TREATMENT EFFECT ACCOUNTING FOR NETWORK CHANGES

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Abstract—Networks may rewire in response to interventions. We propose a measure of the treatment effect when an intervention affects the structure of a social network. We develop a treatment-response model that incorporates dynamic peer effects and provide its identification conditions and the associated instrumental-variable strategy. We illustrate our estimation procedure using a panel data set containing information on a financial network before and after a field experiment that randomized access to savings accounts. Results show that neglecting the network change results in underestimation of the impact of the intervention and the role played by informal networks through which the intervention diffuses.

I. Introduction

A large body of literature has documented how peer effects spread through informal networks.[1] This issue is particularly relevant in the context of policy interventions, where networks may help to spread new products and technologies. One implicit assumption in the literature on peer effects and networks is that pre-existing links matter for economic outcomes (Banerjee et al., 2013; Oster & Thornton, 2012; Cai, de Janvry, & Sadoulet, 2015). This assumption is appropriate in settings where the network is fixed or difficult to change. However, it is also possible that networks rewire in response to changes in the economic environment, such as a policy intervention. If an intervention induces network changes, it is important to reassess both the actual role played by the network and how we measure the impact of this intervention.

In this paper, we study the interplay between network changes and treatment effects by proposing an econometric model in which peer effects spread through a social-interaction structure that changes following the treatment. We build on the intuition that if we observe network changes in a setting where peers matter, then the standard measure of the treatment effect may not capture an indirect (but potentially important) channel through which the intervention affects outcomes. First, we provide identification conditions and an instrumental-variable (IV) strategy that generalize the case of a time-invariant network (Bramoullé, Djebbari, & Fortin, 2009). Then, we propose a novel measure of the treatment effect that accounts for network changes. Next, we show, through a simulation experiment, that this measure outperforms the standard measures of treatment effects whenever the network that mediates the peer effects changes following the intervention.

The identification of treatment response with social interactions is at the frontier of econometric research (Rosenbaum, 2007; Hudgens & Halloran, 2008; Angelucci & De Giorgi, 2009; Manski, 2013).[2] Two recent papers have explored this issue using network data (Dieye, Djebbari, & Osario-Barrera, 2015; Arduini, Patacchini, & Rainone, 2014). Both papers rely on the assumption that the treatment does not change the network topology. Our model relaxes this assumption and identifies network changes as an additional mechanism through which the treatment can affect economic outcomes.

We illustrate our model using data from a field experiment that randomized access to savings accounts in nineteen villages in Nepal. This panel data set contains comprehensive information on all links of regular financial support in these villages before and after the randomized intervention. Our analysis exploits the unique combination of two features: the availability of longitudinal network data and the within-village randomization. Longitudinal network data allow us to assess the change in the network structure produced by the intervention. The randomization design creates exogenous variation in the treatment status of peers within the same village, which allows us to disentangle the direct treatment effect (i.e., the impact of one's own treatment status) and the peer effect diffusing through the network (i.e., the impact of peers' characteristics and treatment status).[3] We illustrate the model using data on household meat consumption. The results suggest that a failure to account for the network change results in underestimates of the overall impact of the intervention and the role played by informal networks through which the intervention spreads.

Our paper contributes to the growing literature that estimates peer effects using network data (Bramoullé et al., 2009; Calvó-Armengol, Patacchini, & Zenou, 2009; De Giorgi, Pellizzari, & Redaelli, 2010) in two ways: it models network changes over time, and it connects to the treatment effects literature. Other network data sources (such as Add Health) follow respondents over time but do not contain longitudinal information on the social network. Our paper uses panel network data to study peer effects and exploits a randomized intervention design to establish the unintended consequences of the treatment on networks and economic outcomes.[4]

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[1] See Jackson and Yariv (2010) for a review.

[2] In the presence of peer effects, the evaluation of a policy intervention is complicated by the fact that the treatment and control groups interact. This invalidates the standard assumption in the program-evaluation literature that one's outcome is invariant to the treatment status of others (the so-called Stable Unit Treatment Value Assumption).

[3] See Banerjee et al. (2018) for evidence on intervention-driven network changes with randomization at the village level.

[4] Our exercise is conceptually closer to Goldsmith-Pinkham and Imbens (2013), who exploit panel network data to first examine a dynamic setting

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The remainder of the paper is organized as follows: section II describes the econometric model, and section III illustrates it using the Nepalese data. Section IV concludes the paper. In addition, appendix A contains the proofs, appendix B describes the simulation exercise, and appendix C reports detailed background information on the experimental setting and the Nepalese data.

II. The Econometric Model

A. Notation

In this section, we introduce our econometric framework for analyzing the relationship between treatment effects and network changes. We frame our problem in the context of peer effects spreading through the social network structure.[5] We begin by describing our longitudinal model of treatment response in which the interaction matrix varies over time, possibly due to the intervention. Then, we provide identification conditions, describe the associated IV strategy, and derive a measure of the treatment effect. Insights from an extended simulation exercise are discussed in appendix B.

We first set out the notation. Column vectors are denoted by lowercase bold letters and matrices by capital bold letters. If $\mathbf{A}$ is an $N \times M$ matrix, a_{ij} indicates its $(i, j)^{\text{th}}$ element. When there is a time index, this is indicated by a superscript to avoid confusion with the entry notation (e.g., we write a^t_{ij} and $\mathbf{A}^t$). For a sample of N individuals, define $\mathbf{y}^t$ as the $N \times 1$ vector of the individual-level outcomes of interest at time t. $\mathbf{itt}$ is the $N \times 1$ intent-to-treat vector, that is, $itt_i = 1$ if individual i was randomized into the treatment group. We call $\boldsymbol{\epsilon}^t$ the $N \times 1$ vector of disturbances, $\boldsymbol{\iota}_N$ the $N \times 1$ vector of 1s, and $\boldsymbol{I}_N$ the $N \times N$ identity matrix. For each period, we observe the social interaction within the sample, represented by an $N \times N$ matrix $\mathbf{G}^t$ of fixed and known structure. $\mathbf{G}^t$ is semi-row-standardized: for non-isolated individuals, its row sums take value 1, while for isolated individuals, they take value 0.[6] Thus, the row sums of $\mathbf{G}^t$ are not constant. Choosing to carry out a (semi-)row-standardization on the interaction matrix implies that we estimate a linear-in-means model, that is, a model in which the individual outcome is affected by the *mean* characteristics and outcomes of peers.[7]

B. Peer Effects with Network Changes

We consider a setting where data for N individuals are collected over two periods ($t = 0, 1$) and there is a randomized intervention at the individual level, which takes place between these two periods.[8] Our peer effects equation for $t = 0$ is

$$\mathbf{y}^0 = \beta_1 \mathbf{G}^0 \mathbf{y}^0 + \boldsymbol{\mu} + \boldsymbol{\epsilon}^0, \tag{1}$$

where the so-called first lag of the dependent variable $\mathbf{G}^0\mathbf{y}^0$ is the mean outcome of the peers, and its coefficient β_1 represents the strength of the peer effect. We denote by $\boldsymbol{\mu} = (\mu_1, \ldots, \mu_N)'$ the $N \times 1$ vector of individual-level heterogeneity, which may be correlated with the regressors. Similarly, for $t = 1$, we have

$$\mathbf{y}^1 = \left(\beta_1 \mathbf{G}^0 + \beta_2 \mathbf{G}^{1-0}\right) \mathbf{y}^1 + \gamma \mathbf{itt} + \left(\delta_1 \mathbf{G}^0 + \delta_2 \mathbf{G}^{1-0}\right) \mathbf{itt} + \boldsymbol{\mu} + \boldsymbol{\epsilon}^1, \tag{2}$$

where $\mathbf{G}^{1-0} = \mathbf{G}^1 - \mathbf{G}^0$ represents the observed change in the network between the two periods. In equation (2), we allow the peer effects to vary by partner type: β_1 is the strength of peer effects from "old" partnerships that predate the intervention, and β_2 is the analogous effect from "new" partners. The specification for $t = 1$ also includes the intent-to-treat vector $\mathbf{itt}$ and its lags, $\mathbf{G}^0\mathbf{itt}$ and $\mathbf{G}^{1-0}\mathbf{itt}$, which represent the treated share of "old" and "new" partners, respectively, and capture the effect of partners' treatment status that does not operate through their outcomes. The corresponding coefficients δ_1 and δ_2 are usually referred to as contextual (peer) effects.[9] Finally, we assume that the interaction matrices are conditionally exogenous:

$$E[\boldsymbol{\epsilon}^t | \mathbf{G}^0, \mathbf{G}^1, \mathbf{itt}, \mu] = 0 \text{ for } t = 0, 1. \tag{3}$$

Conditioning the exogeneity of the interaction matrices on the individual-level effects μ is a remedy for the selection bias stemming from the assortativity of individuals into links (Manski, 1993), as long as correlated unobservables (i.e., unobservables simultaneously affecting link formation and the target regressors) are invariant within the period of study.[10] In section II C, we discuss how this assumption can be partially

of strategic network formation and then estimate a peer effects model using the results from the network formation model. See also the discussions in Bramoullé (2013) and Graham (2013).

[5]Most previous work on peer effects has used data in which individuals are partitioned into mutually exclusive and comprehensive reference groups (e.g., all children in the same school class). By doing so, the assumption is that individuals are equally affected by all other subjects in their group and by no one outside their group. Our model belongs to the class of peer effects models in which interactions are structured through social networks, such that the reference group has individual-level variation: if i and j are connected and j and k are connected, this does not necessarily mean that i and k are also connected.

[6]No self-links are allowed.

[7]Linear-in-means models have a structural interpretation as best-response functions for games with a preference for conformity and strategic complementarities (Kline & Tamer, 2012; Dieye & Fortin, 2016) and are commonly used to model peer effects in educational attainment, consumption, and substance abuse. Our exercise could be extended to a linear-in-sums framework, provided that the invertibility conditions are satisfied, which is generally the case for uniformly bounded interaction matrices (Kelejian & Prucha, 2010).

[8]The model builds on the example of a simple lottery to be consistent with the data described in section III. Nevertheless, it is suitable for all settings where the reference group has individual-level variation and the treatment status is heterogeneous among peers.

[9]In the terminology of Manski (1993), $\mathbf{G}^0\mathbf{y}^1$ and $\mathbf{G}^{1-0}\mathbf{y}^1$ would be called endogenous social effects, and $\mathbf{G}^0\mathbf{itt}$ and $\mathbf{G}^{1-0}\mathbf{itt}$ would be exogenous social effects.

[10]Our strategy accounts for correlated unobservables at the individual level, which is an improvement over the previous literature that allows for assortativity at the level of the entire network only (Bramoullé et al., 2009).

relaxed. Stacking equations (1) and (2) over t, we obtain:

$$\mathbf{y} = \beta_1 \widetilde{\mathbf{G}}^0 \mathbf{y} + \beta_2 \widetilde{\mathbf{G}}^{1-0} \mathbf{y} + \left(\gamma \boldsymbol{I}_{2N} + \delta_1 \widetilde{\mathbf{G}}^0 + \delta_2 \widetilde{\mathbf{G}}^{1-0}\right) \widetilde{\mathbf{itt}} + \iota \boldsymbol{\mu} + \boldsymbol{\epsilon}, \quad (4)$$

where $\mathbf{y} = \begin{bmatrix} \mathbf{y}^0 \\ \mathbf{y}^1 \end{bmatrix}$, $\widetilde{\mathbf{G}}^0 = \begin{bmatrix} \mathbf{G}^0 & 0 \\ 0 & \mathbf{G}^0 \end{bmatrix}$, $\widetilde{\mathbf{G}}^{1-0} = \begin{bmatrix} 0 & 0 \\ 0 & \mathbf{G}^{1-0} \end{bmatrix}$, $\widetilde{\mathbf{itt}} = \begin{bmatrix} 0 \\ \mathbf{itt} \end{bmatrix}$, $\iota = \iota_2 \otimes \boldsymbol{I}_N$ and $\boldsymbol{\epsilon} = \begin{bmatrix} \boldsymbol{\epsilon}^0 \\ \boldsymbol{\epsilon}^1 \end{bmatrix}$.

The reduced form of equation (4) is given by

$$\mathbf{y} = \widetilde{\mathbf{S}}(\beta)^{-1} \left[\left(\gamma \boldsymbol{I}_{2N} + \delta_1 \widetilde{\mathbf{G}}^0 + \delta_2 \widetilde{\mathbf{G}}^{1-0}\right) \widetilde{\mathbf{itt}} + \iota \boldsymbol{\mu}\right] + \widetilde{\mathbf{S}}(\beta)^{-1} \boldsymbol{\epsilon}, \quad (5)$$

where $\widetilde{\mathbf{S}}(\beta) = \left[\boldsymbol{I}_{2N} - \beta_1 \widetilde{\mathbf{G}}^0 - \beta_2 \widetilde{\mathbf{G}}^{1-0}\right]$. This is a model in which peer effects spread through network links and the contextual variable of interest represents a policy intervention. With respect to the standard framework à la Bramoullé et al. (2009), we introduce two dimensions of heterogeneity. First, we add heterogeneity over time in both the individual attributes and the network structure. Second, we allow for heterogeneous peer effects from partners of different types ("old" versus "new" partners), as in Arduini et al. (2014) and Dieye and Fortin (2016). To eliminate the individual effects, we premultiply equation (4) by the standard transformation matrix: $\mathbf{J} = \left[\boldsymbol{I}_2 - \frac{1}{2}\iota_2 \iota_2'\right] \otimes \boldsymbol{I}_N$. Noting that $\mathbf{J}\iota\boldsymbol{\mu} = \mathbf{0}$, equation (4) becomes

$$\mathbf{J}\mathbf{y} = \beta_1 \mathbf{J}\widetilde{\mathbf{G}}^0 \mathbf{y} + \beta_2 \mathbf{J}\widetilde{\mathbf{G}}^{1-0} \mathbf{y} + \mathbf{J}\left(\gamma \boldsymbol{I}_{2N} + \delta_1 \widetilde{\mathbf{G}}^0 + \delta_2 \widetilde{\mathbf{G}}^{1-0}\right) \widetilde{\mathbf{itt}} + \mathbf{J}\boldsymbol{\epsilon}. \quad (6)$$

Equation (6) is our main estimating equation, which we call a "treatment-effects model with dynamic peer effects." It contains two distinct peer effect terms: an "outcome peer effect" and a "network peer effect." The first term, $\mathbf{J}\widetilde{\mathbf{G}}^0\mathbf{y}$, is the outcome peer effect and represents the change in partners' (mean) outcomes holding partners constant. The second term, $\mathbf{J}\widetilde{\mathbf{G}}^{1-0}\mathbf{y}$, is the network peer effect. This reflects the change in partners' (mean) outcomes due to the network change. This quantity is positive if the outcome of the "new" partners is higher than that of the "old" partners at $t = 1$. As these two peer effects terms are correlated, omitting the latter may lead to biased estimates of β_1.[11] Note that as long as there is meaningful variation in the network structure within and across periods, the social-interaction matrices and the transformation matrix do not commute: $\mathbf{J}\widetilde{\mathbf{G}}^0\mathbf{y} \neq \widetilde{\mathbf{G}}^0\mathbf{J}\mathbf{y}$ and $\mathbf{J}\widetilde{\mathbf{G}}^{1-0}\mathbf{y} \neq \widetilde{\mathbf{G}}^{1-0}\mathbf{J}\mathbf{y}$. This is because the row sums of the interaction matrices are not constant, which turns out to be an additional source of identification in our model, as we explain in appendix A. Finally, note that if the network is time-invariant across periods (i.e., $\widetilde{\mathbf{G}}^{1-0} = 0$), equation (6) becomes

$$\mathbf{J}\mathbf{y} = \beta_1 \mathbf{J}\widetilde{\mathbf{G}}^0 \mathbf{y} + \mathbf{J}\left(\gamma \boldsymbol{I}_{2N} + \delta_1 \widetilde{\mathbf{G}}^0\right) \widetilde{\mathbf{itt}} + \mathbf{J}\boldsymbol{\epsilon}. \quad (7)$$

In equation (7), which we call the "treatment-effects model with static peer effects," peer effects appear only through the change in outcome. This specification is an extension of the standard framework developed by Bramoullé et al. (2009), where individual characteristics change over time but the network is assumed to be constant. If we rule out both outcome peer effects *and* network peer effects (by setting $\widetilde{\mathbf{G}}^0 = \widetilde{\mathbf{G}}^1 = 0$), the estimation equation (6) reduces to a standard treatment-response model in panel with no peer effects:[12]

$$\mathbf{J}\mathbf{y} = \gamma \mathbf{J}\widetilde{\mathbf{itt}} + \mathbf{J}\boldsymbol{\epsilon}. \quad (8)$$

C. *Identification and Instrumental Variables*

We now state the conditions under which the model in equation (6) is identified and interpret them in terms of instrumental variables.

Proposition 1. *Suppose that equation (6) holds. If* $|\beta_1| < 1$, $|\beta_2| < 1$, *and* $|\beta_1 - \beta_2| < 1$, *then the matrix* $\widetilde{\mathbf{S}}(\beta)$ *is invertible.*

Proposition 2. *Suppose that equation (6) holds,* $\widetilde{\mathbf{S}}(\beta)$ *is invertible, and* $(\gamma\beta_1 + \delta_1) \neq 0$ *and* $(\gamma\beta_2 + \delta_2) \neq 0$. *If matrices* $\mathbf{I}, \widetilde{\mathbf{G}}^0, \widetilde{\mathbf{G}}^{1-0}, (\widetilde{\mathbf{G}}^0)^2, (\widetilde{\mathbf{G}}^{1-0})^2, \widetilde{\mathbf{G}}^0\widetilde{\mathbf{G}}^{1-0}, \widetilde{\mathbf{G}}^{1-0}\widetilde{\mathbf{G}}^0$ *are linearly independent, then the social effects are identified.*

Proposition 1 sets out the sufficient invertibility conditions for $\widetilde{\mathbf{S}}(\beta)$, which resemble the standard stationarity conditions in spatial and time-series econometrics (Kelejian & Prucha, 1998). Proposition 2 enumerates the minimal identification conditions for the model in equation (6), which are based on restrictions on the parameters and the structure of the interaction matrices. These conditions resemble the conditions stated by Bramoullé et al. (2009) in the context of homogeneous peer effects and by Arduini et al. (2014) and Dieye and Fortin (2016) in the context of heterogeneous peer effects. Appendix A presents the proofs of propositions 1 and 2.

All interaction models exploiting network data are susceptible to endogeneity concerns related to simultaneity, stemming from the fact that the outcomes of an individual and his partners are jointly determined. The terms $\mathbf{J}\widetilde{\mathbf{G}}^0\mathbf{y}$ and $\mathbf{J}\widetilde{\mathbf{G}}^{1-0}\mathbf{y}$ in equation (6) are then correlated with the disturbance vector $\mathbf{J}\boldsymbol{\epsilon}$, which may invalidate OLS inference. As long as individual reference groups are not fully overlapping, the standard solution to this problem is to use "lagged" partners' characteristics (i.e., the exogenous attributes of the partners of

[11] Since $T = 2$, estimating equation (6) is equivalent to estimating the following equation in first differences:

$$\left(\mathbf{y}^1 - \mathbf{y}^0\right) = \beta_1 \mathbf{G}^0 \left(\mathbf{y}^1 - \mathbf{y}^0\right) + \beta_2 \mathbf{G}^{1-0} \mathbf{y}^1 + \gamma \mathbf{itt} + \delta_1 \mathbf{G}^0 \mathbf{itt} + \delta_2 \mathbf{G}^{1-0} \mathbf{itt} + \left(\boldsymbol{\epsilon}^1 - \boldsymbol{\epsilon}^0\right).$$

[12] Note that in our setting, $itt_i = 0$ for $t = 0$.

one's partners) as instruments to address the reflection problem (e.g., Kelejian & Prucha, 1998; Bramoullé et al., 2009; Calvó-Armengol et al., 2009; Drukker, Egger, & Prucha, 2013; Patacchini & Zenou, 2012). The conditions in proposition 2 lead to a novel instrumentation procedure, which adapts the standard lagged-partner strategy to our context. In appendix A, we show how the endogenous regressors can be expressed in terms of a set of internally generated excluded instruments, which represent an "augmented" version of the lagged-partner characteristics exploiting the change in network topology. The associated set of instruments is

$$\begin{aligned}\mathbf{Q}_\infty = \mathbf{J}[&\widetilde{\mathbf{itt}},\ \widetilde{\mathbf{G}}^0\widetilde{\mathbf{itt}},\ \widetilde{\mathbf{G}}^{1-0}\widetilde{\mathbf{itt}},\\ &\widetilde{\mathbf{G}}^0\widetilde{\mathbf{S}}(\beta)^{-1}\widetilde{\mathbf{itt}},\ \widetilde{\mathbf{G}}^0\widetilde{\mathbf{S}}(\beta)^{-1}\widetilde{\mathbf{G}}^0\widetilde{\mathbf{itt}},\\ &\widetilde{\mathbf{G}}^0\widetilde{\mathbf{S}}(\beta)^{-1}\widetilde{\mathbf{G}}^{1-0}\widetilde{\mathbf{itt}},\ \widetilde{\mathbf{G}}^0\widetilde{\mathbf{S}}(\beta)^{-1}\iota,\\ &\widetilde{\mathbf{G}}^{1-0}\widetilde{\mathbf{S}}(\beta)^{-1}\widetilde{\mathbf{itt}},\ \widetilde{\mathbf{G}}^{1-0}\widetilde{\mathbf{S}}(\beta)^{-1}\widetilde{\mathbf{G}}^0\widetilde{\mathbf{itt}},\\ &\widetilde{\mathbf{G}}^{1-0}\widetilde{\mathbf{S}}(\beta)^{-1}\widetilde{\mathbf{G}}^{1-0}\widetilde{\mathbf{itt}},\ \widetilde{\mathbf{G}}^{1-0}\widetilde{\mathbf{S}}(\beta)^{-1}\iota].\end{aligned} \tag{9}$$

By using a series expansion of $\widetilde{\mathbf{S}}(\beta)^{-1}$, we can generate a finite set of instruments at will and derive the corresponding restrictions on the form of the interaction matrices. For instance, the minimal identification conditions in proposition 2 generate four second-order instruments $\mathbf{J}\left(\widetilde{\mathbf{G}}^0\right)^2\widetilde{\mathbf{itt}}$, $\mathbf{J}\left(\widetilde{\mathbf{G}}^{1-0}\right)^2\widetilde{\mathbf{itt}}$, $\mathbf{J}\widetilde{\mathbf{G}}^0\widetilde{\mathbf{G}}^{1-0}\widetilde{\mathbf{itt}}$, and $\mathbf{J}\widetilde{\mathbf{G}}^{1-0}\widetilde{\mathbf{G}}^0\widetilde{\mathbf{itt}}$.[13] Instruments of higher order can be added at the cost of some additional identification requirements (see appendix A). This model can be estimated by 2SLS or GMM (Lee, 2007; Liu & Lee, 2010).

Two notes are in order here. First, among the instruments in equation (9), we have variables that account for the different positions of individuals in the network as described by the centrality measure developed by Bonacich (1987). The details are discussed in appendix A. Second, if we wanted to relax the conditional exogeneity of $\boldsymbol{G}^1$ (equation 3), we could use the *predicted* change in the network as an instrument for the *observed* change in the network.[14]

D. *The Treatment Effect*

In what follows, we define a measure of the treatment effect as a function of the structural parameters of the model. We focus on the situation of interest in which the network change is a function of the randomized intervention, that is, $\mathbf{G}^{1-0} = f(\mathbf{itt})$. In this context, a measure of the treatment effect that accounts for this indirect spillover of the intervention onto individual outcomes may outperform the standard measures.

In the linear treatment-response model with no peer effects (equation 8), the total effect of the treatment is given by the coefficient γ. In models with spatial lags in the dependent variable, the interpretation of the estimated parameters is richer but more complicated: in the presence of peer effects, the treatment status of one individual will affect not only his own outcome (the *direct* effect), but also the outcomes of others (the *indirect* effect). To define the treatment effect of the dynamic model, we begin with the reduced form of equation (5) and derive the closed form of the $N \times N$ matrix of partial derivatives with respect to the treatment, which we call $\frac{\partial E(\mathbf{y}^1|\mathbf{itt})}{\partial \mathbf{itt}}$.[15] The kth column of $\frac{\partial E(\mathbf{y}^1|\mathbf{itt})}{\partial \mathbf{itt}}$ is an $N \times 1$ vector that represents the effect of the treatment of individual $k = 1, \ldots, N$ on the outcomes of all other individuals and is written as

$$\frac{\partial E\left(\mathbf{y}^1|\mathbf{itt}\right)}{\partial itt_k} = \frac{\partial \mathbf{S}(\beta)^{-1}}{\partial itt_k}\mathbf{M} + \mathbf{S}(\beta)^{-1}\frac{\partial \mathbf{M}}{\partial itt_k}, \tag{10}$$

$$\frac{\partial \mathbf{S}(\beta)^{-1}}{\partial itt_k} = \mathbf{S}(\beta)^{-1}\beta_2\frac{\partial \mathbf{G}^{1-0}}{\partial itt_k}\mathbf{S}(\beta)^{-1}, \tag{11}$$

$$\frac{\partial \mathbf{M}}{\partial itt_k} = \gamma\mathbf{e}_k + \delta_1\mathbf{G}^0\mathbf{e}_k + \delta_2\frac{\partial \mathbf{G}^{1-0}}{\partial itt_k}\mathbf{itt} + \delta_2\mathbf{G}^{1-0}\mathbf{e}_k, \tag{12}$$

where $\mathbf{S}(\beta) = \left[\boldsymbol{I}_N - \beta_1\mathbf{G}^0 - \beta_2\mathbf{G}^{1-0}\right]$, $\mathbf{M} = \left[\left(\gamma\boldsymbol{I}_N + \delta_1\mathbf{G}^0 + \delta_2\mathbf{G}^{1-0}\right)\mathbf{itt} + \boldsymbol{\mu}\right]$, and $\mathbf{e}_k$ is an $N \times 1$ vector with 1 at the kth element and 0 elsewhere. The $N \times N$ matrix $\frac{\partial \mathbf{G}^{1-0}}{\partial itt_k}$, which is obtained by differentiating each element of $\mathbf{G}^{1-0}$ with respect to itt_k, represents the effect of the intervention on the matrix of social interactions. Note that equation (10) still incorporates the unobserved heterogeneity terms $\boldsymbol{\mu}$, as commutativity does not hold (see appendix A). Once we have the closed form of equation (10), the treatment effect of the intervention can be calculated following standard practice in spatial econometrics (Le Sage & Pace, 2009; Elhorst, 2014; Hsieh & Lee, 2016): the *direct* treatment effect is the average of the diagonal elements in $\frac{\partial E(\mathbf{y}^1|\mathbf{itt})}{\partial \mathbf{itt}}$, and the *indirect* treatment effect is the average of the column (or row) sums of the nondiagonal elements of $\frac{\partial E(\mathbf{y}^1|\mathbf{itt})}{\partial \mathbf{itt}}$.[16] The indirect treatment effect, which represents social spillovers, operates through two channels: the change in the treatment status of baseline peers (which is, standard in this literature) and the intervention-driven changes in the network (which is, the novelty of our

[13]These instruments represent the share of treated individuals among old partners of old partners, new partners of new partners, old partners of new partners, and new partners of old partners, respectively.

[14]This would require recomputing the instruments by replacing the observed network change $\widetilde{\mathbf{G}}^{1-0}$ with an instrumental matrix, which is its fitted version. Kelejian and Piras (2014) demonstrate the consistency and asymptotic normality of the IV estimator in the context of an endogenous interaction matrix (see also Hsie & Lee, 2016).

[15]Since the treatment affects equilibrium quantities only at $t = 1$, we can simplify the notation by focusing on the right-lower quadrant of $\frac{\partial E(\mathbf{y}|\widetilde{\mathbf{itt}})}{\partial \widetilde{\mathbf{itt}}}$.

[16]The row sum represents the impact of changing the treatment status of all other individuals on the outcome of one particular individual, while the column sum represents the impact of changing the treatment status of one particular individual on the outcomes of all other individuals. These two quantities coincide.

dynamic model). The total treatment effect is then calculated as the sum of the direct and the indirect effects.[17]

Note that assuming $\frac{\partial \mathbf{G}^{1-0}}{\partial itt_k} = 0$ simplifies equation (10) to

$$\frac{\partial E\left(\mathbf{y}^1|\mathbf{itt}\right)}{\partial itt_k} = \left[\boldsymbol{I}_N - \beta_1\mathbf{G}^0\right]^{-1}[\gamma\mathbf{e}_k + \delta_1\mathbf{G}^0\mathbf{e}_k]. \quad (13)$$

We use equation (13) to compute the treatment effect for the model with static peer effects, equation (7), following the same procedure. Here, the indirect treatment effect operates only through the standard channel, which is the change in the treatment status of baseline peers. Observed network changes that are unrelated to the intervention are not considered. Appendix B describes the results of an extensive simulation exercise designed to assess the performance of these measures of the treatment effect under different scenarios regarding the magnitude of the intervention-driven network changes, the sample size, the amount of measurement error, and the type of network data generation process. The results suggest that, as soon as there is any intervention-driven network change, more accurate inference is obtained with the dynamic treatment-effect measure. In addition, the bias of the standard measures increases with the magnitude of the indirect spillovers.

III. Illustration

A. *Data Description*

The remainder of the paper illustrates our model using data that we purposely collected in Nepal. These data come from a randomized field experiment providing access to formal savings accounts to a random sample of poor households in nineteen villages surrounding Pokhara. A baseline survey was conducted in February 2009, where the female heads of all households living in these villages were interviewed.[18] Between the last two weeks of May and the first week of June 2010, half of these women were randomly assigned, through a public lottery held in each village, to the treatment group and offered the option of opening a savings account at the local bank branch office. The remaining half was assigned to the control group and was not given this option. In June 2011, an endline survey of the respondents was conducted. Prina (2015) shows very high take-up and usage rates of these savings accounts.

The sample considered in our study comprises 915 households that completed both survey waves. The network variable is based on the responses to a survey question eliciting repeated financial exchanges within the village sample prior to each wave.[19] On the basis of these responses, we first construct the matrices $\mathbf{Z}^t$ representing binary undirected links among sample households: for each household pair ("dyad") ij, $z^t_{ij} = z^t_{ji} = 1$ if a member of household i or household j mentioned a member of the other household as regular partner at time t.[20] $\mathbf{Z}^t$ is block diagonal as, by construction, only links within a given village are allowed. The resulting networks have a density of 2% (i.e., on average, 2% of the potential within-village links are actually formed). In line with the model described in section II, in the illustration that follows, we compute the semi-row-standardized version of $\mathbf{Z}^t$ that we call $\mathbf{G}^t$. (For further information on the setting and additional descriptive statistics on the data in use, please see appendix C.)

B. *Estimating the Treatment-Response Models*

We now estimate the treatment-response models from section II using our data. The outcome of interest is household meat consumption.[21] In our data, meat is the most expensive food component, and its consumption is fairly common but not ubiquitous.[22] Peer effects in eating behavior have been widely documented, and in our data, meat consumption may reflect conspicuous consumption.[23]

Table 1 reports the results for the three treatment-response models described in section IIB. We assume that the error terms are independent across villages, and we report in parentheses cluster-bootstrapped standard errors. Column 1 reports the estimates from a model with no peer effects; columns 2 and 3 report the estimates from the models with static and dynamic peer effects, respectively, based on a 2SLS IV strategy. We use all the excluded instruments that are internally generated by the model up to the third order.[24]

In column 3, the intent-to-treat dummy and the peer effects terms are positive and statistically significant. The

[17]Note that the estimates of both the direct and indirect effects result from complex interactions between the parameters and the social-interaction structure. For instance, an arbitrary diagonal element $\frac{\partial E(\mathbf{y}^1_i|\mathbf{itt})}{\partial \mathbf{itt}_i}$ does *not* necessarily equal the estimated coefficient γ. This is because the former also includes feedback loops (where observation i affects observation j and observation j also affects observation i) and longer paths that might go from observation i to j to k and back to i. This is because the series expansion of $\mathbf{S}(\beta)^{-1}$ contains terms $\left(\mathbf{G}^0\right)^k$ and $\left(\mathbf{G}^{1-0}\right)^k$ that, for $k \geqslant 2$, have non-zero elements on the diagonal.

[18]Having census data, we avoid making distributional assumptions to deal with sampled dyadic observations (Chandrasekhar & Lewis, 2016).

[19]Compared to hypothetical network data ("*Who would you ask for help in case of need?*"), actual network data ("*Who did you ask for help?*") limit the measurement error due to respondents' subjective evaluations (Comola & Fafchamps, 2014) but may overlook some potential links of mutual support, which were not activated during the period of study (Karlan et al., 2009). In our case, the regular nature of the links elicited should alleviate this concern.

[20]We choose to treat self-declared links as undirected because the survey question is designed to capture repeated episodes of support flowing in one or both directions. Nevertheless, our estimation strategy is compatible with both directed and undirected data. For a discussion of misreporting for discordant network data, see Comola and Fafchamps (2014, 2017).

[21]This variable measures the estimated value in Nepalese rupees of the total consumption of meat in the month prior to the survey. Meat includes goat/lamb and chicken/poultry. Buffalo meat/beef is excluded since this is considered an inferior good in Nepal.

[22]At endline, 33% of households reported no meat consumption during the last week, and the median consumption value was US$10.

[23]See, for example, Angelucci et al. (2019) and Cruwys, Bevelander, and Hermans (2015).

[24]For the descriptive statistics of all variables reported in table 1 plus the instruments, see table C1 of appendix C.

TABLE 1.—TREATMENT-RESPONSE MODELS, MAIN RESULTS

	No PE (1)	Static PE (2)	Dynamic PE (3)
$\mathbf{J}\widetilde{\mathbf{itt}}$	489.73***	399.05***	281.79***
	(81.41)	(83.92)	(86.06)
$\mathbf{J}\widetilde{\mathbf{G}}^0\mathbf{y}$ [outcome PE]		0.39	0.79**
		(0.43)	(0.36)
$\mathbf{J}\widetilde{\mathbf{G}}^{1-0}\mathbf{y}$ [network PE]			0.27**
			(0.11)
$\mathbf{J}\widetilde{\mathbf{G}}^0\mathbf{itt}$		4.41	−24.49
		(252.31)	(247.39)
$\mathbf{J}\widetilde{\mathbf{G}}^{1-0}\mathbf{itt}$			−4.70
			(206.08)
Observations	915	915	915
F-test (weak id.)	–	8.38	10.18

This table reports the estimates of a treatment-response model with no peer effects, static peer effects, and dynamic peer effects. Bootstrapped standard errors in parentheses (100 replications) with village-level clustering. Kleibergen-Paap F-test statistics are shown at the bottom of the table. Statistically significant coefficients are indicated as follows: *10%, **5%, and ***1%.

estimated coefficient on the outcome peer effect $\mathbf{J}\widetilde{\mathbf{G}}^0\mathbf{y}$ suggests that a 1 rupee increase in the average meat consumption of baseline partners increases an individual's own consumption by 0.79 rupee. The network peer-effect term $\mathbf{J}\widetilde{\mathbf{G}}^{1-0}\mathbf{y}$ is also significant: this implies that a 1 rupee increase in average meat consumption at endline by new partners, relative to old partners, translates into an increase of 0.27 rupees in own consumption. These results taken together suggest that greater meat consumption of partners—whether from old or new partners—generates positive peer effects.

The contextual effects $\mathbf{J}\widetilde{\mathbf{G}}^0\mathbf{itt}$ and $\mathbf{J}\widetilde{\mathbf{G}}^{1-0}\mathbf{itt}$ are not statistically significant, suggesting that there is no direct effect of partners' treatment status once their consumption has been taken into account. Finally, note that the coefficient on the intent-to-treat dummy falls from column 1 to column 3. This may be due to omitted variable bias if the intent-to-treat dummy and the peer effects terms are correlated through the intervention-driven network change. If agents strategically rearrange their links after the intervention, treatment status will not be independent of the number or characteristics of partners, which in turn could invalidate inference regarding the direct treatment effect in the presence of peer effects. Thus, in our data illustration, a failure to account for network changes may overestimate the direct treatment effect.

One caveat is in order. The identification strategy based on lagged partner characteristics relies crucially on the assumption that spillovers spread through the observed structure of social interactions. Our estimates of peer effects could then be biased upward if network connections among households were underestimated. This would be the case if peer effects operated via dimensions of social interactions other than regular financial support links. Unfortunately, due to data limitations, we are forced to disregard other potential channels of peer effects beyond the one that we measure.

C. *Estimating the Treatment Effect*

As we argued in section II, if a policy intervention affects network topology and peer effects are at work, a measure of the treatment effect that incorporates the intervention-driven network changes may be attractive. In what follows, first, we provide an estimate of $\frac{\partial \mathbf{G}^{1-0}}{\partial itt_k}$, then use it to evaluate equation (10) in the context of our data illustration. Let us consider the entire sample of within-village dyads and call a dyad "treated" if at least one of the two households involved was offered the savings account, that is, $itt_{ij} = \max(itt_i, itt_j)$. Preliminary statistics on the binary links $\mathbf{Z}^t$ already suggest that our randomized experiment affected the network in our villages by rewiring links from non-treated to treated dyads: despite the important reshuffling of links across waves, treated dyads are more likely to form a binary link at endline if they did not have one beforehand (1% versus 0.8% for non-treated dyads) and are less likely to drop a link at endline if they were already connected (76% versus 81% for non-treated dyads). To evaluate equation (10) we estimate

$$\left(g_{ij}^1 - g_{ij}^0\right) = \vartheta_1 \cdot itt_{ij} + \vartheta_2 \cdot \boldsymbol{X}_{ij} + \left(\varepsilon_{ij}^1 - \varepsilon_{ij}^0\right), \tag{14}$$

where g_{ij}^t is the $(ij)^{\text{th}}$ entry of the row-standardized interaction matrix $\mathbf{G}^t$ and $\boldsymbol{X}_{ij}$ contains dyad-level controls. We retain $\frac{\partial \widehat{\mathbf{G}}^{1-0}}{\partial itt_k} = 0.002$, which corresponds to the estimated coefficient $\widehat{\vartheta}_1$.[25] Equation (14) represents the simplest functional form $f(\cdot)$ to depict intervention-driven network changes. Nevertheless, various parametric or non-parametric models of network evolution can be nested in the current framework.[26]

TABLE 2.—MEASURES OF TREATMENT EFFECT

	(1) No PE	(2) Static PE	(3) Dynamic PE ($\boldsymbol{\mu} = 0$)	(4) Dynamic PE ($\widehat{\boldsymbol{\mu}}$)
Direct	489.7	417.9	342.3	370.9
Indirect	–	94.4	260.9	329.4
Total	489.7	512.3	603.2	700.2

Table 2 combines all of the estimates above to compute the measures of the treatment effect. In the model with no peer effects (column 1), the treatment effect is given by the estimated coefficient $\widehat{\gamma}$ from table 1, column 1. The numerical solution for the model with static peer effects in column 2 is obtained by plugging the estimated coefficients $\widehat{\gamma}$ and $\widehat{\beta}_1$ from table 1, column 2 into equation (13) and solving it recursively. In the model with dynamic peer effects in columns 3 and 4, we solve equation (10) numerically on the basis of the estimated coefficients $\widehat{\gamma}, \widehat{\beta}_1$, and $\widehat{\beta}_2$ from table 1, column 3. We additionally evaluate $\frac{\partial \widehat{\mathbf{G}}^{1-0}}{\partial itt_k} = 0.002$ from equation (14). The difference between columns 3 and 4 relates to the treatment of the household-level effects $\boldsymbol{\mu}$ in equation (10): in column

[25] See table C5 in appendix C for the complete results.

[26] Equation (14) corresponds to a myopic link-formation rule with no externalities from the local network architecture (e.g., no returns from triadic closure). In our context, local network externalities would raise specific econometric challenges (Graham, 2015).

3, we assume that $\mu = 0$,[27] and in column 4, we plug in the estimates of the household-level effects $\widehat{\mu}$.[28]

Overall, the results from table 2 suggest that by neglecting the dynamic peer effects, we underestimate the impact of the intervention. Two remarks are in order. First, the value of the direct treatment effect falls from column 1 to columns 3 and 4. This could be related to the fact that in the presence of intervention-driven network changes, own treatment status is correlated with the peer effects terms: in the context of the dynamic model, this would be the case if treated households tend to both increase their meat consumption and to link among themselves. This issue is relevant for interpretation: our estimates suggest that a sizable share of the overall effect is due to social spillovers rather than direct treatment. In other words, treating a sample of isolated individuals would yield a much lower effect (342.3 or 370.9 versus 417.9). Second, by comparing the results in columns 2 to 4, we can see that by taking into account intervention-driven network changes, we increase the magnitude of the estimated indirect treatment effect, which more than compensates for the decline in the direct effect.

The results from this data illustration suggest that the direct component of the treatment effect is overestimated and that standard measures of peer effects, which neglect network changes, should be revised upward. There is a natural analogy here to standard omitted variable bias: in a framework in which peer effects are positive and there is complementarity between formal savings and network-based interactions, the bias is positive. Our methodology, however, is general and could have produced the opposite results when considering other data if, say, the intervention crowded out network interactions or if the peer effects were negative.

IV. Conclusion

Networks may evolve in response to interventions. This paper develops a structural model of treatment response that allows for time-varying social interactions. We derive a measure of the treatment effect that incorporates intervention-driven network changes. We illustrate our methodology using original data from Nepal, which contain detailed information on the network of regular financial support among households, before and after an exogenous expansion of formal financial access. Our results show that neglecting the intervention-driven network change results in an overestimate of the direct component of the treatment effect and an underestimate of its indirect component that operates through peers. This illustrates the paper's main message that unintentional changes in network topology should be accounted for when evaluating interventions.

Our study provides novel insights into how we should draw inferences based on network data. Some work has sought to manipulate group membership (e.g., Fafchamps & Quinn, 2016; Goette, Huffman, & Meier, 2012; Di Falco et al., 2016), and here, we show that social interactions may well be shaped even by interventions that were a priori not expected to do so. One implicit assumption behind previous work on networks and diffusion is that pre-existing relationships matter for economic outcomes. This assumption is indeed appropriate in a setting where the network is fixed or difficult to change, as in kinship networks. However, it is also possible that certain informal networks can easily be rewired in response to changes in the economic environment and that new links can be formed irrespective of the pre-existing relationships. Networks of financial support are prime examples, as shown in this paper. We hence recommend more caution in interpreting pre-existing links in a causal manner and in drawing policy recommendations based on them.

[27] This is analogous to the treatment of fixed effects in the conditional logit model.

[28] In column 4, the $\widehat{\mu}$ are estimated from a dummy-variable specification, that corresponds to equation (6). For T fixed and $N \to \infty$, these estimates are unbiased but inconsistent.

REFERENCES

Angelucci, M., and G. De Giorgi, "Indirect Effects of an Aid Program: How Do Cash Transfers Affect Ineligibles' Consumption?" *American Economic Review* 99 (2009), 486–508.

Angelucci, M., H. Royer, S. Prina, and A. Savikhin, "Incentives and Unintended Consequences: Spillover Effects in Food Choice," *American Economic Journal: Economic Policy* 11 (2019), 66–95.

Arduini, T., E. Patacchini, and E. Rainone, "Identification and Estimation of Outcome Response with Heterogeneous Treatment Externalities," unpublished (2014).

Banerjee, A., A. Chandrasekhar, E. Duflo, and M. Jackson, "The Diffusion of Microfinance," *Science* 341:6144 (2013), 363–370.

——— "Changes in Social Network Structure in Response to Exposure to Formal Credit Markets," unpublished (2018).

Bonacich, P., "Power and Centrality: A Family of Measures," *American Journal of Sociology* 92 (1987), 1170–1182.

Bramoullé, Y., "Comment," *Journal of Business Economic and Statistics* 31 (2013), 264–266.

Bramoullé, Y., H. Djebbari, and B. Fortin, "Identification of Peer Effects through Social Networks," *Journal of Econometrics* 150:1 (2009) 41–55.

Cai, J., A. de Janvry, and E. Sadoulet, "Social Networks and the Decision to Insure: Evidence from Randomized Experiments in China," *American Economic Journal: Applied Economic* 7 (2015), 81–108.

Calvó-Armengol, A., E. Patacchini, and Y. Zenou, "Peer Effects and Social Networks in Education," *Review of Economic Studies* 76 (2009), 1239–1267.

Chandrasekhar, A., and R. Lewis, "Econometrics of Sampled Networks," unpublished (2012).

Comola, M., and M. Fafchamps, "Testing Unilateral and Bilateral Link Formation," *Economic Journal* 124 (2014), 954–976.

——— "The Missing Transfers: Estimating Misreporting in Dyadic Data," *Economic Development and Cultural Change* 65 (2017), 549–582.

Cruwys, T., K. Bevelander, and R. Hermans, "Social Modeling of Eating: A Review of When and Why Social Influence Affects Food Intake and Choice," *Appetite* 86 (2015), 3–18.

De Giorgi, G., M. Pellizzari, and S. Redaelli, "Identification of Social Interactions through Partially Overlapping Peer Groups," *American Economic Journal: Applied Economics* 2 (2010), 241–75.

Dieye, R., H. Djebbari, and F. Osario-Barrera, "Accounting for Peer Effects in Treatment Response," unpublished (2015).

Dieye, R., and B. Fortin, "Gender Peer Effects Heterogeneity in Obesity," unpublished (2017).

Di Falco, S., F. Feri, P. Pin, and X. Vollenweider, "Ties that Bind: Redistributive Pressure and Economic Decisions in Village Economies," unpublished (2016).

Drukker, D., P. Egger, and I. Prucha, "On Two-Step Estimation of Spatial Autoregressive Models with Autoregressive Disturbances and Endogenous Regressors," *Econometric Reviews* 32 (2013), 686–733.
Elhorst, J., *Spatial Econometrics* (New York: Springer, 2014).
Erdös, P., and Rényi, A., "On Random Graphs," *Publicationes Mathematicae* 6 (1959), 290–297.
Fafchamps, M., and F. Gubert, "The Formation of Risk-Sharing Networks," *Journal of Development Economics* 83 (2007), 326–350.
Fafchamps, M., and S. Quinn, "Networks and Manufacturing Firms in Africa: Initial Results from a Randomised Experiment," *World Bank Economic Review* (2016), lhw057, https://doi.org/10.1093/wber/lhw057.
Goette L., D. Huffman, and S. Meier, "The Impact of Social Ties on Group Interactions: Evidence from Minimal Groups and Randomly Assigned Real Groups," *American Economic Journal: Microeconomics* 4 (2012), 101–115.
Goldsmith-Pinkham, P., and G. Imbens, "Social Networks and the Identification of Peer Effects," *Journal of Business and Economic Statistics* 31 (2013), 253–264.
Graham, B., "Comment," *Journal of Business Economic and Statistics* 31 (2013), 266–270.
——— "Methods of Identification in Social Networks," *Annual Review of Economics* 7 (2015), 465–485.
Hsieh C., and L. Lee, "*A Social Interactions Model with Endogenous Friendship Formation and Selectivity*," *Journal of Applied Econometrics* 31 (2016), 301–319.
Hudgens, M., and E. Halloran, "Toward Causal Inference with Interference," *Journal of the American Statistical Association* 103 (2008), 832–842.
Jackson, M., and L. Yariv, "Diffusion, Strategic Interaction, and Social Structure" (pp. 645–678), in Jess Benhabib, Alberto Bisin, and Matthew Jackson, eds., *Handbook of Social Economics* (Amsterdam: North-Holland Press, 2010).
Karlan, D., M. Mobius, T. Rosenblat, and A. Szeidl, "Trust and Social Collateral," *Quarterly Journal of Economics* 124 (2009), 1307–1361.
Kelejian, H., and G. Piras, "Estimation of Spatial Models with Endogenous Weighting Matrices, and an Application to a Demand Model for Cigarettes," *Regional Science and Urban Economics* 46 (2014), 140–149.
Kelejian, H., and I. Prucha, "A Generalized Spatial Two Stages Least Square Procedure for Estimating a Spatial Autoregressive Model with Autoregressive Disturbances," *Journal of Real Estate Finance and Economic* 17:1 (1998), 99–121.
——— "Specification and Estimation of Spatial Autoregressive Models with Autoregressive and Heteroskedastic Disturbances," *Journal of Econometrics* 157 (2010), 53–67.
Kinnan, C., and R. Townsend, "Kinship and Financial Networks, Formal Financial Access and Risk Reduction," *American Economic Review P&P* 102 (2012), 289–293.
Kline, B., and E. Tamer, "Some Interpretation of the Linear-ln-Means Model of Social Interactions," unpublished (2012).
Le Sage, J., and R. Pace, *Introduction to Spatial Econometrics* (London: CRC Press/Taylor and Francis, 2009).
Lee, L., "GMM and 2SLS Estimation of Mixed Regressive, Spatial Autoregressive Models," *Journal of Econometrics* 137 (2007), 489–514.
Ligon, E., J. Thomas, and T. Worrall, "Mutual Insurance, Individual Savings, and Limited Commitment," *Review of Economic Dynamics* 3 (2000), 216–246.
Liu, X., and L. Lee, "GMM Estimation of Social Interaction Models with Centrality," *Journal of Econometrics* 159 (2010), 99–115.
Manski, C., "Identification of Endogenous Social Effects: The Reflection Problem," *Review of Economic Studies* 60 (1993), 531–542.
——— "Identification of Treatment Response with Social Interactions," *Econometrics Journal* 16 (2013), S1–S23.
Nepal Rastra Bank "*Quarterly Economic Bulletin* (mid-October 2011).
Oster, E., and R. Thornton, "Determinants of Technology Adoption: Private Value and Peer Effects in Menstrual Cup Take-Up," *Journal of the European Economic Association* 10 (2012), 1263–1293.
Patacchini, E., and Y. Zenou, "Juvenile Delinquency and Conformism," *Journal of Law, Economics and Organization* 28:1 (2012), 1–31.
Prina, S., "Banking the Poor via Savings Accounts: Evidence from a Field Experiment," *Journal of Development Economics* 115 (2015), 16–31.
Rosenbaum, P. "Interference between Units in Randomized Experiments," *Journal of the American Statistical Association* 102 (2007), 191–200.
Watts, Duncan J., and Steven H. Strogatz, "Collective of 'Small-World' Networks," *Nature* 393:6684 (1998), 440–442.