NERCCS2021

BURSTY EXPOSURE ON HIGHER-ORDER NETWORKS LEADS TO NONLINEAR INFECTION KERNELS

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

2021/04/01

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





Centre Interdisciplinaire en Modélisation Mathématique de l'Université Laval



Biological contagion modeling

Standard epidemiolocial models predict exponential growth

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

Biological contagion modeling

Standard epidemiolocial models predict exponential growth

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

This is because we assume that the risk of infection is linear

 $heta \propto I$

Superexponential spread of Influenza-Like-Illness¹



^{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.

$\theta \propto I$

- (i) Why assume linearity?
- (ii) When is linearity valid?
- (iii) What other forms could it take?

$\theta \propto I$

- (i) Why assume linearity?
- (ii) When is linearity valid?
- (iii) What other forms could it take?

Take-home message

(iii): Assuming bursty exposure to infection, we should consider

 $\theta \propto I^{\nu}$ with $\nu \in \mathbb{R}^+$.

Model features

- 1. Explicit group interactions
- 2. Heterogeneous temporal patterns
 - Duration of events τ

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



Model features

- 1. Explicit group interactions
- 2. Heterogeneous temporal patterns
 - Duration of events τ

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

3. Minimal infective dose



Feature # 1 : Explicit group interactions – bipartite structure



Feature # 2 : heterogeneous temporal patterns



Feature #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

Feature #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

VOLUME 92, NUMBER 21 PH	YSICAL 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,†}

Infective dose model

- \bigcirc The fraction of infected individuals is ρ
- Individuals receive a dose $\kappa \sim \pi(\kappa; \rho, \tau)$
- The mean dose received is

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

○ An infection is triggered if $\kappa \ge K$, with probability

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



Bursty exposure

Because of the heavy-tailed distribution $P(\tau) \propto \tau^{-\alpha-1}$



Bursty exposure

Because of the heavy-tailed distribution $P(\tau) \propto \tau^{-\alpha-1}$



The probability of getting infected in an environment

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

The probability of getting infected in an environment

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

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

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

Weibull dose distribution

(a) Dose distribution

(b) Infection kernel



Frechet dose distribution



Asymptotically power-law duration of events distribution





Conditions for the asymptotic analysis partially satisfied



14

When is linearity valid?

- $\bigcirc \alpha = 1$ $(P(\tau) \propto \tau^{-\alpha 1})$
- $\odot~\pi$ is a Poisson distribution and K=1
- \bigcirc Some other limit cases

When is linearity valid?

- $\bigcirc \alpha = 1$ $(P(\tau) \propto \tau^{-\alpha 1})$
- $\odot~\pi$ 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 and discontinuous phase transition



Superexponential spread of Influenza-Like-Illness²



18

^{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, e.g., $\theta(\rho) \propto \rho^{\nu}$ with $\nu \in \mathbb{R}^+$

Why assume linearity for the risk of infection?

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

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

For a standard SIR model, this could look like

$$\frac{\mathrm{d}S}{\mathrm{d}t} \approx -\beta S I^{\nu} \ .$$

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

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

.