

Generalized Contagion

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Principles of Complex Systems, Vols. 1, 2, & 3D
CSYS/MATH 6701, 6713, & a pretend number,
2023–2024 | @pocsvox

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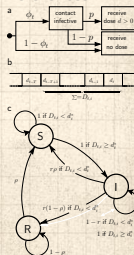
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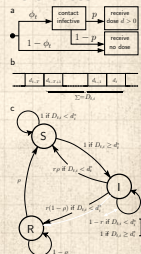
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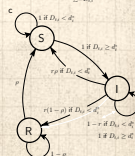
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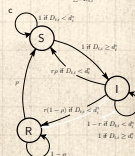
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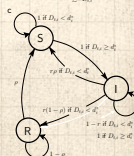
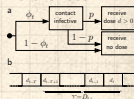
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“Universal Behavior in a Generalized Model of Contagion” ↗

Dodds and Watts,
Phys. Rev. Lett., **92**, 218701, 2004. [5]



“A generalized model of social and biological contagion” ↗

Dodds and Watts,
J. Theor. Biol., **232**, 587–604, 2005. [6]

Generalized contagion model

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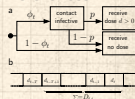
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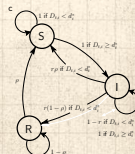
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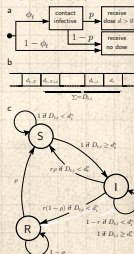


Basic questions about contagion


- How many types of contagion are there?
- How can we categorize real-world contagions?
- Can we connect models of disease-like and social contagion?
- Focus:** mean field models.




Mathematical Epidemiology (recap)





The standard SIR model^[11]


 The basic model of disease contagion


 Three states:

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

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

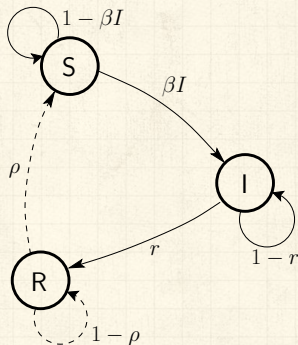
 Presumes random interactions (mass-action principle)

 Interactions are independent (no memory)

 Discrete and continuous time versions

Independent Interaction Models

Discrete time automata example:

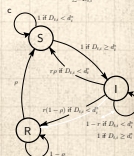
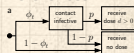


Transition Probabilities:

β for being infected given
contact with infected

r for recovery

ρ for loss of immunity



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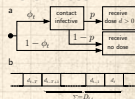
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Original models attributed to



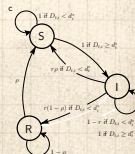
1920's: Reed and Frost



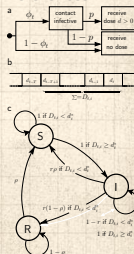
1920's/1930's: Kermack and McKendrick [8, 10, 9]



Coupled differential equations with a mass-action principle



Independent Interaction models



Differential equations for continuous model


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


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

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

β , r , and ρ are now **rates**.

Reproduction Number R_0 :

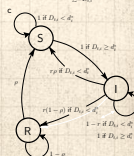
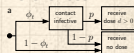
 R_0 = expected number of infected individuals resulting from a single initial infective

 Epidemic threshold: If $R_0 > 1$, 'epidemic' occurs.

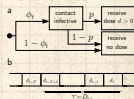
Reproduction Number R_0

Discrete version:


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



Reproduction Number R_0




Discrete version:

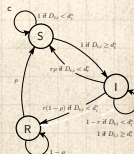
 Expected number infected by original Infective:

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

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

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

 Similar story for continuous model.



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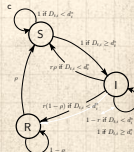
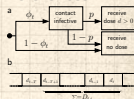
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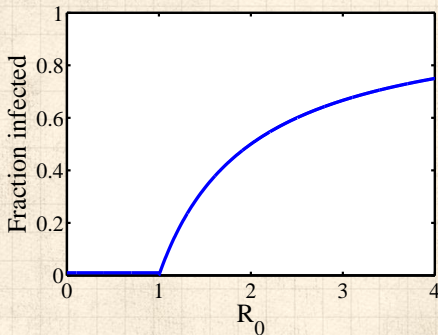
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Example of epidemic threshold:



Continuous phase transition.

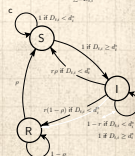


Fine idea from a simple model.

Simple disease spreading models

Valiant attempts to use SIR and co. elsewhere:

- Adoption of ideas/beliefs (Goffman & Newell, 1964) [7]
- Spread of rumors (Daley & Kendall, 1964, 1965) [3, 4]
- Diffusion of innovations (Bass, 1969) [1]
- Spread of fanatical behavior (Castillo-Chávez & Song, 2003) [2]



Granovetter's model (recap of recap)

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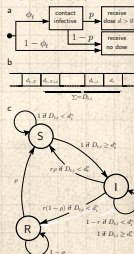
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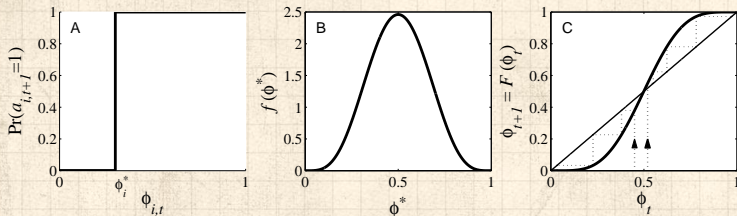
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 Action based on perceived behavior of others.




 Two states: S and I.

 Recovery now possible (SIS).

 ϕ = fraction of contacts 'on' (e.g., rioting).

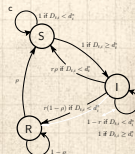
 Discrete time, synchronous update.

 This is a **Critical mass model**.

 **Inter**dependent interaction model.

Some (of many) issues

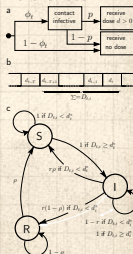
- ❏ Disease models assume independence of infectious events.
- ❏ Threshold models only involve proportions:
 $3/10 \equiv 30/100$.
- ❏ Threshold models ignore exact sequence of influences
- ❏ Threshold models assume immediate polling.
- ❏ Mean-field models neglect network structure
- ❏ Network effects only part of story:
media, advertising, direct marketing.



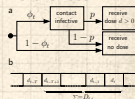
Generalized model

Basic ingredients:

- ✉ Incorporate memory of a contagious element [5, 6]
- ✉ Population of N individuals, each in state S, I, or R.
- ✉ Each individual randomly contacts another at each time step.
- ✉ ϕ_t = fraction infected at time t
= probability of contact with infected individual
- ✉ With probability p , contact with infective leads to an exposure.
- ✉ If exposed, individual receives a dose of size d drawn from distribution f . Otherwise $d = 0$.



Generalized model—ingredients



$$S \Rightarrow I$$

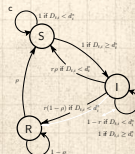
Individuals 'remember' last T contacts:

$$D_{t,i} = \sum_{t'=t-T+1}^t d_i(t')$$

Infection occurs if individual i 's 'threshold' is exceeded:

$$D_{t,i} \geq d_i^*$$

Threshold d_i^* drawn from arbitrary distribution g at $t = 0$.



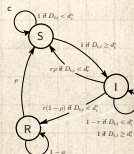
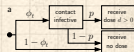
Generalized model—ingredients

I \Rightarrow R

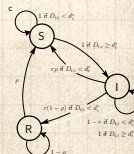
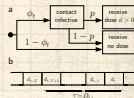
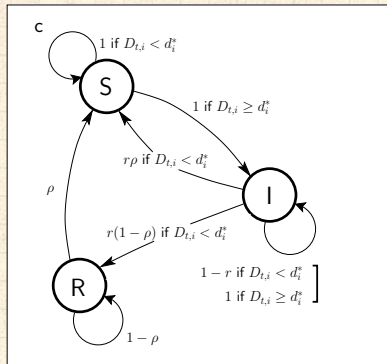
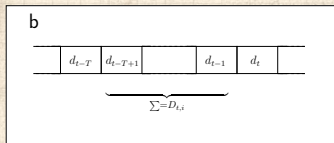
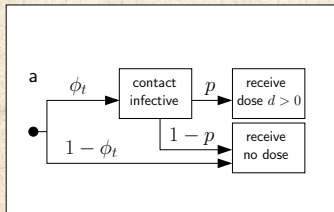
When $D_{t,i} < d_i^*$,
individual i recovers to state R with probability r .

R \Rightarrow S

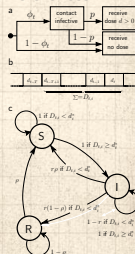
Once in state R, individuals become susceptible again
with probability ρ .



A visual explanation



Generalized mean-field model



Study SIS-type contagion first:

Recovered individuals are immediately susceptible again:

$$\rho = 1.$$

Look for steady-state behavior as a function of exposure probability p .

Denote fixed points by ϕ^* .





Homogeneous version:

All individuals have threshold d^*

All dose sizes are equal: $d = 1$

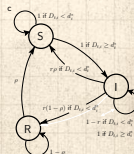
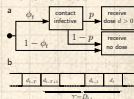
Homogeneous, one hit models:

Fixed points for $r < 1$, $d^* = 1$, and $T = 1$:

-  $r < 1$ means recovery is probabilistic.
-  $T = 1$ means individuals forget past interactions.
-  $d^* = 1$ means one positive interaction will infect an individual.
-  Evolution of infection level:

$$\phi_{t+1} = \underbrace{p\phi_t}_a + \underbrace{\phi_t(1 - p\phi_t)}_b \underbrace{(1 - r)}_c.$$

- a: Fraction infected between t and $t + 1$, independent of past state or recovery.
- b: Probability of being infected and not being reinfected.
- c: Probability of not recovering.



Homogeneous, one hit models:

Fixed points for $r < 1$, $d^* = 1$, and $T = 1$:

🧱 Set $\phi_t = \phi^*$:

$$\phi^* = p\phi^* + (1 - p\phi^*)\phi^*(1 - r)$$

$$\Rightarrow 1 = p + (1 - p\phi^*)(1 - r), \quad \phi^* \neq 0,$$

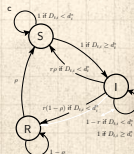
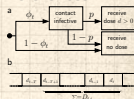
$$\Rightarrow \phi^* = \frac{1 - r/p}{1 - r} \quad \text{and} \quad \phi^* = 0.$$

🧱 Critical point at $p = p_c = r$.

🧱 Spreading takes off if $p/r > 1$







🧱 Find continuous phase transition as for SIR model.

🧱 Goodness: Matches $R_o = \beta/\gamma > 1$ condition.

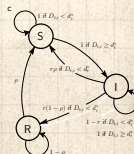
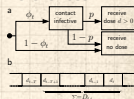


Simple homogeneous examples

Fixed points for $r = 1$, $d^* = 1$, and $T > 1$

-  $r = 1$ means recovery is immediate.
-  $T > 1$ means individuals remember at least 2 interactions.
-  $d^* = 1$ means only one positive interaction in past T interactions will infect individual.
-  Effect of individual interactions is independent from effect of others.
-  Call ϕ^* the steady state level of infection.
-  $\Pr(\text{infected}) = 1 - \Pr(\text{uninfected})$:

$$\phi^* = 1 - (1 - p\phi^*)^T.$$



Homogeneous, one hit models:

Fixed points for $r = 1$, $d^* = 1$, and $T > 1$

🧱 Closed form expression for ϕ^* :

$$\phi^* = 1 - (1 - p\phi^*)^T.$$

🧱 Look for critical infection probability p_c .

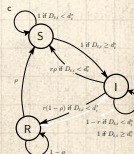
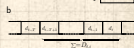
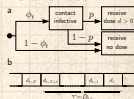
🧱 As $\phi^* \rightarrow 0$, we see

$$\phi^* \simeq pT\phi^* \Rightarrow p_c = 1/T.$$

🧱 Again find continuous phase transition ...

🧱 Note: we can solve for p but not ϕ^* :

$$p = (\phi^*)^{-1} [1 - (1 - \phi^*)^{1/T}].$$



Homogeneous, one hit models:

Fixed points for $r \leq 1$, $d^* = 1$, and $T \geq 1$

Start with $r = 1$, $d^* = 1$, and $T \geq 1$ case we have just examined:

$$\phi^* = 1 - (1 - p\phi^*)^T.$$

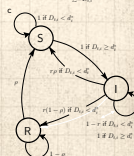
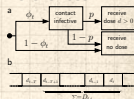
For $r < 1$, add to right hand side fraction who:

1. Did not receive any infections in last T time steps,
2. And **did not recover** from a previous infection.

Define corresponding dose histories. Example:


$$H_1 = \{\dots, d_{t-T-2}, d_{t-T-1}, 1, \underbrace{0, 0, \dots, 0, 0}_{T \text{ 0's}}\},$$

With history H_1 , probability of being infected (not recovering in one time step) is $1 - r$.




Homogeneous, one hit models:

Fixed points for $r \leq 1$, $d^* = 1$, and $T \geq 1$

 In general, relevant dose histories are:

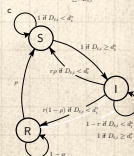
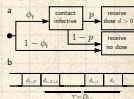
$$H_{m+1} = \{ \dots, d_{t-T-m-1}, 1, \underbrace{0, 0, \dots, 0, 0}_m \text{ 0's}, \underbrace{0, 0, \dots, 0, 0}_T \text{ 0's} \}.$$

 Overall probabilities for dose histories occurring:

$$P(H_1) = p\phi^*(1 - p\phi^*)^T(1 - r),$$


$$P(H_{m+1}) = \underbrace{p\phi^*}_a \underbrace{(1 - p\phi^*)^{T+m}}_b \underbrace{(1 - r)^{m+1}}_c.$$

- a: Pr(infection $T + m + 1$ time steps ago)
- b: Pr(no doses received in $T + m$ time steps since)
- c: Pr(no recovery in m chances)




Homogeneous, one hit models:

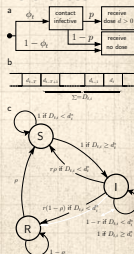
Fixed points for $r \leq 1$, $d^* = 1$, and $T \geq 1$

 Pr(recovery) = Pr(seeing no doses for at least T time steps and recovering)

$$\begin{aligned} &= r \sum_{m=0}^{\infty} P(H_{T+m}) = r \sum_{m=0}^{\infty} p\phi^*(1-p\phi^*)^{T+m}(1-r)^m \\ &= r \frac{p\phi^*(1-p\phi^*)^T}{1-(1-p\phi^*)(1-r)}. \end{aligned}$$


 Using the probability of not recovering, we end up with a fixed point equation:

$$\phi^* = 1 - \frac{r(1-p\phi^*)^T}{1-(1-p\phi^*)(1-r)}.$$




Homogeneous, one hit models:

Fixed points for $r \leq 1$, $d^* = 1$, and $T \geq 1$

 Fixed point equation (again):


$$\phi^* = 1 - \frac{r(1 - p\phi^*)^T}{1 - (1 - p\phi^*)(1 - r)}.$$

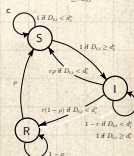
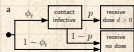
 Find critical exposure probability by examining above as $\phi^* \rightarrow 0$.



$$\Rightarrow p_c = \frac{1}{T + 1/r - 1} = \frac{1}{T + \tau}.$$

where $\tau =$ mean recovery time for simple relaxation process.

 Decreasing r keeps individuals infected for longer and decreases p_c .



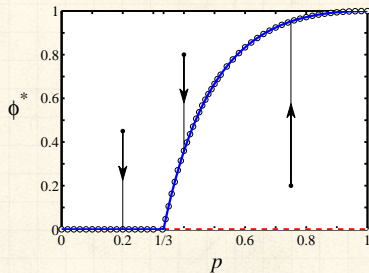
Epidemic threshold:

Fixed points for $d^* = 1$, $r \leq 1$, and $T \geq 1$

$$\phi^* = 1 - \frac{r(1-p\phi^*)^T}{1-(1-p\phi^*)(1-r)}$$

$$\phi^* = 0$$

$$p_c = 1/(T + \tau)$$



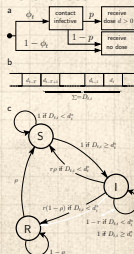
Example details: $T = 2$ & $r = 1/2 \Rightarrow p_c = 1/3$.

Blue = stable, red = unstable, fixed points.







$\tau = 1/r - 1 =$ characteristic recovery time = 1.

$T + \tau \simeq$ average memory in system = 3.


Phase transition can be seen as a **transcritical bifurcation**. [12]

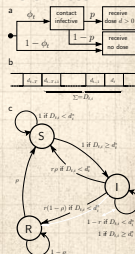


Homogeneous, multi-hit models:

-  All right: $d^* = 1$ models correspond to simple disease spreading models.
-  What if we allow $d^* \geq 2$?
-  Again first consider SIS with immediate recovery ($r = 1$)
-  Also continue to assume unit dose sizes ($f(d) = \delta(d - 1)$).
-  To be infected, must have at least d^* exposures in last T time steps.
-  Fixed point equation:


$$\phi^* = \sum_{i=d^*}^T \binom{T}{i} (p\phi^*)^i (1 - p\phi^*)^{T-i}.$$


-  As always, $\phi^* = 0$ works too.

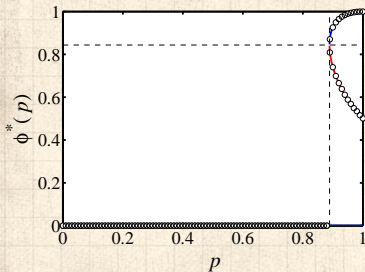



Homogeneous, multi-hit models:

Fixed points for $r = 1$, $d^* > 1$, and $T \geq 1$


 Exactly solvable for small T .


 e.g., for $d^* = 2$, $T = 3$:




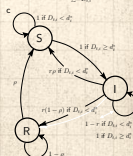
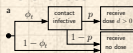
 Fixed point equation:

$$\phi^* = 3p^2 \phi^{*2} (1 - p\phi^*) + p^3 \phi^{*3}$$

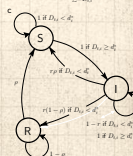
 See new structure: a **saddle node bifurcation**^[12] appears as p increases.

 $(p_b, \phi^*) = (8/9, 27/32)$.

 Behavior akin to output of Granovetter's threshold model.

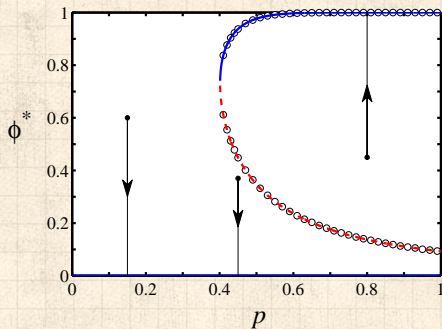


Homogeneous, multi-hit models:



Another example:


Critical Mass Models

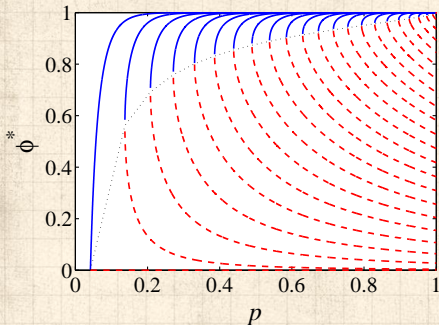



$r = 1, d^* = 3, T = 12$


Saddle-node bifurcation.


Fixed points for $r = 1$, $d^* \geq 1$, and $T \geq 1$

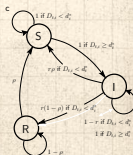
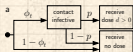
 $T = 24$, $d^* = 1, 2, \dots, 23$.




 See **either** simple phase transition or saddle-node bifurcation, nothing in between.

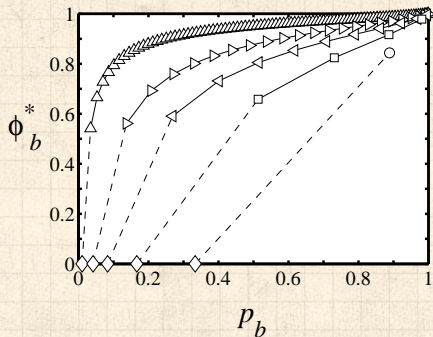
 $d^* = 1 \rightarrow d^* > 1$:
jump between
continuous
phase transition
and pure critical
mass model.


 Unstable curve
for $d^* = 2$ **does**
not hit $\phi^* = 0$.



Fixed points for $r = 1$, $d^* > 1$, and $T \geq 1$


 Bifurcation points for example fixed T , varying d^* :




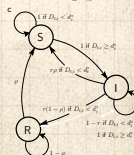
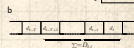
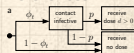
 $T = 96$ (.),

 $T = 24$ (\triangleright),

 $T = 12$ (\triangleleft),

 $T = 6$ (\square),

 $T = 3$ (\circ),



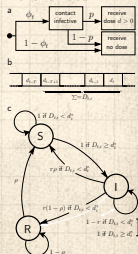
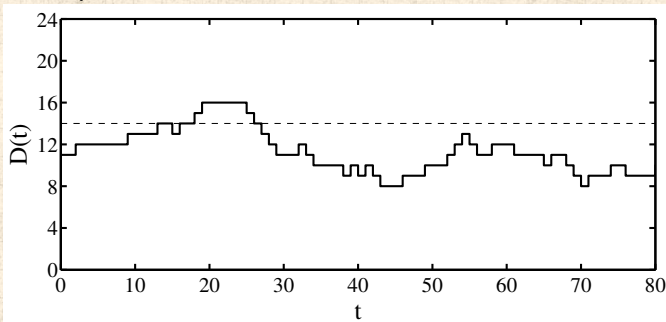
Fixed points for $r < 1$, $d^* > 1$, and $T \geq 1$

For $r < 1$, need to determine probability of recovering as a function of time since dose load last dropped below threshold.


Partially summed random walks:


$$D_i(t) = \sum_{t'=t-T+1}^t d_i(t')$$

Example for $T = 24$, $d^* = 14$:




Fixed points for $r < 1$, $d^* > 1$, and $T \geq 1$

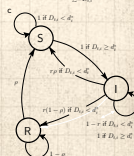
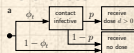
 Define γ_m as fraction of individuals for whom $D(t)$ last equaled, and has since been below, their threshold m time steps ago,

 Fraction of individuals below threshold but not recovered:

$$\Gamma(p, \phi^*; r) = \sum_{m=1}^{\infty} (1-r)^m \gamma_m(p, \phi^*).$$

 Fixed point equation:

$$\phi^* = \Gamma(p, \phi^*; r) + \sum_{i=d^*}^T \binom{T}{i} (p\phi^*)^i (1-p\phi^*)^{T-i}.$$



Fixed points for $r < 1$, $d^* > 1$, and $T \geq 1$

Example: $T = 3$, $d^* = 2$

- Want to examine how dose load can drop below threshold of $d^* = 2$:

$$D_n = 2 \Rightarrow D_{n+1} = 1$$

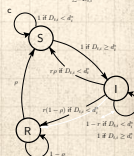
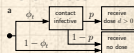
- Two subsequences do this:

$$\{d_{n-2}, d_{n-1}, d_n, d_{n+1}\} = \{1, 1, 0, 0\}$$
$$\text{and } \{d_{n-2}, d_{n-1}, d_n, d_{n+1}, d_{n+2}\} = \{1, 0, 1, 0, 0\}.$$


- Note: second sequence includes an extra 0 since this is necessary to stay below $d^* = 2$.


- To stay below threshold, observe acceptable following sequences may be composed of any combination of two subsequences:

$$a = \{0\} \quad \text{and} \quad b = \{1, 0, 0\}.$$



Fixed points for $r < 1$, $d^* > 1$, and $T \geq 1$

 Determine number of sequences of length m that keep dose load below $d^* = 2$.

 N_a = number of $a = \{0\}$ subsequences.


 N_b = number of $b = \{1, 0, 0\}$ subsequences.

$$m = N_a \cdot 1 + N_b \cdot 3$$

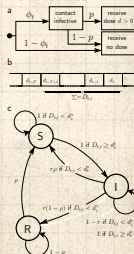
Possible values for N_b :

$$0, 1, 2, \dots, \left\lfloor \frac{m}{3} \right\rfloor.$$

where $\lfloor \cdot \rfloor$ means floor.

 Corresponding possible values for N_a :

$$m, m - 3, m - 6, \dots, m - 3 \left\lfloor \frac{m}{3} \right\rfloor.$$



Fixed points for $r < 1$, $d^* > 1$, and $T \geq 1$

How many ways to arrange N_a a 's and N_b b 's?

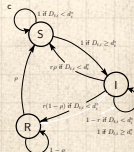
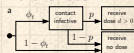
Think of overall sequence in terms of subsequences:

$$\{Z_1, Z_2, \dots, Z_{N_a + N_b}\}$$

$N_a + N_b$ slots for subsequences.

Choose positions of either a 's or b 's:

$$\binom{N_a + N_b}{N_a} = \binom{N_a + N_b}{N_b}$$



Fixed points for $r < 1$, $d^* > 1$, and $T \geq 1$

🧱 Total number of allowable sequences of length m :

$$\sum_{N_b=0}^{\lfloor m/3 \rfloor} \binom{N_b + N_a}{N_b} = \sum_{k=0}^{\lfloor m/3 \rfloor} \binom{m - 2k}{k}$$

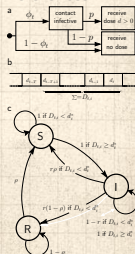
where $k = N_b$ and we have used $m = N_a + 3N_b$.

🧱 $P(a) = (1 - p\phi^*)$ and $P(b) = p\phi^*(1 - p\phi^*)^2$


🧱 Total probability of allowable sequences of length m :


$$\chi_m(p, \phi^*) = \sum_{k=0}^{\lfloor m/3 \rfloor} \binom{m - 2k}{k} (1 - p\phi^*)^{m-k} (p\phi^*)^k.$$

🧱 Notation: Write a randomly chosen sequence of a 's and b 's of length m as $D_m^{a,b}$.



Fixed points for $r < 1$, $d^* > 1$, and $T \geq 1$

 Nearly there ...must account for details of sequence endings.

 Three endings \Rightarrow Six possible sequences:

$$D_1 = \{1, 1, 0, 0, D_{m-1}^{a,b}\}$$

$$D_2 = \{1, 1, 0, 0, D_{m-2}^{a,b}, 1\}$$

$$D_3 = \{1, 1, 0, 0, D_{m-3}^{a,b}, 1, 0\}$$

$$D_4 = \{1, 0, 1, 0, 0, D_{m-2}^{a,b}\}$$

$$D_5 = \{1, 0, 1, 0, 0, D_{m-3}^{a,b}, 1\}$$

$$D_6 = \{1, 0, 1, 0, 0, D_{m-4}^{a,b}, 1, 0\}$$

$$P_1 = (p\phi)^2(1-p\phi)^2\chi_{m-1}(p, \phi)$$

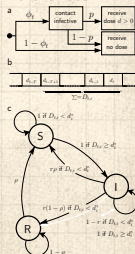
$$P_2 = (p\phi)^3(1-p\phi)^2\chi_{m-2}(p, \phi)$$

$$P_3 = (p\phi)^3(1-p\phi)^3\chi_{m-3}(p, \phi)$$

$$P_4 = (p\phi)^2(1-p\phi)^3\chi_{m-2}(p, \phi)$$

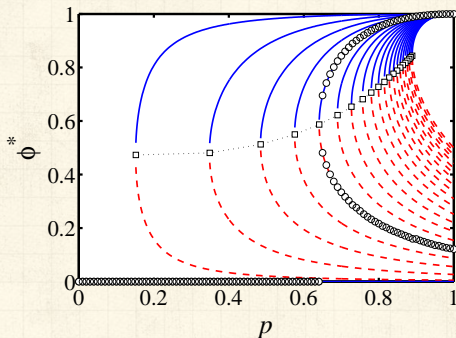
$$P_5 = (p\phi)^3(1-p\phi)^3\chi_{m-3}(p, \phi)$$


$$P_6 = (p\phi)^3(1-p\phi)^4\chi_{m-4}(p, \phi)$$



Fixed points for $r < 1$, $d^* > 1$, and $T \geq 1$

$$T = 3, d^* = 2$$



 $r = 0.01, 0.05, 0.10, 0.15, 0.20, \dots, 1.00$.

The PoCVerse
Generalized
Contagion
45 of 65

Introduction

Independent
Interaction
models

Interdependent
interaction
models

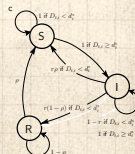
Generalized
Model

Homogeneous version
Heterogeneous version

Nutshell

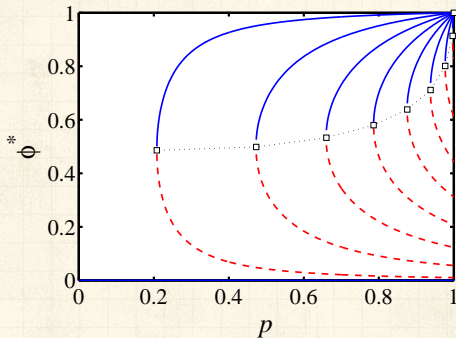
Appendix


References




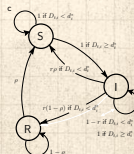
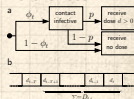
Fixed points for $r < 1$, $d^* > 1$, and $T \geq 1$

$$T = 2, d^* = 2$$



 $r = 0.01, 0.05, 0.10, \dots, 0.3820 \pm 0.0001$.

 No spreading for $r \gtrsim 0.382$.



What we have now:



Two kinds of contagion processes:

1. Continuous phase transition: **SIR-like**.
2. Saddle-node bifurcation: **threshold model-like**.



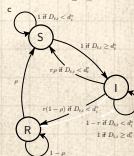
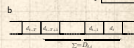
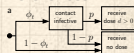
$d^* = 1$: spreading from small seeds possible.



$d^* > 1$: critical mass model.



Are other behaviors possible?



Generalized model

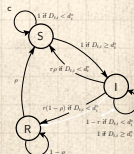
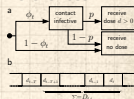
Now allow for general dose distributions (f) and threshold distributions (g).

Key quantities:

$$P_k = \int_0^{\infty} dd^* g(d^*) P \left(\sum_{j=1}^k d_j \geq d^* \right) \text{ where } 1 \leq k \leq T.$$

P_k = Probability that the threshold of a randomly selected individual will be exceeded by k doses.

e.g.,
 P_1 = Probability that one dose will exceed the threshold of a random individual
= Fraction of most vulnerable individuals.



Generalized model—heterogeneity, $r = 1$

Fixed point equation:

$$\phi^* = \sum_{k=1}^T \binom{T}{k} (p\phi^*)^k (1 - p\phi^*)^{T-k} \underline{P}_k$$

Expand around $\phi^* = 0$ to find when spread from single seed is possible:

$$pP_1T \geq 1$$

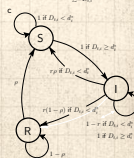
or

$$\Rightarrow p_c = 1/(TP_1)$$

Very good:

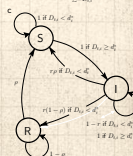
1. P_1T is the expected number of vulnerables the initial infected individual meets before recovering.
2. pP_1T is \therefore the expected number of successful infections (equivalent to R_0).

Observe: p_c may exceed 1 meaning no spreading from a small seed.




Heterogeneous case


- Next: Determine slope of fixed point curve at critical point p_c .
- Expand fixed point equation around $(p, \phi^*) = (p_c, 0)$.
- Find slope depends on $(P_1 - P_2/2)$ [6] (see Appendix).
- Behavior near fixed point depends on whether this slope is
 - positive: $P_1 > P_2/2$ (continuous phase transition)
 - negative: $P_1 < P_2/2$ (discontinuous phase transition)
- Now find **three** basic universal classes of contagion models ...




Heterogeneous case

Example configuration:

 Dose sizes are lognormally distributed with mean 1 and variance 0.433.


 Memory span: $T = 10$.

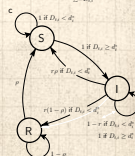
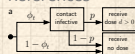
 Thresholds are uniformly set at

1. $d_* = 0.5$

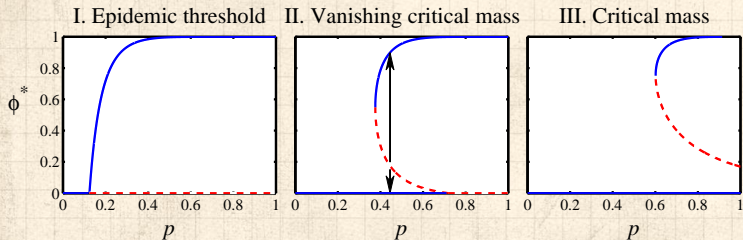
2. $d_* = 1.6$

3. $d_* = 3$


 Spread of dose sizes matters, details are not important.



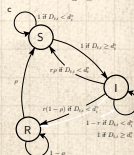
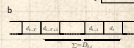
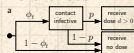
Three universal classes



 Epidemic threshold: $P_1 > P_2/2, p_c = 1/(TP_1) < 1$

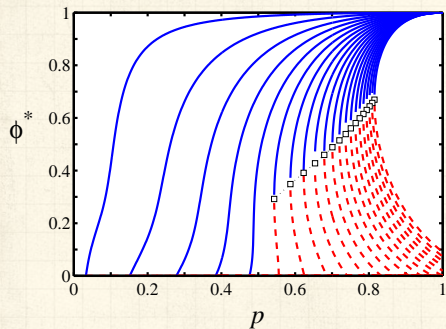
 Vanishing critical mass: $P_1 < P_2/2, p_c = 1/(TP_1) < 1$

 Pure critical mass: $P_1 < P_2/2, p_c = 1/(TP_1) > 1$



Heterogeneous case

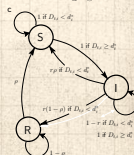
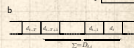
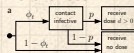
Now allow $r < 1$:



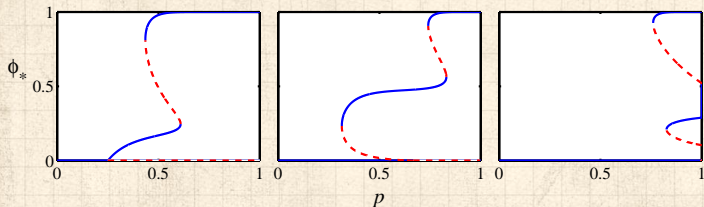
II-III transition generalizes: $p_c = 1/[P_1(T + \tau)]$
where $\tau = 1/r - 1 =$ expected recovery time






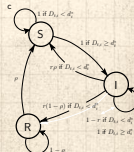
I-II transition less pleasant analytically.



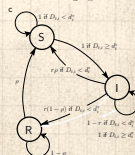
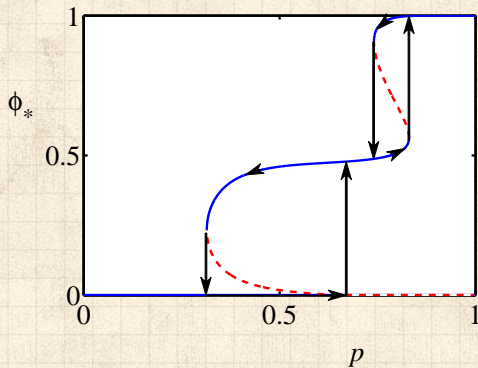
More complicated models



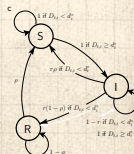
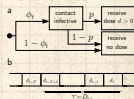
-  Due to heterogeneity in individual thresholds.
-  Three classes based on behavior for small seeds.
-  Same model classification holds: I, II, and III.





Hysteresis in vanishing critical mass models



Nutshell (one half)




 Memory is a natural ingredient.


 Three universal classes of contagion processes:


I. Epidemic Threshold

II. Vanishing Critical Mass

III. Critical Mass

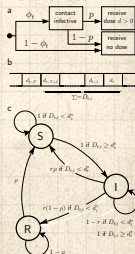
 Dramatic changes in behavior possible.

 To change kind of model: 'adjust' memory, recovery, fraction of vulnerable individuals (T , r , ρ , P_1 , and/or P_2).

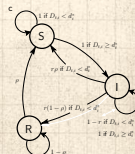
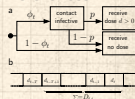
 To change behavior given model: 'adjust' probability of exposure (p) and/or initial number infected (ϕ_0).

Nutshell (other half)

- Single seed infects others if $pP_1(T + \tau) \geq 1$.
- Key quantity: $p_c = 1/[P_1(T + \tau)]$
- If $p_c < 1 \Rightarrow$ contagion can spread from single seed.
- Depends only on:
 - System Memory ($T + \tau$).
 - Fraction of highly vulnerable individuals (P_1).
- Details unimportant: Many threshold and dose distributions give same P_k .
- Another example of a model where vulnerable/gullible population may be more important than a small group of super-spreaders or influentials.

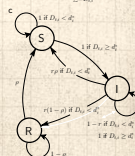


Appendix: Details for Class I-II transition:



$$\begin{aligned}
 \phi^* &= \sum_{k=1}^T \binom{T}{k} P_k (p\phi^*)^k (1 - p\phi^*)^{T-k}, \\
 &= \sum_{k=1}^T \binom{T}{k} P_k (p\phi^*)^k \sum_{j=0}^{T-k} \binom{T-k}{j} (-p\phi^*)^j, \\
 &= \sum_{k=1}^T \sum_{j=0}^{T-k} \binom{T}{k} \binom{T-k}{j} P_k (-1)^j (p\phi^*)^{k+j}, \\
 &= \sum_{m=1}^T \sum_{k=1}^m \binom{T}{k} \binom{T-k}{m-k} P_k (-1)^{m-k} (p\phi^*)^m, \\
 &= \sum_{m=1}^T C_m (p\phi^*)^m
 \end{aligned}$$

Appendix: Details for Class I-II transition:




$$C_m = (-1)^m \binom{T}{m} \sum_{k=1}^m (-1)^k \binom{m}{k} P_k,$$

since


$$\begin{aligned} \binom{T}{k} \binom{T-k}{m-k} &= \frac{T!}{k!(T-k)!} \frac{(T-k)!}{(m-k)!(T-m)!} \\ &= \frac{T!}{m!} \frac{m!}{k!(m-k)!} \\ &= \binom{T}{m} \binom{m}{k}. \end{aligned}$$

Appendix: Details for Class I-II transition:


 Linearization gives

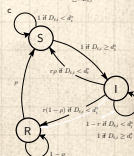
$$\phi^* \simeq C_1 p \phi^* + C_2 p_c^2 \phi^{*2}.$$

where $C_1 = TP_1 (= 1/p_c)$ and
 $C_2 = \binom{T}{2}(-2P_1 + P_2)$.

 Using $p_c = 1/(TP_1)$:

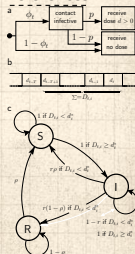
$$\phi^* \simeq \frac{C_1}{C_2 p_c^2} (p - p_c) = \frac{T^2 P_1^3}{(T-1)(P_1 - P_2/2)} (p - p_c).$$

 Sign of derivative governed by $P_1 - P_2/2$.



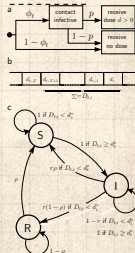
References I

- [1] F. Bass.
A new product growth model for consumer durables.
[Manage. Sci.](#), 15:215–227, 1969. pdf ↗
- [2] C. Castillo-Chavez and B. Song.
Models for the Transmission Dynamics of Fanatic Behaviors, volume 28, chapter 7, pages 155–172.
[SIAM](#), 2003.
- [3] D. J. Daley and D. G. Kendall.
Epidemics and rumours.
[Nature](#), 204:1118, 1964. pdf ↗
- [4] D. J. Daley and D. G. Kendall.
Stochastic rumours.
[J. Inst. Math. Appl.](#), 1:42–55, 1965.



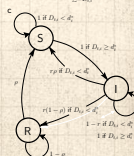
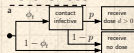
References II

- [5] P. S. Dodds and D. J. Watts.
Universal behavior in a generalized model of contagion.
[Phys. Rev. Lett., 92:218701, 2004. pdf](#)
- [6] P. S. Dodds and D. J. Watts.
A generalized model of social and biological contagion.
[J. Theor. Biol., 232:587–604, 2005. pdf](#)
- [7] W. Goffman and V. A. Newill.
Generalization of epidemic theory: An application to the transmission of ideas.
[Nature, 204:225–228, 1964. pdf](#)



References III

- [8] W. O. Kermack and A. G. McKendrick.
A contribution to the mathematical theory of epidemics.
[Proc. R. Soc. Lond. A, 115:700–721, 1927. pdf](#) ↗
- [9] W. O. Kermack and A. G. McKendrick.
A contribution to the mathematical theory of epidemics. III. Further studies of the problem of endemicity.
[Proc. R. Soc. Lond. A, 141\(843\):94–122, 1927. pdf](#) ↗
- [10] W. O. Kermack and A. G. McKendrick.
Contributions to the mathematical theory of epidemics. II. The problem of endemicity.
[Proc. R. Soc. Lond. A, 138\(834\):55–83, 1927. pdf](#) ↗



References IV

- [11] J. D. Murray.
Mathematical Biology.
Springer, New York, Third edition, 2002.
- [12] S. H. Strogatz.
Nonlinear Dynamics and Chaos.
Addison Wesley, Reading, Massachusetts, 1994.

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Appendix

References

