SIAM DS 2021

Heterogeneous exposure on higher-order networks leads to nonlinear infection kernels

Guillaume St-Onge, Hanlin Sun, Antoine Allard,
Laurent Hébert-Dufresne & Ginestra Bianconi

2021/05/23

Département de physique, de génie physique, et d'optique
Université Laval, Québec, Canada

Université Laval

CIMMUL
Centre Interdisciplinaire en Modélisation Mathématique de l'Université Laval

Standard epidemiolocial models predict exponential growth

For a whole population, with I the fraction of infectious,

$$\frac{\mathrm{d}I}{\mathrm{d}t} \approx \lambda\, I \qquad\qquad (I \ll 1)$$

$$\implies I \propto e^{\lambda t}$$

Standard epidemiolocial models predict exponential growth

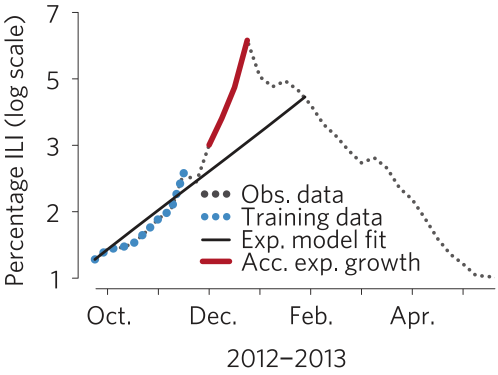
For a whole population, with I the fraction of infectious,

$$\frac{\mathrm{d}I}{\mathrm{d}t} \approx \lambda I \qquad (I \ll 1)$$
$$\implies I \propto e^{\lambda t}$$

But this is because we assume that the risk of infection is linear

$$\theta(I) \propto I$$

Superexponential spread of Influenza-Like-Illness [1]



1. Scarpino, S. V., Allard, A., & Hébert-Dufresne, L. (2016). The effect of a prudent adaptive behaviour on disease transmission. Nature Physics, 12(11), 1042-1046.

$$\theta(I) \propto I$$

(i) Why assume linearity ?

(ii) When is linearity valid ?

(iii) What other forms could it take ?

$$\theta(I) \propto I$$

(i) Why assume linearity ?

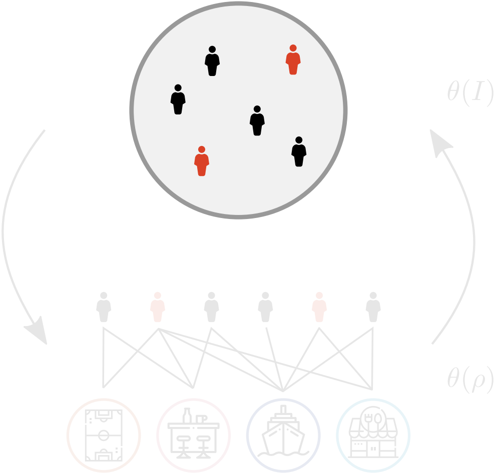
(ii) When is linearity valid ?

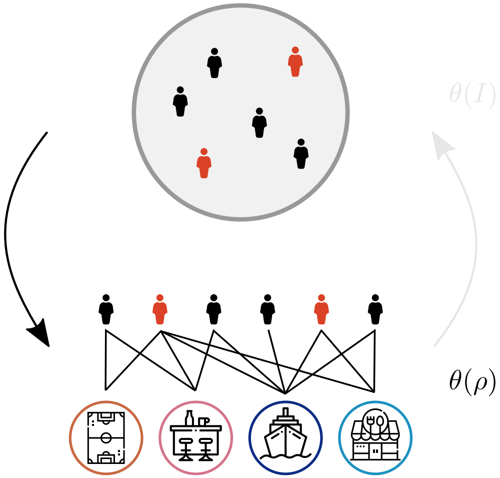
(iii) What other forms could it take ?

Take-home message

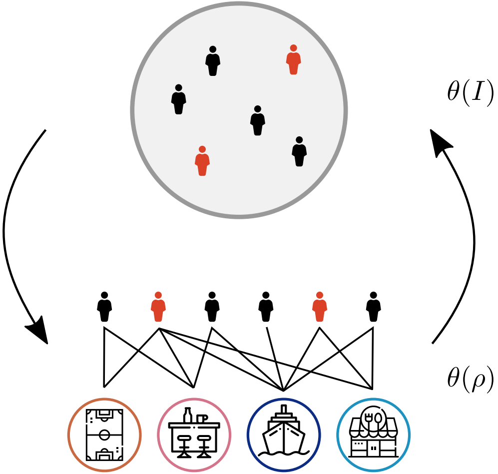
(iii) : For not too small I and heterogeneous exposure, we should consider

$$\theta(I) \propto I^{\nu} \quad \text{with } \nu \in \mathbb{R}^+$$

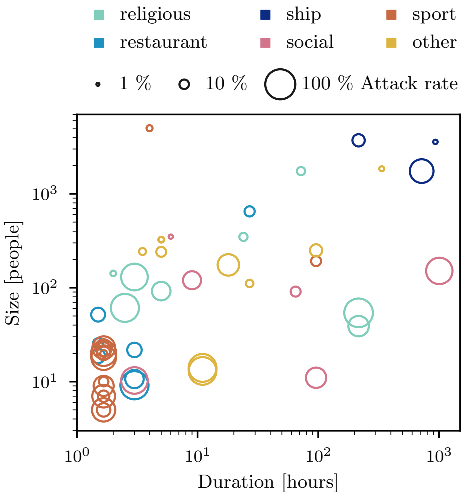

$\theta(I)$
$\theta(\rho)$



$\theta(I)$
$\theta(\rho)$


$\theta(I)$
$\theta(\rho)$

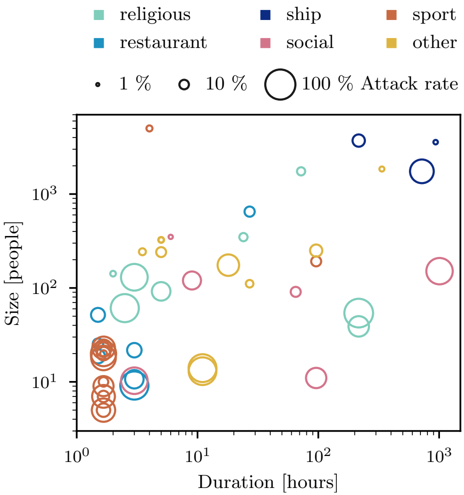
Framework for contagion at the level of environments



Model properties

1. Explicit group interactions in *environments*
2. Heterogeneous temporal patterns
   - Participation time τ

$$P(\tau) \propto \tau^{-\alpha-1}$$



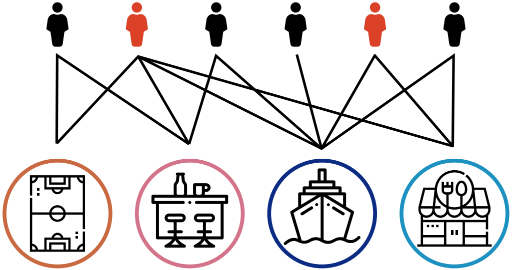
Model properties

1. Explicit group interactions in *environments*
2. Heterogeneous temporal patterns
   - Participation time τ

$$P(\tau) \propto \tau^{-\alpha-1}$$

3. *Minimal infective dose*

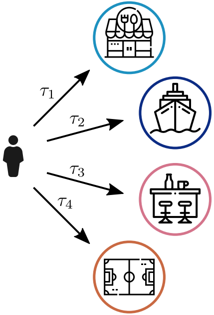
Property # 1 : Explicit group interactions – bipartite structure



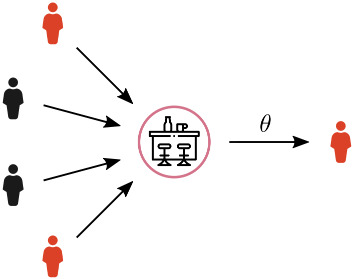
Icons made by Freepik from www.flaticon.com

Property # 2 : heterogeneous temporal patterns – discrete-time contagion

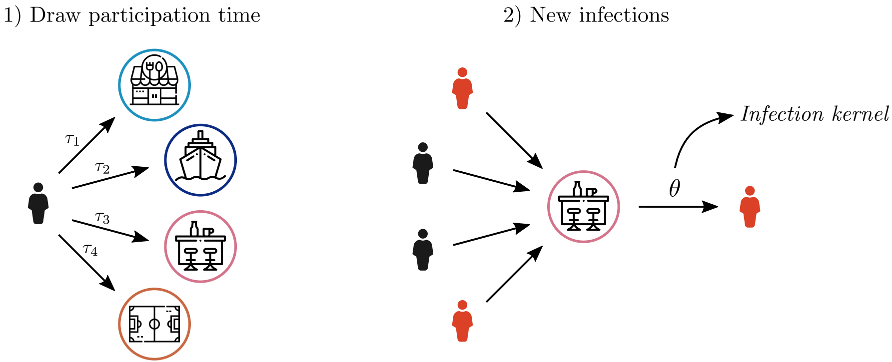
1) Draw participation time



2) New infections



Property # 2 : heterogeneous temporal patterns – discrete-time contagion



θ : probability of infection (per environment) during one time step

Property #3 : minimal infective dose

- Our immune system is able to fight mild challenges
- A certain minimal dose of virus or bacteria is required to trigger an infection

Property #3 : minimal infective dose

- Our immune system is able to fight mild challenges
- A certain minimal dose of virus or bacteria is required to trigger an infection
- *Threshold models*

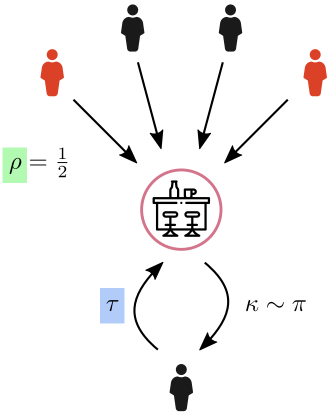
VOLUME 92, NUMBER 21 PHYSICAL REVIEW LETTERS week ending 28 MAY 2004

Universal Behavior in a Generalized Model of Contagion

Peter Sheridan Dodds[1,*] and Duncan J. Watts[2,3,†]

Infection through dose accumulation

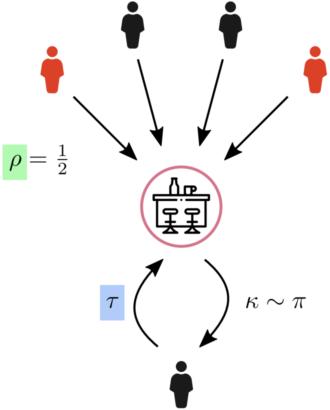
- The fraction of infectious participants is ρ
- Individual receives a dose $\kappa \sim \pi(\kappa; \rho, \tau)$



Infection through dose accumulation

- The fraction of infectious participants is ρ
- Individual receives a dose $\kappa \sim \pi(\kappa; \rho, \tau)$
- The mean dose received is

$$\langle \kappa \rangle \propto \rho\tau$$



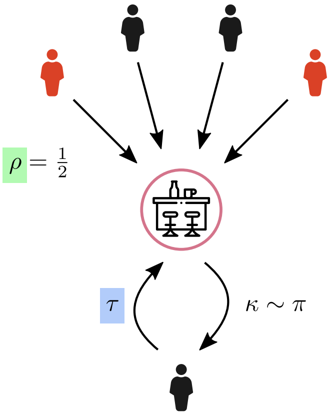
Infection through dose accumulation

- The fraction of infectious participants is ρ
- Individual receives a dose $\kappa \sim \pi(\kappa; \rho, \tau)$
- The mean dose received is

$$\langle \kappa \rangle \propto \rho\tau$$

- An infection is triggered if $\kappa \geq K$, with probability

$$\bar{\Pi}(K; \rho, \tau) = \int_K^\infty \pi(\kappa; \rho, \tau) \mathrm{d}\kappa$$
$$\neq \theta$$



Universal nonlinear infection kernel

The infection kernel is

$$\theta(\rho) = \int P(\tau)\bar{\Pi}(K; \rho, \tau)\mathrm{d}\tau \ .$$

The infection kernel is

$$\theta(\rho) = \int P(\tau)\bar{\Pi}(K;\rho,\tau)\mathrm{d}\tau \ .$$

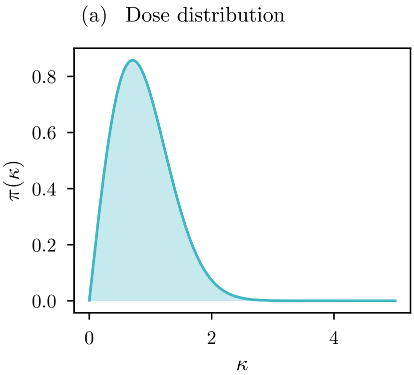
Assuming :

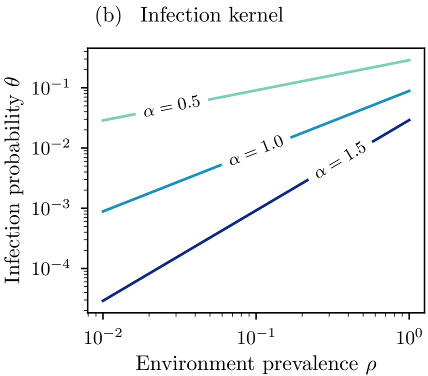
1. $P(\tau) \propto \tau^{-\alpha-1}$;
2. *Some technical conditions for the asymptotic analysis ;*

for a large class of dose distribution π, we recover the *universal* infection kernel

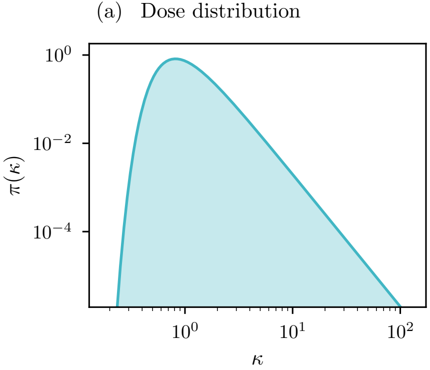
$$\boxed{\theta(\rho) \propto \rho^{\alpha}}$$

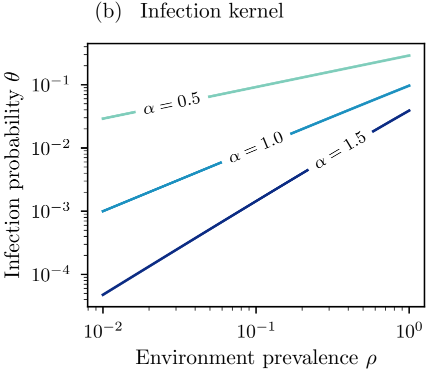
Weibull dose distribution





Fréchet dose distribution





When is linearity valid at the *level of environments*?

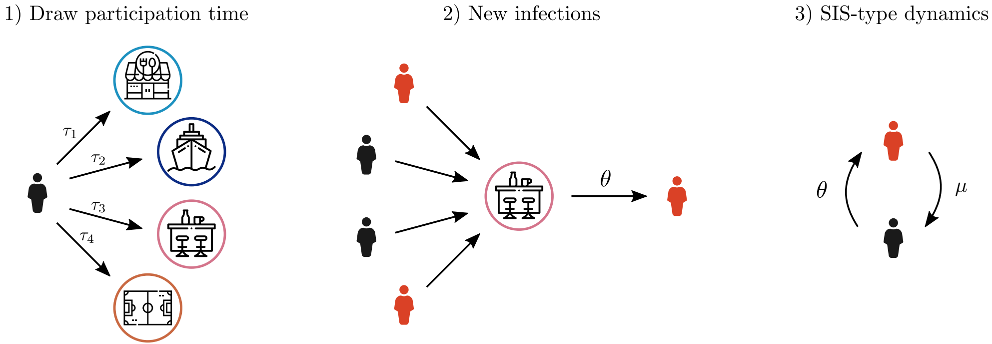
- $\alpha = 1 \quad [P(\tau) \propto \tau^{-\alpha-1}]$
- π is a Poisson distribution and $K = 1$
- Some other limit cases

When is linearity valid at the *level of environments*?

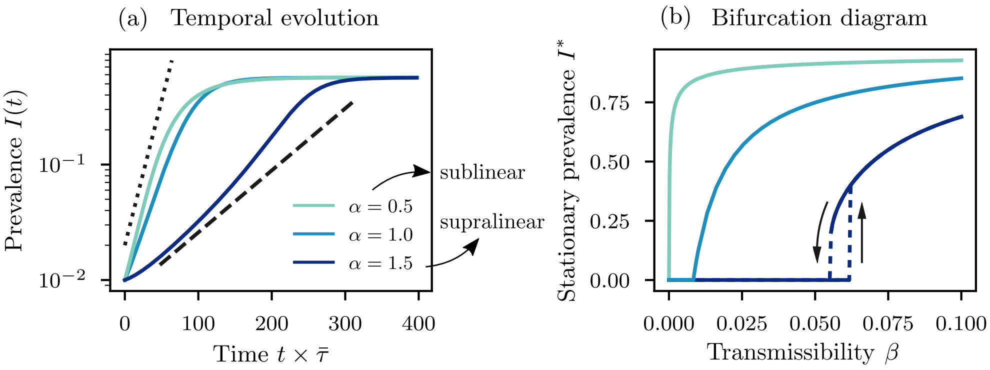
- $\alpha = 1 \quad [P(\tau) \propto \tau^{-\alpha-1}]$
- π is a Poisson distribution and $K = 1$
- Some other limit cases

LINEAR INFECTION KERNELS ARE THE EXCEPTION RATHER THAN THE NORM

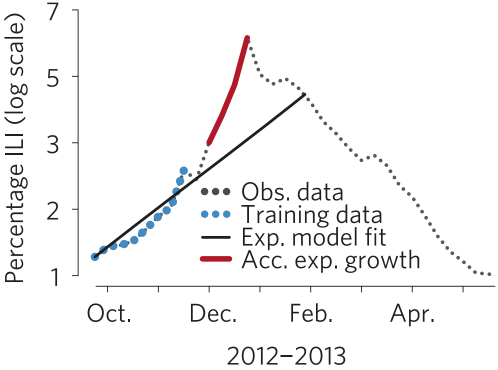
Consequences of nonlinear infection kernel



Superexponential spread and discontinuous phase transition



Superexponential spread of Influenza-Like-Illness [2]



2. Scarpino, S. V., Allard, A., & Hébert-Dufresne, L. (2016). The effect of a prudent adaptive behaviour on disease transmission. Nature Physics, 12(11), 1042-1046.

Why assume linearity for the risk of infection ?

Why assume linearity for the risk of infection ?

MAYBE WE SHOULDN'T, maybe we should adopt more general forms.

Why assume linearity for the risk of infection?

MAYBE WE SHOULDN'T, maybe we should adopt more general forms.

At the level of *environments*, we found

$$\theta(\rho) \propto \rho^{\nu} \quad \text{with } \nu \in \mathbb{R}^{+}$$

Why assume linearity for the risk of infection ?

MAYBE WE SHOULDN'T, maybe we should adopt more general forms.

At the level of *environments*, we found

$$\theta(\rho) \propto \rho^{\nu} \quad \text{with } \nu \in \mathbb{R}^{+}$$

If we coarse-grain at the level of a *whole population*,

$$\theta(I) \propto \begin{cases} I & \text{if } I \ll 1 \\ I^{\nu} & \text{otherwise} \end{cases}$$

For a standard SIR model, this could look like

$$\frac{\mathrm{d}I}{\mathrm{d}t} \approx \beta(1 - I)\,\theta(I) - \mu I \ ,$$

Aknowledgments

Thanks to my collaborators

Hanlin Sun, Antoine Allard, Laurent Hébert-Dufresne & Ginestra Bianconi

Preprint

arXiv:2101.07229

Funding and computational resources

Appendix

Mathematical description for $N \to \infty$

We track $\rho_k(t)$ the fraction of infected nodes of membership k using

$$\rho_k(t+1) = (1-\mu)\rho_k(t) + (1-\rho_k(t))\Theta_k \ ,$$

where

$$\Theta_k(\bar{\rho}) = 1 - [1-\bar{\theta}(\bar{\rho})]^k \ , \quad \bar{\rho}(t) = \sum_k \rho_k(t)\frac{k\tilde{P}(k)}{\langle k\rangle} \ , \quad \bar{\theta}(\bar{\rho}) = \sum_m \bar{\theta}_m(\bar{\rho})\frac{m\hat{P}(m)}{\langle m\rangle} \ ,$$

and

$$\bar{\theta}_m(\bar{\rho}) = \sum_{i=0}^{m-1} \binom{m-1}{i} \bar{\rho}^i (1-\bar{\rho})^{m-1-i} \, \theta_m\left(\frac{i}{m-1}\right) \ .$$