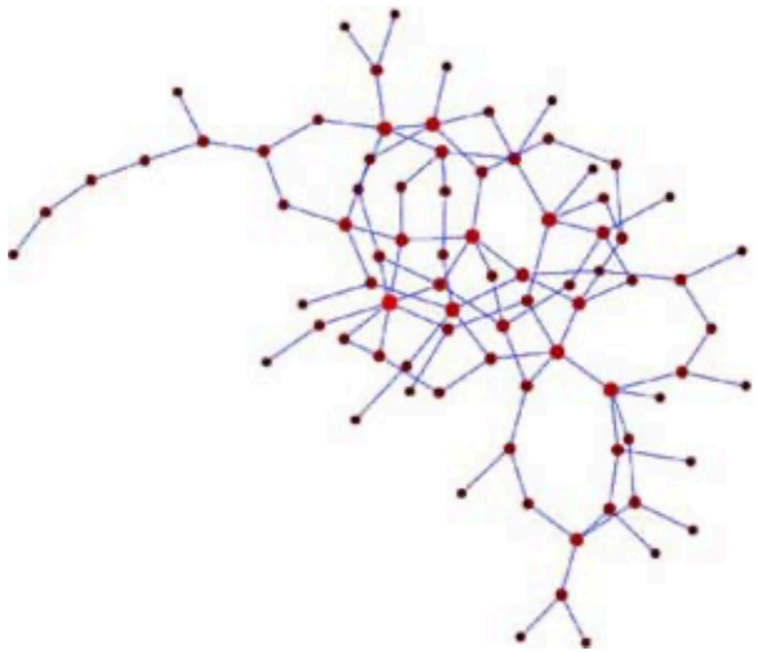
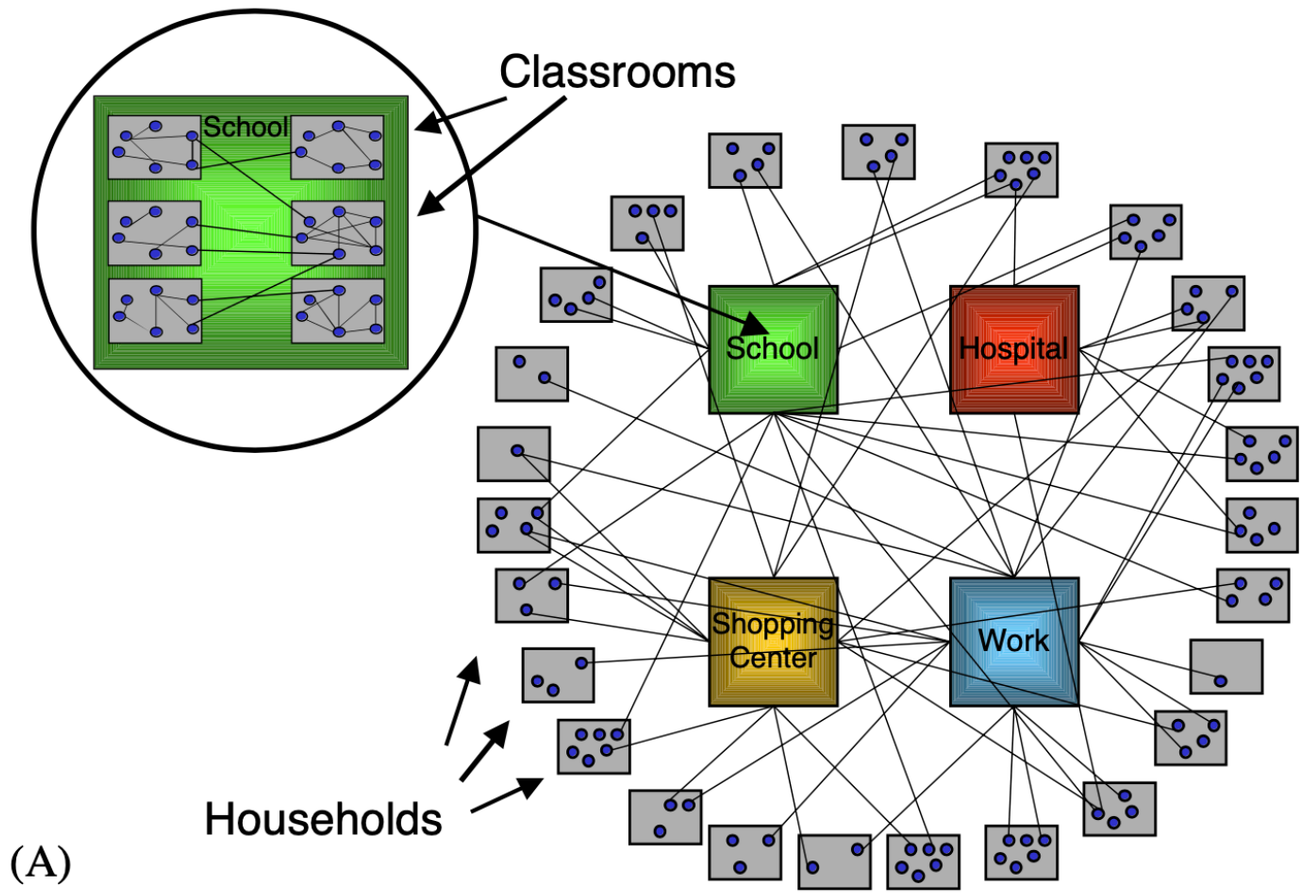
Available online 23 September 2004

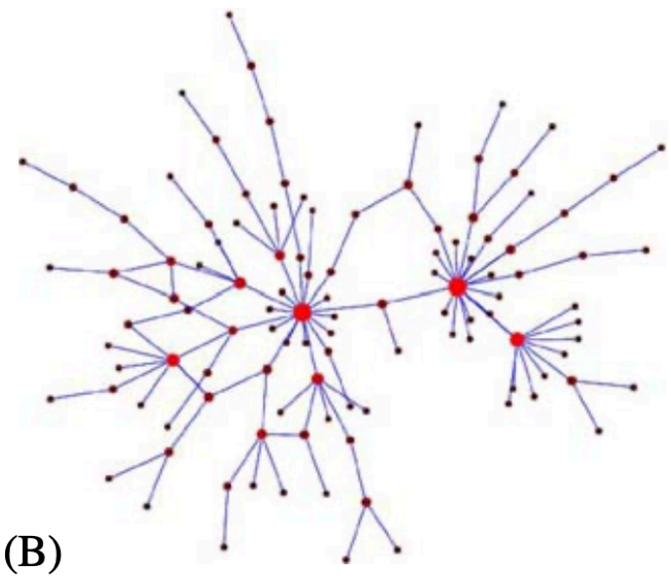
Poisson social contact network

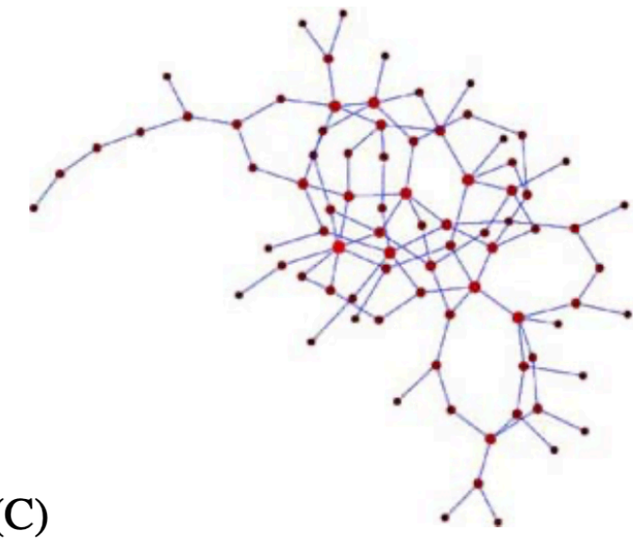


Meyers et al. 2004

Real-world social contact networks

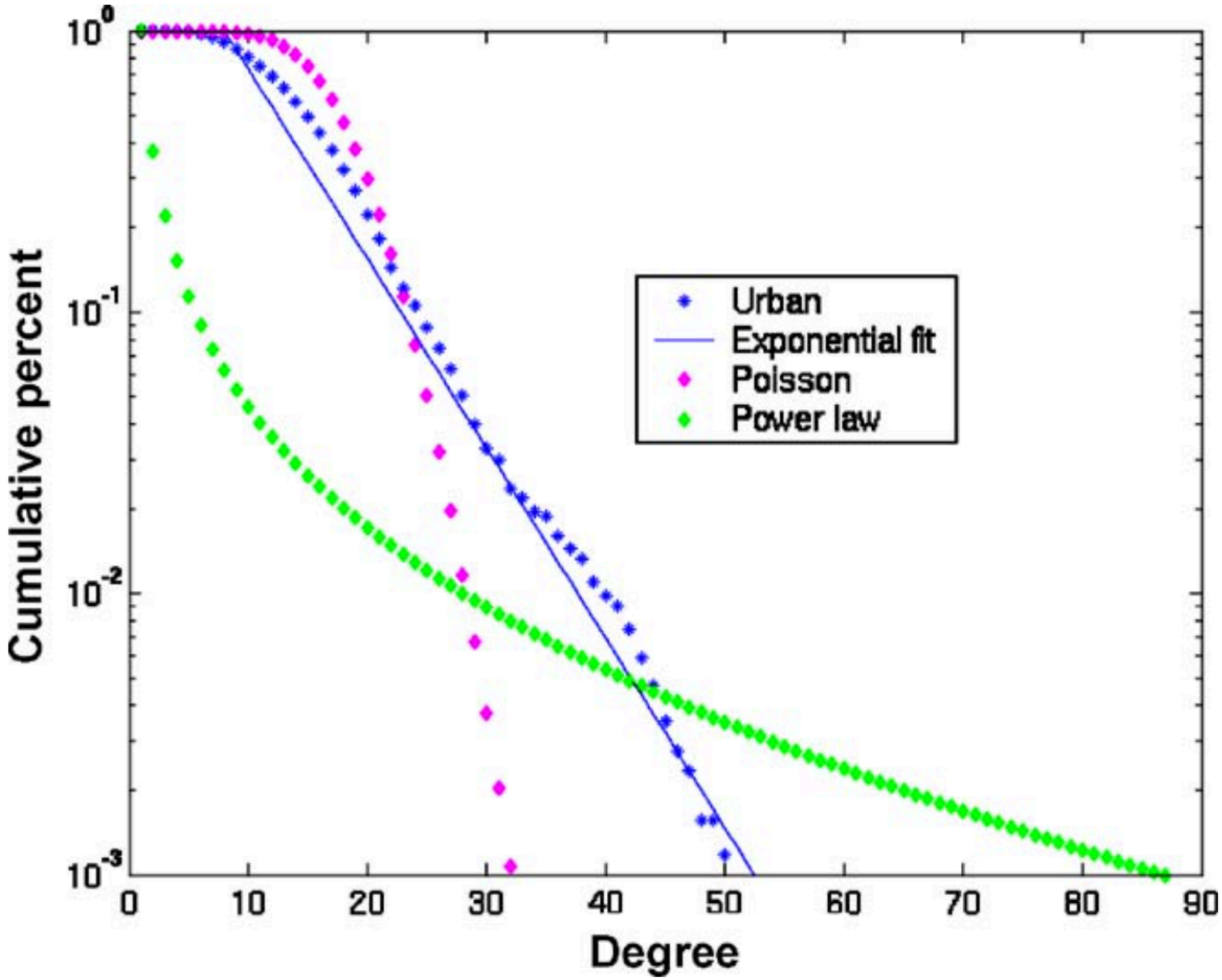






Meyers et al. 2004

Degree distributions of real-world social networks



Meyers et al. 2004

Epidemic Spreading in Scale-Free Networks

Romualdo Pastor-Satorras[1] and Alessandro Vespignani[2]

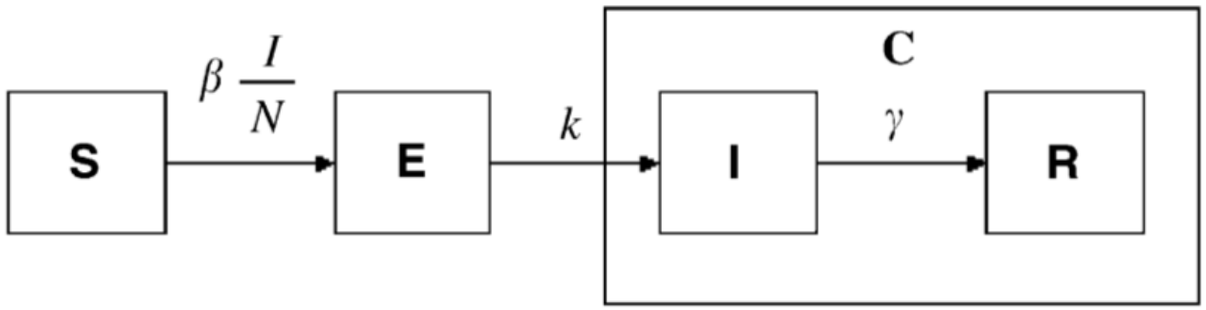
[1]*Departament de Física i Enginyeria Nuclear, Universitat Politècnica de Catalunya, Campus Nord, Mòdul B4, 08034 Barcelona, Spain*

[2]*The Abdus Salam International Centre for Theoretical Physics (ICTP), P.O. Box 586, 34100 Trieste, Italy*

(Received 20 October 2000)

The Internet has a very complex connectivity recently modeled by the class of scale-free networks. This feature, which appears to be very efficient for a communications network, favors at the same time the spreading of computer viruses. We analyze real data from computer virus infections and find the average lifetime and persistence of viral strains on the Internet. We define a dynamical model for the spreading of infections on scale-free networks, finding the absence of an epidemic threshold and its associated critical behavior. This new epidemiological framework rationalizes data of computer viruses and could help in the understanding of other spreading phenomena on communication and social networks.

Lions, Tigers, and Boxes … oh my



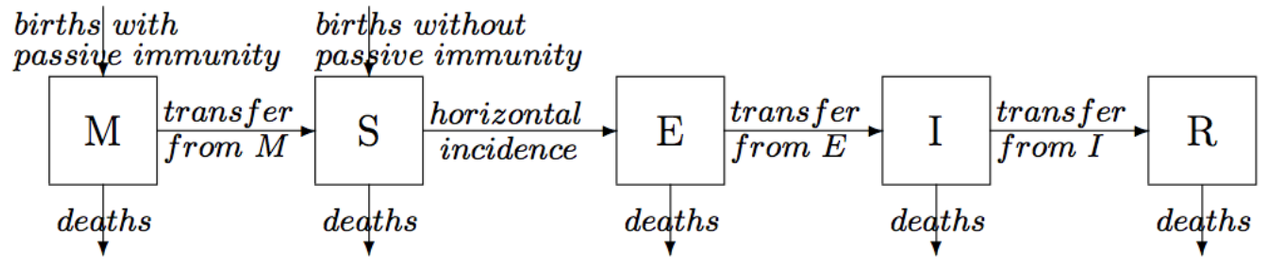


Fig. 1 *The general transfer diagram for the MSEIR model with the passively immune class M, the susceptible class S, the exposed class E, the infective class I, and the recovered class R.*

$$S'(t) = \mu \cdot (1 - wP - aP) - \beta[I_s(t) + I_a(t)]S(t) - \nu S(t) \quad (1)$$

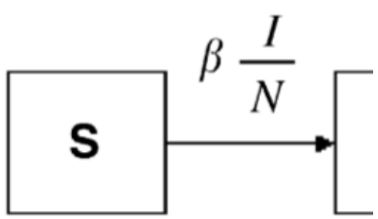
$$I_s'(t) = \beta\sigma[I_s(t) + I_a(t)]S(t) - \gamma_s I_s(t) - \nu I_s(t) \quad (2)$$

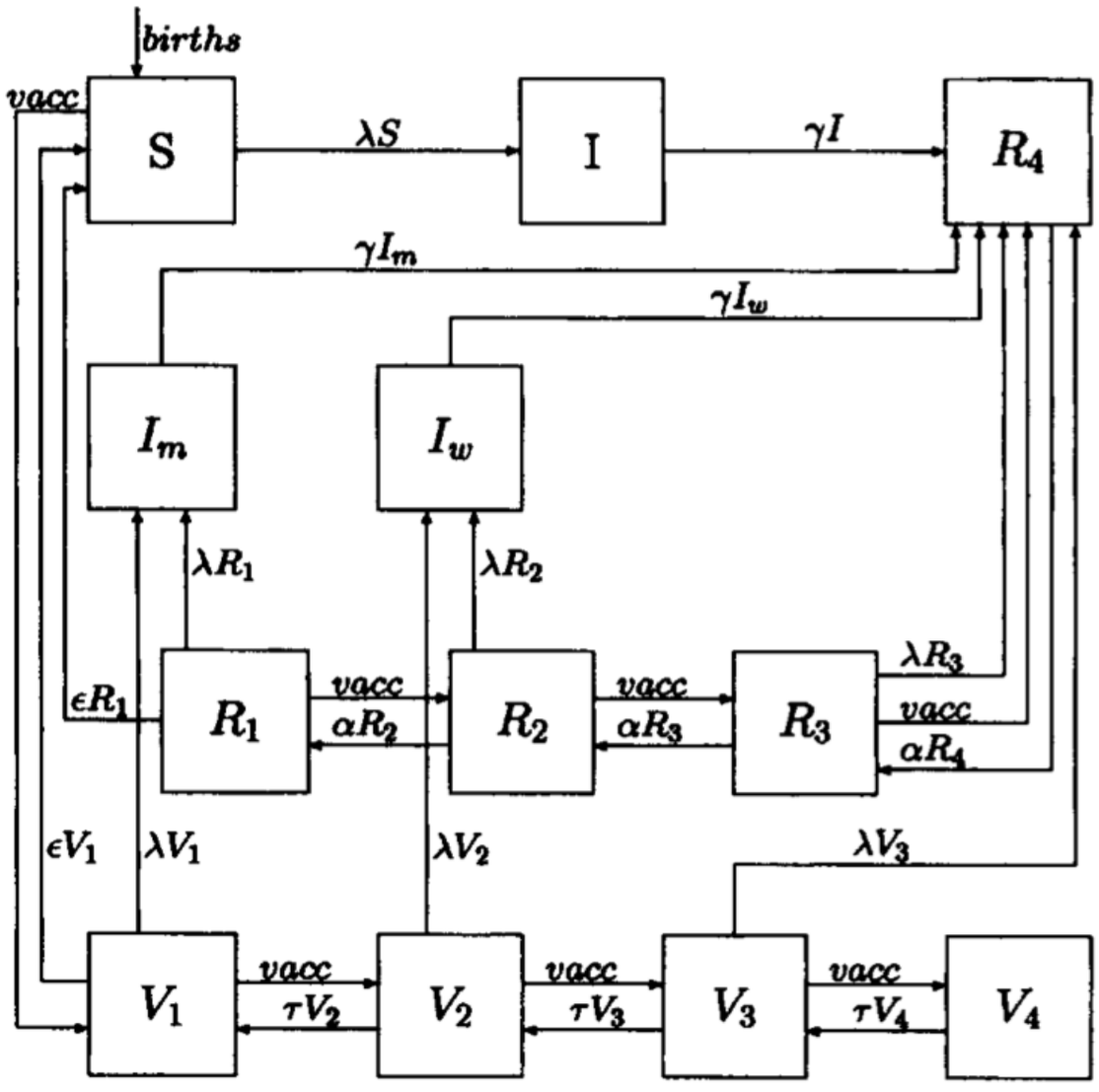
$$I_a'(t) = \beta(1 - \sigma)[I_s(t) + I_a(t)]S(t) + \beta[I_s(t) + I_a(t)]V(t) - \gamma_a I_a(t) - \nu I_a(t) \quad (3)$$

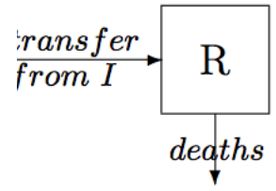
$$V'(t) = \mu \cdot aP - \beta[I_s(t) + I_a(t)]V(t) - \nu V(t) \quad (4)$$

$$R'(t) = \mu \cdot wP + \gamma_s I_s(t) + \gamma_a I_a(t) - \nu R(t) \quad (5)$$

Lions, Tigers, and Boxes … oh my







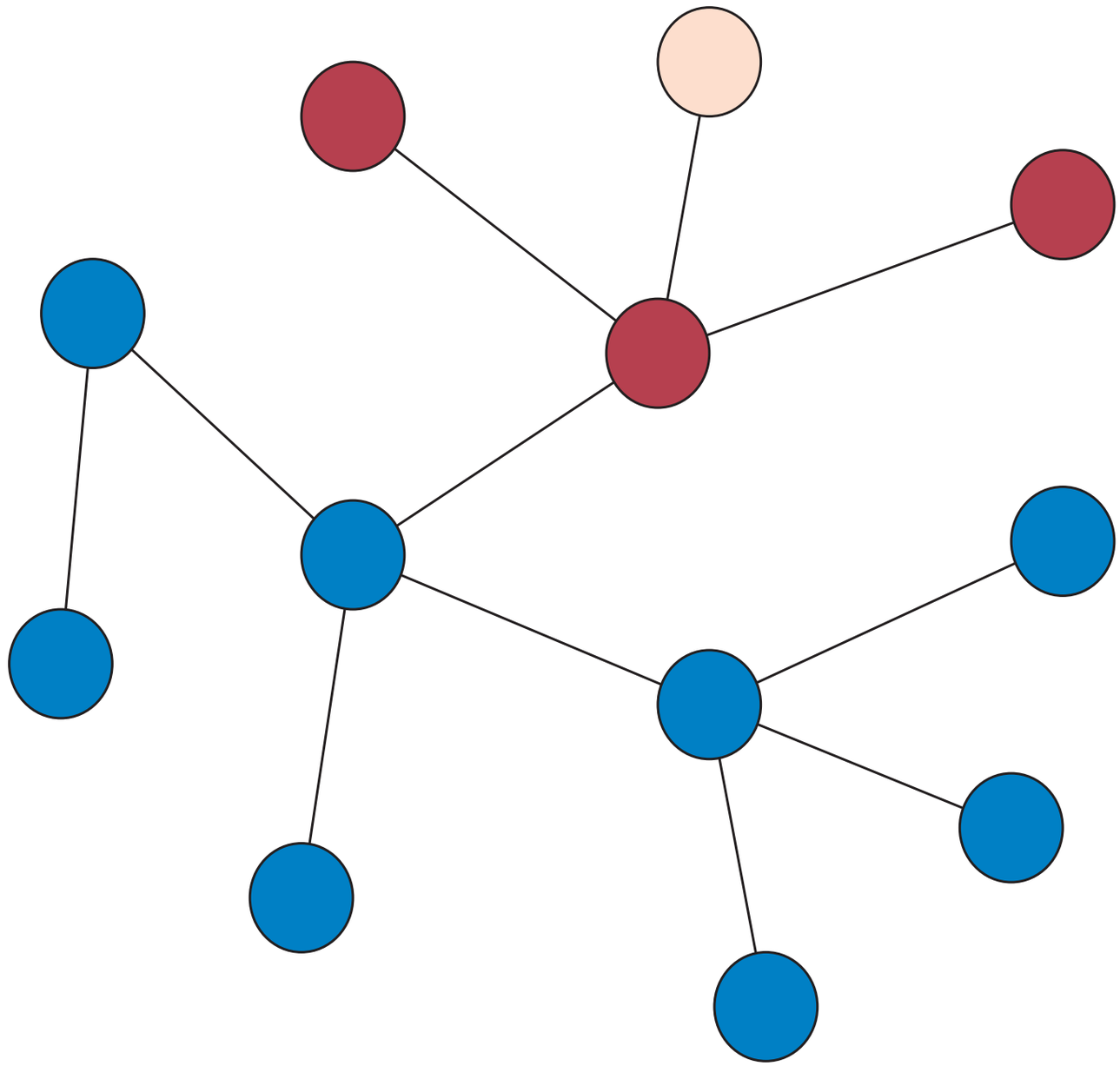
y immune class M, the recovered class R.

$$
\begin{aligned}
S'(t) &= \mu \cdot (1 - \\
I_s'(t) &= \beta\sigma[I_s(t \\
I_a'(t) &= \beta(1 - \epsilon \\
V'(t) &= \mu \cdot aP \\
R'(t) &= \mu \cdot wP
\end{aligned}
$$

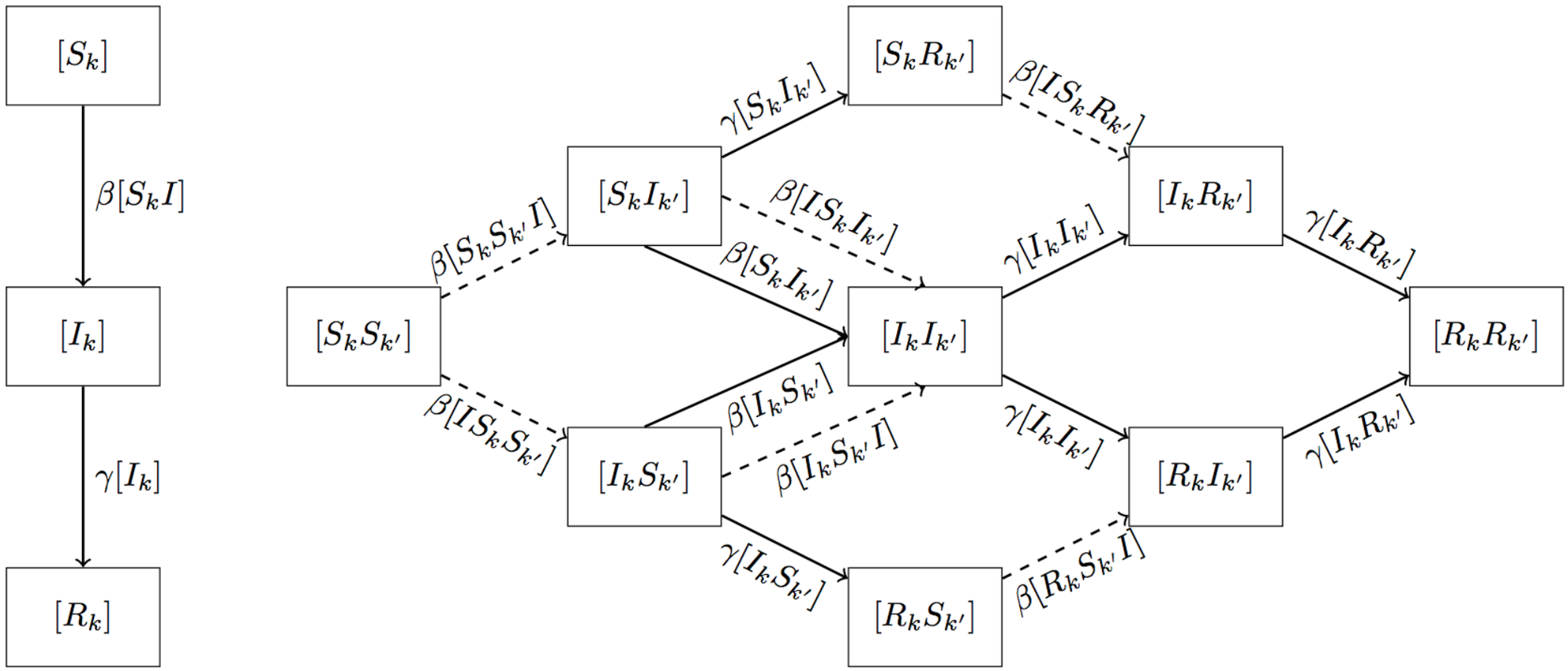
FIG. 3. Transfer diagram for the pertussis model with vaccination.

Model complexity

Network Epidemiology



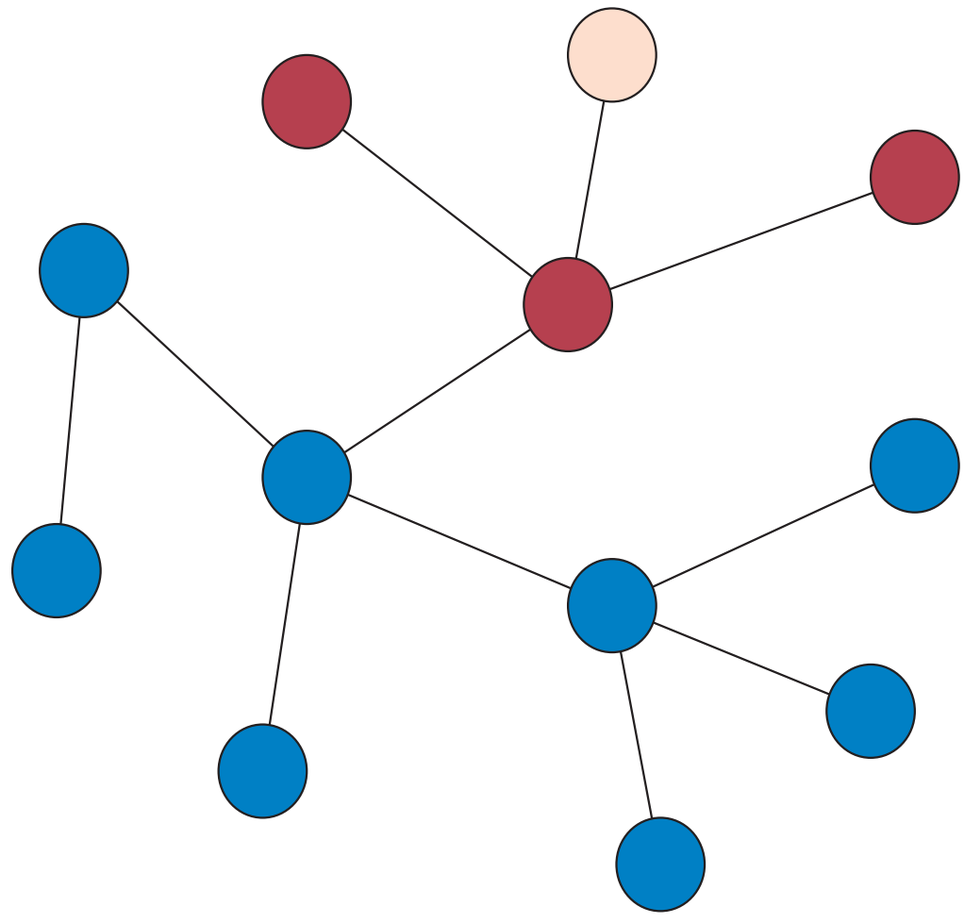
Pairwise



Miller and Kiss 2014

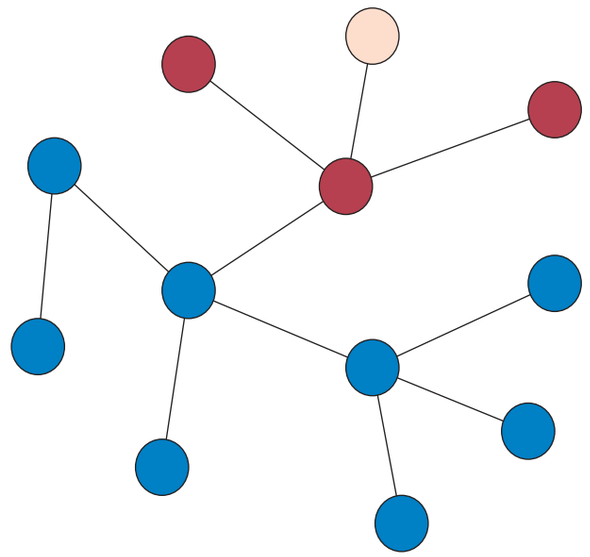
Network Epidemiology

$$R_0 = T\left(\frac{\langle K^2\rangle - \langle K\rangle}{\langle K\rangle}\right)$$



Network Epidemiology Vs. Standard Calculation

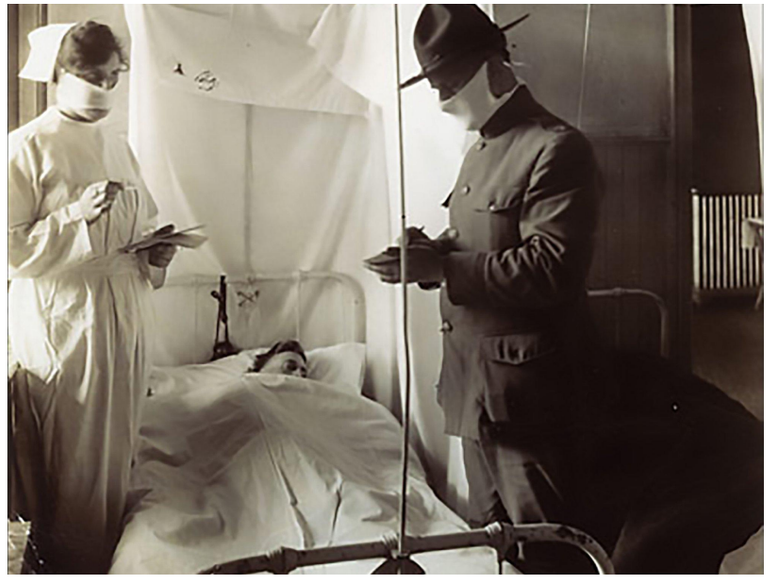
$$R_0 = T\left(\frac{\langle K^2\rangle - \langle K\rangle}{\langle K\rangle}\right)$$



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

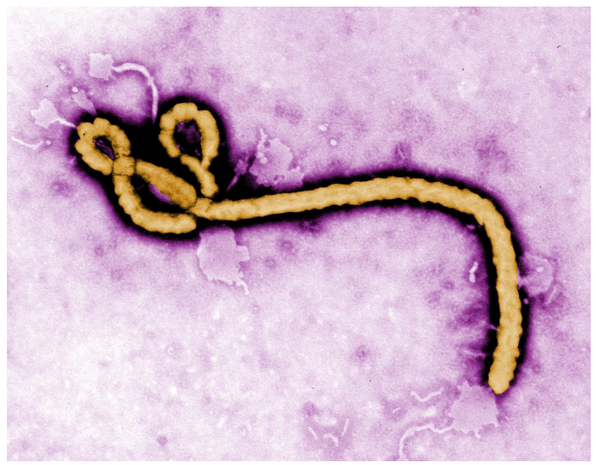
Same R0, very different stories

1918 influenza
R0 ~ 2
500,000,000 infected



2013-16 Ebola
R0 ~ 2
30,000 infected

Why do these problems exist?

1. 

Intrinsic properties of the pathogen

2. 

Behavior patterns of the host

Vol 438|17 November 2005|**doi:10.1038/nature04153**

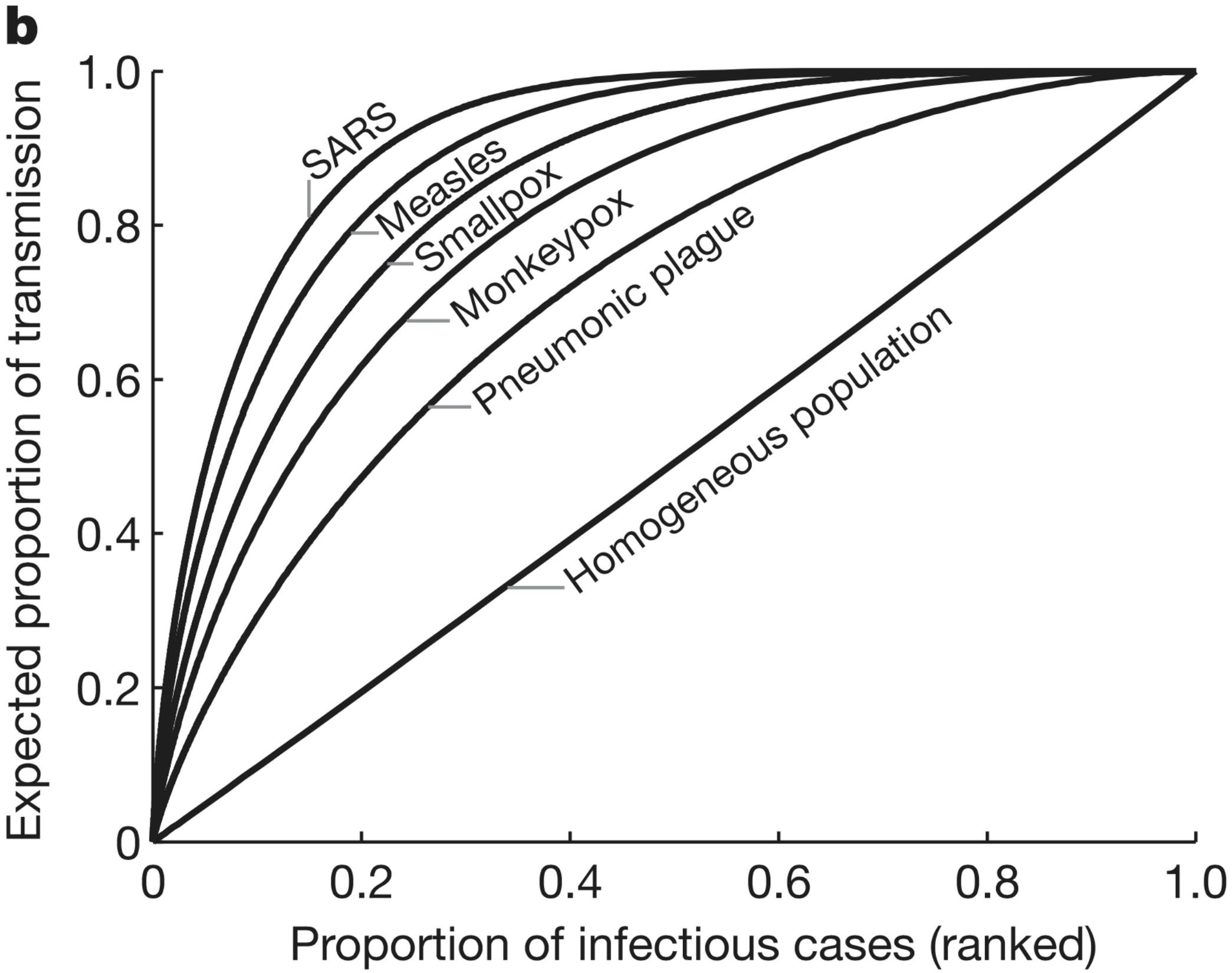
nature

LETTERS

Superspreading and the effect of individual variation on disease emergence

J. O. Lloyd-Smith[1,2], S. J. Schreiber[3], P. E. Kopp[4] & W. M. Getz[1]

b



Lloyd-smith et al. 2005

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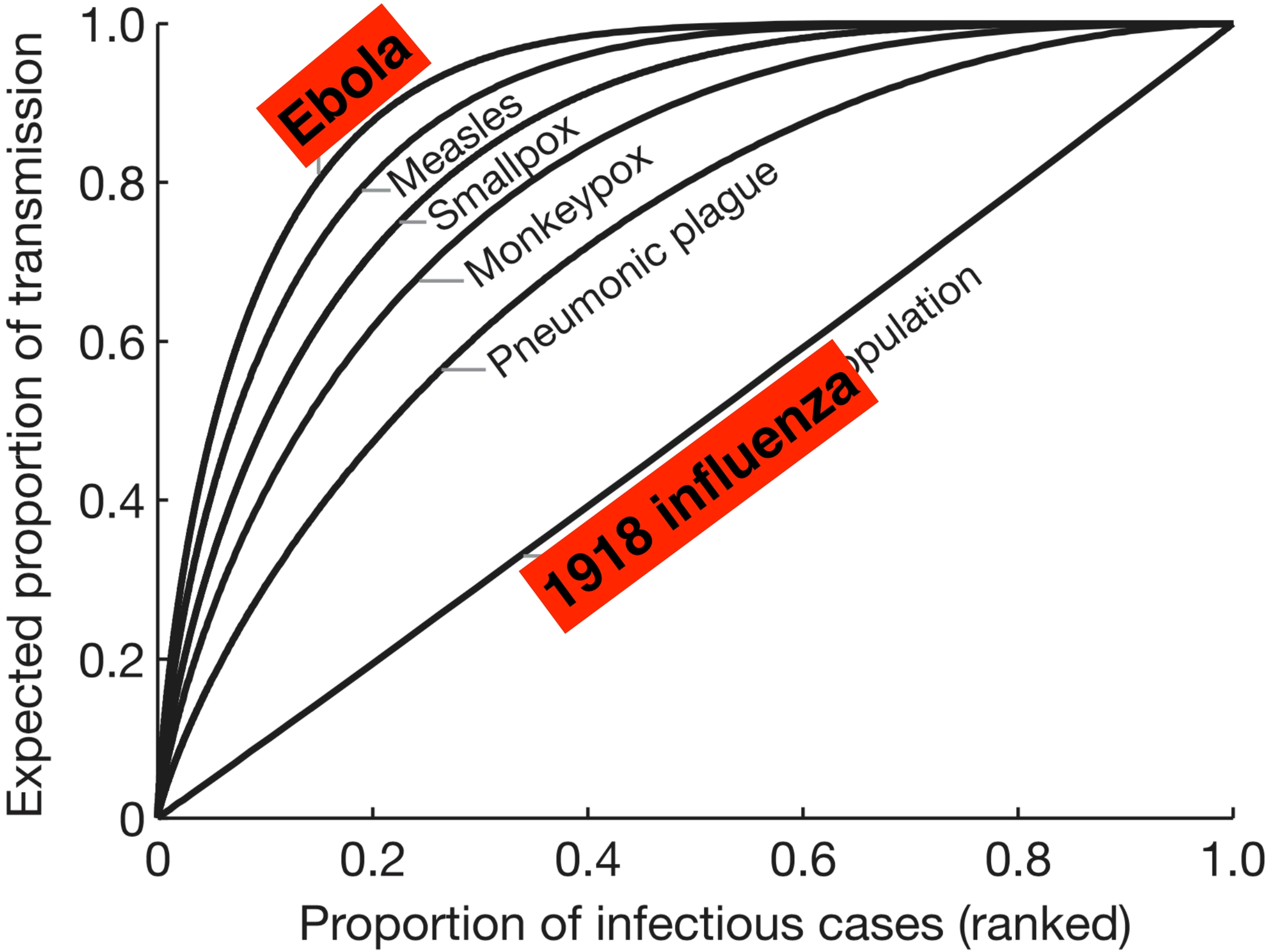

Lloyd-smith et al. 2005

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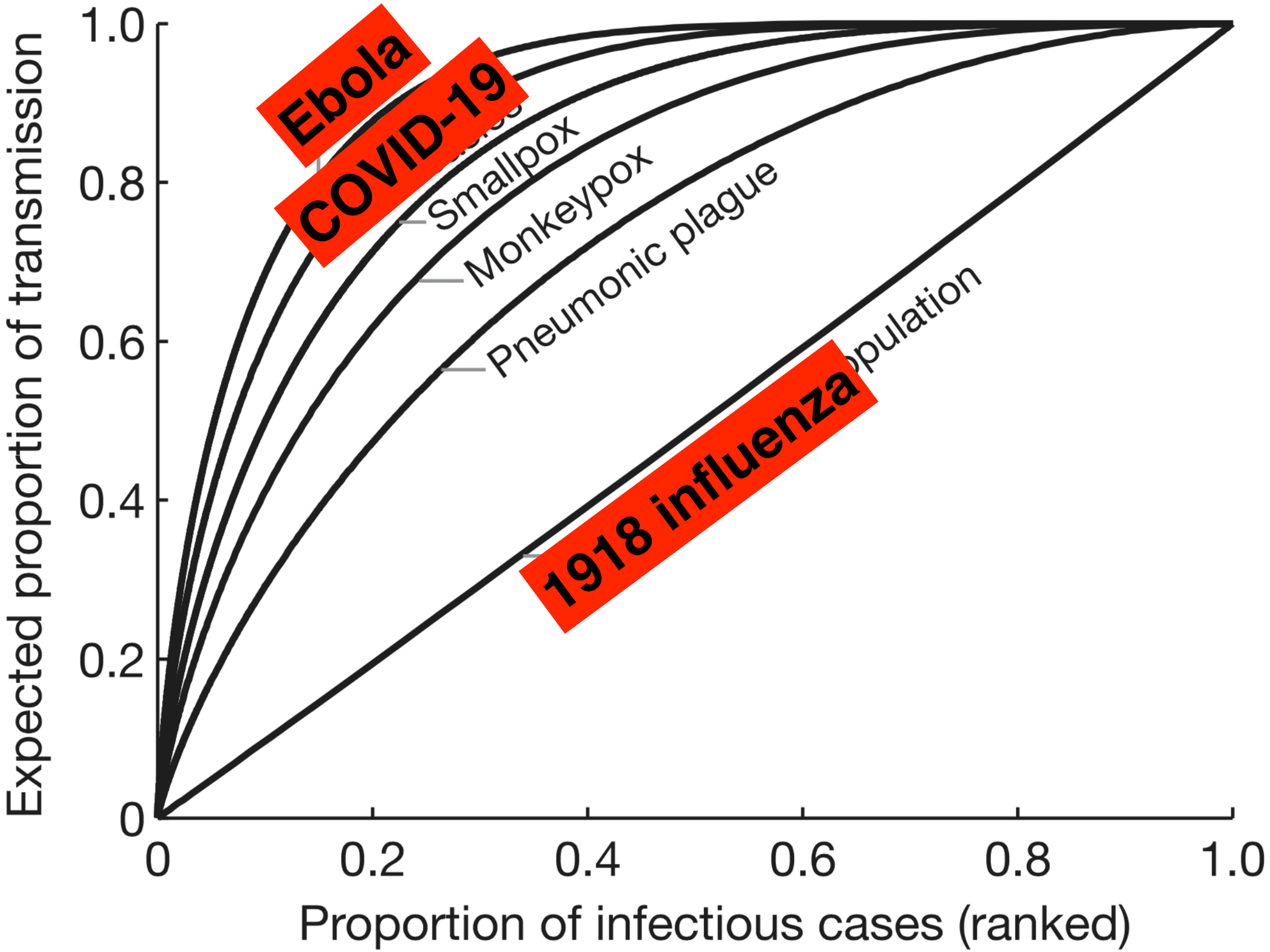


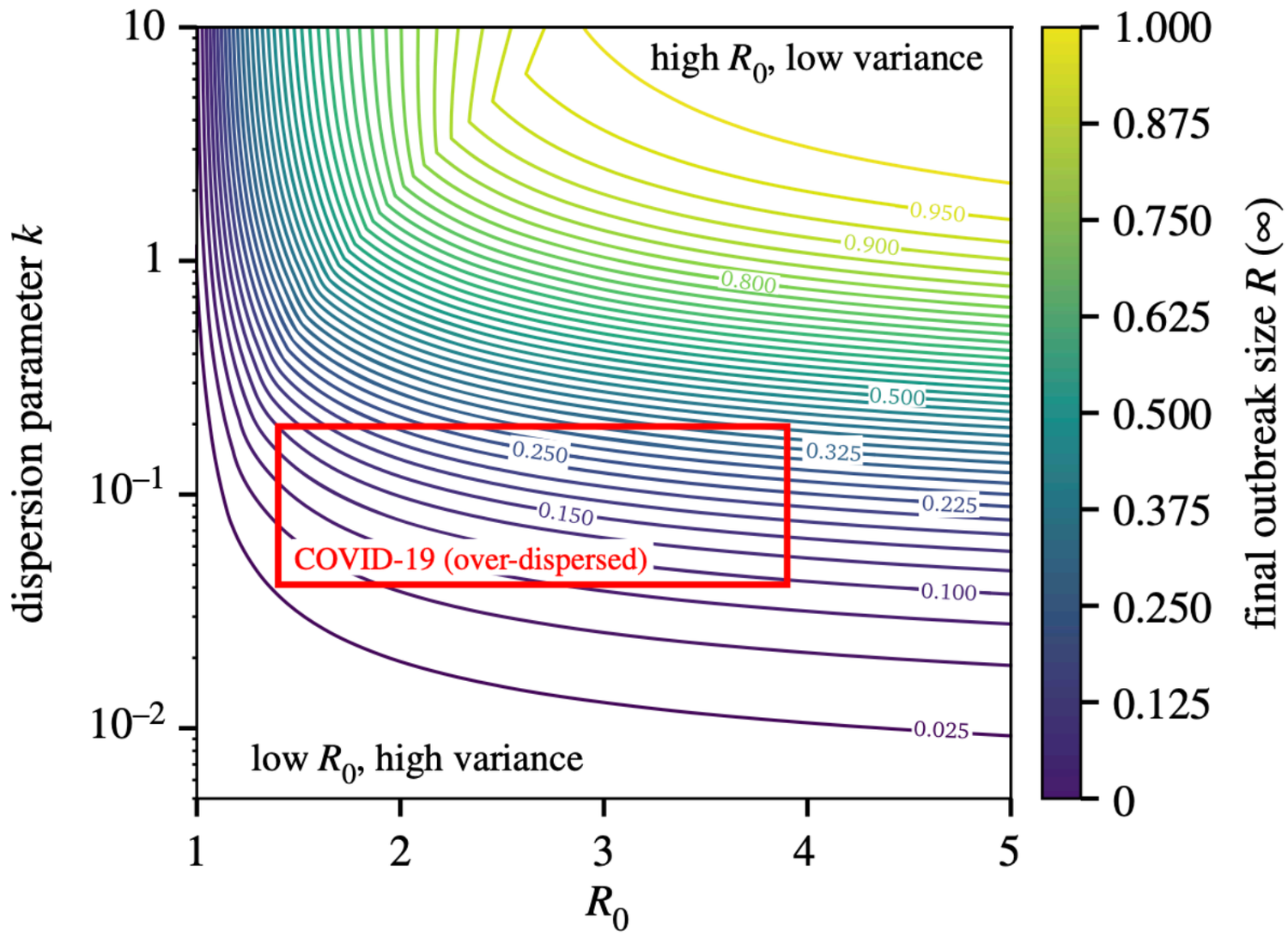
Lloyd-smith et al. 2005

Can we get a more “realistic” estimate of size?

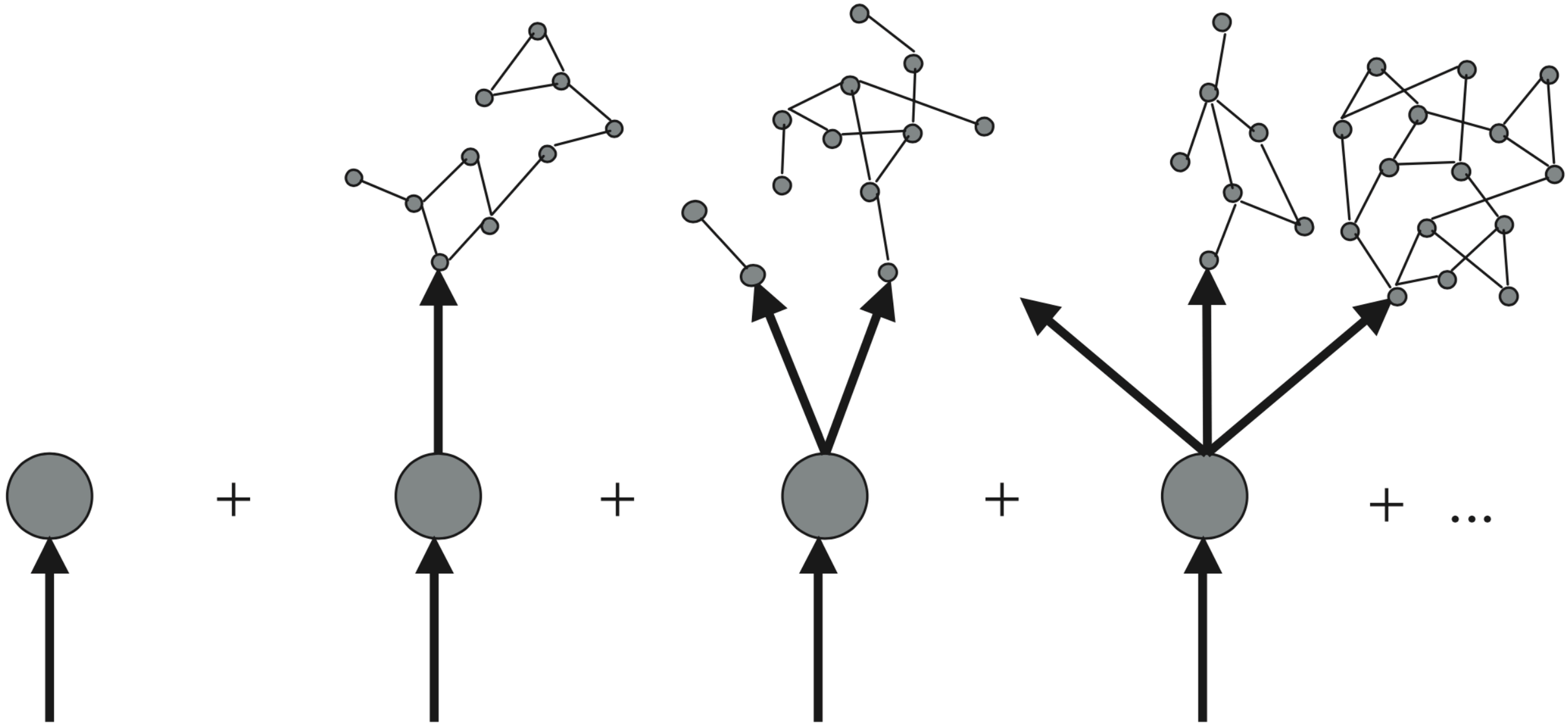
1. the disease results either in complete immunity or death,
2. the disease is transmitted in a closed population,
3. all individuals are equally susceptible,
4. contacts occur according to the law of mass-action,
5. and a deterministic analysis is appropriate.

Beyond R_0: heterogeneity in secondary infections and probabilistic epidemic forecasting

Laurent Hébert-Dufresne[1,2,3], Benjamin M. Althouse[4,5,6], Samuel V. Scarpino[7,8,9,10,11,12] and Antoine Allard[3,13]



CONTACT NETWORK EPIDEMIOLOGY



Meyers 2007

$$u = G_1(u) = \exp\left[\sum_{n=1}^{\infty} \frac{1}{n!} \kappa_n (\ln u)^n\right]$$

$$u = G_1(u) = \exp\left[\sum_{n=1}^{\infty} \frac{1}{n!} \kappa_n (\ln u)^n\right]$$

$$= \exp\left[R_0 |\ln u| - \frac{1}{2}\sigma^2 |\ln u|^2 + \frac{1}{6}\kappa_3 |\ln u|^3 - \frac{1}{24}\kappa_4 |\ln u|^4 \ldots\right]$$

He´bert-Dufresne et al. 2020. Proc. Roy. Soc. Interface

$$= \exp\left[R_0|\ln u| - \frac{1}{2}\sigma^2|\ln u|^2 + \frac{1}{6}\kappa_3|\ln u|^3 - \frac{1}{24}\kappa_4|\ln u|^4 \ldots\right]$$

> “A disease needs a high average number of secondary infections (high $\kappa_1 = R_0$) to spread, but, given that average, a disease with small variance in secondary infections will spread much more reliably and be less likely to stochastically die out. Given a variance, a disease with high skewness (i.e. with positive deviation contributing to most of the variance) will be more stable than a disease with negative skewness (i.e. with most deviations being towards small secondary infections). Given a skewness, a disease will be more stable if it has frequent small positive deviations rather than infrequent large deviations—hence a smaller kurtosis—as stochastic die out could easily occur before any of those large infrequent deviations occur.”

He´bert-Dufresne et al. 2020. Proc. Roy. Soc. Interface

We can connect Lloyd-smith et al. to networks!

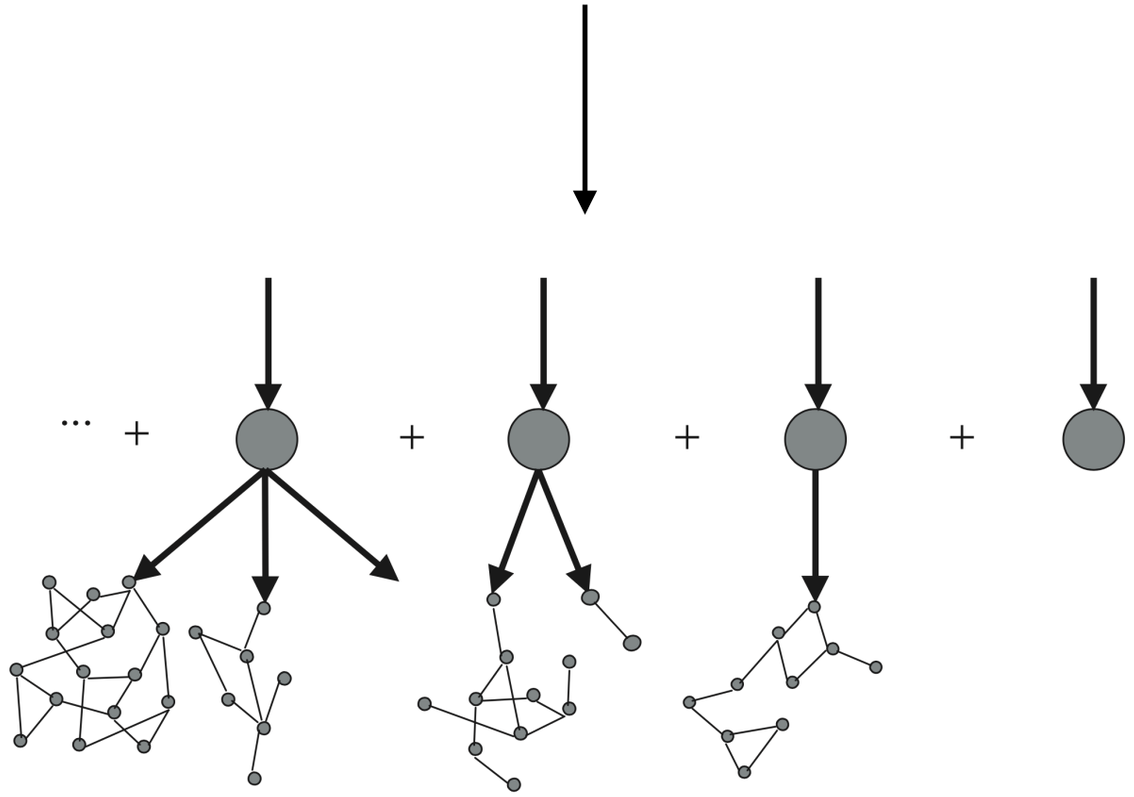
Vol 438|17 November 2005|**doi:10.1038/nature04153**

nature

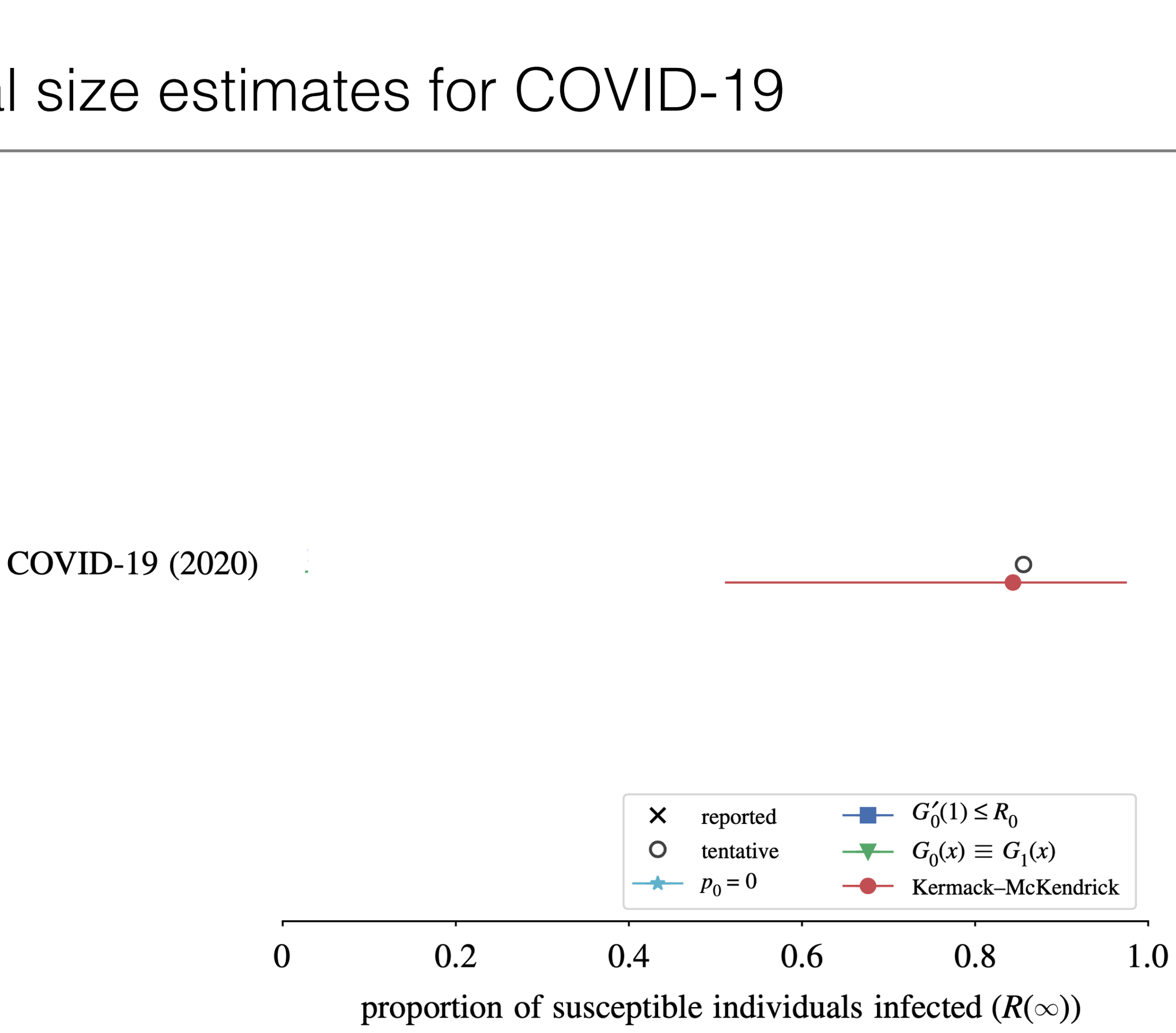
LETTERS

Superspreading and the effect of individual variation on disease emergence

J. O. Lloyd-Smith[1,2], S. J. Schreiber[3], P. E. Kopp[4] & W. M. Getz[1]



Final size estimates for COVID-19



He´bert-Dufresne et al. 2020. Proc. Roy. Soc. Interface

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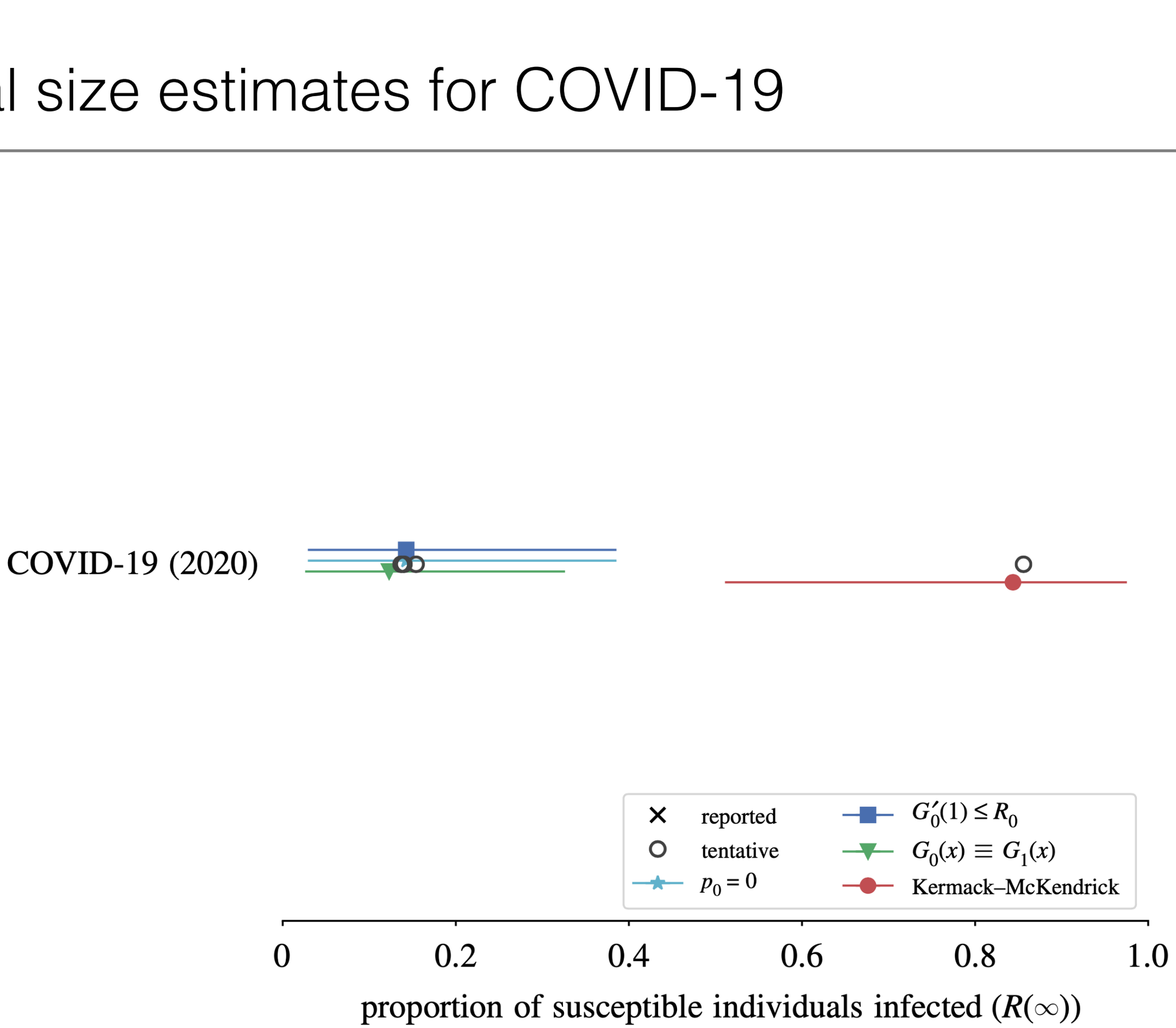
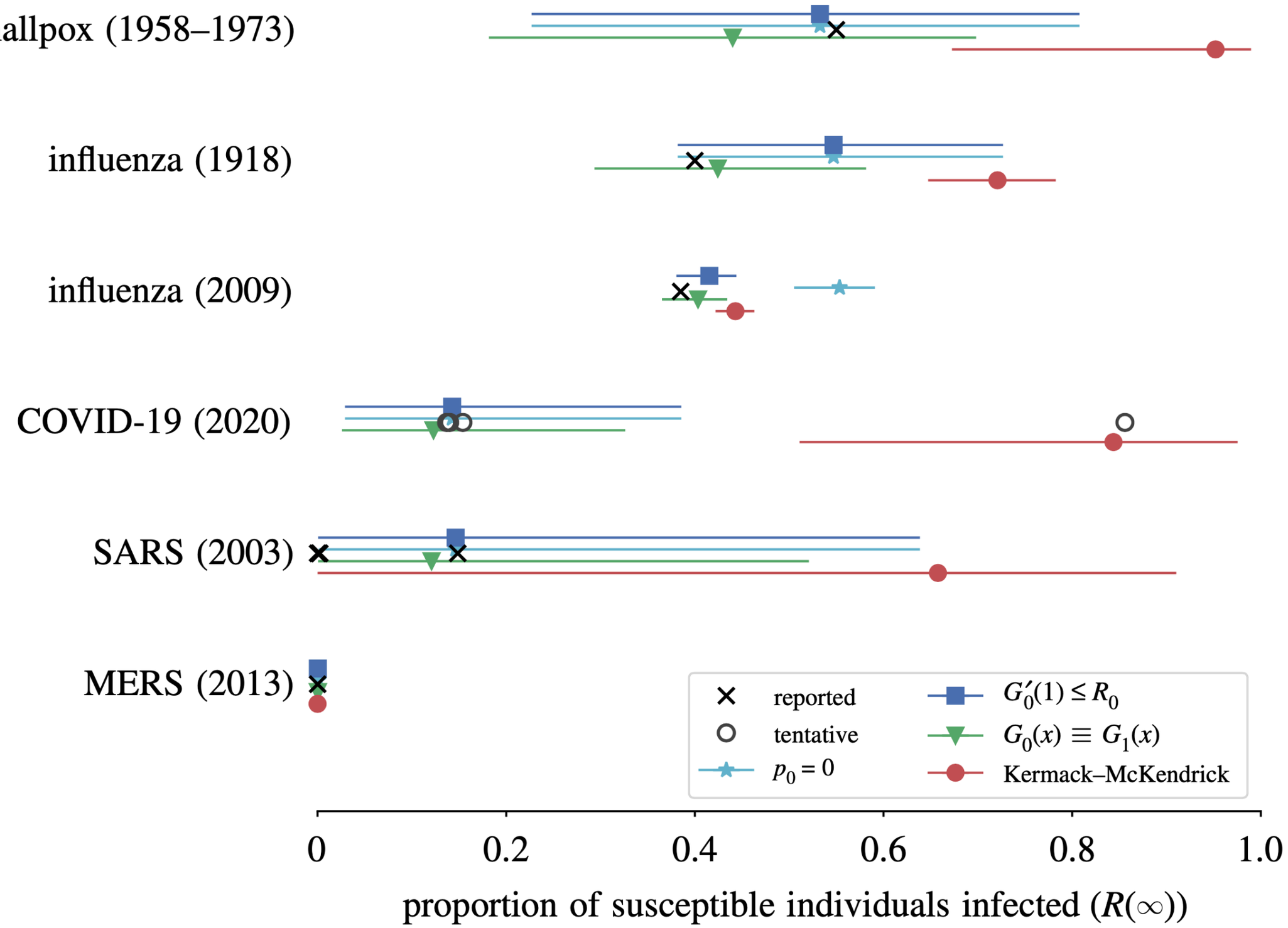


He´bert-Dufresne et al. 2020. Proc. Roy. Soc. Interface

Note the *large* disagreement for COVID-19



He´bert-Dufresne et al. 2020. Proc. Roy. Soc. Interface

There's a *potential* benefit to super spreading

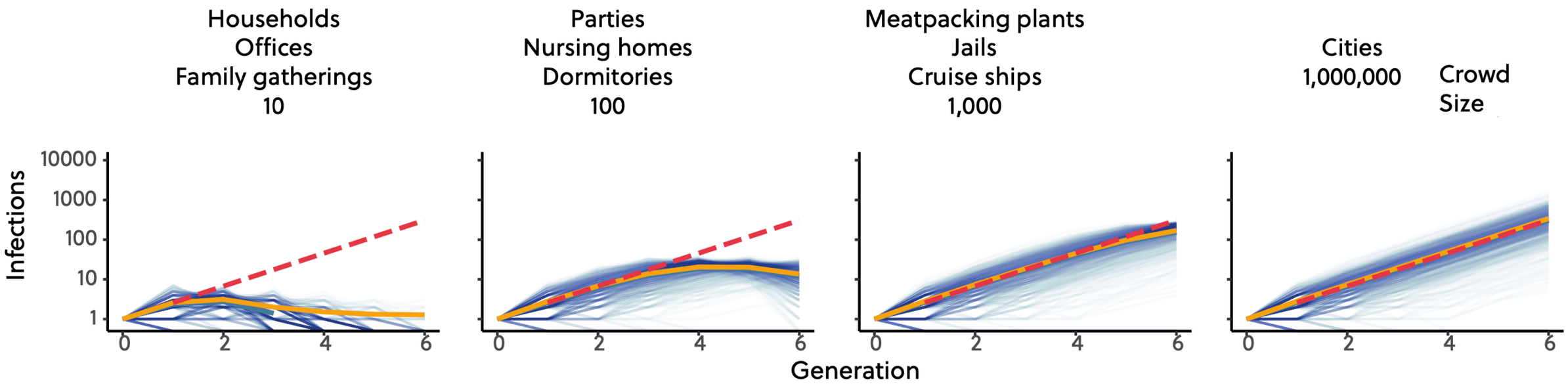
Superspreading events in the transmission dynamics of SARS-CoV-2: opportunities for interventions and control

Benjamin M. Althouse[1,2,3,*,+], Edward A. Wenger[1,+], Joel C. Miller[4], Samuel V. Scarpino[5,6,7,8,9,10], Antoine Allard[11,12], Laurent Hébert-Dufresne[11,13,14], and Hao Hu[15,+]

Althouse et al. 2020. PLoS Biology

There's a *potential* benefit to super spreading

No super-spreading, R0 = 2.6



Althouse et al. 2020. PLoS Biology

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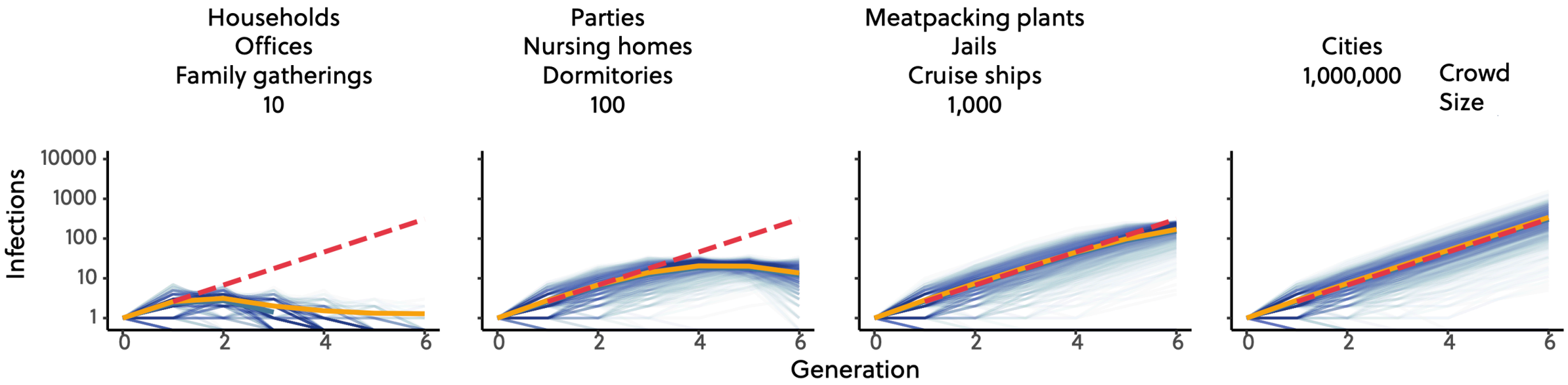
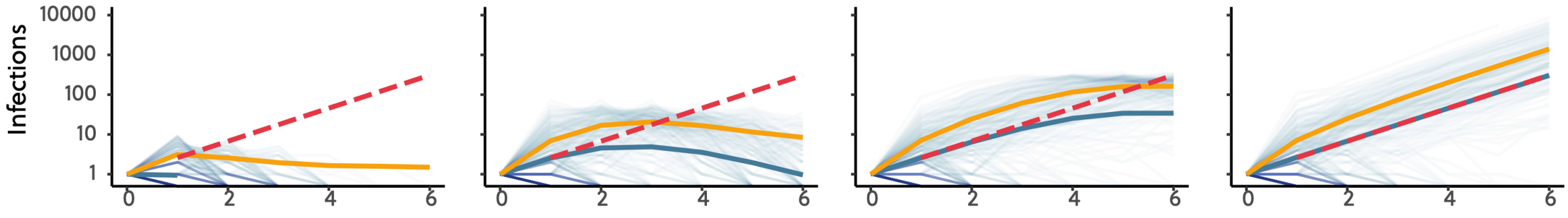


Super-spreading, R0 = 2.6, k = 0.16



Althouse et al. 2020. PLoS Biology

But there's a problem…

The role of directionality, heterogeneity and correlations in epidemic risk and spread

Antoine Allard,[1,2] Cristopher Moore,[3] Samuel V. Scarpino,[4,5]
Benjamin M. Althouse,[6,7,8] and Laurent Hébert-Dufresne[1,9,10]

Allard et al. 2020. *arXiv*

But there's a problem…

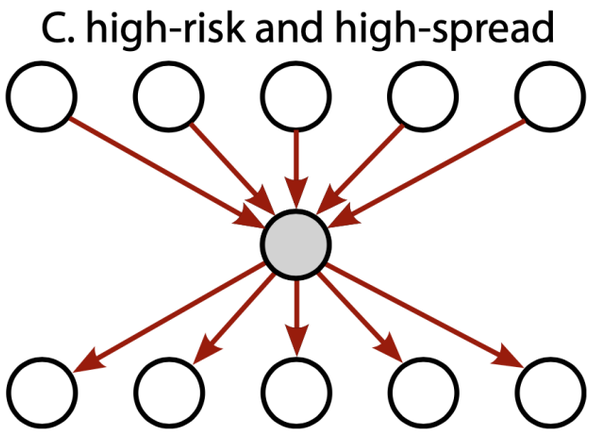
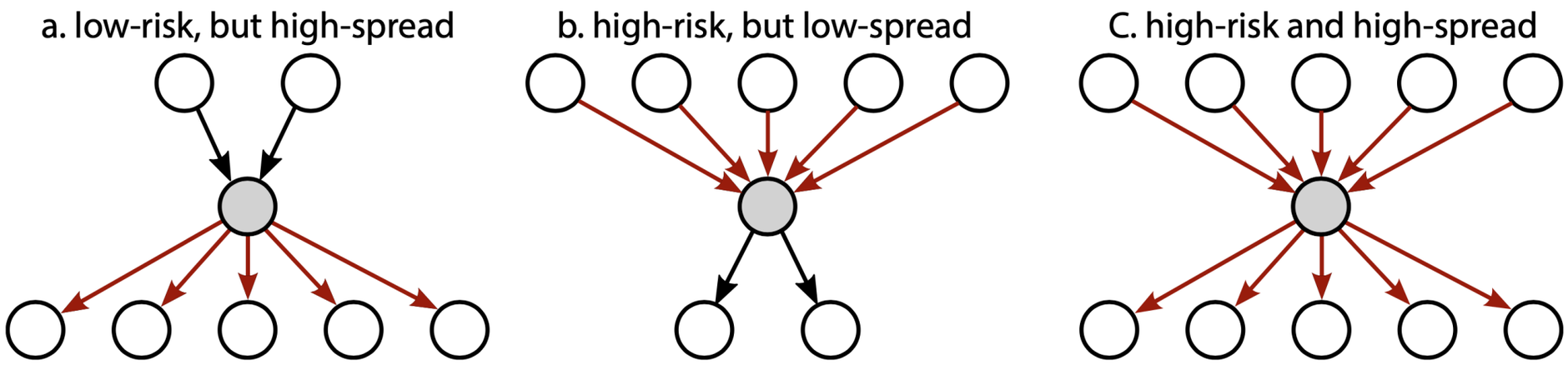


FIG. 1. Different types of individuals in a directed graph model of disease spread: **a.** low-risk, but high-spread, **b.** high-risk, but low-spread, and **c.** high-risk and high-spread.

Allard et al. 2020. *arXiv*

But there's a problem…



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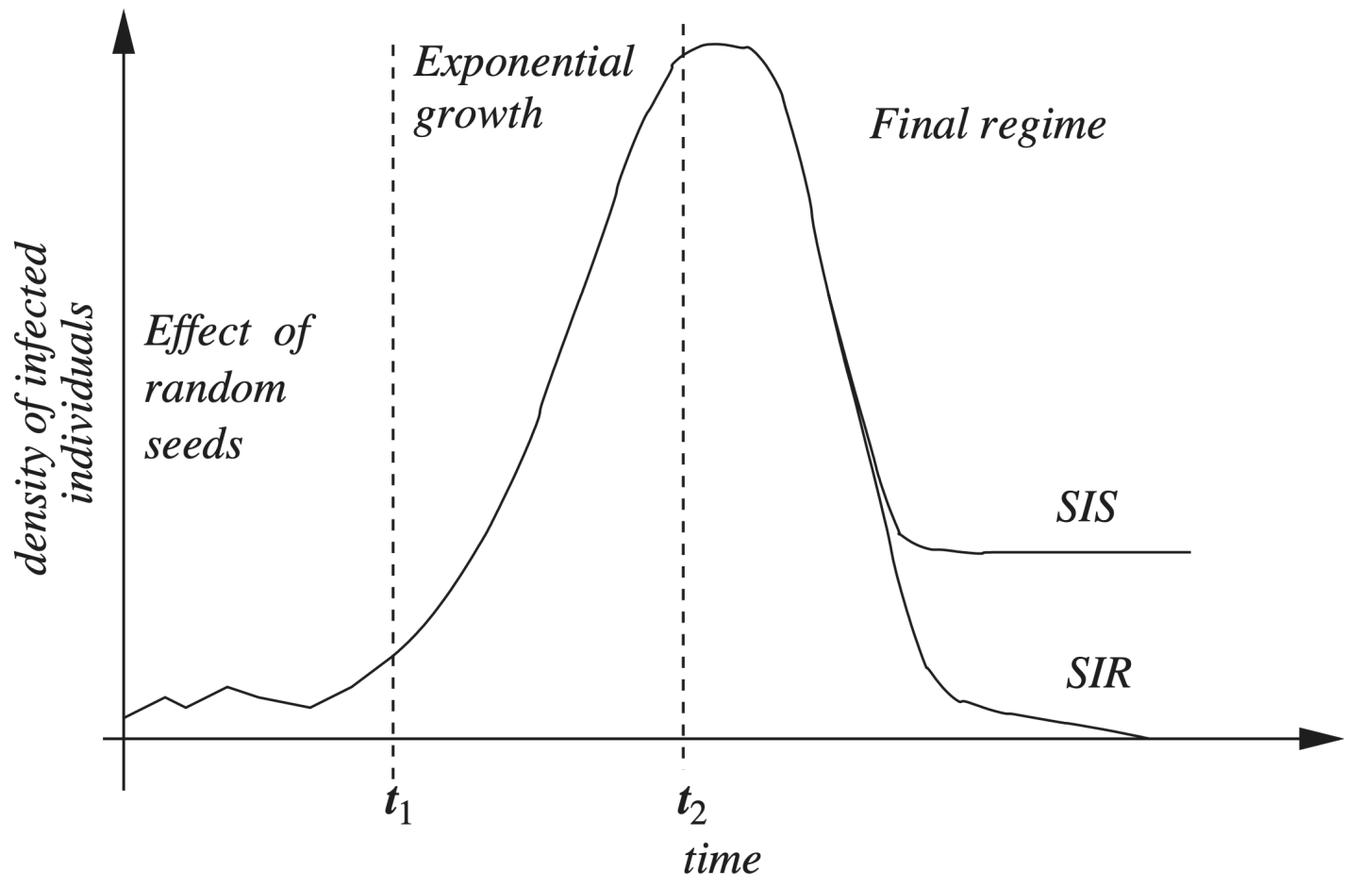
Allard et al. 2020. *arXiv*

But, even with all this, COVID is not so simple

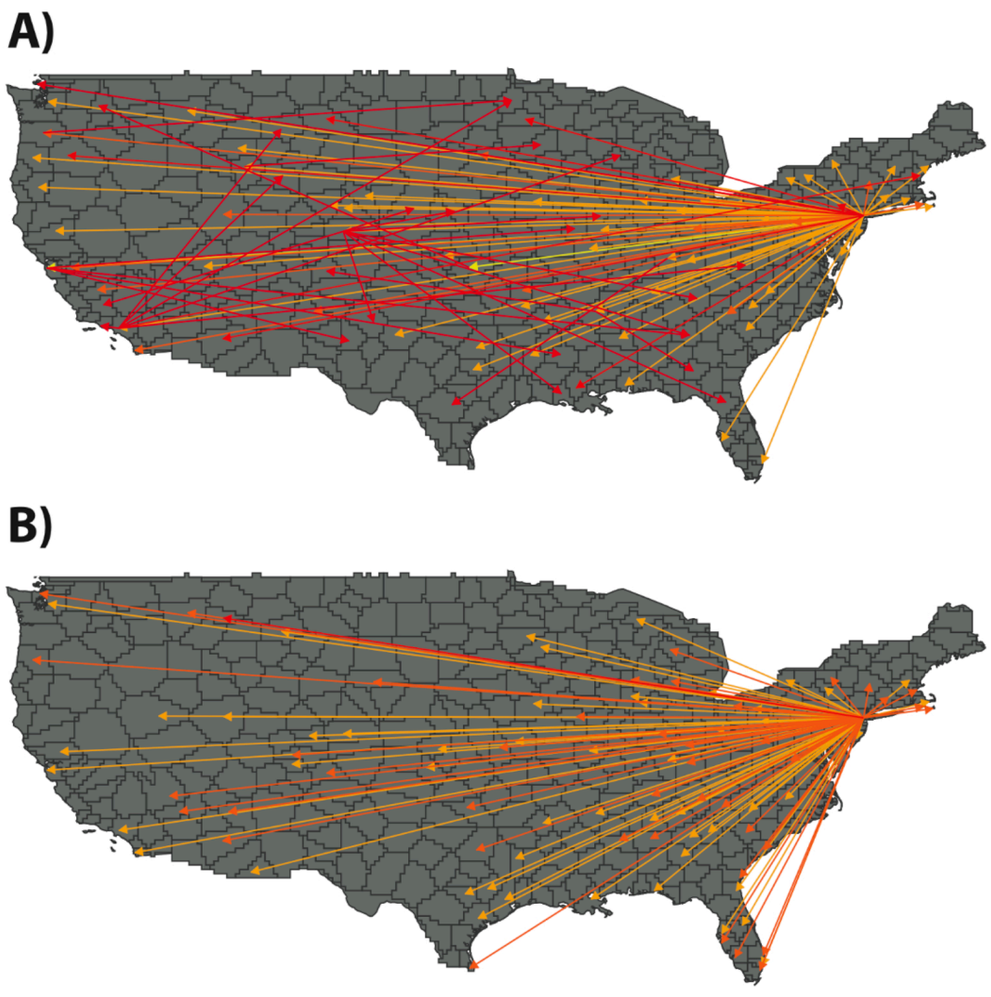
Dynamical patterns of epidemic outbreaks in complex heterogeneous networks

Marc Barthélemy[a,*], Alain Barrat[b], Romualdo Pastor-Satorras[c], Alessandro Vespignani[b,d]

[a]*CEA-Centre d'Etudes de Bruyères-le-Châtel, Département de Physique Théorique et Appliquée BP12, 91680 Bruyères-Le-Châtel, France*
[b]*Laboratoire de Physique Théorique (UMR du CNRS 8627), Bâtiment 210 Université de Paris-Sud, 91405 Orsay, France*
[c]*Departament de Física i Enginyeria Nuclear, Universitat Politècnica de Catalunya Campus Nord, 08034 Barcelona, Spain*
[d]*School of Informatics and Biocomplexity Center, Indiana University, Bloomington, IN 47408, USA*



And human mobility matters for pandemic risk



Modeling human mobility responses to the large-scale spreading of infectious diseases

Sandro Meloni[1,2], Nicola Perra[3,4], Alex Arenas[5,2], Sergio Gómez[5], Yamir Moreno[2,6] & Alessandro Vespignani[3,7]

Mobility mattered early on for COVID-19

Science

RESEARCH ARTICLES

Cite as: M. U. G. Kraemer *et al.*, *Science*
10.1126/science.abb4218 (2020).

The effect of human mobility and control measures on the COVID-19 epidemic in China

Moritz U. G. Kraemer[1,2,3]*, Chia-Hung Yang[4], Bernardo Gutierrez[1,5], Chieh-Hsi Wu[6], Brennan Klein[4], David M. Pigott[7], Open COVID-19 Data Working Group†, Louis du Plessis[1], Nuno R. Faria[1], Ruoran Li[8], William P. Hanage[8], John S. Brownstein[2,3], Maylis Layan[9,10], Alessandro Vespignani[4,11], Huaiyu Tian[12], Christopher Dye[1], Oliver G. Pybus[1,13]*, Samuel V. Scarpino[4]*

Science

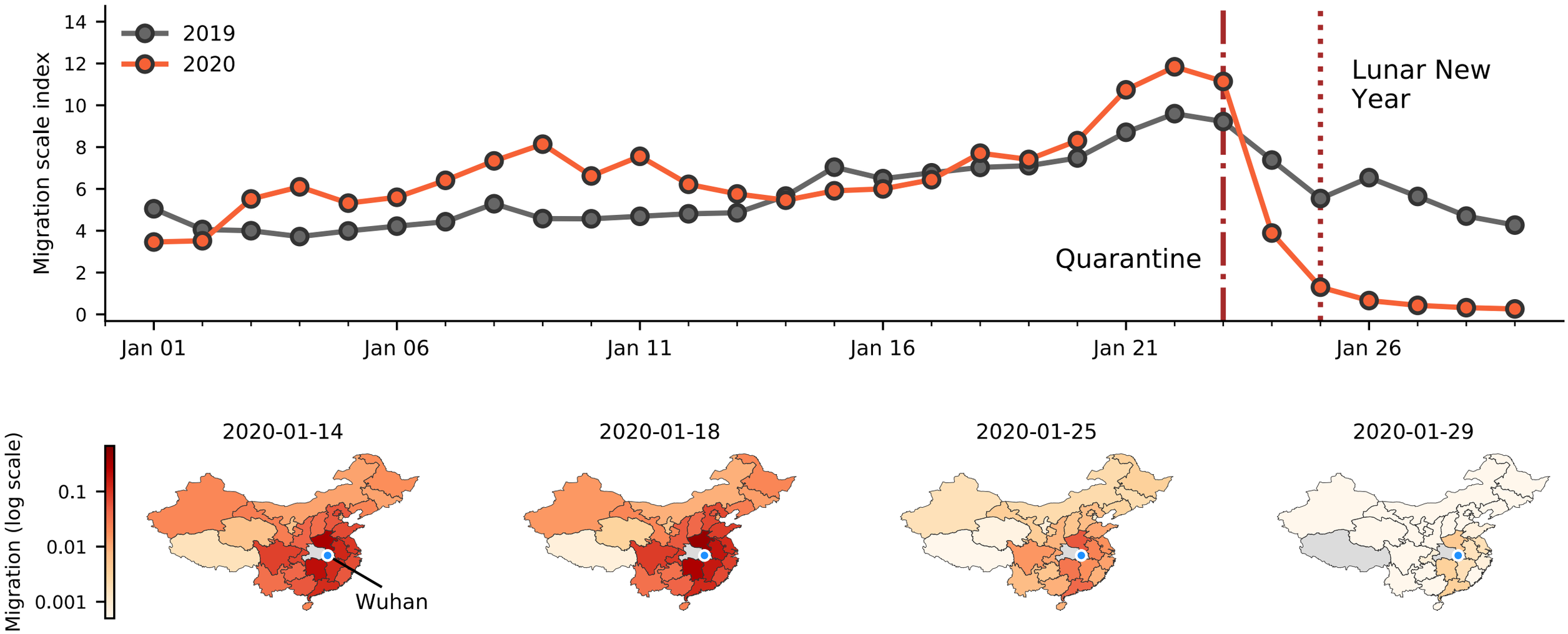
RESEARCH ARTICLES

Cite as: M. Chinazzi *et al.*, *Science*
10.1126/science.aba9757 (2020).

The effect of travel restrictions on the spread of the 2019 novel coronavirus (COVID-19) outbreak

Matteo Chinazzi[1], Jessica T. Davis[1], Marco Ajelli[2], Corrado Gioannini[3], Maria Litvinova[3], Stefano Merler[2], Ana Pastore y Piontti[1], Kunpeng Mu[1], Luca Rossi[3], Kaiyuan Sun[4], Cécile Viboud[4], Xinyue Xiong[1], Hongjie Yu[5], M. Elizabeth Halloran[6,7], Ira M. Longini Jr.[8]*, Alessandro Vespignani[1,3]*

And, restrictions were important

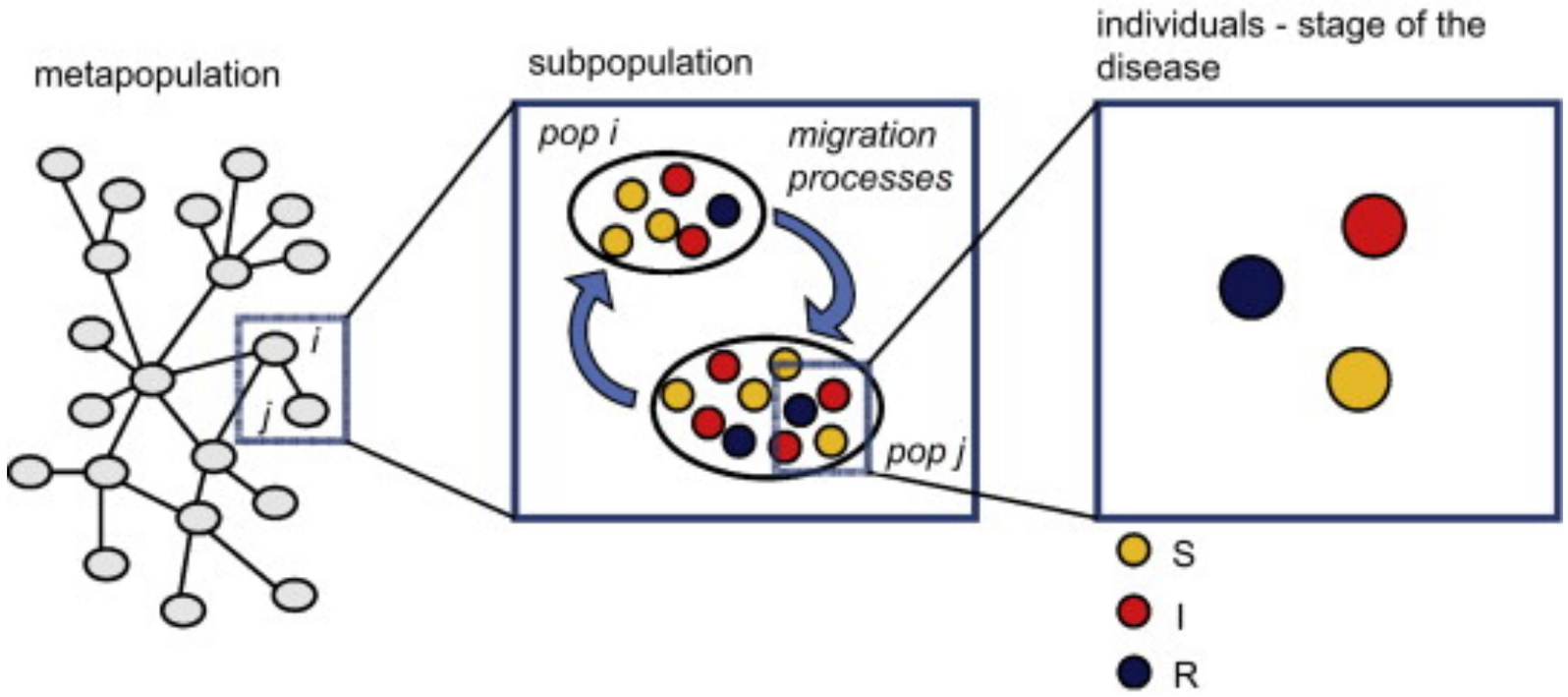


Kraemer et al. 2020 *Science*

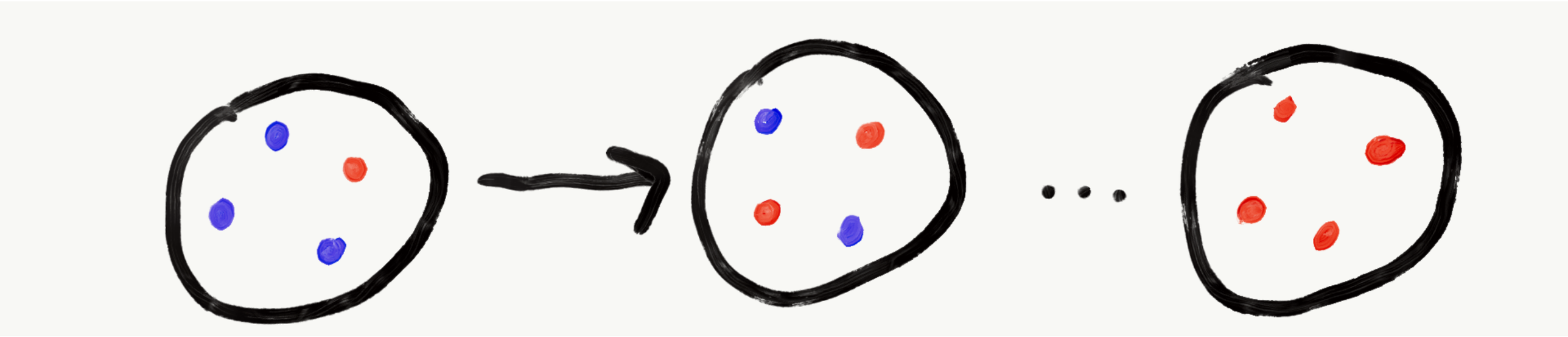
But, our society is a collection of meta-populations!

Epidemic modeling in metapopulation systems with heterogeneous coupling pattern: Theory and simulations

Vittoria Colizza[a,*], Alessandro Vespignani[b,c]



What are the consequences?



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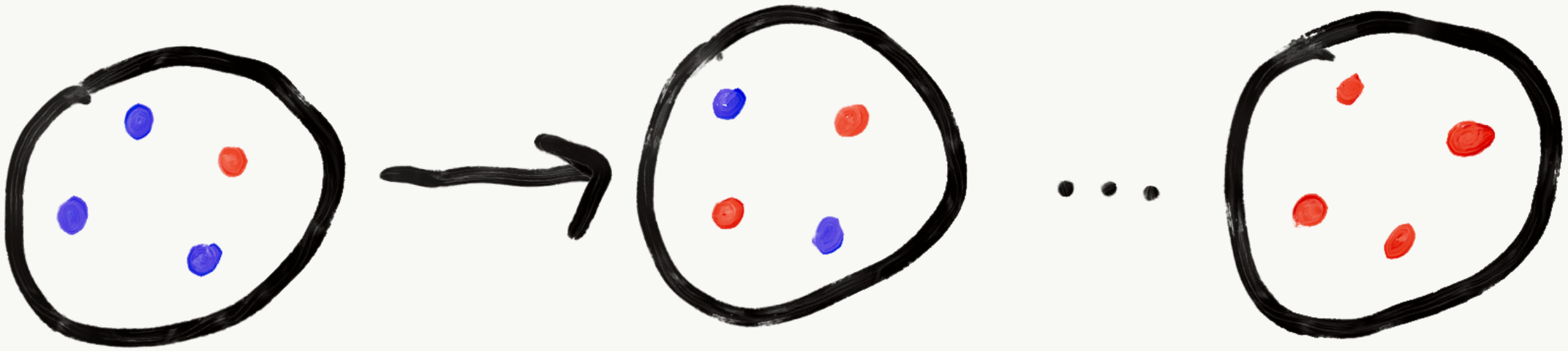
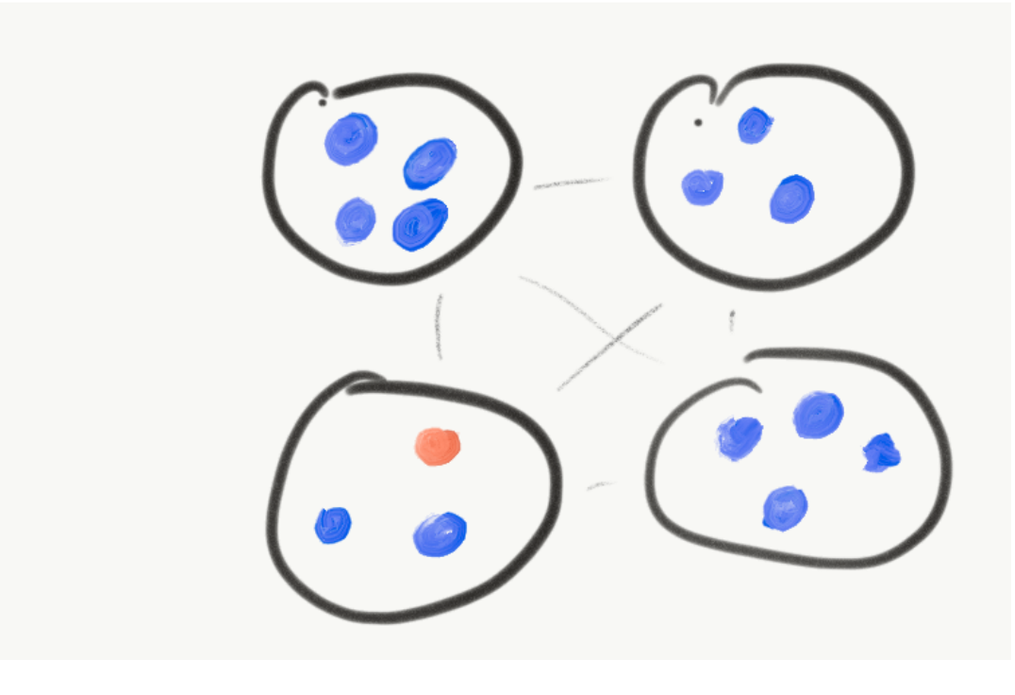


But, that’s not what happened…



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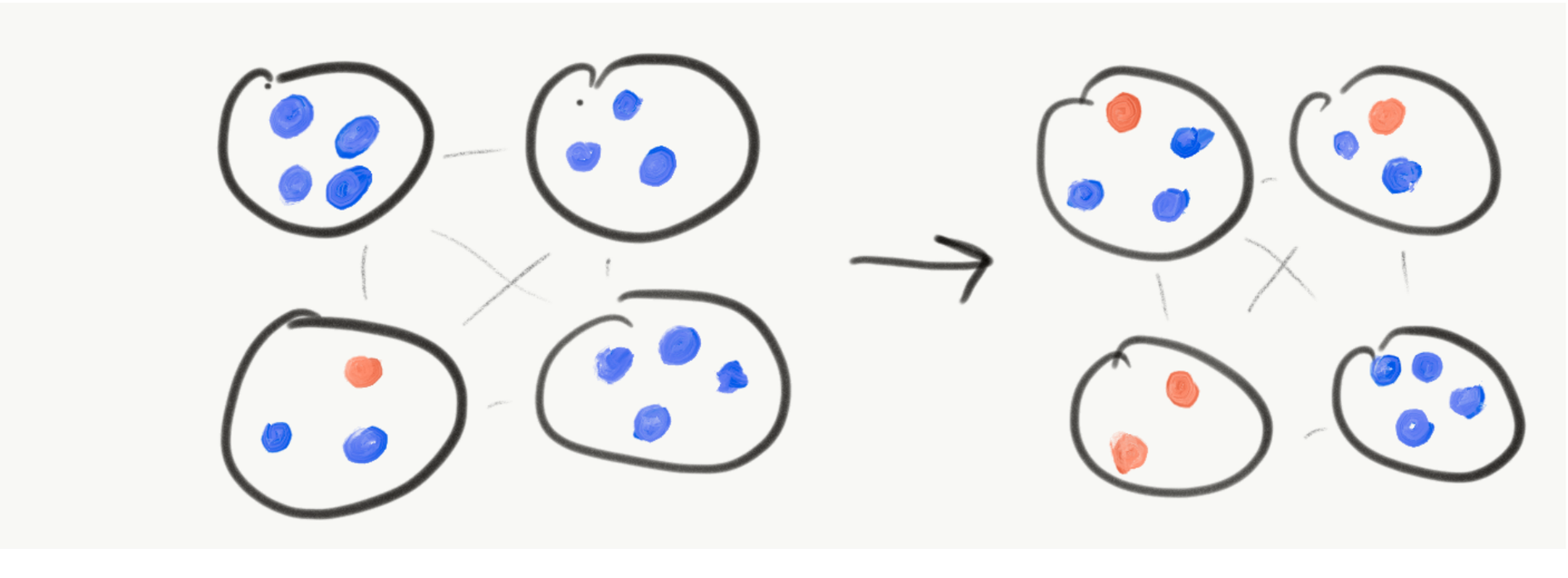

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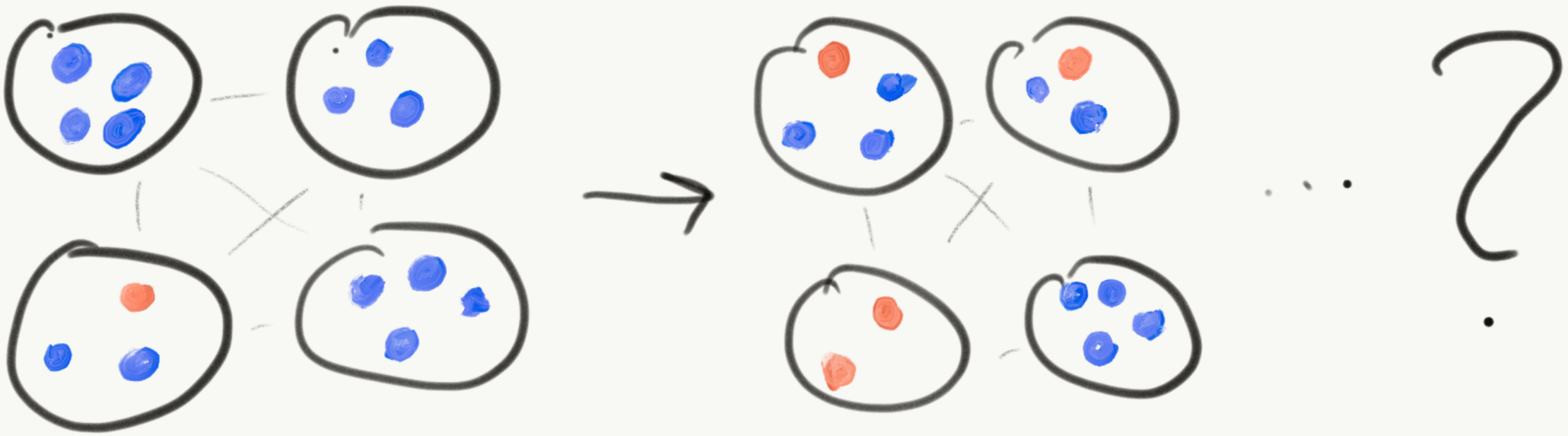

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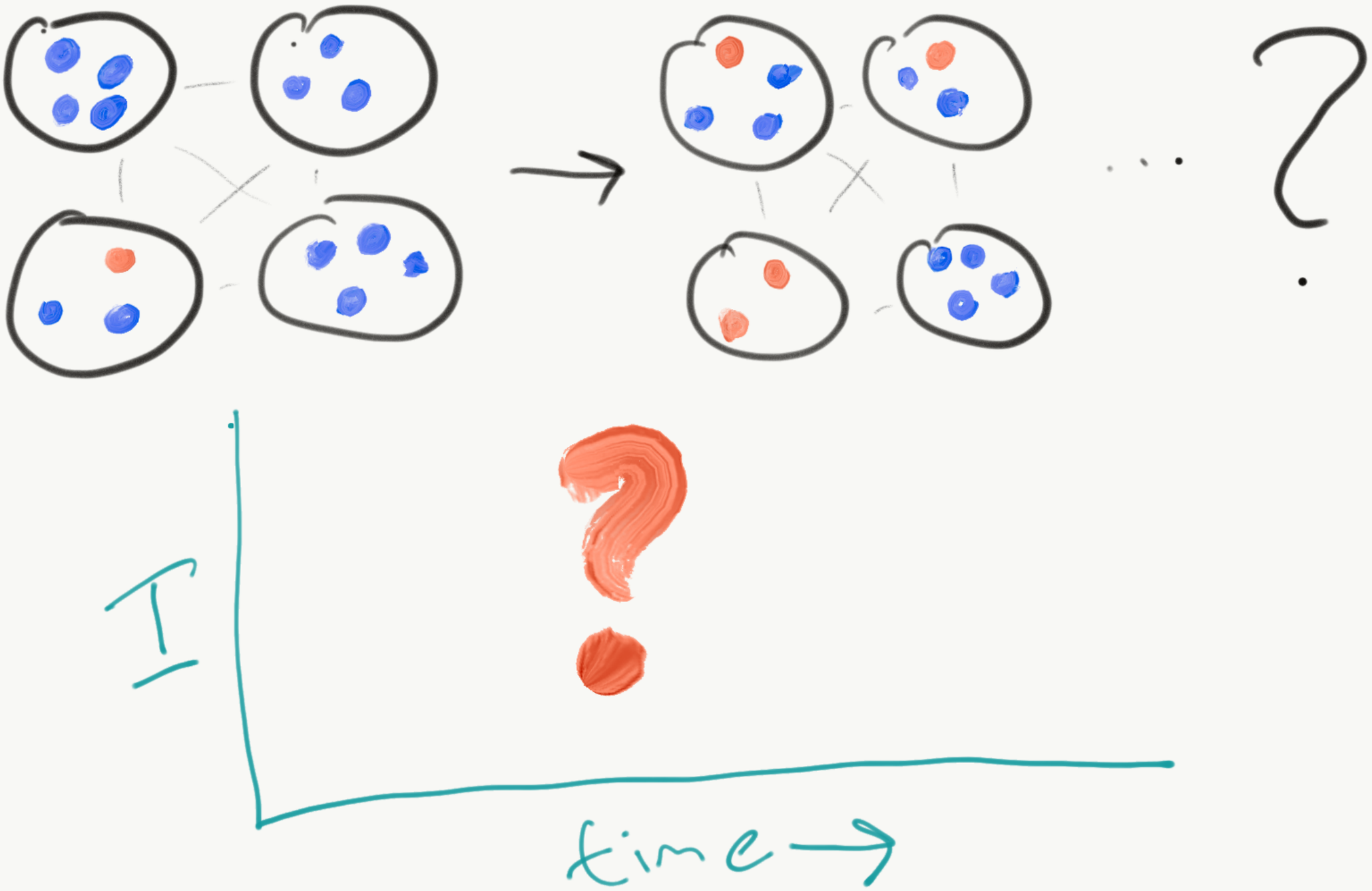
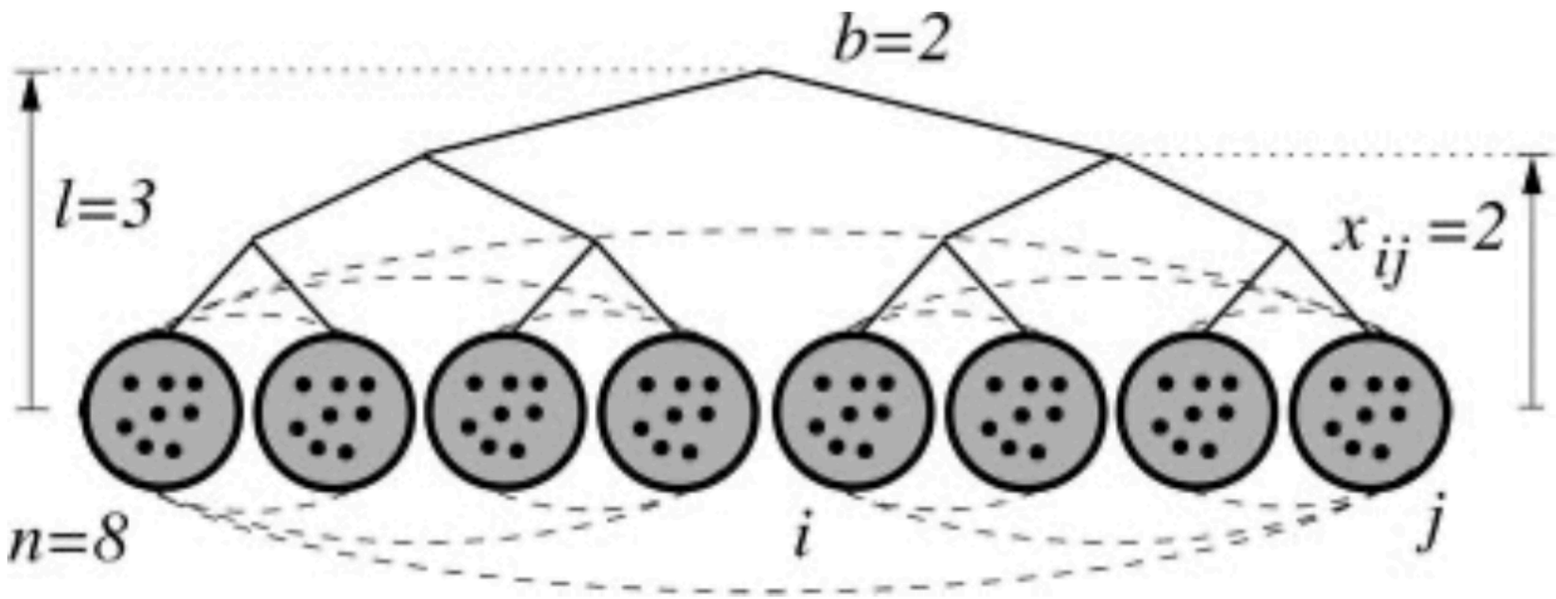
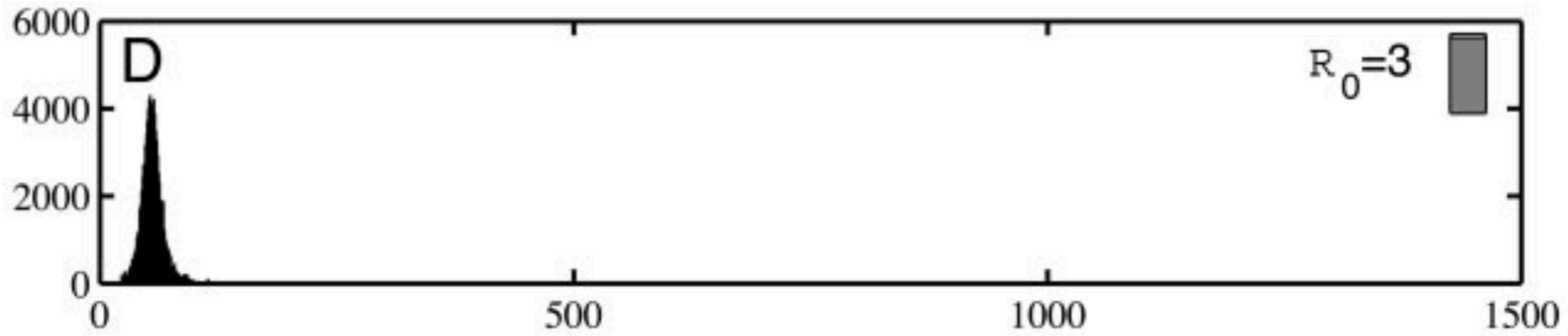


Multiscale, resurgent epidemics in a hierarchical metapopulation model

Duncan J. Watts*†‡§, Roby Muhamad*, Daniel C. Medina¶, and Peter S. Dodds†



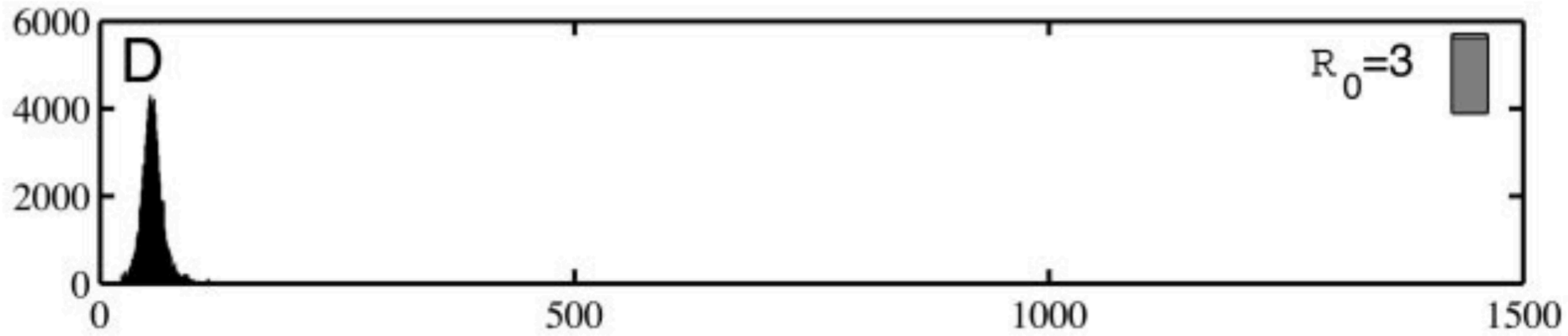
What are the consequences?

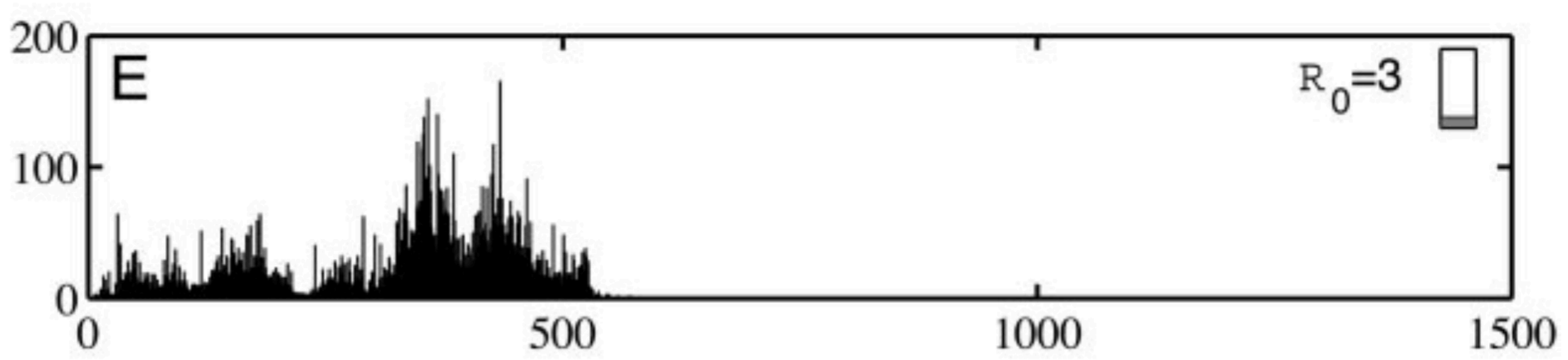


Watts et al. 2005

What are the consequences?

Watts et al. 2005





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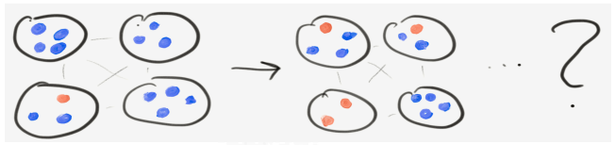
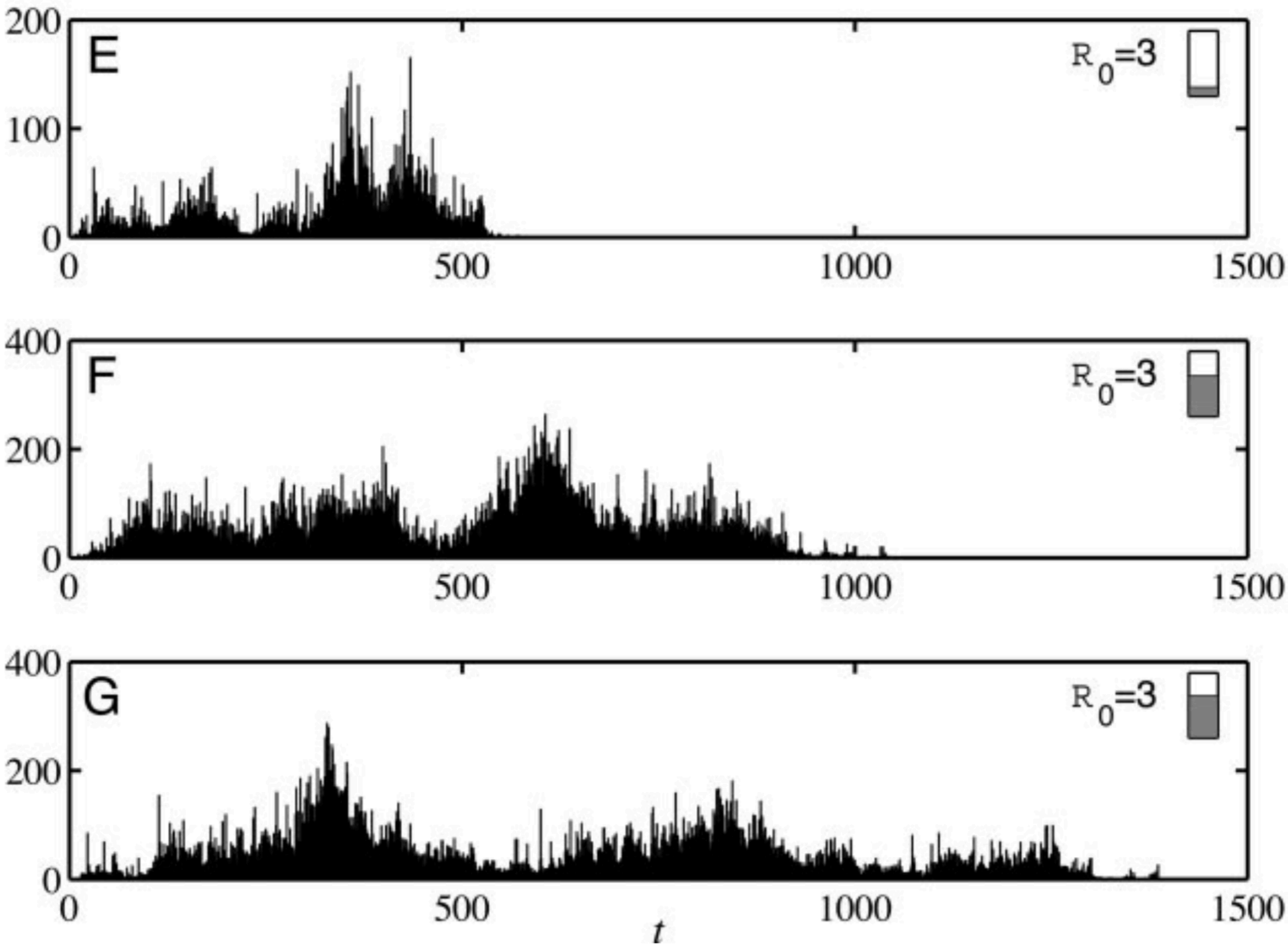


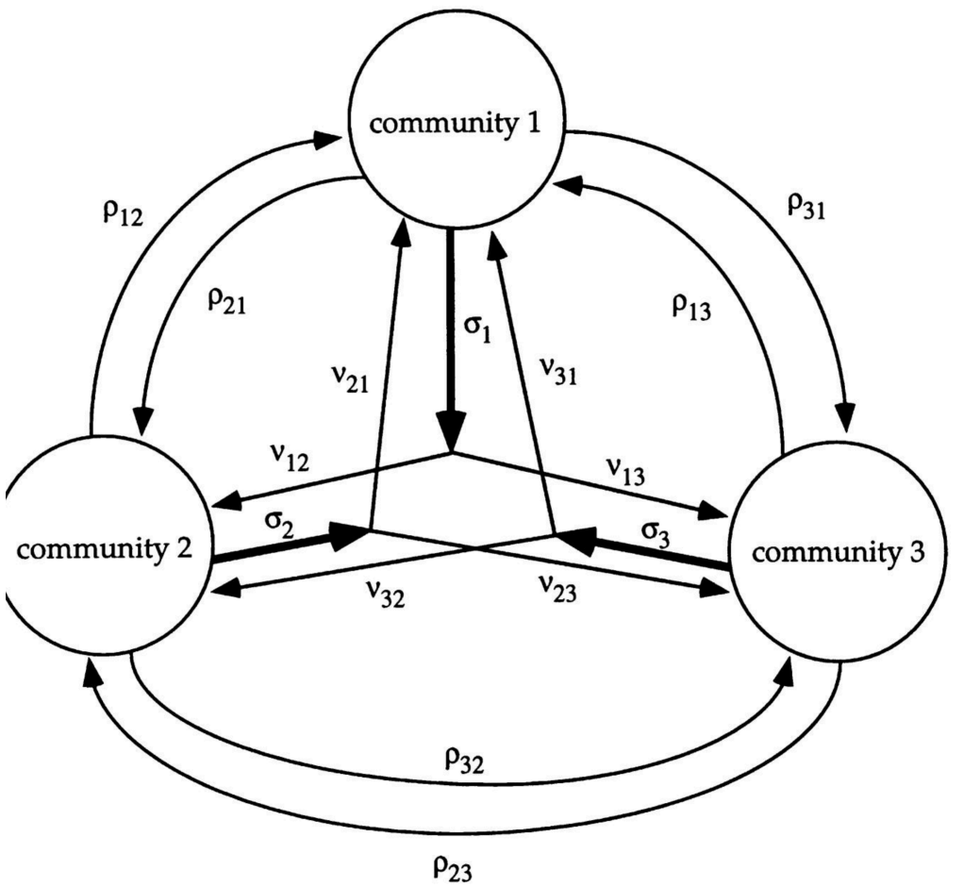
Watts et al. 2005

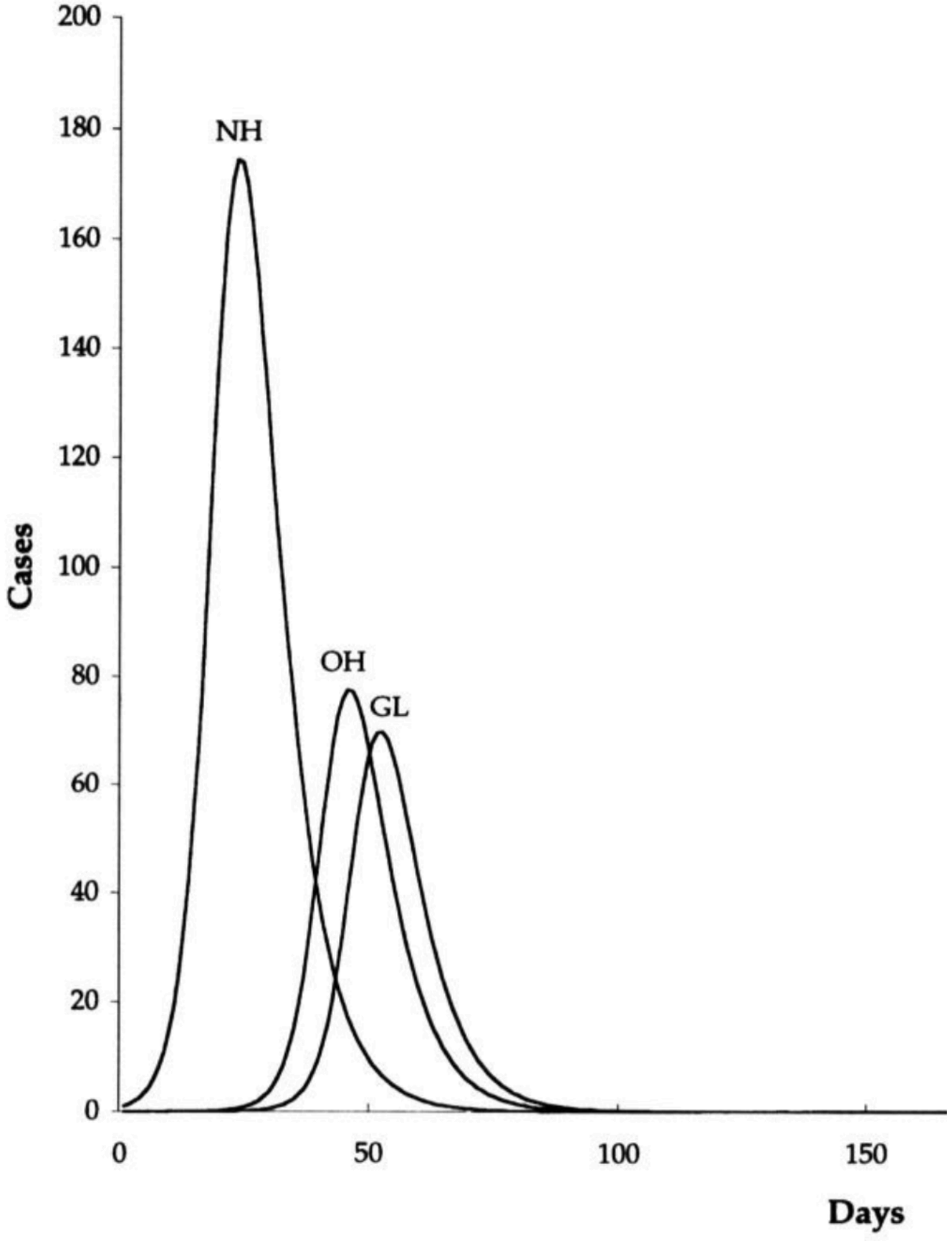


Structured Epidemic Models and the Spread of Influenza in the Central Canadian Subarctic

LISA SATTENSPIEL[1] AND D. ANN HERRING[2]

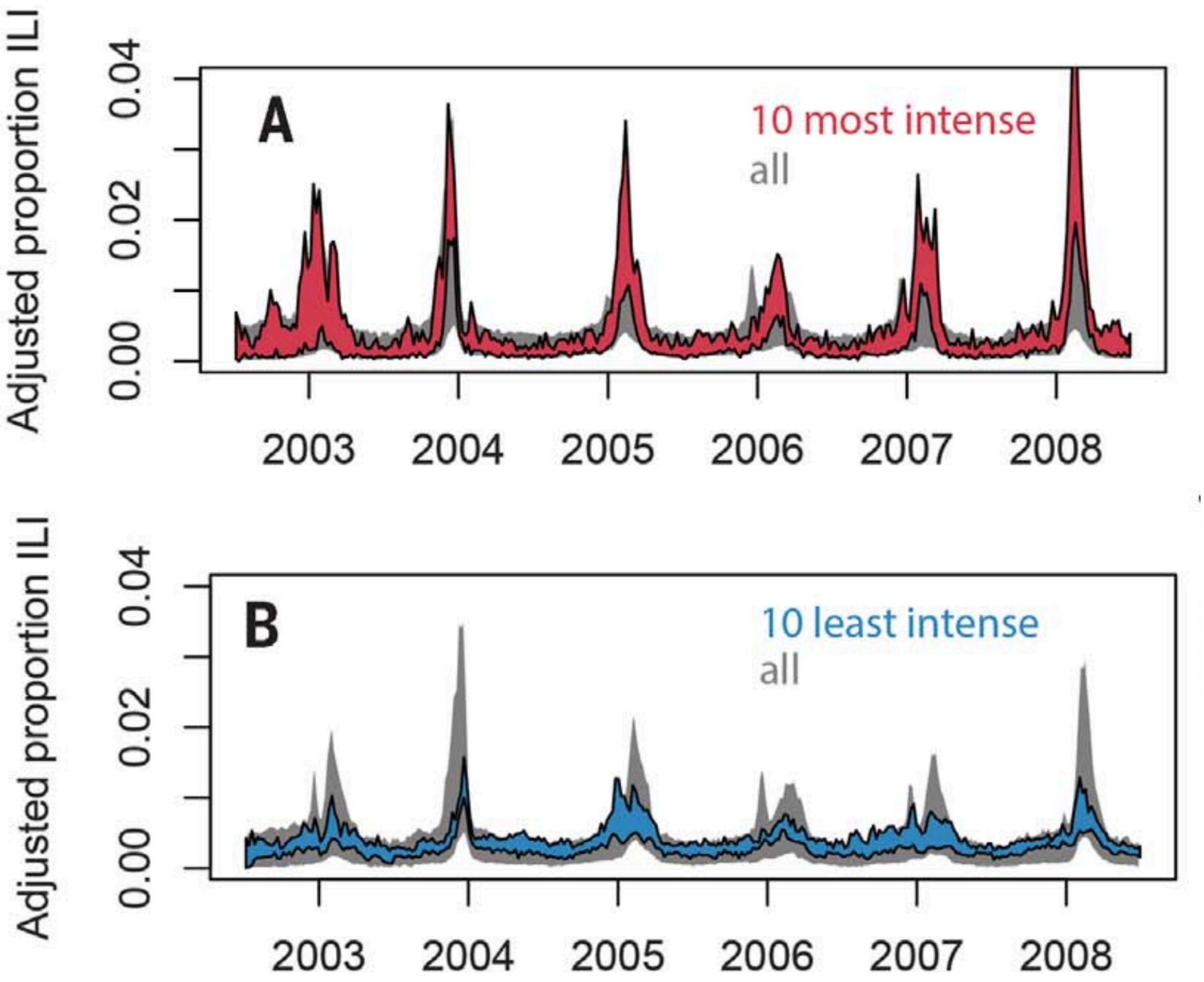
Figure 1. Hudson's Bay Company district divisions during the 1918 influenza pandemic. Redrawn from *Map of the Dominion of Canada Shewing (sic) the Establishments of the Hudson's Bay Company* (Hudson's Bay Company Archives 1922).





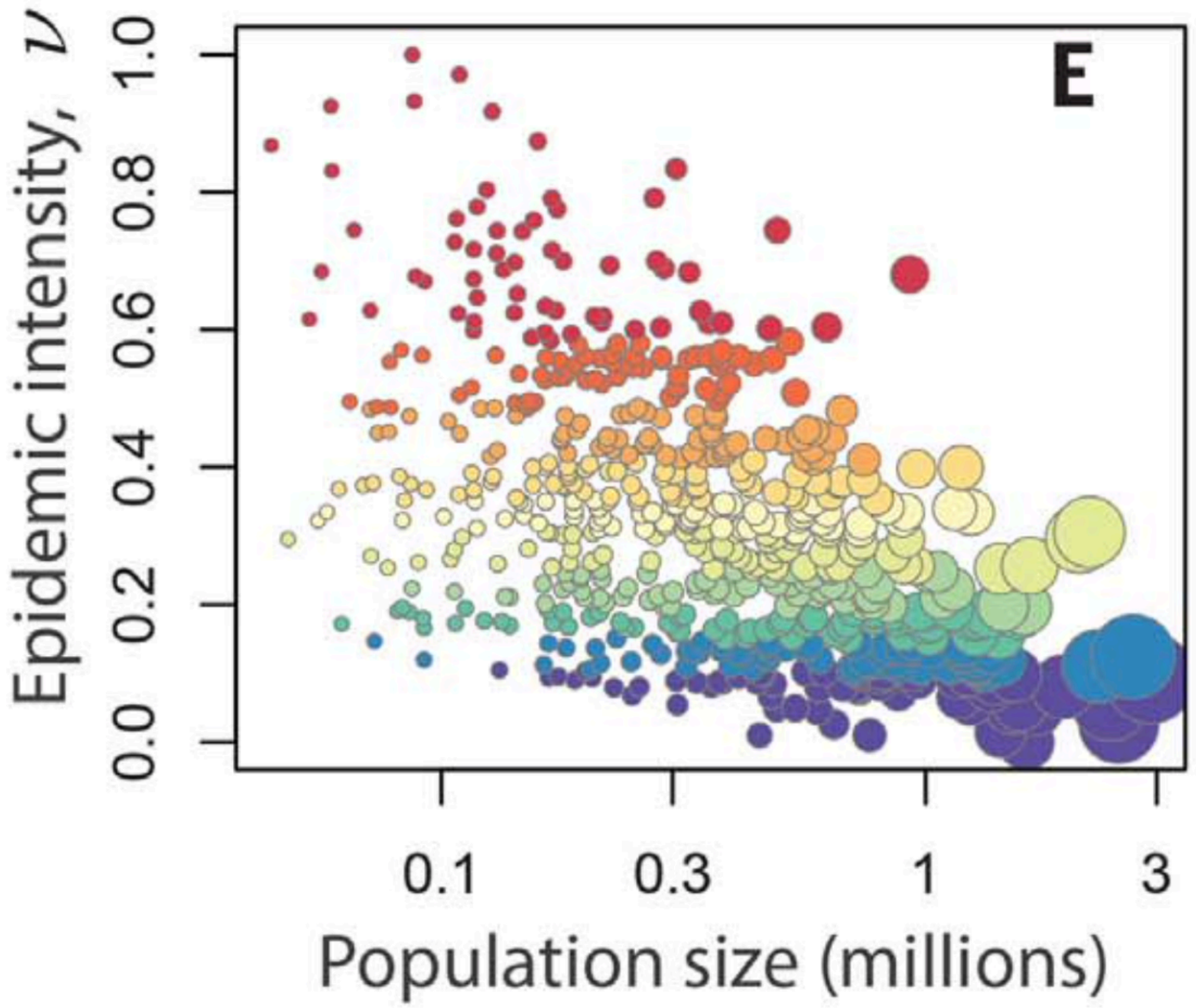
Sattenspiel & Ann Herring 1998

Epidemic “intensity”



Dalziel et al. 2018

Epidemic “intensity” and “urbanisation”



Dalziel et al. 2018

nature medicine

LETTERS

https://doi.org/10.1038/s41591-020-1104-0

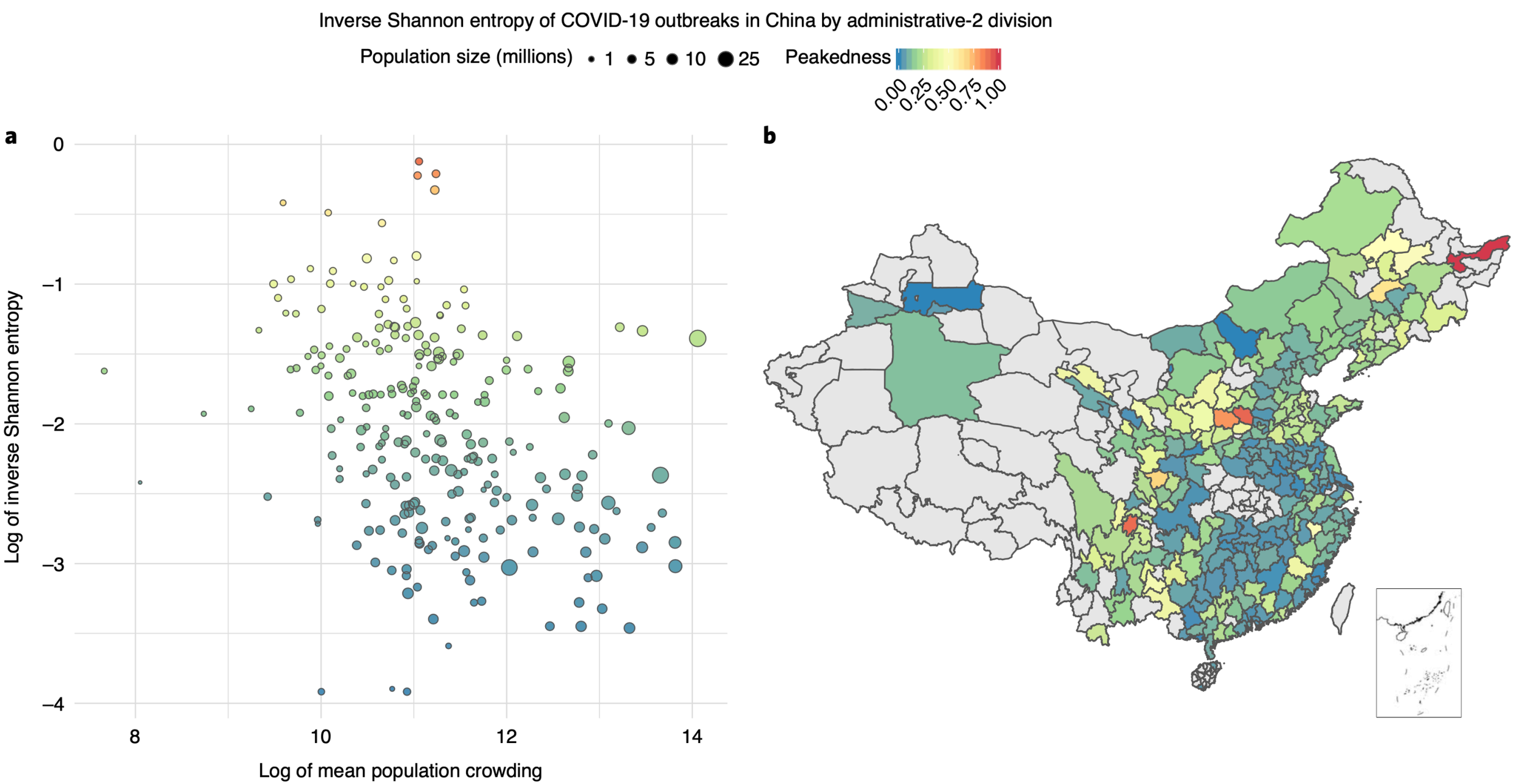
Check for updates

Crowding and the shape of COVID-19 epidemics

Benjamin Rader[1,2,16], Samuel V. Scarpino[3,4,5,16] ✉, Anjalika Nande[6], Alison L. Hill[6,7], Ben Adlam[6], Robert C. Reiner[8,9], David M. Pigott[8,9], Bernardo Gutierrez[10,11], Alexander E. Zarebski[10], Munik Shrestha[3], John S. Brownstein[1,12], Marcia C. Castro[13], Christopher Dye[10], Huaiyu Tian[14], Oliver G. Pybus[10,15] ✉ and Moritz U. G. Kraemer[10] ✉

Rader & Scarpino et al. 2020. Nature Medicine

COVID19: Epidemic “intensity” and “urbanisation”



Rader & Scarpino et al. 2020. Nature Medicine

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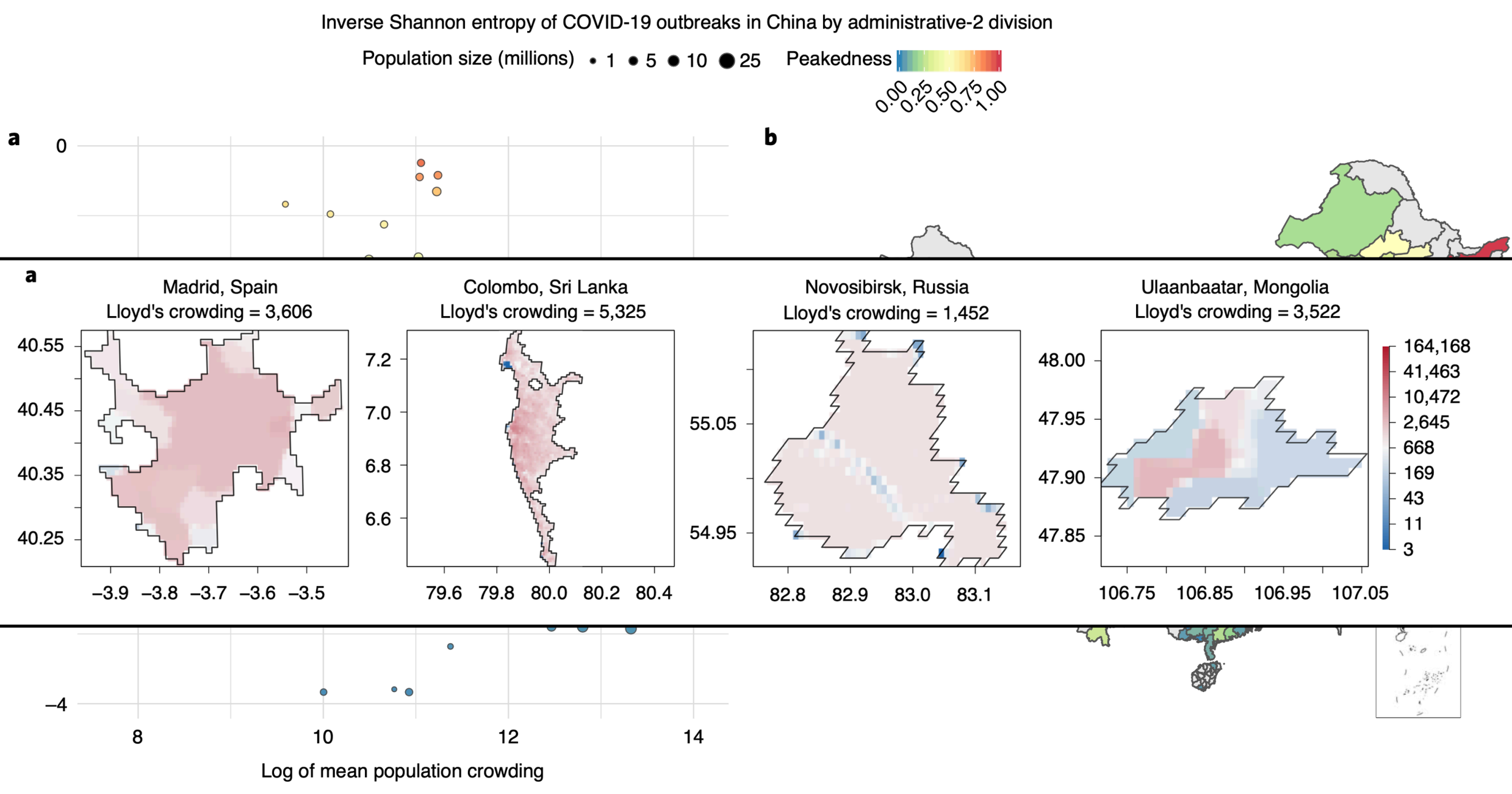

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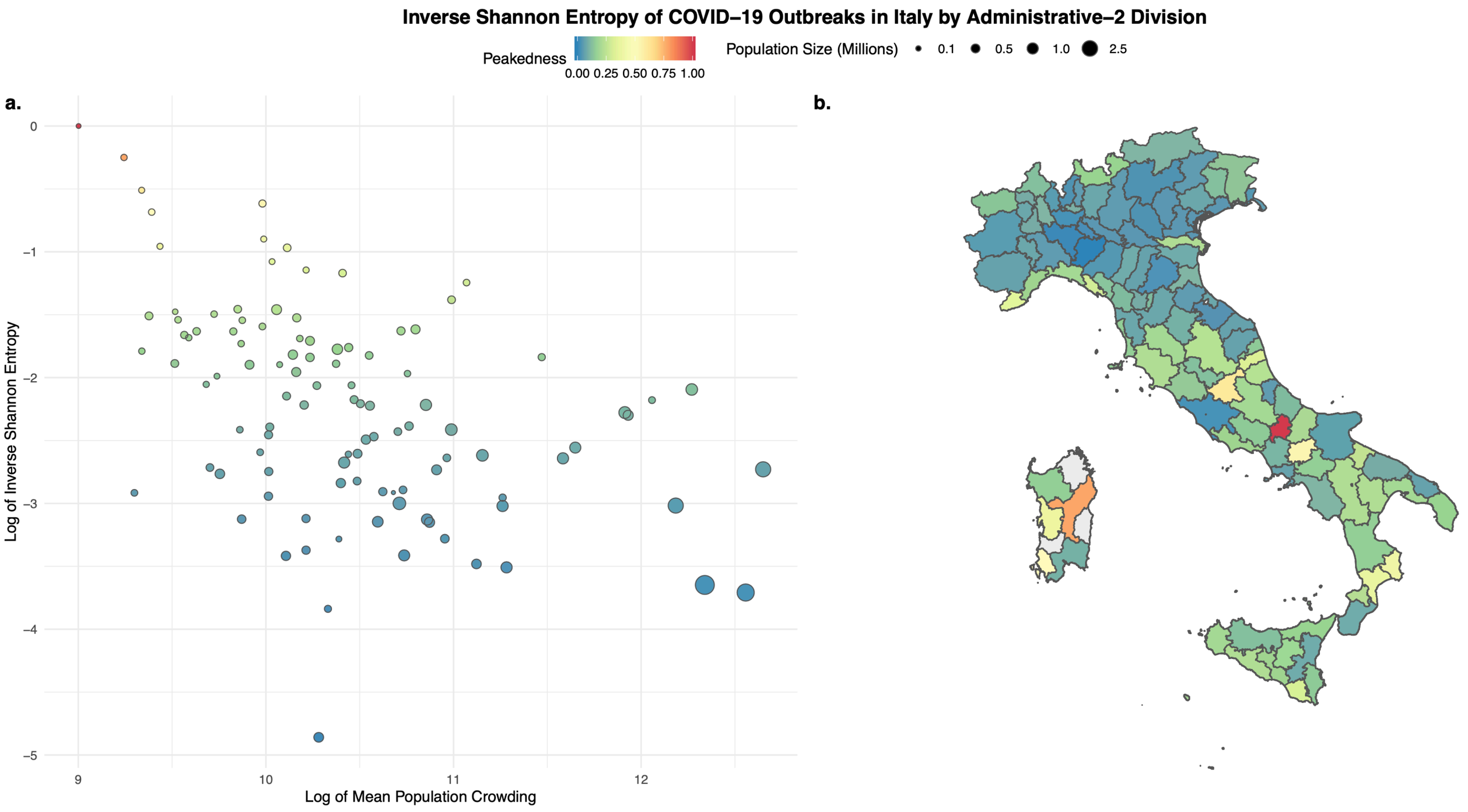
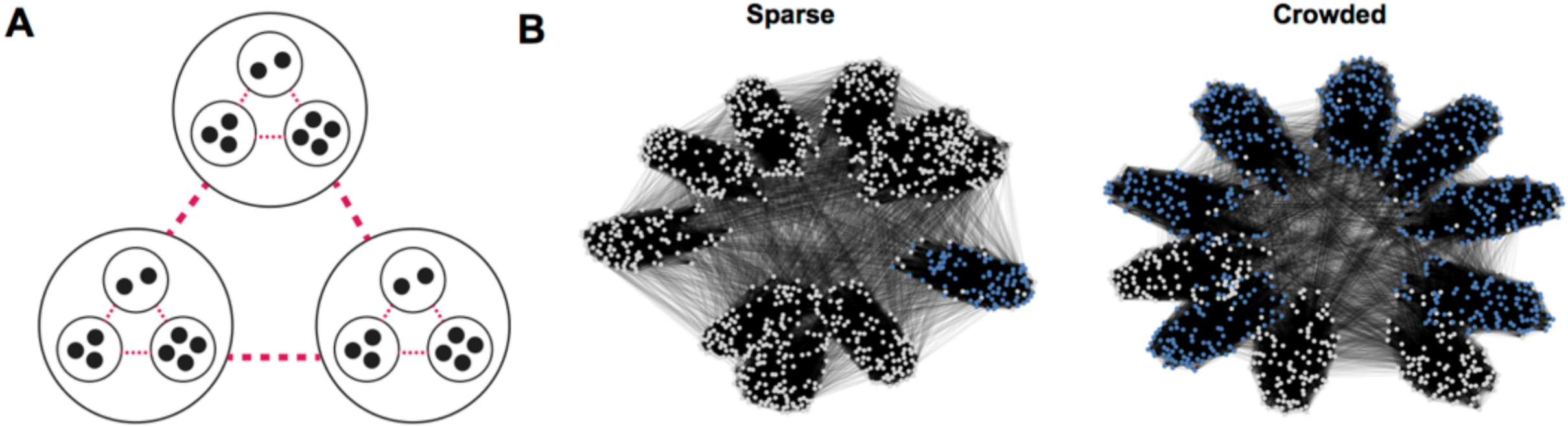


Rader & Scarpino et al. 2020. Nature Medicine

Simulating COVID19 through a meta-population



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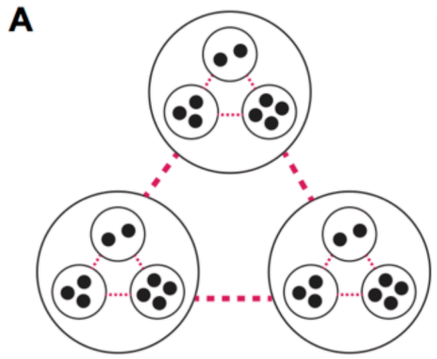
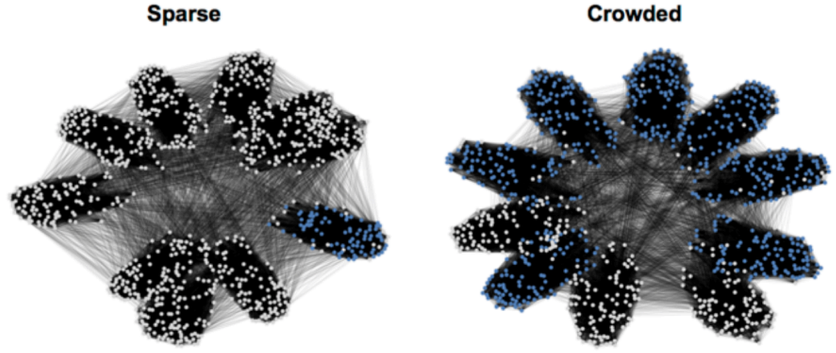
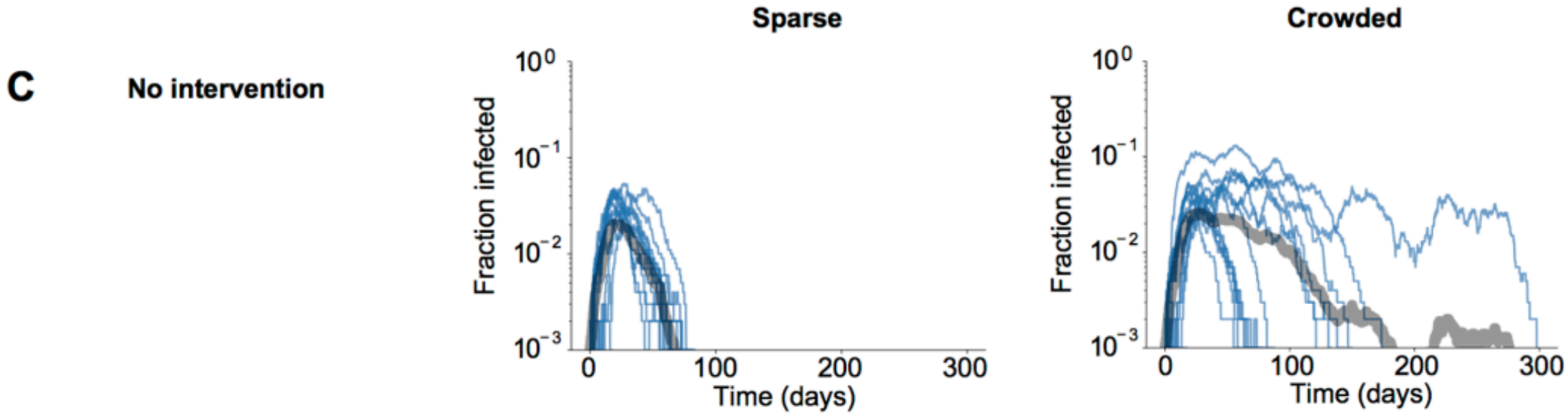
Simulating COVID19 through a meta-population







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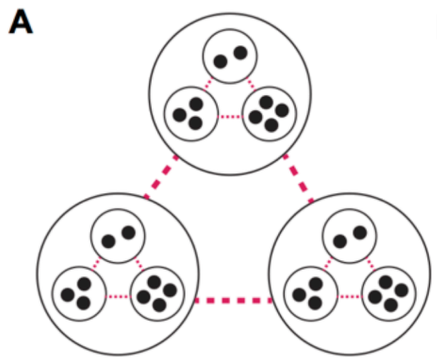
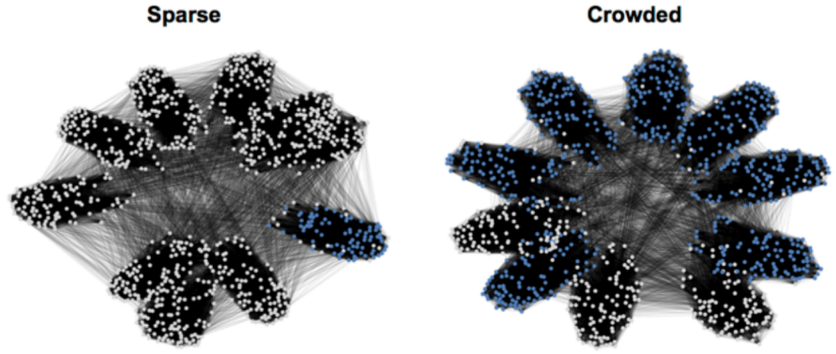
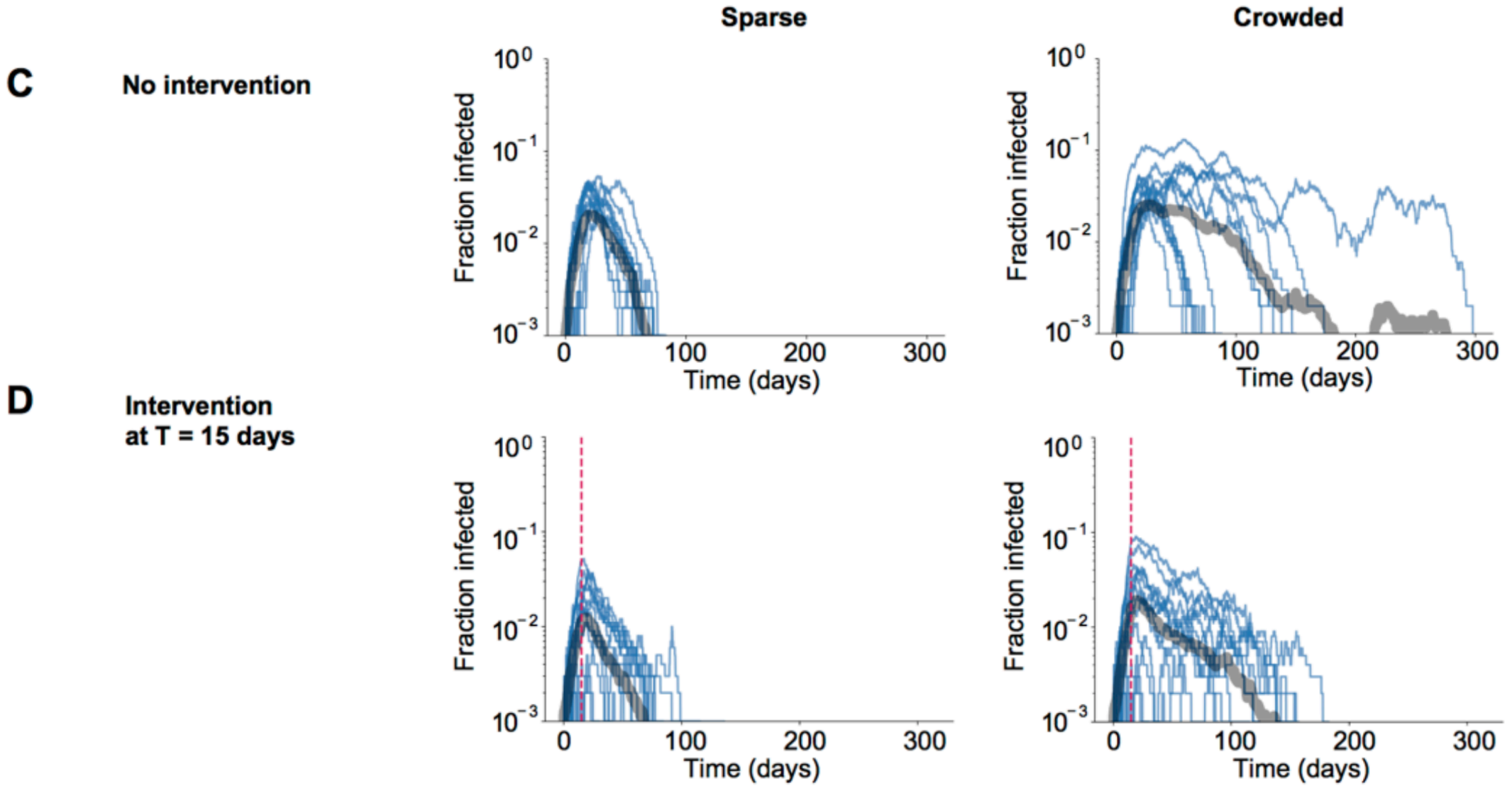
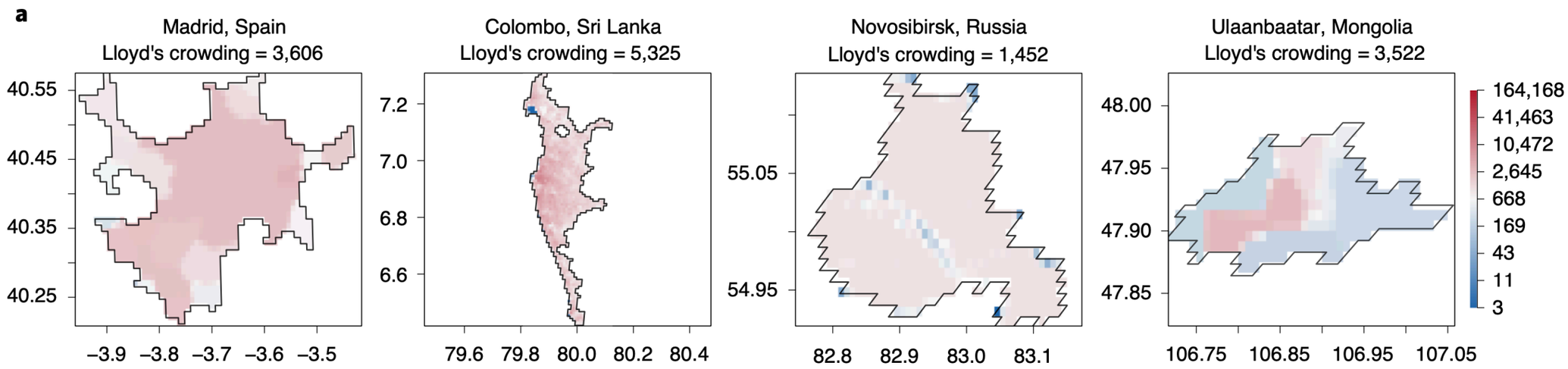
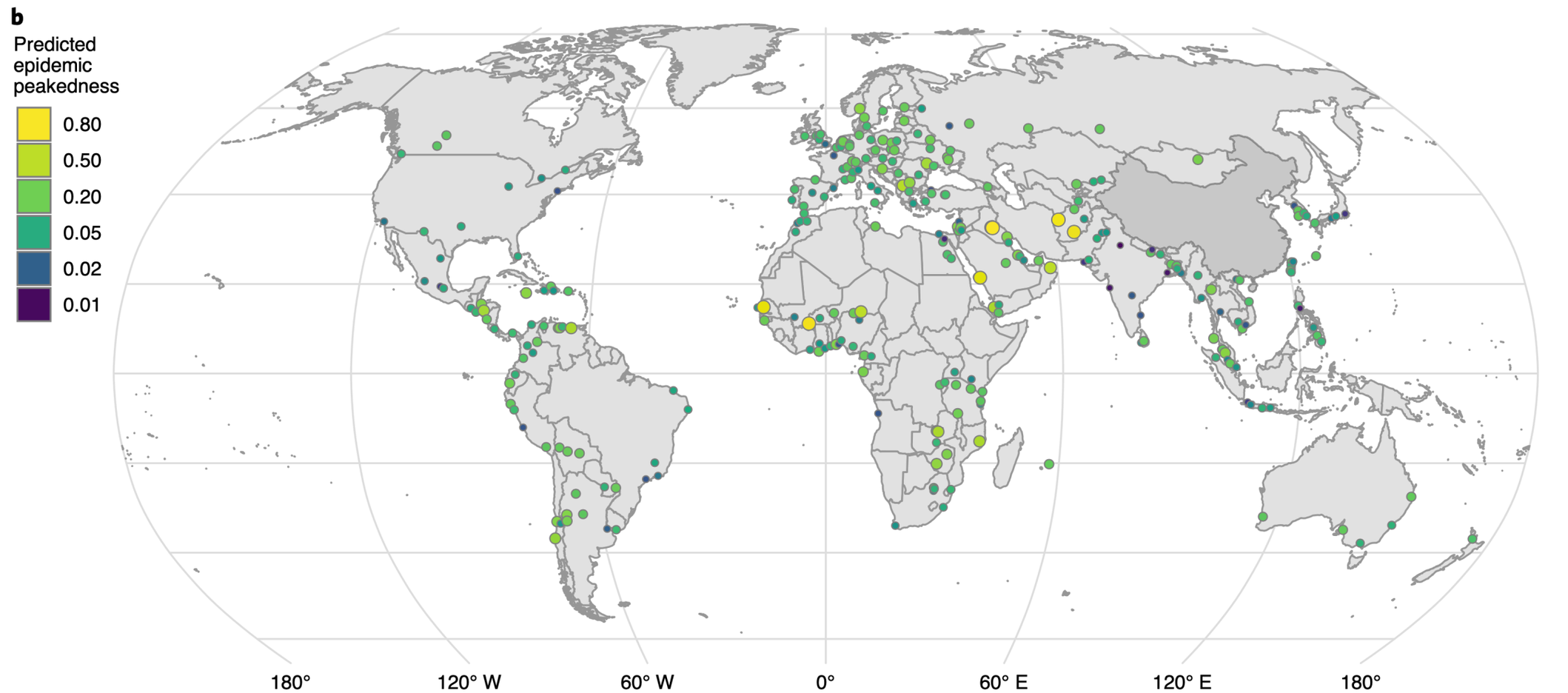






More “intense” outbreaks in more rural areas





Rader & Scarpino et al. 2020. Nature Medicine

But, what is “crowding?”

https://www.nps.gov/grca/planyourvisit/images/crowding_03pair.jpg

Lloyd's mean crowding

By common sense, the degree of crowding that exists at any one moment must be a function, not only of the number of animals present in a given area, but also of their spatial pattern of distribution, their movements, their actions towards each other when they meet, and the physiological repercussions which these meetings bring about.

-Lloyd 1967

And it’s simple to calculate.

50-year anniversary of Lloyd’s “mean crowding”: Ideas on patchy distributions

Michael J. Wade | **Courtney L. Fitzpatrick** | **Curtis M. Lively**

$$m^* = \frac{\sum_i n_i(n_i - 1)}{\sum_i n_i}$$

And should look scarily familiar

BULLETIN (New Series) OF THE
AMERICAN MATHEMATICAL SOCIETY
Volume 44, Number 1, January 2007, Pages 63–86
S 0273-0979(06)01148-7
Article electronically published on October 17, 2006

CONTACT NETWORK EPIDEMIOLOGY: BOND PERCOLATION APPLIED TO INFECTIOUS DISEASE PREDICTION AND CONTROL

LAUREN ANCEL MEYERS

naïve population. In the contact network framework, this is simply the average number of occupied edges emanating from a vertex, that is,

$$R_0 = G_1'(1;T) = TG_1'(1) = T\left(\frac{\langle k^2\rangle - \langle k\rangle}{\langle k\rangle}\right). \tag{22}$$

$$m^* = \frac{\sum_i n_i(n_i - 1)}{\sum_i n_i}$$

Transmission of infectious diseases is...

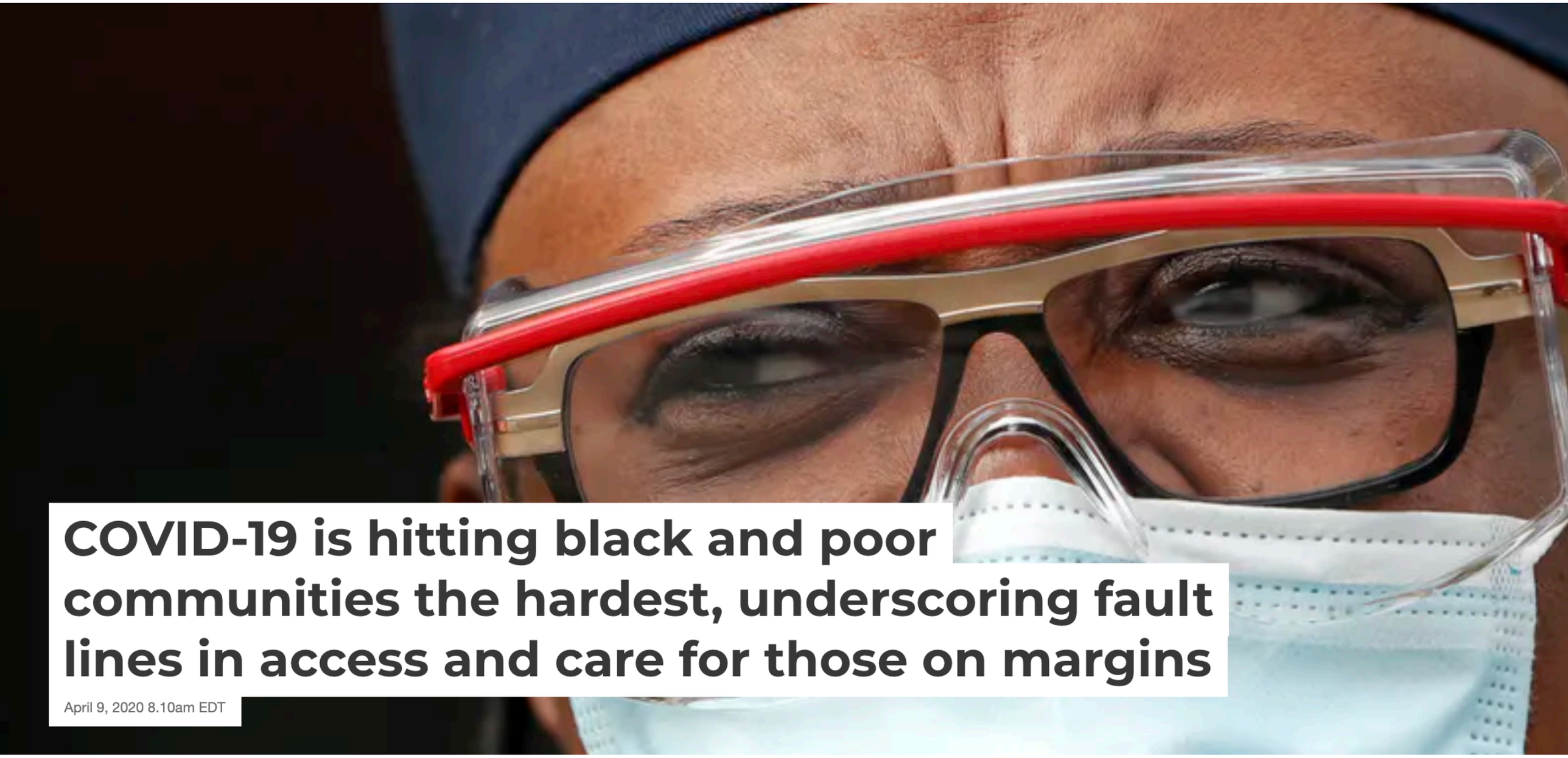
- Heterogeneous (few people infect many, many people infect few)
  - This is especially the case for COVID-19

- Dependent on spatial structure of the underlying population
  - This is especially the case for COVID-19

- Unequal in terms of who is most impacted (age, race, income, etc.)
  - This is especially the case for COVID-19

- Best understood as a *network* phenomenon
  - This is especially the case for COVID-19

Brennan Klein, PhD



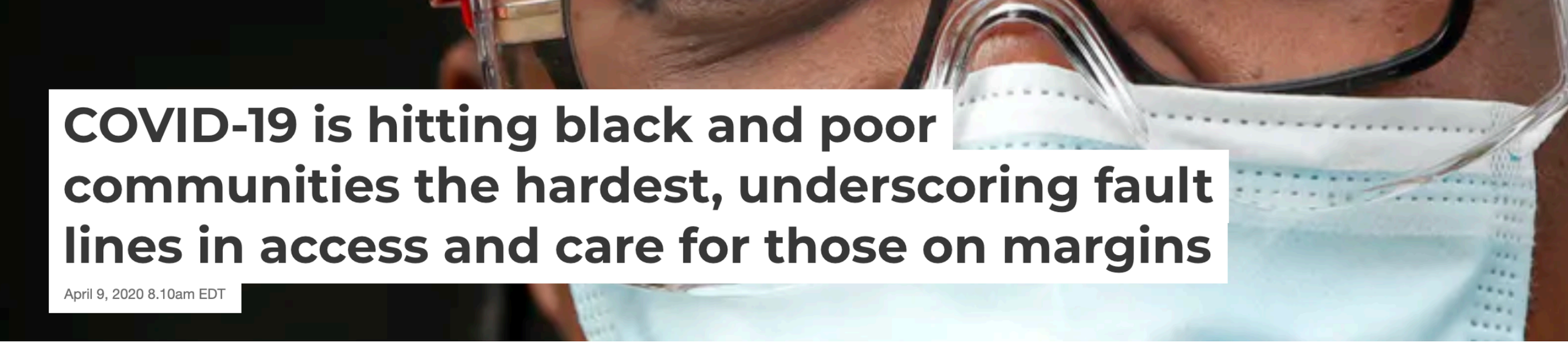
COVID-19 is hitting black and poor communities the hardest, underscoring fault lines in access and care for those on margins

April 9, 2020 8.10am EDT

THE CONVERSATION
Academic rigor, journalistic flair

Author

Grace A. Noppert
Postdoctoral Scholar in Epidemiology, Carolina Population Center, University of North Carolina at Chapel Hill



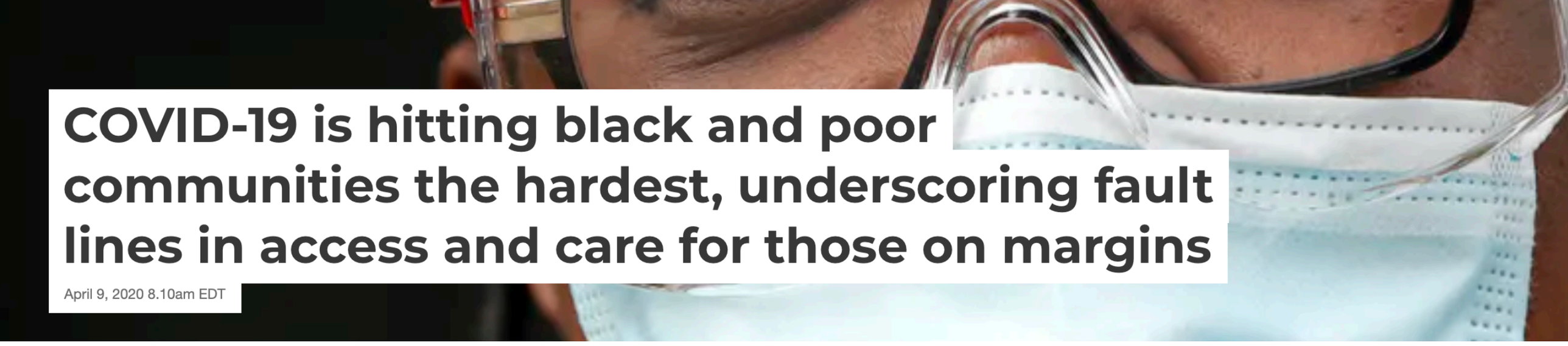
COVID-19 is hitting black and poor communities the hardest, underscoring fault lines in access and care for those on margins

April 9, 2020 8.10am EDT

As the COVID-19 epidemic continues to ravage the American public, an unsurprising story emerges: Poor communities are hot spots for COVID transmission. The death rate from COVID-19 appears to be staggeringly high among African Americans compared to whites. The Washington Post reports, for example, that while 14% of the Michigan population is black, 40% of COVID-19 deaths are among blacks.

Author

Grace A. Noppert
Postdoctoral Scholar in Epidemiology, Carolina Population Center, University of North Carolina at Chapel Hill



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In the context of the current pandemic, blacks are more likely to have low-paying jobs that do not allow remote work options or offer health insurance or paid medical leave. The result of centuries of sidelining by American society plays out most obviously in worse health.

Author

Grace A. Noppert
Postdoctoral Scholar in Epidemiology, Carolina Population Center, University of North Carolina at Chapel Hill

COVID-19 became a pandemic because the world doesn’t understand complex systems (networks)

COVID-19 became a pandemic because the world doesn't understand complex systems (networks)

add to that fundamental Theory in Ecology & Evolution

So what can you do?

So what can you do?

1. **Read and collaborate broadly.** E.g., know the history of our fields, learn what's know in other fields, and stay up-to-date on both theory and empirical work.

Prof. C. Brandon Ogbunu

So what can you do?

1. **Read and collaborate broadly.**

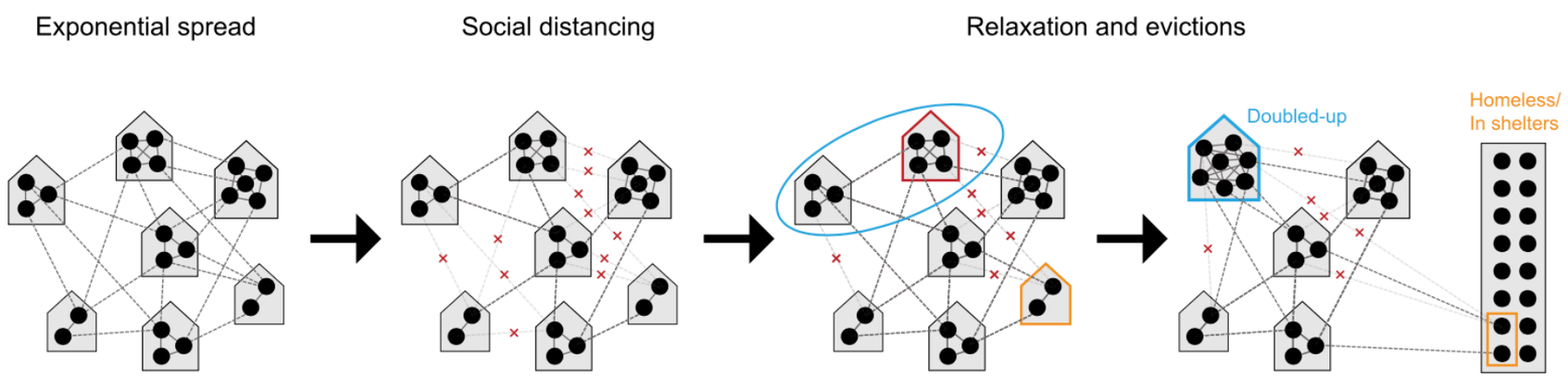
2. **Integrate epidemiological models, policies, behavior, etc.**

ARTICLE

https://doi.org/10.1038/s41467-021-22521-5 OPEN

The effect of eviction moratoria on the transmission of SARS-CoV-2

Anjalika Nande[1,7], Justin Sheen[2,7], Emma L. Walters[3], Brennan Klein[4,5], Matteo Chinazzi[4,5], Andrei H. Gheorghe[1], Ben Adlam[1], Julianna Shinnick[2], Maria Florencia Tejeda[2], Samuel V. Scarpino[4], Alessandro Vespignani[4,5], Andrew J. Greenlee[3], Daniel Schneider[3], Michael Z. Levy[2,8]✉ & Alison L. Hill[1,6,8]✉



So what can you do?

1. **Read and collaborate broadly.**

2. **Put differently, *don’t* stay in your lane. Instead, don’t assume that just because you’re good at one thing, you’ll be good at everything. Nor that you don’t have as much or more to learn from as contribute to a new field.**

So what can you do?

1. **Read and collaborate broadly.**

2. **Integrate epidemiological models, policies, behavior, etc.**

3. **Communicate your work effectively.**

COVID-19 Event Risk Assessment Planning Tool

USA RISK ESTIMATES BY COUNTY | GLOBAL RISK ESTIMATES | REAL-TIME US AND STATE-LEVEL ESTIMATES | USA CONTINUOUS RISK ESTIMATES | TUTORIAL | ABOUT

This map shows the risk level of attending an event, given the event size and location.

You can reduce the risk that one case becomes many by wearing a mask, distancing, and gathering outdoors in smaller groups

The risk level is the estimated chance (0-100%) that at least 1 COVID-19 positive individual will be present at an event in a county, given the size of the event.

Based on seroprevalence data and increases in testing, by default we assume there are three times more cases than are being reported (3:1 ascertainment bias). In places with less testing availability, that bias may be higher. We are evaluating the inclusion of lower ascertainment biases based on increased testing.

Choose an event size and ascertainment bias below.

See our data sources

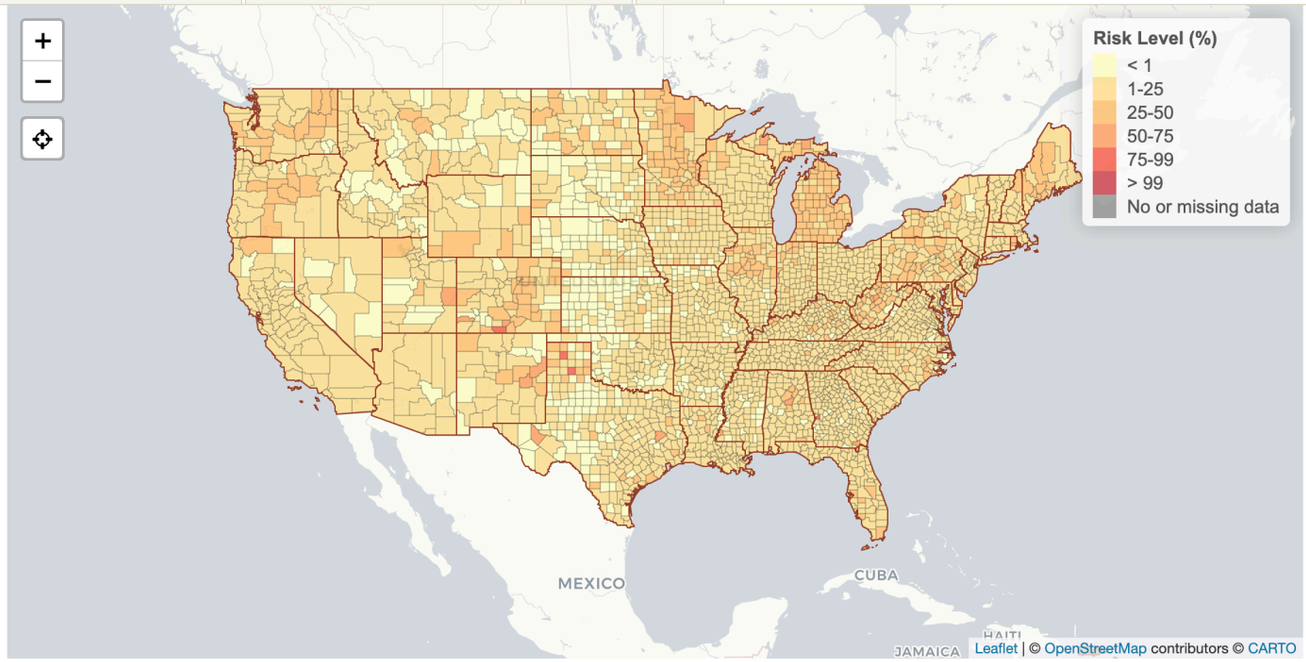
Event Size:

10 50 5000

10 15 20 25 50 100 500 1000 5000

Select Ascertainment Bias

3 5



(Note: This map uses a Web Mercator projection that inflates the area of states in northern latitudes. County boundaries are generalized for faster drawing.)

Explore global risk estimates

So what can you do?

1. **Read and collaborate broadly.**

2. **Integrate epidemiological models, policies, behavior, etc.**

3. **Communicate your work effectively. Which also means moving beyond single measures of risk.**

COVID-19 Event Risk Assessment Planning Tool

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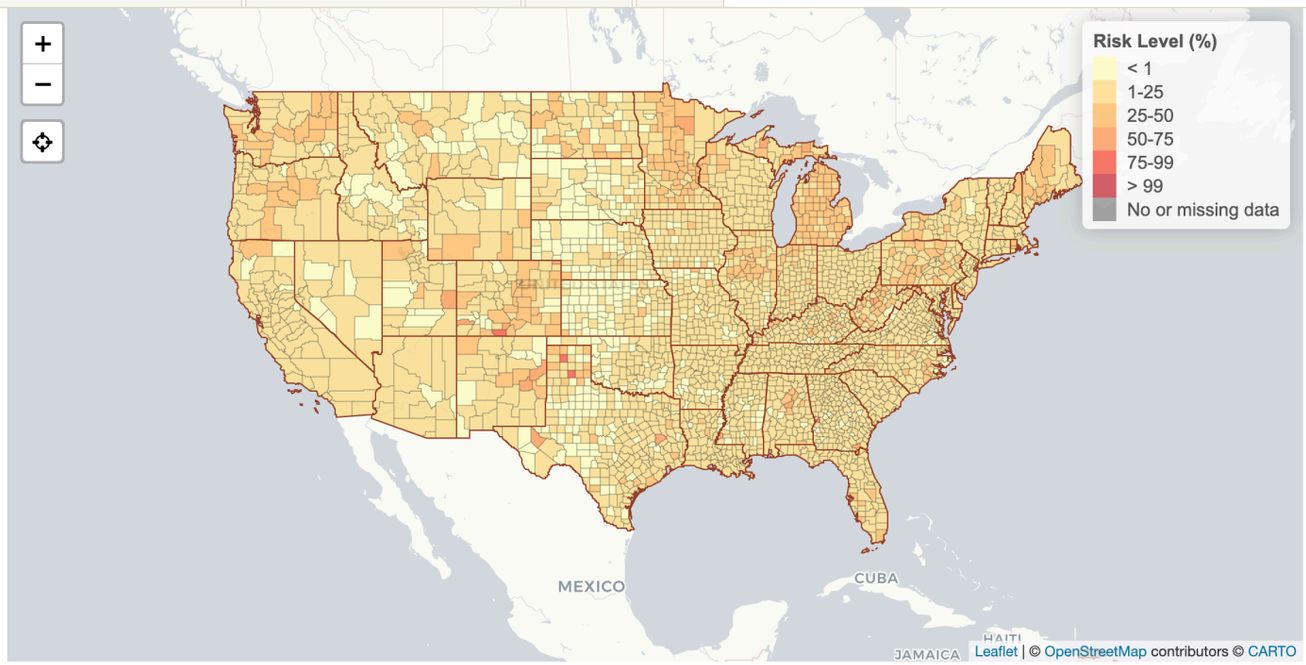
10 15 20 25 50 100 500 1000 5000

Select Ascertainment Bias

3 5



(Note: This map uses a Web Mercator projection that inflates the area of states in northern latitudes. County boundaries are generalized for faster drawing.)

Explore global risk estimates

So what can you do?

1. **Read and collaborate broadly.**

2. **Integrate epidemiological models, policies, behavior, etc.**

3. **Communicate your work effectively.**

4. **Understand that the data *never* “speak for themselves.”**

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BUT, that doesn’t mean we don’t need better data, e.g., on the structure of human social networks.

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6. **Be kind.**

So what can you do?

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4. **Understand that the data *never* “speak for themselves.”**

5. **Build and contribute to diverse, inclusive, and supportive communities.**

6. **Be kind. But resolute in working to improve the culture of academia and our societies.**

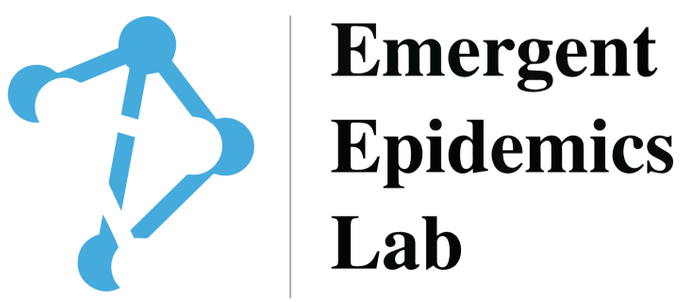
Questions?

Samuel V. Scarpino
Assistant Professor
Northeastern University

External Faculty
Santa Fe Institute
Vermont Complex Systems Center

Co-Founder, Global.health

@svscarpino
scarpino.github.io
s.scarpino@northeastern.edu



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