Generalized Contagion

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Complex Networks | @networksvox
CSYS/MATH 303, Spring, 2018

Prof. Peter Dodds | @peterdodds

Dept. of Mathematics & Statistics | Vermont Complex Systems Center
Vermont Advanced Computing Core | University of Vermont



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Outline

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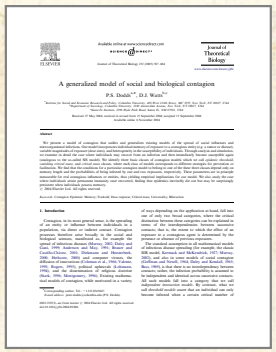
PHYSICAL REVIEW LETTERS

Universal Behavior in a Generalized Model of Contagion

Peter Sheridan Dodds* and Duncan J. Watts†

"Universal Behavior in a Generalized Model of Contagion"

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



Available online at www.sciencedirect.com

Journal of Theoretical Biology

A generalized model of social and biological contagion

P.S. Dodds*, D.J. Watts

"A generalized model of social and biological contagion"

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

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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.

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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

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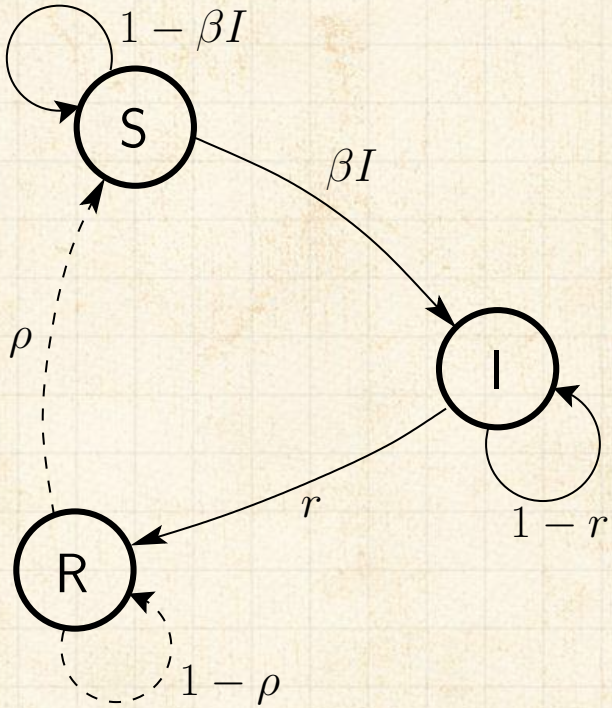
Independent Interaction Models

Discrete time automata example:



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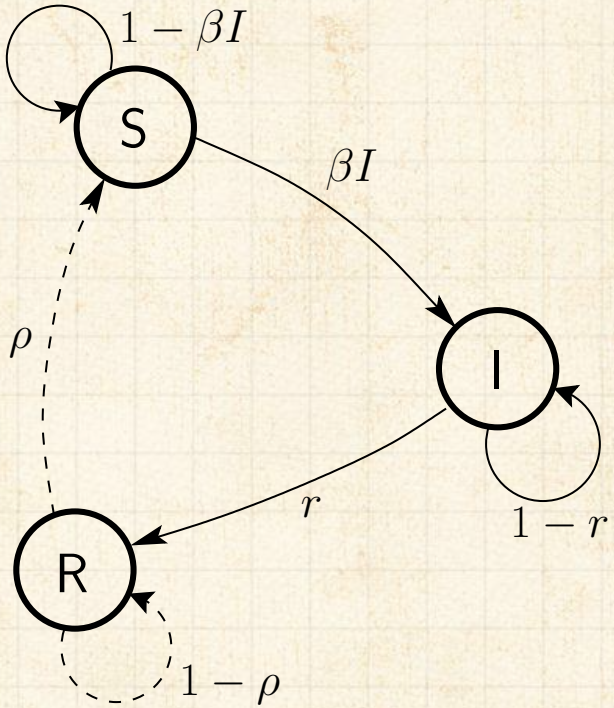

Transition Probabilities:

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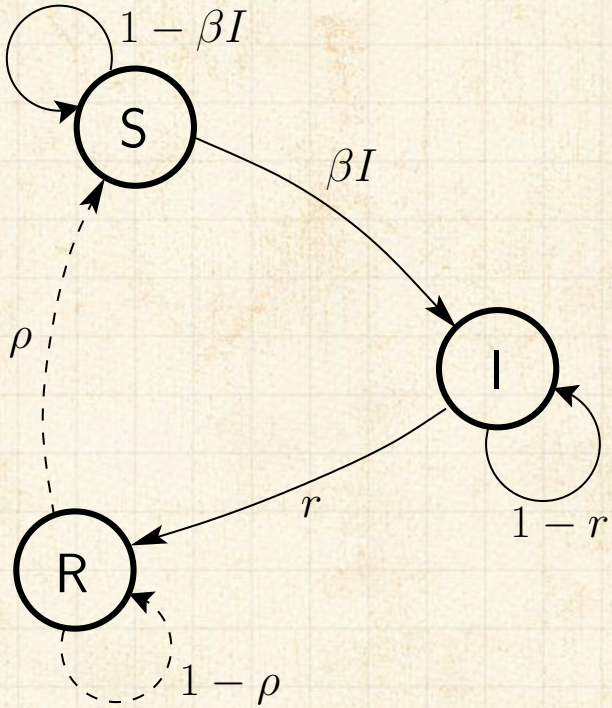

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β for being infected given contact with infected

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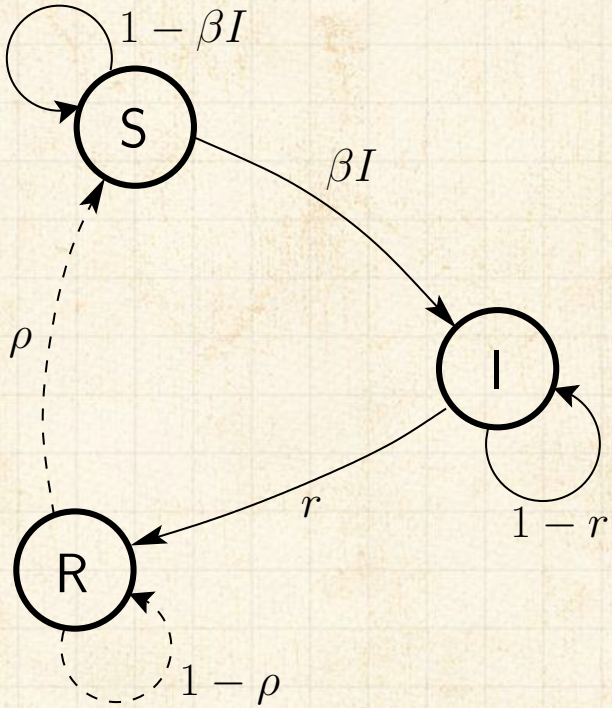

Transition Probabilities:

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r for recovery

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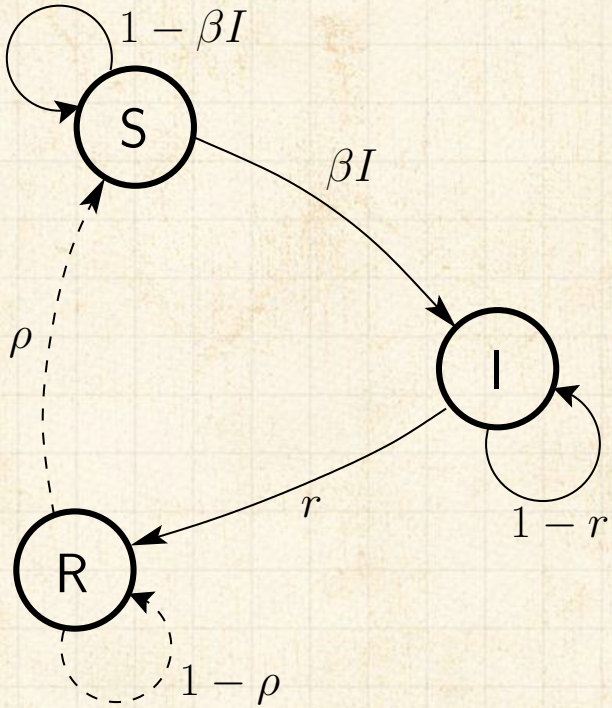

Transition Probabilities:

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, 91]
- Coupled differential equations with a mass-action principle

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Independent Interaction models

Differential equations for continuous model

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

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

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

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.

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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 $(1 - r)^k$

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Reproduction Number R_0

Discrete version:

- Expected number infected by original Infective:

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

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

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

- Similar story for continuous model.

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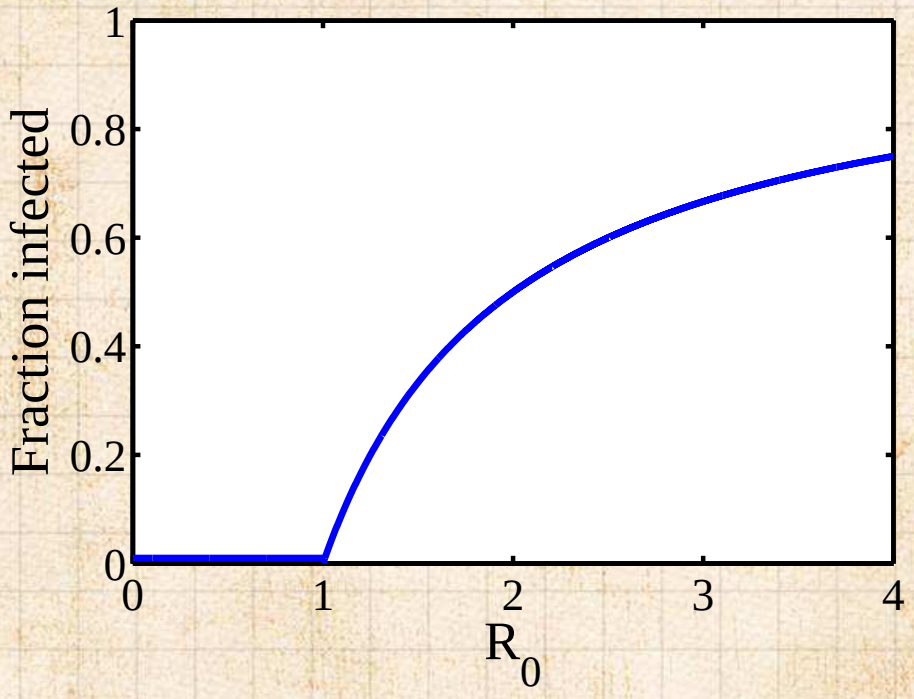
Example of epidemic threshold:



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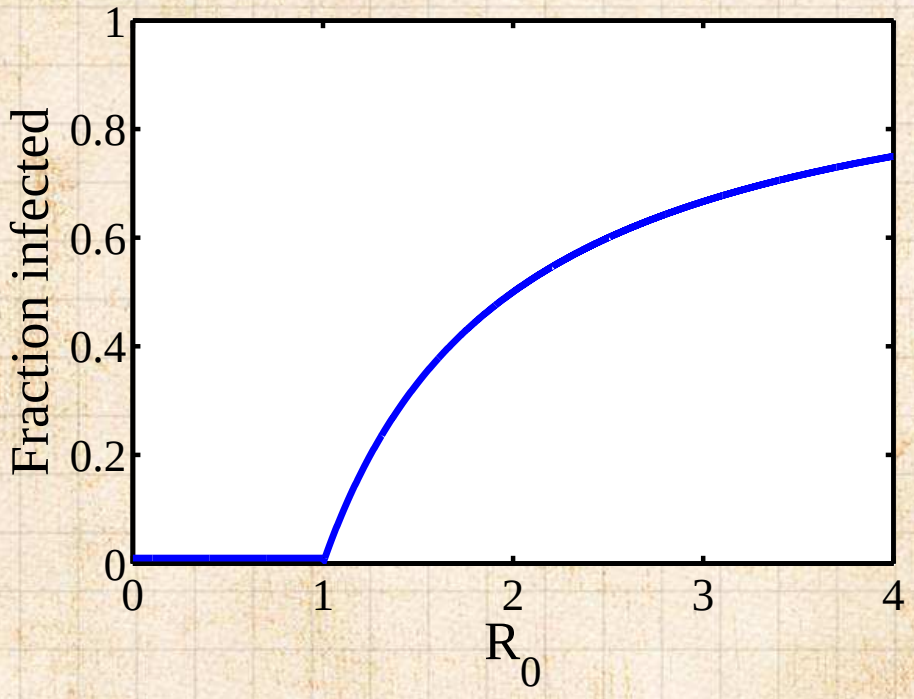

- Continuous phase transition.

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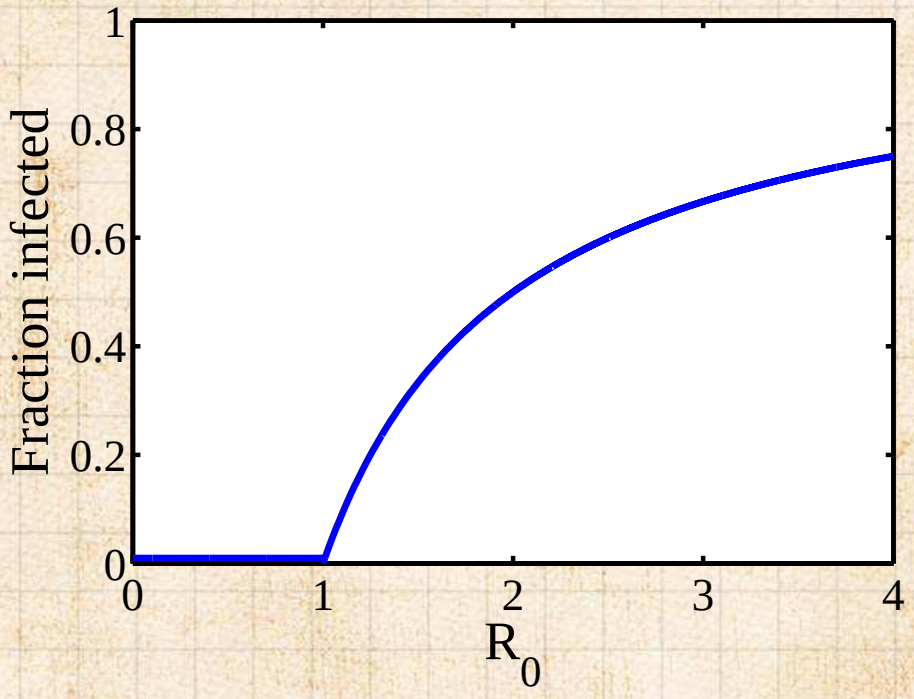

- Continuous phase transition.
- Fine idea from a simple model.

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]

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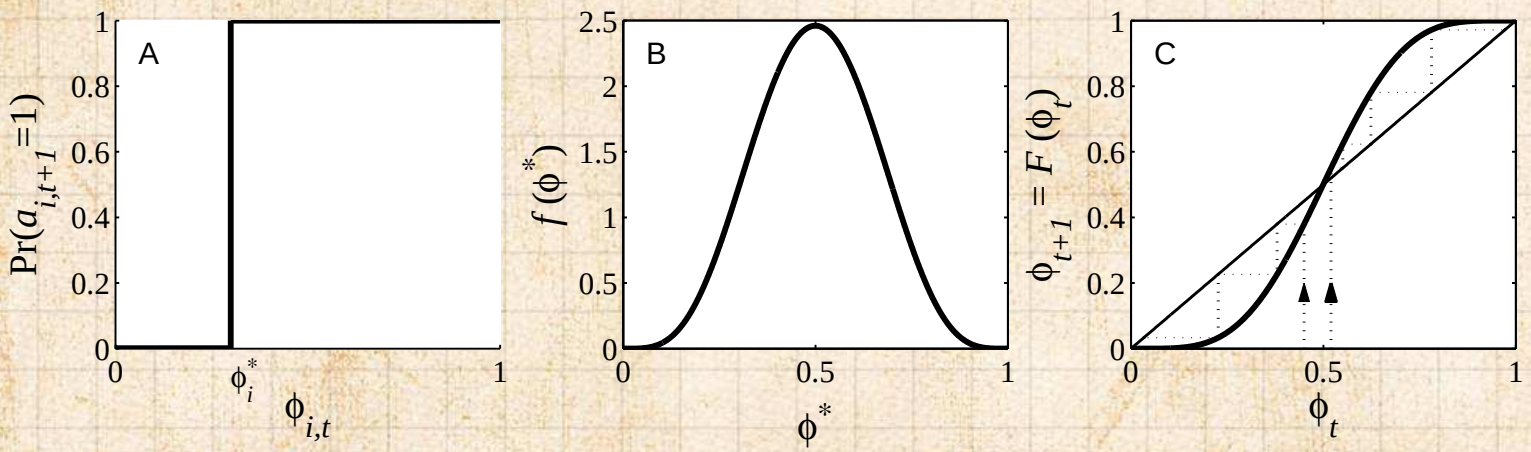
Granovetter's model (recap of recap)

- 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.

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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 $d = 0$.

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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:

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

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Generalized model—ingredients

S ⇒ I

- Individuals 'remember' last T contacts:

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

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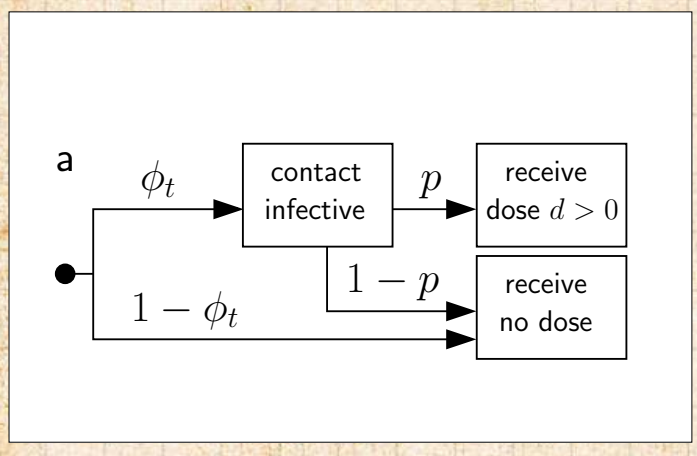
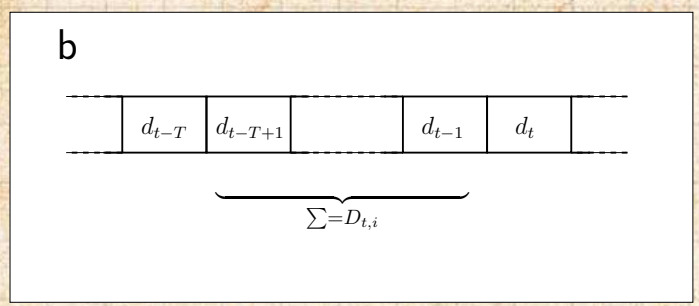
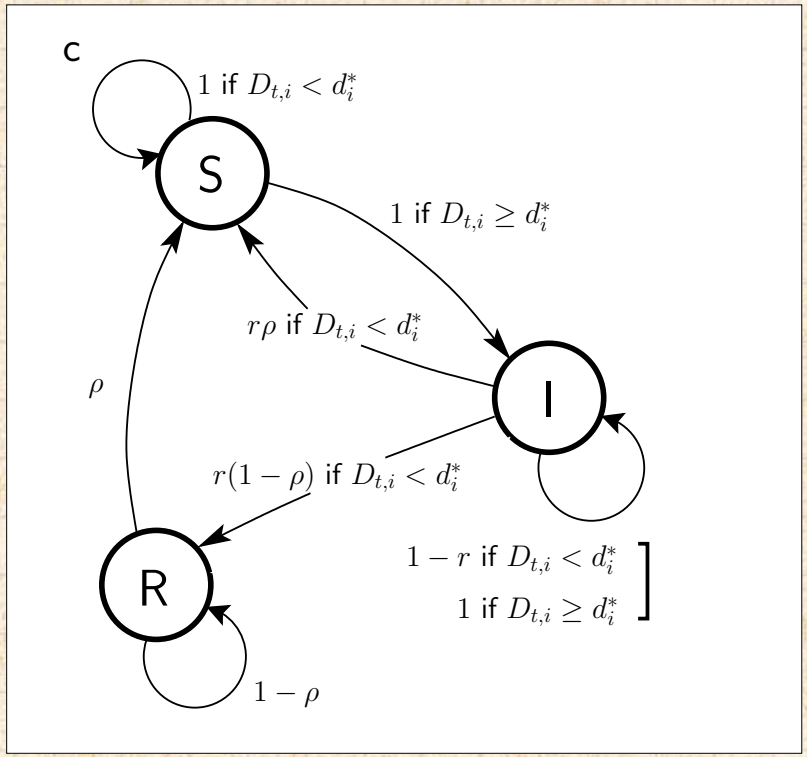
A visual explanation







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Generalized mean-field model

Study SIS-type contagion first:

- Recovered individuals are immediately susceptible again:

$$\rho = 1.$$

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- Denote fixed points by ϕ^*.

Homogeneous version:

- All individuals have threshold a^*
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Homogeneous, one hit models:

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

- $r < 1$ means recovery is probabilistic.
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- Evolution of infection level:

$$\phi_{t+1} = \underbrace{\phi_t}_{a} + \underbrace{\rho(t)}_{b}\underbrace{(1-\phi_t)}_{c}$$

a: Fraction infected between time t and $t+1$.
b: Probability of being infected and not being reinfected.
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$$\Rightarrow \phi^* = \frac{1 - r/p}{1 - r} \quad \text{and} \quad \phi^* = 0.$$

- Critical point at $p = p_c = r$.
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Fixed points for $r = 1$, $d^* = 1$, and $T > 1$

- $r = 1$ means recovery is immediate.
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- Effect of individual interactions is independent from effect of others.
- Call ϕ^* the steady state level of infection.
- Pr(infected) = 1 - Pr(uninfected):

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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

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

- Again find continuous phase transition ...
- Note: we can solve for p but not ϕ^*:

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

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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:

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- 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:

$$H_1 = \{1, 0, 0, \ldots, 0, 0\}$$

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

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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:

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

- In general, relevant dose histories are:

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

- Overall probabilities for dose histories occurring:

$$P(H_{m+1}) = p_{\mathrm{inf}}(1-r)^{m-1}(1-p_{\mathrm{inf}})^{T}(1-r)^{T}$$

$$P(H_{m+1}) = \underbrace{p_{\mathrm{inf}}}_{a}\underbrace{(1-p_{\mathrm{inf}})^{T}}_{b}\underbrace{(1-r)^{m-1}(1-r)^{T}}_{c}$$

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

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

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

where τ = mean recovery time for simple relaxation process.

- Decreasing r keeps individuals infected for longer and decreases p_c.

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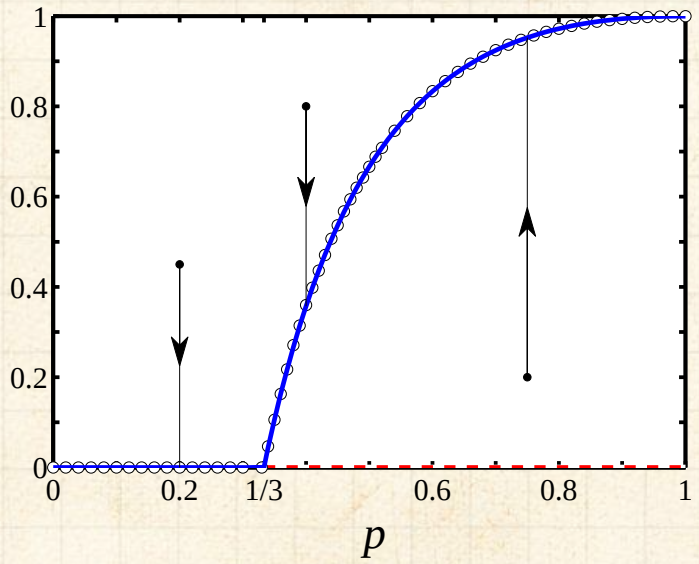
Epidemic threshold:

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]

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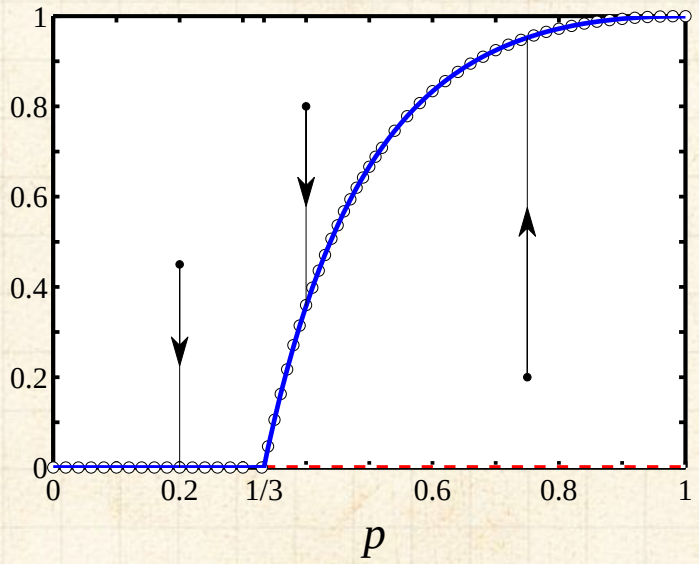

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- All right: $d^* = 1$ models correspond to simple disease spreading models.
- What if we allow $d^* \geq 2$?
- Again first consider SIS with immediate recovery ($\gamma = 1$)
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Fixed points for $r = 1$, $d^* > 1$, 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.
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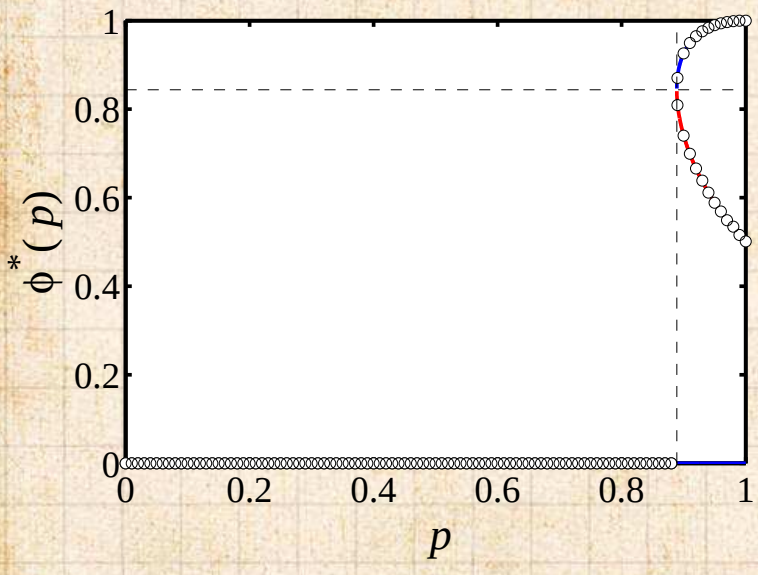

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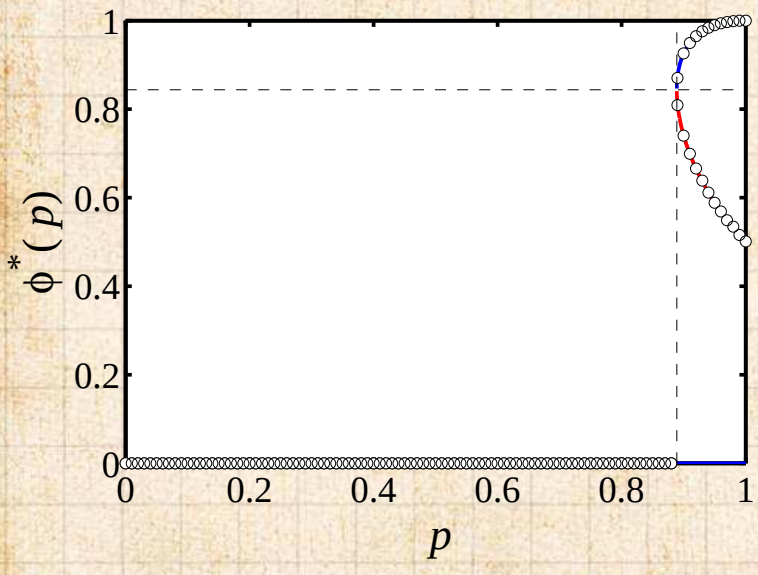

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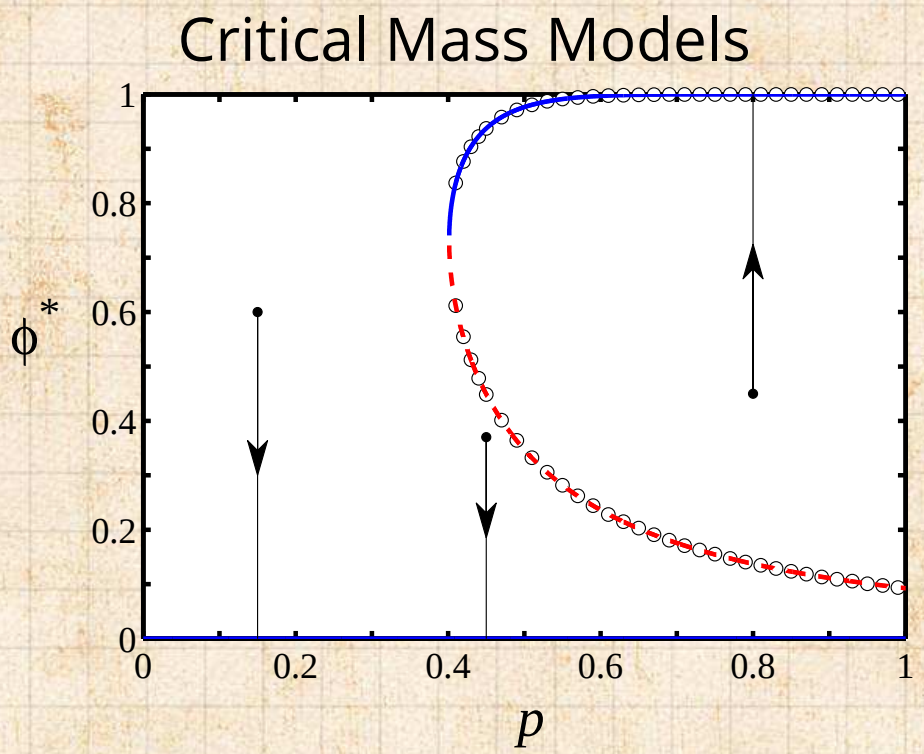
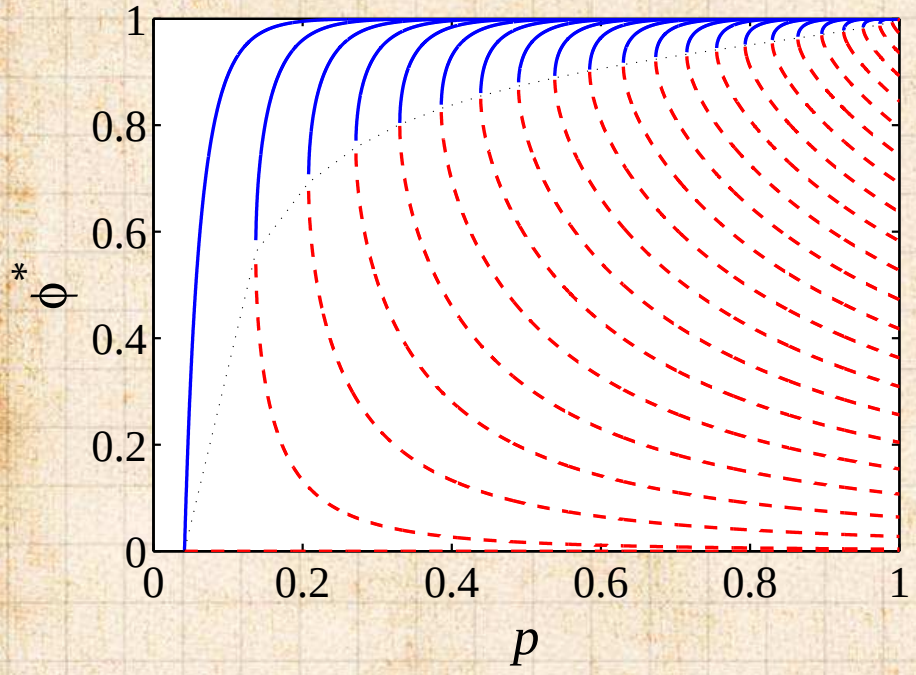
- Another example:



- $r = 1, d^* = 3, T = 12$ 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 $\phi^* = 0$.
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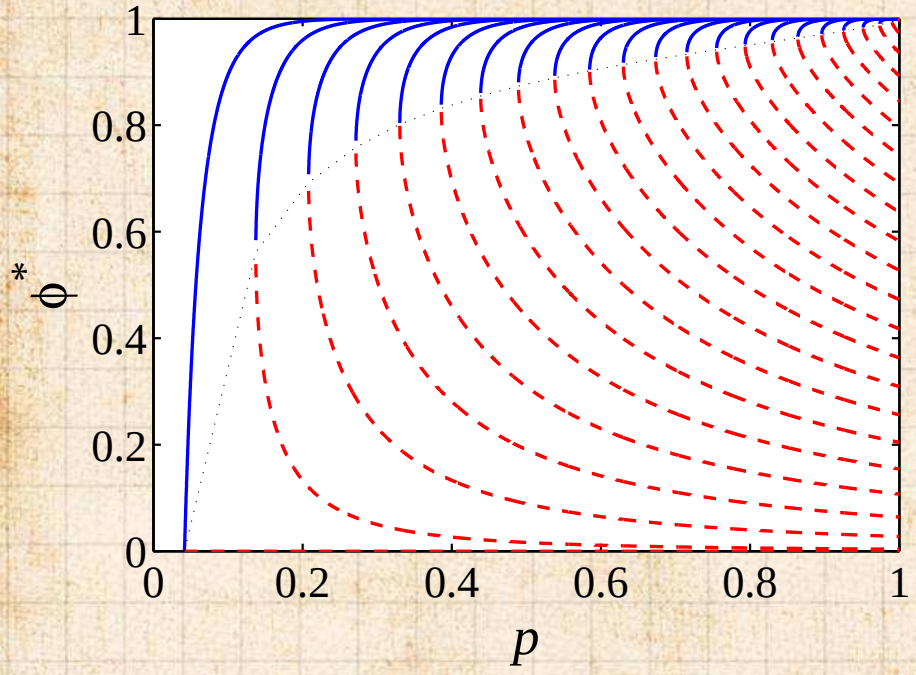

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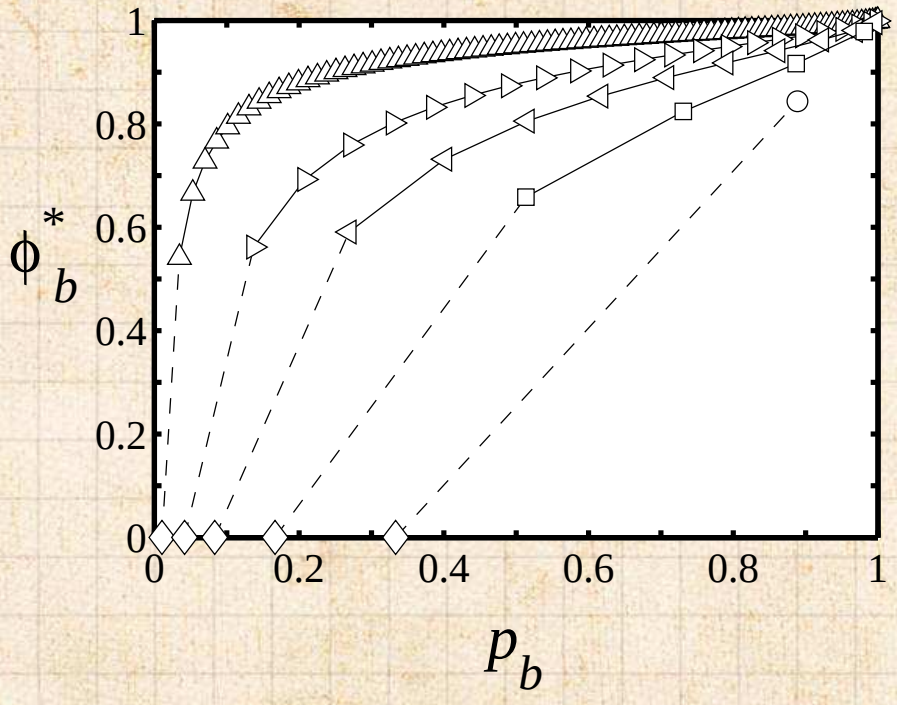
- Bifurcation points for example fixed T, varying d^*:



- $T = 96$ ( ).
- $T = 24$ (▷),
- $T = 12$ (◁),
- $T = 6$ (□),
- $T = 3$ (○),

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:

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

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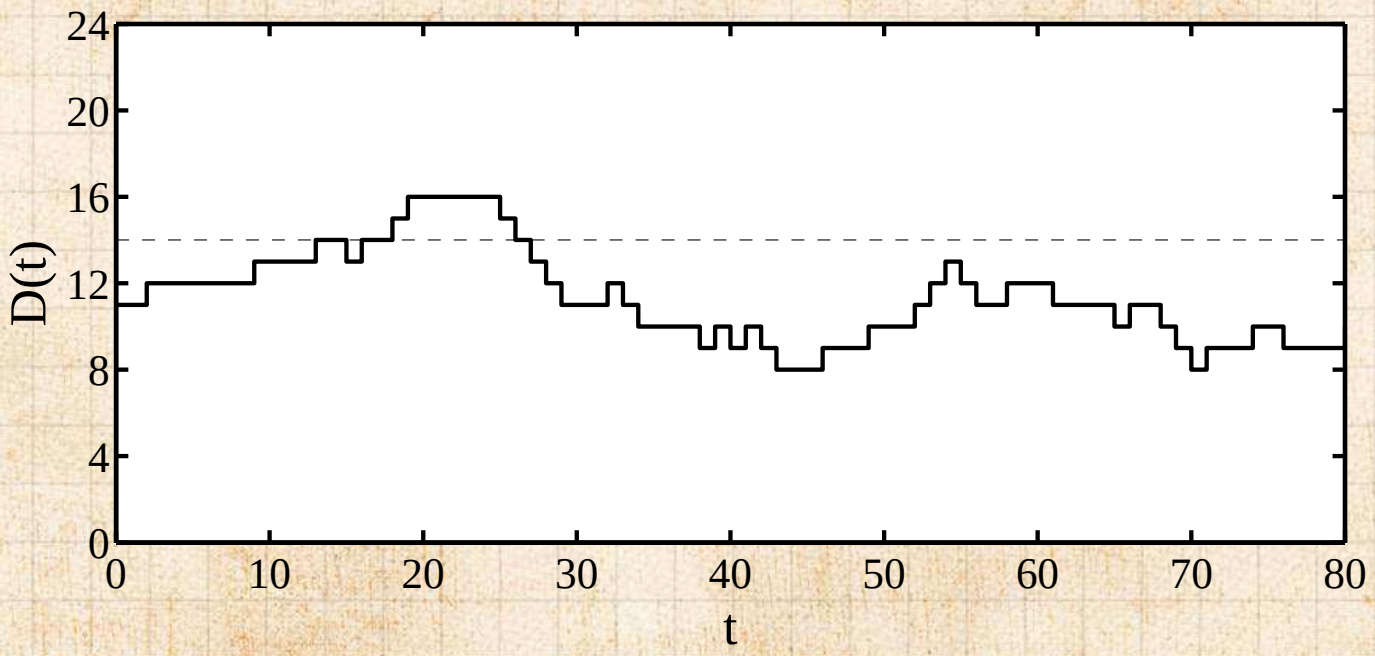

Fixed points for $r < 1$, $d^* > 1$, and $T \geq 1$

- 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 $T \geq 1$

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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:

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

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- 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:

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

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

- Corresponding possible values for N_a:

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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:

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

- $N_a + N_b$ slots for subsequences.
- Choose positions of either a's or b's:

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

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

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

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- Nearly there ...must account for details of sequence endings.
- Three endings $\Rightarrow$ Six possible sequences:

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

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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 $T \geq 1$

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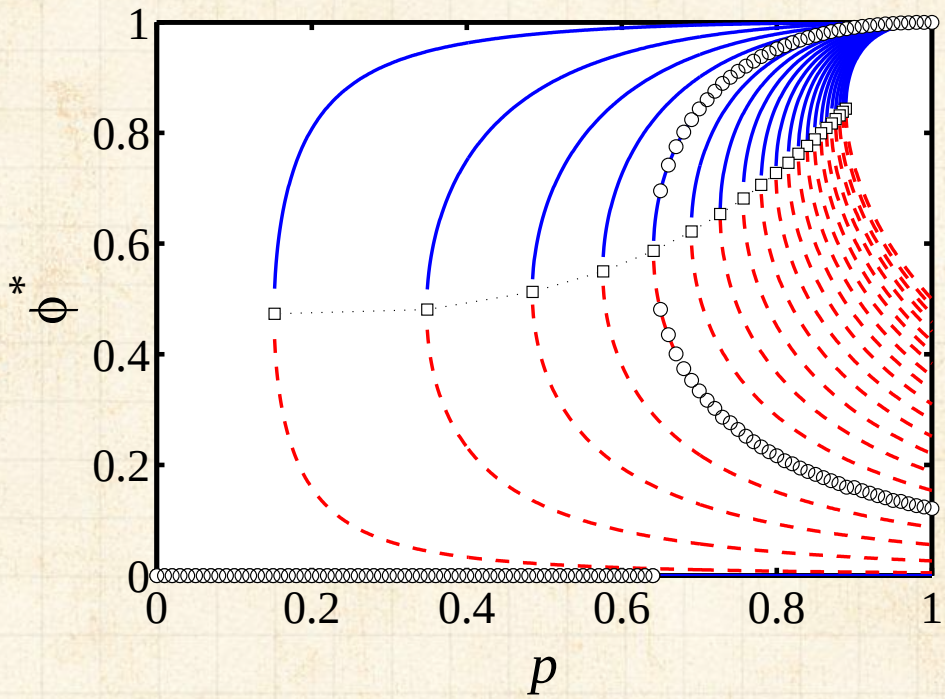
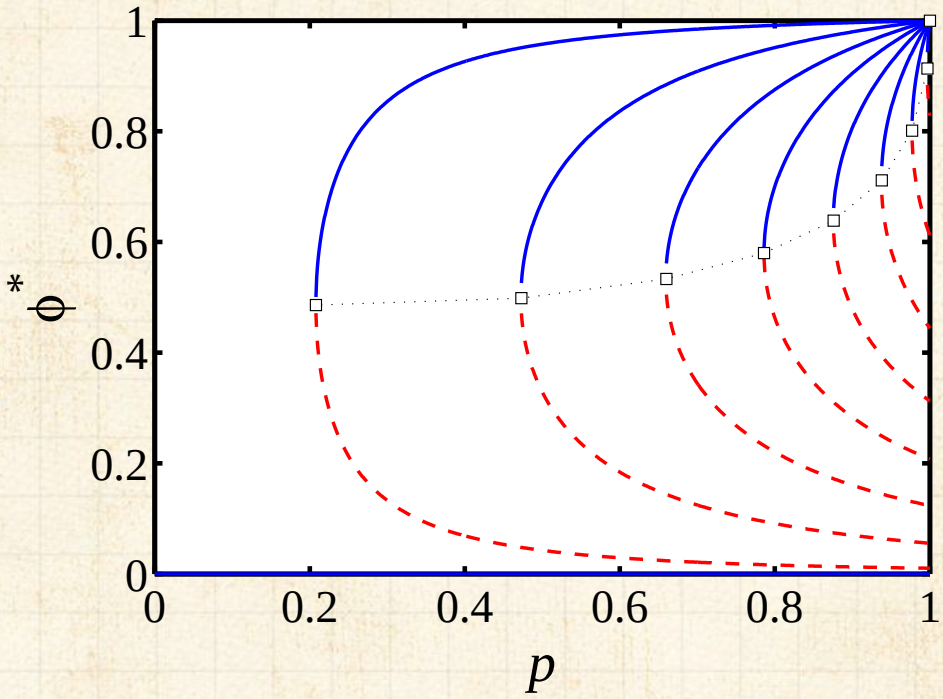


$r = 0.01, 0.05, 0.10, 0.15, 0.20, \ldots, 1.00.$

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 $r \gtrsim 0.382$.

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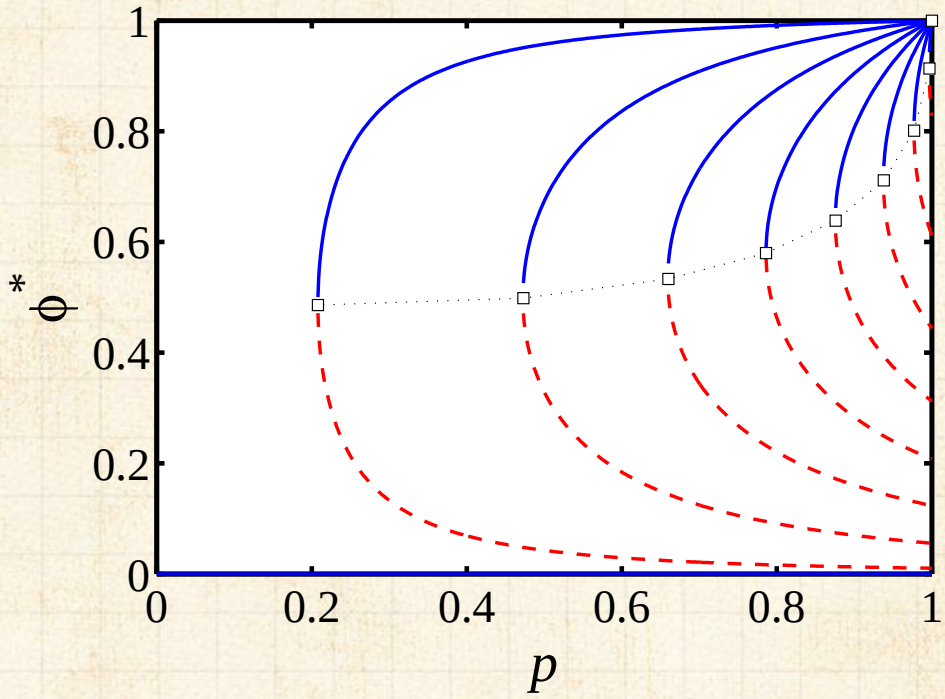

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What we have now:

- 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.
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- Are other behaviors possible?

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Outline

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Generalized model

- Now allow for general dose distributions (f) and threshold distributions (g).
- Key quantities:

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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:

$$pP_1T > 1 \quad \text{or} \quad \Rightarrow p_c = 1/(TP_1)$$

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

Generalized model—heterogeneity, $r = 1$

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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 $(P_1 - P_2/2)$ [5] (see Appendix).
- Behavior near fixed point depends on whether this slope is
  1. positive: $P_1 > P_2/2$ (continuous phase transition)
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Example configuration:

- Dose sizes are lognormally distributed with mean 1 and variance 0.433.
- Memory span: $T = 10$.
- Thresholds are uniformly set at
  1. $\theta = 0$
  2. $\theta = 1.5$
  3. $\theta = 3$
- Spread of dose sizes matters, details are not important.

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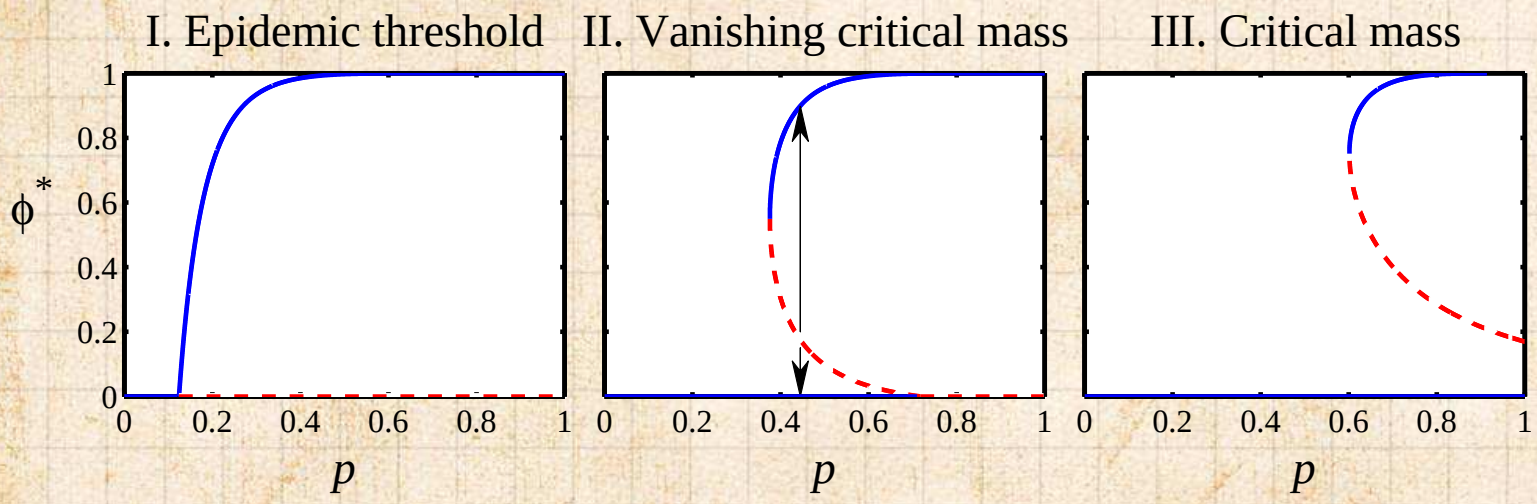
Three universal classes



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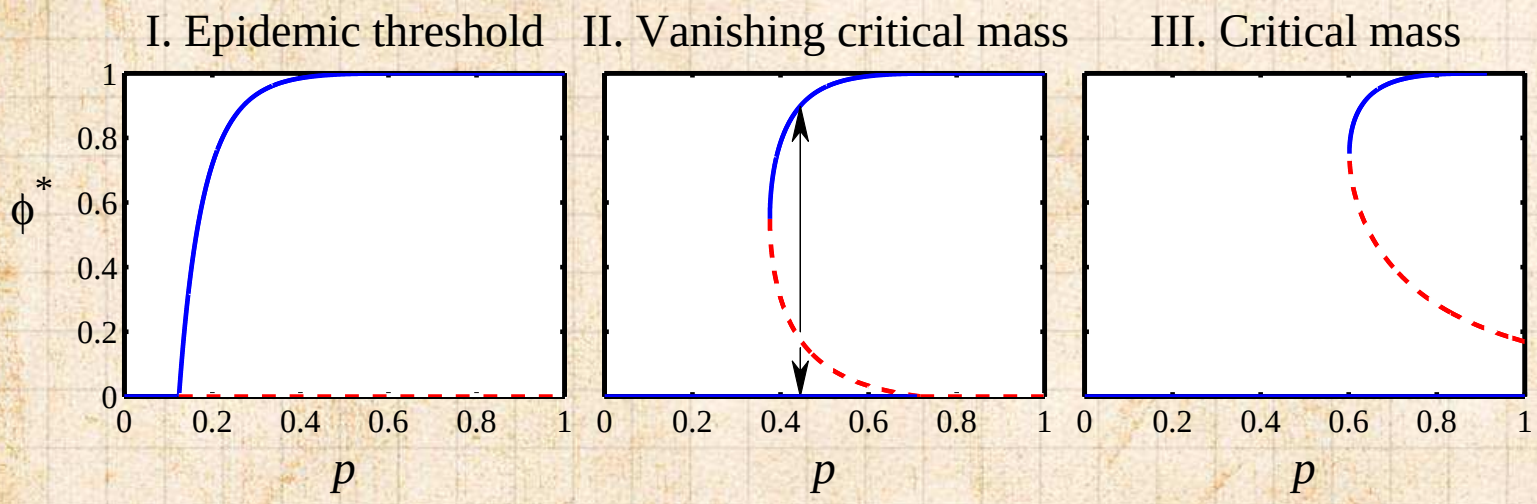

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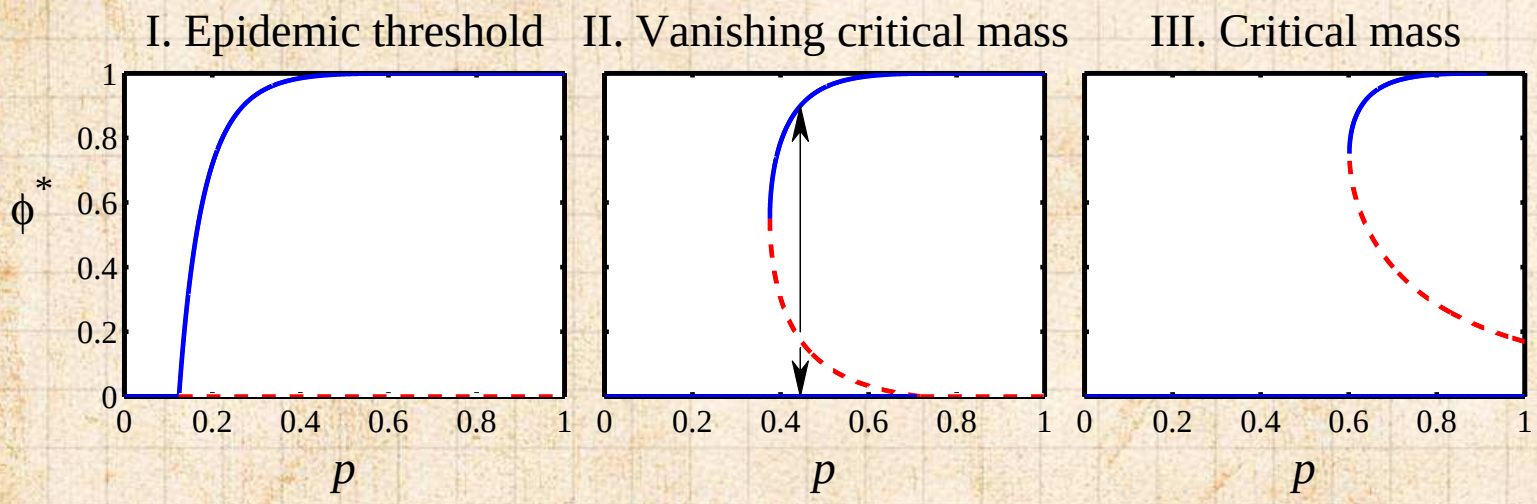

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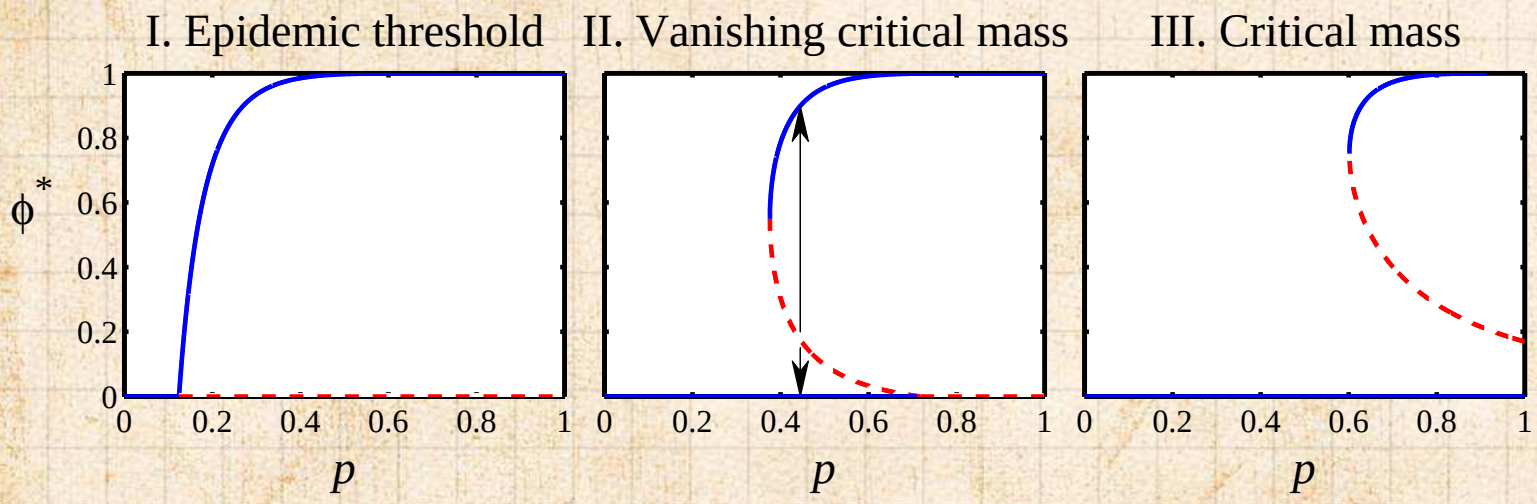

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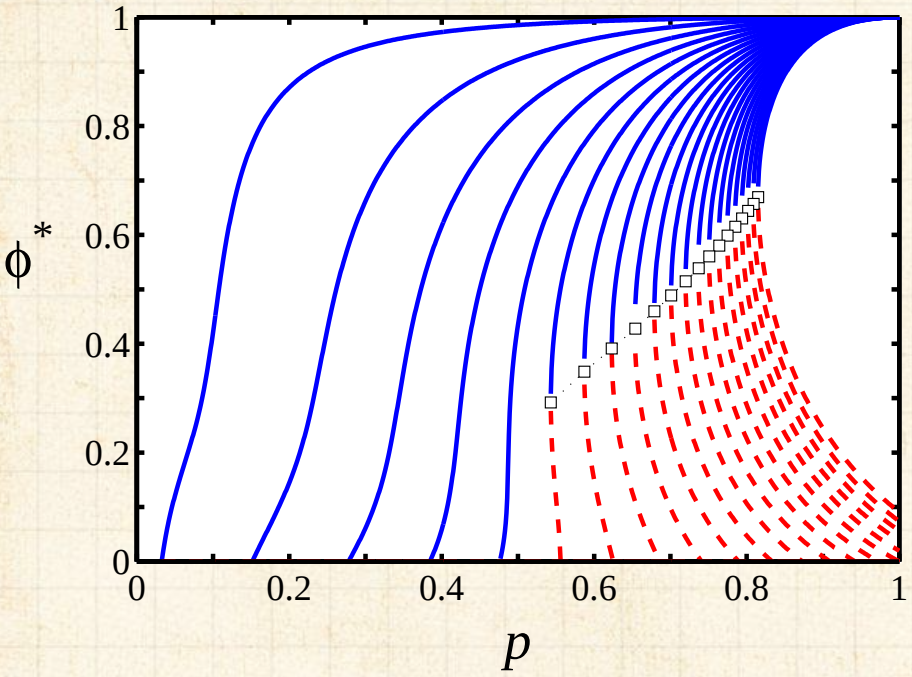
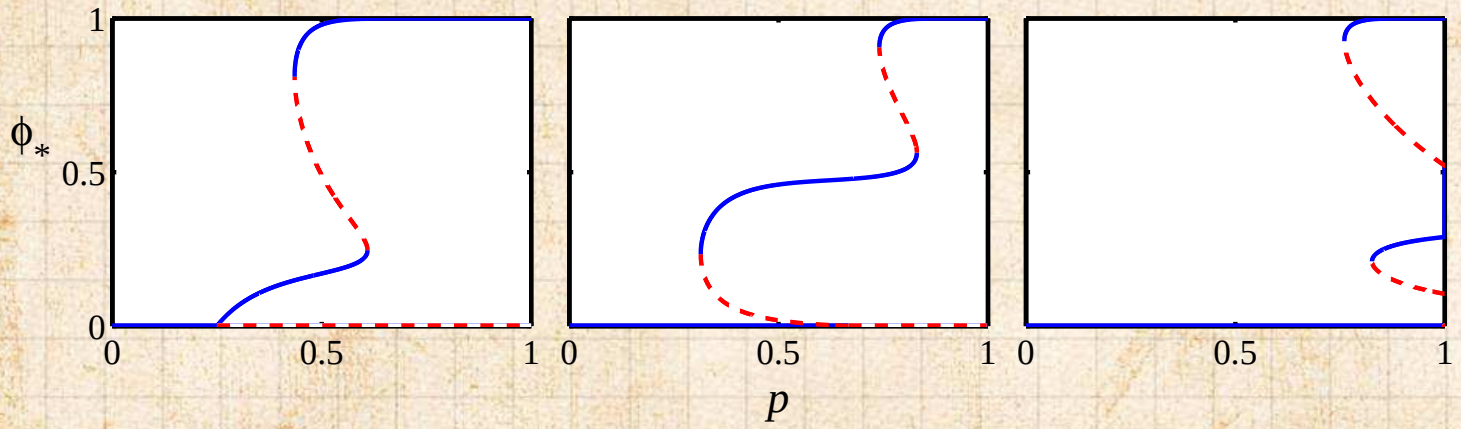
Now allow $r < 1$:



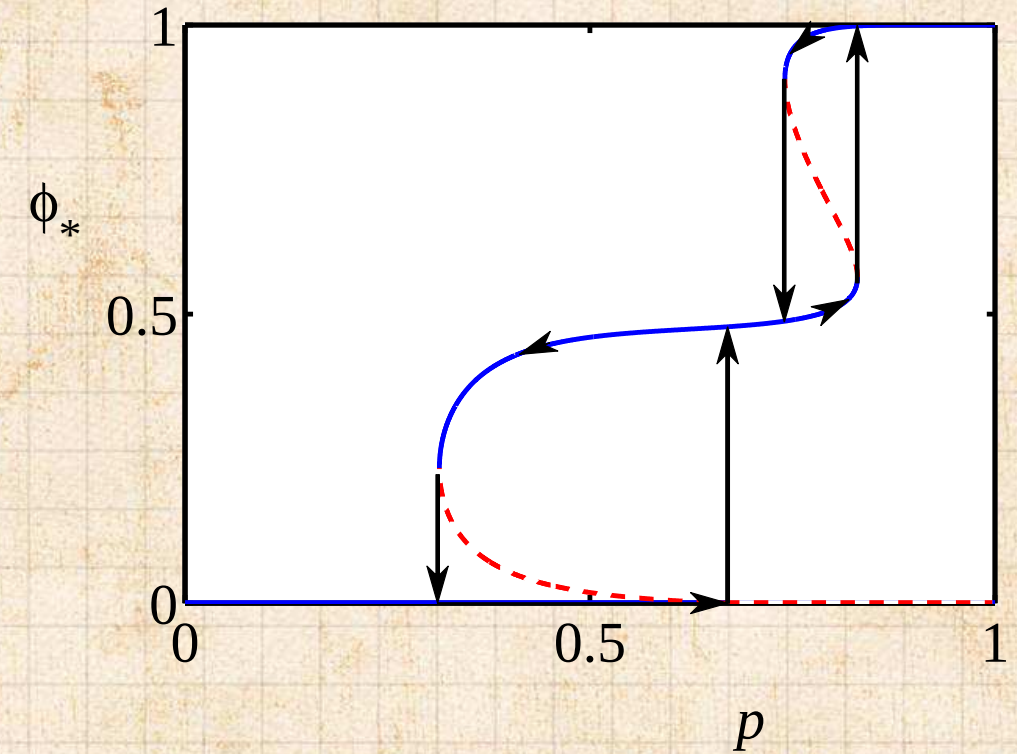
- II-III transition generalizes: $p_c = 1/[P_1(T+\tau)]$ where $\tau = 1/r - 1 =$ expected recovery time
- I-II transition less pleasant analytically.

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:
  1. I. Epidemic Threshold
  2. II. Vanishing Critical Mass
  3. 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).
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Nutshell (other half)

- Single seed infects others if $pP_1(T+\tau) \geq 1$.
- Key quantity: $p_c = 1/[P_1(T+\tau)]$
- If $p_c < 1 \Longrightarrow$ 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.

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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}.
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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 $p_c = 1/(TP_1)$:

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

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