Network Science

Spreading Phenomena

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with

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Questions 1

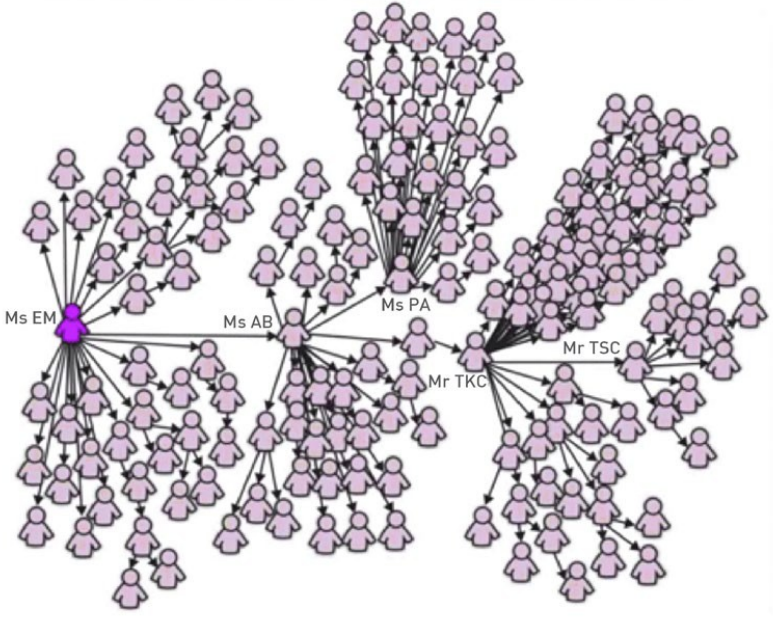
1. Modeling infectious disease. Compartmentalization and Homogeneous mixing assumptions. SI model. Characteristic time.
2. SIS and SIR models. Basic reproductive number (R0). Comparison of qualitative behavior of models.
3. SI model on networks. Assumptions and approximations. Role of degree distribution.
4. SIS model on networks. Epidemic threshold. Random vs. scale free networks. Vanishing epidemic threshold.
5. Contact networks. Cumulative degree distributions. Sexual (and other) hubs.

Questions 2

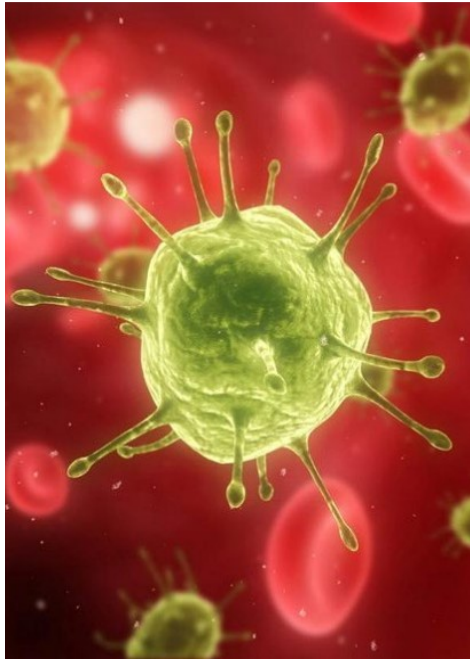
1. Recap of the main results of SI, SIS and SIR models on networks. What is the intrinsic difference between epidemics spreading on random networks vs scale-free networks?
2. Is scale-freeness all that matters?
3. Immunization: random strategy, effects on random networks and scale-free networks
4. Selective Immunization in scale-free networks. What is the connection between immunization strategies and what we know on robustness?
5. Fundamental elements of GLEAM and the H1N1 case
6. Effective distance: what is the better space to look at for the study of diseases nowadays? Why?

Ch 10- Epidemic spreading: Superspreaders

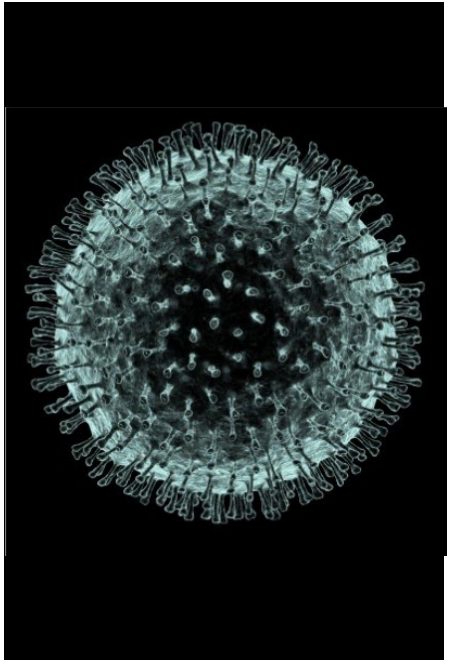
SARS 2003



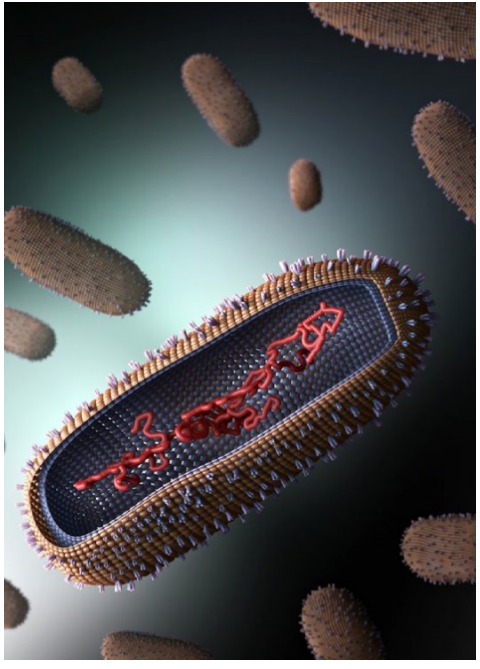
Section 10.1 Biological Pathogens



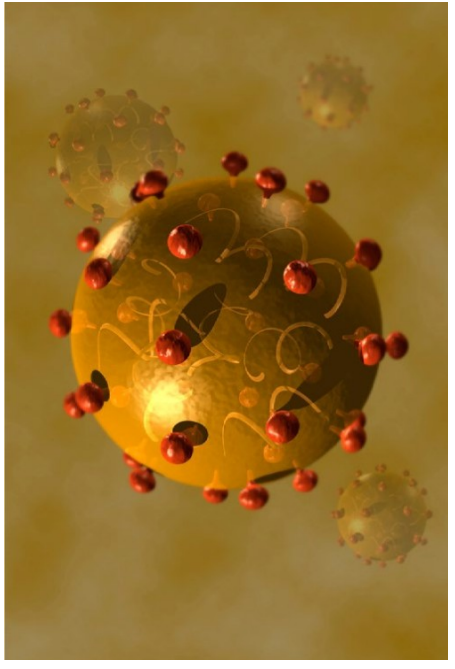
HIV



SARS



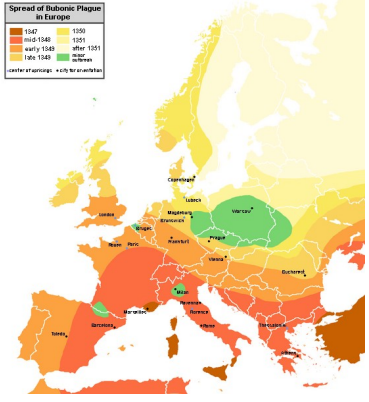
influenza



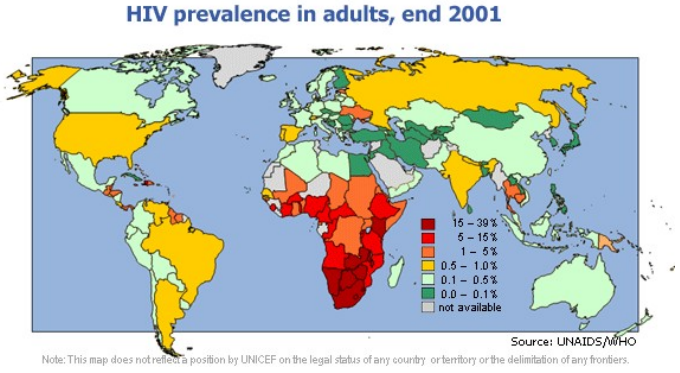
Hepatitis C

Biological: Notable Epidemic Outbreaks

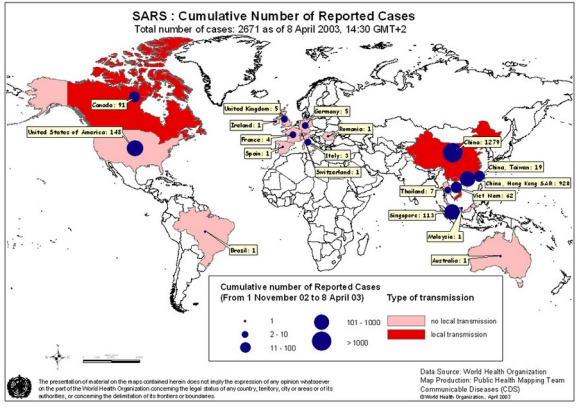
The Great Plague



HIV



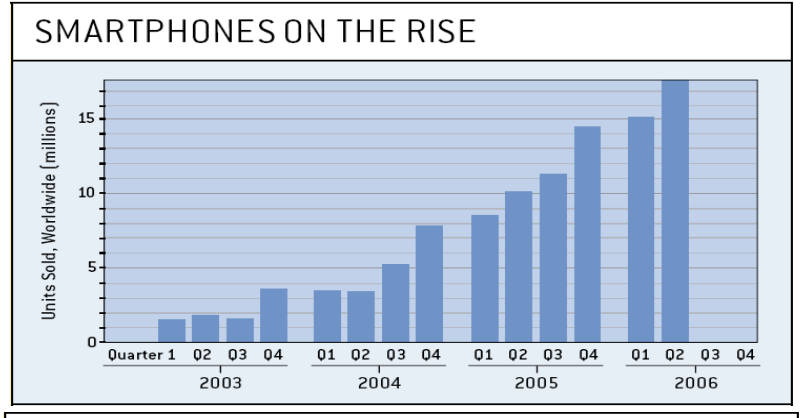
SARS

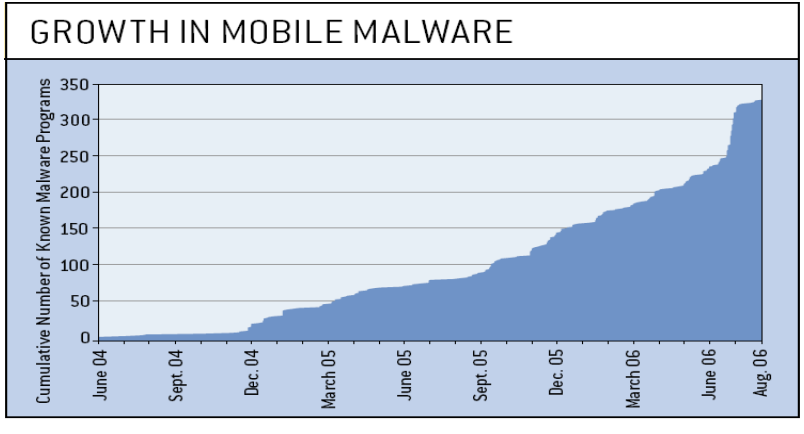


1918 Spanish flu

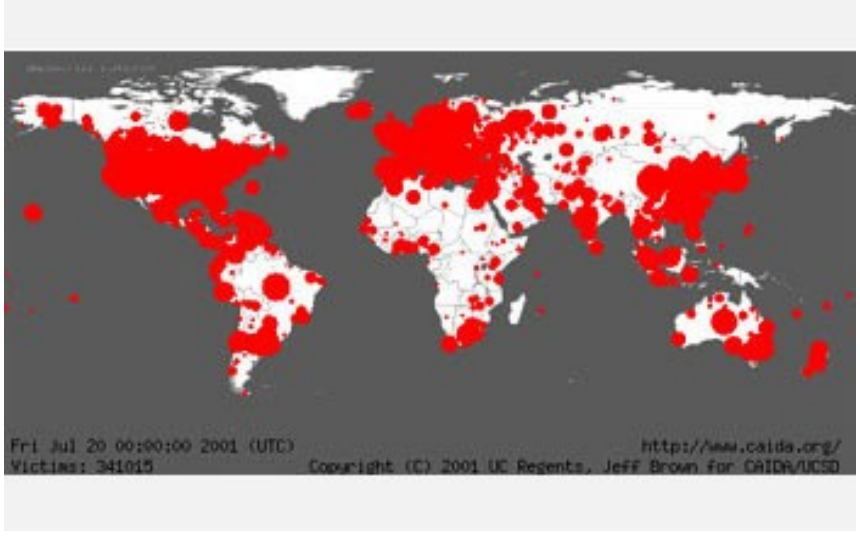
H1N1 flu

Computer Viruses, Worms, Mobile Phone Viruses





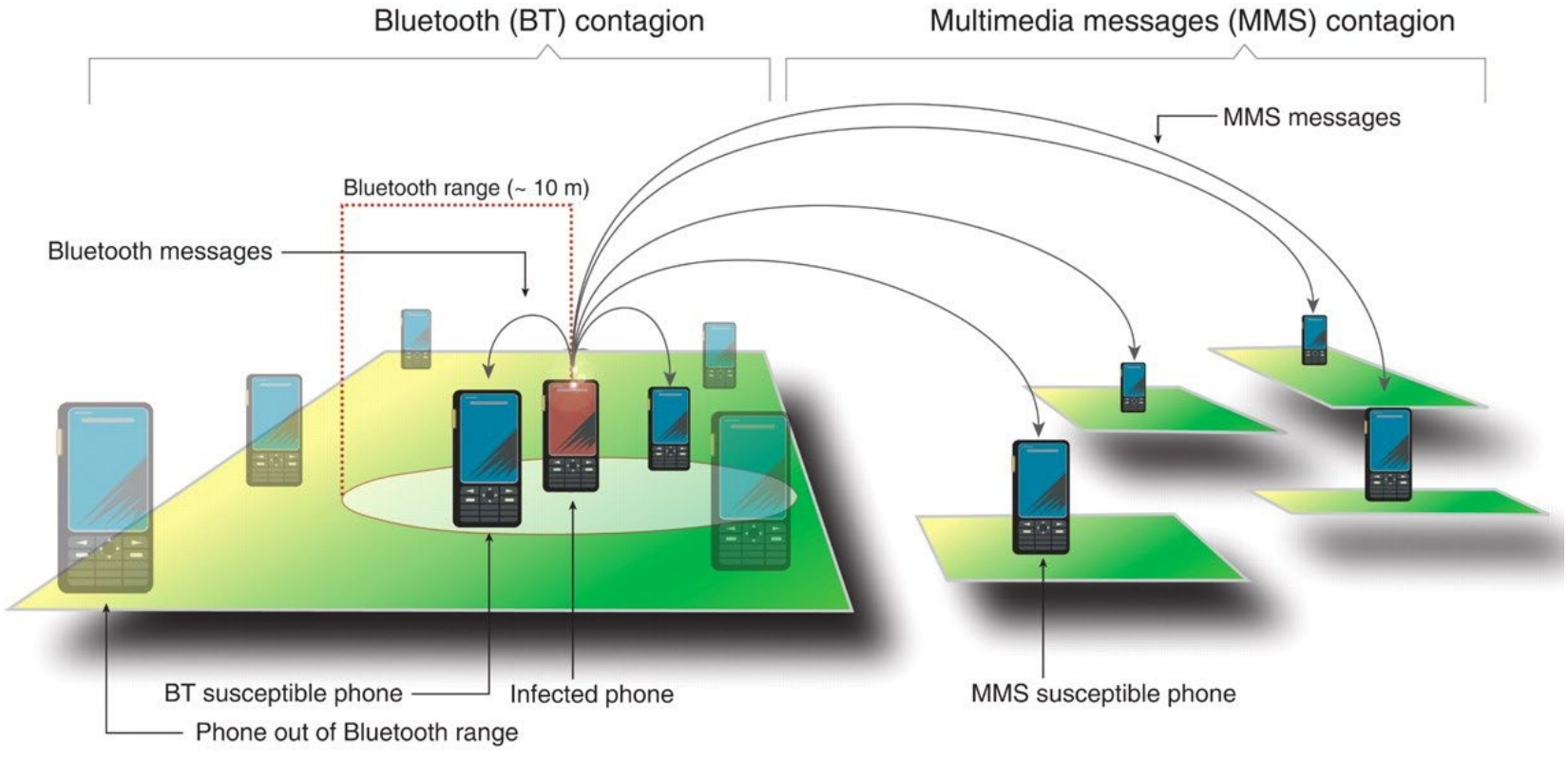
Code Red Worm paralyzed many countries' Internet



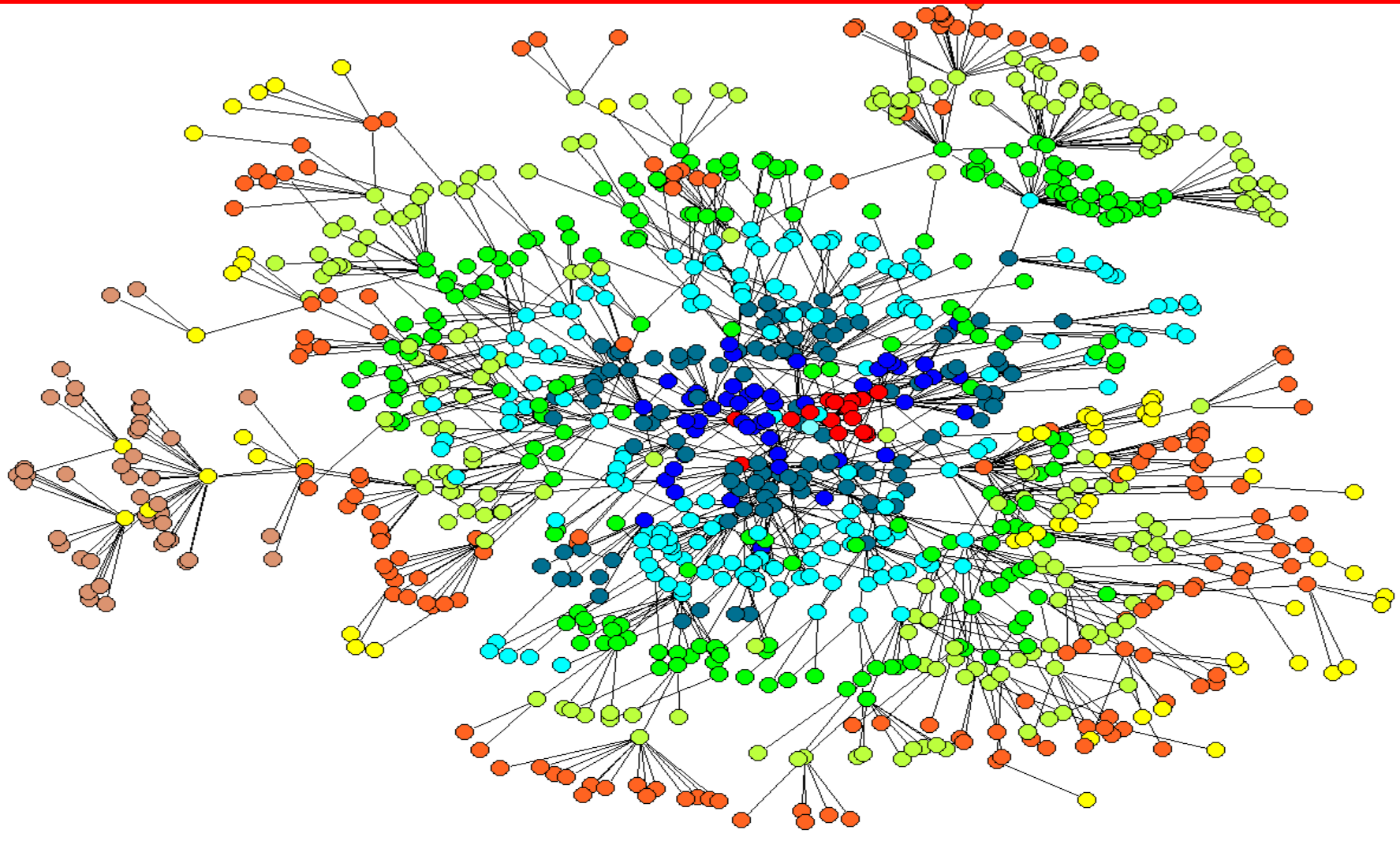
http://www.caida.org/publications/visualizations/

Hypponen M. *Scientific American* Nov. 70-77 (2006).

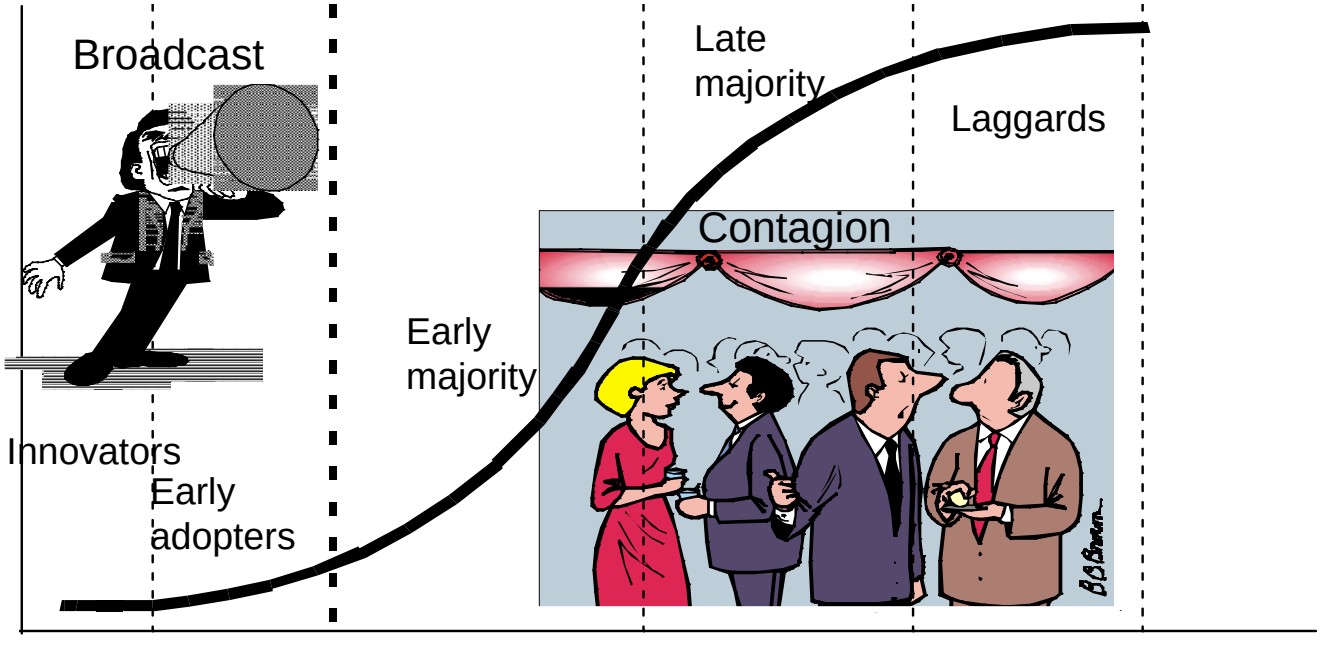
Section 10.1 Digital Viruses



Information Spreading



Diffusion of Innovation – The Adoption Curve



Reference unknown

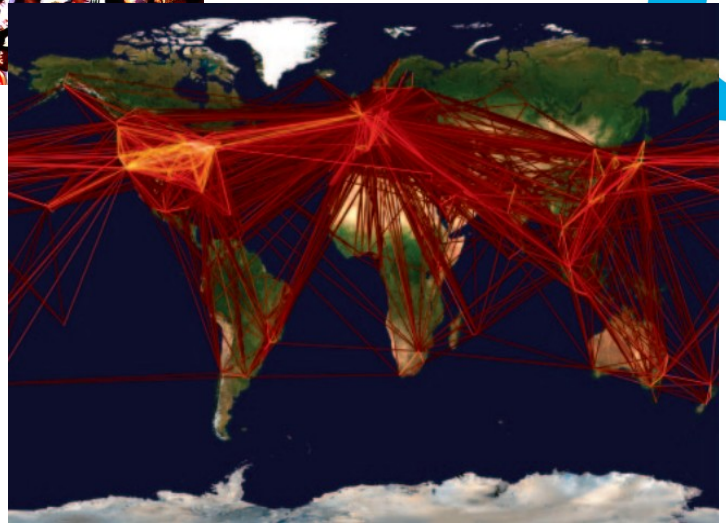
Types of Spreading Phenomena and Networks

Phenomena	Network	Agent
Venereal disease	Sexual network	pathogens
Other infectious disease	Contact network, transport network	pathogens
Rumor spreading	Communication network	Information, memes
Diffusion of innovation	Communication network	Ideas
Internet worms	Internet	Malwares (binary strings)
Mobile phone virus	Social network / proximity network	Malwares (binary strings)
Bedbugs	Hotel – traveler network	Bedbugs
Malaria	Mosquito – Human network	*Plasmodium*

Epidemic spreading – Why does it matter now?

High population density

High mobility



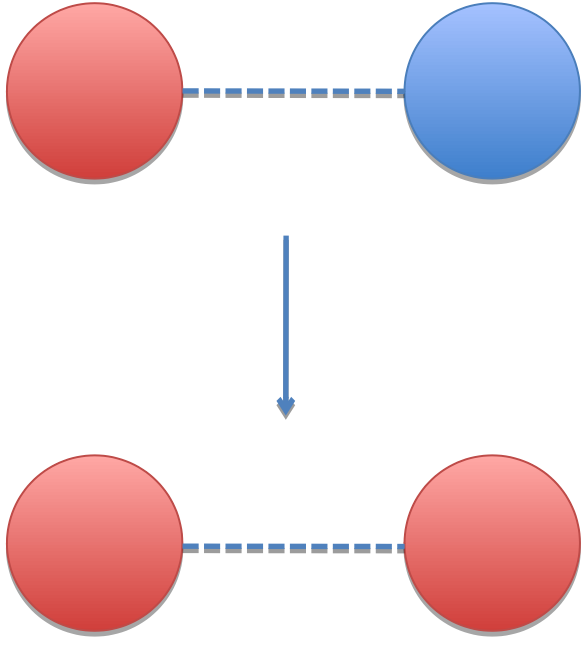
→ perfect conditions for epidemic spreading.

Airline figure: L. Hufnagel et al. *PNAS* **101,** 15124 (2004)

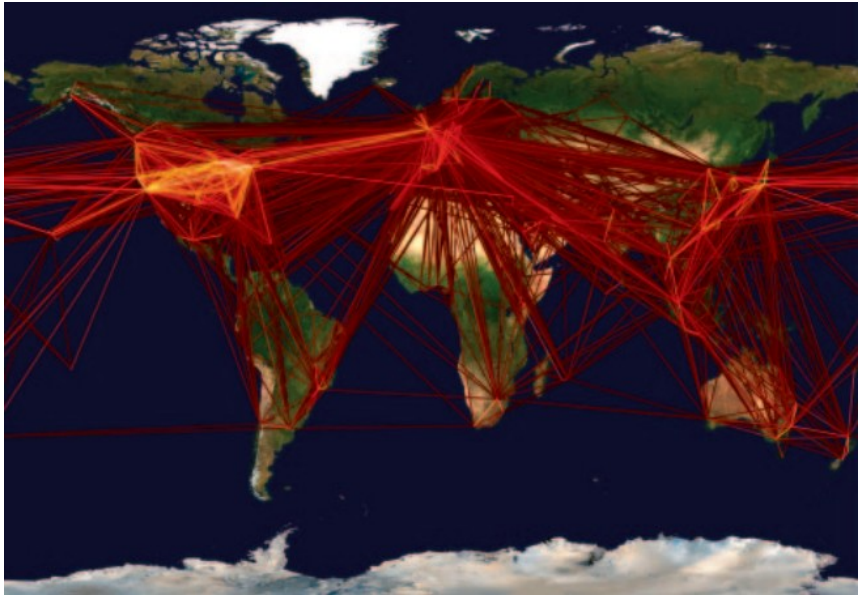
Epidemic Spreading – Network

- Epidemic spreading always implies network structure!

Spreading happens only when the carries of the diseases/virus/idea are **connected to each other**.

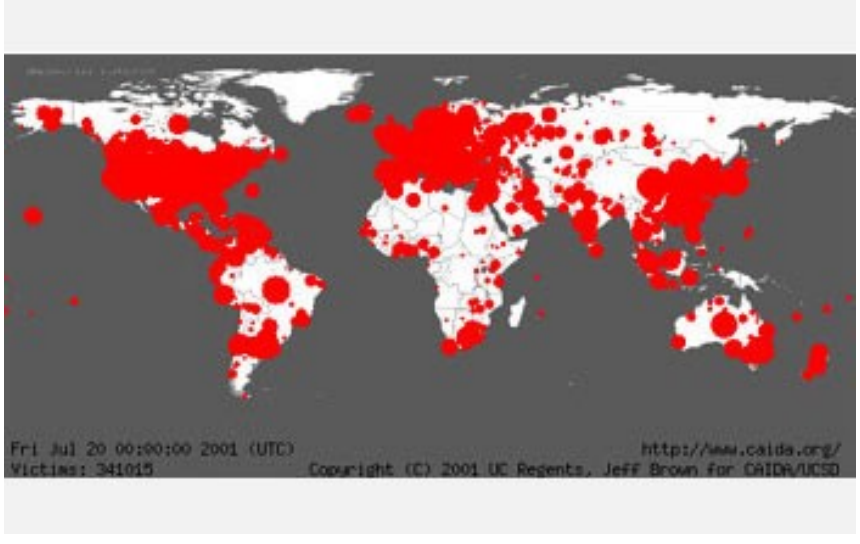


Epidemic Spreading – Network



The transportation network

L. Hufnagel et al. *PNAS* **101,** 15124 (2004)



Internet

http://www.caida.org/publications/visualizations/

Epidemic Modeling (classical models)

Section 10.2 Fundamental assumptions of Epidemic modeling

i. **Compartmentalization**

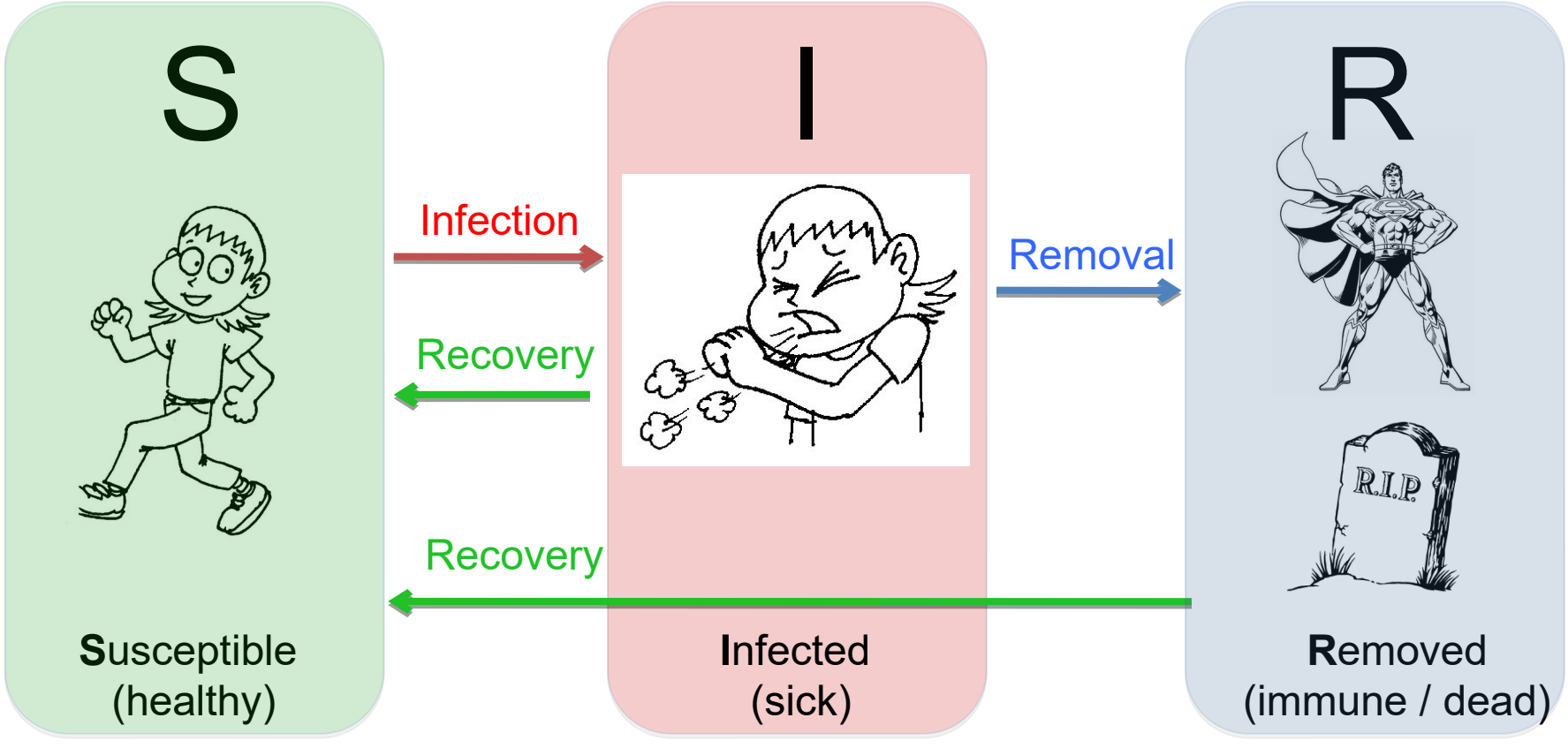
Epidemic models classify each individual into distinct states, based on the stage of the disease affecting them. The simplest classification assumes that an individual can be in one of three states (or compartments):

- *Susceptible (S):* Healthy individuals who have not yet contacted the pathogen.
- *Infectious (I):* Contagious individuals who have contacted the pathogen and hence can infect others.
- *Recovered (R):* Individuals who have been infected before, but have recovered from the disease, hence are not infectious.

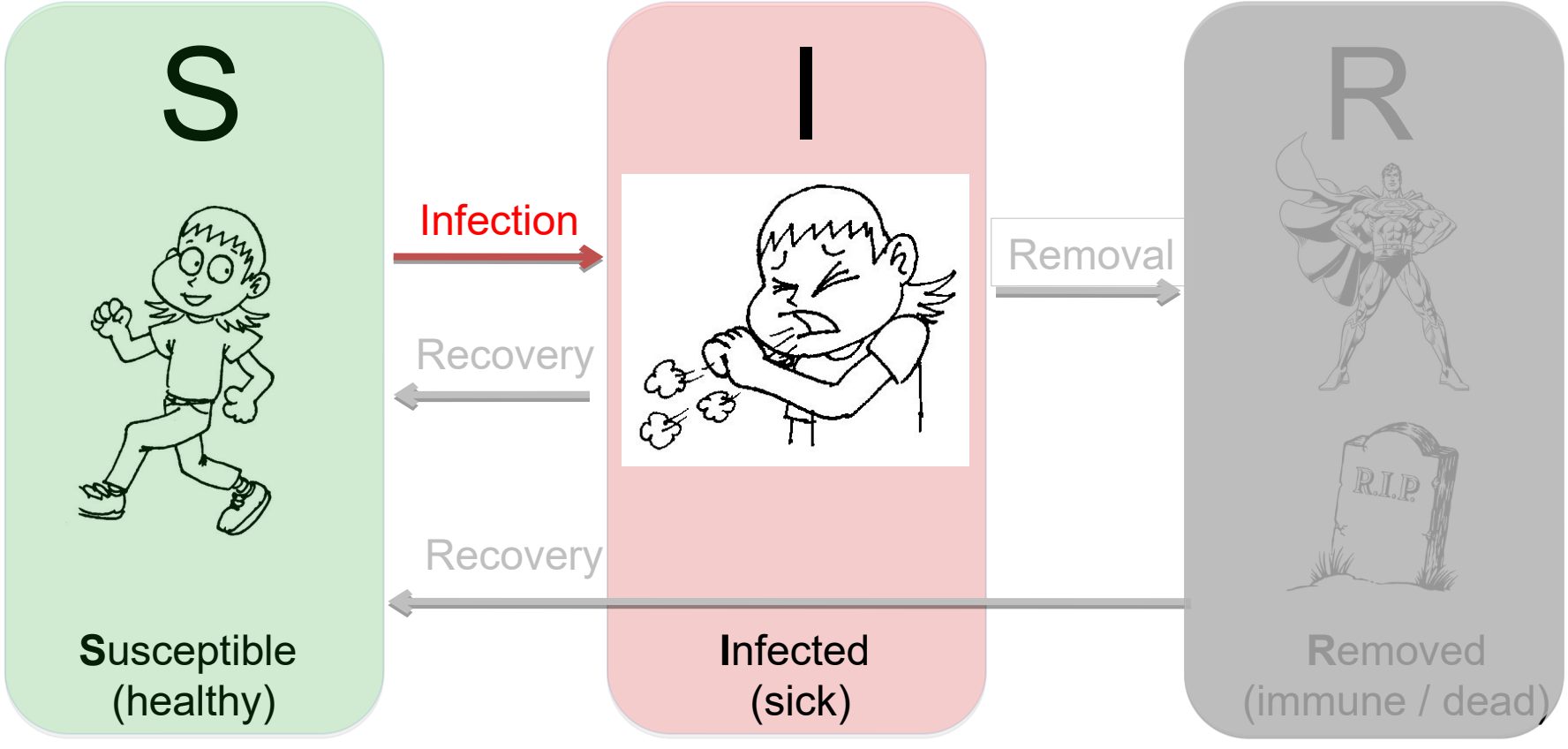
ii. **Homogenous Mixing**

The homogenous mixing hypothesis (also called fully mixed or mass-action approximation) assumes that each individual has the same chance of coming into contact with an infected individual. This hypothesis eliminates the need to know the precise contact network on which the disease spreads, replacing it with the assumption that anyone can infect anyone else.

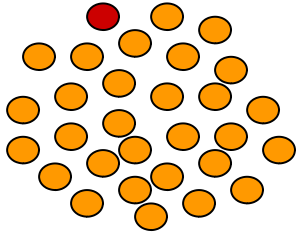
Classical Epidemic Models – Basic States



Simplest Model: SI



SI Model: Homogeneous Mixing (No network)



- Each individual has <k> contacts with randomly chosen others individuals per unit time.
- The likelihood that the disease will be transmitted from an infected to a healthy individual in a unit time: β

If there are I infected individuals and S susceptible individuals, the average rate of new infection is $\beta SI / N$

$$\beta\langle k\rangle \frac{S(t)I(t)}{N} dt.$$

$$\frac{dI(t)}{dt} = \beta\langle k\rangle \frac{S(t)I(t)}{N}.$$

SI Model: Homogeneous Mixing (No network)

$$\frac{dI(t)}{dt} = \beta\langle k\rangle \frac{S(t)I(t)}{N}.$$

$$s = S/N, \qquad i = I/N$$

$$\frac{di}{dt} = \beta si = \beta i(1-i)$$

$\beta\langle k\rangle$ is called the *transmission rate* (or transmissibility).

$$\frac{di}{i} + \frac{di}{(1-i)} = \beta\langle k\rangle dt. \qquad \ln i - \ln(1-i) + c = \beta\langle k\rangle t.$$

$$i(t) = \frac{i_0 \exp(\beta t)}{1 - i_0 + i_0 \exp(\beta t)}$$

SI Model – Behavior

$$\frac{di}{dt} = \beta i(1-i)$$

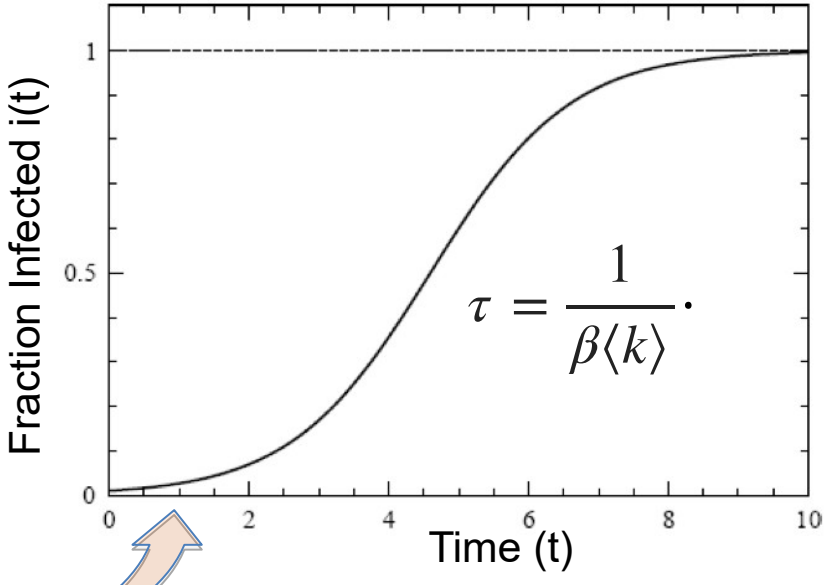
$$i(t) = \frac{i_0 \exp(\beta t)}{1 - i_0 + i_0 \exp(\beta t)}$$

If *i(t)* is small,

$$\frac{di}{dt} \approx \beta i$$

$$i \approx i_0 \exp(\beta t)$$

exponential outbreak



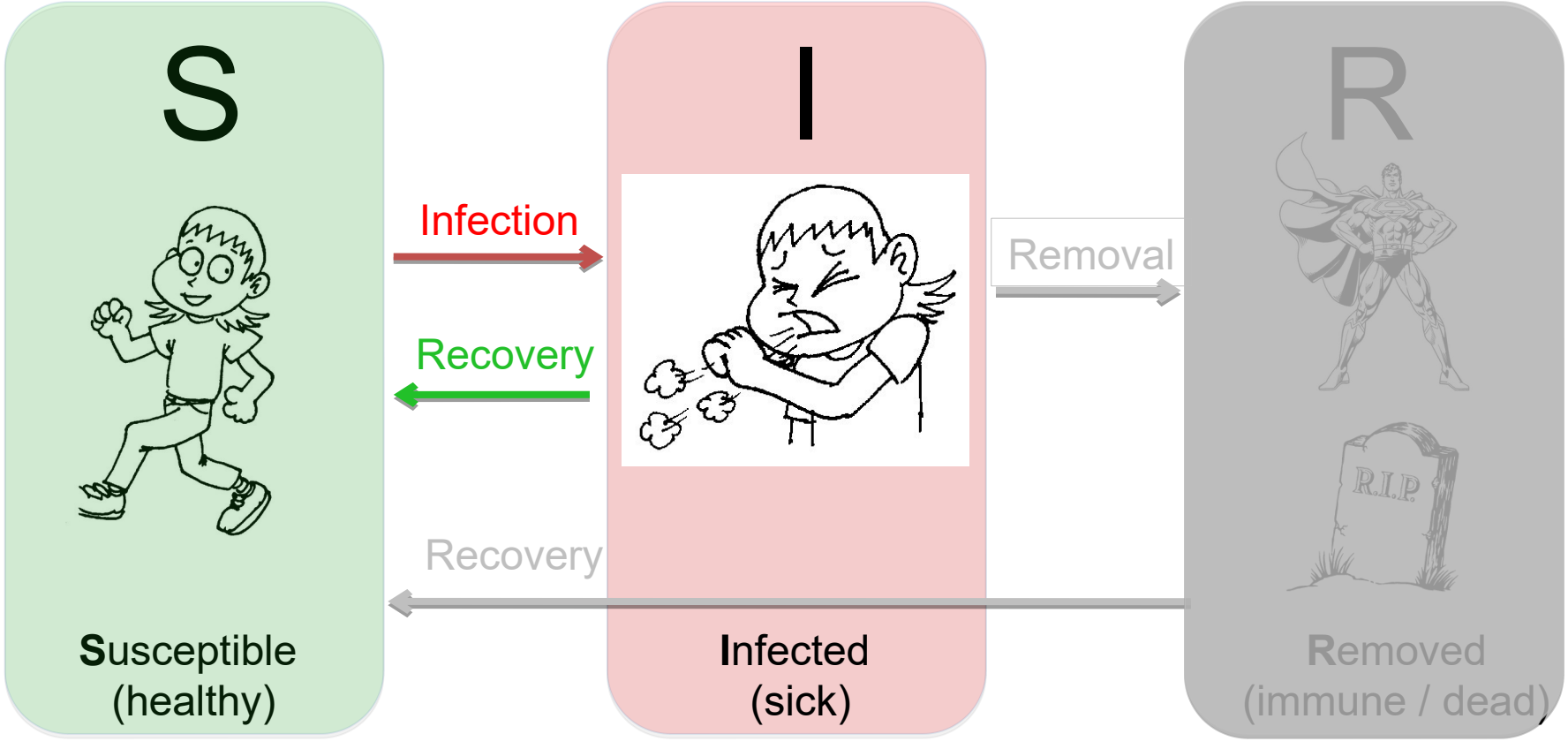
As i(t)→ 1.

$$\frac{di}{dt} \to 0$$

saturation

SI model: the fraction infected increases until everyone is infected.

SIS Model: Common Cold



SIS Model

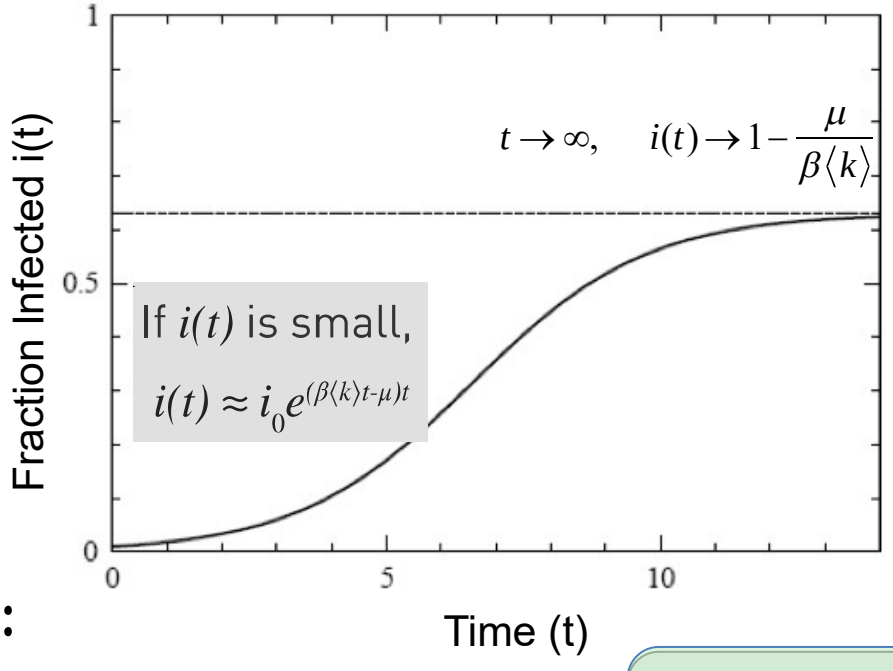
$$\frac{di}{dt} = \beta\langle k\rangle i(1-i) - \mu i$$

I S I→S

$$i = (1 - \frac{\mu}{\beta\langle k\rangle})\frac{Ce^{(\beta\langle k\rangle-\mu)t}}{1 + Ce^{(\beta\langle k\rangle-\mu)t}}.$$

Endemic state ($\mu<\beta\langle k\rangle$):

Disease-free state ($\mu>\beta\langle k\rangle$):



Stationary state:

$$\frac{di}{dt} = \beta i(1-i) - \mu i = 0$$

SIS Model: Reproductive Number

$$i = (1 - \frac{\mu}{\beta\langle k\rangle})\frac{Ce^{(\beta\langle k\rangle-\mu)t}}{1 + Ce^{(\beta\langle k\rangle-\mu)t}}.$$

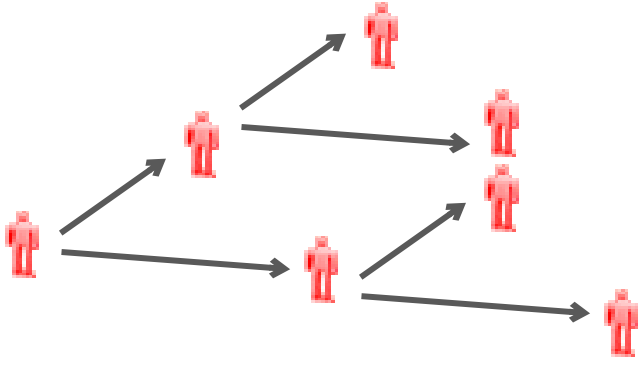
Characteristic Time: $\tau = \frac{1}{\mu(R_0 - 1)}$, $R_0 = \frac{\beta\langle k\rangle}{\mu}$.

reproductive number R_0: average # of infectious individuals generated by one infected in a fully susceptible population.

SIS Model: Reproductive Number

reproductive number $\mathbf{R_0}$: average # of infectious individuals generated by one infected in a fully susceptible population.

e.g. $R_0 = 2$



SIS Model: Reproductive Number

reproductive number R_0: average # of infectious individuals generated by one infected in a fully susceptible population.

DISEASE	TRANSMISSION	R_0
Measles	Airborne	12-18
Pertussis	Airborne droplet	12-17
Diptheria	Saliva	6-7
Smallpox	Social contact	5-7
Polio	Fecal-oral route	5-7
Rubella	Airborne droplet	5-7
Mumps	Airborne droplet	4-7
HIV/AIDS	Sexual contact	2-5
SARS	Airborne droplet	2-5
Influenza (1918 pandemic strain)	Airborne droplet	2-3

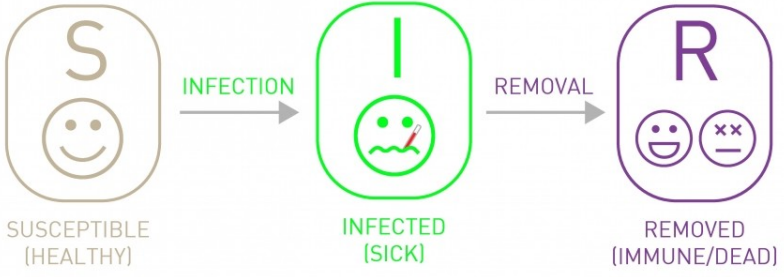
SIR Model

a. Unlike the SIS model, in the SIR model recovered individuals enter a recovered state, meaning that they develop immunity, rather than becoming susceptible again. Flu, SARS, and Plague are diseases with this property. We must use the SIR model to describe their spread.

b. The differential equations governing the time evolution of the fraction of individuals in the susceptible s, infected i, and the removed r state.

c. The time dependent behavior of s, i, and r as predicted by the equations shown in **(b)**. According to the model, all individuals transition from a susceptible (healthy) state to the infected (sick) state and then to the recovered (immune) state.

a.



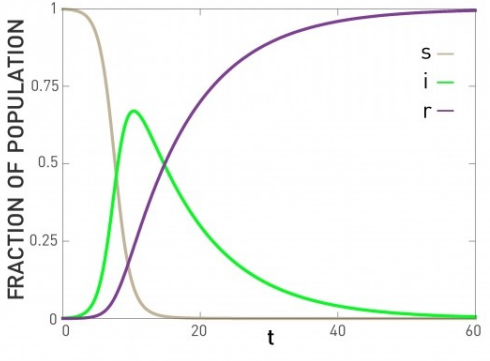
b.

$$\frac{ds}{dt} = -\beta\langle k\rangle i\,[1-r-i]$$

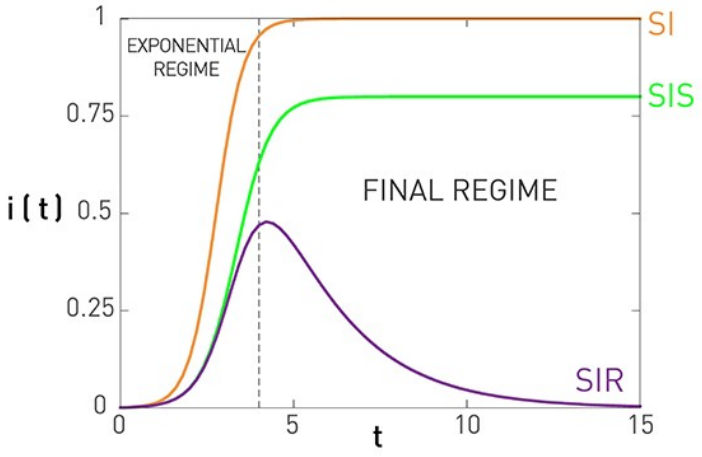
$$\frac{di}{dt} = -\mu i + \beta\langle k\rangle i\,[1-r-i]$$

$$\frac{dr}{dt} = \mu i$$

c.



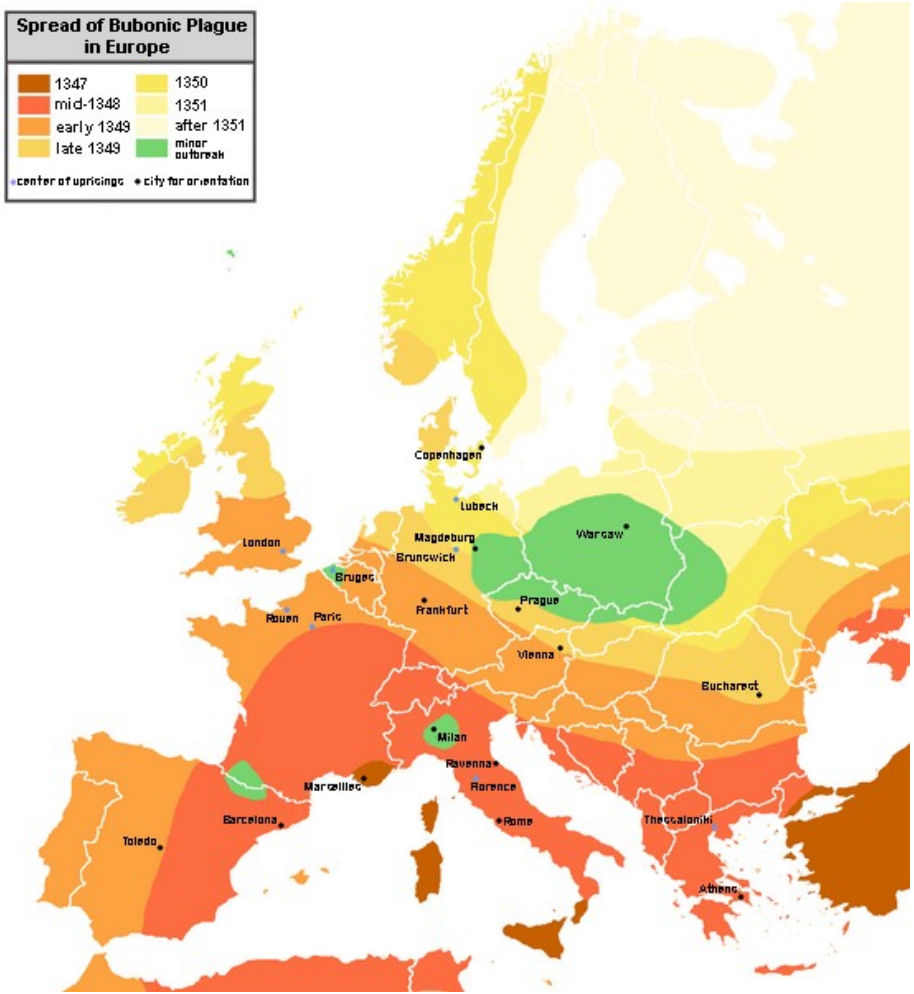
Epidemic Models: Summary



	SI	SIS	SIR
Exponential Regime: Number of infected individuals grows exponentially	$i = \frac{i_0 e^{\beta\langle k\rangle t}}{1 - i_0 + i_0 e^{\beta\langle k\rangle t}}$	$i = \left(1 - \frac{\mu}{\beta\langle k\rangle}\right) \frac{Ce^{(\beta\langle k\rangle - \mu)t}}{1 + Ce^{(\beta\langle k\rangle - \mu)t}}$	No closed solution
Final Regime: Saturation at t→ =∞	$i(\infty) = 1$	$i(\infty) = 1 - \frac{\mu}{\beta\langle k\rangle}$	$i(\infty) = 0$
Epidemic Threshold: Disease does not always spread	No threshold	$R_0 = 1$	$R_0 = 1$

Network Epidemics

14th Century – The Great Plague



4 years from France to Sweden

Limited by the speed of human travel

http://en.wikipedia.org/wiki/Black_Death
http://de.wikipedia.org/wiki/Schwarzer_Tod

Section 10.3 Network Epidemics

The approaches described above have not considered explicitly that the spreading takes place on a network– they assumed *homogenous mixing*, which means that each individual can infect *any* other individual.

In reality, epidemics spread along *links in a network* → we need to explicitly account for the role of the network in the epidemic process.

SI model on a network: Degree Block Approximation

Split nodes by their degrees

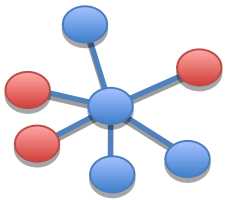
$$i_k = \frac{I_k}{N_k}, \quad i = \sum_k P(k) i_k$$

SI model:

$$\frac{di_k(t)}{dt} = \beta(1 - i_k(t))k\Theta_k(t)$$

Proportional to k

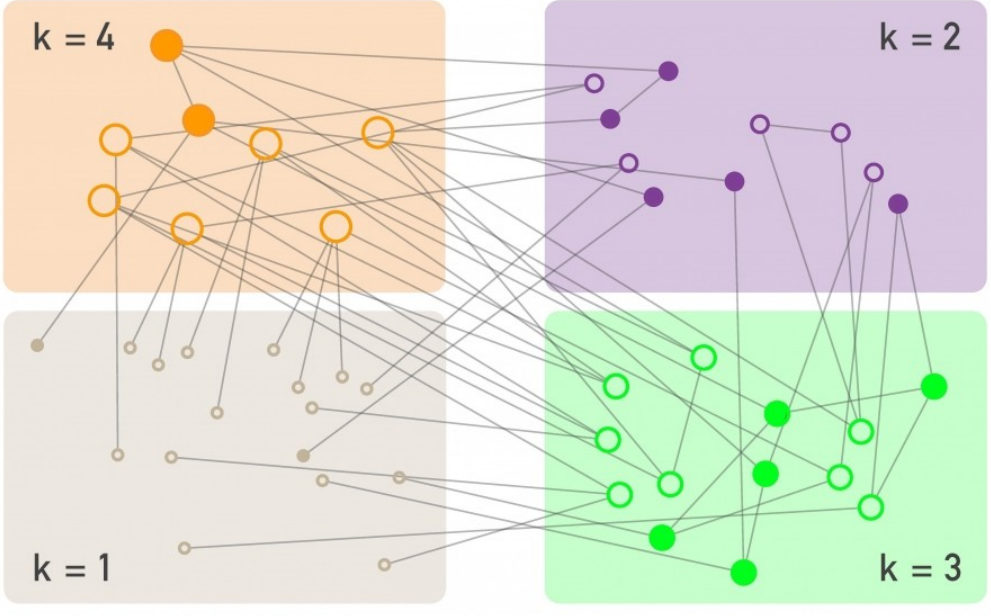
Density of infected neighbors of nodes with degree k



I am susceptible with k neighbors, and $\Theta_k(t)$ of my neighbors are infected.

(Vespignani)

SIS model on a network: Degree Block Approximation



Split nodes by their degrees

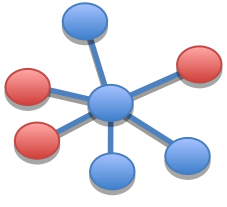
$$i_k = \frac{I_k}{N_k}, \quad i = \sum_k P(k) i_k$$

SIS model:

$$\frac{di_k(t)}{dt} = \beta(1 - i_k(t))k\Theta_k(t) - \mu i_k(t)$$

Proportional to k

Density of infected neighbors of nodes with degree k



I am susceptible with k neighbors, and $\Theta_k(t)$ of my neighbors are infected.

Early time behavior

Early time behavior of an epidemic

Why do we care about the early behavior of an epidemic?

- vaccines, cures, and other medical interventions take months to years to develop

- the best way to stop or slow down an epidemic:
  → early quarantine
  → early vaccination

- SI model is the most relevant for early stages