

# Biological Contagion

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Principles of Complex Systems, Vols. 1, 2, & 3D  
CSYS/MATH 300, 303, & 394, 2022-2023 | @pocsvox

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Santa Fe Institute | University of Vermont



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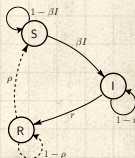
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## Sealie & Lambie Productions



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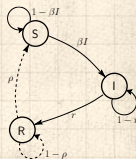
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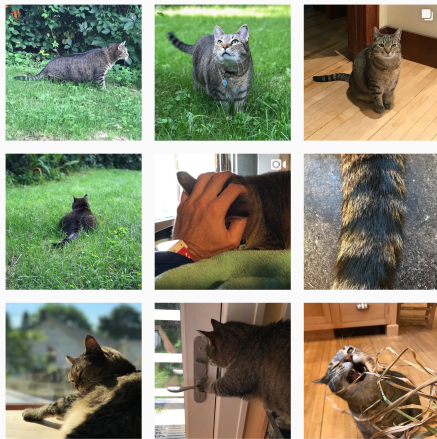
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## Special Guest Executive Producer



 On Instagram at [pratchett\\_the\\_cat](https://www.instagram.com/pratchett_the_cat) 

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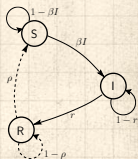
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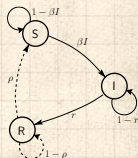
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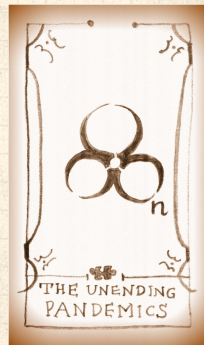
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# An awful recording: Wikipedia's list of epidemics from 430 BC on.

WIKIPEDIA The Free Encyclopedia

Article [Talk](#) Read [Edit](#) [View history](#)

## List of epidemics

From Wikipedia, the free encyclopedia

This article is a **list of epidemics** of **infectious disease**. Widespread and chronic complaints such as **heart disease** and **allergy** are not included if they are not thought to be infectious.

*This list is incomplete; you can help by expanding it.*

Death toll (estimate)	Location	Date	Comment	Disease	Reference
ca. 75,000 - 100,000	<a href="#">Greece</a>	429–426 BC	Known as <b>Plague of Athens</b> , because it was primarily in Athens.	unknown, similar to typhoid	
ca. 30% of population	<a href="#">Europe, Western Asia, Northern Africa</a>	165–180	Known as <b>Antonine Plague</b> , due to the name of the Roman emperor in power at the time.	unknown, symptoms similar to <b>smallpox</b>	
	<a href="#">Europe</a>	250-266 AD	Known as the <b>Plague of Cyprian</b> named after <b>St. Cyprian</b> Bishop of <b>Carthage</b> .	unknown, possibly <b>smallpox</b>	
ca. 40% of population	<a href="#">Europe</a>	541–542	Known as <b>Plague of Justinian</b> , due to the name of the Byzantine emperor in power at the time.	<b>Bubonic plague</b> <sup>[1]</sup>	
30% to 70% of population	<a href="#">Europe</a>	1346–1350	Known as "Black Death" or <b>Second plague pandemic</b> , first return of the plague to Europe after the Justinianic plague of the 6th century.	<b>plague</b> <sup>[2]</sup>	
5-15 million (80% of population)	<a href="#">Mexico</a>	1545-1548	<b>Cocoliztli</b>	<b>viral hemorrhagic fever</b> <sup>[R][F][M]</sup>	
2 - 2.5 million (50% of population)	<a href="#">Mexico</a>	1576	<b>Cocoliztli</b>	<b>viral hemorrhagic fever</b> <sup>[R][F][M]</sup>	
	<a href="#">Seneca nation</a>	1592–1596		<b>measles</b> <sup>[R]</sup>	

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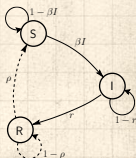
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**THE CONFUSION OF CONTAGIONS**



## A confusion of contagions:

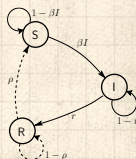
- Did Harry Potter spread like a virus?
- Can disinformation be “infectious”?
- Suicide, violence?
- Morality? Evil? Laziness? Stupidity? Happiness?
- Religion?
- Democracy ...?
- Language? The alphabet?<sup>[10]</sup>
- Stories?

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## Naturomorphisms

- “The feeling was contagious.”
- “The news spread like wildfire.”
- “Freedom is the most contagious virus known to man.”  
—Hubert H. Humphrey, Johnson’s vice president
- “Nothing is so contagious as enthusiasm.”  
—Samuel Taylor Coleridge

## Optimism according to Ambrose Bierce:

The doctrine that everything is beautiful, including what is ugly, everything good, especially the bad, and everything right that is wrong. ... **It is hereditary, but fortunately not contagious.**

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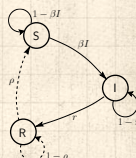
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# Social contagion

Eric Hoffer, 1902–1983

There is a grandeur in the uniformity of the mass. When a fashion, a dance, a song, a slogan or a joke sweeps like **wildfire** from one end of the continent to the other, and a hundred million people roar with **laughter**, sway their bodies in unison, **hum one song** or **break forth in anger and denunciation**, there is the overpowering feeling that in this country we have come nearer the brotherhood of man than ever before.

 Hoffer  was an interesting fellow...

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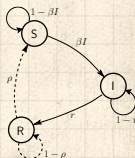
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# The spread of fanaticism

Hoffer's most famous work: "The True Believer: Thoughts On The Nature Of Mass Movements" (1951)<sup>[12]</sup>

## Aphorisms-aplenty:

- ☰ "We can be absolutely certain only about things we do not understand."
- ☰ "Mass movements can rise and spread without belief in a God, but never without belief in a devil."
- ☰ "Where freedom is real, equality is the passion of the masses. Where equality is real, freedom is the passion of a small minority."

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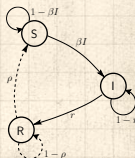
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# Imitation

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despair.com

“When people are free to do as they please, they usually imitate each other.”

—Eric Hoffer  
“The Passionate State of Mind” [13]

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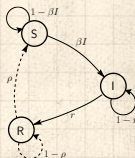
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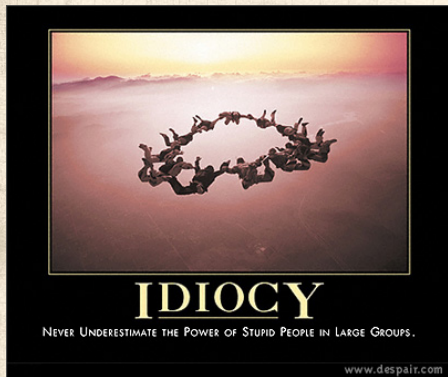
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# The collective...

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“Never Underestimate the Power of Stupid People in Large Groups.”

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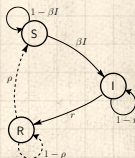
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# Examples of non-disease spreading:

## Interesting infections:

 Spreading of certain buildings in the US:

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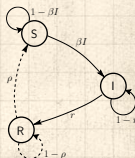
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<http://www.youtube.com/watch?v=EGzHBtoVvpc?rel=0>



# Marbleization of the US:

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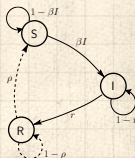
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<http://www.youtube.com/watch?v=9ihSeSToXOw?rel=0>





# The most terrifying contagious outbreak?

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## Google books Ngram Viewer

Graph these comma-separated phrases:   case-insensitive

between  and  from the corpus  with smoothing of  [Search lots of books](#)

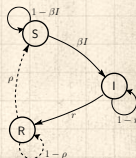


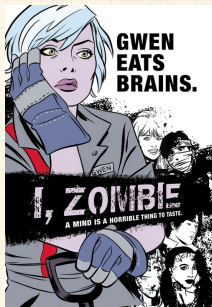
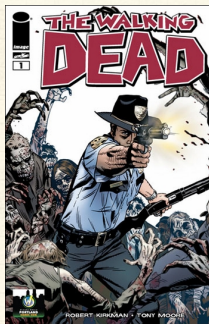
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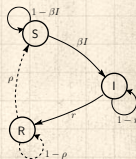


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## Definitions

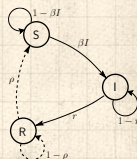
-  (1) The spreading of a quality or quantity between individuals in a population.
-  (2) A disease itself: the plague, a blight, the dreaded lurgi, ...
-  from Latin: *con* = 'with' + *tangere* 'to touch.'
-  Contagion has unpleasant overtones...
-  Just **Spreading** might be a more neutral word
-  But contagion is kind of exciting...

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## Two main classes of contagion

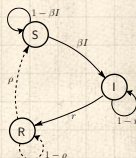
1. **Infectious diseases:**  
tuberculosis, HIV, ebola, SARS, influenza, zombification, ...
2. **Social contagion:**  
fashion, word usage, rumors, uprisings, religion, stories about zombies, ...

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# Community—S2E6: Epidemiology

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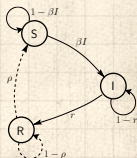
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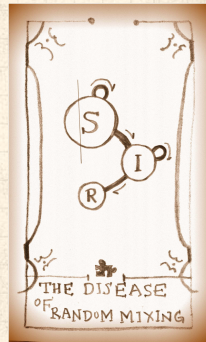
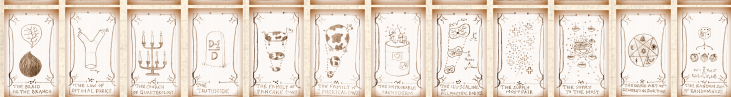
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## The standard SIR model <sup>[18]</sup>


 = basic model of disease contagion


 Three states:

1. S = Susceptible
2. I = Infective/Infectious
3. R = Recovered or Removed or Refractory

  $S(t) + I(t) + R(t) = 1$

 Presumes random interactions (mass-action principle)

 Interactions are independent (no memory)

 Discrete and continuous time versions

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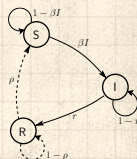
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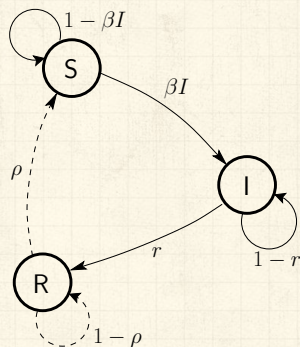
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## Discrete time automata example:



### Transition Probabilities:

$\beta$  for being infected given contact with infected

$r$  for recovery

$\rho$  for loss of immunity

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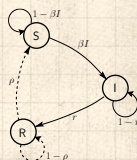
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## Original models attributed to

- 1920's: Reed and Frost
- 1920's/1930's: Kermack and McKendrick [14, 16, 15]
- Coupled differential equations with a mass-action principle

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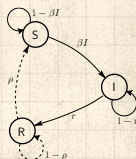
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## Differential equations for continuous model

$$\frac{d}{dt}S = -\beta IS + \rho R$$

$$\frac{d}{dt}I = \beta IS - rI$$

$$\frac{d}{dt}R = rI - \rho R$$

$\beta$ ,  $r$ , and  $\rho$  are now **rates**.

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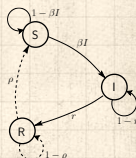
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# Reproduction Number $R_0$

## Reproduction Number $R_0$

-   $R_0$  = expected number of infected individuals resulting from a single initial infective
-  Epidemic threshold: If  $R_0 > 1$ , 'epidemic' occurs.
-  Exponential take off:  $R_0^n$  where  $n$  is the number of generations.
-  Fantastically awful notation convention:  $R_0$  and the  $R$  in  $SIR$ .

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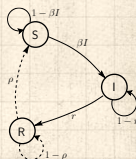
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# Reproduction Number $R_0$

## Discrete version:

- Set up: One Infective in a randomly mixing population of Susceptibles
- At time  $t = 0$ , single infective random bumps into a Susceptible
- Probability of transmission =  $\beta$
- At time  $t = 1$ , single Infective remains infected with probability  $1 - r$
- At time  $t = k$ , single Infective remains infected with probability  $(1 - r)^k$

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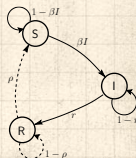
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# Reproduction Number $R_0$

Discrete version:

 Expected number infected by original infective:

$$R_0 = \beta + (1 - r)\beta + (1 - r)^2\beta + (1 - r)^3\beta + \dots$$

$$= \beta(1 + (1 - r) + (1 - r)^2 + (1 - r)^3 + \dots)$$

$$= \beta \frac{1}{1 - (1 - r)} = \beta/r$$

For  $S(0) \simeq 1$  initial susceptibles

$(1 - S(0) = R(0) =$  fraction initially immune):

$$R_0 = S(0)\beta/r$$

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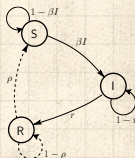
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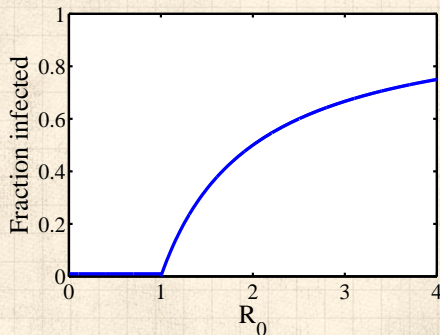
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# Independent Interaction models

Example of epidemic threshold:



- Continuous phase transition.
- Fine idea from a simple model.

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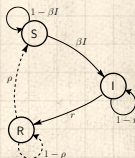
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# Independent Interaction models

For the continuous version

 Second equation:


$$\frac{d}{dt}I = \beta SI - rI$$

$$\frac{d}{dt}I = (\beta S - r)I$$

 Number of infectives grows initially if

$$\beta S(0) - r > 0 \Rightarrow \beta S(0) > r \Rightarrow \beta S(0)/r > 1$$

where  $S(0) \simeq 1$ .

 Same story as for discrete model.

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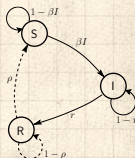
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# Independent Interaction models

## Many variants of the SIR model:

-  **SIS**: susceptible-infective-susceptible
-  **SIRS**: susceptible-infective-recovered-susceptible
-  compartment models (age or gender partitions)
-  more categories such as 'exposed' (**SEIRS**)
-  recruitment (migration, birth)

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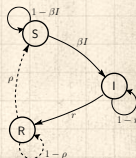
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Watch someone else pretend to save the world:

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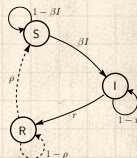
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Save the world yourself: 



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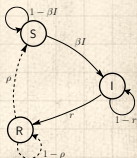
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 And you can be the virus. 

 Also contagious?: Cooperative games ...

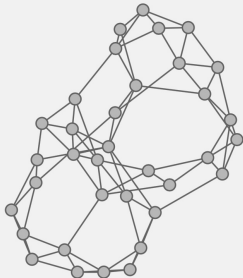


# Neural reboot—Save another pretend world with

Vax: 

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## Lesson 4: Quarantine



Vaccines take time to 'kick in' so they're ineffective if an infection has already begun to spread.

Start >

VAX!

Networks

Epidemics

Vaccines

Quarantine

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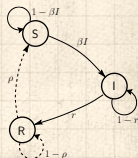
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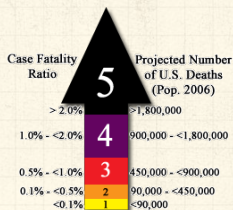
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## Pandemic severity index (PSI)

 Classification during/post pandemic:



Assumes 30% illness rate  
and unmitigated pandemic  
without interventions

CDC

U.S. Gov.



Category based.



1-5 scale.



Modeled on the

Saffir-Simpson hurricane  
scale 

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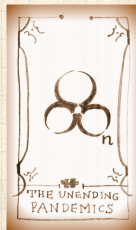
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## For novel diseases:

1. Can we predict the size of an epidemic?
2. How important is the reproduction number  $R_0$ ?

$R_0$  approximately same for all of the following:

-  1918-19 "Spanish Flu" ~ 75,000,000 world-wide, 500,000 deaths in US.
-  1957-58 "Asian Flu" ~ 2,000,000 world-wide, 70,000 deaths in US.
-  1968-69 "Hong Kong Flu" ~ 1,000,000 world-wide, 34,000 deaths in US.
-  2003 "SARS Epidemic" ~ 800 deaths world-wide.

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# Size distributions

As we know, heavy-tailed size distributions are somewhat prevalent in complex systems:

-  earthquakes (Gutenberg-Richter law)
-  city sizes, forest fires, war fatalities
-  wealth distributions
-  'popularity' (books, music, websites, ideas)
-  **Epidemics?**

Power law distributions are common but not obligatory...

Really, what about epidemics?

-  Simply hasn't attracted much attention.
-  Data not as clean as for other phenomena.

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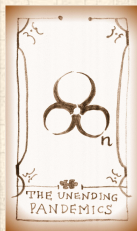
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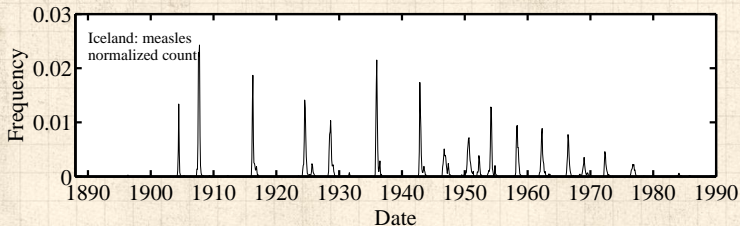
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## Caseload recorded monthly for range of diseases in Iceland, 1888-1990



Treat outbreaks separated in time as 'novel' diseases.

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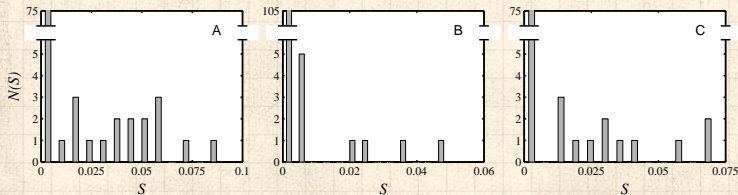
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# Really not so good at all in Iceland

Epidemic size distributions  $N(S)$  for Measles, Rubella, and Whooping Cough.



Spike near  $S = 0$ , relatively flat otherwise.

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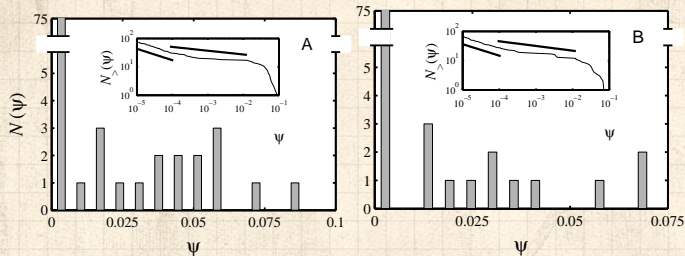
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# Measles & Pertussis



Insert plots:

Complementary cumulative frequency distributions:

$$N(\Psi' > \Psi) \propto \Psi^{-\gamma+1}$$

Limited scaling with a possible break.

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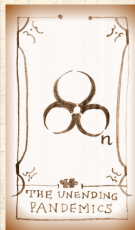
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# Power law distributions


## Measured values of $\gamma$ :

 measles: 1.40 (low  $\Psi$ ) and 1.13 (high  $\Psi$ )

 pertussis: 1.39 (low  $\Psi$ ) and 1.16 (high  $\Psi$ )

 Expect  $2 \leq \gamma < 3$  (finite mean, infinite variance)

 When  $\gamma < 1$ , can't normalize

 Distribution is quite flat.

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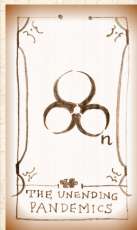
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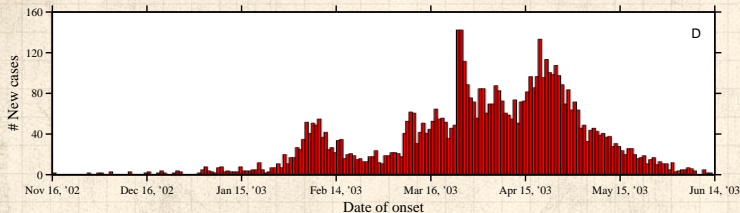
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# Resurgence—example of SARS

PoCS  
@pocsvox  
Biological  
Contagion



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Epidemic slows...

then an infective moves to a new context.



Epidemic discovers new 'pools' of susceptibles:

**Resurgence.**



**Importance of rare, stochastic events.**

# Community—S2E6: Epidemiology

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# The challenge

So... can a simple model produce

1. **broad epidemic distributions**  
and
2. **resurgence ?**

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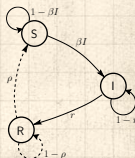
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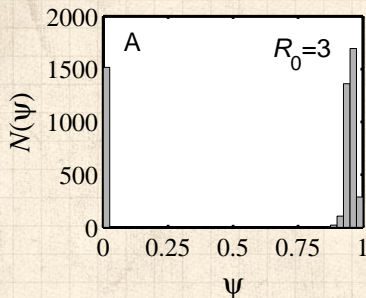
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# Size distributions



Simple models typically produce **bimodal** or **unimodal** size distributions.

 This **includes** network models: random, small-world, scale-free, ...

 Exceptions:

1. Forest fire models
2. Sophisticated metapopulation models

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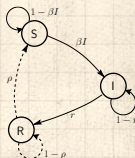
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# Burning through the population

PoCS  
@pocsvox

Biological  
Contagion

## Forest fire models: <sup>[19]</sup>

 Rhodes & Anderson, 1996

 The physicist's approach:

**"if it works for magnets, it'll work for people..."**

## A bit of a stretch:

1. Epidemics  $\equiv$  forest fires spreading on 3-d and 5-d lattices.
2. Claim Iceland and Faroe Islands exhibit power law distributions for outbreaks.
3. Original forest fire model not completely understood.

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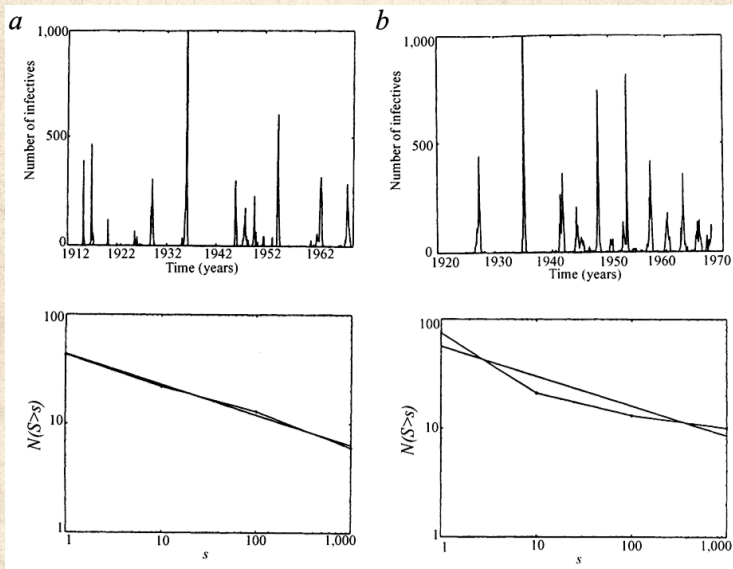
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# Size distributions



From Rhodes and Anderson, 1996.

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# Sophisticated metapopulation models:

- ☰ Multiscale models suggested earlier by others but not formalized (Bailey <sup>[1]</sup>, Cliff and Haggett <sup>[6]</sup>, Ferguson et al.)
- ☰ Community based mixing (two scales)—Longini. <sup>[17]</sup>
- ☰ Eubank et al.'s EpiSims/TRANSIMS —city simulations. <sup>[9]</sup>
- ☰ Spreading through countries—Airlines: Germann et al., Colizza et al. <sup>[7]</sup>


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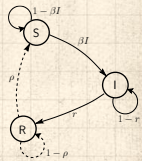
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D\_i^\* = \sum\_j D\_{ij}^{-1} (k\_j - 1) P(k\_j) (1 - R\_0^{-1/k\_j}) \left( 1 - \sum\_k \frac{D\_{ik}^\*}{P\_i} \right)

☰ GLEAM :  
Global pandemic simulations by Vespignani et al.





"The hidden geometry of complex,  
network-driven contagion phenomena"   
Brockmann and Helbing,  
Science, **342**, 1337–1342, 2013. [5]

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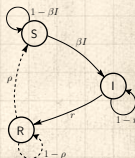
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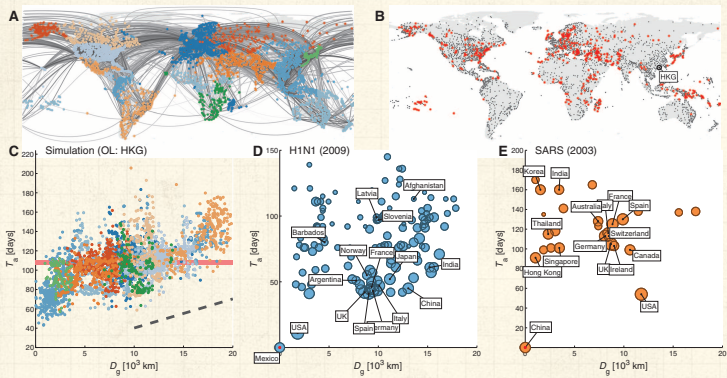
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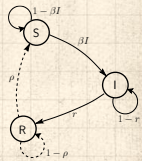
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**Fig. 1. Complexity in global, network-driven contagion phenomena.** (A) The global mobility network (GMN). Gray lines represent passenger flows along direct connections between 4069 airports worldwide. Geographic regions are distinguished by color [classified according to network modularity maximization (39)]. (B) Temporal snapshot of a simulated global pandemic with initial outbreak location (OL) in Hong Kong (HKG). The simulation is based on the metapopulation model defined by Eq. 3 with parameters  $R_0 = 1.5$ ,  $\beta = 0.285 \text{ day}^{-1}$ ,  $\gamma = 2.8 \times 10^{-3} \text{ day}^{-1}$ ,  $\epsilon = 10^{-8}$ . Red symbols depict locations with epidemic arrival times in the time window 105 days  $\leq T_a \leq 110$  days. Because of the multiscale structure of the underlying network, the spatial distribution of disease prevalence (i.e., the fraction of infected individuals) lacks geometric coherence. No clear wave-front is visible, and based on this dynamic state, the OL cannot be easily deduced. (C) For the same simulation as in (B), the panel depicts arrival times  $T_a$  as a function of geographic distance  $D_g$  from the OL [nodes are colored according to geographic region as in (A)] for each of the 4069 nodes in the network. On a

global scale,  $T_a$  weakly correlates with geographic distance  $D_g$  ( $R^2 = 0.34$ ). A linear fit yields an average global spreading speed of  $v_g = 331 \text{ km/day}$  (see also fig. S7). Using  $D_g$  and  $v_g$  to estimate arrival times for specific locations, however, does not work well owing to the strong variability of the arrival times for a given geographic distance. The red horizontal bar corresponds to the arrival time window shown in (B). (D) Arrival times versus geographic distance from the source (Mexico) for the 2009 H1N1 pandemic. Symbols represent 140 affected countries, and symbol size quantifies total traffic per country. Arrival times are defined as the date of the first confirmed case in a given country after the initial outbreak on 17 March 2009. As in the simulated scenario, arrival time and geographic distance are only weakly correlated ( $R^2 = 0.0394$ ). (E) In analogy to (D), the panel depicts the arrival times versus geographic distance from the source (China) of the 2003 SARS epidemic for 29 affected countries worldwide. Arrival times are taken from WHO published data (2). As in (C) and (D), arrival time correlates weakly with geographic distance.



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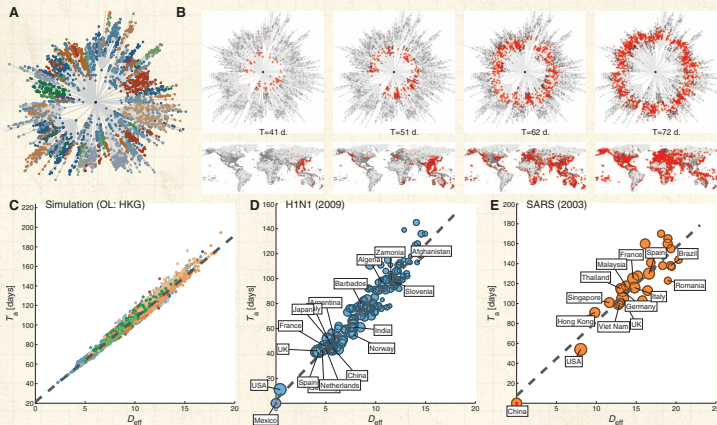
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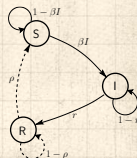
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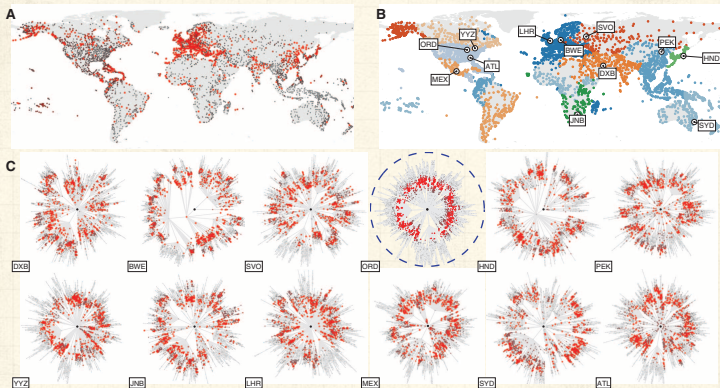
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**Fig. 2. Understanding global contagion phenomena using effective distance.** (A) The structure of the shortest path tree (in gray) from Hong Kong (central node). Radial distance represents effective distance  $D_{\text{eff}}$  as defined by Eqs. 4 and 5. Nodes are colored according to the same scheme as in Fig. 1A. (B) The sequence (from left to right) of panels depicts the time course of a simulated model disease with initial outbreak in Hong Kong (HKG), for the same parameter set as used in Fig. 1B. Prevalence is reflected by the redness of the symbols. Each panel compares the state of the system in the conventional geographic representation (bottom) with the effective distance representation (top). The complex spatial pattern in the conventional view is equivalent to a homoge-

neous wave that propagates outwards at constant effective speed in the effective distance representation. (C) Epidemic arrival time  $T_a$  versus effective distance  $D_{\text{eff}}$  for the same simulated epidemic as in (B). In contrast to geographic distance (Fig. 1C), effective distance correlates strongly with arrival time ( $R^2 = 0.973$ ), i.e., effective distance is an excellent predictor of arrival times. (D and E) Linear relationship between effective distance and arrival time for the 2009 H1N1 pandemic (D) and the 2003 SARS epidemic (E). The arrival time data are the same as in Fig. 1, D and E. The effective distance was computed from the projected global mobility network between countries. As in the model system, we observe a strong correlation between arrival time and effective distance.





**Fig. 3. Qualitative outbreak reconstruction based on effective distance.** (A) Spatial distribution of prevalence  $j_n(t)$  at time  $T = 81$  days for OL Chicago (parameters  $\beta = 0.28 \text{ day}^{-1}$ ,  $R_0 = 1.9$ ,  $\gamma = 2.8 \times 10^{-3} \text{ day}^{-1}$ , and  $\epsilon = 10^{-4}$ ). After this time, it is difficult, if not impossible, to determine the correct OL from snapshots of the dynamics. (B) Candidate OLs chosen from different geographic regions. (C) Panels depict the state of the system shown in (A) from the

perspective of each candidate OL, using each OL's shortest path tree representation. Only the actual OL (ORD, circled in blue) produces a circular wavefront. Even for comparable North American airports [Atlanta (ATL), Toronto (YYZ), and Mexico City (MEX)], the wavefronts are not nearly as concentric. Effective distances thus permit the extraction of the correct OL, based on information on the mobility network and a single snapshot of the dynamics.

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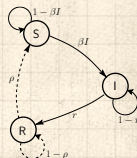
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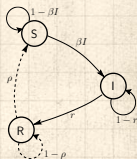
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# Size distributions

- 🧱 Vital work but perhaps hard to generalize from...
- 🧱  $\Rightarrow$  Create a simple model involving multiscale travel
- 🧱 Very big question: **What is  $N$ ?**
- 🧱 Should we model SARS in Hong Kong as spreading in a neighborhood, in Hong Kong, Asia, or the world?
- 🧱 For simple models, we need to know the final size beforehand...

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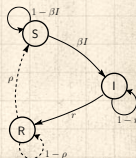
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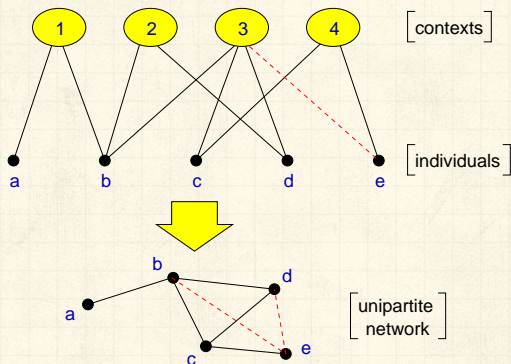
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# Improving simple models

## Contexts and Identities—Bipartite networks



-  boards of directors
-  movies
-  transportation modes (subway)

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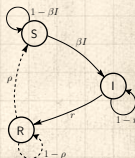
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# Improving simple models

Idea for social networks: incorporate identity

Identity is formed from attributes such as:

-  Geographic location
-  Type of employment
-  Age
-  Recreational activities

Groups are crucial...

-  formed by people with at least one similar attribute
-  Attributes  $\Leftrightarrow$  Contexts  $\Leftrightarrow$  Interactions  $\Leftrightarrow$  Networks. [23]

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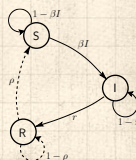
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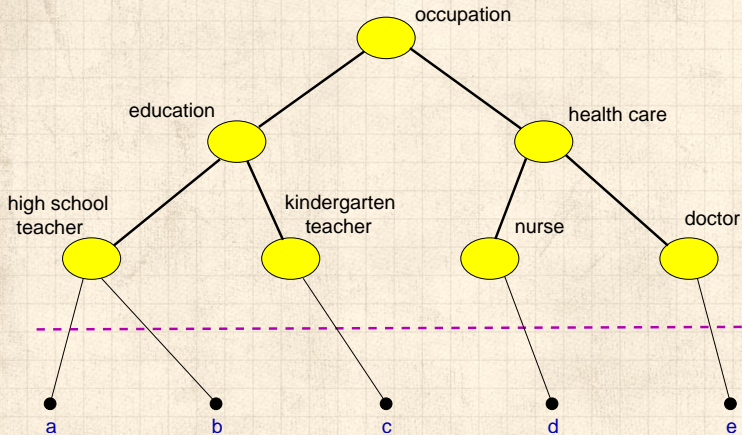
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# Infer interactions/network from identities



Distance makes sense in identity/context space.

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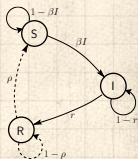
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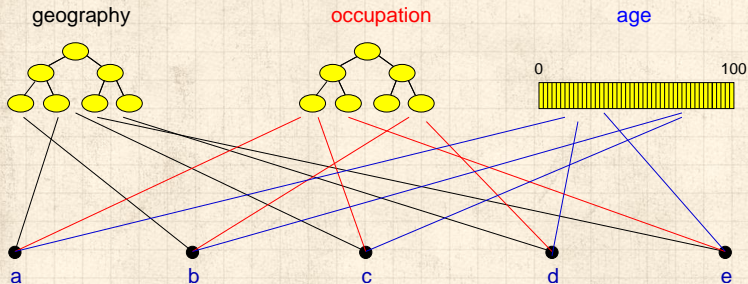
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# Generalized context space



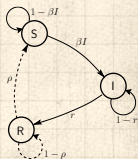
(Blau & Schwartz <sup>[3]</sup>, Simmel <sup>[20]</sup>, Breiger <sup>[4]</sup>)

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# A toy agent-based model:



“Multiscale, resurgent epidemics in a hierarchical metapopulation model” ↗

Watts et al.,

Proc. Natl. Acad. Sci., **102**, 11157–11162, 2005. [24]

Geography: allow people to move between contexts

🧱 Locally: standard SIR model with random mixing

🧱 discrete time simulation

🧱  $\beta$  = infection probability

🧱  $\gamma$  = recovery probability

🧱  $P$  = probability of travel

🧱 **Movement distance:**  $\Pr(d) \propto \exp(-d/\xi)$

🧱  $\xi$  = typical travel distance

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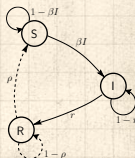
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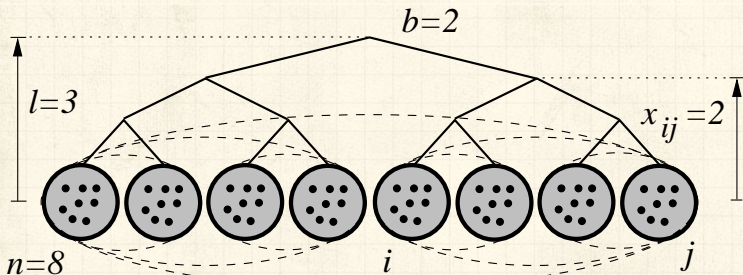
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# A toy agent-based model

## Schematic:



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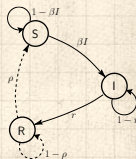
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- Define  $P_0$  = Expected number of infected individuals **leaving** initially infected context.
- Need  $P_0 > 1$  for disease to spread (independent of  $R_0$ ).
- Limit epidemic size by **restricting frequency of travel and/or range**

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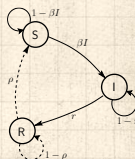
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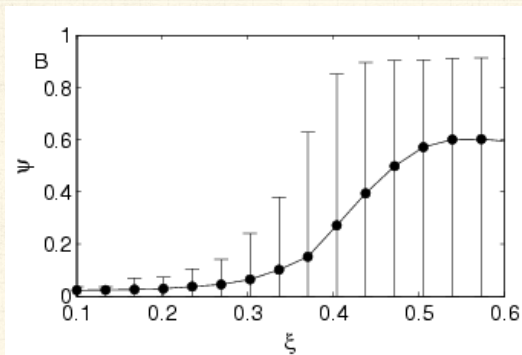
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Varying  $\xi$ :



Transition in expected final size based on typical movement distance (**sensible**)

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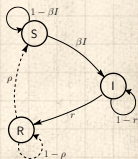
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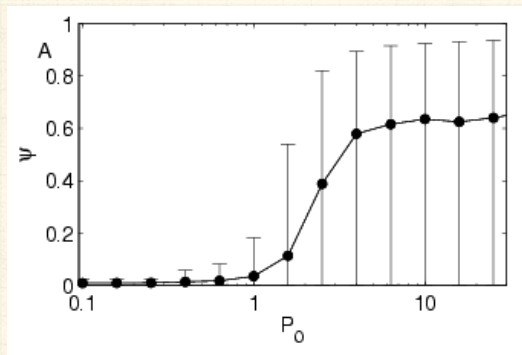
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# Model output

Varying  $P_0$ :



 Transition in expected final size based on typical number of infectives leaving first group (also sensible)

 Travel advisories:  $\xi$  has larger effect than  $P_0$ .

PoCS  
@pocsvox  
Biological  
Contagion

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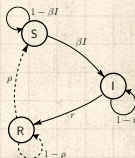
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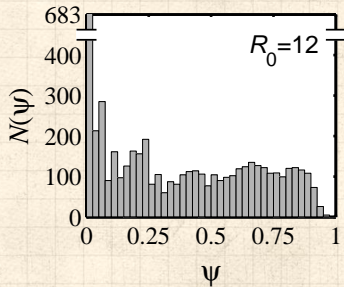
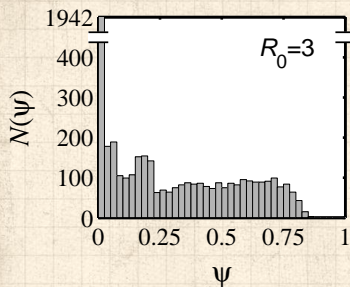
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# Example model output: size distributions



- Flat distributions are possible for certain  $\xi$  and  $P$ .
- Different  $R_0$ 's may produce similar distributions
- Same epidemic sizes may arise from different  $R_0$ 's

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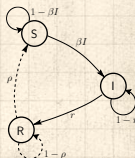
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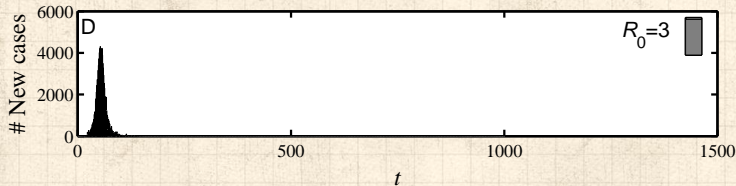
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# Model output—resurgence

## Standard model:

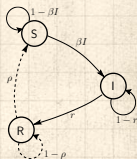


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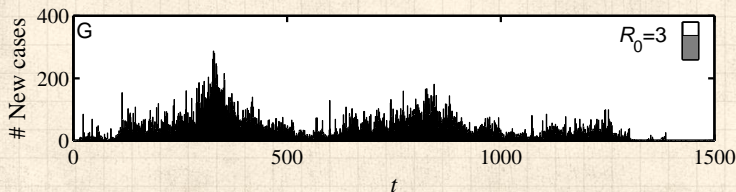
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# Model output—resurgence

## Standard model with transport:

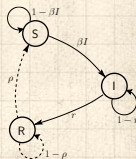


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# The upshot

Simple multiscale population structure

+

stochasticity

leads to

resurgence

+

broad epidemic size distributions

PoCS  
@pocsvox

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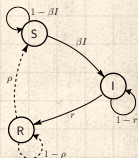
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- For the hierarchical movement model, epidemic size is highly unpredictable
- Model is more complicated than SIR but still simple.
- We haven't even included normal social responses such as travel bans and self-quarantine.
- The reproduction number  $R_0$  is not terribly useful.
- $R_0$ , however measured, is not informative about
  - how likely the observed epidemic size was,
  - and how likely future epidemics will be.
- Problem:  $R_0$  summarises **one** epidemic after the fact and enfolds movement, the price of bananas, everything.

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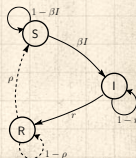
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# Conclusions

- 🧱 Disease's spread is highly sensitive to population structure.
- 🧱 Rare events may matter enormously: e.g., an infected individual taking an international flight.
- 🧱 More support for controlling population movement: e.g., travel advisories, quarantine

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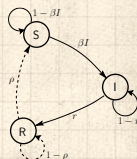
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## What to do:

- Need to separate movement from disease
- $R_0$  needs a friend or two.
- Need  $R_0 > 1$  and  $P_0 > 1$  and  $\xi$  sufficiently large for disease to have a chance of spreading
- And in general: keep building up the kitchen sink models.

## More wondering:

- Exactly how important are rare events in disease spreading?
- Again, what is  $N$ ?

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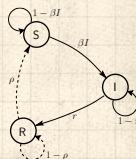
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## Krugman, 1998: "Why most economists' predictions are wrong."



"The growth of the Internet will slow drastically, as the flaw in "Metcalfe's law"—which states that the number of potential connections in a network is proportional to the square of the number of participants—becomes apparent: most people have nothing to say to each other! By 2005 or so, it will become clear that the Internet's impact on the economy has been no greater than the fax machine's."<sup>1</sup>

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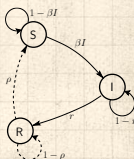
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<sup>1</sup><http://www.redherring.com/mag/issue55/economics.html>

Alan Greenspan (September 18, 2007):

"I've been dealing with these big mathematical models of forecasting the economy ...

If I could figure out a way to determine whether or not people are more fearful or changing to more euphoric,

I don't need any of this other stuff.

I could forecast the economy better than any way I know."



<http://wikipedia.org>

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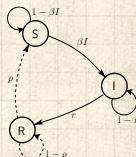
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# Economics, Schmeconomics

Greenspan continues:

"The trouble is that we can't figure that out. I've been in the forecasting business for 50 years. I'm no better than I ever was, and nobody else is. Forecasting 50 years ago was as good or as bad as it is today. And the reason is that human nature hasn't changed. We can't improve ourselves."

Jon Stewart:

"You just bummed the @\*!# out of me."



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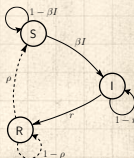
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From the Daily Show  (September 18, 2007)

The full episode is here:

<http://www.cc.com/video-clips/cenrt5/the-daily-show-with-jon-stewart>



# Predicting social catastrophe isn't easy...

## "Greenspan Concedes Error on Regulation"

...humbled Mr. Greenspan admitted that he had put too much faith in the self-correcting power of free markets ...

"Those of us who have looked to the self-interest of lending institutions to protect shareholders' equity, myself included, are in a state of shocked disbelief"

Rep. Henry A. Waxman: "Do you feel that your ideology pushed you to make decisions that you wish you had not made?"

Mr. Greenspan conceded: "Yes, I've found a flaw. I don't know how significant or permanent it is. But I've been very distressed by that fact."

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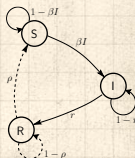
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# Economics, Schmeconomics

James K. Galbraith:

NYT But there are at least 15,000 professional economists in this country, and you're saying only two or three of them foresaw the mortgage crisis? [JKG] Ten or 12 would be closer than two or three.

NYT What does that say about the field of economics, which claims to be a science? [JKG] It's an enormous blot on the reputation of the profession. There are thousands of economists. Most of them teach. And most of them teach a theoretical framework that has been shown to be fundamentally useless.

From the [New York Times, 11/02/2008](#)

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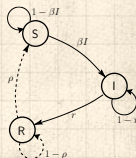
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## Other attempts to use SIR and co. elsewhere:

- Adoption of ideas/beliefs (Goffman & Newell, 1964)<sup>[11]</sup>
- Spread of rumors (Daley & Kendall, 1965)<sup>[8]</sup>
- Diffusion of innovations (Bass, 1969)<sup>[2]</sup>
- Spread of fanatical behavior (Castillo-Chávez & Song, 2003)
- Spread of Feynmann diagrams (Bettencourt et al., 2006)

## Social contagion:

- SIR may apply sometimes ...
- But we need new fundamental models.
- Next up: Thresholds.

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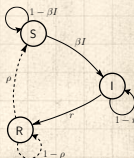
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# We really should know social contagion is different but ...

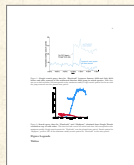



"It's contagious: Rethinking a metaphor dialogically" 

Warren and Power,  
Culture & Psychology, **21**, 359–379,  
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"Facebook will lose 80% of users by 2017, say Princeton researchers"  (Guardian, 2014)



"Epidemiological modeling of online social network dynamics" 

Spechler and Cannarella,  
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<http://arxiv.org/abs/1401.4208>, 2014. [21]

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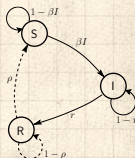
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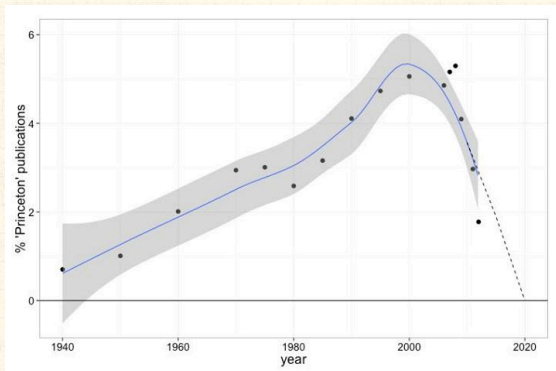
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## The Facebook Data Science team's response



Mike Develin, Lada Adamic, and Sean Taylor.

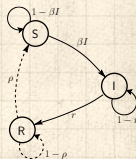
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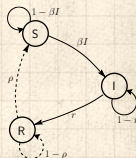
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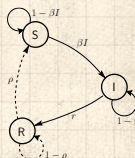
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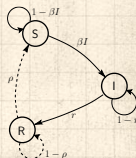
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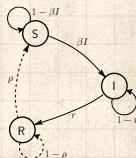
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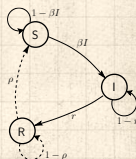
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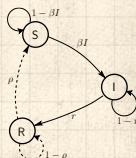
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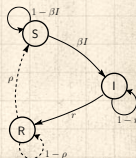
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