Causal Network Analysis

Weihua An, Emory University*
Roberson Beauvile, Emory University
Benjamin Rosche, Cornell University

Abstract

Fueled by recent advancements in statistical modeling and rapid growth of network data, social network analysis has become increasingly popular in sociology and related disciplines. However, a significant amount of work in the field has been descriptive and correlational, which prevents the findings from being more rigorously translated into practices and policies. This paper provides a review of the popular models and methods for causal network analysis with a focus on the casual inference threats (such as measurement error, missing data, network endogeneity, contextual confounding, simultaneity, and collinearity) and potential solutions (such as instrumental variables, specialized experiments, and leveraging longitudinal data). It covers major models and methods for both network formation and network effects and for both sociocentric networks and egocentric networks. Lastly, this review also discusses future directions for causal network analysis.

Keywords: Causal Inference, Network Formation, Network Effects, Social Capital, Social Contagion, Peer Effect

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* Corresponding author: Weihua An, Departments of Sociology & Quantitative Theory and Methods, Emory University, Email: weihua.an@emory.edu. The authors are listed alphabetically and contribute equally to the paper. Major contributions: W.A. designed the study and revised the whole draft, R.B. drafted the part on network formation, and B.R. drafted the part on network effects. We thank Mario Small for the invitation to contribute and the ARS Co-Editor Karen Cook for helpful guidance on the manuscript. We also thank Kenneth Bollen, Bet Caeyers, Ben Cornwell, Felix Elwert, Marcel Fafchamps, James Moody, Alison Schultz, and the anonymous reviewer for helpful comments. This material is based upon work supported by the National Science Foundation under Grant No. 2049207.

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Introduction

Social network analysis (SNA) is a major breakthrough in social science research. It shifts the focus from individual units (e.g., persons, organizations, or words) to their connections (e.g., friendships, collaborations, or co-occurrences). It brings about both theoretical and methodological innovations. Interest in network analysis has exploded especially in the past decade or so, due to new advancements in statistical modeling and rapid growth of network data.

Broad overviews of social network analysis can be found in Borgatti et al. (2009), Lusher et al. (2013), Wasserman and Faust (1994), Yang et al. (2016), among others. The literature predominately focuses on descriptive analysis and correlational analysis. However, in our view, more attention needs to be devoted to causal inference in network analysis, not only as a response to the "causal inference revolution" in social sciences (Angrist and Pischke 2009; Hernán and Robins 2020; Imbens and Rubin 2015; Morgan and Winship 2014), but also for the purpose of improving network analysis itself. For one, the disciplines from which researchers move into the field differ significantly in the amount of knowledge accumulation on causal inference, which sometimes leads to unsubstantiated causal claims. Second, network analysis often entails important practical and policy implications, which naturally requires stronger causal scrutiny.

There have been several prior reviews on the subject (An 2011; VanderWeele and An 2013). This review differs from them in a few aspects. First, unlike prior reviews that only focus on peer effects (An 2011), this one covers both network formation and network effects. Second, unlike prior reviews that only focus on sociocentric network analysis, this one also covers egocentric network analysis. Third, this review presents more updated literature on the subject by incorporating a consideratble amount of new work that has been doen in the last decade. Lastly, this review provides more detailed accounts on the various threats to causal nework analysis, including measurement error, missing data, network endogeneity, contextual confounding, simultaneity, and collinearity, among others. To note, this review focuses on cross-sectional data analysis but also touches on longitudinal data analysis where it is useful to identify causal effects.

The paper proceeds as follow. In the first part, we provide a brief overview of social network analysis to provide some background. In the second part, we review models for studying network formation, in particular, the exponential random graph model. In the third part, we review models for identifying causal network effects, in particular, the social capital model and the social contagion model. Lastly, we provide conclusions and discussions.

1. A Brief History of Social Network Analysis

Broadly speaking, the history of social network analysis can be divided into five periods (Freeman 2004; Scott 1991). First is the Emergence Period (1930s-1950s) in which Moreno (1934) introduced sociograms to represent relations between individual actors in a small group. Second is the Quite Period (1950s-1970s). The most notable achievement of this period is that the Columbia School (e.g., Paul Lazarsfeld, James Coleman, Elihu Katz, and Herbert Menzel, and Everett Rogers) studied social diffusion from a network perspective. Third is the Take-off Period (1970s-1990s) in which Harrison White and his students started the Harvard Revolution in SNA that popularized the use of matrix algebra for network analysis. The fourth period is the Break-through Period (1990s-2000s) which observed the development of advanced statistical network models such as the exponential random graph model and the stochastic actor-oriented model. The last period is the Deepening Period (2010s-present) in which SNA becomes more interdisciplinary and more engaged with causality, network-based interventions, big network analysis, etc.

Network analysis typically employs "egocentric" or "sociocentric" research designs (Marsden 2005). In egocentric network designs, researchers often sample subjects (egos) from a large population and elicit nodes directly connected to the egos (alters). Sociocentric network designs first identify a group of interest and then gather all the ties formed in that group. Researchers typically use observations (e.g., Whyte 1943), surveys (e.g., An 2022), archives (Padgett and Ansell 1993), or digital or social media (Wimmer and Lewis 2010) to collect network data.

2. Network Formation

In this section, we will discuss popular models for modeling sociocentric networks and ego networks and point out potential threats to causal inference and possible solutions.

2.1 Modeling Sociocentric Networks

Suppose we have a sociocentric network or equivalently, an adjacency matrix that describes the ties among actors. See below for an example, where there are five actors, w is the adjacency matrix with 1 indicating a tie from the row actor to the column actor and 0 the absence of the tie. Given the adjacency matrix, the task is to understand the determinants of tie formation, namely, why some cells in the adjacency are ones while others are zeros.

$$\boldsymbol{w} = \begin{bmatrix} 0 & 1 & 1 & 0 & 0 \\ 0 & 0 & 0 & 1 & 0 \\ 1 & 1 & 0 & 1 & 0 \\ 0 & 0 & 0 & 0 & 1 \\ 0 & 1 & 0 & 0 & 0 \end{bmatrix}$$

Theoretical Perspectives

Prior studies tend to model network formation from three perspectives. First, past research has identified various individual covariates that could affect tie formation, such as age (Marsden 1988; McPherson, Smith-Lovin, and Cook 2001), sex and gender (An 2022; Gest et al. 2007), race and ethnicity (Wimmer and Lewis 2010), cultural taste (Lizardo 2006), value and beliefs (Lazarsfeld and Merton 1954), family background (An 2022; Malacarne 2017), and genetic factors (Fowler, Dawes, and Christakis 2009). Past studies typically model three types of covariate effects: (1) receiver effects (i.e., effects of covariates on incoming ties), (2) sender effects (i.e., effects of covariates on outgoing ties), and homophily (i.e., the tendency to affiliate with similar others) (Laumann 1965; Leszczensky and Pink 2015, 2017; McPherson et al. 2001; Wimmer and Lewis 2010). An (2022) further shows how to transform these effects into tie-mixing effects to model status differential and differential homophily.

The second perspective focuses on structural opportunities that dictate how likely and frequently two actors can meet and interact. For example, Feld (1981, 1982) argues that actors with shared activities/affiliations are more likely to form ties with each other. Small & Adler (2019) argue that spatial context (e.g., spatial propinquity, spatial composition, and spatial configuration) plays an important role in tie formation. Wimmer and Lewis (2010) show that college students living in the same dormitories are more likely to be friends than with those living in different residences. Zeng and Xie (2008) show that group composition can also affect friendship formation. In terms of operationalization, most variables describing structural opportunities, such as an individual's affiliation or residence, can be treated as individual covariates.

Third, ties can be formed due to other existing ties, leading to endogenous tie formation (Block 2015; Goodreau, Kitts, and Morris 2009; Kossinets and Watts 2009; Wimmer and Lewis 2010). Prior studies have identified reciprocity (i.e., the tendency to reciprocate ties), transitivity (i.e., the tendency for an ego to link to alters' alters), preferential attachment (i.e., the tendency to link to a popular actor), and differential sociability (i.e., the tendency of some actors to send out relatively large numbers of ties), among others, as important endogenous tie formation processes.

Prior research (An 2022; Wimmer and Lewis 2010) shows that all three perspectives are important elements of a full account of network formation and ignoring any one could lead to inaccurate understanding of the network formation processes. Hence, depending on the type of ties being studied, researchers should include appropriate individual covariates, structural opportunities, and endogenous tie formation processes to prevent omitted variable bias.

Models and Estimation

A simple approach to modelling the (off-diagonal) cells in an adjacency matrix (assuming no self-loops) is to use logistic regression. An example is shown below.

$$\text{logit}\left[P\left(w_{ij}=1 \mid \boldsymbol{w}_{\boldsymbol{ij}}^{\boldsymbol{L}}, x\right)\right] = \theta_1 + \theta_2 \times x_i + \theta_3 \times x_j + \theta_4 \times I_{x_i=x_j} + \theta_5 \times \text{Reciptocity} + \theta_6 \times \text{Transitivity} \quad (1)$$

This model posits that the chance (i.e., log-odds) of having a tie from *i* to *j*, conditioning on $\boldsymbol{w}_{\boldsymbol{ij}}^{\boldsymbol{L}}$ (the local network of the tie w_{ij}) depends on a sender effect from a binary covariate x_i, a receiver effect x_j, and a homophily effect $I_{x_i=x_j}$ which indicates whether *i* and *j* have the same covariate value, as well as two endogenous tie formation processes: reciprocity (as measured by the tie status w_{ji}) and transitivity (as measured by the number of common contacts between *i* and *j*).

The dyadic logistic regression model can account for Markov dependence in tie formation, namely, the dependence between ties that share a common node. However, this model cannot account for higher-order dependence in tie formation, assuming ties are independent from one another conditional on their local dependence. Because of this nature of the model, the estimation of this model is termed as the maximum pseudo-likelihood estimation (MPLE).

Exponential random graph models (ERGMs) model the entire adjacency matrix at once and thus does not make any independence assumption, which makes ERGMs generally preferable to the dyadic logistic regression model. Specifically, an ERGM assumes that the probability of observing a network is given as below (Goodreau 2007; Handcock et al. 2003; Robins et al. 2007; Snijders et al. 2006; Wasserman and Faust 1994).

$$\Pr(\boldsymbol{W}=\boldsymbol{w} \mid \boldsymbol{X}) = \exp\{\theta^T g(\boldsymbol{w}, \boldsymbol{X})\}/K \quad (2)$$

where $\boldsymbol{W}$ represents a random network, $\boldsymbol{w}$ the observed network, $\boldsymbol{X}$ the covariates, $g(\boldsymbol{w}, \boldsymbol{X})$ a list of modeled effects (also called network statistics) such as covariate effects and endogenous tie formation processes, θ the model coefficients, and *K* a normalizing factor to ensure the probabilities sum to one. Hunter et al. (2008) shows that the ERGM is equivalent to a conditional logit model in which the log-odds of a tie from *i* to *j*, conditioning on the rest of the network ($\boldsymbol{w}_{\boldsymbol{ij}}^{\boldsymbol{c}}$), is determined by $\delta_{ij}(\boldsymbol{w}, \boldsymbol{X}) = \Delta g(\boldsymbol{w}, \boldsymbol{X})$, namely, changes in the network statistics when the *i*-*j* tie in the network flips from absence to presence. ERGMs are typically estimated by the Monte Carlo Maximum Likelihood Estimation (MCMLE) method (Hunter and Handcock 2006).

$$\text{logit}\left[P\left(w_{ij}=1 \mid \boldsymbol{w}_{\boldsymbol{ij}}^{\boldsymbol{c}}, \boldsymbol{X}\right)\right] = \theta^T \delta_{ij}(\boldsymbol{w}, \boldsymbol{X}) \quad (3)$$

Scalability

Because the MCMLE relies on Monte Carlo Markov Chains for estimation, it is usually slower than the MPLE used in the dyadic logistic regression. This limitation is especially notable when fitting big networks. Moreover, the MCMLE implicitly assumes that actors form ties with full

knowledge of all other ties, which seems less plausible for bigger networks. Hence, for both computational and theoretical reasons, alternative strategies are needed for fitting ERGMs on big networks. An (2016) presents several strategies for this purpose. They range from speeding up the MCMLE (Calderhead 2014), using the MPLE as an approximation (Wasserman and Pattison 1996; van Duijn, Gile, and Handcock 2009), taking the graph-limit to approximate the normalizing factor (He and Zheng 2015), fitting ERGMs on the subnetworks of a big network and then combining the estimates, to other data reduction and model reparametrization approaches.

Measurement error and missing data

Network data may contain various degrees of measurement error and missing data (An 2011; Handcock and Gile 2007; Marsden 2005). For example, measurement error in network ties can arise if respondents report their ties inaccurately (An and Schramski 2015) or forget about some of their ties (Brewer and Webster 2000), or network data are recorded inaccurately. Generally speaking, (random) measurement error in network ties leads to larger than necessary standard errors, similar to measurement error in the dependent variable. One method to reduce measurement error in network ties is to employ multiple reports on the same network ties to impute the true value (An and Schramski 2015; Krackhardt 1987). Similarly, An & Doan (2015) suggested using multiple peer reports to triangulate and correct self-reports of sensitive behaviors to reduce measurement error in self-reported covariates.

Missing ties can arise when respondents do not share their tie information, some respondents in the network are absent, or there is a cap on the maximum number of ties respondents are allowed to report, etc. The first step in dealing with missing ties is to assess the degree of missingness. For example, if a cap is placed on the number of ties respondents can nominate, then researchers can check the proportion of respondents who have nominated the maximum number of ties to gauge the degree of missingness. If that proportion is small, then the degree of missingness is likely small. Second, researchers should distinguish missing ties from no ties. No ties are coded as zeros in an adjacency matrix but missing ties should be treated as structural zeros in an ERGM to leave them unmodeled. Third, sensitivity analysis can be employed to assess the robustness of the results. An and Doan (2015), for instance, fit ERGMs on the full network and on the reduced networks where only the first few nominated ties per respondent were used to assess whether and how much missing ties impact the results. Lastly, researchers can impute missing ties. For example, one may fit ERGMs on the sub-network with no missing ties, use the estimates to impute missing ties, and then fit ERGMs on the full network with both observed and imputed ties. To note, this approach may not improve the estimates themselves but can help increase the precision of the estimates.

Omitted variable bias, collinearity, and endogeneity

Similar to traditional regression models, researchers must be diligent to include all relevant variables in an ERGM to avoid omitted variable bias in the estimates (Duxbury 2021b). Contextual

confounding is a particular concern because context (e.g., neighborhood characteristics) can affect both tie formation and explanatory variables. Meanwhile, including too many variables, especially if they are highly correlated, can lead to multicollinearity problems (Duxbury 2021a).

ERGMs also face an endogeneity issue if tie formation has a reverse effect on the covariates. Suppose happiness is one of the covariates in an ERGM. While happiness may positively affect tie formation, the reverse could also be true. To identify the causal effect of happiness on tie formation, researchers would need to leverage exogenous variation in happiness through instrumental variables (IVs) (Clarke and Windmeijer 2012) or experiments (Jiang et al. 2014).

Lastly, ERGMs are parametric models that require that the function linking explanatory variables to network formation is correctly specified. The estimates could be severely biased if the functional form is mis-specified. Hence, it would be valuable to explore nonparametric methods, such as matching to estimate counterfactual causal effects on network formation (e.g., by constructing counterfactual groups with same covariate values except the covariate of interest).

2.2 Modeling Egocentric Networks

Perry et al. (2018) offer a comprehensive guide to egocentric network analysis and thus we keep this section brief. Egocentric networks (or ego networks for short) are typically collected via surveys and consist of a focal node (ego) and the nodes to whom ego is directly connected to (alters). Therefore, when modeling ego networks using conventional regression analysis, the clustering of alter ties by egos must be taken into account. This can be done using clustered standard errors by egos (Comfort et al. 2021) or using multilevel regression models with egos at the higher level and alters at the lower level (Perry et al. 2018: 195). In some cases, egos also report ties between alters and thus there are both ego-alter ties and alter-alter ties. Researchers should cluster standard errors by both egos and alters or use cross-classified models with random effects for both egos and alters (Perry et al. 2018: 226; Snijders and Bosker 1999).

Recently, Krivitsky and Morris (2017) have extended ERGMs to model ego networks. Their approach, however, is currently only applicable to undirected ego networks with no alter-alter ties and does not account for any endogenous tie formation processes due to the data structure.

More often than modeling the ego networks themselves, ego network data are used to model the size, composition, or other network features of ego networks. In this case, the statistical problem reduces to conventional regression analysis and, accordingly, the standard challenges of causal inference in regression analysis naturally apply (e.g., confounding and endogeneity).

Compared to sociocentric network data, ego network data have two major advantages. First, it is more efficient to collect ego network data, because researchers do not need to survey all members in a particular group. Second, it is easier to draw random samples of egos and so

representative ego network data are easier to be collected than representative sociocentric network data where researchers have to randomly sample groups instead of individuals.

However, ego network data also face several limitations. First, many ties may be missing by design. For instance, in many ego network datasets, alter-alter ties, alter-ego ties, and indirect ties (alters with path length larger than 1) are usually missing by design. This limits the potential of using ego networks to study network statistics that rely on alter-initiated ties (e.g., reciprocity) or full network information (e.g., betweenness and closeness centrality measures, network density). To address this problem, researchers should use research designs that allow egos to report alter-initiated ties. They may also use ego network statistics to approximate or estimate sociocentric network statistics. For example, Everett and Borgatti (2005) and Marsden (2002) show that under certain conditions, egocentric centrality measures approximate the sociocentric measures well (e.g., betweenness). Smith and Gauthier (2020) use sampled ego network data to estimate global network features such as network density. Second, ego network ties are often recorded as undirected. Thus, only covariate main effects instead of directional effects can be studied. Third, there is probably more measurement error in ego network ties because the ties reported by egos are typically not validated by alters and thus egos may be more liberal in reporting their ties.

In Table 1 (for ERGMs) and Table 2 (for ego networks), we provide readers with concise summaries of the problems, solutions, and examples mentioned in this section.

3. Network Effects

We now turn to models that treat networks as a predictor to examine how social networks affect individual outcomes. Three types of network effects can be differentiated: 1) relational effects from possessing certain network ties; 2) positional effects from occupying certain network positions; and 3) structural effects from the overall network structure (VanderWeele and An 2013).

3.1 Relational effects

The literature presents two popular models for studying relational effects – the social capital model and the social contagion model.

The Social Capital Model

The social capital model examines how social ties provide access to social resources and social support (Cook 2014). Economic sociology has shown that social ties (e.g., knowing someone in a hiring organization) matter for getting a job or higher pay (Bian 1997; Dowd and Pinheiro 2013; Fernandez and Weinberg 1997; Granovetter 1973; Lin 2001; Pinheiro and Dowd 2009) while medical sociology has shown that social ties can provide various forms of social support (or stress) (Perry and Pescosolido 2015; Small 2013; Small and Sukhu 2016; Song 2011; Thoits 2011).

The social capital model typically takes the following form, where y_i represents an outcome of individual i, e.g., wage, and X_i represents of a series of control variables.

$$y_i = \beta_0 + \beta_1 \text{Social Capital}_i + \beta_2 X_i + \varepsilon_i \quad (4)$$

There are three broad critiques of the social capital model. One is how to measure social capital. There has been various measures of (individualized) social capital in social networks, ranging from the existence of a tie, the number and strength of ties (Bian 1997), to the diversity of ties (Uzzi 1999). Different survey instruments are in use to measure the specific resources available through an ego's network (e.g., name, position, and resource generators) (Molina et al. 2020). Selecting measures that have substantive meaning in a specific context is critical, as different measures may lead to different empirical results and levels of statistical significance.

The second critique is about confounding. People with more social capital may differ from those with less social capital in unobserved ways, which will bias the estimated effect of social capital (Mouw 2006). Exogenous variation in social capital, such as by randomization or through instrumental variables are necessary to address this issue.

The finial critique is about reverse causality. In particular with cross-sectional data, it is difficult to discern whether social capital causes the outcome or whether the outcome played some role in forming social capital. If longitudinal data is available, a lagged rather than contemporaneous effect of social capital can be estimated to rule out reverse causality (Shalizi & Thomas 2011; VanderWeele, Ogburn, and Tchetgen 2012). We summarize the critiques of the social capital model and possible solutions in Table 3.

The Social Contagion Model

The social contagion (or social influence or peer effect) model is used to examine whether and how social behaviors and beliefs can transmit through social ties (Friedkin and Johnsen 2011; Marsden and Friedkin 1993). A popular version of this model is the so-called "best friend" model, which is used to study whether an ego's best friend's outcome can socially transmit to the ego (An 2015a; Cohen-Cole and Fletcher 2008). For example, An (2015a) specifies the following model to examine a "best friend" effect on smoking, where *i* has nominated *j* as a best friend.

$$y_i = \beta_0 + \beta_1 y_j + \beta_2 X_i + \beta_3 X_j + \varepsilon_i \quad (5)$$

In this model, β_1 is called the endogenous peer effect, which measures direct social transmission of the outcome, and β_3 is called the exogenous peer effect, which measures how best friend's covariates can affect an ego's outcome (Manski 1993). The conceptual difference between

endogenous and exogenous peer effect is important insofar as the endogenous peer effect can give rise to a "social multiplier" or ripple effect (Boucher and Fortin 2015).

The social contagion model can be extended in several ways. One way is to use the average outcome of all possible peers (e.g., all students in a classroom) instead of just the best friend's outcome. This will lead to the so-called "linear-in-means" model (Manski 1993). Another extension is to use the average outcome of peers to which ego is connected in a network. This will lead to the spatial autoregressive model or the spatial lag model (Friedkin and Cook 1990; O'Malley and Marsden 2008). This approach also allows researchers to model the effect of indirectly connected peers in a network (Christakis and Fowler 2007) or the effects of structurally equivalent peers (e.g., peers with similar network structures) (Burt 1987, Moody & Mucha 2013).

The literature on social contagion has focused on estimating the causal endogenous peer effect and points out four broad challenges for causal inference. (1) Confounding due to peer selection. Rather than y_j causing y_i, their correlation is due to *i* selecting *j* as a friend based on their similarity in the outcome. The linear-in-means model may additionally suffer from a confounding negative correlation between egos and peers because egos, by design in this model, are not part of their own peer group (Caeyers and Fafchamps 2020). (2) Confounding due to omitted variables. This can include omitting the effects of shared contexts (e.g., neighborhood or school features), omitting the effects of common friends of i and j, and omitting relevant exogenous peer effects. (3) Simultaneity. For mutual friends, y_i can cause y_j while the latter also affects the former. This will lead y_j to be correlated with ε_i and cause simultaneity bias in the estimate of β_1. (4) Measurement error, both in the outcome and in the network structure.

There are two broad solutions to the issue of confounding (issues 1 and 2) in the social contagion model – leveraging exogenous variation or correcting for confounding. Exogenous variation can be leveraged through instrumental variables or specifically designed experiments. The instrumental variable strategy is to identify an exogenous variable that directly affects y_j and indirectly affects y_i through its effect on y_j. For example, An (2015a) uses the smoking environment of the best friend as instrument variables for the best friend's smoking status. Bramoullé at al. (2009) use the status of the indirect friend in a transitive triad as an instrument for the directly connected friend's status. O'Malley et al. (2014) use the genetic factors of the best friend as instruments for the best friend's outcome. Estrada et al. (2021) and Reza et al. (2021) use exogenous networks to instrument for the endogenous network.

Experiments with special designs can also be used to identify peer effects (An 2011; VanderWeele and An 2013). The first type of design utilizes randomized peer treatment, where researchers randomly assign treatment to alters but not to egos (An 2015b). If the treatment effect is then observed to diffuse from alters to egos, there is proof of peer effects. An issuewith this design is that egos might be affected by multiple alters simultaneously and the treatment of alters

might interfere with each other, which makes it difficult to cleanly estimate peer effects (see An (2018) for a strategy to address this issue). Another issue with this design is that treatment may, in some cases, alter the underlying social network, leading to a recursive effect on the outcome (An 2015b; Comola and Prina 2021).

The other type of experimental design to identify causal peer effects is the random assignment of peers. Students, for instance, have been randomly assigned to desks (Rohrer, Keller, and Elwert 2021), classrooms (Carrell, Fullerton, and West 2009), or dorm rooms (Sacerdote 2001). Further, quasi-randomizations of social ties based on deaths, expulsions, relocations, or natural disasters have been leveraged in prior work (e.g., Mohnen 2021; Waldinger 2012; Carvalho et al. 2021). A limitation of this design is that an ego's outcome can be affected by multiple behaviors of the randomized alters. Hence, to identify casual peer effects on one behavior, researchers need to control for other competing behaviors of the alters. Moreover, shared environmental factors may affect egos' and alters' behaviors, which are not controlled in this design.

The second broad solution to the issue of confounding are correction strategies, which are employed in the absence of exogenous variation. First, to address peer selection, researchers have proposed modeling tie formation in a first stage and then correcting for peer selection in the network effect model (second stage) using a type of Heckman selection model (Arduini et al. 2015; Goldsmith-Pinkham and Imbens 2013; Hsieh and Lee 2016) or a matching approach (Aral et al. 2009). The state-of-the-art method is to use ERGMs to model tie formation in the first stage to account for complex tie formation processes (An 2011; Hsieh, Lee, and Boucher 2020). Researchers have also used lagged outcomes for both egos and alters to account for peer selection (Christakis and Fowler 2007). Shalizi and Thomas (2011) argue that controlling for lagged outcomes may not be sufficient to eliminate peer selection due to homophily antecedent to the lagged period. Boucher and Fortin (2015), however, show that with a rich set of controls, the impact of latent homophily may be small.

Second, to address contextual confounding, researchers have proposed controlling for the outcomes of neighbors (Christakis and Fowler 2007) or neighborhood characteristics (Block 2011) or using fixed effects model to account for contextual fixed effects (Cohen-Cole and Fletcher 2008; Fortin and Yazbeck 2015). Some researchers criticize the fixed effects model because the within-estimator can generate correlations between the lagged outcomes and the transformed disturbance (Nickell 1981), which may bias the endogenous peer effect estimate.

A third approach to account for peer selection with observational data is to jointly model the evolution of networks and behavior over time with the stochastic actor-oriented model (SAOM) (Snijders 2001, 2011; Steglich, Snijders, and Pearson 2010). SAOMs model network and behavior dynamics with two stochastic processes. The network process is akin to a dynamic ERGM and accounts for peer selection (e.g., homophily). The behavioral process is akin to the social contagion

model in which exogenous and endogenous peer effects can be specified. If the model captures the underlying processes well, SAOM structurally separates selection from influence effects by jointly estimating their effects. Prior work has examined the statistical properties of SAOM (Block et al. 2018; Leifeld and Cranmer 2019) and applied it in various contexts (An 2015b; Schaefer, Haas, and Bishop 2012). Despite its capabilities, SAOM has several limitations: it is computationally expensive as it relies on Monte Carlo Markov Chains for estimation; it makes strong behavioral assumptions (e.g., that actors have full information of the network when making decisions); and omitted variable bias (including contextual confounding) can still bias the estimates.

To circumvent the simultaneity problem (issue 3), researchers can estimate a lagged rather than a contemporaneous peer effect or utilize exogenous variation in peers' behaviors (as provided by instrumental variables or experiments). In the linear-in-means model, the problem of simultaneity additionally causes the "reflection problem" (Manski 1993), namely, the impossibility of separating endogenous from exogenous effects because the mean of peer outcomes and the mean of peer covariates are linearly dependent. Several solutions to the reflection problem have been proposed, including utilizing variation in group size (Davezies, D'Haultfoeuille, and Fougère 2009; Lee 2007), variation in individual-specific network structure (Bramoulle et al. 2009), or via parameter constraints (Arcidiacono et al. 2012; Graham 2008; Rose 2017).

Measurement error (issue 4) in the outcome can be double detrimental as the outcome appears on both sides of the model. It may thus not only attenuate the estimated peer effect but also reduce its precision, making it more difficult to reject the null of no peer effect (An 2015a). Measurement error in network ties can bias the peer effect estimate (Chandrasekhar and Lewis 2011; Micklewright, Schnepf, and Silva 2012). To reduce measurement error, the general advice is to improve the measurement itself (e.g., by refining the survey instruments) or to combine measures from separate reports (An and Doan 2015; An and Schramski 2015).

There are still several open issues in this area of study. In terms of substantive progress, more work is needed to uncover the heterogeneity of peer effects, such as by ego and alter characteristics (e.g., race and gender) and by ego-alter dyadic characteristics (e.g., friendship duration) (Cools, Fernández, and Patacchini 2019; An 2022). More work is also needed to identify the mechanisms underlying peer effects (An 2015a; DiMaggio and Garip 2012; Lin 2001). In terms of methodological progress, more work is needed to provide standard errors that can account for outcome dependence across units beyond social contagion (Advani and Malde 2018b; An 2018, 2021; Lee and Ogburn 2020). Finally, more research is also needed to explore nonparametric identification of network effects (Egami 2020; Ogburn, Sofrygin, et al. 2020; Ogburn, Shpitser, and Lee 2020; Zeleneev 2020).

The threats to causal inference and possible solutions with the social contagion model are summarized in Table 4.

3.2 Positional effects

Positional effects refer to whether and to what degree the position that individuals occupy in a network affects their outcomes. Prior work has identified popularity and isolation effects (Copeland, Bartlett, and Fisher 2017; Schaefer et al. 2010). Other work has demonstrated effects of brokerage positions that bridge units in social networks (Burt 1995; Cornwell 2009; Everett and Valente 2016; Gould and Fernandez 1989; Greenberg 2021), to give a few examples.

In terms of modeling, studying the causal effects of network positions amounts to replacing the social capital indicators with positional measures in the social capital model. Accordingly, network endogeneity (e.g., individuals occupying special positions in a network select into these positions) and contextual confounding (e.g., variables that determine both network positions and the outcome are omitted) are the two major obstacles of causally identifying positional effects.

3.3 Structural effects

Individual outcomes can also be influenced by the macro-structural features of a network, such as cohesion, hierarchy, clustering, and composition. Cohesion describes how densely connected a network is and is measured by the density coefficient or mean geodistance (i.e., the shortest path between two nodes) of a network (Moody and White 2003). Hierarchy describes the extent to which network ties are unevenly distributed across actors and is typically measured by the centralization coefficient and reciprocity coefficient of a network. For example, prior work has shown that more centralized networks facilitate simple diffusion for which one-time exposure is sufficient for adoption (Barrat, Barthélemy, and Vespignani 2008). Clustering describes the degree to which network ties are concentrated in local actors and is measured by the global or local clustering coefficients of a network. It has been shown that local clustering facilitates complex diffusion that requires reinforcement from multiple contacts (Centola 2010). Network compositions (e.g., in terms of race, sex, age and other factors) can also affect individual outcomes (DiMaggio and Garip 2012; McFarland et al. 2014).

Randomization tests have been used to assess whether a network exhibits certain structural features. This can be done by randomly permuting the rows and/or columns of the adjacency matrix and compare selected features of the observed network against the randomized networks (Baldassarri and Diani 2007; Bearman, Moody, and Stovel 2004).

To study causal structural effects, one may use the structural features to replace the social capital indicators in the social capital model. Hence, similar critiques apply. In particular, contextual confounding and reverse causality (i.e., individuals affecting the network structures) are of concern. To improve statistical inferences, one may use a multivariate regression model to account for outcome correlations across units (An 2018, 2021).

4. Conclusion and discussion

Empirical models and methods for studying how social networks are formed and how social networks influence individual outcomes have made tremendous progress in the last decade. However, some of the models and methods are difficult enough to implement that they are currently only accessible to methodologists, hindering their acceptance and applications in the board research community. Hence, more work is needed to help disseminate these knowledge. The field also needs more work to understand how the different models and methods perform under varying degrees of network endogeneity, contextual confounding, and measurement error.

We identify four areas that are critical for future development of causal network analysis. First, more accurate measurement of network ties is crucial to research on both network formation and network effects. More precision in the meaning of ties and use of multiple sources of data including reports from multiple respondents, timed interaction data, social media data, and multiplex tie information can help improve measurement of network ties (An and Doan 2015; An and Schramski 2015; Kitts and Quintane 2020). Moreover, future research should employ sensitivity analysis (An 2015a; VanderWeele 2011) and other methods to address uncertain or missing ties (Fisher 2019).

Second, more work is needed to understand the mechanisms underlying network formation and network effects. Regarding network formation mechanisms, Wimmer and Lewis (2010) argue that endogenous tie formation processes can be competing or mediating mechanisms for covariate effects. Future work is needed to distinguish between these two different types of mechanisms. Moreover, the mechanisms underlying tie dissolution should be examined more as they can differ from tie formation mechanisms (McDermott, Fowler, and Christakis 2013). Regarding mechanisms for network effects, An (2015a), for instance, shows that sharing cigarettes can be one potential mechanism for peer influence on smoking. More of such work will aid our understanding of how behavioral imitation, normative pressure, and social exchange can explain social contagion. In addition, future research may study network effects by including relational, positional, and structural effects in one integrated model to examine how these effects simultaneously operate.

Third, more experimental work (especially with the random treatment design) is needed to identify causality in network analysis and triangulate findings across different settings. Network experiments may also help to design more effective interventions (An 2015b).

Lastly, more work is necessary to model and explain the tremendous heterogeneity in network formation and network effects. This includes allowing modelled effects to change across time, space, covariate values, different types of ties, and different regions in a network. Theories on network ecology (Entwisle et al. 2007; McFarland et al. 2014; Small 2007) may help researchers to explore certain causes of such heterogeneity.

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Table 1. Causal Inference Threats to Exponential Random Graph Models and Selected Solutions

Problems	Selected Solutions	Examples
A. Measurement error	(1) Improve survey instruments. (2) Combine multiple sources of data.	An & Schramski (2015) and Krackhardt (1987) use multiple-reports to impute the true network ties. An & Doan (2015) use peers' observations to refine the self-reported smoking measure.
B. Missing ties	(1) Distinguish missing ties from none ties. (2) Sensitivity analysis. (3) Impute missing ties.	Missing ties should be treated as structural zeros in ERGMs. Researchers can check if results from ERGMs with randomly removed ties (An 2015a) or based on the first few nominated ties (An & Doan 2015) are similar to the original ERGMs. Researchers can fit an ERGM on the sub-network with no missing ties and then impute the missing ties based on the estimated ERGM.
C. Reverse causality	(1) Utilize some exogenous variation in the predictor if available. (2) Use lagged predictor.	Researchers may use experiments to introduce exogenous variation in the predictor of interest (Jiang et al 2015).
D. Multicollinearity	Build the model from simple to complex. Include fewer endogenous tie formation processes.	Duxbury (2021a) provides examples for diagnosing multicollinearity in ERGMs.
E. Omitted variables bias	Use instrumental variables or experiments to introduce exogenous variation in the predictor.	Clarke and Windmeijer (2012) describe a series of instrumental variables methods for binary outcomes. The methods are generally applicable to ERGMs.

Table 2. Causal Inference Threats to Ego Network Models and Selected Solutions

Problems	Selected Solutions	Examples
A. Clustering by egos when there are only ego-alter ties.	(1) Cluster standard errors by egos. (2) Multilevel regression models.	For clustered standard errors by egos, see Comfort et al. (2021). Perry et al. (2018) use multilevel models with egos at the higher level and alters at the lower level.
B. Clustering by egos and alters when there are both ego-alter ties and alter-alter ties.	(1) Cluster standard errors by both egos and alters. (2) Cross-classified models with random effects for both egos and alters.	Please see Perry et al. (2018) and Snijders and Bosker (1999) for examples of cross-classified models.
C. Missing ties.	(1) Use research designs that allow egos to report alter-initiated ties. (2) Use ego network statistics to approximate or estimate sociocentric network statistics. (3) Sensitivity analysis.	Everett and Borgatti (2005) and Marsden (2002) show that egocentric centrality measures may approximate the sociocentric measures well. Smith and Gauthier (2020) use sampled ego network data to estimate global network features such as network density. Researchers can check the robustness of their results by randomly removing certain ties (An 2015a) or based on the first few nominated ties (An & Doan 2015).
D. Other problems such as measurement error, omitted variable bias, and endogeneity.	Consult Table 1 for similar solutions.	

Table 3. Causal Inference Threats to the Social Capital Model and Selected Solutions

Problems	Selected Solutions	Examples
A. Measurement of social capital: Different measures can lead to different results	Selecting a measure that is substantively meaningful and accurately measurable in a specific context.	See Bian (1997), Molina et al. (2020), and Uzzi (1999) for selected measures and survey instruments of social capital.
B. Confounding: Social capital may be correlated with other unobserved covariates.	Utilize exogenous variation in social capital created by experiments or instrumental variables.	Random assignment of social ties (Carrell et al. 2009; Rohrer et al. 2021; Sacerdote 2001) or quasi-randomizations of social ties (Carvalho et al. 2021; Mohnen 2021; Waldinger 2012).
C. Reverse causality: The outcome affects social capital.	Use lagged social capital as a predictor to rule out reverse causality.	See Shalizi & Thomas (2011) and VanderWeele et al. (2012) for examples.

Note: Please also consult Table 4 for other related problems and similar solutions.

Table 4. Causal Inference Threats to the Social Contagion Model and Selected Solutions

Problems	Selected Solutions	Examples
A. Confounding due to peer selection	(1) Utilize exogenous variation in peer's behaviors provided by instrumental variables or experiments. (2) Model peer selection and control for it in the social contagion model.	For the instrumental variables approach, see An (2015a), Bramoulle et al. (2009), and O'Malley et al. (2014). For randomized peer treatment, see An (2015b). For random assignment of peers, see Carrell et al. (2009), Rohrer et al. (2021) and Sacerdote (2001) and for quasi-randomizations of peers, see Carvalho et al. (2021), Mohnen (2021), and Waldinger (2012). For the correction approaches, see Aral et al. (2009), Hsieh and Lee (2016), the dynamic panel model, (Christakis and Fowler 2007), or the stochastic actor-oriented model (Steglich et al. 2010; Snijders 2011).
B. Confounding due to omitted variables (e.g., contextual factors)	(1) Utilize exogenous variation in peers' behaviors provided by instrumental variables or experiments. (2) Control for contextual confounding directly or statistically.	For the approaches of instrumental variables and experiments, see part A of this table. To address contextual confounding, researchers have proposed controlling for outcomes of neighbors (Christakis and Fowler 2007) or neighborhood characteristics (Block 2011) or using fixed effects model to account for contextual fixed effects (Cohen-Cole and Fletcher 2008; Fortin and Yazbeck 2015).
C. Simultaneity bias	(1) Utilize exogenous variation in peer's behaviors provided by instrumental variables or experiments. (2) Use lagged peers' behaviors.	For the approaches of instrumental variables and experiments, see part A of this Table. For estimating the lagged peer effect, see Yeung and Nguyen-Hoang (2016) and VanderWeele et al. (2012).
D. Measurement error	(1) Improve survey instruments. (2) Combine multiple sources of data.	An & Schramski (2015) and Krackhardt (1987) use multiple-reports to impute the true network ties. An & Doan (2015) use peers' observations to refine the self-reported smoking measure.