Identification and sensitivity analysis of contagion effects in randomized placebo-controlled trials

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Summary. In social science research, interference between units is the rule rather than the exception. Contagion represents one key causal mechanism of such spillover effects, where one's treatment affects the outcome of another individual indirectly by changing the treated unit's own outcome. Alternatively, the treatment of one individual can affect the outcome of another person through other mechanisms. We consider the identification and sensitivity analysis of contagion effects. We analyse a randomized placebo-controlled trial of the get out the vote campaign, in which canvassers were sent to randomly selected households with two registered voters but encouraged only one voter within each household to turn out in an upcoming election. To address the problem of non-compliance, the experiment includes a placebo arm, in which canvassers encourage voters to recycle. We show how to identify and estimate the average contagion and direct effects by decomposing the average spillover effect. Our analysis examines whether canvassing increases the turnout of a non-contacted voter by altering the vote intention of a contacted voter or through other mechanisms. To address the potential violation of key identification assumptions, we propose non-parametric and parametric sensitivity analyses. We find robust contagion effects among some households.

Keywords: Contagion effect; Infectious effect; Interference between units; Non-compliance; Spillover effect

1. Introduction

Over the last decade, a major methodological advancement in the causal inference literature has been made by considering the possible interference between units, in which the treatment assignment of one unit affects the outcome of another unit (see, for example, Halloran and Struchiner (1995), Sobel (2006), Rosenbaum (2007), Hudgens and Halloran (2008), Tchetgen Tchetgen and VanderWeele (2010), Manski (2013), Aronow and Samii (2017), Athey et al. (2018) and Imai et al. (2018), among many others). When studying such spillover effects as the main scientific quantities of interest, applied researchers often wish to understand the underlying causal mechanisms through which interference arises. Specifically, the treatment assignment of one individual can influence the outcome of another person directly or indirectly through the outcome of the treated individual. For example, this distinction between contagion and direct interference has been found useful in studies of infectious diseases (VanderWeele et al., 2012, 2014; Ogburn and VanderWeele, 2014, 2017).

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In this paper, we study the identification and sensitivity analysis of contagion and direct effects in the presence of non-compliance and measurement error by using randomized placebo-controlled trials (RPCTs). The RPCTs refer to randomized experiments that include a placebo arm as well as the treatment and control arms to deal with non-compliance effectively. We make several methodological contributions to the existing literature. First, we extend the work of VanderWeele et al. (2012) and Yamamoto (2013) on interference and mediation respectively, to the setting where missing data are present and a placebo condition is available. We decompose the average spillover effect into the average contagion and direct effects and establish their non-parametric identification. Second, we derive the bounds on the causal effects and propose a non-parametric sensitivity analysis for the possible existence of non-differential measurement error. Third, we propose a parametric sensitivity analysis for the coexistence of the measurement error and unobserved confounders, which generalizes the sensitivity analysis of Imai et al. (2010) to account for measurement error of the mediator.

The methodology proposed is motivated by an RPCT of the get out the vote (GOTV) campaign (Nickerson, 2008). In this experiment, the researcher conducted a randomized evaluation of face-to-face canvassing, targeting households with two registered voters. Groups of randomly selected households were visited, and voters who answered the door were exposed either to a GOTV appeal or an encouragement to recycle waste, which is a placebo treatment. The third group of randomly selected households received no visit. After the election, the researcher collected the turnout records of all registered voters of these households from administrative data and examined whether contacting one registered voter affects the turnout of the other non-contacted registered voter in the same household.

There are two possible ways in which canvassing one registered voter can influence the turnout of the other registered voter within the same household. As hypothesized in the original study, the mechanism of contagion implies that a canvasser first persuades one voter to turn out who in turn convinces the other voter in the same household to vote (Nickerson, 2008). However, it is also possible that canvassing directly affects the turnout of the non-contacted voter by heightening the salience of the election without affecting the turnout of the contacted individual. For example, a canvasser may talk to a voter who would turn out regardless of being contacted but still can influence the other voter of the same household by prompting conversation about the election. The original analysis ignores the unreachable households where no voter answered the door and assumes away the direct effect of canvassing. In this paper, we use the potential outcomes framework to define the causal estimands formally and to clarify the identification assumptions. Our analysis finds that there are robust contagion effects among some households, including those whose contacted voter is a Democrat and non-contacted voter is a Republican.

In the study of elections, political scientists have also examined the spillover effects of voter mobilization within and across households (e.g. Nickerson (2008), Rogowski and Sinclair (2012), Sinclair (2012) and Sinclair et al. (2012)). All these studies, however, focus on spillover effects. In the current paper, we adopt a framework which is similar to that in VanderWeele et al. (2013), which considers spillover as a mediating mechanism, and we study the decomposition of spillover effects into contagion and direct effects while utilizing a placebo treatment to address non-compliance.

The rest of the paper is organized as follows. Section 2 introduces an RPCT of the GOTV campaign that motivates the methodology proposed. In Section 3, we establish the non-parametric identification conditions and propose parametric and non-parametric estimation strategies. We also develop non-parametric and parametric sensitivity analyses to examine the consequences of potential violations of key identification assumptions. In Section 4, we apply the proposed methodology to the data that are described in Section 2. Finally, Section 5 concludes.
The replication archive for this article is published as Imai and Jiang (2019) and the data that are analysed in the paper and the programs that were used to analyse them can be obtained also from


2. Randomized placebo-controlled trial of the get out the vote campaign

2.1. The experiment

In an influential study, Nickerson (2008) conducted an RPCT of the GOTV campaign during the 2002 Congressional Primary Election and examined how the effect of canvassing spreads within a household. This election had low salient races with little mobilization efforts by campaigns. In each of the two cities, Denver and Minneapolis, where the experiment was conducted, canvassers were sent to a total of 956 randomly selected households with two registered voters (562 in Denver and 394 in Minneapolis). There were three arms in this experiment—treatment, placebo and control—and each household was randomly assigned to one of the conditions. In the treatment condition, a canvasser was instructed to encourage a registered voter who answered the door to turn out in the upcoming election. In the placebo condition, a canvasser asked the voter to recycle waste without mentioning voting. Finally, the households in the control condition received no canvasser. After the election, the researcher collected the turnout information of all voters in the experiment, including those who were not contacted by a canvasser, from the voter records, which are publicly available.

The placebo condition is useful in this experiment because canvassers could not talk to some voters. Indeed, in part because many voters were not at home, the proportion of households where a registered voter answered the door was only slightly above 30% in Denver and a little more than 40% in Minneapolis. Throughout the paper, we use the phrase ‘contacted voters’ to refer to those who would answer the door if a canvasser visits their households, and we use the phrase ‘non-contacted voters’ to refer to those who would not answer the door. Since those who answered the door systematically differ from those who did not, a naive comparison between the contacted voters in the treatment group and all voters in the control group will result in a biased estimate of the average treatment effect. However, if we assume that the recycling message has no effect on turnout, then it is possible to compare the contacted voters between the treatment and placebo arms to ascertain the causal effect of the GOTV campaign. Moreover, by utilizing the placebo group, we can also reliably estimate the average treatment effect among those in the treatment group who were not contacted by canvassers, representing the spillover effect of the GOTV campaign within households.

Table 1 reproduces the results that were reported in the original study. Table 1 shows the turnout rates separately for contacted and non-contacted voters within each of the three groups—the treatment (GOTV), placebo (recycling) and control groups—where the standard errors are given in parentheses. We observe that the average treatment effect of the GOTV campaign is estimated to be 9.4 percentage points among the contacted voters in the pooled analysis.

A more interesting finding is that the estimated average treatment effect among the non-contacted voters, which represents the estimated average within-household spillover effect, is equal to 5.7 percentage points. This estimate is obtained by computing the difference in the turnout rate of the non-contacted voters between the GOTV and placebo conditions. Although the standard errors are relatively large, this result is consistent with the findings of several observational studies that cohabiting spouses have a great influence on each other’s political
<table>
<thead>
<tr>
<th></th>
<th>Results for Denver</th>
<th>Results for Minneapolis</th>
<th>Results for pooled sample</th>
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<tr>
<td></td>
<td>Contacted</td>
<td>Non-contacted</td>
<td>Contacted</td>
</tr>
<tr>
<td>Turnout rates</td>
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<td></td>
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<tr>
<td>GOTV</td>
<td>0.477</td>
<td>0.424</td>
<td>0.272</td>
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<td></td>
<td>(0.030)</td>
<td>(0.029)</td>
<td>(0.031)</td>
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<tr>
<td>Recycling waste</td>
<td>0.391</td>
<td>0.369</td>
<td>0.162</td>
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<tr>
<td></td>
<td>(0.029)</td>
<td>(0.029)</td>
<td>(0.027)</td>
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<tr>
<td>Control</td>
<td>0.384</td>
<td>0.172</td>
<td>0.312</td>
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<tr>
<td></td>
<td>(0.012)</td>
<td>(0.013)</td>
<td>(0.009)</td>
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<td>Causal effects</td>
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<tr>
<td>Treatment effect</td>
<td>0.086</td>
<td>0.055</td>
<td>0.110</td>
</tr>
<tr>
<td></td>
<td>(0.042)</td>
<td>(0.041)</td>
<td>(0.041)</td>
</tr>
<tr>
<td>Placebo effect</td>
<td>0.006</td>
<td>0.005</td>
<td>0.005</td>
</tr>
<tr>
<td></td>
<td>(0.017)</td>
<td>(0.018)</td>
<td>(0.113)</td>
</tr>
<tr>
<td>Number of households</td>
<td>1124</td>
<td>786</td>
<td>1910</td>
</tr>
</tbody>
</table>

†The estimated average treatment effect is calculated as the difference in turnout rate between the treatment (GOTV) and placebo (recycling) groups, separately for contacted and non-contacted voters. The estimated average placebo effect is computed as the difference in turnout rate between the placebo and control groups. The numbers in parentheses represent the standard errors.

views (e.g. Huckfeldt and Sprague (1991), Mutz (1998) and Zuckerman et al. (2005)). Finally, consistent with the assumption that was made in the original study, the average placebo effect is found to be statistically indistinguishable from zero.

### 2.2. Causal mechanisms of the spillover effect

Nickerson (2008) attributed the positive average treatment effect among the non-contacted voters as evidence for contagion within households, i.e. canvassing first increased the contacted voter’s intention to turn out, which in turn made it more likely for the non-contacted voter within the same household to vote in the upcoming election. Although this is certainly possible, another scenario is that canvassing directly influenced the non-contacted voter’s turnout without altering the vote intention of the contacted voter. For example, a canvasser may talk to a voter who would turn out regardless of being contacted but still can influence the other voter who lives in the same household by heightening the salience of the election and prompting conversation about politics. The goal of this paper is to consider these two potential causal mechanisms in the formal framework of causal inference with interference.

Fig. 1 presents the directed acyclic graph (DAG) depicting the two potential causal mechanisms of the spillover effect that was discussed above. Fig. 1 shows that a canvasser first visits a household at time $t_0$, and then a possible contact to one of the registered voters in the household occurs at time $t_0$. This contact may affect the vote intention of the contacted voter immediately after being contacted, which we denote as time $t_0 +$. Since we define the mediator to be the vote intention of a contacted voter immediately after the canvassing, we essentially eliminate the possibility that the non-contacted partner of the same household influences the vote intention of the contacted voter. Finally, the turnout of each voter is recorded on the election day, which is
Contagion Effects in Randomized Placebo-controlled Trials

Fig. 1. DAG depicting the two potential causal mechanisms of the spillover effect in the randomized placebo-controlled trial of the GOTV campaign: the subscript for vote intention and turnout indexes the two registered voters in the same household where voter 1 or voter 2 is respectively the voter contacted or not contacted by a canvasser; the assumed timing of realization of each variable is denoted by \( t_{0-} < t_0 < t_{0+} < t_1 \) where \( t_0 \) represents the time immediately after \( t_{0-} \) and before \( t_{0+} \); the causal paths (a) and (b) represent the contagion and direct effects respectively.

Denoted by time \( t_1 \). Because voters cast their ballot on the election day, we ignore the subtle and unobserved difference in the time of voting that may exist, assuming that one voter’s turnout has no direct causal effect on the turnout of the other voter.

In Fig. 1, path (a) represents the contagion effect that was hypothesized by Nickerson (2008), in which canvassing influences the turnout of the non-contacted voter of the visited household by changing the vote intention of the contacted voter. In contrast, path (b) represents the direct effect, in which canvassing impacts the turnout of the non-contacted voter without changing the vote intention of the voter who answered the door and talked to a canvasser. This direct effect includes all possible causal mechanisms other than the contagion effect. Our goal is to identify the contagion and direct effects.

There are several challenges in identifying the contagion and direct effects. First, there may be unobserved confounders that affect the vote intention of the contacted voter and the turnout of the non-contacted voter. An example of such an unobserved confounder is the level of interest in politics that is shared by both voters. Fortunately, we need not entertain the possibility that these unobserved confounders are affected by canvassing because we define the vote intention of the contacted voter to be the intention that is formed immediately after the canvassing. Second, we do not observe the vote intentions of voters although we observe their turnout. Directly asking voters about their vote intention, even if possible, may not be desirable because self-reported vote intention may systematically differ from the true vote intention. Finally, voters may change their mind between the time of canvassing and the election day, and hence we must account for possible measurement error.

3. The methodology proposed

In this section, we describe the methodology proposed. We first derive the non-parametric identification condition for the average contagion and direct effects and propose a non-parametric sensitivity analysis. To widen the application, we then show how to use parametric modelling for the estimation and sensitivity analyses.

3.1. The set-up

For each household \( i = 1, 2, \ldots, N \), let \( Z_i \) denote the assigned treatment where \( Z_i = 1 \) if the household is assigned to the GOTV appeal condition, \( Z_i = 2 \) for being assigned to the recycling waste pitch condition and \( Z_i = 0 \) for being assigned to the control condition of no canvassing. Each household in the sample has two registered voters, and a canvasser will try to contact one of them if the household is assigned to either the treatment or placebo condition. Thus, for a household with \( Z_i = 1 \) or \( Z_i = 2 \), we use \( D_i = 1 \) to represent a successful contact whereas \( D_i = 0 \) denotes a failure, which may occur, for example, if neither voter answers the door.
Let $D_i(z)$ denote the potential value of the actual contact variable under the treatment condition $z = 0, 1, 2$ where the observed contact variable is given by $D_i = D_i(Z_i)$. Since no voter in the control group will be contacted, we always have $D_i(0) = 0$. Furthermore, the placebo-controlled design implies that the success of contact does not depend on the type of canvassing message, i.e. $D_i(1) = D_i(2)$, because before contact a voter does not know the message that they will receive from a canvasser. Throughout this paper, as already implicit in this notation, we assume no interference across households (but not between two voters within each household) and the same version of treatments (Rubin, 1980), i.e. the treatment assignment of one household is assumed not to affect the actual contact and turnout of voters in another household.

Under this setting, we define two principal strata by using the potential contact variable $D_i(z)$ (Frangakis and Rubin, 2002). One principal stratum consists of the complier households. A complier household is defined as the household whose voter would answer the door when visited by a canvasser, i.e. $D_i(1) = D_i(2) = 1$. The other principal stratum represents the never-taker households whose voters would not answer the door, i.e. $D_i(1) = D_i(2) = 0$. We use $G_i = c$ to denote a complier household whereas $G_i = n$ represents a never-taker household. There are no always-taker households in this experiment because canvassers do not visit the households in the control group $Z_i = 0$.

Similarly to the truncation-by-death problem (Zhang and Rubin, 2003; Imai, 2008), our causal estimands of interest are defined only for the complier households $G_i = c$. Under the causal model that is depicted in Fig. 1, if a canvasser visits a complier household, one of the registered voters who answers the door forms a vote intention immediately after the canvassing. We use $Y_{i1}^*(z)$ for $z = 0, 1, 2$, to represent the potential value of this latent binary variable, which is equal to 1 if this contacted voter plans to turn out in the upcoming election and is equal to 0 otherwise. Note that this variable is unobservable and defined only for the contacted voter in a complier household who would answer the door when visited by a canvasser. The realized but unobserved value of this vote intention variable is denoted by $Y_{i1}^* = Y_{i1}^*(Z_i)$.

Finally, we observe the turnout for all voters across households from publicly available voter records. For a complier household, we use $Y_{i1}$ and $Y_{i2}$ to represent the observed binary turnout variable for the contacted and non-contacted voters respectively. For the contacted voter in a complier household, we define the potential turnout variable as a function of treatment assignment, i.e. $Y_{i1}(z)$. In contrast, for the non-contacted voter in the same household, we define the potential turnout variable as a function of both treatment assignment and the vote intention of the contacted voter $Y_{i1}^* = Y_{i1}^*$, i.e. $Y_{i2}(z, Y_{i1}^*)$ where $Y_{i1}^* = 0, 1$. This set-up implies that the turnout of the non-contacted voter may be influenced by canvassing either directly or through the change in the vote intention of the contacted voter within the same household. Therefore, the observed turnout variables for these voters are given by $Y_{i1} = Y_{i1}(Z_i)$ and $Y_{i2} = Y_{i2}(Z_i, Y_{i1}^*(Z_i))$.

In addition to these variables, the data contain several pretreatment covariates for each voter including their age, gender, partisanship and turnout history. We use $X_i$ to denote these pretreatment covariates for each household $i$. For the reminder of the paper, we assume that the vector $\{Z_i, X_i, \{D_i(z), Y_{i1}^*(z), Y_{i1}(z), Y_{i2}(z)\}_{z=0}^2\}$ is independently sampled from a superpopulation for $i = 1, 2, \ldots, N$, and thus the observed data vector $\{Z_i, D_i, Y_{i1}, Y_{i2}, X_i\}$ is also independently and identically distributed.

### 3.2. Causal quantities of interest

We now define the causal quantities of interest by focusing on complier households. We begin by defining the average spillover effect of the GOTV appeal on a voter in a complier household who would not be directly contacted by a canvasser (e.g. Halloran and Struchiner (1995), Sobel (2006) and Hudgens and Halloran (2008)):
\[ \theta = \mathbb{E}[Y_{12}\{1, Y_{i1}^*(1)\} - Y_{12}\{0, Y_{i1}^*(0)\}|G_i = c]. \]

This parameter quantifies how canvassing may influence a voter in a complier household by contacting the other voter. In this paper, we consider the causal mechanisms of this average spillover effect by using the idea from the causal mediation literature (e.g. Robins and Greenland (1992), Pearl (2001), Imai et al. (2010) and VanderWeele (2015)).

Specifically, we decompose the average spillover effect into the sum of the contagion effect and direct effect by considering the vote intention of the contacted voter as the mediator. As described earlier, the idea is that a canvasser may influence the turnout of a non-contacted voter in a complier household either directly or indirectly by altering the vote intention of the contacted voter. The contagion effect would then arise if a contacted voter, after being convinced by a canvasser to turn out, influences the turnout of the non-contacted voter in the same household.

Formally, we define the average contagion effect as

\[ \tau(z) = \mathbb{E}[Y_{12}\{z, Y_{i1}^*(z)\} - Y_{12}\{z, Y_{i1}^*(0)\}|G_i = c] \]

for \( z = 0, 1 \). For example, \( \tau(0) \) quantifies how canvassing affects the turnout of non-contacted voters by changing the vote intention of the contacted voter in the same household while holding the treatment assignment constant at the control condition (path (a) of Fig. 1). This effect represents the indirect effect of canvassing through the change in vote intention immediately after contact and does not capture the indirect effect of later changes.

Next, the average direct effect is defined as

\[ \eta(z) = \mathbb{E}[Y_{12}\{1, Y_{i1}^*(z)\} - Y_{12}\{0, Y_{i1}^*(z)\}|G_i = c] \]

for \( z = 0, 1 \). For example, \( \eta(0) \) quantifies how canvassing changes the turnout of a non-contacted voter if the vote intention of the contacted voter within the same household is held unchanged at the same value as the value that would be realized under the control condition (path (b) of Fig. 1). In the epidemiological literature, \( \eta(z) \) is referred to as the infectiousness effect (VanderWeele et al., 2012). Thus, the average spillover effect is equal to the sum of the average contagion and direct effects:

\[ \theta = \tau(1) + \eta(0) = \tau(0) + \eta(1). \]

### 3.3. Assumptions

We next introduce a set of identification assumptions for the average contagion and direct effects. We begin by assuming that the treatment assignment is randomized.

**Assumption 1** (randomization of treatment assignment).

\[ Z_i \perp \perp \{D_i(z), Y_{i1}^*(z), Y_{i2}(z, y_{i1}^*), X_i\} \]

for \( z = 0, 1, 2 \) and \( y_{i1}^* = 0, 1 \).

In our application, this assumption is known to hold because the treatment assignment is randomized. In the on-line appendix S1, we give a weaker version of assumption 1, i.e. strong ignorability, which is sufficient for the results that are derived in our paper. In observational studies, this alternative assumption implies that \( X_i \) includes all the confounders between \( Z_i \) and \( \{D_i, Y_{i1}^*, Y_{i2}\} \).

Next, we introduce an assumption, which was first introduced by Yamamoto (2013), that is analogous to the sequential ignorability assumption of Imai et al. (2010) for the standard causal mediation analysis. The key difference between the two ignorability assumptions is that
the ignorability of the mediator is assumed only among compliers conditionally on the realized value of treatment assignment and the pretreatment covariates. Since the compliance status is unobserved, this assumption represents a type of latent ignorability assumption (Frangakis and Rubin, 1999). In our application, the vote intention of contacted voters is considered as the mediator.

\textit{Assumption 2} (latent ignorability of mediator among compliers).

\[ Y_{i2}(z', y^*_1) \perp\!\!\!\perp Y_{i1}^*(z)|Z_i = z, G_i = c, X_i \]

for \( z, z' = 0, 1 \), and \( y^*_1 = 0, 1 \).

In our application, assumption 2 implies that, among complier households, the potential vote intention of a contacted voter is independent of the potential turnout of the non-contacted voter in the same household once we condition on the treatment assignment status of the household and the pretreatment covariates. This assumption is violated if there is a pretreatment or post-treatment confounder between the vote intention of the contacted voter and the turnout of the non-contacted voter within a complier household even after conditioning on the treatment assignment status and observed pretreatment covariates. In the current application, the assumption of no post-treatment confounding appears to be reasonable because the mediator refers to the vote intention of the contacted voter formed immediately after canvassing. Given the short time span between the realization of the treatment and that of the mediator, it is unlikely that there is another post-treatment variable, which affects the mediator and outcome but is also affected by the treatment. However, we cannot exclude the possible existence of unobserved pretreatment confounders. For example, voters in the same household may share similar political ideology and efficacy that may affect vote intention and turnout, but these factors are often unobservable. To address this potential violation of assumption 2, we develop a sensitivity analysis in Section 3.5.

Because we cannot define \( Y_{i1}^* \) for never-taker households, and both \( Y_{i1}^* \) and \( Y_{i2} \) are unobserved for the complier households in the control group (we do not know which voter can be contacted in these households), we cannot use the identification result of Yamamoto (2013) who relied on the exclusion restriction as well as the latent ignorability of the mediator. Thus, we consider an alternative approach by exploiting the RPCT design of this experiment. First, we assume that among compliers the average placebo effect is zero.

\textit{Assumption 3} (zero average placebo effect among compliers).

\[ \mathbb{E}\{Y_{i1}^*(2)|G_i = c, X_i\} = \mathbb{E}\{Y_{i1}^*(0)|G_i = c, X_i\}, \]

\[ \mathbb{E}\{Y_{i2}(2, y^*_1)|G_i = c, X_i\} = \mathbb{E}\{Y_{i2}(0, y^*_1)|G_i = c, X_i\} \]

for \( y^*_1 = 0, 1 \).

In our application, the assumption implies that among complier households the recycling waste pitch is assumed to have, on average, no effect on either the vote intention of a contacted voter or the turnout of the non-contacted voter. The assumption is violated if, for example, a conversation about recycling with a canvasser leads to an increase in political interest. Because we do not observe the compliance status of households in the control group, assumption 3 is not directly testable. The results in Table 1, however, show that the placebo effect for the whole population (i.e. complier and non-complier households together) is not statistically distinguishable from zero. Since the placebo should have no effect on the turnout of the voters in non-complier households who are never contacted by a canvasser, the results suggest that the placebo is likely
to have little effect on the turnout of complier households, providing empirical support for assumption 3 in this application.

Finally, we assume that the observed turnout of the contacted voter within a complier household serves as a perfect proxy for the unobserved vote intention.

\textit{Assumption 4} (perfect proxy for mediator among compliers).

\[ Y_{i1}^* = Y_{i1} \]

for all \( i \) with \( G_i = c \).

This assumption is violated if a contacted voter changes his or her mind at some time between the time of contact and the election day. Indeed, previous studies show that the actual turnout may systematically differ from the pre-election vote intention for a variety of reasons including the closeness of election and last-minute mobilization efforts by campaigns (e.g. Ansolabehere and Iyengar (1994), Achen and Blais (2010) and Westwood \textit{et al.} (2018)). Although the primary elections under consideration were low salient races that were not close, we address this possibility by considering the following alternative assumption of non-differential measurement error for the mediator.

\textit{Assumption 5} (non-differential measurement error of the mediator among compliers).

\[ \text{Pr}(Y_{i1} = y_1^* | Y_{i1}^* = y_1^*, G_i = c, Y_{i2}, Z_i, X_i) = \text{Pr}(Y_{i1} = y_1^* | Y_{i1}^* = y_1^*, G_i = c, X_i) \]

for \( y_1^* = 0, 1 \).

The assumption states that although the actual turnout of a contacted voter may be different from his or her vote intention immediately after canvassing, it depends on neither the treatment assignment nor on the actual turnout of the non-contacted voter once we condition on the vote intention and pretreatment covariates. Assumption 5 holds, for example, if the actual turnout of a contacted voter depends only on his or her vote intention and the pretreatment covariates. Assumption 5 essentially excludes the possible existence of confounders between the outcome and the proxy variable of the mediator. It also assumes the absence of the causal effect of treatment assignment on the proxy variable other than through the mediator. We follow much of the measurement error literature which assumes that the error does not depend on the outcome after conditioning on covariates (e.g. Carroll \textit{et al.} (2006)). The exception includes Imai and Yamamoto (2010) who studied the consequences of differential measurement error for causal inference. In Section 3.5, we propose a parametric sensitivity analysis to address the potential violation of assumption 5.

### 3.4. Non-parametric identification and sensitivity analysis

We establish the non-parametric identification of the average spillover, contagion and direct effects under the assumptions that were introduced in Section 3.3.

\textit{Theorem 1} (non-parametric identification). Under assumptions 1 and 3, the average spillover effect, i.e. \( \theta \) defined in equation (1), is non-parametrically identified as

\[ \theta = \sum_x \{ \mathbb{E}(Y_{i2}|D_i = 1, Z_i = 1, X_i = x) - \mathbb{E}(Y_{i2}|D_i = 1, Z_i = 2, X_i = x) \} \ \text{Pr}(X_i = x|D_i = 1). \]

Under assumptions 1–4, the average contagion effect, i.e. \( \tau(z) \) defined in equation (2), and the average direct effect, i.e. \( \eta(z) \) defined in equation (3), are non-parametrically identified for \( z = 0, 1 \):
\[\tau(z) = \sum_x [\Pr(X_i = x | D_i = 1) \{m_x(1, 2 - z) - m_x(0, 2 - z)\} \{q_x(1, 1) - q_x(1, 2)\}],\]
\[\eta(z) = \sum_x \left[ \Pr(X_i = x | D_i = 1) \sum_{y=0}^{1} \{m_x(y, 1) - m_x(y, 2)\} q_x(y, 2 - z) \right],\]

where \(m_x(y, z) = \mathbb{E}(Y_i | D_i = 1, Z_i = z, Y_{i1} = y, X_i = x)\) and \(q_x(y, z) = \Pr(Y_{i1} = y | D_i = 1, Z_i = z, X_i = x)\).

A proof is given in the on-line appendix S1. When the covariates \(X_i\) are continuous, we replace summation with integration. For the estimation of the average contagion and direct effects, we can first estimate \(m_x(y, z), q_x(y, z)\) and \(\Pr(X_i = x | D_i = 1)\) from the observed data and then plug in these estimates on the basis of the expressions of \(\tau(z)\) and \(\eta(z)\) that are given in theorem 1.

When \(X\) is discrete and the sample size is sufficiently large, we can non-parametrically estimate these terms by their sample analogues. When \(X\) contains continuous variables and/or the sample size is small, assuming parametric models is convenient.

The non-parametric identification of the average contagion and direct effects in theorem 1 relies on the existence of a perfect proxy for the mediator, i.e. assumption 4. However, this assumption may not be credible in the current application. Thus, we next present a more general non-parametric identification result under an alternative assumption of non-differential measurement error given as assumption 5. Unfortunately, under this assumption, only the average spillover effect is identifiable. To understand the nature of this unidentifiability and to facilitate a sensitivity analysis, we express the average contagion and direct effects as functions of the following unknown parameter as well as identifiable parameters:

\[p_x(y) = \Pr(Y_{i1} = y | Y_{i1}^* = y, G_i = c, X_i = x)\]

for \(y = 0, 1\). This parameter characterizes the magnitude of measurement error and hence the degree to which assumption 4 is violated. Specifically, if \(p_x(y) = 1\) for all \(y\) and \(x\), assumption 5 reduces to assumption 4. The following theorem is a direct generalization of theorem 1.

**Theorem 2** (non-parametric identification under non-differential measurement error). Under assumptions 1, 3 and 5, the average spillover effect, i.e. \(\theta\) defined in equation (1), is identified as in theorem 1. Under assumptions 1–3 and 5, the average contagion effect, i.e. \(\tau(z)\) defined in equation (2), and the average direct effect, i.e. \(\eta(z)\) defined in equation (3), are non-parametrically identified for \(z = 0, 1\) if \(p_x(y)\) is known for \(y = 0, 1\), and all \(x\):

\[\tau(z) = \sum_x [\Pr(X_i = x | D_i = 1) r_x(z) \{m_x(1, 2 - z) - m_x(0, 2 - z)\} \{q_x(1, 1) - q_x(1, 2)\}],\]
\[\eta(z) = \sum_x \left( \Pr(X_i = x | D_i = 1) \sum_{y=0}^{1} \{m_x(y, 1) - m_x(y, 2)\} q_x(y, 2 - z) - \{1 - r_x(1 - z)\} m_x(y, 1 + z) \right.\]
\[\times \left. \{q_x(y, 2) - q_x(y, 1)\} \right),\]

where

\[r_x(z) = \frac{q_x(1, 2 - z) \{1 - q_x(1, 2 - z)\}}{(p_x(1) - q_x(1, 2 - z))(p_x(0) - \{1 - q_x(1, 2 - z)\}).\]

A proof is given in the on-line appendix section S1. Theorem 2 shows that under assumption 5 the average contagion effect is equal to the product of the coefficient \(r_x(z)\) and the average contagion effect under the assumption of no measurement error. It is immediate that when \(p_x(y) = 1\) for all \(y\) and \(x\), which represents the scenario of no measurement error, we have
\[ r_x(z) = 1 \] and hence theorem 2 reduces to theorem 1. This suggests that the coefficient \( r_x(z) \) plays an essential role in the understanding of potential bias in the presence of measurement error. Similarly to theorem 1, if \( p_x(y) \) is known, we can obtain the non-parametric estimates of the average contagion and direct effects by plugging in the sample analogues of \( m_x(y, z), q_x(y, z) \) and \( \Pr(X_i = x \mid D_i = 1) \) based on the expressions of \( \tau(z) \) and \( \eta(z) \) that are given in theorem 2.

In general, \( r_x(z) \) can be rewritten as the product of two terms, both of which are no less than 1:

\[
r_x(z) = \frac{q_x(1, 2 - z)}{q_x(1, 2 - z) - \{1 - p_x(0)\}} \frac{q_x(0, 2 - z)}{q_x(0, 2 - z) - \{1 - p_x(1)\}} \geq 1.
\]

Thus, in the presence of non-differential measurement error, the average contagion effect tends to suffer from attenuation bias. In contrast, the existence of such measurement error is likely to yield an overestimate of the average direct effect. The magnitude of these biases directly depends on that of measurement error, which is represented by \( 1 - p_x(y) \). This is consistent with the conclusion on the natural direct and indirect effects in the context of mediation analysis (Ogburn and VanderWeele, 2012).

This observation naturally leads to a non-parametric sensitivity analysis based on the accuracy of the proxy variable \( p_x(z) \). We consider the sensitivity analysis parameter \( p \), which represents the lower bound of \( p_x(1) + p_x(0) \):

\[
p_x(1) + p_x(0) \geq p \tag{4}
\]

for all \( x \). Thus, the sensitivity parameter \( p \) quantifies the magnitude of measurement error from the maximal amount of measurement error (\( p = 0 \)) to no measurement error (\( p = 2 \)) under the assumption of non-differential measurement error. The following corollary presents the sharp bounds on the average direct effect when \( p \) is reasonably large. To conduct a sensitivity analysis, we examine how the bounds change as we vary the value of the sensitivity parameter \( p \).

**Corollary 1** (non-parametric sensitivity analysis under non-differential measurement error). Suppose that we have \( p > \max\{\frac{3}{2}, 1 + \max_x q_x(1, 2 - z), 2 - \min_x q_x(1, 2 - z)\} \) where \( p \) is the sensitivity analysis parameter defined in equation (4). Then, under assumptions 1–3 and 5, the sharp bounds for \( \tau(z) \) are given by

\[
\tau(z) \in \left( \sum_x \Pr(X_i = x \mid D_i = 1) \left[ I\{Q_x(z) \geq 0\} Q_x(z) + I\{Q_x(z) < 0\} Q_x(z) u_x(z) \right] \right) \sum_x \Pr(X_i = x \mid D_i = 1) \left[ I\{Q_x(z) \geq 0\} Q_x(z) u_x(z) + I\{Q_x(z) < 0\} Q_x(z) \right]
\]

where

\[
Q_x(z) = \left\{ m_x(1, 2 - z) - m_x(0, 2 - z) \right\} \{q_x(1, 1) - q_x(1, 2)\},
\]

\[
u_x(z) = \max \left\{ \frac{q_x(1, 2 - z)}{p + q_x(1, 2 - z) - 2}, \frac{1 - q_x(1, 2 - z)}{p - q_x(1, 2 - z) - 1} \right\}.
\]

A proof is given in the on-line appendix section S1. The upper and lower bounds are obtained by respectively first maximizing and minimizing the average contagion effect for the subpopulation with \( X_i = x \) and then averaging it over the distribution of \( X_i \) given \( D_i = 1 \). According to theorem 2, the average contagion effect for this subpopulation equals the product of \( r_x(z) \) and the average contagion effect under the perfect proxy assumption, i.e. \( Q_x(z) \). Thus, the bounds of \( \tau(z) \) follow directly from the bounds of \( r_x(z) \), which are given by \([1, u_x(z)]\) under the conditions...
of theorem 1. We note that, if the sensitivity parameter does not satisfy the requirements that were described above, then the resulting bounds are completely non-informative.

3.5. Parametric modelling and sensitivity analysis

Although the non-parametric identification conditions and sensitivity analysis that were presented above are useful, applied researchers may use a parametric model especially when the dimensionality of $X_i$ is high.

To facilitate practical applications including that presented in this paper, we propose parametric estimation and sensitivity analyses. On the basis of the DAG in Fig. 1, we begin by assuming the following models of the binary variables $Y_{i1}^*$ and $Y_{i2}$ for complier households $G_i = c$:

$$
\begin{align*}
Y_{i1}^*(z) &= 1\{\tilde{Y}_{i1}(z) > 0\}, \\
\tilde{Y}_{i1}(z) &= g(z, X_i) + \epsilon_{i1}, \\
Y_{i2}(z, y_{i1}^*) &= 1\{\tilde{Y}_{i2}(z, y_{i1}^*) > 0\}, \\
\tilde{Y}_{i2}(z, y_{i1}^*) &= f(z, y_{i1}^*, X_i) + \epsilon_{i2},
\end{align*}
$$

(5)

where $\epsilon_{ij} \sim_{\text{IID}} N(0, 1)$ for $j = 1, 2$. Although different distributions such as logistic and $t$-distributions can also be used for the two error terms, we choose the standard normal distribution, which makes it easier to develop a parametric sensitivity analysis. In this model, assumption 2 is satisfied because of the conditional independence between $\epsilon_{i1}$ and $\epsilon_{i2}$ given $X_i$ and $G_i = c$.

By treating $\tilde{Y}_{i1}$ and $\tilde{Y}_{i2}$ as missing data, we can use the expectation–maximization algorithm (Dempster et al., 1977) to estimate the parameters of this model. We use the estimated coefficients from the probit regressions of $Y_{i1}$ on $(Z_i, X_i)$ and $Y_{i2}$ on $(Z_i, Y_{i2}, X_i)$ as starting values. In the expectation step, we compute the expected values of $\tilde{Y}_{i1}$ and $\tilde{Y}_{i2}$ given the observed data and the current values of parameters, and, in the maximization step, we fit two regression models for $\tilde{Y}_{i1}$ and $\tilde{Y}_{i2}$ to update the parameter values. We repeat these two steps until convergence. Once we have obtained the maximum likelihood estimates of the model parameters, we use the formulae that were derived in theorem 1 to estimate the average spillover, contagion and direct effects.

In Section 3.4, non-parametric identification is achieved under assumptions 1–4. Whereas assumptions 1 and 3 are justified in randomized experiments, assumptions 2 and 4 may not be credible. In particular, assumption 2 is violated if there are unobserved confounders (e.g. shared interests in politics) between the vote intention of the contacted voter and the turnout of the non-contacted voter within the same household. Moreover, assumption 4 fails to hold if the vote intention of a contacted voter changes over time. Below, we develop a parametric sensitivity analysis for each case. In both cases, we modify the aforementioned expectation–maximization algorithm to conduct a sensitivity analysis. The details of the algorithm are given in the on-line appendix section S2.

Assumption 2 is violated if there is a latent confounder $U_1$ affecting both the vote intention of the contacted voter and the turnout of the non-contacted voter. This scenario is depicted as a DAG in Fig. 2 for complier households. This DAG contains no edge from the latent confounder $U_1$ to contact because contact is determined by canvassing and compliance behaviour. To develop a parametric sensitivity analysis for assumption 2, we borrow an idea from Imai et al. (2010) and introduce the correlation between two error terms as a sensitivity parameter:

$$
\left(\begin{array}{c}
\epsilon_{i1} \\
\epsilon_{i2}
\end{array}\right) \sim_{\text{IID}} N_2\left(\left(\begin{array}{c}0 \\
0\end{array}\right), \Sigma = \left(\begin{array}{cc}1 & \rho \\
\rho & 1\end{array}\right)\right)
$$

(6)
The variances of the error terms are set to 1 for the identification of the parameters. If $\rho = 0$, then assumption 2 holds because $Y_{i1}^* (z) \perp Y_{i2} (z, y_1^*) (Z_i = z, G_i = c, X_i)$. A non-zero value of $\rho$ can be interpreted as the existence of unobserved confounders among complier households even after conditioning on the observed pretreatment covariates. A positive or negative value of $\rho$ implies that the unobserved confounders affect $Y_{i1}^*$ and $Y_{i2}$ in respectively the same or opposite direction. As shown by Imai et al. (2010), we can also use an alternative parameterization based on the coefficient of determination rather than the correlation of error terms for this sensitivity analysis.

Next, we develop a parametric sensitivity analysis for assumption 4, which requires the actual turnout of a contacted voter to be identical to their vote intention at the time of contact. In particular, we assume the following additive measurement error structure:

$$Y_{i1} (z) = I\{Y_{i1}^* (z) + \zeta_i > 0\} \quad \zeta_i \overset{\text{IID}}{\sim} N(0, \sigma^2).$$  \hspace{1cm} (7)

The sensitivity parameter is $\sigma^2$, which represents the magnitude of the difference in vote propensity between the actual turnout and vote intention. Assumption 4 holds when $\sigma^2 = 0$ whereas a greater value of $\sigma^2$ implies a larger magnitude of measurement error. Unlike the traditional additive measurement error which is independent of all other variables, we introduce the correlation between the measurement error and the error term $\epsilon_{i2}$ in the outcome model for $Y_{i2}$ to account for unmeasured common causes affecting the turnout of two voters in the same household:

$$\begin{pmatrix} \zeta_i \\ \epsilon_{i2} \end{pmatrix} \overset{\text{IID}}{\sim} N_2 \left\{ \begin{pmatrix} 0 \\ 0 \end{pmatrix}, \Sigma = \begin{pmatrix} \sigma^2 & \rho_{e} \sigma \\ \rho_{e} \sigma & 1 \end{pmatrix} \right\}. \hspace{1cm} (8)$$

Fig. 3 shows the DAG for complier households in this sensitivity analysis. A non-zero value of $\rho_e$ can be interpreted as the existence of unobserved common causes affecting the turnout of two voters within a complier household. These common causes may include weather conditions and family obligations. A positive or negative value of $\rho_e$ implies that the unobserved common causes affect $Y_{i1}$ and $Y_{i2}$ in respectively the same or opposite direction. Note that equation (7) does not assume that the measurement error is non-differential, violating assumption 5.
is because, under this model, the turnout of the contacted voter in a complier household can depend on the treatment assignment and the turnout of the non-contacted voter conditionally on the covariates.

4. Empirical findings

In this section, we apply the proposed methodology to the data that were described in Section 2. We have developed non-parametric estimation and a sensitivity analysis in Section 3.4. Unfortunately, this methodology is not applicable to this particular study because of its limited sample size relative to the number of available covariates. In particular, assumption 2 demands that we condition on all pretreatment confounders, making non-parametric adjustment difficult. Therefore, we shall focus on the analysis by using the parametric model given in equation (5) with the following specification:

\[
\begin{align*}
g(z, x) &= \alpha_0 + \alpha_Z z + x^T \alpha_X + z x^T \alpha_{ZX}, \\
f(z, y_1, x) &= \beta_0 + \beta_Z z + \beta_Y y_1 + \beta_{ZY} z y_1 + x^T \beta_X + z x^T \beta_{ZX} + y_1 x^T \beta_{YX}.
\end{align*}
\]

We conduct a separate analysis for each city. For Denver, the pretreatment covariates \(x\) include age, squared age, gender (male or female), partisanship (Democrat or Republican) and turnout in the previous primary election. Because the gender and partisanship information is not available in Minneapolis, we do not adjust for them in the analysis of the Minneapolis data. To avoid strong functional form assumptions, we include all two-way interaction terms. Using this model, we first estimate the average spillover, contagion and direct effects under assumptions 1–4. In addition to the results for each city, we also present the pooled results by computing the weighted average of the city-specific estimates where the weights are proportional to the sample size in each city. We then conduct the proposed sensitivity analyses to examine the robustness of some findings in the presence of possible violations of assumptions 2 and 4.

4.1. Estimated average spillover, contagion and direct effects

Under assumptions 1–4, we separately fit the two probit models to the households with \(D_i = 1\). Once we have obtained the maximum likelihood estimates of the model parameters, we use the formulae of theorem 1 to obtain the estimated average contagion and direct effects for each value of pretreatment covariates \(x\). We then use the empirical distribution of \(X_i\) to obtain the estimated average effects. The non-parametric bootstrap with 1000 replicates is used to compute the confidence intervals.

Table 2 presents the estimated average spillover, contagion and direct effects under assump-

<table>
<thead>
<tr>
<th>Effect</th>
<th>Results for Denver</th>
<th>Results for Minneapolis</th>
<th>Results for pooled sample</th>
</tr>
</thead>
<tbody>
<tr>
<td>Spillover</td>
<td>(\theta)</td>
<td>0.069 (−0.004, 0.139)</td>
<td>0.073 (0.007, 0.141)</td>
</tr>
<tr>
<td>Contagion</td>
<td>(\tau(1))</td>
<td>0.052 (0.010, 0.093)</td>
<td>0.068 (0.027, 0.115)</td>
</tr>
<tr>
<td></td>
<td>(\tau(0))</td>
<td>0.054 (0.011, 0.098)</td>
<td>0.057 (0.019, 0.106)</td>
</tr>
<tr>
<td>Direct</td>
<td>(\eta(1))</td>
<td>0.016 (−0.042, 0.073)</td>
<td>0.016 (−0.042, 0.073)</td>
</tr>
<tr>
<td></td>
<td>(\eta(0))</td>
<td>0.017 (−0.036, 0.070)</td>
<td>0.005 (−0.057, 0.066)</td>
</tr>
</tbody>
</table>

†The estimated average contagion and direct effects are presented under the treatment (\(\tau(1)\)) and control (\(\tau(0)\) and \(\eta(0)\)) conditions.
tions 1–4. We find that the estimated average spillover effect is approximately 7 percentage points, and much of this effect appears to be attributable to contagion. There is little evidence for the existence of direct effects. This suggests that canvassing affects the turnout of the non-contacted voter in the household by first modifying the vote intention of the contacted voter. This pattern is consistent across the two cities.

We next estimate the average contagion and direct effects in various subpopulations of interest by using the data from Denver where a richer set of covariates is available. Although the findings that are presented below need to be interpreted with caution (because of multiple testing and other issues), they provide useful insights about how the interpersonal influence operates within a household. First, we examine how the magnitude of these effects depends on the turnout of voters in the previous election. Fig. 4(a) presents the results. We present only the estimated average contagion and direct effects under the treatment condition, i.e. $\tau(1)$ and $\eta(1)$, since the corresponding effects under the control condition, i.e. $\tau(0)$ and $\eta(0)$, are similar. In Fig. 4(a), for example, the grey dotted line labelled ‘N/Y’ or ‘N/N’ represents the estimated effects among the households, in which the contacted voter did not vote and the non-contacted voter respectively did or did not vote in the previous election.

We find that for the ‘N/N’ households the average spillover effect is most precisely estimated and appears to be mostly attributable to contagion. This is consistent with the idea that the voters in those households have a low vote propensity and therefore may be susceptible to canvassing. In contrast, for the ‘Y/N’ households, the average direct effect is estimated to be the greatest and appears to account for much of the spillover effect. Although the confidence intervals of these estimates are wide given a small sample size, this finding makes intuitive sense because the contacted voter tends to have a high vote propensity in the first place and hence may be less likely to be influenced by canvassing.

Figs 4(b) and 4(c) present the estimated average spillover, contagion and direct effects for subpopulations that are defined by partisanship and gender. There appears to be a negative contagion effect when the contacted voter is a Republican and the non-contacted voter is Democratic (‘R/D’ households indicated by grey lines). In contrast, the contagion effect is estimated to be positive and greatest when the treated voter is a Democrat and the untreated voter is a Republican. Finally, a statistically significant contagion effect is found among the households in which the contacted voter is female whereas the non-contacted voter is male (‘F/M’ households represented by grey lines).

4.2. Sensitivity analysis
We next conduct the parametric sensitivity analysis that was described in Section 3.5 by examining the robustness of the empirical findings that were presented above to the potential violation of assumptions 2 and 4. Specifically, we focus on two large estimated contagion effects: one for the households in which the contacted voter is Democratic and the non-contacted voter is Republican (‘D/R’ in Fig. 4(b)) and the other for the households in which the contacted voter is female and the non-contacted voter is male (‘F/M’ in Fig. 4(c)).

Fig. 5 presents the results of the sensitivity analysis. In Figs 5(a) and 5(b), we show the estimated contagion effects across different values of the sensitivity parameter $\rho$ for the possible violation of assumption 2 (see Fig. 2). As shown in equation (6), the sensitivity parameter $\rho$ represents the correlation between the error terms that is induced by unobserved confounders among complier households. The assumed absence of such correlation implies $\rho = 0$. We vary this sensitivity parameter from $-0.75$ to 0.75 and examine how the estimated average contagion effects change. We find that the results are quite robust in a sense that for both types (‘D/R’ and ‘F/M’) of households, even when the correlation is as large as 0.5, the 95% confidence
Fig. 4. Estimated spillover, contagion and direct effects by (a) turnout history, (b) party and (c) gender with 95% confidence intervals: for each subgroup estimate, the first letter represents the value of a variable for the contacted voter and the second letter indicates the value of a variable for the non-contacted voter; for turnout history, for example, a grey line labelled with ‘N/Y’ represents an estimate for households in which the contacted voter did not turn out (‘N’) in the previous election and the non-contacted voter did (‘Y’); similarly, for party, ‘R’ and ‘D’ represent a Republican and Democratic voter respectively; for gender, ‘F’ and ‘M’ indicate female and male voters respectively.
Fig. 5. Sensitivity analyses for the estimated average contagion effects in two subpopulations: (a), (b) scenario of latent confounding using the sensitivity parameter $\rho$ for the possible violation of assumption 2; (c), (d) scenario of measurement error based on the sensitivity parameter $\sigma$ with $\rho_e = 0$; (e), (f) scenario of unmeasured common causes based on the sensitivity parameter $\rho_e$ with $\sigma = 0.5$; (a), (c), (e) households in which a contacted voter is Democratic (female) and the non-contacted voter is Republican (male); (b), (d), (f) households in which a contacted voter is female and the non-contacted voter is male.
interval does not overlap with zero. When the correlation is negative (i.e. the effect of unobserved confounders on the vote intention of the contacted voter is opposite to that on the turnout of the non-contacted voter), the estimated average contagion effect will be even greater than the estimate that was obtained under assumption 2.

Figs 5(c) and 5(d) show the results of the sensitivity analysis for the potential violation of assumption 4 due to measurement error. As shown in equation (7), the sensitivity parameter $\sigma$ corresponds to the standard deviation of the unobserved measurement error for the vote intention of the contacted voter. If the actual turnout of this voter is a perfect proxy for vote intention, $\sigma$ will be equal to 0, corresponding to assumption 4. A larger value of $\sigma$ implies a greater degree of measurement error. We fix $\rho_e$ at 0 and vary this sensitivity parameter and examine how the estimated average contagion effects change. We find that the estimated average contagion effect becomes larger as the amount of measurement error increases. Note that this finding differs from the result of non-parametric sensitivity analysis, which leads to attenuation bias under the assumption of non-differential measurement error. The difference arises because the measurement error under this sensitivity analysis is no longer non-differential, i.e. $Y_{i1}$ depends on $Z_i$ conditionally on $Y_{i1}$ and $X_i$.

Figs 5(e) and 5(f) show the results of the sensitivity analysis for the unobserved common causes of the turnouts of two voters (see Fig. 3). This analysis is characterized by the sensitivity parameter $\rho_e$ that is defined in equation (8). Because the common causes are likely to affect the turnout of two voters in the same direction, we vary $\rho_e$ from 0 to 0.5 while fixing $\sigma$ at 0.5 and examine how the estimated average contagion effects change. We find that the existence of such unobserved common causes tends to attenuate the average contagion effect. However, the results are robust in a sense that the 95% confidence interval does not overlap with zero for the range of values that we consider for the sensitivity parameters.

5. Concluding remarks

In this paper, we show how to decompose the average spillover effect into the sum of the average contagion and direct effects in the presence of non-compliance. Contagion effects represent the causal path, in which one’s treatment affects another unit’s outcome by changing the outcome of the treated unit. Such a decomposition is useful when understanding the causal mechanism behind the spillover effects because it may yield different policy recommendations. In the GOTV experiment, the robust presence of contagion effects among some households implies that it is possible for one canvasser’s visit to change the turnout of two voters by contacting only one. This suggests that a GOTV mobilization strategy should be designed to increase the turnout of contacted voters. In contrast, if the direct effect is responsible for spillover, then the goal of a GOTV mobilization strategy should be something different because increasing the turnout of contacted voters may not affect the turnout of non-contacted voters.

Although we focus on a specific application, the methodology proposed is applicable in other contexts as well. One prominent application is a study of vaccine trials, in which a vaccination given to an individual can indirectly protect other individuals by reducing the probability of infection through contagion rather than, for example, increasing the awareness of contagious diseases. Indeed, there is a large methodological literature that examines how to identify contagion and direct effects in vaccine trials (e.g. Datta et al. (1999), Halloran and Hudgens (2012) and VanderWeele et al. (2012)). For example, in a setting that was similar to ours, VanderWeele et al. (2012) applied a mediation analysis to study the contagion and direct effects in households with two people. Their methodology can be viewed as a special case of ours without unobserved data and sensitivity analysis.
In social sciences, interference is often present because people routinely interact with each other. The contagion and direct effects represent a key to understanding causal mechanisms of interference. For example, in the well-known moving to the opportunity experiment where households in poor neighbourhoods are randomly encouraged to move to low poverty areas, it has been pointed out that the encouragement of one household is likely to affect the moving decision of another household (e.g. Sobel (2006)). Such an interference may arise because one household’s decision to move may influence the decision of its neighbours. Another possible mechanism is that people may discuss the pros and cons of moving with their friends and neighbours. Our method can be applied to separate these mechanisms.

In education, the mechanism of spillover effects is also of interest. For example, Basse et al. (2017) studied the within-household spillover effect of an intervention targeting student absenteeism in the School District of Philadelphia. In this experiment, parents in the treatment group received the attendance information only about one randomly chosen child in their household. The researchers showed that this intervention reduces the number of days absent from school for the other children in the same household. We can apply the proposed methodology to estimate the contagion and direct effects. The contagion effect characterizes how the behaviour of one child affects that of another child in the same household. In contrast, the direct effect may arise if, for example, parents who received the attendance information about one child decide to encourage their other children to attend the school.

In these applications, understanding causal mechanisms of spillover effects leads to a better policy recommendation. The presence of contagion effects implies that an intervention should target the outcome of an individual who receives the intervention because it will then affect the outcome of other individuals, e.g. developing an effective vaccine rather than increasing the awareness of contagious diseases in the vaccination example, convincing some households to move instead of encouraging a discussion of a move among a group of households in the moving to the opportunity experiment and encouraging parents to address the absentee problem of a particular child rather than informing them of the benefits of school attendance in general. We believe that our methodology can help researchers to disentangle these important subtleties.

Acknowledgements

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References


Supporting information
Additional ‘supporting information’ may be found in the on-line version of this article: ‘Supplementary materials’.