Homophily, Contagion, Confounding: Pick Any Three

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Joint work with Andrew Thomas, CMU Statistics

Details: Shalizi and Thomas (2011)

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5. yes: because sometimes jumping off a bridge is the only sane thing to do (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

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

Causal Inference

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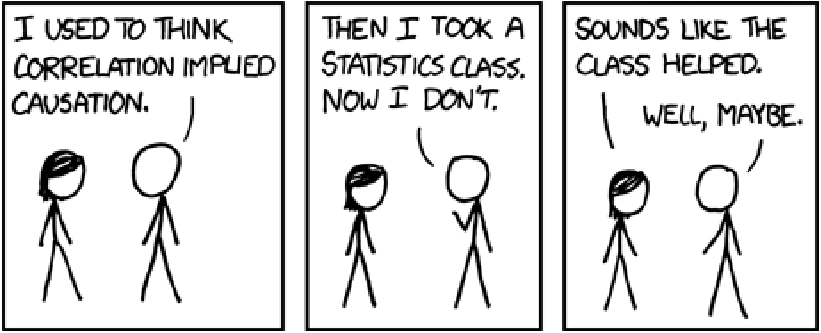

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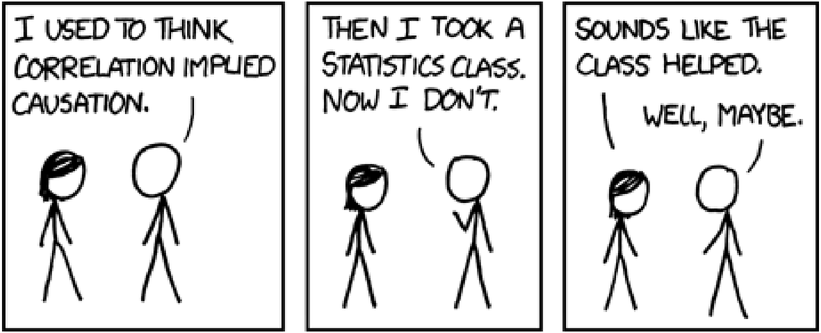

"Correlation doesn't imply causation, but it does waggle its eyebrows suggestively and gesture furtively while mouthing 'look over there'"

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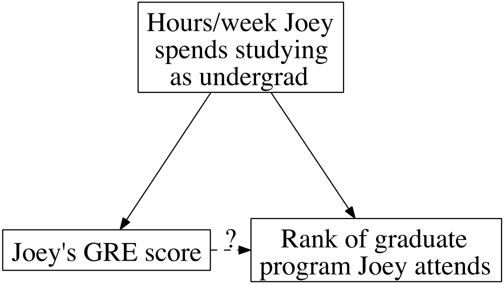
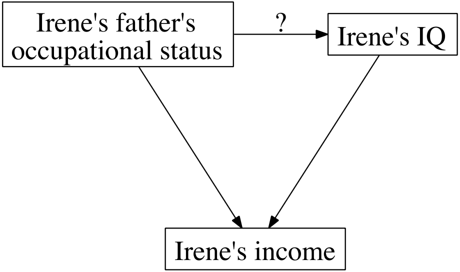
Do controls *activate* indirect paths?

Separate question: what causal diagrams are compatible with the correlation pattern? (Spirtes *et al.*, 2001)



CONTROL: Conditioning on hours studied lets us estimate the effect of GRE scores on college admission



CONFOUNDING: Conditioning on child's income *makes* child's IQ and father's status dependent

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

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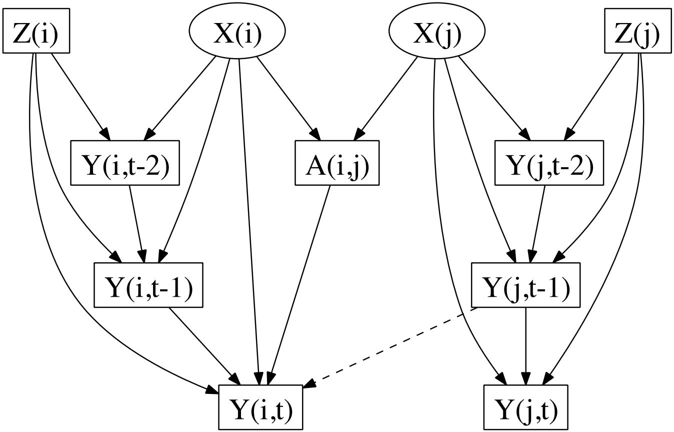
- $Y(j, t - 1)$ *may* have a direct influence on $Y(i, t)$, but only if $A(i, j) = 1$
- Homophily: $X(i)$ and $X(j)$ both directly influence $A(i, j)$


Z(i)
X(i)
X(j)
Z(j)
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A(i,j)
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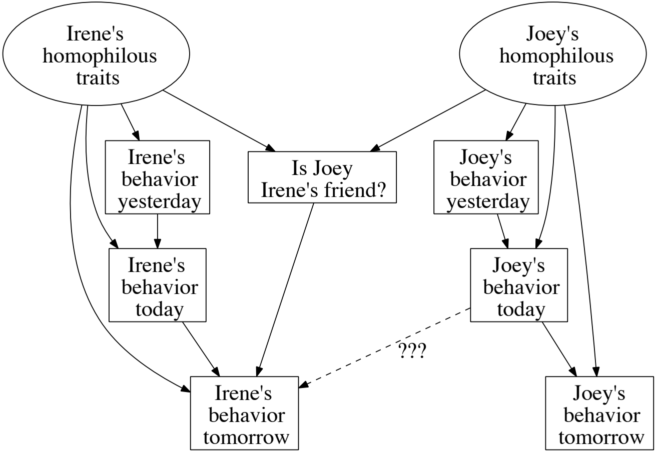
Irene's homophilous traits
Joey's homophilous traits
Irene's behavior yesterday
Is Joey Irene's friend?
Joey's behavior yesterday
Irene's behavior today
Joey's behavior today
???
Irene's behavior tomorrow
Joey's behavior tomorrow

Contagion Effects are Nonparametrically Unidentifiable

Informally:

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3. Irene's traits have information about Irene's behavior today
4. ∴ Joey's behavior yesterday predicts Irene's behavior today

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3. Irene's traits have information about Irene's behavior today
4. $\therefore$ Joey's behavior yesterday predicts Irene's behavior today *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)

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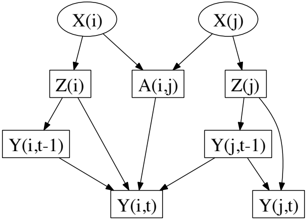
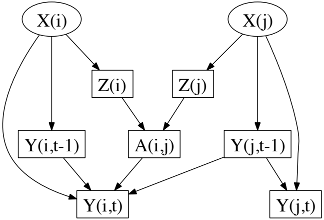
Time-varying edges don't help (more spaghetti; cf. Noel and Nyhan (2010))

Getting Identifiability

Parametric assumptions *might* 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, $i \rightarrow j$, $j \nrightarrow i$

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Doesn't this argue for direct influence?

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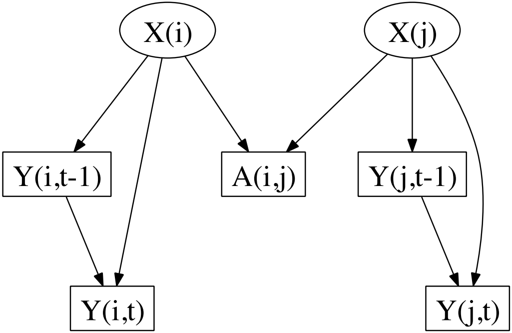
...fails if senders and receivers have systematically different values of X, with different local relations to Y

Toy Example

Try to predict $Y(i,t)$ from $Y(j,t)$ and vice versa when
$A_{ij} = 1, A_{ji} = 0$
$X(i) \sim \mathcal{U}(0,1)$
Edges form with probability $\propto \mathrm{logit}^{-1}(-3|X(i) - X(j)|)$
i nominates j from among neighbors, $\propto \mathrm{logit}^{-1}(-|X(j) - 0.5|)$

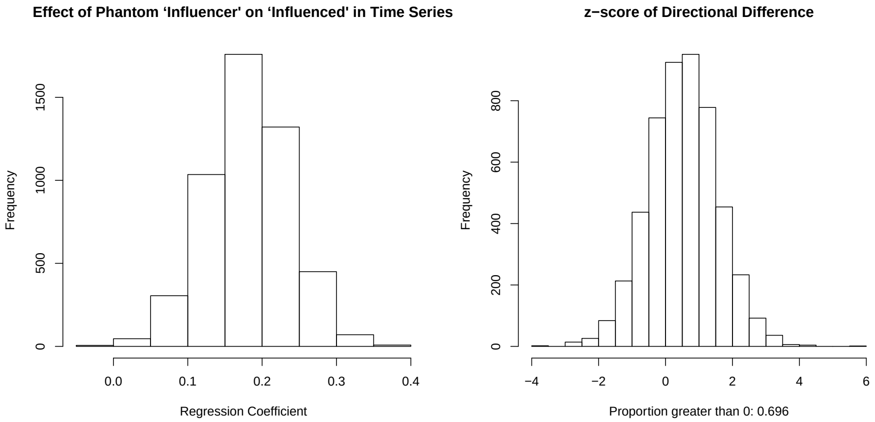
$$
\begin{aligned}
Y(i,0) &= (X(i) - 0.5)^3 + \mathcal{N}(0,(0.02)^2) \\
Y(i,1) &= Y(i,0) + 0.3X_i + \mathcal{N}(0,(0.02)^2)
\end{aligned}
$$



Causal graph of the model with no contagion, but asymmetry in regression coefficients

Results:

- $Y(i, 1)$ is well-predicted from $Y(j, 0)$
- *Nominees* are disproportionately in the middle; $i \rightarrow j, j \not\rightarrow i$ suggests i is more peripheral
- For asymmetric pairs, regression of sender on receiver differs from that of receiver on sender


Effect of Phantom 'Influencer' on 'Influenced' in Time Series
Frequency
0
500
1000
1500
0.0
0.1
0.2
0.3
0.4
Regression Coefficient
z−score of Directional Difference
Frequency
0
200
400
600
800
−4
−2
0
2
4
6
Proportion greater than 0: 0.696

Making homophily and contagion look like causation

A central theme of social science:
Long-term, hard-to-change social/economic status variables explain short-term, malleable cultural / political / consumer variables
Culture and choices express (reflect, serve, ...) social/economic interests or experiences

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

Quantitatively: use differences in demographics to predict differences in wedding gowns (or survey answers)

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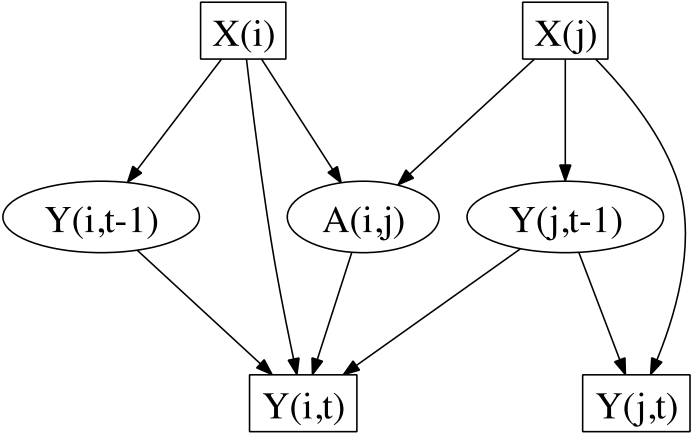
BUT usually ignores social networks and just looks at surveys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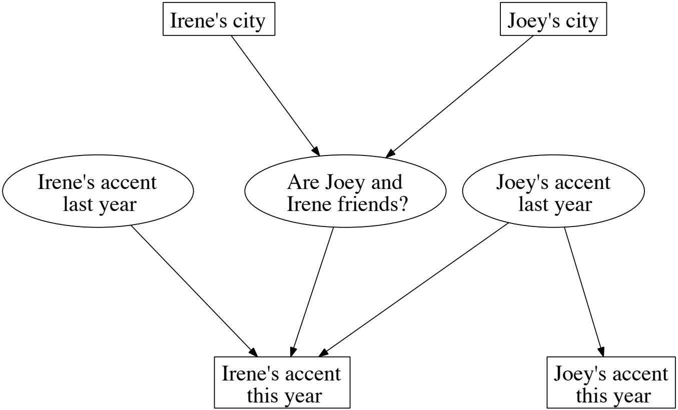
Irene's city
Joey's city
Irene's accent last year
Are Joey and Irene friends?
Joey's accent last year
Irene's accent this year
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4. $\therefore X(i) \not\perp Y(i,t)$ even if no direct influence

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

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- Work out expected behavior of this model
- Data departing from neutral model $\Rightarrow$ evidence of adapation

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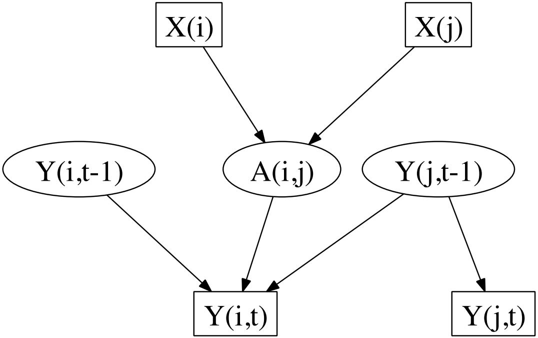
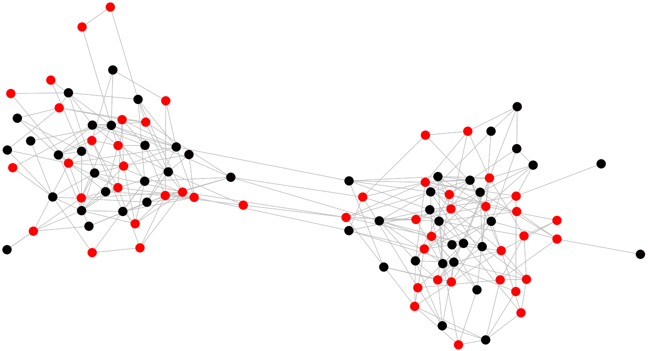
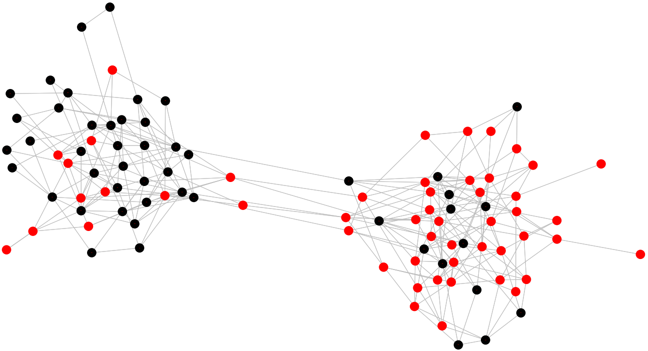
(= "voter model" of statistical mechanics)



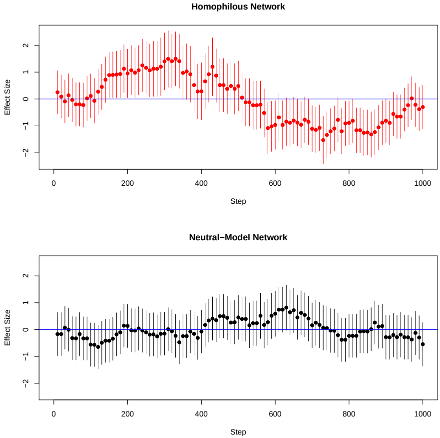
Graph for the voter model of neutral cultural evolution



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



After 300 updates



Coefficients for logistic regression of choice on status, $\pm$ 95% confidence intervals. Red, homophilous network; black, matched non-assortative network

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- Deconfound by conditioning on previous Y_j of neighbors

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Could we bound contagion effects when there is latent homophily?

Work in progress, only negative results so far

Actual contagion effect cannot be bounded from regression coefficient even in linear Gaussian model

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When does that hold?

What about other block models?

Tighter bounds, even if not identified?

Conclusion

1. Social linkage creates causal confounding
2. Homophily + causal influence looks like contagion
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4. *May* be possible to *limit* confounding

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