"Disease Spreading Processes in Multilayer Networks"

Yamir Moreno
Complex Systems & Networks Lab (COSNET)
Institute for Biocomputation and Physics of Complex Systems (BIFI)
University of Zaragoza, Spain.
ISI Foundation, Turin, Italy.

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:

- Competing/interacting Diseases (J. Sanz, C.-Y. Xia, S. Meloni, Y. Moreno, **Physical Review X 4**, 041005, 2014).

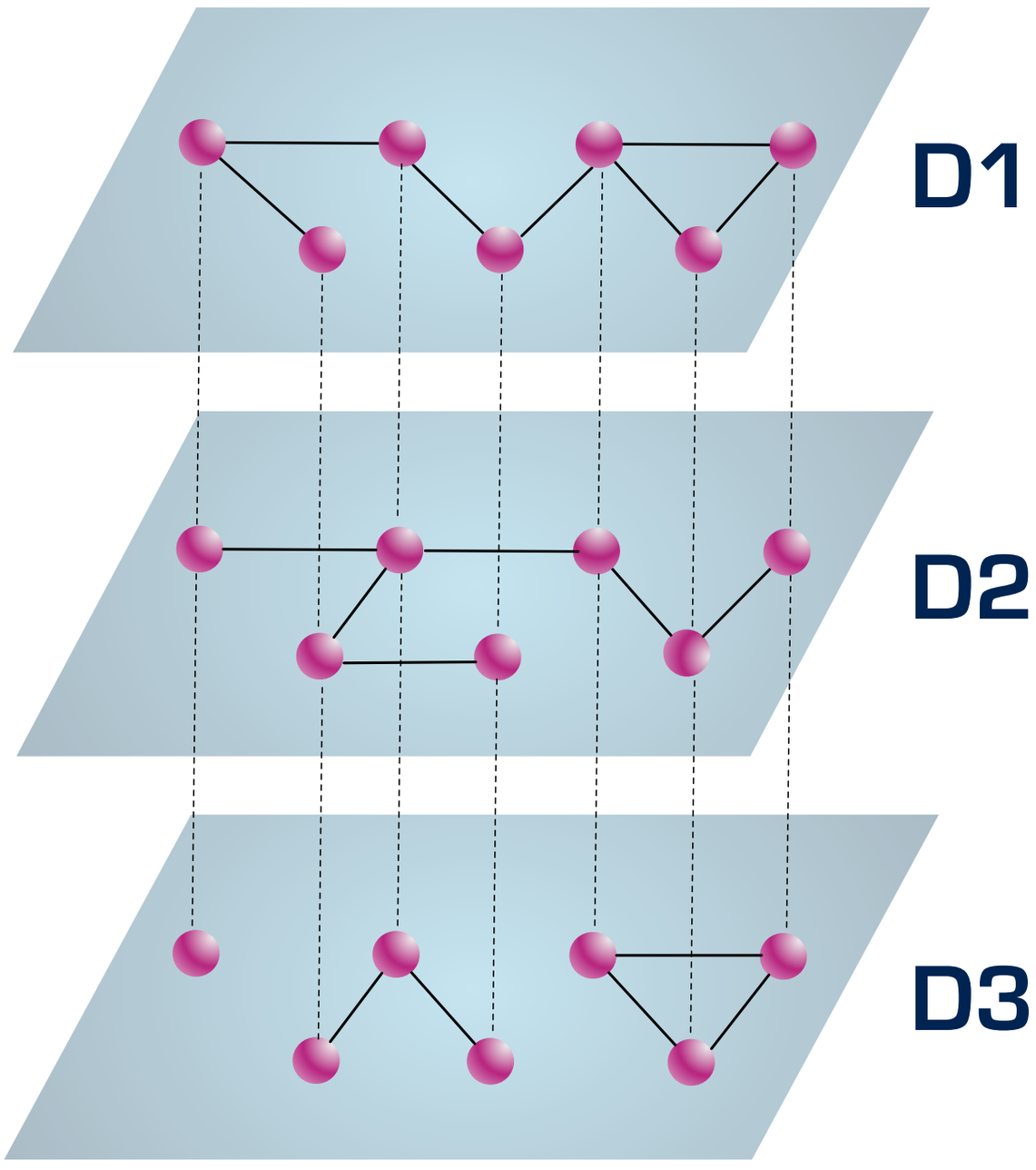
- Different strains of the same disease (C. Poletto, SM, V. Colizza, Y. Moreno, A. Vespignani, **PloS Comp. Bio. 9 (8)**: e1003169, 2013).

How can we deal with both the disease natural history and the networks of interactions?

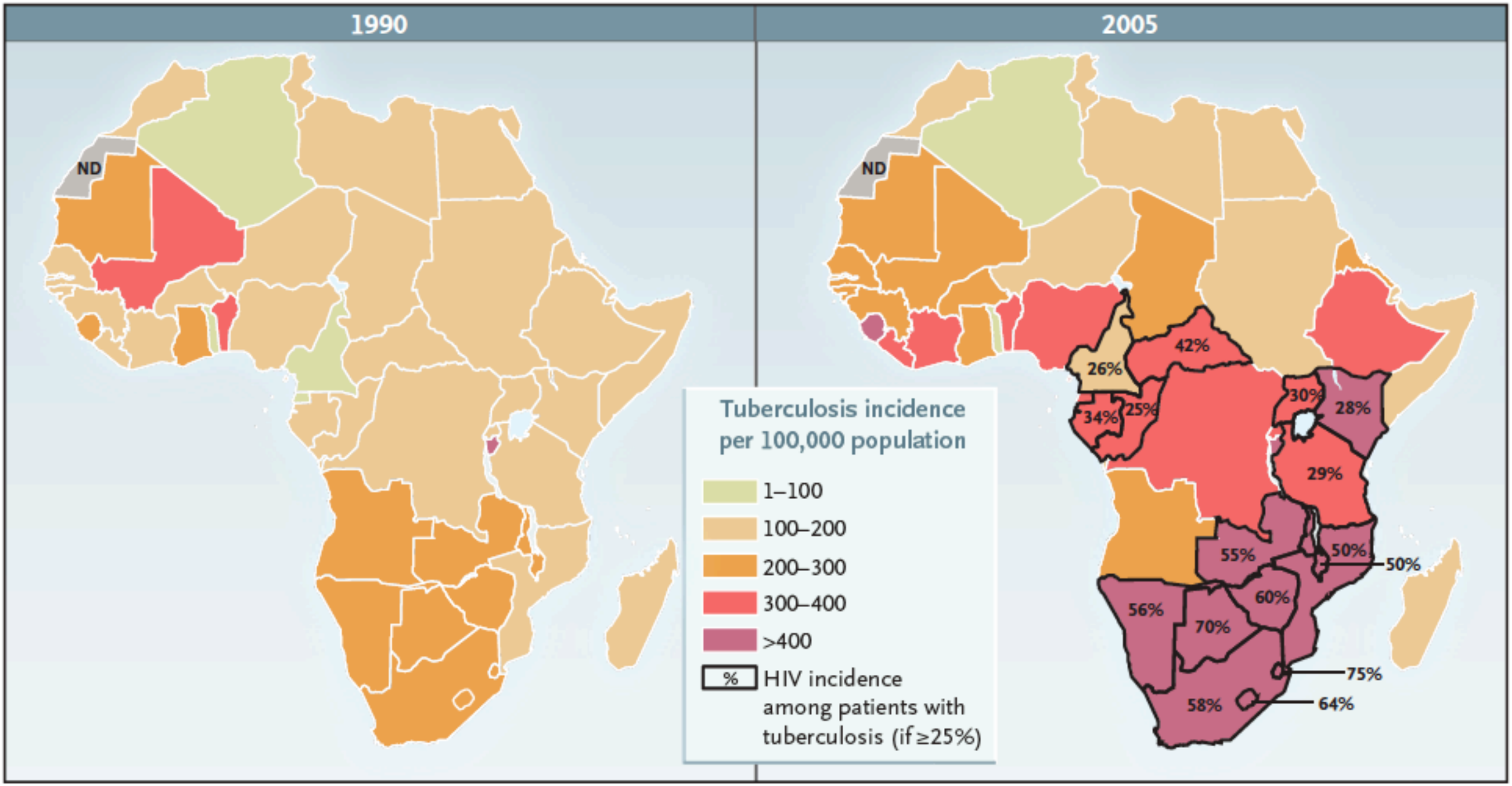
Layers account for different networks of contacts through which diseases spread

Original, aggregate network

Host Population



Co-occurrence TB-HIV:



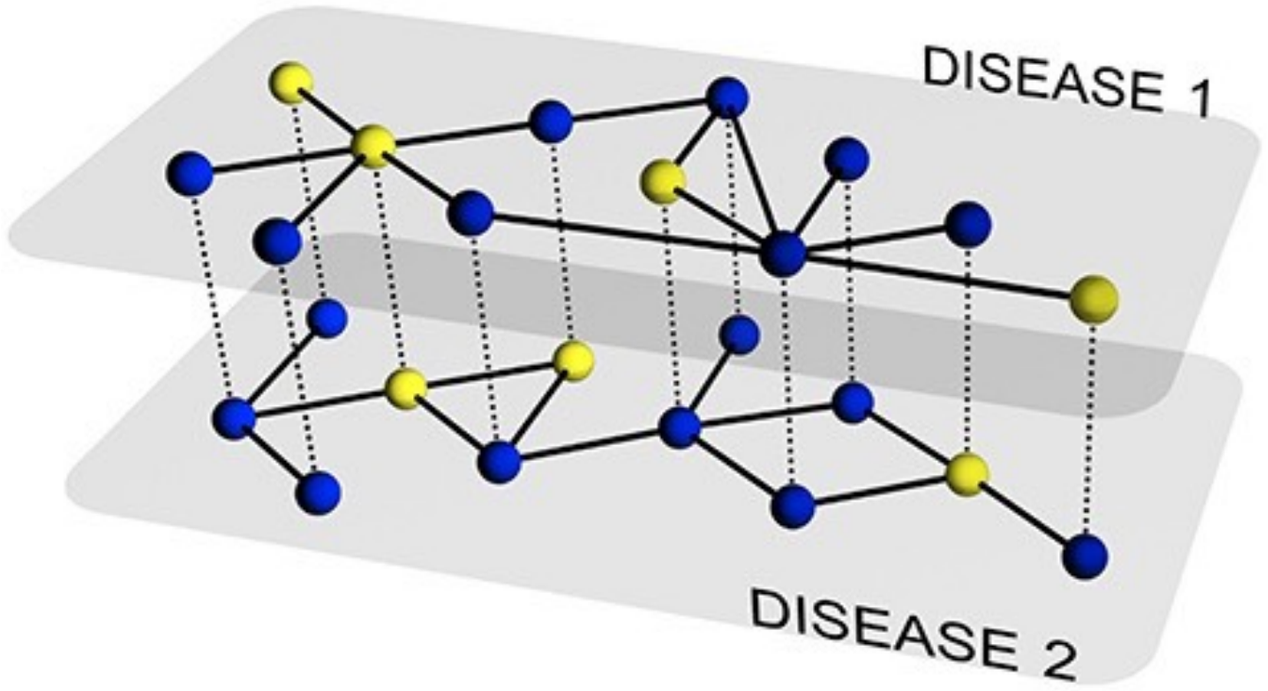
Estimated Incidence of Tuberculosis per 100,000 Population in African Countries in 1990 and 2005.
Data are from the World Health Organization. ND denotes no data.

From "**Tuberculosis in Africa, combating an HIV-driven Crisis**" Chaisson, R.E. & Martinson, N.A., New Eng. J. Med., March 2008.

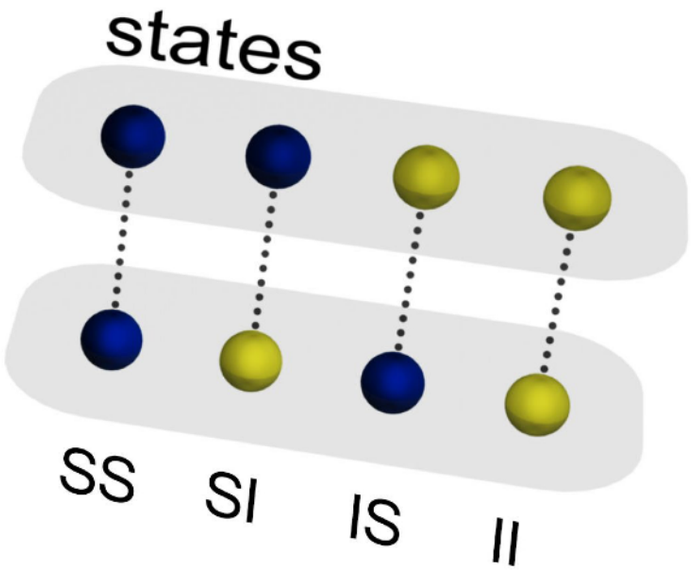
How?

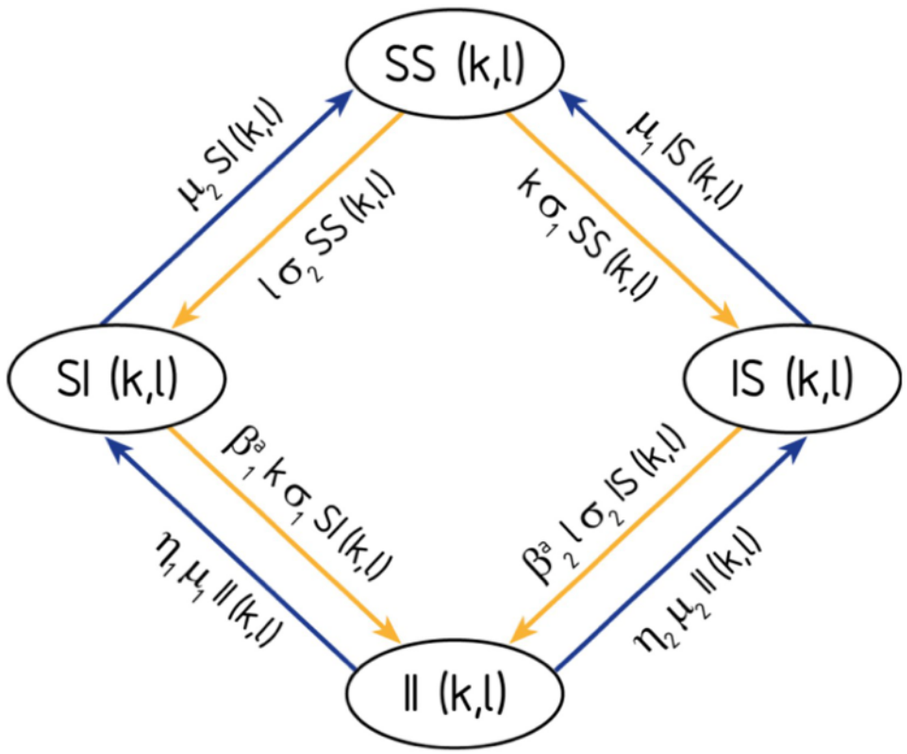
J. Sanz, C.-Y. Xia, S. Meloni, Y.Moreno,
Physical Review X 4, 041005 (2014).

- Two interconnected networks:



- Two coupled epidemic models:





How?

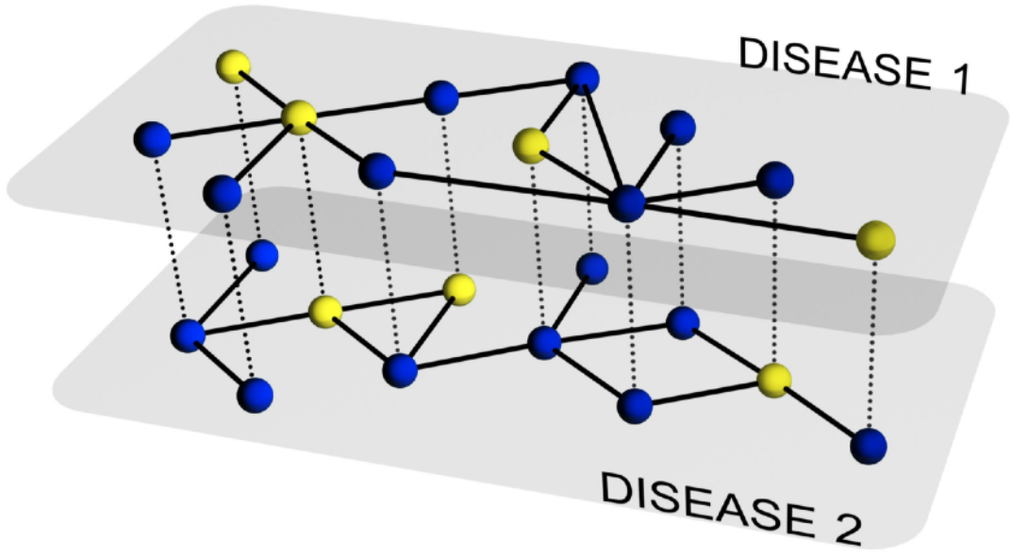
Two interconnected networks:

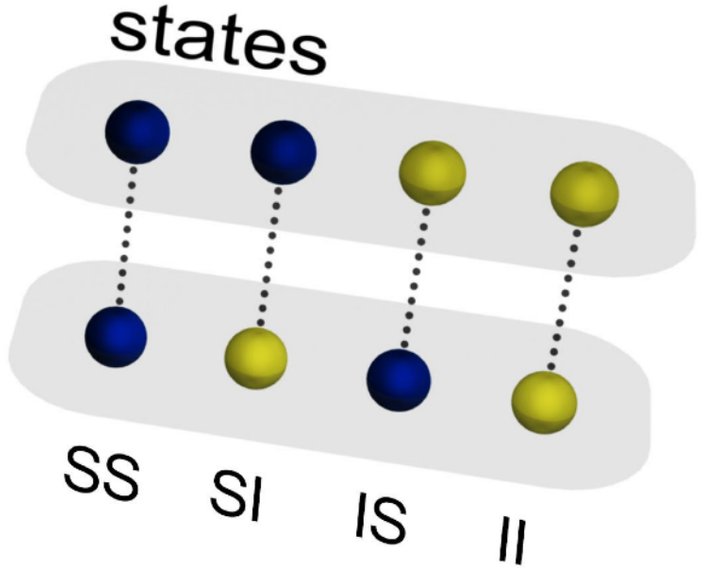
- Networks could be either “scale-free”, homogeneous or even a well-mixed scenario.
- This essentially depends on how the disease spreads.

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

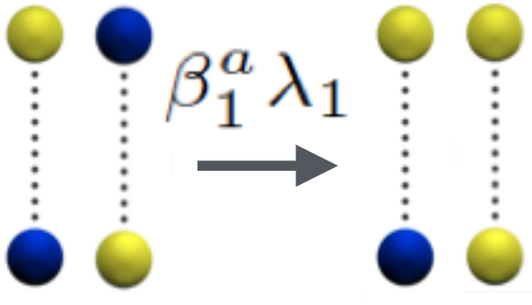
Modified susceptibility

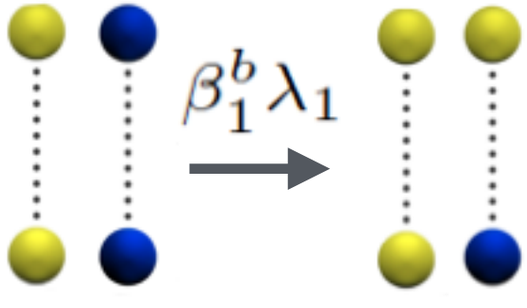
Modified recovery rate

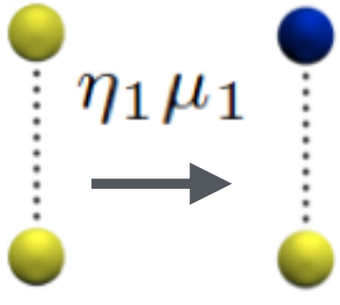
β_1^a

β_1^b

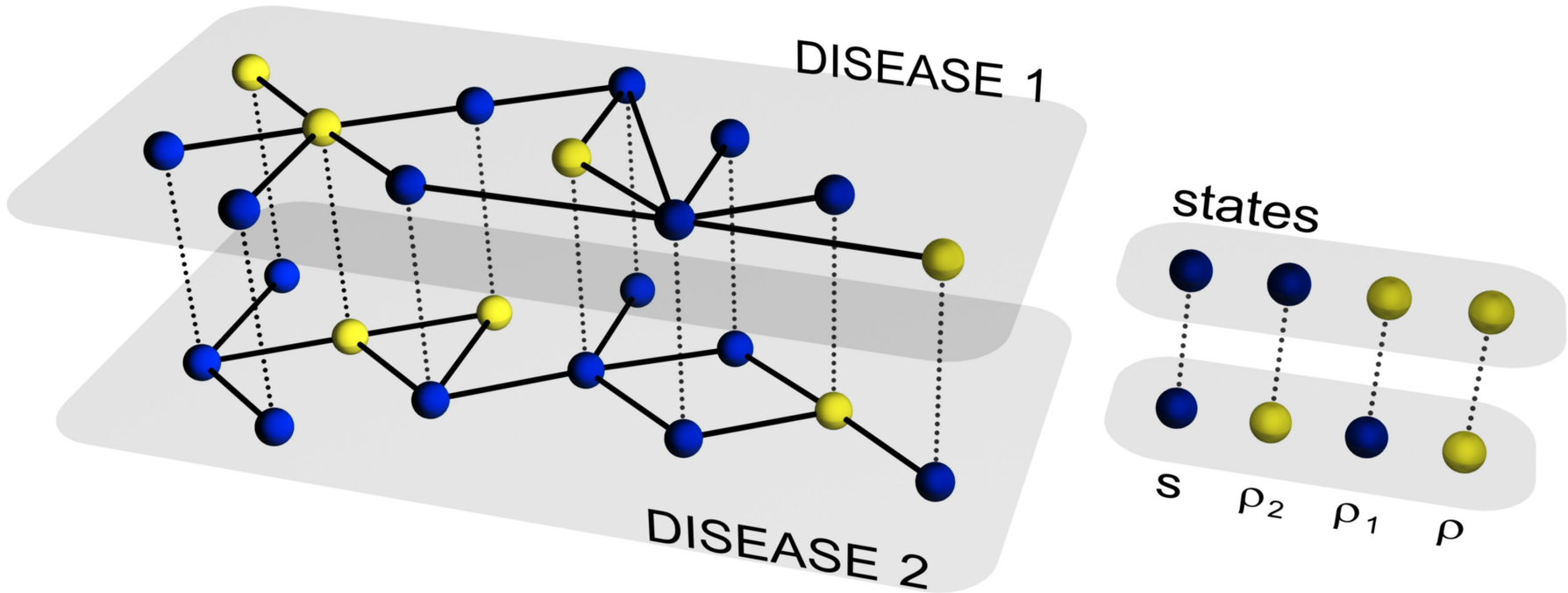
η_1



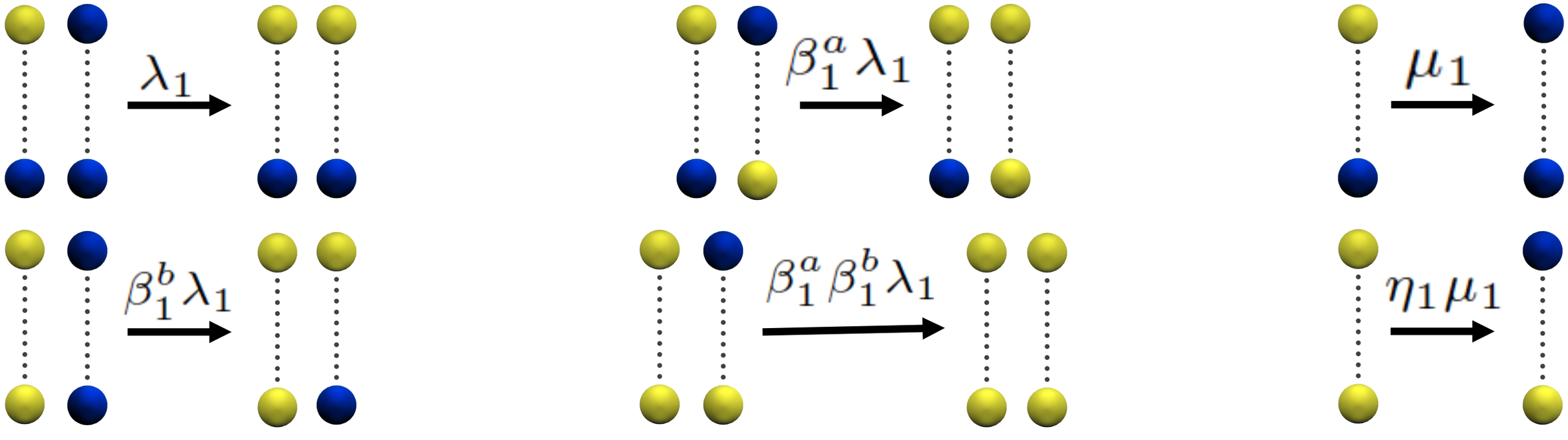




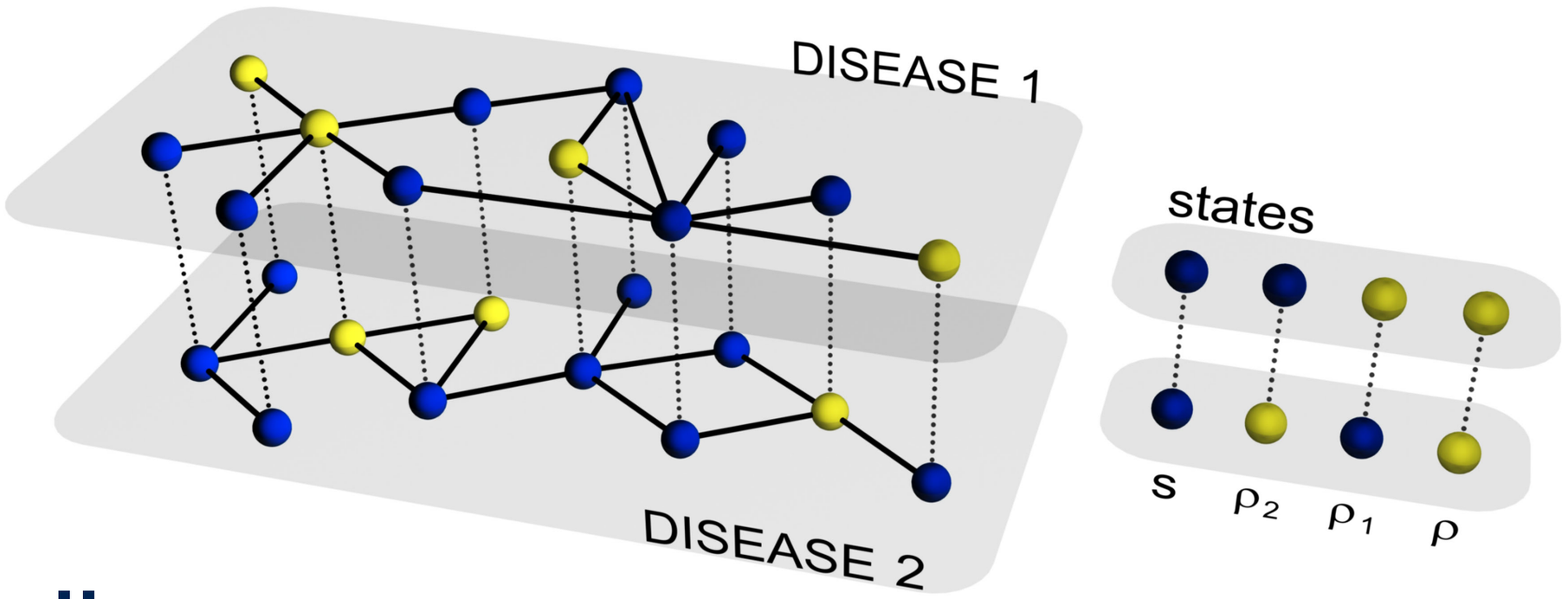
Summing up



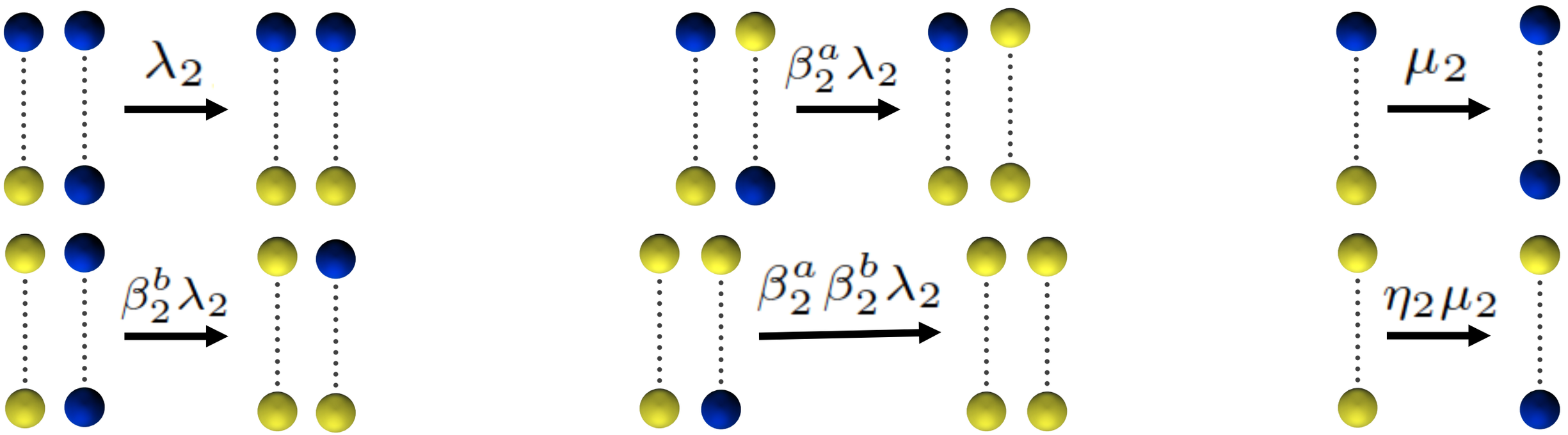
Disease I



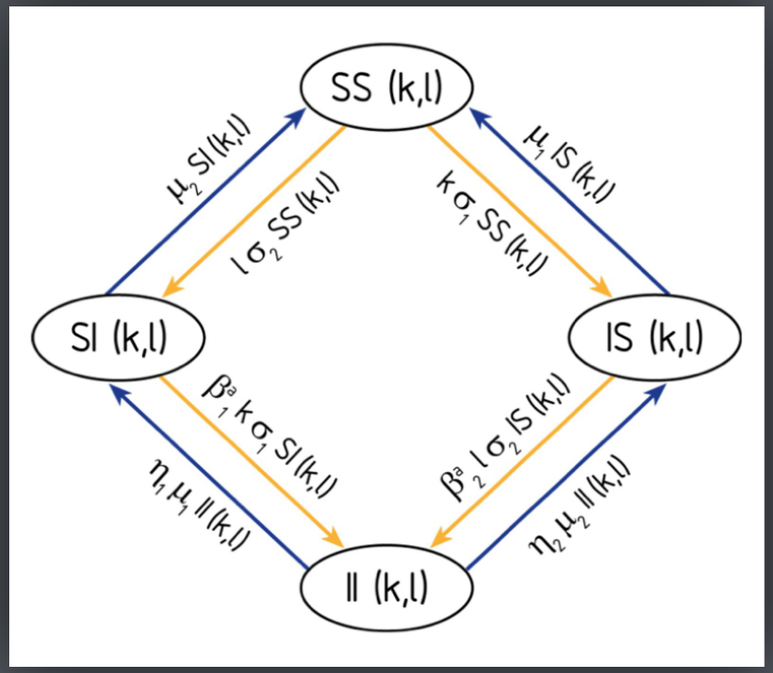
Summing up



Disease II



Heterogenous Mean-field Formulation…



Equations

$$\dot{SS}(k,l) = -(k\sigma_1 + l\sigma_2)SS(k,l) + \mu_1 IS(k,l) + \mu_2 SI(k,l)$$
$$\dot{IS}(k,l) = k\sigma_1 SS(k,l) - l\beta_2^a\sigma_2 IS(k,l) - \mu_1 IS(k,l) + \eta_2\mu_2 II(k,l)$$
$$\dot{SI}(k,l) = l\sigma_2 SS(k,l) - k\beta_1^a\sigma_1 SI(k,l) - \mu_2 SI(k,l) + \eta_1\mu_1 II(k,l)$$
$$\dot{II}(k,l) = k\beta_1^a\sigma_1 SI(k,l) + l\beta_2^a\sigma_2 IS(k,l) - (\eta_1\mu_1 + \eta_2\mu_2)II(k,l)$$

with

$$\sigma_1 = \lambda_1(\theta_1^{IS} + \beta_1^b\theta_1^{II}) \qquad \sigma_2 = \lambda_2(\theta_2^{SI} + \beta_2^b\theta_2^{II})$$

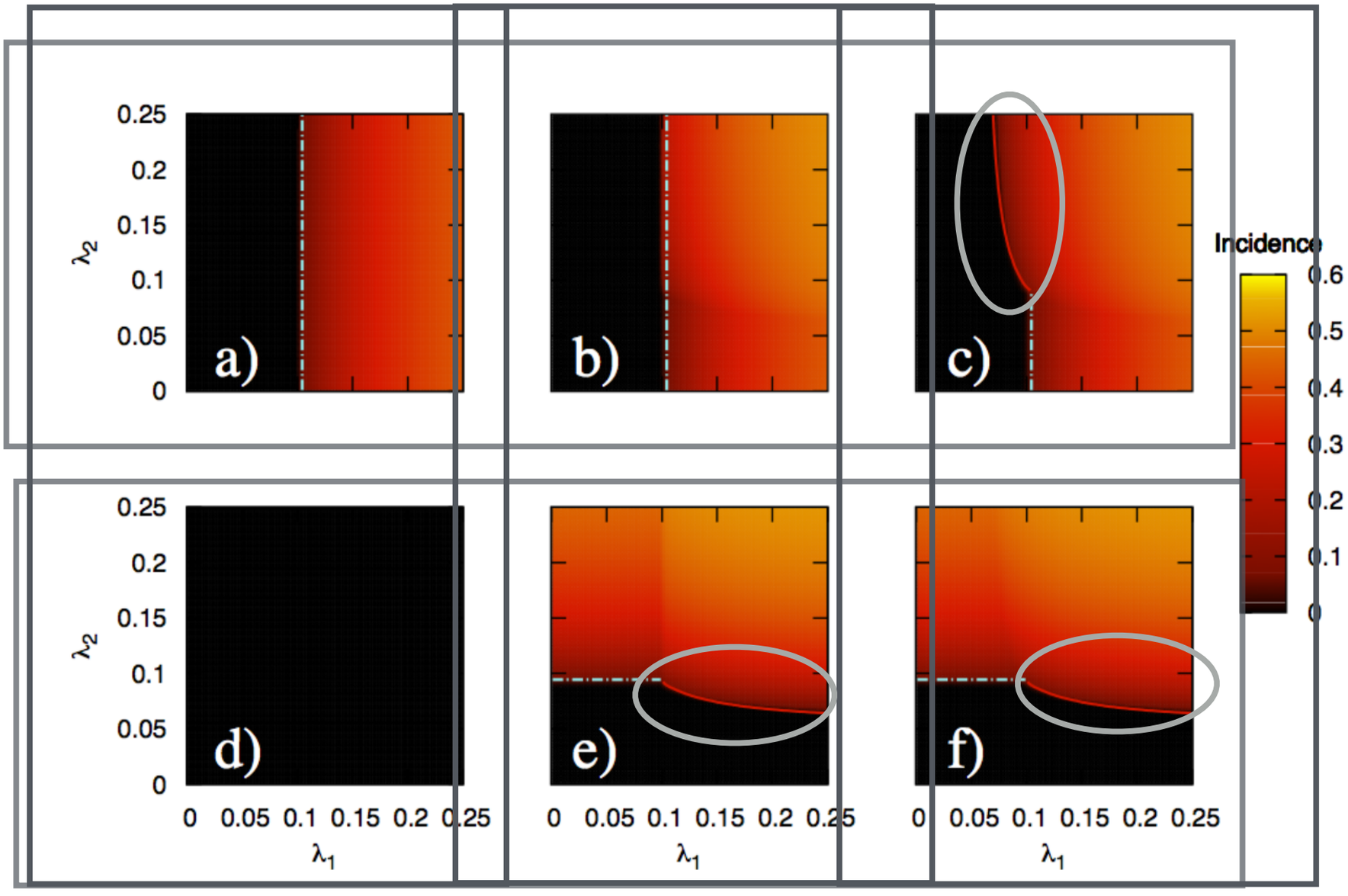
The Threshold

$$\lambda_1^c(\sigma_2) = \mu_1 \frac{\langle k\rangle}{\sum_{k,l} P(k,l)k^2 \frac{l^2\sigma_2^2\beta_2^a\beta_1^a\beta_1^b + l\sigma_2(\eta_2\mu_2\beta_1^a + \beta_1^b(\beta_1^a\mu_1 + \beta_2^a\mu_2)) + \mu_2(\eta_1\mu_1 + \eta_2\mu_2)}{l^2\sigma_2^2\beta_2^a\eta_1 + l\sigma_2(\eta_1\mu_1 + \eta_2\mu_2 + \beta_2^a\eta_1\mu_2) + \mu_2(\eta_1\mu_1 + \eta_2\mu_2)}}$$

Mutual enhancement: Homogeneous contact patterns

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

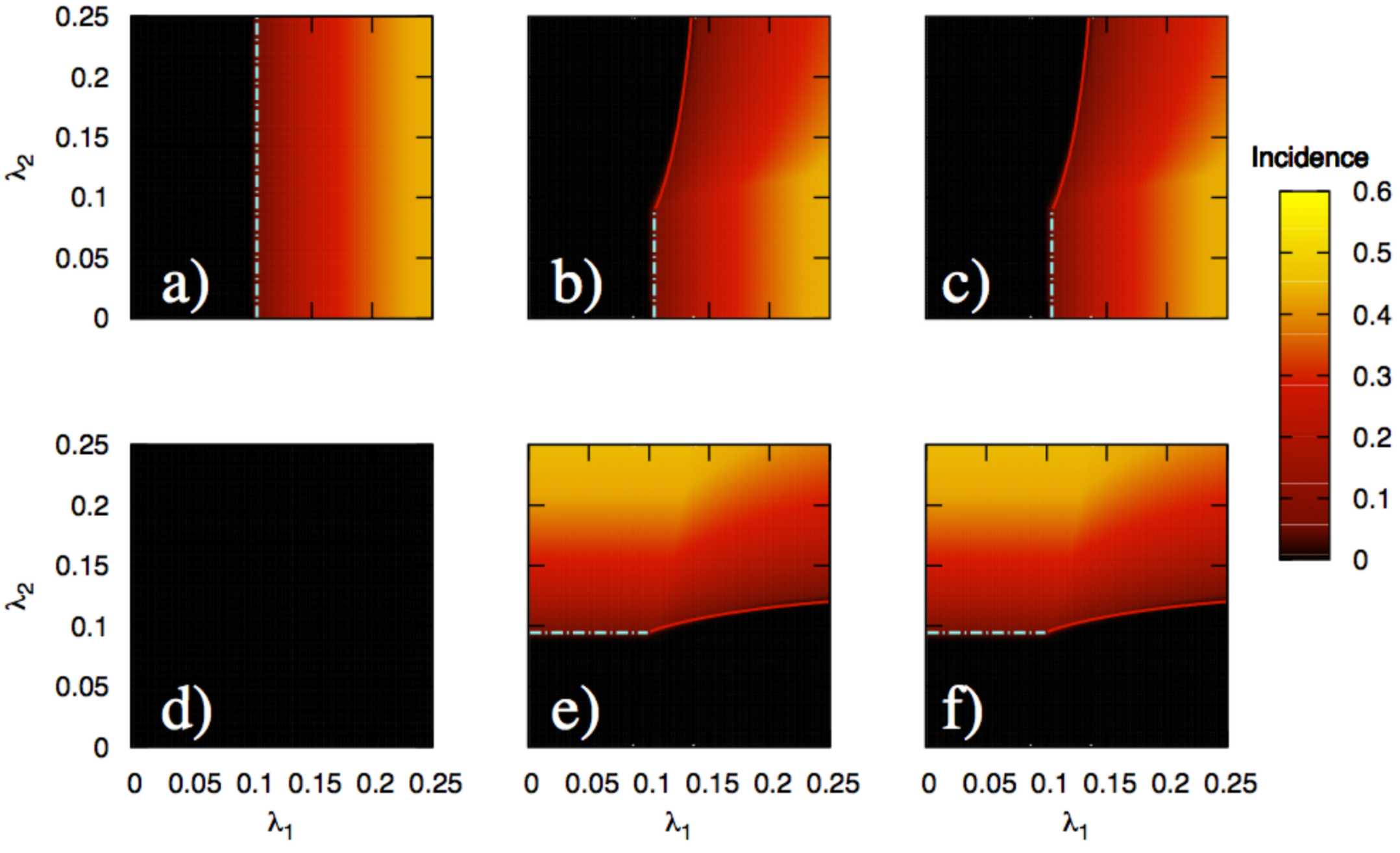
disease 2



Regions where a disease becomes endemic only after the installation of the other disease on the population

Partial Cross Immunity: Homogeneous contact patterns

$\beta < 1.0 \qquad \eta > 1.0$



Partial Cross Immunity: Homogeneous contact patterns

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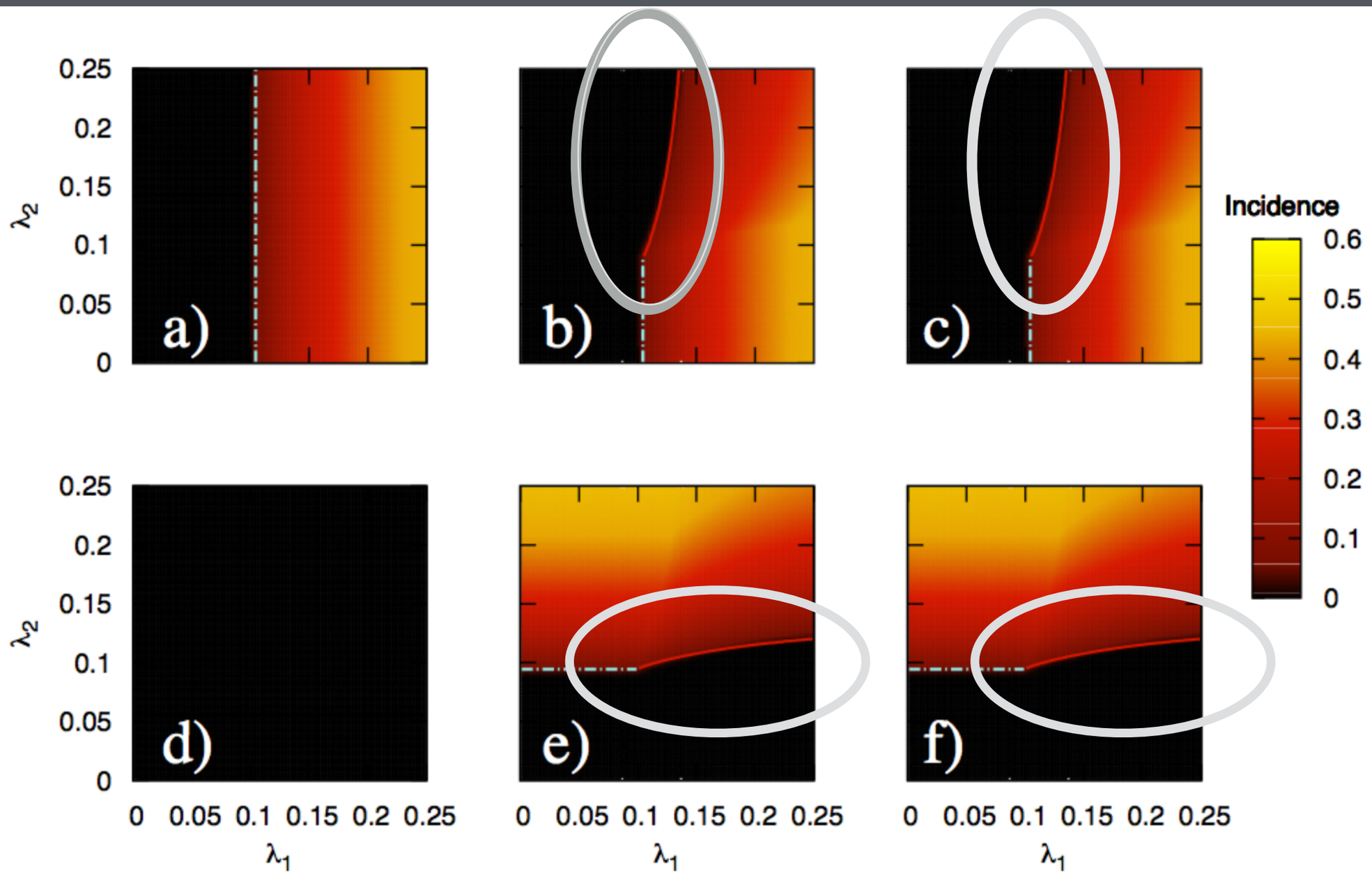
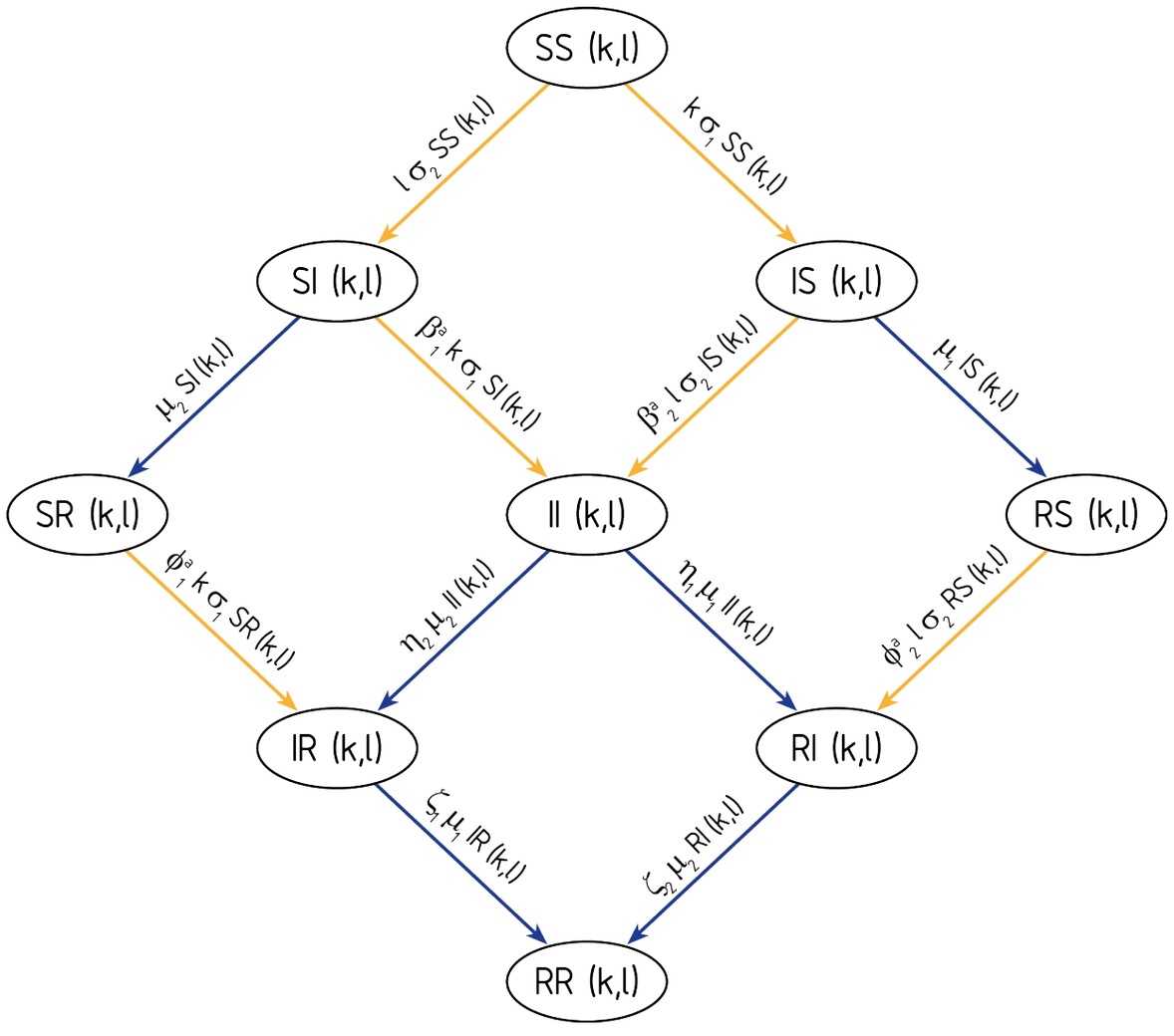


Regions where a disease can be eradicated only after the installation of the conjugate disease on the population

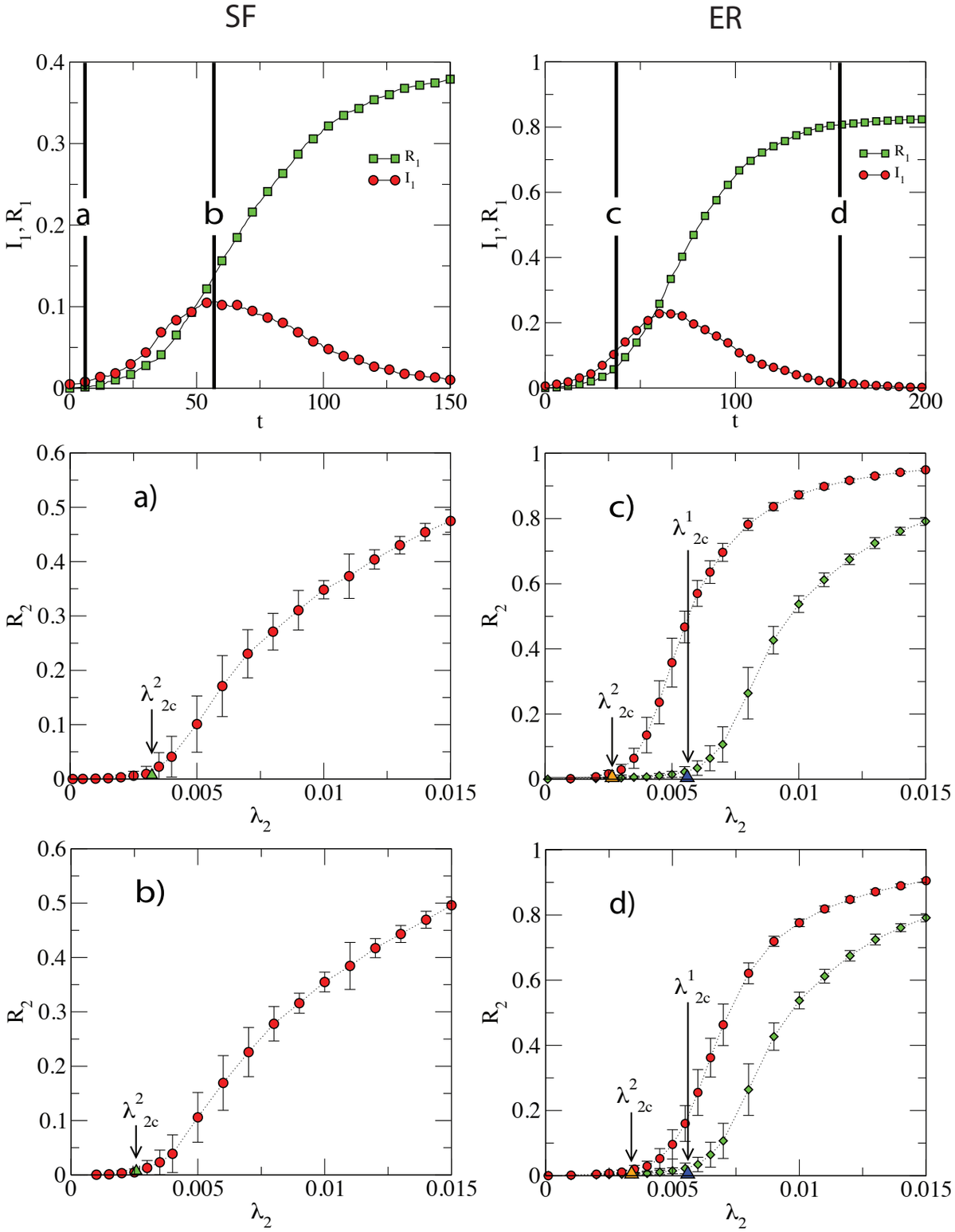
Two coupled SIR dynamics



Few more parameters

$\phi^a_{1,2}$	$S_{1,2}$ recovered from 2(1)
$\phi^b_{1,2}$	$I_{1,2}$ recovered from 2(1)
$\zeta_{1,2}$	recovery rate due to $R_{2(1)}$

Two coupled SIR dynamics



→ Temporal evolution of disease 1

The threshold depends on the time evolution of the other disease

Social Contagion

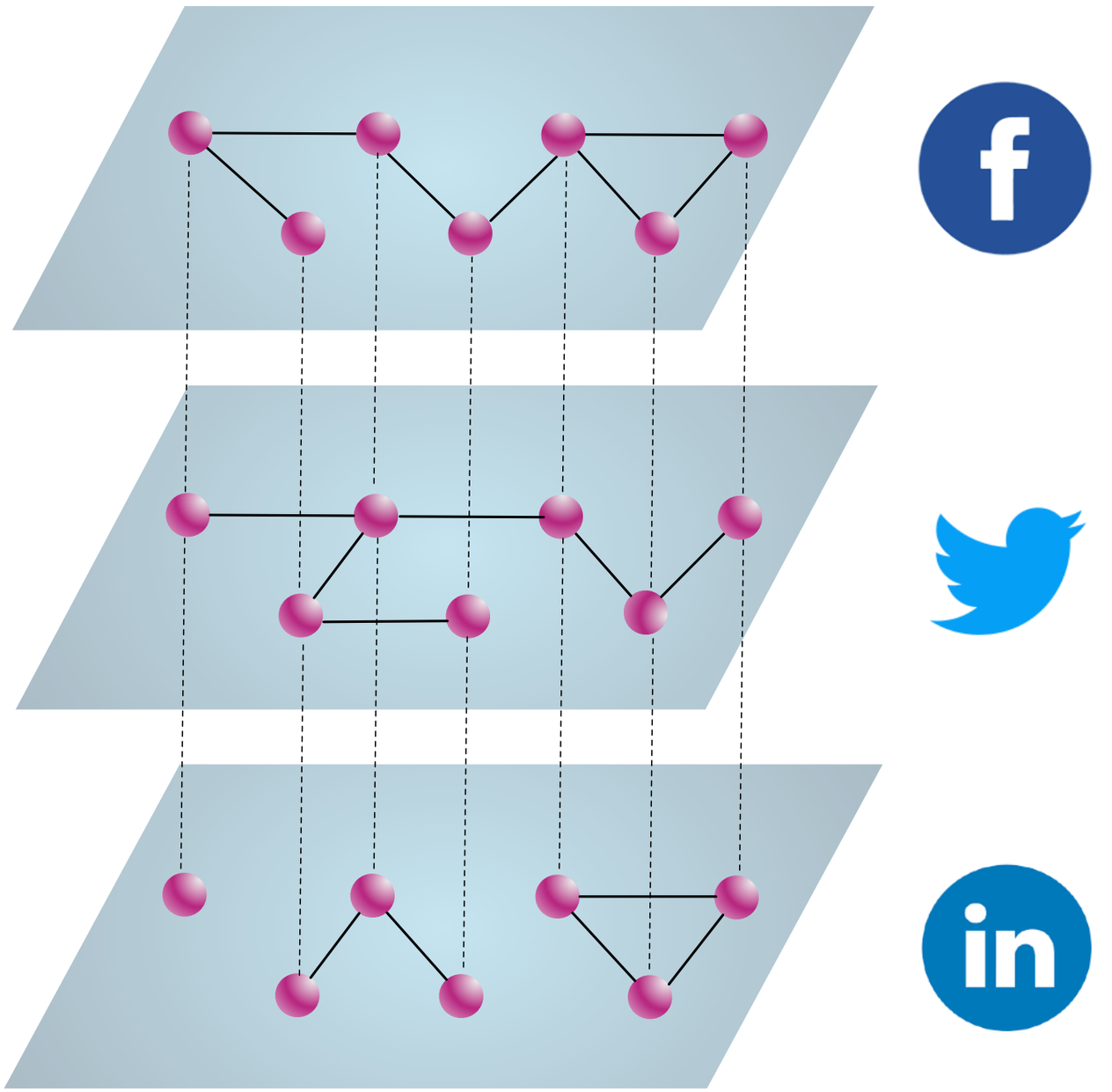
Social Movements

Belief Adoption

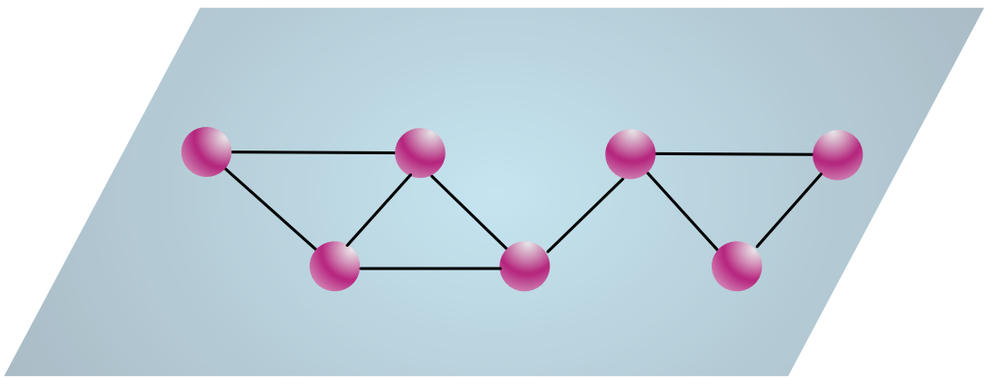
Viral spreading

Multilayer Networks: Social Systems

When unfolded, layers appear

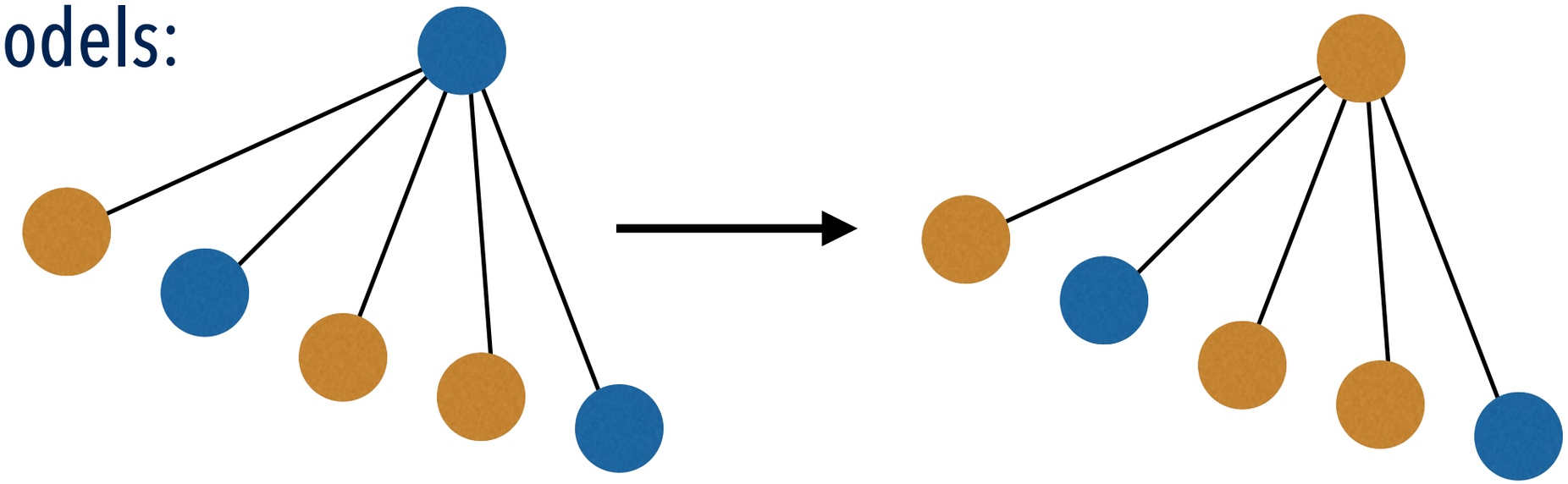


Original, aggregate network

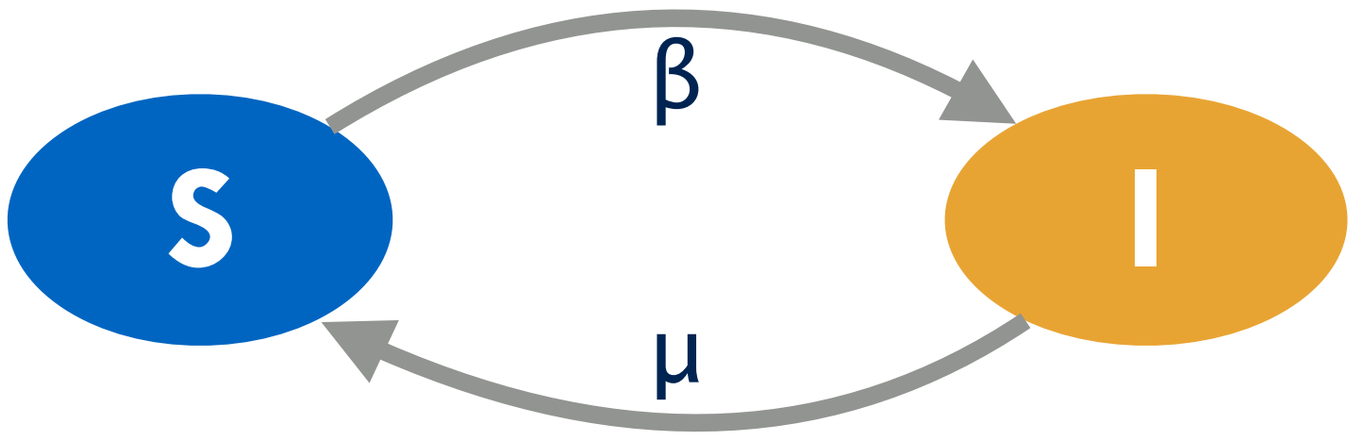


Models

- Threshold models:



Information like a pathogen: SIS



L. Weng, F. Menczer, Y.-Y. Ahn, **Virality Prediction and Community Structure in Social Networks**, Sci. Rep. 02522 (2013)

Single layer Microscopic Markov Chain

$$p_i(t+1) = (1 - q_i(t))(1 - p_i(t)) + (1 - \mu)p_i(t) + \mu(1 - q_i(t))p_i(t)$$

Threshold

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

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

Probability of not being infected by any neighbor

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

Contacts Matrix

Contacts per time

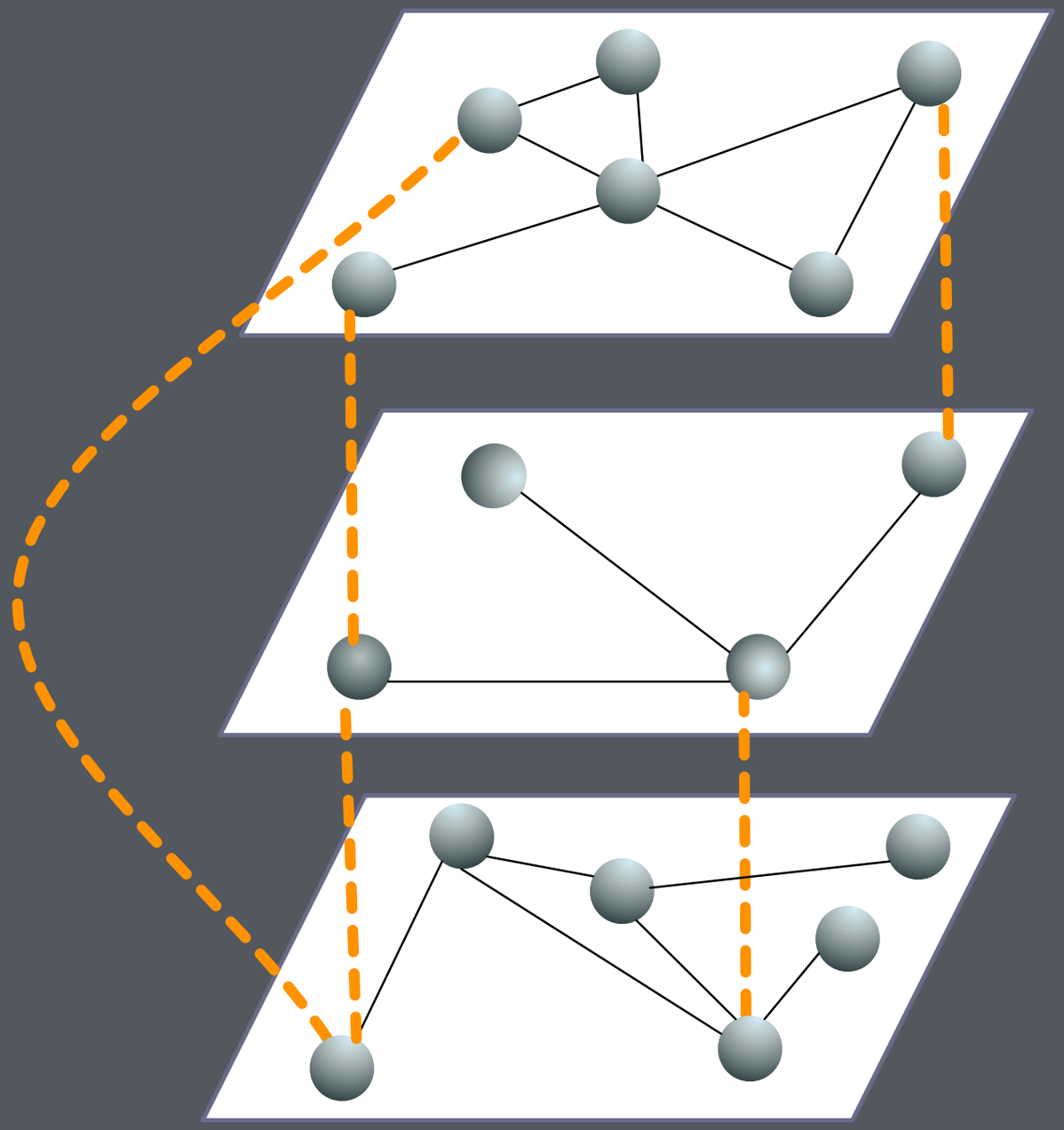
S. Gómez et al., Europhys. Lett. 89, 38009 (2010)

How to represent it

Supra-Adjacency Matrix

$$\bar{A} = \bigoplus_{\alpha} A_\alpha + C = A + C$$

$$\bar{A} = \left(\begin{array}{c|c|c} A_1 & C_{1,2} & C_{1,3} \\ \hline C_{2,1} & A_2 & C_{2,3} \\ \hline C_{3,1} & C_{3,2} & A_3 \end{array}\right)$$



A_i Layer adjacency matrix

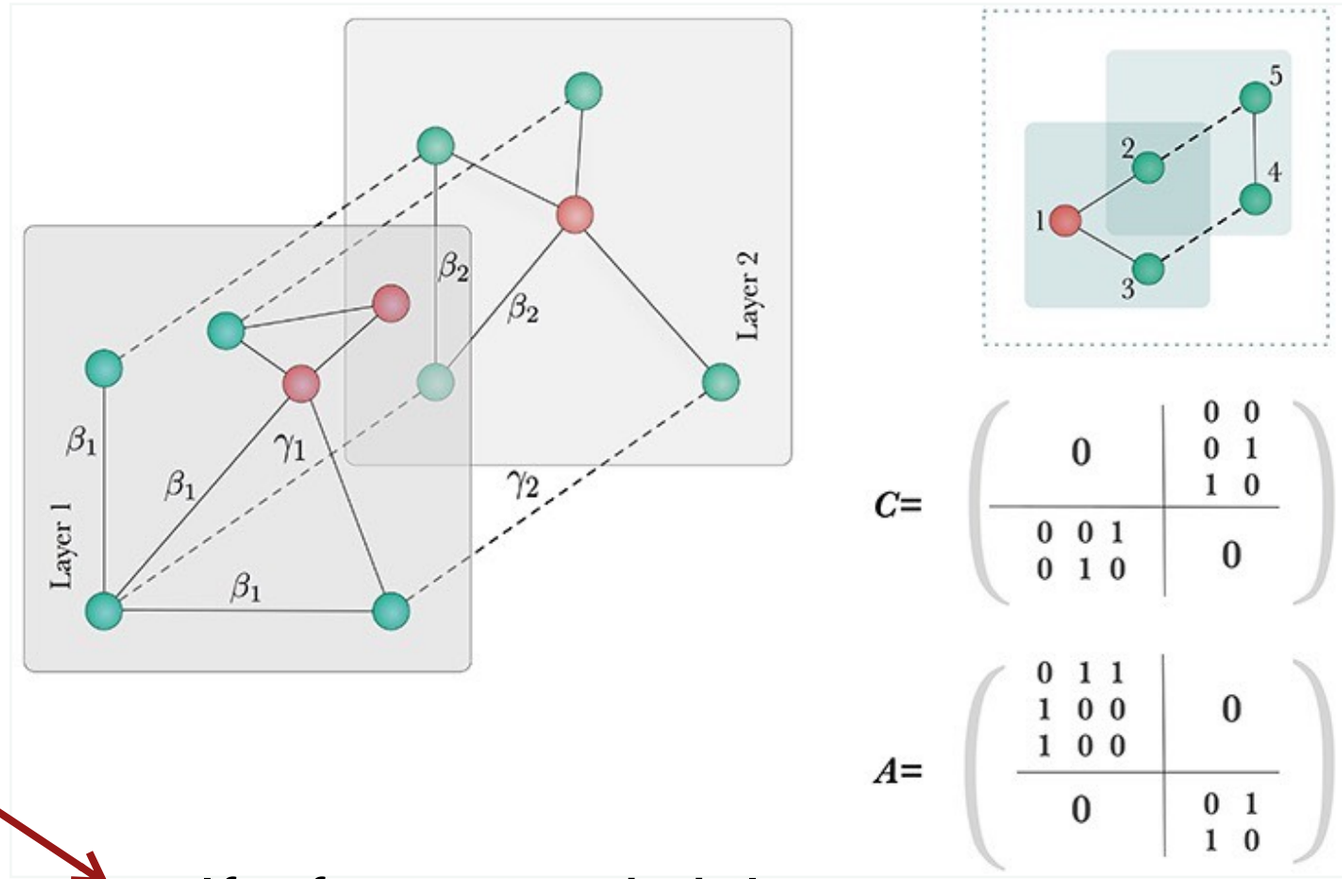
$C_{i,j}$ Coupling matrix

Microscopic Markov Chain on Multiplex

$$\vec{p}(t+1) = (\vec{1} - \vec{p}(t)) * (\vec{1} - \vec{q}(t)) + (\vec{1} - \vec{\mu}) * \vec{p}(t)\vec{\mu} * (\vec{1} - \vec{q}(t)) * \vec{p}(t)$$

Supra-Contacts Matrix

$$\bar{R} = \bigoplus_{\alpha} R_\alpha + \left(\frac{\vec{\gamma}}{\beta}\right)^T C$$



"self infection" probability

$$(R_\alpha)_{ij} = 1 - \left(1 - \frac{(A_\alpha)_{ij}}{k_{\alpha i}}\right)^{\lambda_{\alpha i}}$$

Cozzo et al. **Phys. Rev. E 88**, 050801(R) (2013)

Solving it

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

$$\left(\frac{\beta}{\mu}\right)_c = \frac{1}{\bar{\Lambda}_{max}}$$

The largest eigenvalue of $\bar{R}$ sets the critical value but…

What does $\bar{\Lambda}_{max}$ look like?

The largest eigenvalue of $\bar{R}$

Perturbative Analysis

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

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

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

$$\triangle \Lambda_{max} = \frac{\vec{v}^T C \vec{v}}{\vec{v}^T \vec{v}}$$

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

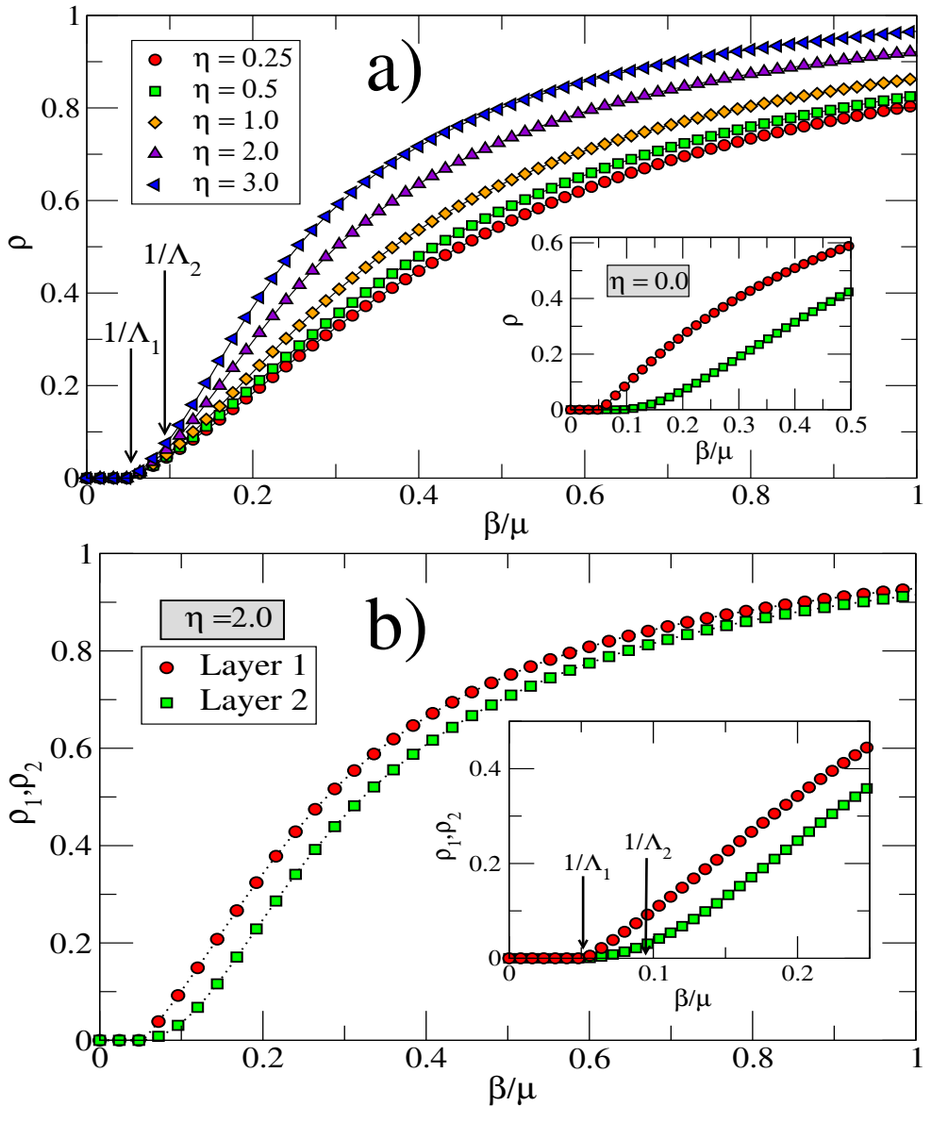
$$\vec{v} = \begin{pmatrix} \vec{v}_{(1)} \\ 0 \end{pmatrix} \rightarrow \triangle \Lambda = 0$$

At first order:

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

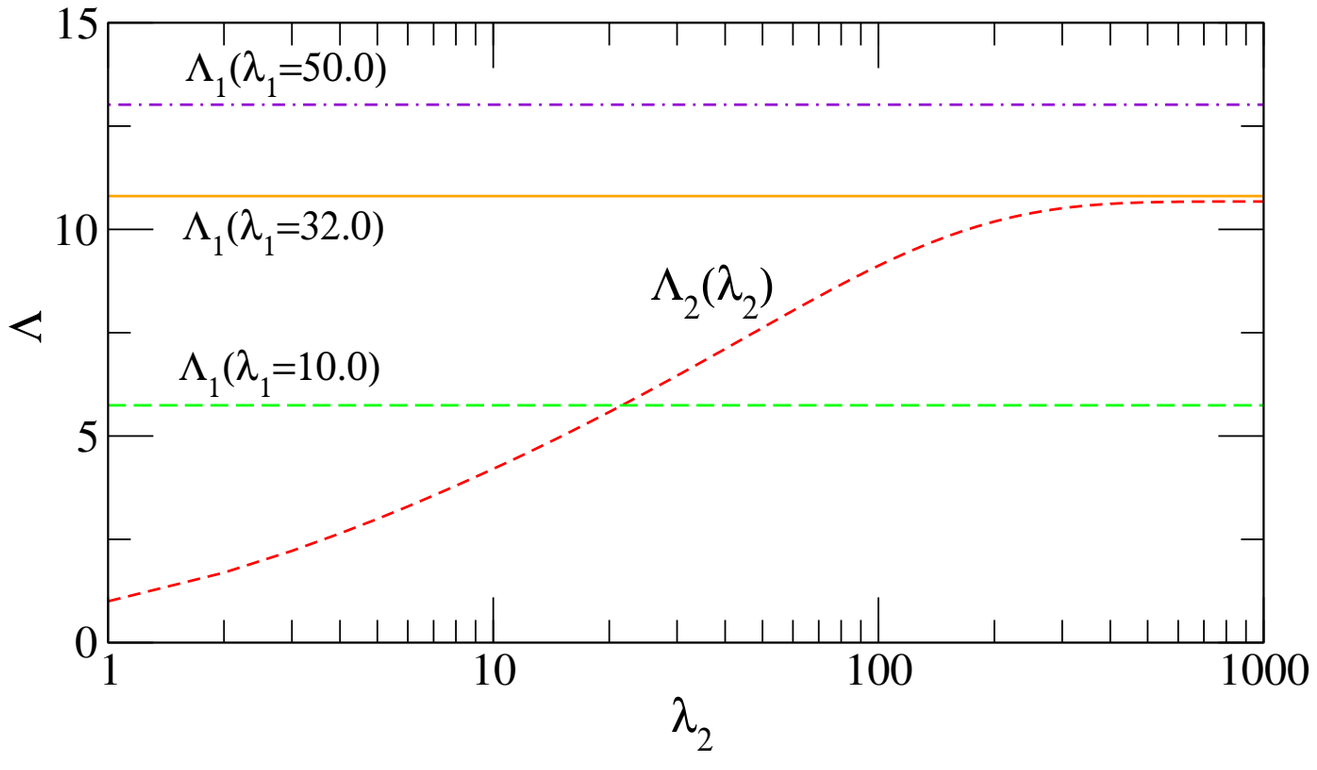
Dominant Layer

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_α:
  a measure of the uncertainty of the interactions

- Dominant layer: least constrained interaction network

The least constrained interaction network drives the social contagion process

G. F. de Arruda et al **Physical Review X 7**, 011014 (2017)

Indeed, one can go a bit more abstract:

(continuous) dynamics on a single layer network:

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

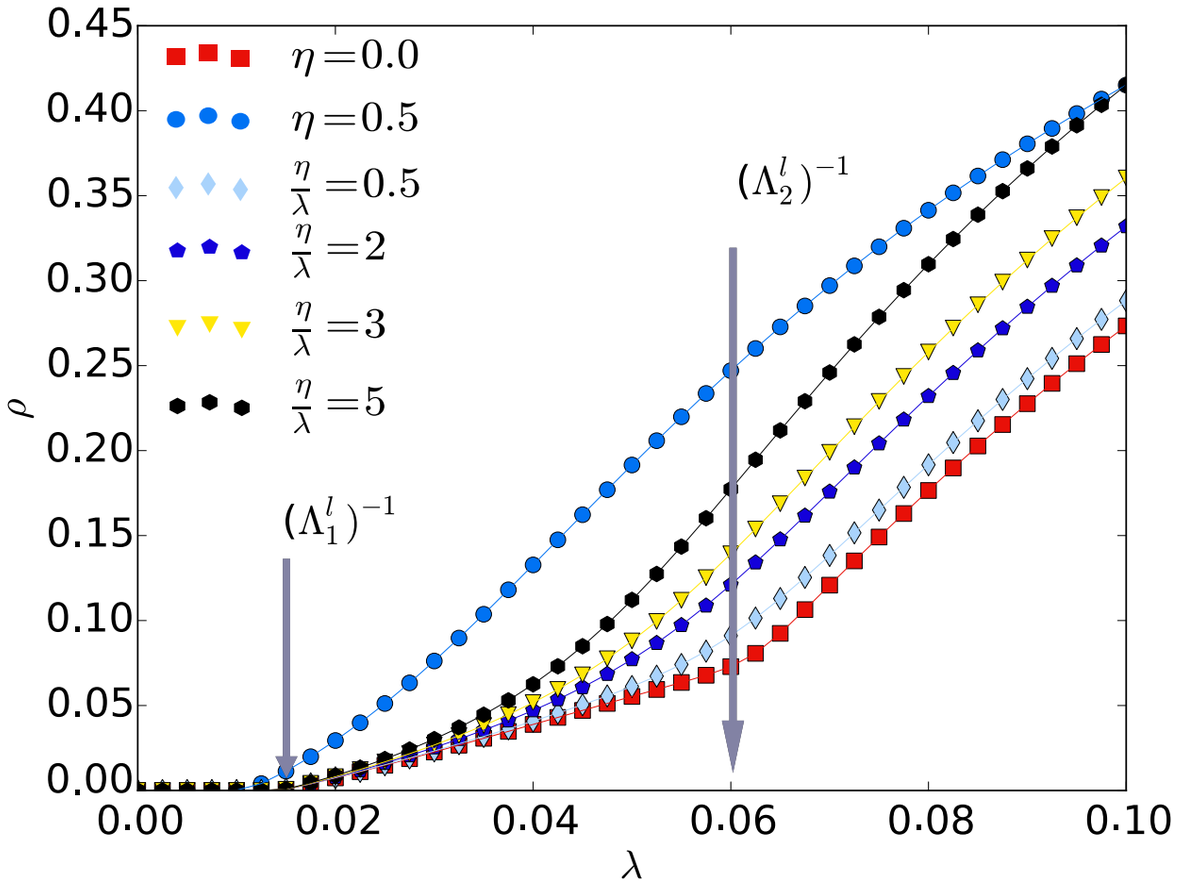
(continuous) dynamics on a multilayer network:

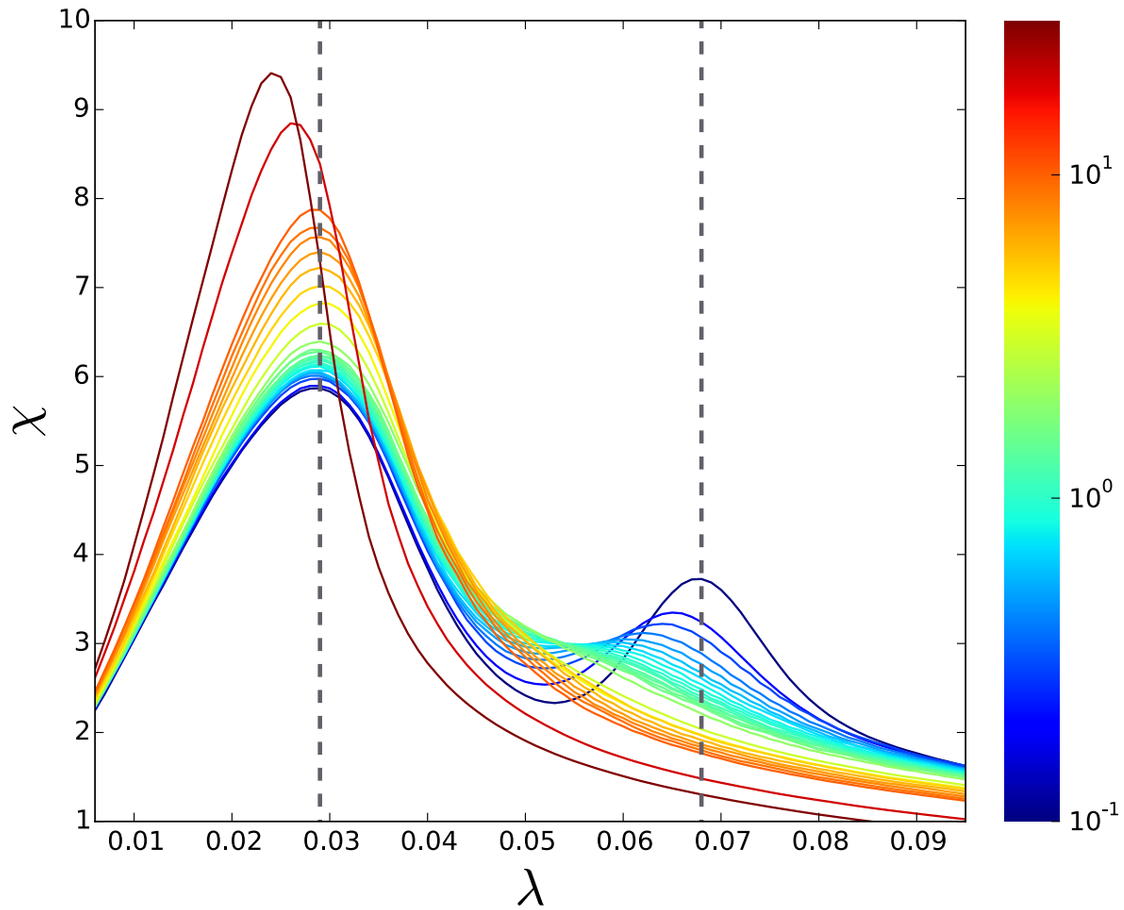
$$\frac{dX_{\beta\tilde{\delta}}}{dt} = -\mu X_{\beta\tilde{\delta}} + (1 - X_{\beta\tilde{\delta}})\lambda \mathcal{R}^{\alpha\tilde{\gamma}}_{\beta\tilde{\delta}}(\lambda,\eta) X_{\alpha\tilde{\gamma}}$$

$$\mathcal{R}^{\alpha\tilde{\gamma}}_{\beta\delta}(\lambda,\eta) = M^{\alpha\tilde{\eta}}_{\beta\tilde{\sigma}} E^{\tilde{\sigma}}_{\tilde{\eta}}(\tilde{\gamma}\tilde{\delta})\delta^{\tilde{\gamma}}_{\tilde{\delta}} + \frac{\eta}{\lambda} M^{\alpha\tilde{\eta}}_{\beta\tilde{\sigma}} E^{\tilde{\sigma}}_{\tilde{\eta}}(\tilde{\gamma}\tilde{\delta})(U^{\tilde{\gamma}}_{\tilde{\delta}} - \delta^{\tilde{\gamma}}_{\tilde{\delta}})$$

intra inter

Dominant layer



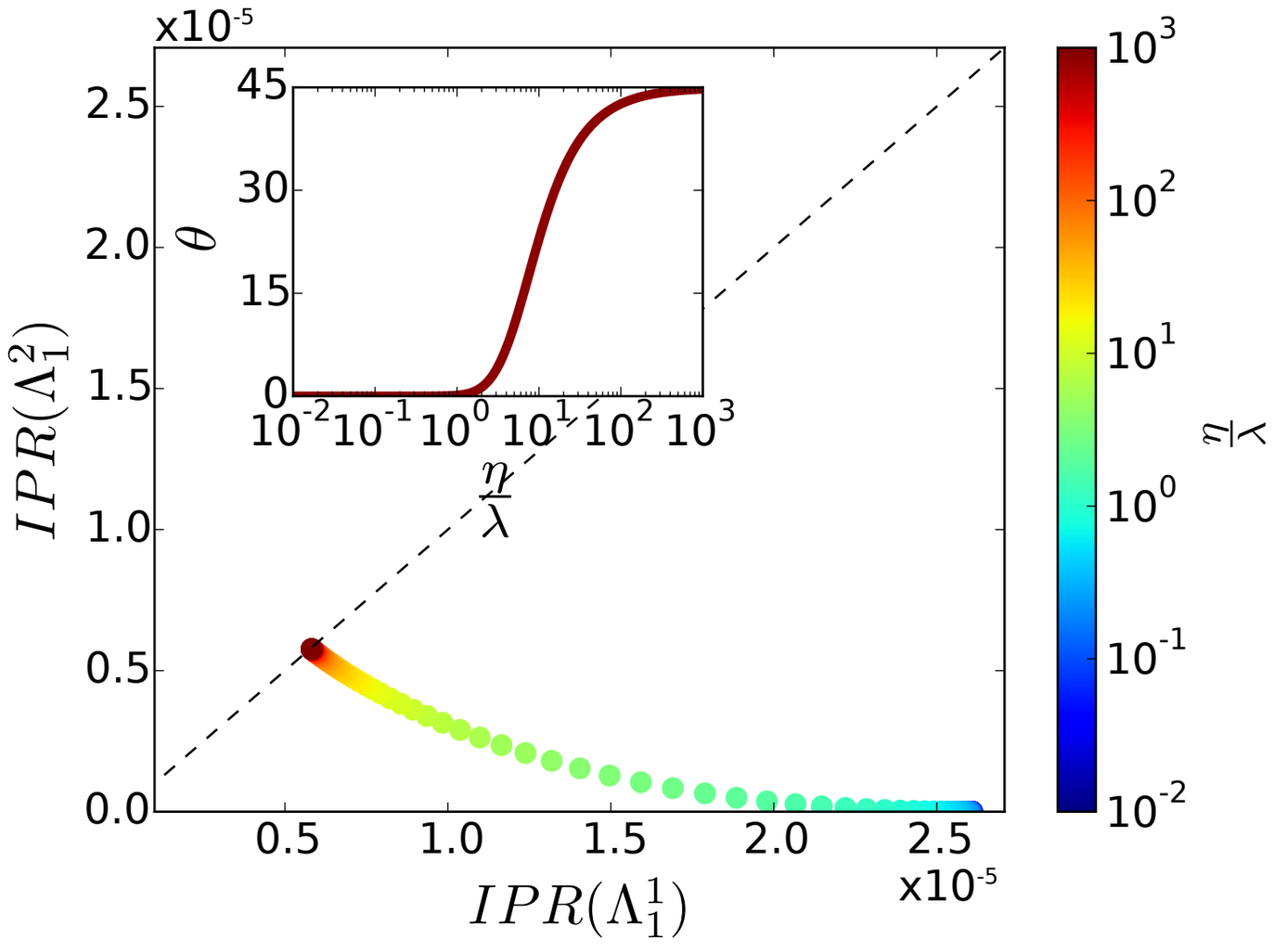


The layer with the largest eigenvalue sets the critical properties of the whole multilayer system

G. F. de Arruda et al **Physical Review X 7**, 011014 (2017)

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

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.



G. F. de Arruda et al **Physical Review X 7**, 011014 (2017)

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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