

ORIGINAL ARTICLE

Sensitivity analysis for network observations with applications to inferences of social influence effects

Ran Xu^{1,*}  and Kenneth A. Frank² 

¹Department of Allied Health Sciences, University of Connecticut, Storrs, CT 06269, USA and ²Department of Counseling, Educational Psychology and Special Education, Michigan State University, East Lansing, MI 48824, USA
(email: kenfrank@msu.edu)

*Corresponding author. Email: ran.2.xu@uconn.edu

Action Editor: Stanley Wasserman

Abstract

The validity of network observations is sometimes of concern in empirical studies, since observed networks are prone to error and may not represent the population of interest. This lack of validity is not just a result of random measurement error, but often due to systematic bias that can lead to the misinterpretation of actors' preferences of network selections. These issues in network observations could bias the estimation of common network models (such as those pertaining to influence and selection) and lead to erroneous statistical inferences. In this study, we proposed a simulation-based sensitivity analysis method that can evaluate the robustness of inferences made in social network analysis to six forms of selection mechanisms that can cause biases in network observations—random, homophily, anti-homophily, transitivity, reciprocity, and preferential attachment. We then applied this sensitivity analysis to test the robustness of inferences for social influence effects, and we derived two sets of analytical solutions that can account for biases in network observations due to random, homophily, and anti-homophily selection.

Keywords: sensitivity analysis; network observations; selection mechanisms; social influence

1. Introduction

Though much progress has been made in modeling social network data, the validity of the network observations is still relatively a blind spot in available methods for social network analysis (Steglich et al., 2010; Moffitt, 2001). Most or all currently available statistical analysis methods assume that network observations are perfectly accurate and fully representative, while we know that social network data are sometimes unreliable and prone to error, especially network relations (Marsden, 1990; 2005). And this lack of validity in network observations is not just a result of simple random measurement errors, but often due to systematic bias that can lead to the misinterpretation of actors' preferences for network selection. Furthermore, these biases in network observations can directly translate into an omitted variable problem, and as such are related to statistical inferences. As a consequence, these biases in observed networks could directly decrease the validity of a study and limit the inference we can draw from the data, such as those pertaining to social influence effects.

In this study, we employ a sensitivity analysis framework (Rosenbaum & Rubin, 1983; Frank et al., 2013; VanderWeele, 2011) for making inferences when there are potential biases in the network observations. First, we will discuss the sources of the biases in the network observations and how they relate to the misinterpretation of various selection mechanisms; next, we will introduce

the idea of simulation-based sensitivity analysis through the rewiring of observed network relations and consider different mechanisms for rewiring that represent some of the sources of biases; after that we will apply our proposed methods to quantify the robustness of inferences for social influence effects and give specific examples; finally, we will derive two sets of analytical solutions for sensitivity analysis of social influence effects under random, homophily and anti-homophily rewiring.

1.1 Bias in networks and misinterpretation of selection mechanisms

The validity of network observations is often of concern in empirical studies, since observed networks are prone to error and may not represent the population of interest. This misinterpretation can occur for various reasons as we described below.

1.1.1 Observation errors

While measurement errors exist in most social science data, the accuracy of observations in network relations is especially of concern (Marsden, 1990). The most common self-reported measures of network relations are known to suffer from cognitive bias (Freeman et al., 1987; Feld & Carter, 2002). For example, Freeman et al. (1987) show that peoples' perception of social ties will be biased toward the routine and frequent interactions. And other studies have shown that self-reported measures of network relations often are biased toward self, group structure, balance, routine interaction, etc. (Marsden, 2005). Studies by Bernard, Killworth, and Sailer (Killworth & Bernard, 1976; Bernard & Killworth, 1977; Bernard et al., 1981, 1982) showed that there are discernable differences between social ties data obtained via questionnaires and behavioral records obtained via various methods including diaries, monitoring of radio communication, observers, or electronic monitoring. Later studies have found a higher (80%) agreement between network questions in surveys and interviews (Pitts & Spillane, 2009). However, in general, observation errors in network relations are often a mixture of both random measurement errors and systematic bias that is driven by many known or unknown mechanisms in actors' preferences for network selection.

1.1.2 Mismatch between the frequency of interaction and functions of the network

Even when network observations are 100% accurate, the validity of the observations still depends on the functions of the network. For example in an information flow network, the frequency of interaction does not necessarily represent how much or how valuable certain information/resources are that flow through a tie, as weak ties are known to be more useful in terms of delivering novel information than strong ties (Granovetter et al., 1973). Furthermore, the observed frequency/importance of interaction is often not the same as that which actors actually perceive (Casciaro, 1998). For example, in the context of influence, actors may perceive more influence from those with whom they share similar interests but have less frequent interactions, compared with those with whom they share less similar interests but talk more often. This is because it is more likely that similar actors share the same identity and find it easier to talk to homophilous others, and in contrast, they find it more difficult and thus have to spend a longer time communicating with dissimilar others while actually conveying less information (Byrne, 1971; Mark, 1998; Carley, 1991). As a result, the frequency of interaction may not be the best representation of an actor's perception of the importance of their alters in terms of influence.

1.1.3 Sampling bias

The observed networks may not represent the population of interest. This can occur both on the network level and actors' level. On a network level, the observed network can be seen as one realization from a set of possible networks that are generated by a single underlying stochastic process

(Robins et al., 2007). It is possible that the one realization we observe does not represent the actual underlying preferences of actors in the network. At the actor level, if the actors in the study sample have preferences for network selection which are different than those in the population of interest, the observed network will be biased and will not represent the population network of interest as well.

Due to the potential sources of bias in network measures described above, by using the observed networks in an analysis, actors' actual preferences for interactions are often misinterpreted. And this misinterpretation may have a direct impact on issues related to both internal validity—whether the observed relationship is confounded by the unobserved mechanisms that drive network selection, and external validity—whether the observed network best represents the population of interest. Furthermore, these issues in observed network observations will likely bias the estimation of many common network models, such as those pertaining to influence and selection, which subsequently lead to erroneous inferences.

The purpose of this study is not to propose methods that can reduce the error in network observations or decrease the bias in the estimation process, rather, we follow a sensitivity analysis framework and investigate how bias in network observations can affect estimation and inferences from network models. Specifically, we propose that instead of stating that an inference drawn from the study is invalid because of unobserved bias in network relations, one should really ask how much bias, and what kind, must have occurred in the network data to invalidate an inference. We focus on sensitivity analyses of network relations for several reasons:

1. Recent work in sensitivity analysis recognizes the near impossibility of controlling for all potential confounders but find a productive scientific path forward by quantifying how much and in what ways the assumption of no omitted variables must be violated to invalidate an inference. The approach proposed in this paper follows this stream of research (Rosenbaum & Rubin, 1983; Frank, 2000; Pan & Frank, 2004; VanderWeele, 2011), as biases in network observations can essentially be framed as an omitted variable bias problem. However, it is unique in this context because we will consider how the omitted variable could change as a result of network selection, and, correspondingly, how strong the various mechanisms of selection must be to invalidate an inference.
2. Sensitivity analysis of the bias in network relations helps to frame external validity issues in social network studies. As network data usually contain the whole population of interest, external validity issues are rarely of concern. But as the observed network can also be treated as one realization from a set of possible networks that are generated by the same underlying stochastic process (Robins et al., 2007), a natural question to ask is to what extent the observed network can represent the underlying stochastic process. So, sensitivity analysis helps to express the validity of network observations into an external validity issue. While the sampling bias of observed networks can also be translated into a sample replacement problem (Frank et al., 2013), social network data usually have unique characteristics like non-random sampling and dependent observations (Manski, 1993; ?) that poses additional challenges for sensitivity analysis and calls for alternative methods that account for unique features of network relations.
3. The impact of some forms of observation errors/bias in network relations (missing data for example) on network outcomes has been studied (Robins et al., 2004; Kossinets, 2006), and relevant methods to detect, replace missing networks, as well as inferences from incomplete network have been developed (Guimera & Sales-Pardo, 2009; Handcock & Gile, 2010; Huisman, 2009). However, the impact of many other forms of errors/bias in network relations is rarely considered and largely unknown. Thus, we contribute to the literature by exploring various mechanisms that can generate observation errors in network relations and their impact on inferences through a sensitivity analysis framework.

2. Theoretical framework

Biases in the observed networks inevitably lead to misinterpretations of network selection mechanisms, and as a consequence, the estimation of many other processes is also biased (e.g. influence). Hence, a natural question to ask is “had the misinterpretations of the network selection been corrected, would the statistical inferences based on the corrected network still hold?” Our sensitivity analysis builds on this logic by constructing a network including counterfactual data that is generated by the “corrected” network selection mechanism and assesses if the inference still holds on the counterfactual network.¹ In particular, we calculate how different the network including the counterfactual data has to be from the observed network to invalidate an inference and use that as an indicator of the robustness of an inference. Specifically, we assess what percentage observed ties would have to be replaced by ties that are generated by a specific selection mechanism to invalidate the inference. For example, if one suspects that the true network is more homophilous than the observed network, one can assess to what extent observed ties would have to be replaced with more homophilous ties to invalidate the inference. Next, we formalize this approach by describing the replacement of ties in terms of a rewiring of the network, as well as the various mechanisms for rewiring.

2.1 Sensitivity analysis through rewiring networks

The sensitivity analysis we propose is a combination of analytical and simulation-based approaches that operate through the rewiring of a currently observed network. Our basic model assumes that actors control their outgoing ties (e.g. who they nominate as friends) and can rewire their ties based on various mechanisms given the observed network. The out-degree of each actor is kept as a constant, which assumes the bias/error in network relations lies in misreporting but not under-reporting or over-reporting. With these assumptions, we can express the magnitude of bias in terms of rewiring and assess the extent to which errors/bias in network structure can bias estimates and ultimately alter inferences. This approach allows us to ground analysis in theory and to test specific forms of bias existing in network relations as represented in actors’ preferences for network selection. This is different from previous research in which errors are assumed to be random, or ties are rewired at random (e.g. the Quadratic Assignment Procedure (QAP) test (Krackhardt, 1987)). Through this framework, we want to contribute to the discussion of validity in studies of social network analysis by focusing on the strength of evidence relative to theoretically plausible alternatives, which is the key to making a strong inference in any field of science (Platt, 1964). Essentially we are asking (1) “what percentage of network relations have to be rewired to invalidate an inference?” and (2) “what forms of bias must exist in network relations to invalidate a current inference?”²

2.2 Mechanisms for rewiring

While there are many potential mechanisms that drive an interaction between actors (thus also bias in observed networks), we follow a long tradition of “structure versus agency” (Emirbayer & Goodwin, 1994), or as Mayhew (1980) referred to as “structuralist” and “individualistic” views of the world. Structure, representing the social-organizational structure in which actors are embedded, limits the choices and opportunities available for network ties. Agency represents the capacity of actors to act freely, based on their own preferences and intentions. Studies have found that both can play an important part in shaping humans’ interactions, sometimes reinforcing each other (Kossinets & Watts, 2009). In our proposed methods, we include some widely studied factors from both views of the world. However, for the purpose of sensitivity analysis, we also want to separate each mechanism and explore how our inferences are sensitive to the specific form of bias resulting from each mechanism. Thus, in our sensitivity analysis, we only rewire an observed network based on one mechanism at a time. Next, we will introduce six mechanisms that can possibly bias network observations, and we categorize them into either agency or structure. Specifically for agency

we have random, homophily, and anti-homophily; for structure we interpret it as the network structure, in the form of reciprocity, transitivity, and preferential attachment.

2.2.1 Agency rewiring

For agency rewiring, first we have random rewiring. In this case, we assume that there are random measurement errors in an observed network. As is similar to the QAP test (Krackhardt, 1987), we rewire network ties randomly among nodes, but we preserve nodes' out-degree. In our application, the purpose is not to simulate the distribution of estimates in a random network, but to assess the extent to which an inference is robust to random errors in networks. Each time we rewire a certain percentage of observed ties randomly and re-estimate our model of interest. We repeat this many times to get an average estimate and compare with a pre-set threshold (e.g., associated with p value $< .05$) for inference. For example, if we assume 30% of our network observations are due to random error, we would randomly rewire 30% of observed ties. We do this repeatedly over a series of simulation rounds. Then we compare our average estimates with a pre-set threshold to decide if our inference is altered (for the analytic expressions this is analogous to taking an expectation).

Next we have homophily rewiring. Homophily, or "birds of a feather flock together," refers to a pervasive phenomenon that people tend to seek similar others for interaction (McPherson et al., 2001). It is an important network-generating mechanism that sometimes produces clustered networks or segregation (Schelling, 1971). Here particularly we focus on the agency of actors and refer to this type of behavior as a result of "choice homophily" as noted in Kossinets & Watts (2009), who attribute the choice of similar other as results of individual, psychological preferences. This is different from "induced homophily," where the choice of similar others is a consequence of the homogeneity of structural opportunities for interaction, as in neighborhoods, schools, workplaces, and friendship circles (Feld, 1981). Thus, in our model, as actors rewire their ties, they tend to choose other actors who are most similar to themselves without being subject to structural constraints. And here, homophily can be broadly defined to be based on various attributes available in the observed data. For example, in a study of social influence effects examining best friends' smoking behavior on actors' smoking behavior (An, 2015), if we suspect that networks of interest are more homophilous based on smoking behavior than observed, we can rewire a certain percentage of observed ties based on homophily. That is, we rewire a certain percentage of observed ties to connect actors with those of most similar smoking behavior who are not previously connected, re-estimate our model and compare average estimates with a pre-set threshold to decide if our inference is altered.

For agency, we also have anti-homophily rewiring. Given the importance of homophily, it would make sense to consider the opposite of homophily for both practical and theoretical reasons. Practically, given the predominant evidence that homophily exists in networks, a natural question to ask is "what if the observed network is too homophilous?," or how to account for errors/bias that result from processes counter to homophily. Theoretically, anti-homophily, or "heterophily" in a broader sense, reflects the tendency for people to interact with dissimilar or diverse others. People might choose to interact with non-homophilous others to seek new information or perspectives (Granovetter, 1973). There is agency in heterophily as heterophilous ties are mostly formed voluntarily (Rivera et al., 2010), and they are found to be more and more common over time in situations such as team building and scientific collaboration (Moody, 2004; Page, 2007). Thus, it would make sense to include anti-homophily as an alternative network-generating mechanism and possible source of bias. In our model, if we need to assess how our inference is robust to "anti-homophily," we would rewire a certain percentage of observed ties to connect actors with the most dissimilar attributes of concern, then re-estimate our model and compare average estimates with a pre-set threshold to decide if our inference is altered.

2.2.2 Structure rewiring

Next we turn to a set of structural mechanisms that account for errors/bias in networks. The first is transitivity, or “triadic closure,” which refers to the phenomenon that people tend to become friends with the friends of their friends (Rivera et al., 2010). This is found to be true across various social settings such as corporate board members (Davis et al., 2003), Hollywood movie actors (Watts, 1999), Broadway musical artists (Uzzi & Spiro, 2005), inventors (Fleming et al., 2007), and scientists (Newman, 2001b). There are various motivations for transitivity (increased encounter opportunities—Granovetter, 1973), decreased risk and uncertainty (Burt & Knez, 1995), and third-party introductions, with important implications for network structure. For example, Jin, Girvan and Newman (2001) found that if a pair has more mutual friends, they will have higher probability to meet, and the resulting network exhibits high levels of clustering and strong community structure.

Thus, we include transitivity as an important source of bias and rewire networks based on shared numbers of friends, updating a graph sequentially. For example, if we were to rewire certain observed ties based on transitivity, we create an order list for the ties to be rewired, then we rewire the first tie to the alter node who is not connected to the ego in the current graph but shares most common friends³ with ego. Then we update the graph and recalculate the network measures (the number of common friends shared by each pair in the updated graph), and do the same thing for the second tie to be rewired, and so forth. In this way, the order of movement matters since actors are more Markovian driven, and at the same time, it explores the whole space in terms of simulation results. As an illustration, consider Figure 1(a) as our original network, which is an unweighted random directed network without loops, where network size is 50 and density is 0.1. Figure 1(b) is one example of the resulting network if we rewire 100% of the ties based on transitivity, which contains many more triads and exhibits a community structure.

Reciprocity represents bi-directional connections (if i selects j , j will also select i) in a directed network. It has been found in friendship networks among students in various grade levels (Runger & Wasserman, 1980; Mollica et al., 2003). Possible reasons for the occurrence of reciprocity include people tending to like others who like them (Newcomb, 1956; Backman & Secord, 1959; Sprecher, 1998; Montoya & Insko, 2008) and reciprocation relative to a first advance of friendship (Goffman, 1963; Leifer, 1988). Reciprocity also has important implications for network structure such as stabilizing networks over time (Rivera et al., 2010). In our model, we include reciprocity as an alternative mechanism for rewiring, following similar sequential steps as in the transitivity case, except that actors will rewire their out-going ties to create more reciprocated ties. Figure 1(c) is one example of the network if we rewire 100% of the ties of the network in Figure 1(a) based on reciprocity, thus creating many more reciprocated relations. Figure 1(c) appears less dense than Figure 1(a) because the ties that are present are mostly reciprocated indicating two-way flows within pairs who interact, but fewer pairs who interact.

Finally, we consider preferential attachment. Preferential attachment states that social connections tend to accrue to those who already have them, also known as “rich get richer” or the “Matthew Effect” (Merton, 1968). Preferential attachment may be driven by people’s tendency to use others’ degree as a proxy for their own fitness, status, power, etc. (Zerubave et al., 2015). Empirical and simulation results suggest that preferential attachment can generate a core-periphery structure or power-law degree distribution in networks (Barabási & Albert, 1999; Newman, 2001), which is found in many settings such as online friendship networks, scientific collaborations, and sexual contact networks. (Golder et al., 2007; Moody, 2004; Newman, 2001; Liljeros et al., 2001). For the reasons above, we include preferential attachment as a possible mechanism for rewiring that accounts for errors/bias in networks. The steps we use to rewire are the same as for previous mechanisms, except that actors will now rewire to others who possess a higher in-degree for the current graph. Figure 1(d) is one example of the network if we rewire 100% of the ties of the network in Figure 1(a) based on preferential attachment, thus generating a clear core-periphery structure (Borgatti & Everett, 2000).⁴

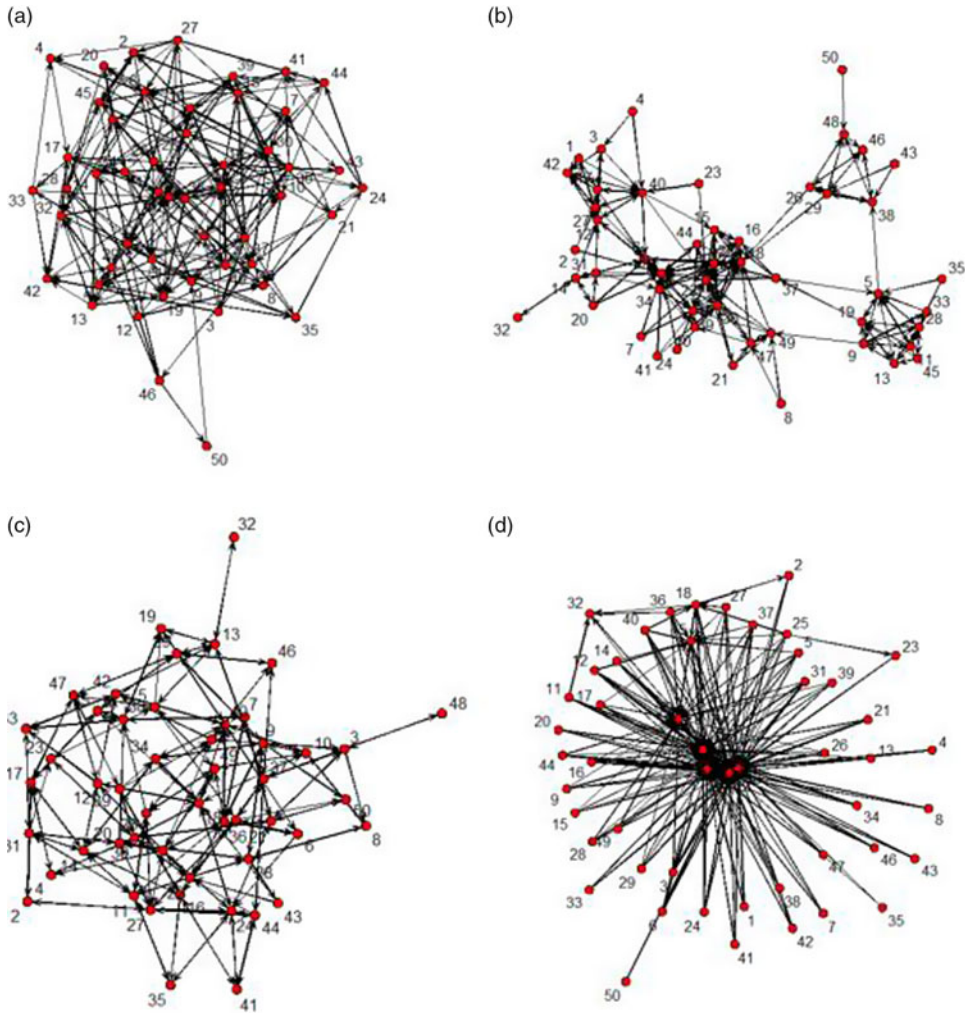


Figure 1. Structural rewiring example. (a) Original network. (b) 100% ties rewired based on transitivity. (c) 100% ties rewired based on reciprocity. (d) 100% ties rewired based on preferential attachment.

3. Applying sensitivity analysis to inferences for social influence effects

After establishing our sensitivity analysis method and various rewiring mechanisms, in this section we give some specific examples on how it can be applied to empirical data. Note that although the sensitivity analysis method we propose could potentially be applied to many different types of models, we are particularly interested in making inferences from social influence models for several reasons: (1) Social influence effects, which are defined as the propensity of an individual to behave in some way varying with the prevalence of that behavior in the network neighbors of the individual (Manski, 1993), have received considerable attention and have been widely studied (Kandel, 1978; Marsden & Friedkin, 1993; Doreian, 2001; An, 2011) as they have potential implications for various issues in different fields such as health behavior (e.g. obesity and smoking), information diffusion, and teacher practice changes, among others (Christakis & Fowler, 2007; 2008, Valente, 2005, 1996; Frank et al., 2004). (2) There are many challenges in identifying social influence effects, as they are often confounded with other unobserved variables (individual attributes, social-environmental factors, etc), especially homophily in the selection process

(Aral et al., 2009; Frank & Xu, 2020; Shalizi & Thomas, 2011). Many sophisticated statistical models have been developed to identify social influence effects (Christakis & Fowler, 2007; Steglich et al., 2010; An, 2011), including the more recent development in the latent space adjusted approach (Xu, 2018). However, in general, there is still debate about the validity of these methods and more research need to be done (VanderWeele & An, 2013; Lyons, 2011; Frank & Xu, 2020). Sensitivity analysis has been proposed as an alternative to deal with the impact of unobserved variables (Rosenbaum & Rubin, 1983; Frank, 2000 2004; VanderWeele, 2011). However, the validity of observed network ties has not been carefully considered. In this context, our proposed methods can contribute to questions such as how inference about social influence effects is robust to errors/bias in networks generated by various mechanisms (e.g., homophily). (3) While how network errors affect other outcomes (e.g., centrality) have been studied (Martin & Niemeyer, 2019), the robustness of social influence effect to network errors is less explored. As outcomes and identification of social influence effects are critically contingent upon the network structure or to whom individuals are exposed (Friedkin & Johnsen, 1999), it is vital to investigate how inference of social influence effects is robust to alternatives or possible errors in the measurement of network ties.⁵

3.1 Theoretical derivation of the direction of the bias due to various selection mechanisms

In this section, we formally frame the bias/error in network observations as an omitted variable problem and provide some algebraic intuition for the direction of the bias of the coefficient for social influence effects under different selection mechanisms.

Within a given social network, where node represents person and a directed tie from A to B represents A nominates B as a network partner (e.g. friendship nomination), we assume people are influenced by those that they nominate. One form of models that characterizes the influence process can be written as (assume predictors are centered about their grand mean so we ignore the constant term).

$$Y = \beta_1 Y_{t-1} + \beta_2 W_{t-1} Y_{t-1} + u_t \quad (1)$$

here Y represents an $n \times 1$ vector of the behavior of nodes, and W_{t-1} represents an $n \times n$ weighting matrix based on observed network ties that pertain to the relationships or ties that occur between $t - 1$ and t . The error term u_t is uncorrelated with $W_{t-1} Y_{t-1}$. For example, Y can represent teachers' teaching practices, and W represents the weighting matrix based on teachers' interactions with one another. We choose this form of linear model over other alternatives as they have greater flexibility to include covariates, and we only use lagged endogenous variables, which to us is a more realistic assumption that there is some lag in the transmission of influence effects. In addition, such formulations require less strict conditions for identification of social effects (Manski, 1993). It has been shown to be able to recover the true influence effect under reasonable assumptions (Frank & Xu, 2020).⁶ This influence model has its origin in Friedkin & Johnsen (1990) (see other discussions in Leenders, 2002; Valente, 2005; An, 2011). The model and its variation has been applied to study many different phenomena, such as health policy implementation (Valente et al., 2019), physician technology use (Zheng et al., 2010), new product diffusion (Iyengar et al., 2011), adolescent smoking status (An, 2015), and teachers' professional practices (Frank et al., 2013; Penuel et al., 2002; Sun et al., 2013).

For the ease of notation, assume that W does not change over time, and there is bias/error in network observations of teachers' interactions so that the true network is

$$\tilde{W} = W + E \quad (2)$$

where we represent the network change based on unobserved mechanism as E . So that

$$Y_t = \beta_1 Y_{t-1} + \beta_2 \tilde{W} Y_{t-1} + u - \beta_2 E Y_{t-1} \quad (3)$$

where $u - \beta_2 EY_{t-1}$ is the new unobserved error term. Next, we estimate this new model and derive the bias of β_2 .

For ease of notation, let $\tilde{Y} = \tilde{W}Y_{t-1}$. Using the Frisch–Waugh–Lovell theorem, we can get

$$\hat{\beta}_2 = (\tilde{Y}'M_{Y_{t-1}}\tilde{Y})^{-1}\tilde{Y}'M_{Y_{t-1}}Y_t = \beta_2 + (\tilde{Y}'M_{Y_{t-1}}\tilde{Y})^{-1}\tilde{Y}'M_{Y_{t-1}}(u - \beta_2 EY_{t-1}) \tag{4}$$

where $M_{Y_{t-1}} = I - Y_{t-1}(Y'_{t-1}Y_{t-1})^{-1}Y'_{t-1}$. Define bias as $\hat{\beta}_2 - \beta_2$, since u is uncorrelated with all the independent variables, substituting M into equation above we have

$$bias = \hat{\beta}_2 - \beta_2 = -\beta_2(\tilde{Y}'M_{Y_{t-1}}\tilde{Y})^{-1}\tilde{Y}'EY_{t-1} + (\tilde{Y}'M_{Y_{t-1}}\tilde{Y})^{-1}\tilde{Y}'Y_{t-1}(Y'_{t-1}Y_{t-1})^{-1}Y'_{t-1}EY_{t-1} \tag{5}$$

This result has several implications:

- (1) For random rewiring in networks,

$$bias = \hat{\beta}_2 - \beta_2 = -\beta_2(\tilde{Y}'M_{Y_{t-1}}\tilde{Y})^{-1}\tilde{Y}'EY_{t-1} \tag{6}$$

as Y_{t-1} and EY_{t-1} are uncorrelated. And as $\tilde{Y} = \tilde{W}Y_{t-1} = (W + E)Y_{t-1}$, which is positively correlated with EY_{t-1} , so bias < 0 if $\beta_2 > 0$, bias > 0 if $\beta_2 < 0$. This means with random rewiring $\hat{\beta}_2$ will be biased toward 0 when compared with β_2 .

- (2) For homophily rewiring, Y_{t-1} and EY_{t-1} will be positively correlated for $\beta_2 > 0$, so the second part of the bias term is larger than 0. As a result, intuitively for the same % of ties rewired as the random case, $\hat{\beta}_2$ under homophily rewiring should have a smaller attenuation than $\hat{\beta}_2$ under random rewiring.
- (3) For anti-homophily rewiring, Y_{t-1} and EY_{t-1} will be negatively correlated for $\beta_2 > 0$, so the second part of the bias term is smaller than 0. As a result, intuitively for the same % of ties rewired, $\hat{\beta}_2$ under anti-homophily rewiring should have a larger attenuation than $\hat{\beta}_2$ under random rewiring.
- (4) For other structure rewiring, the direction of bias is generally unknown.

Thus, we frame the bias/error in network observations as an omitted variable problem and show the direction of the bias under different selection mechanisms. Next we illustrate this by performing our sensitivity analysis on a simulated dataset.

3.2 Simulation example: applying sensitivity analysis to quantify the robustness of an inference for social influence effects

Next, we use a simulated dataset to illustrate how to apply our proposed methods to empirical data to test the robustness of an inference about social influence effects to various errors/bias in networks. (i) We start by estimating a lagged linear-in-mean social influence model and acquire model estimates. (ii) Next, we calculate thresholds to alter our inference for each parameter, using the sensitivity analysis method described in Frank et al. (2013). (iii) Assuming that there are errors in the observed networks, we then rewire the observed networks based on various mechanisms (random, homophily, anti-homophily, transitivity, reciprocity, and preferential attachment, respectively) repeatedly and re-estimate the influence model to obtain new estimates for each parameter. (iv) Finally, we compare the average of the new estimates in step (iii) with the thresholds set in step (ii) to decide what percentage of networks (and under what mechanisms) need to be rewired to invalidate our inference.

Specifically, we construct a simulated network dataset using a random unweighted directed network without self-loops, where network size is 50 and density is 0.2. This allows us to see how influence estimates change with various rewiring mechanisms over the full spectrum of

Table 1. Regression results from the influence model example

	Estimates	Standard error	t-value	Pr
$\hat{\beta}_1$	0.41834	0.07563	5.532	1.37e-06***
$\hat{\beta}_2$	0.83638	0.28675	2.917	0.00541**

p < .01; *p < .001.

network change (i.e. 0% to 100%). We used a linear-in-mean variation of model (1) to represent the influence process:

$$y_{it} = \beta_0 + \beta_1 y_{it-1} + \beta_2 \frac{\sum w_{ijt-1} y_{jt-1}}{\sum w_{ijt-1}} + e_{it} \quad (7)$$

where y_{it} represents the behavioral outcome of interest for person i at time t , w_{ijt-1} is a binary variable representing whether person i has a network relationship with person j at time $t-1$ (1 if yes and 0 otherwise), and e_{it} is an error term following a normal distribution with mean 0 and standard deviation of 0.2. Together, $\frac{\sum w_{ijt-1} y_{jt-1}}{\sum w_{ijt-1}}$ represents the average behavior of person i 's network partners. For example, this model could characterize how teacher's teaching practice is influenced by other teachers he/she has interaction with. To identify social influence effects, we simulated the influence process over two time points and estimated the influence model as in (7); estimated parameters are shown in Table 1. The ratio between the estimate of the prior term (β_1) and the estimate of the network exposure term (β_2) is comparable to what have been reported in several empirical studies, including one that studies how teachers' computer uses are influenced by colleagues (Frank et al., 2011). Furthermore, we calculate the thresholds to alter the inference for each parameter, following Frank et al. (2013).

To explore how our estimates are robust to various errors in the observed network, we then rewired a different percentage of existing ties (varying from 10 to 90%) based on the following:

- (1) Random selection: For each tie that is randomly chosen to be rewired, the new tie is chosen randomly from the unestablished ties;
- (2) Homophily: For each tie that is randomly chosen to be rewired, the new tie is chosen from the unestablished ties with the smallest value of $|y_{it-1} - y_{jt-1}|$;⁷
- (3) Anti-homophily: For each tie that is randomly chosen to be rewired, the new tie is chosen from the unestablished ties with the largest value of $|y_{it-1} - y_{jt-1}|$;
- (4) Transitivity: For each tie that is randomly chosen to be rewired, the new tie is chosen from others whom the actor is not previously connected to but share most common friends with;
- (5) Reciprocity: For each tie that is randomly chosen to be rewired, the new tie is chosen such that the connections are mutual;
- (6) Preferential attachment: For each tie that is randomly chosen to be rewired, the new tie is chosen such that the higher an actor's existing in-degree, the more likely the actor is to be chosen to replace a previous tie.

Note that in each case existing ties to be rewired are selected randomly and actors preserve their out-degree. For each configuration, we simulate 500 times and re-estimate model (7) after each simulation, and for each simulation, we record the estimates for β_1 , β_2 and correlation between the prior term and the network exposure term. And we compare the mean estimates with the pre-set threshold to determine if our inference should be invalidated.

Results are shown in Figure 2. Figures 2(a) and 2(b) plots the average estimates of prior and network exposure vs % of ties rewired, respectively. Black lines in each graph represent the threshold to alter the inference (as defined by statistical significance for $\alpha = .05$), which is calculated as in Frank et al. (2013). Figure 2(a) shows that the estimates of the prior are generally not influenced by various mechanisms of rewiring, except when rewiring by homophily or anti-homophily.

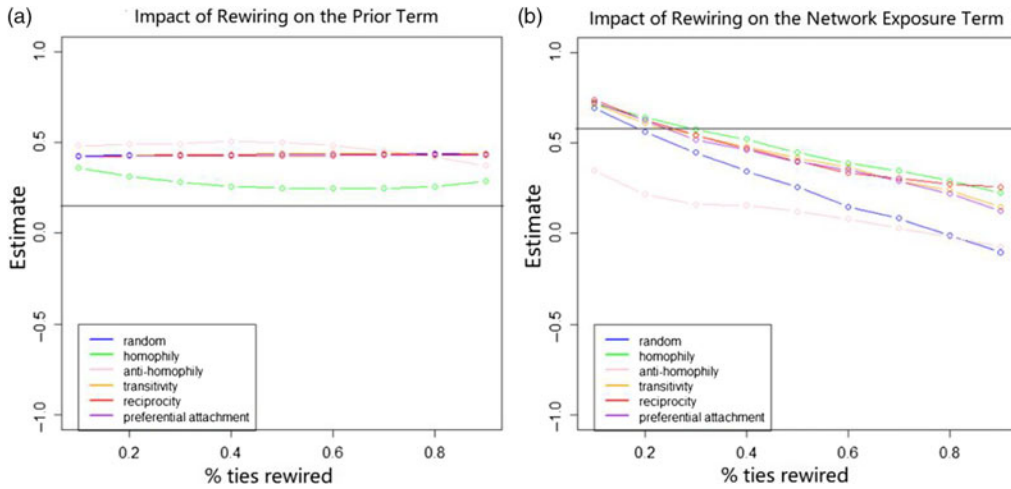


Figure 2. Impact of rewiring on the estimates.

Nevertheless, estimates of the prior are all above the threshold, and the inference is robust to all mechanisms of rewiring.

Estimates of network exposure effects exhibit more interesting patterns. Figure 2(b) shows that 20% of the ties need to be rewired randomly to invalidate the inference of a social influence effect. And as expected, estimates for the social influence effects suffer from a larger attenuation under anti-homophily rewiring; the inference is invalidated even if only 10% of ties are rewired. There is a smaller attenuation of the estimates for the social influence effect under homophily rewiring with 30% of the ties needing to be rewired to invalidate the inference. For other structural types of rewiring (transitivity, reciprocity, preferential attachment), around 20–30% of the ties need to be rewired to invalidate the inference, and the attenuation is smaller than random rewiring but larger than homophily rewiring. Note that a calculation from Frank et al. (2013) indicates that 31% of the estimates of social influence effects have to be due to bias in order to invalidate the inference. Thus, in this example, social influence effects are less robust to errors/bias in networks than simply replacing cases with null hypothesis where the influence effect is assumed to be 0.

4. Analytic solutions for sensitivity analysis of the social influence effects

While these simulation-based sensitivity analysis methods are intuitive and easy to implement, they still have several limitations: (1) Sometimes these simulation-based methods are computationally expensive, especially when the network size is large or when there are many actors' characteristics/variables of interest; (2) to fully understand the behavior/performance of these sensitivity analysis methods under various conditions, it would be helpful if we could derive some sort of closed form/analytic solutions, such as solutions that can be represented as functions of observed networks and correlations between variables.

Thus, in this section, we develop analytical solutions for our sensitivity analysis of the social influence effects based on three rewiring mechanisms, namely, random rewiring, homophily rewiring, and anti-homophily rewiring.

For simplicity, we assume that there are only three variables in our influence model as in (7): dependent variable Y , a prior term Z , and a network exposure term X (although it is possible to extend this analysis to models with more covariates). And the key relationship of interest is the relationship between network exposure X and dependent variable Y .

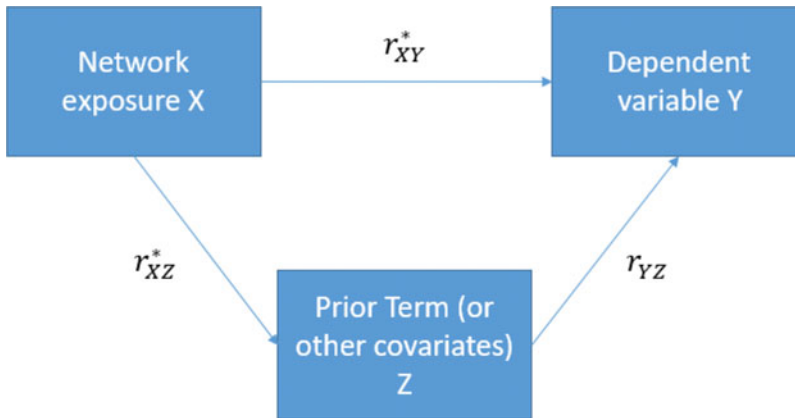


Figure 3. Analytical solution framework.

To determine the impact of different rewiring mechanisms on the inferences about network influence, we follow a partial correlation framework as in Frank (2000). The robustness of an inference is essentially decided by the partial correlation between the dependent variable Y and the network exposure term X , conditional on other covariates, such as $r_{XY|Z}$, as shown in Figure 3. By rewiring the network relations, we are only creating a new exposure term X without changing the correlation between the dependent variable Y and the prior term Z , given by r_{YZ} . As a result, we only need to consider how rewiring changes the correlation r_{XY} between the network exposure and the dependent variable, and r_{XZ} , the correlation between network exposure and prior term (or other covariates), and how these new correlations generate the new partial correlation $r_{XY|Z}^*$.

In this section, we will derive two sets of analytical solutions. The first set is called “node-level rewiring,” where for $(1-p)^*$ 100% of ties rewired, we assume $(1-p)^*$ 100% of nodes rewire all of their ties. The second set is called “tie-level rewiring,” where for $(1-p)^*$ 100% of ties rewired we assume ties rewired are distributed evenly across all nodes. Note that the first set is more intuitive, and the second set is more technically challenging, but the operations of our simulations are in principle more similar to the second set of analytical solutions. And as we will show later, the analytical solutions for tie-level rewiring will have a better fit to raw correlations r_{XY} , r_{XZ} after rewiring, but in terms of goodness of fit to partial correlations between network exposure and the dependent variable $r_{XY|Z}$ (which is the sufficient statistic determining the robustness of inference), the two sets of analytical solutions perform equally well.

Finally, note that in this study, we have not derived analytical solutions for structural rewiring such as transitivity rewiring, preferential attachment rewiring, or reciprocity rewiring. This is because in these mechanisms we need to know the full network and exact distributions of the variables of interest (rather than their correlations) to calculate the new partial correlations. We leave this topic for future work.

The rest of the section is organized as follows: first, we derive the analytical solutions for node-level rewiring where we assume a certain percentage of actors rewires all their ties while others rewire none. Next we derive the analytical solution for tie-level rewiring where we assume ties rewired on average are evenly distributed across all actors. Finally, we will give some simulated examples to examine how well the two derived analytical solutions fit the simulation results.

4.1 Analytical solution for node-level rewiring

In the first set of analytical solutions, we assume only that certain actors in the network have errors in their networks while other actors’ network is perfectly measured. For example, in a network with evenly distributed degrees, if there are $(1-p)$ 100% of the observed ties that need to

be rewired due to errors/biases, we assume that there is zero accuracy in observed networks for $(1 - p)100\%$ of the actors, all of whom have to rewire all their networks. And for the remaining $p*100\%$ actors, their networks are perfectly measured and are 100% accurate. In empirical cases, this may not be realistic, unless some actors identify the wrong primary social group, misread the network question, or deliberately sabotage the study by reporting all their network relations incorrectly. However, for the derivation of an analytical solution, this is more intuitive and analytically accessible. In addition, the imperfect approximation of r_{XY} and r_{XZ} might cancel out in the calculation of partial correlation, and it is possible that we can still achieve approximately consistent estimates of the partial correlation, which is the sufficient statistic for statistical inference. Thus, we derive analytical solutions for random, homophily, and anti-homophily rewiring under this assumption.

Random rewiring. Assume that we randomly rewire $(1 - p)*100\%$ of ties in the observed network, the variance of network exposure will not change, and the new raw correlation between the network exposure X and a dependent variable Y after rewiring $-r_{XY}^*$ would become (assume all variables are grand mean centered)

$$r_{XY}^* = \frac{p * \frac{1}{n} \sum XY}{\sqrt{\text{var}(X)\text{var}(Y)}} + (1 - p)H_0 = pr_{XY} + (1 - p)*0 = pr_{XY} \tag{8}$$

Here, H_0 is the hypothesized correlation between the exposure after rewiring and Y . For the rewired ties, the new correlation is zero from random rewiring. For $(1 - p)*100\%$ actors their r_{XY} has changed to 0, while for $p*100\%$ actors who have not rewired their ties, their r_{XY} remain unchanged. The new correlation becomes a weighted average between r_{XY} and 0.

For a similar reason, the new raw correlation between the network exposure X and the prior term Z after rewiring $-r_{XZ}^*$ would become a weighted average between r_{XZ} and 0:

$$r_{XZ}^* = \frac{p * \frac{1}{n} \sum XZ}{\sqrt{\text{var}(X)\text{var}(Z)}} + (1 - p)H_0 = pr_{XZ} + (1 - p)*0 = pr_{XZ} \tag{9}$$

After deriving the two new raw correlations after rewiring, we now derive the key partial correlation $r_{XY|Z}^*$ after rewiring (Z represents the prior here, which can also include other covariates) have the form:

$$r_{XY|Z}^* = \frac{r_{XY}^* - r_{XZ}^*r_{YZ}}{\sqrt{1 - r_{XZ}^{*2}}\sqrt{1 - r_{YZ}^2}} = \frac{pr_{XY} - pr_{XZ}r_{YZ}}{\sqrt{1 - p^2r_{XZ}^2}\sqrt{1 - r_{YZ}^2}} \tag{10}$$

Note r_{YZ} is unchanged as rewiring does not change the correlation between dependent variable and the prior/covariate. As this shows, we can now represent the new partial correlation in terms of p (the percentage of ties retained) and the original correlations in the observed data.

To understand the robustness of inference, we need to know one more thing—the threshold of inference. In this case, the threshold of partial correlation $r_{XY|Z} - r^\#$ (based on statistical significance) can be calculated as

$$r^\# = \frac{t^\#}{\sqrt{t^{\#2} + \text{res.df}}} \tag{11}$$

where $t^\#$ is the critical value of t used to make an inference, and res.df represents the residual degrees of freedom.

Thus to invalidate the observed inference, we need to randomly rewire $(1 - p)\%$ of ties in order to get

$$r_{XY|Z}^* = \frac{pr_{XY} - pr_{XZ}r_{YZ}}{\sqrt{1 - p^2r_{XZ}^2}\sqrt{1 - r_{YZ}^2}} = r^\# \tag{12}$$

To write p as function of other variables, we have

$$p = \frac{r^\# \sqrt{1 - r_{YZ}^2}}{\sqrt{r^{\#2} r_{XZ}^2 (1 - r_{YZ}^2) + (r_{XY} - r_{YZ} r_{XZ})^2}} \tag{13}$$

Homophily rewiring. Next we derive an analytical solution for homophily rewiring. Assume that homophily is based on the prior (this can also extend to homophily based on a prior and other covariates), and for ties, that rewired people will rewire to others who engage in exactly the same behavior. The underlying assumption here is that the network is big and diverse enough so that everyone can find others who engage in exactly the same behavior.

Then for a large network where everyone can find perfectly homophilous others based on the prior, to rewire $(1 - p)^*100\%$ of ties, we assume $(1 - p)^*100\%$ of nodes rewire all their ties (so that on average $(1 - p)^*100\%$ of the total ties are rewired), and the new raw correlation between network exposure X and the prior term Z after rewiring $-r_{XZ}^*$ becomes a weighted average between r_{XZ} and 1.

$$\begin{aligned} r_{XZ}^* &= \frac{\frac{1}{n} \left(\sum_1^{np} XZ + \sum_1^{n(1-p)} X'Z \right)}{\sqrt{\text{var}(X)\text{var}(Z)}} = \frac{\frac{p}{np} \sum_1^{np} XZ + \frac{1-p}{n(1-p)} \sum_1^{n(1-p)} X'Z}{\sqrt{\text{var}(X)\text{var}(Z)}} \\ &= pr_{XZ} + (1 - p)^*H_0 = pr_{XZ} + (1 - p)^*1 \end{aligned} \tag{14}$$

Here, X' represents the new network exposure after rewiring, and for the $(1 - p)^*100\%$ of nodes who rewire to perfectly homophilous others based on the prior, their new correlation between network exposure and prior term, as stated in H_0 , should be 1.

For a similar reason, the new raw correlation between network exposure X and the dependent variable Y after rewiring $-r_{XY}^*$ would become a weighted average between r_{XY} and r_{YZ} .

$$r_{XY}^* = \frac{\frac{1}{n} \left(\sum_1^{np} XY + \sum_1^{n(1-p)} X'Y \right)}{\sqrt{\text{var}(X)\text{var}(Y)}} = \frac{\frac{p}{np} \sum_1^{np} XY + \frac{1-p}{n(1-p)} \sum_1^{n(1-p)} YZ}{\sqrt{\text{var}(X)\text{var}(Y)}} = pr_{XY} + (1 - p)^*r_{YZ} \tag{15}$$

Note that here for the $(1 - p)^*100\%$ of nodes who rewire all their ties to perfectly homophilous others, their network exposure becomes exactly the same in their prior term. As a result, for these people, the new correlation between network exposure X and the dependent variable Y after rewiring becomes the correlation between the prior term Z and the dependent variable $Y - r_{YZ}$.

After deriving the two new raw correlations after rewiring, we now derive the key partial correlation $r_{XY|Z}^*$ after rewiring, which has the form:

$$r_{XY|Z}^* = \frac{r_{XY}^* - r_{XZ}^* r_{YZ}}{\sqrt{1 - r_{XZ}^{*2}} \sqrt{1 - r_{YZ}^2}} = \frac{(pr_{XY} + (1 - p)^*r_{YZ}) - (pr_{XZ} + (1 - p))^*r_{YZ}}{\sqrt{1 - (pr_{XZ} + (1 - p))^2} \sqrt{1 - r_{YZ}^2}} \tag{16}$$

As this shows, we can now represent the new partial correlation in terms of p (the percentage of ties retained) and the correlations in the observed data.

Using $r^\#$ as defined in (11), to invalidate the observed inference, we need to rewire $(1 - p)^*100\%$ of ties based on homophily in order to get

$$r_{XY|Z}^* = \frac{(pr_{XY} + (1 - p)^*r_{YZ}) - (pr_{XZ} + (1 - p))^*r_{YZ}}{\sqrt{1 - (pr_{XZ} + (1 - p))^2} \sqrt{1 - r_{YZ}^2}} = r^\# \tag{17}$$

To write p as function of other variables, we have

$$p = \frac{2(r_{XZ} - 1)(r_{YZ}^2 - 1)r^{\#2}}{(r_{XY} - r_{XZ}r_{YZ})^2 - r_{XZ}^2r_{YZ}^2r^{\#2} + r_{XZ}^2r^{\#2} + 2r_{XZ}r_{YZ}^2r^{\#2} - 2r_{XZ}r^{\#2} - r_{YZ}^2r^{\#2} + r^{\#2}} \tag{18}$$

Anti-homophily rewiring. Next, we derive an analytical solution for anti-homophily rewiring. Assume that anti-homophily is based on the prior (this can also be extended to other covariates), and for ties, that rewired people will rewire to others who hold the most dissimilar behavior/belief.

As before, to rewire $(1 - p)^*$ 100% of ties, we assume that $(1 - p)^*$ 100% of nodes rewire all their ties (so that on average $(1 - p)^*$ 100% of the total ties are rewired), and the new raw correlation between network exposure X and the prior term Z after rewiring $-r_{XZ}^*$ becomes a weighted average between r_{XZ} and -1 :

$$r_{XZ}^* = \frac{\frac{1}{n} \left(\sum_1^{np} XZ + \sum_1^{n(1-p)} X'Z \right)}{\sqrt{\text{var}(X)\text{var}(Z)}} = \frac{\frac{p}{np} \sum_1^{np} XZ + \frac{1-p}{n(1-p)} \sum_1^{n(1-p)} X'Z}{\sqrt{\text{var}(X)\text{var}(Z)}} = pr_{XZ} + (1 - p)H_0 \approx pr_{XZ} - (1 - p) \tag{19}$$

x' represents the new network exposure after rewiring and for the $(1 - p)^*$ 100% of nodes who rewire to the most dissimilar others based on the prior, and their new correlation between network exposure and the prior term, as stated in H_0 , should approximately be -1 . Note that for each different distribution of the prior term, the correlation r_{xz}^* after rewiring will be different. Assuming prior is a binary variable (e.g. whether actors smoke or not), and when all actors connect to others who have different behavior to themselves, the correlation between network exposure and the prior would be -1 .

For a similar reason, the new raw correlation between network exposure X and the dependent variable Y after rewiring $-r_{XY}^*$ would become a weighted average between r_{XY} and $-r_{YZ}$:

$$r_{XY}^* = \frac{\frac{1}{n} \left(\sum_1^{np} XY + \sum_1^{n(1-p)} X'Y' \right)}{\sqrt{\text{var}(X)\text{var}(Y)}} = \frac{\frac{p}{np} \sum_1^{np} XY + \frac{1-p}{n(1-p)} \sum_1^{n(1-p)} (-Z)Y'}{\sqrt{\text{var}(X)\text{var}(Y)}} = pr_{XY} - (1 - p)r_{YZ} \tag{20}$$

Note that here for the $(1 - p)^*$ 100% of nodes who rewire all their ties to the most dissimilar others, their new network exposure term and their prior term have a perfect negative correlation of -1 . As a result, for these people, the new correlation between network exposure X and the dependent variable Y after rewiring becomes $-r_{YZ}$ and the correlation between the prior term Z and the dependent variable Y with a negative sign.

After deriving the two new raw correlations after rewiring we now derive the key partial correlation $r_{XY|Z}^*$ after rewiring, which has the form:

$$r_{XY|Z}^* = \frac{(pr_{XY} - (1 - p)r_{YZ}) - (pr_{XZ} - (1 - p))^*r_{YZ}}{\sqrt{1 - (pr_{XZ} - (1 - p))^2}\sqrt{1 - r_{YZ}^2}} \tag{21}$$

As this shows, we can now represent the new partial correlation in terms of p (the percentage of ties retained) and the correlations in the observed data.

Again, using $r^{\#}$ as defined in (11), to invalidate the observed inference, we need to randomly rewire $(1 - p)\%$ of ties to obtain

$$r_{XY|Z}^* = \frac{(pr_{XY} + (1 - p)^*r_{YZ}) - (pr_{XZ} + (1 - p))^*r_{YZ}}{\sqrt{1 - (pr_{XZ} + (1 - p))^2}\sqrt{1 - r_{YZ}^2}} = r^{\#} \tag{22}$$

To write p as a function of other variables we have

$$p = \frac{-2(r_{XZ} + 1)(r_{YZ}^2 - 1)r^{\#2}}{(r_{XY} - r_{XZ}r_{YZ})^2 - r_{XZ}^2 r_{YZ}^2 r^{\#2} + r_{XZ}^2 r^{\#2} - 2r_{XZ}r_{YZ}^2 r^{\#2} + 2r_{XZ}r^{\#2} - r_{YZ}^2 r^{\#2} + r^{\#2}} \tag{23}$$

4.2 Analytical solutions for tie-level rewiring

The previous derivation is based on the assumption that we rewire all ties for $(1 - p) \cdot 100\%$ of the nodes. However, in our simulation we are essentially rewiring $(1 - p)100\%$ of ties evenly across all nodes. Here we give a simulation example where we randomly rewire 30% of the total ties from a random network. Figure 4 shows the distribution of the percentage of the ties rewired on an individual level. As this shows, the percentage of ties rewired for each individual varies from 10% to 80%, but on average each node has rewired 30% of its original ties. Compared with the assumptions in the analytical solutions of the node-level rewiring, the assumption that rewiring occurs evenly across all of the nodes is more reasonable, as network measurements for each actor can be imperfect for various reasons, such as bias toward self, group structure, balance, and routine interaction (Marsden, 2005). So in this section, we derive a new set of analytical solutions by assuming that rewiring occurs evenly for each node. This approach would have more similarity to the empirical situation and the actual simulation, but is more difficult to derive. Here we provide some intuition for this scenario. Note that here we only derive the analytical solutions for homophily rewiring and anti-homophily rewiring, since in random rewiring the result will not be affected much by the level at which we rewire the ties, as long as we simulate a sufficient number of times. See Appendix A (Supplementary Material) for the details of derivation.

4.3 Validation

In this section, we provide three simulation examples to evaluate how well our analytical solutions.

In each example, we construct a simulated network dataset where network size is 100 and the density of ties = 0.1. As above, the network is a random unweighted directed network without self-loops, and the influence process is as follows:

$$y_{it} = \beta_0 + \beta_1 y_{it-1} + \beta_2 \frac{\sum w_{ijt-1} y_{jt-1}}{\sum w_{ijt-1}} + e_{it} \tag{24}$$

where y_{it} represents the behavioral outcome of interest, w_{ijt-1} is a binary variable representing a network relationship, and e_{it} is an error term following a normal distribution with mean 0 and standard deviation 0.2. We simulated the influence process based on model (24) for two time points. In the three simulated examples, we fix the correlation between the prior and the dependent variable (r_{YZ}) to be ~ 0.6 , the correlation between the prior and the network exposure (r_{XZ}) to be $0.1 \sim 0.2$, but vary the correlation between network exposure and the dependent variable (r_{XY}) to be > 0.4 (strong influence/inference), 0.3 (moderate influence/inference), and < 0.2 (weak influence/inference).

To explore how our estimates are robust to various errors in the observed network, we then rewire a different percentage of existing ties (varying from 10 to 90%) based on (1) random selection; (2) homophily, that is, actors will rewire to unestablished ties with the smallest value of $|y_{it-1} - y_{jt-1}|$; or (3) anti-homophily, that is, actors will rewire to unestablished ties with the largest value of $|y_{it-1} - y_{jt-1}|$.

In each configuration, we simulate 1000 times and calculate the mean of (1) the new raw correlation r_{XZ} between the prior and the network exposure after rewiring; (2) the new raw correlation r_{XY} between network exposure and the dependent variable after rewiring; and (3) the partial correlation $r_{XY|Z}$ between network exposure and the dependent variable after rewiring. We then compare the simulation results with the two sets of analytical solutions below.

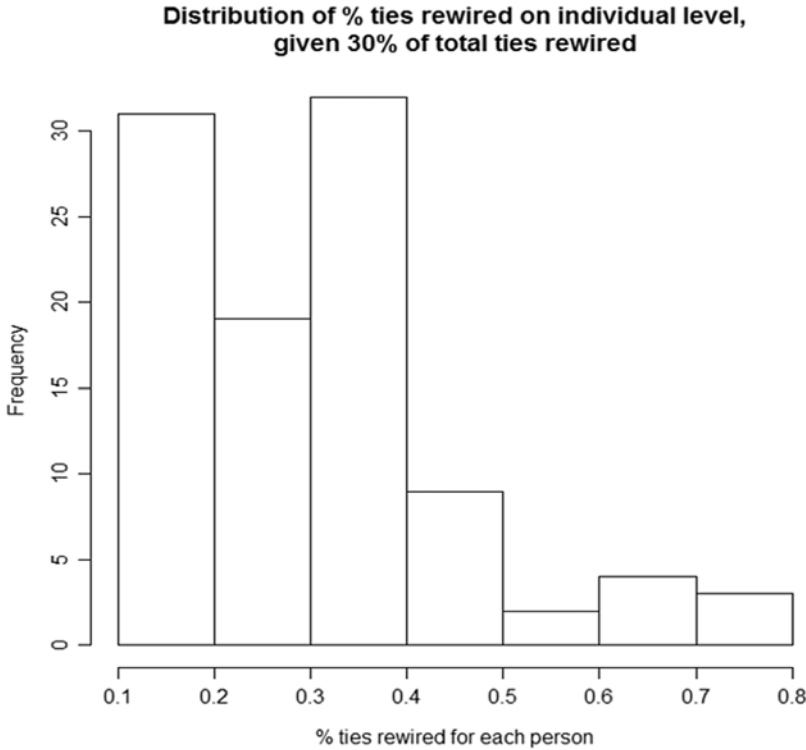


Figure 4. Distribution of percent of the ties rewired.

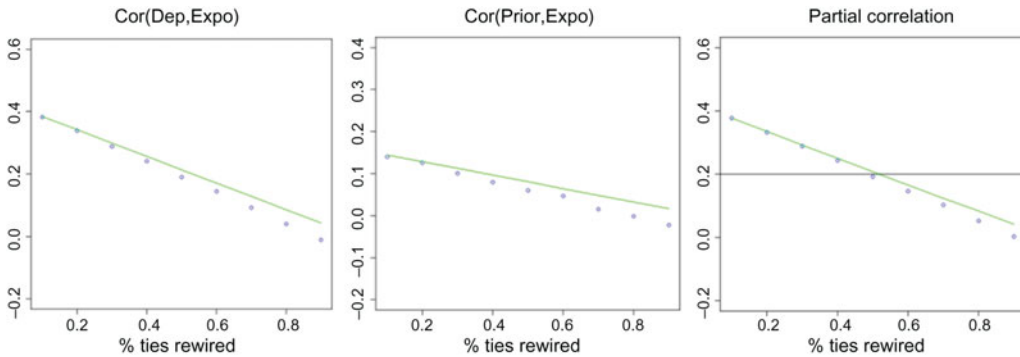


Figure 5. Strong influence—random rewiring example.

Example 1 is a case when there is strong influence/inference. $r_{XY} > 0.4$. Random rewiring results are shown in Figure 5.

The graphs from left to right, respectively, show (1) raw correlations r_{XY} between network exposure and the dependent variable after rewiring, (2) raw correlations r_{XZ} between the prior and network exposure after rewiring; and (3) partial correlations $r_{XY|Z}$ between network exposure and the dependent variable after rewiring. The X axis shows the percentage of ties rewired. The Y axis shows the values of correlation. Blue dots are the results from simulations (each point is a result of 1000 simulations), the green line is the result from the analytical solutions for the

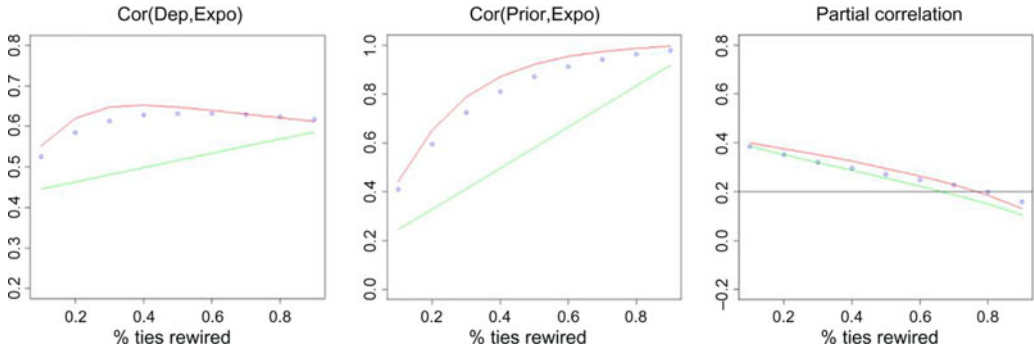


Figure 6. Strong influence—homophily rewiring example.

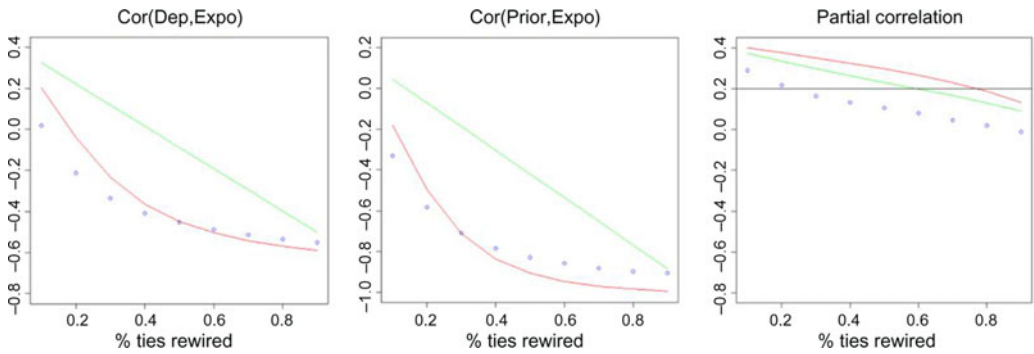


Figure 7. Strong influence—anti-homophily rewiring example.

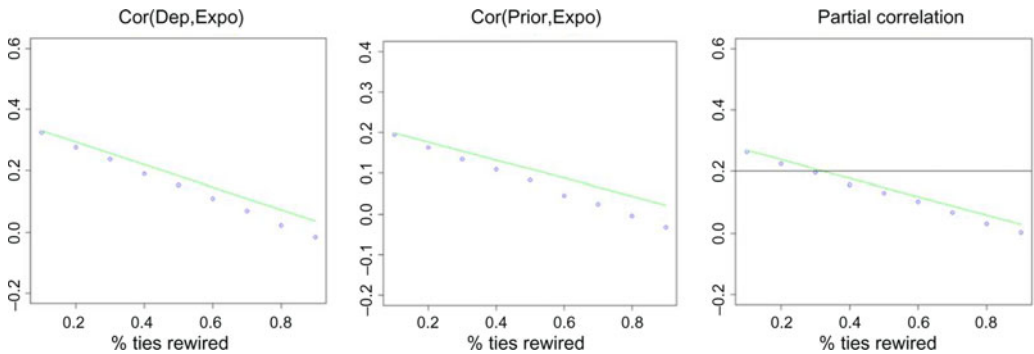


Figure 8. Moderate influence—random rewiring example.

node-level rewiring, and the red line is the result from the analytical solutions for the tie-level rewiring. The black line represents the threshold to change the inference. The same description applies to all the graphs set out below. Homophily rewiring results are shown in Figure 6, where anti-homophily results are shown in Figure 7.

Example 2 is a case when there is moderate influence/inference, $r_{XY} \approx 0.3$. As before random rewiring results are shown in Figure 8, homophily rewiring results are shown in Figure 9, and anti-homophily results are shown in Figure 10.

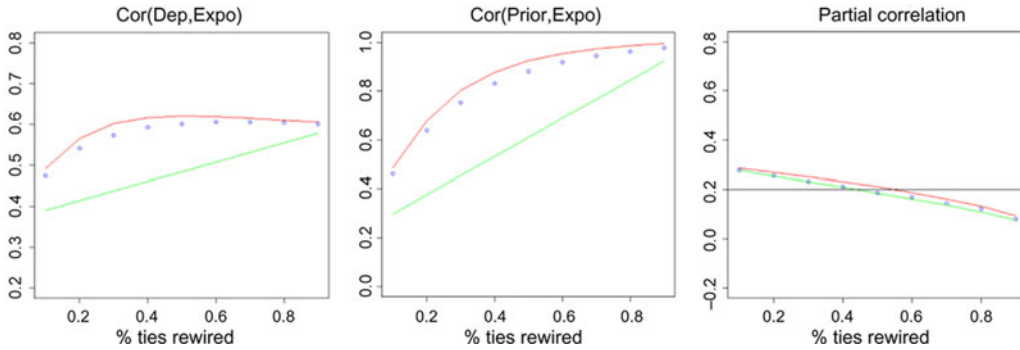


Figure 9. Moderate influence—homophily rewiring example.

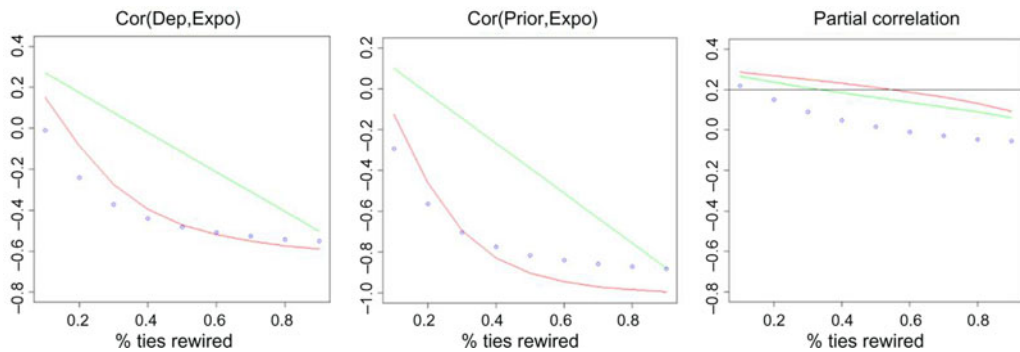


Figure 10. Moderate influence—anti-homophily rewiring example.

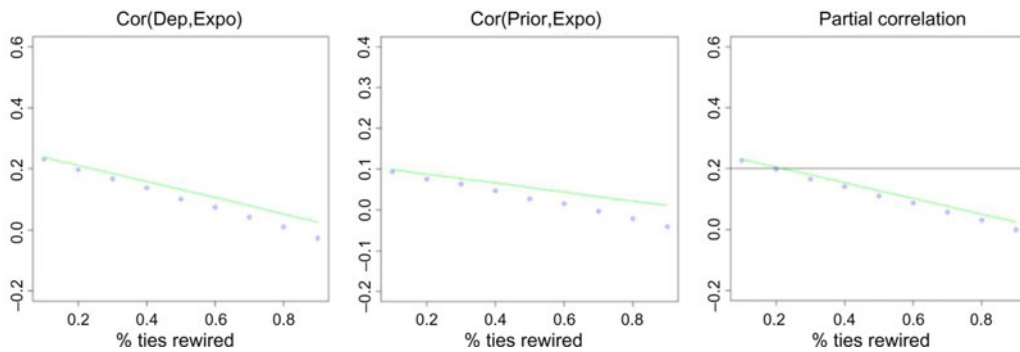


Figure 11. Weak influence—random rewiring example.

Example 3 is a case when we have weak influence/inference, $r_{XY} < 0.2$. As before random rewiring results are shown in Figure 11, homophily rewiring results are shown in Figure 12, anti-homophily rewiring results are shown in Figure 13.

As the three examples show, the analytical solutions for node-level rewiring do not fit well to the actual raw correlations r_{XY} and r_{XZ} after rewiring, but the analytical solutions for the tie-level rewiring fit quite well to the raw correlations r_{XY} and r_{XZ} from the simulations, mainly due to the fact that the calculation of the tie-level rewiring approximates the actual simulation process better.

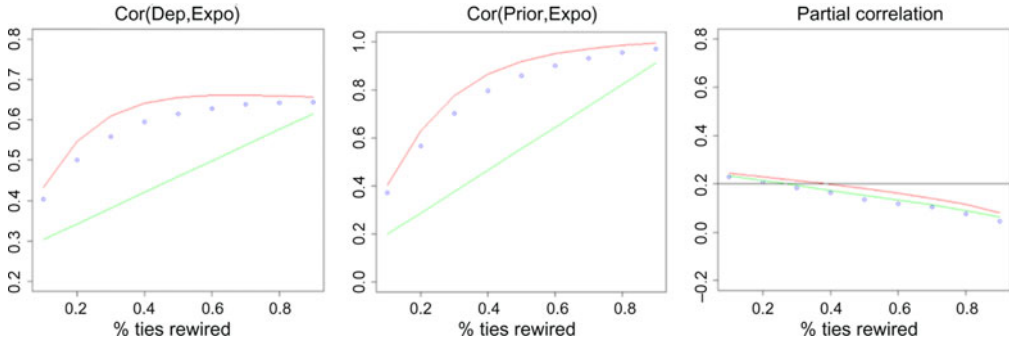


Figure 12. Weak influence—homophily rewiring example.

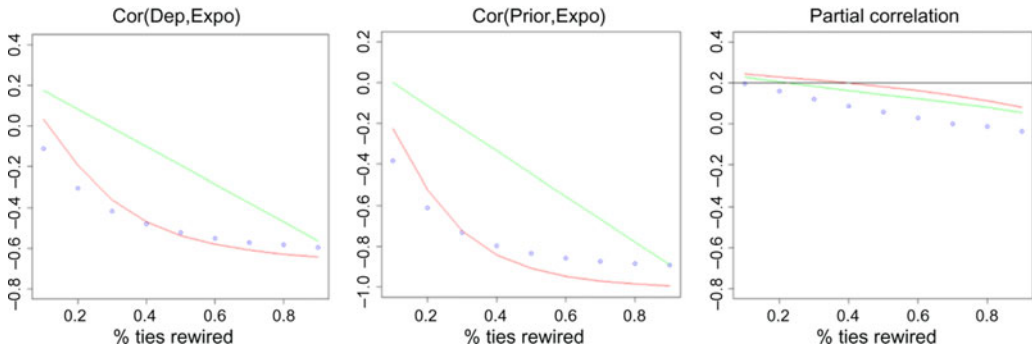


Figure 13. Weak influence—anti-homophily rewiring example.

However, in terms of determining the robustness of inference, both sets of analytical solutions are equally useful and fit very well with the partial correlation $r_{XY|Z}$ calculated from the simulation, except for anti-homophily rewiring, where the analytical solution overestimates the partial correlation to some extent. One possible reason could be that for the anti-homophily rewiring, we assume the new correlation r_{XZ} between network exposure and prior after rewiring is -1 . But the magnitude of the actual correlation is usually smaller than 1, depending on the distribution of the prior term. As a result, our analytical solutions for anti-homophily rewiring are likely to provide an upper bound for the actual partial correlation of interest. In Figure S1 in the Appendix C (Supplementary Material), we give another simulation example where the prior term is a binary variable, so that the new correlation between network exposure and the prior after rewiring should be -1 . As Figure S1 shows (Supplementary Material), in this case, most of the bias is eliminated and the analytical solutions fit very well with the simulation results.

Finally, note that in the homophily rewiring, we assume that actors can rewire to others who hold exactly the same behavior. This generally requires a large network where everyone can find perfectly homophilous others. It should not be surprising that the fit between the analytical solution and simulation would be worse with a smaller size of the network, or higher density of ties, in which cases the actors are forced to form ties with others who are different from themselves. And indeed, examples (Figures S2 and S3) in Appendix D (Supplementary Material) show a worse fit between the analytical solution and homophily rewiring when we reduce the network size or increase the density of the network.

5. Discussion and conclusion

This study proposes a set of simulation-based sensitivity analysis methods that can test the robustness of inferences made in social network analysis to six forms of selection mechanisms that can cause biases in network observations, namely, random, homophily, anti-homophily, transitivity, reciprocity, and preferential attachment. Specifically, we show how these approaches are useful in testing the robustness of inferences for social influence effects. Besides, we have also derived two sets of analytical solutions for sensitivity analysis methods that can account for selection mechanisms based on random, homophily, and anti-homophily. Examples show that the analytical solutions generally fit well to the simulation results under reasonable assumptions.

The principle under which these sensitivity analysis methods operate closely relate to the sensitivity analysis literature that account for the omitted variable problem, as biases in the network observations can essentially be framed as an omitted variable problem. However, the methods proposed in this paper are unique in this context because they consider the specific properties of the omitted variable and how it could change as a result of network selection and, correspondingly, how strong the various mechanisms of selection must be to invalidate an inference.

The simulation-based sensitivity analysis methods developed in this chapter can be easily adapted and applied to various study designs (e.g. observational studies and randomized experiments) and many different forms of network analysis, such as a one-mode selection model (e.g. P2, ERGM (Van Duijn et al., 2004; Robins et al., 2007)), bipartite graph analysis, and models that deal with co-evolution between behavior and networks (SIENA (Snijders et al., 2007)). Nevertheless, our focus has been on the robustness of inference in influence models, partly due to the wide interest of behavioral outcomes under the network process. Our sensitivity analysis methods essentially re-construct the network exposure terms by rewiring the observed interaction matrix W , and different rewiring mechanisms have distinct implications for the network structure and the distribution of the network exposure term, as follows.

1. Different agency rewiring (random, homophily, and anti-homophily) can create distinct distributions of network exposure. For example, random rewiring can create a network exposure that is close to the overall mean of the actor's prior belief/behavior distribution; homophily rewiring (in the extreme) can create a network exposure that is exactly the same as the actor's prior belief/behavior distribution; and anti-homophily rewiring often creates a network exposure with a polarized bimodal distribution, as actors seek most dissimilar others in this case, and their network exposure will be clustered around those with most dissimilar behaviors/beliefs.
2. Structural rewiring (transitivity, reciprocity, and preferential attachment) can create distinct network structures. For example, if observed ties are completely rewired based on transitivity, we would see a network with a strong community feature and local clustering; if observed ties are completely rewired based on reciprocity we would see a network with fewer paths but much more bi-directional interactions; finally, if observed ties are completely rewired based on preferential attachment, we would see a network with a core-periphery structure or a network with a power-law degree distribution.

Besides simulation-based sensitivity analysis methods, in this study, we have also developed analytical solutions for random, homophily, and anti-homophily rewiring. However, it should be noted that the motivation behind the analytical solutions in this study is not to replace the simulation-based rewiring, but to better understand how simulation-based rewiring methods work and how they affect the inference of social influence effects in various scenarios. Furthermore, the analytical solutions can help readers to locate a range of percent of ties to be rewired when only some key statistics (instead of raw data) from a study is available, or when it is computationally expensive to run though the whole range of percent of ties to be rewired

in the simulation. In general, the goodness of fit between analytical solutions and simulation-based rewiring depends on the observed network structure and the distribution of covariates of interest (e.g. prior, individual characteristics such as gender, age, etc.), some of which have direct sociological implications, as follows.

1. The analytical solution for homophily rewiring requires that actors rewire to perfectly homophilous others, and this fits better to simulation results when we have a larger network, as is plausible since actors are more likely to find similar others in a large social structure with a lot of heterogeneity (Blau, 1977). In contrast, selection constraint is more salient in smaller networks as actors' choices will be more limited. And it will be even more difficult if actors are seeking similar others on multiple dimensions (age, race, etc.). Furthermore, the analytical solution also fits better to simulation results when we have a sparser network, as for each individual it is easier to find and maintain a small homogenous social group than a large one.
2. The analytical solution for anti-homophily rewiring assumes that the new correlation between network exposure and the prior term after rewiring should approximately be -1 . This assumption is more likely to hold when the distribution for actors' prior belief/behavior is binary or bimodal. For example, if actors seek dissimilar others based on whether they smoke or not (1 yes, 0 no), the resulting correlation after rewiring between network exposure and the prior term should be exactly -1 . Or if the variable of interest is a continuous measure of political ideas/belief and actors' prior orientation are clustered around two polarizing political ideas/beliefs, the resulting correlation after rewiring between network exposure and the prior term should be approximately -1 as well because actors can fulfill their selection preferences. As a result, the analytical solution for anti-homophily rewiring will fit better with simulation results in cases where there are two ideologically polarized groups.

Finally, note that there are several limitations in this study which introduce future avenues of research.

1. In our study, we represent network relations using binary variables, either there is a relation or there is no relation. However, there are many other representations of network relations, using descriptors such as ranks and weights. The sensitivity analyses developed in this study can be extended to include these cases.
2. We have only applied our sensitivity analyses to inference for social influence effects. Although our simulation-based sensitivity analyses can potentially be applied to any case using network data, in our study, we only give examples for the inference for social influence effects, and our analytical solutions also only apply to one type of social influence effect. Future research may explore the analytical solutions of the sensitivity analysis as applied to other network outcome (e.g. centrality (Martin & Niemeyer, 2019)) and influence models.
3. Our sensitivity analysis methods only deal with observed variables in networks, not unobserved variables. For example, for the homophily and anti-homophily rewiring, our sensitivity analysis methods only apply to homophily/anti-homophily based on observed variables. That is, our methods estimates and inferences are vulnerable to unobserved/confounding variables, which are the focus of many other sensitivity analysis methods (Frank, 2000; VanderWeele, 2011). For example, Frank (2000) characterized the strength of correlations between the omitted variable and the exposure term and between the omitted variable and the outcome necessary to change the inference. Potentially, this limits how useful our methods are in terms of testing the internal validity of the inference for social influence effects,

since latent homophily/shared environmental factors are usually the biggest concerns. Some other potential remedies include conducting randomized experiments, or utilizing latent social positions and latent social distances generated from latent space models (Hoff et al., 2002) to account for omitted variable bias due to latent homophily/shared environmental factors or observation error/sampling error in networks (Xu, 2018; Sweet et al., 2020). Future research may explore how randomized network experiments or latent space adjusted approaches perform and how they interact with the sensitivity analysis methods proposed here.

4. Our current methods only focus on rewiring of misreported ties in network studies and we assume the out-degree for each person is constant. Although misreported ties are important concerns and can occur due to various reasons (e.g. bias due to cognitive priming and satisficing in multiplex network measurements, misreport of aspirational ties (Berndt & Murphy, 2002; Pustejovsky & Spillane, 2009)), unreported or underreported ties are generally more prevalent in network studies. Future research may explore how to generate unreported ties based on various mechanisms mentioned in this paper and their implications for statistical inferences.

Nevertheless, we consider the sensitivity analyses developed here to be important steps in terms of understanding how misinterpretation of actors' preferences that are manifest in observed networks can affect the robustness of inferences. And they are also useful as empirical tools that allow us to quantify the robustness of our inferences by devising clear and testable alternative hypotheses, which are the keys to making strong inference in any field of science (Platt, 1964).

Conflicts of interest. None.

Supplementary materials. For supplementary material for this article, please visit <https://doi.org/10.1017/nws.2020.36>.

Notes

1 The idea is similar to replacing unsampled population as in Frank & Min (2007), where ties are the unit of analysis. In their original cases, the estimate of interest based on ideal sample can be represented as $\hat{\beta}_1 = (1 - \pi)\hat{\beta}_1^{ob} + \pi\hat{\beta}_1^{un}$. However, what is unique in our case is that the observed sample and unobserved sample are generally not independent. As a consequence, the inference based on ideal sample is much more complicated than the weighted average of the estimates based on two samples and thus less tractable.

2 In principle, our proposed methods are analogous to a community detection algorithm (Girvan & Newman, 2002) that identifies edges with high betweenness (Freeman, 1977) that need to be removed to create separable components in the graph.

3 Our network is directed, but we define common friends as in an undirected graph to capture various definitions of transitivity or triads. For details see Davis & Leinhardt (1972) on triad censuses, or Wasserman and Faust (1994, p 243) and Robins et al. (2007).

4 Note that for random and homophily-based rewiring, the maximum percentage of network relations that can be rewired is constrained by the density of the network, since observed ties must be rewired to different pairs. However, for structure-based rewiring, there are no such constraints, and observed ties do not necessarily have to be rewired to different pairs, as actors can choose whichever pairs maximize their utility.

5 This in principle is very similar to the case in Frank (2000) where he talked about the attenuation bias due to measurement errors in confounding variables. Here we discuss how measurement errors in networks (and as a result in exposure terms) can attenuate our inference.

6 Some have raised potential concerns for only modeling influence because it ignores the selection process occurring at the same time which might bias the influence estimates (An, 2011). However, as Shalizi & Thomas (2011) showed that the selection bias can be translated into an omitted variable bias problem. And Frank & Xu (2020) showed as long as we have controlled for the confounding variable that affects both selection and influence, the true influence effect can still be identified using model 1 or its equivalent (see also Xu, 2018).

7 Note that in empirical data, we can incorporate variables other than Y for rewiring based on homophily.

References

- An, W. (2011). Models and methods to identify peer effects. In J. Scott & Peter J. Carrington (Eds.), *The Sage handbook of social network analysis* (pp. 515–532). London: Sage.
- An, W. (2015). Instrumental variables estimates of peer effects in social networks. *Social Science Research*, 50, 382–394.
- Aral, S., Muchnik, L., & Sundararajan, A. (2009). Distinguishing influence-based contagion from homophily-driven diffusion in dynamic networks. *Proceedings of the National Academy of Sciences*, 106(51), 21544–21549.
- Backman, C. W., & Secord, P. F. (1959). The effect of perceived liking on interpersonal attraction. *Human Relations*, 12, 379–84.
- Barabási, A. L., & Albert, R. (1999). Emergence of scaling in random networks. *Science*, 286(5439), 509–512.
- Bernard, H. R., & Killworth, P. D. (1977). Informant accuracy in social network data II. *Human Communication Research*, 4(1), 3–18.
- Bernard, H. R., Killworth, P. D., & Sailer, L. (1982). Informant accuracy in social-network data V. An experimental attempt to predict actual communication from recall data. *Social Science Research*, 11(1), 30–66.
- Bernard, H. R., Killworth, P., & Sailer, L. (1981). Summary of research on informant accuracy in network data and the reverse small world problem. *Connections*, 4(2), 11–25.
- Berndt, T. J., & Murphy, L. M. (2002). Influences of friends and friendships: Myths, truths, and research recommendations. *Advances in child development and behavior*, 30, 275–310.
- Blau, P. M. (1977). Inequality and heterogeneity: A primitive theory of social structure (Vol. 7). New York: Free Press.
- Borgatti, S. P., & Everett, M. G. (2000). Models of core/periphery structures. *Social Networks*, 21(4), 375–395.
- Burt, R. S., & Knez, M. (1995). Kinds of third-party effects on trust. *Rationality and Society*, 7(3), 255–292.
- Byrne, D. E. (1971). *The attraction paradigm* (Vol. 11). Cambridge, MA: Academic Press.
- Carley, K. M. (1991). Designing organizational structures to cope with communication breakdowns: A simulation model. *Organization & Environment*, 5(1), 19–57.
- Casciaro, T. (1998). Seeing things clearly: Social structure, personality, and accuracy in social network perception. *Social Networks*, 20(4), 331–351.
- Christakis, N. A., & Fowler, J. H. (2007). The spread of obesity in a large social network over 32 years. *New England Journal of Medicine*, 357(4), 370–379.
- Christakis, N. A., & Fowler, J. H. (2008). The collective dynamics of smoking in a large social network. *New England Journal of Medicine*, 358(21), 2249–2258.
- Davis, G. F., Yoo, M., & Baker, W. E. (2003). The small world of the American corporate elite, 1982–2001. *Strategic Organization*, 1(3), 301–326.
- Davis, J. A. and Leinhardt, S. (1972): The structure of positive interpersonal relations in small groups. In J. Berger, M. Zelditch Jr., & B. Anderson (Eds.), *Sociological Theories in Progress* (Vol. 2, pp. 218–251). Boston: Houghton Mifflin.
- Doreian, P. (2001). Causality in social network analysis. *Sociological Methods & Research*, 30(1), 81–114.
- Emirbayer, M., & Goodwin, J. (1994). Network analysis, culture, and the problem of agency. *American Journal of Sociology*, 1411–1454.
- Feld, S. L. (1981). The focused organization of social ties. *American Journal of Sociology*, 1015–1035.
- Feld, S. L., & Carter, W. C. (2002). Detecting measurement bias in respondent reports of personal networks. *Social Networks*, 24(4), 365–383.
- Fleming, L., King III, C., & Juda, A. I. (2007). Small worlds and regional innovation. *Organization Science*, 18(6), 938–954.
- Frank, K. A. (2000). Impact of a confounding variable on a regression coefficient. *Sociological Methods & Research*, 29(2), 147–194.
- Frank, K. A., Zhao, Y., & Borman, K. (2004). Social capital and the diffusion of innovations within organizations: The case of computer technology in schools. *Sociology of Education*, 77(2), 148–171.
- Frank, K. A., Zhao, Y., Penuel, W. R., Ellefson, N., & Porter, S. (2011). Focus, fiddle, and friends: Experiences that transform knowledge for the implementation of innovations. *Sociology of education*, 84(2), 137–156.
- Frank, K. A., Maroulis, S. J., Duong, M. Q., & Kelcey, B. M. (2013). What would it take to change an inference? Using Rubin's causal model to interpret the robustness of causal inferences. *Educational Evaluation and Policy Analysis*, 35(4), 437–460.
- Frank, K. & Min, K. S. (2007). 10. Indices of Robustness for Sample Representation. *Sociological Methodology*, 37(1), 349–392.
- Frank, K. A. & Xu, R. (2020). Causal Inference for Social Network Analysis. In R. Light & J. Moody (Eds.), *The Oxford Handbook of Social Networks*. 288–310. New York, Oxford: Oxford University Press.
- Freeman, L. C. (1977). A set of measures of centrality based on betweenness. *Sociometry*, 40(1), 35–41.
- Freeman, L. C., Romney, A. K., & Freeman, S. C. (1987). Cognitive structure and informant accuracy. *American Anthropologist*, 89(2), 310–325.
- Friedkin, N. E., & Johnsen, E. C. (1990). Social influence and opinions. *Journal of Mathematical Sociology*, 15(3–4), 193–206.
- Friedkin, N. E., & Johnsen, E. C. (1999). Social influence networks and opinion change. *Advances in Group Processes*, 16(1), 1–29.

- Girvan, M., & Newman, M. E. (2002). Community structure in social and biological networks. *Proceedings of the National Academy of Sciences*, 99(12), 7821–7826.
- Goffman E. (1963). Behavior in public places: Notes on the social organization of gatherings. New York: Free Press Glencoe. 248 pp.
- Golder, S. A., Wilkinson, D. M., & Huberman, B. A. (2007). Rhythms of social interaction: Messaging within a massive online network. In C. Steinfield, B. T. Pentland, M. Ackerman, & N. Contractor (Eds.), *Communities and technologies 2007* (pp. 41–66). London: Springer.
- Granovetter, M. S. (1973). The strength of weak ties. *American Journal of Sociology*, 1360–1380.
- Guimerà, R., & Sales-Pardo, M. (2009). Missing and spurious interactions and the reconstruction of complex networks. *Proceedings of the National Academy of Sciences*, 106(52), 22073–22078.
- Handcock, M. S., & Gile, K. J. (2010). Modeling social networks from sampled data. *The Annals of Applied Statistics*, 4(1), 5.
- Hoff, P. D., Raftery, A. E., & Handcock, M. S. (2002). Latent space approaches to social network analysis. *Journal of the American Statistical Association*, 97(460), 1090–1098.
- Huisman, M. (2009). Imputation of missing network data: Some simple procedures. *Journal of Social Structure*, 10(1), 1–29.
- Iyengar, R., Van den Bulte, C., & Valente, T. W. (2011). Opinion leadership and social contagion in new product diffusion. *Marketing Science*, 30, 195–212.
- Jin, E. M., Girvan, M., & Newman, M. E. (2001). Structure of growing social networks. *Physical Review E*, 64(4), 046132.
- Kandel, D. B. (1978). Homophily, selection, and socialization in adolescent friendships. *American Journal of Sociology*, 84(2), 427–436.
- Killworth, P., & Bernard, H. (1976). Informant accuracy in social network data. *Human Organization*, 35(3), 269–286.
- Kossinets, G. (2006). Effects of missing data in social networks. *Social Networks*, 28(3), 247–268.
- Kossinets, G., & Watts, D. J. (2009). Origins of homophily in an evolving social network. *American Journal of Sociology*, 115(2), 405–450.
- Krackhardt, D. (1987). QAP partialling as a test of spuriousness. *Social Networks*, 9(2), 171–186.
- Leenders, R. T. A. (2002). Modeling social influence through network autocorrelation: constructing the weight matrix. *Social networks*, 24(1), 21–47.
- Leifer, E. M. (1988). Interaction preludes to role setting: Exploratory local action. *American Sociological Review*, 53(6), 865.
- Liljeros, F., Edling, C. R., Amaral, L. A. N., Stanley, H. E., & Åberg, Y. (2001). The web of human sexual contacts. *Nature*, 411(6840), 907–908.
- Lyons, R. (2011). The spread of evidence-poor medicine via flawed social-network analysis. *Statistics, Politics, and Policy*, 2(1).
- Manski, C. F. (1993). Identification of endogenous social effects: The reflection problem. *The Review of Economic Studies*, 60(3), 531–542.
- Mark, N. (1998). Beyond individual differences: Social differentiation from first principles. *American Sociological Review*, 309–330.
- Marsden, P. V. (1990). Network data and measurement. *Annual Review of Sociology*, 435–463.
- Marsden, P. V., & Friedkin, N. E. (1993). Network studies of social influence. *Sociological Methods & Research*, 22(1), 127–151.
- Marsden, P. V. (2005). Recent developments in network measurement. *Models and Methods in Social Network Analysis*, 8, 30.
- Martin, C., & Niemeier, P. (2019). Influence of measurement errors on networks: Estimating the robustness of centrality measures. *Network Science*, 7(2), 180–195.
- Mayhew, B. H. (1980). Structuralism versus individualism: Part 1, shadowboxing in the dark. *Social Forces*, 59(2), 335–375.
- McPherson, M., Smith-Lovin, L., & Cook, J. M. (2001). Birds of a feather: Homophily in social networks. *Annual Review of Sociology*, 415–444.
- Merton, R. K. (1968). The Matthew effect in science. *Science*, 159(3810), 56–63.
- Mollica, K. A., Gray, B., & Trevino, L. K. (2003). Racial homophily and its persistence in newcomers' social networks. *Organization Science*, 14(2), 123–136.
- Montoya, R. M., & Insko, C. A. (2008). Toward a more complete understanding of the reciprocity of liking effect. *European Journal of Social Psychology*, 38(3), 477–498.
- Moffitt, R. A. (2001). Policy interventions, low-level equilibria, and social interactions. *Social Dynamics*, 4(45–82), 6–17.
- Moody, J. (2004). The structure of a social science collaboration network: Disciplinary cohesion from 1963 to 1999. *American Sociological Review*, 69(2), 213–238.
- Newcomb, T. M. (1956). The prediction of interpersonal attraction. *American psychologist*, 11(11), 575.
- Newman, M. E. (2001). The structure of scientific collaboration networks. *Proceedings of the National Academy of Sciences*, 98(2), 404–409.
- Page, S. E. (2007). *The difference: How the power of diversity creates better groups, firms, schools, and societies*. Princeton, NJ: Princeton Univ. Press.
- Pan, W., & Frank, K. A. (2004). An approximation to the distribution of the product of two dependent correlation coefficients. *Journal of Statistical Computation and Simulation*, 74(6), 419–443.
- Penuel, W. R., Sun, M., Frank, K. A., & Gallagher, H. A. (2012). Using social network analysis to study how collegial interactions can augment teacher learning from external professional development. *American Journal of Education*, 119(1), 103–136.

- Pitts, V. M., & Spillane, J. P. (2009). Using social network methods to study school leadership. *International Journal of Research & Method in Education*, 32(2), 185–207.
- Platt, J. R. (1964). Strong inference. *Science*, 146(3642), 347–353.
- Pustejovsky, J. E., & Spillane, J. P. (2009). Question-order effects in social network name generators. *Social networks*, 31(4), 221–229.
- Rivera, M. T., Soderstrom, S. B., & Uzzi, B. (2010). Dynamics of dyads in social networks: Assortative, relational, and proximity mechanisms. *Annual Review of Sociology*, 36, 91–115.
- Robins, G., Pattison, P., & Woolcock, J. (2004). Missing data in networks: Exponential random graph (p^*) models for networks with non-respondents. *Social Networks*, 26(3), 257–283.
- Robins, G., Pattison, P., Kalish, Y., & Lusher, D. (2007). An introduction to exponential random graph (p^*) models for social networks. *Social Networks*, 29(2), 173–191.
- Rosenbaum, P. R., & Rubin, D. B. (1983). Assessing sensitivity to an unobserved binary covariate in an observational study with binary outcome. *Journal of the Royal Statistical Society. Series B (Methodological)*, 212–218.
- Rosenbaum, P. R., & Rubin, D. B. (1983). The central role of the propensity score in observational studies for causal effects. *Biometrika*, 70(1), 41–55.
- Runger, G., & Wasserman, S. (1980). Longitudinal analysis of friendship networks. *Social Networks*, 2(2), 143–154.
- Schelling, T. C. (1971). Dynamic models of segregation. *Journal of Mathematical Sociology*, 1(2), 143–186.
- Shalizi, C. R., & Thomas, A. C. (2011). Homophily and contagion are generically confounded in observational social network studies. *Sociological Methods & Research*, 40(2), 211–239.
- Snijders, T. A., Steglich, C. E., & Schweinberger, M. (2007). Modeling the co-evolution of networks and behavior. Longitudinal models in the behavioral and related sciences, 31(4), 41–71.
- Sprecher, S. (1998). Insiders' perspectives on reasons for attraction to a close other. *Social Psychology Quarterly*, 287–300.
- Steglich, C., Snijders, T. A., & Pearson, M. (2010). Dynamic networks and behavior: Separating selection from influence. *Sociological Methodology*, 40(1), 329–393.
- Sun, M., Penuel, W. R., Frank, K. A., Gallagher, H. A., & Youngs, P. (2013). Shaping professional development to promote the diffusion of instructional expertise among teachers. *Educational Evaluation and Policy Analysis*, 35, 344–369.
- Sweet, T., & Adhikari, S. (2020). A Latent Space Network Model for Social Influence. *Psychometrika*, 1–24.
- Uzzi, B., & Spiro, J. (2005). Collaboration and creativity: The small world Problem1. *American Journal of Sociology*, 111(2), 447–504.
- Valente, T. W. (1996). Social network thresholds in the diffusion of innovations. *Social networks*, 18(1), 69–89.
- Valente, T. W. (2005). Network models and methods for studying the diffusion of innovations. In P. J. Carrington, J. Scott, & Stanley (Eds.), *Models and methods in social network analysis*, 28, 98. Cambridge: Cambridge university press.
- Valente, T. W., Pitts, S., Wipfli, H., & Yon, G. G. V. (2019). Network influences on policy implementation: Evidence from a global health treaty. *Social Science & Medicine*, 222, 188–197.
- VanderWeele, T. J. (2011). Sensitivity analysis for contagion effects in social networks. *Sociological Methods & Research*, 40(2), 240–255.
- VanderWeele, T. J., & An, W. (2013). Social networks and causal inference. In *Handbook of causal analysis for social research* (pp. 353–374). Springer, Dordrecht. Edited by Stephen L. Morgan.
- Van Duijn, M. A., Snijders, T. A., & Zijlstra, B. J. (2004). p2: A random effects model with covariates for directed graphs. *Statistica Neerlandica*, 58(2), 234–254.
- Wasserman, S. & Faust, K. (1994). *Social network analysis: Methods and applications* (Vol. 8). Cambridge university press, Cambridge.
- Watts, D. J. (1999). Networks, dynamics, and the small-world phenomenon. *American Journal of sociology*, 105(2), 493–527.
- Xu, R. (2018). Alternative estimation methods for identifying contagion effects in dynamic social networks: A latent-space adjusted approach. *Social Networks*, 54, 101–117.
- Zerubavel, N., Bearman, P. S., Weber, J., & Ochsner, K. N. (2015). tracking popularity in real-world social networks. *Proceedings of the National Academy of Sciences*, 112(49), 15072–15077.
- Zheng, K., Padman, R., Krackhardt, D., Johnson, M. P., & Diamond, H. S. (2010). Social networks and physician adoption of electronic health records: Insights from an empirical study. *Journal of the American Medical Informatics Association*, 17, 328–336.