LIFE DATA EPIDEMIOLOGY

Lecture 6: Complex contagions

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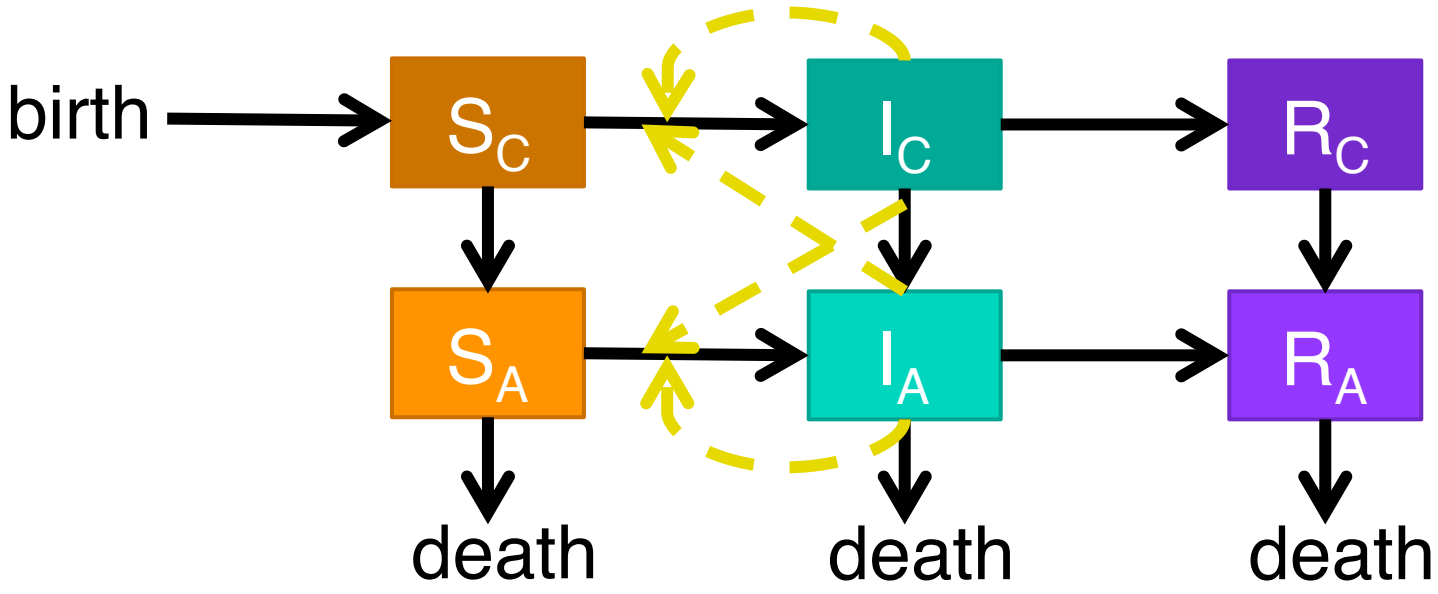
Age structure

- Other diseases have a different behavior at various ages, so consider population with (C)hildren and (A)dults



Age structure

- We can still write WAIFW matrix $\boldsymbol{\beta}$

$$\boldsymbol{\beta} = \begin{pmatrix} \beta_{CC} & \beta_{AC} \\ \beta_{AC} & \beta_{AA} \end{pmatrix}$$

- Hypotheses that are still reasonable
  - assortative mixing: $\beta_{ii} > \beta_{ij}$ with $i \neq j$
  - symmetry $\beta_{AC} = \beta_{CA}$
- But now it is unclear what class is more at risk, and also class sizes change in time

Age structure

- We may actually be tempted to treat age as a continuous parameter
  - but that makes the equations hard to solve
  - and at the same time, we do not have this many parameters
- Better replicate compartments and divide the population in age classes
  - model is more tractable and we can exploit standard assumptions (e.g. memoryless)

Control by vaccination

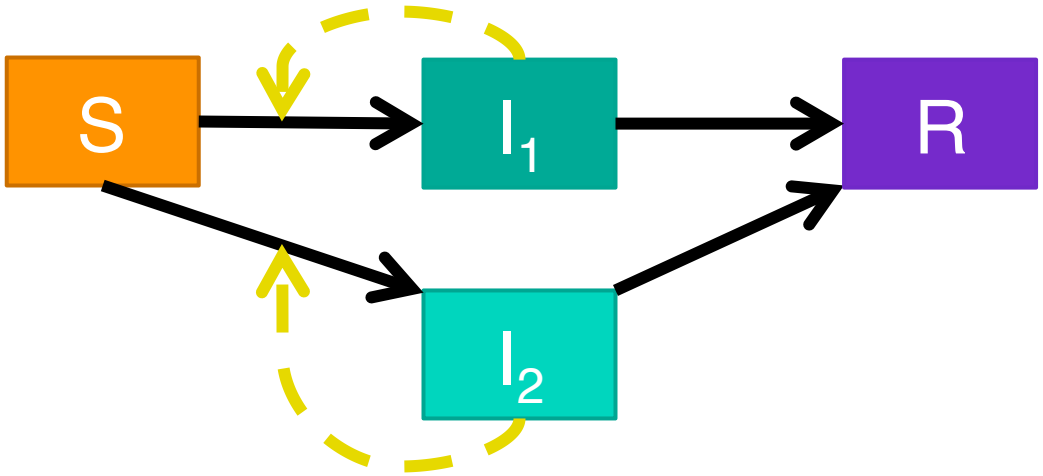
- For risk structure, it was more efficient to vaccinate or quarantine high-risk elements as this reduces the actual value of R_0
- In age structure, we do not have a class with "higher risk" (it can be both)
  - individuals partake in the entire dynamics (we expect them to be C, then A in due time)
  - yet, $s_\infty^{(a)} / n_\infty^{(a)} \geq s_\infty^{(b)} / n_\infty^{(b)}$ if $a < b$ (younger than)

Control by vaccination

- This implies that to control the disease spread, most of the individual's lifespan should be immune; thus, it is generally best to vaccinate the **youngest** group
  - just a general rule, though (it depends on the numbers and also on waning immunity)
- If children are vaccinated, $1-1/R_0$ still well approximates the minimal vaccination rate
  - often used for measles and baby-diseases

Multi-pathogen

- Consider an SIR with 2 (or more) strains of disease within the same population
- Cross-immunity relates to individuals becoming infected with only one strain.
- Simplest case is complete cross-immunity



Multi-pathogen

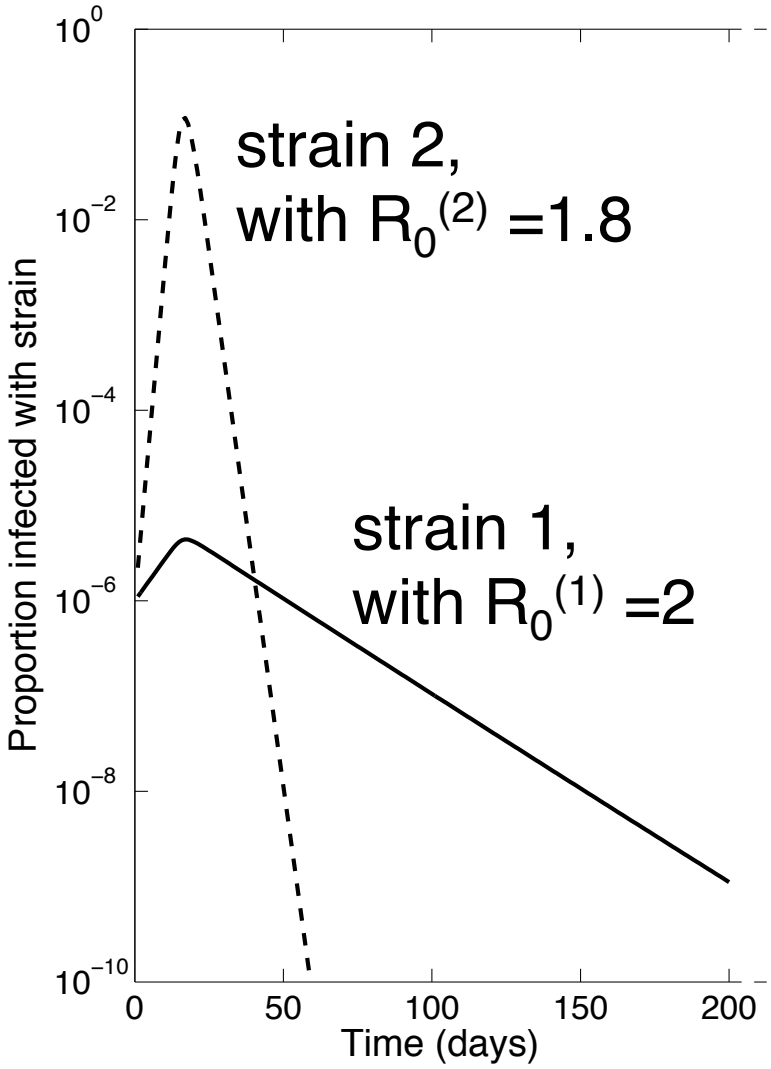
- Class I_j only helps creating I_j members
  - we can write per-class $R_0^{(j)}$ as per definition
  - without loss of generality, say $R_0^{(1)} > R_0^{(2)}$
- Now write + solve equations as usual or...
  - use a principle: 2 infections cannot coexist!
  - we can show that the equilibrium in case of complete cross-immunity, is the same as SIR with just one strain (because in the end only one strain is there)

Multi-pathogen

- Proof: we expect $x(t)$ to grow and reach a peak (when $s\,R_0 = 1$) and then go down
  - as we have 2 different R_0s, if we believe to be at the endemic point for $R_0^{(2)}$, we still have a higher $R_0^{(1)}$ causing x to still grow
  - → thus, we cannot be at equilibrium
- The only possibility is that at equilibrium, one strain survived, the other is eliminated

Implications

- Relationship $R_0^{(1)} > R_0^{(2)}$ does not mean that strain 2 cannot have faster dynamics (being stronger than strain 1 in the short term)



Evolutionary implications

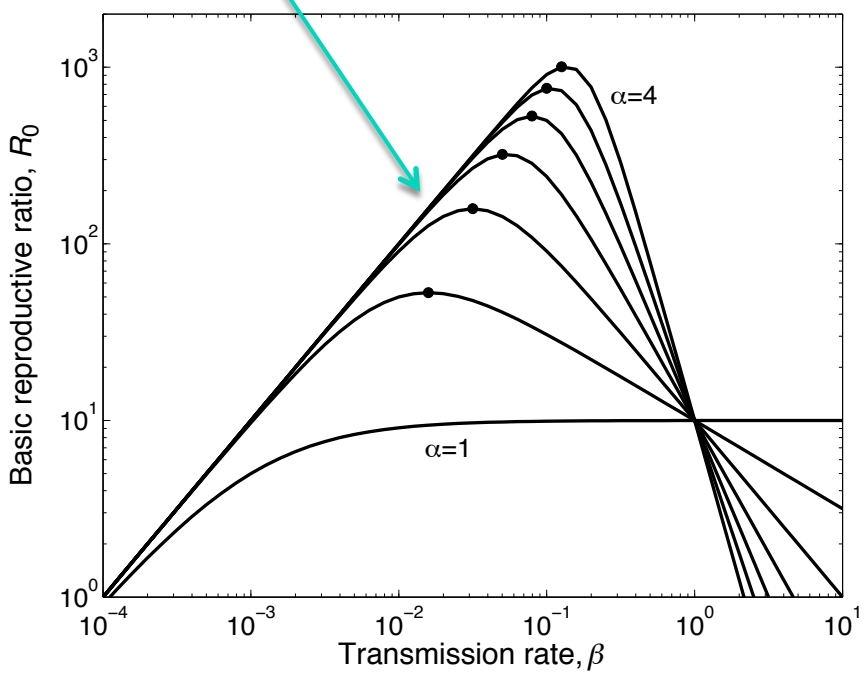
- Why do not we have a single dominant disease with extremely high R_0?
  - that would be the limit of a spotted equilibrium where mutants keep appearing with higher R_0
  - also this would be a harmless disease (why it has to be harmful to the host?)
- Biologists would answer that there is a natural tradeoff of transmission (how contagious) vs virulence (how deadly)

Evolutionary implications

- If a disease produces many pathogens, it spreads rapidly but is also harmful to the host; thus, we cannot have very high β without μ being very high too
  - e.g., STDs have long infected time $1/\mu$ but also are not very contagious, compared to measles (high β, but $1/\mu$ is short)

A power law?

- The tradeoff between β and μ is generally taken as a power law $\mu = k\,\beta^{\alpha}$
- If this holds, R_0 cannot grow arbitrarily
  - it can only reach these peaks
- Why power law?
  - easy (but hard to validate choice of α)
  - other functions may fit better

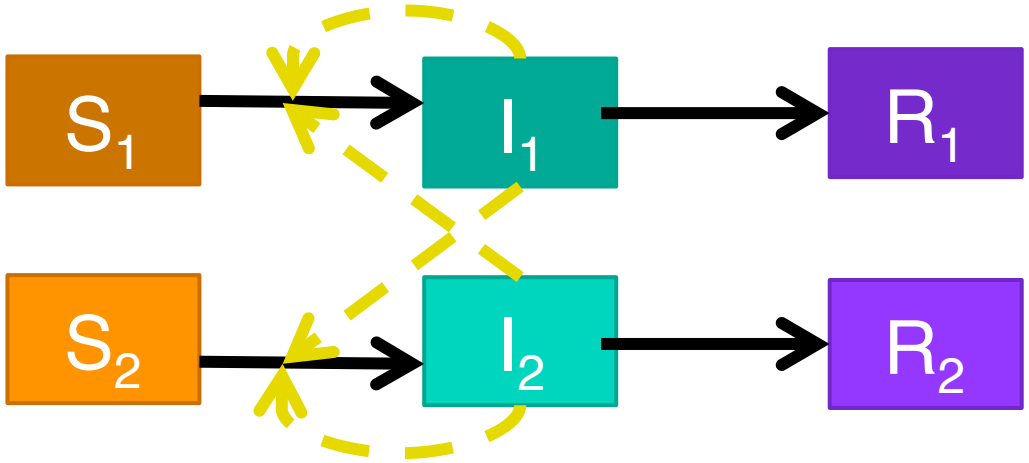


Without cross-immunity

- This situation has two extreme cases
  - simultaneous infections are frequent, and in the end it is just two separate strains evolving orthogonally on same population
  - or multiply infected individuals are rare (e.g., because when you are sick you control yourself and avoid further infections) → this is just like a single infection

Multi-host

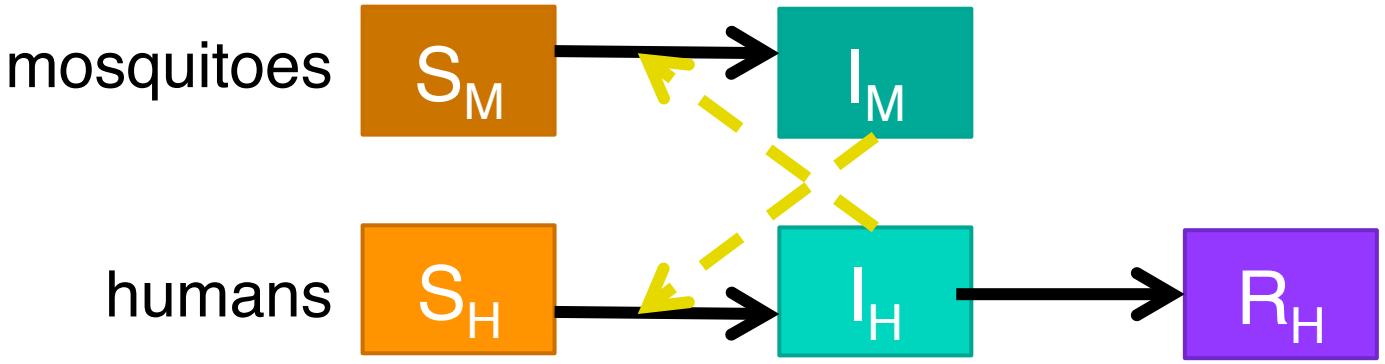
- We can connect this to risk-structure



  - main difference: now we have two distinct populations (of different species! so, we will not require symmetry of WAIFW)
  - some special cases are notable

Vectored transmissions

- This is the case for many diseases carried by mosquitoes (or similar insects)
  - $S_M \rightarrow I_M$: susceptible mosquito bites infected
  - $S_H \rightarrow I_H$: infected mosquito bites susceptible



Vectored transmissions

- No intra-species infection, thus WAIFW is

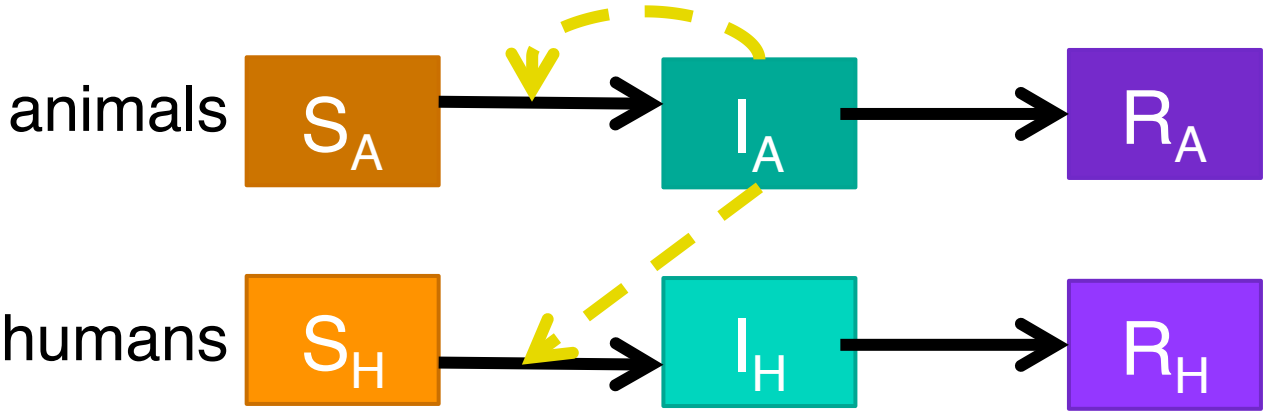
$$\beta = \begin{pmatrix} 0 & \beta_{MH} \\ \beta_{HM} & 0 \end{pmatrix}$$

  - can be related to "bite rate" of mosquitoes
    r = bites per sec/ N_H

- Usually, transmission mechanism is:
  - frequency-dependent for human population
  - density-dependent for mosquitoes
  - i.e. mosquitoes bite at same rate, humans get bitten more often if mosquito-density ↑

Zoonoses

- Here, the disease is mostly active in animals, but can affect humans
  - yet, negligible contagion rate from humans



  - e.g. brucellosis, Ebola, rabies, toxoplasmosis

Zoonoses

- Now the WAIFW matrix is $\beta = \begin{pmatrix} \beta_{AA} & \beta_{AH} \\ 0 & 0 \end{pmatrix}$
  - simpler model to study
  - yet, epidemics hard to eradicate as we estimate R_0 and enact countermeasures in humans but spreading happens in animals
- Also, combined cases: vectored zoonosis
  - spreading in animals (birds/mammals) can extend to humans via insects bites: Lyme, Chagas, leishmaniasis, bubonic plague

Temporal forcing

- It has been observed that some diseases have a cyclic behavior (seasonal flu, outbreak of measles/smallpox in schools)
  - the oscillatory pattern of the endemic equilibrium of classic SIR model is insufficient to explain these trend
- Some models introduce a temporal forcing to explain these phenomena

Temporal forcing

- A simple example
  - we have birth rate λ and recovery rate μ but we neglect deaths in classes S and I

$$\frac{ds}{dt} = \lambda - \beta(t)\ sx$$

$$\frac{dx}{dt} = \beta(t)\ sx - \mu x$$

  - $\beta(t)$ represents seasonal variability in the contact rate (e.g., school time of children)
- E.g., $\beta(t)$=constant term + sinusoid

$$\beta(t) = \beta_0 [1+\beta_1 \cos(2\pi f_1 t)]$$

Temporal forcing

- If $X = X_\infty(1+\chi)$, χ=small perturbation, we get

$$\frac{d^2\chi}{dt^2} + \lambda R_0 \frac{d\chi}{dt} + \lambda\beta_0(t)\chi = -\omega_1 \beta_1 \mu \sin(\omega_1 t)$$

- Oscillatory with frequency f_1 and amplitude

$$M = \omega_1 \beta_1 \mu \left[(\lambda\beta_0 - \omega_1^2)^2 + (\lambda R_0 \omega_1)^2 \right]^{-0.5}$$

- Generally, $M \gg \beta_1$ so natural oscillations are highly amplified (**resonance**)

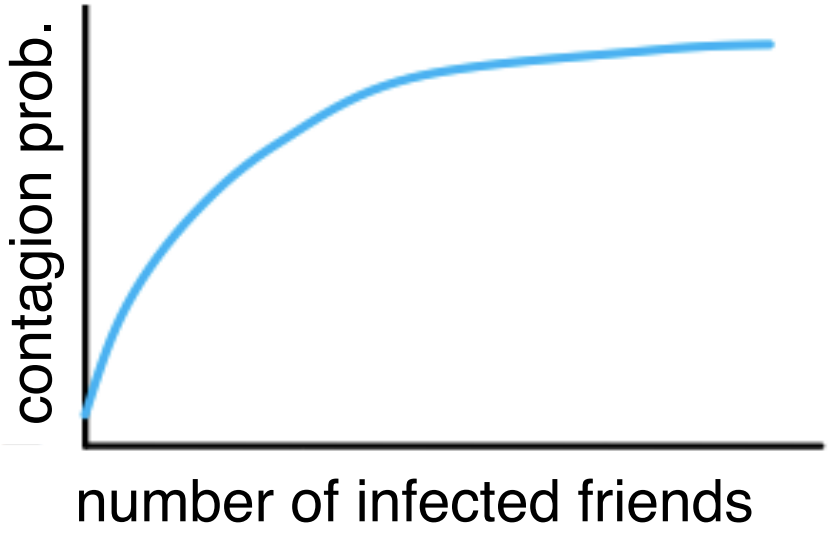
Complex contagion

- What if infection occurs after exposure to multiple sources? → **complex contagion**
- Diseases (a single contact is enough) vs:
  - adoption of innovation
  - consensus over a policy
  - spreading of a news (or urban legend)

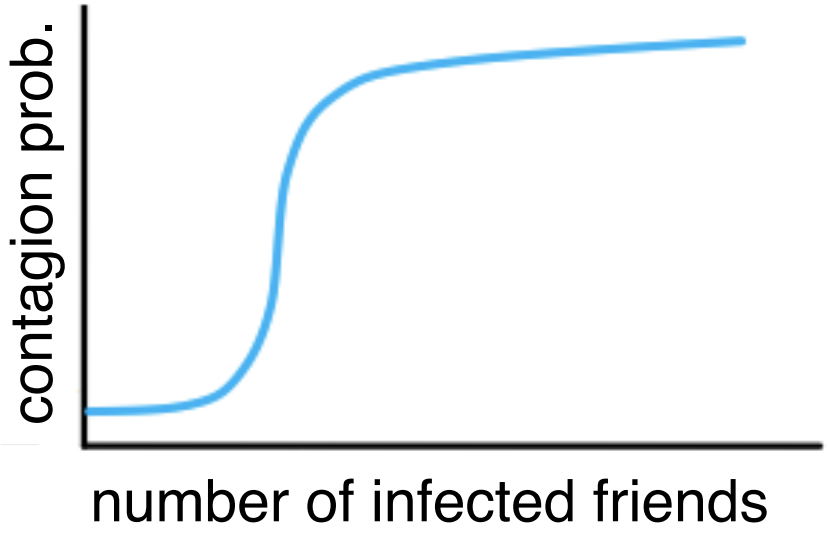
  these usually requires multiple sources!
- No consolidated analytical models ☹
  - how can we quantify this anyways?

Exposure

- Driving process=**exposure** (passive influence)
  → expect a monotonic increasing trend



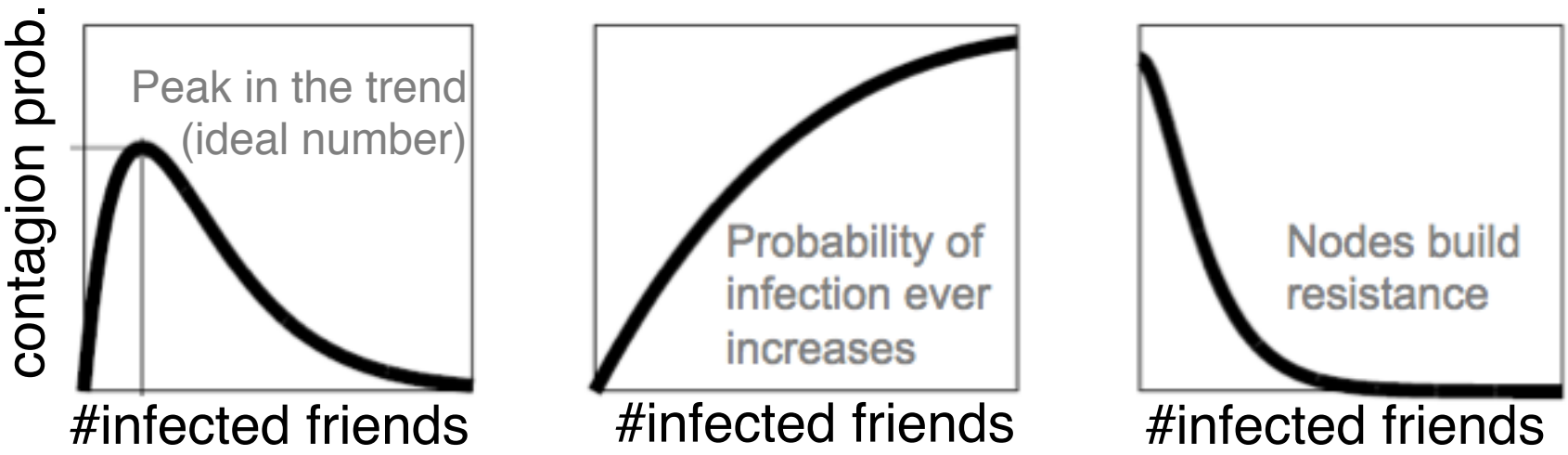
diminishing law
(prob⟶1 but saturates)



activation threshold
(min #infected neighbors)

Adoption

- Or we require an active agreement to accepts the contagion (**adoption**)

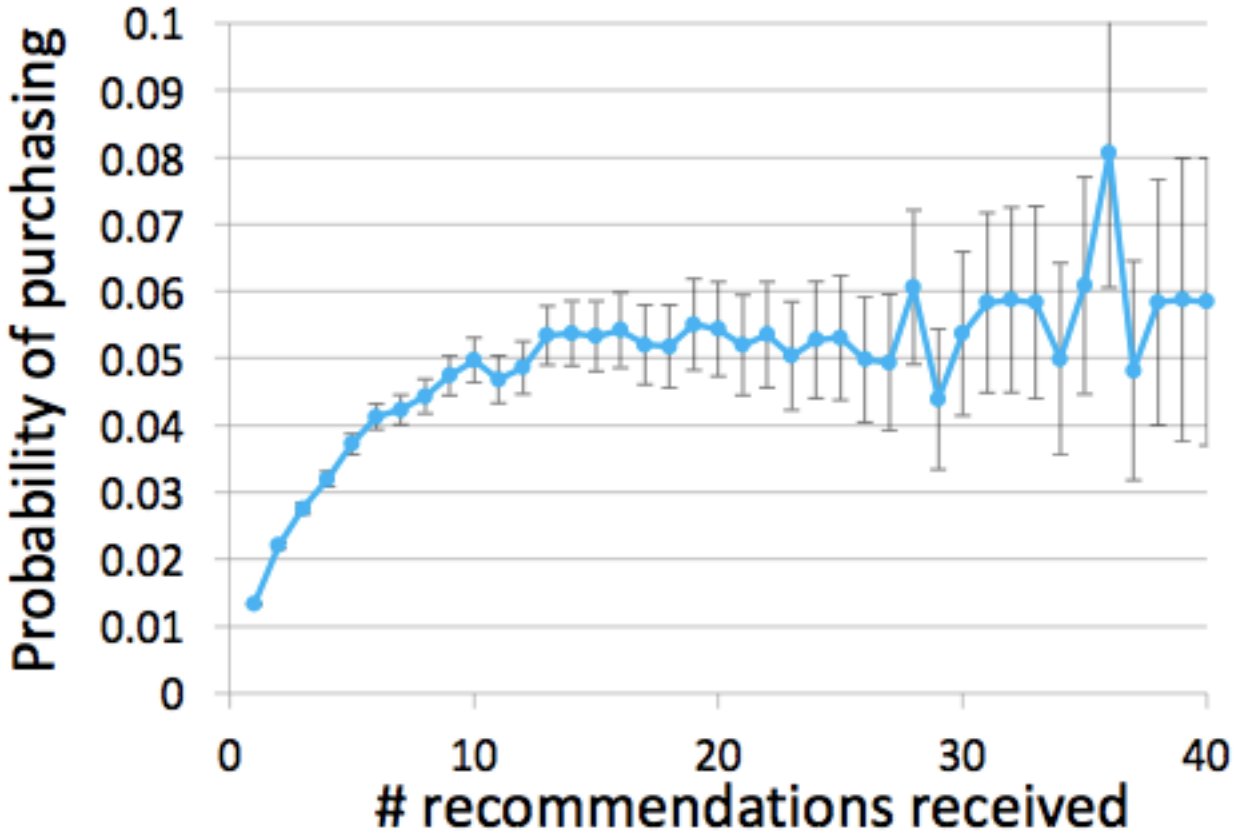


Exposure - Adoption

- We can also combine two behaviors
  - the higher #infected neighbors, the higher the number of contagious interactions
  - this is taken as the input for adoption
- Applications
  - some friends of yours are joining a social network: do you join it too?
  - how many ads needed to trigger a buy?
  - how many views to check a viral video?

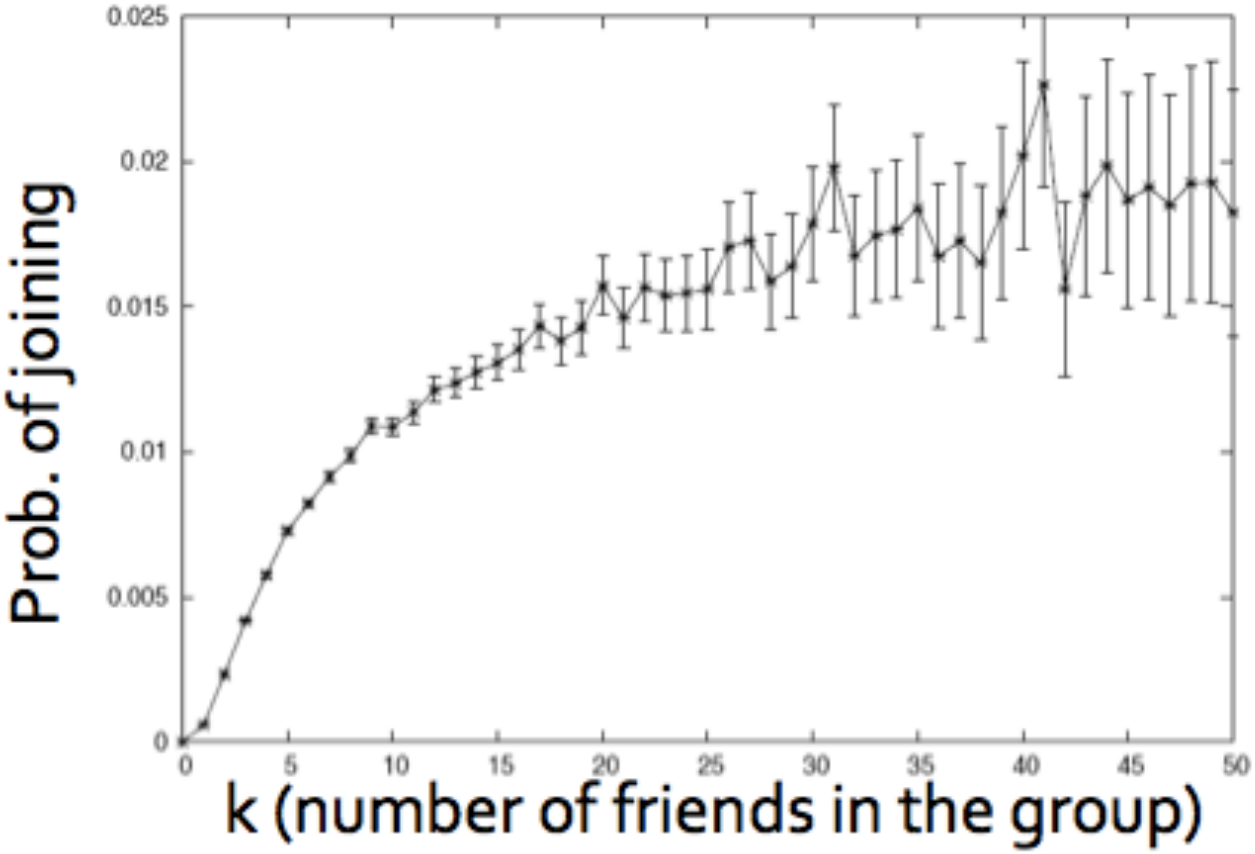
Example of application

- DVD Recommendations



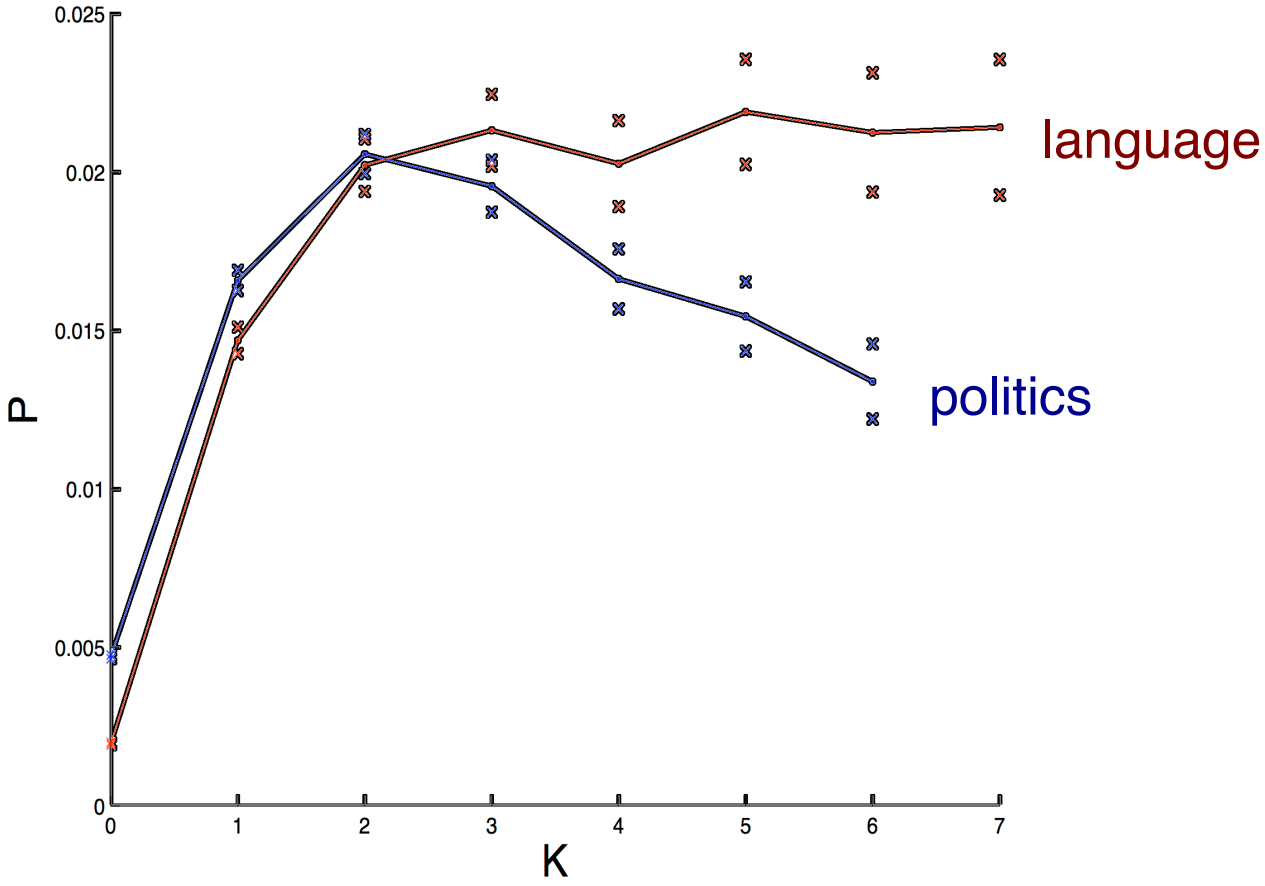
Example of application

- LiveJournal membership



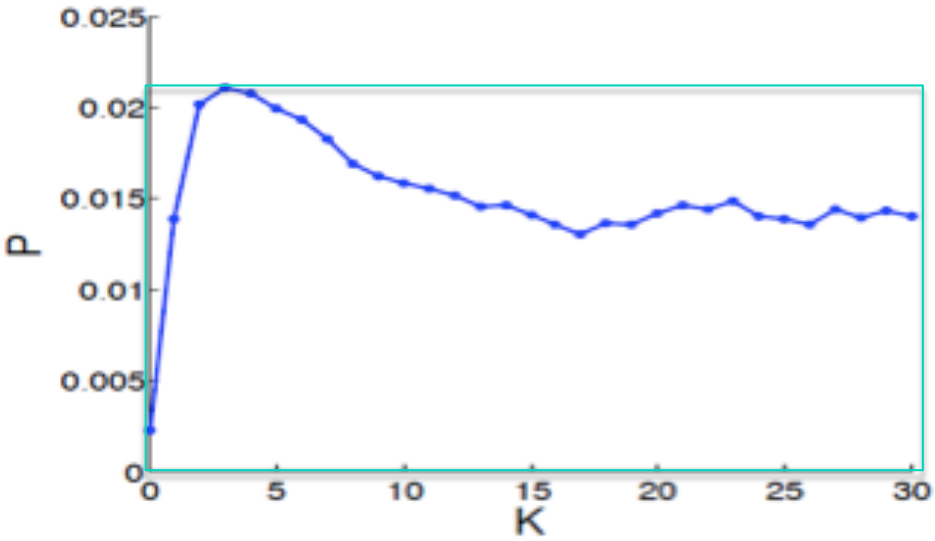
Example of application

- Retweeting probability



Spreading on Twitter

- **Persistence**: ratio $\int_0^{k_{max}}$ curve / $\int_0^{k_{max}}$ peak



- **Stickiness**: just the peak (assumes we exploit contagion at its top strength)