Behavioural responses and epidemic spread on networks

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(joint work with David Juher)

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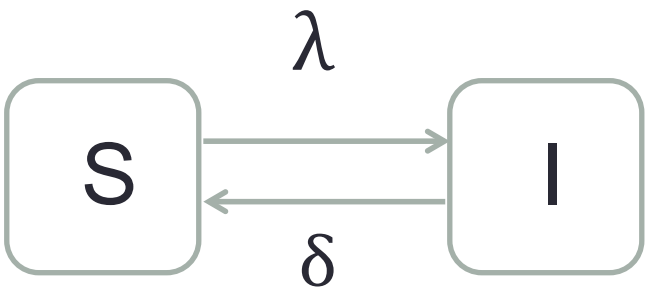
Outline of the talk

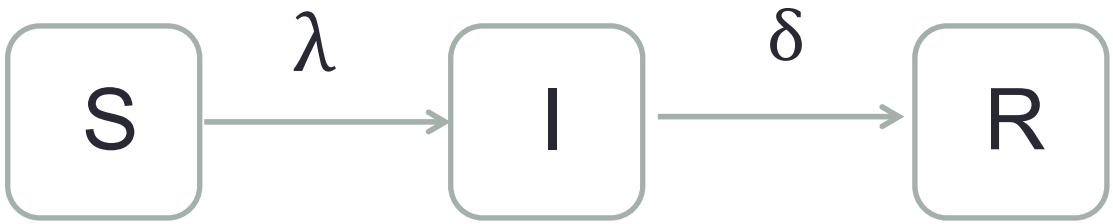
1. Deterministic epidemic models: Basic hypotheses
2. Networks and epidemic models
3. Awareness and epidemics: multilayer networks
4. A toy model for studying the impact of the overlap between layers on epidemics

1. Deterministic epidemic models



- $\lambda =$ rate at which susceptible individuals S get infected (*force of infection*)
  - Proportional to the number of infectious contacts
- $\delta =$ recovery rate

1. Deterministic epidemic models



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Basic hypotheses

- *Homogeneous mixing:*
  - The same *contact rate* c for everybody
  - Uniformly random election of a partner
- Constant *transmission* probability per contact
- Duration of the infectious *period* T ~ *Exp*(δ):

$$\mathrm{E}(T) = 1/\delta$$

An example: SIS model

$$\frac{dI}{dt} = \lambda S - \delta I, \quad S + I = N$$

- λ = rate at which susceptible individuals S get infected (*force of infection*)
  - Proportional to the number of infectious contacts
- δ = recovery rate

Homogeneous SIS model

$$\frac{dI}{dt} = c\beta S\frac{I}{N} - \delta I, \quad S + I = N$$

- $\lambda = \beta$ c I/N → Homogeneous mixing
- β : probability of transmission
- δ = recovery rate

Homogeneous SIS model

$$\frac{di}{dt} = (c\beta s - \delta)i, \quad s + i = 1$$

$$(s = S/N, \;\; i = I/N)$$

$$\left.\frac{di}{dt}\right|_{t=0} > 0 \;\; \text{when} \;\; s(0) = 1 \Leftrightarrow R_0 := \frac{c\beta}{\delta} > 1$$

(epidemic threshold = 1)

Basic reproduction number R_0

- R_0 = Average number of infections produced by a *typical* infectious individual in a totally susceptible population

  $= \beta\, c\, T = \beta\, c\, 1/\delta$ under the MF hypotheses

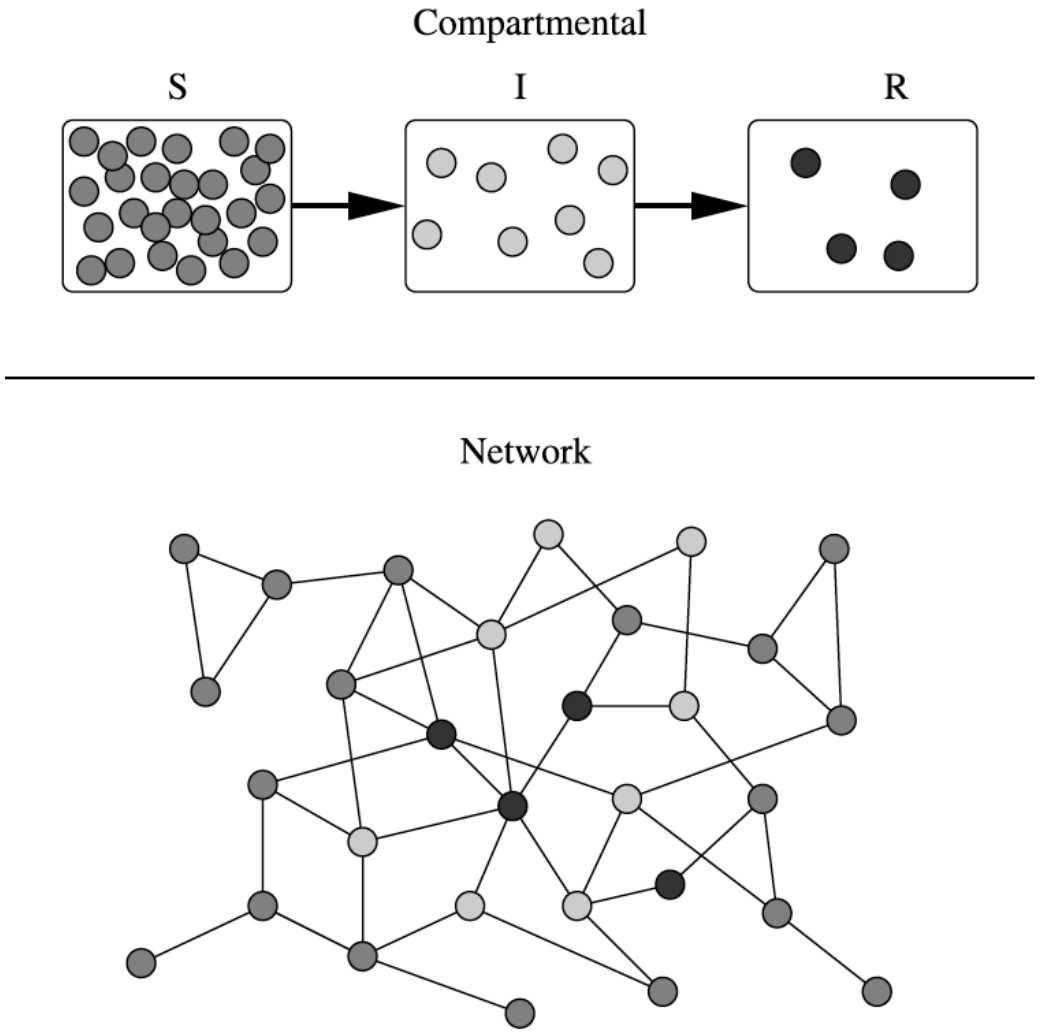
- Deterministic SIS and SIR have the same R_0

- When mixing is *non-homogeneous*,

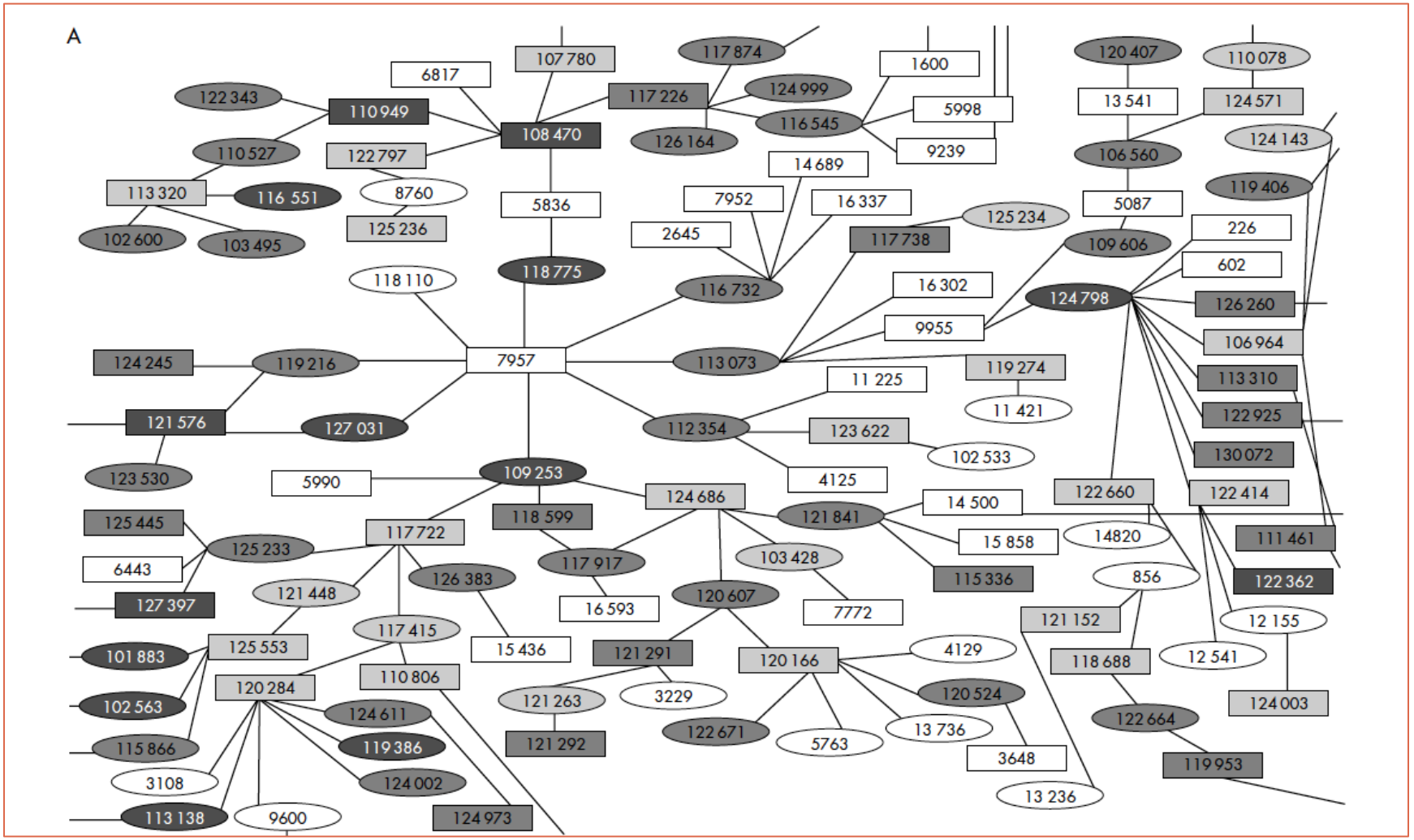
  c ~ *structure* of the *contact pattern*

  → Consider the probability of reaching an infectious individual **through a contact**

2. Networks and epidemic models



A contact network of STDs



(*Sex. Transm. Infect.* 2002)

Gonorrhoea and chlamydia core groups and sexual networks in Manitoba

A M Jolly, J L Wylie

Sex Transm Infect 2002;**78**(Suppl I):i145–i151

This paper summarises the results of the R_0 equation in sexually transmitted infection (STI) repeaters in Manitoba, Canada, in the early 1990s, with both concurrent and more recent descriptions of sexual networks in the same population. The research presented provides empirical network and sex partner data to refine definitions of sexual networks and core groups in phase IV epidemics. New challenges for both practice and research are also discussed.

In an effort to reduce the burden and cost of illness of sexually transmitted infections, legislation, policy guidelines, and programmes have been implemented in the developed world. Syphilis and gonorrhoea have been notifiable diseases in many jurisdictions for decades.[1] Chlamydia was reported in most provinces in Canada by 1991,[2] and in the USA 46 states reported chlamydial infections to the Centers for Disease Control (CDC) by 1992.[3] Guidelines on the diagnosis, treatment, and management of gonorrhoea and chlamydia have been published regularly in the USA and Canada. Screening programmes to detect asymptomatic patients with disease have decrease of infections in the years immediately following.[12]

The basic formula for describing the success of a disease in a population (that is, whether it propagates or dies out), is:

$$R_0 = \beta c D$$

R_0 is the reproductive number of infection and represents the "average number of secondary infections produced when one infected individual is introduced into a host population where everyone is susceptible."[13] If R_0 is greater than 1, such as within the core group, the disease will spread in a population; if it is consistently less than 1, the disease will eventually cease to exist in the population.[14] The term β is the probability of transmission from one infected individual to a susceptible partner; D denotes the duration of infectiousness, and c is the measurement of random sexual contact between infectious individuals and susceptible individuals given by:

$$c = m + \frac{\sigma^2}{m}$$

Deterministic models on networks

- *Node-based models* explicitly include the contacts in the network by means of the *adjacency matrix*
- *Heterogeneous mean-field models* consider a statistical description of the contact pattern in the network (degree distribution, degree-degree correlations, etc.)
  - → ODEs for the number of nodes with the same degree and state
  - → assume the so-called *proportionate* mixing
- *Pairwise models* → time evolution of the number of pairs of disease status: S-S, S-I, I-I, …

Heterogeneous mean-field models

- Approach developed by May and Anderson in the 80s for modelling STDs (and reintroduced in the early 2000s by physics community working on computer viruses)
- *Heterogeneous* means that individuals are not identical but characterized by their number of contacts (degree)
- *Mean field* means that individuals of the same degree behave in the same way and experience the same environment

Heterogeneous mean-field models

- Lead to a good estimate of the epidemic threshold for the SIR model *on networks* but not so good for the SIS model

PHYSICAL REVIEW LETTERS

Thresholds for Epidemic Spreading in Networks

Claudio Castellano[1] and Romualdo Pastor-Satorras[2]

PRL **105,** 218701 (2010)

A heterogeneous mean-field model

- I_k: number of infectious nodes of degree k

$$\frac{dI_k}{dt} = k\beta_k S_k \Theta_I - \delta I_k, \qquad S_k = N - I_k$$

$$\Theta_I := \frac{\sum_k kI_k}{\langle k \rangle N}$$

= fraction of (oriented) links pointing to infectious nodes

= prob. that a randomly chosen link points to an infectious node

Proportionate mixing

R_0 for the heterogeneous SIR/SIS models

Linearizing the system at the DFE for $\beta_k = \beta$, it follows

$$R_0 = \frac{\beta}{\delta}\frac{\langle k^2 \rangle}{\langle k \rangle} = \frac{\beta}{\delta}\left(\langle k \rangle + \frac{\sigma^2}{\langle k \rangle}\right)$$

(May & Anderson 1988; Diekmann & Heesterbeek 2000)
(Pastor-Satorras & Vespignani 2001, Newmann 2002)

The contact rate c in networks

- Susceptible nodes are reached via a randomly selected link ⟶ degree distribution of nodes *reached by following a randomly chosen link*:

$$q_k = \frac{k p_k}{\langle k \rangle} \quad (p_k\text{: degree distribution})$$

- So,

$$c = \bar{q} = \sum_k k q_k = \frac{\langle k^2 \rangle}{\langle k \rangle} = \langle k \rangle \left(1 + \frac{\mathrm{var}(k)}{\langle k \rangle^2} \right)$$

Gonorrhoea and chlamydia core groups and sexual networks in Manitoba

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What is the impact of human behaviour on the progress of an epidemic?

SARS epidemics 2002-2003

What is the impact of human behaviour on the progress of an epidemic?

Awareness *vs* unawareness

What is the impact of human behaviour on the progress of an epidemic?

Awareness *vs* unawareness

3. Awareness and epidemics

OPEN ACCESS Freely available online

PLoS one

Coupled Contagion Dynamics of Fear and Disease: Mathematical and Computational Explorations

Joshua M. Epstein[1,2,3]*, Jon Parker[1], Derek Cummings[4], Ross A. Hammond[1] (2008)

The spread of awareness and its impact on epidemic outbreaks

Sebastian Funk[a,1], Erez Gilad[a], Chris Watkins[b], and Vincent A. A. Jansen[a]

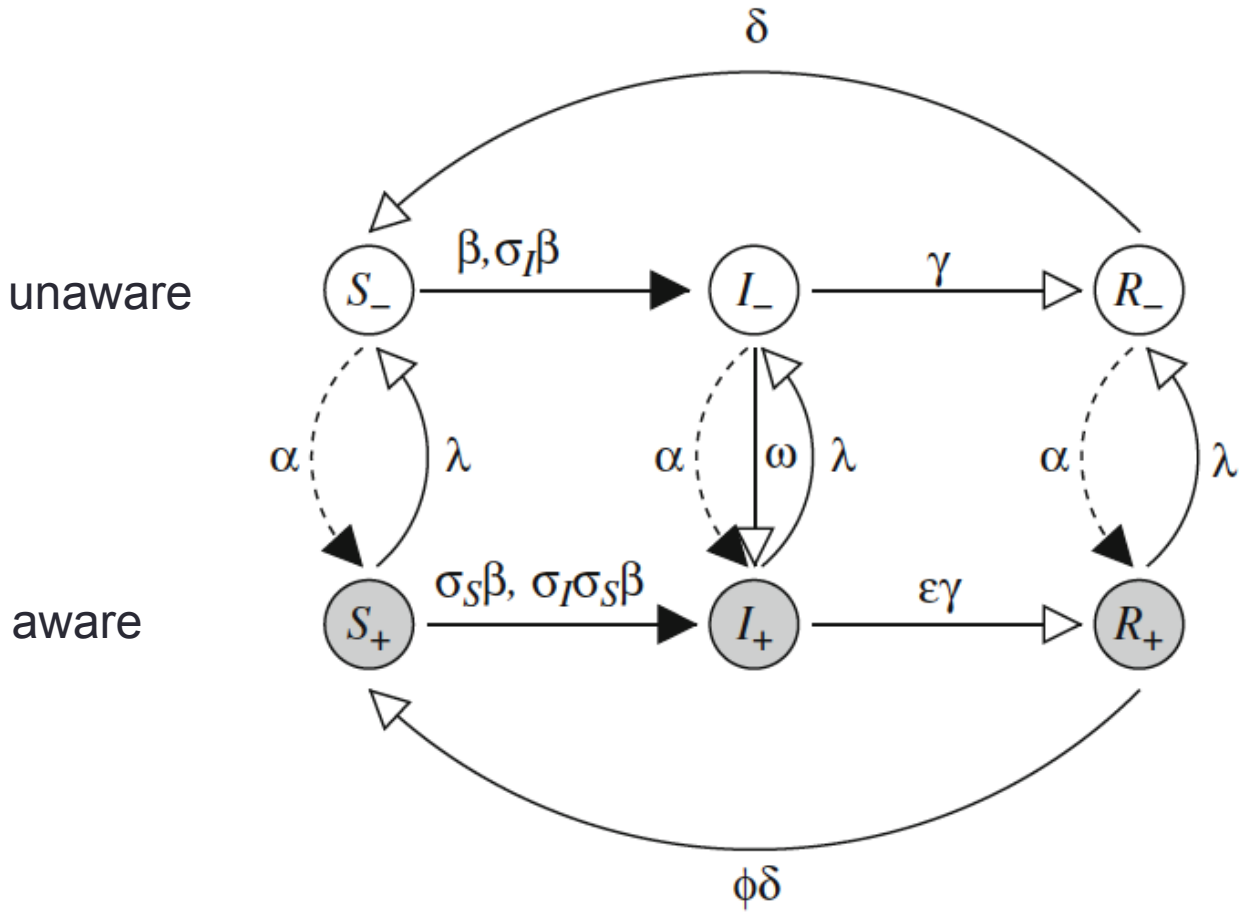
6872–6877 | PNAS | April 21, 2009 | vol. 106 | no. 16

The impact of information transmission on epidemic outbreaks

Istvan Z. Kiss[a,*], Jackie Cassell[b], Mario Recker[c], Péter L. Simon[d]

Mathematical Biosciences 225 (2010) 1–10

An extended compartmental model



(Funk et al., *JTB* 2010)

Conclusions from these (and other) works

- In models where the appearance of aware nodes is only based on local generation of information arising from the presence of the disease, there is no change in epidemic threshold but a reduction of the final epidemic size.
- When awareness spreads as an infection (aware nodes “infect” susceptible ones), epidemic threshold changes.
- In IBM models with different networks for the transmission of infection and information, the degree of overlapping plays an important role but no analytic expression of the epidemic threshold involving the latter is available.

Competing contagious processes

- Awareness propagation is a contagious process and, so, its dissemination in the presence of an epidemic can be embedded into the class of competing spreading processes.

- Recent papers deal with the simultaneous progress of competitive viral species and study conditions for their coexistence

Competing contagious processes

Effect of the interconnected network structure on the epidemic threshold

Huijuan Wang,[1,2,*] Qian Li,[2] Gregorio D'Agostino,[3] Shlomo Havlin,[4] H. Eugene Stanley,[2] and Piet Van Mieghem[1]

PHYSICAL REVIEW E **88**, 022801 (2013)

PHYSICAL REVIEW E **89**, 062817 (2014)

Competitive epidemic spreading over arbitrary multilayer networks

Faryad Darabi Sahneh[*] and Caterina Scoglio

Awareness and epidemics - 2

PRL 111, 128701 (2013) PHYSICAL REVIEW LETTERS week ending 20 SEPTEMBER 2013

Dynamical Interplay between Awareness and Epidemic Spreading in Multiplex Networks

Clara Granell,[1] Sergio Gómez,[1] and Alex Arenas[1,2]
[1]*Departament d'Enginyeria Informàtica i Matemàtiques, Universitat Rovira i Virgili, 43007 Tarragona, Spain*
[2]*IPHES, Institut Català de Paleoecologia Humana i Evolució Social, C/Escorxador s/n, 43003 Tarragona, Spain*

Individual-based Information Dissemination in Multilayer Epidemic Modeling

Faryad Darabi Sahneh[a], Fahmida N. Chowdhury[b], Gary Brase[c] and Caterina M. Scoglio[a, 1]

Math. Model. Nat. Phenom. (2014)

Generalized Epidemic Mean-Field Model for Spreading Processes Over Multilayer Complex Networks

Faryad Darabi Sahneh, *Student Member, IEEE*, Caterina Scoglio, *Member, IEEE*, and Piet Van Mieghem, *Member, IEEE*

IEEE/ACM TRANSACTIONS ON NETWORKING (2013)

Two-layer networks

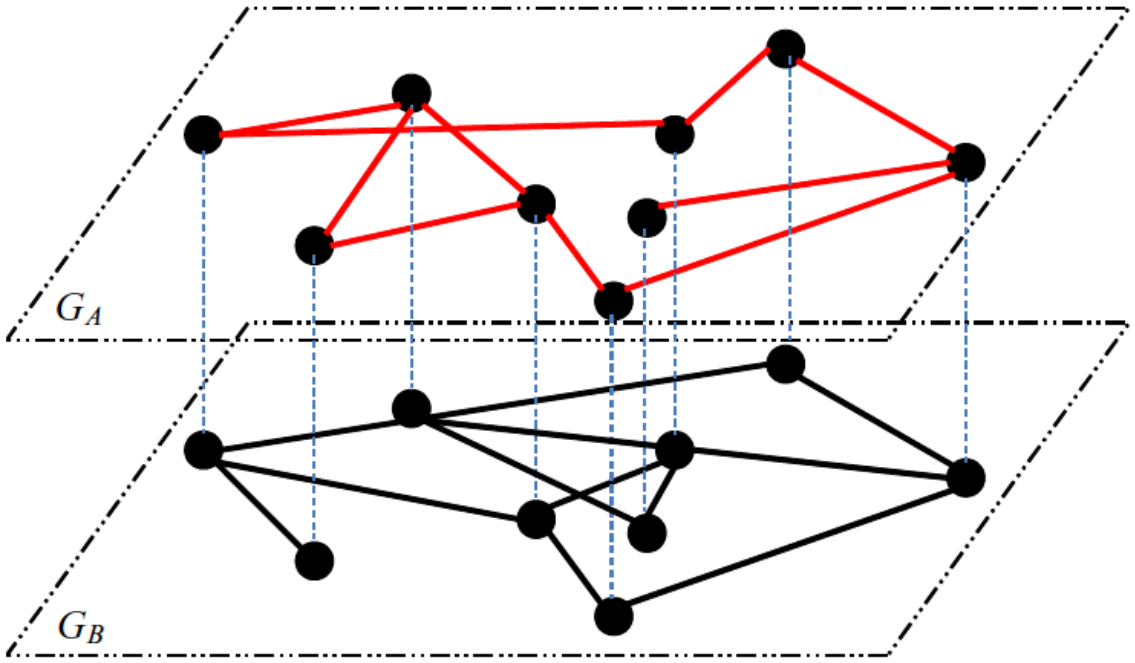


FIG. 1. (Color online) Schematic of two-layer contact topology $\mathcal{G}(V, E_A, E_B)$, where a group of nodes shares two distinct interactions.

(Sahneh & Scoglio, *PRE* (2014))

Multiplex networks

PHYSICAL REVIEW X **3**, 041022 (2013)

Mathematical Formulation of Multilayer Networks

Manlio De Domenico,[1] Albert Solé-Ribalta,[1] Emanuele Cozzo,[2] Mikko Kivelä,[3] Yamir Moreno, Mason A. Porter,[6] Sergio Gómez,[1] and Alex Arenas[1]

PHYSICAL REVIEW E **89**, 032804 (2014)

Structural measures for multiplex networks

Federico Battiston,[1] Vincenzo Nicosia,[1,2] and Vito Latora[1,2,3]

Questions arising from such processes

- What characteristics of two-layer networks allow for coexistence of competing contagious processes?
- How to characterize the interrelation between layers in a meaningful way for the dynamics of processes defined on them?

An interesting analytical result

Theorem 1. In the SI_1SI_2S model [Eqs. (1) and (2)] for competitive epidemics over multilayer networks, if the two network layers G_A and G_B are identical, coexistence is impossible; i.e., a virus with even a slightly larger effective infection rate dominates and completely removes the other virus. Otherwise, if node-degree vectors of G_A and G_B are not parallel, i.e., $\boldsymbol{d}_A \nparallel \boldsymbol{d}_B$, or if normalized dominant eigenvectors of G_A and G_B do not completely overlap, i.e., $\boldsymbol{v}_A \neq \boldsymbol{v}_B$, the multilayer structure of the underlying topology allows a nontrivial coexistence region.

(Sahneh & Scoglio, *PRE* 2014)

Interrelation between network layers

- *Overlap* and *inter-layer degree-degree correlation* have been highlighted as important features

The spread of awareness and its impact on epidemic outbreaks

Sebastian Funk[a,1], Erez Gilad[a], Chris Watkins[b], and Vincent A. A. Jansen[a]

6872–6877 | PNAS | April 21, 2009 | vol. 106 | no. 16

- The relationship of the overlap with previous analytical results about coexistence of competing processes is not clear

4. A model for studying the overlap impact

- Let us extend the heterogeneous SIS/SIR model by assuming the following hypotheses:

1) Links of the two layers *uniformly* overlap over the set of nodes: the fraction of overlapped links is independent of the degree

2) Intra-layer degree correlations are not present (proportionate mixing within each layer)

A model for studying the overlap impact

- According to these assumptions, we can write

$$\frac{dI_k}{dt} = k\beta(1-p_{B|A})S_k\Theta_I + k\beta_c p_{B|A} S_k \Theta_I - \mu I_k$$

$p_{B|A}$ = prob. that two nodes connected by a randomly chosen link of layer *A* are also connected in layer *B*

$$\Theta_I = \frac{1}{\langle k \rangle N}\sum_k kI_k$$

A model for studying the overlap impact

- To introduce the overlap α into the model, we have to relate it to the conditional probability $p_{B|A}$
- Defining the overlap as $\alpha := \frac{L_{A\cap B}}{L_{A\cup B}}$ it follows

$$
\begin{aligned}
p_{B|A} &= \frac{L_{A\cap B}}{L_A} = \frac{L_{A\cap B}}{L_{A\cup B}} \frac{L_{A\cup B}}{L_A} \\
&= \alpha \frac{L_A + L_B - L_{A\cap B}}{L_A} \\
&= \alpha \left(1 + \frac{\langle k_B \rangle}{\langle k_A \rangle} - p_{B|A}\right)
\end{aligned}
$$

A model for studying the overlap impact

- Introducing this relationship into the model and if we use the fraction of nodes that are both infectious and of degree k, $i_k = I_k / N$, we have

$$\begin{aligned}\frac{di_k}{dt} &= \frac{k}{1+\alpha}\left(\beta\left(1-\frac{\langle k_B\rangle}{\langle k_A\rangle}\alpha\right)\right. \\ &+ \left.\beta_c\left(1+\frac{\langle k_B\rangle}{\langle k_A\rangle}\right)\alpha\right)\left(p_A(k)-i_k\right)\Theta_I - \mu i_k\end{aligned}$$

$$\left(p_A(k) = N_k / N = i_k + s_k\right)$$

(Juher & J.S., *arXiv* 2015)

A model for studying the overlap impact

If $\langle k_B \rangle > \langle k_A \rangle$, the non-negativity of the factor multiplying β is guaranteed because the overlapping α is bounded from above by

$$\alpha = \frac{L_{A \cap B}}{L_{A \cup B}} \leq \frac{\langle k_A \rangle N}{\langle k_A \rangle N + \langle k_B \rangle N - \langle k_A \rangle N} = \frac{\langle k_A \rangle}{\langle k_B \rangle}$$

In general, we have:

$$\alpha \leq \frac{\min\left\{\langle k_A \rangle, \langle k_B \rangle\right\}}{\max\left\{\langle k_A \rangle, \langle k_B \rangle\right\}}$$

A model for studying overlap impact

- It can be considered as an extension of the classic heterogeneous mean-field SIS model, so similar results follows
- For instance, linearizing around the DFE, it follows:

$$R_0(\alpha) = \frac{\langle k_A^2 \rangle}{\langle k_A \rangle (1+\alpha)\,\mu} \left(\beta \left(1 - \frac{\langle k_B \rangle}{\langle k_A \rangle} \alpha \right) + \beta_c \left(1 + \frac{\langle k_B \rangle}{\langle k_A \rangle} \right) \alpha \right).$$

Predicted R_0 *vs* overlap

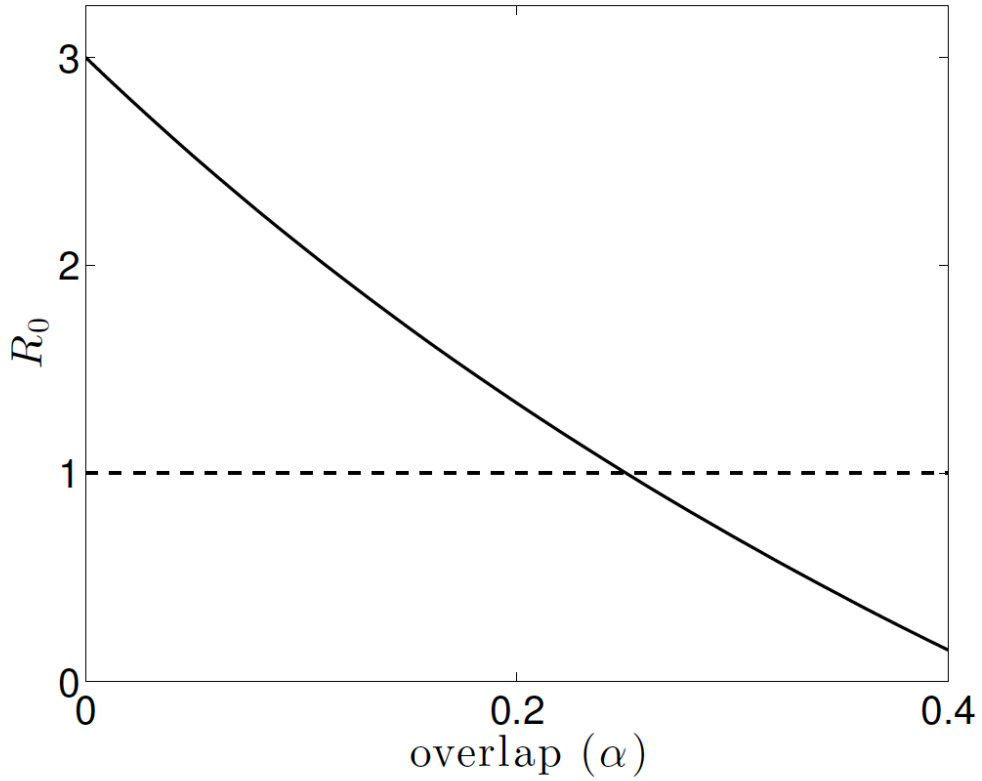


Figure 1: R_0 of the mean-field SIS model as a function of the overlap α between network layers. Parameters values: $\mu = 1$, $\beta = 0.1$, $\beta_c = 0.005$, $\langle k_A \rangle = 20$, $\langle k_A^2 \rangle = 600$, and $\langle k_B \rangle = 50$. For these mean degrees, $\alpha \in [0, 2/5]$.

Stochastic simulations

- Develop an algorithm that, given two degree distributions, allows a the maximum range of possible overlaps
- Based on a cross-rewiring process: the degree distribution of each layer remains unchanged
- Given the degree sequences $\{k_i\}$, $\{k_i'\}$ of each layer, a more accurate upper bound of the maximum overlap between them is

$$\alpha_{\max} \leq \frac{\sum_i \min\left\{k_i, k_i'\right\}}{\sum_i \max\left\{k_i, k_i'\right\}}$$

Overlap between degree distributions

	Regular	Poisson	SF	Exponential
Regular	1 1	0.739020 0.7761	0.564752 0.6112	0.448772 0.5004
Poisson		0.993035 0.994325	0.654052 0.7180	0.583859 0.6345
SF			0.97987 0.98575	0.665794 0.7095

TABLE 3. Maximum overlap generated using the CR Algorithm (first row) vs the maximum value permitted by Theorem 4.4 (second row). In all cases, $N = 10000$ and $\langle k \rangle = 10$.

R_0 computed from stochastic simul's

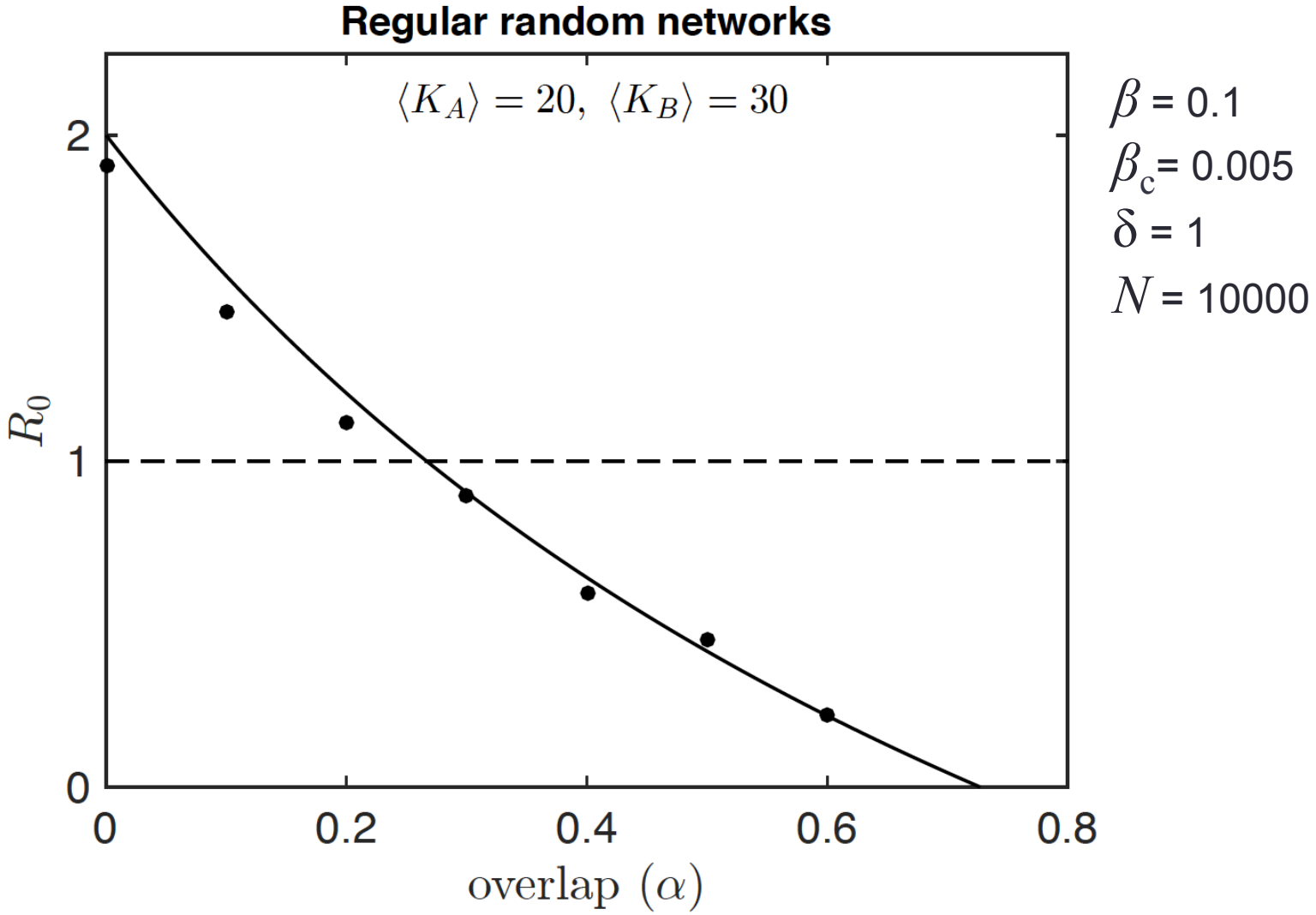
- R_0 computed as the mean number of new infections produced by "typical" individuals at the beginning of an outbreak, i.e., by those who have been infected by primary cases

  (Britton, Juher & J.S., *arXiv* 2015, to appear in *J.Theor. Biol.*)

- Primary cases are chosen uniformly at random (i.e., independently of their degree)

- Results correspond to averages over 250 runs using different sets of 10 primary cases

Comparison of R_0: preliminary results



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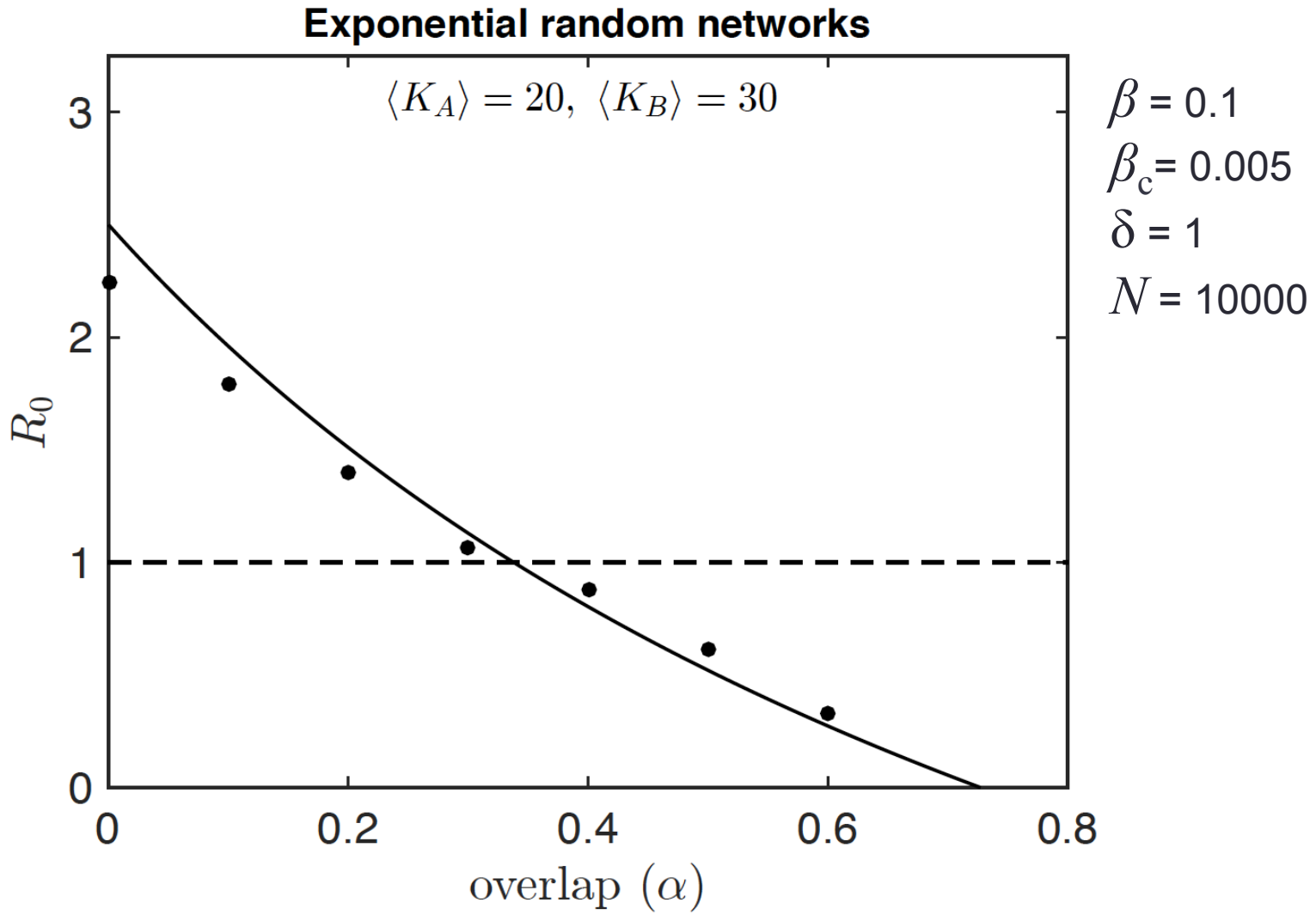

Conclusions of the extended SIS model

- A simple model to analyse the impact of network overlap on the initial epidemic growth is derived
- An algorithm to control the desired overlap between layers without intra-layer degree correlations is implemented
- Simulations with different degree distributions show the importance of having uniform overlap over the whole set of nodes for the accuracy of the model predictions

Thanks for your attention !!

https://sites.google.com/site/min2016girona/

Creating and controlling overlap in two-layer networks. Application to a mean-field SIS epidemic model with awareness dissemination. In: arXiv:1504.02031 [physics.soc-ph]