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# Multilevel meta network analysis with application to studying network dynamics of network interventions

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ABSTRACT

In this paper, I introduce new methods for multilevel meta network analysis. The new methods can combine results from multiple network models, assess the effects of predictors at network or higher levels and account for both within- and cross-network correlations of the parameters in the network models. To demonstrate the new methods, I studied network dynamics of a smoking prevention intervention that was implemented in 76 classes of six middle schools in China. The results show that as compared to random intervention (i.e., that targets random students), smokers' popularity was significantly reduced in the classes with network interventions (i.e., those target central students or students with their friends together). The findings highlight the importance of examining network outcomes in evaluating social and health interventions, the role of social selection in managing social influence, and the potential of using network methods to design more effective interventions.



## 1. Introduction

In a seminal paper, Snijders and Baerveldt (2003) describe meta-analysis methods for combining results from multiple network models. The methods consist of two steps. In the first step, a network model (e.g., the Exponential Random Graph Model) is fitted on multiple networks. In the second step, the estimated parameters from the multiple networks are combined via meta analysis. Such meta network analysis can not only provide inferences on the population averages of the estimated parameters, but also test the equality or joint significance of the estimated parameters across the networks.

In light of the latest advances in meta analysis (e.g., Viechtbauer, 2010; White, 2011; Gasparrini et al., 2012), the methods documented in Snijders and Baerveldt (2003), however, may be updated from several aspects. First, the methods can be extended to incorporate network level and higher levels of predictors. This extension essentially converts simple meta-analysis to multilevel meta-regressions. The extension is important because it helps to provide a more complete characterization of social and network processes. If the network models in the first step account for network dependence, including appropriate higher levels of predictors helps to adjust for spatial dependence (e.g., area), larger group dependence without specific dependence structure (e.g., school), or differences in other network characteristics (e.g., treatment status). This extension is particularly important, if the research interest is examining the effects of network level or higher levels of predictors.

Second, the meta network analysis may be extended to incorporate cross-network variations in the estimated parameters. Previous meta network analysis, maybe except the Fisher's method for combining independent *P*-values (Snijders and Bosker, 2012; Ripley et al., 2014), mostly assumes that the estimated parameters for a particular variable in the network models are generated by a common effect. This fixed effect assumption holds well when the networks can be viewed as being sampled from the same population. However, it may not hold when there are important characteristics that differ across networks and are unaccounted for in the network models. In such cases, it may be more appropriate to assume that the estimated parameters for a variable come from different underneath effects. For parsimonious reasons, however, these different underneath effects can be assumed to come from the same distribution. This new assumption leads to what is so-called the random effects model. It can help to examine cross-network variations in the estimated parameters in the network models.

Third, previous univariate meta network analysis may be extended to multivariate cases. First, the multivariate fixed effects model can help to account for within-network correlations in the estimated parameters. For example, active actors (i.e, those nominate a lot friends) also tend to be popular actors (i.e, those receive a lot friend nominations). Previous univariate meta network analysis assumes such correlations are zero while in contrast,

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the multivariate fixed effects model utilizes the covariance matrix of the estimated parameters in the network models to facilitate estimating the underneath effects in the meta analysis. Extending meta analysis to the multivariate cases is also important because sometimes the estimated parameters in the network models may be correlated across networks. This can result from, for example, spillover effects of implemented interventions, etc. The multivariate random effects model can help to account for cross-network correlations in the estimated parameters in such cases.

In this paper, I introduce the latest advances in meta analysis for multilevel network research and provide an overview of multilevel meta regressions in both univariate and multivariate cases and in both fixed effects and random effects models. To demonstrate the new methods, I applied them to studying network dynamics of a smoking prevention intervention that was implemented to students from 76 classes of six middle schools in China. The 76 classes were randomly assigned into one of four treatment conditions: control condition in which no students received the intervention, random intervention in which a quarter of students were randomly selected to participate in the intervention, central intervention in which a quarter of central students (i.e., those received a lot friend nominations from their classmates) were selected to participate in the intervention, and group intervention in which a quarter of students and their close friends were selected to participate in the intervention. The goal is to study whether network dynamics related to smokers significantly differ between the random intervention and the network interventions (i.e., both the central intervention and the group intervention). More specifically, it is hypothesized that smokers would become less popular in the network interventions than in the random intervention, as the treated students in the network interventions had more leverages to sever their ties to smokers if they choose to do so.

During data analysis, first I fit a stochastic actor-oriented model (SAOM) (Snijders, 2001; Steglich et al., 2010) on the friendship network in each class in order to characterize the network dynamics before and after the intervention. In the second step, I use multilevel meta-regressions to examine the effects of network interventions in contrast to the random intervention. The univariate meta-regressions show that network interventions (including both the central intervention and the group intervention) are more effective than the random intervention in reducing smoker's popularity. Friendship ties directed to smokers in the network intervention classes are only about half as likely to continue as those in the random intervention classes. Results of the multilevel multivariate regressions show similar patterns. But the evidence is probably more robust for the central intervention than for the group intervention. Overall, both the meta network analysis and the substantive findings in this paper shed lights on future network studies.

This paper proceeds as follows. In Section 2, I introduce the multilevel meta-regressions for meta network analysis. In Section 3, I describe the data and the analytical strategies used to demonstrate the multilevel meta network analysis. Section 4 presents the empirical results. Last, I conclude.

## 2. Models for multilevel meta network analysis

### 2.1. Multilevel univariate meta-regressions

One approach to extending the univariate meta network analysis is to specify a multilevel model that can include network level (or even higher levels) of predictors. Incorporating these predictors is important because it helps to account for special dependence in the data that goes beyond network dependence. It is particularly important if the research interest is assessing the effects of network or high levels of predictors. More formally, this extension can be expressed as follows:

$$\hat{\theta}_{ki} = \theta_i + \boldsymbol{x}'_{\boldsymbol{k}}\boldsymbol{\beta_i} + e_{ki}, \tag{1}$$

where it is assumed that $I$ estimated parameters are available from each of $K$ networks, $\hat{\theta}_{ki}$ denotes the $i$th estimated parameter in the $k$th network, $\theta_i$ a common effect (or population-average) for the $i$th estimated parameter, $\boldsymbol{x_k}$ a $(p \times 1)$ vector containing the $p$ dimensions of characteristics of the $k$th network, $\boldsymbol{\beta_i}$ a $(p \times 1)$ vector of coefficients reflecting the associations of the network characteristics with the $i$th estimated parameter, and $e_{ki}$ an error term with a zero mean and a variance that equals the variance of the $i$th estimated parameter $\hat{\sigma}^2_{ki}$. Assuming independence and normality of the error terms, the model can also be expressed as:

$$\hat{\theta}_{ki} \sim \text{Normal}(\theta_i + \boldsymbol{x}'_{\boldsymbol{k}}\boldsymbol{\beta_i}, \hat{\sigma}^2_{ki}). \tag{2}$$

In words, the $i$th estimated parameter in the $k$th network is assumed to follow a Normal distribution with a mean of $(\theta_i + \boldsymbol{x}'_{\boldsymbol{k}}\boldsymbol{\beta_i})$ and a variance of $\hat{\sigma}^2_{ki}$. Since the estimated parameters are assumed to have been generated by a common effect, formulation (2) is often called the fixed effects meta-regression. The statistical problem is to estimate $\theta_i$ and $\boldsymbol{\beta_i}$ with information on $\boldsymbol{x_k}$, $\hat{\theta}_{ki}$, and $\hat{\sigma}^2_{ki}$. Recall that both $\hat{\theta}_{ki}$, and $\hat{\sigma}^2_{ki}$ are assumed known from the network models in the first step analysis.

Formulation (2) can be revised to account for the fact that the underneath effect for each estimated parameter is not a fixed quantity, but a random quantity that follows a hyper-distribution.

$$\hat{\theta}_{ki} \sim \text{Normal}(\theta_i + \boldsymbol{x}'_{\boldsymbol{k}}\boldsymbol{\beta_i}, \hat{\sigma}^2_{ki}), \text{ where } \theta_i \sim \text{Normal}(\mu_i, \upsilon^2_i) \tag{3}$$

Or, in a compact way,

$$\hat{\theta}_{ki} \sim \text{Normal}(\mu_i + \boldsymbol{x}'_{\boldsymbol{k}}\boldsymbol{\beta_i}, \hat{\sigma}^2_{ki} + \upsilon^2_i), \tag{4}$$

where $\mu_i$ is the mean of the underneath effects for the $i$th estimated parameter and $\upsilon^2_i$ measures the between-network variation of the estimated parameter. Correspondingly, this model represents the random effects meta-regression. The statistical problem is to estimate $\mu_i$, $\boldsymbol{\beta_i}$, and $\upsilon^2_i$ with information on $\boldsymbol{x_k}$, $\hat{\theta}_{ki}$, and $\hat{\sigma}^2_{ki}$.

### 2.2. Multilevel multivariate meta-regressions

Both the fixed effects and random effects models aforementioned can be extended to multivariate cases. Unlike the univariate meta-regressions, multivariate meta-regressions do not assume independence of the estimated parameters in each network. In the multivariate fixed effects model, the estimated parameters in the $k$th network are assumed to follow a multivariate normal distribution of dimension $I$ (i.e., the number of shared parameters in the network models).

$$\boldsymbol{\hat{\theta}_k} \sim \text{Normal}_{\boldsymbol{I}}(\boldsymbol{\theta} + \boldsymbol{X}'_{\boldsymbol{k}}\boldsymbol{\beta}, \boldsymbol{\Sigma_k}), \tag{5}$$

where $\boldsymbol{\hat{\theta}_k}$ represents a $(I \times 1)$ vector of the estimated parameters in the $k$th network, $\boldsymbol{\theta}$ a $(I \times 1)$ vector containing the common effects for the parameters in the network model, and $\boldsymbol{X_k}$ a $(Ip \times I)$ block matrix derived from the Kronecker product of an identity matrix of dimension $I$ and the characteristics of the $k$th network $\boldsymbol{x_k}$ (Gasparrini et al., 2012). The $(Ip \times 1)$ vector $\boldsymbol{\beta}$ represents the associations between the network characteristics and the estimated parameters. Last, $\boldsymbol{\Sigma_k}$ is the $(I \times I)$ variance-covariance matrix of the estimated parameters in the $k$th network. The statistical problem is to estimate $\boldsymbol{\theta}$, $\boldsymbol{\beta}$ with information on $\boldsymbol{\hat{\theta}_k}$, $\boldsymbol{X_k}$, and $\boldsymbol{\Sigma_k}$.

Sometimes the underneath effects $\boldsymbol{\theta}$ may be correlated across networks. In such cases, a multivariate random effects model may be more appropriate.

$$\boldsymbol{\hat{\theta}_k} \sim \text{Normal}_{\boldsymbol{I}}(\boldsymbol{\theta} + \boldsymbol{X}'_{\boldsymbol{k}}\boldsymbol{\beta}, \boldsymbol{\Sigma_k}), \text{ where } \boldsymbol{\theta} \sim \text{Normal}_{\boldsymbol{I}}(\boldsymbol{\mu}, \boldsymbol{\Omega}) \tag{6}$$

In this model, each parameter is assumed to have been generated from different underneath effects. These underneath effects, however, are assumed to have been generated from the same distribution with mean $\boldsymbol{\mu}$ and covariance $\boldsymbol{\Omega}$. The matrix $\boldsymbol{\Omega}$ represents the between-network covariation of the underneath effects. Formulation (6) can also be condensed and expressed in a different way.

$$\hat{\boldsymbol{\theta}}_{\boldsymbol{k}} \sim \text{Normal}_{\boldsymbol{I}}(\boldsymbol{\mu} + \boldsymbol{X}'_{\boldsymbol{k}}\boldsymbol{\beta}, \boldsymbol{\Sigma}_{\boldsymbol{k}} + \boldsymbol{\Omega}) \tag{7}$$

The statistical problem is to estimate $\boldsymbol{\mu}$, $\boldsymbol{\beta}$, and $\boldsymbol{\Omega}$ with information on $\hat{\boldsymbol{\theta}}_{\boldsymbol{k}}$, $\boldsymbol{X}_{\boldsymbol{k}}$, and $\boldsymbol{\Sigma}_{\boldsymbol{k}}$.

### 2.3. Model comparison and estimation

As stated above, fixed effects models assume there is a common underneath effect for each set of parameters in the network model. This is reasonable when the networks can be viewed as being drawn from the same population. However, when the networks are collected under heterogeneous conditions, the underneath effects may differ across networks (while they may still follow a common distribution). In such cases, random effects models may be preferred.

In practice, it is often a good idea to fit both random effects and fixed effects models and examine how much the estimates differ. There are three statistics that are useful for model selection. The first is an overall model fitness measure, like Akaike's Information Criterion (AIC) or Bayesian Information Criterion (BIC). In general, the smaller a information criterion is, the better the model fits the data. The second is Cochran $Q$ test for residual heterogeneity. The test statistics is $Q = \sum_k \hat{e}'_k \Sigma_k^{-1} \hat{e}_k$, where $\hat{e}_k$ containing the residuals in the fixed effects model of the $k$th network (Gasparrini et al., 2012). This statistic can be used to test the null hypothesis that the estimated parameters are equal across networks or the covariance matrix (i.e., $\boldsymbol{\Omega}$) is a zero matrix in the multivariate models (Gasparrini et al., 2012). Rejecting the null hypothesis favors the random effects model. Third, the $I^2$ statistic, defined as $(1 - \frac{K-p-1}{Q})$, shows the proportion of variation in the estimated parameters across networks that is attributable to heterogeneity rather than sampling error (Higgins and Thompson, 2002; Higgins et al., 2003; Gasparrini et al., 2012). The larger the $I^2$ statistic is, the more preferred the random effects model is.

Unlike univariate meta-regressions, multivariate meta-regressions do not assume the covariances of the estimated parameters in each network are zero. Instead, they exploit the variance-covariance matrix of the estimated parameters in the network models to estimate the coefficients of the underneath effects in the meta analysis. Sometimes this can lead to efficiency gains. But the computation in multivariate models tends to be time-consuming and instable, especially when it involves a large number of estimated parameters. Thus in conducting multivariate meta-regressions it is often a good idea to focus on only a small subset of the estimated parameters in the network models.

The fixed effects meta-regressions can be estimated by generalized least squares. The random effects meta-regressions can be estimated by maximum likelihood, restricted maximum likelihood, methods of moments, or method of variance components. These estimation methods have been detailed in prior literature (e.g., White, 2011; Gasparrini et al., 2012) and implemented in statistical packages such as "metafor" (Viechtbauer, 2010) and "mvmeta" (Gasparrini et al., 2012) in R and "mvmeta" in Stata (White, 2011). Also, the "RSiena" package in R has recently introduced a Bayesian method for random coefficient meta analysis (Ripley et al., 2014). In short, interested readers can consult the references listed here for more details of the estimation methods.

## 3. Data and analytical strategies

### 3.1. Data

To demonstrate the multilevel meta network analysis, I applied them to analyzing network dynamics of network interventions. The data comes from a two-wave survey of over 4000 students from 76 classes in six middle schools in China, conducted between November, 2010 and February, 2011. In both surveys, students were asked to provide information about up to ten of their closes friends in their school. Since the unit of analysis is class in this study, only close friendships within a student's classroom are used to construct the friendship networks at the two time points.

Between the two surveys, a smoking prevention intervention with a partial treatment design was implemented. Specifically, within each of the three grades (7, 8, and 9) of each school, four comparable classes were randomly assigned into one of four treatment conditions: control condition in which none of the students received the intervention, random intervention in which a quarter of random students were selected to receive the intervention, central intervention in which a quarter of central students (i.e., those received a lot friend nominations from classmates) were selected to participate in the intervention, and group intervention in which a quarter of students and their close friends were selected to participate in the intervention.[1]

Fig. 1 depicts the partial treatment design. Such a design is appropriate because it helps to evaluate a well-known conjecture in social network analysis, namely, whether treating a small number of carefully selected subjects can spread the benefits of social interventions to a larger group (Sobel, 2006; Valente, 2012). In addition, unlike previous network interventions (e.g., Kelly et al., 1991; Latkin, 1998; Campbell et al., 2008), this study includes a random intervention. Thus unlike previous studies that contrasted network interventions with only the control condition, this study can benchmark network interventions against random intervention and so can provide a fairer evaluation of the effectiveness of network interventions.

The smoking prevention intervention consisted of two waves. In the first wave (implemented in December, 2010), brochures containing information about the negative effects of smoking were distributed to the selected students in each selected class. In the second wave (implemented in January, 2011), two health professionals held a workshop in each school (except the second) presenting to the treated students the health and economic cost of smoking. Since the intervention was mostly informational, presumably it might not have much impact of changing student's smoking behavior. But it is an empirical (and under-explored) question whether the intervention would have discernible impacts of changing student's friendship networks. In particular, if the students became more aware of the negative effects of smoking, would they be motivated to consolidate their ties to smokers? If so, since the treated students in the network interventions have more leverages to manage their ties to smokers than their counterparts in the random intervention, would smokers in the network interventions become less popular than their counterparts in the random intervention? These considerations lead to the following central hypothesis.

**Central hypothesis**: Compared to smokers in the random intervention classes, smokers in the network intervention classes are less likely to be nominated by others as friends. This is possible because the treated students in the network interventions can more

[1] The ninth grade of the third school has 4 additional classes participating in the experiment. Thus in total 76 classes participated in the experiment, of which 57 received the intervention.

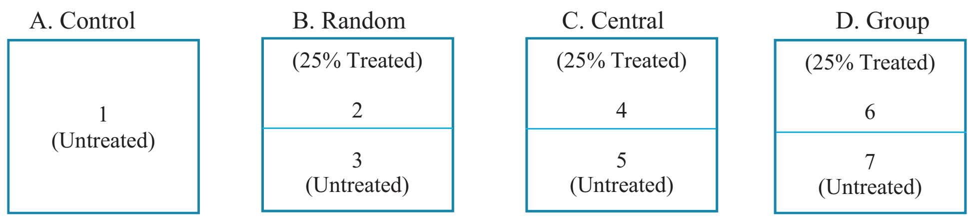


**Fig. 1.** The partial treatment design. *Note*: Groups 2, 4 and 6 correspond to the students who received the intervention in the random, central, and group conditions, respectively, while groups 1, 3, 5 and 7 are their classmates who did not receive the intervention.

easily sever their connections to smokers (due to their relative high centrality in the friendship networks) than their counterparts in the random intervention classes.

In addition, I also examine whether network interventions are more effective than random intervention in de-motivating smokers to connect to others and in maintaining or creating homophilious ties between students with the same smoking status.

### 3.2. Analytical strategies

#### 3.2.1. Modeling network dynamics through SAOM

The analysis is divided into two steps. In the first step, I fitted a stochastic actor-oriented model (Snijders, 2001; Steglich et al., 2010) on the friendship network in each class. The computation was done via the "RSiena" package (Ripley et al., 2014) in R. The SAOM included the following three functions to depict friendship formations (Snijders, 2001; Steglich et al., 2010; Ripley et al., 2014).

$$\text{Evaluation function}: f_j^{\text{net}}(w, w', z, ) = \sum_i \beta_i^{\text{net}} S_i^{\text{net}}(j, w, w', z), \quad (8)$$

$$\text{Maintenance function}: m_j^{\text{net}}(w, w', z, ) = \sum_i \zeta_i^{\text{net}} S_i^{\text{net}}(j, w, w', z), \quad (9)$$

$$\text{Creation function}: c_j^{\text{net}}(w, w', z, ) = \sum_i \gamma_i^{\text{net}} S_i^{\text{net}}(j, w, w', z), \quad (10)$$

where $w$ represents the current network and $w'$ a new network that student $j$ can choose to form, $z$ the covariates, and $i$ the number of parameters in the model. Thus, student $j$ chooses a tie to change in order to maximize the objective function. The evaluation function depicts the overall probability of tie formation. The maintenance function (or the endowment function as called in the "RSiena" manual) depicts the probability of the maintenance of existing ties. The creation function depicts the probability of creating new ties. To avoid collinearity, no more than two of the three functions can be simultaneously included in the SAOM for the same attribute or dynamics. The SAOM imposes that actor $j$ chooses the parameter values that maximize the tie-formation functions, respectively.

In each SAOM, I included five terms to account for basic network dynamics such as density, reciprocity (i.e., ties are more likely to be reciprocated over time), the number of transitive triplets (i.e., friends of friends are more likely to be friends over time), indegree-popularity (i.e., popular students become more popular over time), and outdegree-popularity (i.e., students who have nominated many friends become more active to do so over time). I also included alter effects and homophily effects for a number of demographic and social attributes, such as sex (1 = boy; 0 = girl), height, academic ranking (1 = ranked top twenty in the class; 0 = otherwise), personality (1 = optimistic; 0 = not optimistic), family economic condition (1 = good; 0 = not good), and treatment status (1 = treated; 0 = untreated).[2] A positive estimate of the alter effect for an attribute indicates that students with that attribute receive more friend nominations over time than those without the attribute. A positive estimate of the homophily effect indicates that students with the same attribute are more likely to be friends over time than those with different attributes. Smokers are defined as those who regularly smoke or who have recently smoked.[3] To better capture the network dynamics surrounding smokers, I separated the network dynamics related to smokers into two parts: maintenance of old ties and creation of new ties. Besides alter effects and homophily effects, I also included ego effects for smokers. A positive estimate of the ego effects indicates that smokers are more likely to nominate others as friends over time than nonsmokers.[4] The estimates from the SAOM models are used as outcomes in the meta analysis. I refer to them as "parameters" and refer to the estimates in the meta analysis as "coefficients".

#### 3.2.2. Multilevel meta network analysis

In the second step, I fitted a series of meta-regressions on the estimated parameters of the converged SAOMs. The computation was done via the "mvmeta" package (Gasparrini et al., 2012) in R. Since the central interest of this study is on the network dynamics surrounding smokers, I only focus on meta analysis of the six estimated parameters that are related to smokers. The basic multilevel univariate meta-regression takes the following form.

$$\hat{\theta}_{ki} = \theta_i + \sum_{t!=2} \delta_t \times T_t^k + \sum_{s!=1} \eta_s \times S_s^k + e_{ki}, \quad (11)$$

where $\hat{\theta}_{ki}$ is the $i$th estimated parameter in class $k$. $T_t^k$ is a dummy indicator for the treatment condition the $k$th class was assigned into. $T_1^k = 1$ only if the class was assigned into control, $T_2^k = 1$ only if the class was assigned into the random intervention, $T_3^k = 1$ only if the class was assigned into the central intervention, and $T_4^k = 1$ only if the class was assigned into the group intervention. To avoid collinearity, $T_2^k$ is omitted. Thus the coefficients for $T_t^k$ reflect the effects of the control condition and the network interventions on network dynamics in contrast to that of the random intervention. In a similar vein, $S_s^k$ is a dummy indicator for the school that the $k$th class is in. I use the first school as the reference group. Including a school indicator helps to account for variations in the estimates across schools. Last, $e_{ki}$ is a random error term with a zero mean

2 For students in the control condition, since there is no difference in the treatment status, the variable of treatment status was omitted from the model.

3 Regular smokers are defined as those who smoke at least weekly and have smoked for over a year. Recent smokers are defined as those who have smoked within the past 30 days before the second survey. To address possible under-reports of smoking (Kenkel et al., 2003), a self-reported nonsmoking student was identified as a recent smoker if three or more peers have seen him or her smoking within the past 30 days before the second survey.

4 I also experimented with several other variants of the SAOMs. One of them included behavioral dynamics on smoking. But the estimated parameters and standard errors are unusually large, indicating poor model convergence. This may be because there is less variation in smoking behavior over time and the data is not informative enough for studying such effects. In another variant of the SAOMs, smoking was treated as a time-varying covariate, as there is a baseline measure of smoking from the first survey. But the model also fitted poorly.

**Table 1**
SAOM results for the network dynamics in a selected class.

| | Function | Dynamics | OR | 95% CI | | CT |
|---|---|---|---|---|---|---|
| 1 | Evaluation | Outdegree (density) | 0.08 | −3.54 | 0.25 | 0.08 |
| 2 | Evaluation | Reciprocity | 4.85 | 4.24 | 14.36*** | 0.00 |
| 3 | Evaluation | Transitive triplets | 1.48 | 1.37 | 4.38*** | −0.01 |
| 4 | Evaluation | Indegree – popularity (sqrt) | 0.93 | 0.42 | 2.75 | −0.08 |
| 5 | Evaluation | Outdegree – popularity (sqrt) | 0.66 | 0.08 | 1.95 | 0.00 |
| 6 | Evaluation | Boy alter | 1.13 | 0.81 | 3.35 | 0.00 |
| 7 | Evaluation | Same boy | 3.58 | 3.01 | 10 59*** | −0.02 |
| 8 | Evaluation | Height alter | 1.01 | 1.00 | 3.00 | −0.05 |
| 9 | Evaluation | Height similarity | 1.54 | 0.50 | 4.55 | 0.04 |
| 10 | Evaluation | Ranking alter | 1.60 | 1.26 | 4.73** | 0.03 |
| 11 | Evaluation | Same ranking | 1.40 | 1.08 | 4.15* | −0.02 |
| 12 | Evaluation | Personality alter | 1.09 | 0.76 | 3.22 | 0.01 |
| 13 | Evaluation | Same personality | 0.83 | 0.55 | 2.45 | 0.01 |
| 14 | Evaluation | Family alter | 0.92 | 0.38 | 2.71 | 0.00 |
| 15 | Evaluation | Same family | 1.63 | 1.19 | 4.83* | −0.01 |
| 16 | Maintenance | Smoking alter | 1.47 | −1.14 | 4.34 | −0.04 |
| 17 | Creation | Smoking alter | 0.19 | −7.61 | 0.56 | 0.02 |
| 18 | Maintenance | Smoking ego | 14.96 | 11.60 | 44.27 | −0.01 |
| 19 | Creation | Smoking ego | 0.02 | −4.90 | 0.06 | 0.03 |
| 20 | Maintenance | Same smoking | 1.15 | −1.32 | 3.42 | −0.04 |
| 21 | Creation | Same smoking | 0.21 | −7.23 | 0.63 | 0.06 |
| 22 | Evaluation | Treat alter | 0.70 | 0.24 | 2.07 | 0.04 |
| 23 | Evaluation | Same treat | 0.95 | 0.63 | 2.80 | 0.01 |

*Note*: The shown estimates are odds ratios and their 95% confidence intervals. The last column shows the convergence *t*-ratios. An absolute value smaller than 0.1 generally indicates a good convergence (Ripley et al., 2014). Significance pattern:
* $P < 0.05$.
** $P < 0.01$.
*** $P < 0.001$.

and a variance that is equal to the estimated variance of the *i*th estimated parameter. The fixed effects model assumes that $\theta_i$ does not vary across networks. The random effects model assumes that $\theta_i$ follows a Normal distribution with a zero mean and an unknown variance. In the analysis, I estimated the fixed effects model by generalized least squares and the random effects model by restricted maximum likelihood.

I also conducted multivariate meta-regressions on the six parameters related to smokers.[5] Similar to the univariate case, the fixed effects models are estimated by generalized least squares while the random effects models by restricted maximum likelihood. To address possible estimation issues (e.g., due to the small sample size), I also specified two smaller multivariate models. Since the smoking-alter effects seem to be the most significant effects consistently in previous models, I specified a bivariate meta regression model only for the two smoking-alter effects. Furthermore, since the school effects are generally insignificant, I specified another model that excluded the school effects. These smaller models should be less likely to encounter estimation issues.

## 4. Results

### 4.1. SAOM results

Among the 76 friendship networks, SAOM converged well in 55 of them, including 13 control classes, 13 random intervention classes, 14 central intervention classes, and 15 group intervention classes.[6] Table 1 shows an example of the fitted SAOM in a selected class. The shown estimates are odds ratios and their 95% confidence intervals (CIs). The last column shows the convergence *t*-ratios. An absolute value smaller than 0.1 generally indicates a good convergence (Ripley et al., 2014).

In this example, the results indicate that ties are more likely to reciprocate and to form triangles (i.e., to be transitive) over time. Specifically, the odds of a tie which will reciprocate a previous friendship nomination is almost 5 times the odds of a tie which does not reciprocate a previous nomination (Odds ratio = 4.85, $P < 0.001$.). Similarly, a tie that would close a triangle is almost 50% more likely to occur than a tie does not close a triangle (Odds ratio = 1.48, $P < 0.001$.). There is also strong evidence for homophily. A tie between students of the same sex is almost 2.58 times more likely to occur than a tie between students of different sex (Odds ratio = 3.58, $P < 0.001$.). In addition, ties between students with similar academic ranking and similar family economic status are more likely to form than ties between students who differ in these dimensions (Odds ratio = 1.4, $P < 0.05$, and Odds ratio = 1.63, $P < 0.05$, respectively). Last, students with higher academic ranking (i.e, better academic performance) receive more connections than those with lower academic ranking. A tie to a higher ranked student is about 60% more likely to occur than a tie to a lower ranked student (Odds ratio = 1.6, $P < 0.01$).

None of the network dynamics related to smokers is statistically significant in this example. Fig. 2 presents the estimated parameters (the log odds ratios) and their 95% CIs for the network dynamics related to smokers across the classes.[7] Many of the estimates are indistinguishable from zero. Part of the reason may be that there is only a few smokers in many classes and so there is not enough information to estimate the parameters in a precise way. Meta analysis provides a way to synthesize the information from individual classes and so may help to reveal more significant patterns.

### 4.2. Results of the univariate meta-regressions

I conducted meta analysis only on the 55 classes for which the SAOM converged well. Table 2 shows the results of the univariate

[5] Modeling the six parameters related to smokers rather than all the parameters in the network models helps to stabilize the computation and address the high-dimensionality problem in meta analysis.

[6] A SAOM is judged as converging well if no more than two of the convergence *t*-ratios are larger than 0.1 in absolute value.

[7] For clarify, estimates with standard errors larger than 10 are omitted from the graph.

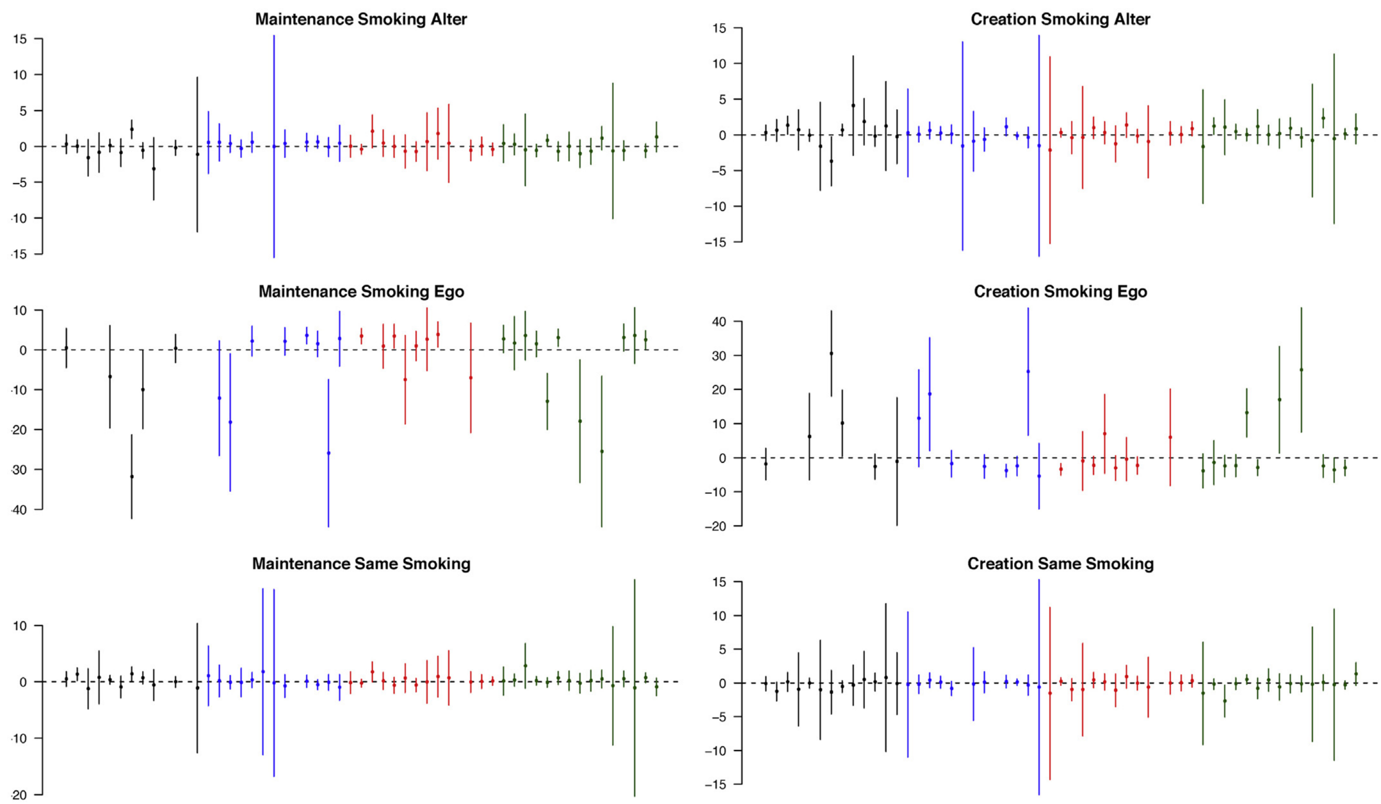


**Fig. 2.** Estimated parameters for network dynamics related to smokers across the classes. *Note*: Each vertical line shows the 95% confidence intervals of an estimated parameter for a selected network dynamics in a class. Estimates with standard errors larger than 10 are omitted from the graph for clarity. Estimates are colored black in control classes, blue in random intervention classes, red in central intervention classes, and dark green in group intervention classes. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

**Table 2**
Results of the univariate meta-regressions for network dynamics related to smokers.

| | (1) | | | (2) | | | (3) | | | (4) | | | (5) | | | (6) | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Maintenance alter | | | Creation alter | | | Maintenance ego | | | Creation ego | | | Maintenance same | | | Creation same | | |
| | OR | 95% CI | | OR | 95% CI | | OR | 95% CI | | OR | 95% CI | | OR | 95% CI | | OR | 95% CI | |
| *Fixed effects model* | | | | | | | | | | | | | | | | | | |
| Control | 0.56 | 0.28 | 1.11 | 1.21 | 0.65 | 2.24 | 0.01 | 0.00 | 0.27** | 9.92 | 0.50 | 2.E+2 | 1.67 | 0.85 | 3.29 | 0.75 | 0.41 | 1.38 |
| Random intervention | | | | | | | | | | | | | | | | | | |
| Central intervention | 0.49 | 0.26 | 0.91* | 1.18 | 0.67 | 2.08 | 11.48 | 0.89 | l.E+2 | 0.10 | 0.01 | 1.21 | 1.04 | 0.56 | 1.93 | 1.20 | 0.71 | 2.05 |
| Group intervention | 0.53 | 0.28 | 0.99* | 1.43 | 0.85 | 2.39 | 2.83 | 0.31 | 25.68 | 0.40 | 0.05 | 3.27 | 1.41 | 0.76 | 2.60 | 0.90 | 0.56 | 1.46 |
| *Random effects model* | | | | | | | | | | | | | | | | | | |
| Control | 0.55 | 0.25 | 1.21 | 1.21 | 0.65 | 2.24 | 1.08 | 0.00 | 3.E+4 | 9.92 | 0.50 | 2.E+2 | 1.67 | 0.85 | 3.29 | 0.75 | 0.41 | 1.38 |
| Random intervention | | | | | | | | | | | | | | | | | | |
| Central intervention | 0.52 | 0.25 | 1.06 | 1.18 | 0.67 | 2.08 | 2.E+2 | 0.03 | 2.E+6 | 0.10 | 0.01 | 1.21 | 1.04 | 0.56 | 1.93 | 1.20 | 0.71 | 2.05 |
| Group intervention | 0.53 | 0.26 | 1.09 | 1.43 | 0.85 | 2.39 | 58.22 | 0.01 | 2.E+5 | 0.40 | 0.05 | 3.27 | 1.41 | 0.76 | 2.60 | 0.90 | 0.56 | 1.46 |
| *Model fitness* | | | | | | | | | | | | | | | | | | |
| AIC (fixed effects) | **206** | | | **180** | | | 486 | | | 458 | | | **189** | | | **161** | | |
| AIC (random effects) | 214 | | | 191 | | | **440** | | | **445** | | | 199 | | | 173 | | |
| Cochran *Q*-test | 0.70 | | | 0.83 | | | 0.00 | | | 0.00 | | | 1.00 | | | 1.00 | | |
| *I*-square statistic | 0.01 | | | 0.01 | | | 0.63 | | | 0.59 | | | 0.01 | | | 0.01 | | |
| Samples | 55 | | | 55 | | | 55 | | | 55 | | | 55 | | | 55 | | |

*Note*: The shown estimates are odds ratios and their 95% confidence intervals. The fixed effects models are estimated by generalized least squares while the random effects models by restricted maximum likelihood. The meta analysis is conducted only on the 55 classes for which the SOAM converged well. Significance pattern:
* $P < 0.05$.
** $P < 0.01$.

meta-regressions for network dynamics related to smokers.[8] With the AIC as a criterion, the fixed effects model fits better on four of the six outcomes: the two smoking-alter effects (i.e., ties to smokers are more likely to maintain or create over time) and the two smoking-homophily effects (i.e., ties between students with the same smoking status are more likely to form than other ties). The random effects model fits relatively better on the smoking-ego effects (i.e., smokers are more likely to maintain or create ties).

[8] For conciseness, the results for the school effects are presented in Table A1 in the online supplementary materials.

**Table 3**
Results of the multivariate random effects meta-regression for network dynamics related to smokers.

| | (1) | | | (2) | | | (3) | | | (4) | | | (5) | | | (6) | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Maintenance alter | | | Creation alter | | | Maintenance ego | | | Creation ego | | | Maintenance same | | | Creation same | | |
| | OR | 95% CI | | OR | 95% CI | | OR | 95% CI | | OR | 95% CI | | OR | 95% CI | | OR | 95% CI | |
| *Treatment status* | | | | | | | | | | | | | | | | | | |
| Control | 0.57 | 0.31 | 1.05 | 1.02 | 0.59 | 1.79 | 1.E+3 | 0.02 | 6.E+7 | 0.00 | 0.00 | 37.90 | 0.92 | 0.52 | 1.63 | 1.01 | 0.62 | 1.63 |
| Random intervention | | | | | | | | | | | | | | | | | | |
| Central intervention | 0.52 | 0.28 | 0.95* | 1.30 | 0.75 | 2.24 | 7.E+2 | 0.02 | 3.E+7 | 0.00 | 0.00 | 71.07 | 0.71 | 0.41 | 1.22 | 1.38 | 0.88 | 2.18 |
| Group intervention | 0.67 | 0.38 | 1.20 | 1.25 | 0.75 | 2.09 | 2.E+3 | 0.09 | 7.E+7 | 0.00 | 0.00 | 12.65 | 1.05 | 0.62 | 1.78 | 1.10 | 0.72 | 1.68 |
| *Model fitness* | | | | | | | | | | | | | | | | | | |
| AIC | 1173 | | | | | | | | | | | | | | | | | |
| Cochran *Q*-test | 0.00 | | | | | | | | | | | | | | | | | |
| *I*-square statistic | 0.90 | | | | | | | | | | | | | | | | | |
| *Between-class covariance* | | | | | | | | | | | | | | | | | | |
| Standard deviations | 0.34 | | | 0.35 | | | 11.99 | | | 11.97 | | | 0.25 | | | 0.14 | | |
| Correlations | | | | | | | | | | | | | | | | | | |
| Creation alter | **−0.53** | | | | | | | | | | | | | | | | | |
| Maintenance ego | −0.43 | | | 0.56 | | | | | | | | | | | | | | |
| Creation ego | 0.43 | | | −0.53 | | | **−1.00** | | | | | | | | | | | |
| Maintenance same | 0.25 | | | 0.10 | | | −0.73 | | | 0.75 | | | | | | | | |
| Creation same | 0.47 | | | −0.58 | | | 0.28 | | | −0.29 | | | −0.72 | | | | | |
| Samples | 55 | | | 55 | | | 55 | | | 55 | | | 55 | | | 55 | | |

*Note*: The random effects models are estimated by restricted maximum likelihood. The first part of this table shows the estimated odds ratios and their 95% confidence intervals. The second part shows the model fitness measures. The last part shows the between-class covariance components, of which the first row presents the standard deviations of the estimated parameters across the classes while the remaining rows present the correlations among them. The meta analysis is conducted only on the 55 classes for which the SOAM converged well. Significance pattern:

* $P < 0.05$.

Using the Cochran *Q* test and the $I^2$ statistic to assess model fitness leads to the same conclusions. Thus the smoking-alter effects and the smoking-homophily effects are similar across the classes while the smoking-ego effects may vary greatly across the classes. However, regardless which models are used, the results are similar.

The fixed effects model (column 1 of Table 2) shows that compared to smokers in the random intervention classes, friendship ties directed to smokers in the central intervention classes and in the group intervention classes are both only half (odds ratio = 0.49, $P < 0.05$ and odds ratio = 0.53, $P < 0.05$, respectively) as likely to be maintained over time while the pattern is not statistically different in the control condition. Table 2 also shows that other network dynamics related to smokers are not statistically different between the random intervention and the network interventions. This is true in both the fixed effects models and the random effects models.

Two other features of the results are also worth noting. First, the coefficients of the smoking-ego effects (columns 3 and 4 in Table 2) seem to have unusually wide 95% confidence intervals, suggesting that the sample size may be insufficient to estimate these effects precisely. Second, the fixed effects model (column 3 in Table 2) suggests that smokers in the intervention classes were more likely to maintain their ties to others than their counterparts in the control classes. However, the better fitting random effects model suggest that the differences are not statistically significant.

### 4.3. Results of the multivariate meta-regressions

Table 3 presents the results of the multivariate random effects model. The results (column 1 in Table 3) suggest that smokers in the network intervention classes became less popular as compared to smokers in the random intervention classes. But the effect seems to be only statistically significant in the central intervention. Namely, friendship ties directed to smokers in the central intervention classes are only half as likely to be maintained as those in the random intervention classes (odds ratio = 0.52, $P < 0.05$). The effect in the group intervention classes is similar in magnitude, but statistically insignificant (odds ratio = 0.67, $P > 0.05$), probably because the intervention effect there is mostly restricted within the treated groups rather than being dispersed to outside members. In addition, similar to the univariate meta-regressions, the results of the multivariate random effects model show that there is no statistically significant difference between the random intervention and the network interventions in any other network dynamics related to smokers. Furthermore, across all outcomes, there is no statistically significant difference between the random intervention and the control condition.

Model fitness measures like AIC, the Cochran *Q* test, and the $I^2$ statistic all suggest that the multivariate random effects model is preferred to the multivariate fixed effects model. In addition, the coefficients of the multivariate fixed effects model are found to be unusually large and statistically significant, suggesting that the model may have run into some estimation issues. Thus the results are omitted for conciseness.

The last part of Table 3 shows the estimated between-class covariance (i.e., $\mathbf{\Omega}$). The first row shows the standard deviations of the estimated parameters across the classes while the remaining rows show the correlations among them. There appears to be a high level of variation across the classes in smokers' propensity to maintain and create ties to others as well as a moderate level of variation in students' propensity to maintain or create ties to the smokers. But the smoking-homophily effects seem not varying much across the classes. In addition, there also appears to be a moderate to high level of correlations among the parameters. In particular, the smoking-ego effects have a nearly −1 correlation coefficient.

The nearly perfect negative correlation between the smoking-ego effects and their unusually wide 95% confidence intervals make the model suspicious. Although the results could be due to finite sample bias alone and so does not necessarily indicate estimation issues (Riley et al., 2007), it is useful to fit a smaller model to check the robustness of the main results. In that vein, Table 4 shows the results of the bivariate meta-regressions on the two smoking-alter effects only. I specified two models: one included school effects and the other not. The one without school effects have fewer coefficients to estimate and so should be computationally more stable. For each model, I provided both the fixed effects

**Table 4**
Results of the bivariate meta-regressions for selected network dynamics related to smokers.

| | Model I: with schools | | | | | | Model II: without schools | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | (1) | | | (2) | | | (1) | | | (2) | | |
| | Endowment alter | | | Creation alter | | | Endowment alter | | | Creation alter | | |
| | OR | 95% CI | | OR | 95% CI | | OR | 95% CI | | OR | 95% CI | |
| *Fixed effects models* | | | | | | | | | | | | |
| Control | 0.52 | 0.28 | 0.98* | 1.09 | 0.63 | 1.88 | 0.66 | 0.38 | 1.15 | 1.18 | 0.73 | 1.89 |
| Random intervention | | | | | | | | | | | | |
| Central intervention | 0.49 | 0.27 | 0.89* | 1.23 | 0.72 | 2.07 | 0.57 | 0.33 | 0.99* | 1.30 | 0.82 | 2.04 |
| Group intervention | 0.51 | 0.28 | 0.92* | 1.43 | 0.88 | 2.31 | 0.62 | 0.36 | 1.06 | 1.35 | 0.86 | 2.11 |
| *Random effects models* | | | | | | | | | | | | |
| Control | 0.50 | 0.24 | 1.05 | 1.12 | 0.63 | 1.98 | 0.65 | 0.34 | 1.23 | 1.20 | 0.72 | 2.00 |
| Random intervention | | | | | | | | | | | | |
| Central intervention | 0.52 | 0.26 | 1.04 | 1.22 | 0.71 | 2.13 | 0.60 | 0.32 | 1.14 | 1.29 | 0.79 | 2.10 |
| Group intervention | 0.50 | 0.25 | 0.99* | 1.43 | 0.86 | 2.38 | 0.61 | 0.33 | 1.13 | 1.37 | 0.84 | 2.23 |
| *Between-class covariance* | | | | | | | | | | | | |
| Standard deviations | 0.35 | | | 0.14 | | | 0.33 | | | 0.16 | | |
| Between-class correlations | −0.64 | | | | | | −0.50 | | | | | |
| *Model comparison* | | | | | | | | | | | | |
| AIC (fixed effects) | 344 | | | | | | 339 | | | | | |
| AIC (random effects) | 370 | | | | | | 357 | | | | | |
| Cochran Q-test | 0.67 | | | | | | 0.52 | | | | | |
| *I*-square statistic | 0.01 | | | | | | 0.01 | | | | | |
| Samples | 55 | | | | | | 55 | | | | | |

*Note*: The shown estimates are odds ratios and their 95% confidence intervals. The fixed effects models are estimated by generalized least squares while the random effects models by restricted maximum likelihood. The meta analysis is conducted only on the 55 classes for which the SOAM converged well. Significance pattern:
* $P < 0.05$

and random effects estimates. In both model I and model II the fixed effects estimations outperform the random effects ones according to the model fitness measures. Hence, I focus on the fixed effects estimates to review the results.

The fixed effects estimates in model I (column 1 in Table 4) suggest that as compared to friendship ties directed to smokers in the random intervention classes, friendship ties directed to smokers in the network intervention classes (and in the control condition) are only about half (odds ratio ≈0.5, all $P < 0.05$) as likely to continue over time. There is no statistically significant difference in terms of ties being created toward smokers across these conditions (column 2 in Table 4). Thus, with this simpler model, we confirm the main findings in the more complicated multivariate meta-regressions shown above. However, the fact that smokers' popularity is reduced significantly even in the control condition when compared to the random condition seems suspicious. In the second model where the relatively insignificant school effects are excluded, the suspicious effect of the control condition disappears. The effect of the group intervention also reduces to insignificance. But the effect of the central intervention remains substantial and statistically significant (odds ratio = 0.57, $P < 0.05$). Model II has a smaller AIC than model I and so fits the data relatively better. Hence, according to the more credible results in model II, the effect of the central intervention seems more robust than that of the group intervention. This makes sense, because the effect of the group intervention is likely to operate mostly within the group rather than outside of the group. Last, note that the 95% CIs of the effects of the central intervention and the group intervention (and even the control condition) overlap with one another quite substantially. Thus perhaps a larger sample will help to provide clearer evidence on their relative performance.

## 5. Conclusion and discussion

In this paper, I introduced the latest advances in meta analysis for multilevel meta network. The new methods can not only combine results from multiple network models but also assess the effects of predictors at network or higher levels. In addition, unlike previous methods that mostly hold a fixed effects assumption, the new methods can accommodate both fixed effects and random effects. The random effects model assumes the underneath effect for each estimated parameter is a random quantity following a Normal distribution with a common mean and an unknown variance. Thus the random effects model helps to account for cross-network variation in the underneath effect for each parameter. Furthermore, unlike previous methods that are mostly for univariate meta analysis, the new methods incorporate multivariate meta analysis. The multivariate meta analysis can account for both within- and cross-network correlations among the estimated parameters while the univariate meta analysis assumes such correlations are zero. In short, the new methods provide more sophisticated options for conducting multilevel meta network analysis.

I applied the new methods to studying network dynamics of a smoking prevention intervention implemented to students in 76 classes of six middle school in China. First, I fitted a stochastic actor-oriented model on the friendship network in each of the 76 classes. Then I used meta-regressions to analyze the SAOM results. The univariate meta-regressions suggest that network interventions (including both the central intervention and the group intervention) are more effective than the random intervention in reducing smokers' popularity. Friendship ties directed to smokers in the network intervention classes are only about half as likely to be maintained as those in the random intervention classes. The multivariate meta-regressions confirm the above finding while also suggesting that the evidence is probably more robust for the central intervention than for the group intervention.

Overall, this paper introduces new and more sophisiticated methods for multilevel meta network analysis. The findings of the empirical example point out the importance of examining network outcomes, not just attitudinal or behavioral outcomes, in evaluating social and health interventions. This study also highlights that actors may chose to alter their social connections in order to resist (or manage) social influence. It suggests a new mechanism by which social influence and social selection can intertwine to make the causal analysis of network effects both more challenging and more intriguing. Last, the paper also indicates the great potential of using network methods to elevate intervention effects.

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## Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at http://dx.doi.org/10.1016/j.socnet.2015.03.006

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