Modeling Interference Via Symmetric Treatment

Decomposition

BY ILYA SHPITSER

Department of Computer Science
Johns Hopkins University
Baltimore, MD 21218
ilyas@cs.jhu.edu

ERIC TCHETGEN TCHETGEN

Department of Biostatistics
Harvard T.H. Chan School of Public Health
Boston, MA 02115
etchetge@hsph.harvard.edu

AND RYAN ANDREWS

Department of Mental Health
Johns Hopkins Bloomberg School Of Public Health
Baltimore, MD 21218
randre23@jhu.edu

SUMMARY
Classical causal inference assumes a treatment meant for a given unit does not have an effect on other units. When this “no interference” assumption is violated, new types of spillover causal effects arise, and causal inference becomes much more difficult. In addition, interference introduces a unique complication where outcomes may transmit treatment influences to each other, which is a relationship that has some features of a causal one, but is symmetric. In settings where detailed temporal information on outcomes is not available, addressing this complication using statistical inference methods based on Directed Acyclic Graphs (DAGs) (Ogburn & VanderWeele, 2014) leads to conceptual difficulties. In this paper, we develop a new approach to decomposing the spillover effect into direct (also known as the contagion effect) and indirect (also known as the infectiousness effect) components that extends the DAG based treatment decomposition approach to mediation found in (Robins & Richardson, 2010) to causal chain graph models (Lauritzen & Richardson, 2002). We show that when these components of the spillover effect are identified in these models, they have an identifying functional, which we call the symmetric mediation formula, that generalizes the mediation formula in DAGs (Pearl, 2011). We further show that, unlike assumptions in classical mediation analysis, an assumption permitting identification in our setting leads to restrictions on the observed data law, making the assumption empirically falsifiable. Finally, we discuss statistical inference for the components of the spillover effect in the special case of two interacting outcomes, and discuss a maximum likelihood estimator, and a doubly robust estimator.

Some key words: Chain graphs; Graphical models; Interference; Mediation analysis;

1. INTRODUCTION

A standard assumption in causal inference is lack of unit interference, which asserts that giving treatment to a particular unit only affects the response of that unit. This is a sensible assumption
in many settings, for instance administering a fertilizer to a particular field to increase yield will likely not affect the yield in other fields. However, there are settings where this assumption is not reasonable. A classic example from epidemiology is herd immunity: vaccinating a subset of a population may grant immunity to the whole population, including those not vaccinated themselves.

Interference introduces a number of conceptual difficulties. First, unlike classical causal inference, treatments and outcomes for every experimental unit can no longer be viewed as independent realizations of some underlying distribution. Second, new types of causal effects called spillover effects arise, which quantify the degree to which treatments for one unit affect the outcome of another unit. Like total causal effects from classical causal inference, it may be desirable to decompose spillover effects into direct and indirect components. In the context of infectious disease epidemiology, the direct spillover effect is called the infectiousness effect, and the indirect spillover effect is called the contagion effect. For example, a vaccine administered to one person may prevent infection of another, giving a non-zero spillover effect. This effect may occur either because the vaccination stops the inoculated from being infected and thus passing the infection on (the contagion effect), or because the vaccination may not stop infection itself, but may render the pathogen weaker and thus less infectious (the infectiousness effect). One such decomposition was proposed by a direct mapping onto mediation analysis ideas (VanderWeele et al., 2012). This was motivated by the intuition that the contagion effect of vaccinating person one on infection of person two is mediated by the infection status of person one. What makes interference more complex than classical mediation analysis is that unlike a mediator and an outcome, the roles of outcomes for person one and two may be switched. This symmetry makes a direct application of ideas from directed acyclic graph models conceptually problematic.
We propose an alternative approach to interference that places dependent outcomes “on the same footing.” Our model can viewed as either a symmetric version of classical mediation analysis models (Robins & Greenland, 1992; Pearl, 2001), or as a generalization of the treatment decomposition model on DAGs advocated in (Robins & Richardson, 2010) to causal chain graph models (Lauritzen & Richardson, 2002).

We show that the spillover effect, and its direct and indirect portions can be formally represented as potential outcomes in our model. We further show how a “cross-world” assumption necessary for identification in mediation analysis settings generalizes to interference settings, and employ it to derive a functional of the observed data corresponding to direct and indirect spillover effects. We call this functional the symmetric mediation formula, due to the fact that it can be viewed as an appropriate generalization of the mediation formula (Pearl, 2011) to chain graphs.

In addition, we demonstrate that the identifying assumption in our model imposes restrictions on the observed data law, which leads to falsifiability (but not testability) of our model, a desirable feature not present in the classical mediation setting. Finally, we consider estimation of the symmetric mediation formula as an inference problem in statistical chain graph models. We derive a maximum likelihood estimator that is straightforward to implement. In settings when outcomes are labeled, e.g parent and child or husband and wife, we describe a doubly robust estimator.

2. Notation and Preliminaries

A successful framework for causal inference from observational data is based on potential outcomes (Neyman, 1923; Rubin, 1974), that is responses to hypothetical treatment assignments. Here we describe potential outcome notation suitable for interference settings, introduce graphi-
2.1. Potential Outcomes in Partial Interference Settings

Assume we are analyzing data from a randomized control trial with \( M \) blocks with \( N \) units each. Equal sized blocks is not a necessary assumption for the kinds of interference problems we consider, but we make it to simplify our notation.

We distinguish two settings: labeled and unlabeled units. In the labeled unit setting we are interested in effects of treatments applied to a certain privileged subset of units on outcomes for another privileged subset of units. Obtaining spillover effects of child vaccinations on mothers is an example of such a setting. In the unlabeled unit setting we are interested in the effect summary averaged over all units. Estimating effects of a marketing intervention applied to users of a social network on their friends is an example of such a setting. Here we are not interested in privileging any subsets of users, but wish to obtain a summary of such effects across all users. The choice of setting does not influence our notation, but changes the target of inference, and may have implications for statistical inference, as we later show.

For each unit \( i \) in block \( j \), denote the response variable as \( Y^i_j \), the binary treatment variable as \( A^i_j \), with the control group treatment value denoted by 0, active treatment value denoted by 1, and treatment values generally by lowercase letters: \( a^i_j \). That is, \( a^i_j \) is an element of the state space of \( A^i_j \), or \( a^i_j \in X_{A^i_j} \).

We define the following notation. For each block \( j \), let \( \bar{Y}_j \equiv (Y^1_j, \ldots, Y^N_j) \), \( \bar{A}_j \equiv (A^1_j, \ldots, A^N_j) \). That is, \( \bar{Y}_j \) and \( \bar{A}_j \) are vectors of responses and treatments in block \( j \). The state space of \( \bar{A}_j \) is defined in the usual way as the Cartesian product of the state spaces of its elements: \( X_{\bar{A}_j} \equiv \otimes_{i=1}^{N} X_{A^i_j} \). The state space of \( \bar{Y}_j \) is defined similarly. For the vector of \( i \)th units across all blocks, let \( \bar{Y}^i \equiv (Y^i_1, \ldots, Y^i_M), \bar{A}^i \equiv (A^i_1, \ldots, A^i_M) \). Finally, with a slight abuse of
notation, define \( \vec{A} \equiv (A_1, \ldots, A_M) = (\vec{A}^1, \ldots, \vec{A}^N) \), and \( \vec{Y} \equiv (Y_1, \ldots, Y_M) = (\vec{Y}^1, \ldots, \vec{Y}^N) \),
with \( X_{\vec{A}} = \otimes_{j=1}^{M} X_{\vec{A}^j} \), and \( X_{\vec{Y}} = \otimes_{j=1}^{M} X_{\vec{Y}^j} \).

For any \( \vec{a} \in X_{\vec{A}} \), define \( Y_j^i(\vec{a}) \) to be the potential response of unit \( i \) in block \( j \) to a hypothetical treatment assignment of \( \vec{a} \) to \( \vec{A} \). Note that in general the distribution of the random variable \( Y_j^i(\vec{a}) \) differs from that of \( Y_j^i \) conditional on \( \vec{a} \), due to issues of confounding of the treatment assignment mechanism. That is, it may be the case that the design of the study assigns particular treatments within \( \vec{A} \) in a way that depends on unobserved confounders which may also influence \( Y_j^i \). We define \( \vec{Y}_j(\vec{a}) \) and \( \vec{Y}(\vec{a}) \) in the natural way as vectors of responses, given a hypothetical treatment assignment to \( \vec{A} \), either for units in block \( j \) or for all units, respectively.

Let \( \vec{a}_{(j)} \) be a vector of values of \( \vec{A} \), where values assigned to units in block \( j \) are free variables, and other values are bound variables. Furthermore, for any \( \vec{a}'_j \in X_{\vec{A}^j} \), let \( \vec{a}_{(j)}[\vec{a}'_j] \) be a vector of values which agrees on all bound values with \( \vec{a}_{(j)} \), but which assigns \( \vec{a}'_j \) to all units in block \( j \) (e.g. which binds free variables in \( \vec{a}_{(j)} \) to \( \vec{a}'_j \)). Throughout this manuscript we will assume interblock non-interference, also known as partial interference in (Sobel, 2006; Tchetgen & VanderWeele, 2012), where for any block \( j \), treatments assigned to units in a block other than \( j \) do not affect the responses of any unit in block \( j \). Formally, this is stated as

\[
(\forall j, \vec{a}_{(j)}, \vec{a}'_j, \vec{a}^l_j, \vec{Y}_j(\vec{a}_{(j)}[\vec{a}'_j])) = \vec{Y}_j(\vec{a}'_j[\vec{a}^l_j]).
\]

Due to this assumption, we will typically write potential responses within a particular block as only depending on treatments assigned within that block. That is, for any \( \vec{a}_{(j)} \), \( \vec{Y}_j(\vec{a}_{(j)}[\vec{a}'_j]) \equiv \vec{Y}_j(\vec{a}'_j) \). We allow treatments within a single block to affect units within that block in an arbitrary way.
2-2. Classical Causal Inference

Classical causal inference assumes blocks of size $N = 1$, so $\vec{Y}_j = (Y_j)$, $\vec{A}_j = (A_j)$, $\vec{a}_j = (a_j)$ are vectors of size 1. Interblock non-interference allows us to view realizations of $Y_j$ and $A_j$ for all $j$ as independent realizations of single random variables $Y$ and $A$, respectively.

In this setting we can quantify the effect of treatment by a contrast, for instance a difference of mean outcomes of units had they been assigned to the active group, and of units had they been assigned to the control group:

$$\mathbb{E}\{Y(1)\} - \mathbb{E}\{Y(0)\}.$$

This is called the *average causal effect* (ACE).

The goal of causal inference is estimation of counterfactual contrasts, like the ACE, from observed data in our study. The difficulty with the ACE is that it is a function of responses that occur contrary to fact. The fundamental problem of causal inference is that we only observe the response actually assigned. A link between counterfactual contrasts such as the ACE, and observed data is typically made by means of the *consistency assumption*, and some version of the *ignorability assumption*.

Consistency provides a link between observed and counterfactual outcomes by asserting that the random variable representing the outcome $Y$ is equal to the random variable representing the outcome $Y(A)$ where $A$ is set to whatever value it actually obtained. The simplest version of ignorability states that the treatment assignment probability $A$ is independent of the potential outcome under either treatment. This can be ensured to hold by randomizing the treatment assignment. Under these assumptions, we have

$$\mathbb{E}\{Y(1)\} - \mathbb{E}\{Y(0)\} = \mathbb{E}\{Y(1) \mid A = 1\} - \mathbb{E}\{Y(0) \mid A = 0\} = \mathbb{E}(Y \mid A = 1) - \mathbb{E}(Y \mid A = 0),$$

where the first equality is by ignorability, and the second by consistency.
Without active randomization, ignorability is an unrealistic assumption, and more complex assumptions are used to obtain identification of counterfactual contrasts in terms of observed data. A general theory of identification, in terms of a graphical representation of potential outcomes, exists (Tian & Pearl, 2002; Shpitser & Pearl, 2006b,a).

Consistency and treatment non-interference (or interblock non-interference for blocks of size 1) are often grouped together as the Stable Unit Treatment Value Assumption (SUTVA) (Rubin, 1974). In this paper, which deals with interference within blocks of size > 1, we relax the non-interference part of SUTVA, but keep consistency.

2.3. Classical Mediation Analysis

Assuming blocks of size 1, and given the overall effect, as quantified by the ACE, we may wish to decompose it into components corresponding to a direct effect and an indirect effect (mediated by a third variable on a causal pathway from treatment to outcome). Defining such a decomposition and recovering it from observed data is the goal of mediation analysis. Originally such a decomposition was considered in the context of linear regression models (Baron & Kenny, 1986), where it was established that the total effect can be decomposed into a sum of direct and indirect effects, and this decomposition has a particularly simple representation in terms of regression coefficients.

However, this representation breaks down in the presence of non-linearities and interactions. A general representation of direct and indirect effects in terms of nested potential outcomes was proposed in (Robins & Greenland, 1992). The key idea is to consider a contrast between the response to a treatment value, for example $Y(1)$, and a response to a hypothetical experiment where treatment is set to an active value, but the mediator is set to whatever value it would have attained under a baseline intervention, or $Y(1, M(0))$. The intuition is that setting the treatment to baseline for the purposes of the mediator “turns off” the causal pathway mediated by the
treatment, and leaves active the direct causal pathway from treatment to the outcome. Given this intuition, we can define an indirect effect contrast as \( E[Y(1)] - E[Y(1, M(0))] \) (subtracting off the active direct path from the overall effect), and a direct effect contrast as \( E[Y(1, M(0))] - E[Y(0)] \) (subtracting off the baseline from the active direct path). The ACE decomposes into a sum of these contrasts:

\[
E\{Y(1)\} - E\{Y(0)\} = [E\{Y(1, M(0))\} - E\{Y(0)\}] + [E\{Y(1)\} - E\{Y(1, M(0))\}].
\]

As we saw above, consistency and ignorability assumptions suffice for identifying the counterfactual responses \( Y(1) \) and \( Y(0) \). However, identifying the counterfactual response \( Y(1, M(0)) \) is a more difficult task since it is a response to contradictory treatment assignments.

One simple set of assumptions that allow identification of this response is that the set of counterfactuals \( \{Y(a, m), M(a'), A\} \) are mutually independent for all \( a, a' \in X_A, m \in X_M \). Given this set of assumptions, we obtain the following derivation for the indirect effect (and a similar one for the direct effect):

\[
E\{Y(1, M(0))\} - E\{Y(0)\} = \sum_m [E\{Y(1, m)\} - E\{Y(0, m)\}] p(M(0) = m)
= \sum_m \left[ E\{Y(1, m) | A = 1, M(1) = m\} - E\{Y(0, m) | A = 0, M(0) = m\} \right] p(M(0) = m)
= \sum_m \{E(Y | 1, m) - E(Y | 0, m)\} p(m | 0).
\]

This is known as the mediation formula (Pearl, 2011).

2.4. Graph Theory and Graphical Models

Before reviewing graphical representations of mediation analysis, and our causal model for interference problems, we briefly review necessary graph theory. We consider mixed graphs containing directed (\( \to \)), and undirected (\( - \)) edges, such that at most one edge connects two
vertices. A sequence of non-repeating vertices \((V_1, \ldots, V_k)\) in such a graph is called a \textit{path} if for every \(i = 1, \ldots, k - 1\), \(V_i\) and \(V_{i+1}\) are connected by an edge.

A path is called partially directed if all directed edges in the path point towards the vertex with a larger index. A partially directed path is called directed if it contains no undirected edges. A mixed graph is said to contain a partially directed cycle if it contains a partially directed path from \(V_1\) to \(V_k\), and a directed edge from \(V_k\) to \(V_1\). A mixed graph of the above form without a partially directed cycle is called a \textit{chain graph} (CG). A chain graph without undirected edges is called a \textit{directed acyclic graph} (DAG).

If an edge \(A \rightarrow B\) exists in a CG \(\mathcal{G}\), we say \(A\) is a parent of \(B\), and \(B\) is a child of \(A\). If an edge \(A - B\) exists in a CG \(\mathcal{G}\), we say \(A\) is a neighbor of \(B\) (and vice versa). If there is a directed
path from $A$ to $B$, $A$ is an ancestor of $B$ and $B$ is a descendant of $A$. If there is a partially directed path from $A$ to $B$, $A$ is in the anterior of $B$. By convention, for any $A$ in any CG, $A$ is its own ancestor and descendant, and is in its own anterior. A non-descendant of $A$ is any node that is not a descendant of $A$. We denote the sets of parents, children, ancestors, descendants, non-descendants, and neighbors of $A$ in $\mathcal{G}$ by $\text{pa}_G(A)$, $\text{ch}_G(A)$, $\text{an}_G(A)$, $\text{de}_G(A)$, $\text{nd}_G(A)$, $\text{nb}_G(A)$. The set of vertices in the anterior of $A$ in $\mathcal{G}$ is $\text{ant}_G(A)$. We define these sets on sets of vertices disjunctively, e.g. for a set of vertices $\vec{A}$, $\text{pa}_G(\vec{A}) \equiv \bigcup_{A \in \vec{A}} \text{pa}_G(A)$. For any $\vec{A}$, define the family set as $\text{fa}_G(\vec{A}) = \text{pa}_G(\vec{A}) \cup \vec{A}$.

A connected component in an edge subgraph of a CG $\mathcal{G}$ that drops all directed edges and retains undirected edges is called a block. The set of blocks in a CG $\mathcal{G}$ will be denoted by $\mathcal{B}(\mathcal{G})$. This set clearly partitions the set of vertices in $\mathcal{G}$. Given $\vec{B} \in \mathcal{B}(\mathcal{G})$, let $\mathcal{G}^a_{\text{fa}_G(\vec{B})}$ be an undirected graph that contains only vertices in $\text{fa}_G(\vec{B})$, and an edge between any two vertices that are either adjacent in $\mathcal{G}$, or lie in $\text{pa}_G(\vec{B}) \setminus \vec{B}$. In an undirected graph $\mathcal{G}$, a clique is a fully connected set of vertices. A maximal clique is a clique such that no superset of it is also a clique. Let the set of maximal cliques in $\mathcal{G}$ be $\mathcal{C}(\mathcal{G})$. Note that unlike $\mathcal{B}(\mathcal{G})$, $\mathcal{C}(\mathcal{G})$ is not necessarily a partition of vertices in $\mathcal{G}$.

2.5. Graphical Causal Models and Graph Representations of Mediation

The simple mediation setting we are discussing is often represented by means of a causal diagram shown in Fig. 1 (a). Here vertices represent variables of interest, the treatment $A$, mediator $M$, and outcome $Y$, and directed edges represent direct causal relationships. Thus, $A$ directly causes $M$ and $Y$, and $M$ directly causes $Y$. Formally, a causal diagram corresponds to a set of independence assumptions on potential outcome random variables. For mediation problems, it is common to assume, explicitly or implicitly, the non-parametric structural equation model with independent errors (NPSEM-IE) of Pearl (Pearl, 2009).
This model associates a set of variables and a set of vertices $\vec{V} = \{V_1, \ldots, V_k\}$ in a DAG, and for each variable $V_i \in \vec{V}$ assumes a noise variable $\epsilon_{V_i}$, and an arbitrary, invariant causal mechanism $f_{V_i} : \mathcal{X}_{\text{pa}_G(V_i) \cup \{\epsilon_{V_i}\}} \rightarrow \mathcal{X}_{V_i}$. It is assumed $f_{V_i}$ determines the value of $V_i$ regardless of how the values of $\text{pa}_G(V_i)$ were assigned. Moreover, it is assumed the noise variables are mutually independent: $p(\epsilon_{V_1}, \ldots, \epsilon_{V_k}) = \prod_{i=1}^{k} p(\epsilon_i)$. The arbitrary nature of $f_{V_i}$ justifies the word “non-parametric,” and this property justifies the phrase “independent errors” in the name of the model. Interventions, including conflicting interventions that arise in mediation analysis, are represented by replacing certain mechanisms by constant values.

The three node example in Fig. 1 (a) is represented by three functions $f_A : \mathcal{X}_{\epsilon_A} \rightarrow \mathcal{X}_A$, $f_M : \mathcal{X}_{\{A, \epsilon_M\}} \rightarrow \mathcal{X}_M$, and $f_Y : \mathcal{X}_{\{A, M, \epsilon_Y\}} \rightarrow \mathcal{X}_Y$, and the counterfactual $Y(1, M(0))$ is the random variable $f_Y(A = 1, f_M(A = 0, \epsilon_M), \epsilon_Y)$ induced by $\epsilon_Y$ and $\epsilon_M$. The mutual independence of $Y(a, m) = f_Y(A = a, M = m, \epsilon_Y)$, $M(a') = f_M(A = a', \epsilon_M)$ and $A = f_A(\epsilon_A)$ is induced by mutual independence of $\epsilon_Y$, $\epsilon_M$, and $\epsilon_A$.

An alternative definition of the NPSEM-IE model for a DAG $G$ with a vertex set $\vec{V}$, given in (Richardson & Robins, 2013), uses one step ahead counterfactuals of the form $V(\vec{a}_V)$, for any $\vec{a}_V \in \mathcal{X}_{\text{pa}_G(V)}$, to define all other variable, factual or counterfactual, using recursive substitution. Specifically, for any $\vec{A} \subseteq \vec{V}$, and any $\vec{a} \in \mathcal{X}_{\vec{A}}$, we have for every $V \in \vec{V}$

$$V(\vec{a}) \equiv V(\vec{a}_{\text{pa}_G(V)}, \{\text{pa}_G(V) \ \backslash \ \vec{A}\}(\vec{a}))$$  \hspace{1cm} (1)

Since all variables in the NPSEM-IE are defined in terms of one step ahead counterfactuals, all restrictions made by the NPSEM-IE are entailed by a set of assumptions on these counterfactuals, as a kind of causal version of the local Markov property. These assumption state that

“variables in the set $\left\{\left\{V(\vec{a}_V) \mid \vec{a}_V \in \mathcal{X}_{\text{pa}_G(V)}\right\} \mid V \in \vec{V}\right\}$ are mutually independent.”  \hspace{1cm} (2)
It has been shown in (Richardson & Robins, 2013) that (1) and (2) entail that the observed data law obeys the standard Markov factorization with respect to $G$:

$$p(\vec{V}) = \prod_{V \in \vec{V}} p(V | pa_G(V)) = \prod_{V \in \vec{V}} p(V | pa_G(V))$$  \hspace{1cm} (3)

One difficulty with these NPSEM-IE assumptions is they include independence of variables like $Y(1,m)$ and $M(0)$ above, which cannot be tested, since these variables involve conflicting value assignments to $A$. However, there exists an alternative formulation of mediation problems which avoids this issue (Robins & Richardson, 2010). This formulation views the mediation counterfactual like $Y(1, M(0))$ as representing a decomposition of the effect of treatment $A$ into two components, one associated only with a direct effect on the outcome, and one only with the effect through the mediator. For example, assume smoking ($A$) affects a health outcome $Y$ either directly via smoke or indirectly via nicotine content, mediated by cardiovascular disease $M$. Note that while both components of the treatment are present in smokers, we can, in principle, imagine intervening on these components separately, by means of smoke less cigarettes or nicotine patches (see discussion in Section 5 of (Robins & Richardson, 2010)).

We can represent these components explicitly in a larger causal diagram shown in Fig. 1 (b), where nicotine and smoke components of smoking are represented as “copies” $A', A''$ of $A$ that in ordinary circumstances (normal smoking) have the same value as $A$, but whose values can in principle be set separately. Moreover, $A'$ only affects $M$ and $A''$ only affects $Y$. The counterfactual $Y(A = 1, M(A = 0))$ in the causal model corresponding to Fig. 1 (a) would be represented by the counterfactual $Y(A' = 0, A'' = 1)$ in the causal model represented by Fig. 1 (b), which is a response to interventions that are no longer in conflict, by construction.

The larger causal model can be viewed as a standard NPSEM-IE (in fact a weaker FFRCISTG model of Robins (Robins, 1986) suffices for our purposes), with a deterministic relationship between $A'$, $A''$ and $A$. Though it might appear that Fig. 1 (b) is a simple recoding of Fig. 1 (a),
this is not the case. In fact, while (2) entails an untestable independence assumption \( Y(1, m) \perp \perp M(0) \) in Fig. 1 (a), (2) entails \( M(A' = 0) \perp \perp Y(A'' = 1, m) \) in Fig. 1 (b). This assumption can, in principle, be tested by intervening on \( A' \) and \( A'' \) separately.

To contrast the model given by Fig. 1 (b) with our model, described later, we restate the constraints corresponding to this model directly on the joint distribution over potential outcomes \( p(Y(a', a''), M(a', a'')) \). They state, in particular, that for any values \( a', a'' \in \mathcal{X}_A \),

\[
p\{Y(a', a'') \mid M(a', a'')\} \text{ is only a function of } Y, M, A''\tag{4}
\]
\[
p\{M(a', a'')\} \text{ is only a function of } M, A'\tag{5}
\]

Equation (3) for the observed data law of the NPSEM-IE of Fig. 1 (b) is

\[
p(Y, M, A', A'', A) = p(Y \mid M, A'')p(M \mid A')p(A' \mid A)p(A'' \mid A)p(A),\tag{6}
\]

where factors \( p(A' \mid A) \) and \( p(A'' \mid A) \) are deterministic.

3. Decomposing Spillover Effects Using Classical Mediation Analysis

With a block size \( > 1 \), realizations of responses \( \vec{Y}_j \) in block \( j \) can no longer be thought of as realizations of a single random variable. We instead assume that response and treatment vectors \( A_1^i, \ldots, A_M^i \) and \( Y_1^i, \ldots, Y_M^i \) are realization of random variables \( A^i \) and \( Y^i \), respectively. For \( i \neq k \), \( A^i, Y^i \) and \( A^k, Y^k \) are distinct and potentially dependent random variables.

To simplify the notation, for the remainder of the paper, we will define \( \vec{A} \equiv (A_1, \ldots, A_N) \), and \( \vec{Y} \equiv (Y_1, \ldots, Y_N) \). For any \( \vec{a} \in \mathcal{X}_{\vec{A}} \), let \( \vec{a}(i) \) be a vector of values of \( \vec{A} \) where the treatment value assigned to the \( i \)th unit is a free variable, and \( \vec{a}(i)[a^i] \) be a vector of values which agrees on all bound values with \( \vec{a}(i) \), but which assigns \( a^i \) to the treatment for unit \( i \).

Since we allow treatment on one unit to affect another unit within a block, a treatment administered to unit \( i \) may affect the response of unit \( i \), or the responses of other units \( j \) within a block.
Following (Halloran & Struchiner, 1995; Tchetgen & VanderWeele, 2012), we define the *direct intrablock or main effect* (on the mean difference scale) of treatment $A^i$ on $Y^i$ as

$$ME(\vec{a}^{(i)}) \equiv \mathbb{E}\{Y^i(\vec{a}^{(i)}[1])\} - \mathbb{E}\{Y^i(\vec{a}^{(i)}[0])\}.$$ 

Similarly, we define the *indirect intrablock or spillover effect* (on the mean difference scale) of treatments other than $A^i$ on $Y^i$ as

$$SE(\vec{a}^{(i)}, \vec{a}'^{(i)}) \equiv \mathbb{E}\{Y^i(\vec{a}^{(i)}[0])\} - \mathbb{E}\{Y^i(\vec{a}'^{(i)}[0])\}.$$ 

We denote these effects as intrablock effects to distinguish them from direct and indirect effects that arise in mediation analysis.

Consistency and ignorability generalize to the partial interference setting in the natural way. In particular, for any $Y^i$, consistency states that for any $Y^i$, $Y^i(\vec{A}) = Y^i$, and ignorability states that $Y^i(\vec{a}) \perp \perp \vec{A}$.

### 3.1 Infectiousness and Contagion Components of the Spillover Effect

Assume the simplest case that departs from the classical causal inference setting, namely one with blocks of size 2. As an example, consider a vaccine trial discussed in (VanderWeele et al., 2012), where one-year-olds at a day-care center are randomized to receive a pneumococcal conjugate vaccine. Pneumococcus is prevalent in children attending day care, but mothers are also likely to be infected, and moreover are much more likely to be infected from their child, rather than other transmission sources. We are interested in the effect of vaccinating the children on the infection rates of their mothers.

Here, let $Y^1$ and $A^1$ be the response and treatment of the child, and $Y^2$ and $A^2$ be the response and treatment of the mother. We are interested in the spillover effect of vaccinating the child on
the mother’s infection status, or

\[ SE \equiv \mathbb{E}\{Y^2(A^1 = 1, A^2 = 0)\} - \mathbb{E}\{Y^2(A^1 = 0, A^2 = 0)\}. \]

Since \( A^2 \) is always 0 (treatment is not given), we suppress it from the notation, to yield the contrast

\[ \mathbb{E}\{Y^2(A^1 = 1)\} - \mathbb{E}\{Y^2(A^1 = 0)\}. \]

Using the ignorability and consistency assumptions defined above, we can identify this contrast as a function of observed data as:

\[ \mathbb{E}(Y^2 \mid A^1 = 1) - \mathbb{E}(Y^2 \mid A^1 = 0). \]

This estimated contrast is then a measure of the protective effect of the child’s vaccine on the mother. This effect might operate in one of two ways. It might prevent the child from getting infected, and then passing on the infection to the mother, or it might weaken the child’s infection and make it less transmittable. The former component of the spillover effect was called the contagion effect, and the latter the infectiousness effect (VanderWeele et al., 2012). To generalize from the infectious disease setting, in this paper, we call these effects the indirect spillover effect, and direct spillover effect, respectively.

One counterfactual approach for modeling contagion and infectiousness effects is to mirror the mediation approach, and treat the outcome of the mother, \( Y^2 \), as the “outcome of interest,” and the outcome of the child, \( Y^1 \), as the “mediator.” Specifically, it was proposed in (VanderWeele et al., 2012) to define the indirect spillover (contagion) effect as the contrast

\[ ISE \equiv \mathbb{E}\{Y^2(A^1 = 0, Y^1(A^1 = 1))\} - \mathbb{E}\{Y^2(A^1 = 0, Y^1(A^1 = 0))\}, \]

and the direct spillover (infectiousness) effect as

\[ DSE \equiv \mathbb{E}\{Y^2(A^1 = 1, Y^1(A^1 = 1))\} - \mathbb{E}\{Y^2(A^1 = 0, Y^1(A^1 = 1))\}. \]
The justification is quite sensible in a setting where only unit 1 is vaccinated, e.g. \( A^1 \) is set to 1, while \( A^2 \) is kept at 0. For instance the indirect spillover effect contrast will only be non-zero if \( Y^1(A^1 = 1) \) differs from \( Y^1(A^1 = 0) \), and this change effect unit 2 that was not given a vaccine. This is precisely a formalization of the intuitive definition of the indirect spillover effect.

Reformulating these contrasts using the split treatment formulation of mediation discussed in section 2.5, yields the spillover effect contrast of

\[
SE \equiv \mathbb{E}\{Y^2(A^1 = 1)\} - \mathbb{E}\{Y^2(A^1 = 0)\} = \mathbb{E}\{Y^2(A' = 1, A'' = 1)\} - \mathbb{E}\{Y^2(A' = 0, A'' = 0)\}
\]

and contrasts

\[
ISE \equiv \mathbb{E}\{Y^2(A' = 0, A'' = 1)\} - \mathbb{E}\{Y^2(A' = 0, A'' = 0)\}
\]

as the indirect spillover effect, and

\[
DSE \equiv \mathbb{E}\{Y^2(A' = 1, A'' = 1)\} - \mathbb{E}\{Y(A' = 0, A'' = 1)\}
\]

as the direct spillover effect. Given these definitions, independence assumptions employed in classical mediation analysis yield identification of these contrasts from observed data. For example, the indirect spillover effect is identified via the mediation formula (Pearl, 2011)

\[
ISE = \sum_{y^1} \{ \mathbb{E}(Y^2 \mid A'' = 1, y^1) - \mathbb{E}(Y^2 \mid A'' = 0, y^1) \} \cdot p(y^1 \mid A' = 0).
\]

The difficulty with this approach is the fact that responses often have a symmetric influence on each other, unlike a typical causal relationship where a mediator affects the outcome, but not vice versa. Specifically, had we vaccinated mothers and studied the effect on children, we would expect a similar analysis, only now with a counterfactual of the form \( Y^1(A^2 = 0, Y^2(A^2 = 1)) \). This implies that the underlying causal model for the interaction allows both \( Y^2(A^1 = 0, Y^1(A^1 = 1)) \neq Y^2(A^1 = 0) \) and \( Y^1(A^2 = 0, Y^2(A^2 = 1)) \neq Y^1(A^2 = 0) \), and in addition uses the following independences for identification: \( Y^2(A^1 = 0, y^1) \perp \perp Y^1(A^1 = 1) \), \( Y^1(A^2 = 0, y^2) \perp \perp Y^2(A^2 = 1) \) for all \( y^1, y^2 \).
It is not clear how one might specify a structural equation model where all these properties always hold. For instance, a natural approach of specifying an NPSEM-IE with functions of the form $f_{Y_1} : \mathcal{X}_{\{A^2, Y_1, \epsilon_{Y_1}\}} \rightarrow \mathcal{X}_{Y_1}$, and $f_{Y_2} : \mathcal{X}_{\{A^1, Y_1, \epsilon_{Y_2}\}} \rightarrow \mathcal{X}_{Y_2}$ will result in above constraints not being satisfied for most types of $f_{Y_1}, f_{Y_2}$. This is well known as endogeneity or simultaneity bias in econometrics models of supply and demand (see discussion of Fig. 1.5, and Chapter 5 in (Pearl, 2009), or discussion of cyclic econometrics models in (Wooldridge, 2015)). These models are special cases of more general cyclic models considered in (Spirtes, 1995).

This difficulty is resolvable if we are able to collect very detailed information on the temporal order in which treatments and outcomes influence each other, in which case the problem can be well-modeled by a DAG “unrolled” in time. This is an approach sometimes taken in the analysis of longitudinal data with interference (Ogburn & VanderWeele, 2014). However, in practice such detailed temporal information is rarely available, and instead information on outcomes is collected in such a way that detailed information on transmission dynamics is lost. In this paper, we present an alternative causal model for reasoning about outcomes without detailed temporal information, one where identifying constraints that treat outcomes symmetrically are satisfied by construction.

4. Chain Graph Causal Models and Symmetric Spillover Effect Decomposition

We wish to model the decomposition of an indirect effect within a block into an infectiousness and contagious components in such a way that no ordering on outcomes $\vec{Y}_j$ within any block $j$ is imposed. This means we cannot use standard causal models representable by DAGs, such as the NPSEM-IE. In particular, we wish to define the decomposition of any spillover effect within a block in a coherent way, such that any outcome may act either as mediation or outcome.
Since we must avoid orderings on outcomes, we cannot use nested counterfactuals of the form \( Y(a, M(a')) \) that arise in mediation analysis.

We propose the following generalization of mediation analysis in this paper. First, we generalize the FFRCISTG and NPSEM-IE model definitions using one step ahead counterfactuals in (Richardson & Robins, 2013) to causal models that imply saturated observed data distributions using one step ahead \textit{blocks}. This proposal can be viewed as a counterfactual version of the structural equation model in (Lauritzen & Richardson, 2002). Next, we impose additional restrictions on these models that would allow us to model effect decompositions, and interference settings where not all spillover effects, or their components, are present. We then use these restrictions to generalize the treatment decomposition approach to mediation in (Robins & Richardson, 2010) to our models. Finally, we use our approach to decompose indirect intrablock effects into infectiousness and contagion effects, and consider identification and estimation of these effects.

4.1. \textit{The Saturated Chain Graph Causal Model}

Consider a CG \( G \) with vertex set \( \tilde{V} \), where any two \( V_1, V_2 \in \tilde{V} \) are adjacent (in other words \( G \) is a complete graph). We consider settings where only interventions on entire blocks are allowed. That is we only consider a set of interventions on \( \tilde{A} \) to be valid if for any \( \tilde{B} \in B(G) \), if \( A \in \tilde{A} \cap \tilde{B} \), then \( \tilde{B} \subseteq \tilde{A} \). For any block \( \tilde{B} \in B(G) \), we assume the existence of a \textit{one step ahead block} \( \tilde{B}(\tilde{A}_{\tilde{B}}) \equiv \{ B(\tilde{A}_{\tilde{B}}) \mid B \in \tilde{B} \} \), for any \( \tilde{A}_{\tilde{B}} \in X_{\text{pa}_G(\tilde{B})} \), which is a set of potential outcomes defined jointly. We now show that in a complete CG, all one step ahead blocks are defined using valid intervention sets.

**Lemma 1.** If a CG \( G \) with vertex set \( \tilde{V} \) is complete, then for any \( V, W \in \tilde{V}, W \in \text{pa}_G(V) \) or \( W \in \text{nb}_G(V) \) or \( W \in \text{ch}_G(V) \).
 LEPRO T. G. TCHETGEN TCHETGEN AND RYAN ANDREWS

LEMMA 2. If a CG $\mathcal{G}$ with vertex set $\tilde{V}$ is complete, then the set of treatments $\tilde{A}_{\tilde{B}}$ of any one step ahead block $\mathcal{B}(\tilde{A}_{\tilde{B}})$ is valid.

Proof. Fix $A \in \text{pa}_G(\tilde{B})$, and let $\tilde{B}_A \in \mathcal{B}(\mathcal{G})$ such that $A \in \tilde{B}_A$. Then for any $W \in \tilde{B}_A$ such that $W \neq A$, if $W \notin \text{pa}_G(\tilde{B})$, then by Lemma 1, $\mathcal{G}$ is not a CG, which is a contradiction. □

All other responses to valid interventions are defined analogously to (1). To do so, we show that this definition only involves valid intervention sets.

LEMMA 3. If a CG $\mathcal{G}$ with vertex set $\tilde{V}$ is complete, and $\tilde{A}$ is a valid intervention set, then for any $\tilde{B} \in \mathcal{B}(\mathcal{G})$, a subset of $\mathcal{B}(\mathcal{G})$ partitions $\text{pa}_G(\tilde{B}) \setminus \tilde{A}$.

Proof. Since $\tilde{A}$ is valid, a subset of $\mathcal{B}(\mathcal{G})$ partitions $\tilde{A}$. If a subset of $\mathcal{B}(\mathcal{G})$ does not partition $\text{pa}_G(\tilde{B})$, then $\mathcal{G}$ cannot be a CG. □

For any $\tilde{B} \in \mathcal{B}(\mathcal{G})$, and any intervention on a valid set of treatments $\tilde{A}$,

$$\tilde{B}(\tilde{A}) \equiv \tilde{B}(\text{pa}_G(V), \{\tilde{B}_p(\tilde{A}) \mid \tilde{B}_p \in \mathcal{B}(\mathcal{G}), \tilde{B}_p \subseteq \text{pa}_G(V) \setminus \tilde{A}\}) \quad (10)$$

We define counterfactual independence restrictions on our model by analogy with (2):

“blocks in the set $\{\tilde{B}(\tilde{A}_{\tilde{B}}) \mid \tilde{A}_{\tilde{B}} \in \text{pa}_G(\tilde{B})\}$ are mutually independent.” \quad (11)

For example, in the mother/child vaccination example, represented by Fig. 1 (c), this assumption states that the counterfactual corresponding to the mother/child outcome set, given a hypothetical treatment $a$, $\{Y^1, Y^2\}(a) \equiv \{Y^1(a), Y^2(a)\}$, is independent of the treatment assignment variable $A$. This is just a simple version of the ignorability assumption generalized to blocks, as discussed in section 3.

Weaker restrictions defining the generalization of the FFRCISTG model to blocks can be derived similarly, though we leave them aside in the interests of space. For the remainder of the paper, we assume every one step ahead block distribution is positive.
Having defined the model using a complete CG, we now wish to allow restrictions into our model corresponding to missing edges in a CG. In a DAG missing edges are always directed and correspond to exclusion restrictions, or absences of a direct effect. In a CG missing edges are either directed, which we interpret as a generalization of an absence of a direct effect, or undirected, which we interpret as an absence of direct outcome interference.

For a complete CG $G$, fix $\vec{B} \in B(G)$. Consider an edge subgraph $G^\dagger$ of $G$, such that any edge missing in $G^\dagger$ is “associated with $\vec{B}$,” that is, is either between elements of $\vec{B}$, or a directed arrow into an element of $\vec{B}$. Then we define the causal submodel associated with $G^\dagger$ as the chain graph causal model associated with $G$, with the following additional restrictions. For every $B \in B$, and every $\vec{A} \in X_{pa_G(B)}$, and $\vec{A}_B$ a restriction of $\vec{A}_B$ to values of $pa_G(B)$,

$$p(B(\vec{A}_B) \mid \{\vec{B} \setminus \{B\}\}(\vec{A}_B)) \text{ is only a function of } \vec{A}_B, B, \text{ and } nb_{G^\dagger}(B).$$

Submodels associated with an arbitrary edge subgraph of the model associated with a complete CG $G$ can be defined similarly, since missing edges can be partitioned by their association with elements of $B(G)$.

These models are defined using potential outcomes, and not using structural equations. An appropriate generalization of the structural equation definition, along with a data generating process for these models, based on an equilibrium process of a Markov chain defined via a Gibbs sampler, was given in (Lauritzen & Richardson, 2002). In the remainder of the paper, we refer to our model as the “chain graph causal model” (CGM).

A saturated CGM (trivially) corresponds to existing models for partial interference (Tchetgen & VanderWeele, 2012) that correspond to a saturated observed data distribution. A CGM that is not saturated corresponds to an edge subgraph of a complete chain graph. Such models
are appropriate in interference settings where some contagion effects, infectiousness effects, or spillover effects are absent. For instance, settings where the outcome of unit \( i \) is independent of treatments given to other units and “non-neighboring” unit outcomes, conditional on treatment for unit \( i \) and “neighboring” outcomes of unit \( i \) can be naturally represented by a chain graph model. These assumptions are quite natural for instance in social network settings.

We now generalize existing results on the observed data distribution factorization and the g-formula from DAG models to CG models.

**Lemma 4.** Under the CGM associated with a CG \( \mathcal{G} \) with a vertex set \( \vec{V} \), the observed data distribution factorizes as

\[
p(\vec{V}) = \prod_{\vec{B} \in \mathcal{B}(\mathcal{G})} \frac{1}{Z_{\vec{B}}(\text{pa}_G(\vec{B}))} \left( \prod_{\vec{C} \in \mathcal{C}((\text{fa}_G(\vec{B}))^a), \vec{C} \not\subseteq \text{pa}_G(\vec{B})} \phi_{\vec{C}}(\vec{C}) \right),
\]

where \( \phi_{\vec{C}}(\vec{C}) \) are unnormalized potential functions mapping \( \mathcal{X}_{\vec{C}} \) to non-negative real numbers.

**Proof.** If \( \mathcal{G} \) is complete, the result is a generalization of results in (Richardson & Robins, 2013). If \( \mathcal{G} \) is an edge subgraph of a complete CG \( \mathcal{G}^* \), then \( p(\vec{B}(\vec{A}_\vec{B})) = p(\vec{B} \mid \text{pa}_G(\vec{B}) = \vec{A}_\vec{B}) \), and (12) gives exactly the local Markov property for chain graphs for \( \mathcal{G} \). Since we assumed positivity for every factor, \( p(\vec{V}) \) is positive, and local Markov property implies that \( p(\vec{V}) \) obeys the chain graph factorization with respect to \( \mathcal{G} \), which gives our conclusion. \( \square \)

A generalization of the truncated factorization or g-formula holds for all responses to valid treatments in the CGM.

**Lemma 5.** For any valid \( \vec{A} \) in a CGM associated with a CG \( \mathcal{G} \) with vertex set \( \vec{V} \),

\[
p(\{\vec{V} \setminus \vec{A}\}(\vec{A}) = \vec{v}) = \prod_{\vec{B} \in \mathcal{B}(\mathcal{G}), \vec{B} \cap \vec{A} = \emptyset} \frac{\Pi_{\vec{C} \in \mathcal{C}((\text{fa}_G(\vec{B}))^a), \vec{C} \not\subseteq \text{pa}_G(\vec{B})} \phi_{\vec{C}}(\vec{A} \cap \vec{C}, \vec{v} \setminus \vec{A})}{Z_{\vec{B}}(\vec{A} \cap \text{pa}_G(\vec{B}), \vec{v} \setminus \text{pa}_G(\vec{B}))}. \tag{13}
\]
Similarly, for a subset \( \mathbf{\bar{Y}} \subset \mathbf{\bar{V}} \setminus \mathbf{\bar{A}} \),

\[
p(\mathbf{\bar{Y}}(\mathbf{\bar{A}}) = \mathbf{\bar{v}}_\mathbf{\bar{Y}}) = \sum_{\mathbf{\bar{v}}_\mathbf{\bar{V}} \setminus \mathbf{\bar{Y}}} \prod_{\mathbf{\bar{B}} \in \mathbf{\bar{B}}} \phi_{\mathbf{\bar{C}}} (\mathbf{\bar{A}}_{\mathbf{\bar{B}} \cap \mathbf{\bar{A}}} \setminus \mathbf{\bar{v}}_{\mathbf{\bar{C}} \setminus \mathbf{\bar{A}}}) Z_{\mathbf{\bar{G}}} (\mathbf{\bar{A}}_{\mathbf{\bar{B}} \cap \mathbf{\bar{A}}} \setminus \mathbf{\bar{v}}_{\mathbf{\bar{B}} \cap \mathbf{\bar{A}}}) \prod_{\mathbf{\bar{C}} \in \mathbf{\bar{C}}} (\phi_{\mathbf{\bar{G}}} (\mathbf{\bar{A}}_{\mathbf{\bar{C}} \cap \mathbf{\bar{A}}} \setminus \mathbf{\bar{v}}_{\mathbf{\bar{C}} \setminus \mathbf{\bar{A}}})^\mathbf{\bar{C}} \setminus \mathbf{\bar{A}}) \prod_{\mathbf{\bar{D}} \in \mathbf{\bar{D}}} (\mathbf{\bar{v}}_{\mathbf{\bar{D}} \setminus \mathbf{\bar{A}}}) (14)
\]

**Proof.** This follows by the generalization of the argument proving propositions 11 and 16 in (Richardson & Robins, 2013) from singleton nodes to blocks. Note that restricting attention to the “outer” factorization of a CG resembling the DAG factorization defined on elements of \( \mathbf{\bar{B}}(\mathbf{\bar{G}}) \) suffices for the argument.

\[\square\]

### 4.3. Treatment Decomposition and a Two Outcome Example

Having given an appropriate causal model for interference within blocks, we now generalize the treatment decomposition approach to mediation analysis advocated in (Robins & Richardson, 2010) to our model. We illustrate our proposal with a simple partial interference setting with two outcomes exhibiting interference, shown in Fig. 1 (c). These outcomes are sometimes called *dyads* (Kenny et al., 2006). More complex versions of our proposal applicable to general network data without the assumption of partial interference are possible given a CG model, but we leave them to future work.

In our example, we have blocks with two outcomes, \( Y^1 \) and \( Y^2 \), and a single treatment \( A \) administered to \( Y^1 \). As before, we split \( A \) into two components, \( A' \) and \( A'' \), that always occur together normally, but can in principle be intervened on separately. Furthermore, \( A' \) only influences \( Y^1 \) “directly,” and \( Y^2 \) “indirectly via \( Y^1 \), while \( A'' \) only influences \( Y^2 \) “directly” and \( Y^1 \) “indirectly” via \( Y^2 \). Since we insist on a symmetric relation of \( Y^1 \) and \( Y^2 \), we can no longer conceptualize “directly” and “indirectly” using causal DAG models, and instead use a CG model, shown in Fig. 1 (d).
Submodel restrictions given in Section 4.2 applied to the block \( \{Y^1, Y^2\} \) in the graph in Fig. 1 (d) are:

\[
p\{Y^1(a', a'') \mid Y^2(a', a'')\} \text{ is only a function of } Y^1, A', Y^2
\]

(15)

\[
p\{Y^2(a', a'') \mid Y^1(a', a'')\} \text{ is only a function of } Y^2, A'', Y^1.
\]

(16)

These can be viewed as symmetric versions of constraints (4) and (5).

Lemma 4 applied to the observed data law of the CGM in Fig. 1 (d) gives the following factorization:

\[
p(Y^1, Y^2, A', A'', A) = \frac{\phi_{Y^1, Y^2}(y^1, y^2)\phi_{Y^1, A'}(y^1, a')\phi_{Y^2, A''}(y^2, a'')}{Z(a', a'')} p(A' \mid A)p(A'' \mid A)p(A),
\]

(17)

where \( Z(a', a'') \) is a normalizing constant, and, as before, factors \( p(A' \mid A) \) and \( p(A'' \mid A) \) are deterministic.

Given our split treatment formulation, and a single treatment \( A \) meant for \( Y^1 \), the spillover effect \( \mathbb{E}[Y^2 \mid A = 1] - \mathbb{E}[Y^2 \mid A = 0] \) decomposes into precisely the same contagion and infectiousness effects as (8) and (7). However, the identifying formula implied by (13) for counterfactuals involved is different. For instance,

\[
p\{Y^2(A' = 0, A'' = 1) = y^2\} = \sum_{y^1} p\{Y^2(A' = 0, A'' = 1) = y^2, Y^1(A' = 0, A'' = 1) = y^1\}
\]

\[
= \sum_{y^1} p(Y^2 = y^2, Y^1 = y^1 \mid A' = 0, A'' = 1)
\]

\[
= \sum_{y^1} \frac{\phi_{Y^1, Y^2}(y^1, y^2)\phi_{Y^1, A'}(y^1, 0)\phi_{Y^2, A''}(y^2, 1)}{Z(0, 1)},
\]

(18)

where the first equality is by definition, and the other two by Lemma 5. We call expression (18) the symmetric mediation formula, and it is a special case of the CG version of the \( g \)-formula on an appropriately expanded CG in Fig. 1 (d), just as regular mediation formula is a special case of the \( g \)-formula on an appropriately expanded DAG in Fig. 1 (b).
5. Statistical Inference For The Symmetric Mediation Formula

We now consider two approaches to statistical inference for the symmetric mediation formula in the dyad (block of size 2) setting, one based on maximum likelihood estimation, and one on doubly robust semi-parametric estimators. Assume a dataset with \( n \) dyads, with labeled outcomes \( \mathbf{Y} = (Y^1, Y^2) \), and baseline covariates \( \mathbf{C} = (C^1, C^2) \) for each of the two units. For simplicity, we assume only a single treatment \( A \) is assigned. Without loss of generality, assume \( A \) is assigned to unit 2. We also assume conditional ignorability for treatment assignment, specifically \( \{Y^1(a), Y^2(a)\} \perp \perp A \mid \mathbf{C} \) for any \( a \in \mathcal{X}_A \). A graphical representation of this model is shown in Fig. 1 (e). We wish to estimate direct and indirect components of the spillover effect of \( A \) on \( Y_1 \) “mediated” by \( Y_2 \), in the sense described above.

Extending restrictions in Section 4·3 to the setting with baseline covariates \( \mathbf{C} \) leads to exclusion restrictions

\[
p\{Y^1(a', a'') \mid Y^2(a', a''), \mathbf{C}\} \text{ is only a function of } Y^1, A', Y^2, \mathbf{C}
\]

\[
p\{Y^2(a', a'') \mid Y^1(a', a''), \mathbf{C}\} \text{ is only a function of } Y^2, A'', Y^1, \mathbf{C}.
\]

These constraints are represented by an extension of the CG in Fig. 1 (e), shown in Fig. 1 (f), where treatments are split.

5.1. Maximum likelihood inference

Equation (13) applied to Fig. 1 (f) implies

\[
p\{Y^1(a', a'')\} = \sum_{\mathbf{c}, y^2} p(y^1, y^2 \mid a', a'', \mathbf{c}) p(\mathbf{c}),
\]

and

\[
p(y^1, y^2 \mid a', a'', \mathbf{c}) = \frac{\phi_{Y^1, Y^2, \mathbf{C}}(y^1, y^2, \mathbf{c}) \phi_{Y^1, A, \mathbf{C}}(y^1, a', \mathbf{c}) \phi_{Y^2, A, \mathbf{C}}(y^2, a'', \mathbf{c})}{Z(a', a'', \mathbf{c})}.
\]
To estimate $E \{ Y^1(a', a'') \} = \sum_{\vec{c}, y^2} p(y^1, y^2 \mid a', a'', \vec{c}) p(\vec{c})$, we can rewrite $E \{ Y^1(a', a'') \}$ as

$$
\psi(a', a'') = \sum_{\vec{c}} \theta(a', a'' , \vec{c}) p(\vec{c}),
$$

where $\sum$ may be interpreted as integration for continuous variables, and

$$
\theta(a', a'' , \vec{c}) = \frac{\beta_{\psi}(a', a'', \vec{c})}{\beta_1(a', a'', \vec{c})},
$$

where $\beta_h(a', a'', \vec{c})$ is defined as follows for any function $h(y^1, y^2)$,

$$
\beta_h(a', a'', \vec{c}) = \sum_{y^1, y^2} h(y^1, y^2) f(y^1 \mid a', Y^2 = 0, \vec{c}) \gamma(y^1, y^2 \mid \vec{c}) f(y^2 \mid a'', Y^1 = 0, \vec{c})
$$

(20)

This type of parameterization is described in more detail in (Chen, 2007). An advantage of this type of parameterization is that it decomposes the joint outcome distribution into variation independent conditional distributions, which are easy to specify using standard regression models. Another alternative for binary models is a standard log-linear parameterization. Note that the the parameterization in (20) is in the form (19), with $\gamma(y^1, y^2 \mid \vec{c})$ serving as $\phi_{Y^1,Y^2,\vec{c}}$, $f(y^1 \mid a', Y^2 = 0, \vec{c})$ serving as $\phi_{Y^1,A,\vec{c}}$, $f(y^2 \mid a'', Y^1 = 0, \vec{c})$ serving as $\phi_{Y^2,A,\vec{c}}$, while $\beta_1(a', a'', \vec{c})$ serves as the normalizing function $Z(a', a'', \vec{c})$.

Maximum likelihood estimation of $\psi(a', a'')$ requires correct specification of models for $f(y^1 \mid a', Y^2 = 0, \vec{c}), \gamma(y^1, y^2 \mid \vec{c})$ and $f(y^2 \mid a'', Y^1 = 0, \vec{c})$. Specifically, suppose that one has specified parametric models $f(y^1 \mid a', Y^2 = 0, \vec{c}; \omega_1), \gamma(y^1, y^2 \mid \vec{c}; \nu)$ and $f(y^2 \mid a'', Y^1 = 0, \vec{c}; \omega_2)$ for $f(y^1 \mid a', Y^2 = 0, \vec{c}), \gamma(y^1, y^2 \mid \vec{c})$ and $f(y^2 \mid a'', Y^1 = 0, \vec{c})$ respectively. Given a data matrix $D$, the maximum likelihood estimator $(\hat{\omega}_1, \hat{\omega}_2, \hat{\nu})$ of $(\omega_1, \omega_2, \nu)$ maximizes the following log-likelihood $\log L_{Y,A,\hat{\nu}}(D; \{\omega_1, \omega_2, \nu\})$

$$
\sum_{i=1}^n \log \frac{f(y^1_i \mid a_i, Y^2 = 0, \vec{c}_i; \omega_1) \gamma(y^1_i, y^2_i \mid \vec{c}_i; \nu)}{f(y^1_i \mid a_i, Y^2 = 0, \vec{c}_i; \omega_2)} \frac{f(y^2_i \mid a_i, Y^1 = 0, \vec{c}_i; \omega_2)}{f(y^2_i \mid a_i, Y^1 = 0, \vec{c}_i; \omega_1)}.
$$
The corresponding maximum likelihood estimator of $\psi(a', a'')$ is given by

$$\hat{\psi} = n^{-1} \sum_{i=1}^{n} \theta (a', a'', \bar{c}_i; \hat{\omega}_1, \hat{\omega}_2, \hat{\nu})$$

where $\theta(a', a'', \bar{c}; \hat{\omega}_1, \hat{\omega}_2, \hat{\nu})$ is equal to $\theta(a', a'', \bar{c})$ evaluated at $\gamma(y^1, y^2 | \bar{c}, \hat{\nu})$, $f(y^1 | a', Y^2 = 0, \bar{c}; \hat{\omega}_1)$, and $f(y^2 | a'', Y^1 = 0, \bar{c}; \hat{\omega}_2)$. Under standard regularity conditions, $n^{1/2}(\hat{\psi} - \psi)$ is approximately normal for large $n$ with mean zero and variance given by $\hat{\psi} \hat{I}^{-1} \hat{\psi}^T$, where

$$\hat{\psi} = n^{-1} \sum_{i=1}^{n} \frac{\partial \theta (a', a'', \bar{c}_i; \hat{\omega}_1, \hat{\omega}_2, \hat{\nu})}{\partial (\hat{\omega}'_1, \hat{\omega}'_2, \hat{\nu}')}$$

and $\hat{I}$ is the second derivative of $\log \mathcal{L}_{Y, A, C^2} \{ D; (\omega_1, \omega_2, \nu) \}$ with respect of $(\omega_1, \omega_2, \nu)$ evaluated at $(\hat{\omega}_1, \hat{\omega}_2, \hat{\nu})$.

5.2. Towards doubly robust inference

We consider some preliminary results on robust statistical inferences about $\psi$. We consider a setting in which $\bar{Y}$ is continuous and $A$ is randomized, so that $\bar{C}$ can be taken as the empty set.

We develop an estimator of $\beta_h(a', a'')$ that is consistent under the semiparametric union model which assumes that (i) a parametric model $\gamma(y^1, y^2; \nu)$ for $\gamma(y^1, y^2)$ is correctly specified; and (ii) either (ii.a) $f(Y^1 | A, Y^2 = 0; \omega_1)$ or (ii.b) $f(Y^2 | A, Y^1 = 0; \omega_2)$ is correctly specified but not necessarily both. Therefore the proposed estimator is doubly robust since it offers the analyst two opportunities to obtain a consistent estimator of $\beta_h(a, a^2)$, and therefore of $\psi(a, a^2)$. In order to exhibit such an estimator requires successfully completing the following tasks:

1. First, obtaining a consistent estimator of $\gamma(y^1, y^2; \nu)$ under (i) and (ii).
2. Second, obtaining a consistent estimator of $\beta_h(a', a'')$ under (i) and (ii).

Tchetgen Tchetgen et al (2010) have previously characterized a large class of doubly robust estimators that accomplish 1, in the sense that any estimator of $\gamma(y^1, y^2; \nu)$ in their class (which includes a semiparametric locally efficient estimator) is guaranteed to remain consistent and
asymptotically normal under (i) and (ii). Specifically, let \( \tilde{\omega}_1 (\nu) \) denote the conditional MLE that maximizes the conditional log likelihood \( \sum_i \log f(y_i^1 \mid a_i, y_i^2; \omega_1, \nu) \), where

\[
f(y_i^1 \mid a_i, y_i^2; \omega_1, \nu) = \frac{f(y_i^1 \mid a_i, Y_i^2 = 0; \omega_1) \gamma(y_i^1, y_i^2; \nu)}{\sum_{y_i^1} f(y_i^1 \mid a_i, Y_i^2 = 0; \omega_1) \gamma(y_i^1, y_i^2; \nu)}.
\]

Likewise, let \( \tilde{\omega}_2 (\nu) \) denote the corresponding conditional MLE of \( \omega_2 \). Tchetgen Tchetgen et al (2010) proved that the solution \( \hat{\nu}_{dr} \) to the following class of estimating equations is doubly robust, i.e. consistent and asymptotically normal under (i) and (iii):

\[
0 = \sum_{i=1}^n h(\tilde{\epsilon}_i) \gamma(y_i^1, y_i^2; \hat{\nu}_{dr})^{-1} \left[ y_i^1 - E \left\{ y_i^1 \mid a_i, Y^2 = 0; \tilde{\omega}_1 (\hat{\nu}_{dr}) \right\} \right] \\
\times \left[ y_i^2 - E \left\{ y_i^2 \mid a_i, Y^1 = 0; \tilde{\omega}_2 (\hat{\nu}_{dr}) \right\} \right]
\]

where \( h \) is a user-specified function of dimension matching that of \( \nu \). Tchetgen Tchetgen et al (2010) developed a more general class of doubly robust estimators including locally semiparametric efficient estimators for polytomous, count or continuous \( Y \), we refer the reader to the original manuscript for more details.

Next, we turn to task 2. Consider the following estimating equation for \( \beta_h (a_y, a_m) \):

\[
U_1 \{ h; \beta_h, \tilde{\omega}_1 (\nu) \} = \frac{I(A = a'')}{\gamma(y_i^1, y_i^2; \nu)} \sum_{y_i^1} h(y_i^1, y_i^2) f \left\{ y_i^1 \mid a', Y^2 = 0; \tilde{\omega}_1 (\hat{\nu}_{dr}) \right\} \gamma(y_i^1, y_i^2; \nu) - \beta_h (a', a'')
\]

and let \( \tilde{\beta}_{h,1} \) denote the solution to the estimating equation \( \sum U_1 \{ h; \tilde{\beta}_{h,1}, \tilde{\omega}_1 (\hat{\nu}_{dr}) \} = 0 \). Likewise, let \( \tilde{\beta}_{h,2} \) denote the solution to the estimating equation \( \sum U_2 \{ h; \tilde{\beta}_{h,2}, \tilde{\omega}_2 (\hat{\nu}_{dr}) \} = 0 \) where \( U_2 \) is defined as \( U_1 \) with \( I(A = a'') \) replaced by \( I(A = a') \) and \( f \{ y_i^1 \mid a', Y_i^2 = 0; \tilde{\omega}_1 (\hat{\nu}_{dr}) \} \) replaced by \( f \{ y_i^2 \mid a'', Y_i^1 = 0; \tilde{\omega}_2 (\hat{\nu}_{dr}) \} \). It is straightforward to
show that $\tilde{\beta}_{h,1}$ is consistent under (i) and (ii.a); while $\tilde{\beta}_{h,2}$ is consistent under (i) and (ii.b). Let

$$
U_{dr,i} \{h; V \kappa, \tilde{\omega}_1 (\tilde{\nu}_{dr}), \tilde{\omega}_2 (\tilde{\nu}_{dr}) \} = I \left( A_i = a' \right) f \left\{ y_i \mid \alpha', Y^1 = 0; \tilde{\omega}_2 (\tilde{\nu}_{dr}) \right\}
$$

$$
\gamma (y_i^1, y_i^2; \tilde{\nu}_{dr}) f \left\{ y_i^1 \mid \alpha, Y^1 = 0; \tilde{\omega}_2 (\tilde{\nu}_{dr}) \right\}
$$

$$
\times \left\{ h \left( y_i^1, y_i^2 \right) \gamma \left( y_i^1, y_i^2; \tilde{\nu}_{dr} \right) - \tilde{\beta}_{h,dr} (a', a'') \\
- \sum_{y_i^1} h \left( y_i^1, y_i^2 \right) f \left\{ y_i^1 \mid \alpha, Y_i^1 = 0; \tilde{\omega}_1 (\tilde{\nu}_{dr}) \right\} \gamma \left( y_i^1, y_i^2; \tilde{\nu}_{dr} \right) + \tilde{\beta}_{h,1} (a', a'') \\
- \sum_{y_i^2} h \left( y_i^1, y_i^2 \right) f \left\{ y_i^2 \mid a'', Y_i^1 = 0; \tilde{\omega}_2 (\tilde{\nu}_{dr}) \right\} \gamma \left( y_i^1, y_i^2; \tilde{\nu}_{dr} \right) + \tilde{\beta}_{h,2} (a', a'') \right\}
$$

In the appendix, we establish that the solution $\tilde{\beta}_{h,dr}$ to the following estimating equation is doubly robust in the sense of being consistent under (i) and (ii),

$$
0 = \sum_i U_{dr,i} \left\{ h_1; \tilde{\beta}_{h,dr}, \tilde{\omega}_1 (\tilde{\nu}_{dr}), \tilde{\omega}_2 (\tilde{\nu}_{dr}) \right\}
$$

Confidence intervals for these estimates can be obtained via the standard nonparametric bootstrap.

### 5.3. Model Falsifiability

One advantage of the treatment decomposition approach to classical mediation analysis is that assumptions necessary for identification may in principle be tested by a randomized experiment on treatment components. The same is true in our symmetric treatment decomposition model represented by causal chain graphs. However, an additional desirable property holds in causal CGs, but not in causal DAGs – identifying assumptions can be falsified using observed data. This is because the factorization (17) implies a restriction on the observed data law (where $A = A' = A''$). Specifically, for the observed data law, (17) reduces to

$$
p(Y^1, Y^2, A) = \frac{\phi_{Y^1,Y^2}(Y^1, Y^2)\phi_{Y^1,A}(Y^1, A)\phi_{Y^2,A}(Y^2, A)}{Z(A)} p(A),
$$

(21)
which is not the saturated observed data factorization, given by

\[
p(Y^{1}, Y^{2}, A) = \frac{\phi_{Y^{1}, Y^{2}, A}(Y^{1}, Y^{2}, A)\phi_{Y^{1}, A}(Y^{1}, A)\phi_{Y^{2}, A}(Y^{2}, A)}{Z(A)}p(A)
= \frac{\phi_{Y^{1}, Y^{2}, A}(Y^{1}, Y^{2}, A)}{Z(A)}p(A).
\]

In particular, the first term in the numerator of (21) does not depend on \( A \).

This implies that given a parameterization of the observed data factorization, for instance a standard log-linear parameterization for binary models, or the parameterization described in section 5.1, we may falsify our model by the likelihood ratio test, which would entail rejecting the submodel given by (21). The rejection of the submodel would imply that it is not possible to set up a randomized controlled trial, where the treatment \( A \) is decomposable in such a way that the appropriate exclusion restrictions, represented by missing edges in Fig. 1 (d), hold. We contrast this situation with what happens with mediation analysis in a DAG model. In such a model, assumptions underlying identification of mediation functionals in DAG models do not place any restrictions on the observed data law. This implies that decomposability of the treatment \( A \) into components that satisfy exclusion restrictions, represented by missing edges in Fig. 1 (b), must be verified entirely using background knowledge.

Another implication of the fact that in our setting contagion and infectiousness components of the spillover effect are only identified in causal models consistent with a strict submodel of the saturated observed data model is that if the observed data law does not lie in this submodel, the identifying functionals corresponding to contagion and infectiousness effects do not add up to the spillover effect. This is in contrast to classical mediation analysis settings where functionals given by the mediation formula corresponding to natural direct and indirect effects always add up to the functional corresponding to the average causal effect (this follows by a simple telescoping
sum argument), even in cases where direct and indirect effects are not identifiable, and thus not equal to those functionals.

6. CONCLUSIONS

In this paper, we proposed a new approach for decomposing the spillover effect in causal inference problems with partial treatment interference among two interacting outcomes. We decompose the spillover effect into direct and indirect components using an approach that considers outcomes to be on the same footing. In particular, our approach allows, in a coherent way, for any one of the interacting outcomes to serve as the “outcome” for the spillover effect, with the other acting as the “mediator.”

To achieve this property, we use a generalization of causal models of the DAG (Pearl, 2009) to chain graphs (Lauritzen & Richardson, 2002), which allow both directed causal relationships between treatments and outcomes, and symmetric relationships between outcomes that arise in interference problems. Given a causal chain graph model, we propose to view mediation analysis as “splitting,” or decomposition of treatments, as a generalization of the approach to mediation analysis described in (Robins & Richardson, 2010).

We show that under our model functionals corresponding to direct and indirect components of the spillover effects are identified via the symmetric mediation formula, and that some of the assumptions that identification relies on can be falsified from observed data. This falsifiability property is not present in mediation analysis of DAG models. Finally, we describe statistical inference for components of the spillover effect in our setting. We propose two estimators, one based on maximizing the log likelihood, and one which exhibits double robustness in a restricted version of our problem.
Then for all $\omega_2$, 

$$
\sum_{y^1, y^2} f (y^1 | a', Y^2 = 0; \omega_1) h (y^1, y^2) f (y^2 | a'', Y^1 = 0; \omega_2) \gamma (y^1, y^2; \nu).
$$

We prove that $\tilde{\beta}_{h, dr}$ is doubly robust in the sense of being consistent under the union model where either (i) and (ii.a) or (i) and (ii.b) from section 5-2 hold. Note that under (i) and (ii), $\tilde{\nu}_{dr}$ is consistent. Furthermore, suppose that (ii.a) holds such that $\tilde{\omega}_1 (\tilde{\nu}_{dr})$ is also consistent. Note that under this submodel $\tilde{\beta}_{h, 1}$ is consistent but $\tilde{\beta}_{h, 2}$ is consistent for 

$$
E \{ U_{dr, i} \} (h; \beta_h, \omega_1, \omega_2) \mid a_i, y_i^1
$$

$$
\times \frac{\gamma (y^1, y^2; \nu) h (y^1, y^2) - \beta_h (a', a'')}{\gamma (y^1, y_i^2; \nu)}
$$

$$
\times \left\{ \begin{array}{l}
\sum_{y^1} f (y^1 | a', Y^2 = 0; \omega_1) \gamma (y^1, y_i^2; \nu) h (y^1, y_i^2) - \beta_h (a', a'') \\
- \sum_{y^1} h (y^1, y_i^2) f (y^1 | a', Y_i^2 = 0, \omega_1) \gamma (y^1, y_i^2; \nu) + \beta_h (a', a'') \\
- \sum_{y^2} h (y^1, y_i^2) f (y^2 | a', Y_i^2 = 0, \omega_2) \gamma (y^1, y_i^2; \nu) + \beta_{h, 2} (a', a'')
\end{array} \right\}
$$

$$
= 0.
$$
Next, suppose that (i) and (ii.b) hold. Then \( \tilde{\beta}_{h,2} \) is consistent but \( \tilde{\beta}_{h,1} \) is consistent for
\[
\sum_{y^1, y^2} f (y^1 | a', Y^2 = 0; \omega_1^2) h (y^1, y^2) f (y^2 | a''', Y^1 = 0; \omega_2) \gamma (y^1, y^2; \nu). \]
Then for all \( \omega_1^2 \),
\[
E \{ U_{i\nu,i} (h; \beta_h, \omega_1^2, \omega_2) | a_i, y_i \}
\]
\[
\propto \sum_{y^2} \frac{\gamma (y^1, y^2) f (y^2 | a', Y^1 = 0) f (y^2 | a'', Y^1 = 0; \omega_2)}{\gamma (y^1, y^2; \nu) f (y^2 | a', Y^1 = 0; \omega_2)} \left\{ \gamma (y^1, y^2; \nu) h (y^1, y^2) - \beta_h (a', a'') \right\}
\]
\[
\times \left\{ - \sum_{y^1} h (y^1, y^2) f (y^1 | a', Y^1 = 0; \omega_1^2) \gamma (y^1, y^2; \nu) + \beta_{h,1} (a', a'', c_i) \right\}
\]
\[
- \sum_{y^2} h (y^1, y^2) f (y^2 | a'', Y^1 = 0; \omega_2) \gamma (y^1, y^2; \nu) + \beta_h (a', a'', c_i; \kappa) \right\}
\]
\[
= - \sum_{y^1, y^2} f (y^2 | a'', Y^1 = 0; \omega_2) h (y^1, y^2) f (y^1 | a', Y^2 = 0; \omega_1^2) \gamma (y^1, y^2; \nu) + \beta_{h,1} (a', a'') \right\}
\]
\[
- \sum_{y^2} h (y^1, y^2) f (y^2 | a'', Y^1 = 0, c_i; \omega_2) \gamma (y^1, y^2; \nu) + \beta_h (a', a'') \right\}
\]
\[
= 0,
\]
proving the result.

REFERENCES

BARON, R. M. & KENNY, D. A. (1986). The moderator-mediator variable distinction in social psychology research: Conceptual, strategic, and statistical considerations. Journal of Personality and Social Psychology 51, 1173–1182.

CHEN, H. Y. (2007). A semiparametric odds ratio model for measuring association. Biometrics 63, 413–421.

HALLORAN, M. E. & STRUCHINER, C. J. (1995). Causal inference for infectious diseases. Epidemiology 6, 142–151.

KENNY, D. A., KASHY, D. A. & COOK, W. L. (2006). Dyadic Data Analysis. Guilford Press New York.

LAURITZEN, S. L. & RICHARDSON, T. S. (2002). Chain graph models and their causal interpretations (with discussion). Journal of the Royal Statistical Society: Series B 64, 321–361.

NEYMAN, J. (1923). Sur les applications de la thar des probabilitie s aux experiences agaricales: Essay des principle. excerpts reprinted (1990) in English. Statistical Science 5, 463–472.

OGBURN, E. L. & VANDERWEELE, T. J. (2014). Causal diagrams for interference. Statistical Science 29, 559–578.

PEARL, J. (2001). Direct and indirect effects. In Proceedings of the Seventeenth Conference on Uncertainty in Artificial Intelligence (UAI-01). Morgan Kaufmann, San Francisco.

PEARL, J. (2009). Causality: Models, Reasoning, and Inference. Cambridge University Press, 2nd ed.
I. Shpitser, E.J. Tchetgen Tchetgen AND Ryan Andrews

Pearl, J. (2011). The causal mediation formula – a guide to the assessment of pathways and mechanisms. Tech. Rep. R-379, Cognitive Systems Laboratory, University of California, Los Angeles.

Richardson, T. S. & Robins, J. M. (2013). Single world intervention graphs (SWIGs): A unification of the counterfactual and graphical approaches to causality. preprint: http://www.csss.washington.edu/Papers/wp128.pdf.

Robins, J. M. (2011). A new approach to causal inference in mortality studies with sustained exposure periods – application to control of the healthy worker survivor effect. Mathematical Modeling 7, 1393–1512.

Robins, J. M. & Greenland, S. (1992). Identifiability and exchangeability of direct and indirect effects. Epidemiology 3, 143–155.

Robins, J. M. & Richardson, T. S. (2010). Alternative graphical causal models and the identification of direct effects. Causality and Psychopathology: Finding the Determinants of Disorders and their Cures.

Rubin, D. B. (1974). Estimating causal effects of treatments in randomized and non-randomized studies. Journal of Educational Psychology 66, 688–701.

Shpitser, I. & Pearl, J. (2006a). Identification of conditional interventional distributions. In Proceedings of the Twenty Second Conference on Uncertainty in Artificial Intelligence (UAI-06). AUAI Press, Corvallis, Oregon.

Shpitser, I. & Pearl, J. (2006b). Identification of joint interventional distributions in recursive semi-Markovian causal models. In Proceedings of the Twenty-First National Conference on Artificial Intelligence (AAAI-06). AAAI Press, Palo Alto.

Sobel, M. E. (2006). What do randomized studies of housing mobility demonstrate? causal inference in the face of interference. Journal of the American Statistical Association 101.476, 1398–1407.

Spirtes, P. (1995). Directed cyclic graphical representations of feedback models. In Proceedings of the Eleventh Conference on Uncertainty in Artificial Intelligence (UAI-95). AUAI Press.

Tchetgen, E. J. T. & VanderWeele, T. J. (2012). On causal inference in the presence of interference. Statistical Methods in Medical Research 21, 55–75.

Tian, J. & Pearl, J. (2002). On the testable implications of causal models with hidden variables. In Proceedings of the Eighteenth Conference on Uncertainty in Artificial Intelligence (UAI-02), vol. 18. AUAI Press, Corvallis, Oregon.

VanderWeele, T. J., Tchetgen, E. J. T. & Halloran, M. E. (2012). Components of the indirect effect in vaccine trials: identification of contagion and infectiousness effects. Epidemiology 23, 751–761.

Wooldridge, J. M. (2015). Introductory Econometrics: A Modern Approach. South-Western, Mason, OH, 5th ed.
Biometrika style

[Received September 2017. Revised October 2017]