Confounder Selection: Objectives and Approaches

F. Richard Guo, Anton Rask Lundborg and Qingyuan Zhao

Abstract. Confounder selection is perhaps the most important step in the design of observational studies. A number of criteria, often with different objectives and approaches, have been proposed, and their validity and practical value have been debated in the literature. Here, we provide a unified review of these criteria and the assumptions behind them. We list several objectives that confounder selection methods aim to achieve and discuss the amount of structural knowledge required by different approaches. Finally, we discuss limitations of the existing approaches and implications for practitioners.

Key words and phrases: Causal graphical model, Causal inference, Covariate adjustment, Common causes, Study design, Variable selection.

1. INTRODUCTION

When designing an observational study to estimate the causal effect of a treatment on an outcome, possibly the most important task is to select covariates that should be measured and controlled for. A variety of criteria and methods have been proposed, sometimes as formal algorithms but most often as loosely stated principles. These proposals differ quite substantially in their objectives and approaches, so it is not uncommon that methods developed for one objective fail in achieving another, thus causing a great deal of confusion for methodologists and practitioners.

To get a sense of the variety of criteria for confounder selection in the literature, a brief literature review is helpful. In a highly cited tutorial of propensity score methods, Austin (2011, p. 414) suggested that there are four possible sets of confounders to control for: “all measured baseline covariates, all baseline covariates that are associated with treatment assignment, all covariates that affect the outcome (i.e., the potential confounders), and all covariates that affect both treatment assignment and the outcome (i.e., the true confounders).” Citing previous simulation studies (Austin, Grootendorst and Anderson, 2007; Brookhart et al., 2006), Austin concluded that “there were merits to including only the potential confounders or the true confounders in the propensity score model”. The idea that only controlling for the “true confounders” is sufficient, if not superior, is commonplace in practice (Glymour, Weuve and Chen, 2008) and methodological development (Ertefaie, Asgharian and Stephens, 2017; Shortreed and Ertefaie, 2017; Koch et al., 2020). We will call this the “conjunction heuristic”; other authors have referred to it as the “common cause principle”. It is well known that this approach can select too few covariates if some confounders are not observed (VanderWeele, 2019).

Another widely used criterion for confounder selection is to simply use all observed pre-treatment covariates. This is primarily driven by the heuristic that an observational study should be designed to emulate a randomized experiment, as the latter stochastically balances all pre-treatment covariates, observed or unobserved. This consideration led Rubin (2009, p. 1421) to conclude that “I cannot think of a credible real-life situation where I would intentionally allow substantially different observed distributions of a true covariate in the treatment and control groups.” There, Rubin was defending this “pre-treatment heuristic” against counterexamples stemming from the graphical theory of causality (Shrier, 2008; Pearl, 2009a; Sjölander, 2009). In these counterexamples, conditioning on some pre-treatment covariates introduces “collider bias” to the causal analysis because conditioning on a variable can induce dependence between its parents in a causal graph; see Fig. 1(a) below. This is often seen as a dispute in the long-standing friction between the graphical and the potential outcome (or counterfactual) approaches towards causal inference: the mathematical counterexample seems to make it easy for the graphical approach to claim a victory. However, the target trial emulation perspective may still be very useful in practice (Hernán and Robins, 2016; Hernán and Robins, 2020).
In response to the drawbacks of the common cause and pre-treatment heuristics, VanderWeele and Shpitser (2011) proposed the “disjunctive cause criterion” that selects pre-treatment covariates that are causes of the treatment, the outcome, or both (throughout this article, causes include both direct and indirect causes). They proved a remarkable property that the proposed subset is sufficient to control for confounding if the observed covariates contains at least one subset that is sufficient to control for confounding. This criterion was later revised in VanderWeele (2019) to additionally account for (1) instrumental variables, which tend to reduce efficiency under the causal model assumption or amplify bias in misspecified models, and (2) proxies of unmeasured confounders, which tend to reduce bias when controlled for.

Another approach for confounder selection in causal inference is statistical variable selection: rather than using substantive knowledge about the causes of the treatment and the outcome, can we select confounders using statistical tests (usually of conditional independence) with observational data? Besides the methods mentioned previously that use the conjunction heuristic, Robins (1997, Theorem 4.3) implied that one can sequentially discard covariates that are irrelevant to determining the treatment or the outcome, which may be viewed as another instance of the conjunction heuristic; see also Greenland, Pearl and Robins (1999a, §3.3) and Hernán and Robins (2020, §7.4). Later, de Luna, Waernbaum and Richardson (2011) extended this approach and devised an iterative algorithm that converges to a minimal subset of covariates that suffice to control for confounding; see Section 3.3 and also Persson et al. (2017). Others have proposed a more global approach that attempts to learn the causal graph (or its Markov equivalence class) first and then use the learned graph to determine the set of covariates to control for (Maathuis and Colombo, 2015; Häggström, 2017); however, the learned graph class typically does not yield a unique set of covariates to control for. A statistical version of the disjunctive cause criterion, albeit motivated by some completely different considerations, can be found in Belloni, Chernozhukov and Hansen (2013). There, the authors showed that using the union of variables selected in the treatment model and the outcome model is critical to coping with incorrectly selected variables in high-dimensional problems.

More recently, an emerging literature considers the problem of selecting the optimal set of covariates to control for that maximizes the statistical efficiency in estimating the causal effect of interest (Kuroki and Miyakawa, 2003; Henckel, Perković and Maathuis, 2022; Rotnitzky and Smucler, 2020; Guo, Perković and Rotnitzky, 2022). These algorithms require knowing the structure of the underlying causal graph.

At this point, it should have become abundantly clear that there are many different objectives and approaches in confounder selection, which often lead to conflicting views and criteria. In the rest of this article, we will give a more in-depth discussion of these objectives (Section 2) and existing approaches (Section 3), in hope that a clear description of the considerations in the literature will reveal their merits and limitations. We conclude the article with some further discussion in Section 4. Technical background and proofs are deferred to the Appendix. Throughout the paper, random vectors are sometimes viewed as sets, allowing us to use set operations to describe confounder selection.

2. OBJECTIVES OF CONFOUNDER SELECTION

We consider the canonical problem of estimating the causal effect of a binary treatment $A$ on an outcome $Y$. For $a = 0, 1$, let $Y_a$ denote the potential outcome had the treatment been administered at level $a$. Let $S$ be the set of pre-treatment covariates that are already measured or can be potentially measured. To be practical, we require any selected set of confounders to be a subset of $S$.

We use $Z$ to denote the superset of $S$ that consists of all the pre-treatment covariates, observable or not, that are relevant to estimating the causal effect of $A$ on $Y$. Formally, we assume that $Z \cup \{A, Y\}$ is causally closed in the sense that every non-trivial common cause of two distinct variables in $Z \cup \{A, Y\}$ also belongs to the set itself. Here, a common cause of two variables is said to be non-trivial if it affects either variable directly, i.e., not only through the other common causes of the two variables. Without this restriction, the set $Z$ may be too large because any cause of a common cause of $A$ and $Y$ is still a common cause of $A$ and $Y$. For a concrete choice of $Z$, we can take it to be the smallest superset of $S$ such that $Z \cup \{A, Y\}$ is causally closed, given by

$$Z = S \cup \{A, Y\} \setminus \{A, Y\},$$

where $S \cup \{A, Y\}$ is the causal closure of $S \cup \{A, Y\}$. It can be shown that this choice of $Z$ is pre-treatment (Proposition B.1). The reader is referred to Appendix B.6 for a precise definition of causal closure using causal graphical models.

2.1 Primary objectives

Naturally, the primary objective of confounder selection is to identify a subset of covariates $C \subseteq S$ that suffices to control for confounding. That is, we would like to select a subset $C$ such that every potential outcome is independent of the treatment in every stratum defined by $C$.

**DEFINITION 1.** A set $C$ of variables is said to control for confounding or be a sufficient adjustment set if

$$Y_a \perp A \mid C \quad \text{for } a = 0, 1.$$
The condition (2) is known as weak conditional ignorability (Rosenbaum and Rubin, 1983; Greenland and Robins, 2009) or conditional exchangeability (Hernán and Robins, 2020, §2.2) in the literature. Depending on the problem, there may be no, one, or multiple sufficient adjustment sets.

When a sufficient adjustment set \( C \) exists, under the positivity or overlap assumption that \( 0 < P(A = 1 \mid C) < 1 \) holds with probability one, we can identify the marginal distribution of the potential outcomes by “controlling” or “adjusting” for \( C \) in the following way:

\[
P(Y_a = y) = E\left[P(Y = y \mid A = a, C)\right], \quad a = 0, 1.
\]

This is also known as the back-door formula (Pearl, 1993). Equation (3) assumes the outcome \( Y \) is discrete and can be easily extended to the continuous case. It follows that functionals defined by the distributions of \( Y_a \) for \( a = 0, 1 \) (such as the average treatment effect \( E[Y_1] - E[Y_0] \)) can be identified from the observed distribution of \((A, C, Y)\).

In this article, we will focus on confounder adjustment in the sense given by (3), as this is by far the most popular approach in practice. Before proceeding, we mention some alternative approaches when no such set \( C \) exists. Besides using the back-door formula (3), the causal effect may be identified using the front-door formula (Pearl, 1995) and other graph-based formulae (Tian and Pearl, 2002; Shpitser and Pearl, 2006; Shpitser, Richardson and Robins, 2022). With additional assumptions, causal identification may be possible by using instrumental variables (Imbens, 2014; Wang and Tchetgen Tchetgen, 2017) and proxies of the confounders (Miao, Geng and Tchetgen Tchetgen, 2018; Tchetgen et al., 2020). Note that the last approach requires using a different adjustment formula for proxy confounders; a different strand of literature has investigated the bias-reducing effect of adjusting for proxy confounders via (3) (Greenland, 1980; Ogburn and VanderWeele, 2012; Ogburn and VanderWeele, 2013).

Much of the disagreement and confusion about confounder selection can be attributed to not distinguishing the observable set of pre-treatment covariates \( S \) from the full set of pre-treatment covariates \( Z \), or more broadly speaking, not distinguishing the design of observational studies from the analysis of observational studies. In fact, the common cause and the pre-treatment heuristics both achieve the primary objective if \( S = Z \) (because we assume \( Z \cup \{A, Y\} \) is causally closed); see Appendix B. This may explain why these criteria are often disputed when different parties make different assumptions about \( S \) and \( Z \).

### 2.2 Secondary objectives

Besides controlling for confounding, there are many other objectives for confounder selection. An incomplete list includes

1. dimension reduction for better transparency, interpretability, accuracy or stability (de Luna, Waernbaum and Richardson, 2011; Vansteelandt, Bekaert and Claeskens, 2012; Belloni, Chernozhukov and Hansen, 2013; Greenland, Daniel and Pearce, 2016; Loh and Vansteelandt, 2021),
2. robustness against misspecified modeling assumptions, especially when a posited confounder can be an instrumental variable (Bhattacharya and Vogt, 2007; Wooldridge, 2016; Myers et al., 2011; Ding, VanderWeele and Robins, 2017),
3. statistical efficiency in estimating the causal effect (Kuroki and Miyakawa, 2003; Brookhart et al., 2006; Henckel, Perković and Maathuis, 2022; Rotnitzky and Smucker, 2020; Guo, Perković and Rotnitzky, 2022; Tang et al., 2022), and
4. ethical and economic costs of data collection (Smucker and Rotnitzky, 2022).

Generally speaking, these considerations are not as crucial as the primary objective of controlling for confounding bias. Nonetheless, they are still very important and may decide whether an observational study is successful.

In the following section, we will give a review of various approaches to confounder selection. As these methods are usually developed to achieve or optimize just one objective, practitioners must balance achieving the different objectives for their problem at hand.

### 3. APPROACHES TO CONFOUNDER SELECTION

As suggested by VanderWeele (2019), confounder selection methods can be based on substantive knowledge or statistical analysis. Substantive knowledge of the causal structure is usually represented by a causal directed acyclic graph (DAG) \( G \) over the vertex set \( \{A, Y\} \cup Z \).

A directed edge in the graph such as \( u \rightarrow v \) represents a direct causal effect from \( u \) to \( v \). Let \( \Lambda_G(A) \) be the set of ancestors of \( A \) in \( G \), including \( A \) itself. That is, \( \Lambda_G(A) \) consists of \( A \) and all the variables in \( G \) that have a causal path to \( A \).

Given \( G \), we assume that the distribution of the potential outcomes obeys the single-world intervention graphs derived from \( G \) (Richardson and Robins, 2013), which implies that the distribution of the observed variables

\[ \text{This causal graph } G \text{ can be the latent projection (Verma and Pearl, 1990) of a larger ground causal DAG } \tilde{G} \text{ onto } \{A, Y\} \cup Z. \text{ Because we assume that } \{A, Y\} \cup Z \text{ is causally closed with respect to the underlying } \tilde{G} \text{ (see Appendix B.6), the latent projection } G \text{ does not contain bidirected edges and is hence a DAG.} \]
\{A, Y\} \cup Z \text{ obeys the Bayesian network model represented by } \mathcal{G} \text{ (Pearl, 1988). The conditional independencies imposed by the model can then be read off from } \mathcal{G} \text{ with the d-separation criterion. In the context of confounder selection, a useful concept is the faithfulness assumption, which posits the reverse: conditional independence in the observed distribution implies the corresponding d-separation in } \mathcal{G}. \text{ A short primer on causal graphical models can be found in Appendix B.}

We shall always assume that \(A\) temporally precedes \(Y\), which implies \(Y \notin \text{An}_\mathcal{G}(A)\), since otherwise \(A\) cannot have any causal effect on \(Y\) and the problem is trivial. Recall that \(Z\) consists of observable and unobservable pre-treatment covariates such that \(S \subseteq Z\) and \(Z \cup \{A, Y\}\) is causally closed. Therefore, \(\mathcal{G}\) contains not only the observable variables \(\{A, Y\} \cup S\) but also the unobservable variables \(Z \setminus S\).

Next, we give a review of a number of confounder selection methods, which are organized according to their assumptions on the amount of structural knowledge of \(\mathcal{G}\).

### 3.1 Methods that assume full structural knowledge

With full structural knowledge, achieving the primary objective of confounding control is a solved problem by the well-known back-door criterion due to Pearl (1993, 2009b):

**Proposition 1** (Back-door criterion). A set \(C\) of pre-treatment covariates controls for confounding if \(C\) blocks all the back-door paths from \(A\) to \(Y\) in \(\mathcal{G}\). Furthermore, the reverse is true under the faithfulness assumption.\(^2\)

Here, a path from \(A\) to \(Y\) in \(\mathcal{G}\) is called a back-door path if it starts with an edge into \(A\). Note that given a sufficient adjustment set \(C\), a superset of \(C\) need not continue to block all the back-door paths. This is known as the “collider bias” or “M-bias” (Greenland, Pearl and Robins, 1999b) and an example is given in Fig. 1(a).

For the generalization that allows adjusting for post-treatment variables, see Shpitser, VanderWeele and Robins (2010); for the extension to graphs that describe Markov equivalence classes, see also Perkovic et al. (2017).

When full structural knowledge is available, the graph can also inform secondary objectives of confounder selection. In particular, there is a line of work that selects the adjustment set to minimize the asymptotic variance in estimating the causal effect (Kuroki and Miyakawa, 2003; Henckel, Perkovic and Maathuis, 2022; Witte et al., 2020; Rotnitzky and Smucler, 2020). When all pre-treatment covariates are observed (i.e. \(Z = S\)), there is an optimal choice of \(C\) that minimizes the variance of the causal effect estimator under linear and semiparametric models. This so-called optimal adjustment set consists of parents of the outcome \(Y\) and parents of all mediators between \(A\) and \(Y\), excluding \(A\) and any descendant of a mediator; see Henckel, Perković and Maathuis (2022, §3.4).

### 3.2 Methods that assume partial structural knowledge

Of course, the full graph \(\mathcal{G}\) is rarely known in practice. With just partial knowledge of the causal graph, there exist several methods for confounder selection. It is worthwhile to scrutinize the assumptions that guarantee the validity of these methods.

**Assumption 1.** All relevant pre-treatment covariates are (or can be) measured, i.e., \(S = Z\).

**Assumption 2.** \(S\) is a sufficient adjustment set.

**Assumption 3.** At least one subset of \(S\) is a sufficient adjustment set.

The assumptions are listed from the strongest to the weakest: Assumption 1 implies Assumption 2 (see Proposition B.2 in the appendix), and Assumption 2 trivially implies Assumption 3. Assumption 3 is the weakest possible in the sense that without this assumption, it is impossible to achieve the primary objective of confounding control.

In graphical terms, the conjunctive (or common) cause criterion selects

\[ C_{\text{conj.cause}} \equiv S \cap \text{An}_\mathcal{G}(A) \cap \text{An}_\mathcal{G}(Y), \]

and the pre-treatment criterion selects

\[ C_{\text{pre.treatment}} \equiv S. \]

Although the pre-treatment criterion does not use any structural knowledge other than the temporal order, it is included here to facilitate comparison with other criteria.

**Proposition 2.** Under Assumption 1, \(C_{\text{conj.cause}}\) is a sufficient adjustment set.

**Proposition 3.** Under Assumption 2, \(C_{\text{pre.treatment}}\) is a sufficient adjustment set.

Proposition 3 is just a tautology. Proposition 2 is proved in Appendix B. The assumptions stated in these two propositions are the weakest possible on our list. Indeed, the conjunctive cause can fail under Assumption 2: consider \(S = \{X_2\}\) in Fig. 1(b), which leads to \(C_{\text{conj.cause}} = \emptyset\) and fails to block the back-door path. Also, the pre-treatment criterion may not hold under Assumption 3: for \(S = \{L\}\) in Fig. 1(a), while \(\emptyset \subset S\) is a sufficient adjustment set, \(S\) is not.

---

\(^2\)Here the faithfulness is with respect to the corresponding SWIG; see Appendix B.2.
To address these issues, Guo and Zhao (2023) recently introduced a new procedure to select confounders that does not require pre-specifying the graph or the set $S$. It is based on a reformulation of the back-door criterion (Proposition 1) under latent projection. In fact, this procedure inverts the process of latent projection by iteratively expanding the causal graph as more and more structural knowledge is elicited from the user. This is repeated until a sufficient adjustment set is found or it can be determined that no sufficient adjustment set exists. Compared to the “static” criteria $C_{\text{disj.cause}}$, $C_{\text{conj.cause}}$ and $C_{\text{pre.treatment}}$, this new procedure is able to elicit structural knowledge in an interactive and economical way.

3.3 Methods that assume no structural knowledge

Without any knowledge about the causal structure, it must be assumed that the primary objective is already satisfied by a given set of covariates because the ignorability condition (2) cannot be tested using observational data alone. Without loss of generality, we assume $S$ is a sufficient adjustment set; that is, for this subsection, we shall proceed under Assumption 2.

In this case, data-driven approaches can be useful to achieve some of the secondary objectives, especially if one would like to reduce the dimension of $C$. This is typically accomplished by considering two types of conditional independencies.

**DEFINITION 2** (Treatment/outcome Markov blankets and boundary). For any $V \subseteq S$, the collection of treatment Markov blankets in $V$ is defined as

$$\mathcal{R}_{A}(V) \equiv \{V' : A \perp V \setminus V' | V'\}$$

and the collection of outcome Markov blankets in $V$ is defined as

$$\mathcal{R}_{Y|A}(V) \equiv \{V' : Y \perp V \setminus V' | A, V'\}.$$  

Further, we define the treatment Markov boundary

$$R_{A}(V) \equiv \bigcap_{V' \in \mathcal{R}_{A}(V)} V'$$

and the outcome Markov boundary

$$R_{Y|A}(V) \equiv \bigcap_{V' \in \mathcal{R}_{Y|A}(V)} V'.$$

As a convention, conditional independence always holds with an empty vector, so $V \in \mathcal{R}_{A}(V)$ and $V \in \mathcal{R}_{Y|A}(V)$. For the Markov blankets to have desirable properties, we require the positivity of the distribution over $(A, S)$ in the following sense, which warrants the application of the intersection property of conditional independence; see also Studený (2004, §2.3.5). In below, let $P_A$ be the marginal distribution of $A$ and $P_v$ be the marginal distribution of $v$ for each $v \in S$. 

Fig. 1. (a) M-bias: $U_1, U_2$ are unobserved and $S = \{L\}$. It holds that $A \perp Y(a)$ for $a = 0, 1$ unconditionally. However, controlling for $L$ opens the back-door path $A \leftarrow U_1 \rightarrow L \leftarrow U_2 \rightarrow Y$ and hence would introduce bias. (b) Suppose $S = \{X_1, X_2\}$ and we have $R_{A}(S) = \{X_1\}$, $R_{Y|A}(S) = \{X_2\}$. Using the definitions from Section 3.3, we note that the conjunctive $C_{\cap}(S) = R_{A}(S) \cap R_{Y|A}(S) = \emptyset$ fails to control for confounding. Meanwhile, $C_{Y|A}(S) = \{X_1\}$, $C_{Y|A}(S) = \{X_2\}$ and $C_{\cup}(S) = \{X_1, X_2\}$ all suffice to control for confounding.

Alternatively, one may use the disjunctive cause criterion:

$$C_{\text{disj.cause}} \equiv S \cap [\text{Anc}(A) \cup \text{Anc}(Y)].$$

VanderWeele and Shpitser (2011) proved the following remarkable result regarding this criterion.

**PROPOSITION 4.** Under Assumption 3 and the faithfulness assumption, $C_{\text{disj.cause}}$ is a sufficient adjustment set.

As pointed out by Richardson, Robins and Wang (2018), this result follows almost immediately from a general result on inducing paths; for completeness, a proof is provided in Appendix B.4.

In view of the last three propositions, the disjunctive cause criterion is superior to the pre-treatment criterion: as a subset of $C_{\text{pre.treatment}}$, $C_{\text{disj.cause}}$ is guaranteed to control for confounding under an even weaker assumption. Both the conjunctive and disjunctive cause criteria require substantive knowledge about the causes of $A$ and $Y$, though much less demanding than knowing the full graph. Between the two, the disjunctive cause criterion is usually preferred because it achieves the primary objective under a weaker assumption. That being said, when Assumption 3 fails to hold, using $C_{\text{disj.cause}}$ may lead to a larger bias than $C_{\text{pre.treatment}}$; see Richardson, Robins and Wang (2018, §2.1).

While the aforementioned criteria only require partial structural knowledge, verifying the corresponding assumptions requires much more information and can be very difficult. Further, these criteria are formulated with a pre-specified set of observed variables $S$. Hence they are not suitable for the design of observational studies where one must choose a set of covariates for measurement. This is an important limitation because design is often more consequential than analysis in observational studies (Rubin, 2008).
ASSUMPTION 4 (Positivity). The distribution over \((A, S)\) admits a density that is almost everywhere positive with respect to the product measure \(P_A \prod_{v \in S} P_v\).

**Lemma 1.** Under Assumption 4, \(\mathcal{R}_A(V)\) and \(\mathcal{Y}_{VA}(V)\) are closed under intersection for every \(V \subseteq S\).

Hence, under positivity \(R_A(V)\) and \(R_{Y/A}(V)\) are the minimum of \(\mathcal{R}_A(V)\) and \(\mathcal{Y}_{VA}(V)\), respectively. Moreover, the following result suggests that the order of reduction within \(\mathcal{R}_A(V)\) and \(\mathcal{Y}_{VA}(V)\) is irrelevant.

**Lemma 2 (Reduction).** Under Assumption 4, we have
\[
R_A(V) = R_A(V') \quad R_{Y/A}(V) = R_{Y/A}(V'')
\]
for every \(V' \in \mathcal{R}_A(V)\), \(V'' \in \mathcal{Y}_{VA}(V)\) and \(V \subseteq S\).

Given that \(S\) is a sufficient adjustment set, the following two key lemmas state that every treatment or outcome Markov blanket in \(S\) is also a sufficient adjustment set. The proof of these lemmas applies graphoid properties of conditional independence listed in Appendix A.1 and is deferred to Appendix A.2.

**Lemma 3 (Soundness of \(\mathcal{R}_A\)).** Under Assumption 2, every set in \(\mathcal{R}_A(S)\) controls for confounding.

**Lemma 4 (Soundness of \(\mathcal{Y}_{VA}\)).** Under Assumptions 2 and 4, every set in \(\mathcal{Y}_{VA}(S)\) controls for confounding.

**Remark 1 (Binary treatment).** That \(A\) is binary is necessary for Lemma 4 to hold. When \(A\) has more than two levels, under the same assumptions, for every \(S_{Y/A} \in \mathcal{Y}_{VA}(S)\) one can still show
\[
Y_a \indep \{A = a\} \mid S_{Y/A}
\]
for every level \(a\). This is weaker than \(Y_a \indep A \mid S_{Y/A}\). Nevertheless, equation (6) still ensures the identification of the distribution of \(Y_a\):
\[
\mathbb{P}(Y_a = y) = \mathbb{E} [\mathbb{P}(Y_a = y \mid S_{Y/A})] = \mathbb{E} [\mathbb{P}(Y_a = y \mid A = a, S_{Y/A})] = \mathbb{E} [\mathbb{P}(Y_a = y \mid A = a, S_{Y/A})].
\]
However, (6) is not sufficient to identify \(\mathbb{E}[Y_a \mid A = a']\) for \(a \neq a'\) (required for identifying the treatment effect on the treated) when \(A\) has more than two levels.

We can use \(\mathcal{R}_A\) and \(\mathcal{Y}_{VA}\) to find subsets of \(S\) that control for confounding. There are at least four possible ways to do this:

**Conjunctive** \(C_\cap(S) \equiv R_A(S) \cap R_{Y/A}(S)\),

**Disjunctive** \(C_\cup(S) \equiv R_A(S) \cup R_{Y/A}(S)\),

**AY-sequential** \(C_{AY}(S) \equiv R_{Y/A}(R_A(S))\),

**YA-sequential** \(C_{YA}(S) \equiv R_A(R_{Y/A}(S))\).

More generally, the Markov boundaries \(R_A\) and \(R_{Y/A}\) above can be replaced by any set from the corresponding collection of Markov blankets \(\mathcal{R}_A\) and \(\mathcal{Y}_{VA}\). For example, the YA-sequential rule can take any set from \(\mathcal{R}_A(S_{Y/A})\) for any \(S_{Y/A} \in \mathcal{Y}_{VA}(S)\).

**Example 1.** Consider Figure 1(a) with \(S = \{U_1, U_2, L\}\). We have \(R_A(S) = \{U_1\}, R_{Y/A}(S) = \{U_2\}\) and hence
\[
C_\cap(S) = \emptyset, \quad C_\cup(S) = \{U_1, U_2\}.
\]
Further, we have \(C_{AY}(S) = R_{Y/A}(\{U_1\}) = \emptyset\) and \(C_{YA}(S) = R_A(\{U_2\}) = \emptyset\).

**Example 2.** Consider the case of Figure 1(b) with \(S = \{X_1, X_2\}\). By d-separation, \(A \not\perp \perp X_2 \mid X_1\) and \(Y \not\perp \perp X_1 \mid A, X_2\), so
\[
\mathcal{R}_A(S) = \{\{X_1\}, \{X_1, X_2\}\}, \quad R_A(S) = \{X_1\},
\]
\[
\mathcal{Y}_{VA}(S) = \{\{X_2\}, \{X_1, X_2\}\}, \quad R_{Y/A}(S) = \{X_2\}.
\]
It follows that \(C_\cap(S) = \emptyset, C_\cup(S) = \{X_1, X_2\}, C_{AY}(S) = \{X_1\}\) and \(C_{YA}(S) = \{X_2\}\), among which only \(C_\cap(S)\) fails to control for confounding.

Among these four methods, it is clear from Example 2 that the conjunctive criterion \(C_\cap(S)\) can fail to control for confounding, even when \(S \cup \{A, Y\}\) is causally closed. The validity of the other three criteria directly follows from Lemmas 3 and 4 and applying the weak union property of conditional independence.

**Theorem 1.** Under Assumptions 2 and 4, \(C_{AY}(S)\), \(C_{YA}(S)\) and \(C_\cap(S)\) are sufficient adjustment sets.

For the rest of this section, suppose Assumption 4 (positivity) holds. By applying the soundness of \(\mathcal{R}_A\) and \(\mathcal{Y}_{VA}\) (Lemmas 3 and 4) sequentially, one can show that \(R_{A}(C_{AY}), R_{Y/A}(R_{A}(C_{AY}))\) and so forth are sufficient adjustment sets too. Furthermore, consider alternating between \(R_A\) and \(R_{Y/A}\) and let \(C_{AY}(S)\) and \(C_{YA}(S)\) be, respectively, the limit of
\[
S \xrightarrow{R_A} C_A \xrightarrow{R_{Y/A}} C_{AY} \xrightarrow{R_A} C_{AYA} \xrightarrow{R_{Y/A}} \ldots
\]
and
\[
S \xrightarrow{R_{Y/A}} C_Y \xrightarrow{R_A} C_{YA} \xrightarrow{R_{Y/A}} C_{YAY} \xrightarrow{R_A} \ldots
\]
The iterations terminate in a finite number of steps, when \(|R_A| = 1\) or \(|\mathcal{Y}_{VA}| = 1\). Then \(C_{AY}(S)\) and \(C_{YA}(S)\) are also sufficient adjustment sets and cannot be further reduced using these operations.
LEMMA 5 (Stability). Under Assumption 4, for $C^* \in \{C^*_A(Y), C^*_Y(S)\}$, we have $R_{A}(C^*) = R_{Y|A}(C^*) = C^*$.

EXAMPLE 1 (continued). $C^*_A(Y) = C^*_Y(S) = \emptyset$ in Figure 1(a).

EXAMPLE 2 (continued). $C^*_A(Y) = \{X_1\}, C^*_Y(S) = \{X_2\}$ in Figure 1(b).

In fact, from a graphical perspective, $C^*_A(Y)$ and $C^*_Y(S)$ are minimal sufficient adjustment sets in the sense that no proper subset can control for confounding, or in view of Proposition 1, can block all the back-door paths between $A$ and $Y$.

THEOREM 2 (Minimal sufficient adjustment sets). Suppose Assumptions 2 and 4 hold. The set $C^* \in \{C^*_A(Y), C^*_Y(S)\}$ controls for confounding.

Further, let $\mathcal{G}$ be a causal DAG over variables $Z \cup \{A, Y\}$. For $a = 0, 1$, suppose the distribution of $(A, Y_a, Z)$ is Markov to and faithful to the SWIG $\mathcal{G}(a)$. Then, for $C^* \in \{C^*_A(Y), C^*_Y(S)\}$ there is no proper subset of $C^*$ that also controls for confounding.

The soundness of $C^*_A(Y)$ and $C^*_Y(S)$ does not rely on the assumption of a causal graph; nor is structural knowledge of the graph required for computing $C^*_A(Y)$ and $C^*_Y(S)$. In relation to the results above, see also de Luna, Waernbaum and Richardson (2011, §4.2), who used weak transitivity to show that no single element can be removed from $C^*_A(Y)$ or $C^*_Y(S)$ such that the remaining set continues to control for confounding. Yet, that result is weaker than Theorem 2 as it does not rule out an even smaller subset controlling for confounding. To show Theorem 2, we prove a strengthened version of weak transitivity; see Appendix B.5.

To compute the Markov boundaries, a principled approach can be developed using the following result.

PROPOSITION 5. Under Assumption 4, for $V \subseteq S$, it holds that

$$R_{A}(V) = \{v \in V : A \perp\!\!\!\!\perp v \mid V \setminus \{v\}\},$$

$$R_{Y|A}(V) = \{v \in V : Y \perp\!\!\!\!\perp v \mid A, V \setminus \{v\}\}.$$

See Appendix A.2 for its proof. As an alternative to Proposition 5, by Lemma 2, we can also perform the conditional independence tests sequentially and only use the already reduced set as $V$. This leads to a backward stepwise procedure in Algorithm 1 to compute $R_{A}(V)$ and $R_{Y|A}(V)$; see also VanderWeele and Shpitser (2011, §4).

For practical use, one must employ a parametric or non-parametric test for conditional independence. As a caveat, if the statistical test used has low power to detect conditional dependence (i.e., claiming false conditional independencies), the Markov boundaries selected by the aforementioned methods will be too small and not sufficient to control for confounding. The same issue underlies many other constraint-based structure learning methods as well (Orton and Maathuis, 2017; Strobl, Spirtes and Visweswaran, 2019). For estimating the causal effect with a data-driven set of confounders, the quality of effect estimation depends on the estimator, the set $S$, the statistical procedure for selecting $S$, as well as the underlying data generating mechanism. The reader is referred to Witte and Didelez (2019) for simulation studies on these aspects.

4. DISCUSSION

Confounder selection is a fundamental problem in causal inference, and the backdoor criterion solves this problem when full structural knowledge is available. In practice, however, we almost always have only partial knowledge of the causal structure. This is when different principles and objectives of confounder selection clash the most. More theoretical and methodological development is needed when only partial knowledge of the causal structure is available.

Compared to the widely used conjunctive cause and pre-treatment criteria, the disjunctive cause criterion achieves the primary objective of confounding control under the weakest possible assumption. However, as argued by Richardson, Robins and Wang (2018, §2.1), the appeal of the disjunctive cause criterion diminishes when we consider the secondary objectives that arise in practical situations.

In view of the propositions in Section 3.2, the conjunctive/common cause criterion appears to require the strongest assumption when a set $S$ of observed or potentially observable covariates is given. Nor is taking the conjunction useful in the context of data-driven variable selection (Example 2). Thus, there is little theoretical support for the conclusions in Austin (2011) as described in...
the introduction. However, as mentioned in Section 3.2, this may not be how confounder selection is or should be conceptualized in the design of observational studies. In that process, investigators try to directly come up with potential confounders, usually through arguing about common causes, before reviewing whether or how those confounders can be measured.

Although the conjunctive/common cause heuristic has little value in analyzing observational studies, we believe that they are still indispensable in designing observational studies. This can be seen from the iterative graph expansion procedure in Guo and Zhao (2023), where the common cause heuristic is formalized as identifying the so-called primary adjustment sets that block all immediately confounding paths between two variables. However, Guo and Zhao (2023) do not take this as a one-shot decision; instead, they show that by iterating the common cause heuristic one can obtain all the minimal sufficient adjustment sets.

Although data-driven confounder selection can be used without any structural knowledge, it hinges on the premise that the set of variables under consideration is already a sufficient adjustment set. In any case, substantive knowledge is essential in observational studies as conditional ignorability cannot be empirically verified with observational data alone. Moreover, in general, substantive knowledge cannot be replaced by structure learning methods that learn the causal graph before selecting confounders (Maathuis and Colombo, 2015; Nandy, Maathuis and Richardson, 2017; Perkovic et al., 2017), because (1) the underlying causal graph is typically not identified from data due to Markov equivalence (Frydenberg, 1990; Verma and Pearl, 1990); (2) causal structure learning methods themselves rely on strong assumptions about the causal structure (such as no hidden variables or faithfulness).

Greenland (2007) and Greenland, Daniel and Pearce (2016) challenged the usefulness of data-driven confounder selection (without structural knowledge) and suggested that other modern techniques such as regularization and model averaging are more suitable than variable selection. The same philosophy underlies influence-function-based causal effect estimators such as double machine learning (Chernozhukov et al., 2018), one-step corrected estimation (Kennedy, 2022) and targeted maximum likelihood estimation (van der Laan and Rose, 2011). In view of the secondary objectives in Section 2, this criticism is reasonable if we assume the full set S already controls for confounding, because statistical confounder selection simplifies the final estimator (especially if one uses matching-based methods) at the cost of sacrificing robustness or stability. That being said, statistical variable selection could still be useful in other scenarios by complementing partial structural knowledge.

APPENDIX A: CONDITIONAL INDEPENDENCE AND MARKOV BLANKET

In the following, when a set operation is applied to a random vector, it means the random vector consisting of entries from the set operation applied to the coordinates.

A.1 Graphoid properties of conditional independence

Let W, X, Y, Z be random variables. Conditional independence satisfies the following graphoid properties:

Symmetry \( X \perp Y \mid Z \implies Y \perp X \mid Z \).

Decomposition \( X \perp Y, W \mid Z \implies X \perp Y \mid Z \) and \( X \perp W \mid Z \).

Weak union \( X \perp Y, W \mid Z \implies X \perp Y \mid W, Z \).

Contraction \( X \perp Y \mid Z \) and \( X \perp W, Y \mid Z \implies X \perp W \mid Y, Z \).

Intersection \( X \perp Y \mid W, Z \) and \( X \perp W \mid Y, Z \implies X \perp W, Y \mid Z \).

Among the above, the intersection property additionally requires the positivity of \( P_{W,Y,Z} \) in the sense that \( P_{W,Y,Z} \) admits a density with respect to \( P_W P_Y P_Z \) that is almost everywhere positive; see also Studený (2004, §2.3.5). For properties of conditional independence, see also Pearl (2009b, §1.1.5).

A.2 Markov blanket and boundary

PROOF OF LEMMA 2. We prove \( R_A(V) = R_A(V') \) for every \( V' \in R_A(V) \); the proof for \( R_{Y|A} \) follows similarly. We first show \( R_A(V) \subseteq R_A(V') \). By definition of \( R_A \), it suffices to show \( R_A(V) \supseteq R_A(V') \). Take \( V \in R_A(V') \) so we have

\[ A \perp V' \setminus \bar{V} \mid \bar{V}. \]

Since \( V' \in R_A(V) \), we also have

\[ A \perp \bar{V} \mid V'. \]

It follows from contraction that

\[ A \perp \bar{V} \setminus \bar{V} \mid \bar{V} \]

and hence \( V \in R_A(V) \). Now we show \( R_A(V) \supseteq R_A(V') \). This is true because \( R_A(V) \in R_A(V') \), which follows by weak union. \[ \square \]

PROOF OF LEMMA 3. Fix \( S_A \in R_A(S) \) and \( a \in \{0,1\} \). By contraction, \( A \perp Y_a \mid S \) and \( A \perp S \setminus S_A \mid S_A \) together imply \( A \perp Y_a, S \setminus S_A \mid S_A \), which further gives \( A \perp Y_a \mid S_A \) by decomposition. \[ \square \]

PROOF OF LEMMA 4. Fix \( S_{Y|A} \in R_{Y|A}(S) \) and \( a \in \{0,1\} \). That \( S \) controls for confounding implies

\[ Y_a \perp \{A = a\} \mid \text{in} \setminus S_Y | A, S_{Y|A}. \]
By definition of $\mathcal{R}_{Y|A}$, we have $Y \perp S \setminus S_{Y|A} \mid A = a, S_{Y|A}$, which by consistency becomes

$$Y_a \perp S \setminus S_{Y|A} \mid A = a, S_{Y|A}.$$  

Under positivity, by applying the intersection property to the previous two displays, we have

$$Y_a \perp \{A = a\} \setminus S \setminus S_{Y|A} \mid S_{Y|A},$$

which by decomposition further implies $Y_a \perp \{A = a\} \mid \cup S_{Y|A}$, which is equivalent to $Y_a \perp A \mid S_{Y|A}$ since $A$ is binary.

**Proof of Theorem 1.** To see $C_{\cup}(S)$ controls for confounding, note $R_{A}(S) \cup R_{Y|A}(S) \in \mathcal{R}_{A}(S)$ by weak union and then apply Lemma 3. That $C_{\cap}(S)$ and $C_{\cap}(S)$ control for confounding under positivity follows from sequentially applying Lemmas 3 and 4.

**Proof of Lemma 5.** Markov boundaries $R_{A}(\cdot)$ and $R_{Y|A}(\cdot)$ are well-defined under Assumption 4. By the termination condition, either $R_{A}(C) = C^*$ or $R_{Y|A}(C) = C^*$ holds already. First, suppose $R_{A}(C) = C^*$ holds. We prove $R_{Y|A}(C) = C^*$ by contradiction. Since $R_{Y|A}(C) \subseteq C^*$, suppose $R_{Y|A}(C) = C^* \subseteq C^*$. By the last iteration, $C^* = R_{Y|A}(\bar{C})$ for some $\bar{C} \supseteq C^*$. Writing $C^* = C^* \cup (C^* \setminus C')$, we have

$$Y \perp C^* \setminus C' \mid A, C'$$

and

$$Y \perp \bar{C} \setminus C^* \mid A, C', C^* \setminus C'.$$

Applying contraction, we have

$$Y \perp \bar{C} \setminus C' \mid A, C',$$

which contradicts $C^* = R_{Y|A}(\bar{C})$ and Eq. (5) because $C'$ is a proper subset of $C^*$. The other case follows similarly.

**Proof of Lemma 1.** It directly follows from the next lemma and the fact that the positivity of $(A, S)$ implies the positivity of $S$.

**Lemma A.1.** Let $X, Z$ be two random variables and let $S$ be a finite-dimensional random vector. Define

$$\mathcal{R}_{X|Z}(S) \equiv \{S' \subseteq S : X \perp S \setminus S' \mid S', Z\}.$$

Suppose the distribution of $(Z, S)$ is positive in the sense that $P_{Z,S}$ admits a density with respect to $P_{Z} \prod_{v \in S} P_{v}$ that is almost everywhere positive. It holds that $\mathcal{R}_{X|Z}(S)$ is closed under intersection.

**Proof.** Take $S_1, S_2 \in \mathcal{R}_{X|Z}(S)$. By definition,

$$X \perp S \mid S_1 \mid X, Z,$$

$$X \perp S \mid S_2 \mid X, Z$$

which can be rewritten as

$$X \perp S \setminus S_1 \mid S \setminus (S_1 \cup S_2) \mid S_1 \cap S_2, S \setminus S_1, Z,$$

$$X \perp S \setminus S_2 \mid S \setminus (S_1 \cup S_2) \mid S_1 \cap S_2, S \setminus S_2, S \setminus S_1, Z.$$

Further by decomposition,

$$X \perp S \setminus S_1 \mid S \setminus (S_1 \setminus S_2) \setminus S_2 \setminus S_1, Z,$$

Applying intersection under positivity, we have

$$X \perp S \setminus S_1 \mid S_1 \setminus S_2, S_2 \setminus S_1, S \setminus S_1 \cup S_2, Z,$$

which by decomposition implies

$$X \perp S \setminus (S_1 \setminus S_2) \setminus S_2 \setminus S_1, Z.$$

Meanwhile, note that (7) can be rewritten as

$$X \perp S \setminus S_1 \mid S \setminus (S_1 \setminus S_2) \setminus S_2 \setminus S_1, Z.$$  

Applying contraction to the two previous displays, we get

$$X \perp S \setminus S_1 \setminus S_2 \setminus S_1 \setminus S_2, Z,$$

i.e.,

$$X \perp S \setminus (S_1 \cup S_2) \setminus S_1 \setminus S_2, Z.$$  

Hence, $S_1 \cap S_2 \in \mathcal{R}_{X|Z}(S)$.}

**Proof of Proposition 5.** We show $R_{A}(V) = \Gamma_{A}(V)$, where $\Gamma_{A}(V) \equiv \{v \in V : A \not\perp v \mid V \setminus \{v\}\}$. First, we show $\Gamma_{A}(V) \subseteq R_{A}(V)$. Fix $v \in \Gamma_{A}(V)$. Suppose $v \notin R_{A}(V)$. We can then rewrite $A \perp V \setminus R_{A}(V) \mid R_{A}(V)$ as

$$A \perp v, V \setminus (R_{A}(V) \cup \{v\}) \mid R_{A}(V).$$

By weak union, it follows that $A \perp v \mid V \setminus \{v\}$, contradicting $v \in \Gamma_{A}(V)$. Now we show $R_{A}(V) \subseteq \Gamma_{A}(V)$. Fix $v \in R_{A}(V)$. By definition of $R_{A}(V)$, we have

$$A \perp v \mid R_{A}(V) \setminus R_{A}(V) \mid \{v, v\}.$$

Suppose $v \notin \Gamma_{A}(V)$, i.e., $A \not\perp v \mid V \setminus \{v\}$, which can be rewritten as

$$A \not\perp v \mid R_{A}(V) \setminus \{v\}, V \setminus R_{A}(V).$$

Note that Assumption 4 implies the distribution over $A$ and $V$ is also positive. Applying intersection to the previous two displays, we get

$$A \not\perp v, R_{A}(V) \setminus \{v\}, V \setminus R_{A}(V),$$

which contradicts the minimality of $R_{A}(V)$ in $\mathcal{R}_{A}(V)$ in Eq. (4).

By $P(A = a, Y, V) = P(A = a)P(Y, V \mid A = a) = P(A = a)P(Y, V \mid A = a)$ for $a = 0, 1$, Assumption 4 implies that the distribution over $A, Y, V$ is also positive. The proof for the equality on $R_{Y|A}(V)$ follows similarly.
APPENDIX B: GRAPHICAL RESULTS

B.1 DAG and d-separation

We use standard graphical terminology; see also, e.g., Pearl (2009b, §1.2). In particular, for two vertices \( u \) and \( v \), we say \( u \) is an ancestor of \( v \), or equivalently \( v \) is a descendant of \( u \), if either \( u = v \) or there is a causal path \( u \rightarrow \cdots \rightarrow v \). The set of ancestors of vertex \( u \) in graph \( G \) is denoted as \( \text{An}_G(u) \). Similarly, the set of descendants is denoted as \( \text{De}_G(u) \). By definition, \( u \in \text{An}_G(u) \) and \( u \in \text{De}_G(u) \). We use symbol \( \text{Nd}_G(u) \) for non-descendants of \( u \), i.e., the complement of \( \text{De}_G(u) \). The definitions of relational sets extend disjunctively to a set of vertices, e.g.,

\[
\text{An}_G(L) \equiv \bigcup_{v \in L} \text{An}_G(v), \quad \text{De}_G(L) \equiv \bigcup_{v \in L} \text{De}_G(v).
\]

For two vertices \( u \) and \( v \), a path \( \pi \) between them consists of a sequence of distinct vertices such that consecutive vertices are adjacent in the graph. For vertices \( w, z \) also on path \( \pi \), we use notation \( \pi(w, z) \) for the subpath between \( w \) and \( z \). A non-endpoint vertex \( k \) is called a collider on the path if it is of the form \( \cdots \rightarrow k \leftarrow \cdots \); otherwise \( k \) is called a non-collider.

**Definition B.1** (d-connecting path). A path \( p \) between \( u \) and \( v \) is called d-connecting given a set of vertices \( L \) \( (u, v \notin L) \) if (1) every non-collider on \( p \) is excluded from \( L \) and (2) every collider on \( p \) is in \( L \) or is an ancestor of some vertex in \( L \).

When a path \( p \) is not d-connecting given \( L \), we also say \( L \) blocks the path \( p \).

**Definition B.2** (d-separation). If there is no d-connecting path between \( u \) and \( v \) given \( L \) \( (u, v \notin L) \), we say that \( u \) and \( v \) are d-separated given \( L \). Similarly, for disjoint vertex sets \( U, V, L \), we say that \( U \) and \( V \) are d-separated given \( L \), if there is no d-connecting between \( u \) and \( v \) given \( L \) for \( u \in U \), \( v \in V \).

We use symbol \( u \perp_G v \mid L \) to denote that \( u \) and \( v \) are d-separated by \( L \) in graph \( G \). A similar notation applies to the d-separation between sets. It holds that d-separation shares the graphoid properties of conditional independence listed in Appendix A.1, where symbol \( \perp \) is replaced with \( \perp_G \). In addition, d-separation satisfies certain properties that are in general not obeyed by abstract conditional independence, including the following (Pearl, 1988, Theorem 11), where \( W, X, Y, Z \) are disjoint sets of vertices of a directed acyclic graph \( G \).

**Composition** \( X \perp_G Y \mid Z \) and \( X \perp_G W \mid Z \) \( \implies \) \( X \perp_G W \perp_Y Z \).

**Weak transitivity** \( X \perp_G Y \mid Z \) and \( X \perp_G Y \mid Z \), \( s \) for vertex \( s \) \( \implies \) either \( s \perp_G X \mid Z \) or \( s \perp_G Y \mid Z \).

We strengthen weak transitivity in Theorem B.1.

DAG \( G \) defines the Bayesian network model over the variables \( V \) in the graph. We say a distribution \( P \) follows the Bayesian network model, or \( P \) is Markov to \( G \), if \( P \) factorizes according to the graph:

\[
p(V) = \prod_v p(v \mid \text{Pa}_G(v)),
\]

where \( p \) is the density of \( P \) with respect to some product dominating measure. It can be shown that \( P \) is Markov to \( G \) if and only if \( P \) obeys the global Markov property implied by \( G \): for disjoint subsets \( X, Y, Z \) of \( V \),

\[
X \perp_G Y \mid Z \implies X \perp Y \mid Z \quad \text{under } P.
\]

See, e.g., Lauritzen (1996, Theorem 3.27). If the reverse holds, i.e., for disjoint subsets \( X, Y, Z \),

\[
X \perp Y \mid Z \quad \text{under } P \implies X \perp_G Y \mid Z,
\]

we say \( P \) is faithful to \( G \).

B.2 Causal model and SWIGs

A causal DAG \( G \) can be associated with different assumptions on the distribution of factual and counterfactual random variables, depending on how we interpret graph \( G \) (Robins and Richardson, 2011). Most notably, Robins (1986) introduced the “finest fully randomized causally interpretable structured tree graph” (FFRISTG) model, while Pearl (1995) interpreted \( G \) as positing a non-parametric structural equation model with independent errors (NPSEM-IE). The conditional independencies implied by the FFRISTG model can be read off from the corresponding single-world intervention graph (SWIG) using d-separation (Richardson and Robins, 2013). For the same graph \( G \), the NPSEM-IE model is a submodel of the FFRISTG model, as the former additionally posits cross-world independencies (Richardson and Robins, 2013; Shpitser, Richardson and Robins, 2022).

B.3 Conjunctive cause criterion

We prove \( A \perp Y(a) \mid C_{\text{conj.cause}} \) holds under Assumption 1 for the FFRISTG potential outcome model (and hence the stronger NPSEM-IE model) represented by graph \( G \).

**Proof of Proposition 2.** Let \( G(a) \) be the SWIG corresponding to intervening on \( A \) and imposing value \( a \). Graph \( G(a) \) is formed from \( G \) by splitting \( A \) into a random part \( A \) and a fixed part \( a \), where \( A \) inherits all the edges into \( A \) and \( a \) inherits all the edges out of \( A \). Additionally, any descendant \( V_i \) of \( A \) is labelled as \( V_i(a) \) in \( G(a) \). In particular, \( Y \) is labelled as \( Y(a) \) in \( G(a) \). To prove our result, it suffices to show that \( A \) and \( Y(a) \) are d-separated in \( G(a) \) given \( C_{\text{conj.cause}} \).
Under $Z = S$, by the fact that $G$ is a DAG over $Z \cup \{A, Y\}$, we have $C_{\text{conj.cause}} = \text{An}_G(A) \cap \text{An}_G(Y) \setminus \{A\}$. Suppose there is a path $p$ in $G(a)$ that $d$-connects $A$ and $Y(a)$ given $C_{\text{conj.cause}}$. First, observe that this is impossible if $p$ does not contain any collider, since by construction of $G(a)$ (no edge stems out of $A$) and $Y \notin \text{An}_G(A)$, $p$ must be of the form $A \leftarrow \cdots \rightarrow Y(a)$ and is thus blocked by $C_{\text{conj.cause}}$. Hence, $p$ must contain at least one collider. Further, again by construction of $G(a)$, $p$ must also contain non-colliders. Let $\gamma$ be the collider on $p$ that is closest to $A$. Also let $\delta$ be the vertex that precedes $\gamma$ on the subpath $p(A, \gamma)$. Vertex $\delta \neq A$ is a non-collider on $p$. For $p$ to $d$-connect, $\gamma$ is an ancestor of some $v \in C_{\text{conj.cause}}$. However, this implies that $\delta \in C_{\text{conj.cause}}$ and hence $p$ is blocked. Therefore, such a $d$-connecting path $p$ cannot exist.

**B.4 Disjunctive cause criterion**

The following proof of Proposition 4 is due to Richardson, Robins and Wang (2018), which is based on the following result on inducing paths.

**Definition B.3.** Consider vertices $u, v$ and a vertex set $L \subset V \setminus \{u, v\}$. A path between $u$ and $v$ is called an inducing path relative to $L$ if every non-endpoint vertex on the path that is not in $L$ is (1) a collider and (2) an ancestor of $u$ or $v$.

**Lemma B.1 (Verma and Pearl (1990)).** Let $G$ be a DAG over vertices $V$. Fix two vertices $u, v$ and a set $L \subset V$ such that $u, v \notin L$. Then $u$ and $v$ cannot be $d$-separated by any subset of $V \setminus \{L \cup \{u, v\}\}$ if and only if there exists an inducing path between $u$ and $v$ relative to $L$ in $G$.

**Proof of Proposition 4.** Let DAG $G$ over vertices $V$ be the underlying causal graph. We prove the statement by contradiction. Suppose $C_{\text{disj.cause}}$ is not a sufficient adjustment set. By Proposition 1, there exists some $d$-connecting backdoor path $\pi$ between $A$ and $Y$ given $S \cap (\text{An}_G(A) \cup \text{An}_G(Y))$. Remove edges out of $A$ from $G$ and call the resulting graph $\tilde{G}$. Observe that $\text{An}_{\tilde{G}}(A) \cup \text{An}_{\tilde{G}}(Y) = \text{An}_G(A) \cup \text{An}_G(Y)$, and the paths between $A$ and $Y$ in $\tilde{G}$ exactly correspond to the back-door paths between $A$ and $Y$ in $G$. Moreover, the path $\pi$ $d$-connects $A$ and $Y$ in $\tilde{G}$ given $S \cap (\text{An}_{\tilde{G}}(A) \cup \text{An}_{\tilde{G}}(Y))$.

We claim that $\pi$ is an inducing path between $A$ and $Y$ in $\tilde{G}$ relative to $L \equiv V \setminus (S \cup \{A, Y\})$. To see this, let $k$ be any non-endpoint vertex on $\pi$. Suppose $k$ is a non-collider. Because $\pi$ is $d$-connected given $S \cap (\text{An}_{\tilde{G}}(A) \cup \text{An}_{\tilde{G}}(Y))$, it follows from Lemma B.2 that $k$ must be either an ancestor of $A$, an ancestor of $Y$, or an ancestor of $S \cap (\text{An}_{\tilde{G}}(A) \cup \text{An}_{\tilde{G}}(Y))$. However, in any case, $k$ is included in the conditioning set and $\pi$ would be blocked. Hence, $k$ is a collider. Further, by the $d$-connection of $\pi$, $k$ must be an ancestor of $S \cap (\text{An}_{\tilde{G}}(A) \cup \text{An}_{\tilde{G}}(Y))$, which implies $k \in \text{An}_{\tilde{G}}(A) \cup \text{An}_{\tilde{G}}(Y)$. Hence, $\pi$ is an inducing path between $A$ and $Y$ relative to $L$. By Lemma B.1, $A$ and $Y$ cannot be $d$-separated in $\tilde{G}$ by any subset of $S$. This contradicts the existence of a sufficient adjustment set postulated by Assumption 3, which by Proposition 1 is equivalent to the existence of a subset $S$ blocking all back-door paths from $A$ to $Y$ under the faithfulness assumption.

**B.5 Minimality of $C_{AV}^*$ and $C_{YA}^*$**

We first strengthen the weak transitivity property of $d$-separation (cf Pearl, 1988, Theorem 12).

**Theorem B.1 (Weak transitivity, strengthened).** Let $G$ be a DAG. Let $x, y$ be two vertices and $W, Z$ be two disjoint vertex sets such that $x, y \notin W \cup Z$. Suppose $x \perp_G y \mid W$ and $x \perp_G y \mid W, Z$. Then there exists $z \in Z$ such that either $z \perp_G x \mid W$ or $z \perp_G y \mid W$ holds.

To prove Theorem B.1, we need the following two lemmas on $d$-connecting paths.

**Lemma B.2.** Let $\pi$ be a $d$-connecting path between $x$ and $y$ given $W$. Then, every vertex on $\pi$ is in $\text{An}(\{x, y\} \cup W)$. In consequence, if $z$ on the path is not an ancestor of $W$, then either $\pi(z, x)$ or $\pi(z, y)$ is a causal path from $z$ to the endpoint.

**Proof.** It follows from the fact that every vertex on a $d$-connecting path is an ancestor of either endpoint or a collider.

**Lemma B.3.** Let $G$ be a DAG. Suppose path $\pi_1$ $d$-connects $x$ and $z$ given $W$ and path $\pi_2$ $d$-connects $y$ and $z$ given $W$. Suppose $z \in \text{An}_G(W)$. Then $x$ and $y$ are $d$-connected given $W$.

**Proof.** Let $t$ be the first vertex after $x$ on $\pi_1$ that is also on $\pi_2$. Let $\pi^*$ be the path formed by concatenating $\pi_1(x, t)$ and $\pi_2(t, y)$ (one can check that there are no duplicating vertices in $\pi^*$). Observe that every non-endpoint vertex on $\pi^*$ has the same collider status as the same vertex on $\pi_1$ or $\pi_2$, with possible exception of $t$. Thus, it suffices to show that $\pi^*$ is not blocked by $t$.

Suppose $t \neq y$, otherwise $\pi^*$ is obviously $d$-connected. There are two scenarios:

(i) The vertex $t$ is a collider on $\pi^*$; it suffices to show $t \in \text{An}_G(W)$. This is immediately true if $t = z$. Otherwise, suppose $t \neq z$ and $t \notin \text{An}_G(W)$. Then by Lemma B.2, that $\pi(x, t)$ terminates with an arrow into $t$ implies that $\pi_1(t, z)$ must be of the form $t \rightarrow \cdots \rightarrow z$, which gives $t \in \text{An}_G(W)$ and hence a contradiction.
(ii) The vertex $t$ is a non-collider on $\pi^*$. It suffices to show $t \not\in W$. This is immediately true if $t \neq z$. Otherwise, $t$ must be a non-collider on either $\pi_1$ or $\pi_2$ (in order for $t$ to be a non-collider on the concatenated path $\pi^*$). The d-connectedness of $\pi_1$ and $\pi_2$ also implies $t \not\in W$.

**Proof of Theorem B.1.** We prove the statement by contradiction. Suppose for every $z \in Z$, there exist path $p_z$ that d-connects $x$ and $z$ given $W$ and path $q_z$ that d-connects $y$ and $z$ given $W$. Observe that $p_z$ cannot be a causal path from $z$ to $x$ and $q_z$ cannot be a causal path from $z$ to $y$, since otherwise by concatenating the two paths at the first vertex they intersect (which is a non-collider), $x$ and $y$ are d-connected given $W$, which contradicts our assumption.

We claim that $Z \cap An_G(W) = \emptyset$. Otherwise, suppose $z' \in Z$ is an ancestor of $W$. Then by Lemma B.3 and the existence of paths $p_{z'}, q_{z'}$, vertices $x$ and $y$ are d-connected given $W$, which contradicts our assumption.

Choose $z^0$ from $Z$ such that no other element of $Z$ is an ancestor of $z^0$. Consider path $p_{z^0}$ between $x, z^0$ and path $q_{z^0}$ between $y, z^0$. Both paths are d-connecting given $W$. Let $t$ be first vertex after $x$ on $p_{z^0}$ that is also on $q_{z^0}$, with $t = z^0$ as a special case. Let $\pi$ be the path formed by concatenating $p_{z^0}(x, t)$ and $q_{z^0}(t, y)$. There are two cases.

(i) Vertex $t$ is a collider on $\pi$. By $x \perp_G y \mid W$ and Lemma B.3, $t$ is not an ancestor of $W$. Then by Lemma B.2, $p_{z^0}(t, z^0)$ must be of the form $t \rightarrow \cdots \rightarrow z^0$. We claim that $\pi$ between $x$ and $y$ is d-connecting given $Z$ and $W$, which would contradict $x \perp_G y \mid Z, W$. To see this, let us inspect every non-endpoint vertex $v$ on $\pi$ and verify the condition on $v$ for $\pi$ to d-connect given $W, Z$. If $v = t$, then $v$ is a collider and $v$ is an ancestor of $Z$ and hence of $W \cup Z$.

Now suppose $v \neq t$. The status (collider or non-collider) of $v$ is the same as its status on $p_{z^0}$ or $q_{z^0}$. We show $v$ does not block $\pi$. If $v$ is a collider, then it is an ancestor of $W$ and hence of $W \cup Z$. Otherwise, we know $v \not\in W$ and it suffices to show $v \not\in Z$. For a contradiction, suppose $v \in Z$ is a non-collider on $\pi$. As observed in the beginning of the proof, $\pi(v, x)$ and $\pi(v, y)$ cannot be causal paths from $v$ to the other endpoint. It follows that on the subpath $\pi(x, t)$ or $\pi(y, t)$ that contains $v$, vertex $v$ must be an ancestor of a collider (and hence of $W$) or an ancestor of $t$ (and hence of $z^0$), neither of which is possible: we showed $Z \cap An_G(W) = \emptyset$ and we choose $z^0$ such that no other vertex in $Z$ is an ancestor of $z^0$. Hence, $\pi$ d-connects $x$ and $y$ given $W$, which contradicts our assumption $x \perp_G y \mid Z, W$.

(ii) Vertex $t$ is a non-collider on $\pi$. Then $t$ is a non-collider on either $p_{z^0}$ or $q_{z^0}$, which implies $t \not\in W$. Because the status of any other non-endpoint vertex on $\pi$ remains the same as on $p_{z^0}$ or $q_{z^0}$, we know $\pi$ d-connects $x$ and $y$ given $W$, which contradicts our assumption $x \perp_G y \mid W$.

We are ready to prove that $C^*_A(Y) = C^*_Y(A)$ are minimal sufficient adjustment sets.

**Proof of Theorem 2.** That $C^*$ is a sufficient adjustment set follows from iteratively applying Lemmas 3 and 4 under Assumptions 2 and 4.

Now under the additional assumption of a causal graph $G$ and the faithfulness with respect to SWIG $G(a)$, we show no proper subset of $C^*$ is a sufficient adjustment set. Suppose $C' \subset C^*$ controls for confounding. By faithfulness, we have

\begin{equation}
A \perp_{G(a)} Y_a \mid C'
\end{equation}

and

\begin{equation}
A \perp_{G(a)} Y_a \mid C^*.
\end{equation}

By Theorem B.1, we know there exists $s \in (C^* \setminus C')$ such that either (or both) of the following holds.

(i) $A \perp_{G(a)} s \mid C'$. Given also Eq. (9), by composition of d-separation (see Appendix B.1), we have $A \perp_{G(a)} s, Y_a \mid C'$. By weak union, it follows that $C'' = C' \cup \{s\}$ also controls for confounding.

(ii) $Y_a \perp_{G(a)} s \mid C'$. Given also Eq. (9), by composition of d-separation, we have $Y_a \perp_{G(a)} s, A \mid C'$. By weak union, it follows that $C'' = C' \cup \{s\}$ also controls for confounding.

In either case, we see that $C'$ can be replaced by a set $C''$ whose size is increased by one. Iterating this argument until we get $\tilde{C} = C^* \setminus \{\tilde{s}\}$ for some element $\tilde{s} \in C^*$. We have

\begin{equation}
A \perp_{G(a)} Y_a \mid \tilde{C}.
\end{equation}

Applying Theorem B.1 again to Eqs. (10) and (11), at least one of the following holds.

(i) $A \perp_{G(a)} C^* \setminus \tilde{C} \mid \tilde{C}$. By Lemma 2, this means the treatment Markov boundary of $C^*$ is contained in $\tilde{C}$, contradicting Lemma 5.

(ii) $Y_a \perp_{G(a)} C^* \setminus \tilde{C} \mid \tilde{C}$. Combining it with Eq. (11) and applying composition, we have $Y_a \perp_{G(a)} A, C^* \setminus \tilde{C} \mid \tilde{C}$, which by weak union implies

\begin{equation}
Y_a \perp C^* \setminus \tilde{C} \mid A = a, \tilde{C}.
\end{equation}
Using consistency, we have

\[ Y \perp C^* \setminus \overline{C} \mid A = a, \overline{C}, \quad a = 0, 1. \]

By Lemma 2, this means the outcome Markov boundary of \( C^* \) is contained in \( \overline{C} \), which again contradicts Lemma 5.

**B.6 Causal closure**

In this subsection, we discuss the notion of causal closure with respect to a fixed ground DAG \( \mathcal{G} \), which is the underlying causal DAG that includes every variable in the system. For two distinct vertices \( v_1, v_2 \) on \( \mathcal{G} \), we define their non-trivial common ancestors to be

\[ \text{An}_{\mathcal{G}}^*(v_1, v_2) \equiv \{ u \in \text{An}_{\mathcal{G}}(v_1) \cap \text{An}_{\mathcal{G}}(v_2) : u \text{ has a causal path to } v_1 \text{ or } v_2 \text{ not through } \text{An}_{\mathcal{G}}(v_1) \cap \text{An}_{\mathcal{G}}(v_2) \setminus \{u\} \}. \]

For example, if \( \mathcal{G} \) is \( Z \to X \to Y \), then \( Z \) is a common ancestor of \( (X, Y) \) but not a non-trivial common ancestor: \( Z \) causally goes to \( X \) and \( Y \) only through \( \text{An}(X) \cap \text{An}(Y) \setminus \{Z\} = \{X\} \).

**Definition B.4 (causal closure).** A vertex set \( H \) is causally closed if

\[ v_1, v_2 \in H, v_1 \neq v_2 \implies \text{An}_{\mathcal{G}}^*(v_1, v_2) \subseteq H. \]

Further, the causal closure of \( H \) is

\[ \mathcal{T} \equiv \bigcap \{H' \supseteq H : H' \text{ is causally closed}\}. \]

Observe that a causal closure is always causally closed. Causal closure is defined with respect to those non-trivial common ancestors so pre-treatment variables irrelevant to the effect need not be included. In the example of Fig. B.1, the causal closure of \( \{S_1, S_2, A, Y\} \) is \( \{Z_1, Z_3, S_1, S_2, A, Y\} \).

**Lemma B.4.** For \( u \in \mathcal{T} \), either \( u \in H \) or \( u \) is a common ancestor of two distinct vertices in \( H \).

**Proof.** We prove the statement by contradiction. Suppose \( u \in \mathcal{T} \) and \( \text{De}(u) \cap H \) is empty or a singleton. Consider \( H' = \mathcal{T} \setminus (\text{De}(u) \setminus H) \). By construction, \( H \subseteq H' \subset \mathcal{T} \). Further, observe that \( H' \) is causally closed. However, this contradicts the definition of \( \mathcal{T} \).

**Proposition B.1.** The set \( Z \) given by Eq. (1) is pretreatment.

**Proof.** Eq. (1) states that

\[ Z = S \cup \{A, Y\} \setminus \{A, Y\}. \]

Because \( S \) is pre-treatment, we shall show every \( z \in Z \setminus S \) is pre-treatment. By Lemma B.4, \( z \) is a common ancestor to two distinct vertices \( v_1, v_2 \in S \cup \{A, Y\} \). It follows that either \( v_1 \) or \( v_2 \) is in \( S \cup \{A\} \). It is clear that \( z \) is an ancestor of \( S \cup \{A\} \) and hence pre-treatment.

**Proposition B.2.** Suppose \( \mathcal{G} \) is a DAG in which \( A \in \text{An}_{\mathcal{G}}(Y) \). Let \( Z \) be a vertex set that contains no descendant of \( A \). If \( Z \cup \{A, Y\} \) is causally closed, then \( Z \) is a sufficient adjustment set.

**Proof.** We prove by contradiction. Suppose \( Z \) is not a sufficient adjustment set. Then, by Proposition 1, there exists a back-door path \( \pi \) between \( A \) and \( Y \) that is d-connected given \( Z \). There are two cases. If \( \pi \) contains no collider, then one can show that there exists a non-collider vertex \( L \) on \( \pi \) such that \( L \in \text{An}_{\mathcal{G}}^*(A, Y) \); otherwise, one can show that there exists a non-collider vertex \( L \) on \( \pi \) such that \( L \in \text{An}_{\mathcal{G}}^*(A, z) \cup \text{An}_{\mathcal{G}}^*(z, Y) \) for some \( z \in Z \). In either case, because \( \pi \) d-connects \( A \) and \( Y \) given \( Z \), it holds that \( L \notin Z \cup \{A, Y\} \). However, this contradicts our assumption that \( Z \cup \{A, Y\} \) is causally closed.

**References**

Austin, P. C. (2011). An introduction to propensity score methods for reducing the effects of confounding in observational studies. *Multivariate Behavioral Research* 46 399-424.

Austin, P. C., Grootendorst, P. and Anderson, G. M. (2007). A comparison of the ability of different propensity score models to balance measured variables between treated and untreated subjects: a Monte Carlo study. *Statistics in Medicine* 26 734-753. https://doi.org/10.1002/sim.2580

Belloni, A., Chernozhukov, V. and Hansen, C. (2013). Inference on treatment effects after selection among high-dimensional controls. *The Review of Economic Studies* 81 608-650. https://doi.org/10.1093/restud/rdt044

Bhattacharya, J. and Vogt, W. B. (2007). Do instrumental variables belong in propensity score models? Working Paper No. 343, National Bureau of Economic Research. https://doi.org/10.3386/w10343

Brookhart, M. A., Schneeweiss, S., Rothman, K. J., Glynn, R. J., Avorn, J. and Stürmer, T. (2006). Variable selection for propensity score models. *American Journal of Epidemiology* 163 1149-1156. https://doi.org/10.1093/aje/kwj149
Chernozhukov, V., Chetverikov, D., Demirer, M., Duflo, E., Hansen, C., Newey, W. and Robins, J. (2018). Double/debiased machine learning for treatment and structural parameters. The Econometrics Journal 21 C1-C68. https://doi.org/10.1111/ectj.12097

De Luna, X., Wainwright, I. and Richardson, T. S. (2011). Covariate selection for the nonparametric estimation of an average treatment effect. Biometrika 98 861–875.

Ding, P., VanderWeele, T. J. and Robins, J. M. (2017). Instrumental variables as bias amplifiers with general outcome and confounding. Biometrika 104 291-302. https://doi.org/10.1093/biomet/ass009

Drton, M. and Maathuis, M. H. (2017). Structure learning in graphical modeling. Annual Review of Statistics and Its Application 4 365–393.

Ertefaie, A., Asgharian, M. and Stephens, D. A. (2017). Variable selection in causal inference using a simultaneous penalization method. Journal of Causal Inference 6 nil. https://doi.org/10.1515/jci-2017-0010

Frydenberg, M. (1990). The chain graph Markov property. Scandinavian Journal of Statistics 333–353.

Glymour, M. M., Ruwe, J. and Chen, J. T. (2008). Methodological challenges in causal research on racial and ethnic patterns of cognitive trajectories: measurement, selection, and bias. Neuropsychology Review 18 194-213. https://doi.org/10.1007/s11065-008-9096-x

Greenland, S. (1980). The effect of missclassification in the presence of covariates. American Journal of Epidemiology 112 564-569. https://doi.org/10.1093/oxfordjournals.aje.a113025

Greenland, S. (2007). Invited commentary: variable selection versus shrinkage in the control of multiple confounders. American Journal of Epidemiology 167 523-529. https://doi.org/10.1093/aje/kwm355

Greenland, S., Daniel, R. and Pearl, N. (2016). Outcome modelling strategies in epidemiology: traditional methods and basic alternatives. International Journal of Epidemiology 45 565-575.

Greenland, S., Pearl, J. and Robins, J. M. (1999a). Confounding and collapsibility in causal inference. Statistical Science 14 29-46. https://doi.org/10.1214/ss/1009228216

Greenland, S., Pearl, J. and Robins, J. M. (1999b). Causal diagrams for epidemiologic research. Epidemiology 10 37–48.

Greenland, S. and Robins, J. M. (2009). Identifiability, exchangeability and confounding revisited. Epidemiologic Perspectives & Innovations 6 1–9.

Guo, F. R., Perkovic, E. and Rotnitzky, A. (2022). Variable elimination, graph reduction and the efficient g-formula. Biometrika 110 739-761. https://doi.org/10.1093/biomet/asac062

Guo, F. R. and Zhao, Q. (2023). Confounder selection via iterative graph expansion. arXiv preprint arXiv:2309.06053.

Haggstrom, J. (2017). Data-driven confounder selection via Markov and Bayesian networks. Biometrics 74 389-398. https://doi.org/10.1111/biomet.12788

Hencel, L., Perkovic, E. and Maathuis, M. H. (2022). Graphical criteria for efficient total effect estimation via adjustment in causal linear models. Journal of the Royal Statistical Society: Series B (Statistical Methodology) 84 579-599.

Hernán, M. A. and Robins, J. M. (2016). Using big data to emulate a target trial when a randomized trial is not available. American Journal of Epidemiology 183 758–764.

Hernán, M. A. and Robins, J. M. (2020). Causal Inference: What If. Chapman & Hall/CRC, Boca Raton.

Imbens, G. W. (2014). Instrumental variables: an econometrician’s perspective. Statistical Science 29 323-358. https://doi.org/10.1214/14-sts480

Kennedy, E. H. (2022). Semiparametric doubly robust targeted double machine learning: a review. arXiv preprint arXiv:2203.06469.

Koch, B., Vock, D. M., Wolfson, J. and Vock, L. B. (2020). Variable selection and estimation in causal inference using Bayesian spike and slab priors. Statistical Methods in Research 29 2445-2469. https://doi.org/10.1177/0962280219898497

Kuroki, M. and Miyakawa, M. (2003). Covariate selection for estimating the causal effect of control plans by using causal diagrams. Journal of the Royal Statistical Society: Series B (Statistical Methodology) 65 209–222.

Lauritzen, S. L. (1996). Graphical Models. Oxford University Press, New York.

Loh, W. W. and Vansteelandt, S. (2021). Confounder selection strategies targeting stable treatment effect estimators. Statistics in Medicine 40 607-630.

Maathuis, M. H. and Colombo, D. (2015). A generalized backdoor criterion. The Annals of Statistics 43 1060–1088.

Miao, W., Geng, Z. and Tchetgen Tchetgen, E. J. (2018). Identifying causal effects with proxy variables of an unmeasured confounder. Biometrika 105 987-993. https://doi.org/10.1093/biomet/asy038

Myers, J. A., Rassen, J. A. G., Gagne, J. J., Huybrechts, K. F., Schneeweiss, S., Rothman, K. J., Joffe, M. M. and Glynn, R. J. (2011). Effects of adjusting for instrumental variables on bias and precision of effect estimates. American Journal of Epidemiology 174 1213-1222. https://doi.org/10.1093/aje/kwr364

Nandy, P., Maathuis, M. H. and Richardson, T. S. (2017). Estimating the effect of joint interventions from observational data in sparse high-dimensional settings. The Annals of Statistics 45 647–674.

Ogburn, E. L. and VanderWeele, T. J. (2012). On the nondifferential missclassification of a binary confounder. Epidemiology 23 433-439. https://doi.org/10.1097/ede.0b013e318234d1f3

Ogburn, E. L. and VanderWeele, T. J. (2013). Bias attenuation results for nondifferentially mismeasured ordinal and coarsened confounders. Biometrika 100 241-248. https://doi.org/10.1093/biomet/ass054

Pearl, J. (1988). Probabilistic Reasoning in Intelligent Systems: Networks of Plausible Inference. Morgan Kaufmann Publishers Inc., San Francisco, CA, USA.

Pearl, J. (1993). Comment: graphical models, causality and intervention. Statistical Science 8 266–269.

Pearl, J. (1995). Causal diagrams for empirical research. Biometrika 82 669–688.

Pearl, J. (2009a). Remarks on the method of propensity score. Statistics in Medicine 28 1415-1416.

Pearl, J. (2009b). Causality: Models, Reasoning, and Inference, 2nd ed. Cambridge University Press.

Perkovic, E., Textor, J., Kalisch, M. and Maathuis, M. H. (2017). Complete graphical characterization and construction of adjustment sets in Markov equivalence classes of ancestral graphs. Journal of Machine Learning Research 18 8132–8193.

Persson, E., Haggstrom, J., Waernbaum, I. and de Luna, X. (2017). Data-driven algorithms for dimension reduction in causal inference. Computational Statistics & Data Analysis 105 280-292. https://doi.org/10.1016/j.csda.2016.08.012

Richardson, T. S. and Robins, J. M. (2013). Single world intervention graphs (SWIGs): a unification of the counterfactual and graphical approaches to causality. Center for the Statistics and the Social Sciences, University of Washington Series. Working Paper 128.

Richardson, T. S., Robins, J. M. and Wang, L. (2018). Discussion of “Data-driven confounder selection via Markov and Bayesian networks” by Häggström. Biometrika 74 403–406.
Robins, J. M. (1986). A new approach to causal inference in mortality studies with a sustained exposure period-application to control of the healthy worker survivor effect. *Mathematical Modelling* **7**, 1393–1512.

Robins, J. M. (1997). *Causal inference from complex longitudinal data*. In *Latent Variable Modeling and Applications to Causality*, Lecture Notes in Statistics 69-117. Springer New York. [https://doi.org/10.1007/978-1-4612-1842-5_4](https://doi.org/10.1007/978-1-4612-1842-5_4)

Robins, J. M. and Richardson, T. S. (2011). *Alternative graphical causal models and the identification of direct effects*. In *Causality and Psychopathology*, Causality and Psychopathology. Oxford University Press. [https://doi.org/10.1093/oso/9780199754649.003.0011](https://doi.org/10.1093/oso/9780199754649.003.0011)

Rosenbaum, P. R. and Rubin, D. B. (1983). The central role of the propensity score in observational studies for causal effects. *Biometrika* **70** 41-55. [https://doi.org/10.1093/biomet/70.1.41](https://doi.org/10.1093/biomet/70.1.41)

Rotnitzky, A. and Schuler, E. (2020). Efficient adjustment sets for population average causal treatment effect estimation in graphical models. *Journal of Machine Learning Research* **21** 1–86.

Rubin, D. B. (2008). For objective causal inference, design trumps analysis. *The Annals of Applied Statistics* **2** 808-840.

Rubin, D. B. (2009). Should observational studies be designed to allow lack of balance in covariate distributions across treatment groups? *Statistics in Medicine* **28** 1420-1423.

Shortreed, S. M. and Ertefaie, A. (2017). Outcome-adaptive lasso: variable selection for causal inference. *Biometrics* **73** 1111-1122. [https://doi.org/10.1111/biom.12679](https://doi.org/10.1111/biom.12679)

Spirtes, P. and Pearl, J. (2006). Identification of joint interventional distributions in recursive semi-Markovian causal models. In *Proceedings of the 21st National Conference on Artificial Intelligence - Volume 2*. AAAI’06 1219–1226. AAAI Press.

Spirtes, P., Richardson, T. S. and Robins, J. M. (2022). *Multivariate counterfactual systems and causal graphical models*. In *Probabilistic and Causal Inference: The Works of Judea Pearl* 1 ed. 813–852. Association for Computing Machinery, New York, NY, USA.

Spirtes, P., Van der Weele, T. and Robins, J. M. (2010). On the validity of covariate adjustment for estimating causal effects. In *Proceedings of the Twenty-Sixth Conference on Uncertainty in Artificial Intelligence*. UAI’10 527–536. AUAI Press, Arlington, Virginia, USA.

Shrier, I. (2008). Letter to the Editor. *Statistics in Medicine* **27** 2740-2741.

Siolander, A. (2009). Propensity scores and M-structures. *Statistics in Medicine* **28** 1416-1420.

Smucler, E. and Rotnitzky, A. (2022). A note on efficient minimum cost adjustment sets in causal graphical models. *Journal of Causal Inference* **10** 174–189.

Strob, E. V., Spirtes, P. L. and Visweswaran, S. (2019). Estimating and controlling the false discovery rate of the PC algorithm using edge-specific p-values. *ACM Transactions on Intelligent Systems and Technology (TIST)* **10** 1–37.

Studenty, M. (2004). *Probabilistic Conditional Independence Structures: With 42 Illustrations (Information Science and Statistics)*. Springer-Verlag TELOS, Santa Clara, CA, USA.

Tang, D., Kong, D., Pan, W. and Wang, L. (2022). Ultra-high dimensional variable selection for doubly robust causal inference. *Biometrics* **79** 903–914.

Tchetgen, E. J. T., Ying, A., Cui, Y., Shi, X. and Miao, W. (2020). An introduction to proximal causal learning. *arXiv preprint arXiv:2009.10982*.

Tian, J. and Pearl, J. (2002). A general identification condition for causal effects. In *Eighteenth National Conference on Artificial Intelligence* 567–573. American Association for Artificial Intelligence, USA.