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. To construct this measure, 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 unique panel dataset that contains information on a financial support network before and after a field experiment that randomized access to savings accounts in Nepal. Our results show that neglecting the network change results in an underestimation of the impact of the intervention and the role played by informal networks through which the intervention diffuses.

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1 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, Chandrasekhar, Duflo, and Jackson 2013; Oster and Thornton 2012; Cai, de Janvry, and 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 and 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 and Halloran 2008; Angelucci and De Giorgi 2009; Manski 2013).[2] Two recent papers have explored this issue using network data

[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

(Dieye, Djebbari and Osario-Barrera 2015; Arduini, Patacchini and 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 19 villages in Nepal. This panel dataset 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, and Zenou 2009; De Giorgi, Pellizzari, and 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 inter-

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, Chandrasekhar, Duflo, and Jackson (2018) for evidence on intervention-driven network changes with randomization at the village level.

vention design to establish the unintended consequences of the treatment on networks and economic outcomes.[4]

The remainder of the paper is organized as follows: Section 2 describes the econometric model, and Section 3 illustrates it using the Nepalese data. Section 4 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.

2 The econometric model

2.1 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.

[4]Our exercise is conceptually closer to Goldsmith-Pinkham and Imbens (2013), who exploit panel network data to first examine a dynamic setting of strategic network formation and then estimate a peer effects model using the results from the network formation model. See also the discussion 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.

We first set out the notation. Column vectors are denoted by lower-case bold letters and matrices by capital bold letters. If $\mathbf{A}$ is an $N \times M$ matrix, a_{ij} indicates its $(i,j)^{th}$ 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_{ij}^t 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. **itt** is the $N \times 1$ intent-to-treat vector, *i.e.*, $itt_i = 1$ 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 ones, 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 one, while for isolated individuals, they take value zero.[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, *i.e.*, a model in which the individual outcome is affected by the *mean* characteristics and outcomes of peers.[7]

2.2 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

[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 and Tamer 2012; Dieye and 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 and Prucha 2010).

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 $\boldsymbol{\mu} = (\mu_1, ..., \mu_N)'$ 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 **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:

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

[8] The model builds on the example of a simple lottery to be consistent with the data described in Section 3. 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.

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 2.3, we discuss how this assumption can be partially relaxed. Stacking Equations (1) and (2) over t, we obtain:

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

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:

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

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' vs. 'new' partners), as in Arduini *et al.* (2014) and Dieye and Fortin (2016). To eliminate the individual effects, we pre-multiply Equation (4) by the standard

[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).

transformation matrix: $\mathbf{J} = \left[\boldsymbol{I}_2 - \frac{1}{2}\boldsymbol{\iota}_2\boldsymbol{\iota}_2'\right] \otimes \boldsymbol{I}_N$. Noting that $\mathbf{J}\iota\boldsymbol{\mu} = \mathbf{0}$, Equation (4) becomes:

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

Equation (6) is our main estimation 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 and is positive, as long as at $t = 1$, the outcome of the 'new' partners is higher than that of the 'old' partners. 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: $\mathbf{J}\widetilde{\mathbf{G}}^0\mathbf{y} \neq \widetilde{\mathbf{G}}^0\mathbf{Jy}$ and $\mathbf{J}\widetilde{\mathbf{G}}^{1-0}\mathbf{y} \neq \widetilde{\mathbf{G}}^{1-0}\mathbf{Jy}$. 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:

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

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

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

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

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]

$$\mathbf{Jy} = \gamma \widetilde{\mathbf{Jitt}} + \mathbf{J}\boldsymbol{\epsilon}. \tag{8}$$

2.3 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 and 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

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

long as individual reference groups are not fully overlapping, the standard solution to this problem is to use "lagged" partners' characteristics (that is, the exogenous attributes of the partners of one's partners) as instruments to address the reflection problem (*e.g.*, Kelejian and Prucha 1998; Bramoullé *et al.* 2009; Calvó-Armengol *et al.* 2009; Drukker, Egger, and Prucha 2013; Patacchini and 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:

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

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 and 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

[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.

use the *predicted* change in the network as an instrument for the *observed* change in the network.[14]

2.4 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, *i.e.,* $\mathbf{G}^{1-0} = f(\mathbf{itt})$. 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 $\frac{\partial E\left(\mathbf{y}^1|\mathbf{itt}\right)}{\partial \mathbf{itt}}$.[15] The k^{th} column of $\frac{\partial E\left(\mathbf{y}^1|\mathbf{itt}\right)}{\partial \mathbf{itt}}$ is an $N \times 1$ vector that represents the effect of the treatment of individual $k = 1, ..., N$ on the outcomes of all other

[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 and 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 $\frac{\partial E\left(\mathbf{y}|\widetilde{\mathbf{itt}}\right)}{\partial \mathbf{itt}}$.

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 $\mathbf{S}(\beta) = [\boldsymbol{I}_N - \beta_1\mathbf{G}^0 - \beta_2\mathbf{G}^{1-0}]$, $\mathbf{M} = [(\gamma\boldsymbol{I}_N + \delta_1\mathbf{G}^0 + \delta_2\mathbf{G}^{1-0})\,\mathbf{itt} + \boldsymbol{\mu}]$, and $\mathbf{e}_k$ is an $N \times 1$ vector with 1 at the k^{th} 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 and Pace 2009; Elhorst 2014; Hsieh and Lee 2016): the *direct* treatment effect is the average of the diagonal elements in $\frac{\partial E\left(\mathbf{y}^1|\mathbf{itt}\right)}{\partial \mathbf{itt}}$, and the *indirect* treatment effect is the average of the column (or row) sums of the non-diagonal elements of $\frac{\partial E\left(\mathbf{y}^1|\mathbf{itt}\right)}{\partial \mathbf{itt}}$.[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 dynamic model). The *total* treatment effect is then calculated as the sum of the direct and the indirect effects.[17]

[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.

[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 $\frac{\partial E\left(\mathbf{y}_i^1|\mathbf{itt}\right)}{\partial \mathbf{itt}_i}$ 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.

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

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

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.

3 Illustration

3.1 Data description

The remainder of the paper illustrates our model using data purposely collected by the authors in Nepal. These data come from a randomized field experiment providing access to formal savings accounts to a random sample of poor households in 19 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

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

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, $z_{ij}^t = z_{ji}^t = 1$ 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% (that is, on average, 2% of the potential within-village links are actually formed). In line with the model described in Section 2, 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.

[19]Vis-a-vis 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 and Fafchamps 2014) but may overlook some potential links of mutual support, which were not activated during the period of study (Karlan, Mobius, Rosenblat, and Szeidl.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 and 2017).

3.2 Estimating the treatment-response models

We now estimate the treatment-response models from Section 2 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 2.2. 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]

[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 10 USD.

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

[24] For the descriptive statistics of all variables reported in Table 1 plus the instruments, see Table C3 of Appendix C.

Table 1: Treatment-response models, main results

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

Notes: 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%.

In column (3), the intent-to-treat dummy and the peer effects terms are positive and statistically significant. The estimated coefficient on the outcome peer effect $\mathbf{J\widetilde{G}^0y}$ suggests that a one-rupee increase in the average meat consumption of baseline partners increases an individual's own consumption by 0.79 rupees. The network peer-effect term $\mathbf{J\widetilde{G}^{1-0}y}$ is also significant: this implies that a one-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 $\mathbf{J\widetilde{G}^0itt}$ and $\mathbf{J\widetilde{G}^{1-0}itt}$ 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 upwards 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.

3.3 Estimating the treatment effect

As we argued in Section 2, 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, *i.e.*, $itt_{ij} = max(itt_i, itt_j)$. 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 $(ij)^{th}$ 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 combines the all 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 (3), we assume that $\boldsymbol{\mu} = 0$,[27] and in column (4), we plug in the estimates of the household-level effects $\widehat{\boldsymbol{\mu}}$.[28]

[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).

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

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

Table 2: Measures of treatment effect

	(1)	(2)	(3)	(4)
	No PE	Static PE	Dynamic PE ($\boldsymbol{\mu}$ =0)	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

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 *vs.* 417.9). Second, by comparing the results in columns (2)-(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 upwards. 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.

4 Concluding remarks

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 and Quinn 2016; Goette, Huffman, and Meier 2012; Di Falco, Feri, Pin, and Vollenweider 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.

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Appendix A: proofs

Proof of Proposition 1

The following steps are required for the proof of Proposition 1.

1. Since $\mathbf{G}^{1-0} = \mathbf{G}^1 - \mathbf{G}^0$, we can rewrite $\widetilde{\mathbf{S}}(\beta)$ as:

$$\widetilde{\mathbf{S}}(\beta) = \left[I_{2N} - \overline{\mathbf{G}}\right]$$

where $\overline{\mathbf{G}} = (\beta_1 - \beta_2)\,\widetilde{\mathbf{G}}^0 + \beta_2 \widetilde{\mathbf{G}}^1$ and $\widetilde{\mathbf{G}}^1 = \begin{bmatrix} \mathbf{G}^0 & 0 \\ 0 & \mathbf{G}^1 \end{bmatrix}$.

2. The degree vector contains the sum of the rows of matrix $\overline{\mathbf{G}}$. Since $\widetilde{\mathbf{G}}^0$ and $\widetilde{\mathbf{G}}^1$ are semi row-standardized, the degree vector of $\overline{\mathbf{G}}$ can take four unique values only:

   - if individual k has partners at both baseline and endline, his two entries (*i.e.,* the k^{th} and $(N+k)^{th}$ elements of the degree vector) are both β_1;
   - if k has partners at baseline only, his wo entries are β_1 and $(\beta_1 - \beta_2)$, respectively;
   - if k has partners at endline only, his two entries are 0 and β_2, respectively; and
   - if k is isolated throughout, his two entries are both 0.

3. Let $\lambda_1 \geq \lambda_2 \geq ... \geq \lambda_{2N}$ be the spectrum of $\overline{\mathbf{G}}$. We can write the determinant of $\widetilde{\mathbf{S}}(\beta)$ as $det\left(\widetilde{\mathbf{S}}(\beta)\right) = \prod_{i=1}^{2N}(1-\lambda_i)$.

4. We know that the maximum eigenvalue of a graph is smaller that the maximum degree of a graph: $\lambda_1 \leq \Delta\overline{\mathbf{G}}$.

5. A sufficient condition for $\widetilde{\mathbf{S}}(\beta)$ to be invertible is that its determinant be positive. In conjunction with the conditions above, this holds if $|\beta_1| < 1$, $|\beta_2| < 1$ and $|\beta_1 - \beta_2| < 1$.

Proof of Proposition 2

The following steps are required for the proof of Proposition 2.

1. We develop the geometric series expansion of $\widetilde{\mathbf{S}}(\beta)^{-1}$ for $k = 0, 1$ using Newton's binomial formula:

$$\begin{aligned}\widetilde{\mathbf{S}}(\beta)^{-1} &= \sum_{k=0}^{\infty} \widetilde{\mathbf{S}}_k(\beta) \\ &= \mathbf{I}_{2N} + \sum_{k=1}^{\infty} \widetilde{\mathbf{S}}_k(\beta) \\ &= \mathbf{I}_{2N} + \beta_1 \widetilde{\mathbf{G}}^0 + \beta_2 \widetilde{\mathbf{G}}^{1-0} + \sum_{k=2}^{\infty} \widetilde{\mathbf{S}}_k(\beta)\end{aligned}$$

   where $\widetilde{\mathbf{S}}_k(\beta) = \sum_{i=0}^{k} \binom{k}{i} \left(\beta_1 \widetilde{\mathbf{G}}^0\right)^{(k-i)} \times \left(\beta_2 \widetilde{\mathbf{G}}^{1-0}\right)^{i}$.

2. We substitute this series expansion into the reduced form of the model in Equation (5) and obtain:

$$\begin{aligned}\mathbf{y} &= \widetilde{\mathbf{S}}(\beta)^{-1}\widetilde{\mathbf{M}} + \widetilde{\mathbf{S}}(\beta)^{-1}\boldsymbol{\epsilon} \qquad (15)\\ &= \left(\mathbf{I}_{2N} + \beta_1 \widetilde{\mathbf{G}}^0 + \beta_2 \widetilde{\mathbf{G}}^{1-0}\right)\widetilde{\mathbf{M}} + \sum_{k=2}^{\infty} \widetilde{\mathbf{S}}_k(\beta)\widetilde{\mathbf{M}} + \widetilde{\mathbf{S}}(\beta)^{-1}\boldsymbol{\epsilon} \\ &= \left[\gamma \mathbf{I}_{2N} + (\delta_1 + \gamma\beta_1)\,\widetilde{\mathbf{G}}^0 + (\delta_2 + \gamma\beta_2)\,\widetilde{\mathbf{G}}^{1-0}\right]\widetilde{\mathbf{itt}} \\ &+ \left[\beta_1\delta_1\left(\widetilde{\mathbf{G}}^0\right)^2 + \beta_2\delta_2\left(\widetilde{\mathbf{G}}^{1-0}\right)^2 + \beta_1\delta_2\widetilde{\mathbf{G}}^0\widetilde{\mathbf{G}}^{1-0} + \beta_2\delta_1\widetilde{\mathbf{G}}^{1-0}\widetilde{\mathbf{G}}^0\right]\widetilde{\mathbf{itt}} \\ &+ \left(\mathbf{I}_{2N} + \beta_1 \widetilde{\mathbf{G}}^0 + \beta_2 \widetilde{\mathbf{G}}^{1-0}\right)\iota\boldsymbol{\mu} \\ &+ \sum_{k=2}^{\infty} \widetilde{\mathbf{S}}_k(\beta)\widetilde{\mathbf{M}} + \widetilde{\mathbf{S}}(\beta)^{-1}\boldsymbol{\epsilon}\end{aligned}$$

   where

$$\widetilde{\mathbf{M}} = \left[\left(\gamma \mathbf{I}_{2N} + \delta_1 \widetilde{\mathbf{G}}^0 + \delta_2 \widetilde{\mathbf{G}}^{1-0}\right)\widetilde{\mathbf{itt}} + \iota\boldsymbol{\mu}\right].$$

3. We take the conditional expectation of the expression above and pre-multiply it by $\widetilde{\mathbf{G}}^0$

and $\widetilde{\mathbf{G}}^{1-0}$ to yield:

$$
\begin{aligned}
E(\widetilde{\mathbf{G}}^{0}\mathbf{y} \quad |\widetilde{\mathbf{itt}}) = \quad & \left[\gamma\widetilde{\mathbf{G}}^{0} + (\delta_1 + \gamma\beta_1)\left(\widetilde{\mathbf{G}}^{0}\right)^{2} + (\delta_2 + \gamma\beta_2)\,\widetilde{\mathbf{G}}^{0}\widetilde{\mathbf{G}}^{1-0}\right]\widetilde{\mathbf{itt}} \\
& + \left[\beta_1\delta_1\left(\widetilde{\mathbf{G}}^{0}\right)^{3} + \beta_2\delta_2\widetilde{\mathbf{G}}^{0}\left(\widetilde{\mathbf{G}}^{1-0}\right)^{2} + \beta_1\delta_2\left(\widetilde{\mathbf{G}}^{0}\right)^{2}\widetilde{\mathbf{G}}^{1-0} + \beta_2\delta_1\widetilde{\mathbf{G}}^{0}\widetilde{\mathbf{G}}^{1-0}\widetilde{\mathbf{G}}^{0}\right]\widetilde{\mathbf{itt}} \\
& + \left[\widetilde{\mathbf{G}}^{0} + \beta_1\left(\widetilde{\mathbf{G}}^{0}\right)^{2} + \beta_2\widetilde{\mathbf{G}}^{0}\widetilde{\mathbf{G}}^{1-0}\right]\iota\boldsymbol{\mu} \\
& + \widetilde{\mathbf{G}}^{0}\sum_{k=2}^{\infty}\widetilde{\mathbf{S}}_k(\beta)\widetilde{\mathbf{M}}
\end{aligned}
\tag{16}
$$

$$
\begin{aligned}
E(\widetilde{\mathbf{G}}^{1-0}\mathbf{y} \quad |\widetilde{\mathbf{itt}}) = \quad & \left[\gamma\widetilde{\mathbf{G}}^{1-0} + (\delta_1 + \gamma\beta_1)\,\widetilde{\mathbf{G}}^{1-0}\widetilde{\mathbf{G}}^{0} + (\delta_2 + \gamma\beta_2)\left(\widetilde{\mathbf{G}}^{1-0}\right)^{2}\right]\widetilde{\mathbf{itt}} \\
& + \left[\beta_1\delta_1\widetilde{\mathbf{G}}^{1-0}\left(\widetilde{\mathbf{G}}^{0}\right)^{2} + \beta_2\delta_2\left(\widetilde{\mathbf{G}}^{1-0}\right)^{3} + \beta_1\delta_2\widetilde{\mathbf{G}}^{1-0}\widetilde{\mathbf{G}}^{0}\widetilde{\mathbf{G}}^{1-0} + \beta_2\delta_1\left(\widetilde{\mathbf{G}}^{1-0}\right)^{2}\widetilde{\mathbf{G}}^{0}\right]\widetilde{\mathbf{itt}} \\
& + \left(\widetilde{\mathbf{G}}^{1-0} + \beta_1\widetilde{\mathbf{G}}^{1-0}\widetilde{\mathbf{G}}^{0} + \beta_2\left(\widetilde{\mathbf{G}}^{1-0}\right)^{2}\right)\iota\boldsymbol{\mu} \\
& + \widetilde{\mathbf{G}}^{1-0}\sum_{k=2}^{\infty}\widetilde{\mathbf{S}}_k(\beta)\widetilde{\mathbf{M}}
\end{aligned}
\tag{17}
$$

By pre-multiplying Equations (16) and (17) by the transformation matrix $\mathbf{J}$, we can express the two endogenous regressors in terms of the excluded instruments that are internally generated by the model. According to Equation (15), the best IV matrix for Equation (6) is given by:

$$\mathbb{Q} = \mathbf{J}\left[\widetilde{\mathbf{itt}}, \widetilde{\mathbf{G}}^{0}\widetilde{\mathbf{itt}}, \widetilde{\mathbf{G}}^{1-0}\widetilde{\mathbf{itt}}, \widetilde{\mathbf{G}}^{0}\widetilde{\mathbf{S}}(\beta)^{-1}\widetilde{\mathbf{M}}, \widetilde{\mathbf{G}}^{1-0}\widetilde{\mathbf{S}}(\beta)^{-1}\widetilde{\mathbf{M}}\right]$$

even though $\mathbb{Q}$ is unfeasible (as it involves unknown parameters such as γ, $\boldsymbol{\beta}$, $\boldsymbol{\delta}$, and $\boldsymbol{\mu}$), it is a linear combination of the associated set of all instruments, which is:

$$
\begin{aligned}
\mathbf{Q}_{\infty} \quad = \quad & \mathbf{J}[\widetilde{\mathbf{itt}},\ \widetilde{\mathbf{G}}^{0}\widetilde{\mathbf{itt}},\ \widetilde{\mathbf{G}}^{1-0}\widetilde{\mathbf{itt}}, \\
& \widetilde{\mathbf{G}}^{0}\widetilde{\mathbf{S}}(\beta)^{-1}\widetilde{\mathbf{itt}},\ \widetilde{\mathbf{G}}^{0}\widetilde{\mathbf{S}}(\beta)^{-1}\widetilde{\mathbf{G}}^{0}\widetilde{\mathbf{itt}},\ \widetilde{\mathbf{G}}^{0}\widetilde{\mathbf{S}}(\beta)^{-1}\widetilde{\mathbf{G}}^{1-0}\widetilde{\mathbf{itt}},\ \widetilde{\mathbf{G}}^{0}\widetilde{\mathbf{S}}(\beta)^{-1}\iota, \\
& \widetilde{\mathbf{G}}^{1-0}\widetilde{\mathbf{S}}(\beta)^{-1}\widetilde{\mathbf{itt}},\ \widetilde{\mathbf{G}}^{1-0}\widetilde{\mathbf{S}}(\beta)^{-1}\widetilde{\mathbf{G}}^{0}\widetilde{\mathbf{itt}},\ \widetilde{\mathbf{G}}^{1-0}\widetilde{\mathbf{S}}(\beta)^{-1}\widetilde{\mathbf{G}}^{1-0}\widetilde{\mathbf{itt}},\ \widetilde{\mathbf{G}}^{1-0}\widetilde{\mathbf{S}}(\beta)^{-1}\iota]
\end{aligned}
\tag{18}
$$

In Equations (16) and (17), we show how to use a series expansion of $\widetilde{\mathbf{S}}(\beta)^{-1}$ to generate

a finite set of instruments and how to state the corresponding restrictions on the form of the interaction matrices. Note that the instruments are of two types: lagged-partner characteristics and centrality instruments. Using as instruments the exogenous attributes of the lagged partners who are excluded from one's own reference group is a standard strategy in network-interaction models. For instance, our series expansion for $k = 0$ generates four lagged-partner instruments of second order: $\mathbf{J}\left(\widetilde{\mathbf{G}}^{0}\right)^{2}\widetilde{\mathbf{itt}}$, $\mathbf{J}\left(\widetilde{\mathbf{G}}^{1-0}\right)^{2}\widetilde{\mathbf{itt}}$, $\mathbf{J}\widetilde{\mathbf{G}}^{0}\widetilde{\mathbf{G}}^{1-0}\widetilde{\mathbf{itt}}$, and $\mathbf{J}\widetilde{\mathbf{G}}^{1-0}\widetilde{\mathbf{G}}^{0}\widetilde{\mathbf{itt}}$. Therefore, as stated in Proposition 2, the minimal identification conditions of our model based on second-order lagged-partner instruments are $(\gamma\beta_1 + \delta_1) \neq 0$ and $(\gamma\beta_2 + \delta_2) \neq 0$ if the matrices $\mathbf{I}$, $\widetilde{\mathbf{G}}^{0}$, $\widetilde{\mathbf{G}}^{1-0}$, $\left(\widetilde{\mathbf{G}}^{0}\right)^{2}$, $\left(\widetilde{\mathbf{G}}^{1-0}\right)^{2}$, $\widetilde{\mathbf{G}}^{0}\widetilde{\mathbf{G}}^{1-0}$, *and* $\widetilde{\mathbf{G}}^{1-0}\widetilde{\mathbf{G}}^{0}$ are linearly independent. Note that the linear independence of $\mathbf{G}^{0}$ and $\widetilde{\mathbf{G}}^{1-0}$ (and their higher-order products) generally holds for non-degenerate network topologies with meaningful variation across time. Similarly, the series expansion for $k = 1$ produces eight possible lagged-partner instruments of third order: $\mathbf{J}\left(\widetilde{\mathbf{G}}^{0}\right)^{3}\widetilde{\mathbf{itt}}$, $\mathbf{J}\left(\widetilde{\mathbf{G}}^{1-0}\right)^{3}\widetilde{\mathbf{itt}}$, $\mathbf{J}\left(\widetilde{\mathbf{G}}^{0}\right)^{2}\widetilde{\mathbf{G}}^{1-0}\widetilde{\mathbf{itt}}$, $\mathbf{J}\left(\widetilde{\mathbf{G}}^{1-0}\right)^{2}\widetilde{\mathbf{G}}^{0}\widetilde{\mathbf{itt}}$, $\mathbf{J}\widetilde{\mathbf{G}}^{0}\left(\widetilde{\mathbf{G}}^{1-0}\right)^{2}\widetilde{\mathbf{itt}}$, $\mathbf{J}\widetilde{\mathbf{G}}^{1-0}\left(\widetilde{\mathbf{G}}^{0}\right)^{2}\widetilde{\mathbf{itt}}$, $\mathbf{J}\widetilde{\mathbf{G}}^{0}\widetilde{\mathbf{G}}^{1-0}\widetilde{\mathbf{G}}^{0}\widetilde{\mathbf{itt}}$, and $\mathbf{J}\widetilde{\mathbf{G}}^{1-0}\widetilde{\mathbf{G}}^{0}\widetilde{\mathbf{G}}^{1-0}\widetilde{\mathbf{itt}}$. If we wish to include them, we need to add all of the corresponding third-order interaction matrices $\left(\widetilde{\mathbf{G}}^{0}\right)^{3}$, $\left(\widetilde{\mathbf{G}}^{1-0}\right)^{3}$, ... to the list of linearly independent matrices above. Following the same procedure, we can further develop the series expansion of $\widetilde{\mathbf{S}}(\beta)^{-1}$ to derive instruments of higher order (*i.e.*, fourth order and above) and the corresponding identification requirements.

As mentioned in Section 2, Equations (16) and (17) also include a set of IVs that are the products of the interaction matrices $\widetilde{\mathbf{G}}^{0}$ and $\widetilde{\mathbf{G}}^{1-0}$ (and their higher-order matrix multiplications) with the vector ι. Their presence reflects that the row sums of the interaction matrices are not constant (for instance, because some households are isolated).[29] As explained by Liu and Lee (2010) and Dieye and Fortin (2016), these instruments account for Bonacich (1987) centrality and may provide an additional source of identification. However,

[29]When dealing with correlated effects, Bramoullé *et al.* (2009) assume that no individual is isolated, which guarantees commutativity in the cross-sectional model with homogeneous peer effects. In our context, were $\widetilde{\mathbf{G}}^{0}$ and $\mathbf{J}$ to be commutable, the term $\mathbf{J}\widetilde{\mathbf{G}}^{0}\iota\boldsymbol{\mu}$ would not appear in the transformed equation.

the number of centrality instruments rises with the number of groups. Liu and Lee (2010) perform a group-level transformation and therefore calculate their centrality at the group level. With our transformation, we obtain household-level centrality measures, which we do not include in the empirical illustration to avoid many-instrument bias.[30]

Appendix B: simulations

We now describe the simulation exercise we design to evaluate the performance of the proposed method. The objective is twofold. First, we wish to assess the performance of the estimators of the treatment effect of Equation (10) (direct, indirect and total treatment effect: DTE, ITE, and TTE, respectively). To so do, we compute their mean squared errors (MSE) under different scenarios regarding the data generating process and the magnitude of the intervention-driven network changes. Second, we investigate the coverage probability of different confidence intervals for the parameters of Equation (6) in the presence of cluster-level correlation.

We begin by exploring the performance of the estimators of the treatment effect. Since Equations (13) and (10) need to be evaluated at a specific value of the network structure and the intervention allocation, we compute the 'integrated' MSE of the estimates over various draws of these independent variables. The procedure is divided in two steps:

Step 1: Generate the estimated peer effects coefficients

For a given value of the parameter λ, representing intervention-driven network changes, we generate multiple estimates of the peer effects coefficients by using the following procedure:

[30]Note that not all of these instruments are meaningful in our context. For instance, $\widetilde{\mathbf{G}}^0$ has the same block $\boldsymbol{G}^0$ repeated on the main diagonal; therefore, $\widetilde{\mathbf{G}}^0\iota$ is constant over time and is cancelled out by the transformation $\mathbf{J}\widetilde{\mathbf{G}}^0\iota = 0$ and $\mathbf{J}\left(\widetilde{\mathbf{G}}^0\right)^2\iota = 0$. This is not the case for $\widetilde{\mathbf{G}}^{1-0}$, so that all variables involving the product of $\widetilde{\mathbf{G}}^{1-0}$ and ι may yield meaningful variation. To illustrate, $\mathbf{J}\widetilde{\mathbf{G}}^{1-0}\iota$ is a $2N \times N$ matrix whose i^{th} column accounts for the new links of household i (as deviations from i's two-period average).

1.1. Generate the independent variables $X^s = \{\boldsymbol{itt}, \boldsymbol{G}^0, \boldsymbol{G}^1\}$

We generate a dataset of $N = 50$ nodes and a treatment vector $\boldsymbol{itt}$ such that each node has an independent 50% probability of receiving the binary treatment. Then, we generate the interaction matrices as follows. First, we draw a binary baseline network Z^0, which is an Erdős-Renyi random graph of type $G(50, 0.1)$, *i.e.*, where each link among 50 nodes exists independently with a probability $p = 0.1$ (Erdős and Renyi, 1959). Then, we draw an Erdős-Renyi graph $Z^z \sim G(50, 0.1 + \lambda)$, where λ is a parameter representing intervention-driven network changes, and we build the binary endline network Z^1 according to the following rule: $Z^1 = Z^0$ if $itt_{ij} = max(itt_i, itt_j) = 0$, and $Z^1 = Z^z$ if $itt_{ij} = 1$.[31] Finally, we reshuffle 1% of the links to account for measurement error,[32] and we row-standardize the resulting matrices to obtain G^0, G^1.

1.2. Generate the dependent variable $\boldsymbol{y}^s$:

We generate the vector $\boldsymbol{y}^s$ on the basis of Equation (6), using the independent variables X^s, setting the population parameters $\gamma = 10$, $\beta_1 = 0.5$, $\beta_2 = 0.2$, $\delta_1 = \delta_2 = 0$ and $\boldsymbol{\mu}$ =0, and adding i.i.d. error terms $\boldsymbol{\epsilon}^0, \boldsymbol{\epsilon}^1$ such that $\epsilon_i^1 - \epsilon_i^0 \sim N(0, 1)$;

1.3. Estimate the peer effects coefficients:

We estimate the three peer effects models (Equations 8, 7, 6) and save the coefficients $\widehat{\boldsymbol{\beta}}^s$ =($\widehat{\gamma}_{ols}, \widehat{\gamma}_{stat}, \widehat{\beta}_{1stat}, \widehat{\beta}_{2stat}, \widehat{\gamma}_{dyn}, \widehat{\beta}_{1dyn}, \widehat{\beta}_{2dyn}, \widehat{\vartheta}$).

We repeat the procedure for $s = 1, ..., 100$ to obtain 100 vectors of estimated coefficients $\widehat{\boldsymbol{\beta}}^s$.

Step 2: compute the MSE

[31] Note that, in absence of measurement error, the binary links of non-treated dyads would stay the same.

[32] The measurement error is constructed as follows for Z^0: we reshuffle all binary links across dyads to generate Z_r^0, such that the overall share of non-zero outcomes and the symmetry is preserved ($Z_{r,ij}^0 = Z_{r,ji}^0$) while the links are by construction uncorrelated with the treatment status. We then draw a random subsample of 1% of dyads for which we replace the 'real' matrix Z^0 with the reshuffled version Z_r^0. We repeat the procedure for Z^1 (independently, *i.e.*, both the reshuffling scheme and the random 1% subsample are drawn independently across periods).

Next we assess the performance of the estimators of the treatment effects (DTE, ITE, and TTE) by computing an 'integrated' MSE (IMSE) as an average of the MSE over various draws of the network and the intervention allocation. This is to ensure that the estimator behaves well in different parts of the latent distribution of network structures. We proceed as follows.

2.1 Compute the MSE for a given network draw: We generate a new draw X^d from the same data generating process explained in Step 1.1 above and use it to compute the MSE of the treatment effects. For example, the MSE of the ITE for the static peer effects model based on draw d is defined as $MSE_d^{ITE,stat} = \frac{1}{100}\sum_{s=1}^{100}\left(\widehat{ITE}_{s,d}^{stat} - ITE^d\right)^2$, where $\widehat{ITE}_{s,d}^{stat}$ is obtained by evaluating Equation (13) on the basis of the independent variables X^d plus the estimated parameters in $\widehat{\boldsymbol{\beta}}^s$, while the benchmark value ITE^d is obtained by evaluating Equation (10) on the basis of the 'true' population parameters γ, β_1, β_2 and the generated data X^d. We also compute the standard decomposition of MSE into variance and bias.[33]

2.2. Compute the integrated MSE over multiple network draws: We repeat step 2.1 for $d = 1, ..., 100$ and average out the MSE over these 100 draws to obtain the final values of the IMSE (and variance and squared bias) 'integrated' over the various network configurations.

Columns (1)-(4) of Table B1 report the resulting integrated statistics of MSE, variance and squared bias (IMSE, IVar and IBias in the table) for the three peer-effects models (no PE, static PE, dynamic PE) and the three measures of treatment effects (TTE, ITE, DTE) for four different values of the parameter $\lambda \in \{0, 0.02, 0.05, 0.1\}$, ranging from no effect of the intervention to an increase of 100% in expected degree with respect to the baseline $p = 0.1$. The results suggest that, as soon as there is any intervention-driven network change

[33]For example, $Var_d^{ITE,stat} = \frac{1}{100}\sum_{s=1}^{100}\left(\widehat{ITE}_{s,d}^{stat} - \overline{ITE}_d^{stat}\right)^2$ and $Bias_d^{2\,ITE,stat} = \left(\overline{ITE}_d^{stat} - ITE^d\right)^2$ where $\overline{ITE}_d^{stat} = \frac{1}{100}\sum_{s=1}^{100}\widehat{ITE}_{s,d}^{stat}$.

(*i.e.*, $\lambda > 0$), more accurate inference is obtained with the dynamic model. The bias of the models with static peer effects increases with the magnitude of network changes and is mostly accounted for by the indirect treatment effect. Unsurprisingly, the dynamic model is unbiased but has a larger variance than the other two models, as it incorporates an additional error term at the dyadic level.

Next, we extend the simulation exercise along several dimensions to gather further insights into the performance of the estimator. In columns (5) and (6) of Table B1, we maintain an intermediate value of network changes $\lambda = 0.05$, as in column (3), and we increase the sample size N to 75 and 100, respectively. By construction, while the density of the network is constant, the expected degree is now higher. As expected, the increase in sample size has a slight effect on the variance (with respect to column 3), but overall, the performance of the estimators remains comparable.

In columns (7) and (8), we explore the role of mis-measurement: we reproduce the benchmark exercise in column (3) with $\lambda = 0.05$, with the only difference being that we now increase the amount of measurement error in the networks. In Column (7), we reshuffle 5% of observations independently for the generated binary networks Z^0, Z^1. In column (8), the percentage of reshuffled links is increased to 10%.[34] The results suggest that the dynamic model still outperforms the other models, but its MSE increases through the channel of variance.

Then, we explore the performance of our estimator in the context of a different data generating process. In fact, most real-life networks display a high level of clustering that Erdős-Renyi graphs cannot capture. To see how our estimator performs in the presence of clustering, in columns (9)-(11), we instead use a small-world network generation process (Watts and Strogatz, 1998). To do so, we define Z_s^0 as a Watts Strogatz lattice with 2 linked neighbors per side and Z_s^z as a Watts Strogatz lattice with 3 linked neighbors per side. Then, we explore the results under different hypotheses on the rewiring probability $q \in \{0.1, 0.25, 0.5\}$

[34]Note that a small change in the binary matrices Z^0, Z^1 may result in a much larger change in G^{1-0}.

in columns (9)-(11), respectively: for low q, the graph displays high clustering, but as q increases, it converges to the Erdős-Renyi graphs. The results are overall consistent with the patterns displayed previously: the dynamic model outperforms largely the other models.

Finally, in Table B2, we study the coverage probabilities of the confidence intervals in the presence of cluster-level correlation and few clusters. In fact, several real-life network datasets feature a small number of clusters (*e.g.*, separate villages or schools). This is also the case for the data in our empirical illustration, where we have only 20 clusters. In what follows, we evaluate the performance of different inference methods in such a context. For this scope, we fix a variance parameter σ^2 and repeat the following procedure 300 times:

1. We generate a sample of 1000 data points, divided into 20 clusters of equal size (N=50). For each cluster, we posit the same data generating process as in column (3) of Table B1, with the only difference here being that now the individual-level error term in Equation (6) includes a random effect at the cluster/period level: $\varepsilon_i^0 = \epsilon_i^0 + \alpha_c^0$ and $\varepsilon_i^1 = \epsilon_i^1 + \alpha_c^1$, where $\alpha_c^0, \alpha_c^1 \sim N(0, \sigma^2)$ and $\alpha_c^1 - \alpha_c^0 \sim N(0, 2\sigma^2)$;

2. Then, we use the generated data to estimate the dynamic PE model with four different inference methods (robust, clustered-robust, bootstrapped, and clustered-bootstrapped standard errors),[35] and we store the confidence intervals for $\widehat{\gamma}_{dyn}$, $\widehat{\beta}_{1dyn}$, and $\widehat{\beta}_{2dyn}$.

Table B2 reports the resulting coverage statistics, where coverage is defined as the percentage of times that the 95% confidence interval contains the known value of the parameter—a method thus performs well if it returns a coverage rate close to 95%. The three columns of Table B2 correspond to $\sigma^2 \in \{0.1, 0.5, 1\}$, which result in different levels of within-cluster correlation. The results suggest that clustered bootstrap standard errors perform rather well across all scenarios, which motivates our inference approach in the empirical illustration in Section 3.

[35] 'Robust' refers to the Huber/White estimator, 'bootstrap' performs non-parametric bootstrap with replacement, and 'cluster-bootstrap' extends the bootstrap such that each replication is based on a bootstrap sample of clusters.

Table B1: Simulation results

			(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)
					λ			N		meas. error (%)		Watts Strogatz (q)	
			0	**0.02**	**0.05**	**0.1**	**75**	**100**	5	**10**	**0.1**	**0.25**	**0.5**
		IMSE	23.52	36.22	50.26	72.19	48.17	48.26	48.81	47.24	45.11	47.28	52.45
no PE	TTE	Ibias2	22.83	35.6	49.67	71.6	47.73	48.02	48.21	46.67	44.45	46.71	51.76
		Ivar	0.69	0.62	0.59	0.59	0.44	0.24	0.60	0.57	0.66	0.58	0.69
		IMSE	0.17	1.86	5.13	12.18	3.94	3.65	4.75	4.29	3.95	4.19	5.78
static PE	TTE	Ibias2	0.00	1.63	4.91	11.95	3.84	3.54	4.53	4.06	3.8	4.06	5.57
		Ivar	0.17	0.23	0.22	0.23	0.1	0.11	0.22	0.23	0.14	0.13	0.2
		IMSE	0.04	0.1	0.24	0.85	0.33	0.39	0.22	0.18	0.32	0.26	0.27
static PE	DTE	Ibias2	0.00	0.00	0.15	0.76	0.29	0.36	0.13	0.09	0.26	0.22	0.21
		Ivar	0.04	0.1	0.09	0.08	0.05	0.03	0.09	0.09	0.06	0.05	0.06
		IMSE	0.15	1.76	3.51	6.86	2.16	1.77	3.34	3.14	2.2	2.54	3.81
static PE	ITE	Ibias2	0.00	1.56	3.34	6.68	2.03	1.65	3.15	2.94	2.07	2.4	3.6
		Ivar	0.15	0.2	0.18	0.18	0.13	0.11	0.18	0.2	0.13	0.14	0.21
		IMSE	0.31	1.28	1.23	1.52	0.73	0.48	1.51	1.75	0.63	0.93	1.24
dynamic PE	TTE	Ibias2	0.00	0.01	0.01	0.01	0.00	0.00	0.01	0.00	0.00	0.00	0.00
		Ivar	0.31	1.28	1.22	1.51	0.73	0.48	1.50	1.75	0.63	0.93	1.24
		IMSE	0.05	0.11	0.11	0.15	0.07	0.04	0.13	0.15	0.09	0.1	0.13
dynamic PE	DTE	Ibias2	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
		Ivar	0.05	0.11	0.11	0.15	0.07	0.04	0.13	0.15	0.09	0.1	0.13
		IMSE	0.22	0.76	0.74	0.87	0.48	0.33	0.90	1.02	0.4	0.57	0.76
dynamic PE	ITE	Ibias2	0.00	0.00	0.01	0.00	0.00	0.01	0.00	0.00	0.00	0.01	0.00
		Ivar	0.22	0.76	0.73	0.87	0.48	0.32	0.90	1.02	0.4	0.56	0.76

Table B2: Coverage

coeff	C. I.	σ^2		
		0.1	0.5	1
γ	robust	0.96	0.98	0.96
γ	cluster	0.95	0 .94	0.92
γ	boot	0.96	0.97	0.97
γ	boot clust	0.93	0.93	0.96
β_1	robust	0.88	0.45	0.35
β_1	cluster	0.94	0.94	0.92
β_1	boot	0.88	0.44	0.34
β_1	boot clust	0.93	0.93	0.92
β_2	robust	0.92	0.81	0.74
β_2	cluster	0.92	0 .92	0.91
β_2	boot	0.91	0.81	0.74
β_2	boot clust	0.91	0.90	0.90

Notes: "robust," as in robust standard errors; "cluster," as in clustered-robust standard errors; "boot," as in bootstrapped; and "boot cluster," as in clustered-bootstrapped standard errors.

Appendix C: background information

The intervention

The accounts offered in the contexts of our randomized experiment have all the characteristics of any formal savings account. The bank does not charge any opening, maintenance, or withdrawal fees and pays 6% nominal yearly interest, similar to the average alternatives available in the Nepalese market (Nepal Rastra Bank, 2011).[36] In addition, the savings account does not have a minimum balance requirement.[37] Customers can make transactions at the local bank-branch offices in the villages, which are open twice a week for approximately three hours, or at the bank's main office, located in downtown Pokhara, during regular business hours. There are no additional benefits to opening an account (*e.g.*, customers with a savings account were not eligible for credit or a lower interest rate on loans).

As shown by Prina (2015), the take-up and usage rates of the savings accounts offered to the treatment group were very high. In particular, over 84% of the treatment households opened an account and used it actively, depositing an average of 8% of their baseline weekly household income almost once a week for the first year of the intervention. Access to the savings account did not considerably increase total assets but raised household investments in health and education and improved their perceived financial situation.

Data description

Of the 1,009 households who completed the baseline survey in 2009, 915 were interviewed at endline. Appendix Table C1 reports the summary statistics for these 915 households, separately for the treatment and control groups, and shows that randomization generally led

[36]The International Monetary Fund Country Report for Nepal (2011) indicates a 10.5% rate of inflation during the intervention period.

[37]Money deposited in a savings account is fully liquid for withdrawal. The savings account operates without any commitment to save a given amount or to save for a specific purpose.

to balance among the baseline characteristics.

The women in the sample are very poor. They have on average 2.5 years of schooling and live in households with an average weekly income of 1,500 Nepalese rupees ($\simeq$ \$20) and with assets valued at approximately 44,000 rupees ($\simeq$ \$630).[38] Households have on average 4.5 members, two of whom are children. Only 15% of the households had a bank account before the intervention.

During each survey wave, the female household head was asked to provide a list of people (inside or outside the village) who she and her family rely on most (and/or who could rely on them most) for help in cash or in kind and with whom they *regularly* exchanged gifts and/or loans (even if only in one direction). Respondents could list as many partners as they wished. Special attention was devoted to accurately matching the declared partners' identities to sampled households and circumventing homonymy between different individuals living in the same village.[39]

The bottom part of Table C1 contains the network descriptive statistics at baseline by treatment status. On average, households reported having 1.42 partners, of whom 0.64 lived in the village. We also collected information on the number and size of loans and gifts to partners in the year before the survey. Loans seem to be more frequent than gifts: the declared numbers of gifts and loans exchanged with their partners were 0.79 and 1.90, respectively. Overall, at baseline, the treatment and comparison groups were well balanced along all network characteristics. Table C2 reports the attrition regressions for the sample of 1,009 households who completed the baseline survey: the probability of completing the endline survey does not seem to depend on either the treatment or network characteristics.

Figure 1 depicts $\mathbf{Z}^0$ and $\mathbf{Z}^1$, that is, the network of binary links at baseline and endline

[38] In 2010-2011, 70 Nepalese rupees equalled approximately one U.S. dollar.

[39] At the end of each interview, the enumerator used the updated village roster to determine, jointly with the respondent, the household identity code of the partners mentioned. The partners' unique identifiers were thus coded into the questionnaire in the field with the respondent, rather than during the data-cleaning process.

in the 19 villages. The links in green are observed at baseline only, those in red at endline only, and those in black both at baseline and endline. The average number of links is 0.72 for both baseline and endline, and 312 households (34% of the sample) are isolated.[40] The network density is 2% (that is, on average, 2% of the potential within-village links are actually formed).[41] The number of binary links remains the same across waves (656 at baseline vs. 658 at endline), but the network underwent an important reshuffling, as described in Section 3.3. Although there is considerable heterogeneity across villages, these networks tend to be sparse and divided into small groups (the average village-level clustering coefficient is 0.2).

Dyadic estimates

Table C4 reports the descriptive statistics of the dyadic sample, and Table C5 reports the results from estimating the dyadic equation (14) described in Section 3.3, that is:

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

In line with the model in Section 2, as dependent variable, we use the semi row-standardized version of $\mathbf{Z}^t$ that we call $\mathbf{G}^t$. Hence, $\left(g_{ij}^1 - g_{ij}^0\right)$ is continuous, and the estimation sample is directed ($N = 56,308$ dyads). The controls $\boldsymbol{X}_{ij}$ include the following time-varying dyadic characteristics, all computed in first differences: marital status (equal to one if both female household heads are married), the absolute difference in the number of children (under 16)

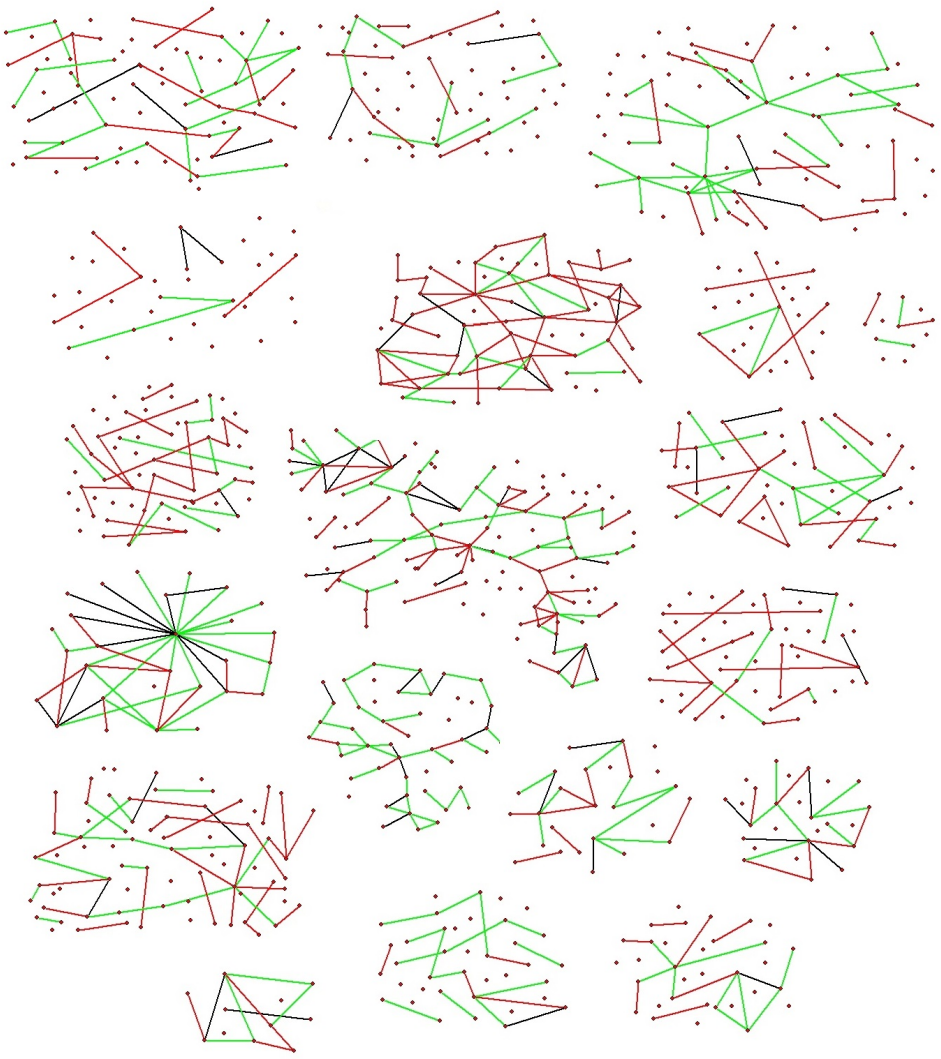
[40] The average number of links reported here is slightly greater than the number of self-reported links in Table C1 (0.64) because to build $\mathbf{Z}^t$, we use the maximum report out of the two parties involved whenever discrepancies arise.

[41] These statistics compare well to other examples of network data described in the literature: Kinnan *et al.* (2019) report an average degree of 0.65 for the financial network of the Townsend Thai Monthly Survey. Dizon *et al.* (forthcoming) find that while respondents list on average 2.46 potential partners, they only sent money to 0.8 partners within the 3 months prior to the study. Our density (2%) is the same as that for the median *Add Health* school as reported by Bramoullé *et al.* (2009).

and household members, two shock dummies (equal to one if the household either suffered the death of a member or a livestock loss in the six months prior to the survey wave), and a time trend. Standard errors in first differences $\left(\varepsilon_{ij}^{1} - \varepsilon_{ij}^{0}\right)$ are corrected to account for dyadic dependence (Fafchamps and Gubert 2007).

These estimated coefficients in Table C5 may seem small at first glance because, by construction, in dyadic samples, only a small share of all potential links are actually formed. Nevertheless, the estimated coefficient of itt_{ij} represents an increase of 30% over the mean outcome at baseline. These findings are of interest *per se* because they suggest some complementarity between formal savings and regular financial support links. *Ex ante*, it is unclear how the network would change as a result of the randomized intervention. On the one hand, access to a savings account might allow households to accumulate a buffer stock, which may offer a partial substitute for informal financial arrangements. As a result, informal transactions may be crowded out (Ligon, Thomas, and Worrall 2000). On the other hand, households that have accumulated assets may increase transfers to others, either because of altruism or *via* fear of social sanctions (Comola and Fafchamps 2017; Kinnan and Townsend 2012). This topic however, goes beyond the scope of this paper.

Figure 1: **The 19 within-village networks**



Notes: links in green were declared at baseline only, those in red at endline only, and those in black at both baseline and endline.

Table C1: Household descriptive statistics at baseline

	Sample N=915	**Control N=447**	**Treatment N=468**	**T-stat**
Age of the female household head	36.80	36.77	36.82	0.05
	(12.51)	(12.16)	(12.85)	
Years of education of the female hh. head	2.52	2.44	2.59	0.79
	(2.82)	(2.67)	(2.96)	
Percent married/living with partner	0.89	0.88	0.90	0.77
	(0.32)	(0.33)	(0.31)	
Household size	4.55	4.58	4.52	-0.51
	(1.66)	(1.68)	(1.64)	
Number of children	2.21	2.26	2.18	-0.86
	(1.30)	(1.30)	(1.29)	
Total income last week	1 494.73	1 472.84	1 515.64	0.13
	(4,833.91)	(4,598.50)	(5,053.36)	
Total assets	44 469.26	42,510.10	46 340.51	1.14
	(50,891.76)	(45,540.07)	(46,340.51)	
% of households with money in a bank	0.15	0.14	0.17	0.89
	(0.36)	(0.35)	(0.37)	
% of households with outstanding loans	0.90	0.88	0.91	1.42
	(0.31)	(0.32)	(0.29)	
No. of declared partners - total	1.42	1.39	1.45	0.61
	(1.37)	(1.35)	(1.39)	
No. of declared partners - village	0.64	0.62	0.65	0.53
	(0.92)	(0.94)	(0.89)	
No. of declared partners - out of village	0.79	0.77	0.80	0.32
	(1.07)	(1.02)	(1.12)	
No. of gifts declared	0.79	0.72	0.86	1.32
	(1.57)	(1.47)	(1.66)	
No. of loans declared	1.90	1.84	1.96	0.83
	(2.12)	(2.11)	(2.13)	

Notes: the last column shows the t-statistic from the two-way test of the equality of means across the treatment and control groups. Differences are statistically significant at the *10%, **5% and ***1% levels.

Table C2: Attrition regressions

	Completed endline		
	(1)	(2)	(3)
itt	0.009	0.008	0.008
	(0.015)	(0.015)	(0.015)
No. of declared partners – total		-0.009	
		(0.015)	
No. of declared partners - village			-0.001
			(0.016)
No. of declared partners – out of village			-0.010
			(0.015)
No. of gifts declared		0.002	0.001
		(0.004)	(0.004)
No. of loans declared		0.014	0.013
		(0.011)	(0.011)
Constant	0.919***	0.914***	0.915***
	(0.007)	(0.009)	(0.009)
Village dummies	yes	yes	yes
Observations	1,009	1,009	1,009
R-squared	0.056	0.061	0.061

Notes: Robust standard errors appear in parentheses, clustered at the village level. Statistically significant coefficients are indicated as follows: * 10%, ** 5% and *** 1%. All regressors are calculated at $t = 0$. *itt* represents the intent-to-treat dummy, which takes a value of one if the household was offered the savings account.

Table C3: Descriptive statistics for the individual sample

	Mean	**Min**	**Max**	**Std. Dev.**
$\mathbf{Jy}$	466.38	-7520	10800	1509.0
$\mathbf{J}\widetilde{\mathbf{itt}}$	0.51	0	1	0.5
$\mathbf{J}\widetilde{\mathbf{G}}^{0}\mathbf{y}$	245.54	-5120	9800	976.2
$\mathbf{J}\widetilde{\mathbf{G}}^{1-0}\mathbf{y}$	71.16	-8000	10800	1323.7
$\mathbf{J}\widetilde{\mathbf{G}}\widetilde{\mathbf{itt}}$	0.22	0	1	0.4
$\mathbf{J}\widetilde{\mathbf{G}}^{1-0}\widetilde{\mathbf{itt}}$	0.04	-1	1	0.5
IV1: $\mathbf{J}\left(\widetilde{\mathbf{G}}^{0}\right)^{2}\widetilde{\mathbf{itt}}$	0.22	0	1	0.4
IV2: $\mathbf{J}\left(\widetilde{\mathbf{G}}^{1-0}\right)^{2}\widetilde{\mathbf{itt}}$,	0.18	-1.5	2	0.5
IV3: $\mathbf{J}\widetilde{\mathbf{G}}^{0}\widetilde{\mathbf{G}}^{1-0}\widetilde{\mathbf{itt}}$	-0.06	-1	1	0.3
IV4: $\mathbf{J}\widetilde{\mathbf{G}}^{1-0}\widetilde{\mathbf{G}}^{0}\widetilde{\mathbf{itt}}$	-0.08	-1	1	0.4
IV5: $\mathbf{J}\left(\widetilde{\mathbf{G}}^{0}\right)^{3}\widetilde{\mathbf{itt}}$	0.22	0	1	0.4
IV6: $\mathbf{J}\left(\widetilde{\mathbf{G}}^{1-0}\right)^{3}\widetilde{\mathbf{itt}}$	0.03	-3.5	3	0.7
IV7: $\mathbf{J}\left(\widetilde{\mathbf{G}}^{0}\right)^{2}\widetilde{\mathbf{G}}^{1-0}\widetilde{\mathbf{itt}}$	-0.08	-1	1	0.3
IV8: $\mathbf{J}\left(\widetilde{\mathbf{G}}^{1-0}\right)^{2}\widetilde{\mathbf{G}}^{0}\widetilde{\mathbf{itt}}$	0.11	-1.3	2	0.5
IV9: $\mathbf{J}\widetilde{\mathbf{G}}^{0}\left(\widetilde{\mathbf{G}}^{1-0}\right)^{2}\widetilde{\mathbf{itt}}$	0.12	-1.4	2	0.4
IV10: $\mathbf{J}\widetilde{\mathbf{G}}^{1-0}\left(\widetilde{\mathbf{G}}^{0}\right)^{2}\widetilde{\mathbf{itt}}$	-0.07	-1	1	0.4
IV11: $\mathbf{J}\widetilde{\mathbf{G}}^{0}\widetilde{\mathbf{G}}^{1-0}\widetilde{\mathbf{G}}^{0}\widetilde{\mathbf{itt}}$	-0.11	-1	1	0.3
IV12: $\mathbf{J}\widetilde{\mathbf{G}}^{1-0}\widetilde{\mathbf{G}}^{0}\widetilde{\mathbf{G}}^{1-0}\widetilde{\mathbf{itt}}$	0.04	-2	1.5	0.3

Notes: $N = 915$.

Table C4: Descriptive statistics for the dyadic sample

	t	**Mean**	**Min**	**Max**	**Std. Dev.**
g_{ij}	0	0.01	0	1	0.07
g_{ij}	1	0.01	0	1	0.08
itt_{ij}	1	0.76	0	1	0.43
Marital status	0	0.79	0	1	0.41
Marital status	1	0.73	0	1	0.44
Abs. diff. children	0	1.41	0	7	1.18
Abs. diff. children	1	1.29	0	6	1.06
Abs. diff hh members	0	1.80	0	11	1.53
Abs. diff hh members	1	1.82	0	14	1.61
Shock livestock	0	0.15	0	1	0.36
Shock livestock	1	0.04	0	1	0.20
Shock death	0	0.02	0	1	0.15
Shock death	1	0.01	0	1	0.11

Notes: $t = 0$ refers to baseline, and $t = 1$ refers to endline. $N = 56,308$.

Table C5: Dyadic regressions

itt_{ij}	0.0021*
	(0.0013)
marital status$_{ij}$	-0.0014
	(0.0013)
Abs. diff. children$_{ij}$	0.0000
	(0.0004)
Abs. diff. hh members$_{ij}$	0.0001
	(0.0003)
shock livestock$_{ij}$	0.0015
	(0.0012)
shock death$_{ij}$	-0.0013
	(0.0019)
$t = 1$	-0.0009
	(0.0011)
Observations	56,308

Notes: This table reports the OLS estimates of the dyadic intent-to-treat regressions over the row-standardized interaction matrix. Robust standard errors account for dyadic dependence (Fafchamps and Gubert, 2007). Statistically significant coefficients are indicated as follows: * 10%, ** 5%, *** 1%.