Generalized Control Functions via Variational Decoupling

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Abstract

Causal estimation relies on separating the variation in the outcome due to the confounders from that due to the treatment. To achieve this separation, practitioners can use external sources of randomness that only influence the treatment called instrumental variables (IVs). Traditional IV-methods rely on structural assumptions that limit the effect that the confounders can have on both outcome and treatment. To relax these assumptions we develop a new estimator called the generalized control-function method (GCFN). GCFN’s first stage called variational decoupling (VDE) recovers the residual variation in the treatment given the IV. In the second stage, GCFN regresses the outcome on the treatment and residual variation to compute the causal effect. We evaluate GCFN on simulated data and on recovering the causal effect of slave export on community trust. We show how VDE can help unify IV-estimators and non-IV-estimators.

1 Introduction

Many disciplines use observational data to estimate causal effects: economics [6], sociology [18], psychology [22], epidemiology [31], and medicine [34]. Estimating causal effects with observational data requires care due to the presence of variables that influence both treatment assignment and outcome called confounders. Observational causal estimators deal with confounders in one of two ways. One, they assume that all confounders are observed; an assumption called ignorability. Two, they assume a source of external randomness that has a direct influence only on the treatment. Such a source is called an instrumental variable (IV) [5, 13]. Examples of IVs include natural disasters like hurricanes or distance to the closest health center.

Two common IV-based causal estimation methods are the two-stage least-squares method (2SLS) [17, 3, 4] and the control-function method (CFN) [13, 39, 37, 8]. Both methods have a common first stage: learn a distribution over the treatment conditioned on the IV. In the second stage, 2SLS regresses the outcome on simulated treatments from the first stage. On the other hand, CFN’s second stage regresses the outcome on the true treatment and the error in the prediction of treatment from the first stage. The prediction error acts as an estimate of unobserved confounders. Though widely used to estimate causal effects, both 2SLS and CFN breakdown under certain conditions like when the outcome depends on multiplicative interactions of treatment and confounders. Further, CFN requires an additional assumption about the correlations between the prediction errors and the outcome.

To relax some of these assumptions we propose the generalized control-function method (GCFN). GCFN’s first stage constructs an estimate of the unobserved confounder that is marginally independent of the IV, but together with the IV reconstructs treatment. This first-stage is called variational decoupling (VDE). VDE is a type of autoencoder, where the encoder estimates the
unobserved confounder, and the decoder reconstructs treatment with that estimate and the IV. The core challenge in VDE lies in ensuring the estimated confounder guarantees ignorability. The difficulty stems from the fact that marginal independence of random variables does not imply joint independence. To ensure the estimated confounders guarantee ignorability, GCFN requires assumptions on the treatment generation process such as additivity or invertibility. These assumptions dictate the structure of VDE’s decoder. GCFN’s second stage computes the causal effect using VDE’s confounder estimate. VDE’s confounder estimate can be used with any method that assumes ignorability to compute causal estimates. GCFN works without making the assumptions on the true outcome model.

Beyond IV-based causal estimation, VDE can help compute causal effects from observational data where ignorability holds. Variational decoupling (VDE) treats confounders and IVs symmetrically. This symmetry means that given data where all the confounders are measured and ignorability holds, VDE can be used to estimate an IV. Then any IV-method can be run on the VDE-estimated IVs to compute causal effects on data where only confounders are known and ignorability holds. Further, we outline a single general procedure for both kinds of observational data, whether it satisfies ignorability, or with a strong IV but no ignorability, or a mixture of both.

We evaluate GCFN’s prediction of causal effects on simulated data with the outcome, treatment, and IV observed. We demonstrate how GCFN produces correct causal estimates without assumptions on the true outcome process, whereas 2SLS and CFN fail to produce the correct estimate. We also demonstrate how data points with observed confounders can be combined with GCFN. Then, we illustrate how VDE enables the usage of an IV-method on simulated data with only known confounders. Finally, we show recovery of the causal effect of slave export in the colonial era on current societal trust [24].

Related Work. IVs are well-studied in econometrics and causal inference [4, 5, 40]. One of the earliest methods that uses IVs is the Wald estimator [36]. While simple, the Wald estimator is limited to linear and logistic models of outcome and treatment. Two methods extend Wald to more general models: two-stage least-squares method (2SLS) [3, 4, 17] and the control-function method (CFN) [8, 13, 39, 37]. 2SLS’s estimation could be biased when the outcome model has multiplicative interactions between treatment and confounders. We demonstrate this empirically in the experiments and discuss the issue theoretically in appendix A.5. Guo and Small [11] proved that, under some assumptions, CFN improves upon 2SLS. They show that the CFN method is better by proving its equivalence to 2SLS with an augmented set of instrumental variables. Wooldridge [39] discusses extensions of regression residuals for non-linear models under distributional assumptions about the noise in the treatment model. GCFN uses the residual information in treatment as estimates of the unobserved confounder. Recent work has illustrated the usage of deep models for the estimation of causal effects. Louizos et al. [19] use a variational-autoencoder type setup to construct a confounder-estimate using proxies of the true confounder. Hartford et al. [12] developed DeepIV where they parametrize both stages of 2SLS with deep networks. Like 2SLS, DeepIV needs the additive noise assumption for the true outcome process.

2 Generalized Control-Function Method

To define the causal effect we use causal graphs [25]. In causal graphs, each variable is represented by a node, and each causal relationship is a directed arrow from the cause to the effect. Causal graphs get transformed by interventions with the do-operator. The shared relationships between the graphs before and after the do-operation make causal estimation possible. The causal effect of giving a treatment \( t = a \) on an outcome \( y \) is \( E[y \mid \text{do}(t = a)] \). The causal graph in Figure 1 describes a broad class of instrumental variable problems. The difficulty of causal estimation in this graph stems from the unobserved confounder \( z \). The instrumental variable (IV) \( \epsilon \) can be used to adjust for the unobserved confounder.

Two IV methods are the two-stage least-squares method (2SLS) and control-function method (CFN). We follow the CFN setup from Guo and Small [11]. In this setup the true outcome and treatment functions have additive zero-mean noise called \( \eta_y \) and \( \eta_t \) that may be correlated because of the unobserved confounder \( z \):

\[
y = f(t) + \eta_y, \quad t = g(\epsilon) + \eta_t.
\] (1)
To estimate the causal effect, the CFN method constructs an estimate of the unobserved confounder with the regression residual \( t -  \hat{g}(\epsilon) \). Then, CFN regresses the regression residual and the treatment \( \hat{\epsilon} \) on the outcome \( y \). The causal effect is the estimate of the function \( f(t) \). For this estimate to be valid, the CFN method requires that \( \eta_t, \eta_y \) satisfy the following property for some constant \( \rho \):

\[
E[\eta_y | \eta_t] = \rho \eta_t,
\]

assumption A4 in [11]. This property restricts the applicability of the CFN method by limiting how confounding affects the outcome and the treatment. Consider the following example with additive noise, where \( \mathcal{N} \) is the normal distribution:

\[
\epsilon, z \sim \mathcal{N}(0, 1), t = z + \epsilon, y \sim \mathcal{N}(t^2 + z^2, 1).
\]

Here \( \eta_y = z^2 \) and \( \eta_t = z \) meaning that \( E[\eta_y | \eta_t] = \eta_t^2 \), violating the assumption in eq. (2). Note that \( E[z t^2] = E[z z^2] = 0 \), however \( E[t^2 z^2] > 0 \). This means regressing \( y \) on \( t^2 \) and \( z \), i.e., with correct models for \( f(t) \) and \( g(\epsilon) \), would result in an inflated coefficient of \( t^2 \), which is an incorrect causal estimate. Equation (2) is required because some specified function \( t \) could be correlated with an unspecified function of \( z \), resulting in a biased causal estimate. See appendix A.5 for an example where 2SLS fails to recover the true causal effect.

The CFN assumption eq. (1) restrict the confounder’s influence to be additive on both the treatment and outcome. Further, CFN requires that the additive influence the confounder has on the outcome is a scaled version of the confounder’s influence on the treatment (eq. (2)). Such assumptions may not hold in real data. For example, the effect of a medical treatment on patient’s lifespan is confounded by the patient’s current health. This confounder influences treatment through a human decision process, while it influences the outcome through a physiological process making it unlikely to meet CFN’s assumptions.

### 2.1 Generalizing the Control-Function Method

With an estimate of the unobserved confounder, causal estimation reduces to regression of the outcome on the treatment and the confounder estimate. Three relationships in the IV graph Fig. 1 provide an avenue for the estimation of the unobserved confounder.

1. (Estimability) The confounder \( z \) is conditionally dependent on the IV given treatment.
2. (Reconstruction) The confounder \( z \), IV \( \epsilon \), and some independent noise \( \delta \) determine \( t \) through a deterministic function \( g : g(z, \delta, \epsilon) = t \). Here the independent noise is jointly independent of \( z, \epsilon : \delta \perp (z, \epsilon) \).
3. (Independence) The confounder \( z \) is marginally independent of the IV.

A variable \( \hat{z}(t, \epsilon) \) that satisfies these three relationships can be used to estimate the causal effect:

**Theorem 1.** We state the theorem for a discrete \( t \). Let \( F(t, \epsilon, y) \) be the true data distribution. Assume we have access to a confounder estimate \( \hat{z} \), which is sampled from a distribution conditioned on the pair \( t, \epsilon \); estimated confounder \( \hat{z}(t, \epsilon) \sim q(\hat{z} | t, \epsilon) \). Similarly, \( \hat{z}(t) \sim q(\hat{z} | t) = E_{F(t, \epsilon)}q(\hat{z} | t, \epsilon) \) and \( \hat{z} \sim q(\hat{z}) = E_{F(t, \epsilon)}q(\hat{z} | t, \epsilon) \). Assume the following:

1. (A1) The estimate confounder \( \hat{z} \) satisfies the estimability, reconstruction, and independence relationships: 1. (Estimability) \( \hat{z}(t) \perp \epsilon | t \). 2. (Reconstruction) \( \exists d, \forall t, \exists \hat{z}, \epsilon \text{ s.t. } t = d(\hat{z}, \epsilon) \). 3. (Independence) \( \hat{z} \perp \epsilon \).
2. (A2) Strong IV \( \epsilon \). Let \( g \) be some deterministic function, \( \delta \) be independent noise, and \( F \) be the true probability distribution over \( t, z, \epsilon \), s.t. \( t = g(z, \epsilon, \delta) \). Assume \( \exists c, F(t | z, \delta) > c > 0 \).
3. (A3) The IV is jointly independent of reconstructed confounder, true confounder, and independent noise $\delta$: $e \independent (\hat{z}, \delta) \implies e \independent (z, \hat{z})$.

Then, $\hat{z}$ satisfies ignorability, $q(y \mid t = a, \hat{z}) = q(y \mid do(t = a), \hat{z})$, and positivity, $q(\hat{z}), F(t) > 0 \implies q(t \mid \hat{z}) > 0$. So we can estimate the causal effect as follows:

$$E_\hat{z}[y \mid t = a, \hat{z}] = E_\hat{z}[y \mid do(t = a), \hat{z}] = E[y \mid do(t = a)].$$

The assumption A2 of a strong IV provides positivity. Theorem 1 also holds when $t$ is continuous under the assumption that the true outcome process is continuous everywhere. Though, Theorem 1 allows for stochastic $\hat{z}$, Appendix A details how a deterministic $\hat{z}$ is sufficient to meet the three criteria of estimability, reconstruction, and independence. Theorem 1 requires an additional assumption over the three criteria given by Fig. 1: joint independence of the instrument with the estimated and true confounder. This requirement will impose further conditions beyond those in the causal graph Fig. 1 and is the central challenge in developing two-stage IV-estimators.

**Why is joint independence $e \independent (z, \hat{z})$ needed?** A potential outcome $y_t$ is the outcome that would be observed if a unit is given treatment $t$. The potential outcome $y_t$ follows the distribution of $y$ under the $do$ operator and only depends on the true confounder $z$. For ignorability with respect to $\hat{z}$, we need $y_t$ to be independent of $t$, given $\hat{z}$. By reconstruction, given $\hat{z}$, $t$ is purely a function of $e$. This means ignorability with respect to the estimated confounder $\hat{z}$ requires that the true confounder and instrument be independent given the estimated confounder. Thereby, in addition to the marginal independence $e \independent \hat{z}$, ignorability requires joint independence $e \independent (z, \hat{z})$.

The causal graph Fig. 1 with $y$ marginalized out can be represented with two sources of randomness one from the unobserved confounder and one from the instrument; the extra randomness in $t$ denoted as $\delta$ can be absorbed into $z$. In this setup, the treatment and confounder estimates are deterministic functions of the unobserved confounder and instrument. With only two sources of randomness, joint independence means the confounder estimate $\hat{z}$ needs to only be a function of the true unobserved confounder $z$. However stochastic independence of $\hat{z}$ and $e$ does not imply joint independence, so a $\hat{z}$ that meets the estimability, reconstruction, and independence criteria may fail to yield ignorability. We construct such an example.

First, we build an example of a function of two independent variables $a, b$ that is marginally independent of both. Let $1_e$ be one if $e$ is true and zero otherwise, then let

$$a, b \sim \text{uniform}(0, 1), \quad c(a, b) = \begin{cases} 1_{a+b>1}(a+b-1) + 1_{a+b\leq 1}(a+b) \end{cases}.$$

First, $c$ is marginally a uniform variable. The distribution $c \mid a = x$ can be obtained by translating the distribution of $b$ up by $x$, then translating the part greater than one down to zero, meaning $c \mid a$ is uniformly distributed. Thus $p(c \mid a) = p(c)$ meaning $c \independent a$. However, $c$ is a deterministic function of $a$ and $b$. Therefore, while $c \mid a$ is uniformly distributed, $c \mid (a, b)$ is a dirac-delta distribution, meaning $p(c \mid a, b) \neq p(c \mid a)$ implying $c \independent a \mid b$. Note that $b$ can be constructed back from $c, a$ up to measure-zero as $b = c - a$ if $c > a$ and $b = c - a + 1$ if $c \leq a$; i.e., $c$ is almost everywhere invertible for each fixed $a$.

Now imagine that $a = e, b = z, c = \hat{z}$ and that $t = z + e$ or $t = z - e$. Note that the pairs $z, e$ and $\hat{z}, e$ can both reconstruct $t$ almost everywhere and are pairs of marginally independent variables: $z \independent e$ and $\hat{z} \independent e$. Thus both pairs $z, e$ and $\hat{z}, e$ satisfy estimability, reconstruction, and independence. Further, $t$ and $\hat{z}$ are invertible functions of $z$ for every fixed value of $e$. But conditioning on $\hat{z}$ creates dependence between $z$ on $e$, thereby making $y_t$, a function of $z$, and $t$, a function of $e$ and $\hat{z}$, dependent. This means that $\hat{z}$ fails to satisfy ignorability: $y_t \independent t \mid \hat{z}$, meaning that casual estimation with $\hat{z}$ will be biased.

This construction with uniform random variables can be generalized to other continuous distributions by inverse transform sampling. Any marginal density of $a, b$ can be bijectively mapped to a uniform density over $[0, 1]$. Then $c$ can be computed as above and then $a, b, c$ can be bijectively mapped back; $c$ could be mapped back with the CDF of $b$. Conditional dependence is unaffected by bijective transformations and therefore the issue remains. Similar constructions exist with discrete random variables. In general, assumptions on the true data generating process will be needed to ensure joint independence.

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1. $P(c < x) = P(a + b < x) + P(1 < a + b < 1 + x) = 0.5(x^2 - 1) + 1 - 0.5(1-x)^2 = x.$
What assumptions lead to $\epsilon \perp (z, \hat{z})$? Assumptions on the form of the data generating process of the treatment can lead to joint independence. One such assumption is when the treatment is a sum of a function of the confounder and a function of the instrument: $t = f(z) + g(\epsilon)$. By construction we have $\hat{z} = t - \mathbb{E}[t \mid \epsilon] = f(z) - \mathbb{E}_z f(z)$. This means that $\hat{z}$ is purely a function of $z$ and not $\epsilon$, giving the required joint independence $\epsilon \perp (z, \hat{z})$. Assuming treatment gets generated from other known invertible functions $g(z, \epsilon)$, such as multiplication $t = f(z) * g(\epsilon)$, also leads to joint independence.

In the next section, we describe a procedure that constructs $\hat{z}$ by directly encoding the three required properties that $\hat{z}$ must satisfy into an optimization problem along with the assumption needed for joint independence.

2.2 Generalized Control-Function Method

The generalized control-function method (GCFN) has two phases. The first phase constructs an estimate of the unobserved confounder using an autoencoder. The second phase builds a model from confounder and treatment to the outcome.

Variational Decoupling. Using the three relationships in the IV graph in fig. 1, we build an estimator. From the estimability relationship in the IV graph, the confounder estimate is a stochastic function of the treatment and the IV; so the estimator with parameter $\theta$ should be $q_\theta(\hat{z} \mid t, \epsilon)$. From the reconstruction relationship, the estimated confounder and the IV determine treatment implying that with parameter $\phi$, $p_\phi(t \mid \hat{z}, \epsilon)$ should be maximized for $\hat{z} = \hat{z}(t, \epsilon)$. The reconstruction $p_\phi(t \mid \hat{z}, \epsilon)$ can also encode the relationships needed for identification such as additivity. Together these form the parts of an autoencoder where an estimated confounder is sampled conditioned on the treatment and IV, while the treatment is reconstructed from the same estimated confounder and IV.

An autoencoder without any regularization would just memorize its inputs. However the independence relationship in the IV graph enforces regularization via the marginal independence of the estimated confounder $\hat{z}$ and the IV. Let the true data distribution be $F(t, \epsilon)$ and $I$ denote mutual information. We define a constrained optimization problem to construct $\hat{z}$, called variational decoupling (VDE):

$$\text{(VDE)} \max_{\theta, \phi} \mathbb{E}_{F(t, \epsilon)} \mathbb{E}_{q_\theta(\hat{z} \mid t, \epsilon)} \log p_\phi(t \mid \hat{z}, \epsilon) \quad s.t \quad I_\theta(\hat{z}; \epsilon) = 0. \quad (3)$$

VDE is converted to an unconstrained optimization problem by absorbing the independence constraint into the optimization problem via the Lagrange multipliers trick with $\lambda$,

$$\max_{\theta, \phi, \nu} \mathbb{E}_{F(t, \epsilon)} \mathbb{E}_{q_\theta(\hat{z} \mid t, \epsilon)} \log p_\phi(t \mid \hat{z}, \epsilon) - \lambda I_\theta(\hat{z}; \epsilon).$$

Estimation of mutual information requires $q_\theta(\hat{z} \mid \epsilon)$. Instead, we upper bound the mutual information by introducing an auxiliary distribution $r_\nu(\hat{z})$. This yields a tractable objective:

$$\max_{\theta, \phi, \nu} \mathbb{E}_{F(t, \epsilon)} [(1 + \lambda)\mathbb{E}_{q_\theta(\hat{z} \mid t, \epsilon)} \log p_\phi(t \mid \hat{z}, \epsilon) - \lambda \text{KL}(q_\theta(\hat{z} \mid t, \epsilon) \| r_\nu(\hat{z}))]. \quad (4)$$

A full derivation can be found in Appendix A.2. The lower bound is tight when $r_\nu(\hat{z}) = q_\theta(\hat{z})$. For example, when $q_\theta(\hat{z} \mid t, \epsilon)$ is categorical, setting $r(\hat{z})$ to categorical makes the bound eq. (4) tight. The parameters $\theta, \phi, \nu$ can be learned via stochastic optimization. VDE can also be solved by regression, when the form of $p(t \mid \epsilon)$ is known. This procedure can be adapted to the case with covariates by conditioning on the covariates where needed.

The decoder structure can be constructed using the assumptions made on the true treatment generating process to guarantee joint independence. As an example, consider an additive treatment function $t = z + h(\epsilon)$. For this assumption, the decoder would be additive with functions $g, g'$:

$$p_\phi(t \mid \hat{z}, \epsilon) = p_\phi(t - g_\phi(\hat{z}) - g'_\phi(\epsilon)),$$

where $p_\phi$ is some likelihood that can put all its mass at zero. This decoder allows for perfect reconstruction when $h$ lives in $g'$. Thus when VDE is solved, $g_\phi(\hat{z})$ can only depend on $z$ because

$$g_\phi(\hat{z}) - \mathbb{E}_z[g_\phi(\hat{z})] = t - \mathbb{E}[t \mid \epsilon] = z - \mathbb{E}_z[z].$$
We give an example to illustrate how we can use the discussion so far has only seen outcome estimation using either \( \hat{\theta} \). Thus, after solving \( VDE \), the first stage of \( GCFN \), constructs a confounder estimate when given data with an \( IV \). The \( VDE \)-estimated confounder satisfies ignorability, so instead of regression in the second stage, \( IV \)-estimate given data with ignorability. Given this \( IV \)-estimate, we can compute causal effects using \( IV \)-estimate, we can compute causal effects using \( SLS \) and \( CFN \) exploit this relationship between assumptions on the true outcome and treatment models by limiting the function classes to simple parametric functions. Under strong ignorability, confounder estimates \( \hat{\epsilon} \) need to satisfy \( y \perp \epsilon | t, \hat{z} \). This additional independence can be baked into \( VDE \) as a regularizer leading to joint independence depending on assumptions involving the outcome and the confounder.

### 3 Properties and Unification

The first stage of \( GCFN \), \( VDE \), constructs a confounder estimate when given data with an \( IV \). The second stage uses the confounder estimate to compute a causal effect with regression. The \( VDE \)-estimated confounder satisfies ignorability, so instead of regression in the second stage, \( GCFN \) could use other ignorability-based estimators like propensity methods \([9, 30]\), balancing and matching methods \([15, 30, 16, 29, 9]\), extensions of these methods that use deep neural networks \([33, 35, 19]\), and Bayesian nonparametrics \([14, 2]\). In this section, we show the counterpart: \( VDE \) can be used to construct an \( IV \)-estimate given data with ignorability. Given this \( IV \)-estimate, we can compute causal effects using \( IV \)-methods, like the Wald or \( 2SLS \). Finally, we describe a single general procedure for causal inference for a mixture of data with IVs or with confounders where ignorability holds.

#### Symmetry of variational decoupling and interchangeability of estimators.

When all the confounders have been measured and ignorability holds, we can use \( VDE \) to construct an \( IV \)-estimate by switching the places of the confounder \( z \) and \( \epsilon \) in \( VDE \) in eq. (3):

\[
\max_{\theta, \phi} E_{f(t,z)} E_{q_\theta(\epsilon | t, z)} \log p_\phi(t \mid \hat{\epsilon}, z) \quad s.t. \quad I_\theta(\hat{\epsilon} ; z) = 0. \tag{5}
\]

We give an example to illustrate how we can use \( IV \) methods on data with confounders. Consider that we are given data with only the confounders and no \( IV \). We can use \( VDE \) to construct an \( IV \)-estimate \( \hat{\epsilon} \) and then compute the \( 2SLS \) estimate using \( \hat{\epsilon} \). Without further analysis of \( VDE \), it is unclear whether traditional ignorability methods are better for causal estimation with confounders where ignorability holds, compared to running \( 2SLS \) on \( VDE \)'s \( IV \)-estimate.

#### General observational estimation via disentangled effect estimation.

With \( VDE \), given any observational dataset, we can construct a new dataset that contains both confounders and IVs. On this new dataset, we can use either \( IV \) methods or ignorability methods. Furthermore, consider an example where we have a dataset where some samples have IVs, and the others have confounders that guarantee ignorability. We can use \( VDE \) to fill in the confounder and the \( IV \) when needed. Thus, after solving \( VDE \), we can use any observational effect estimator, thereby providing a single view of observational causal estimation.

The discussion so far has only seen outcome estimation using either \( \hat{\epsilon}, t \) or \( \hat{\epsilon}, t \). There is a third class of outcome estimators that can learn the outcome model by mapping the instrument and...
confounder to the outcome. We call this new class that maps \((\hat{z}, \hat{\epsilon}) \to y\), disentangled estimators, where the confounder estimate is referred to as \(\hat{z}\) and the IV-estimate as \(\hat{\epsilon}\).

Using disentangled estimators, we can combine both kinds of observational causal estimation, IV methods and ignorability methods, into a single general unified procedure:

1. Construct confounder-estimates using VDE, giving us both \(\hat{z}\) and \(\hat{\epsilon}\).

2. Use disentangled estimators to learn the map \(\hat{z}, \hat{\epsilon} \to y\).

Using the map \(\hat{z}, \hat{\epsilon} \to y\), causal estimation can be done in general. As an illustrative example, consider the case where we have a deterministic function \(d\) that constructs \(t\) from the confounder and IV estimates: \(d(\hat{z}, \hat{\epsilon}) = t\). Assume that for every confounder estimate value \(\hat{z}\), \(d(\hat{z}, \cdot) = t\) is invertible. With it, we compute the causal effect as follows:

\[
E[y \mid \text{do}(t = t^\ast)] = E_{\hat{z}}E[y \mid t = t^\ast, \hat{z}] = E_{\hat{z}}E[y \mid \hat{\epsilon} = d^{-1}(t^\ast, \hat{z}), \hat{z}].
\]

We note that disentangled estimation for causal inference was first discussed in [27]. Recent work has shown that disentangled representations are often better for learning algorithms [41, 21, 7]. For example, in linear prediction, a disentangled representation means that the covariance matrix is diagonal, which helps optimization. In our problem, by construction, we have independence \(\hat{z} \perp \hat{\epsilon}\). This means mapping \(\hat{z}, \hat{\epsilon} \to y\) is learned on disentangled inputs \(\hat{z}, \hat{\epsilon}\), and therefore gains the advantages of disentangled representations. A good disentangled representation often means a simpler model suffices for prediction, resulting in easier estimation and better performance.

4 Experiments

We evaluate GCFN on simulated data, where the true causal effects are known, and on recovering the effect of slave export on community trust [24] where we compare GCFN’s estimate to the estimate reported in [24]. For GCFN, we let the estimated confounder \(\hat{z}\) be a categorical variable. The encoder in VDE, \(f_\theta\), is a 2-hidden-layer neural network, such that \(q_\theta(z = i \mid t, \epsilon) \propto \exp(f_\theta(t, \epsilon, i))\) is a categorical likelihood. The decoder is also a 2-hidden-layer network, but the reconstructed likelihood of \(t\) is different for different experiments. In all experiments, the hidden layers in both encoder and decoder networks have 100 units and use ReLU activations. The outcome model is also a 2-hidden-layer neural network with ReLU activations. For the simulated data, the hidden layers in the outcome model have 50 hidden units. In estimating the effect of slave export, the hidden layers in the outcome model have only 10 hidden units; larger width resulted in overfitting.

For all experiments we use 5000 samples and a batch size of 500 for optimizing both VDE and the outcome stages. We optimize both VDE and the outcome-stage for 60 epochs with Adam; starting with a learning rate of \(10^{-2}\) and halving it every 10 epochs if the training error goes up. We found that setting the mutual-information coefficient \(\kappa = \lambda/(1 + \lambda)\) between 0.1 — 0.3 worked without further tuning.

4.1 Simulation Study

We compare GCFN’s performance against 2SLS and CFN and show that GCFN out-performs both methods when the functional form of the treatment is known. We consider two settings with continuous outcome, treatment and confounders where the assumptions of 2SLS and CFN fail: 1) with additive treatment model but a multiplicative outcome model and 2) with a multiplicative treatment model with an additive outcome model. For both settings, the causal effect is the same \(E[y \mid \text{do}(t = a)] = a\). The confounder estimate \(\hat{z}\) is set to have 50 categories. We used the python package statsmodels for 2SLS and our own CFN-implementation. We compare their point-predictions to GCFN’s in figs. 2 and 3.

Multiplicative outcome model + Additive treatment model. Let \(N\) be the normal distribution and \(a\) be a parameter to control the confounding strength. We generate data as:

\[
z, \epsilon \sim N(0, 1), \quad t = (z + \epsilon)/\sqrt{2}, \quad y \sim N(t + a t^2 z, 0.1).
\]
The larger the absolute value of \( a \), the more the confounding. In economics terminology, the treatment noise and the outcome noise are \( \eta_t = z \) and \( \eta_y = at^2z + \text{noise} \) respectively. The generation process above violates the linear noise relation assumption, \( \mathbb{E}[\eta_y | \eta_t] \propto \eta_t \), that \text{CFN} requires [11]. \text{GCFN}, on the other hand, does not require this assumption. In this experiment, \text{VDE} has an additive decoder which specifies a gaussian reconstruction likelihood: 
\[
t \sim \mathcal{N}(g_{\phi}^e(\hat{z}) + g_{\phi}^p(e), 1)
\]
where both \( g_{\phi}^e \) and \( g_{\phi}^p \) are 2-hidden-layer neural networks with ReLU activations and 100 hidden units in each layer.

We found, as expected, that \text{GCFN} out-performs \text{CFN} and 2SLS. We repeated the experiment for multiple \( a \)’s (0.5, 1, 2) and found that \text{GCFN} is robust to different strengths of confounding. Overall, \text{GCFN} obtained an RMSE of 0.48 ± 0.3 while the \text{CFN} and 2SLS only managed to obtain an RMSE of 1.2 ± 0.01 and 1.05 ± 1.1 respectively, despite a correctly specified model. We plot the mean and standard deviation of point-predictions of causal effect in fig. 2. \text{GCFN}’s predictions near \( |t| \approx 2 \) are uncertain because few samples have this magnitude.

**Multiplicative treatment model + Additive outcome model.** For this simulation, we generate data as:
\[
z, \epsilon \sim \mathcal{N}(0, 1), \quad t = az, \quad y \sim \mathcal{N}(t + az, 0.1).
\]

In this experiment, \text{VDE} has a multiplicative decoder which specifies a gaussian reconstruction likelihood with 
\[
t = \mathcal{N}(g_{\phi}^e(\hat{z}) \cdot g_{\phi}^p(e), 1). \quad \text{Note that the 2SLS function from statsmodels uses a linear model } t = \beta \epsilon + \eta_t \text{ which will correctly predict that } \mathbb{E}[t | \epsilon] = 0.
\]

We found, as expected, that \text{GCFN} significantly out-performs 2SLS. We repeated the experiment for multiple \( a \)’s (0.5, 1, 2) and found that \text{GCFN} is robust to different strengths of confounding. Overall, \text{GCFN} obtained an RMSE of 0.3 ± 0.2 while 2SLS only managed to obtain an RMSE of 1.0 ± 1.2. We plot the mean and standard deviation of point-prediction in fig. 3.

![Additive decoder/Additive t](image1.png) ![Multiplicative decoder/Multiplicative t](image2.png)

**Figure 2:** Comparing mean and std of point-predictions of \text{GCFN}, 2SLS, and \text{CFN}, when the additive outcome model assumption is violated; averaged over 3 \( a \)’s, and 5 random seeds. \text{GCFN}’s predictions are better.

**Figure 3:** Comparing mean and std of point-predictions of \text{GCFN} and 2SLS, when the additive treatment model assumption is violated; averaged over 3 \( a \)’s, and 5 random seeds. \text{GCFN}’s predictions are better.

### 4.2 Estimation with an observed subset of the confounder.

In this experiment, we demonstrate that \text{GCFN} does not need outcome or treatment model assumptions if the confounder \( z \) is observed on a subset of the data. Let \( \rho \) be the fraction of data that \( z \) is observed on and \( \mathcal{B} \) be the Bernoulli distribution. We generate data as follows:
\[
\epsilon, z \sim \mathcal{N}(0, 1), m \sim \mathcal{B}(\rho), \quad t = \epsilon z, \quad y \sim \mathcal{N}(t + tz, 0.1)
\]

Let \( z' = z + m \). The observed data is \((y, t, \epsilon, z', m)\). The decoder uses a categorical reconstruction likelihood: \( \rho(t = i | \hat{z}, \epsilon) \propto \exp(g_{\phi}(z, \epsilon, i)) \). Each category of \( t \) corresponds to one of the 50 equally-sized bins in the interval \([-3.5, 3.5]\). In this setup, \text{VDE}’s objective has an additional loss term on the samples with observed \( z' \)’s. The confounder \( z \) is discretized to 50 equal bins in \([-3.5, 3.5]\). The additional loss term for the \( i^{th} \) sample is the categorical log-likelihood of
We demonstrate the recovery of the causal effect of slave export on the trust in the community with the estimated IV.

The observed data is the outcome, treatment, confounder triple: \( (y, t, z) \). To use the Wald estimator, the outcome and treatment need be linear functions of the IV. With a binary IV, VDE recovers the true IV, up to permutation of labels which doesn’t affect the Wald estimate. So we use VDE to construct a binary IV-estimate whose distribution is \( q_0(\hat{e} | t, z) \). For this experiment, VDE has a general decoder \( g_\theta \) which is a 2-hidden-layer neural network with 100 hidden units in each layer and ReLU activations. The decoder \( g_\theta \) specifies a categorical reconstruction likelihood: \( p_\phi(t = i | z, \hat{e}) \propto \exp(g_\theta(z, \epsilon, i)) \). Each category of \( t \) corresponds to one of the 50 equally-sized bins in the interval [0, 2]. In this experiment, the Wald estimator replaces the outcome stage. With the estimated IV \( \hat{e} \), we have the ATE estimate: \( \text{ATE}_{Wald} = \text{Cov}(y, \hat{e}) / \text{Cov}(t, \hat{e}) \).

Figure 5 plots the ATE vs magnitude of correlation between the \( t, \epsilon \) in the data generating process set by \( \gamma \in \{2.0, 1.5, 0.5, -0.5, -1.0\} \). The experiments were repeated for 5 different seeds each to estimate the standard deviation. Figure 5 shows the Wald estimates of the true ATE are good, through the VDE-estimated IV, when the observed data contains no instruments. The error bars reflect how the Wald-estimation degrades with increasing confounding.

Figure 4: Causal effect prediction RMSE vs. percentage of samples with \( z \)'s observed. Plot shows mean/std. of the RMSE of the GCFN-predicted causal effects for 5 different percentages, averaged over 5 different seeds each. GCFN predicts well at 2% or more.

Figure 5: ATE Prediction using Wald with a VDE-estimated IV, when the data has no instruments. Plot shows mean/std. of the ATE estimate for 5 \( \gamma \)'s, averaged over 5 random seeds. The errors show increasing quality of estimation with decreasing confounding.

### 4.3 Wald without IV when the confounders are known

This experiment demonstrates the interchangeability of estimators. Here we use an IV-method, the Wald estimator, on data where the confounders are observed but no IV is observed. Let \( U, \mathcal{B} \) be the uniform and bernoulli distributions respectively and let \( \gamma \) be a parameter that controls the strength of the confounder. The data is generated as

\[
e_n \sim \mathcal{B}(0.5), \quad z_n = U[0, 1], \quad t_n = 2(\gamma z_n + (1-\gamma)\epsilon_n), \quad y_n = \mathcal{N}(t_n + z_n, 0.5).
\]

The observed data is the outcome, treatment, confounder triple: \( \{y_n, t_n, z_n\} \). To use the Wald estimator, the outcome and treatment need be linear functions of the IV. With a binary IV, VDE recovers the true IV, up to permutation of labels which doesn’t affect the Wald estimate. So we use VDE to construct a binary IV-estimate whose distribution is \( q_0(\hat{e} | t, z) \). For this experiment, VDE has a general decoder \( g_\theta \) which is a 2-hidden-layer neural network with 100 hidden units in each layer and ReLU activations. The decoder \( g_\theta \) specifies a categorical reconstruction likelihood: \( p_\phi(t = i | z, \hat{e}) \propto \exp(g_\theta(z, \epsilon, i)) \). Each category of \( t \) corresponds to one of the 50 equally-sized bins in the interval [0, 2]. In this experiment, the Wald estimator replaces the outcome stage. With the estimated IV \( \hat{e} \), we have the ATE estimate: \( \text{ATE}_{Wald} = \text{Cov}(y, \hat{e}) / \text{Cov}(t, \hat{e}) \).

### 4.4 Recovering the effect of Slave Export on Community Trust

We demonstrate the recovery of the causal effect of slave export on the trust in the community and society [24]. Nunn and Wantchekon [24] pooled surveys and historical records to get sub-ethnicity and tribe level data from the period of slave trade. The data was used to study the long-term effects of slave-trade, as measured by the 2005 Afrobarometer survey.
46 covariates and
We thank Xintian Han, Mukund Sudarshan, Mark Goldstein, and David Brandfonbrener for their thoughtful feedback and comments.

We predict the effect of the treatment $t = \ln(1 + \text{slave-export/area})$ on the outcome of interest, $y = \text{trust in neighbors}$. Nunn and Wantchekon [24] use a linear model for the outcome $y$ and use the distance to sea as an IV for each community. The authors claim that the distance to sea cannot causally affect how individuals trust each other, but it affects the chance of coming in contact with colonial slave-traders and being shipped to the Americas. They control for urbanization, fixed effects for precolonial sophistication, the number of precolonial jurisdictional political hierarchies beyond the community, integration with the colonial rail network, contact with precolonial European explorers, and the number of missions during colonial rule. The dataset has 6932 samples with 59 features. After filtering out missing values, we preprocessed 46 covariates and IV to have mean 0 and maximum 1, and $t$ to lie in $[0, 2]$.

For this experiment, VDE’s decoder $g_{\theta}$ specifies a categorical reconstruction likelihood as $p_{q}(t = i | \hat{z}, \varepsilon) \propto \exp(g_{\phi}(z, \varepsilon, i))$. Each category of the treatment corresponds to one of 50 equally-sized bins in the interval $[0, 2]$. For the outcome stage, we use a partially linear model $y = \beta t + h_{\theta}(\hat{z})$ so that the effect we recover is of comparable nature to the effect reported in the paper. The outcome-stage network $h_{\theta}$ is a 2-layer network with 10 hidden units per layer and ReLU activations.

The GCFN-estimate of $\beta$ matches the qualitative claims made in Nunn and Wantchekon [24]. Averaged over 4 random seeds each, GCFN’s estimate of $\beta$ was $-0.21 \pm 0.04$ compared with $-0.27 \pm 0.10$, as reported in Nunn and Wantchekon [24].

5 Discussion and Future

In this paper, we propose the generalized control-function method. The first stage of GCFN called VDE constructs confounder estimates, which can be used to learn causal effects in a second stage regression. GCFN removes assumptions on the true outcome process at the cost of assumptions to ensure joint independence between the confounder, estimated confounder pair and the instrument. VDE allows the usage of either IV methods or ignorability methods for observational data with IVs or confounders. Finally, we discuss a single general procedure for observational causal estimation. We expand on potential future extensions.

Studying the general procedure for causal estimation. This paper outlines a general procedure for observational causal inference defined by disentangling the instrument and confounder variation in treatment. Formalizing the assumptions of this procedure and understanding its sample complexity will help practitioners. The bridge that VDE provides can be a channel to theoretically and empirically compare existing IV-methods and ignorability methods.

Information-based causal estimability. In causal estimation, parametric assumptions can be traded-off with assumptions of strength of IV or positivity. Consider a setting where $\varepsilon$ is binary. For every possible confounder value, only two values of the treatment are observed. Thus it is impossible to estimate a quadratic function of $t$ for each fixed value of the confounder. In this setting, one cannot identify the true function $y|t$ from data unless it is linear in $t$. This insight indicates the existence of a fine tuned information-theoretic understanding of the trade-off between violations of positivity or strength of IV, and parametric assumptions.

"Semi-Supervised" causal inference. Consider the setup where we have a large observational dataset with treatment and confounders but very few outcomes are seen. This is a common problem in electronic health records in hospitals, where there can be a gap of years between the observation of covariates and treatment, and the observation of outcomes. Disentangled estimation often allows for the usage of simpler models, which in settings like the electronic health record where few outcomes are measured.

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A Details and Proofs

A.1 The general IV causal graph with covariates/observed confounders

Figure 6: IV Causal Graph with hidden confounder $z$, outcome $y$, instrumental variable $\epsilon$, treatment $t$ and covariates (also called observed confounders) $x$.

Figure 6 is the general version of the IV problem where the instrumental variable property holds true after conditioning on $x$. This is sometimes called a conditional instrument. All our proofs and results carry over to the situation with covariates after conditioning all estimables and distributions on $x$. VDE in this setting with covariates is re-written as:

$$
\max_{\hat{\theta}, \hat{\phi}} E_{F(t, \epsilon, x)} E_{q_{\hat{\theta}}(\hat{z} | t, \epsilon, x)} \log p_{\hat{\phi}}(t | \hat{z}, \epsilon, x) - \lambda I_\theta(\hat{z}; \epsilon | x) \tag{6}
$$

A.2 Mutual Information lower bound

Here we show the full derivation of the lower bound for mutual-information. We derive the lower bound for the general case where there are both observed and unobserved confounders. Notice that a simple lower bound is possible by using $H(\hat{z} | \epsilon, x) \geq H(\hat{z} | \epsilon, t, x)$ but approximating this quantity requires multiple samples; further, this is not a tight lower bound. Instead we introduce two auxiliary distributions $r_c(\hat{z} | x)$ and $p_c(t | \epsilon, \hat{z}, x)$, following the work in variational inference [28, 1, 32, 20] and causal inference [27].

Recall that $F(t, \epsilon, x, y)$ is the true data distribution and $q_{\theta}(\hat{z} | t, \epsilon, x)$ is the confounder-estimate distribution. We overload notation and use $q_{\theta}$ to refer to any distribution that involves operations with $q_{\theta}(\hat{z} | t, \epsilon, x)$. We use $\hat{\cdot}$ to denote that the LHS and RHS are equal up to constants that are ignored during optimization. Note that $H(t, \epsilon | x)$, $H(t | x)$ are both instance-specific constants with respect to the parameters of interest $\phi, \theta, \nu$ and we will drop them from the lower bound when encountered. For a given $x$, we lower-bound the negative instantaneous conditional mutual information as follows:

$$
-\lambda I(\hat{z}; \epsilon | x) = -\lambda KL(q_{\theta}(\hat{z}, \epsilon | x) \parallel q_{\theta}(\hat{z} | x) F(\epsilon | x))
$$

$$
= -\lambda \left[ E_{q_{\theta}(\epsilon, \hat{z} | x)} [\log q_{\theta}(\epsilon | \hat{z}, x) - \log F(\epsilon | x)] \right]
$$

$$
= -\lambda \left[ E_{q_{\theta}(\epsilon, \hat{z} | x)} [\log q_{\theta}(\epsilon | \hat{z}, x)] + H(\epsilon | x) \right]
$$

$$
\bar{\lambda} \leq -\lambda \left[ E_{q_{\theta}(\epsilon, \hat{z} | x)} [KL(q_{\theta}(\hat{z} | x) \parallel q_{\theta}(\hat{z} | x)) + KL(q_{\theta}(t | \epsilon, \hat{z}, x) \parallel q_{\theta}(t | \epsilon, \hat{z}, x))
$$

$$
+ \log q_{\theta}(\epsilon | \hat{z}, x)] \right]
$$

$$
\bar{\lambda} \leq -\lambda \left[ E_{q_{\theta}(\epsilon, \hat{z} | x)} [KL(q_{\theta}(\hat{z} | x) \parallel q_{\theta}(\hat{z} | x)) + KL(q_{\theta}(t | \epsilon, \hat{z}, x) \parallel p_{\theta}(t | \epsilon, \hat{z}, x))
$$

$$
+ \log q_{\theta}(\epsilon | \hat{z}, x)] \right]
$$

$$
\bar{\lambda} \leq -\lambda \left[ E_{q_{\theta}(\epsilon, \hat{z} | x)} [\log q_{\theta}(\hat{z}, \epsilon | x)] + E_{q_{\theta}(t | \epsilon, \hat{z}, x)} \log q_{\theta}(t | \epsilon, \hat{z}, x)
$$

$$
- E_{q_{\theta}(\hat{z} | x)} \log r_c(\hat{z} | x) - E_{q_{\theta}(t, \epsilon, \hat{z}, x)} \log p_{\theta}(t | \epsilon, \hat{z}, x)] \right]
$$

$$
\bar{\lambda} \leq -\lambda \left[ E_{q_{\theta}(\epsilon, \hat{z}, t | x)} [\log q_{\theta}(\hat{z}, \epsilon, t | x)] - E_{q_{\theta}(\hat{z} | x)} \log r_c(\hat{z} | x)
$$

$$
- E_{q_{\theta}(t, \epsilon, \hat{z}, x)} \log p_{\theta}(t | \epsilon, \hat{z}, x)] \right]
$$

$$
\bar{\lambda} \leq -\lambda \left[ E_{F(t, \epsilon, x)} E_{q_{\theta}(\epsilon, t, x)} [\log q_{\theta}(\hat{z} | t, \epsilon, x) - \log p_{\theta}(t | \epsilon, \hat{z}, x)] - H(t, \epsilon | x) \right]
$$
We overload notation here for setting without covariates. The proof adapts to the setting with covariates.

Theorem 1. We state the theorem for a discrete $t$. Let $F(t, e, y)$ be the true data distribution. Assume we have access to a confounder estimate $\hat{z}$, which is sampled from a distribution conditioned on the pair $t, e$: estimated confounder $\hat{z}(t, e) \sim q(\hat{z} | t, e)$. Similarly, $\hat{z}(t) \sim q(\hat{z} | t) = E_{F(t, e | t)}q(\hat{z} | t, e)$ and $\hat{z} \sim q(\hat{z}) = E_{F(t, e)}q(\hat{z} | t, e)$. Assume the following:

1. (A1) The estimate confounder $\hat{z}$ satisfies the estimability, reconstruction, and independence relationships: 1. (Estimability) $\hat{z}(t) \perp \epsilon | t$. 2. (Reconstruction) $3d, \forall t, 3\hat{z}, \epsilon$ s.t. $t = d(\hat{z}, \epsilon)$. 3. (Independence) $\hat{z} \perp \epsilon$.
2. (A2) Strong IV $e$. Let $g$ be some deterministic function, $\delta$ be independent noise, and $F$ be the true probability distribution over $t, z, e$, s.t. $t = g(z, e, \delta)$. Assume $3c, F(t | z, \delta) > c > 0$.
3. (A3) The IV is jointly independent of reconstructed confounder, true confounder, and independent noise $\delta$: $e \perp (\hat{z}, z, \delta) \implies e \perp (z, \hat{z})$.

Then, $\hat{z}$ satisfies ignorability, $q(y | t = a, \hat{z}) = q(y | do(t = a), \hat{z})$, and positivity, $q(\hat{z}), F(t) > 0 \implies q(t | \hat{z}) > 0$. So we can estimate the causal effect as follows:

$$E_{\hat{z}}[y | t = a, \hat{z}] = E_{\hat{z}}[y | do(t = a), \hat{z}] = E[y | do(t = a)].$$

We prove this for the setting without covariates. The proof adapts to the setting with covariates (observed confounders) by conditioning all terms on them.

Proof. (Theorem 1) The proof shows that reconstruction (A1) and joint independence(A3) together imply ignorability, and strength of IV (A2) together with the joint independence(A3) imply positivity.

Ignorability. To establish ignorability we need to show that $y_t \perp t | \hat{z}$ where $y_t$ is the potential outcome for a unit when the treatment given is $t$. In fig. 1, this variable $y_t$ would be on the path from the confounder $z$ to the observed outcome $y$. Treatment $t$ selects the corresponding $y_t$ to produce $y$.

We overload notation here for $q$. While $q(\hat{z})$ can either be a density function or a probability distribution $\hat{z}$, $q(t | \hat{z}), F(t)$ are probability distributions.
By assumption A3 where, we have the joint independence $\epsilon \perp (z, \hat{z})$.

$$e \perp (z, \hat{z}) \iff e \perp z | \hat{z},$$

for all $q(\hat{z}) > 0$.

Note that by the reconstruction property (from assumption A1) $t = d(\hat{z}, \epsilon)$. So given $\hat{z}$, $t$ is purely a function of $\epsilon$. Thus, given $\hat{z}$, $t$ satisfies the same conditional independence as $e$: $e \perp z | \hat{z}$.

Using this, we have

$$e \perp z | \hat{z} \implies d(\hat{z}, \epsilon) \perp z | \hat{z} \implies t \perp z | \hat{z}.$$  

The potential outcome $y_t$ depends only on $z$ and some noise $\eta_t$ that is jointly independent of all other variables. This means for some function $m_t$ that $y_t = m_t(z, \eta_t)$.

$$t \perp z | \hat{z} \implies t \perp m_t(z, \eta_t) | \hat{z} \implies t \perp y_t | \hat{z}.$$  

This shows ignorability.

**Strength of IV and Positivity.** To show positivity, we need to show that the

$$q(\hat{z}) > 0, F(t) > 0 \implies q(t | \hat{z}) > 0.$$  

We start with $q(t | \hat{z})$ and expand it as an integral over the full joint:

$$q(t | \hat{z}) = \int q(t | z, \hat{z}, \epsilon, \delta)q(\epsilon | z, \hat{z}, \delta)q(z, \delta | \hat{z})dzd\delta d\epsilon$$

$$= \int q(t | z, \epsilon, \delta)q(\epsilon | z, \hat{z}, \delta)q(z, \delta | \hat{z})dzd\delta d\epsilon \quad \{ \text{by } t = g(z, \delta, \epsilon) \}$$

$$= \int q(t | z, \epsilon, \delta)q(\epsilon | z, \hat{z}, \delta)q(z, \delta | \hat{z})dzd\delta d\epsilon \quad \{ \text{by A3: } e \perp (z, \delta, \hat{z}) \}$$

$$= \int \left[ \int q(t | z, \epsilon, \delta)q(\epsilon | z, \hat{z}, \delta)d\epsilon \right]q(z, \delta | \hat{z})dzd\delta$$

$$= \int q(t | z, \hat{z})q(z, \delta | \hat{z})dzd\delta \quad \{ \text{by A2: } q(t | z, \delta) > c \}$$

Note that $q(z, \delta | \hat{z})$ is a valid density over $(z, \delta)$. This proves positivity:

$$q(t | \hat{z}) \geq c \int q(z, \delta | \hat{z})dzd\delta > 0.$$  

**Estimated Causal Effect.** We have the following estimated effect by ignorability:

$$E_{q(\hat{z})}E[y | \hat{z}, t = a] = E_{q(\hat{z})}E[y | \hat{z}, do(t = a)] = E[y | do(t = a)]$$

Assumptions for continuous $t$. When $t$ has non-zero density rather than non-zero probability given the estimated confounder, the true outcome model must be smooth to estimate the causal effect.

---

2If $q(z, \delta | \hat{z}) = 0$ everywhere then no pair $(z, \delta)$ maps to $\hat{z}$ and $\hat{z}$ cannot be observed and we cannot condition on it. But $\hat{z}$ is constructed explicitly as part of the algorithm, so it’s observed. Thus $q(z, \delta | \hat{z})$ is a valid conditional density.
Does \( \hat{z}(t, \epsilon) \) have to be a stochastic function? We will show by construction that there exists a deterministic map \( t, \epsilon \to \hat{z} \). Define the following collection of sets indexed by \( t, \epsilon \):

\[
A_{t, \epsilon} = \{(z, \delta) : t = g(z, \delta, \epsilon)\}
\]

Note that for \( t \neq t' \), by construction of \( A_{t, \epsilon} \), we have \( A_{t, \epsilon} \cap A_{t', \epsilon} = \emptyset \). Thus the collection \( \{A_{t, \epsilon}\} \) separates the \((z, \delta)\)-space into disjoint sets, and we can construct a map \( k \) as:

\[
(z, \delta) \in A_{t, \epsilon}, \text{ and } (z', \delta') \in A_{t, \epsilon} \iff k(z, \delta) = k(z', \delta')
\]

We construct \( \hat{z} \) from \( z, \delta \) as \( \hat{z} = k(z, \delta) \). Then by the marginal independence \( z, \delta \perp \epsilon \) implies that \( \hat{z} \perp \epsilon \), meaning we have the independence relationship. Notice that \( \hat{z} \) can be computed from \( t, \epsilon \) and \( t \) can be constructed from \( \hat{z}, \epsilon \). Define \( d'(t, \epsilon) \) and \( d(\hat{z}, \epsilon) \) to be:

\[
\hat{z} = d'(t, \epsilon) := k(z, \delta), \text{ and } t = d(\hat{z}, \epsilon) := g(z, \delta, \epsilon), \forall (z, \delta) \in A_{t, \epsilon}
\]

The maps \( d, d' \) can be constructed because for a given \( \epsilon \)

\[(z, \delta), (z', \delta') \in A_{t, \epsilon} \iff g(z, \delta, \epsilon) = g(z', \delta', \epsilon), k(z, \delta) = k(z', \delta').\]

In other words, we can define the maps \( d, d' \) because all \( (z, \delta) \in A_{t, \epsilon} \) result in the same \( t \), through \( g \), and also in the same \( \hat{z} \), through the function \( k \). The maps \( d', d \) give us the estimability relationship and the reconstruction relationship respectively.

The constructed function above \( \hat{z} = d'(t, \epsilon) \) is a deterministic function of \( t, \epsilon \). Thus \( \hat{z} \) can satisfy ignorability and positivity without being a stochastic function of \( t, \epsilon \).

A.4 From additive treatment function to joint independence

In this setting we assume that \( t = f(z) + g(\epsilon) \). The decoder structure can be constructed using this assumption to guarantee joint independence. For the assumption \( t = f(z) + g(\epsilon) \), the decoder would be additive with functions \( g, g' \):

\[
P_{\phi}(t | \hat{z}, \epsilon) = p_{\phi}(t - g_{\phi}(\hat{z}) - g'_{\phi}(\epsilon)),
\]

where \( p_{\phi} \) is some likelihood that can put all its mass at zero. This decoder allows for perfect reconstruction when \( h \) lives in \( g' \). When VDE is solved, \( g_{\phi}(\hat{z}) \) can only depend on \( f(z) \) because

\[
g_{\phi}(\hat{z}) - E_\epsilon[g_{\phi}(\hat{z})] = t - E[t | \epsilon] = f(z) - E_\epsilon[f(z)].
\]

Therefore for some constant \( c \), \( g_{\phi}(\hat{z}) = f(z) + c \). By the independence, \( \hat{z} \perp \epsilon \), we have

\[
q(\hat{z}, f(z) | \epsilon) = q(\hat{z}, g_{\phi}(\hat{z}) - c | \epsilon) = q(\hat{z}, g_{\phi}(\hat{z}) - c) = q(\hat{z}, f(z)).
\]

Thus we have \((\hat{z}, f(z)) \perp \epsilon \).

Now, we prove that joint independence \((\hat{z}, z) \perp \epsilon \) holds.

\[
q(\hat{z} | z, \epsilon) = \int q(\hat{z} | z, \epsilon, t)q(t | \epsilon, z)dt \quad \{\text{full joint expansion}\}
\]

\[
= \int q(\hat{z} | \epsilon, t)q(t | \epsilon, z)dt \quad \{\hat{z} \perp z | \epsilon, t\}
\]

\[
= \int q(\hat{z} | \epsilon, t)q(t | \epsilon, f(z))dt \quad \{t = f(z) + g(\epsilon)\}
\]

\[
\leq \int q(\hat{z} | \epsilon, t, f(z))q(t | \epsilon, f(z))dt \quad \{f(z) \perp \hat{z} | t, \epsilon\}
\]

\[
= q(\hat{z} | f(z), \epsilon)
\]

\[
= q(\hat{z} | f(z)) \quad \{(\hat{z}, f(z)) \perp \epsilon\}
\]

Integrating both sides with respect to \( q(\epsilon | z) \) we get

\[
q(\hat{z} | f(z)) = \int q(\hat{z} | f(z))q(\epsilon | z)d\epsilon = \int q(\hat{z} | z, \epsilon)q(\epsilon | z)d\epsilon = q(\hat{z} | z)
\]

So, by eqs. (8) and (9) respectively,

\[
q(\hat{z} | z, \epsilon) = q(\hat{z} | f(z)) = q(\hat{z} | z)
\]

Thus given \( z \perp \epsilon \), joint independence \((z \perp \hat{z}) \perp \epsilon \) holds. Since joint independence holds, no assumptions are needed on true outcome process to estimate causal effects with IVs.
A.5 Estimation with the Two-stage least-squares method

We first describe the general version of two-stage least-squares method (2SLS). Let the outcome, treatment and IV be \( y, t', e \) respectively and the true data distribution be \( p(t',y,e) \).

1. In the first-stage, 2SLS learns the distribution \( q(t'|e) \). Given some class of distributions \( Q \), the first-stage can be framed as a maximum-likelihood problem:

\[
q = \arg \max_{q \in Q} E_p(t'|e) \log q(t'|e)
\]

In our setup, \( t \) is the synthetic treatment sampled from the conditional distribution \( q \) estimated in the first stage.

2. In the second-stage, 2SLS learns the conditional distribution of the outcome \( y \) given the synthetic treatment \( t \) sampled from the conditional \( q(t|e) \) from the first stage. Given some class of distributions \( G \), 2SLS’s second-stage can be framed as a maximum-likelihood problem:

\[
g = \arg \max_{g \in G} E_p(t,y) E_{q(t|e)} \log g'(y|t).
\]

The causal effect is then computed as:

\[
h(t) = E_g(y|t)[y|t]
\]

Note: Typically in settings with continuous \( y, t, e \), both stages of 2SLS are framed and implemented as least-squares regressions instead of maximum-likelihood problems. See Kelejian [17] for an overview of classical vs. Bayesian two-stage least-squares method.

In this section, we derive an alternate expression for 2SLS’s causal effect estimate \( h(t) \). Recall that \( t \) is the synthetic treatment sampled from the conditional distribution \( q \) estimated in the first stage. We assume that both stages of 2SLS are perfectly solved. Note that \( t \) is independently sampled conditioned on \( e \). This imposes the following conditional independences:

\[ y \perp t | e, t' \text{ and } t' \perp t | e \]

We marginalize out \( t', e \) from the joint \( q(y,t,t',e) \) to get the dependence of \( y \) on \( t \):

\[
h(t) = E[y|t] = \int_{t',e} y q(y,t',e|t) d\epsilon dy dt'
\]

\[
= \int_{t',e} y p(y|t',e,t) q(e|t) p(t'|e,t) d\epsilon dy dt'
\]

\[
= \int_{t',e} y p(y|t',e) q(e|t) p(t'|e) d\epsilon dy dt' \quad [t \perp t'|e \text{ and } t \perp y|t',e],
\]

which yields

\[
h(t) = \int_{t',e} y p(y|t',e) q(e|t) p(t'|e) d\epsilon dy dt' = E_{q(e|t)} E_{p(t'|e)} E[y|t',e]. \tag{11}
\]

This shows that the effect estimated by 2SLS can be rewritten as

\[
h(t) = E[y|t] = E_{q(e|t)} E_{p(t'|e)} E[y|t',e]
\]

With this, we show that 2SLS’s estimation is biased when the outcome model might have multiplicative interactions between treatment and confounders. Consider this data generation:

\[ e, z \sim \mathcal{N}(0, 1), \quad t = e + z, \quad y = t + t^2 z. \]

Let \( p(t|e) \) be the learned conditional treatment distribution from a perfectly solved first-stage. We use the reverse conditional \( p(e|t) \). 2SLS’s causal effect estimate can be rewritten as \( h(t) = E_{e|t} E_{t'|e} E[y|t',e] \). The true causal effect is \( h^*(t) = E_{z|t} [t + t^2 z | do(t)] = t \). Note that \( E[e|t] = E_{z \sim \mathcal{N}(0,1)} [t - z] = t \). The 2SLS-estimate is \( 3t \neq t = h^*(t) \):

\[
E_{e|t} E_{t'|e} E[y|t',e] = E_{e|t} E_{e} E[y|t' = z + e, e] = E_{e|t} E_{e} [e + z + (e + z)^2 z] = 3t
\]

This shows 2SLS needs to assume properties of the true outcome and treatment functions.
A.6 DeepIV and connections to Variational Decoupling

DeepIV [12] extends the two-stage least-squares method to use neural networks in both stages of treatment and outcome estimation. For simplicity, we ignore the covariates $x$. The first stage of DeepIV estimates the conditional density of treatment given the IV. Assuming the first-stage of DeepIV is solved and we have an estimate $p_θ(t | e)$, the outcome stage of DeepIV solves the following to obtain an estimate $h_φ(t)$ for the true effect function $h^*(t) = E[y | do(t)]$:

$$\min_φ E_{y,e}[y - E_{p_θ(t | e)}h_φ(t)]^2.$$  \hspace{1cm} (12)

This optimization eq. (12) has a subtle issue. We will show that there exist different functions that solve the optimization problem, thereby resulting in different treatment-effect estimates. Assume that the first stage was solved with $t \sim p(t | e)$. The trouble lies in the fact that eq. (12) averages the function $h_φ(t)$ over the distribution $p(t | e)$. If there exists a function $h' \neq h^*$, $h' \neq 0$ such that $E_{p(t | e)}h'(t) = 0$, both $h$ and $h + h'$ solve the optimization problem in Equation (12). As there is no way to separate $h$ from functions like $h + h'$, we face a non-identifiability issue.

We show that multiplicative interactions between $e, z$ in the true treatment function is a sufficient condition for such functions $h'$ to exist. Consider the following data generation:

$$e, z \sim \mathcal{N}(0, 1), \ t = ze, \ y = t^2.$$  

Here $h^*(t) = t^2$. We will show that $E_{p(t | e)}h^*(t) = E_{p(t | e)}(h^*(t) + t)$, meaning that both $h^*(t)$ and $h^*(t) + t$ solve the optimization problem eq. (12). Notice that $E[t | e] = 0$ and

$$E_{p(t | e)}(h^*(t) + t) = E[t^2 + t | e] = E[t^2 | e] + E[t | e] = E[t^2 | e] = E_{p(t | e)}[h^*(t)].$$

For any constant $a$, the function $t^2 + at$ also solves the optimization problem in eq. (12). This means that multiple solutions to DeepIV exist.

One potential reason that DeepIV does not run into this non-identifiability issue is that an upper bound of the original proposed objective is solved instead. To compute gradients for the original optimization two independent expectations are needed, which is not sample-efficient; this is called the double-sample problem. So, [12] optimize an upper bound (via Jensen’s):

$$E_{y,e}[y - E_{p_θ(t | e)}h_φ(t)]^2 \leq E_{y,e} E_{p_θ(t | e)}[y - h_φ(t)]^2.$$ \hspace{1cm} (13)

The RHS above is a log-likelihood problem with a Gaussian likelihood. A general form of this is $E_{y,e} E_{p_θ(t | e)} \log p_θ(y | t)$; where $p_θ$ is the distribution of the outcome under do $(t)$. Finally, as DeepIV is based on 2SLS, DeepIV assumes an additive outcome model to avoid the issues in the previous section.

A.7 Information preserving maps and additional utility constraints

A bijective map is one that maps each element in its domain to a unique element in its range. No information can be lost in this process, resulting in bijective transformations being called information-preserving maps. Information-preserving maps preserve computations that only involve conditioning and expectations; meaning that the causal effect estimate $E_z E[y | t, \hat{z}]$ is preserved. Therefore we can impose additional distributions utility constraints satisfied by bijective transformations of confounder-estimate $\hat{z}$, without losing the properties of ignorability.

Coupled with flexible over-parametrized modelling, information-preserving maps give us the ability to enforce utility constraints on the latent space of $\hat{z}$. If there is an outcome-model that works well with data drawn from a normal distribution, one can add an additional term to VDE’s objective that is the KL divergence between the distribution of $\hat{z}$ and a normal distribution. If we wanted information about continuity in $t$ to be preserved in $\hat{z}$, we could enforce linear interpolation. Similarly, we could force an estimated $\hat{t}$ to have a monotonic relation with $t$. One could enforce multiple constraints from a combination of distances, divergences, ordering and modality constraints. When used correctly, these constraints trade optimization complexity between the outcome-stage to the VDE-stage.
B Additional experiments

The following experiment is done with a full decoder even though the true data generation process has only additive interactions for the treatment model. We compare against vanilla CFN to demonstrate that GCFN doesn’t require the same assumptions. Let \( \mathcal{N} \) be the normal distribution and \( \alpha \) be a parameter to control the confounding strength. We generate data as:

\[
z_n, \epsilon_n \sim \mathcal{N}(0, 1), \quad t_n = (z_n + \epsilon_n)/\sqrt{2}, \quad y_n \sim \mathcal{N}(t_n^2 + \alpha z_n^2, 0.1).
\]

The larger the absolute values of \( \alpha \), the more the confounding. In economics terminology, the treatment noise and the outcome noise are \( \eta_t = z \) and \( \eta_y = \alpha z^2 + \text{noise} \) respectively. We use 5000 samples and a batch size of 500. The 50 categories for the reconstruction and confounder estimate are equally spaced in \([-3.5, 3.5]\). We compare against vanilla CFN with both stages correctly specified as functions of \( t \). The generation process in eq. (14) violates assumption A4 in Guo and Small [11] for the CFN that requires \( \mathbb{E}[\eta_y|\eta_t] \propto \eta_t \). GCFN does not require this assumption.

![Figure 7](image.png)

**Figure 7**: Comparing the quality of predicted causal effects of GCFN and CFN, when the linear noise relation is violated. The plot shows mean of point-predictions of the causal effect for 4 different \( \alpha \)’s, and 5 random seeds. Unlike CFN, GCFN is unaffected by the strength of confounding(\( \alpha \)).

We find, as expected, that GCFN significantly out-performs CFN. Over 5 runs, for \( \alpha = 1 \), we obtain an RMSE of \( 0.3 \pm 0.1 \) while the CFN only manages to obtain an RMSE of \( 1.5 \pm 0.1 \) despite having the correctly specified model for \( t^2 \). We plot in fig. 7, the average of point-predictions over 5 runs and 4 different \( \alpha \)s : \{–2, –1, 1, 2\}. While the error-bands of GCFN are similar for all values of \( t \), CFN significantly varies showing the effect of the strength of confounding \( \alpha \).