Causal Bias Quantification for Continuous Treatment

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Abstract

In this work we develop a novel characterization of marginal causal effect and causal bias in the continuous treatment setting. We show they can be expressed as an expectation with respect to a conditional probability distribution, which can be estimated via standard statistical and probabilistic methods. All terms in the expectations can be computed via automatic differentiation, also for highly non-linear models. We further develop a new complete criterion for identifiability of causal effects via covariate adjustment, showing the bias equals zero if the criterion is met. We study the effectiveness of our framework in three different scenarios: linear models under confounding, overcontrol and endogenous selection bias; a non-linear model where full identifiability cannot be achieved because of missing data; a simulated medical study of statins and atherosclerotic cardiovascular disease.

1 Introduction

Motivation. Our goal is to compare the value of different treatments in a counterfactual setting. Typically, this involves a controlled experiment constructed such that no causal bias is introduced and, hence, any estimated association is also causal. However, in practice we may not be able to control for all confounders or cannot avoid observing factors that introduce other types of causal bias, such as overcontrol or endogenous selection. In such cases we must infer the causal effect from past observational data. This however requires more complex models as we must not only explain the relation of treatment and outcome, but also all other factors explaining the treatment assignment as well as any other observation causing or caused by the treatment and the outcome. In the language of probabilistic machine learning, we require a generative model of treatments and outcomes. While we can always state a model free of causal bias, this would require to make non-credible assumptions about the data generating process. Arguably, we must balance the suitability of modelling assumptions and the introduced causal bias. In particular, we would like to decide between any two models that explain the observed data equally well. This in turn requires to quantify the causal bias of a model.

Contribution. In this paper we offer a complete criterion for identifiability of causal effects via covariate adjustment in the framework of structural equation models (SEMs). We use it to develop closed-form characterizations of marginal causal effect $C$ and marginal causal bias $B$ in a continuous treatment framework, proving the bias must be zero if such criterion holds. The term marginal refers to small variations of the treatment, which can be expressed in terms of derivatives.

Given their characterizations, estimation of $C$ and $B$ reduces to the standard statistical task of estimating an expectation with respect to a conditional probability density. All terms within such expectations can be conveniently computed via automatic differentiation, which makes estimation feasible even for highly non-linear machine learning models.
Related work. While large part of the literature in causal inference focuses on discrete, primarily, binary treatments \cite{16,3}, a continuous treatment framework has been developed \cite{14,33}, closely related to the work on dose-response \cite{2,39}. Although in the literature it might assume several different names, the concept of marginal causal effect is not novel either \cite{37,15,31}. However, most existing works on continuous treatments assume that the causal effect is identifiable and, hence, the causal bias is zero. In this work we consider the scenario where the causal effect might be non-identifiable and study the mathematical relation between marginal association, marginal causal effect, and marginal causal bias in a continuous framework. Concretely, we develop a characterization of the bias that can be used to evaluate how far a model is from being causal. While different in methodology, a similar purpose can be found in \cite{58,12}. In addition, it can be used to perform sensitivity analysis in the presence of unobserved confounders in a completely non-linear framework, similarly to what is proposed in \cite{9} for the linear case.

We observe that the criterion for identifiability via covariate adjustment that we develop in the theory can be extended seamlessly. We further denote latent variables other than treatment by \(X\) throughout this paper we consider \(Pa(Vᵢ)\), \(An(Vᵢ)\) and \(De(Vᵢ)\) denote parents, ancestors and descendants of \(Vᵢ\) in \(D\), which by definition do not include \(Vᵢ\) (see \cite{24}).

We denote \(f_{Vᵢ}\) to be differentiable with respect to the treatment, plus some extra differentiability and invertibility assumptions in Theorem \cite{2,3} with inverse functions required for estimation. We remark, however, that many machine learning models satisfy these requirements, including highly non-linear models like invertible deep neural networks and normalizing flows \cite{28}.

Setting. We assume the model induces a directed acyclic graph (DAG) \(D\). Each node \(Vᵢ ∈ D\) corresponds to a random variable, possibly multivariate. \(Pa(Vᵢ)\), \(An(Vᵢ)\) and \(De(Vᵢ)\) denote parents, ancestors and descendants of \(Vᵢ\) in \(D\), which by definition do not include \(Vᵢ\) (see \cite{24}).

We denote \(f_{Vᵢ}\) to be deterministic functions that map \(Pa(Vᵢ)\) and some background random variable \(U_{Vᵢ}\) to \(Vᵢ\), that is \(Vᵢ = f_{Vᵢ}(Pa(Vᵢ), U_{Vᵢ})\). Such functions always exist (see \cite{30}, Proposition 4.1). The variables \(U_{Vᵢ}\) are latent and cannot be observed. We denote \(D^+\) to be the extension of \(D\) that also includes all variables \(U_{Vᵢ}\) and arrows from \(U_{Vᵢ}\) to \(Vᵢ\). \(Pa₊(Vᵢ)\), \(An₊(Vᵢ)\) and \(De₊(Vᵢ)\) denote parents, ancestors and descendants of \(Vᵢ\) in \(D^+\). Because of how \(D^+\) is constructed, we have \(Pa₊(U_{Vᵢ}) = An₊(U_{Vᵢ}) = ∅\) and \(U_{Vᵢ} ⊥ ⊥ Vᵢ\) for \(i ≠ j\). Furthermore, we assume there exists a probability density \(p\) associated to \(D^+\) such that \(p(U_{Vᵢ}) > 0\) for all values of \(U_{Vᵢ}\).

Among the variables \(Vᵢ\), we denote treatment by \(X\) and outcome by \(Y\). For sake of presentation, throughout this paper we consider \(X\) and \(Y\) to be single scalar or multivariate nodes in \(D\); though, the theory can be extended seamlessly. We further denote latent variables other than treatment by \(L_j\) \((≠ X, Y)\) and observed variables other than treatment and outcome by \(O_k\) \((≠ X, Y)\); hence, each node \(Vᵢ ∈ D\) corresponds exactly to either \(X, Y\), one \(L_j\), or one \(O_k\).

With a slight abuse of notation, we use capital letters without indices to denote both sets and concatenated arrays of random variables. Specifically, we denote \(U, U_L, U_O, V, L, O\) for sets and concatenations of \(U_{Vᵢ}, U_{Lᵢ}, U_{Oᵢ}, Vᵢ, Lᵢ, Oᵢ\), respectively. \(X\) and \(Y\) also denote the sets of themselves. We denote observed variables that are not descendants of \(X\) by \(O = O \setminus De(X)\), similarly to the notation of mutilated graph in \cite{29}. Their respective background variables are denoted by \(U_{O}, f_L\) and \(f_O\) denote concatenations of \(f_{Lᵢ}\) and \(f_{Oᵢ}\), respectively. For convenience, we may decompose \(V = [L, X, O]\) and \(U = [U_L, U_X, U_O]\).

We denote realizations of random variables by lowercase, e.g. \(vᵢ\) for realizations of \(Vᵢ\). For a generic function \(h\) and variable \(Vᵢ\), the Jacobian of \(h\) with respect to \(Vᵢ\) is denoted by \(h_{Vᵢ}\) if it exists, where in this case the use of lowercase \(vᵢ\) is just for readability. Note that if \(h\) is a scalar function, \(h_{Vᵢ}\) denotes a transposed gradient. We use \(E, Cov\) and \(Var\) for expectation, covariance and variance.
We may use subscripts to specify the underlying probability density, e.g. $E_{Y|x,o}$ means that the expectation is taken with respect to $p(Y|x,o)$.

## 2 Identification, quantification and estimation

### 2.1 A characterization of identifiability via covariate adjustment

This section offers a complete criterion for identifiability of causal effects \[29\] via covariate adjustment \[35\] in the framework of the extended DAG $D^+$. By covariate adjustment we mean deciding which subset of variables $V_i$ to observe. The idea behind our approach is to construct a set $C$ of background random variables $U_{V_i}$ that, together with $X$ and $O$, completely characterize the randomness of an outcome $Y$. In terms of potential outcome $Y^x$ \[3\], $C$ can be thought as a set of random variables generating the $\sigma$-algebra of $Y^x$ given $O$.

**Definition 2.1.** We define a causal set from $X$ to $Y$ in $D^+$ as the set of all the background random variables that are ancestors of $Y$ and that are not independent of $Y$ given $X$ and $O$:

$$C = \{U_{V_i} \in U \cap \text{An}^+(Y) : U_{V_i} \not\perp\!\!\!\!\perp Y|X,O\}. \quad (1)$$

**Example 2.1.** Figure 1 is an example of DAG $D$ where gray circles represent observed variables in $O$. Figure 2 is the corresponding extended DAG $D^+$ where dashed arrows highlight that the transitions are deterministic given all inputs. In this example, it is well understood that the unobserved variable $V_1$ and the observed variables $V_2$ and $V_3$ each compromise identifiability of the causal effect from $X$ to $Y$. In fact, they respectively induce confounding, overcontrol and endogenous selection bias \[11\]. In terms of causal set, we have $C = \{U_{V_1}, U_{V_2}, U_Y\}$. We observe that $U_{V_1} \not\perp\!\!\!\!\perp X$, $U_{V_2} \not\perp\!\!\!\!\perp X|V_2$, $U_Y \not\perp\!\!\!\!\perp X|V_3$, which we will see in Theorem 2.1 to be the very reasons why the causal effect is not identifiable.

**Theorem 2.1.** Given a causal set $C$ from $X$ to $Y$ in $D^+$, the causal effect of $X$ on $Y$ can be identified by covariate adjustment if and only if

$$C \perp\!\!\!\!\perp X|O. \quad (2)$$

**Proof.** See Appendix [A.1]

**Corollary 2.1.** With the notation in Theorem 2.1, if (2) holds and $p$ is differentiable in $X$, then

$$\nabla_x \log p(C|x,o) = 0. \quad (3)$$

**Proof.** If (2) holds, then $p(C|x,o) = p(C|o)$, which implies the result.

### 2.2 Quantification of marginal causal effect and bias

The following definition introduces a notation that emulates the one of potential outcome.

**Definition 2.2.** Given a function $f(V_1, \ldots, V_n)$ and a set of random variables $A$, we denote $f^a$ to indicate that all arguments of $f$ in $A$ have been assigned to their respective observations $a$, that is

$$f^a = f(V_1, \ldots, V_n)|_{A=a}. \quad (4)$$

Analogously, if $A$ and $B$ are disjoint sets of random variables, we denote $f^{a,b}$ for both assignments of $a$ and $b$ simultaneously.
Example 2.2. Consider again the DAG in Example 2.1. For instance, we have
\[ f_Y^x = f_Y(x, V_1, f_{V_2}, x, U_{V_2}), \quad f_{V_3}^{x,o} = f_{V_3}(x, f_Y(x, V_1, o_2, U_Y), U_{V_3}). \]
If derivatives are well-defined, this implies \( \nabla u_{V_2} f_{V_3}^{x,o} = 0 \), while in general \( \nabla u_{V_2} f_{V_3} \neq 0 \).

Proposition 2.1. Given the DAG \( D^{+} \), consider the causal set \( C \) from \( X \) to \( Y \). Suppose that \( f_Y \) and \( p \) are differentiable with respect to \( X \) and satisfy sufficient regularity assumptions. Then
\[ \nabla_x \mathbb{E}_{Y|X,o} [Y] = \mathbb{E}_{C|X,o} [\nabla_x f_Y^x] + \mathbb{E}_{C|X,o} [f_Y^{x,y} \nabla_x \log p(C|X,o)] \quad (5) \]
Furthermore, if the causal effect of \( X \) on \( Y \) can be identified by covariate adjustment then \( B(x, o) = 0 \).

Proof. See Appendix A.2

Remark 2.1. We address \( A, C \) and \( B \) in (5) as marginal association, causal effect and bias, where the term marginal stands for small variations of the treatment. Indeed, one can decompose
\[ \mathbb{E}[Y|X = x + h] - \mathbb{E}[Y|X = x] = \mathbb{E}[Y^{x+h} - Y^{x}| X = x + h] + \mathbb{E}[Y|X = x + h] - \mathbb{E}[Y|X = x]. \]
While the association measures the difference in average outcome after two treatments, the conditional average treatment effect (CATE) (1) measures the average difference between outcomes in a treatment group if only the treatment changed but all other variables stayed the same. Thus, the CATE is a measure of causation. If we condition on \( O \), divide both sides by \( h \) and take the limit for \( h \to 0 \), we get \( A, B \) and \( C \) in (5).

Remark 2.2. The expressions for \( C(x, o) \) and \( B(x, o) \) in (5) highlight the connection with Theorem 2.1. However, in practice we do not need to find the causal set \( C \) in order to compute them. Indeed, because \( \nabla_x f_Y^{x,y} \) does not depend on \( U \setminus C \) by definition of \( C \), via marginalization we can write
\[ C(x, o) = \mathbb{E}_{U|X,o} [\nabla_x f_Y^x], \quad (6) \]
where \( C \) does not appear any longer and we are free to remove the assignment of \( O \). Regarding \( B \), the main challenge of the expression in (5) is to compute the term \( \nabla_x \log p(C|X,o) \), since \( x \) appears in the normalization constant. Theorem 2.2 provides an elegant formulation of the bias that is easier to compute in practice. Under a few more assumptions Theorem 2.3 provides an alternative formulation of \( B \) that is automatically differentiable and does also not depend on \( C \).

Theorem 2.2. Under the assumptions of Proposition 2.1 the bias in (5) can be rewritten as
\[ B(x, o) = \text{Cov}_{C|X,o} (f_Y^{x,y}, \nabla_x \log p(C, x, o)). \quad (7) \]

Proof. See Appendix A.3

Remark 2.3. The expression of the bias in Theorem 2.2 is a large improvement compared to the one in (5) because the joint \( p(C, x, o) \) does not include a normalization constant involving \( x \). If no mediators between \( X \) and \( Y \) are observed (e.g. \( V_2 \) in Example 2.1), (7) can already provide a practical way to estimate the bias. Otherwise it may hide a difficulty: if the factorization of \( p(C, x, o) \) includes a Dirac delta distribution depending on \( x \), computing its gradient with respect to \( x \) requires some extra analysis. For instance, consider Example 2.1. The joint factorizes as
\[ p(c, x, o) = p(v_3|x, v_2, u_Y)p(v_2|x, u_{V_2})p(x|u_{V_1})p(u_{V_1})p(u_Y). \]
However, the factor \( p(v_2|x, u_{V_2}) \) is a Dirac delta distribution. How can we then compute \( \nabla_x \log p(c, x, o) ? \) Theorem 2.3 further manipulates the bias to remove this obstacle.

Theorem 2.3. Suppose that \( f_Y \) is differentiable in \( X \) and \( O \), and that \( f_X \) and \( f_{O_i} \) are respectively differentiable and invertible in \( U_X \) and \( U_{O_i} \). Then the bias in (5) can be rewritten as
\[ B(x, o) = - \sum_{V_i \in X \cup O} \mathbb{E}_{U|X,o} \left[ (\nabla_{u_{V_i}} f_Y^x + (f_Y^x - \mathbb{E}_{U|X,o}[f_Y^x]) \nabla_{u_{V_i}} \log p(U_{V_i})) (\nabla_{u_{V_i}} f_{V_i}^{x,o})^{-1} \nabla_x (f_{V_i}^{x,o} - v_i) \right] \quad (8) \]

Proof. See Appendix A.4
Remark 2.4. (i) All terms in the expectation of (8) can be computed via automatic differentiation. (ii) $B(x, o)$ can be decomposed in the contributions given by $X$ (confounding bias) and by each variable $O_i$ (overcontrol or endogenous selection bias). (iii) The term $\nabla_{uv, f_{U, V}^x}$ in $B(x, o)$ is null if $V_i$ is $X$ or if it is a descendant of $Y$. (iv) If $U_{V_i}$ is modeled as a multivariate standard Gaussian, then $\nabla_{uv, \log p(U_{V_i})} = -\nabla_{V_i}$. The inverse Jacobian $(\nabla_{uv, f_{V_i}^{x, o}})^{-1}$ is easy to compute if $V_i$ is low-dimensional, or in high-dimensions if $f_{V_i}$ has, for example, a normalizing flow structure [28]. (vi) The term $\nabla_x (f_{V_i}^{x, o} - v_i)$ is $-1$ if $V_i$ is $X$, otherwise it is just $\nabla_x f_{V_i}^{x, o}$.

2.3 Estimation of marginal causal effect and bias

Sections 2.1 and 2.2 provide closed-form expressions for marginal causal effect and bias, namely (6) and (8). Their numerical estimation reduces to a mere statistical inference task: the approximation of an expectation with respect to the probability density $p(u|x, o)$. We further notice that

$$p(u|x, o) = p(u_L|x, o) \delta(u_X - u_X^*) \delta(u_O - u_O^*),$$

where $u_X^*$ and $u_O^*$ are uniquely determined by the assumptions in Theorem 2.3 that $f_X$ and $f_O$ are respectively invertible in $U_X$ and $U_O$ given $U_L$. Hence we can restrict our attention to infer $p(u_L|x, o)$.

To this purpose, the literature is vast; according to scalability and computational constraints, one may resort to sample-based strategies, e.g. MCMC [6], SMC [10] and importance sampling [27], or to optimization-based ones, e.g. Laplace approximation [36], variational inference [21, 5] and normalizing flows [32, 28]. Let us quickly review Laplace approximation and importance sampling in our framework, as they will be used in Sections 3.2 and 3.3 respectively.

**Laplace approximation.** Let us assume that $u_L$ belongs to a continuous unbounded space, or that we can transform the space to be as such. The Laplace approximation is a Gaussian approximation of $p(u_L|x, o)$ around its mode. In order to compute it, we first notice that via automatic differentiation we have immediate access to $\nabla_{u_L} \log p(u_L|x, o)$, which we can employ in any gradient-based optimization to compute a Maximum-a-Posteriori (MAP) estimate [26] of $p(u_L|x, o)$. Let us denote the latter by $u_L^{\text{MAP}}$. Furthermore, automatic differentiation also gives access to the Hessian $\nabla_{u_L}^2 \log p(u_L|x, o)$ evaluated at the MAP, or to any scalable approximation of it (e.g. diagonal approximation); denote this by $H(u_L^{\text{MAP}})$. Whenever $p(u_L|x, o)$ has a well-defined MAP, $-H(u_L^{\text{MAP}})$ is positive-definite, hence we can approximate

$$p(u_L|x, o) \approx N(u_L^{\text{MAP}}, -H^{-1}(u_L^{\text{MAP}})) (u_L),$$

where in general $N(\mu, \Sigma)(v)$ denotes a multivariate Gaussian density with mean $\mu$, covariance matrix $\Sigma$, evaluated at $v$. Then we can sample $u_L$ from the approximated distribution in (10), get $u_X^*$ and $u_O^*$ via the respective inverse maps of $f_X^{x, o}$ and $f_O^{x, o}$, then produce Monte Carlo estimators of $\hat{C}(x, o)$ and $B(x, o)$ in (6) and (8), respectively.

**Importance sampling.** Importance sampling is a way to change the density underlying an expectation. In causal inference it might be better known under the name of inverse propensity weighting [20]. Consider any treatable density $q(u_L) > 0$, which we address as importance density. Also, consider a generic integrable function $h(u_L, u_X, u_O)$. We have

$$\mathbb{E}_{U|x, o}[h(U_L|U_X, U_O)] = \mathbb{E}_{U_L|x, o}[h(U_L, u_X^*, u_O^*)] = \mathbb{E}_{U_L}[h(U_L, u_X^*, u_O^*)] w(U_L|x, o),$$

where $\mathbb{E}_{U_L}$ denotes the expectation with respect to the density $q(U_L)$ and

$$w(u_L|x, o) = \frac{p(u_L|x, o)}{q(u_L)} \mathbb{E}_{U_L} \left[ \frac{p(U_L|x, o)}{q(U_L)} \right].$$

The quantities in (12) are known as importance weights. Then we can first sample $u_L^{(i)}$ from $q(U_L)$ for $i = 1, \ldots, N$, compute the unnormalized weights $\tilde{w}^{(i)} = \frac{p(u_L^{(i)}, x, o)}{q(u_L^{(i)})}$, then estimate the importance weights as $w^{(i)} = \frac{\tilde{w}^{(i)}}{\sum_{i=1}^{N} \tilde{w}^{(i)}}$. Finally, using the same samples we can compose a Monte Carlo estimator of the right-hand-side of (11). Thus we can estimate $\hat{C}(x, o)$ and $B(x, o)$ by applying such technique for $h$ being the respective expressions in (6) and (8). Notice that if we take $q(U_L)$ to be the prior $p(U_L)$, the unnormalized weight simplifies to be the likelihood, i.e. $\frac{p(u_L|x, o)}{p(u_L)} = p(x, o|u_L)$. 

5
3 Experiments

3.1 Confounding, overcontrol and endogenous selection bias

![Diagram of a causal graph showing confounding bias]

\[ V_1 = U_{V_1} \]
\[ X = \alpha V_1 + U_X \]
\[ Y = \beta X + \gamma V_1 + U_Y \]

![Diagram of a causal graph showing overcontrol bias]

\[ X = U_X \]
\[ V_1 = \alpha X + U_{V_1} \]
\[ Y = \beta X + \gamma V_1 + U_Y \]

![Diagram of a causal graph showing endogenous selection bias]

\[ X = U_X \]
\[ V_1 = \beta X + \gamma Y + U_{V_1} \]

Figure 3: Confounding bias  
Figure 4: Overcontrol bias  
Figure 5: End. selection bias

In this section we study simple linear models exhibiting the three possible types of bias: confounding, overcontrol and endogenous selection \([11]\). Studying linear models is useful because we can compute \(A, C\) and \(B\) in closed-form, checking our results and building up intuition. For all models, we will consider \(\alpha, \beta, \gamma\) and \(\delta\) to be scalar parameters and \(U_{V_1}, U_X, U_Y \sim N(0, 1)\). For detailed proofs of the following results, see Appendix \([B]\).

**Confounding bias.** Given the model in Figure 3 we can compute

\[ A(x, o) = \beta + \frac{\gamma \alpha}{1 + \alpha^2}, \quad C(x, o) = \beta, \quad B(x, o) = \frac{\gamma \alpha}{1 + \alpha^2}. \]

Notice that because the model is linear, \(C(x, o)\) is independent of \(p(u|x, o)\). If \(\gamma \neq 0\) then \(U_{V_1} \in C\), and if \(\alpha \neq 0\) then \(U_{V_1} \not\perp \!\!\!\perp X\). Thus (2) is violated and \(B(x, o) \neq 0\).

**Overcontrol bias.** Given the model in Figure 4 we can compute

\[ A(x, o) = \beta, \quad C(x, o) = \beta + \gamma \alpha, \quad B(x, o) = -\gamma \alpha. \]

Again, because the model is linear, \(C(x, o)\) is independent of \(p(u|x, o)\). If \(\gamma \neq 0\) then \(U_{V_1} \in C\), and if \(\alpha \neq 0\) then \(U_{V_1} \not\perp \!\!\!\perp X\). Thus (2) is violated and \(B(x, o) \neq 0\).

**Endogenous selection bias.** Given the model in Figure 5 we can compute

\[ A(x, o) = \frac{\alpha - \gamma \beta}{1 + \gamma^2}, \quad C(x, o) = \alpha, \quad B(x, o) = -\frac{\gamma(\beta + \gamma \alpha)}{1 + \gamma^2}. \]

Once more, because the model is linear, \(C(x, o)\) is independent of \(p(u|x, o)\). If \(\gamma \neq 0\) and either \(\alpha \neq 0\) or \(\beta \neq 0\), then \(U_Y \not\perp \!\!\!\perp X|V_1\). Since \(U_Y \in C\), this violates (2) and \(B(x, o) \neq 0\).

3.2 The lesser of two evils: covariate adjustment with missing data

![Diagram of a causal graph with missing data]

\[ V_1 = U_{V_1} \]
\[ X = \alpha \exp(V_1) + U_X \]
\[ V_2 = \beta X + \gamma V_1^2 + U_{V_2} \]
\[ Y = \delta V_2 + U_Y \]

Figure 6: Confounding vs. overcontrol bias. If \(V_1\) cannot be observed, should we observe \(V_2\) or not?

In most real applications we cannot adjust for all confounders. For example, take the DAG in Figure 6. Whenever we model a mediator \(V_2\), there is likely to exist some variable \(V_1\) for which we do not have data acting as a confounder between \(X\) and \(V_2\). Theorem \([2, 1]\) says that in order to achieve identifiability we would need to observe \(V_1\) and not observe \(V_2\). However, as we cannot observe \(V_1\), we are left with the following question: should we observe \(V_2\) and incur overcontrol bias, or not observe it and have confounding bias?
Let us bring this question to the non-linear model in Figure 6, where we take $\alpha, \beta, \gamma, \delta$ to be scalar parameters and $U_{V_1}, U_X, U_{V_2}, U_Y \sim N(0, 1)$. Here the marginal causal effect is given by $C(x, o) = \beta \delta$ independently whether we observe $V_2$ or not, therefore it does not help us towards a decision. Rather, we want to compute the absolute marginal causal bias $|B(x, o)|$ and pick the case where it is smaller. To this purpose, here we first estimate $p(u_L|x, o)$ via a Laplace approximation as described in Section 2.3, using an ADAM optimizer [23] to compute the MAP; then we compose $p(u|x, o)$ as in (9). Figure 7 shows its marginal distributions for an arbitrary configuration of parameters and for $x = 1$, when $V_2$ is unobserved and when $V_2 = 2$.

![Figure 7: Marginal distributions of $p(u|x, o)$](image)

Given approximate posterior samples, we can compute a Monte Carlo estimator of the bias in (8). Figure 8 shows the estimated $|B(x, o)|$ both when $V_2$ is unobserved and when $V_2 = 2$, for two different configurations of parameters $\alpha, \beta, \gamma, \delta$ and for $x \in (-20, 20)$. We can see that the estimated $|B(x, o)|$ is constant over $x$ when $V_2$ is observed, which is expected since for this model the overcontrol bias equals $\beta \delta$. The confounding bias, however, is non-linear in $x$. Just increasing $\alpha$ from 1 to 5, the range of values of $x$ for which observing $V_2$ is better than not observing it changes drastically. As a sanity check, we also make sure that the estimated value of $B(x, o)$ is close to 0 if we could hypothetically observe only $X$ and $V_1$; this turns out in the order of $10^{-13}$, confirming that the estimation procedure is sensible. In conclusion, as expected the decision depends on specific observations and parameters, potentially learned from data. If we can model missing confounders, feasible bias estimation provides a quantitative tool to take concrete decisions under missing data.

3.3 A simulated study of statins and atherosclerotic cardiovascular disease

We follow the example in [41] and conduct a simulation study of statins and subsequent atherosclerotic cardiovascular disease (ASCVD). We adapt the data generation procedure as presented by the authors to satisfy the requirements of Theorem 2.3. In particular, we assume a continuous treatment variable $X$ representing the strength of each prescribed medicine with support in the range $[0, 1]$. This dose intensity of statin is relative to the maximal dose, that is, 80 mg for atorvastatin and 40 mg for rosuvastatin. This better reflects reality as a typical daily dose varies depending on the patient characteristics and other confounders: age $A$, pre-treatment low-density lipoprotein $L$, frailty $F$, diabetes $D$, and ASCVD risk score $R$. We consider another measurement $M$ of low-density lipoprotein that happens post-treatment. The outcome $Y$ indicates the observed incidence of ASCVD. Finally, we record whether a patient had severe headache $H$, which can be an adverse reaction to statin and/or caused by ASCVD.

We implement the data generation process as a $\theta$-parameterized joint distribution of $X$ and $Y$ as well as covariates $V = \{A, L, F, R, D, M, H\}$. The model in use is complex and non-linear; see Appendix C. Figure 9 shows some distributions and statistics recovered from the data, which, although the model is not exactly the same, are relatively close to those reported in [41].

In order to estimate $C(x, o)$ and $B(x, o)$, here we adopt an importance sampling technique as described in Section 2.3 choosing the prior $p(u_L)$ as importance density and $N = 10^5$. Figure 10 shows
distributions of estimated $C(x, o)$ and $B(x, o)$ with $x$ and $o$ randomly generated from the model, for different sets of observed covariates $O$; the table shows their mean estimates, with standard deviation in brackets. When $O = \{A, L, F, D\}$ the bias distribution is peaked around zero. This is expected, since all confounders are observed and only them, so the model should exhibit no bias. When $O = \emptyset$ we have confounding bias, which we see to be very pronounced. In comparison to the case before, here the distribution of $C(x, o)$ is very peaked, showing the large impact of the confounders. This is also the case studied in [41], where a true causal effect of $-0.1081508$ is reported in the GitHub repository and is very close to what we estimate for our modified model. When $O = \{A, L, F, D, M\}$ we have overcontrol bias, but this does not seem particularly impactful. If $O = \{A, L, F, D, H\}$ we have endogenous selection bias, which is smaller then the confounding bias but stronger than the overcontrol one. Finally, if $O = \{H, M\}$ all biases interact together. Interestingly, in this case the observation of post-treatment effects such as $H$ and $M$, while themselves contributing to induce bias, partially mitigates the effect of confounding bias.

| $O$ | $O = \{A, L, F, D\}$ | $O = \emptyset$ | $O = \{A, L, F, D, M\}$ | $O = \{A, L, F, D, H\}$ | $O = \{H, M\}$ |
|-----|---------------------|------------------|-----------------|-----------------|----------------|
| effect bias | $-0.112 (+0.045)$ | $-0.113 (+0.004)$ | $-0.112 (+0.045)$ | $-0.112 (+0.047)$ | $-0.112 (+0.027)$ |
| bias | $0.000 (+0.000)$ | $0.417 (+0.895)$ | $-0.005 (+0.005)$ | $-0.088 (+0.048)$ | $0.100 (+0.608)$ |

Figure 10: Distributions and means (standard deviations) of marginal causal effect and bias

4 Conclusion and future direction

In this work we developed a new complete criterion for identifiability of causal effects via covariate adjustment; we provided a mathematical relation between marginal association, causal effect and bias, showing the bias is zero if such criterion holds; we developed a formulation of the bias where all terms are automatically differentiable; we demonstrated the usefulness of our results over several experiments. A natural future work direction is to exploit the bias characterization as a causal regularizer for the training of highly non-linear machine learning models where we cannot easily control for causal bias.
References

[1] Jason Abrevaya, Yu-Chin Hsu, and Robert P Lieli. “Estimating conditional average treatment effects”. In: *Journal of Business & Economic Statistics* 33.4 (2015), pp. 485–505.
[2] Bernard Altshuler. “Modeling of dose-response relationships.” In: *Environmental health perspectives* 42 (1981), pp. 23–27.
[3] Joshua D Angrist and Jörn-Steffen Pischke. *Mostly harmless econometrics: An empiricist’s companion*. Princeton university press, 2008.
[4] Peter C Austin. “Assessing covariate balance when using the generalized propensity score with quantitative or continuous exposures”. In: *Statistical methods in medical research* 28.5 (2019), pp. 1365–1377.
[5] David M Blei, Alp Kucukelbir, and Jon D McAuliffe. “Variational inference: A review for statisticians”. In: *Journal of the American statistical Association* 112.518 (2017), pp. 859–877.
[6] Steve Brooks et al. *Handbook of markov chain monte carlo*. CRC press, 2011.
[7] Victor Chernozhukov et al. *Double/debiased machine learning for treatment and structural parameters*. 2018.
[8] Hugh A Chipman, Edward I George, Robert E McCulloch, et al. “BART: Bayesian additive regression trees”. In: *The Annals of Applied Statistics* 4.1 (2010), pp. 266–298.
[9] Carlos Cinelli and Chad Hazlett. “Making sense of sensitivity: Extending omitted variable bias”. In: *Journal of the Royal Statistical Society: Series B (Statistical Methodology)* 82.1 (2020), pp. 39–67.
[10] Arnaud Doucet, Nando De Freitas, and Neil Gordon. “An introduction to sequential Monte Carlo methods”. In: *Sequential Monte Carlo methods in practice*. Springer, 2001, pp. 3–14.
[11] Felix Elwert. “Graphical causal models”. In: *Handbook of causal analysis for social research*. Springer, 2013, pp. 245–273.
[12] Alexander Gain and Ilya Shpitser. “Structure learning under missing data”. In: *International Conference on Probabilistic Graphical Models*. PMLR. 2018, pp. 121–132.
[13] Douglas Galagate. “Causal inference with a continuous treatment and outcome: alternative estimators for parametric dose-response functions with applications.” PhD thesis. 2016.
[14] Richard D Gill and James M Robins. “Causal inference for complex longitudinal data: the continuous case”. In: *Annals of Statistics* (2001), pp. 1785–1811.
[15] James J Heckman and Edward Vytlacil. “Structural equations, treatment effects, and econometric policy evaluation 1”. In: *Econometrica* 73.3 (2005), pp. 669–738.
[16] Miguel A Hernán and James M Robins. *Causal inference: what if*. 2020.
[17] Jennifer L Hill. “Bayesian nonparametric modeling for causal inference”. In: *Journal of Computational and Graphical Statistics* 20.1 (2011), pp. 217–240.
[18] Keisuke Hirano and Guido W Imbens. “The propensity score with continuous treatments”. In: *Applied Bayesian modeling and causal inference from incomplete-data perspectives* 226164 (2004), pp. 73–84.
[19] Kosuke Imai and David A Van Dyk. “Causal inference with general treatment regimes: Generalizing the propensity score”. In: *Journal of the American Statistical Association* 99.467 (2004), pp. 854–866.
[20] Guido W Imbens. “The role of the propensity score in estimating dose-response functions”. In: *Biometrika* 87.3 (2000), pp. 706–710.
[21] Michael Jordan, Jaakkola, and Lawrence Saul. “An Introduction to Variational Methods for Graphical Models”. In: *Machine Learning* 37 (1999), pp. 183–233.
[22] Edward H Kennedy et al. “Nonparametric methods for doubly robust estimation of continuous treatment effects”. In: *Journal of the Royal Statistical Society. Series B, Statistical Methodology* 79.4 (2017), p. 1229.
[23] Diederik P Kingma and Jimmy Ba. “Adam: A method for stochastic optimization”. In: *arXiv preprint arXiv:1412.6980* (2014).
[24] Steffen L Lauritzen. “Causal inference from graphical models”. In: *Complex stochastic systems* (2001), pp. 63–107.
[25] Roderick JA Little and Donald B Rubin. *Statistical analysis with missing data*. Vol. 793. John Wiley & Sons, 2019.
[26] Kevin P Murphy. *Machine learning: a probabilistic perspective*. MIT press, 2012.
[27] Radford M Neal. “Annealed importance sampling”. In: *Statistics and computing* 11.2 (2001), pp. 125–139.
[28] George Papamakarios et al. “Normalizing flows for probabilistic modeling and inference”. In: *arXiv preprint arXiv:1912.02762* (2019).
[29] Judea Pearl. *Causality*. Cambridge university press, 2009.
[30] Jonas Peters, Dominik Janzing, and Bernhard Schölkopf. *Elements of causal inference: foundations and learning algorithms*. The MIT Press, 2017.
[31] Marc Ratkovic and Dustin Tingley. “Causal inference through the method of direct estimation”. In: *arXiv preprint arXiv:1703.05849* (2017).
[32] Danilo Rezende and Shakir Mohamed. “Variational inference with normalizing flows”. In: *International Conference on Machine Learning*. PMLR. 2015, pp. 1530–1538.
[33] Paul R Rosenbaum et al. *Design of observational studies*. Vol. 10. Springer, 2010.
[34] Paul R Rosenbaum and Donald B Rubin. “The central role of the propensity score in observational studies for causal effects”. In: *Biometrika* 70.1 (1983), pp. 41–55.
[35] Ilya Shpitser, Tyler VanderWeele, and James M Robins. “On the validity of covariate adjustment for estimating causal effects”. In: *arXiv preprint arXiv:1203.3515* (2012).
[36] Zhenming Shun and Peter McCullagh. “Laplace approximation of high dimensional integrals”. In: *Journal of the Royal Statistical Society: Series B (Methodological)* 57.4 (1995), pp. 749–760.
[37] Ross M Stolzenberg. “The measurement and decomposition of causal effects in nonlinear and nonadditive models”. In: *Sociological methodology* 11 (1980), pp. 459–488.
[38] Dustin Tran et al. “Model criticism for bayesian causal inference”. In: *arXiv preprint arXiv:1610.09037* (2016).
[39] Jixian Wang. *Exposure-Response Modeling: Methods and Practical Implementation*. Vol. 84. CRC press, 2015.
[40] Xiao Wu et al. “Matching on generalized propensity scores with continuous exposures”. In: *arXiv preprint arXiv:1812.06575* (2018).
[41] Paul N Zivich and Alexander Breskin. “Machine learning for causal inference: on the use of cross-fit estimators”. In: *Epidemiology* 32.3 (2021), pp. 393–401.
A Proofs

A.1 Proof of Theorem 2.1

Let us first report the adjustment criterion introduced in [35], adapted to our framework where, for sake of simplicity, we required $X$ to be a single node. We stress that, unlike the definition in [35], by convention in this work we defined parents, ancestors and descendants of a variable not to include the variable itself (see [24]).

**Definition A.1.** Given a DAG $D$, a set of nodes $O \in V \setminus \{X,Y\}$ satisfies the adjustment criterion relative to $(X,Y)$ in $D$ if:

- $O$ does not include descendants of any variable laying on a causal path from $X$ to $Y$, that is a path from $X$ to $Y$ where all arrows point away from $X$;
- $O$ blocks all non-causal paths from $X$ to $Y$ in $D$.

The adjustment criterion was shown in [35] to be complete for identifiability of causal effects via covariate adjustment. We now show that such criterion and (2) are equivalent. In the proof, we will use graphical concepts such as blocked/unblocked path, fork and collider; see [29] for their definitions.

Let us proceed by contradiction. Suppose there exists $O_j \in O \cap \text{De}(V_i) \cup \{V_i\}$ with $V_i$ laying on a causal path from $X$ to $Y$. Without loss of generality, suppose that a causal path from $X$ to $V_i$ is unblocked, otherwise we could repeat the argument for the first variable that blocks the path instead. Analogously, without loss of generality, suppose that a causal path form $V_i$ to $O_j$ is unblocked. Then we have $U_{V_i} \not\perp \!\!\!\perp X|O$. Since $U_{V_i} \subset C$, this contradicts (2).

Now suppose instead there exists an unblocked non-causal path from $X$ to $Y$. If the last arrow of such path points away from $Y$, then $U_Y \not\perp \!\!\!\perp X|O$. Since $U_Y \subset C$, this contradicts (2). Vice versa, if the last arrow of such path points into $Y$, in order to be unblocked and non-causal the path must contain a fork $V_i \not\in O$ such that $V_i \in \text{An}(Y)$. Then $U_{V_i} \not\perp \!\!\!\perp X|O$ and $U_{V_i} \subset C$, contradicting (2).

This completes the proof that (2) implies the adjustment criterion. Let us now show the other direction. By contradiction, suppose (2) does not hold. Then there exists a $U_{V_i} \subset U \cap \text{An}^+(Y)$ such that $U_{V_i} \not\perp \!\!\!\perp Y|X,O \setminus \text{De}(X)$ and $U_{V_i} \not\perp \!\!\!\perp X|O$. Note that $V_i \neq X$ because $U_X \perp \!\!\!\perp Y|X,O \setminus \text{De}(X)$. Also, we must have $V_i \in \text{An}(Y)$ or $V_i = Y$.

Suppose that $V_i = Y$. Then there exists some observed variable $O_j \in \text{De}(Y)$ such that a path from $X$ to $O_j$ is unblocked, otherwise $U_Y \perp \!\!\!\perp X|O$. Consequently, there exists an unblocked path from $X$ to $Y$. If the latter is non-causal, this contradicts the second item of the adjustment criterion. Otherwise, since also the path from $Y$ to $O_j$ is causal by definition of descendant, we have a contradiction with the first item instead.

Now suppose instead that $V_i \in \text{An}(Y) \setminus X$ and $V_i \in O$. Since $U_{V_i} \not\perp \!\!\!\perp X|O$ there exists an unblocked path from $X$ to $V_i$ with last arrow pointing into $V_i$. If, in addition, $V_i \not\in O \setminus \text{De}(X)$, then there exists an unblocked path from $V_i$ to $Y$ with first arrow into $V_i$, otherwise $U_{V_i} \perp \!\!\!\perp Y|X,O \setminus \text{De}(X)$. Then there is an unblocked non-causal path from $X$ to $Y$ with observed collider $V_i$, which contradicts the second item of the adjustment criterion. If, vice versa, $V_i \in O \setminus \text{De}(X)$, then there is a causal path from $X$ to $V_i$ and one from $V_i$ to $Y$ with $V_i$ observed, which contradicts the first item instead.

On the other hand, suppose $V_i \in \text{An}(Y) \setminus X$ and $V_i \not\in O$. Since $U_{V_i} \not\perp \!\!\!\perp X|O$ there exists an unblocked path from $X$ to $V_i$ with last arrow pointing away from $V_i$. If a causal path from $V_i$ to $Y$ is unblocked, then there is an unblocked non-causal path from $X$ to $Y$ with unobserved fork $V_i$, which contradicts the second item of the adjustment criterion. If a causal path from $V_i$ to $Y$ is blocked by some variable in $O \cap \text{De}(X)$, then there is a blocked causal path from $X$ to $Y$, which contradicts the first item. Otherwise, since $U_{V_i} \subset C$ there must be a non-causal path from $V_i$ to $Y$, with first arrow pointing away from $V_i$, that is unblocked when observing variables in $O \setminus \text{De}(X)$. If this path is also unblocked by $O$, then there is a contradiction with the second item of the adjustment criterion. Otherwise, take the last variable $O_j \in O \cap \text{De}(X)$ that is blocking the path. This can only happen if $O_j \in \text{An}(Y)$, because otherwise there would need to be $O_k \in O \cap \text{De}(O_j)$ that is either an observed collider or an observed descendant of a collider, which would make the path from $V_i$ to $Y$ blocked when observing variables in $O \setminus \text{De}(X)$. Hence we have a contradiction with the first item of the adjustment criterion and conclude the proof.
A.2 Proof of Proposition 2.1

Conditioned over \( X = x \) and \( O = o \), we have \( Y = f_Y = f^{x,o}_Y \), where \( = \) stands for equality in distribution. By Definition 2.1, the causal set \( C \) uniquely determines \( f^{x,o}_Y \), as it includes all background random variables that are ancestors of \( Y \) and are not independent of \( Y \) given \( x \) and \( o \). Then by the Law of the Unconscious Statistician we can write

\[
\mathbb{E}_{Y|x,o}[Y] = \int y p(y|x, o) \, dy = \int f^{x,o}_y p(c|x, o) \, dc.
\]

Next, as we assumed \( f_Y \) differentiable with respect to \( X \) and satisfying sufficient regularity assumptions, by integral Leibniz rule we have

\[
\nabla_x \mathbb{E}_{Y|x,o}[Y] = \int \nabla_x \left( f^{x,o}_Y p(c|x, o) \right) \, dc
\]

\[
= \int \left( \nabla_x f^{x,o}_Y + f^{x,o}_Y \nabla_x \log p(c|x, o) \right) p(c|x, o) \, dc
\]

\[
= \mathbb{E}_{C|x,o} \left[ \nabla_x f^{x,o}_Y \right] + \mathbb{E}_{C|x,o} \left[ f^{x,o}_Y \nabla_x \log p(C|x, o) \right],
\]

where we used product rule and the fact that \( \nabla_x p(c|x, o) = p(c|x, o) \nabla_x \log p(c|x, o) \). Finally, if the causal effect of \( X \) on \( Y \) can be identified via covariate adjustment then Corollary 2.1 holds, which trivially implies \( B(x, o) = 0 \).

A.3 Proof of Theorem 2.2

First, we observe that \( \nabla_x \log p(c|x, o) = \nabla_x \log p(C, x, o) - \nabla_x \log p(x, o) \). Then we have

\[
\nabla_x \log p(x, o) = \frac{1}{p(x, o)} \nabla_x p(x, o)
\]

\[
= \frac{1}{p(x, o)} \nabla_x \int p(c, x, o) \, dc
\]

\[
= \frac{1}{p(x, o)} \int \nabla_x p(c, x, o) \, dc
\]

\[
= \frac{1}{p(x, o)} \int \nabla_x \log p(c, x, o) p(c, x, o) \, dc
\]

\[
= \int \nabla_x \log p(c, x, o) p(c|x, o) \, dc
\]

\[
= \mathbb{E}_{C|x,o} \left[ \nabla_x \log p(C, x, o) \right].
\]

Thus the bias can be rewritten as

\[
B(x, o) = \mathbb{E}_{C|x,o} \left[ f^{x,o}_y \nabla_x \log p(C|x, o) \right]
\]

\[
= \mathbb{E}_{C|x,o} \left[ f^{x,o}_y \nabla_x \log p(C, x, o) \right] - \mathbb{E}_{C|x,o} \left[ f^{x,o}_y \nabla_x \log p(x, o) \right]
\]

\[
= \mathbb{E}_{C|x,o} \left[ f^{x,o}_y \nabla_x \log p(C, x, o) \right] - \mathbb{E}_{C|x,o} \left[ f^{x,o}_y \nabla_x \log p(x, o) \right]
\]

\[
= \mathbb{E}_{C|x,o} \left[ f^{x,o}_y \nabla_x \log p(C, x, o) \right] - \mathbb{E}_{C|x,o} \left[ f^{x,o}_y \nabla_x \log p(C, x, o) \right]
\]

\[
= \text{Cov}_{C|x,o} \left( f^{x,o}_y, \nabla_x \log p(C, x, o) \right).
\]

A.4 Proof of Theorem 2.3

Let us start by the expression of the bias in (7). We first note that \( \bar{f}^{x,o}_y = \int \bar{f}_y u \, du \), where \( p(u_Q|c, o) \) is a Dirac delta distribution. In particular the latter does not depend on \( x \) by definition of \( O \). By further marginalization, this also implies that \( \mathbb{E}_{C|x,o} \left[ \bar{f}^{x,o}_y \right] = \mathbb{E}_{u|C,x,o} \left[ f^{x}_y \right] \). For sake of notation, we will denote \( \bar{f}^{x,o}_y = f^{x,o}_y - \mathbb{E}_{C|x,o} \left[ f^{x}_y \right] \) and \( \bar{f}_y = f^{x}_y - \mathbb{E}_{U|x,o} \left[ f^{x}_y \right] \). Also, denote \( u = [c, u_Q, \bar{u}] \), where \( c \) is a realization of the causal set and \( \bar{u} \) contains all variables in \( u \) that are neither in \( c \) nor in
We have
\[
B(x, o) = \text{Cov}_{C|z, o}(f_Y^{z, o}, \nabla_x \log p(c, x, o))
\]
\[
= \mathbb{E}_{C|z, o}[f_Y^{z, o} \nabla_x \log p(c, x, o)]
\]
\[
= \frac{1}{p(x, o)} \int f_Y^{z, o} \nabla_x p(c, x, o) \, dc
\]
\[
= \frac{1}{p(x, o)} \int \left( \int f_Y^{z} p(u_{o}|c, o) \, du_{o} \right) \nabla_x \left( \int p(c, \tilde{u}, x, o) \, d\tilde{u} \right) \, dc
\]
\[
= \frac{1}{p(x, o)} \int f_Y^{z} \nabla_x p(u_{o}|c, o) \, du_{o}
\]
\[
= \frac{1}{p(x, o)} \int f_Y^{z} \nabla_x p(u, x, o) \, du
\]
where (i) we marginalized \(p(c, x, o)\) over variables \(\tilde{u}\); (ii) we moved the integrals outside, which is possible because \(f_Y^{z}\) does not depend on \(\tilde{u}\) by definition of \(C\); (iii) we moved \(p(u_{o}|c, o)\) inside the gradient with respect to \(x\), which is allowed since it is independent of it; (iv) we rewrote the density factorization as \(p(u, x, o)\), that is correct because \(p(u_{o}|c, o) = p(u_{o}|c, x, o, \tilde{u})\) by definition of \(O\).

Again for sake of notation, let us denote \(z = [x, o]\). We indicate by \(z_i\) a generic variable \(x\) or \(o_i\), whereas \(z_{-i}\) corresponds to all variables in \(z\) except \(z_i\). Without loss of generality, we further order the indices \(i\) from parents to children, so that a variable \(z_i\) may depend on \(z_{i-1}\) but not on \(z_{i+1}\). We write \(z_{<i} = [z_1, \ldots, z_{i-1}]\). Then we can factorize
\[
p(u, z) = p(u) \prod_i p(z_i|z_{<i}, u) = p(u) \prod_i \delta(g_{z_i}),
\]
where \(\delta\) denotes a Dirac delta distribution and we define \(g_{z_i} = f_{Z_i}^{x, o} - z_i\). Replacing in the derivation above, we have
\[
\frac{1}{p(x, o)} \int f_Y^{z} \nabla_x p(u, x, o) \, du = \frac{1}{p(x, o)} \sum_i \int f_Y^{z} p(u)p(z_{-i}|u) \nabla_x \delta(g_{z_i}) \, du
\]
\[
= \frac{1}{p(x, o)} \sum_i \int f_Y^{z} p(u)p(z_{-i}|u) \nabla_{g_{z_i}} \delta(g_{z_i}) \nabla_x g_{z_i} \, du,
\]
where in the last step we used chain rule. Let us now focus on each term of the sum. We decompose \(u = [u_{Z_i}, u_{-Z_i}]\), where \(u_{-Z_i}\) contains all variables in \(u\) but \(u_{Z_i}\). Observe that \(p(z_{-i}|u) = p(z_{-i}|u_{Z_i})\) because \(z_{-i}\) is independent of \(u_{Z_i}\). We then proceed with a change of variable, i.e. \(p(u_{Z_i}) \, du_{Z_i} = p(g_{z_i}) \, dg_{z_i}\). Replacing above, we get
\[
\int f_Y^{z} p(u)p(z_{-i}|u) \nabla_{g_{z_i}} \delta(g_{z_i}) \nabla_x g_{z_i} \, du
\]
\[
\int f_Y^{z} p(u_{Z_i})p(u_{-Z_i})p(z_{-i}|u_{-Z_i}) \nabla_{g_{z_i}} \delta(g_{z_i}) \nabla_x g_{z_i} \, du_{Z_i} \, du_{-Z_i}
\]
\[
\int f_Y^{z} p(g_{z_i})p(u_{Z_i})p(z_{-i}|u_{Z_i}) \nabla_{g_{z_i}} \delta(g_{z_i}) \nabla_x g_{z_i} \, dg_{z_i} \, du_{-Z_i}
\]
\[
= - \int \nabla_{g_{z_i}} \left( f_Y^{z} p(g_{z_i}) \nabla_x g_{z_i} p(u_{-Z_i})p(z_{-i}|u_{-Z_i}) \right) \delta(g_{z_i}) \, dg_{z_i} \, du_{-Z_i},
\]
where in the last step we used the definition of derivative of a Dirac delta, i.e. \(\int h(a) \nabla_a \delta(a) \, da = - \int \nabla_a h(a) \delta(a) \, da\), where \(h\) is a smooth function. This is possible because we assumed \(f_Y\) to be differentiable in \(X\) and \(O_i\). Note that \(\nabla_{g_{z_i}} \nabla_x g_{z_i} = \nabla_x \nabla_{g_{z_i}} g_{z_i} = 0\); furthermore, \(\nabla_{g_{z_i}} p(u_{-Z_i}) = 0\) and \(\nabla_{g_{z_i}} p(z_{-i}|u_{-Z_i}) = 0\) because they do not depend on \(u_{Z_i}\). Also, notice that
\[
\nabla_{g_{z_i}} p(g_{z_i}) = p(g_{z_i}) \nabla_{g_{z_i}} \log p(g_{z_i}) = p(g_{z_i}) \left( \nabla_{g_{z_i}} \log p(u_{Z_i}) - \nabla_{g_{z_i}} \log |\det(\nabla_{u_{Z_i}} g_{z_i})| \right).
\]
We can always express $\det(\nabla_{u_x} g_z) = \sum_\rho \text{sign}(\rho) \prod_k \partial_{u_{z_i}} [g_{z_i}]_{\rho(k)}$, where $\rho$ is a permutation over the indices, $\text{sign}(\rho) \in \{+1, -1\}$ is its sign and $[g_{z_i}]_{\rho(k)}$ denotes the $\rho(k)$-component of $g_{z_i}$.

Because $\partial_{u_{z_i}} \nabla_{g_{z_i}} [g_{z_i}]_{\rho(i)} = 0$, we conclude that $\nabla_{g_{z_i}} \log |\det(\nabla_{u_x} g_z)| = 0$ and $\nabla_{g_{z_i}} p(g_z) = p(g_z) \nabla_{g_{z_i}} \log p(u_{z_i})$. By replacing in the derivation above, we have

$$- \int \nabla_{g_{z_i}} \left( \frac{\partial f}{\partial g} p(g_z) \nabla_{x} g_z \ p(u_x) \ p(z_{-i} | u_Z) \right) \delta(g_z) \ dg_z \ du_{-Z},$$

where in the last two steps we undid the change of variables and reassembled the joint density.

We now sum together all terms over $i$, scale by $p(z)$ and remember that we defined $z = [x, o]$, $f^i = f^i - E_{U|x,o}[f^i]$ and $g_{v_i} = f_{v_i} - v_i$ for $V_i \in X \cup O$. We have

$$B(x, o) = - \frac{1}{p(z)} \sum_i \int \left( \nabla_{g_{z_i}} \nabla_{x} g_z \ p(u_x) \ p(z_{-i} | u_z) \right) \nabla_{x} g_z \ p(u, z) \ du$$

$$- \sum_i \int \left( \nabla_{v_i} f_{v_i} \ nabla_{x} g_z \ p(u_x) \ p(z_{-i} | u_z) \right) \nabla_{x} g_z \ p(u_x, o) \ du$$

$$- \sum_{V_i \in X \cup O} \sum_{U \in X \cup O} \left[ \nabla_{f_{v_i}^{x,o}} f_{v_i} \ nabla_{x} g_z \ p(u_x) \ p(z_{-i} | u_z) \right) \nabla_{x} (f_{v_i}^{x,o} - v_i)$$

where we named $V_i$ to be a generic variable in $X \cup O$ and we expressed the integral as an expectation under the probability density $p(u_x, o)$. We can finally observe that for a generic function $h$ differentiable in $f_{v_i}^{x,o}$, by chain rule we have $\nabla_{u_{v_i}} h = \nabla_{f_{v_i}^{x,o}} h \nabla_{u_{v_i}} f_{v_i}^{x,o}$. Because $f_{v_i}^{x,o}$ is invertible with respect to $U_{v_i}$ for $V_i \in X \cup O$, the Jacobian $\nabla_{u_{v_i}} f_{v_i}^{x,o}$ is also invertible and we can write $\nabla_{f_{v_i}^{x,o}} h = \nabla_{u_{v_i}} h \left( \nabla_{u_{v_i}} f_{v_i}^{x,o} \right)^{-1}$. Replacing in the derivation above, we conclude that

$$B(x, o) = - \sum_{V_i \in X \cup O} \sum_{U \in X \cup O} \left[ \nabla_{u_{v_i}} f_{v_i} \ nabla_{x} g_z \ p(u_x) \ p(z_{-i} | u_z) \right) \nabla_{x} (f_{v_i}^{x,o} - v_i)$$

### B Confounding, overcontrol and endogenous selection bias

#### B.1 Confounding bias

Let us consider the model in Figure 3. Following [6], the marginal causal effect can be expressed as

$$C(x, o) = E_{U|x,o}[\nabla_{x} f_{v_i}^{x,o}] = E_{U|x,o}[\beta] = \beta.$$ 

For the marginal causal bias, we have $\nabla_{u_{v_i}} f_{v_i}^{x,o} = 0$, $\nabla_{u_{v_i}} log p(U_X) = -U_X$, $\nabla_{u_{v_i}} f_{v_i}^{x,o} = 1$, and $\nabla_{x}(f_{v_i}^{x,o} - x) = -1$. Then [6] gives

$$B(x, o) = - E_{U|x,o}[f_{v_i}^{x} - E_{U|x,o}[f_{v_i}^{x}]] U_X].$$ 

Notice that $p(u_{V_i}, u_X, u_Y | x) = p(u_{V_i}, u_X | x)p(u_Y)$, which implies that $U_Y|X = x$ and $U_X|X = x$ are independent. This yields

$$- E_{U|x,o}[f_{v_i}^{x} - E_{U|x,o}[f_{v_i}^{x}]] U_X].$$

$$= -E_{U|x,o}[\beta X + U_Y - E_{U|x,o}[\beta X + U_Y]] U_X] - E_{U|x,o}[\gamma U_{V_1} - E_{U|x,o}[\gamma U_{V_1}]] U_X]$$

$$= -E_{U|x,o}[\beta X + U_Y - E_{U|x,o}[\beta X + U_Y]] E_{U|x,o}[U_X] - \gamma E_{U|x,o}[U_{V_1} - E_{U|x,o}[U_{V_1}]] U_X]$$

$$= -\gamma E_{U|x,o}[U_{V_1} - E_{U|x,o}[U_{V_1}]] U_X]$$

$$= -\gamma \text{Cov}_{U|x,o}[U_{V_1}, U_X].$$
since $\mathbb{E}_{U|x,o}[\beta x + U_Y - \mathbb{E}_{U|x,o}[\beta x + U_Y]] = 0$. With some simple calculation, one can show that $p(u_X|x) = N\left(\frac{x}{1 + \alpha^2}, \frac{\alpha^2}{1 + \alpha^2}\right)(u_X)$ and $p(u_{V_1}|x) = N\left(\frac{\alpha^2}{1 + \alpha^2}, \frac{1}{1 + \alpha^2}\right)(u_{V_1})$, where $N(\mu, \sigma^2)(a)$ generally denotes a Gaussian probability density with mean $\mu$, variance $\sigma^2$, evaluated at $a$. We then have that $\mathbb{E}_{U|x,o}[U_X] = \frac{\alpha^2}{1 + \alpha^2}$, $\mathbb{E}_{U|x,o}[U_{V_1}] = \frac{\alpha^2}{1 + \alpha^2}$ and $\text{Var}_{U|x,o}(U_{V_1}) = \frac{1}{1 + \alpha^2}$. Furthermore, we have

$$\mathbb{E}_{U|x,o}[U_{V_1}|U_X] = \int u_{V_1} u_x p(u_{V_1}, u_X|x) du_x du_{V_1}$$

$$= \int u_{V_1} \int u_x p(u_X|u_{V_1}, x) du_x p(u_{V_1}|x) du_{V_1}$$

$$= \int u_{V_1} \int u_x \delta(u_x + \alpha u_{V_1} - x) du_x p(u_{V_1}|x) du_{V_1}$$

$$= \int u_{V_1} (x - \alpha u_{V_1}) p(u_{V_1}|x) du_{V_1}$$

$$= x \mathbb{E}_{U|x,o}[U_{V_1}] - \alpha \mathbb{E}_{U|x,o}[U_{V_1}^2]$$

$$= x \mathbb{E}_{U|x,o}[U_{V_1}] - \alpha (\text{Var}_{U|x,o}(U_{V_1}) + \mathbb{E}_{U|x,o}[U_{V_1}]^2).$$

Then we have all the elements to compute

$$B(x, o) = -\gamma (\mathbb{E}_{U|x,o}[U_{V_1}|U_X] - \mathbb{E}_{U|x,o}[U_{V_1}][\mathbb{E}_{U|x,o}[U_X]]) = \frac{\gamma \alpha}{1 + \alpha^2}.$$

Thus

$$C(x, o) + B(x, o) = \beta + \frac{\gamma \alpha}{1 + \alpha^2}.$$

Let us check that the latter matches the association $A(x, o)$ when computed directly. We have

$$\mathbb{E}_{Y|x,o}[Y] = \beta x + \gamma \mathbb{E}_{V_1|x,o}[V_1] + \mathbb{E}_{U|x,o}[U_Y]$$

$$= \beta x + \gamma \mathbb{E}_{U|x,o}[U_{V_1}]$$

$$= \beta x + \frac{\gamma \alpha}{1 + \alpha^2} x.$$

Then $A(x, o) = \nabla_x \mathbb{E}_{Y|x,o}[Y] = \beta + \frac{\gamma \alpha}{1 + \alpha^2}$, which verifies the statement.

### B.2 Overcontrol bias

Let us consider the model in Figure 4. Following (6), we have

$$C(x, o) = \mathbb{E}_{U|x,o}[\beta + \alpha \gamma] = \beta + \gamma \alpha.$$

For the bias, we have $\nabla_u f^{x,o}_Y = 0$, $\nabla_u \log p(U_X) = -U_X$, $\nabla_u f^{x,o}_X = 0$ and $\nabla_x (f^{x,o}_X - x) = -1$. Then the contribution given by $X$ to the bias is null, since

$$\mathbb{E}_{U|x,o}[(f^{x}_Y - \mathbb{E}_{U|x,o}[f^{x}_Y]) U_X] = \mathbb{E}_{U|x,o}[f^{x}_Y - \mathbb{E}_{U|x,o}[f^{x}_Y]] \mathbb{E}_{U|x,o}[U_X] = 0,$$

where we used that $f^{x}_Y$ is independent of $U_X$ given $X = x$.

Regarding the contribution to the bias given by $V_1$, we have $\nabla_{u_{V_1}} f^{x}_Y = \gamma$, $\nabla_x f^{x,o}_V = \beta$ and $\nabla_{u_{V_1}} f^{x,o}_V = 1$. Moreover, $U_Y|V_1$ is independent of $X$. Then

$$B(x, o) = -\gamma \alpha + \alpha \mathbb{E}_{U|x,o}[(f^{x}_Y - \mathbb{E}_{U|x,o}[f^{x}_Y]) U_{V_1}]$$

$$= -\gamma \alpha + \alpha \mathbb{E}_{U|x,o}[\beta x + \gamma V_1 + U_Y - \mathbb{E}_{U|x,o}[\beta x + \gamma V_1 + U_Y]][\mathbb{E}_{U|x,o}[U_{V_1}]]$$

$$= -\gamma \alpha$$

since $\mathbb{E}_{U|x,o}[\beta x + \gamma V_1 + U_Y - \mathbb{E}_{U|x,o}[\beta x + \gamma V_1 + U_Y]] = 0$. Then

$$C(x, o) + B(x, o) = \beta + \gamma \alpha - \gamma \alpha = \beta.$$

Let us check the result by computing the marginal association directly. We have $p(y|x, v_1) = N(\beta x + \gamma v_1, 1)(y)$, whence $\mathbb{E}_{Y|x,o}[Y] = \beta x + \gamma v_1$ and $A(x, o) = \nabla_x \mathbb{E}_{Y|x,o}[Y] = \beta.$
B.3 Endogenous selection bias

Let us consider the model in Figure 5. Following (6), we have

$$C(x, o) = E_{U|x, o}[\nabla_x f_Y^2] = E_{U|x, o}[\alpha] = \alpha.$$ 

For the bias, we can argue that the contribution of $X$ is null for exactly the same reason as in the overcontrol bias case (see Appendix B.2). Regarding the contribution of $V_1$, we have $\nabla_{u_{V_1}} f_Y^2 = 0$, $\nabla_{u_{V_1}} \log p(U_{V_1}) = -U_{V_1}$, $\nabla_{u_{V_1}} f_{V_1}^2 = 1$ and $\nabla_x (f_{V_1}^2 - v_1) = \beta + \gamma \alpha$. Then

$$B(x, o) = (\beta + \gamma \alpha)E_{U|x, o}[(f_Y^2 - E_{U|x, o}[f_Y^2]) U_{V_1}] = (\beta + \gamma \alpha)E_{U|x, o}[(U_Y - E_{U|x, o}[U_Y]) U_{V_1}] = (\beta + \gamma \alpha)\text{Cov}_{U|x, o}(U_Y, U_{V_1}).$$

One can compute $p(u_Y | x, v_1) = N(\gamma(1 + \gamma \gamma)(v_1 - (\beta + \gamma \alpha)x), \frac{1}{1 + \gamma \gamma}(u_Y)$ and $p(u_{V_1} | x, v_1) = N(\gamma \beta \gamma(1 + \gamma \gamma)(v_1 - (\beta + \gamma \alpha)x), \frac{\gamma^2}{1 + \gamma \gamma}(v_1 - \beta - \gamma \alpha)$, which respectively imply $E_{U|x, o}[U_Y] = \frac{\gamma}{1 + \gamma \gamma}(v_1 - \beta - \gamma \alpha)$ and $\text{Var}_{U|x, o}(U_{V_1}) = \frac{\gamma^2}{1 + \gamma \gamma}$. If $\gamma = 0$, then $E_{U|x, o}[U_Y | V_1] = 0$. Otherwise

$$E_{U|x, o}[U_{V_1}] = \int u_{V_1} u_Y p(u_{V_1}, u_Y | x, v_1) du_Y du_{V_1} = \int u_{V_1} \int u_Y p(u_Y | u_{V_1}, x, v_1) du_Y p(u_{V_1} | x, v_1) du_{V_1} = \int u_{V_1} u_Y \delta(\beta + \gamma \alpha) x + \gamma u_Y + u_{V_1} - v_1) du_Y p(u_{V_1} | x, v_1) du_{V_1} = \int u_{V_1} \left(\frac{1}{\gamma}(v_1 - (\beta + \gamma \alpha)x) - \frac{1}{\gamma} u_{V_1}\right) du_{V_1} = \frac{1}{\gamma}(v_1 - (\beta + \gamma \alpha)x) E_{U|x, o}[U_{V_1}] = \frac{1}{\gamma}(\text{Var}_{U|x, o}(U_{V_1}) + E_{U|x, o}[U_{V_1}]).$$

Then we have all the elements to compute

$$B(x, o) = (\beta + \gamma \alpha) (E_{U|x, o}[U_{V_1} U_Y] - E_{U|x, o}[U_{V_1}] E_{U|x, o}[U_Y]) = \frac{\gamma(\beta + \gamma \alpha)}{1 + \gamma ^2}.$$ 

Thus

$$C(x, o) + B(x, o) = \frac{\alpha - \gamma \beta}{1 + \gamma ^2}.$$ 

Once more, we check that this is the same as computing the association directly. Since $p(y|x, v_1) = N(\gamma v_1 + (\alpha - \beta \gamma)x, \frac{1}{1 + \gamma \gamma}(y)$, we have $E_{Y|x, o}[Y] = \frac{1}{1 + \gamma \gamma}(\gamma v_1 + (\alpha - \beta \gamma)x)$, whence

$$A(x, o) = \nabla_x E_{Y|x, o}[Y] = \frac{\alpha - \gamma \beta}{1 + \gamma ^2}.$$
The model is given by
\[
A = U_A \\
L = \theta_0^L A + \theta_1^L + e^{\theta_2^L} U_L \\
F = \text{sigmoid}\left(\theta_0^F + \theta_1^F (A + \theta_2^F) + \theta_3^F A^2 + e^{\theta_4^F} U_F\right) \\
D = \text{sigmoid}\left(\theta_0^D + \theta_1^D L + \theta_2^D A + \theta_3^D A^2 + e^{\theta_4^D} U_D\right) \\
R = \text{sigmoid}\left(\theta_0^R + \theta_1^R D + \theta_2^R \log(A) + \theta_3^R \log^2(A) + \theta_4^R L + \theta_5^R \log(A) L + \theta_6^R F\right) \\
X = \text{sigmoid}\left(\theta_1^X \mathbb{1}_{0.05 \leq R < 0.075} + \theta_2^X \mathbb{1}_{0.075 \leq R < 0.2} + \theta_3^X \mathbb{1}_{R \geq 0.2} + \theta_4^X + \theta_5^X \mathbb{1}_{D \geq 0.5} \right) \\
\quad + \theta_6^X L + \theta_7^X \mathbb{1}_{L > \log(160)} + \theta_8^X (A + \theta_9^X) + \theta_0^X (A + \theta_0^X)^2 + e^{\theta_{10}^X} U_X\right) \\
M = L + \theta_0^M + \theta_1^M X (\theta_2^M - L) \mathbb{1}_{L < \log(130)} + e^{\theta_{11}^M} U_M \\
Y = \text{sigmoid}\left(\theta_3^Y + \theta_4^Y X + \theta_5^Y M + \theta_6^Y \sqrt{A + \theta_7^Y} + \theta_8^Y D + \theta_9^Y e^{1+R} \right) \\
\quad + \theta_0^Y (L + \theta_1^Y \text{sigmoid} (10(L + \theta_2^Y)) L^2) + e^{\theta_{10}^Y} U_Y\right) \\
H = \text{sigmoid}\left(\theta_0^H + \theta_1^H X + \theta_2^H Y + e^{\theta_{11}^H} U_H\right),
\]

with
\[
\theta^L = [0.005, \log(100), \log(0.18)] \\
\theta^F = [-5.5, 0.05, -20, 0.001, \log(1.1)] \\
\theta^D = [-4.23, 0.03, -0.02, 0.0009, \log(1.6)] \\
\theta^R = [4.3, 3.5, -2.07, 0.05, 4.09, -1.04, 0.01] \\
\theta^X = [-30, 0.273, 1.592, 2.461, -3.471, 1.39, 0.112, 0.973, -0.046, 0.003, \log(1.7)] \\
\theta^M = [0.1, -3.5, 5, 0] \\
\theta^Y = [-39, 1.4, -\log(110), -6.25, -0.75, -0.1, 0.45, 1.75, 0.29, 0.1, \log(0.9)] \\
\theta^H = [-1.7, 0.8, 1.5, \log(0.5)].
\]

We take \( p(U_A) \) to be a trapezoidal distribution with bottom base from 40 to 75 and top base from 40 to 60. All other distributions of background random variables are taken to be standard Gaussians, that is \( U_L, U_F, U_D, U_X, U_M, U_Y, U_H \sim N(0, 1) \).