Structure and dynamics of cities

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Outline – part 2

- Multilayer networks
  - Fragility
  - Anatomy of multimodal trips/Synchronization
  - Optimal coupling

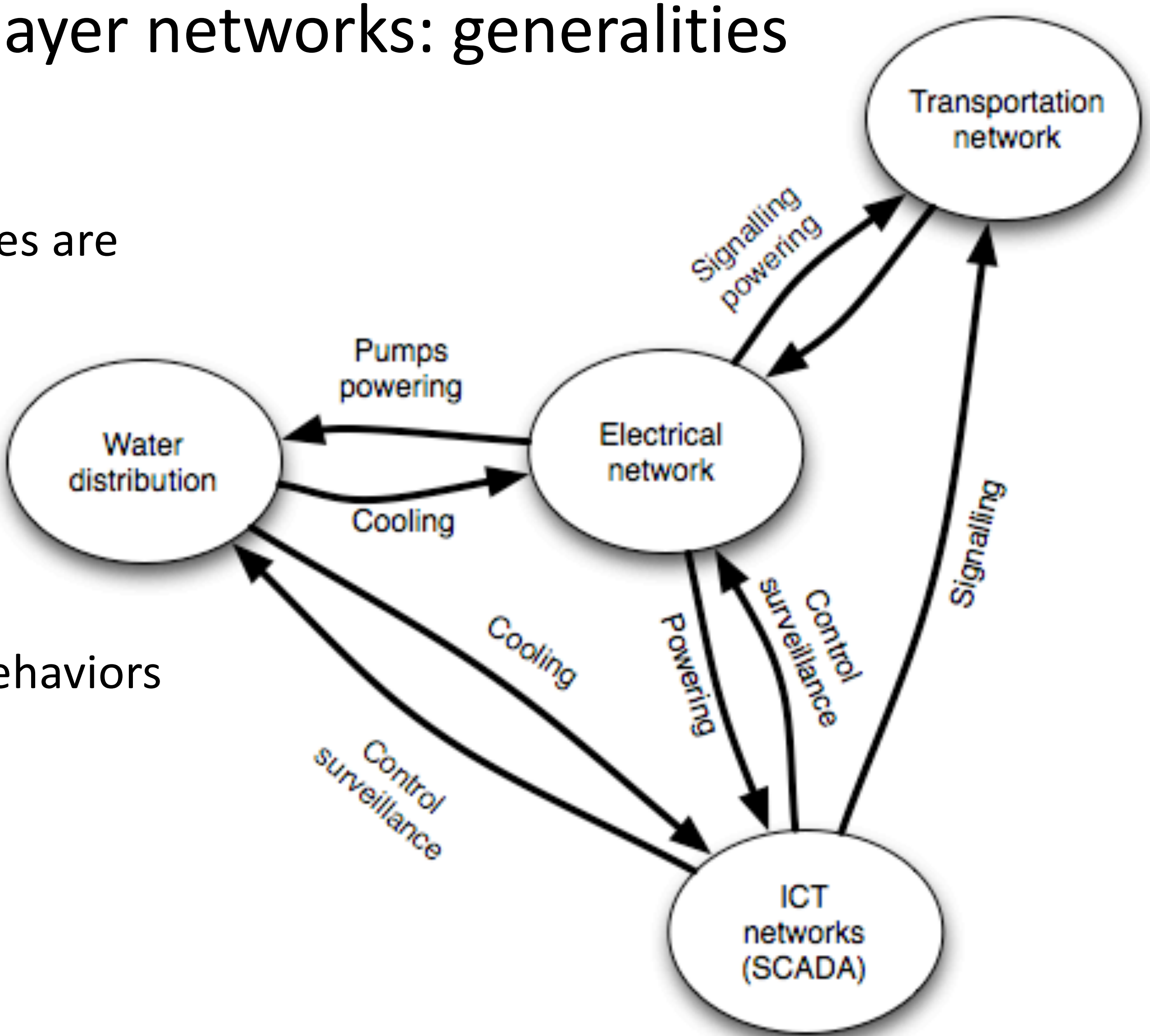
- Spread of infectious diseases
  - Modeling in epidemiology
  - Epidemic on networks and in cities
  - Pandemic spread

Coupling in multilayer networks: generalities

Most critical infrastructures are

- ❑ Networks
- ❑ Coupled

The coupling implies a variety of (unexpected) behaviors



Multilayer networks

- The coupling implies a variety of (unexpected) behaviors, in particular: Enhanced fragility (Buldyrev et al, 2010).

- From a 2nd order transition to a 1st order one

- Shows that it is dangerous to consider networks isolated

H. E. STANLEY

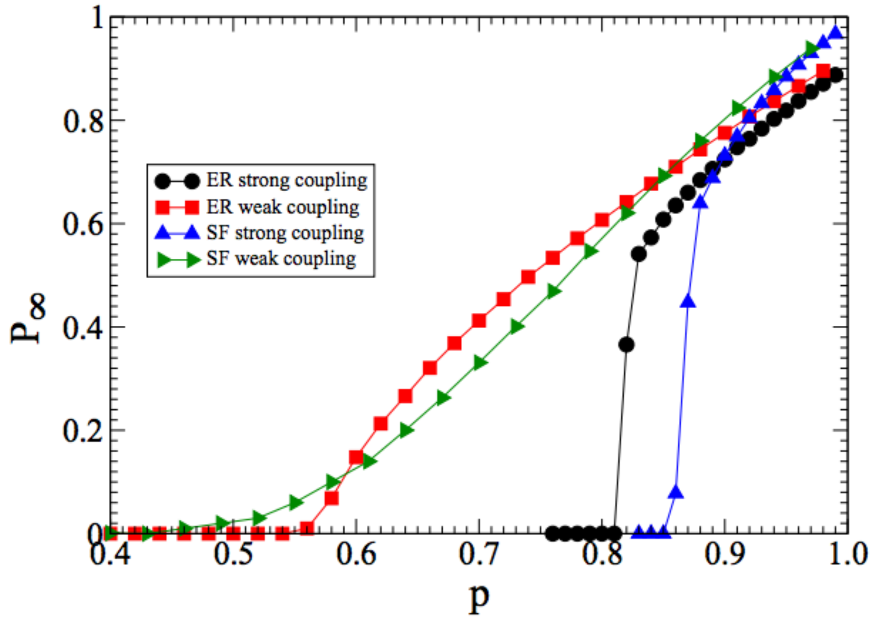
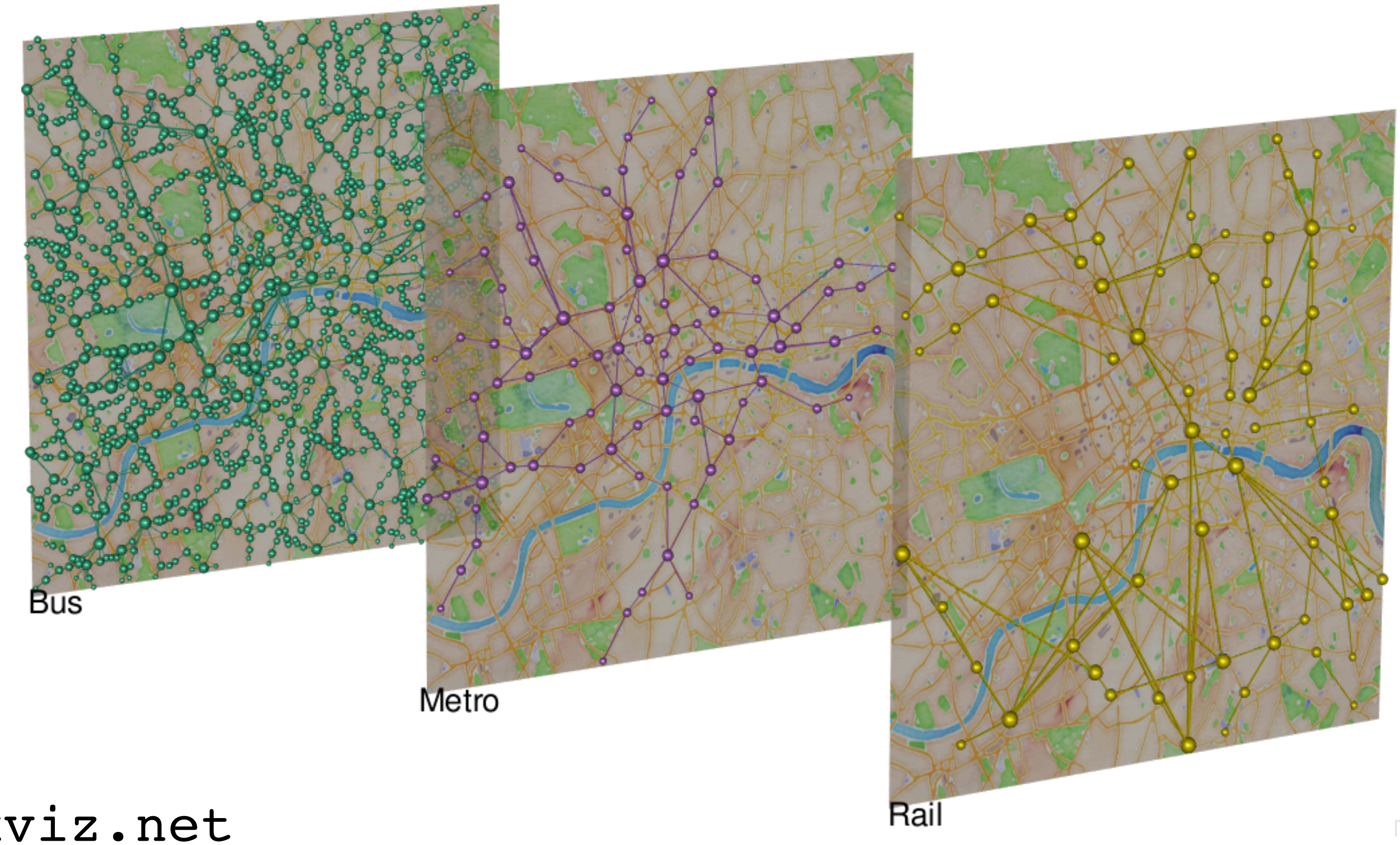


Fig. 8. – Order parameter P_∞ as a function of the fraction of nodes left p for Erdős-Rényi and scale-free network ($\gamma = 2.7$) with strong and weak coupling. Both systems contain 5×10^4 nodes. For both network types, first-order transitions occurs for strong coupling in contrast to second order transition in weak coupling. (After Parshani *et al.* [16]).

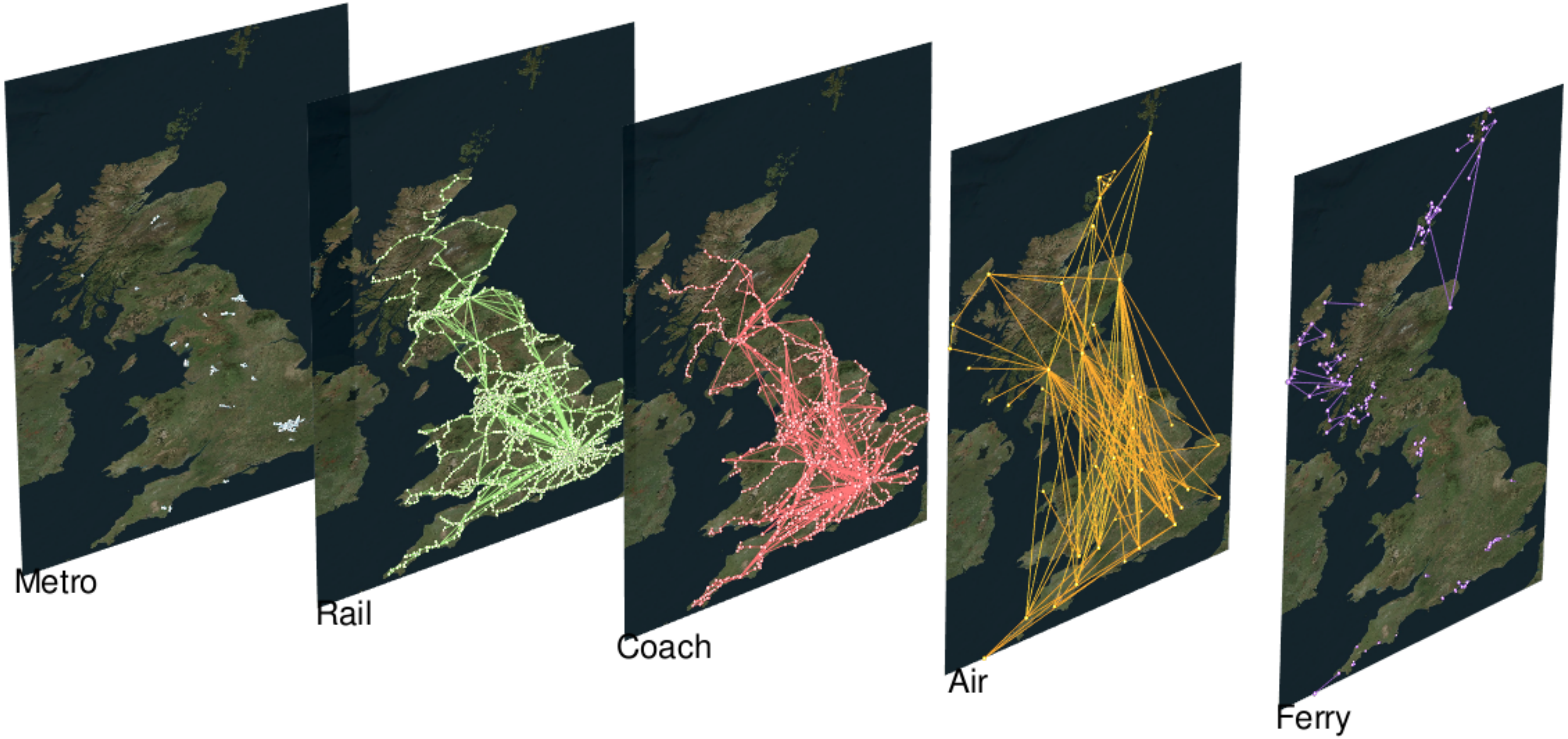
Multilayer transport networks

- Each mode is represented by a layer
- Couplings represent connections between different modes (bus-subway, subway-train, etc). Usually done by walk



MUX VIZ
http://www.muxviz.net

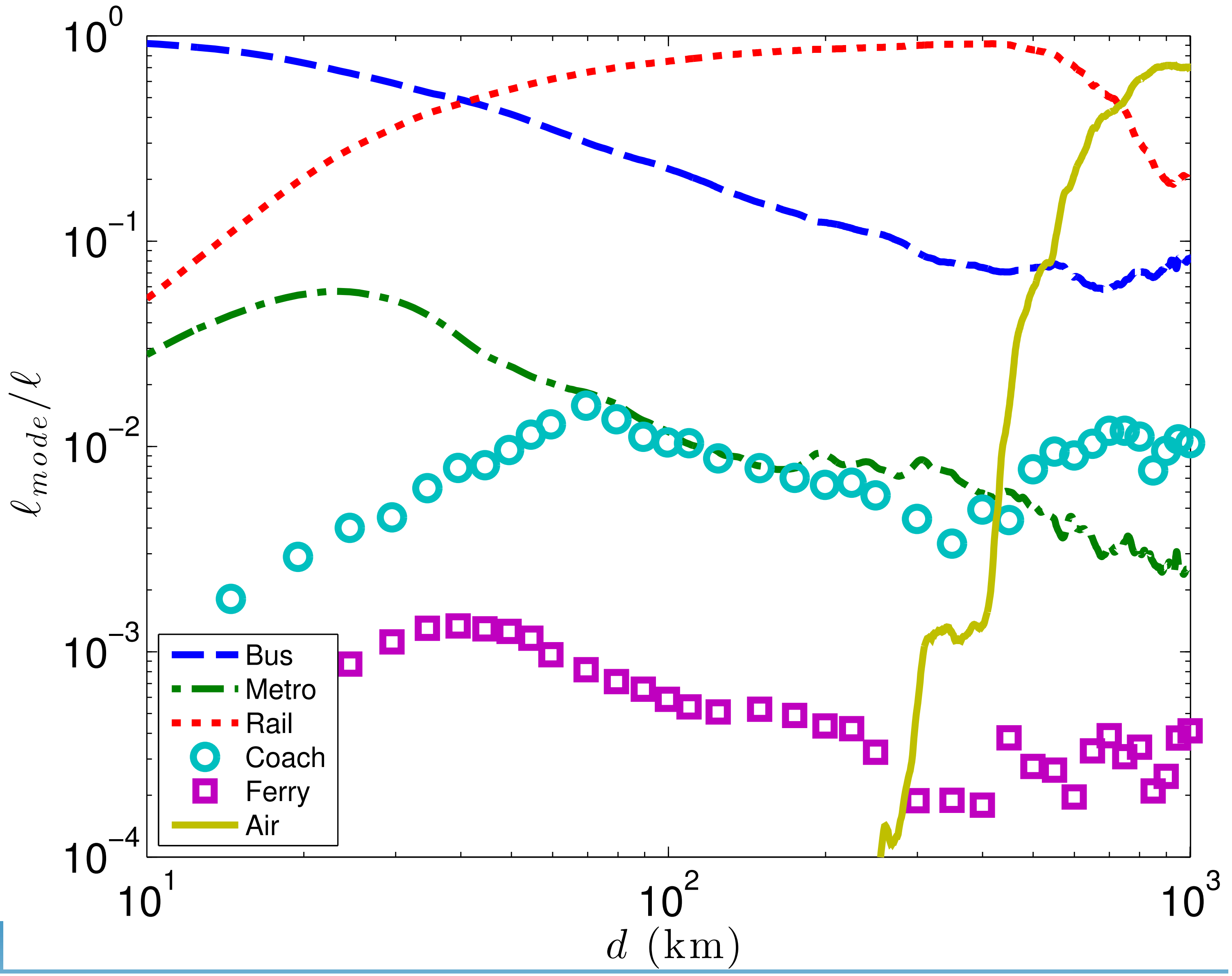
UK Multilayer transport networks



http://www.muxviz.net

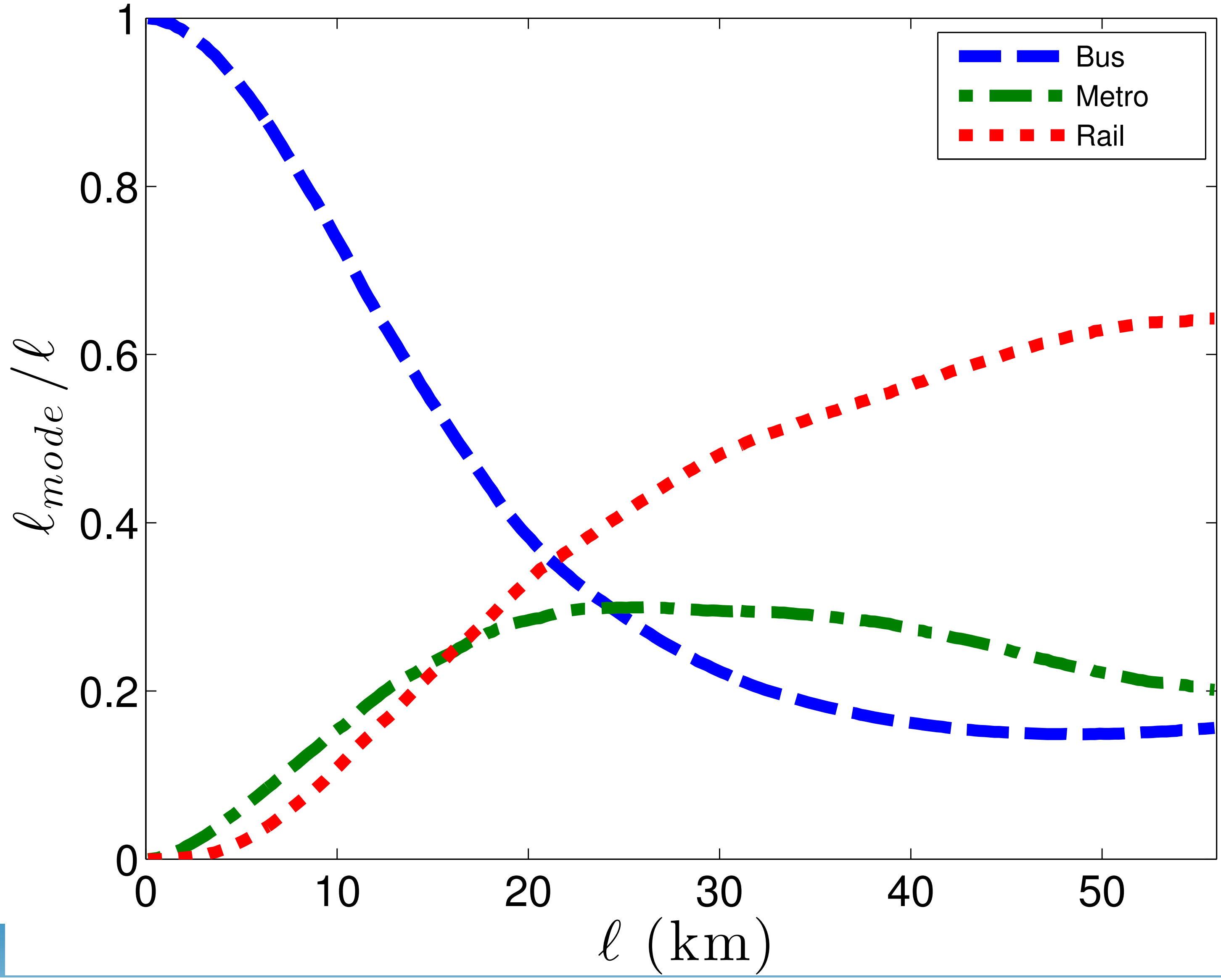
UK Multilayer transport networks

- Modal use (national level)



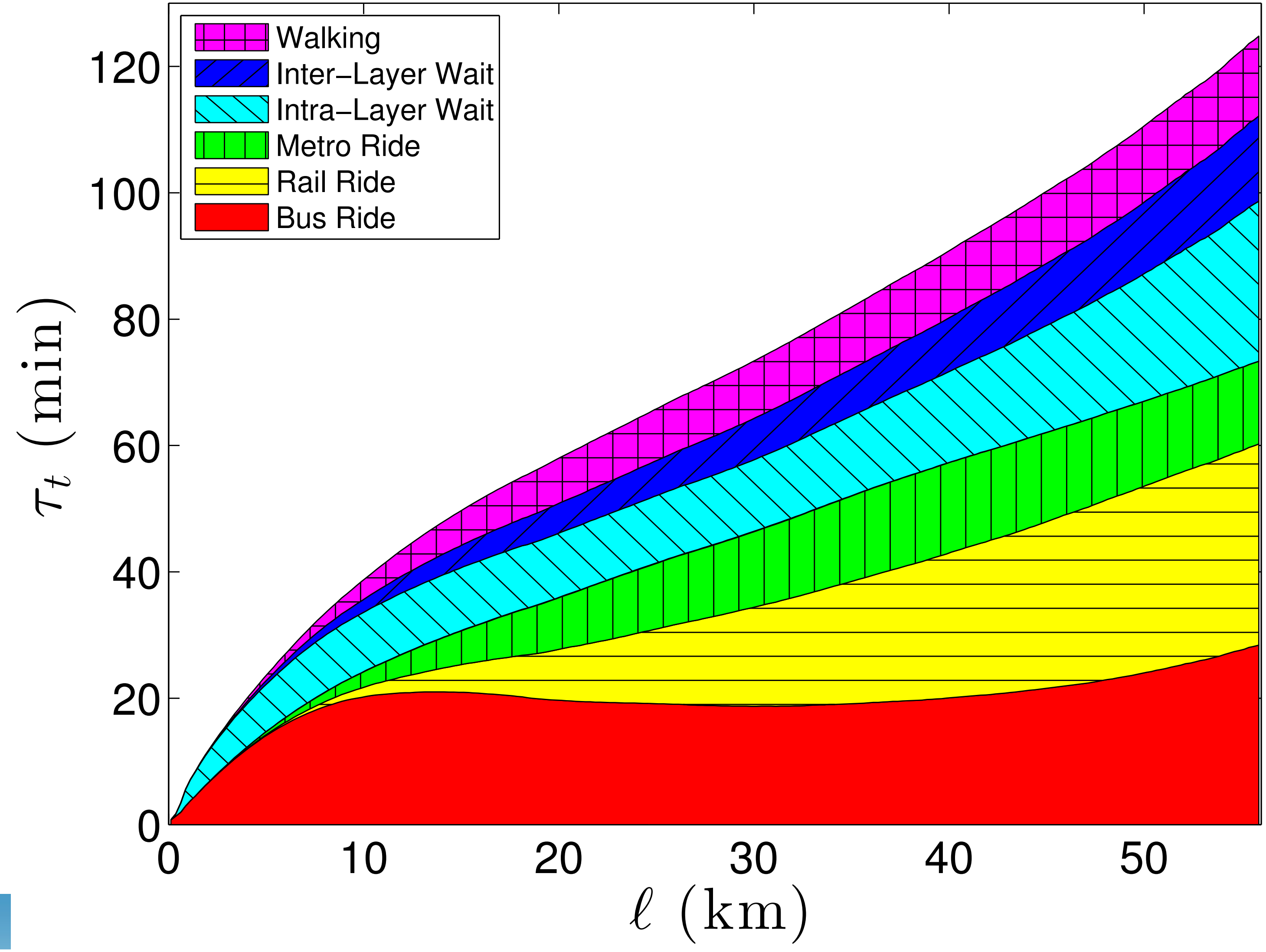
UK Multilayer transport networks

Modal use at the urban level: London



UK Multilayer transport networks

- Anatomy of travel trips (London)

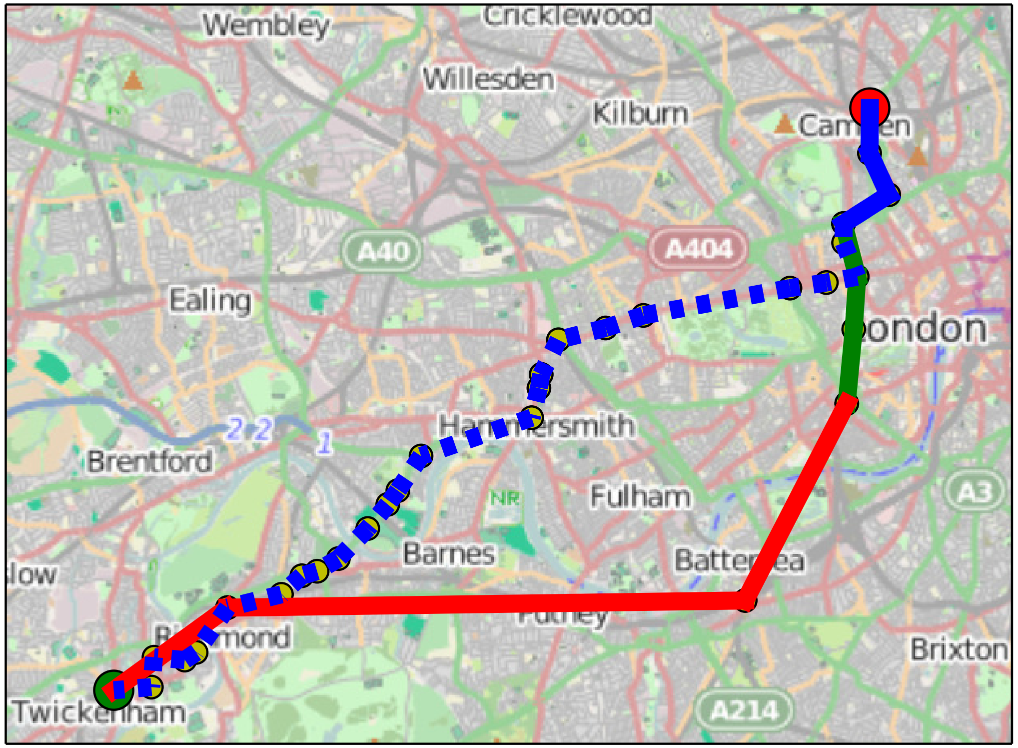


R. Gallotti, MB, 2014

UK Multilayer transport networks

- In a temporal, transportation network, there are many paths and two are important:
  - Quickest path (weights are time)
  - ‘Time-respecting’ quickest path

- The comparison of these paths tells something about the coupling between modes and the efficiency of the system



UK Multilayer transport networks

- The efficiency of synchronization between modes can be characterized by

$$\delta(i,j) = \frac{\tau_t(i,j)}{\tau_m(i,j)} - 1$$

- $\tau_m(i,j)$ Travel time on the quickest path
- $\tau_t(i,j)$ Travel time on the time-respecting path
- Average $\delta(\ell)$
  - Maximal for short trips
  - Decreases with the length ℓ of trips

R. Gallotti, MB, 2014

UK Multilayer transport networks

- What controls the average $\overline{\delta}$?

- The efficiency of the system should be related to the frequency of stops and the number of layers.

- A natural global quantity characterizing the transportation system of a city is then the number of stop events per unit time

$$\Omega = \frac{\sum_\alpha \Omega_\alpha}{\Delta t}$$

where Ω_α is the number of stops in the layer α during Δt

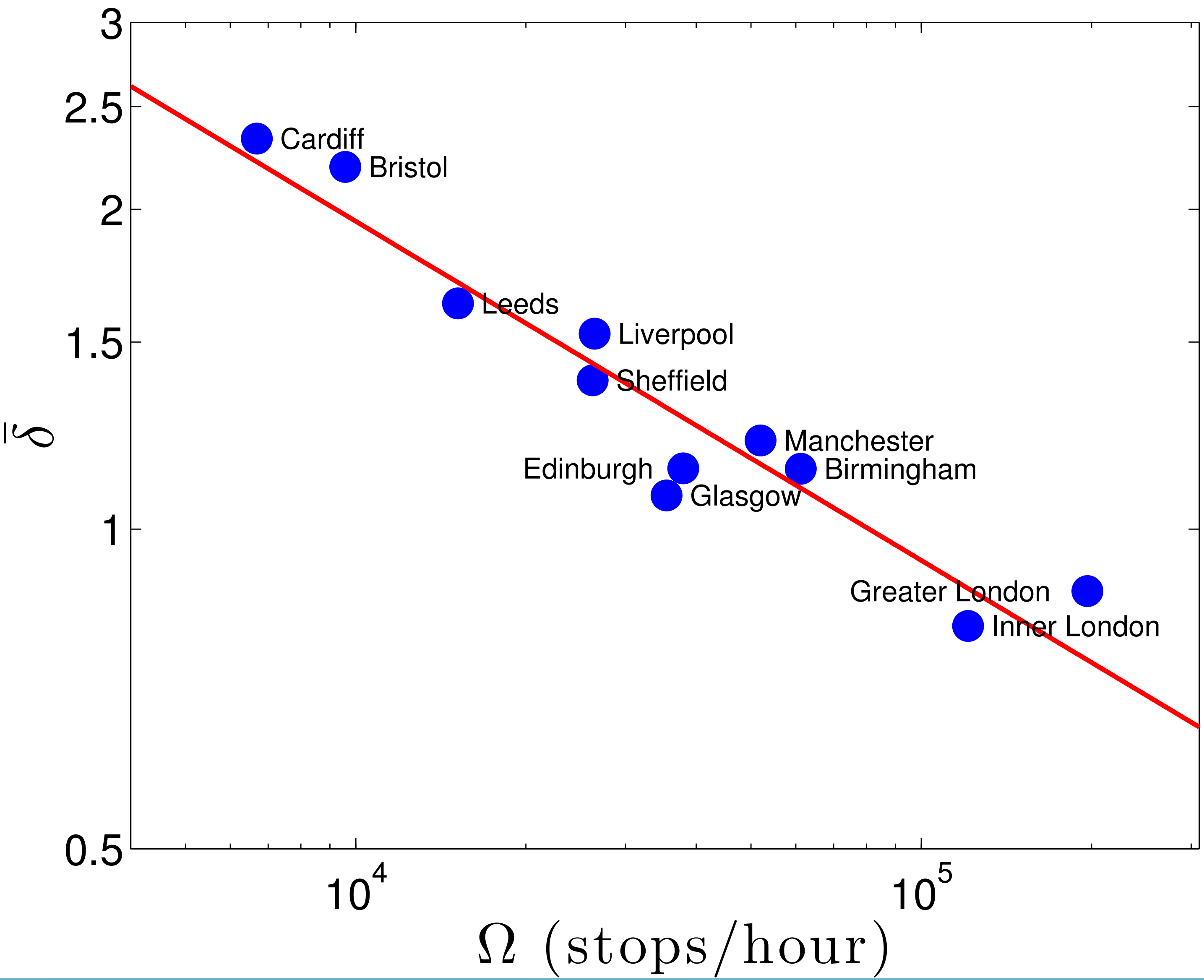
Multilayer transport networks: efficiency

- What controls $\overline{\delta}$?

$$\overline{\delta} \sim \Omega^{-\mu}$$

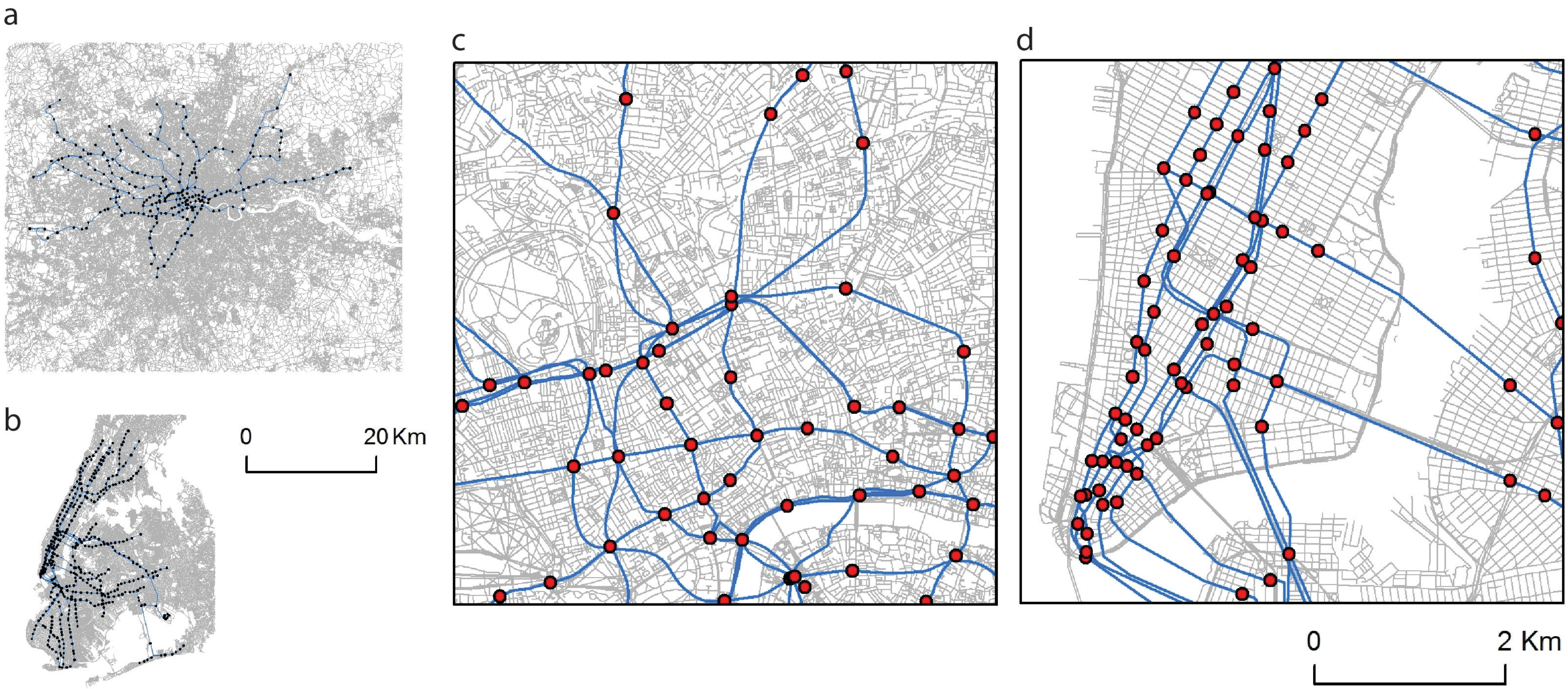
$$\text{with} \quad \mu \approx 0.3$$

- The small value of μ indicates a poor optimization !



R. Gallotti, MB, 2014

Another example: comparison of London and NYC (Streets+subway)



Streets+subway: London and NY

$$\text{nodes(subway)} \subset \text{nodes(street)}$$

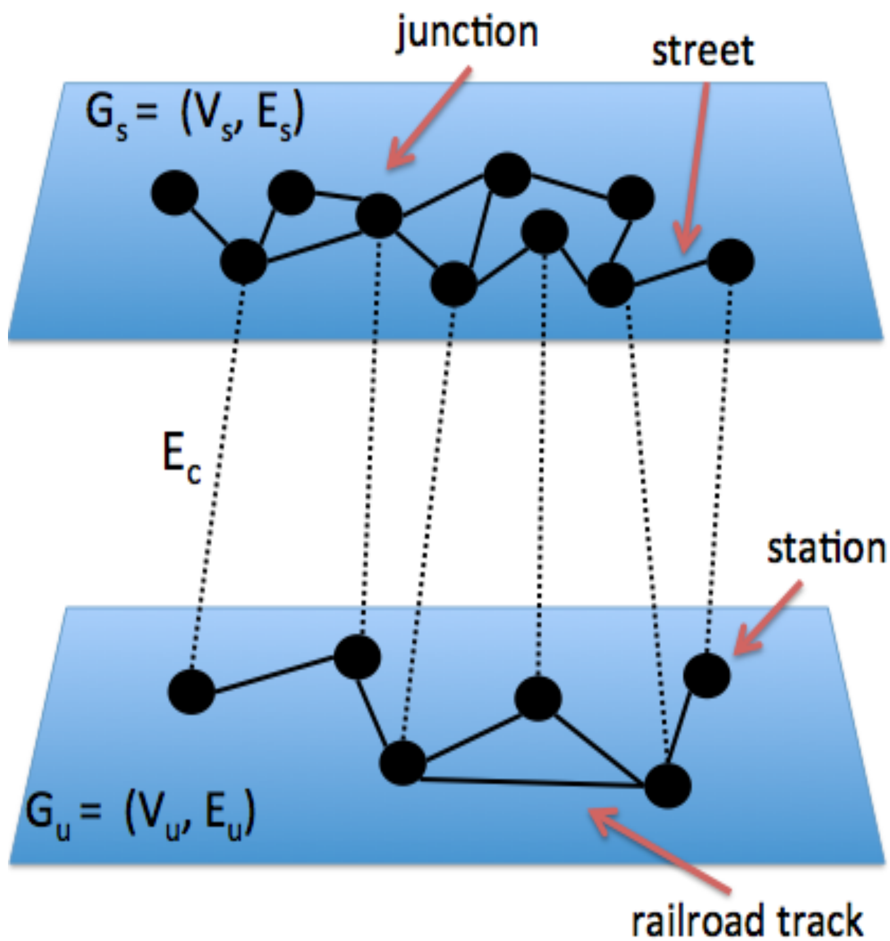
$$\Rightarrow \text{“Multiplex”}$$

- Street network- average velocity:

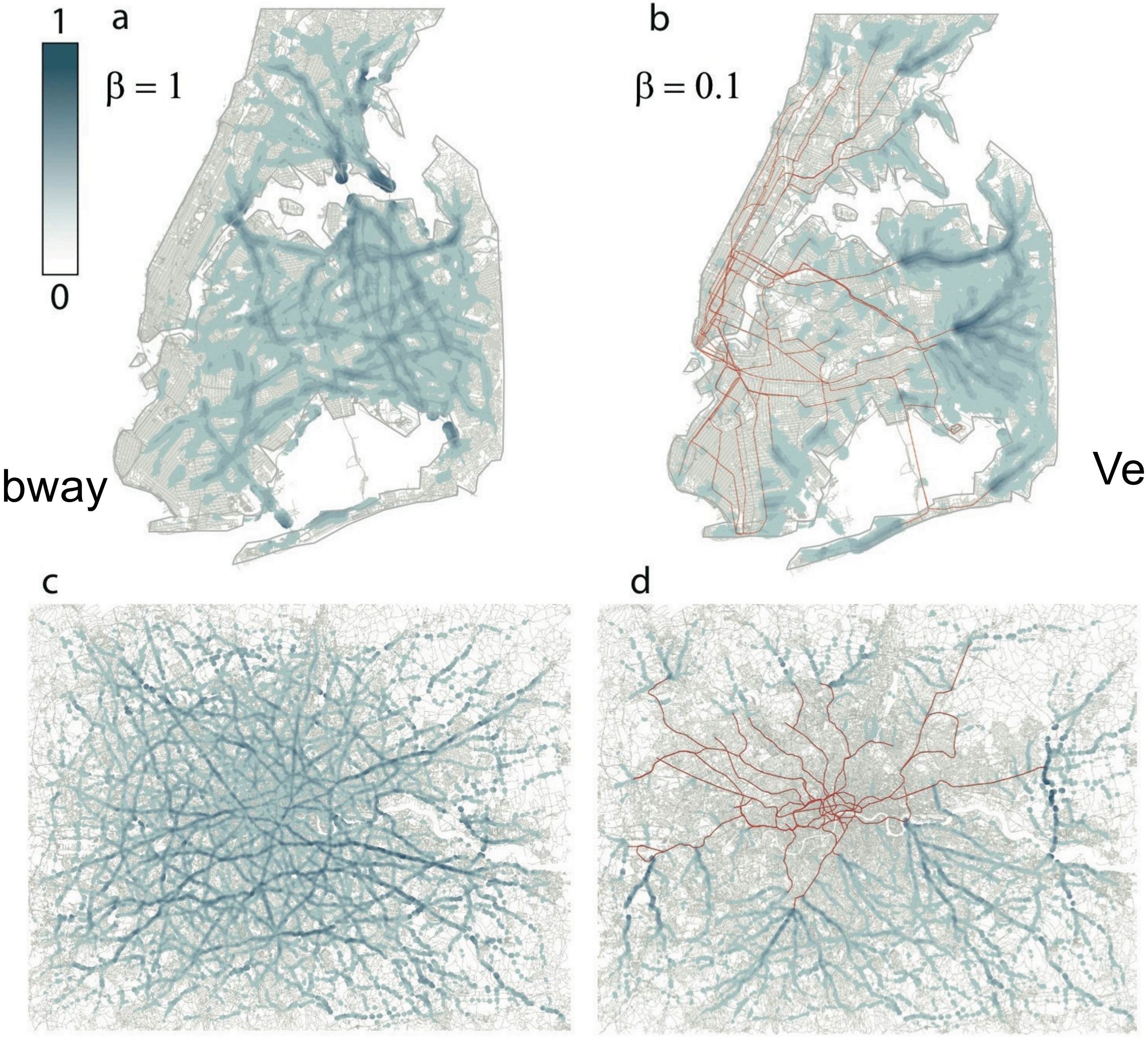
$$v$$

- Subway network (faster):

$$v/\beta \quad \text{with} \quad \beta < 1$$



Spatial distribution of traffic (bc)

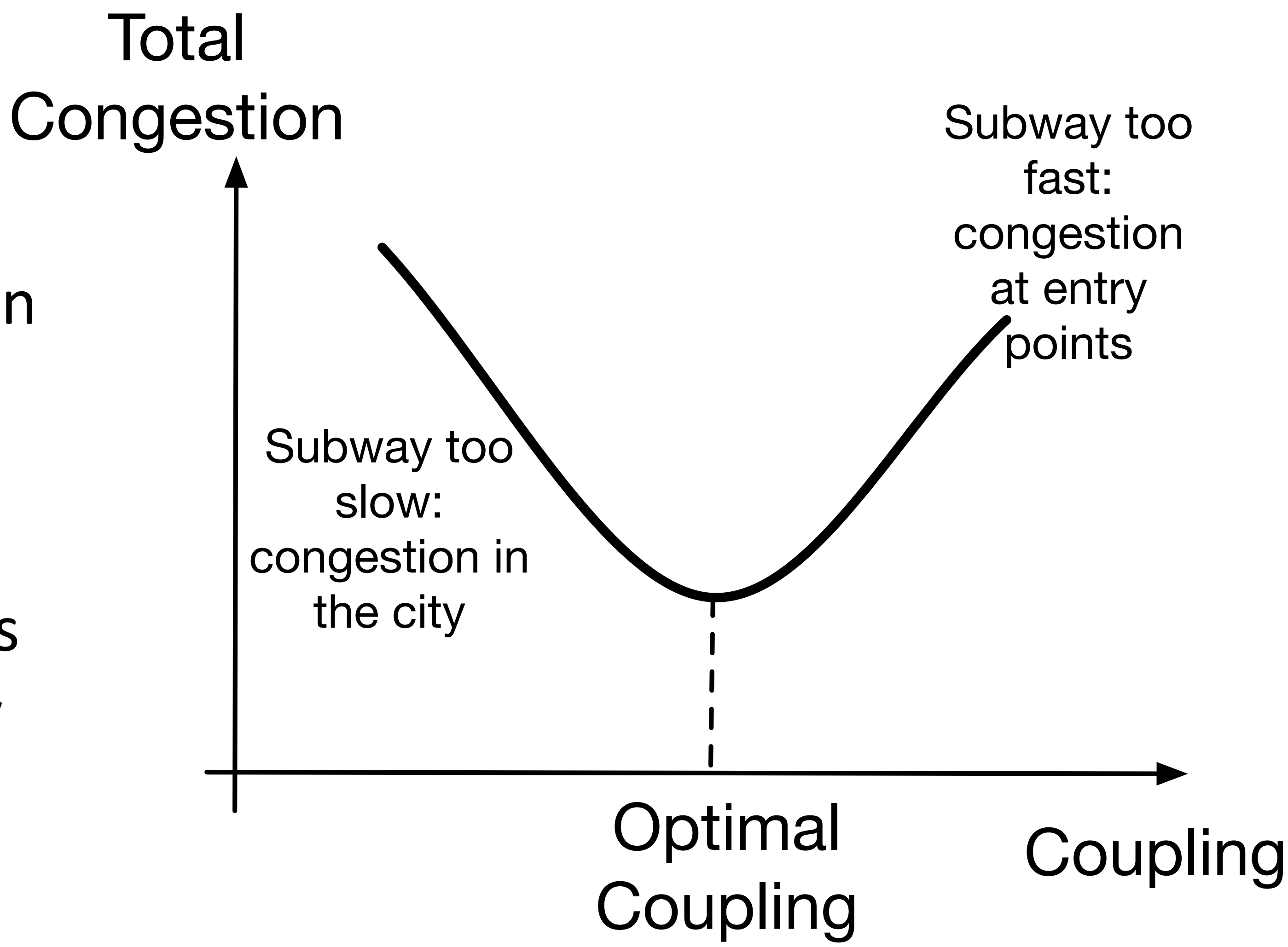


Slow subway

Very fast subway

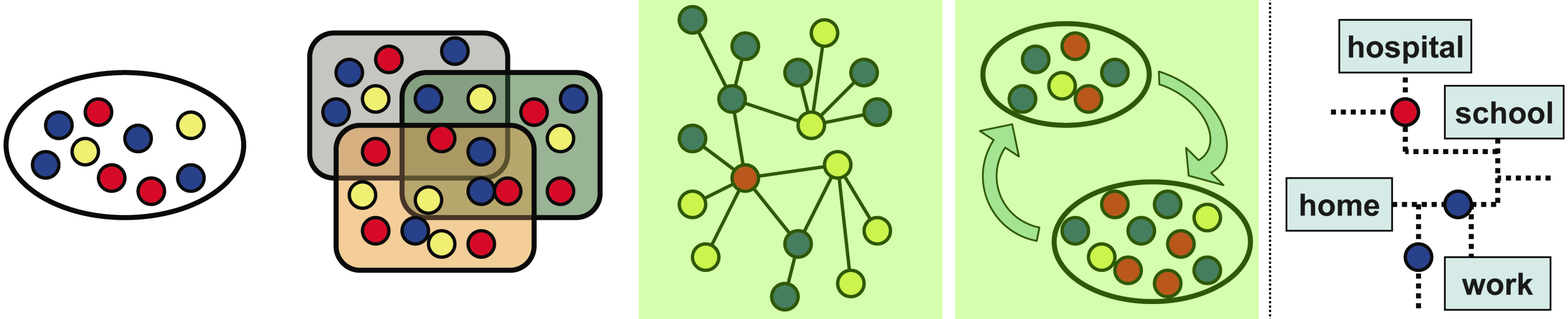
Nontrivial optimal coupling

Trade-off between congestion on roads and congestion in intermodal points : optimal velocity for the subway



Morris and Barthelemy *PRL* **109**, 128703, 2013).

Modeling in Epidemiology



Homogeneous mixing

Social structure

Contact network models

Multi-scale models

Agent Based models

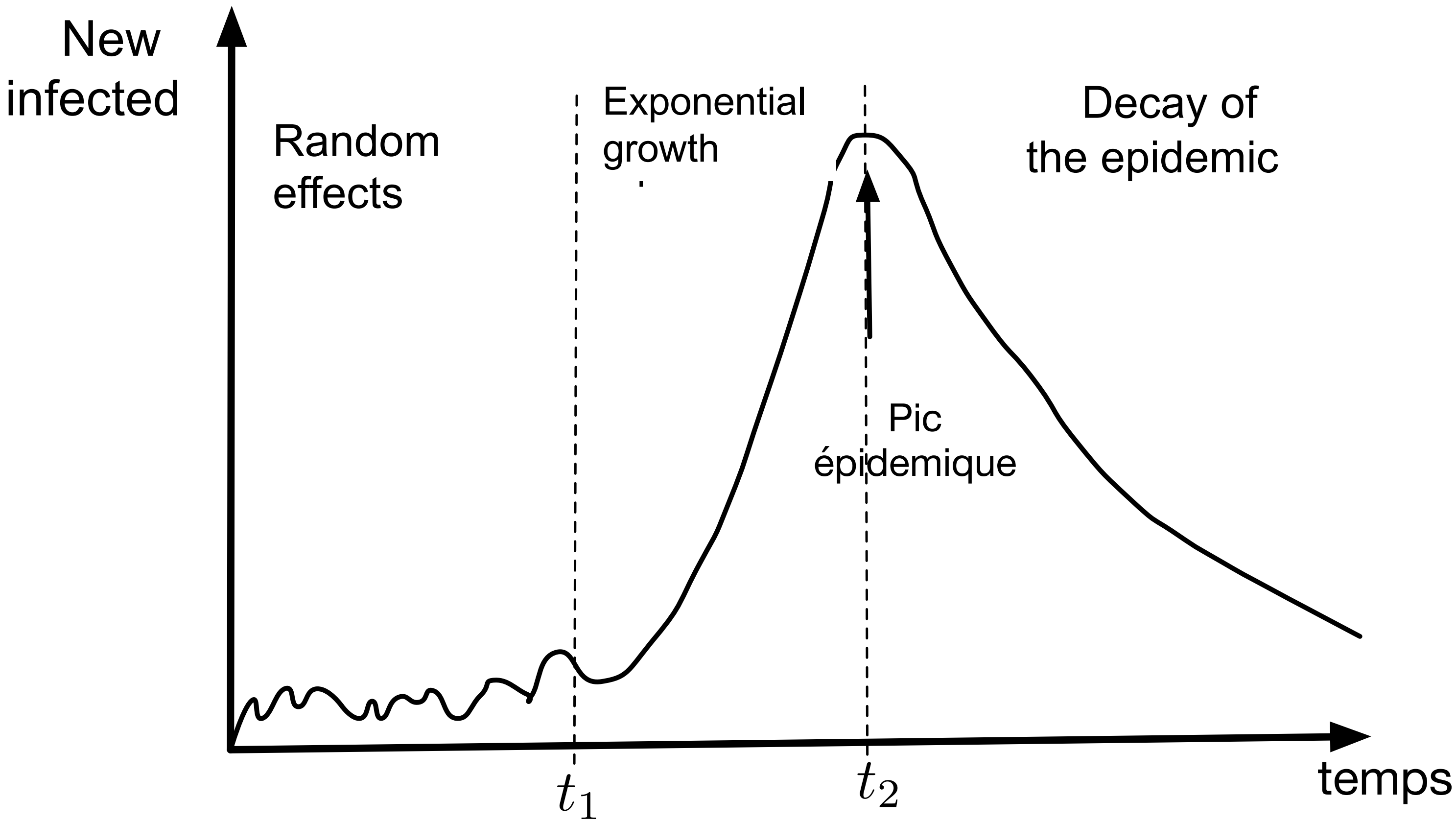
Theoretical Studies
(pre-network era)

Introducing some realism
(city/country level)

Different approximations at different scales

Level of details?
Validity ?

General dynamics of spreading



- Natural variables: number of infected I(t), susceptibles S(t)
- New cases per unit time: attack rate (incidence)
- Total cases=prevalence

Simple Models of Epidemics

Stochastic compartmental model:

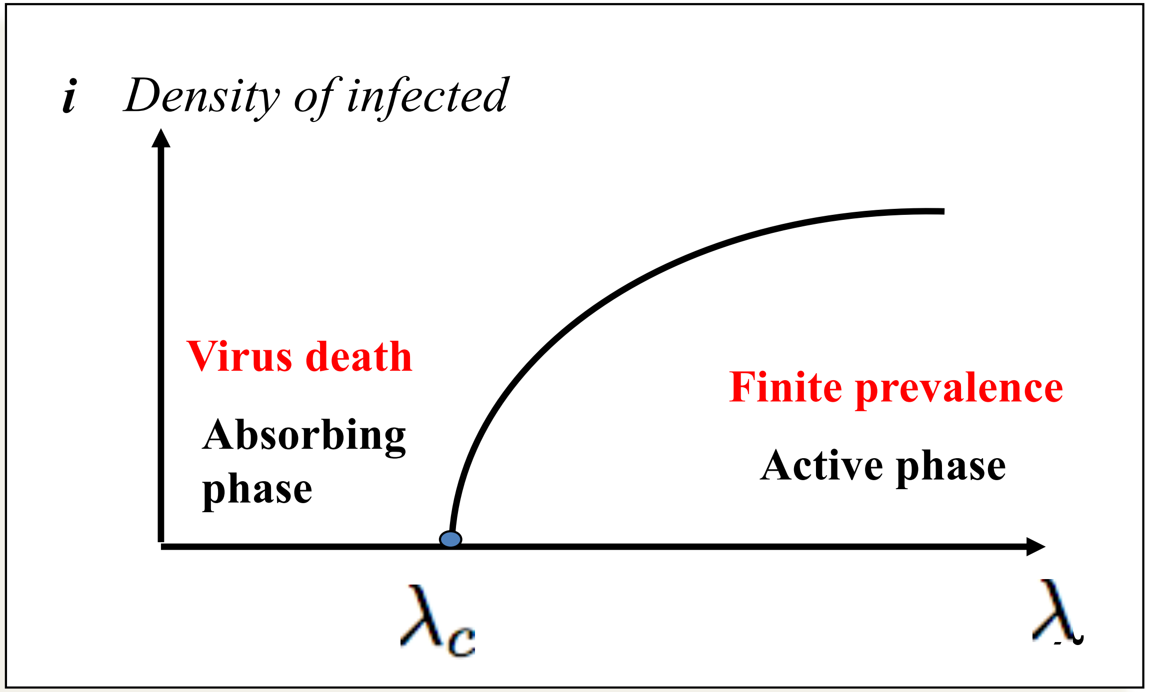
- SIS model: $$S \xrightarrow{\lambda} I \xrightarrow{\mu} S$$
- SIR model: $$S \xrightarrow{\lambda} I \xrightarrow{\mu} R$$
- SI model: $$S \xrightarrow{\lambda} I$$

λ: proba. per unit time of transmitting the infection

μ: proba. per unit time of recovering

Epidemic Threshold λ_c

The epidemic threshold is a general result (SIS, SIR,…)



- Epidemic threshold = critical point
- Prevalence i =order parameter

The question of thresholds in epidemics is central

Basic reproductive number

- Assume a homogeneously mixed population
- Introduce one infected in a population of susceptibles
- Average number of secondary cases: R_0
- Evolution of the number of newly infected:

$$I_{new}(t+1) = I_{new}(t) * R_0$$
$$\Rightarrow I_{new}(t) = I_0 R_0^t$$

- If R_0>1 the epidemic spreads. This is equivalent to $\lambda > \lambda_c$

1. Epidemic on contact networks

Epidemics on networks

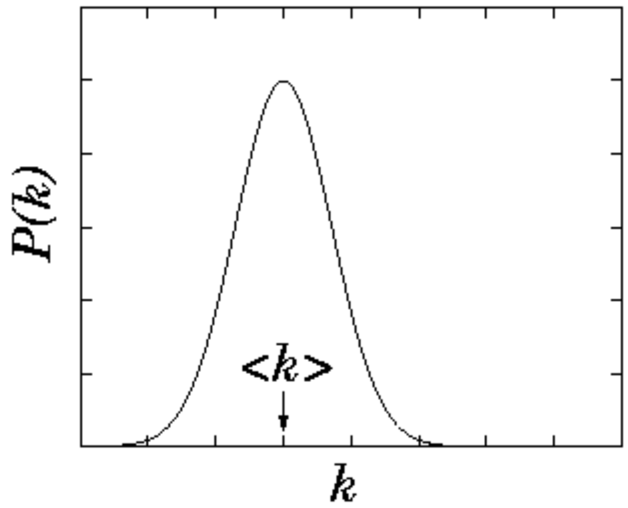
Contact network: the pattern of contacts along which infections spread in population is identified by a network

- Each node represents an individual
- Each link is a connection along which the virus can spread

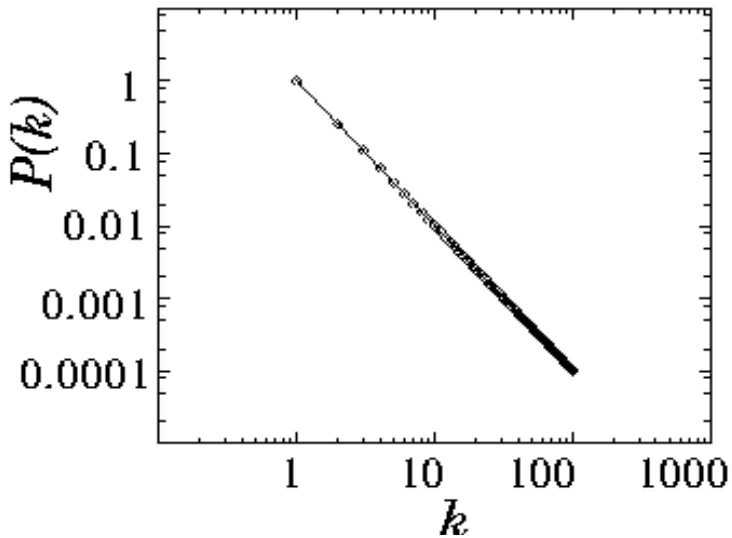
Two classes of networks: degree distribution

Degree k = number of neighbors; Distribution P(k)

Exponentially decreasing distribution



Power-law distribution



"Homogeneous" networks
Existence of constraints
(age, cost, space, …)

$$\lambda_c = \frac{\mu}{\langle k \rangle}$$

"Scale-free" networks
Existence of hubs with
very large degrees

$$\lambda_c \sim \frac{\langle k \rangle}{\langle k^2 \rangle} \to 0$$

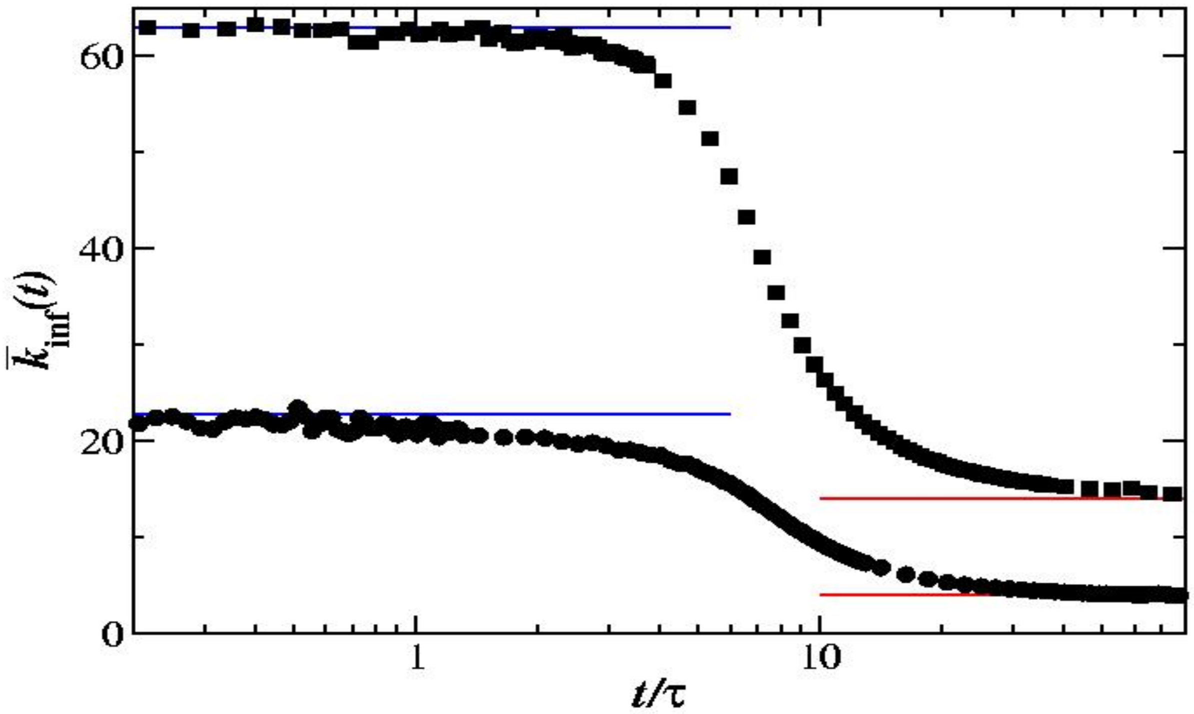
Consequence: immunization

- When hubs are absent: random immunization is working

- For scale-free network ($\lambda_c \sim 0$) with hubs
random immunization is ineffective: targeted
strategy needed ! (find the hubs)

Dynamics: Cascade

Which nodes are infected ?
What is the infection scenario ?



Seeds -> Hubs -> Intermediate -> Small k

Modeling epidemic spread in cities

Modelling disease outbreaks in realistic urban social networks

Stephen Eubank[1], Hasan Guclu[2], V. S. Anil Kumar[1], Madhav V. Marathe[1], Aravind Srinivasan[3], Zoltán Toroczkai[4] & Nan Wang[5]

Nature **429**, 180–184(2004)

Construction (mostly with simulations) of the contact network among people and movemements between locations

Existence of hub (locations highly visited): allows highly efficient outbreak detection by placing sensors at these locations

Enable to analyse the merits of proposed mitigation strategies (smallpox spread): Outbreaks can be contained by a strategy of targeted vaccination combined with early detection (without resorting to mass vaccination)

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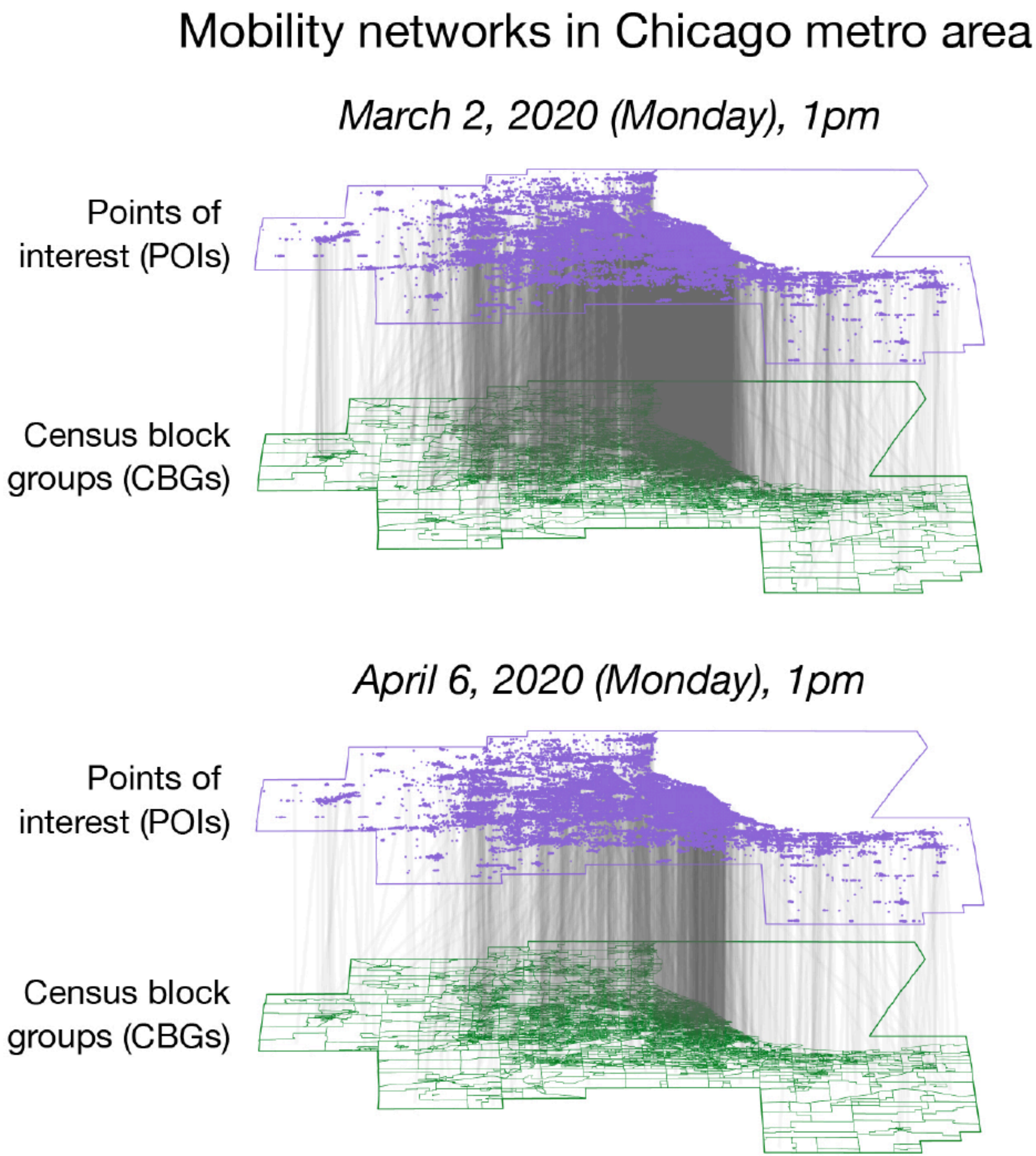
Modeling epidemic spread in cities

Cell phone data allows to construct the mobility network: individuals from a census block to a point of interest (restaurants, etc)

98 million people
57k CBGs to 553k POIs
with 5.4 billion hourly edges

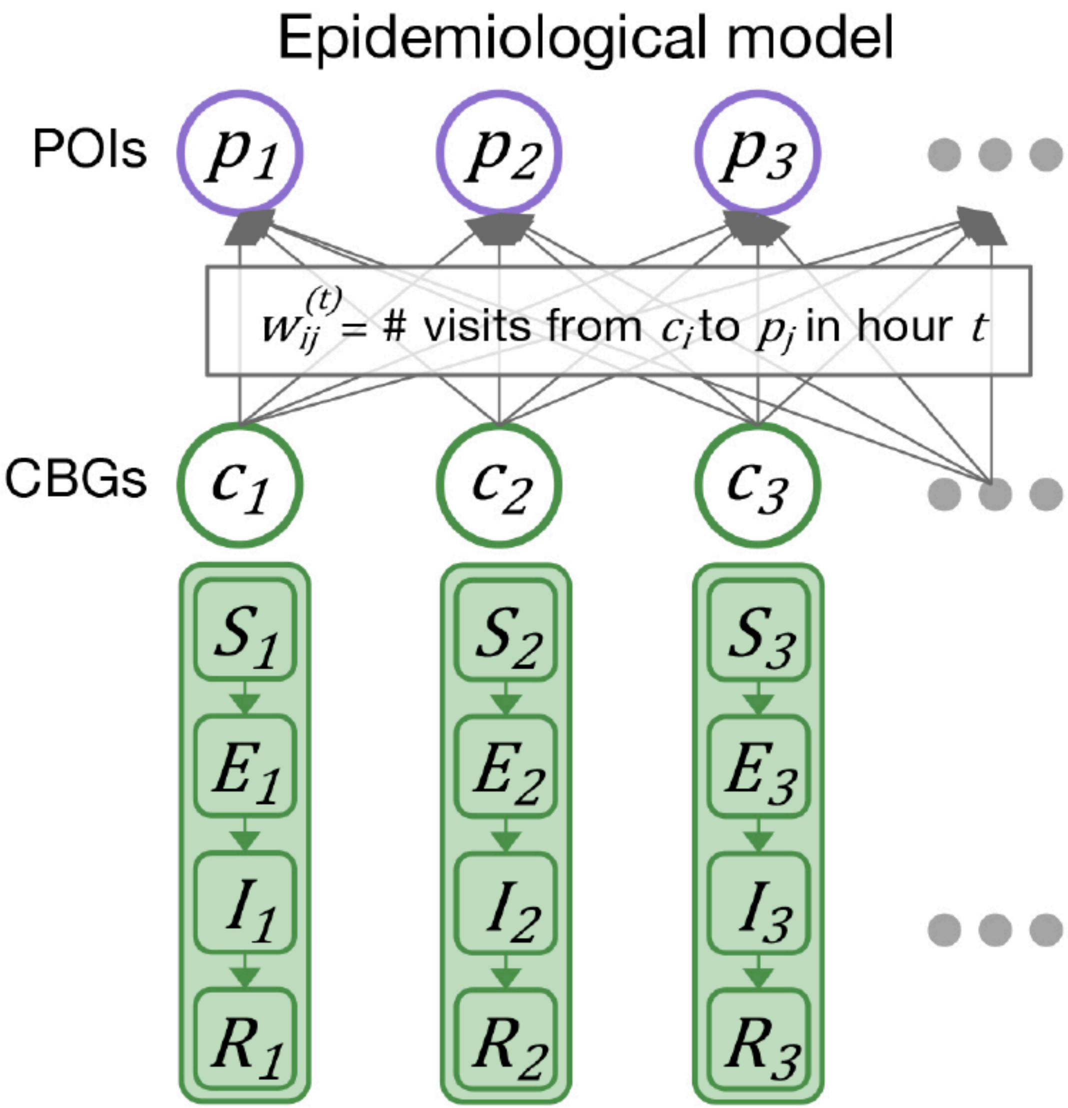


Chang et al, Nature 2020

Modeling epidemic spread in cities

Use this mobility data for constructing an epidemiological model

- Able to reproduce observations
- Shows that a small minority of "superspreader" POIs account for a large majority of infections
- Restricting maximum occupancy at each POI is more effective than uniformly reducing mobility

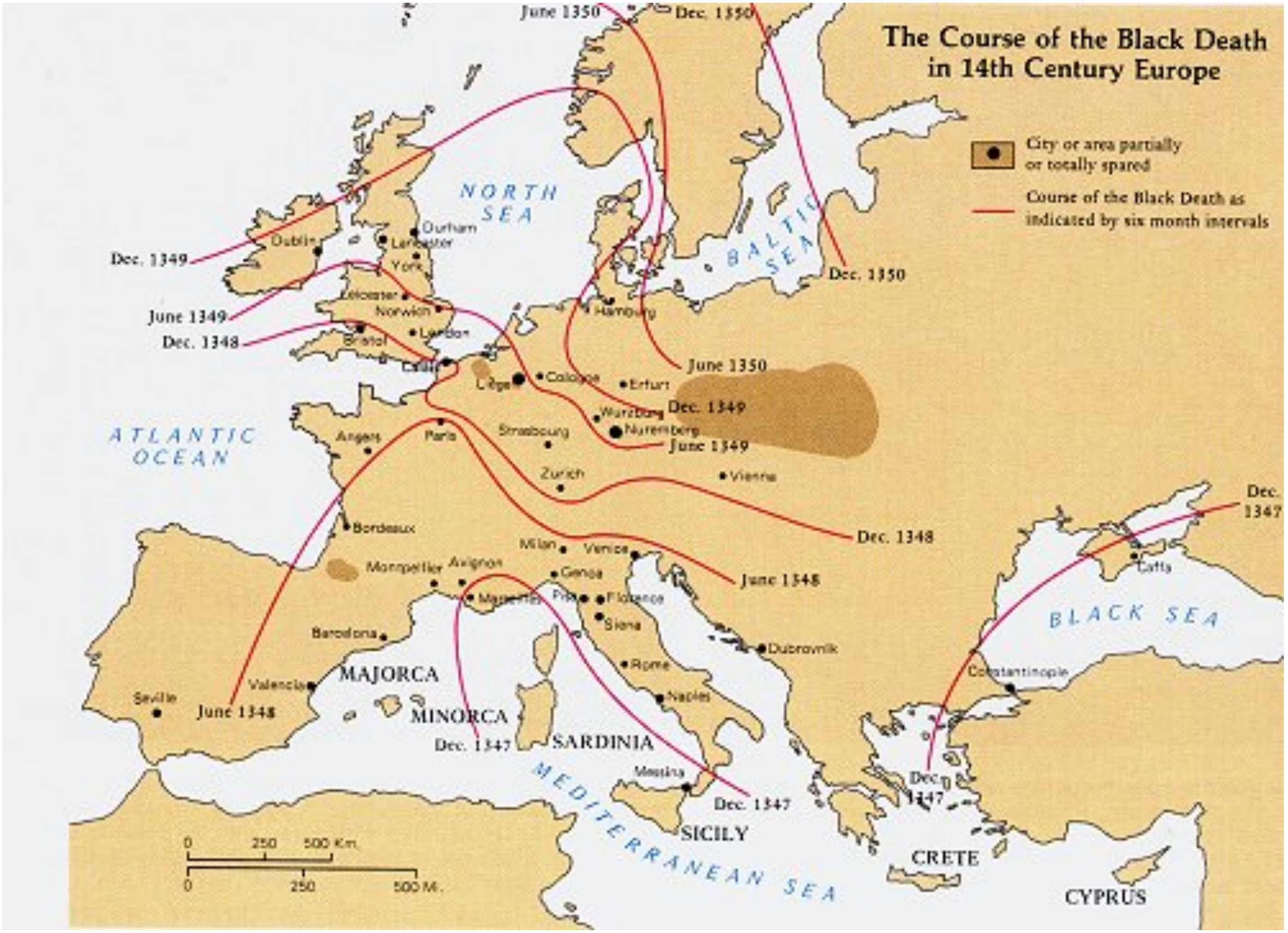


2. Metapopulation models (between cities)

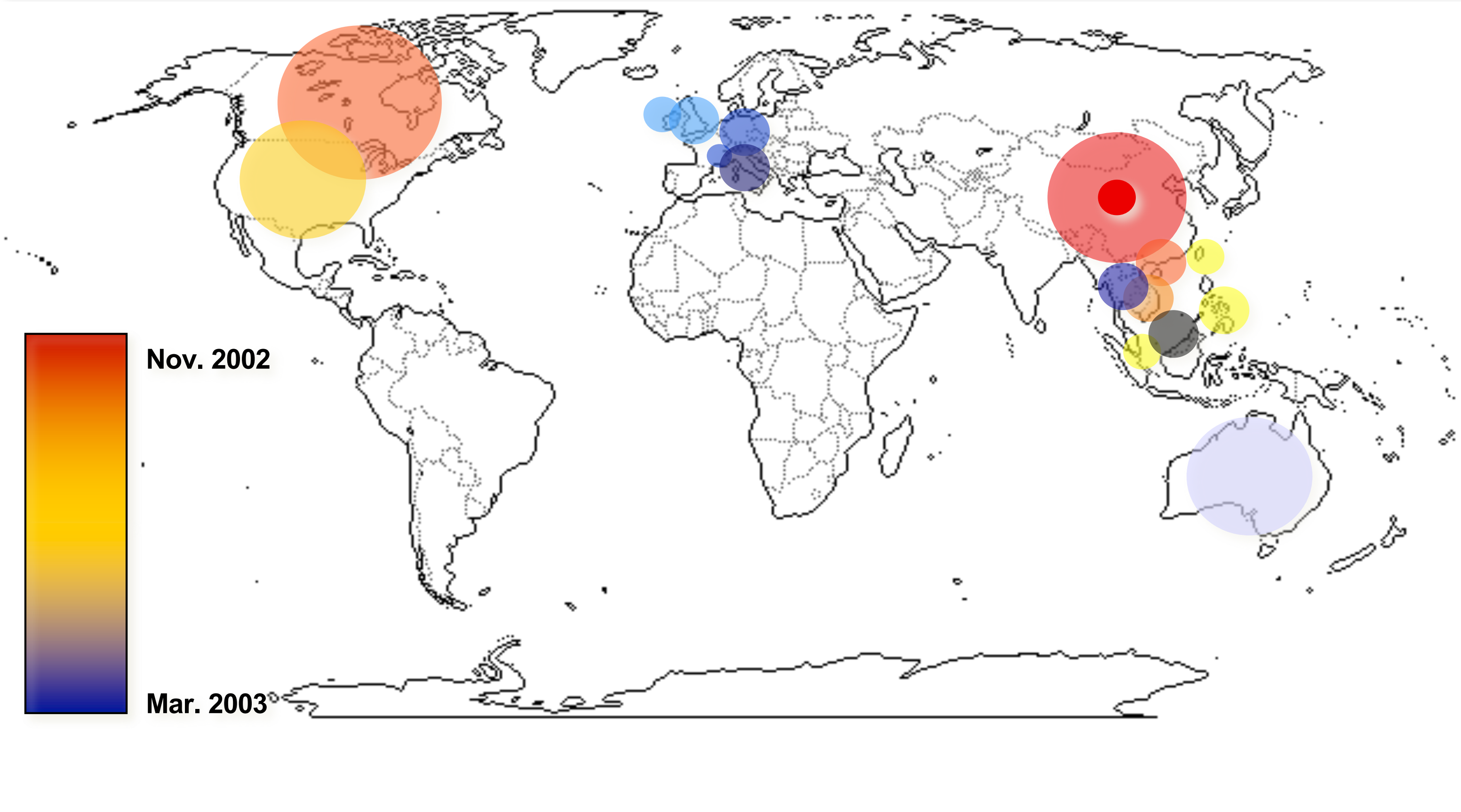
Epidemiology: past and current

Black death
25 millions victims
~50% population
V~100-200kms/an

SIR model with spatial diffusion allows to understand this

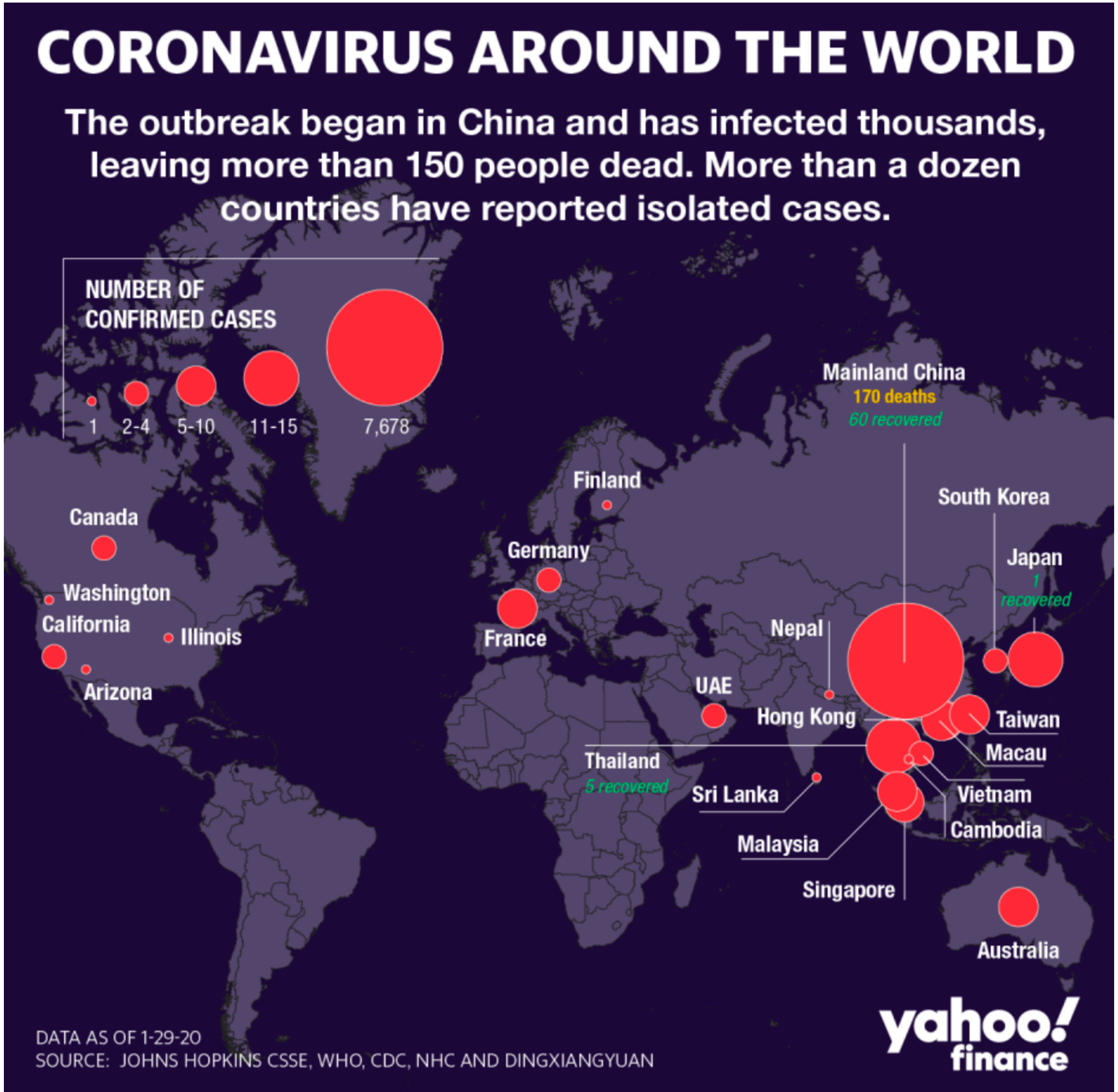


Epidemiology: past and current

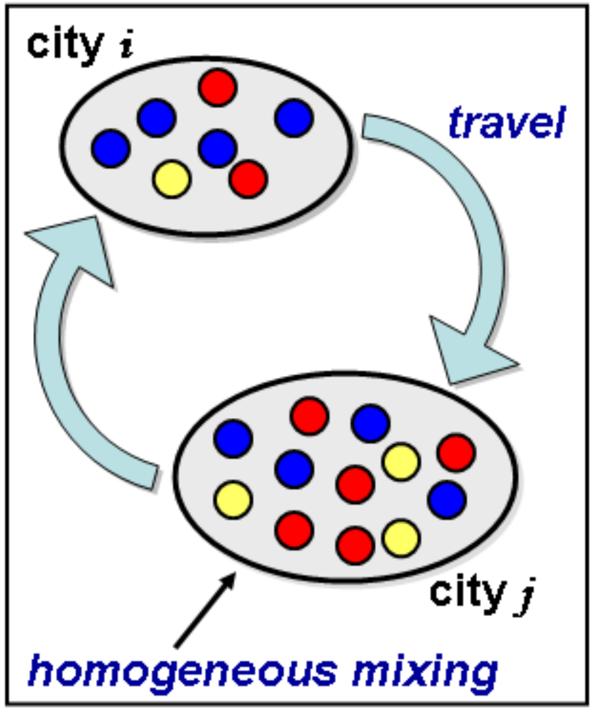


- Complex movement patterns: different means, different scales (SARS): Importance of networks

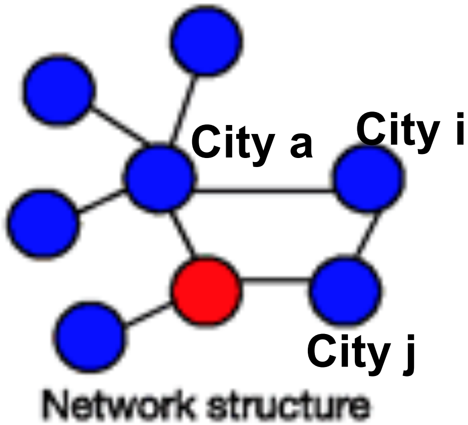
Epidemiology: present… 2019-nCoV



Metapopulation models



- Each node: internal structure (cities/countries)
- Links: transport/traffic



- Baroyan et al, 1969: ≈40 russian cities
- Rvachev & Longini, 1985: 50 airports worldwide
- Grais et al, 1988: 150 airports in the US
- Hufnagel et al, 2004: 500 top airports worldwide
- Colizza, Barrat, Barthelemy & Vespignani, PNAS (2006): 3000+ airports

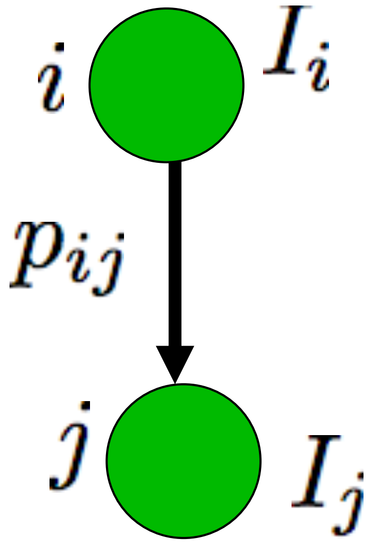
Metapopulation model

- Rvachev Longini (1985)

$$\partial_t I_i(t) = K_i[I_i(t)] + \Omega_i(t)$$

Inner city term

Travel term



- Transport operator:

$$\Omega_i(t) = \sum_{j\in\Gamma_i} p_{ji} I_j(t) - p_{ij} I_i(t)$$

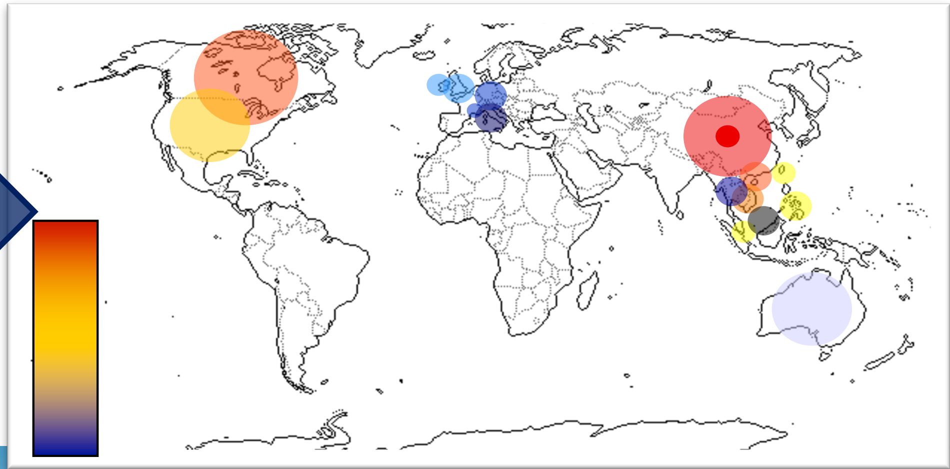
Flahault & Valleron (1985); Hufnagel et al, PNAS 2004, Colizza, Barrat, Barthelemy, Vespignani PNAS 2006, BMB, 2006. Theory: Colizza & Vespignani, Gautreau & al, …

Airline network and pandemic spread

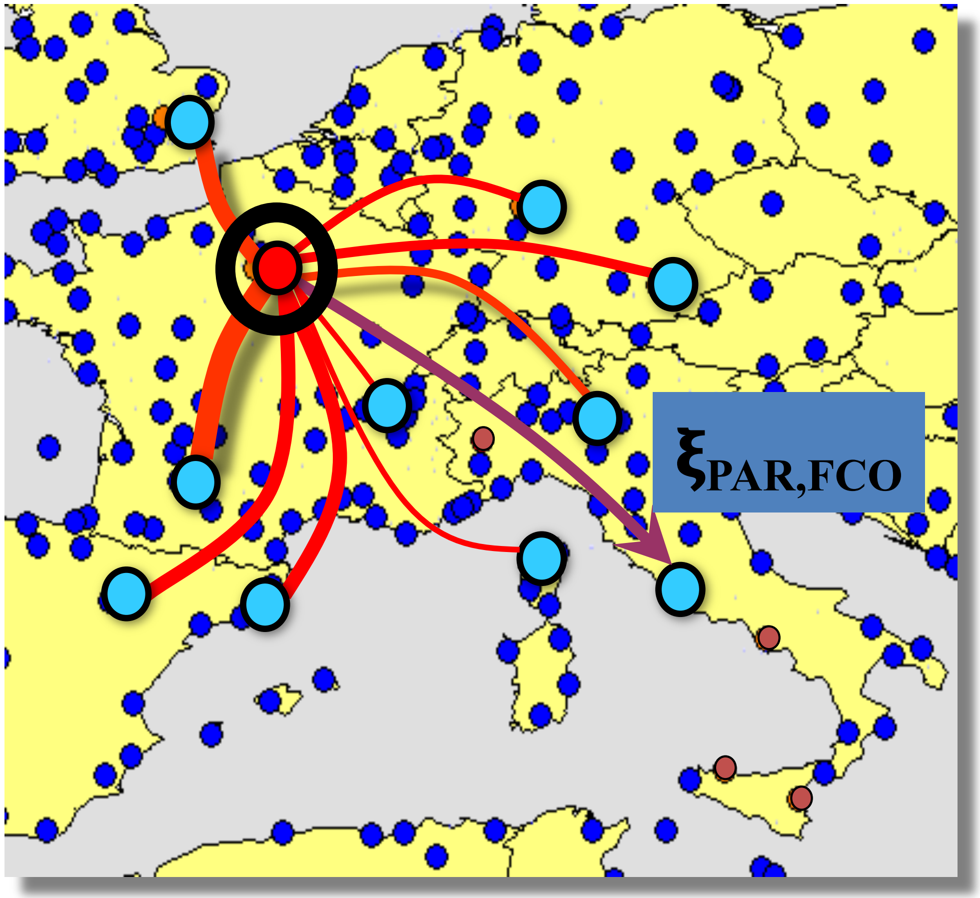
- Node: airport
- Link: existence of a direct flight

Complete IATA database:

- 3100 airports worldwide
- 220 countries
- ≈ 20,000 connections
- w_{ij} #passengers on connection i-j
- >99% total traffic



Stochastic model: travel term



Travel probability from PAR to FCO:

$$p_{PAR,FCO} = \frac{\xi_{PAR,FCO}}{N_{PAR}}$$

$\xi_{PAR,FCO}$ # passengers from PAR to FCO (Stochastic variable, multinomial distr.)

Discrete stochastic Model

$$S \xrightarrow{\beta} I \xrightarrow{\mu} R$$

- Data input: airline network, initial conditions, disease parameters

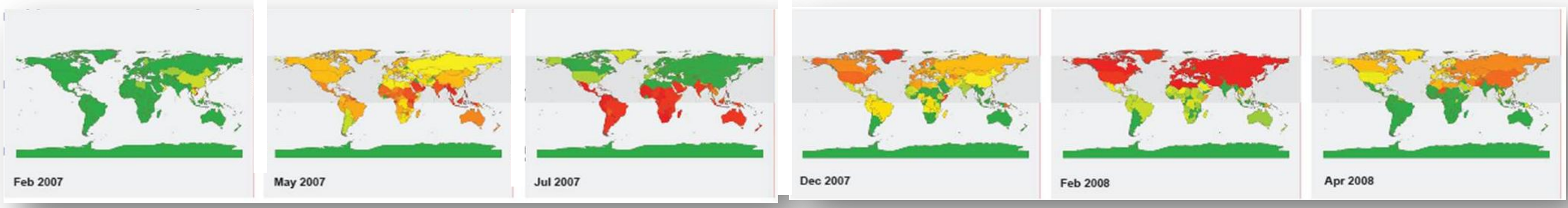
- Write evolution equations for each city (for S, I, R)

- Solve the 3100 x 3 differential coupled stochastic equations

Colizza, Barrat, Barthélemy, Vespignani, *PNAS* 103, 2015 (2006); *Bull. Math. Bio.* (2006)

Predictability

One outbreak realization:

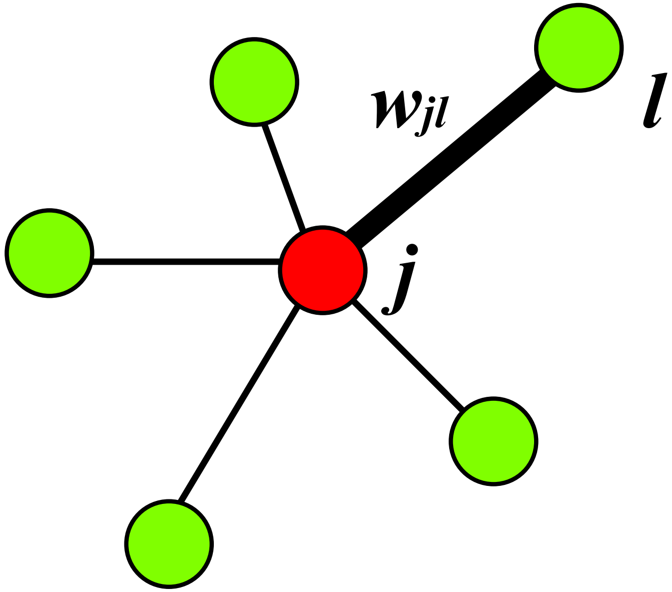


Another outbreak realization ? Effect of noise ?

?	?	?	?	?

Predictability and airport hubs

- Effect of heterogeneity:



- degree heterogeneity: decreases predictability

- Weight heterogeneity: increases predictability !

Good news: Existence of preferred channels !

Epidemic forecast, risk analysis of **containment strategies**

SARS - Epidemic Pathways

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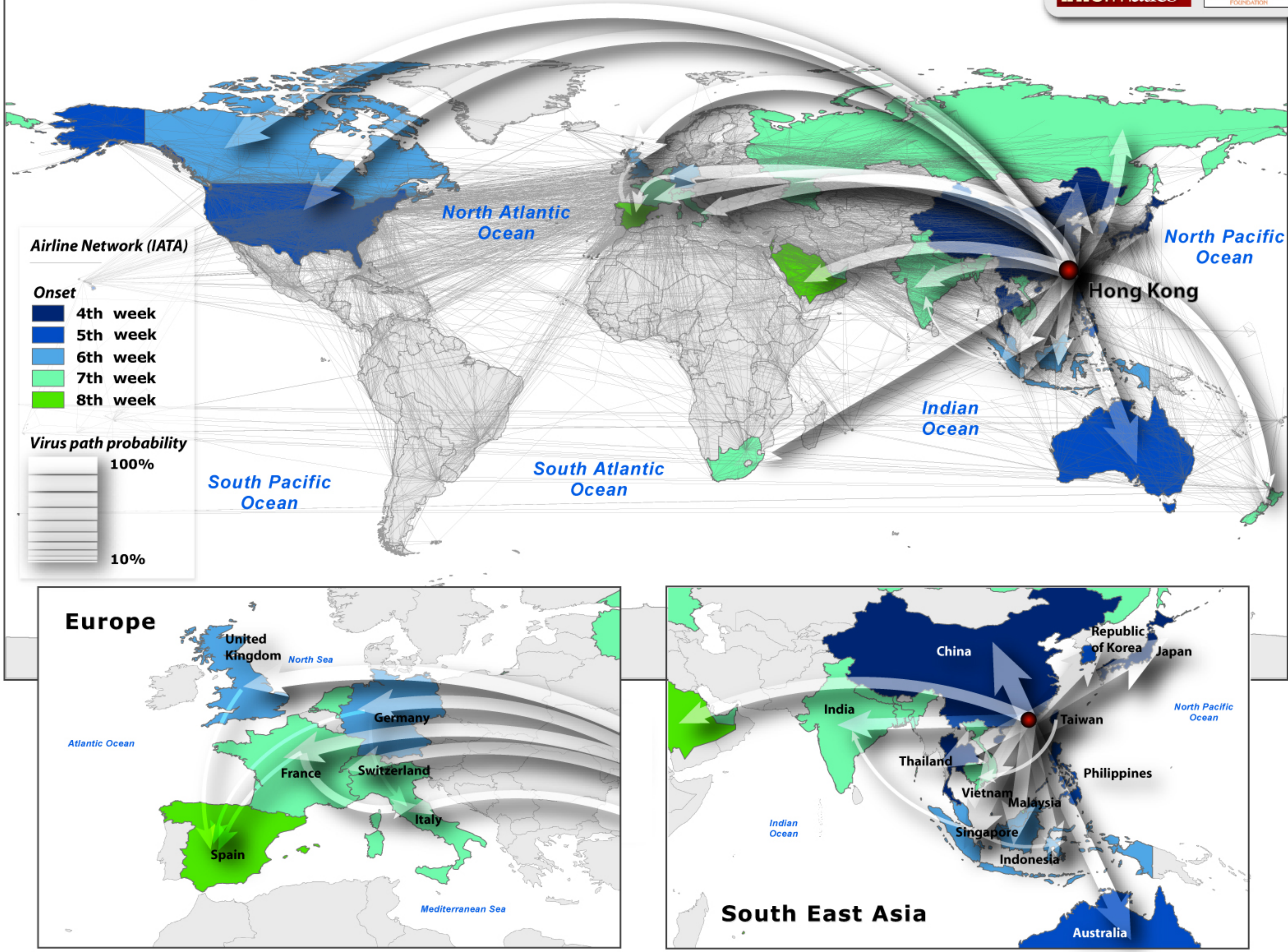
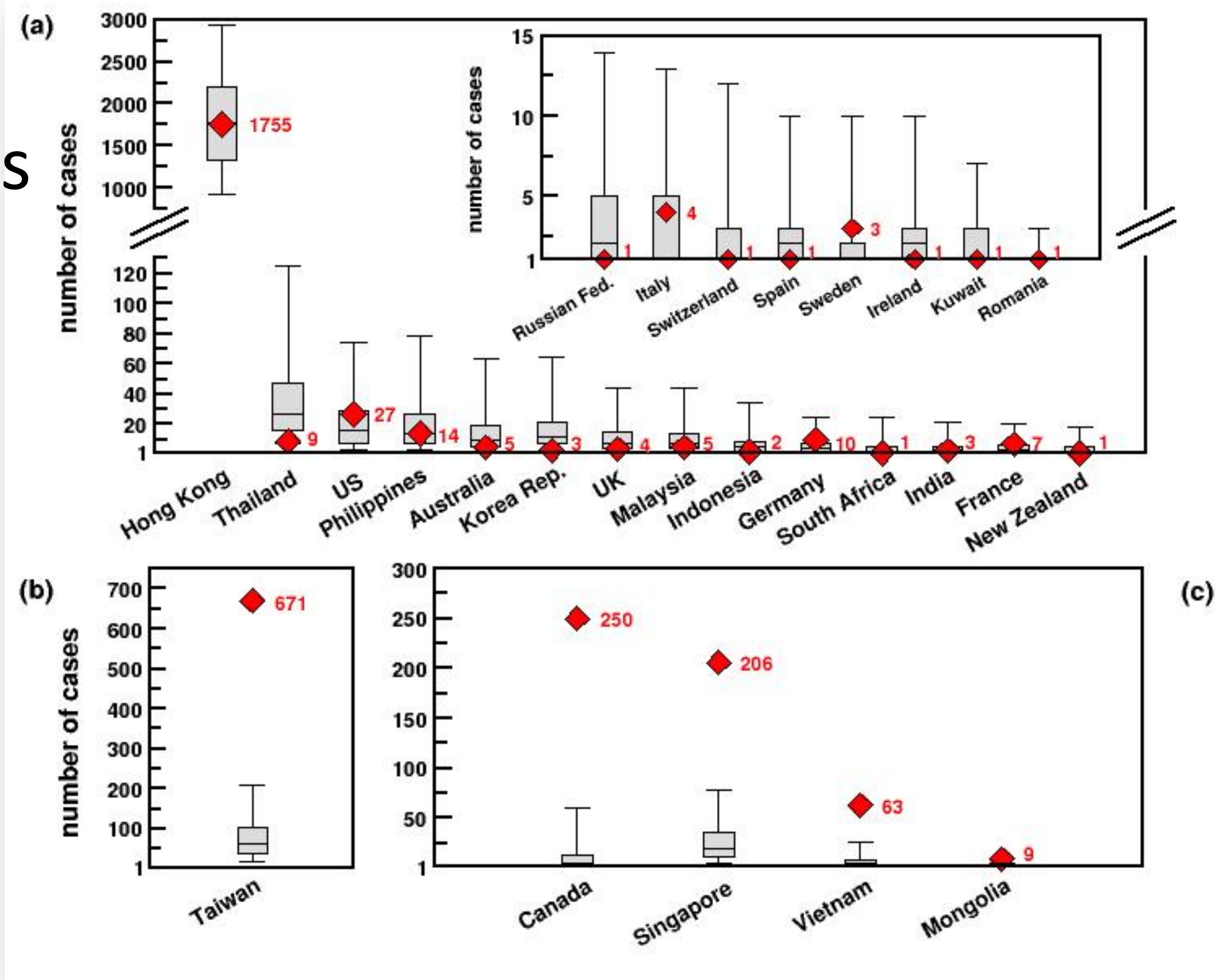
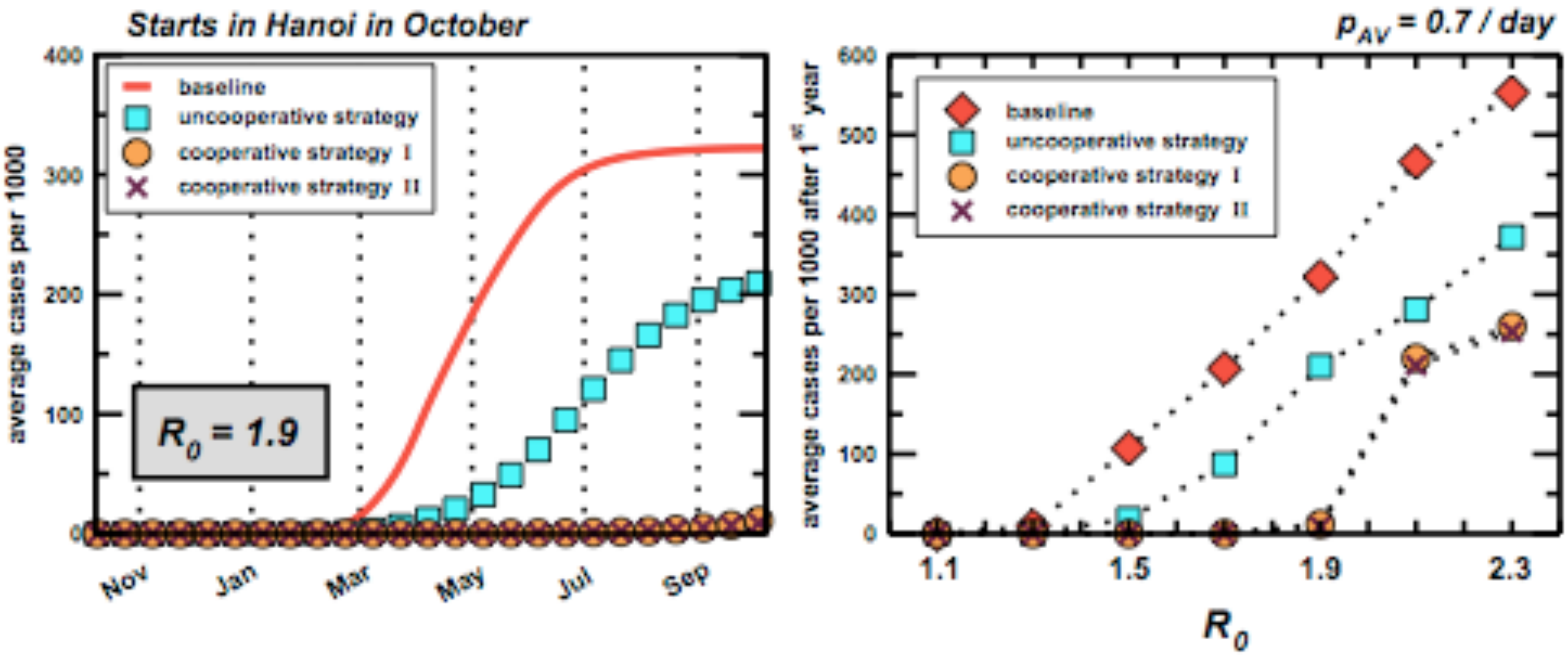


SARS: predictions



Colizza, Barrat, Barthelemy & Vespignani, bmc med (2007)

Effect of antivirals: Strategy comparison



Best strategy: Cooperative !

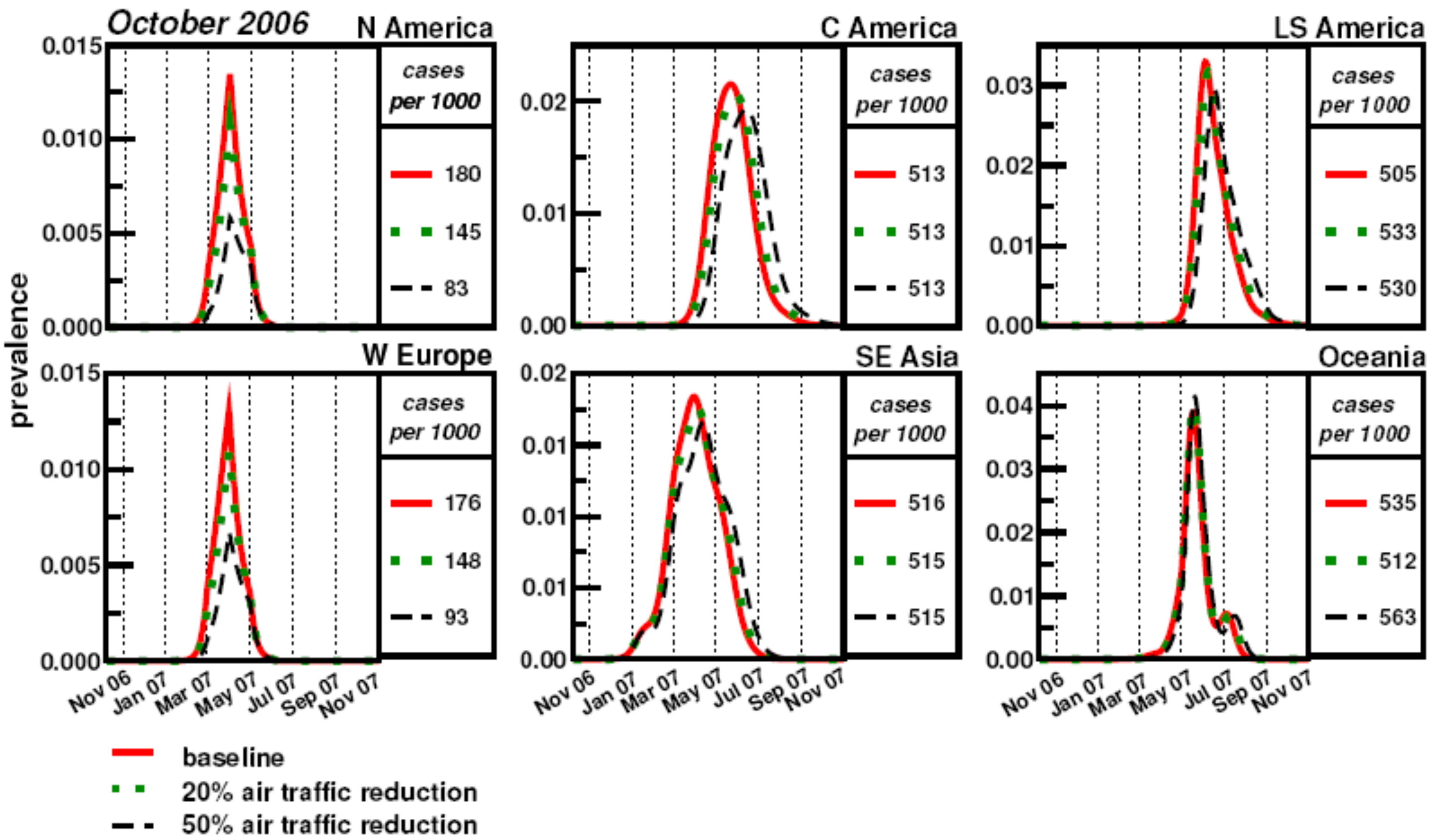
Theory: Pandemic threshold

- Condition for a pandemic spread ?

- Necessary condition: $R_0>1$ (spread inside a country)
- With mild assumptions, due to the structure of the airline network it can be shown that:

The pandemic always spreads !
=> Travel restrictions inefficient !

Travel restrictions inefficient

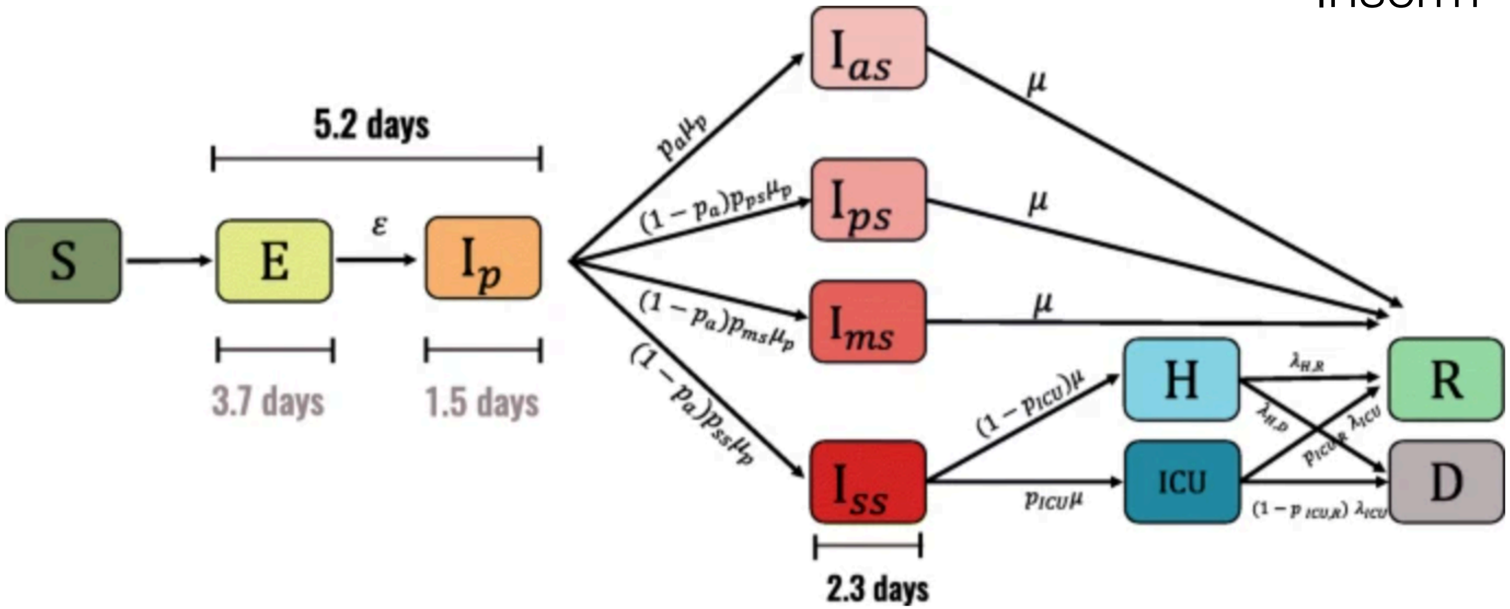


Colizza, Barrat, Barthelemy, Valleron, Vespignani. *PLoS Medicine* (2007)

Discussion: Covid19

Group of
V. Colizza
Inserm

Fig. 2



Compartmental model. S, susceptible; E, exposed; I_p, infectious in the prodromic phase (the length of time including E and I_p stages is the incubation period); I_a, asymptomatic infectious; I_{ps}, paucysymptomatic infectious; I_{ms}, symptomatic infectious with mild symptoms; I_{ss}, symptomatic infectious with severe symptoms; ICU, severe case admitted to ICU; H, severe case admitted to the hospital but not in intensive care; R, recovered; D, deceased

Discussion: Covid19

Impact of lockdown
Group of
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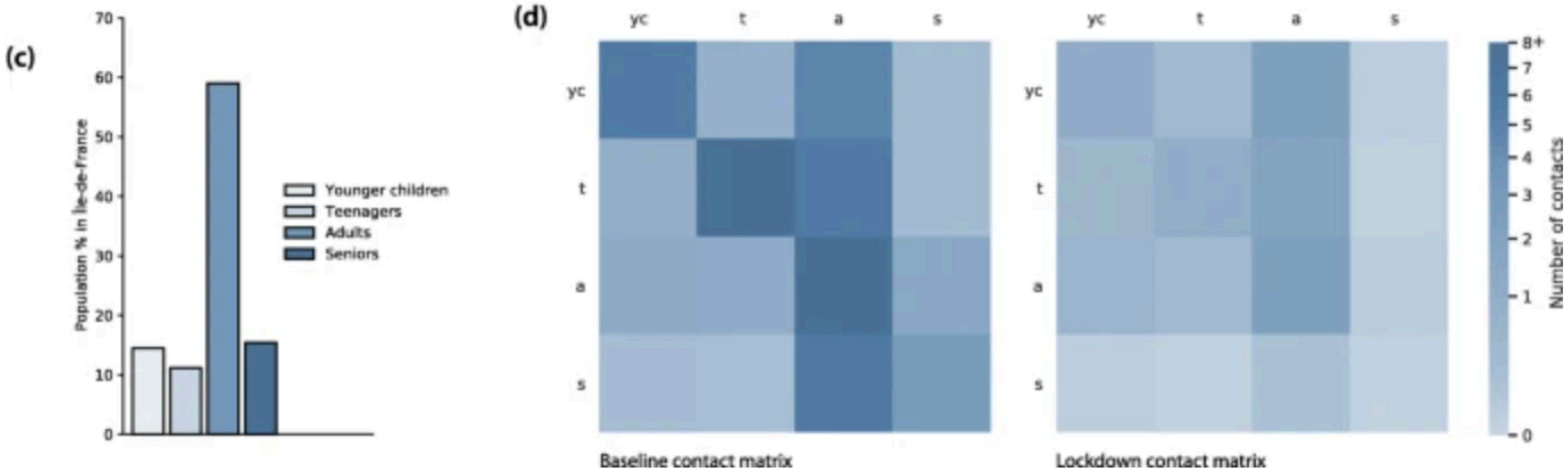

c Age profile in Île-de-France region corresponding to younger children, teenagers, adults, seniors (0, 11; 11, 19; 19, 65; and 65+ years old, respectively). **d** Contact matrices in the baseline scenario (no intervention) obtained from data [17] (left) and estimated for lockdown (right)

Some remarks about the Covid19

- Early studies: Predictions of exportation to Europe, etc. worked quite well

- At the national level, things are more complicated. In general, various predictions (effect of masks, impact of lockdown, timing, etc) didn't work very well…

Some remarks about the Covid19

- Possible problems:
  - Larger number of parameters
  - (Very) Large number of asymptomatic individuals
  - Very strong heterogeneity of transmission and symptoms: needs for a non mean-field model
  - Most of the modeling approaches have been based on coarse-grained data about the network structure
  - R_0 meaningless ?
  - Precise structure of the contact network needed but (new tech ? Tracing apps?)

Summary and Perspectives

- Maybe surprisingly it is easier to model the spread from a country to another
- More difficult at a smaller spatial level: national, and even more so for cities
- However increasing availability of individual data (phone, GPS) gives hope for constructing the contact network in cities and to be able to make better predictions for disease spread in urban areas…

End