Homophily, Contagion, Confounding: Pick Any Three

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My interest: non-parametric reconstruction of dynamical systems from the behavior they generate

Perspective: Yet another ex-physicist

$\therefore$ Social networks are "just" large coupled dynamical systems

Apologies in advance for social-scientific and graphological naivete

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5. yes: because you're both on it when it starts collapsing and that's the only way off (external causation)

Wikipedia, s.v. “Tacoma Narrows Bridge (1940)”

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Manski (1993) suggests this is just not identifiable, but does not quite settle the problem

Influence due to group average vs. individuals

Contagion, Influence

Whether i does something at time t is well-predicted by whether i's neighbors had already done it at $t-1$

- Diffusion of innovations
- Infectious diseases
- Not-obviously-infectious conditions (e.g., obesity) ...

This *can* be due to influence or contagion

Analogy of ideas to diseases is very old: Pliny used it in 110 (*Epistles* X 96)

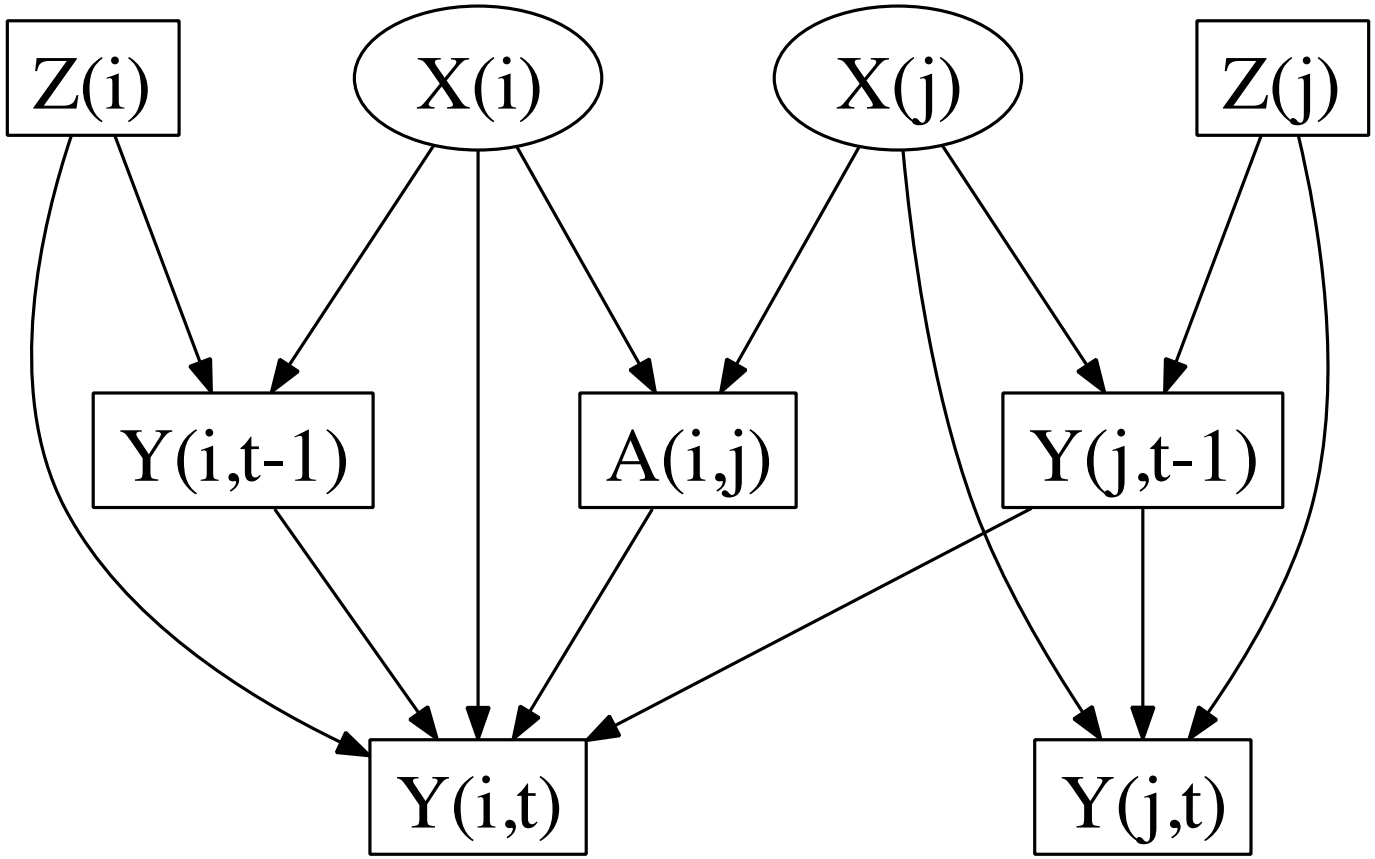
Can the same *observational* consequences can follow from latent homophily?

Notation:

- $Y(i,t) =$ does node i show condition/behavior at time t?
- $X(i) =$ *latent* persistent trait of i
- $Z(i) =$ other, manifest persistent traits
- $A(i,j) =$ whether there is an edge from j to i

We suppose that:

- $Y(i,t-1)$ has a direct influence on $Y(i,t)$
- $X(i)$ has a direct influence on whether/when i adopts
- $Z(i)$ has a direct influence on $Y(i,t)$ (possibly null)
- $Y(j,t-1)$ *may* have a direct influence on $Y(i,t)$, but only if $A(i,j) = 1$
- Homophily: $X(i)$ and $X(j)$ both directly influence $A(i,j)$


Z(i)
X(i)
X(j)
Z(j)
Y(i,t-1)
A(i,j)
Y(j,t-1)
Y(i,t)
Y(j,t)

Contagion Effects are Nonparametrically Unidentifiable

Informally:

1. $Y(j, t-1)$ is informative about $X(j)$
2. $X(j)$ is informative about $X(i)$ if i and j are neighbors
3. $X(i)$ is informative about $Y(i, t)$
4. $\therefore Y(i, t) \not\perp\!\!\!\perp Y(j, t-1)$, even if there is no direct causal effect
5. $\therefore$ Latent homophily is confounded with contagion

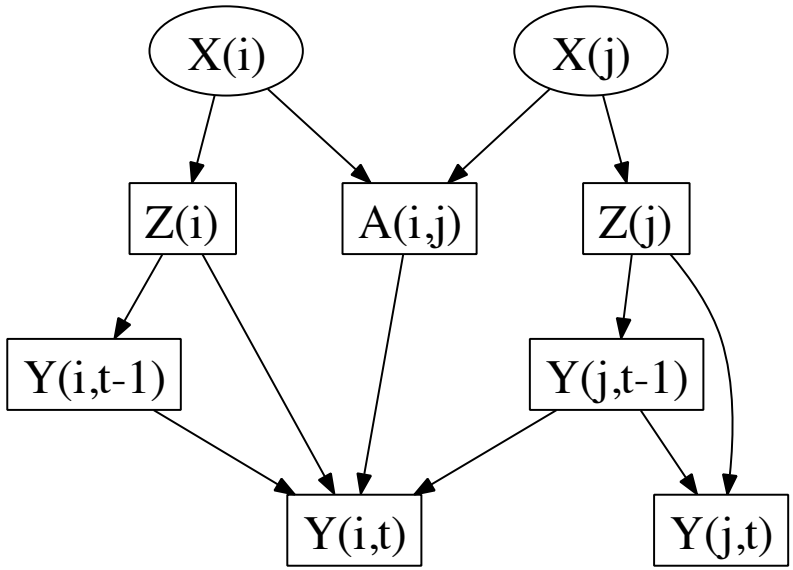
More formally:

1. $Y(i,t) \leftarrow X(i) \rightarrow A(i,j)$ is a confounding path from $Y(i,t)$ to $A(i,j)$
2. Likewise $Y(j,t-1) \leftarrow X(j) \rightarrow A(i,j)$ is a confounding path from $Y(j,t-1)$ to $A(i,j)$
3. $\therefore$ the direct effect of $Y(j,t-1)$ on $Y(i,t)$ is not identifiable (Pearl, 2009, §3.5, pp. 93–94)

Adding conditioning on $Y(i,t-1)$ and $Y(j,t)$ does not remove the confounding paths
Neither does adding conditioning on $Z(i), Z(j)$
Argument still goes through with time-varying edges (more spaghetti)

Getting Identifiability

Parametric assumptions *can* suffice
Better: condition on X; or find Z which block paths from Y to X
Explicit modeling as in Leenders (1995); Steglich *et al.* (2004) does both



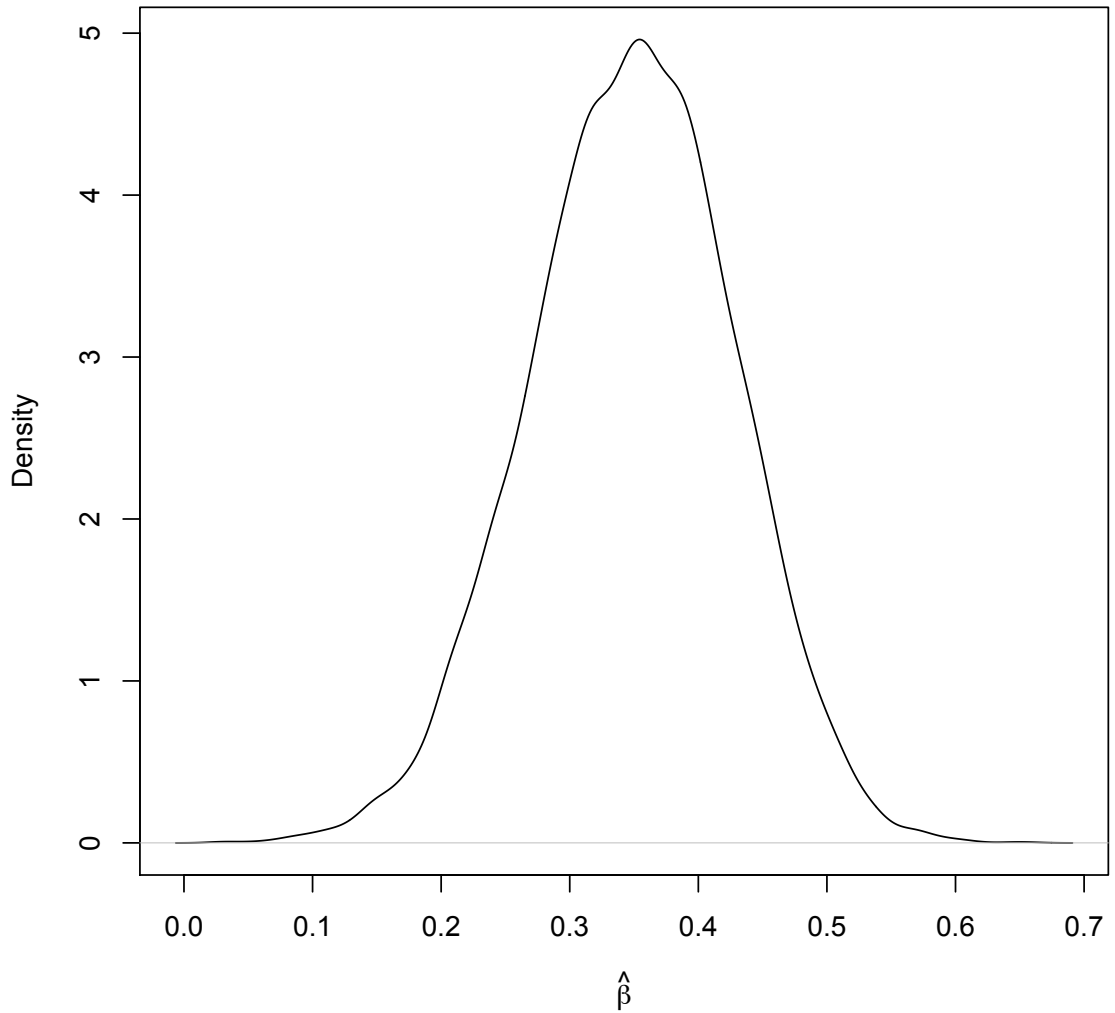
The Argument from Asymmetry

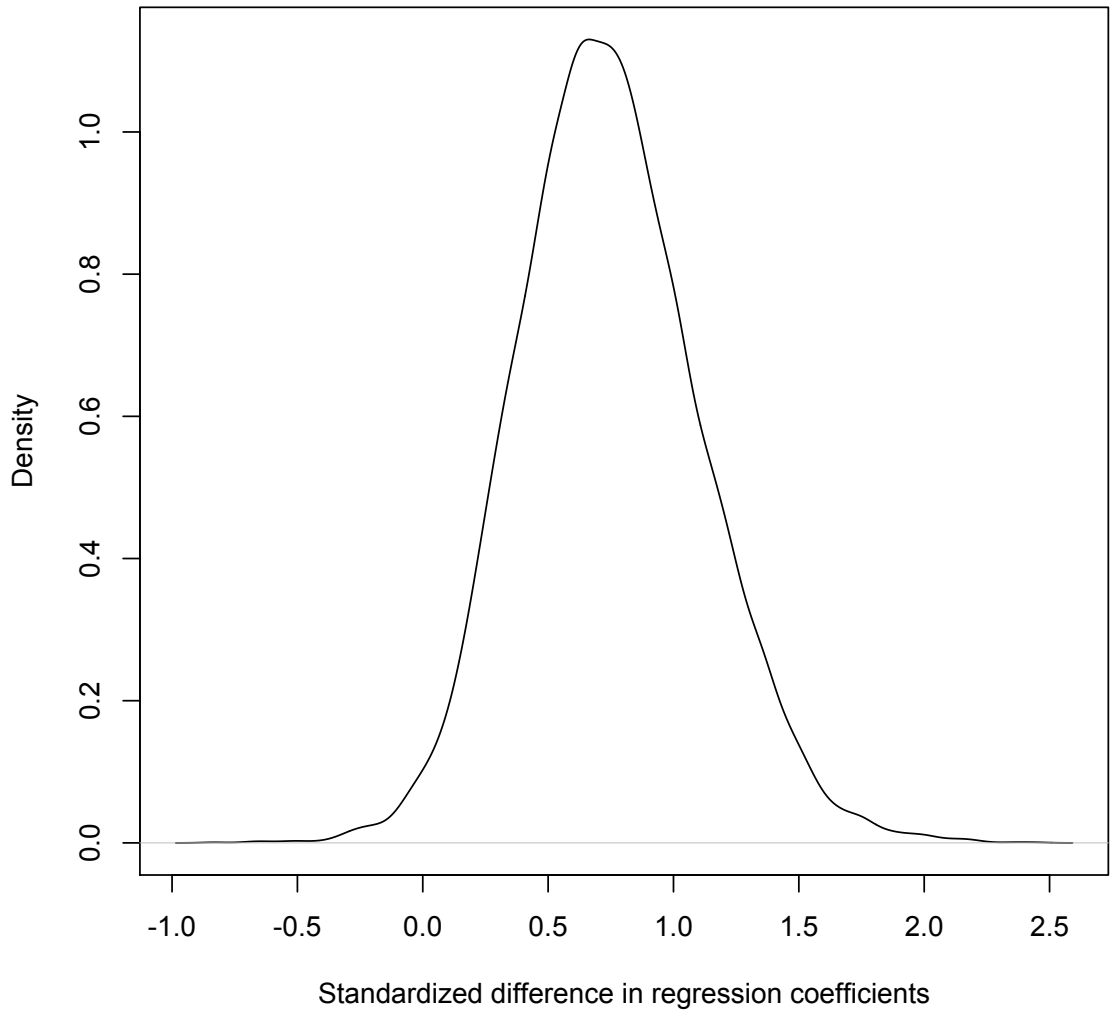
Focus on unreciprocated edges, $i \rightarrow j$, $j \not\rightarrow i$
Suppose $Y(i,t)|Y(j,t-1)) \not\sim Y(j,t)|Y(i,t-1)$
Doesn't this argue for direct influence?
Considerable *prima facie plausibility*
Argument breaks down if senders and receivers have systematically different values of X, with different local relations to Y

Toy Example

Ignore time-dependence; try to predict $Y(i)$ from $Y(j)$ and vice versa when $A_{ij} = 1, A_{ji} = 0$
$X(i) \sim \mathcal{U}(0,1)$
Edges form with probability $\propto \text{logit}^{-1}(-3|X(i) - X(j)|)$
i nominates j from among neighbors, $\propto \text{logit}^{-1}(-|X(j) - 0.5|)$
$Y(i) = 10(X(i) - 0.5)^3 + \mathcal{N}(0, 0.1)$
Results:

- $Y(i)$ is well-predicted from $Y(j)$
- *Nominees* are disproportionately in the middle; $i \to j, j \not\to i$ suggests i is more peripheral
- For asymmetric pairs, regression of sender on receiver differs from that of receiver on sender


Sampling distribution: regression coefficient of Y(i) on Y(j)
Density
0
1
2
3
4
5
0.0
0.1
0.2
0.3
0.4
0.5
0.6
0.7
$\hat{\beta}$


Asymmetry from preferential nomination
Density
0.0
0.2
0.4
0.6
0.8
1.0
-1.0
-0.5
0.0
0.5
1.0
1.5
2.0
2.5
Standardized difference in regression coefficients

Making homophily and contagion look like causation

Long-term, hard-to-change social/economic status explains more short-term, malleable cultural / political / consumer variables

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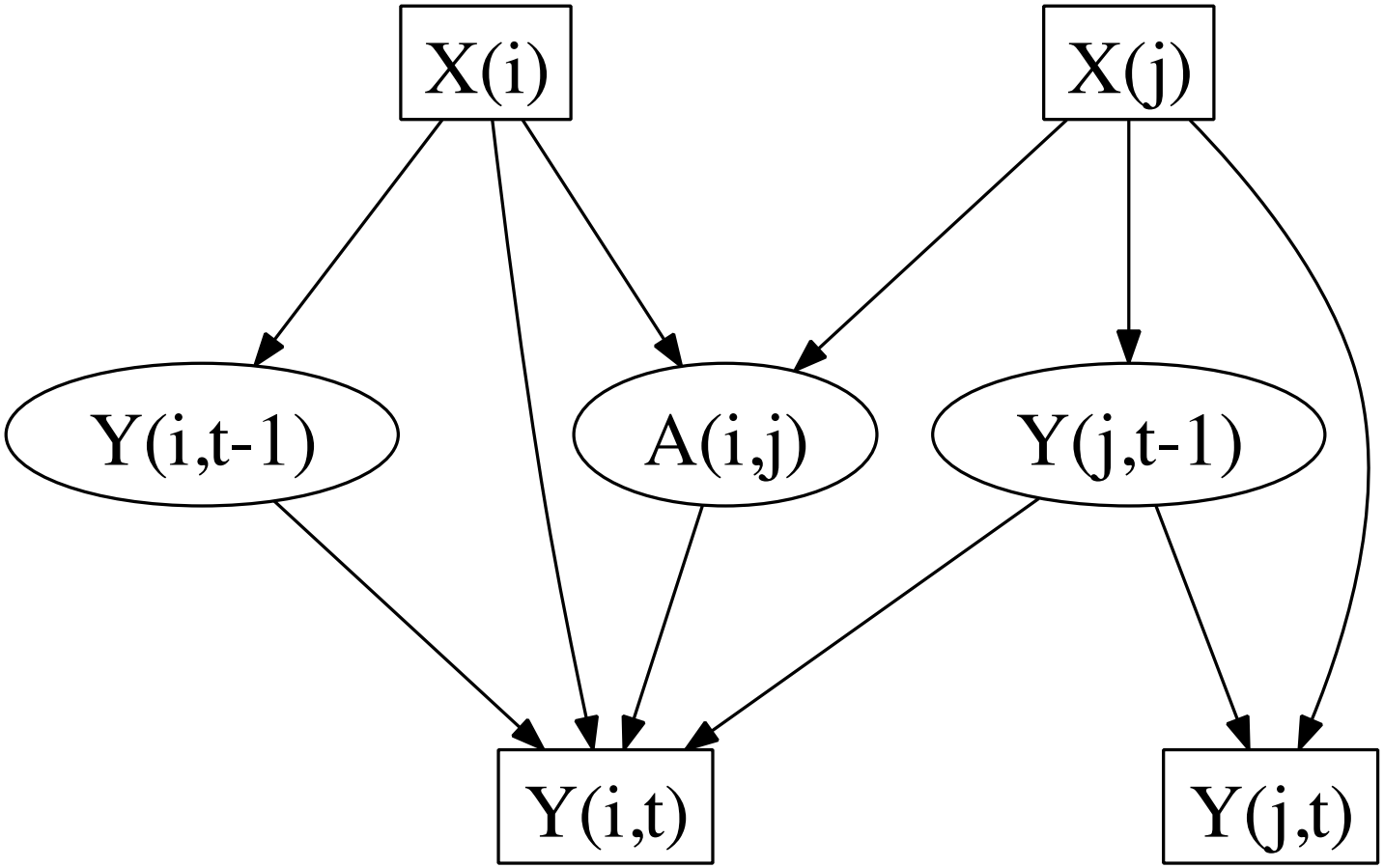
> *Gellner: "Social structure is who you can marry, culture is what you wear at the wedding."*

What's the evidence?

- The stories sound good
- Casual empiricism
- Correlation/regression analyses; cultural choices are predictable from social positions (e.g. Bourdieu (1984))

Probably even true a lot of the time
BUT usually ignores social networks and just looks at surveys


X(i)
X(j)
Y(i,t-1)
A(i,j)
Y(j,t-1)
Y(i,t)
Y(j,t)

More Confounding

Direct influence of $X(i)$ on $Y(i,t)$ is confounded with contagion:

1. $X(i)$ is a cue about who i's friends are, i.e. $A(i,j)$
2. $\therefore X(i)$ is a cue about what i's friends think, $Y(j,t-1)$
3. contagion: $Y(j,t-1)$ influences $Y(i,t)$ if $A(i,j)=1$
4. $\therefore X(i) \not\perp Y(i,t)$ even if no direct influence

Responsible Just-So Story-telling

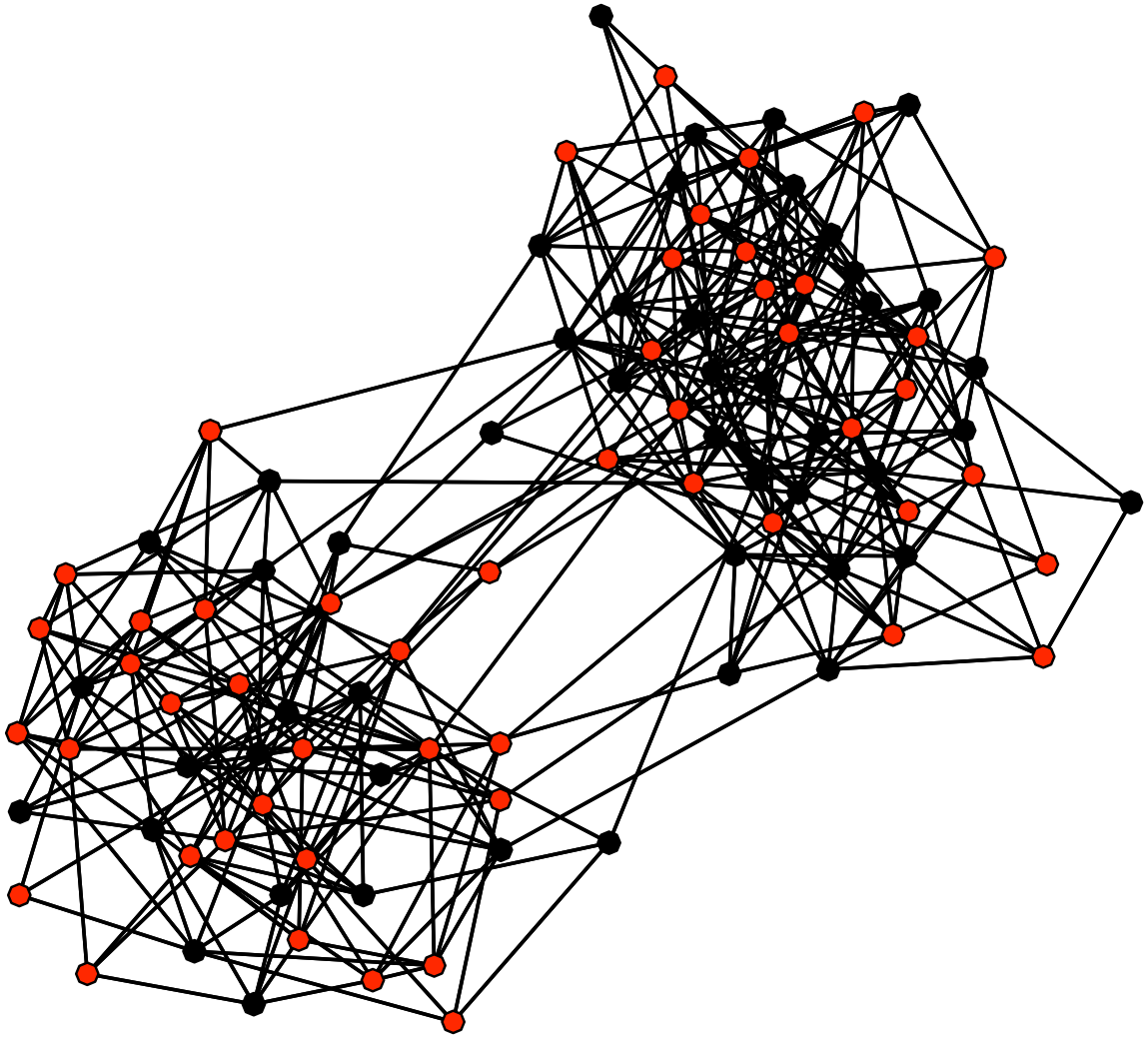
These accounts are usually adaptationist/functionalist
At the very least they are causal accounts
We should really check them
Biology suggests: a **neutral model**

- Include all the evolutionary processes *except* adaptation
- Work out expected behavior of this model
- Data departing from neutral model $\Rightarrow$ evidence of adapation

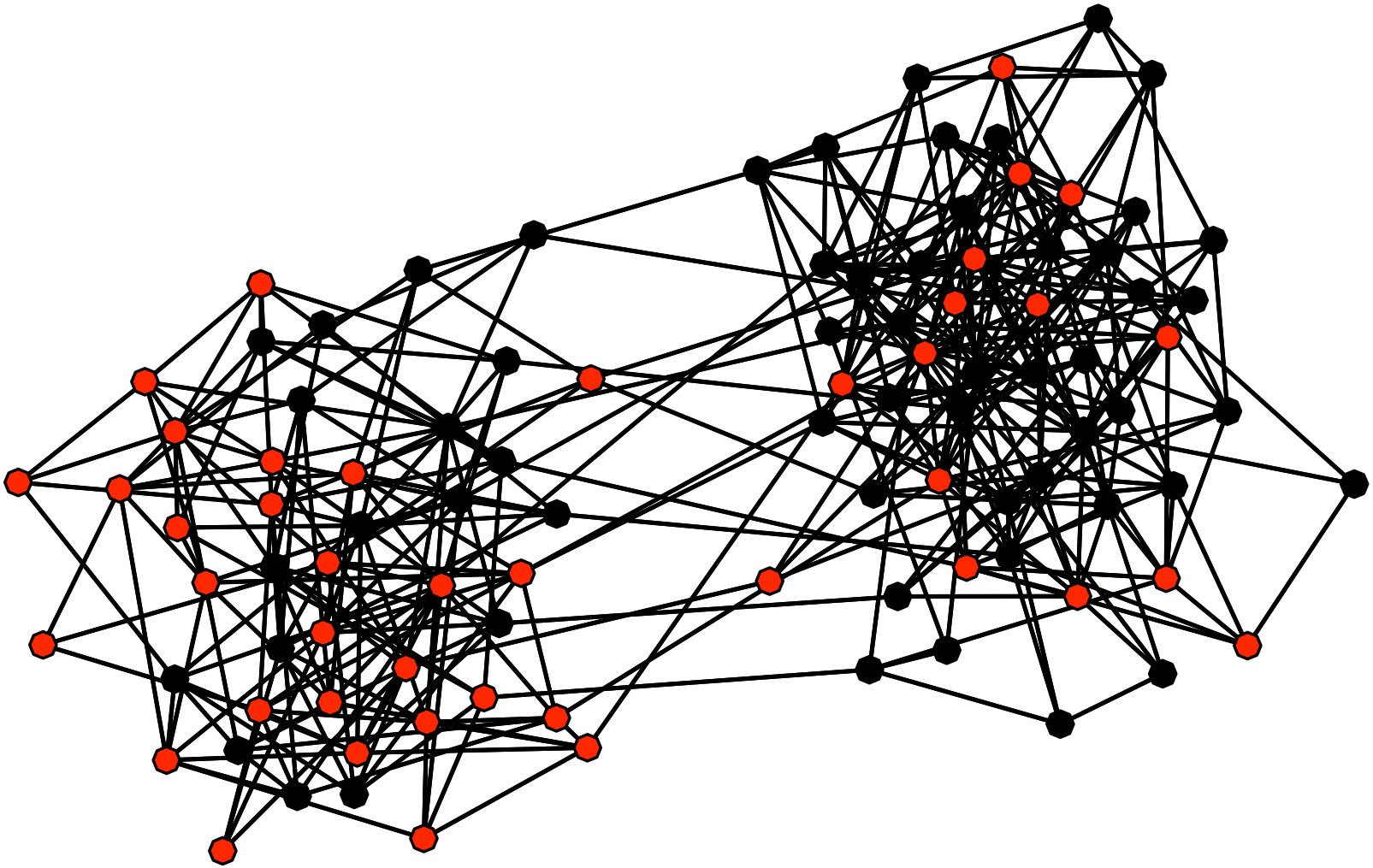
Caricature Neutral Model of Cultural Evolution

- $X(i) =$ unchanging status variable for node i ("social")
- Network is assortative on X (minimal departure from Erdős-Rényi)
- $Y(i,t) =$ rapidly changing choice variable for i ("cultural")
  1. At each t, pick a random i, and a random neighbor j
  2. Set $Y(i,t) = Y(j,t-1)$
  3. Go to (1)
- $Y(\cdot, 0) =$ Bernoulli(1/2) process

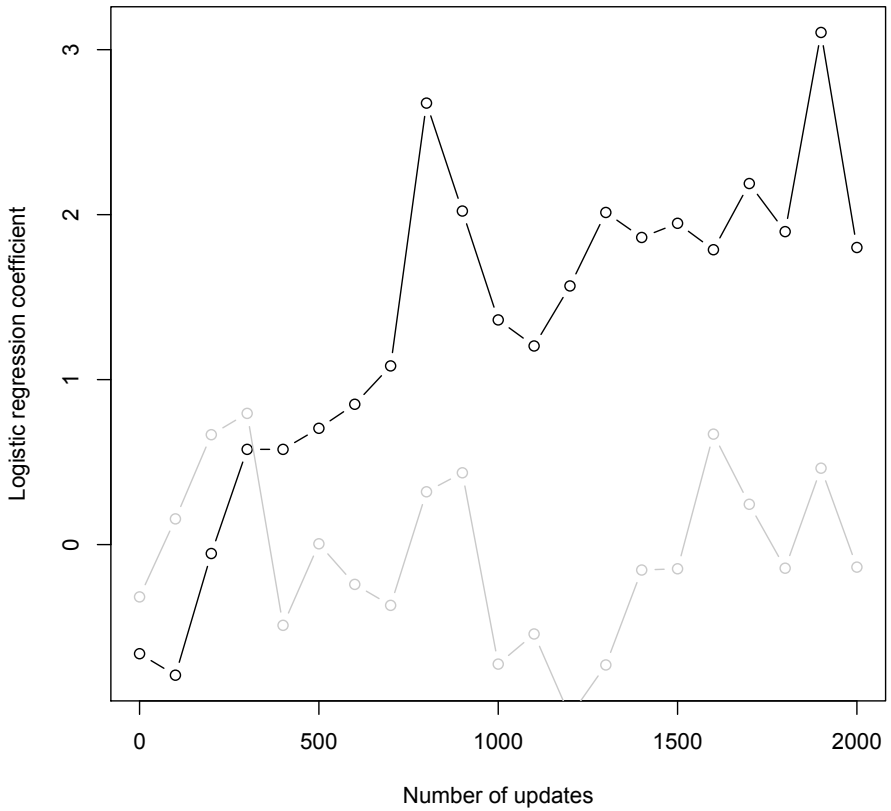
(= "voter model" of statistical mechanics)



100 node network, homophily for status (2 groups), initial choices



After 1000 updates



100 node network

Logistic regression of choice on status
black = assortative
grey = non-assortative

- Neutral diffusion + homophily looks like a real connection between social status and cultural choices
- Problem is *not* the ecological fallacy (red-state/blue-state fallacy) (not using aggregated data)
- Problem is that choices are not independent conditional on statuses
- Deconfound by conditioning on previous Y_j of neighbors

Partial Control by Clustering?

If the problem is latent heterogeneity, why not try to identify the latent trait?
Latent homophily $\Rightarrow$ you tend to resemble your neighbors
$\Rightarrow$ Especially likely if you all have lots of neighbors in common who all have lots of neighbors in common, etc.
$\Rightarrow$ modules/communities
Can't remove confounding but *might* reduce it
. . . or make it worse if the latent relationship isn't simple homophily (e.g. block models)

An Analogy For Community Control

Gene association studies: does having this genetic variant influence this trait/change this risk?

Real populations are structured

Sub-populations differ (due to reproductive isolation etc.)

$\Rightarrow$ genes are correlated

$\Rightarrow$ random biases and inflated variances (vs. usual formulas)

$\Rightarrow$ many bogus results

Population structure substantial even for e.g. Germany (Steffens *et al.*, 2006) or Italy, never mind "white Americans"

Responses: (1) pedigrees; (2) "genomic control" by estimating over-dispersion empirically (Devlin *et al.*, 2001); (3) clustering — the diffusion maps in Lee *et al.* (2009) look *a lot* like Newman (2006)

Conclusion

1. Homophily + causal influence looks like contagion
2. Homophily + contagion looks like causal influence
3. Of course contagion + causality looks like (is?) homophily

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