“Disease Spreading Processes in Multilayer Networks”

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Modeling and Understanding Disease Spreading

- Theoretical Models for Single Layers
- Meta-population Approaches
- Data driven simulation/analysis
- Agent Based Modeling
- Digital Epidemiology

See Alex Vespignani’s talk
There are however several less explored -an increasingly important- problems:


How can we deal with both the disease natural history and the networks of interactions?
Layers account for different networks of contacts through which diseases spread

Original, aggregate network

Host Population

D1

D2

D3
Co-occurrence TB-HIV:

How?

- Two interconnected networks:


- Two coupled epidemic models:
Two interconnected networks:

- Networks could be either “scale-free”, homogeneous or even a well-mixed scenario.
- This essentially depends on how the disease spreads.

Two coupled epidemic models:

- SIS or SIR Scenarios, but when dealing with real diseases, more complex compartmental models should be used.
Two coupled SIS

Interaction

Modified susceptibility

Modified infectivity

\[ \beta_1^a \lambda_1 \]

\[ \beta_1^b \lambda_1 \]

\[ \eta_1 \mu_1 \]
Summing up

Disease I

\[ \lambda_1 \]

\[ \beta_1^a \lambda_1 \]

\[ \beta_1^b \lambda_1 \]

\[ \beta_1^a \beta_1^b \lambda_1 \]

\[ \mu_1 \]

\[ \eta_1 \mu_1 \]
Summing up

Disease II

\[ \lambda_2 \]
\[ \beta_2^a \lambda_2 \]
\[ \beta_2^b \lambda_2 \]
\[ \beta_2^a \beta_2^b \lambda_2 \]
\[ \mu_2 \]
\[ \eta_2 \mu_2 \]
Heterogenous Mean-field Formulation...

Equations

\[
\begin{align*}
\dot{S}_S(k, l) &= -(k\sigma_1 + l\sigma_2)S_S(k, l) + \mu_1 I_S(k, l) + \mu_2 I_I(k, l) \\
\dot{I}_S(k, l) &= k\sigma_1 S_S(k, l) - l\beta_2^a\sigma_2 I_S(k, l) - \mu_1 I_S(k, l) + \eta_2\mu_2 I_I(k, l) \\
\dot{S}_I(k, l) &= l\sigma_2 S_S(k, l) - k\beta_1^a\sigma_1 I_S(k, l) - \mu_2 I_I(k, l) + \eta_1\mu_1 I_I(k, l) \\
\dot{I}_I(k, l) &= k\beta_1^a\sigma_1 I_S(k, l) + l\beta_2^a\sigma_2 I_S(k, l) - (\eta_1\mu_1 + \eta_2\mu_2) I_I(k, l)
\end{align*}
\]

with

\[
\begin{align*}
\sigma_1 &= \lambda_1(\theta_1^{IS} + \beta_1^b\theta_1^{II}) & \sigma_2 &= \lambda_2(\theta_2^{SI} + \beta_2^b\theta_2^{II})
\end{align*}
\]

The Threshold

\[
\lambda_1^c(\sigma_2) = \mu_1 \frac{\langle k \rangle}{\sum_{k, l} P(k, l)k^2 \frac{l^2\sigma_2^2\beta_2^a\beta_1^b + l\sigma_2(\eta_2\mu_2\beta_1^a + \beta_1^b(\beta_1^a\mu_1 + \beta_2^a\mu_2)) + \mu_2(\eta_1\mu_1 + \eta_2\mu_2)}{l^2\sigma_2^2\beta_2^a\eta_1 + l\sigma_2(\eta_1\mu_1 + \eta_2\mu_2 + \beta_2^a\eta_1\mu_2) + \mu_2(\eta_1\mu_1 + \eta_2\mu_2)}}
\]
Mutual enhancement: Homogeneous contact patterns

\[ \beta > 1.0 \quad \eta < 1.0 \]

Regions where a disease becomes endemic only after the installation of the other disease on the population
Partial Cross Immunity: Homogeneous contact patterns

\[ \beta < 1.0 \quad \eta > 1.0 \]
Partial Cross Immunity: Homogeneous contact patterns

$\beta < 1.0 \quad \eta > 1.0$

Regions where a disease can be eradicated only after the installation of the conjugate disease on the population
Two coupled SIR dynamics

Few more parameters

\[ \phi_{1,2}^a \quad \text{S}_{1,2} \text{ recovered from 2(1)} \]

\[ \phi_{1,2}^b \quad \text{I}_{1,2} \text{ recovered from 2(1)} \]

\[ \zeta_{1,2} \quad \text{recovery rate due to R}_{2(1)} \]
Two coupled SIR dynamics

The threshold depends on the time evolution of the other disease.
Social Contagion

Social Movements
Belief Adoption
Viral spreading
Multilayer Networks: Social Systems

Original, aggregate network

When unfolded, layers appear
Models

• Threshold models:

Information like a pathogen: SIS

Single layer Microscopic Markov Chain

\[ p_i(t + 1) = (1 - q_i(t))(1 - p_i(t)) + (1 - \mu)p_i(t) + \mu(1 - q_i(t))p_i(t) \]

\[ q_i(t) = \prod_{j=1}^{N}(1 - \beta r_{ij}p_j(t)) \]

\[ r_{ij} = 1 - \left(1 - \frac{a_{ij}}{k_i}\right) \lambda_i \]

Probability of not being infected by any neighbor

\[ \left(\frac{\beta}{\mu}\right)_c = \frac{1}{\Lambda_{max}} \]

Contacts Matrix

Contacts per time

S. Gómez et al., Europhys. Lett. 89, 38009 (2010)
How to represent it

Supra-Adjacency Matrix

\[
\tilde{A} = \bigoplus_{\alpha} A_{\alpha} + C = A + C
\]

\[
\tilde{A} = \begin{pmatrix}
A_1 & C_{1,2} & C_{1,3} \\
C_{2,1} & A_2 & C_{2,3} \\
C_{3,1} & C_{3,2} & A_3
\end{pmatrix}
\]

\(A_i\) Layer adjacency matrix

\(C_{i,j}\) Coupling matrix
Microscopic Markov Chain on Multiplex

\[ \tilde{p}(t + 1) = (\mathbf{1} - \tilde{p}(t)) \ast (\mathbf{1} - \tilde{q}(t)) + (\mathbf{1} - \tilde{\mu}) \ast \tilde{p}(t) \tilde{\mu} \ast (\mathbf{1} - \tilde{q}(t)) \ast \tilde{p}(t) \]

Supra-Contacts Matrix

\[ \tilde{R} = \bigoplus_{\alpha} R_{\alpha} + \left( \frac{\tilde{\gamma}}{\beta} \right)^T C \]

\[ (R_{\alpha})_{ij} = 1 - \left( 1 - \frac{(A_{\alpha})_{ij}}{k_{\alpha_i}} \right)^{\lambda_{\alpha_i}} \]

Solving it

\[ [\bar{R} - \frac{\mu}{\beta} I] \rho = 0 \]

\[ \left( \frac{\beta}{\mu} \right)_c = \frac{1}{\Lambda_{max}} \]

The largest eigenvalue of \( \bar{R} \) sets the critical value but...

What does \( \Lambda_{max} \) look like?
The largest eigenvalue of $\bar{R}$

Perturbative Analysis

$$\bar{R} = R + \epsilon C$$

$$\bar{\Lambda}_{max} \simeq \Lambda + \epsilon \Delta \Lambda$$

$$\bar{\Lambda}_{max} = \max_{\alpha} \{ \Lambda_{\alpha} \}$$

$$\Delta \Lambda_{max} = \frac{\bar{\nu}^T C \bar{\nu}}{\bar{\nu}^T \bar{\nu}}$$

If $\Lambda_{1_{max}} >> \Lambda_{\alpha_{max}}$

$$\bar{\nu} = \begin{pmatrix} \bar{\nu}^{(1)} \\ 0 \end{pmatrix} \rightarrow \Delta \Lambda = 0$$

At first order:

$$\bar{\Lambda}_{max} = \Lambda_{max}$$

Dominant Layer
The Dominant Layer sets the critical point for the outbreak but…

Dominance depends on both topology and activity

\[(R_\alpha)_{ij} = 1 - \left(1 - \frac{(A_\alpha)_{ij}}{k_{\alpha i}}\right)^{\lambda_{\alpha i}}\]
• Largest eigenvalue of $R_\alpha$: a measure of the uncertainty of the interactions

• Dominant layer: least constrained interaction network

The least constrained interaction network drives the social contagion process
Indeed, one can go a bit more abstract:

(continuous) dynamics on a single layer network:

\[
\frac{dX_i}{dt} = -\mu X_i + (1 - X_i) \lambda \sum_j A(i, j) X_j
\]

(continuous) dynamics on a multilayer network:

\[
\frac{dX_{\beta \tilde{\delta}}}{dt} = -\mu X_{\beta \tilde{\delta}} + (1 - X_{\beta \tilde{\delta}}) X \mathcal{R}_{\beta \tilde{\delta}}^{\alpha \tilde{\gamma}}(\lambda, \eta) X_{\alpha \tilde{\gamma}}
\]

\[
\mathcal{R}_{\beta \tilde{\delta}}^{\alpha \tilde{\gamma}}(\lambda, \eta) = M_{\beta \sigma}^{\alpha \eta} E_{\eta}^{\tilde{\sigma}} (\tilde{\gamma} \tilde{\delta}) \delta_{\tilde{\delta}}^{\tilde{\gamma}} + \frac{\eta}{\lambda} M_{\beta \sigma}^{\alpha \eta} E_{\eta}^{\tilde{\sigma}} (\tilde{\gamma} \tilde{\delta}) (U_{\tilde{\delta}}^{\tilde{\gamma}} - \delta_{\tilde{\delta}})
\]
The layer with the largest eigenvalue sets the critical properties of the whole multilayer system

Disease Localization: \( \text{IPR}(\Lambda) \equiv (f_{\beta \delta}(\Lambda))^4 U^{\beta \delta} \)

In the localized phase, only the entries of the eigentensor associated with the dominant layer are effectively populated, while the entries associated with the other layers are not. In the delocalized phase, all the entries are equally populated.
Multilayer/multiplex networks are a useful conceptual framework for the study of complex disease contagion processes, e.g., interacting or competing diseases.

There is a dominant layer that drives the contagion process. It is the least constrained interaction network.

Disease Localization might be present. At variance with single layer networks, disease localizes on the layers, not on the nodes.
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