Generalized 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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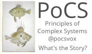

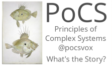

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Outline

"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

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.

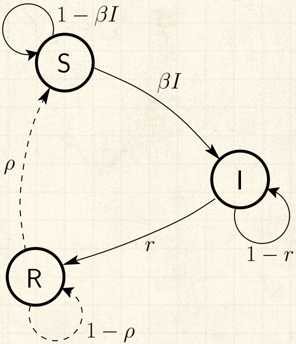
Mathematical Epidemiology (recap)

The standard SIR model [11]

- = 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

Independent Interaction Models

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

$$\frac{\mathrm{d}}{\mathrm{d}t} S = -\beta I S + \rho R$$

$$\frac{\mathrm{d}}{\mathrm{d}t} I = \beta I S - r I$$

$$\frac{\mathrm{d}}{\mathrm{d}t} R = r I - \rho R$$

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:

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

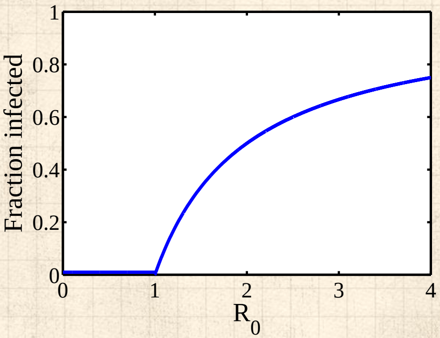
$$= \beta\left(1 + (1-r) + (1-r)^2 + (1-r)^3 + \dots\right)$$

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

- Similar story for continuous model.

Independent Interaction models

Example of epidemic threshold:



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

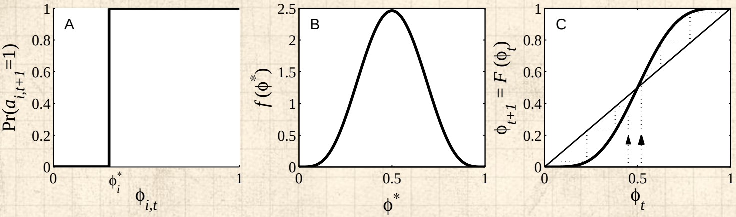
Simple disease spreading models

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)

- 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.
- Interdependent 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

$\boxed{\mathsf{S} \Rightarrow \mathsf{I}}$

- 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

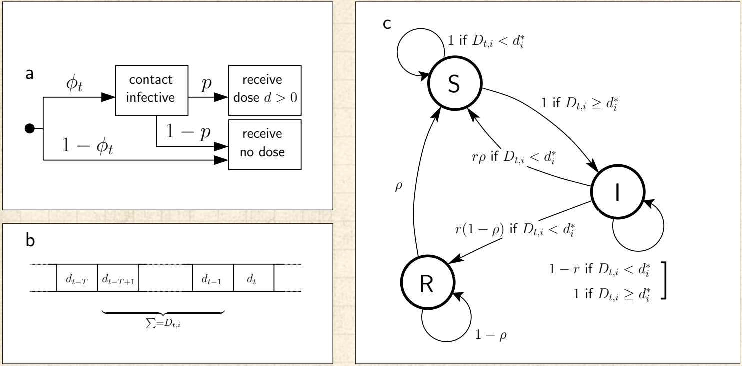
$\boxed{\mathrm{I} \Rightarrow \mathrm{R}}$

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

$\boxed{\mathrm{R} \Rightarrow \mathrm{S}}$

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:

$$\phi_{t+1} = \underbrace{p\phi_t}_{\text{a}} + \underbrace{\phi_t(1 - p\phi_t)}_{\text{b}} \underbrace{(1 - r)}_{\text{c}}.$$

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(infected) = 1 - Pr(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 $\phi^* \to 0$, we see

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

- 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 $r \le 1$, $d^* = 1$, and $T \ge 1$

- Start with $r = 1$, $d^* = 1$, and $T \ge 1$ 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:

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

- 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 $r \le 1$, $d^* = 1$, and $T \ge 1$

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

$$= r \sum_{m=0}^{\infty} P(H_{T+m}) = r \sum_{m=0}^{\infty} p\phi^*(1-p\phi^*)^{T+m}(1-r)^m$$

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

- 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 $r \le 1$, $d^* = 1$, and $T \ge 1$

- Fixed point equation (again):

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

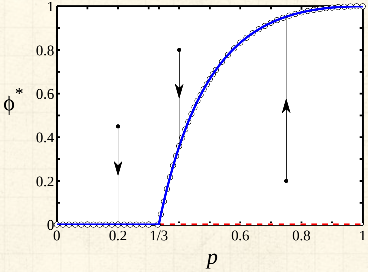
- Find critical exposure probability by examining above as $\phi^* \to 0$.
-
$$\Rightarrow \quad p_c = \frac{1}{T + 1/r - 1} = \frac{1}{T+\tau}.$$

  where τ = 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:

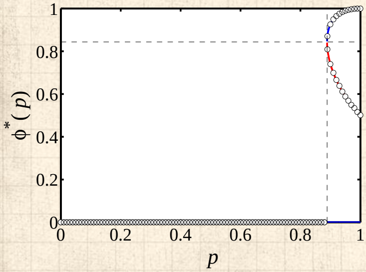
$$\phi^* = \sum_{i=d^*}^{T} \binom{T}{i} (p\phi^*)^i (1 - p\phi^*)^{T-i}.$$

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

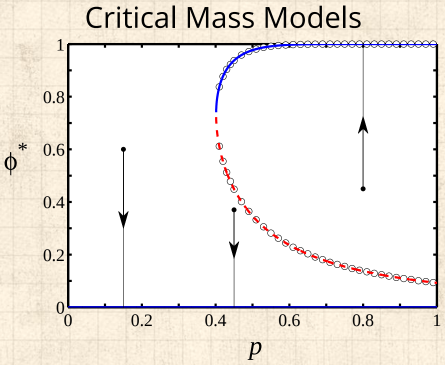


- Fixed point equation: $\phi^* = 3p^2{\phi^*}^2(1 - p\phi^*) + p^3{\phi^*}^3$
- See new structure: a saddle node bifurcation [12] appears as p increases.
- $(p_b, \phi^*) = (8/9, 27/32)$.

- Behavior akin to output of Granovetter's threshold model.

Homogeneous, multi-hit models:

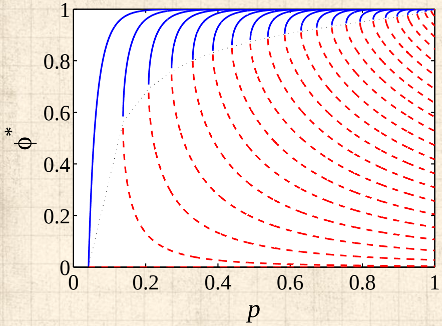
- Another example:



- $r = 1$, $d^{*} = 3$, $T = 12$ Saddle-node bifurcation.

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

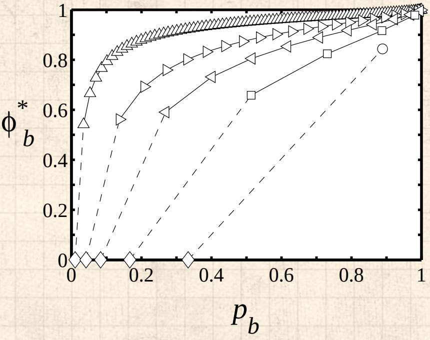
- $T = 24$, d^* = 1, 2, ...23.



- $d^* = 1 \to d^* > 1$: jump between continuous phase transition and pure critical mass model.
- Unstable curve for $d^* = 2$ does not hit $\phi^* = 0$.
- See either simple phase transition or saddle-node bifurcation, nothing in between.

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

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



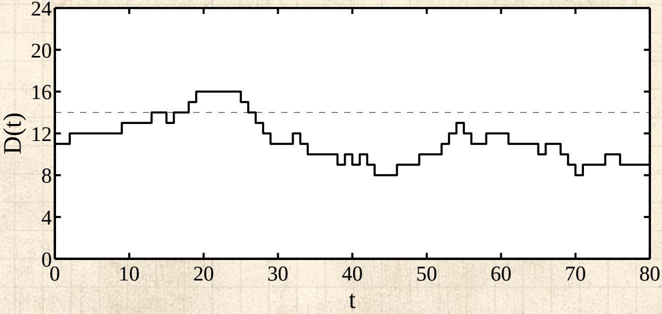
- $T = 96$ ( ).
- $T = 24$ ($\triangleright$),
- $T = 12$ ($\triangleleft$),
- $T = 6$ ($\square$),
- $T = 3$ ($\bigcirc$),

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:
  $\{d_{n-2}, d_{n-1}, d_n, d_{n+1}\} = \{1, 1, 0, 0\}$
  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:

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

- $N_a + N_b$ 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:

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

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

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

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

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

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

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

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

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

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

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

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

Fixed points for $r < 1$, $d^* = 2$, and $T = 3$

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

where $\Gamma(p, \phi^*; r) =$

$$(1-r)(p\phi)^2(1-p\phi)^2 + \sum_{m=1}^{\infty} (1-r)^m (p\phi)^2 (1-p\phi)^2 \times$$

$$[\chi_{m-1} + \chi_{m-2} + 2p\phi(1-p\phi)\chi_{m-3} + p\phi(1-p\phi)^2 \chi_{m-4}]$$

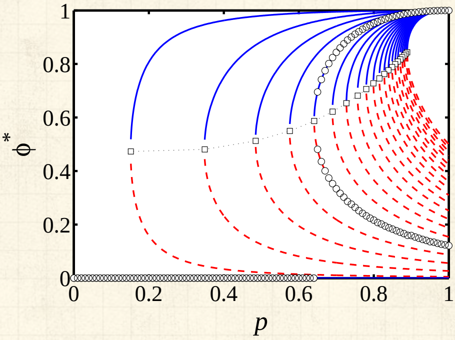
and

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

Note: $(1-r)(p\phi)^2(1-p\phi)^2$ accounts for $\{1, 0, 1, 0\}$ sequence.

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

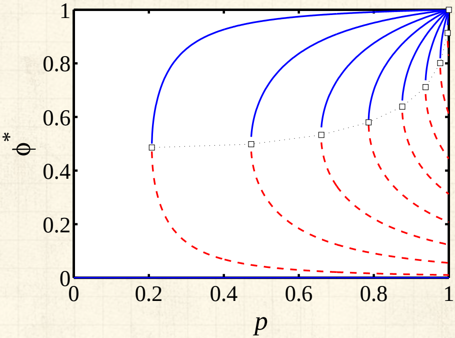
$T = 3$, $d^* = 2$



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

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

$T = 2, d^* = 2$



- $r = 0.01, 0.05, 0.10, \ldots, 0.3820 \pm 0.0001.$
- 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:

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

- 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:

$$\boxed{pP_1 T \geq 1} \quad \text{or} \quad \boxed{\Rightarrow p_c = 1/(TP_1)}$$

- Very good:
  1. $P_1 T$ is the expected number of vulnerables the initial infected individual meets before recovering.
  2. $pP_1 T$ 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

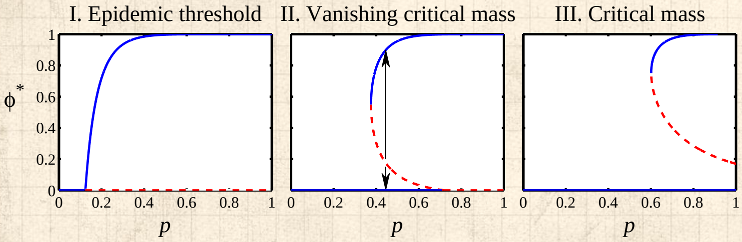
- Next: Determine slope of fixed point curve at critical point p_c.
- Expand fixed point equation around $(p, \phi^*) = (p_c, 0)$.
- Find slope depends on $(P_1 - P_2/2)$ [6] (see Appendix).
- Behavior near fixed point depends on whether this slope is
  1. positive: $P_1 > P_2/2$ (continuous phase transition)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

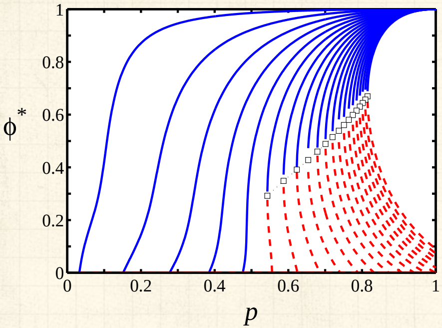
Three universal classes



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

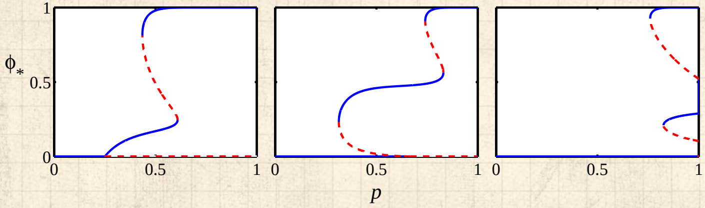
Heterogeneous case

Now allow $r < 1$:



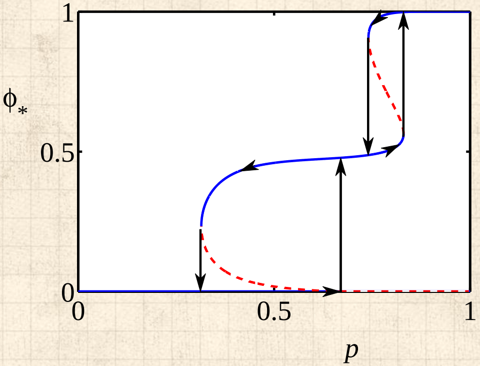
- II-III transition generalizes: $p_c = 1/[P_1(T + \tau)]$ where $\tau = 1/r - 1 =$ expected recovery time
- I-II transition less pleasant analytically.

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:
  1. System Memory ($T+\tau$).
  2. 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

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

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.