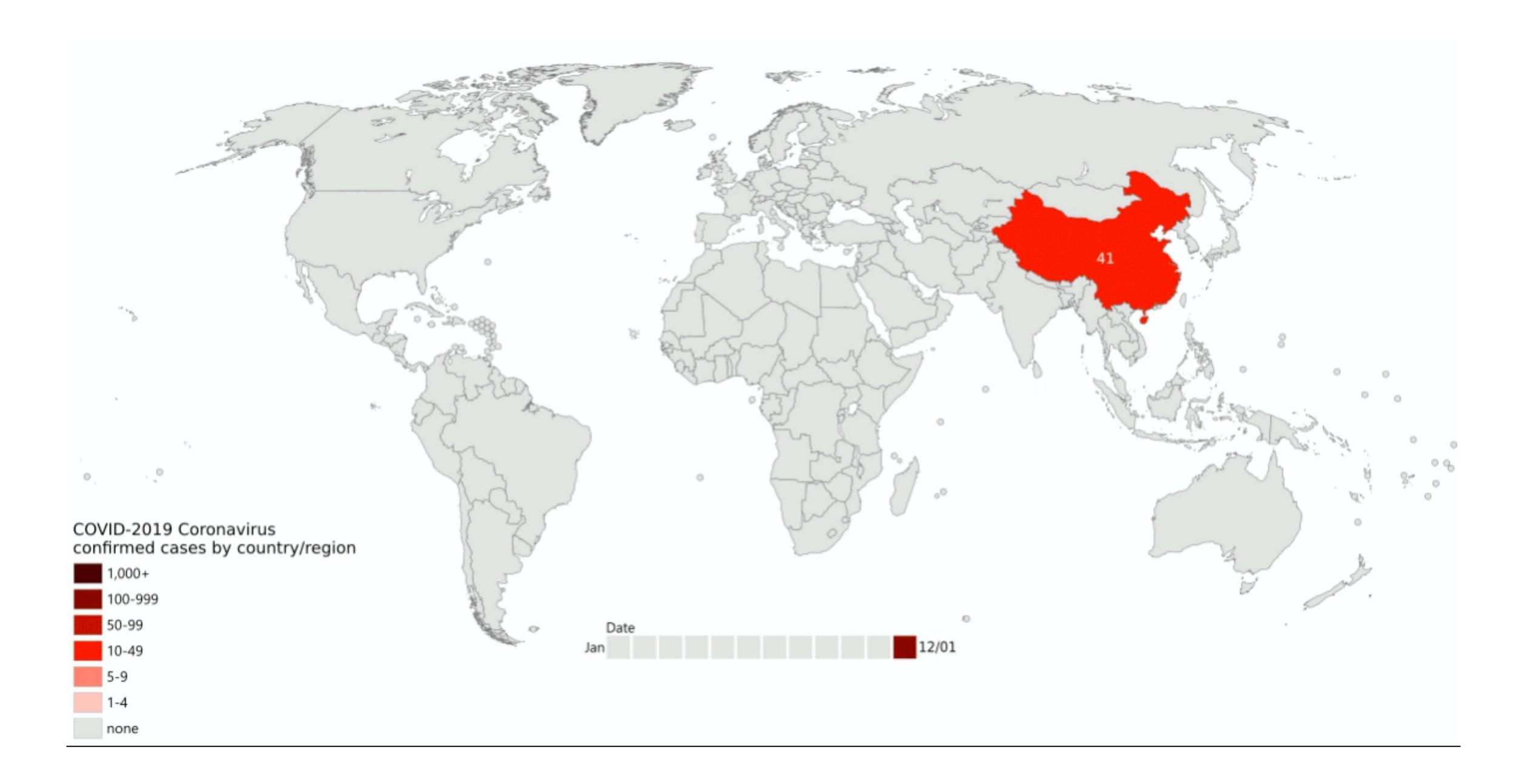
How contact patterns affect the non-equilibrium physics of epidemic outbreaks

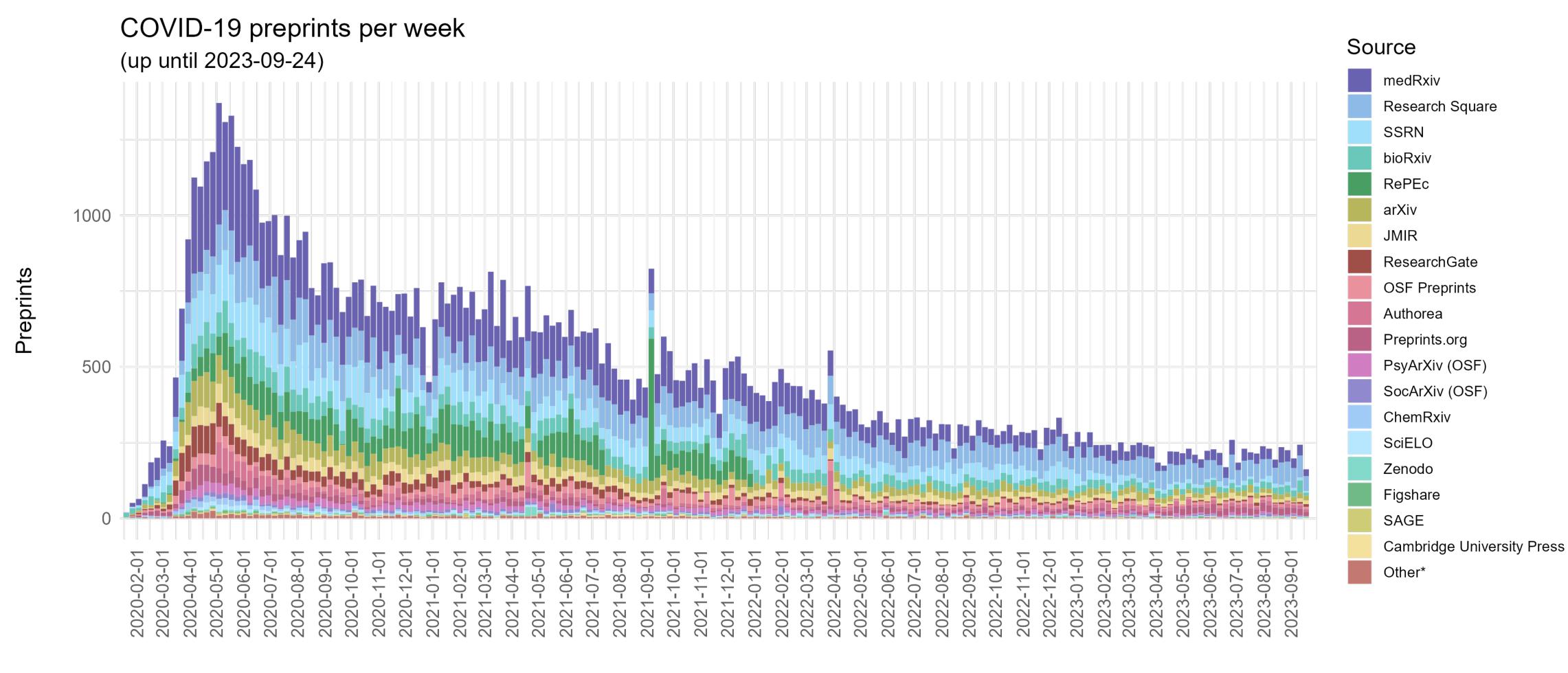
Johannes Zierenberg
Max Planck Institute
for Dynamics and Self-Organization

Theorie Kolloquium Oldenburg, 04/2024



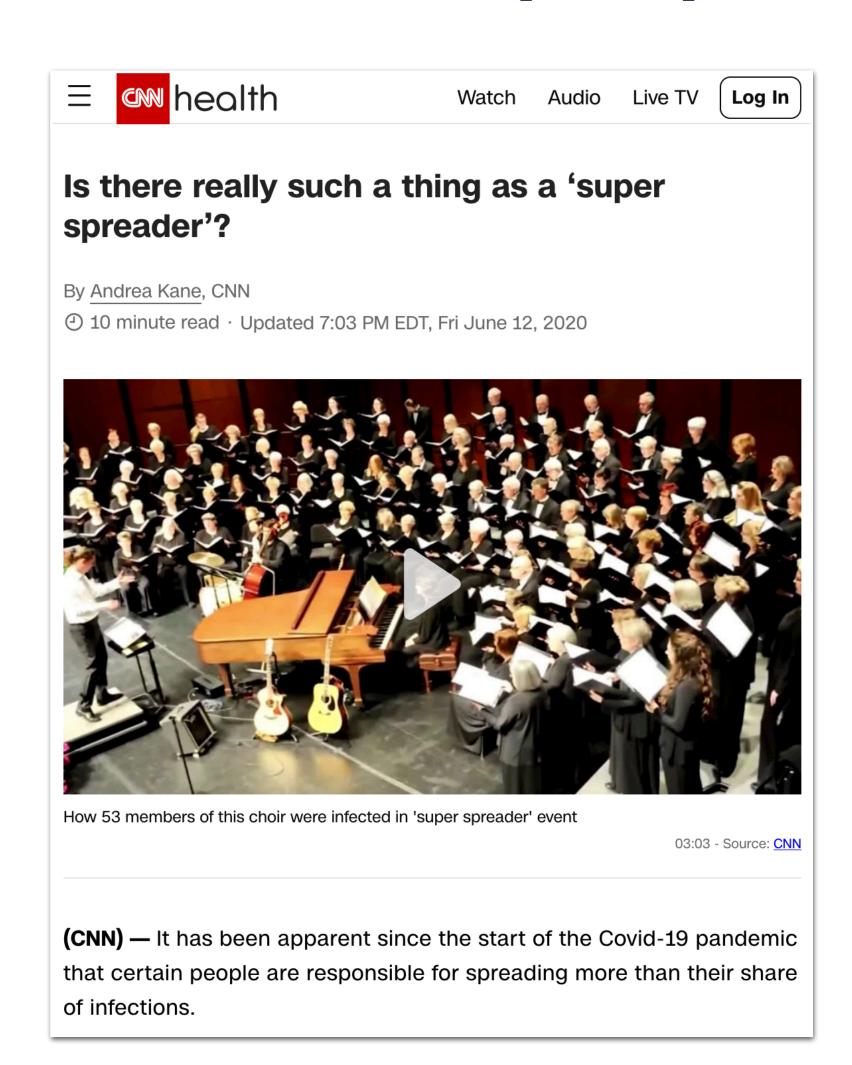


Source: Wikipedia 2



Posted Date (week beginning)

^{* &#}x27;Other' refers to preprint repositories containing <175 total relevant preprints. These include: AfricArXiv (OSF), AgriXiv (OSF), BioHackrXiv (OSF), Copernicus GmbH, ESSOAR, EcoEvoRxiv (OSF), EdArXiv (OSF), Frenxiv (OSF), INA-Rxiv (OSF), IndiaRxiv (OSF), LawArXiv (OSF), MediArXiv (OSF), MetaArXiv (OSF), NutriXiv (OSF), ScienceOpen, SportRxiv (OSF), Techrxiv (IEEE), WHO, engrXiv (OSF).



 Clear evidence that most virus introductions spread little and few cases amplify considerably.

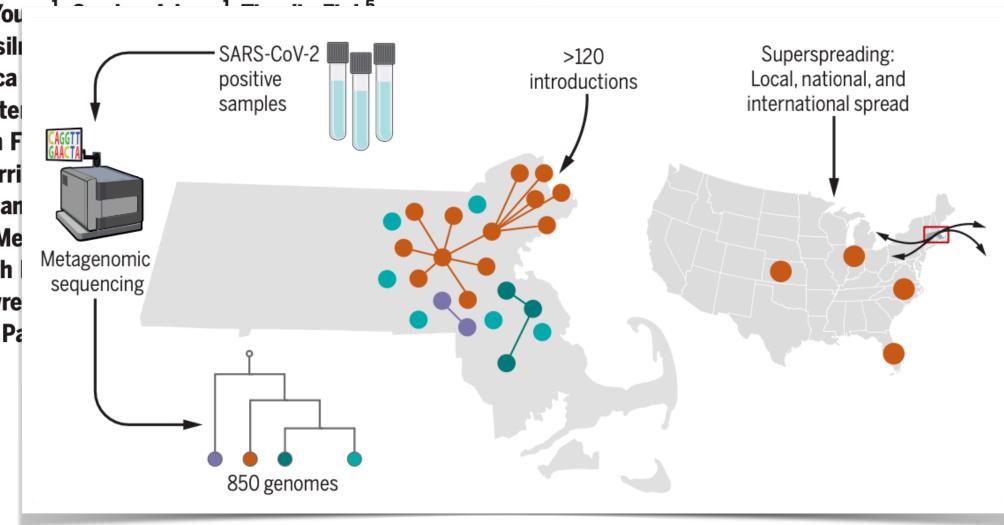
RESEARCH ARTICLE

CORONAVIRUS

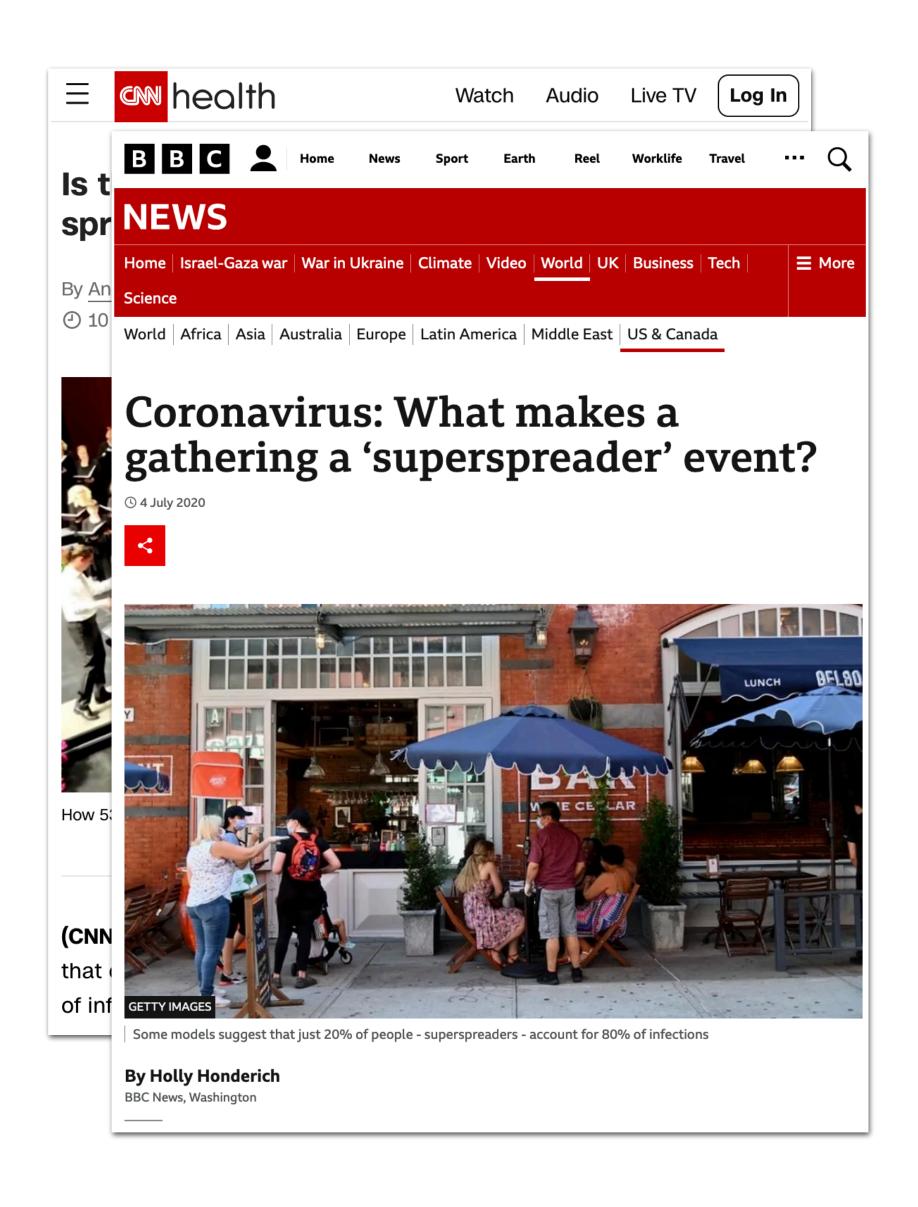
Phylogenetic analysis of SARS-CoV-2 in Boston highlights the impact of superspreading events

Jacob E. Lemieux^{1,2}*†, Katherine J. Siddle^{1,3}*, Bennett M. Shaw^{1,2}, Christine Loreth¹,

Stephen F. Schaffner^{1,3,4}, Adrianne Gladden-You Christopher H. Tomkins-Tinch^{1,3}, Lydia A. Krasili Matthew R. Bauer^{1,6}, Kim A. Lagerborg^{1,6}, Erica Melis N. Anahtar⁸, Aaron E. Lin^{1,3}, Amber Carte Sushma Chaluvadi¹, Caroline Cusick¹, Katelyn F Maha Farhat^{9,10}, Damien Slater², Jason B. Harri Jessie M. Gaeta^{12,13}, Travis P. Baggett^{12,14,15}, Jan Tami D. Lieberman^{1,16}, Anthony Philippakis¹, Me Jeremy Luban^{1,17,18}, Edward T. Ryan^{2,4,15}, Sarah William P. Hanage¹⁹, Glen R. Gallagher⁵‡, Lawre Virginia M. Pierce^{8,21,22}‡, Eric Rosenberg^{2,8}‡, Pa Daniel J. Park¹‡, Bronwyn L. MacInnis^{1,4,18}†‡

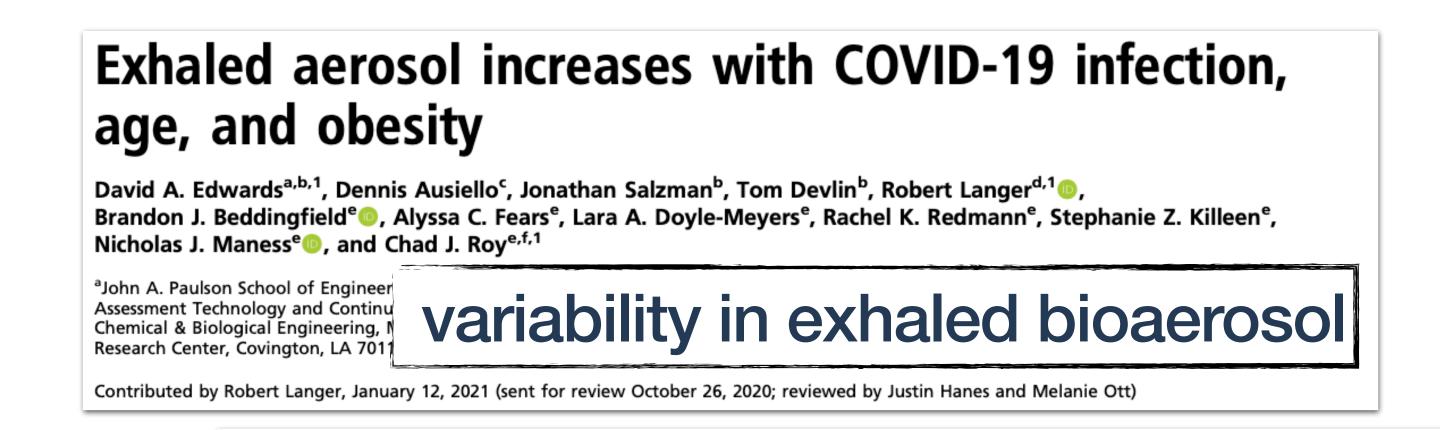


Lemieux et al. Science (2021)



- Clear evidence that most virus introductions spread little and few cases amplify considerably.
- Many mechanisms underlying superspreading!

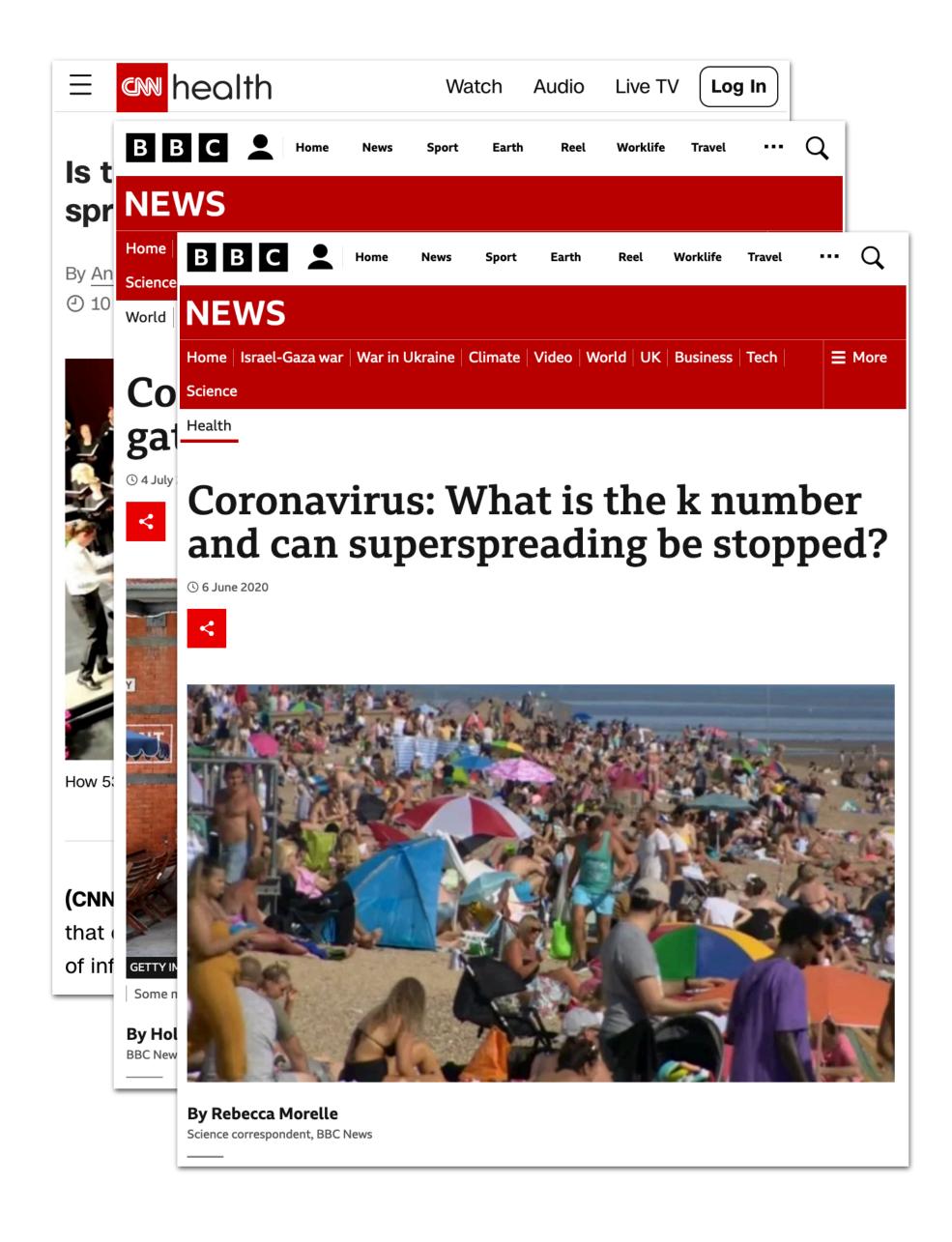
.ETTERS





Maksim Kitsak^{1,2}, Lazaros K. Gallos³, Shlomo Havlin⁴, Fredrik Liljeros⁵, Lev Muchnik⁶, H. Eugene Stanley¹ and Hernán A. Makse³*

nature



- Clear evidence that most virus introductions spread little and few cases amplify considerably.
- Many mechanisms underlying superspreading!
- From a statistical perspective super-spreading can be considered a `rare` event.

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

IFTTFRS

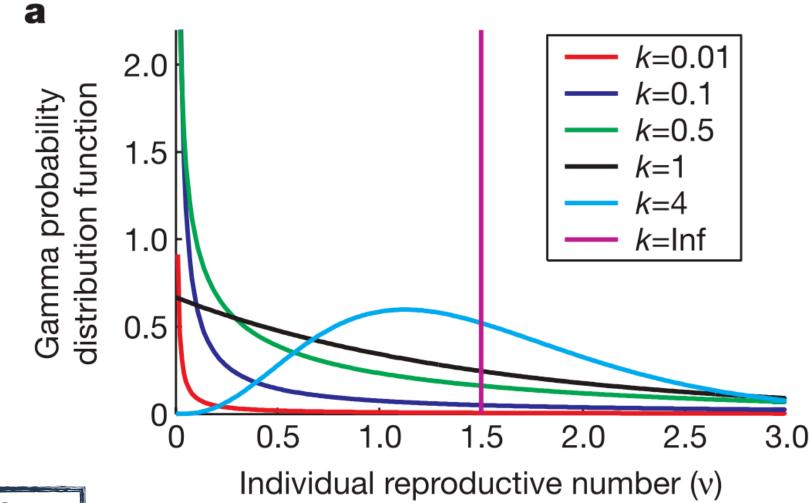
Superspreading and the effect of individual variation on disease emergence a 0.8 [

J. O. Lloyd-Smith^{1,2}, S. J. Schreiber³, P. E. Kopp⁴ & W. M. Getz¹

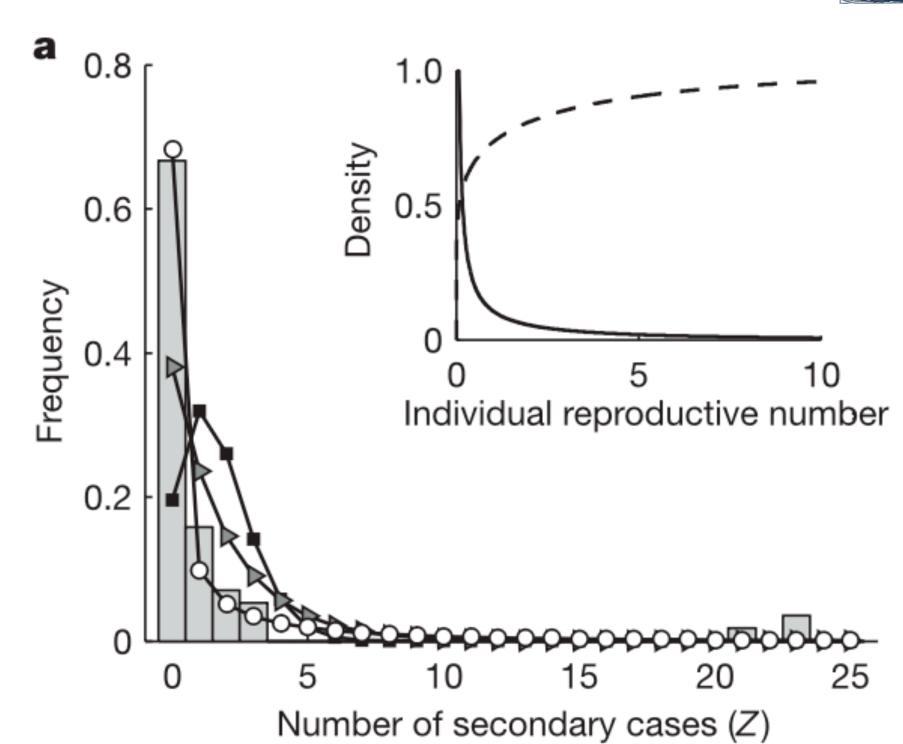
LETTERS

Superspreading and the effect of individual variation on disease emergence

J. O. Lloyd-Smith^{1,2}, S. J. Schreiber³, P. E. Kopp⁴ & W. M. Getz¹



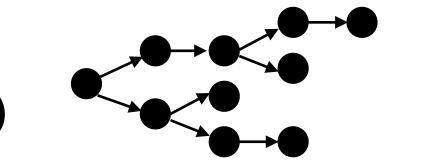
Overdispersion: $\sigma_Z^2 = R_0 + R_0^2/k$



Individual 'reproductive number' $\nu \sim P(R_0)$ with mean R_0

Secondary cases stochastic $Z \sim \text{Poisson}(\nu)$

- Branching Process: $P(R_0) = \delta(\nu - R_0) \text{ then } Z \sim \text{Poisson}(R_0)$



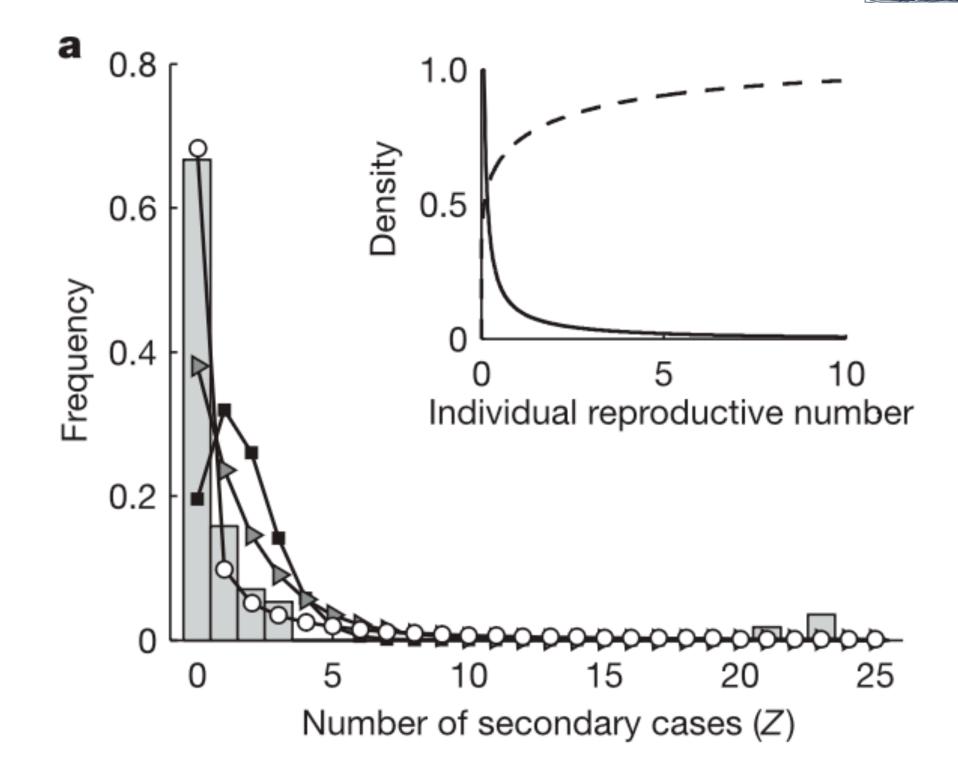
- Homogeneous transmission and recovery rate: $P(R_0) = \operatorname{Exp}(R_0)$ then $Z \sim \operatorname{Geometric}(R_0)$
- More general: $P(R_0) = \operatorname{Gamma}(R_0,k) \text{ then } Z \sim \operatorname{Neg.Binomial}(R_0,k)$

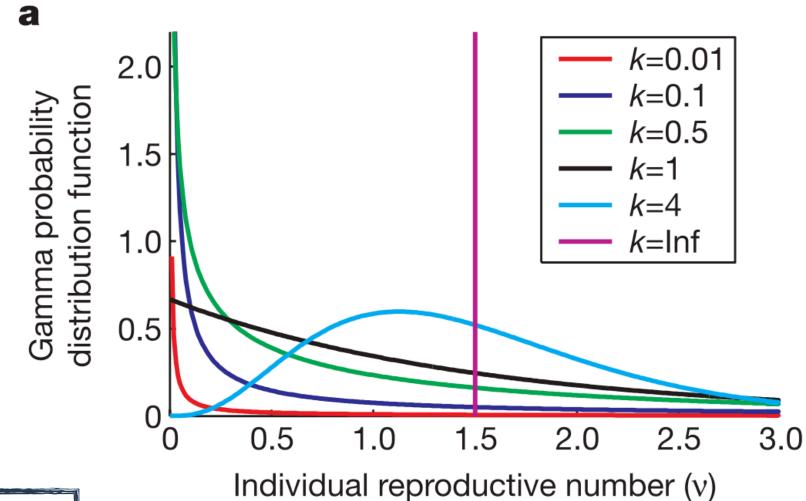
LETTERS

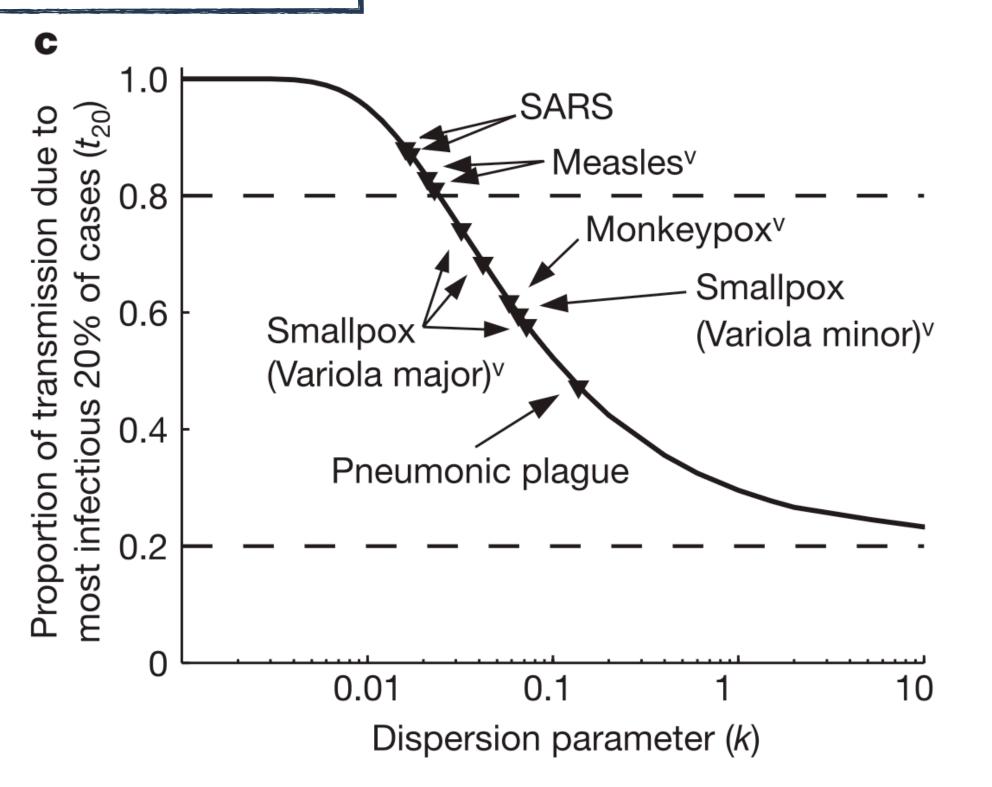
Superspreading and the effect of individual variation on disease emergence

J. O. Lloyd-Smith^{1,2}, S. J. Schreiber³, P. E. Kopp⁴ & W. M. Getz¹

Overdispersion: $\sigma_Z^2 = R_0 + R_0^2/k$





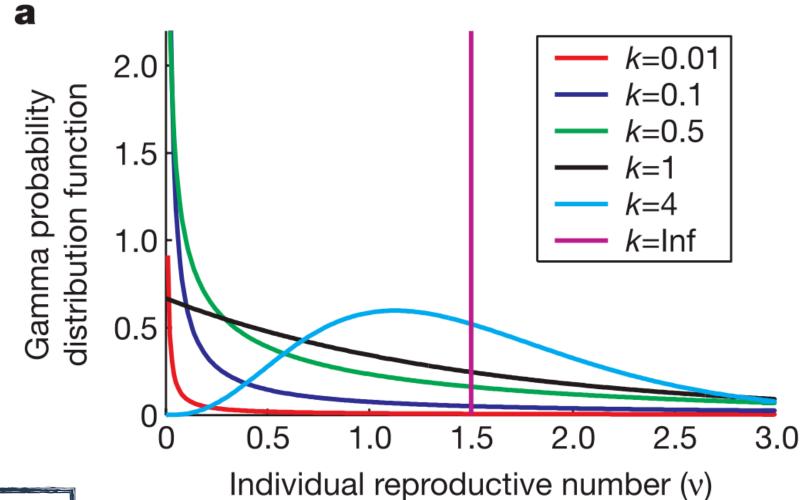


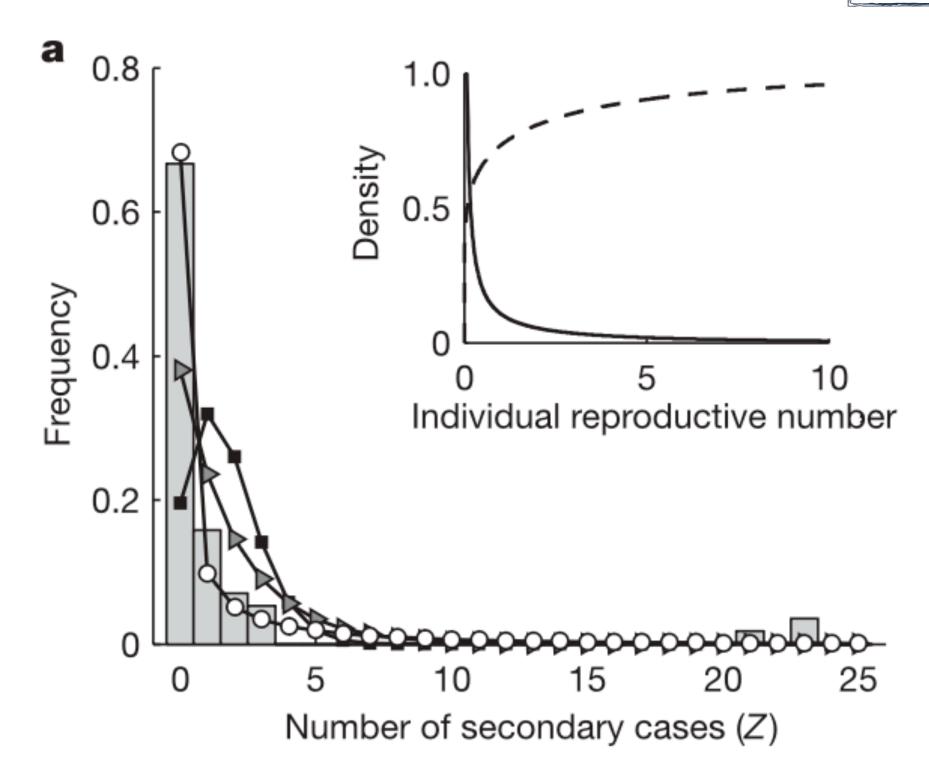
LETTERS

Superspreading and the effect of individual variation on disease emergence

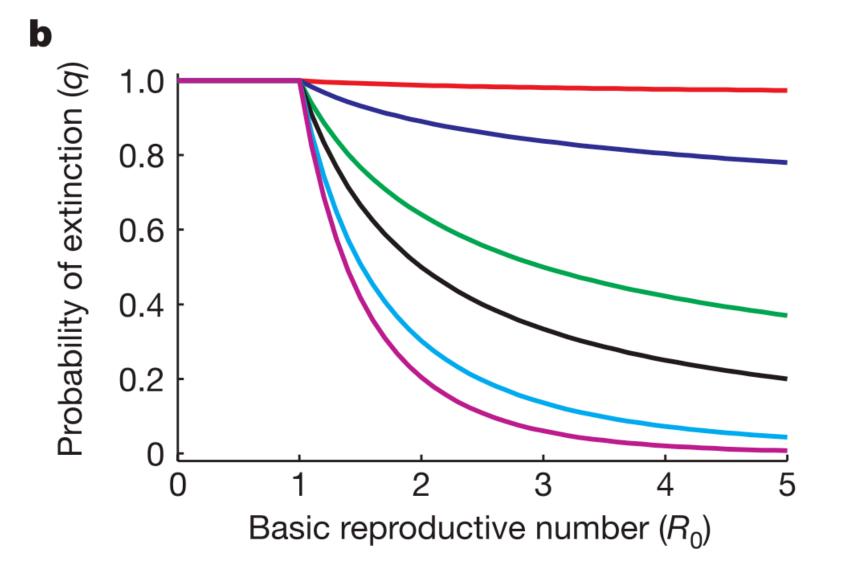
J. O. Lloyd-Smith^{1,2}, S. J. Schreiber³, P. E. Kopp⁴ & W. M. Getz¹

Overdispersion: $\sigma_Z^2 = R_0 + R_0^2/k$





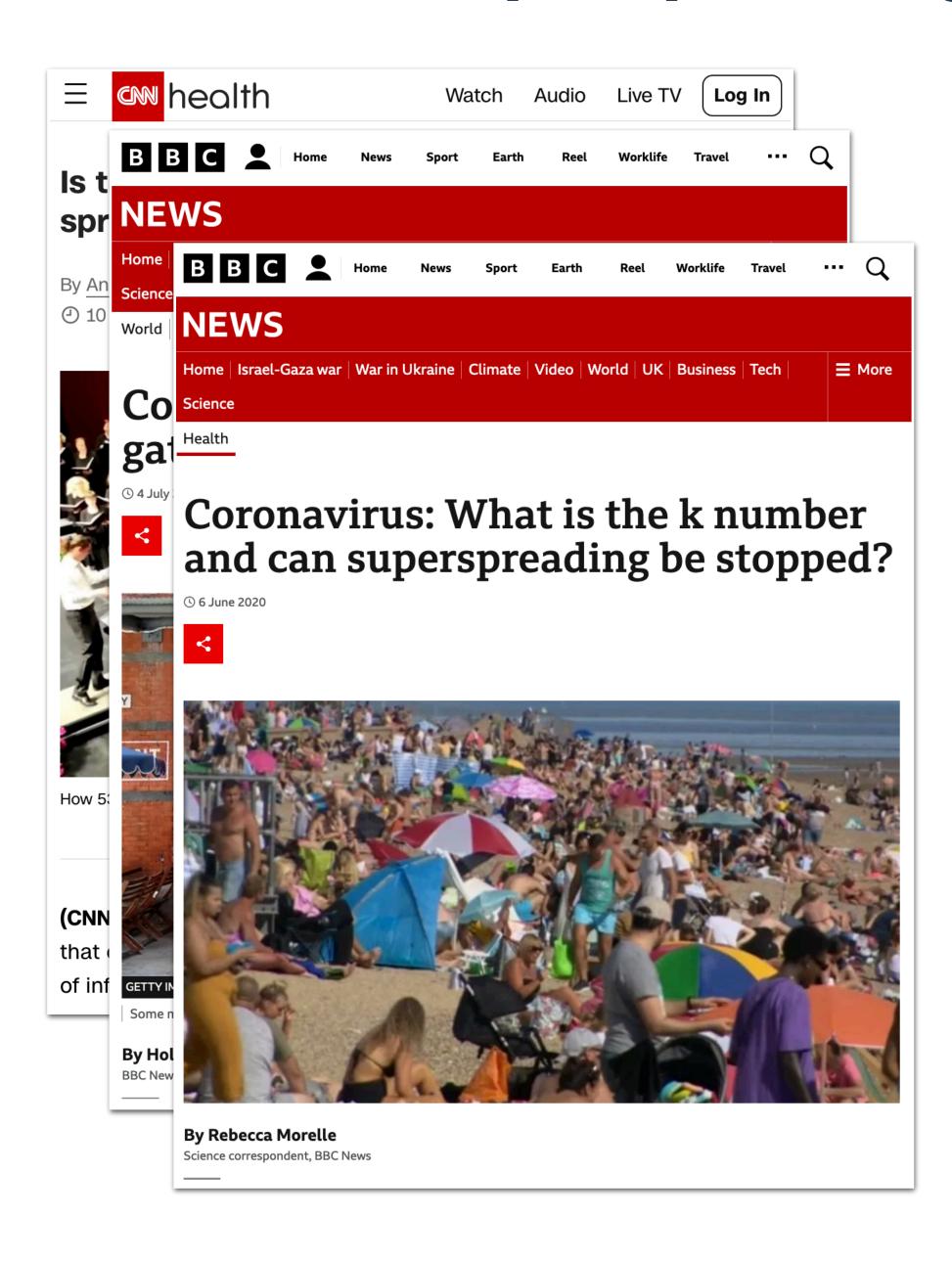
Strong effect on statistics of epidemic outbreaks:





- Clear evidence that most virus introductions spread little and few cases amplify considerably.
- Many mechanisms underlying superspreading!
- From a statistical perspective super-spreading can be considered a `rare` event.

Cumulatively, these observations suggest that behavioural, interventional, general demographic, seasonal, and other environmental factors might affect transmissibility but are not the key determinants of overdispersion for directly transmitted viruses. They suggest instead that k, at least in part, is an intrinsic characteristic of these viruses and, as obligate intracellular parasites, their host interactions. That is, poorly ventilated, crowded spaces with susceptible individuals facilitate superspreading, but whether a virus tends to transmit via large clusters in the first place seems to be intrinsic to that viral infection. Thus, the previous question can be reframed: which virological factors mediate k?



How much do human contact patterns contribute to overdispersion?

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Chen et al. Lancet Infect. Dis. (2021)

Contact patterns and epidemic outbreaks

Statistical analysis of disease spread from human contact patterns



Epidemic phase transition in a toy model of spatiotemporal contacts

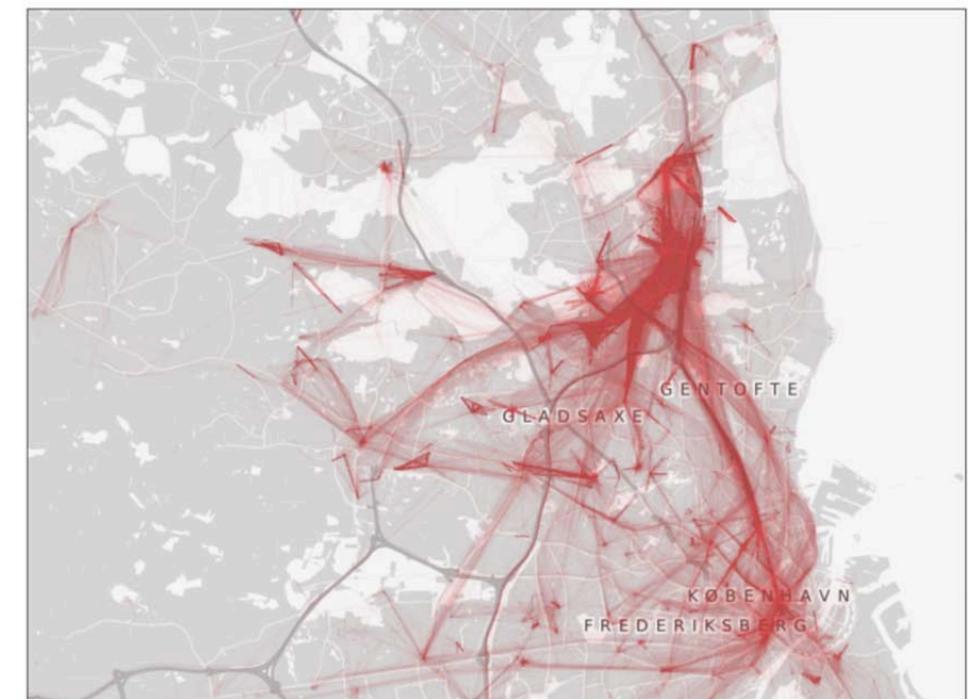
Copenhagen Networks Study

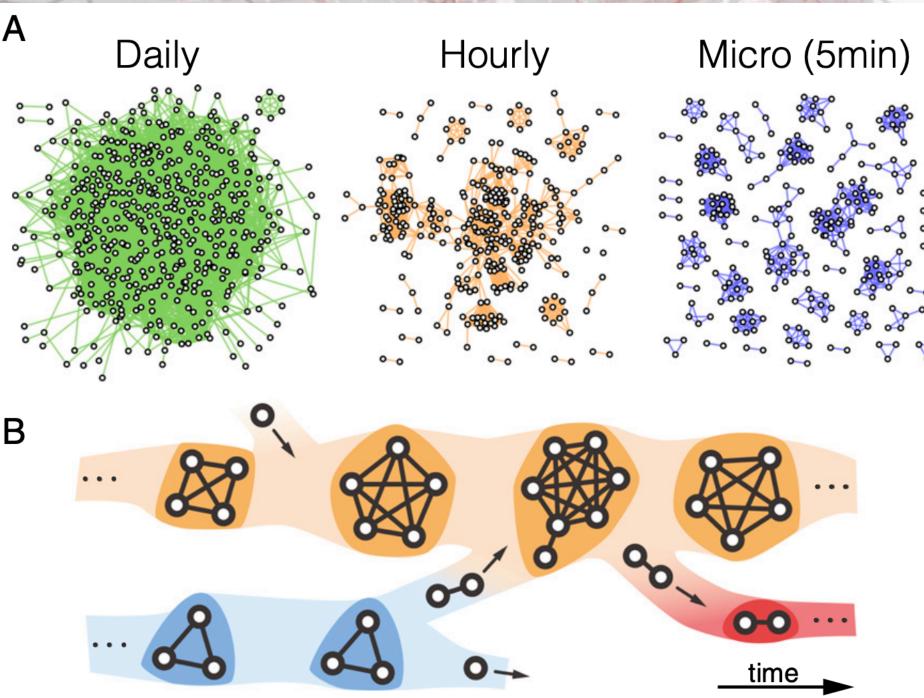
- 1000 participants from DTU with smartphones as social sensors
- Face-to-face interactions, telecommunication, social networks, location, and background information

SCIENTIFIC DATA

OPEN Interaction data from the DATA DESCRIPTOR Copenhagen Networks Study

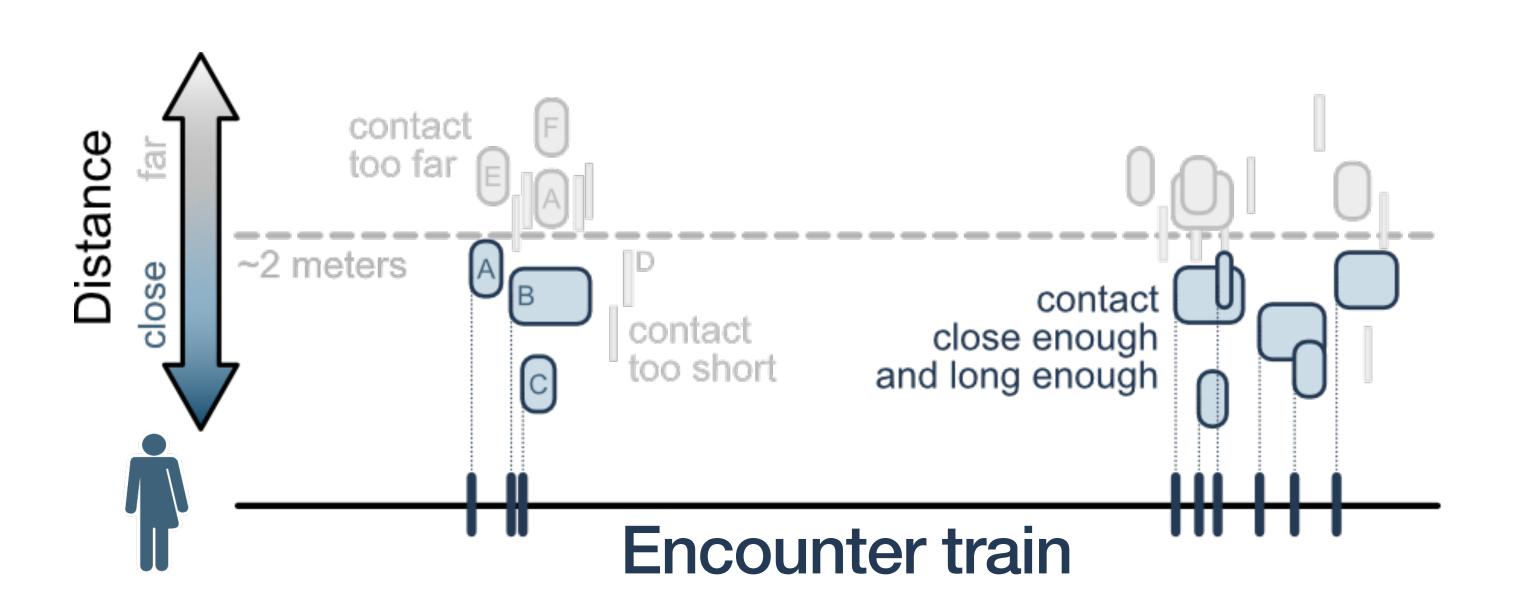
Piotr Sapiezynski¹, Arkadiusz Stopczynski¹, David Dreyer Lassen² & Sune Lehmann (1)^{1,2*}

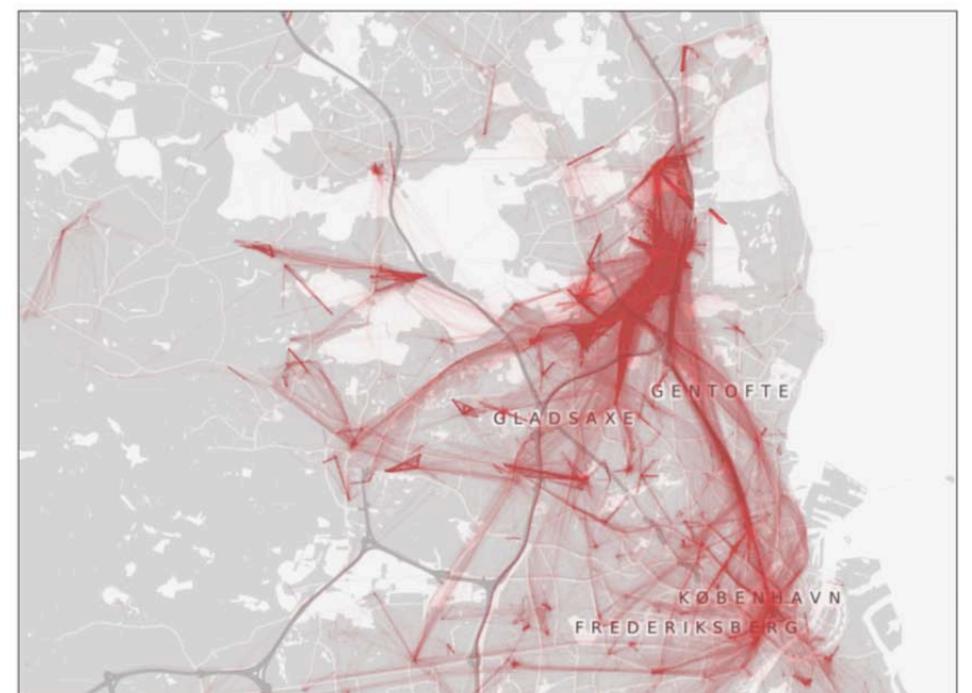


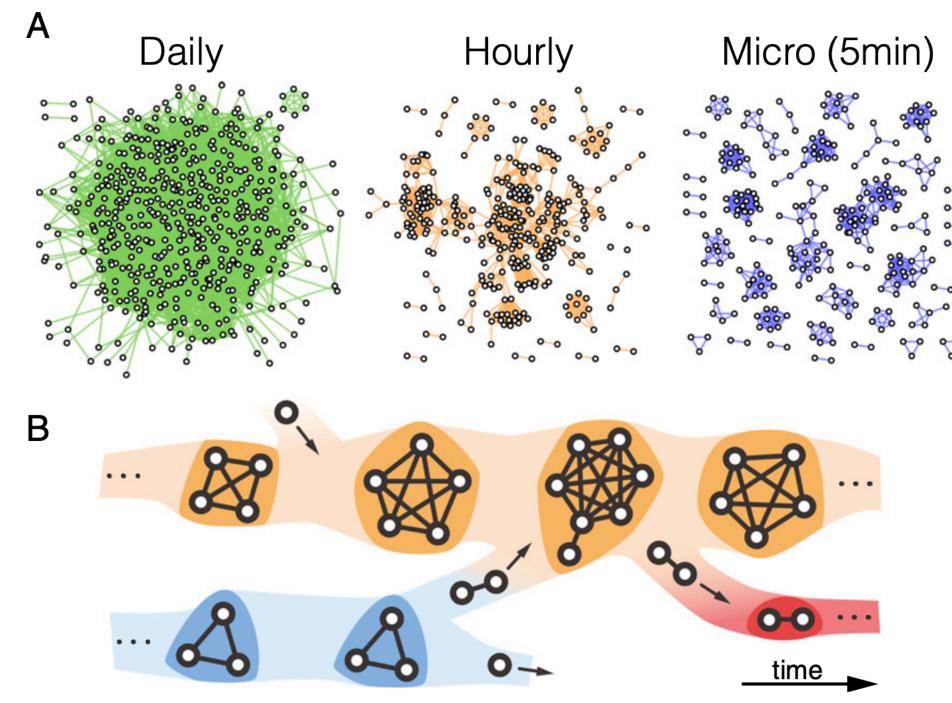


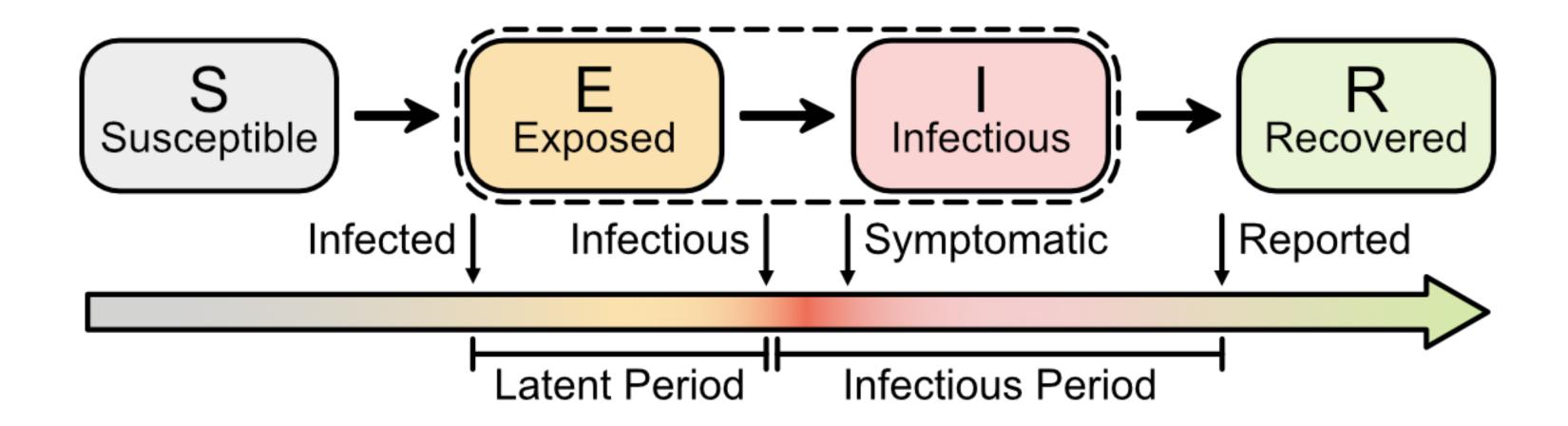
Copenhagen Networks Study

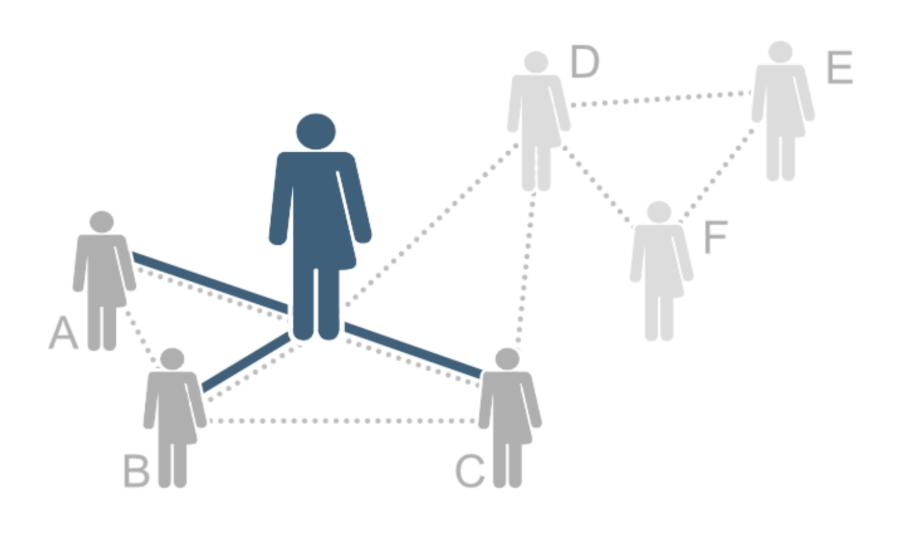
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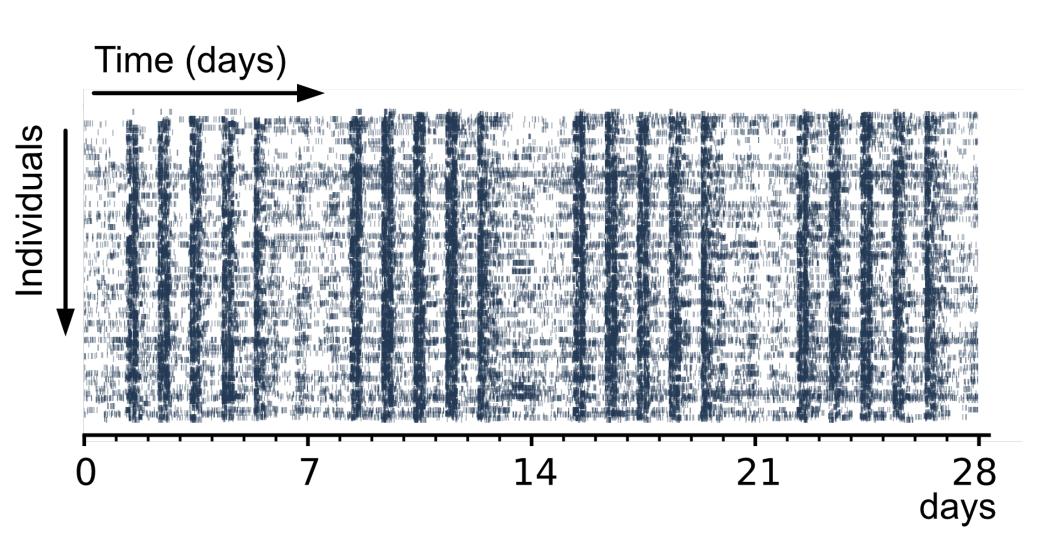


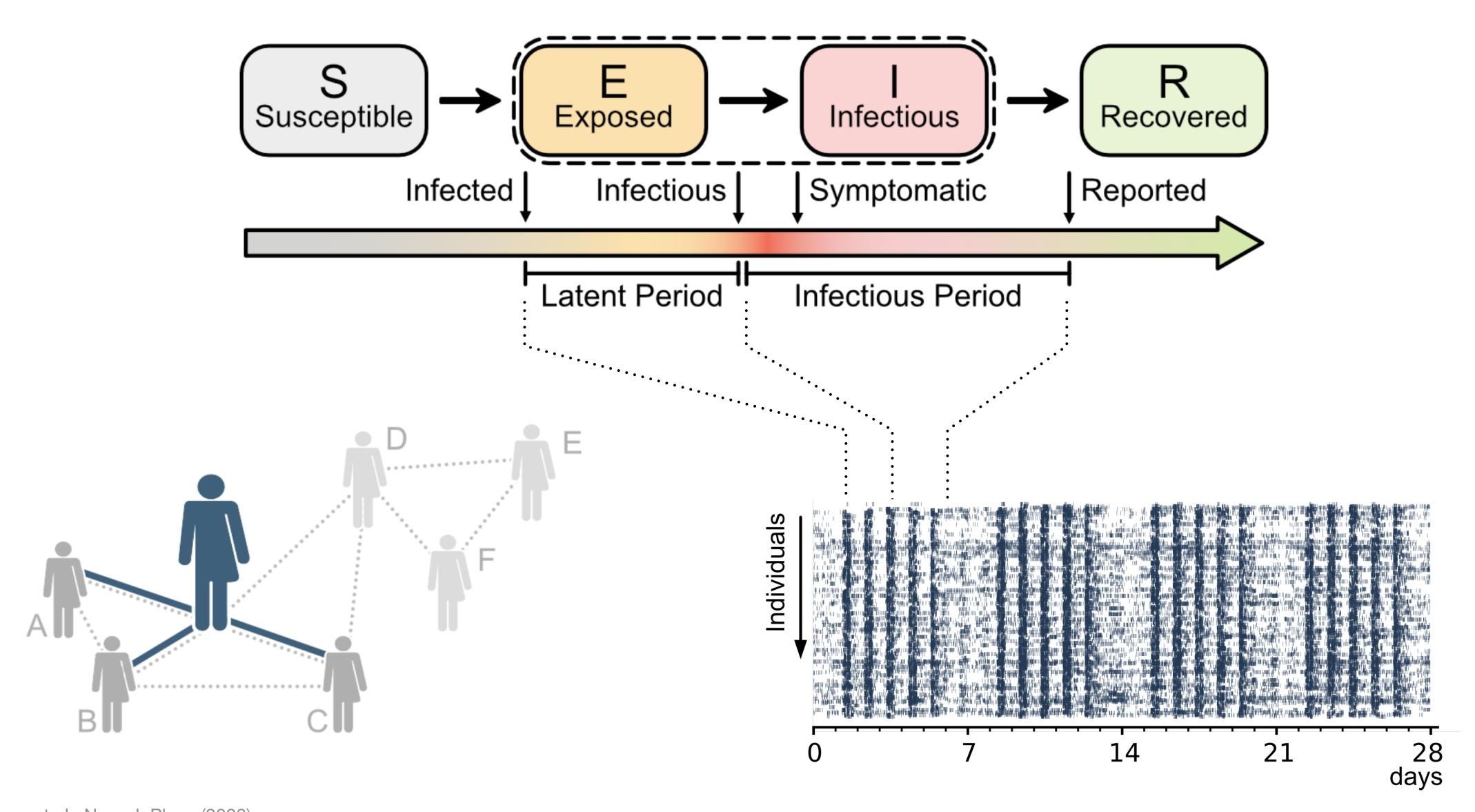


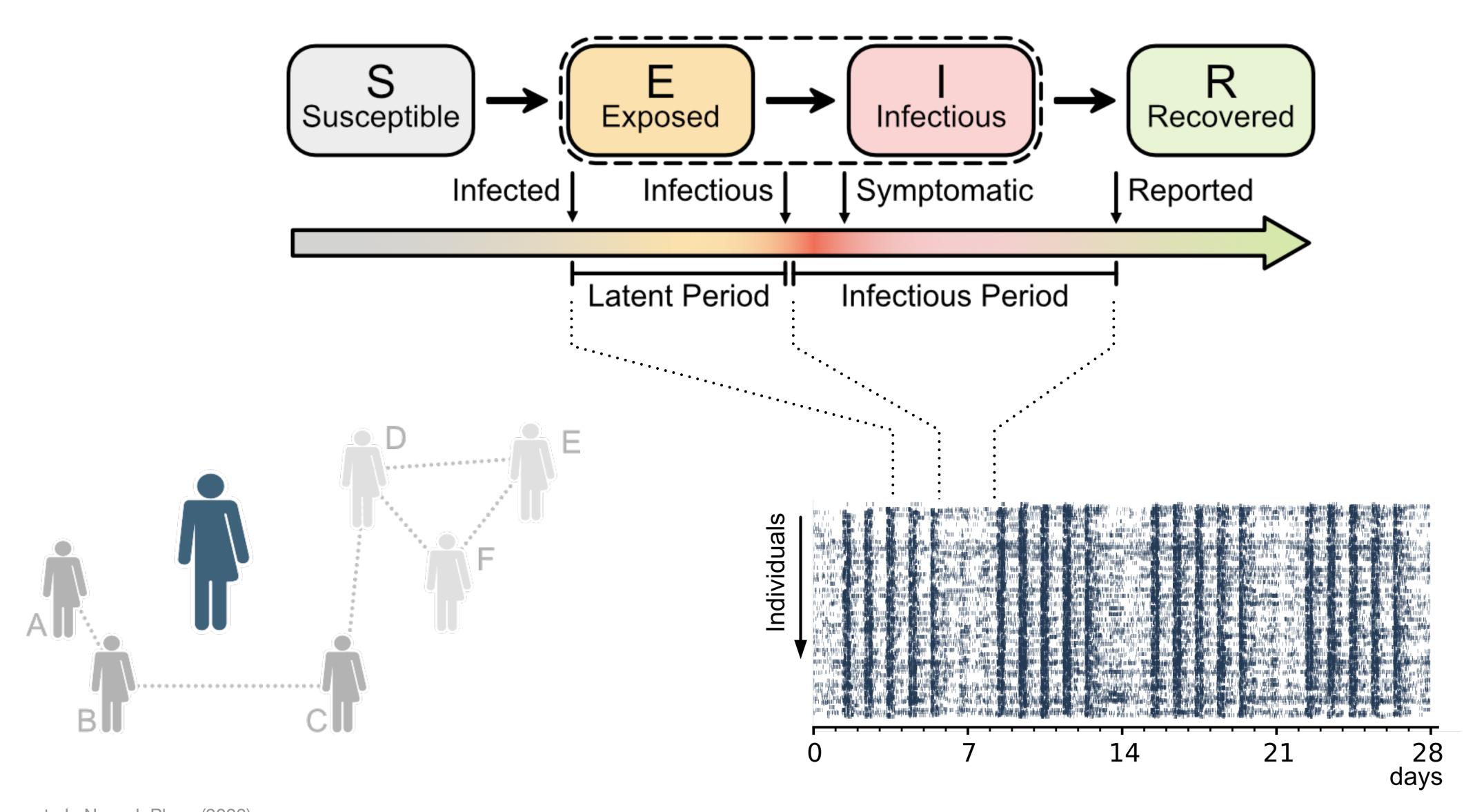


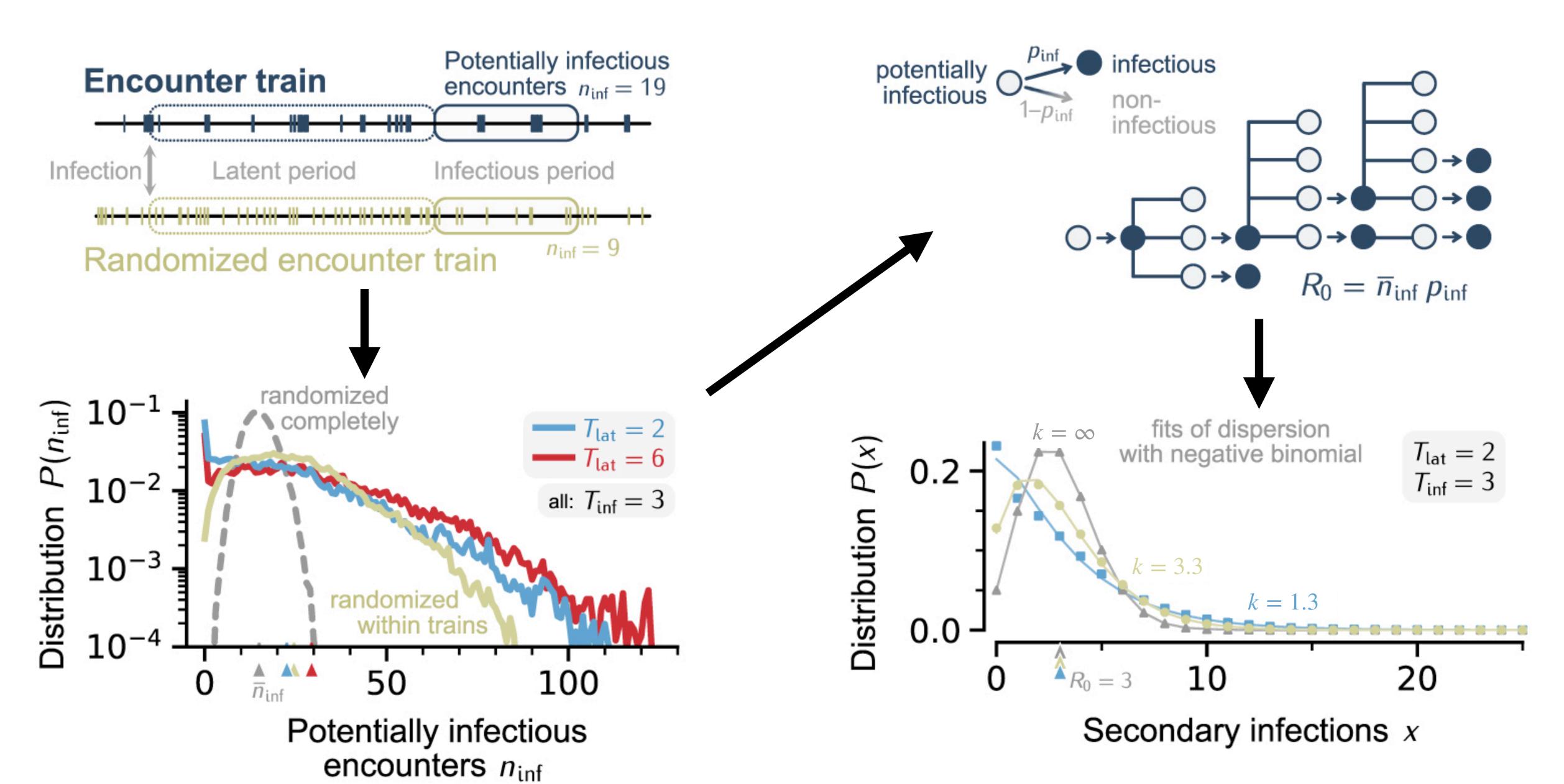


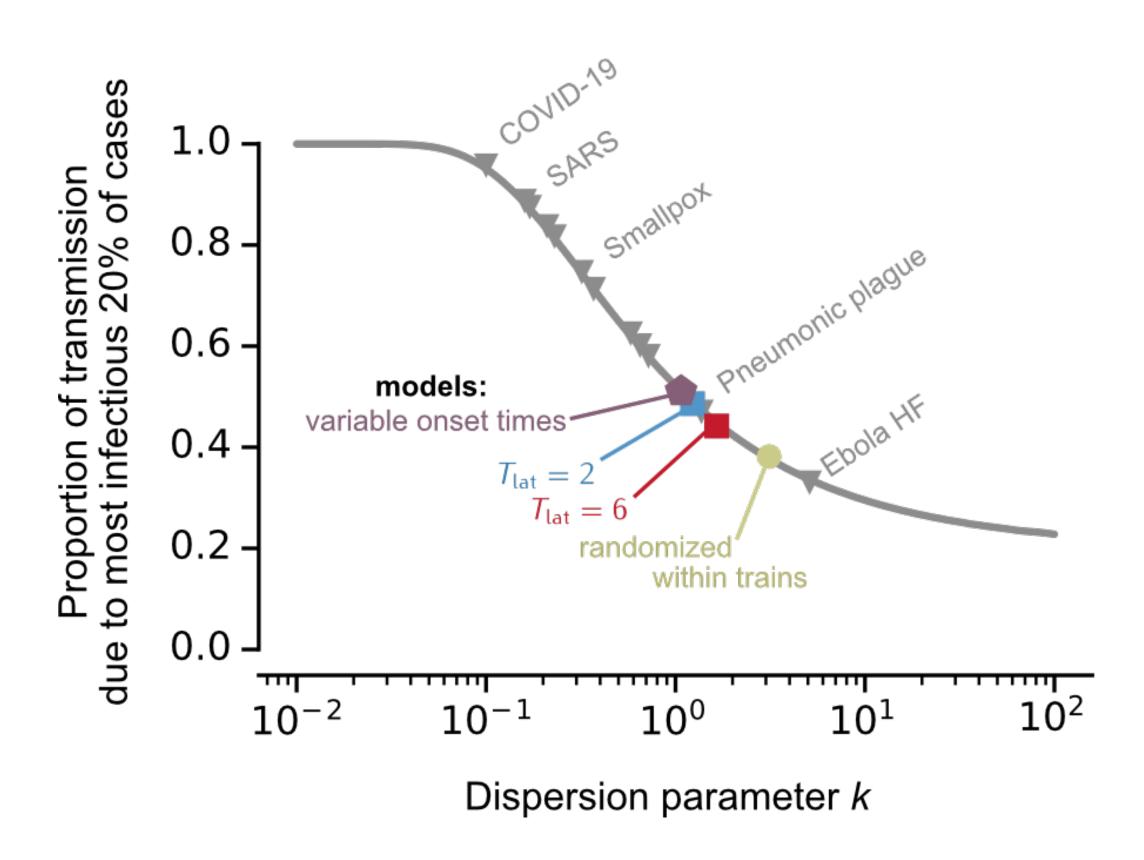












We used a very simple disease model, yet dispersion from contact patterns is in the right regime

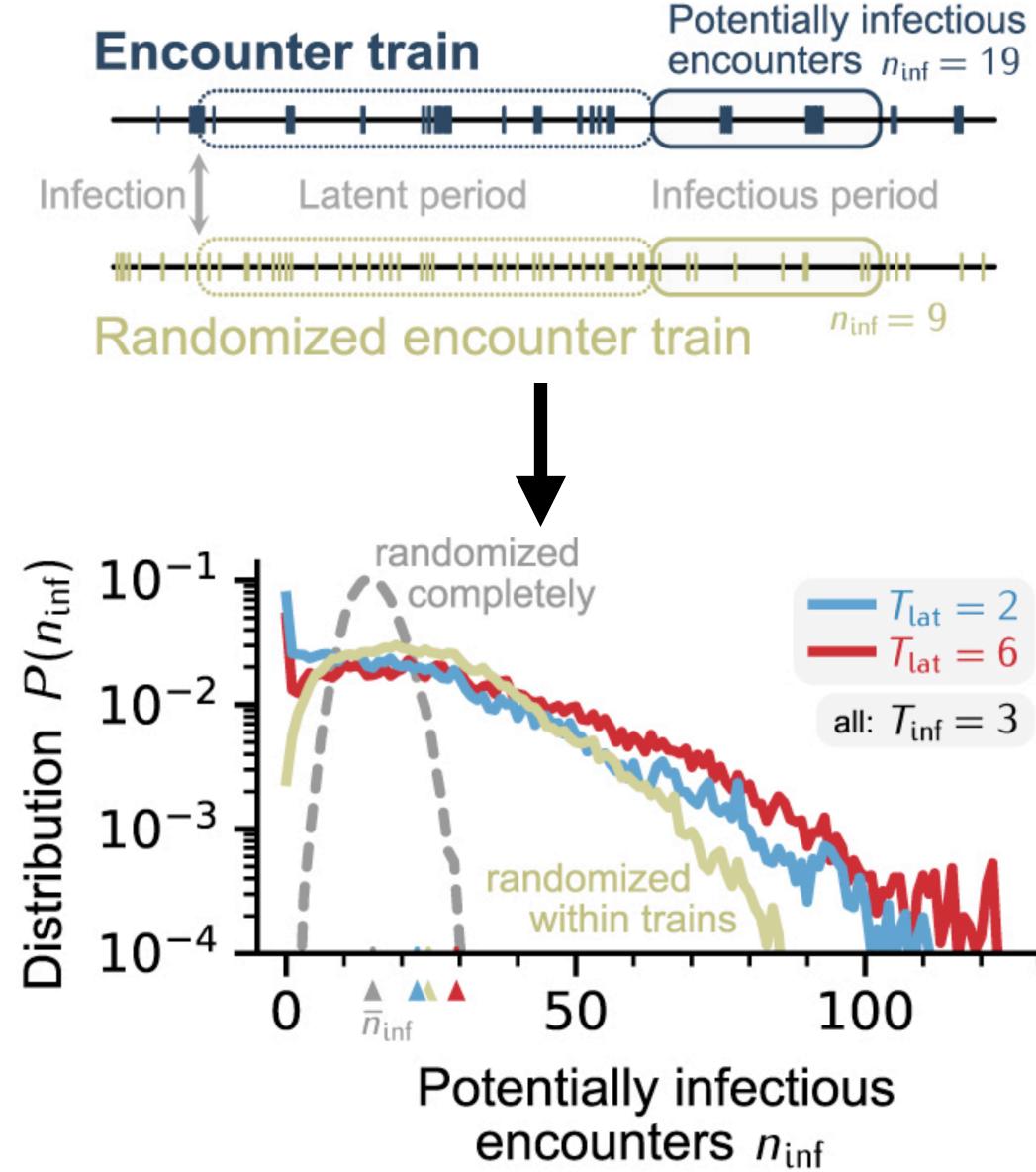
Cumulatively, these observations suggest that behavioural, interventional, general demographic, seasonal, and other environmental factors might affect transmissibility but are not the key determinants of overdispersion for directly transmitted viruses. They suggest instead that k, at least in part, is an intrinsic characteristic of these viruses and, as obligate intracellular parasites, their host interactions. That is, poorly ventilated, crowded spaces with susceptible individuals facilitate superspreading, but whether a virus tends to transmit via large clusters in the first place seems to be intrinsic to that viral infection. Thus, the previous question can be reframed: which virological factors mediate k?

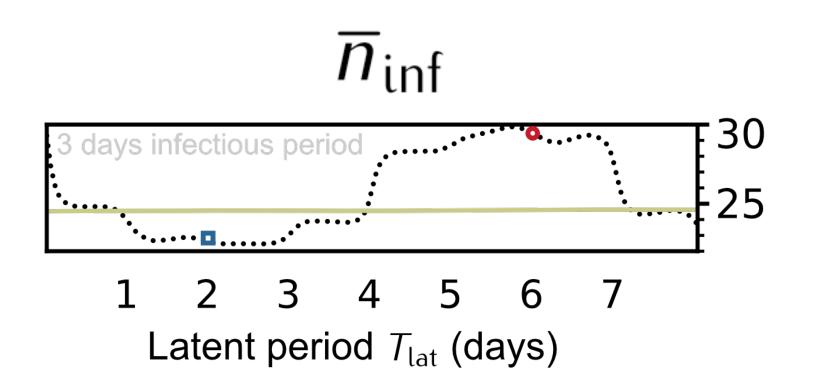
cf. Chen et al. Lancet Infect. Dis. (2021)

Our perspective:

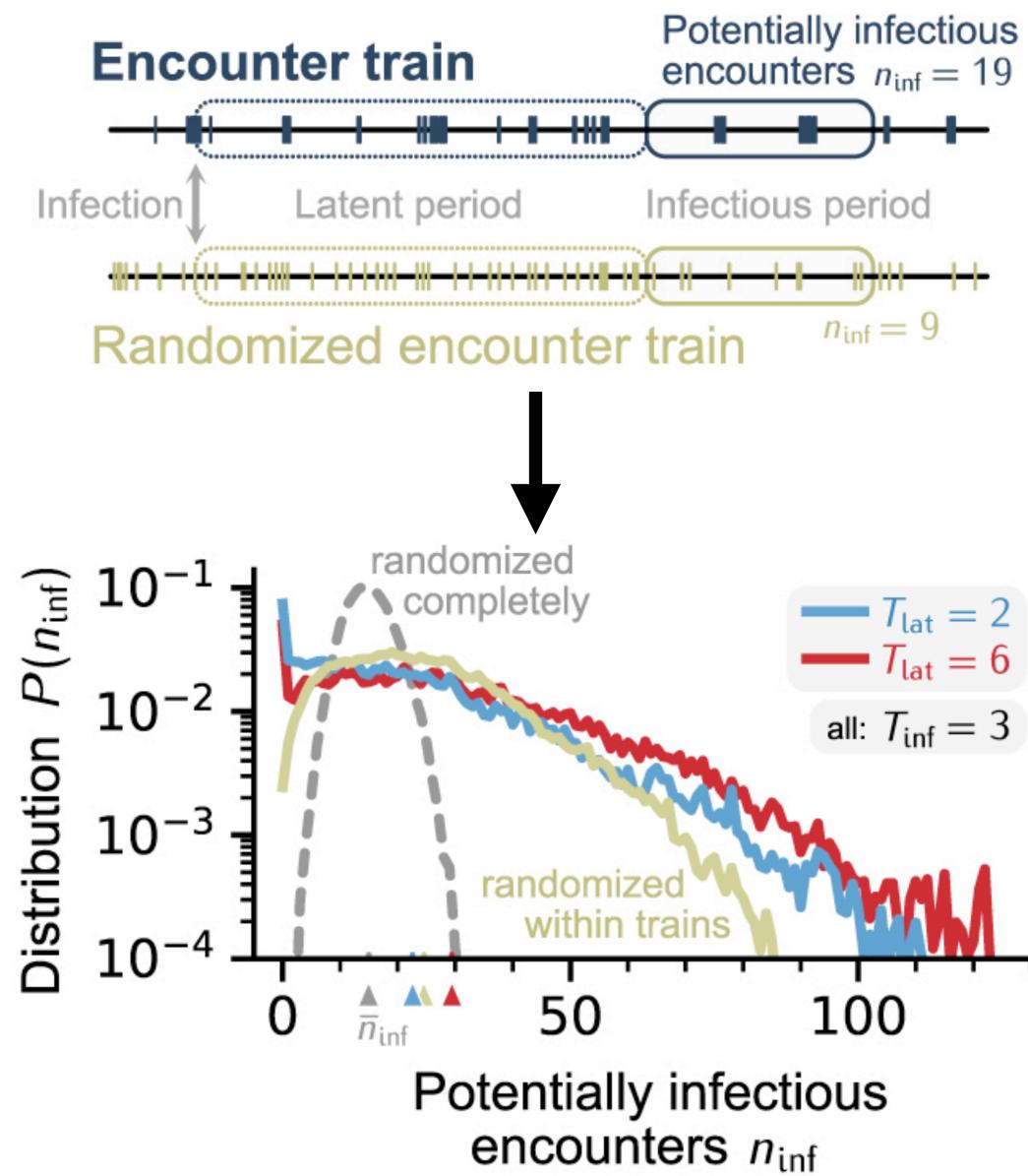
Contact patterns form baseline;
Physiological disease details are higher order

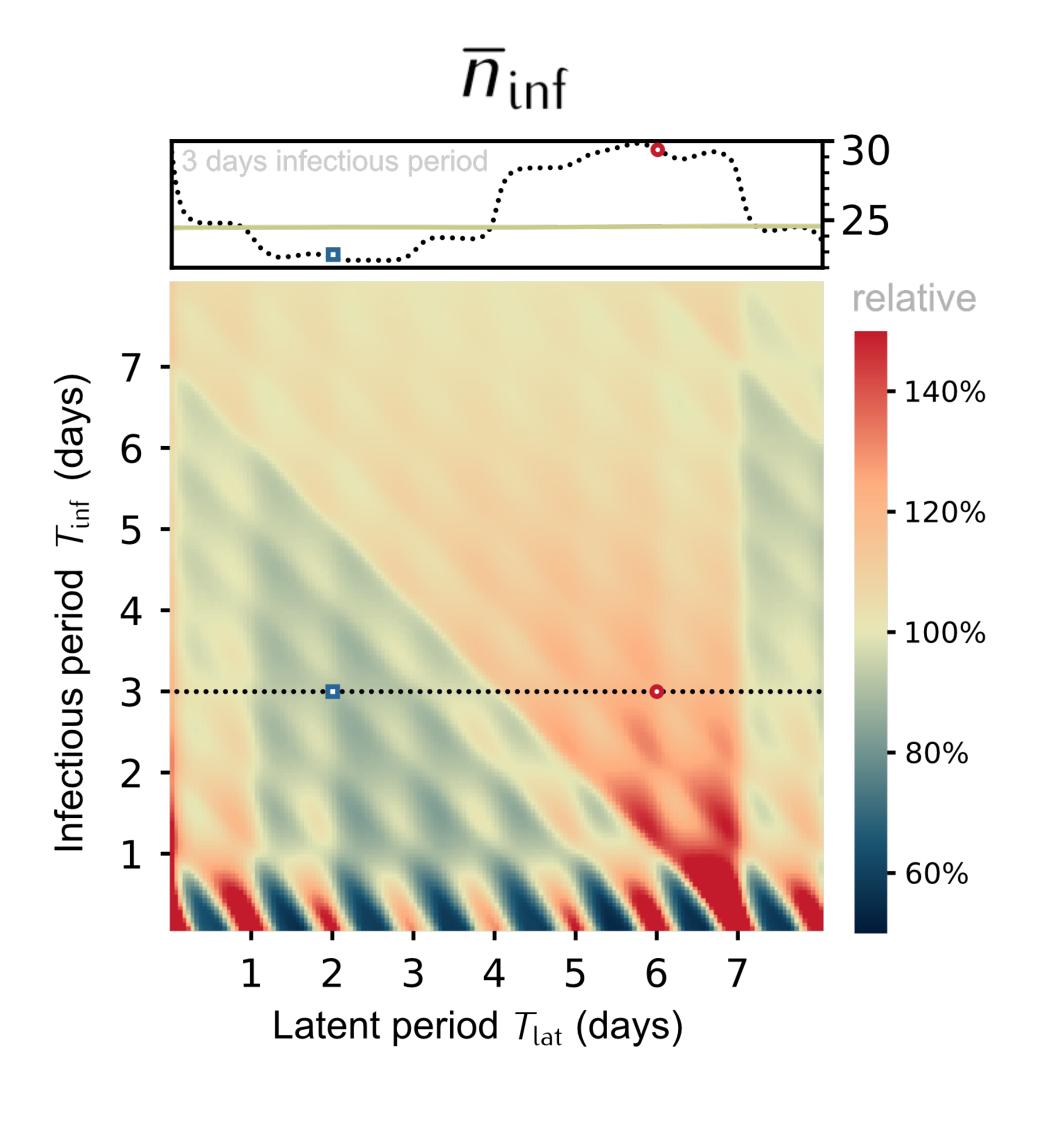
Modulation from human contact patterns



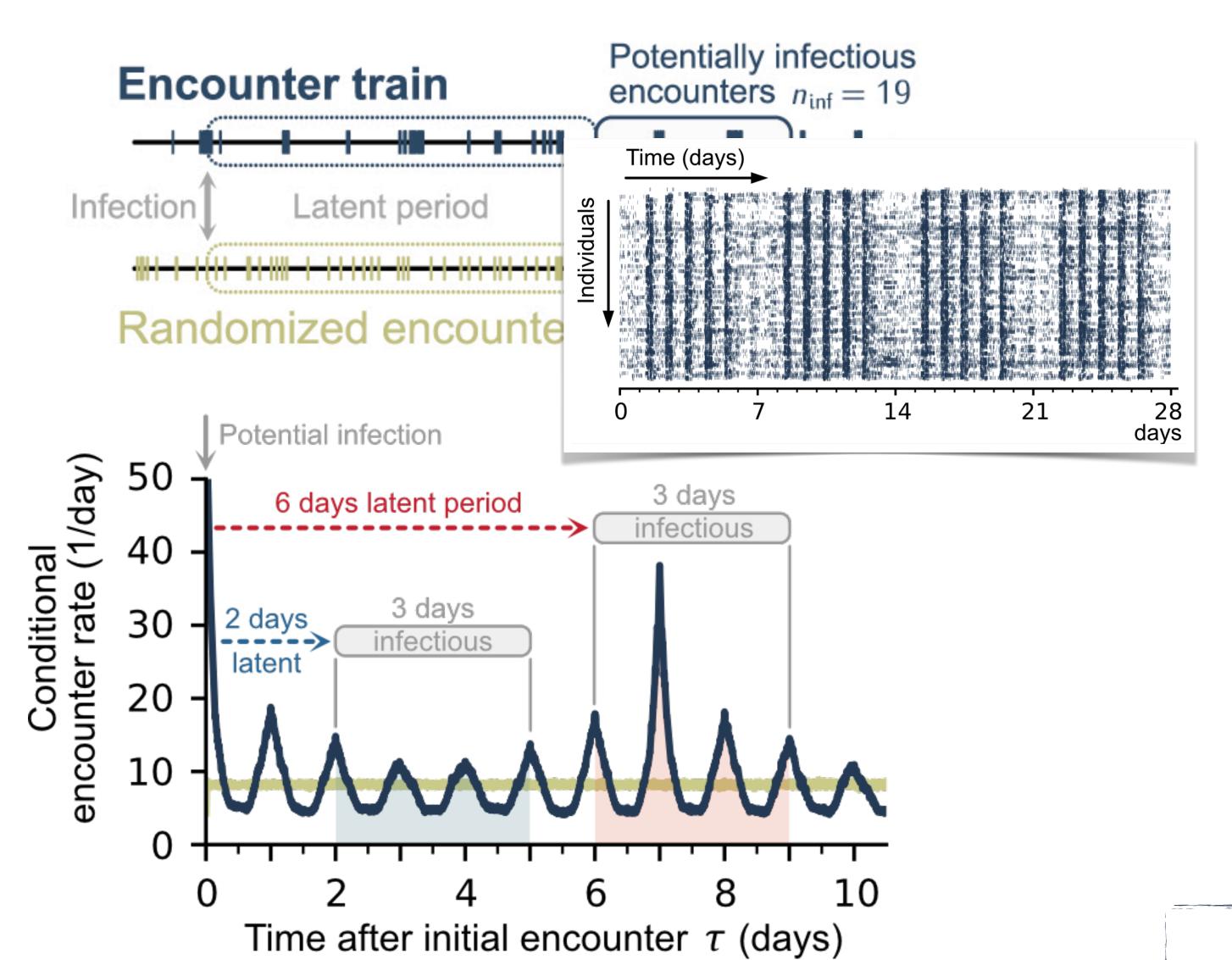


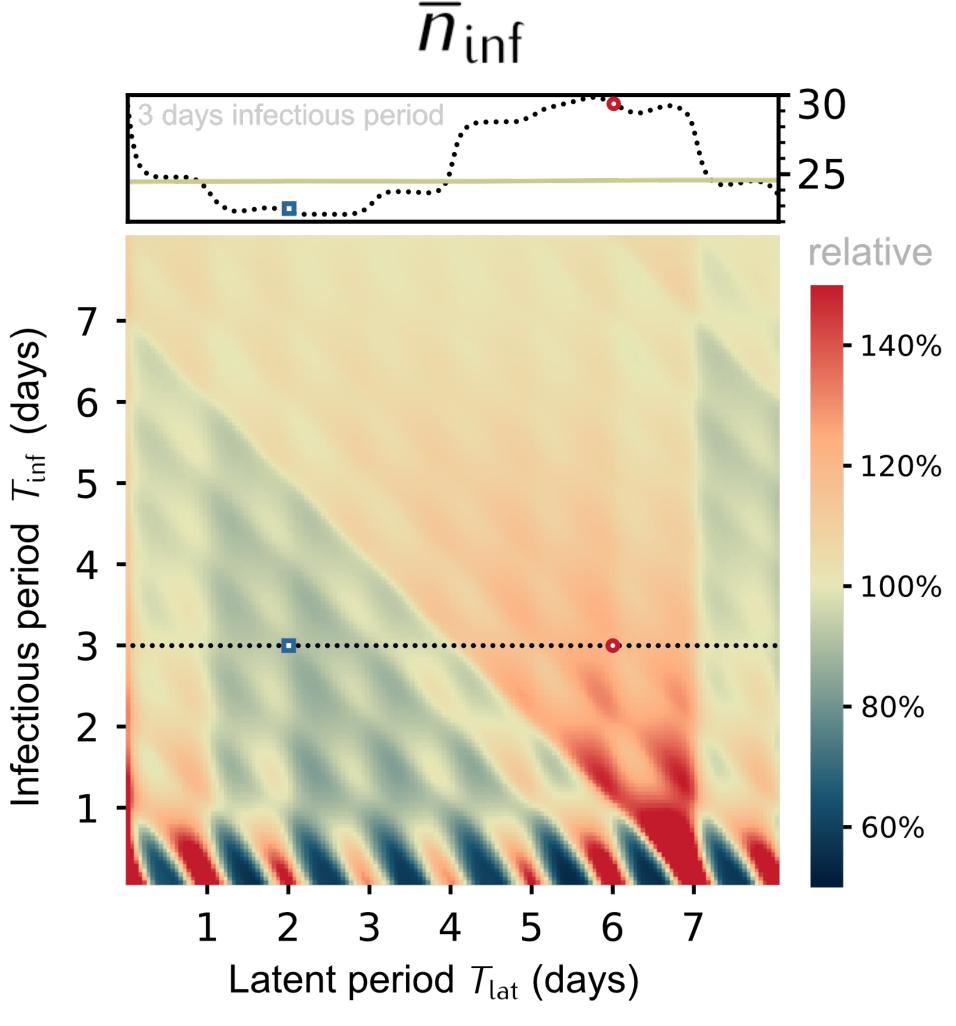
Modulation from human contact patterns





Modulation from human contact patterns





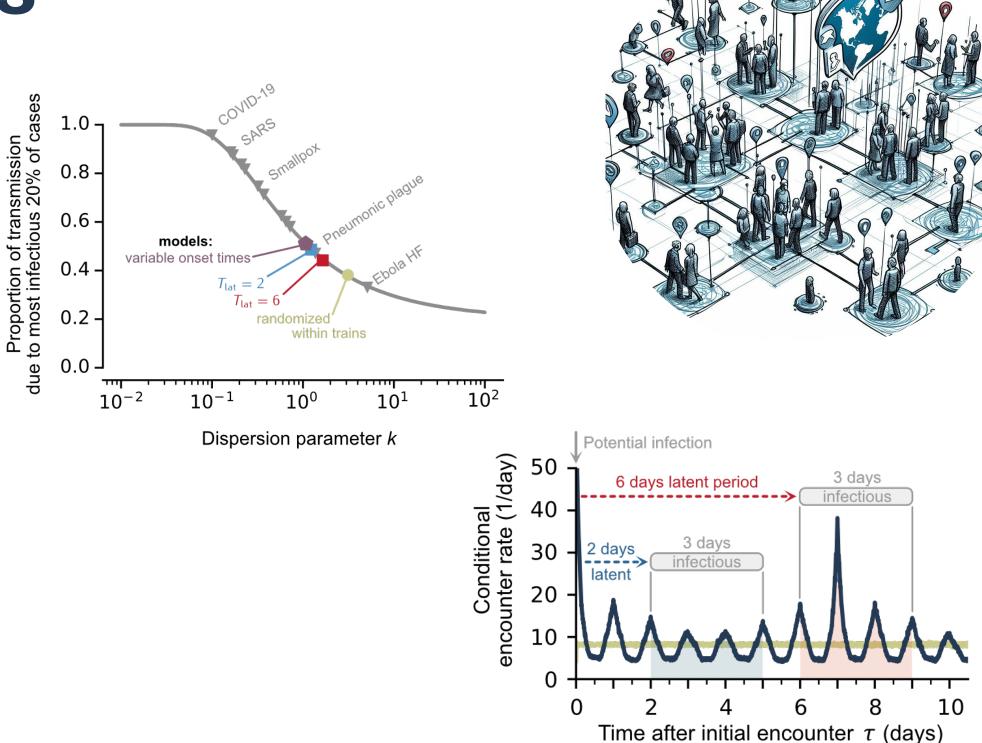
Memory effects in contact patterns can modulate the disease spread (R_0)

Contact patterns and epidemic outbreaks

Statistical analysis of disease spread from human contact patterns

Contact patterns affect the dispersion k and disease spread $R_{\rm 0}$

Epidemic phase transition in a toy model of spatiotemporal contacts

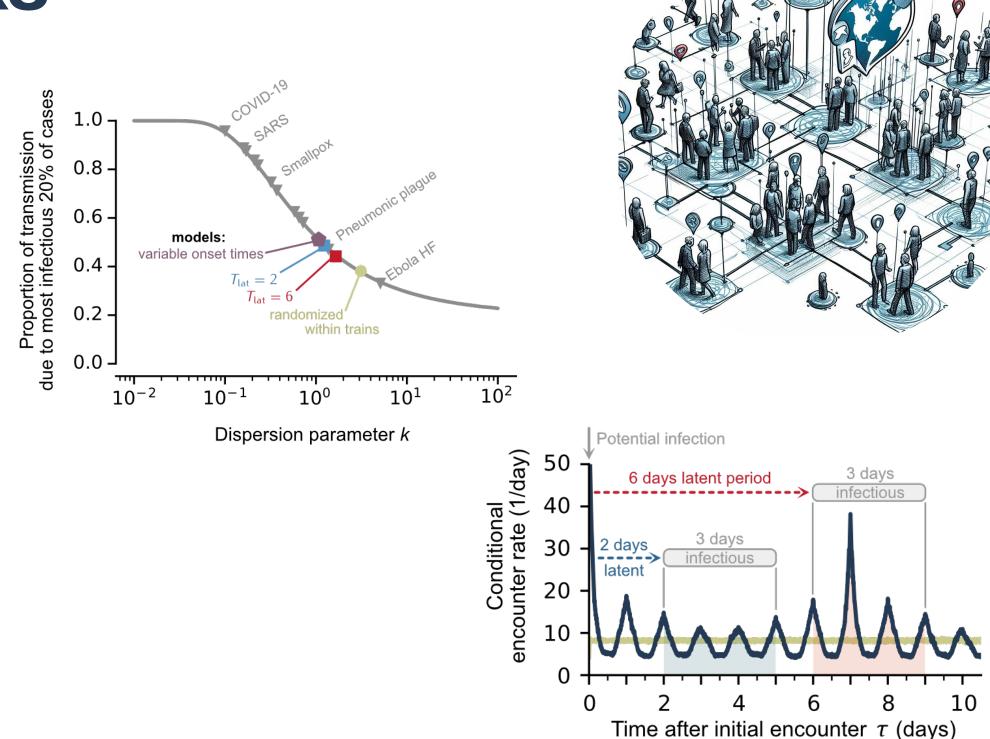


Contact patterns and epidemic outbreaks

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Random walkers with stochastic resetting

Darlo marone de la compansión de la comp

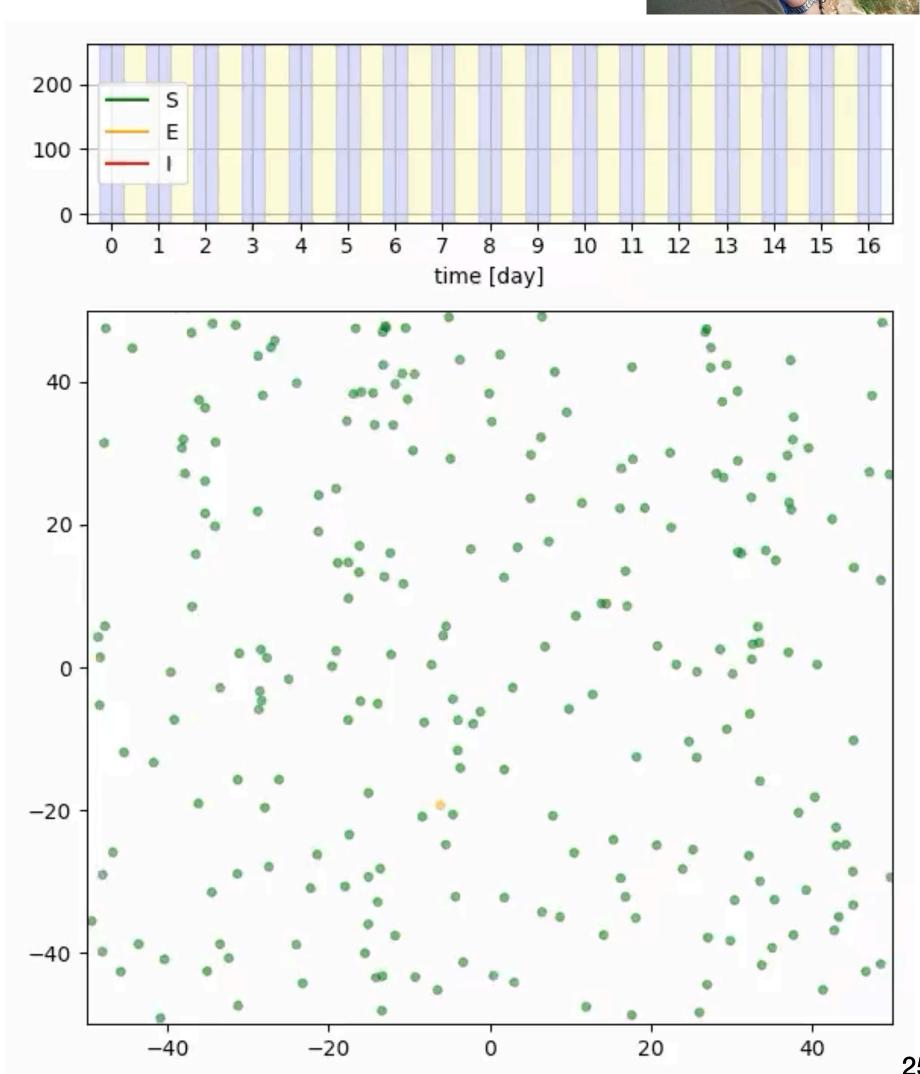
During day: Diffusive motion

During night: reset to individual home location

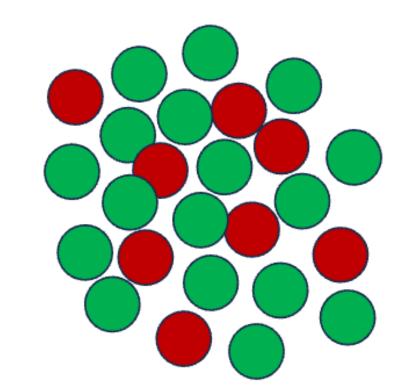
Overdamped Langevin dynamics:

$$\dot{\mathbf{r}}_i = -F(t) \nabla V \left(\mathbf{r}_i - \mathbf{r}_i^0 \right) + \sqrt{2D} \eta_i$$

Non-Markovian Infection dynamics: Upon infection agent is exposed for $\tau_{\rm lat}$ and infectious for $\tau_{\rm inf}$, where they infect only upon close contact.

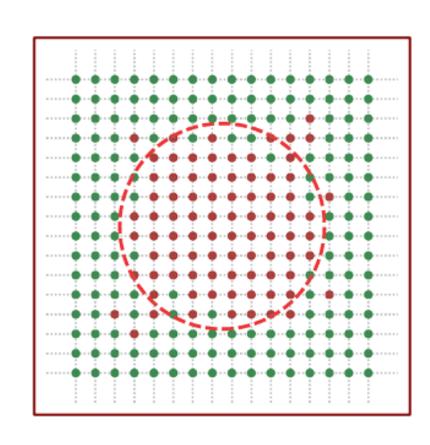


Mean-field model



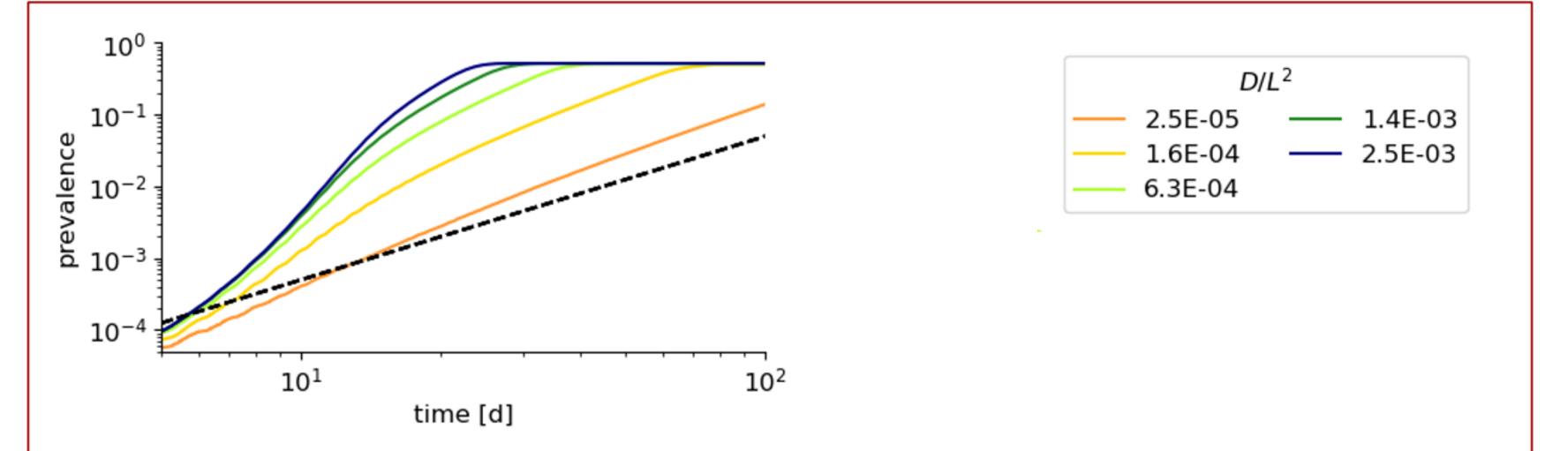
 $\dot{\mathbf{r}}_i = -F(t) \nabla V \left(\mathbf{r}_i - \mathbf{r}_i^0 \right) + \sqrt{2D} \boldsymbol{\eta}_i$

2D lattice



Barone Dario





Cycloactive walkers interpolate between mean-field and spatial dynamics with super-quadratic growth

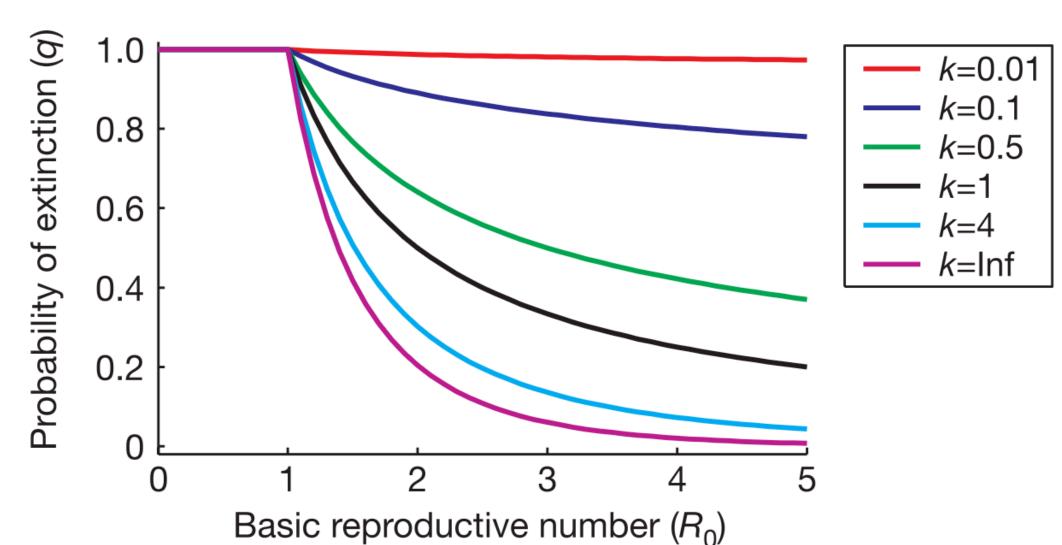
Instantaneous log slope:

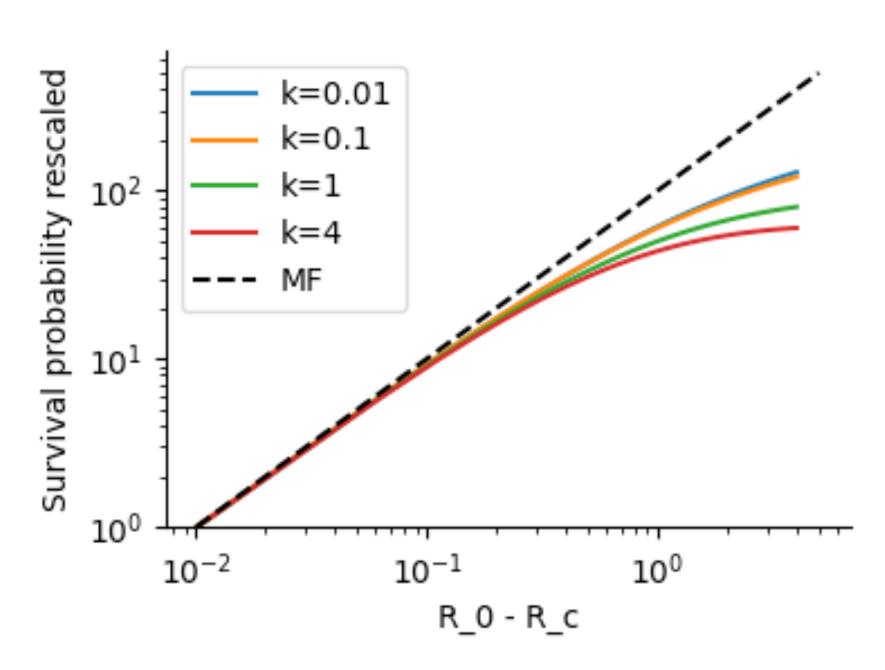
$$\alpha = \frac{d \log I}{d \log t} = \frac{t \, dI}{I \, dt}$$

$$\alpha(t) = kt \to I(t) \propto e^{kt}$$

$$\alpha(t) = k \to I(t) \propto t^k$$

Random walkers with stochastic resetting - scaling



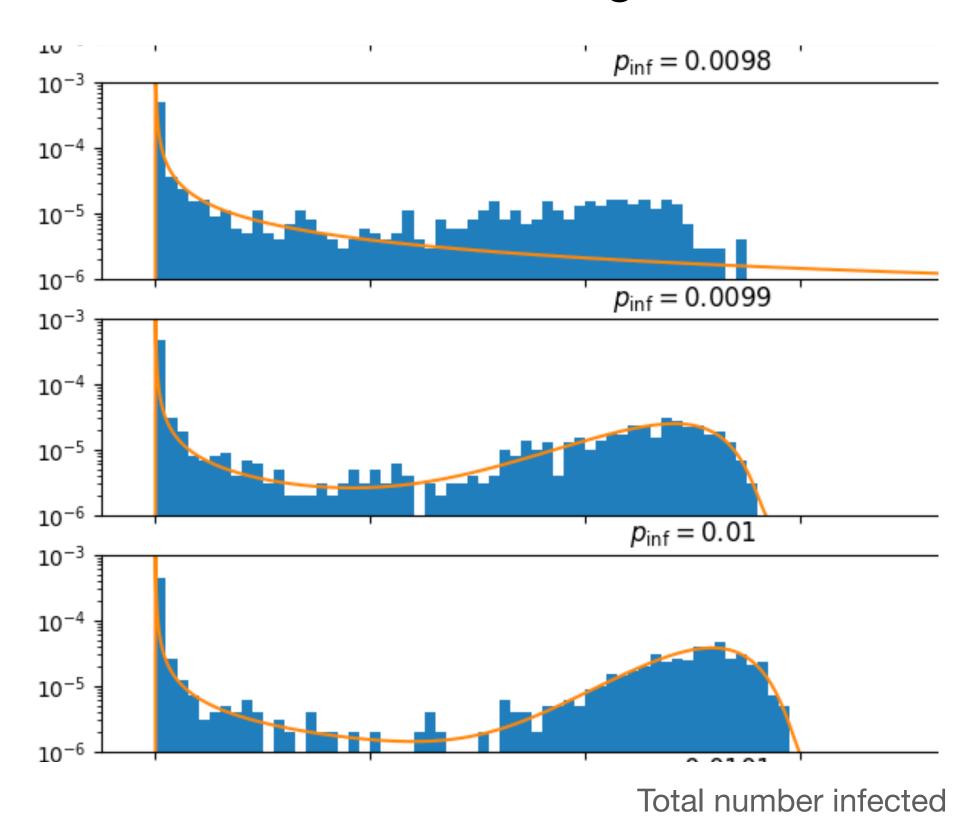


Expected scaling of the survival probability:

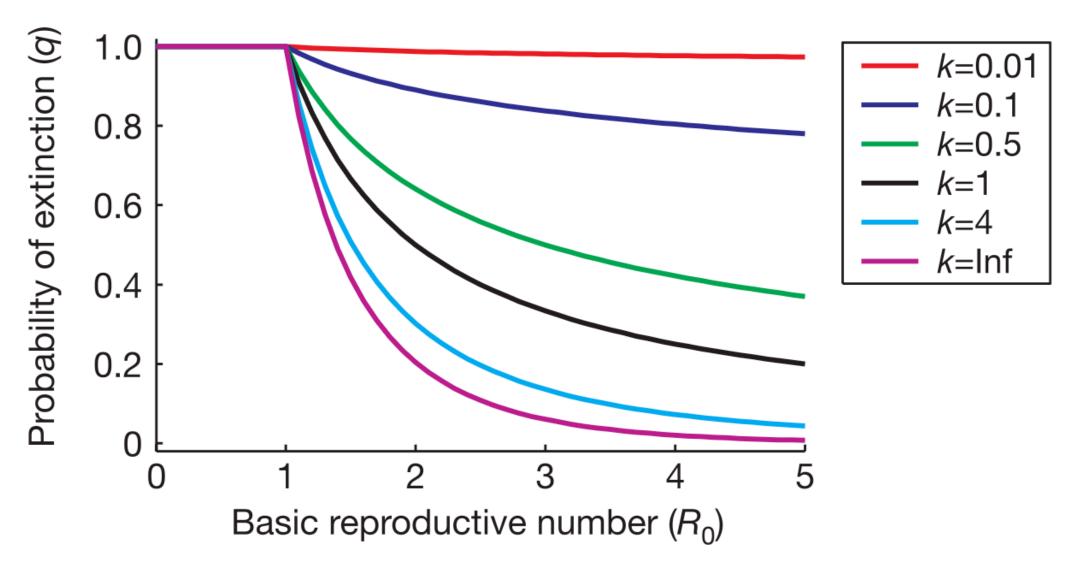
$$1 - p_{\text{ext}} = p_{\text{surv}} \sim (p_{\text{inf}} - p_c)^{\beta'}$$

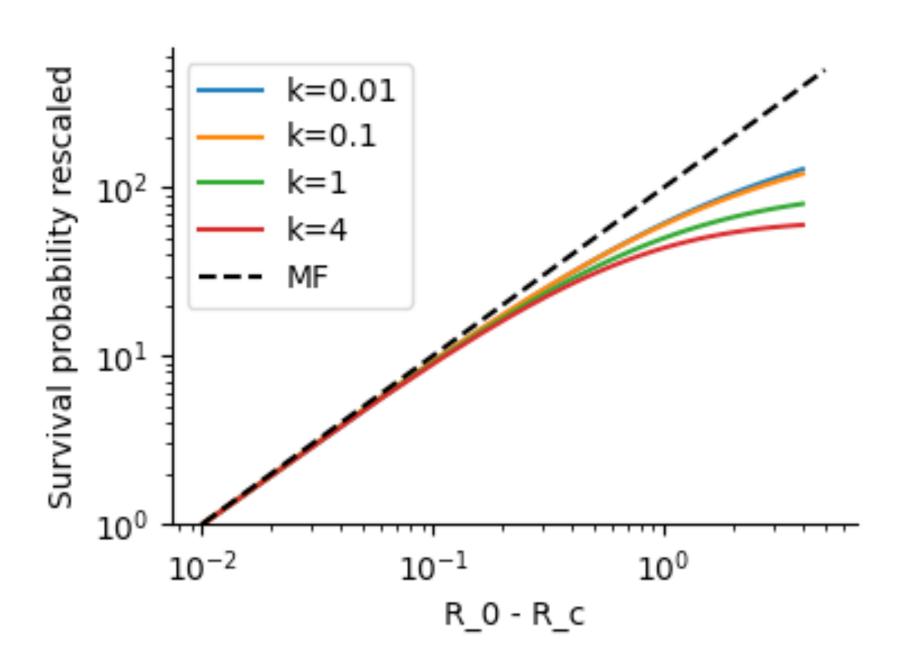


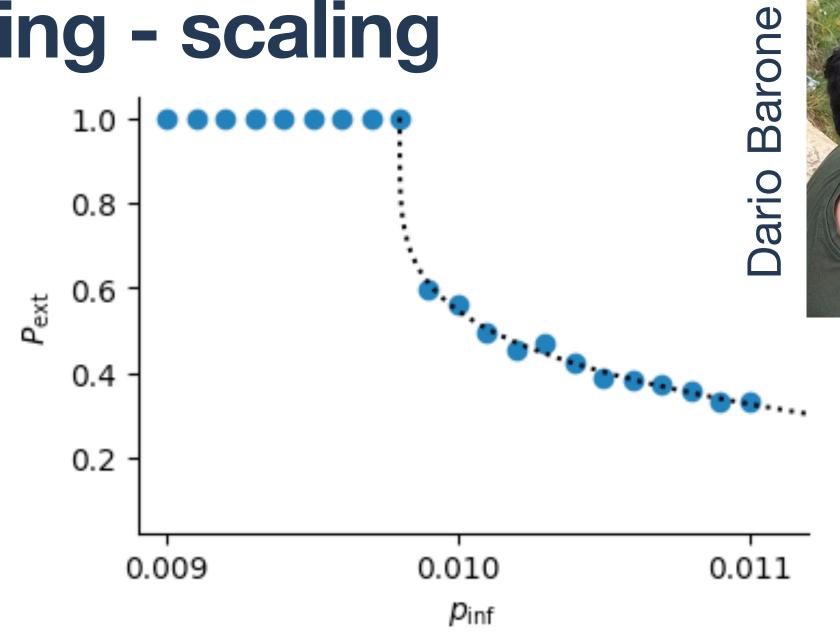
Focus on SEIR and distinguish which runs survive

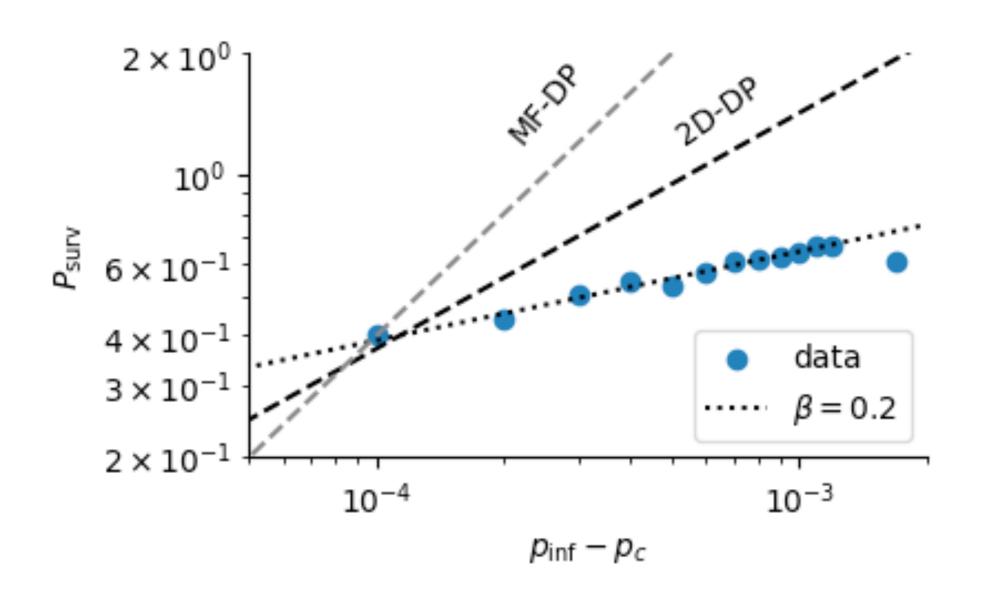


Random walkers with stochastic resetting - scaling









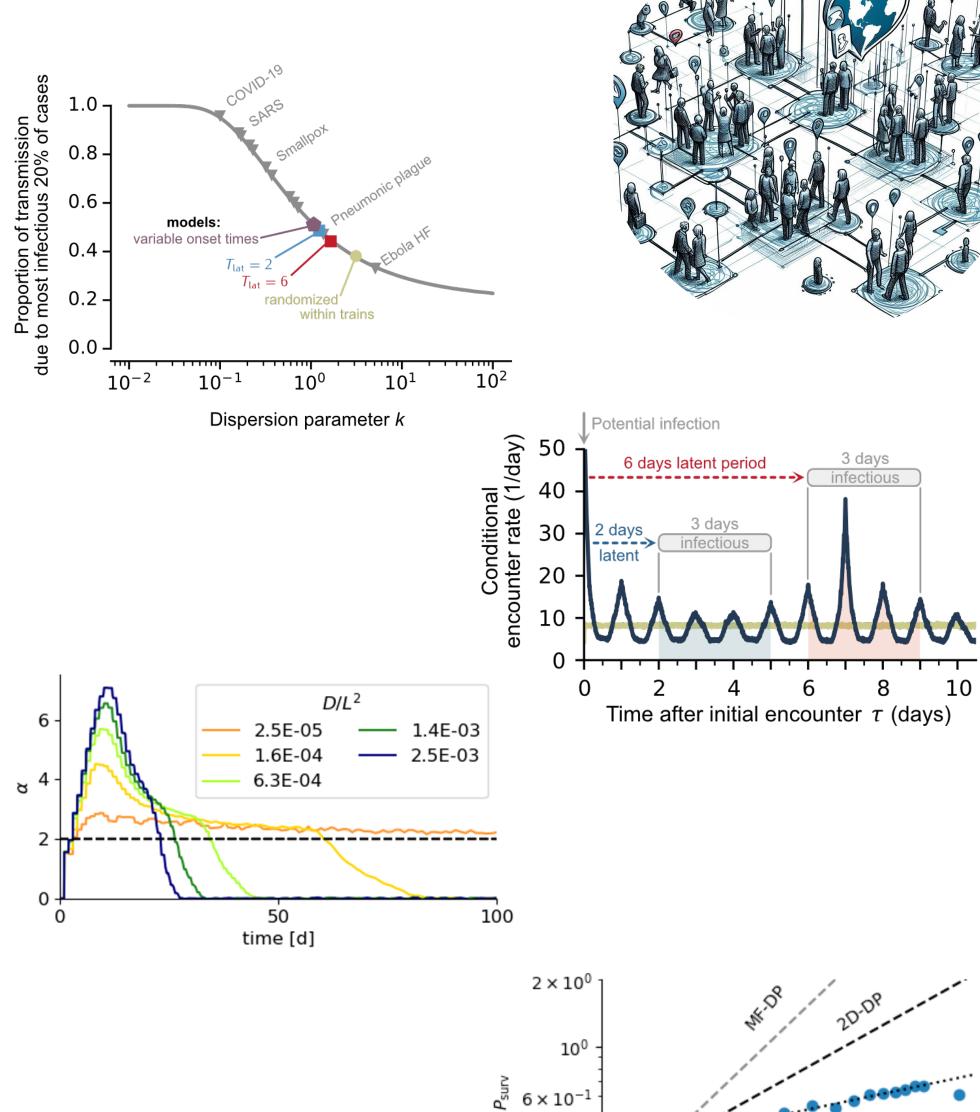
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Epidemic phase transition in a toy model of spatiotemporal contacts

Toy model interpolates between meanfield and spatial dynamics with non-trivial critical exponents.



 4×10^{-1}

 3×10^{-1}

 $2 \times 10^{-}$

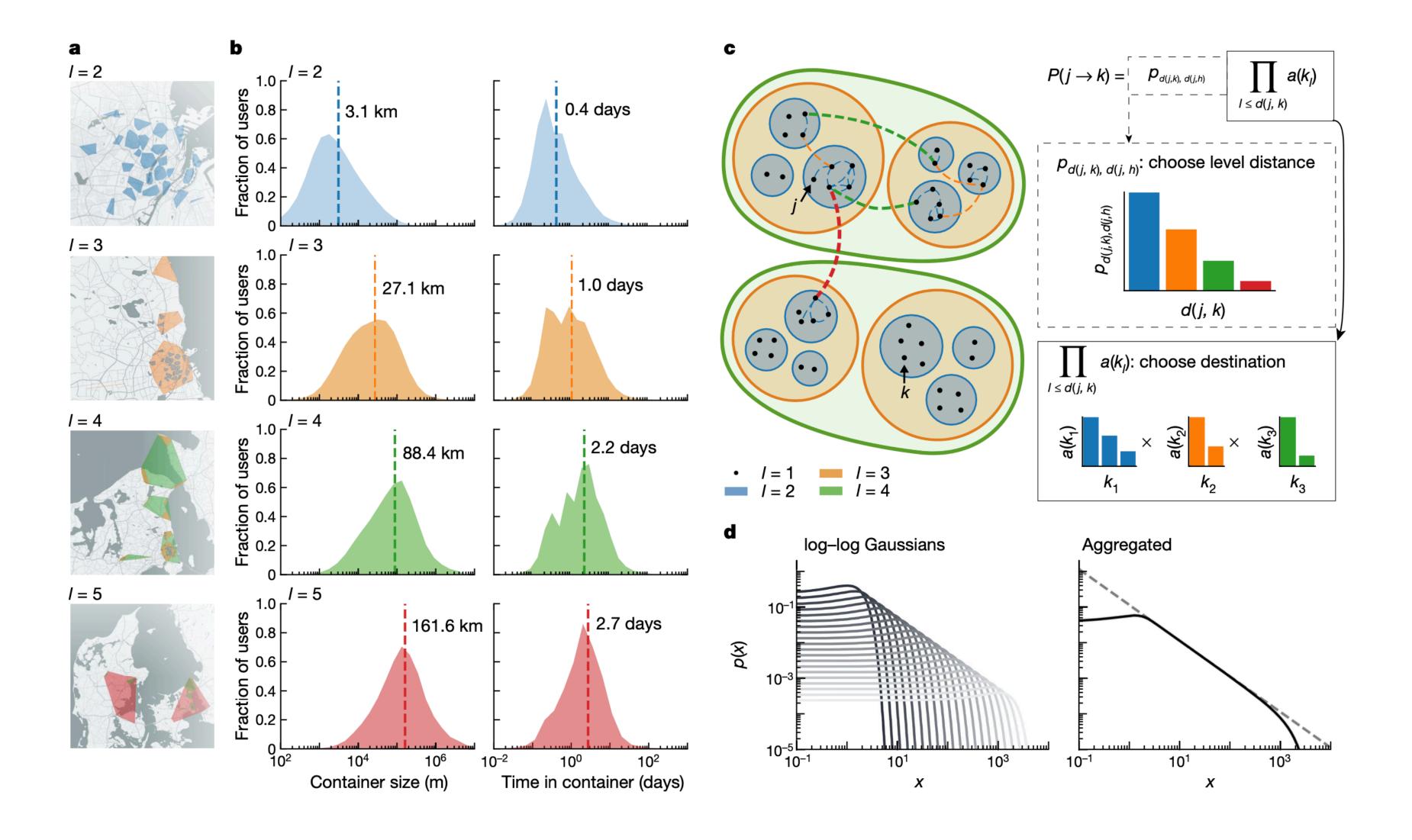
 10^{-4}

 $\beta = 0.2$

 10^{-3}

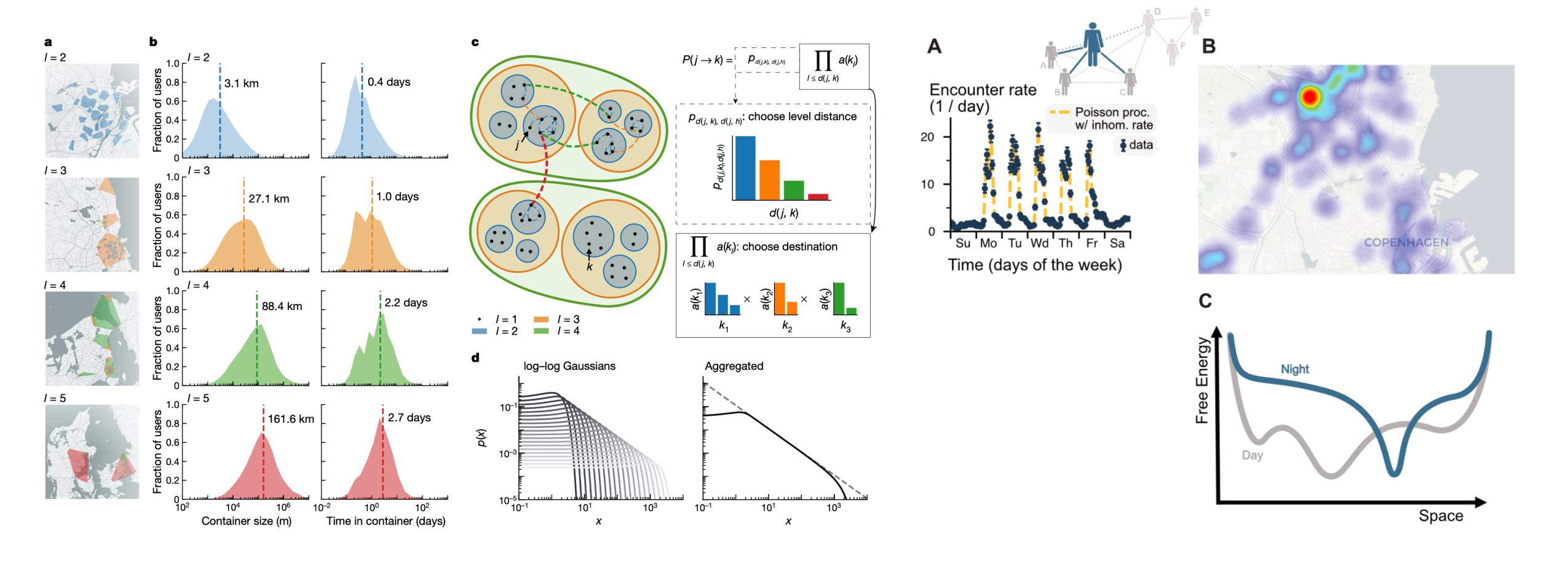
 $p_{inf} - p_c$

Outlook: Hierarchical Mobility



Alessandretti et al., Nature (2020)

Outlook: Hierarchical Mobility



My Future: Cycloactive agents with individual, hierarchical, discrete interaction sites inspired by data

Alessandretti et al., Nature (2020)

Acknowledgements

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Viola Priesemann (MPIDS)

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Seba Contreras (MPIDS)

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Joacim Rocklöv (Heidelberg)

Dario Barone (Padova)

Chiara Poletto (Padova)

Sune Lehmann (DTU)



Funding:





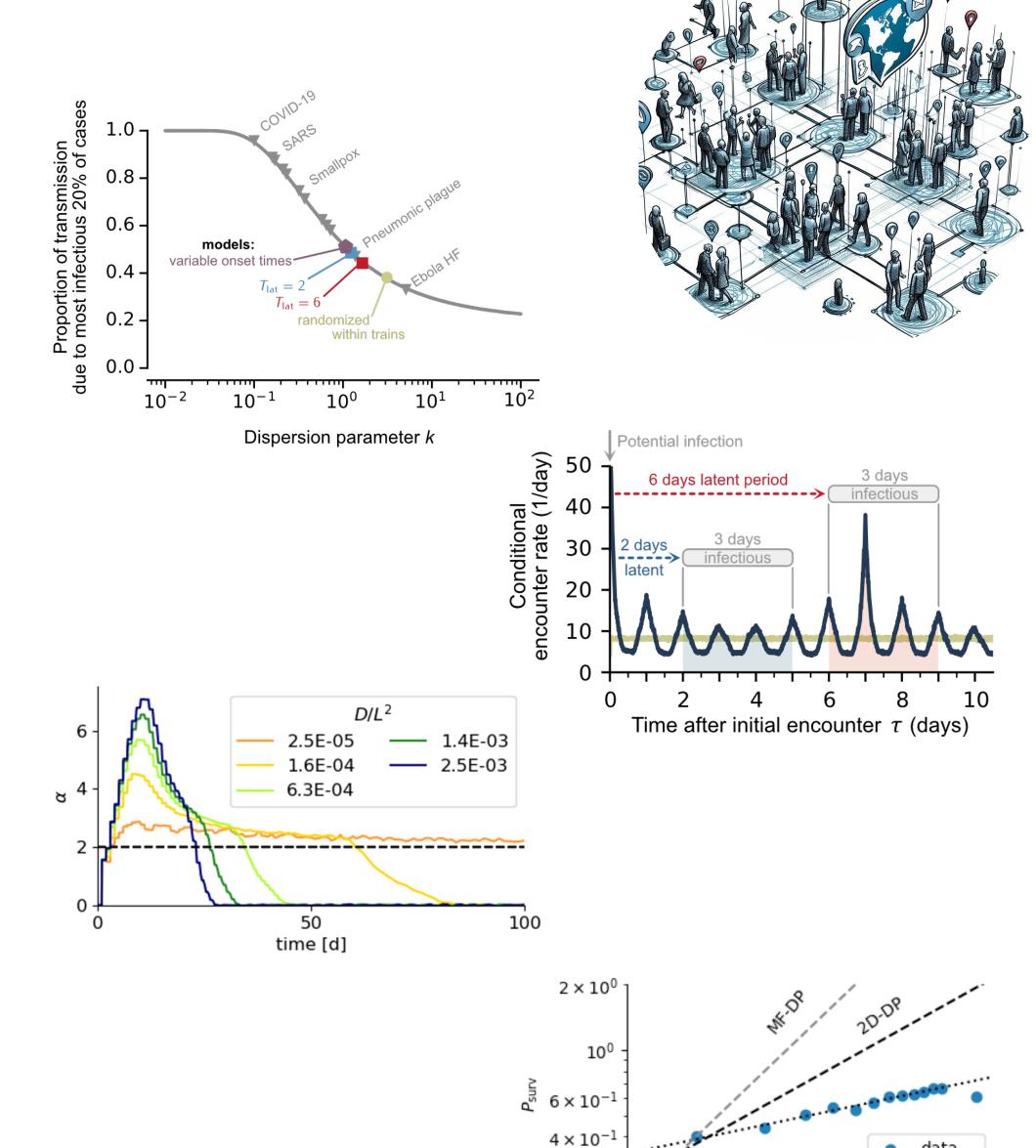
Summary

Statistical analysis of disease spread from human contact patterns

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 $p_{inf} - p_c$