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Comment on “Why and When 'Flawed' Social Network Analyses Still Yield Valid Tests of no Contagion”

Cosma Rohilla Shalizi

Abstract

VanderWeele et al.'s paper is a useful contribution to the on-going scientific conversation about the detection of contagion from purely observational data. It is especially helpful as a corrective to some of the more extreme statements of Lyons (2011). Unfortunately, this paper, too, goes too far in some places, and so needs some correction itself.

KEYWORDS: social networks, causal inference, contagion, social influence

The paper by VanderWeele *et al.* is a useful contribution to the on-going scientific conversation about the detection of contagion from purely observational data. It is especially helpful as a corrective to some of the more extreme statements of Lyons (2011). Unfortunately, this paper, too, goes too far in some places, and so needs some correction itself.

To begin with, Lyons was so unrelentingly hostile in his paper, from the title onwards, that it's quite natural and even laudable to want to defend the objects of his attack. That said, look at exactly what is being offered here as a defense. There is no disputing Lyons's claim that the Christakis and Fowler (2007) model¹ is, in the strictest mathematical sense, simply meaningless, *unless* there is no contagion. The present paper says that estimating this nonsensical model allows one to test, not a clean hypothesis of no contagion, but rather a joint hypothesis of no contagion *and* no latent homophily *and* the complete correctness of the specification for the observed covariates.

Suppose I take my data and test this joint hypothesis with the model, and I reject it. It seems to me that there are two big obstacles in the way of saying that I have really tested the hypothesis of no contagion, rejected it, and so can infer contagion with some modicum of confidence.

1. The power of the test is quite unknown. But unless the test has power, it doesn't provide any *evidence* for an inference (Mayo, 1996, Mayo and Cox, 2006). More exactly, it provides no more evidence than what my teachers called a "Gygax test", which generates an independent random number between 0 and 1, and rejects if the number falls into an appropriately-sized interval². Specifically, one would need to know how much power the test had to detect departures from the null hypothesis in the direction of contagion. Since the model in question cannot, mathematically, be extended to allow for contagion, finding this power seems like a hard thing to do. I don't want to say it's impossible, but it is a pre-requisite for the test to have any scientific value.
2. Assuming the joint null hypothesis is rejected, how is one to know which component is at fault? Leaving latent homophily aside for the moment, in my experience of applied statistics I can recall exactly one case where a generalized linear model has actually passed even moderately severe mis-specification

¹I'll join everyone else in calling it their model, but, if I can decipher their somewhat obscure citations, they actually took it from Valente (2005), which gives the impression that it is common in both the network-epidemiology and diffusion-of-innovations literatures.

²If invoking an independent random number feels like cheating, substitute complicated calculations which are sensitive only to low-significance digits in continuous quantities.

checks³. (Perhaps the authors of the present paper have been luckier than me in this regard.) Unless some guidance can be given for *reliably* locating the problem in the hypothesis of no contagion, it's a stretch to say that this *tests* no contagion, as opposed to a model which posits that, along with a lot of *a priori* most dubious assumptions.

I am happy to agree with VanderWeele *et al.* that matters are better when one uses the model where ego's state at t is supposed to be caused by alter's state at time $t - 1$, and that then some of Lyons's criticisms lose their force. That model at least gives rise to a self-consistent stochastic process when there is contagion, so it is not *necessarily* wrong. One can even give it a coherent causal interpretation, unlike the simultaneous-regression model. (That is why we used the time-delayed model in Shalizi and Thomas (2011).) My point about power above is at least mitigated, since the power of the test proposed, assuming contagion but maintaining the other assumptions, could at be directly approximated by simulation. In all, I can think of no reason for ever using the simultaneous model.

This however still leaves the matter of what one learns from rejecting the joint null hypothesis. The issue of latent homophily returns here. VanderWeele (2011) is a truly ingenious paper, which advanced the field by providing the second approach⁴ to something like partial identification, as called for in Shalizi and Thomas (2011, §4.2). However, it did so under very strong parametric and substantive assumptions, such as, e.g., all latent homophily being due to a *single* binary variable, which interacts with observables in very specific and limiting ways. Proving results under these restrictions is more than anyone else has done, but before one appeals to the results in empirical problems, one needs to either have some scientific reason to think the restrictions hold, or a mathematical reason to think that the conclusions are robust to substantial departures from those assumptions. Since those mathematical reasons are, at least for now, unavailable, we are forced to rely on scientific knowledge. Is anyone prepared to argue that we ought, on biological or sociological grounds, to think that everything relevant to friendship formation and obesity (in suburban Massachusetts) boils down to one binary variable?

To sum up, there seem to me to be three major weaknesses with the argument of the present paper.

1. If the sensitivity analysis of VanderWeele (2011) is to be invoked, the assumptions underlying that analysis must be shown to apply.

³The exception involved a lot of work with the client to craft covariates which were highly nonlinear in the raw data.

⁴After Ver Steeg and Galstyan (2010), who however have to assume that the latent variables have the same relationship to observables at all times, i.e., that aging does not matter.

2. If rejecting the null hypothesis of no-contagion-and-no-latent-homophily-and-completely-correct-specification-of-everything-else is to provide evidence of *contagion*, then
 - (a) it must be shown that the test has power to detect departures from the null in the direction of contagion; and
 - (b) there really ought to be some guidance as to how one tells that the problem with the null is *contagion*, specifically.

I suspect that these weak points can be patched up, but they do need repair.

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