Towards causality-aware predictions in static machine learning tasks: the linear structural causal model case

Elias Chaibub Neto
Sage Bionetworks, Seattle, WA 98109

Abstract: While counterfactual thinking has been used in ML tasks that aim to predict the consequences of different actions, policies, and interventions, it has not yet been leveraged in more traditional/static supervised learning tasks, such as the prediction of discrete labels in classification tasks or continuous responses in regression problems. Here, we propose a counterfactual approach to train “causality-aware” predictive models that are able to leverage causal information in static ML tasks. In applications plagued by confounding, the approach can be used to generate predictions that are free from the influence of observed confounders. In applications involving observed mediators, the approach can be used to generate predictions that only capture the direct or the indirect causal influences. The ability to quantify how much of the predictive performance of a learner is actually due to the causal relations of interest is important to improve the explainability of ML systems. Mechanistically, we train and evaluate supervised ML algorithms on (counterfactually) simulated data which retains only the associations generated by the causal relations of interest. In this paper we focus on linear models, where analytical results connecting covariances to causal effects are readily available. Quite importantly, we show that our approach does not require knowledge of the full causal graph. It suffices to know which variables represent potential confounders and/or mediators, and whether the features have a causal influence on the response (or vice-versa).

1. INTRODUCTION

Machine learning (ML) plays an increasingly pronounced role in society, with ramifications in most areas of human activities including health, law, and business. With the increasing availability of data and improved efficiency of computing, ML systems are becoming more powerful, more complex, and more widely deployed. However, together with the increase in popularity, the ML field is also experiencing a deeper awareness about its limitations. A growing concern of ML practitioners revolves around explainability, transparency, and discrimination issues.

Causal reasoning has been proposed as a possible remedy for some of these issues. Although ML methods have been more traditionally used for prediction, while causal inference methods have usually focused on the estimation of the causal effects from treatments and actions, some researchers have argued that the adoption of causal modeling tools, in particular, causal diagrams and counterfactual thinking has the potential to overcome fundamental limitations of ML
Current approaches operating at the intersection between causality and ML can be roughly split into two different classes. The first, focus on the prediction of the consequences of different actions, policies, and interventions, aiming to improve decision making. These approaches attempt to answer “what if” counterfactual questions such as “What if I had treated a patient differently?”; “What if I had placed an advertisement on a different position of the webpage?”; “What if I had applied a different taxation policy?”. The second class, is largely concerned with the estimation of causal effects and only uses ML techniques as a tool to improve the estimation of causal effects. (In the Related work section, we will review in more detail these two distinct tasks.)

In this paper, we address a different problem. Rather than attempting to predict the consequences of actions/interventions, or to estimate causal effects, we focus on traditional supervised learning tasks, such as building a classifier of disease status in a diagnostic application or predicting the values of a continuous response variable in a regression problem. In these static ML tasks the goal is still to generate good predictions of the output variable given a new set of input data. However, we want to do that in a way that only leverages the associations that are generated by the causal mechanisms of interest.

To this end, we propose a simple counterfactual approach to train “causality-aware” predictive models, where we train and evaluate machine learning algorithms on (counterfactually) simulated data which retains only the associations of interest. For instance, in applications plagued by confounding, we simulate counterfactual data where the total association between the response and the features is only due to the causal relations between features and response, but not to the spurious associations generated by the observed confounders. By training and evaluating ML models on the counterfactual data we are able to generate predictions that are free from the influence of the observed confounders and can be used to answer the counterfactual question: what would the predictive performance of the learner be, had the observed confounders not biased the data? On the other hand, in mediation problems whose interest focuses on the direct causal effect we can simulate counterfactual data to answer the question: what would the predictive performance of the learner be, had the indirect path not contributed to the association between the features and the response? Similarly, if the interest focus on the indirect causal effect, we can also simulate counterfactual data where the association between features and response is only due to the indirect causal paths.

This ability to disentangle how much of the predictive performance of a learner is actually due to the causal relations of interest is key to improve the explainability of ML systems. For instance, our approach can be used to evaluate the importance of sensitive variables in prediction tasks, where it is important to quantify the predictive performance that is free from the influence of the sensitive variable, as well as, the predictive performance that is due exclusively to the sensitive variable. (Here, it is important to point out that there is always a trade-off between explainability and accuracy, and better explainability often leads to a reduction in predictive performance.)
In this paper, we focus on linear models, where analytical results connecting covariances and causal effects are readily available. In practice, the proposed approach is implemented using a modification of Pearl’s three step approach (“abduction, action, prediction”) for the computation of deterministic counterfactuals (Pearl 2000; Pearl, Glymour, Jewell 2016). In the “abduction” step we fit regression models to the data and estimate causal effects and model residuals. In the “action” step we intervene on the causal diagram in order to remove causal paths that generate confounding, or causal paths that lead to direct or indirect effects in mediation tasks. In the “prediction” step we use the modified model to simulate the counterfactual data that will be used for the ML prediction task.

Quite importantly, we clarify that the interventions that we perform in the “action” step are different from Pearl’s \( do(Z = z) \) interventions. While Pearl’s \( do(Z = z) \) intervention corresponds to replacing the structural equation describing a variable \( Z \) by a fixed value \( z \), our interventions correspond to removing the protected variables (i.e., confounders and/or mediators) from the parent set of either the response or the features. Using the twin network approach (Balke and Pearl 1994; Pearl 2000), we show that our proposed intervention captures the covariance that is generated by the causal relations of interest, while interventions of the type \( do(Z = z) \) are often unable to capture these covariances.

At first sight, the proposed approach appears to require the strong assumption that one needs to know the full causal graph describing the data generation process. We point out, however, that this is not the case. Actually, the approach only requires partial domain knowledge about which variables represent potential confounders and/or mediators and whether the prediction goes in the causal direction (i.e., the features have a causal influence on the response), or in the anticausal direction (i.e., the response has a causal influence on the features). Noteworthy, we will present analytical results showing that we can always reparameterize the model in a way that the covariance among the features, among the confounders, and among the mediators is pushed towards the respective error terms, so that we can safely generate counterfactual data without even knowing the causal relations among features, the causal relations among the confounders, and the causal relations among the mediators. In practice, this is an important advantage in applications involving high-dimensional feature spaces and metadata, where it is unlikely that domain knowledge about these causal relationships (specially among the features) will be available. It is also important to point out that, in many applications, it is quite clear if the ML prediction is causal or anti-causal. For instance, health applications where the goal is to predict the severity of a disease, using measured symptoms as inputs, are clearly anticausal prediction tasks since the disease always causes the symptoms.

The rest of this paper is organized as follows. Section 2 reviews previous work on the intersection between causal inference and ML, and describes how they differ from our proposed approach. Section 3 presents notation and key definitions that will be needed latter in the paper. Section 4 describes the proposed approach across several subsections. For the sake of clarity, we first present results
and important remarks in the univariate case, before introducing the reparameterization and counterfactual estimation procedures in the general multivariate case. Section 5 provides final remarks.

2. RELATED WORK

Causal approaches based on counterfactual thinking have been used in the context of ML applications to predict the outcomes of different actions, policies, and interventions using non-experimental data (Bottou et al 2013; Swaminathan and Joachims 2015; Johansson et al 2016; Schulam and Saria 2017). For instance, Bottou et al (2013) have adopted a counterfactual approach to evaluate the influence of advertisement placement in search engines. Schulam and Saria (2017) developed the counterfactual Gaussian process approach for modeling the effects of sequences of actions on continuous-time time series data and improve the reliability of medical decision support tools. The goal of these approaches is to make “what if” predictions of the consequences of different actions in order to guide decisions. These approaches, however, are only applicable in situations where the “treatment” variables correspond to features of the ML model, so that prediction goes in the same direction of the causal effect (i.e., the features have a causal effect on the response variable). Our approach, on the other hand, focus on static ML tasks, and can also be employed in applications where the outcome variable has a causal effect on the features.

Supervised ML has also been extensively used to aid the estimation of causal effects, where it can potentially attenuate model mis specification issues (Kreif and DíazOrdaz 2019). In particular, supervised ML has been used to: (i) improve the calculation of propensity scores (McCaffrey et al 2004; Westreich et al 2010; Lee et al 2010; Wyss et al 2014; Pirracchio et al 2012; Zhu et al 2015); (ii) fit regression approaches to estimate outcome models (Hill 2011; Austin 2012; Hahn et al 2017); and (iii) also for the development of double-robust approaches that combine propensity score and outcome regression approaches together (Gruber and van der Laan 2011; Chernozhukov et al 2017). In this paper, however, we take an opposite strategy where instead of using ML to improve causal inference we leverage causal knowledge to improve the explainability of ML predictions.

3. PRELIMINARIES

Throughout the text we let $X = (X_1, X_2, \ldots, X_n)^T$, $C = (C_1, \ldots, C_n)^T$, and $M = (M_1, \ldots, M_M)^T$ represent, respectively, sets of features, confounders, and mediators, while $Y$ represents the response (outcome) variable. The counterfactual versions of $X$, $M$, and $Y$ are represented, respectively, by $X^*$, $M^*$, and $Y^*$.

Following Pearl (2000), we adopt a mechanism-based approach to causation, where the statistical information encoded in the joint probability distribution of a set of variables is supplemented by a directed acyclic graph (DAG) describing our qualitative assumptions about the causal relation between the variables. In
a DAG, a \textit{path} is defined as any unbroken, nonintersecting sequence of edges, which may go along or against the direction of the arrows. A path is \textit{d-separated} or \textit{blocked} (Pearl, 2000) by a set of nodes $W$ if and only if: (i) the path contains a chain $Z_j \rightarrow Z_m \rightarrow Z_k$ or a fork $Z_i \leftarrow Z_m \rightarrow Z_k$ such that the middle node $Z_m$ is in $W$; or (ii) the path contains an collider $Z_j \rightarrow Z_m \leftarrow Z_k$ such that the middle node $Z_m$ is not in $W$ and no descendant of $Z_m$ is in $W$. Otherwise, the path is \textit{d-connected} or \textit{open}.

We denote prediction tasks where the response influences the features as \textit{anticausal prediction tasks}, whereas tasks where the features influence the response are denoted as \textit{causal prediction tasks}. Figure 1 presents DAGs of general anticausal and causal predictive tasks, where $X$, $C$, and $M$ are organized into arbitrary DAG subdiagrams (see Figure 2 for an example of how $X$, $C$, $M$ are organized into subdiagrams).

Note that an arrow from one set of variables $Z_1$ to another set $Z_2$, $Z_1 \rightarrow Z_2$, implies that at least one variable in $Z_1$ is a parent of at least one variable in $Z_2$. Observe, as well, that $Z_1 \rightarrow Z_2$ is also a notation for a set of open paths that start at an element of $Z_1$ and end at an element of $Z_2$, and might include direct, indirect, and backdoor paths, involving only elements of $Z_1$ and $Z_2^1$. Throughout the text, we will denote these set of paths as \textit{set paths} in order to distinguish it from standard paths. Similarly, set paths such as $Z_1 \rightarrow Z_2 \rightarrow Z_3$

are composed of the union of the indirect paths that start at $Z_1$ and end at $Z_3$ and the backdoor paths that start at $Z_2$, go to $Z_1$, and return to $Z_2$ before ending at $Z_3^2$. On the other hand, set paths such as $Z_1 \leftarrow Z_2 \rightarrow Z_3$ are

\begin{figure}[h]
\centering
\includegraphics[width=\textwidth]{figure1.png}
\caption{DAGs describing anticausal (panel a) and causal (panel b) prediction tasks.}
\end{figure}

\begin{figure}[h]
\centering
\includegraphics[width=\textwidth]{figure2.png}
\caption{Panel a shows an example of the general causal prediction task shown in Figure 1b. Panels b, c, and d show, respectively, the DAG subdiagrams represented by the $X$, $C$, and $M$ nodes in Figure 1b.}
\end{figure}

\begin{figure}[h]
\centering
\includegraphics[width=\textwidth]{figure3.png}
\caption{For instance, for the DAG in Figure 2a we have that the set $X \rightarrow Y$ is composed by the direct paths $X_1 \rightarrow Y$ and $X_3 \rightarrow Y$, the indirect path $X_1 \rightarrow X_3 \rightarrow Y$, and the backdoor paths $X_1 \leftarrow X_2 \rightarrow X_3 \rightarrow Y$ and $X_3 \leftarrow X_2 \rightarrow X_1 \rightarrow Y$. In Figure 2a, the set path $C \rightarrow X \rightarrow Y$ is composed by the indirect paths $C_1 \rightarrow X_1 \rightarrow Y$, $C_1 \rightarrow X_3 \rightarrow Y$, $C_1 \rightarrow X_1 \rightarrow X_3 \rightarrow Y$, $C_2 \rightarrow C_1 \rightarrow X_1 \rightarrow Y$, $C_2 \rightarrow C_1 \rightarrow X_1 \rightarrow X_3 \rightarrow Y$, and $C_2 \rightarrow C_1 \rightarrow X_1 \rightarrow X_3 \rightarrow Y$, and the backdoor paths $X_1 \leftarrow C_1 \rightarrow X_3 \rightarrow Y$ and

\footnotesize{\begin{itemize}
\item[] \textit{For instance, for the DAG in Figure 2a we have that the set $X \rightarrow Y$ is composed by the direct paths $X_1 \rightarrow Y$ and $X_3 \rightarrow Y$, the indirect path $X_1 \rightarrow X_3 \rightarrow Y$, and the backdoor paths $X_1 \leftarrow X_2 \rightarrow X_3 \rightarrow Y$ and $X_3 \leftarrow X_2 \rightarrow X_1 \rightarrow Y$.}
\item[] \textit{In Figure 2a, the set path $C \rightarrow X \rightarrow Y$ is composed by the indirect paths $C_1 \rightarrow X_1 \rightarrow Y$, $C_1 \rightarrow X_3 \rightarrow Y$, $C_1 \rightarrow X_1 \rightarrow X_3 \rightarrow Y$, $C_2 \rightarrow C_1 \rightarrow X_1 \rightarrow Y$, $C_2 \rightarrow C_1 \rightarrow X_1 \rightarrow X_3 \rightarrow Y$, and $C_2 \rightarrow C_1 \rightarrow X_1 \rightarrow X_3 \rightarrow Y$, and the backdoor paths $X_1 \leftarrow C_1 \rightarrow X_3 \rightarrow Y$ and}
\end{itemize}}
\end{figure}
composed of backdoor paths that start at $Z_1$ and end at $Z_3$.  

4. THE PROPOSED APPROACH

4.1. THE UNIVARIATE CASE

For the sake of clarity, we first describe our proposed approach in the special case where $X$, $C$, and $M$ are composed of a single variable. We describe how to use counterfactual reasoning to simulate datasets where the association between the response and the features is due exclusively to the causal effects of interest. For the sake of simplicity, we assume that the data is generated from a standardized linear model, so that the variances of $X$, $C$, $M$, and $Y$ are equal to 1, and the direct causal effect of a variable $Z_j$ on another variable $Z_k$ is represented by the path coefficient (Wright 1934), $\theta_{Z_kZ_j}$.  

The anticausal task presented in Figure 1a is represented by the set of structural equations, $C = U_C$, $Y = \theta_{YC} C + U_Y$, $M = \theta_{MC} C + \theta_{MY} Y + U_M$, and $X = \theta_{XC} C + \theta_{XM} M + \theta_{XY} Y + U_X$, where $U_C$, $U_Y$, $U_M$, and $U_X$ are independent background (residual) variables. Using Wright’s method of path analysis (Wright 1934), we have that the total covariance (correlation) between $X$ and $Y$,

$$
\text{Cov}(X, Y) = \theta_{XY} + \theta_{XM} \theta_{MY} + \theta_{XC} \theta_{YC}.
$$

can be decomposed into the contribution of the direct causal path, $Y \rightarrow X$, the indirect causal path $Y \rightarrow M \rightarrow X$, and the spurious association generated by the backdoor path $X \leftarrow C \rightarrow Y$.

Clearly, the predictive performance of any ML model trained with data generated by this model will be biased by the influence of the confounder $C$ since the learner will leverage the total association between $X$ and $Y$ during training.

Now, suppose that our goal is to build a ML model whose predictive performance is only informed by the direct influence of $Y$ on $X$ and is free from the influence of $C$, as well as, from the indirect influence of $Y$ that is mediated by $M$. To this end, we need to simulate counterfactual data where the association between $X$ and $Y$ is due exclusively to the direct causal effect of $Y$ on $X$.

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In Figure 2a, the set path $X \leftarrow C \rightarrow Y$, is composed by the backdoor paths $X_1 \leftarrow C_1 \rightarrow C_2 \rightarrow C_3 \rightarrow Y$ and $X_3 \leftarrow X_1 \leftarrow C_1 \rightarrow C_2 \rightarrow C_3 \rightarrow Y$. Note that blocked paths such as $X_2 \rightarrow X_1 \leftarrow C_1 \rightarrow C_2 \rightarrow C_3 \rightarrow Y$, which include a collider node ($X_1$ in this example), are not included in the set path. Set paths only include open paths.

Note that any linear model $Z_k = \mu_k + \Sigma_j \beta_{kj} Z_j + U_k$, where $Z_k$ represents the original data, can be reparameterized into its equivalent standardized form $Z_k = \sum_j \theta_{kj} Z_j + U_k$, where $Z_k = (Z_k - E(Z_k))/\sqrt{\text{Var}(Z_k)}$ represent standardized variables with $E(Z_k) = 0$ and $\text{Var}(Z_k) = 1$; $\theta_{Z_kZ_j} = \beta_{Z_kZ_j} \sqrt{\text{Var}(Z_j)/\text{Var}(Z_k)}$ represent the path coefficients; and $U_k = U_k^* / \sqrt{\text{Var}(Z_k^*)}$ represent the standardized error terms. Note that for the standardized model, we have that the covariances are identical to the correlations - i.e., $\text{Cov}(Z_k, Z_j) = \text{Cov}(Z_k, Z_j)$ - since all variables have variance equal to 1.
In other words, we want to simulate counterfactual feature and response data, $X^*$ and $Y^*$, such that $\text{Cov}(X^*, Y^*) = \theta_{XY}$. In theory, this could be done by simulating data according to the twin network\(^5\) (Balke and Pearl 1994; Pearl 2000) in Figure 3a, where the new counterfactual feature data, $X^*$, is generated from the model $X^* = \theta_{XY} Y + U_X$. (In practice, we can estimate $\theta_{XY}$ and $U_X$ by regressing $X$ on $C, M$ and $Y$, and simulate the counterfactual feature data using $\hat{X}^* = \theta_{XY} Y + \hat{U}_X$. In other words, we can employ a variation of Pearl’s “abduction, action, prediction” approach to simulate deterministic counterfactuals. In the next subsection we explain in detail how the proposed approach differs from Pearl’s approach at the “action” step.) Direct calculation of the covariances between $X^*$ and $Y^*$ shows that,

$$\text{Cov}(X^*, Y^*) = \text{Cov}(X^*, Y) = \text{Cov}(\theta_{XY} Y + U_X, Y)$$

$$= \theta_{XY} \text{Var}(Y) + \text{Cov}(U_X, Y) = \theta_{XY}$$

(1)

where the first equality follows from the fact that we only need to simulate counterfactual feature data but not counterfactual response data in this example (so that $Y^* = Y$). (Similarly, for the causal task in Figure 1b, we can simulate counterfactual response data, $Y^*$, according to the twin network in Figure 3b in order to generate datasets where $\text{Cov}(X^*, Y^*) = \text{Cov}(X, Y^*) = \theta_{YX}.\)  

![Diagram of twin network approach](image)

Fig 3. Twin network approach in the case where the direct effect represents the causal effect of interest.

Now, suppose that our goal is to build a ML model whose predictive performance is only informed by the indirect causal effect of $Y$ on $X$. In this case, we simulate data according to the twin network in Figure 4a, so that,

$$\text{Cov}(X^*, Y^*) = \text{Cov}(X^*, Y) = \text{Cov}(\theta_{XM} M^* + U_X, Y)$$

$$= \theta_{XM} \text{Cov}(M^*, Y) = \theta_{XM} \text{Cov}(\theta_{MY} Y + U_M, Y)$$

$$= \theta_{XM} \theta_{MY} \text{Cov}(Y, Y) = \theta_{XM} \theta_{MY}.\)  \(2\)

\(^5\)The twin network approach provides a graphical method for evaluating conditional independence relations between counterfactual and factual variables. The basic idea is to use two networks, one representing the factual world and the other the counterfactual world, which share the same background (residual) variables. The factual network (shown to the left of the residual terms) represents the data generation process for the original data, while the counterfactual network (show to the right of the residual terms) shows the modified causal model.
(Similarly, for the causal task in Figure 1b, we can simulate counterfactual response data, \(Y^*\), according to the twin network in Figures 4b so that \(\text{Cov}(X^*, Y^*) = \text{Cov}(X, Y^*) = \theta_{YM}\theta_{M,X}\).

\[\text{Cov}(X^*, Y^*) = \text{Cov}(\theta_{XC}C + U_X, \theta_{YC}C + U_Y) = \theta_{XC}\theta_{YC}\text{Cov}(C, C) = \theta_{XC}\theta_{YC}. \quad (3)\]

Finally, if the goal is to build a ML model whose predictive performance is only informed by the spurious associations generated by the confounder, we can simulate data according to the twin network in Figure 5, so that,

\[\text{Cov}(X^*, Y^*) = \text{Cov}(\theta_{XC}C + U_X, \theta_{YC}C + U_Y) = \theta_{XC}\theta_{YC}\text{Cov}(C, C) = \theta_{XC}\theta_{YC}. \quad (3)\]

At this point, a natural question is whether alternative interventions would also work. In Section 6 in the Supplement, we show that a requirement for the intervention to work is that \(Y\) is not altered by the intervention in anticausal prediction tasks, whereas \(X\) is not altered by the intervention in causal prediction tasks. Furthermore, in Section 7 in the Supplement we also show that node-splitting transformations in SWIGs (Richardson and Robins 2013) can also be used as alternative interventions.

4.1.1. Remarks

At this point, it is important to highlight that our proposed interventions are different from Pearl’s \(\text{do}(Z = z)\) interventions, and that our counterfactual approach is implemented using a modification of Pearl’s “abduction, action, prediction” procedure for the computation of deterministic counterfactuals. While in Pearl’s approach the action step is enforced by a \(\text{do}(Z = z)\) intervention, where the causal structural model \(Z = f(pa(Z), U_Z)\) is replaced by \(Z = z\),...
our interventions are different. For instance, in the case where the direct effect represents the causal effect of interest, our intervention corresponds to replacing 
\[ X = f_X(pa(X), U_X) \]
by
\[ X = f_X(pa(X) \setminus \{ C \cup M \}, U_X) = f_X(Y, U_X) \]
in anticausal prediction tasks, and to replacing
\[ Y = f_Y(C, M, X, U_Y) \]
by
\[ Y = f_Y(X, U_Y) \]
in causal prediction tasks. (Note that while our interventions at the action step differs from Pearl’s approach, the abduction and prediction steps are still the same.)

From a more “philosophical” point of view, note that even though our proposed interventions represent a different type of microsurgery on the structural causal models, they are still consistent with Lewis’ framework of possible worlds (Lewis 2013). Instead of considering counterfactual worlds that develop from different actions than the actions taken in the factual world, our approach considers counterfactual worlds where the data generation mechanisms/laws are different from the mechanisms/laws of the factual world.

While interventions of the type \( \text{do}(Z = z) \) represent a natural approach when the goal is to estimate the causal effects of a given action/treatment, this is not necessarily so when the interest focus on the associations generated by causal mechanisms, rather than on the causal effects per se.

As a simple example, consider a linear structural causal model where the observed confounder \( C \) is a discrete variable, \( X = \theta_{XC} C + U_X \) and \( Y = \theta_{YX} X + \theta_{YC} C + U_Y \). Now, let \( Y_x \) represent the value the response would have attained, had \( X \) been set to \( x \). Assuming that \( C \) is the only confounder, we have that the direct causal effect of \( X \) on \( Y \) is given by the difference in expectations,
\[
E[Y|\text{do}(X = x')] - E[Y|\text{do}(X = x)] = \theta_{YX},
\]
where \( E[Y|\text{do}(X = x)] \) is computed with respect to the backdoor adjusted distribution \( f(Y_x) = f(Y | \text{do}(X = x)) = \sum_c f(Y | X = x, C = c) P(C = c) \). However, as we show in Section 8 in the Supplement, it turns out that
\[
\text{Cov}(X, Y_x) = \theta_{YC} \theta_{XC} \]
rather than \( \theta_{YX} \).

Given that Pearl’s do calculus was developed to identify causal effects, it is not really surprising that it cannot be readily used when the goal is to estimate associations, rather than causal effects. We point out, nonetheless, that \( \text{do}(Z = z) \) interventions can still be used to estimate associations if one is willing to consider more complicated counterfactuals arising from node-splitting operations in SIWGs, as described in Section 7 in the Supplement.

### 4.2. THE MULTIVARIATE CASE

Now, we extend the results presented in Section 4.1 to the multivariate case, where the nodes \( X, C, \) and \( M \) in Figure 1 represent arbitrary DAG subdiagrams. The main results are presented in Section 4.2.3. But first, we show that

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6As an example, consider an anticausal prediction task described by the DAG \( C \xrightarrow{\rightarrow} X \xrightarrow{\rightarrow} Y \), where \( Y \) represents the severity score of a disease, \( X \) represents a symptom, \( C \) represents age, and where the goal is to predict \( Y \) using \( X \), after removing the spurious association generate by \( C \). In our proposed approach, we consider a counterfactual world, \( C \xrightarrow{\rightarrow} X^* \xrightarrow{\rightarrow} Y \), where age no longer influences the symptom \( X \).
we can always reparameterize linear structural causal models in a way that, in practice, we do not need to know how the DAG subdiagrams are organized in order to estimate the causal effects and the residuals employed in the computation of the counterfactual data.

4.2.1. Reparameterization in linear models

For linear structural causal models, we can always reparameterize any arbitrary DAG model to a simpler model where the covariance structure between the observed variables is “pushed” to the unobserved error terms and no arrows directly connect any two observed variables. Figure 6 provides an illustration. The DAG in panel a represents the actual data generation process for the variables \( \mathbf{X} = (X_1, X_2, X_3)^T \), where the error terms \( \mathbf{U}_X = (U_{X_1}, U_{X_2}, U_{X_3})^T \) are independent, whereas the DAG in panel b shows the reparameterized model with correlated error terms \( \mathbf{W}_X = (W_{X_1}, W_{X_2}, W_{X_3})^T \).

Explicitly, the reparameterization goes as follows. The set of linear structural causal models describing the DAG in Figure 6a is given by,

\[
\begin{pmatrix}
X_1 \\
X_2 \\
X_3
\end{pmatrix} = \begin{pmatrix}
0 & 0 & 0 \\
\theta_{X_2 X_1} & 0 & 0 \\
\theta_{X_3 X_1} & \theta_{X_3 X_2} & 0
\end{pmatrix} \begin{pmatrix}
X_1 \\
X_2 \\
X_3
\end{pmatrix} + \begin{pmatrix}
U_{X_1} \\
U_{X_2} \\
U_{X_3}
\end{pmatrix},
\]

or, in matrix notation, by \( \mathbf{X} = \Theta_{XX} \mathbf{X} + \mathbf{U}_X \). Simple algebraic manipulations show that we can re-express \( \mathbf{X} \) as, \( \mathbf{X} = (I - \Theta_{XX})^{-1}\mathbf{U}_X \). Re-naming the term \( (I - \Theta_{XX})^{-1}\mathbf{U}_X \) as \( \mathbf{W}_X \), we have that \( \mathbf{X} \) can be reexpressed as \( \mathbf{X} = \mathbf{W}_X \),
where the correlated error term,

\[
W_X = \begin{pmatrix}
1 & 0 & 0 \\
\theta_{X_2X_1} & 1 & 0 \\
\theta_{X_3X_1} + \theta_{X_2X_1} \theta_{X_3X_2} & \theta_{X_3X_2} & 1 \\
\end{pmatrix}
\begin{pmatrix}
U_{X_1} \\
U_{X_2} \\
U_{X_3}
\end{pmatrix}
\]

is represented in the DAG in Figure 6b. Quite importantly, note that because model \(X = W_X\) is just a simple reparameterization of model \(X = \Theta_{XX} X + U_X\), we have that the association structure between the \(X_j\) variables is still the same after the model reparameterization.

Now, consider the arbitrary anticausal and causal predictive tasks presented in Figure 1. Panel a shows the DAG for the anticausal model, whose joint distribution is factorized as,

\[
P(C)P(Y | C)P(M | C, Y)P(X | C, M, Y)
\]

Each component of the above factorization is described, respectively, by the linear structural causal models,

\[
\begin{align*}
C &= \Theta_{CC} C + U_C \\
Y &= \Theta_{YC} C + U_Y \\
M &= \Theta_{MM} M + \Theta_{MC} C + \Theta_{MY} Y + U_M \\
X &= \Theta_{XX} X + \Theta_{XC} C + \Theta_{XM} M + \Theta_{XY} Y + U_X
\end{align*}
\]

where \(U_C, U_Y, U_M,\) and \(U_X\) are vectors of independent error terms with zero mean and finite variance; \(\Theta_{CC}, \Theta_{MM},\) and \(\Theta_{MM}\) represent, respectively, square matrices of dimension \(n_C \times n_C\), \(n_M \times n_C\), and \(n_C \times n_C\), containing the path coefficients connecting the confounders among themselves, the mediators among themselves and the features among themselves; and \(\Theta_{MC}, \Theta_{MY}, \Theta_{XC}, \Theta_{XM},\) and \(\Theta_{XY}\), represent rectangular matrices of path coefficients connecting variables from separate sets. (For instance, \(\Theta_{MC}\), corresponds to a \(n_M \times n_C\) matrix of path coefficients connecting confounder variables to mediator variables, whereas \(\Theta_{XY}\), corresponds to a \(n_X \times 1\) matrix of path coefficients connecting the response to the features.)

Using simple algebraic manipulations, we can re-write the above linear structural models as,

\[
\begin{align*}
C &= W_C \\
Y &= \Gamma_{YC} C + W_Y \\
M &= \Gamma_{MC} C + \Gamma_{MY} Y + W_M \\
X &= \Gamma_{XC} C + \Gamma_{XM} M + \Gamma_{XY} Y + W_X
\end{align*}
\]
where $W_C = (I - \Theta_{CC})^{-1}U_C$, $W_Y = U_Y$, $W_M = (I - \Theta_{MM})^{-1}U_M$, $W_X = (I - \Theta_{XX})^{-1}U_X$, and $\Gamma_{YC} = \Theta_{YC}$, $\Gamma_{MC} = (I - \Theta_{MM})^{-1}\Theta_{MC}$, $\Gamma_{MY} = (I - \Theta_{MM})^{-1}\Theta_{MY}$, $\Gamma_{XC} = (I - \Theta_{XX})^{-1}\Theta_{XC}$, $\Gamma_{XM} = (I - \Theta_{XX})^{-1}\Theta_{XM}$, and $\Gamma_{XY} = (I - \Theta_{XX})^{-1}\Theta_{XY}$.

Similarly, for the causal prediction task presented in Figure 1b we have that the joint distribution is factorized as,

$$P(C)P(X \mid C)P(M \mid C, X)P(Y \mid C, M, X)$$,

where each component is described by the structural model,

$$C = \Theta_{CC}C + U_C$$

$$X = \Theta_{XX}X + \Theta_{XC}C + U_X$$

$$M = \Theta_{MM}M + \Theta_{MC}C + \Theta_{MX}X + U_M$$

$$Y = \Theta_{YC}C + \Theta_{YM}M + \Theta_{YX}X + U_Y$$

which can also be reparameterized as,

$$C = W_C$$

$$X = \Gamma_{XC}C + W_X$$

$$M = \Gamma_{MC}C + \Gamma_{MX}X + W_M$$

$$Y = \Gamma_{YC}C + \Gamma_{YM}M + \Gamma_{YX}X + W_Y$$

where $\Gamma_{MX} = (I - \Theta_{MM})^{-1}\Theta_{MX}$, $\Gamma_{YC} = \Theta_{YC}$, $\Gamma_{YM} = \Theta_{YM}$, $\Gamma_{YX} = \Theta_{YX}$, $W_Y = U_Y$, and the other parameters and error terms are given as before.

Note that, in the reparameterized models, we have that the total causal effect between $X$ and $Y$ is given by $\Gamma_{XY} = \Gamma_{XY} + \Gamma_{XM}\Gamma_{MY}$ in the anticausal prediction task, and by $\Gamma_{YX} = \Gamma_{YX} + \Gamma_{YM}\Gamma_{MX}$ in the causal prediction task.

Next, we present an illustrative example based on an anticausal prediction task. Figure 7a represents the actual data generation process, whereas Figure 7b represents the reparameterized model.

Section 10 in the Supplement provides a derivation of $\Gamma_{YC}$, $\Gamma_{MC}$, $\Gamma_{MY}$, $\Gamma_{XC}$, $\Gamma_{XM}$, and $\Gamma_{XY}$ (as well as, of $W_C$, $W_Y$, $W_M$, and $W_X$) for this particular example. Here, we just present the results.

Table 1 presents the direct causal effects in the reparameterized model (represented by the $\gamma$s). Note that the $\gamma$s correspond to sums of direct and indirect effects in the original model (represented by the sum of products of $\theta$s). For instance, $\gamma_{X3Y}$, corresponds to $\theta_{X3Y} + \theta_{X3X2}\theta_{X2Y}$, where $\theta_{X3Y}$ represents the direct causal effect of $Y$ on $X_3$ in the original model, while $\theta_{X3X2}\theta_{X2Y}$ represents the indirect causal effect of $Y$ on $X_3$ that is mediated by $X_2$ via the indirect path $Y \rightarrow X_2 \rightarrow X_3$ in the original model. Observe, as well, that the total causal effects of $Y$ on $X$ (computed as $\Gamma_{XY} = \Gamma_{XY} + \Gamma_{XM}\Gamma_{MY}$), are
\[ \gamma \in \Gamma_{YC} \quad \gamma_{YC} = \theta_{YC} \]
\[ \gamma \in \Gamma_{MC} \quad \gamma_{MC} = \gamma_{YC} = 0 \]
\[ \gamma \in \Gamma_{GY} \quad \gamma_{GY} = \theta_{GY} \]

\[ \gamma \in \Gamma_{MX} \quad \gamma_{MX} = \theta_{MX} \]
\[ \gamma \in \Gamma_{XY} \quad \gamma_{XY} = \theta_{XY} \]

**Table 1**

Direct causal effects in the reparameterized model.

\[ \tilde{\gamma}_{X_1 Y} = \theta_{X_1 M_2} \theta_{M_2 Y} \]
\[ \tilde{\gamma}_{X_2 Y} = \theta_{X_2 Y} + \theta_{X_2 X_1} \theta_{X_1 M_2} \theta_{M_2 Y} \]
\[ \tilde{\gamma}_{X_3 Y} = \theta_{X_3 Y} + \theta_{X_3 X_2} \theta_{X_2 Y} + \theta_{X_3 M_1} \theta_{M_1 M_2} \theta_{M_2 Y} + \theta_{X_3 X_1} \theta_{X_1 M_2} \theta_{M_2 Y} \]

Note that the total causal effect of \( Y \) on \( X_1 \) corresponds to the indirect causal
4.2.2. Estimation of causal effects and residuals in the reparameterized model

In practice, our counterfactual approach requires the estimation of causal effects and residuals using regression models. For an anticausal task, we regress each feature $X_j$, $j = 1, \ldots, n_X$, on the set of observed confounders and mediators using, respectively, \( \hat{\gamma}_{X_iC_k} \), \( \hat{\gamma}_{X_iM_k} \), and residuals \( \hat{W}_{X_j} \), and then generate counterfactual features by adding back the estimated residuals to a linear predictor containing only the causal effects of interest. That is, in order to estimate the predictive performance due to direct causal effects, indirect causal effects, or confounding, we generate counterfactual features using, respectively, \( \hat{X}^* = \tilde{\Gamma}_{XY}Y + \hat{W}_X \), \( \hat{X}^* = \tilde{\Gamma}_{XM} \hat{M} + \hat{W}_X \), or \( \hat{X}^* = \tilde{\Gamma}_{XC}C + \hat{W}_X \).

Similarly, for the causal prediction task, we regress the response on the confounders, mediators, and features,

\[
Y = \sum_{k=1}^{n_C} \gamma_{YC_k}C_k + \sum_{k=1}^{n_M} \gamma_{YM_k}M_k + \sum_{k=1}^{n_X} \gamma_{YX_k}X_k + W_Y,
\]

and then generate the counterfactual response by adding back \( \hat{W}_Y \) to a linear predictor containing only the causal effects of interest. In particular, we can generate counterfactual response data that captures the predictive performance due to direct causal effects, indirect causal effects, or to confounding, using, \( \hat{Y}^* = \tilde{\Gamma}_{YX}X + \hat{W}_Y \), \( \hat{Y}^* = \tilde{\Gamma}_{YM} \hat{M} + \hat{W}_Y \), or \( \hat{Y}^* = \tilde{\Gamma}_{YC}C + \hat{W}_Y \).

Under the assumption that all the confounders and mediators are observed, we can identify the direct and indirect causal effects of response on the features (or vice-versa). In particular, a simple least squares estimation procedure provides consistent estimates of these causal effects. To see why, note that for the reparameterized model, if all confounders and mediators are observed, it
follows from the Markov property of DAGs that $X_j = f_{X_j}(C, M, Y, W_{X_j}) = f_X(pa(X_j), W_{X_j})$ in anticausal prediction tasks, and $Y = f_Y(C, M, X, W_Y) = f_Y(pa(Y), W_Y)$ in causal prediction tasks. (Here, $f_X$ and $f_Y$ represent linear structural causal models). Hence, for the anticausal task, it follows that, when we regress $X_j$ on the elements of $C$, $M$, and $Y$ only the coefficients associated with the parents of $X_j$ will be statistically different from zero (for large enough sample sizes). This shows that, in practice, we don’t need to know before hand which variables are the parents of $X_j$ in the reparameterized model. The parent set will be learned automatically from the data by the regression model fit. Similarly, for the causal task, we can also automatically learn the parent set of $Y$ by the regression model fit.

Finally, observe, as well, that even if the mediators are unobserved, but the confounders are still observed, we can still identify the total causal effects $\tilde{\Gamma}_{XY}$ (in anticausal tasks) and $\tilde{\Gamma}_{YX}$ (in causal tasks). On the other hand, if the mediators are observed, but the confounders are unobserved, then neither the direct, the indirect, or the total causal effects are identifiable. Finally, observe that while we have focused on confounders of the feature/response relationship, it is also possible that the causal relations between features and mediators or between mediators and response are also influenced by confounders. If these confounders are unobserved, then we cannot identify the causal effects $\Gamma_{XM}$ and $\Gamma_{MY}$ in the anticausal prediction task, or $\Gamma_{MX}$ and $\Gamma_{YM}$ in the causal one.

4.2.3. The connection between covariances and causal effects in the multivariate general case

Here, we extend the univariate results of Section 4.1 to the multivariate case. The results for the anticausal and causal prediction tasks are presented, respectively, on Theorems 1 and 2 (which are proven in Section 9 in the Supplement). But, before we present the results, we first clarify that, in the multivariate case, the covariance between two vectors of random variables, $A = (A_1, \ldots, A_{N_A})^T$ and $B = (B_1, \ldots, B_{N_B})^T$, is given by the cross-covariance operator, $\text{Cov}(A, B)$, defined and the $N_A \times N_B$ matrix with elements $\text{Cov}(A_i, B_j)$. For the anticausal prediction task the following results hold.

**Theorem 1.** Consider an anticausal prediction task:

(i) Suppose the interest focus on the causal effects generated by the paths in the set path $Y \rightarrow X$. If $X^*$ is given by $X^* = \Gamma_{XY} Y + W_X$, then $\text{Cov}(X^*, Y) = \Gamma_{XY}$.

(ii) Suppose the interest focus on the causal effects generated by the paths in

---

9For instance, for the example in Figure 7b, when we fit the regression model $X_1 = \sum_{k=1}^3 \gamma_{X_1C_k}C_k + \sum_{k=1}^2 \gamma_{X_1M_k}M_k + \gamma_{X_1Y}Y + W_{X_1}$ we have that only $\hat{\gamma}_{X_1C_2}$ and $\hat{\gamma}_{X_1M_2}$ will be statistically different from 0, since only $C_2$ and $M_2$ are parents of $X_1$ in the DAG in Figure 7b. Since by the Markov property of DAGs we have that given its parents a node is independent of all its non-descendants, it follows that conditional on $C_2$ and $M_2$, $X_1$ is independent of all other nodes and, consequently, all other $\hat{\gamma}$s should vanish towards zero (for large enough sample sizes).
the set path \( Y \rightarrow M \rightarrow X \). If \( X^* \) is given by \( X^* = \Gamma_{XM} M^* + W_X \), and \( M^* = \Gamma_{MY} Y + W_M \), then \( \text{Cov}(X^*, Y) = \Gamma_{XM} \Gamma_{MY} \).

(iii) Suppose the interest focus on the spurious associations generated by the paths in the set path \( X \leftarrow C \rightarrow Y \). If \( X^* \) is given by \( X^* = \Gamma_{XC} C + W_X \), then \( \text{Cov}(X^*, Y) = \Gamma_{XC} \text{Cov}(C) \Gamma_{YC}^T \).

Note that, in the univariate case, results i, ii, and iii above reduce, respectively, to the results shown in equations (1), (2), and (3), since \( \Gamma_{XY} = \gamma_{XY} \), \( \Gamma_{XM} = \gamma_{XM} = \theta_{XM} \), \( \Gamma_{XC} = \gamma_{XC} = \theta_{XC} \), \( \text{Cov}(C) = 1 \), and \( \Gamma_{YC} = \gamma_{YC} = \theta_{YC} \).

Now, for the causal prediction task the following results hold.

**Theorem 2.** Consider a causal prediction task:

(i) Suppose the interest focus on the causal effects generated by the paths in the set path \( X \rightarrow Y \). If \( Y^* \) is given by \( Y^* = \Gamma_{YX} X + W_Y \), then \( \text{Cov}(Y^*, X) = \Gamma_{YX} \text{Cov}(X) \).

(ii) Suppose the interest focus on the causal effects generated by the paths in the set path \( X \rightarrow M \rightarrow Y \). If \( Y^* \) is given by \( Y^* = \Gamma_{YM} M^* + W_Y \), and \( M^* = \Gamma_{MX} X + W_M \), then \( \text{Cov}(Y^*, X) = \Gamma_{YM} \Gamma_{MX} \text{Cov}(X) \).

(iii) Suppose the interest focus on the spurious associations generated by the paths in the set path \( X \leftarrow C \rightarrow Y \). If \( Y^* \) is given by \( Y^* = \Gamma_{YC} C + W_Y \), then \( \text{Cov}(Y^*, X) = \Gamma_{YC} \text{Cov}(C) \Gamma_{XC}^T \).

Again, in the univariate case, results (i) to (iii) in Theorem 2 reduce to the results presented in Section 4.1 (note that \( \text{Cov}(X) \) also reduces to 1). Observe, as well, that results (i) and (ii) show that, in addition to the direct causal effect (\( \Gamma_{YX} \), in result i) and the indirect causal effect (\( \Gamma_{YM} \Gamma_{MX} \), in result ii) the marginal covariances between the elements of \( X \) and \( Y^* \) also depend on \( \text{Cov}(X) \). This makes sense, since \( \text{Cov}(X) \) captures the associations between the elements of \( X^{10} \). Note that for each element \( X_j \) of \( X \), the operation \( \Gamma_{YX} \text{Cov}(X) \) captures not only the association generated by the direct causal path \( X_j \rightarrow Y^* \), but also the association generated by indirect and backdoor paths that start at \( X_j \) and end at \( Y^* \), but where the last node prior to \( Y^* \) is another element \( X_k \) of \( X \).

As an illustration, consider the DAG describing the causal prediction task in Figure 8a, where \( \text{Cov}(X) \),

\[
\begin{pmatrix}
1 \\
\theta_{X_2X_1} + \theta_{X_1C_1} \theta_{X_2C_1} \\
\theta_{X_2X_1} + \theta_{X_1C_1} \theta_{X_2C_1} \\
\theta_{X_2X_1} + \theta_{X_1C_1} \theta_{X_2C_1} \\
\end{pmatrix}
\]

In this example, the association between \( X_1 \) and \( X_2 \),

\[
\text{Cov}(X_1, X_2) = \frac{\theta_{X_2X_1} + \theta_{X_1C_1} \theta_{X_2C_1}}{X_1 \rightarrow X_2} \frac{X_1 \leftarrow C_1 \rightarrow X_2}
\]

\(^{10}\)Note that the associations between the elements of \( X \) might be generated not only by causal relations between the elements of \( X \) among themselves, but also by causal relations between the elements of \( C \) and \( X \). (Recall, that \( C \) is a parent of \( X \) and, as described in Section 3, the set path \( C \rightarrow X \) might contain backdoor paths that start at an element of \( X \), go through \( C \), and then return to another element of \( X \).)
is generated by the paths $X_1 \rightarrow X_2$ and $X_1 \leftarrow C_1 \rightarrow X_2$. In the direct causal

\begin{figure}
\centering
\includegraphics[width=0.8\textwidth]{fig8}
\caption{A causal prediction task illustrative example.}
\end{figure}

effects case (result i) we have that,

$$
\text{Cov}(Y^*, X) = \Gamma_{YX} \text{Cov}(X) = (\theta_{YX_1}, \theta_{YX_2}) \text{Cov}(X)
$$

$$
= \left( \theta_{YX_1} + \theta_{X_2 X_1} \theta_{YX_2} + \theta_{X_1 C_1} \theta_{X_2 C_1} \theta_{YX_2}, \theta_{YX_2} + \theta_{X_2 X_1} \theta_{YX_1} + \theta_{X_2 C_1} \theta_{X_1 C_1} \theta_{YX_1} \right)^T.
$$

Note that the direct application of Wright’s path analysis to the diagram in Figure 8b shows that we can decompose the covariance of $X_1$ and $Y^*$,

$$
\text{Cov}(Y^*, X_1) = \theta_{YX_1} + \theta_{X_2 X_1} \theta_{YX_2} + \theta_{X_1 C_1} \theta_{X_2 C_1} \theta_{YX_2}, \quad X_1 \rightarrow Y
$$

$$
\text{Cov}(Y^*, X_2) = \theta_{YX_2} + \theta_{X_2 X_1} \theta_{YX_1} + \theta_{X_2 C_1} \theta_{X_1 C_1} \theta_{YX_1}, \quad X_2 \rightarrow Y
$$

in terms of the direct path $X_1 \rightarrow Y$, the indirect path $X_1 \rightarrow X_2 \rightarrow Y$, and the backdoor path $X_1 \leftarrow C_1 \rightarrow X_2 \rightarrow Y$. Similarly, the covariance of $X_2$ and $Y^*$,

$$
\text{Cov}(Y^*, X_2) = \theta_{YX_2} + \theta_{X_2 X_1} \theta_{YX_1} + \theta_{X_2 C_1} \theta_{X_1 C_1} \theta_{YX_1}, \quad X_2 \rightarrow Y
$$

$$
\text{Cov}(Y^*, X_2) = \theta_{YX_2} + \theta_{X_2 X_1} \theta_{YX_1} + \theta_{X_2 C_1} \theta_{X_1 C_1} \theta_{YX_1}, \quad X_2 \leftarrow C_1 \rightarrow X_1 \rightarrow Y
$$

can be decomposed in terms of the direct path $X_2 \rightarrow Y$, and the backdoor paths $X_2 \leftarrow X_1 \rightarrow Y^*$ and $X_2 \leftarrow C_1 \rightarrow X_1 \rightarrow Y$. (Note that all the indirect and backdoor paths in this example either start at $X_1$ and end at $X_2$ before connecting to $Y^*$, or start at $X_2$ and end at $X_1$ before connecting to $Y^*$.)

### 5. FINAL REMARKS

As described in Section 4.2.1 we can always reparameterize linear structural causal models in a way that the associations generated by the causal relations among the observed variables in the DAG subdiagrams is “pushed” to the unobserved error terms. Hence, even though the associations among the observed variables are still preserved (i.e., $\text{Cov}(X)$, $\text{Cov}(C)$, and $\text{Cov}(M)$ are still the same in the original or in the reparameterized model), knowledge about the causal relations between the elements of $X$ (and, similarly, for the elements of $C$ and $M$ among themselves) is no longer necessary when we work with the
reparameterized model. Together with the fact that we can obtain consistent estimates of causal effects in the reparameterized model when the confounders and mediators are observed (as described in Section 4.2.2), this implies that, in practice, we only need partial domain knowledge about which variables are potential confounders and mediators, and whether the prediction task goes in the causal or anticausal direction, in order to estimate the necessary causal effects and residuals that will be used to simulate the counterfactual data. Hence, in practice, no domain knowledge about the causal relations between the features, confounders, and mediators among themselves is necessary in order to implement the proposed counterfactual approach.

Hence, while the lack of domain knowledge about the DAG subdiagrams might, at first sight, be perceived as an unsurpassable difficulty, it turns out that the proposed reparameterization makes the approach practical.

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6. A note on alternative model modifications for simulating counterfactual data

In Section 4.1 in the main text we showed how to generate counterfactual data that contains only associations generated by the causal effects of interest. A natural question is whether alternative modifications of the causal diagram (other than the ones presented in Section 4.1) would also lead to counterfactual datasets containing only the associations due to the causal effects of interest. Here, we show that this is sometimes possible, and clarify that, for anticausal prediction tasks, the requirement for the intervention to work is that $Y$ is not altered by the intervention. Similarly, for causal prediction tasks the requirement is that $X$ is not altered by the intervention. (For the sake of brevity this analogous case is not shown here.)

We start with the case where the interest focus on the direct causal effects in anticausal predictive tasks. Here, the goal is to simulate counterfactual data where $\text{Cov}(X^*, Y^*) = \theta_{XY}$. Starting with examples involving confounding alone, consider first an alternative modification where we simulate data with the confounder variable $C$ set to a fixed value $c$, as described in the twin network in Figure S1a. Direct calculation shows that,

$$\text{Cov}(X^*, Y^*) = \text{Cov}(\theta_{XC} c + \theta_{XY} Y^* + U_X, Y^*)$$
$$= \theta_{XY} \text{Cov}(Y^*, Y^*) = \theta_{XY} \text{Var}(Y^*)$$
$$= \theta_{XY} \text{Var}(\theta_{YC} c + U_Y) = \theta_{XY} \text{Var}(U_Y)$$
$$= \theta_{XY}(1 - \theta^2_{YC}),$$

for any chosen value. (Note that $\text{Var}(U_Y) = 1 - \theta^2_{YC}$, since $1 = \text{Var}(Y) = \text{Var}(\theta_{YC} C + U_Y) = \theta^2_{YC} \text{Var}(C) + \text{Var}(U_Y) = \theta^2_{YC} + \text{Var}(U_Y)$.)

![Fig S1](image.png)

Fig S1. Alternative model modifications for the confounding only examples.

Now, consider another alternative modification where we drop the causal link $C \rightarrow Y$ (rather than $C \rightarrow X$) as shown in Figure S1b. Note that direct calculation of $\text{Cov}(X^*, Y^*)$ shows again that,

$$\text{Cov}(X^*, Y^*) = \text{Cov}(\theta_{XY} Y^* + \theta_{XC} C + U_X, Y^*)$$
$$= \theta_{XY} \text{Cov}(Y^*, Y^*)$$
$$= \theta_{XY} \text{Var}(Y^*) = \theta_{XY} \text{Var}(U_Y)$$
$$= \theta_{XY}(1 - \theta^2_{YC}).$$
Hence, we see that for both alternative modifications presented in Figure S1
the covariance between the response and the feature does not equal
$\theta_{XY}$, the
association due to the causal effect of $Y$ on $X$. (Note that in both examples the
intervention altered $Y$ to $Y^*$.)

Now, we show that for the mediation only example, these alternative modifi-
cations still capture the correct covariance because, in this case, these modi-
fications do not alter $Y$. For instance, by setting the mediator $M$ to the fixed
value $m$, as described in Figure S2a, we still have that,

$$\text{Cov}(X^*,Y^*) = \text{Cov}(X^*,Y) =
\text{Cov}(\theta_{XY} Y + \theta_{XM} m + U_X, Y)
= \theta_{XY} \text{Cov}(Y,Y) + \theta_{XM} \text{Cov}(m,Y) + \text{Cov}(U_X,Y)
= \theta_{XY} \text{Var}(Y) = \theta_{XY}. $$

Similarly, note that by dropping the causal link $Y \rightarrow M$ (rather than $M \rightarrow X$),
as described in Figure S2b, we still have that,

$$\text{Cov}(X^*,Y^*) = \text{Cov}(X^*,Y) =
\text{Cov}(\theta_{XY} Y + \theta_{XM} M^* + U_X, Y)
= \theta_{XY} \text{Cov}(Y,Y) + \theta_{XM} \text{Cov}(M^*,Y) + \text{Cov}(U_X,Y)
= \theta_{XY} \text{Var}(Y) = \theta_{XY}. $$

These examples show that for the mediation problem we don’t necessarily
need to simulate counterfactual features by dropping the $M$ as a parent of $X$.
From a practical point of view, however, it is still more advantageous to simulate
counterfactual features by dropping the causal link $M \rightarrow X$ since this approach
only requires the simulation of the counterfactual features, whereas the approach
described in Figure S2a requires us to set $M$ to $m$, and the approach in Figure
S2b requires the simulation of counterfactual mediator data, $M^*$, in addition to
the simulation of counterfactual feature data, $X^*$.

Now, let’s consider indirect causal effects in anticausal prediction tasks. Here,
the goal is to simulate counterfactual data where $\text{Cov}(X^*,Y^*) = \theta_{XM} \theta_{MY}$.
Consider first the alternative intervention where we remove the link $C \rightarrow Y$
(rather than $C \rightarrow X$) shown in Figure S3a.
Note that, in this case, the intervention altered $Y$ and we have that,

\[
\operatorname{Cov}(X^*, Y^*) = \operatorname{Cov}(\theta_{XC}C + \theta_{XM}M^* + U_X, U_Y) \\
= \theta_{XM} \operatorname{Cov}(M^*, U_Y) \\
= \theta_{XM} \operatorname{Cov}(\theta_{MY}Y^* + U_M, U_Y) \\
= \theta_{XM} \theta_{MY} \operatorname{Cov}(Y^*, U_Y) = \theta_{XM} \theta_{MY} \operatorname{Var}(U_Y) \\
= \theta_{XM} \theta_{MY} \left(1 - \theta_{Y|C}^2\right).
\]

Similarly, for the intervention where we set $C$ to $c$ we also altered $Y$ and we have that,

\[
\operatorname{Cov}(X^*, Y^*) = \operatorname{Cov}(\theta_{XC}C + \theta_{XM}M^* + U_X, Y^*) \\
= \theta_{XM} \operatorname{Cov}(M^*, Y^*) \\
= \theta_{XM} \operatorname{Cov}(\theta_{MY}Y^* + U_M, Y^*) \\
= \theta_{XM} \theta_{MY} \operatorname{Var}(Y^*) = \theta_{XM} \theta_{MY} \operatorname{Var}(U_Y) \\
= \theta_{XM} \theta_{MY} \left(1 - \theta_{Y|C}^2\right).
\]

7. Node-splitting transformations as alternative interventions

In this section we show that the adoption of node-splitting transformations (Richardson and Robins 2013) encoded in single world intervention graphs (SWIGs) can also be used as an alternative intervention for the generation of counterfactual data that contains only the associations generated by the causal mechanisms of interest. Here, we present SWIGs that capture exactly the same marginal associations between the counterfactual features and responses, as the twin-networks presented in Figures 3, 4, and 5, in the main text.

Figure S4 presents the SWIGs for the generation of counterfactual features in the anticausal prediction tasks. Here, a node-split operation associated with the intervention $\text{do}(Z = z)$ is represented by splitting the node $Z$ into two elements: $\{\hat{Z}\}$ representing the instantiation of $Z$ to the fixed value $z$; and $\{\hat{Z}\}$ representing the random variable $Z$. 
In Figure S4a we split the $C$ and $M$ nodes in order to obtain a counterfactual feature $X_{c,m,Y}$, whose association with $Y$ is generated by the direct causal effect $\theta_{XY}$, since for any fixed values of $c$ and $m$ we have that,

$$\begin{align*}
\text{Cov}(X_{c,m,Y}, Y) &= \\
&= \text{Cov}(\theta_{XC} c + \theta_{XM} m + \theta_{XY} Y + U_X, Y) \\
&= \theta_{XY} \text{Cov}(Y, Y) = \theta_{XY}.
\end{align*}$$

In Figure S4b we split the $C$ and $Y$ nodes in order to obtain a counterfactual feature $X_{y,c,M_{c,Y}}$, whose association with $Y$ is generated by the indirect causal effect $\theta_{XM} \theta_{MY}$, since for any fixed values of $c$ and $y$ we have that,

$$\begin{align*}
\text{Cov}(X_{y,c,M_{c,Y}}, Y) &= \\
&= \text{Cov}(\theta_{XC} c + \theta_{XY} y + \theta_{XM} M_{c,Y} + U_X, Y) \\
&= \theta_{XM} \text{Cov}(M_{c,Y}, Y) \\
&= \theta_{XM} \text{Cov}(\theta_{MC} c + \theta_{MY} Y + U_M, Y) \\
&= \theta_{XM} \theta_{MY} \text{Cov}(Y, Y) = \theta_{XM} \theta_{MY}.
\end{align*}$$

Finally, in Figure S4c we split the $Y$ and $M$ nodes in order to obtain a counterfactual feature $X_{y,m,C}$, whose association with $Y$, measured by $\theta_{XC} \theta_{YC}$, is generated by the confounder $C$. Note that for any fixed values of $y$ and $m$ we
have that,
\[
\text{Cov}(X_{y,m,C}, Y) = \\
= \text{Cov}(\theta_{XC} C + \theta_{XM} m + \theta_{XY} y + U_X, Y) \\
= \theta_{XC} \text{Cov}(C, Y) \\
= \theta_{XC} \text{Cov}(C, \theta_{YC} C + U_Y) \\
= \theta_{XC} \theta_{YC} \text{Cov}(C, C) = \theta_{XC} \theta_{YC} .
\]

Similarly, Figure S5 shows the SWIGs for the generation of counterfactual responses in the causal prediction tasks. Direct calculation of the covariances shows that, \( \text{Cov}(X, Y_{c,m,X}) = \theta_{YX} \) for the SWIG in panel a, \( \text{Cov}(X, Y_{x,c,M_c,X}) = \theta_{YM} \theta_{MX} \) for the SWIG in panel b, and \( \text{Cov}(X, Y_{x,m,C}) = \theta_{XC} \theta_{YC} \) for the SWIG in panel c.

Note that in the SWIG framework, even when we split the \( Y \) node into \( \{\overline{Y}\} \) and \( \{\overline{\overline{Y}}\} \) in anticausal prediction tasks (e.g., Figure S4b and c), we have that the component \( \{\overline{Y}\} \) still represents the un-altered random variable \( Y \). Similarly, when we split \( X \) node into \( \{\overline{X}\} \) and \( \{\overline{\overline{X}}\} \) in causal prediction tasks (e.g., Figure S5b and c), we still have that the component \( \{\overline{X}\} \) is still un-altered.

Observe, as well, that while the SWIG framework is also able to capture the covariances generated by the causal relations of interest, it requires the adoption of more complicated counterfactual statements than our proposed approach. For instance, for the example in Figure S4a, we have that \( X_{c,m,Y} \) corresponds to the counterfactual statement “the value that \( X \) would have attained, had we set \( C \)
to \( c \), and \( M \) to \( m \), while leaving \( Y \) to assume the value it would have naturally assumed had we not intervened on \( C \). On the other hand, for our approach the counterfactual feature \( X^* \) (generated according to the twin network in Figure 3a) corresponds to the simpler statement “the value that \( X \) would have attained, if \( C \) was not a confounder and \( M \) was not a mediator of the causal effect of \( Y \) on \( X \)”.

When the goal is to obtain static ML predictions, it seems perhaps more natural to think about alternative counterfactual worlds where data generation mechanism is changed, than to adopt the more complicated counterfactuals associated with node splitting. In practice, however, it doesn’t matter which approach seems more natural, since in both cases we can still simulate counterfactual data containing only the associations generated by the causal mechanisms of interest.

8. On the differences from backdoor adjustment for confounding

At this point, a potential question a reader might have is whether our proposed counterfactual distribution is equivalent to the distribution generated by backdoor adjustment in the case that \( C \) is an observed confounder. In this section, we clarify that this is not the case.

Note that in Pearl’s approach, the effect of an intervention (such as setting the value of \( X \) to \( x \)) is modeled by replacing the structural causal equation \( X = f(pa(X),U_x) \) by the equation \( X = x \). Under Pearl’s approach (and under the assumption that the observed confounder \( C \) is the only confounder) we have that the counterfactual probability \( P(Y = y|do(X = x)) = \sum_c P(Y = y|X = x,C = c)P(C = c) \).

However, in our approach the counterfactual distributions are not generated by \( do(X = x) \) interventions. Instead of replacing \( X = f(pa(X),U_X) \) by \( X = x \), we actually remove \( C \) from the parent set of \( X \) by replacing \( X = f(pa(X) \setminus C,U_X) \) in anticausal prediction. (Similarly, we replace \( Y = f(pa(Y),U_Y) \) by \( Y = f(pa(Y) \setminus C,U_Y) \) in causal prediction tasks.)

For concreteness, we now derive the backdoor and our counterfactual distributions for the toy regression task example described in Figure S6a, where \( X \) has a causal effect on \( Y \), and we assume that the model is parameterized by the following set of structural equations,

\[
C = U_C
\]
\[
X = \beta_{XC} C + U_X
\]
\[
Y = \beta_{YC} C + \beta_{XY} X + U_Y
\]

where \( U_C \sim \text{Bernoulli}(p) \), \( U_X \sim N(0,\sigma_X^2) \) and \( U_Y \sim N(0,\sigma_Y^2) \). In addition to showing that the distributions are different, we will also show that for the backdoor-adjusted distribution, the covariance between \( X \) and the counterfactual response, \( Y^*_x \), do not capture the association generated by the causal effect.
of $X$ on $Y$. (Note that here we are using a slightly different notation to differentiate the counterfactual generated by the backdoor adjustment, $Y^b_x$, from the counterfactual generated by our proposed approach, $Y^*$.)

From the backdoor adjustment formula,

$$P(Y|do(X = x)) = P(Y|X = x, C = 0)P(C = 0) + P(Y|X = x, C = 1)P(C = 1),$$

we have that the backdoor distribution $P(Y|do(X = x))$ corresponds to a mixture of the conditional distributions,

$$P(Y|X = x, C = 0) \sim N(\beta_{YX} x, \sigma_Y^2),$$
$$P(Y|X = x, C = 1) \sim N(\beta_{YC} + \beta_{YX} x, \sigma_Y^2),$$

and has mean and variance given by,

$$E[Y^b_x] = E[Y|do(X = x)] = p \beta_{YC} + \beta_{YX} x,$$
$$Var[Y^b_x] = Var[Y|do(X = x)] = \sigma_Y^2 + p(1-p) \beta_{YX}^2.$$

Note that, as expected, the average causal effect of $X$ on $Y$, is given by,

$$ACE = E[Y^b_{x+1}] - E[Y^b_x] \tag{4}$$
$$= E[Y|do(X = x' + 1)] - E[Y|do(X = x')]$$
$$= p \beta_{YC} + \beta_{YX} (x' + 1) - p \beta_{YC} - \beta_{YX} x'$$
$$= \beta_{YX}.$$

Figure S6b shows the modified causal graph for the intervention $do(X = x')$. Figure S6c shows the respective twin network. From the twin network it is easy
to see that

\[
\text{Cov}(X, Y_b^2) = \\
= \text{Cov}(\beta_{XC} C + U_X, \beta_{YX} X' + \beta_{YC} C + U_Y) \\
= \text{Cov}(\beta_{XC} C + U_X, \beta_{YX} X' + \beta_{YC} C + U_Y) \\
= \beta_{XC} \beta_{YC} \text{Var}(C) .
\]

On the other hand, our proposed approach generates a counterfactual response, \(Y^*\), as described by the causal graph on Figure S6d, where \(C\) is no longer a parent of \(Y\). Figure S6e shows the respective twin network. For this model, note that \(Y^*\) can be re-expressed as,

\[
Y^* = \beta_{YX} X + U_Y = \beta_{YX} \beta_{XC} C + \beta_{YX} U_X + U_Y ,
\]

so that its expectation and variance are given by,

\[
E[Y^*] = \mu \beta_{YX} \beta_{XC} \\
\text{Var}[Y^*] = \sigma_Y^2 + \beta^2_{YX} \sigma_X^2 + \beta^2_{XC} \mu(1 - \mu) .
\]

This shows that the distribution of our counterfactual response is different from the backdoor adjusted distribution. Furthermore, from the twin network in Figure S6e we have that,

\[
\text{Cov}(X, Y^*) = \text{Cov}(X, \beta_{YX} X' + U_Y) \\
= \text{Cov}(X, \beta_{YX} X + U_Y) \\
= \beta_{YX} \text{Var}(X) .
\]

Assuming that the data has been standardized (so that \(\text{Var}(C) = \text{Var}(X) = 1\)) we have that \(\text{Cov}(X, Y_b^2) = \theta_{XC} \theta_{YC}\), whereas \(\text{Cov}(X, Y^*) = \theta_{YX}\) (where, as before, \(\theta_{VW} = \beta_{VW} (\text{Var}(W)/\text{Var}(V))^{1/2}\) represents Wright’s path coefficient). These results show that while our counterfactual distribution only captures the association between \(X\) and \(Y\) that is generated by the causal effect of \(X\) on \(Y\) (i.e., \(\theta_{YX}\)), the backdoor adjusted distribution is actually capturing the spurious association generated by the confounder (i.e., \(\theta_{XC} \theta_{YC}\)). This example shows that not only the backdoor adjusted distribution is different from our counterfactual distribution, but it also does not capture the association between \(X\) and \(Y\) that is generated by the causal effect of \(X\) on \(Y\).

While the backdoor adjusted distribution is used to estimate the directed causal effect \(\beta_{YX}\) (or \(\theta_{YX}\) for standardized data), this is done by taking into consideration a difference in expectations from two interventions \(\text{do}(X = x' + 1)\) and \(\text{do}(X = x')\), namely, \(E[Y_b^2_{x'+1}] - E[Y_b^2_{x'}]\), as described in equation (4). The variance of \(Y_b^2\) (or its covariance with \(X\)) does not play a direct role in the estimation of the causal effects. These observations illustrate that the proposed task of building “confounding aware” machine learning predictions is different from performing backdoor adjustment on the observed confounders.
9. Proofs of Theorems 1 and 2

For the proof of Theorems 1 and 2 we will use the following properties of the cross-covariance operator:

1. $\text{Cov}(Z_1 + Z_2, Z_3) = \text{Cov}(Z_1, Z_3) + \text{Cov}(Z_2, Z_3)$,
2. $\text{Cov}(AZ_1, BZ_2) = A \text{Cov}(Z_1, Z_2) B^T$, where $A$ and $B$ are constant matrices
3. $\text{Cov}(Z, Z) = \text{Cov}(Z)$, where $\text{Cov}(Z)$ is the variance covariance matrix of $Z$.

The proofs are straightforward, and follow directly from the above three properties.

9.1. Proof of Theorem 1

Proof.

Result $i$: If $X^* = \Gamma_{XY} Y + W_X$, then,

$$\text{Cov}(X^*, Y) = \text{Cov}(\Gamma_{XY} Y + W_X, Y)$$
$$= \Gamma_{XY} \text{Cov}(Y, Y)$$
$$= \Gamma_{XY}$$

Result $ii$: If $X^* = \Gamma_{XM} M^* + W_X$ and $M^* = \Gamma_{MY} Y + W_M$, then,

$$\text{Cov}(X^*, Y) = \text{Cov}(\Gamma_{XM} M^* + W_X, Y)$$
$$= \Gamma_{XM} \text{Cov}(fM^*, Y)$$
$$= \Gamma_{XM} \text{Cov}(\Gamma_{MY} Y + W_M, Y)$$
$$= \Gamma_{XM} \Gamma_{MY} \text{Var}(Y)$$
$$= \Gamma_{XM} \Gamma_{MY}$$

Result $iii$: If $X^* = \Gamma_{XC} C + W_X$, then,

$$\text{Cov}(X^*, Y) = \text{Cov}(\Gamma_{XC} C + W_X, Y)$$
$$= \Gamma_{XC} \text{Cov}(C, Y)$$
$$= \Gamma_{XC} \text{Cov}(C, \Gamma_{YC} C + W_Y)$$
$$= \Gamma_{XC} \text{Cov}(C, C) \Gamma_{YC}^T$$
$$= \Gamma_{XC} \text{Cov}(C) \Gamma_{YC}^T$$

\qed

9.2. Proof of Theorem 2

Proof.
Result i: If $Y^* = \Gamma_{YX} X + W_Y$, then,

\[ \text{Cov}(Y^*, X) = \text{Cov}(\Gamma_{YX} X + W_Y, X) \]
\[ = \Gamma_{YX} \text{Cov}(X, X) \]
\[ = \Gamma_{YX} \text{Cov}(X) \]

Result ii: If $Y^* = \Gamma_{YM} M^* + W_Y$ and $M^* = \Gamma_{MX} X + W_M$, then,

\[ \text{Cov}(Y^*, X) = \text{Cov}(\Gamma_{YM} M^* + W_Y, X) \]
\[ = \Gamma_{YM} \text{Cov}(X, M^*) \]
\[ = \Gamma_{YM} \text{Cov}(\Gamma_{MX} X + W_M, X) \]
\[ = \Gamma_{YM} \Gamma_{MX} \text{Cov}(X, X) \]
\[ = \Gamma_{YM} \Gamma_{MX} \text{Cov}(X) \]

Result iii: If $Y^* = \Gamma_{YC} C + W_Y$, then,

\[ \text{Cov}(Y^*, X) = \text{Cov}(\Gamma_{YC} C + W_Y, X) \]
\[ = \Gamma_{YC} \text{Cov}(C, X) \]
\[ = \Gamma_{YC} \text{Cov}(C, \Gamma_{XC} C + W_X) \]
\[ = \Gamma_{YC} \text{Cov}(C, C) \Gamma_{XC}^T \]
\[ = \Gamma_{YC} \text{Cov}(C) \Gamma_{XC}^T \]

10. Reparameterization of the anticausal prediction task presented in Figure 7 in the main text

For the anticausal prediction task DAG in Figure 7a, we have that the structural equations,

\[ C = \Theta_{CC} C + U_C, \]
\[ Y = \Theta_{YC} C + U_Y, \]
\[ M = \Theta_{MM} M + \Theta_{MC} C + \Theta_{MY} Y + U_M, \]
\[ X = \Theta_{XX} X + \Theta_{XC} C + \Theta_{XM} M + \Theta_{XY} Y + U_X, \]
are explicitly given by,

\[
\begin{pmatrix}
C_1 \\
C_2 \\
C_3
\end{pmatrix} =
\begin{pmatrix}
0 & \theta_{C_1C_2} & 0 \\
0 & 0 & 0 \\
\theta_{C_3C_1} & \theta_{C_3C_2} & 0
\end{pmatrix}
\begin{pmatrix}
C_1 \\
C_2 \\
C_3
\end{pmatrix}
+ 
\begin{pmatrix}
UC_1 \\
UC_2 \\
UC_3
\end{pmatrix},
\]

\[
Y = \begin{pmatrix}
0 & 0 & \theta_{YC_3}
\end{pmatrix}
\begin{pmatrix}
C_1 \\
C_2 \\
C_3
\end{pmatrix} + U_Y,
\]

\[
\begin{pmatrix}
M_1 \\
M_2
\end{pmatrix} =
\begin{pmatrix}
0 & \theta_{M_1M_2} \\
0 & 0
\end{pmatrix}
\begin{pmatrix}
M_1 \\
M_2
\end{pmatrix}
+ 
\begin{pmatrix}
0 & 0 & 0 \\
0 & 0 & \theta_{M_2C_3}
\end{pmatrix}
\begin{pmatrix}
C_1 \\
C_2 \\
C_3
\end{pmatrix}
+ 
\begin{pmatrix}
0 \\
\theta_{M_2Y}
\end{pmatrix}Y 
+ 
\begin{pmatrix}
UM_1 \\
UM_2
\end{pmatrix},
\]

\[
\begin{pmatrix}
X_1 \\
X_2 \\
X_3
\end{pmatrix} =
\begin{pmatrix}
\theta_{X_2X_1} & 0 & 0 \\
\theta_{X_3X_1} & \theta_{X_3X_2} & 0 \\
\theta_{X_3M_1} & 0 & 0
\end{pmatrix}
\begin{pmatrix}
X_1 \\
X_2 \\
X_3
\end{pmatrix}
+ 
\begin{pmatrix}
0 & \theta_{X_1C_2} & 0 \\
0 & 0 & 0 \\
0 & 0 & 0
\end{pmatrix}
\begin{pmatrix}
C_1 \\
C_2 \\
C_3
\end{pmatrix}
+ 
\begin{pmatrix}
0 \\
\theta_{XM_2}
\end{pmatrix}Y 
+ 
\begin{pmatrix}
UX_1 \\
UX_2 \\
UX_3
\end{pmatrix}.
\]

Using simple algebraic manipulations, we can re-write the above linear structural models as,

\[
C = W_C,
\]

\[
Y = \Gamma_{YC}C + W_Y,
\]

\[
M = \Gamma_{MC}C + \Gamma_{MY}Y + W_M,
\]

\[
X = \Gamma_{XC}C + \Gamma_{XM}M + \Gamma_{XY}Y + W_X,
\]

where,

\[
W_C = (I - \Theta_{CC})^{-1}U_C,
\]

\[
W_Y = U_Y,
\]

\[
W_M = (I - \Theta_{MM})^{-1}U_M,
\]

\[
W_X = (I - \Theta_{XX})^{-1}U_X.
\]
and,

\[ \Gamma_{YC} = \Theta_{YC} \]
\[ \Gamma_{MC} = (I - \Theta_{MM})^{-1}\Theta_{MC} , \]
\[ \Gamma_{MY} = (I - \Theta_{MM})^{-1}\Theta_{MY} , \]
\[ \Gamma_{XC} = (I - \Theta_{XX})^{-1}\Theta_{XC} , \]
\[ \Gamma_{XM} = (I - \Theta_{XX})^{-1}\Theta_{XM} , \]
\[ \Gamma_{XY} = (I - \Theta_{XX})^{-1}\Theta_{XY} . \]

Next, present the explicit form of parameters and error terms for the particular example in Figure 7. Starting with model \( C = W_C \), we have that,

\[ (I - \Theta_{CC})^{-1} = \begin{pmatrix} 1 & \theta_C c_1 c_2 & 0 \\ 0 & 1 & 0 \\ \theta_C c_3 c_1 & \theta_C c_3 c_2 + \theta_C c_3 c_1 \theta_C c_1 c_2 & 1 \end{pmatrix}, \]

so that,

\[ W_C = (I - \Theta_{CC})^{-1} U_C \]
\[ = \begin{pmatrix} 1 & \theta_C c_1 c_2 & 0 \\ 0 & 1 & 0 \\ \theta_C c_3 c_1 & \theta_C c_3 c_2 + \theta_C c_3 c_1 \theta_C c_1 c_2 & 1 \end{pmatrix} \begin{pmatrix} U_C \end{pmatrix} \]
\[ = \begin{pmatrix} U_C + \theta_C c_1 c_2 U_C \\ U_C + \theta_C c_3 c_1 + \theta_C c_3 c_2 + \theta_C c_3 c_1 \theta_C c_1 c_2 + U_C \theta_C c_3 c_1 \\ U_C \end{pmatrix} \]
\[ = \begin{pmatrix} W_C_1 \\ W_C_2 \\ W_C_3 \end{pmatrix}, \]

For the model \( Y = \Gamma_{YC} C + W_Y \), we have that,

\[ \Gamma_{YC} = \Theta_{YC} \]
\[ = \begin{pmatrix} 0 & 0 & \theta_Y c_3 \end{pmatrix} \]
\[ = \begin{pmatrix} \gamma_Y c_1 & \gamma_Y c_2 & \gamma_Y c_3 \end{pmatrix}, \]
\[ W_Y = U_Y. \]

For the model \( M = \Gamma_{MC} C + \Gamma_{MY} Y + W_M \), we have that,

\[ (I - \Theta_{MM})^{-1} = \begin{pmatrix} 1 & \theta_M m_1 m_2 \\ 0 & 1 \end{pmatrix}, \]
\[ \mathbf{\Gamma}_{MC} = (\mathbf{I} - \mathbf{\Theta}_{MM})^{-1} \mathbf{\Theta}_{MC} \]
\[ = \begin{pmatrix} 1 & \theta_{M_1M_2} \\ 0 & 1 \end{pmatrix} \begin{pmatrix} 0 & 0 & 0 \\ 0 & 0 & \theta_{M_2C_3} \end{pmatrix} \]
\[ = \begin{pmatrix} \gamma_{M_1C_1} & \gamma_{M_1C_2} & \gamma_{M_1C_3} \\ \gamma_{M_2C_1} & \gamma_{M_2C_2} & \gamma_{M_2C_3} \end{pmatrix}, \]

and,
\[ \mathbf{\Gamma}_{MY} = (\mathbf{I} - \mathbf{\Theta}_{MM})^{-1} \mathbf{\Theta}_{MY} \]
\[ = \begin{pmatrix} 1 & \theta_{M_1M_2} \\ 0 & 1 \end{pmatrix} \begin{pmatrix} 0 \\ \theta_{M_2Y} \end{pmatrix} \]
\[ = \begin{pmatrix} \theta_{M_1M_2} \theta_{M_2Y} \\ \theta_{M_2Y} \end{pmatrix} = \begin{pmatrix} \gamma_{M_1Y} \\ \gamma_{M_2Y} \end{pmatrix}, \]

and,
\[ \mathbf{W}_M = (\mathbf{I} - \mathbf{\Theta}_{MM})^{-1} \mathbf{U}_M \]
\[ = \begin{pmatrix} 1 & \theta_{M_1M_2} \\ 0 & 1 \end{pmatrix} \begin{pmatrix} \mathbf{U}_{M_1} \\ \mathbf{U}_{M_2} \end{pmatrix} \]
\[ = \begin{pmatrix} \mathbf{U}_{M_1} + \theta_{M_1M_2} \mathbf{U}_{M_2} \\ \mathbf{U}_{M_2} \end{pmatrix} = \begin{pmatrix} \mathbf{W}_{M_1} \\ \mathbf{W}_{M_2} \end{pmatrix}. \]

Finally, for the model \( \mathbf{X} = \mathbf{\Gamma}_{XC} \mathbf{C} + \mathbf{\Gamma}_{XM} \mathbf{M} + \mathbf{\Gamma}_{XY} \mathbf{Y} + \mathbf{W}_X \), we have that,
\[ (\mathbf{I} - \mathbf{\Theta}_{XX})^{-1} = \begin{pmatrix} 1 & 0 & 0 \\ \theta_{X_2X_1} & 1 & 0 \\ \theta_{X_3X_1} + \theta_{X_2X_1} \theta_{X_3X_2} & \theta_{X_3X_2} & 1 \end{pmatrix}, \]

so that,
\[ \mathbf{\Gamma}_{XC} = (\mathbf{I} - \mathbf{\Theta}_{XX})^{-1} \mathbf{\Theta}_{XC} \]
\[ = \begin{pmatrix} 1 & 0 & 0 \\ \theta_{X_2X_1} & 1 & 0 \\ \theta_{X_3X_1} + \theta_{X_2X_1} \theta_{X_3X_2} & \theta_{X_3X_2} & 1 \end{pmatrix} \begin{pmatrix} 0 & 0 & \theta_{X_1C_2} \\ 0 & 0 & 0 \\ 0 & 0 & \theta_{X_1C_2} \theta_{X_2X_1} + \theta_{X_1C_2} \theta_{X_2X_1} \theta_{X_3X_2} \end{pmatrix} \]
\[ = \begin{pmatrix} \gamma_{X_1C_1} & \gamma_{X_1C_2} & \gamma_{X_1C_3} \\ \gamma_{X_2C_1} & \gamma_{X_2C_2} & \gamma_{X_2C_3} \\ \gamma_{X_3C_1} & \gamma_{X_3C_2} & \gamma_{X_3C_3} \end{pmatrix}, \]
and,

$$\Gamma_{XM} = (I - \Theta_{XX})^{-1} \Theta_{XM}$$

$$= \begin{pmatrix}
1 & 0 & 0 \\
\theta_{X_2 X_1} & 1 & 0 \\
\theta_{X_3 X_1} + \theta_{X_2 X_1} \theta_{X_3 X_2} & \theta_{X_3 X_2} & 1
\end{pmatrix}
\begin{pmatrix}
0 & \theta_{X_1 M_2} \\
0 & \theta_{X_1 M_2} \theta_{X_2 X_1} \\
\theta_{X_3 M_1} & \theta_{X_3 M_2} \theta_{X_2 X_1} \theta_{X_3 X_2}
\end{pmatrix}
= \begin{pmatrix}
\gamma_{X_1 M_1} & \gamma_{X_1 M_2} \\
\gamma_{X_2 M_1} & \gamma_{X_2 M_2} \\
\gamma_{X_3 M_1} & \gamma_{X_3 M_2}
\end{pmatrix},$$

and,

$$\Gamma_{XY} = (I - \Theta_{XX})^{-1} \Theta_{XY}$$

$$= \begin{pmatrix}
1 & 0 & 0 \\
\theta_{X_2 X_1} & 1 & 0 \\
\theta_{X_3 X_1} + \theta_{X_2 X_1} \theta_{X_3 X_2} & \theta_{X_3 X_2} & 1
\end{pmatrix}
\begin{pmatrix}
0 & \theta_{X_2 Y} \\
0 & \theta_{X_2 Y} \\
\theta_{X_3 Y} + \theta_{X_3 X_2} \theta_{X_2 Y}
\end{pmatrix}
= \begin{pmatrix}
\gamma_{X_1 Y} \\
\gamma_{X_2 Y} \\
\gamma_{X_3 Y}
\end{pmatrix},$$

and,

$$W_{X} = (I - \Theta_{XX})^{-1} U_{X}$$

$$= \begin{pmatrix}
1 & 0 & 0 \\
\theta_{X_2 X_1} & 1 & 0 \\
\theta_{X_3 X_1} + \theta_{X_2 X_1} \theta_{X_3 X_2} & \theta_{X_3 X_2} & 1
\end{pmatrix}
\begin{pmatrix}
U_{X_1} \\
U_{X_2} \\
U_{X_3}
\end{pmatrix}
= \begin{pmatrix}
U_{X_1} \\
U_{X_1} \theta_{X_2 X_1} + U_{X_2} \\
U_{X_1} (\theta_{X_3 X_1} + \theta_{X_2 X_1} \theta_{X_3 X_2}) + U_{X_2} \theta_{X_3 X_2} + U_{X_3}
\end{pmatrix}
= \begin{pmatrix}
W_{U_1} \\
W_{U_2} \\
W_{U_3}
\end{pmatrix} .$$

Table 1 in the main text lists all the elements of $\Gamma_{YC}$, $\Gamma_{MC}$, $\Gamma_{MY}$, $\Gamma_{XC}$, $\Gamma_{XM}$, and $\Gamma_{XY}$.