

# Universal Behavior in a Generalized Model of Contagion

Peter S. Dodds

Duncan J. Watts

Columbia University

# Outline

- Motivation of model
  - “biological contagion”
  - “social contagion”
  - “generalized contagion”
- Model description
  - General case
  - Special SIS case
- Results
  - Universal classes
  - Transition conditions
- Discussion

# Motivation

- Concept of “contagion” arises quite generally in biological and social sciences
  - Spread of infectious disease
  - Diffusion of innovations
  - Rumor spreading
  - Growth of cultural fads
  - Emergence of collective beliefs
  - Transmission of financial distress
- Roughly speaking, would like to understand in what sense these different kinds of contagion are the same and how they are different

# What do we mean by “contagion”?

- Individuals are in one of (at least) two discrete states:
  - “susceptible” (also inactive, uninformed, non-adopter, etc.)
  - “infected” (also active, informed, adopter, etc.)
- When “susceptibles” come into contact with “infectives”, they too can become infected (with some probability  $p$ ), and not otherwise.
- By this definition, “contagion” is not the same as, say, diffusion (which is continuous), but is still reasonably general

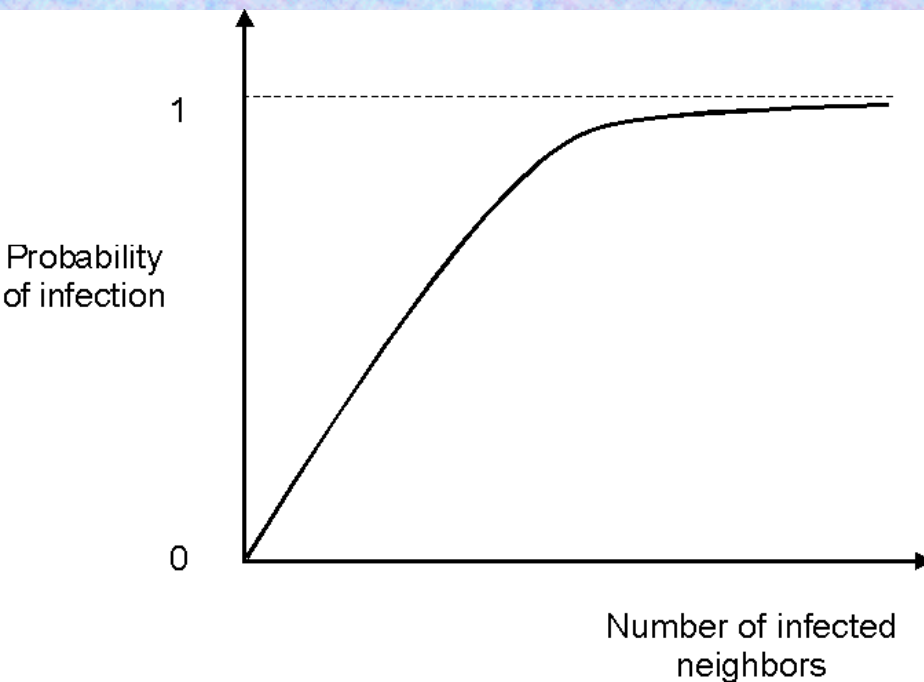
# What kinds of contagion are there?

- Different kinds of contagion could be differentiated according to, for example:
  - More than two states, or classes of individuals, with different interactions between them (also can generalize dynamics of population: birth, death, aging, etc.)
  - Different interaction structure
  - Different choices of “infection probability”  $p$
- Here we discuss only the last distinction

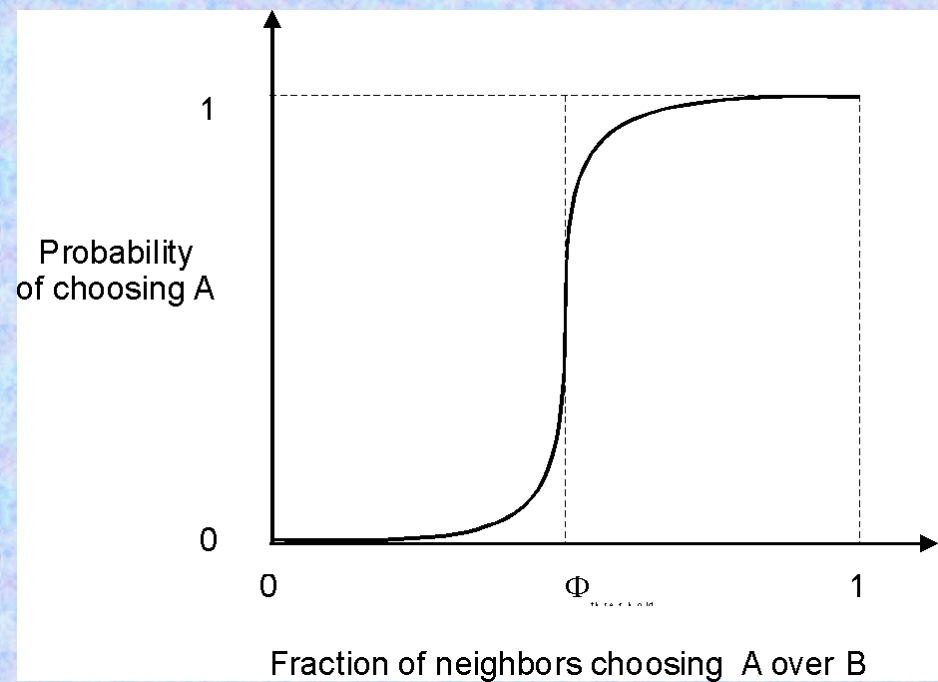
# Classes of Contagion Models

- “Poisson” models
  - Each susceptible-infective interaction (an “exposure”) results in infection with independent (constant) probability  $p$
  - Infection thus regarded as a Poisson process
  - SIR-type models and “Bass” model of diffusion of innovations both examples of Poisson models
- “Threshold” models
  - Infection likely only after a threshold number of doses has been exceeded
  - Threshold gives rise to temporal interdependencies between exposures
  - Many such models in literature on binary decisions, information cascades, fads, etc. (Schelling, Granovetter, Glance and Huberman, Durlauf, Morris, etc.)

# Graphically:



Poisson Model



Threshold Model

# The Problem

- Poisson models assert (implicitly) that infection is memory-less
- Threshold models assert (also implicitly) that infection displays very strong memory dependence
- Neither class offers a means to vary temporal interdependency (i.e. memory) or test its effect on *collective dynamics*
- One result is that our conceptual view of contagion is vague with respect to the underlying model (“everything that spreads is the same”)

# Model Description

- Consider a fixed population of size  $N$
- Each individual is in one of three states:
  - Susceptible (S)
  - Infected (I)
  - Removed (R)
- $S(t)+I(t)+R(t)=1$  for all  $t$ .
- At each time step, each individual ( $i$ ) comes into contact with another individual ( $j$ ) chosen uniformly at random (i.e. uniform mixing)

# Model Description

- If  $i$  is susceptible and  $j$  is infected, then with probability  $p$ ,  $i$  is exposed, receiving a positive dose  $d_i$  drawn randomly from a dose distribution  $f(d)$ . Otherwise  $d_i=0$
- Each individual  $i$  retains a memory of its previous  $T$  doses, recording its cumulative dose

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

- If  $D_i(t) \geq d_i^*$  ( $i$ 's dose threshold, assigned randomly at  $t=0$  from a threshold distribution  $g(d^*)$ ) then  $i$  becomes infected

# Infection probability

- Probability that a susceptible individual who contacts  $K \leq T$  infected individuals in  $T$  time steps will become infected is therefore

$$P_{\text{inf}}(K) = \sum_{k=1}^K \binom{K}{k} p^k (1-p)^{K-k} P_k \quad (1)$$

- Where

$$P_k = \int_0^{\infty} g(u) du P \left[ d^* \leq \sum_{i=1}^k d_i \right] \quad (2)$$

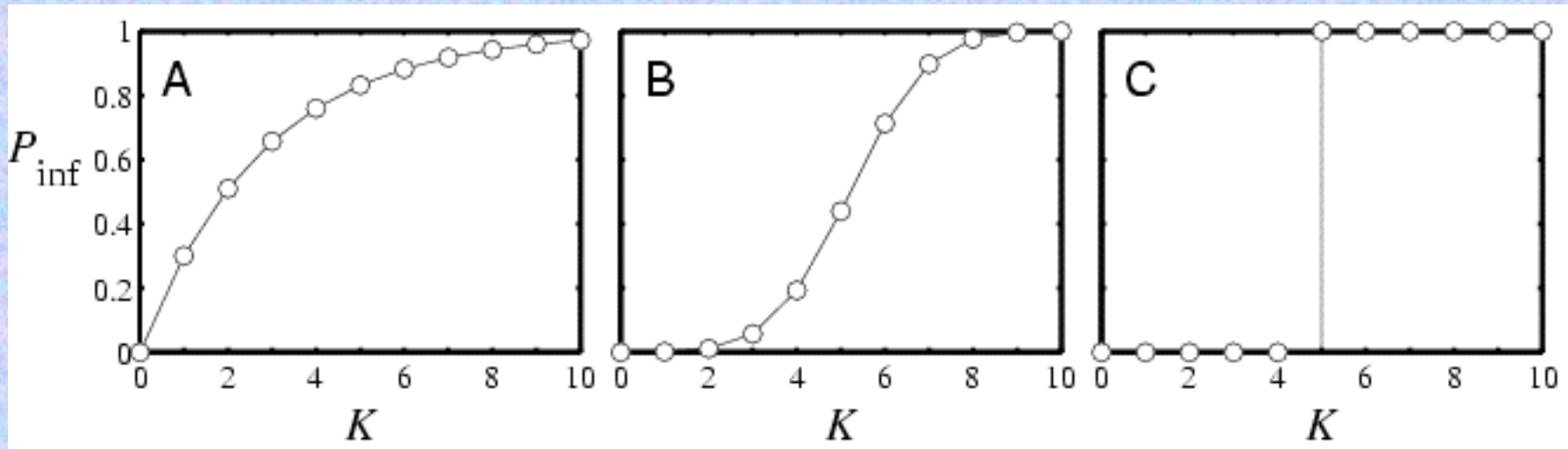
- $P_{\text{inf}}$  can be thought of as a dose response relationship
- Different choices of  $T$ ,  $f(d)$  and  $g(d^*)$  lead to different dose response relationships; hence different contagion models

# Dose Response Examples

Poisson

Stochastic  
Threshold

Deterministic  
Threshold



$p < 1$   
All  $d_i = 1$   
All  $d^* = 1$

$p = 1$   
 $d_i$  log-normally  
distributed (mean 1)  
 $d^* = 4$

$p = 1$   
All  $d_i = 1$   
All  $d^* = 4$

# Recovery and Re-Susceptibility

- Infected individuals recover with probability  $r$  once  $D_i(t)$  falls below  $d_i^*$  (otherwise they remain infected)
- Recovered individuals become re-susceptible again with probability  $\rho$
- Consider special case of  $\rho = 1, r = 1$ 
  - Analogous to SIS dynamics ( $\rho = 1$ ) with instantaneous recovery ( $r = 1$ )
  - Have also considered  $r < 1$  (equivalent to changing time-scale)

# Steady-State Dynamics

- SIS formulation allows us to write down the equation for the steady-state fraction of infectives in the population

$$\phi_* = \sum_{k=1}^T (p\phi_*)^k (1 - p\phi_*)^{T-k} P_k \quad (3)$$

- where

$$P_k = \int_0^{\infty} g(u) du P\left[d^* \leq \sum_{i=1}^k d_i\right]$$

- is the probability of a random individual being infected by k successive (randomly drawn) exposures

# Collective Dynamics

- Have studied the solutions of Equation 3 and also simulated the corresponding dynamics for
  - Homogeneous populations ( $d_i=1$ ;  $d^*\geq 1$ )
  - Heterogeneous populations
    - $d_i$  log-normally distributed with variable mean and variance
    - $d^*$  occupies either a single discrete value or multiple discrete values

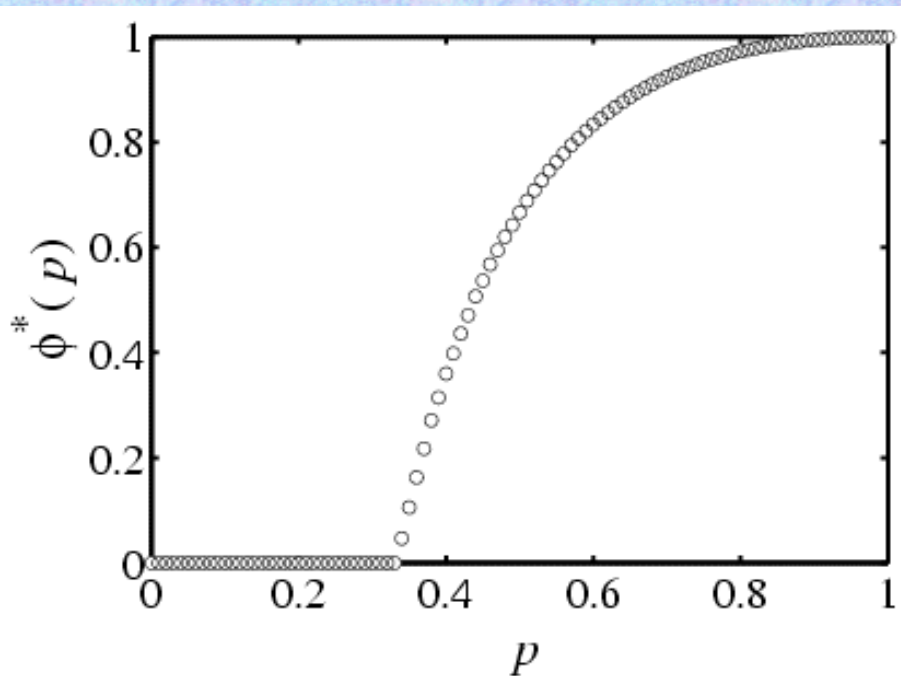
# Results:

## 1. Homogeneous Systems

- Only two classes of collective dynamics possible:
  - Epidemic Threshold Dynamics ( $d^*=1$ )
    - See a transcritical bifurcation at  $p=p_c=1/T$ ,  $\phi_*=0$
    - For  $p<p_c$ , all initial outbreaks die out
    - For  $p>p_c$ , all initial outbreaks grow to occupy finite fraction of population
    - $p_c$  is equivalent to epidemic threshold in SIR-type models
  - Critical Mass Dynamics ( $d^*>1$ )
    - See a saddle-node bifurcation at  $p=p_b$ ,  $\phi_*=\phi_b$
    - For  $p<p_b$ , all initial outbreaks die out
    - For  $p>p_b$ , outbreaks larger than  $\phi_b$  grow; otherwise die out
    - Hence critical mass required for global contagion to take place

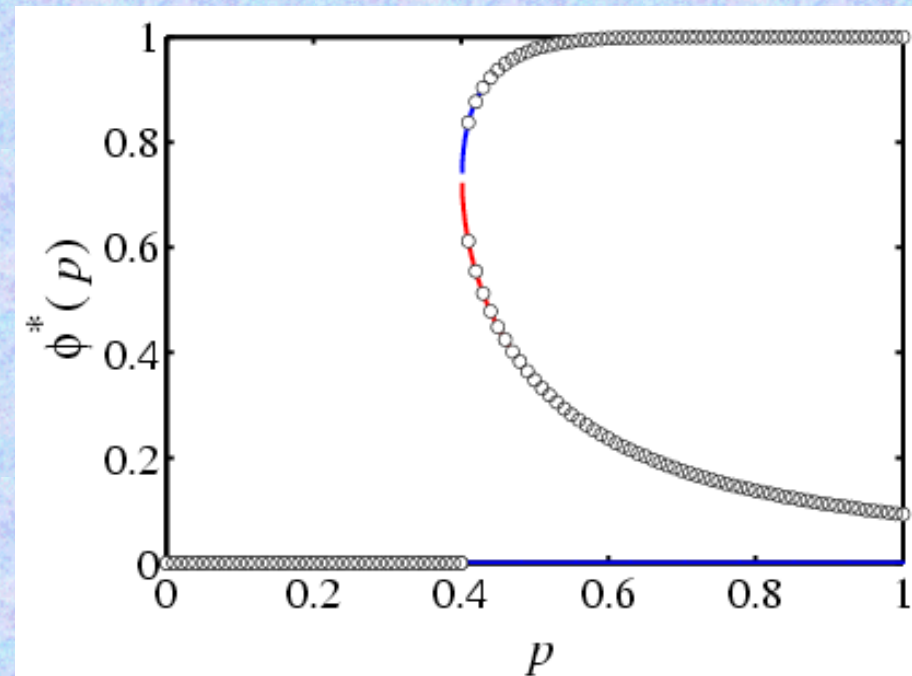
# Graphically

## I. Epidemic Threshold



$$d^*=1$$

## II. Critical Mass



$$d^*>1$$

## 2. Heterogeneous Systems

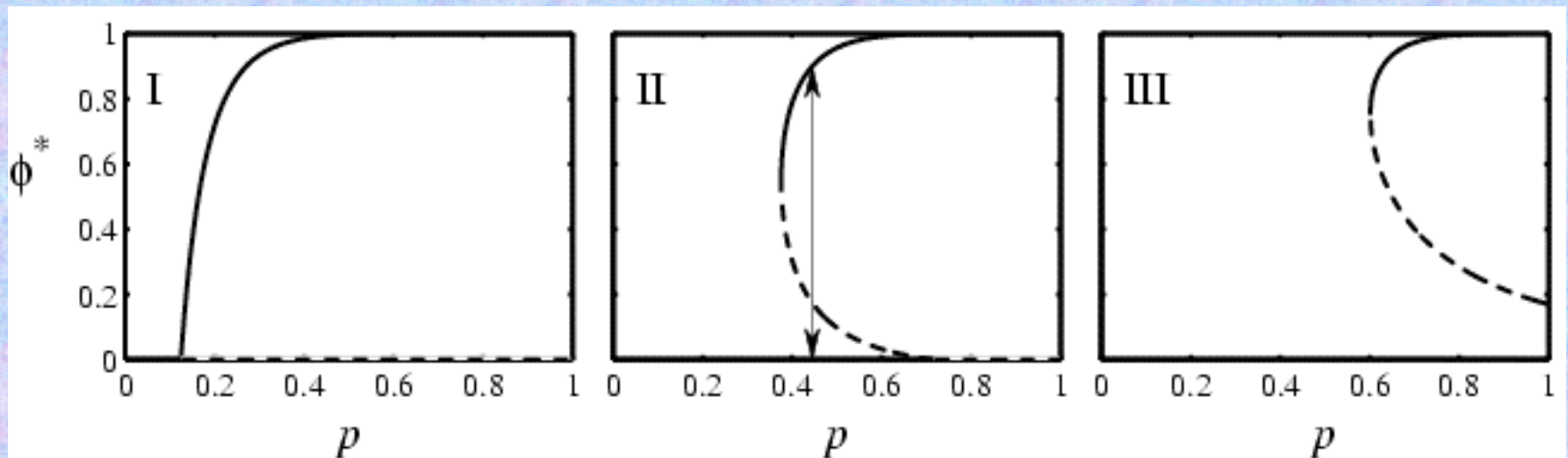
- More complicated; don't have completely general conditions
- However, under reasonably broad conditions, find only three classes of dynamics:
  - Epidemic Threshold (but now,  $p_c = 1/(TP_1)$ )
  - Pure Critical Mass (same as CM in homogeneous case)
  - Vanishing Critical Mass
    - Both saddle-node and transcritical bifurcations present
    - Unstable branch of the SN bifurcation collides with zero axis
    - Hence critical mass “vanishes” at  $p_c$

# Graphically

Epidemic  
Threshold

Vanishing  
Critical Mass

Pure  
Critical Mass

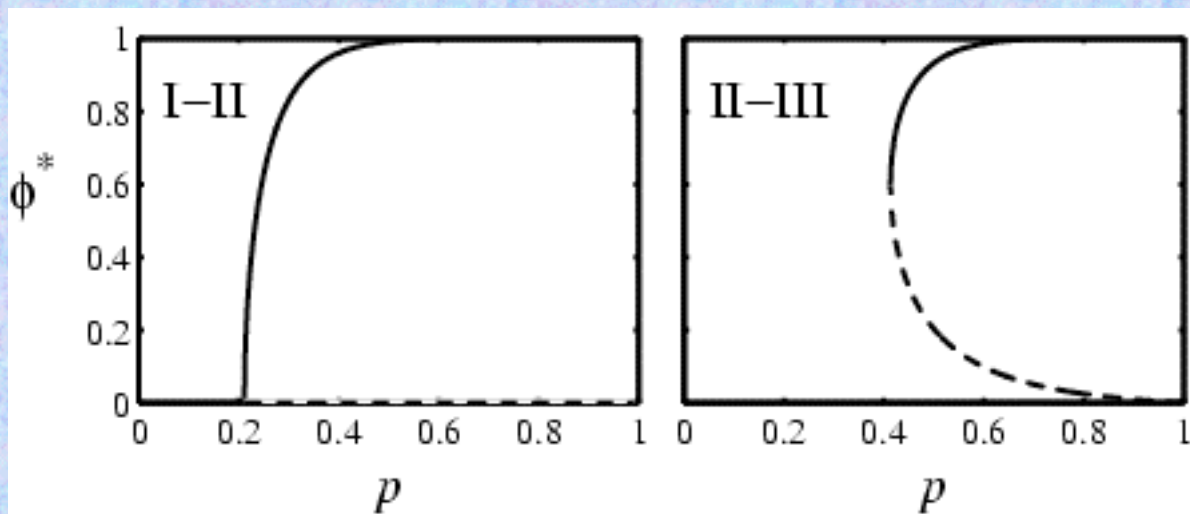


Class II of particular interest because of sensitivity (near unstable branch of SN bifurcation) both to  $p$  and also  $\phi_0$

# Transitions Between Classes

- Also more complicated in heterogeneous case than homogeneous case ( $d^*=1$ )
  - Class I requires  $P_1 > P_2/2$
  - Class II requires  $P_2/2 > P_1 > 1/T$
  - Class II requires  $1/T > P_1$
- Still, conditions are surprisingly simple (Equation 3 depends on all  $P_k$ )

# Graphically



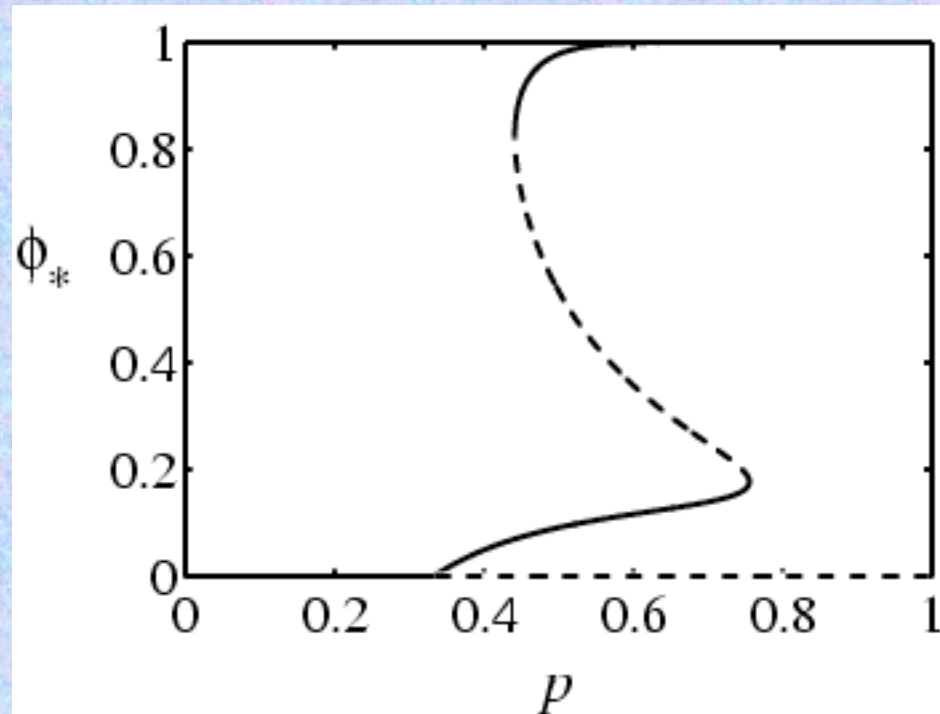
$$P_1 = P_2/2$$

$$P_1 = 1/T$$

# Hybrid Classes

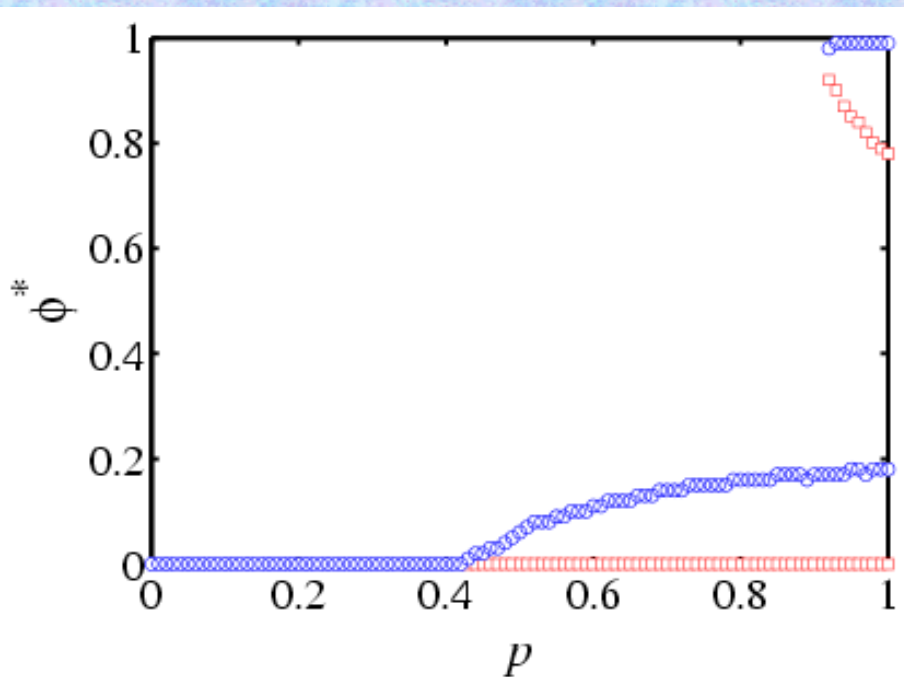
- For some distributions  $g(d^*)$ , we do find additional solutions to Equation 3 (i.e. more bifurcations)
- However
  - It appears that  $g(d^*)$  must be bi-modal with widely separated peaks
  - The new classes can be thought of as combinations of the three basic classes
  - The basic class structure remains (i.e. new bifurcations are added, but the existing ones are classified as before)
- Hence we stick with simple classification scheme

# Example of Hybrid Class (I and III)

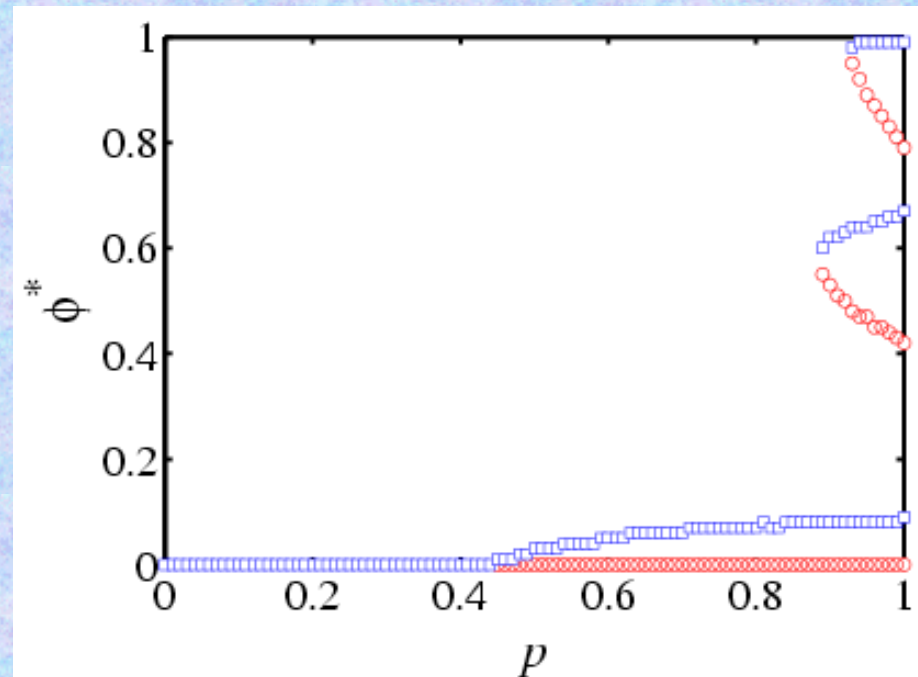


$$T = 20, d_i = 1, P[d^* = 1] = 0.15, P[d^* = 6] = 0.85$$

# More Hybrid Classes



0.2  $T=12$ ;  $d^*=1$  (prob 0.2) or 9 (prob 0.8)



0.2  $T=24$ ;  $d^*=1$  (prob 0.1),  
10 (prob 0.55), or 20 (prob 0.35)

# Conclusions

- Not all contagion is the same
  - If real contagion exhibits temporal interdependencies then model needs to reflect that (if  $P_1 < P_2/2$ )
- But not all contagion is different either
  - Only three universal classes
- Furthermore, simple conditions (on  $P_1$  and  $P_2$ ) predict into which class a given model should fall
- Might have some nice applications
  - Suggests a simple test for real-world contagion
  - Also suggests a possible intervention strategy (shifting individuals from  $P_1$  to  $P_2$ )
  - Finally, suggests that more attention should be paid to the “easily influenced” (rather than “influential”)

# Importance of $P_k$

- For a given choice of  $T$ , the  $P_k$  contain all the information about different choices of model (i.e. Equation 3 solely in terms of  $P_k$ , not  $f(d)$ ,  $g(d^*)$ )
- Suggests that all we need pay attention to is the  $P_k$
- If true
  - Can ignore how they are obtained from micro model
  - Perhaps can test for, and manipulate,  $P_k$ 's directly
  - Model becomes considerably more general
- So far, it's an open question

# Problems and Extensions

- In setting  $\rho=1$  and  $r=1$ , we have studied only the simplest case (and possibly not so interesting from an epidemiological perspective)
- Have considered  $r<1$ 
  - Basically changes position of  $p_c$  from  $1/T$  to  $1/(T+\tau)$  where  $t = (1-r)/r$  (although this relationship appears only to be approximate for heterogeneous case)
- But also need to consider
  - $\rho<1$  (SIRS)
  - $\rho = 0$  (SIR)
- Many other obvious extensions (e.g. Networks)

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