Understanding Causal Inference with Causal Statistical Decision Theory

Abstract

We develop *causal statistical decison theory* (CSDT) a novel theory of causal inference which we derive by introducing the idea that "decisions have consequences" to statistical decision theory. CSDT features *causal theories* as the central object of study. We show that causal Bayesian networks have a natural representation as a causal theory and that potential outcomes models may arguably be represented as causal theories as well. In both cases the resulting theories feature unreasonably rich sets of decisions, which we suggest is because both approaches aim to produce reusable causal models. Using causal theories, we investigate reusability – when can knowledge gained using one causal theory be applied to another – and show that this is possible when the theories are related by a *coarsening*.

1 Introduction

It is widely accepted that causal knowledge and statistical knowledge are distinct. Statistics is concerned with *association* while causation is concerned with *consequences*; a distinction of this type goes back at least to Hume (Morris and Brown, 2019). Statistics nevertheless plays a vital role in causal inference and the languages of *potential outcomes* (Rubin, 2005) and *causal Bayesian networks* (Pearl, 2009) are key tools that, in their somewhat different ways, allow us to bring statistical knowledge to bear on causal questions. Counterfactual random variables and "do-interventions" are unique elements of the potential outcomes and causal Bayesian network approach respectively, and there have been a number of attempts to bring these two views together Richardson and Robins (2013); Shpitser (2008); Pearl (2009), but there doesn't appear to be a consensus about which (if any) of these unifications is successful. Our work takes a different approach: we begin with statistical decision theory and connect it with both.

We develop *causal statistical decision theory*, an approach to causal inference that takes statistical decision theory (Wald, 1950) as a starting point and, with conceptual cues from Savage (1972), replaces losses with utilities and consequences. Causal statistical decsion theory features *causal theories* as the central object of study, playing a role analogous to *statistical experiments* in ordinary statistical decision theory. They differ in that, where a statistical experiment gives you a probability measure, a causal theory gives a stochastic map. A statistical experiment connects observations with an abstract "state", while a causal theory expresses a relationship between observations and consequences.

Causal Bayesian networks themselves have a natural representation as causal theories. Against Pearl's claim that counterfactual models subsume interventional ones, we find that arbitrary choices must be made in order to represent potential outcomes models as causal theories. Nonetheless, we propose a plausible strategy for representing potential outcomes models as causal theories. We find that these two approaches yield very different causal theories from one another, though these differences may disappear when we move from "idealised" theories to more realistic theories that we might actually use to make decisions.

We then turn our attention to a notable feature of the idealised causal theories induced by both approaches: they each allow for unrealistically fine control over many of their consequences. We posit that these theories are intended to inform realistic theories used for making decisions. We show that this is possible if the realistic theories can be derived from the idealised theories via what we term *coarsening*. To our knowledge, this is the first attempt to formalise the notion of "stable knowledge" that is often raised as a key potential advantage of causal understanding over purely statistical learning (Arjovsky et al., 2019; Pearl, 2009; Rubin, 2005).

2 Definitions & Notation

We use the following standard notation: $[n]$ refers to the set of natural numbers $\{1, ..., n\}$. Sets are ordinary capital letters X, σ-algebras are script letters $\mathcal{X}$ while

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random variables are sans serif capitals $\mathsf{X} : \_ \to X$. All sets mentioned are understood to be equipped with measures. The calligraphic $\mathcal{G}$ refers to a directed acyclic graph rather than a σ-algebra. Probability measures are greek letters μ, ξ, γ and stochastic maps are bold capitals $\mathbf{C}, \mathbf{H}$. Sets of probability measures or stochastic maps are script capitals: $\mathcal{H}$, $\mathcal{T}$, $\mathcal{J}$. We write the set of all probability measures on $(X, \mathcal{X})$ as $\Delta(\mathcal{X})$. $\delta_x : (X) \to [0, 1]$ is the probability measure such that $\delta_x(A) = 1$ if $x \in A$ and 0 otherwise.

A stochastic map or Markov kernel is a map $\mathbf{A} : X \to \Delta(\mathcal{Y})$. We write the first argument of a Markov kernel as a subscript; for $x \in X$, $G \in \mathcal{Y}$, $\mathbf{A}_x$ is a probability measure on X and $A_x(G) \in [0, 1]$ is the measure of G. For $\mathbf{A}$ to be a Markov kernel we also require that the function $x \mapsto A_x(G)$ must be measurable for all $G \in \mathcal{Y}$. For $\mathbf{C} : X \times Y \to \Delta(\mathcal{Z})$ and $x \in X$ we will write $\mathbf{C}_x$ for the "curried" map $y \mapsto \mathbf{C}_{x,y}$.

We can use a notation similar to matrix-vector products to represent relationships with Markov kernels. Probability measures $\mu \in \Delta(\mathcal{X})$ can be read as row vectors, Markov kernels as matrices and measurable functions $\mathsf{T} : Y \to T$ as column vectors. Defining $\mathbf{B} : Y \to \Delta(\mathcal{Z})$ we have $\mu\mathbf{A}(G) = \int \mathbf{A}_x(G) d\mu(x)$, $\mathbf{A}\mathbf{B}_x(H) = \int \mathbf{B}_y(H) d\mathbf{A}_x(y)$ and $\mathbf{A}\mathsf{T}(x) = \int \mathsf{T}(y) d\mathbf{A}_x(y)$. The tensor product is $(mathbf{A} \otimes \mathbf{B})_{x,y}(G, H) = \mathbf{A}_x(G)\mathbf{B}_y(H)$ where the product on the left is scalar multiplication. Kernel products are associative and the product of kernels is always a kernel itself (Çinlar, 2011).

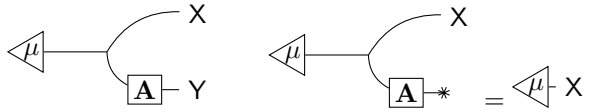
Some elaborate constructions are unwieldly in inline product notation. Here we use string diagrams. String diagrams can always be interpreted as a mixture of kernel products and tensor products of Markov kernels, but we introduce kernels with special notation that helps with interpreting the resulting objects. String diagrams are the subject of a coherence theorem: taking a string diagram and applying a planar deformation or any of a number of graphical rules not used here yields a string diagram that represents the same kernel (Selinger, 2010). For a thorough definition of version of string diagrams used here, see Cho and Jacobs (2019).

A kernel $\mathbf{A} : X \to \Delta(\mathcal{Y})$ is written as a box with input and output wires, probability measures $\mu \in \Delta(\mathcal{X})$ are written as triangles "closed on the left" and measurable functions $\mathsf{T} : Y \to T$ as triangles "closed on the right".

(1)

The identity $\mathbf{Id} : X \to \Delta(X)$ is the Markov kernel $x \mapsto \delta_x$, which we represent with a bare wire. The copy map $\curlyvee : X \to \Delta(X \times X)$ is the Markov kernel $x \mapsto \delta_{(x,x)}$. For $\mathbf{A} : X \to \Delta(Y)$ and $\mathbf{B} : X \to \Delta(Z)$, $\curlyvee(A \otimes B)_x = A_x \otimes B_x$. The discard map $*$ is the Markov kernel $X \to \{\#\}$ given by $x \mapsto \delta_\#$, where $\#$ is some one element set. Placing boxes side by side with connected wires corresponds to taking kernel products as defined above.

We will apply these notions to a couple of example constructions. Given $\mu \in \Delta(X), \mathbf{A} : X \to \Delta(Y)$ as before, the joint distribution on $X \times Y$ given by $\nu(A \times B) = \int_A A(x; B) d\mu(x)$ is given in string diagram on the left of 2. Marginalisation is accomplished with the discard map $*$; hence $\mu\curlyvee(\mathbf{Id} \otimes \mathbf{A}*) = \mu$; this is shown on the right of 2



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3 Statistical Decision Problems and Causal Statistical Decision Problems

A statistical decision problem (SDP) poses the following scenario: suppose we have a set of "states of nature" Θ, a set of decisions D and a loss function $l : \Theta \times D \to \mathbb{R}$. For each state of nature $\theta \in \Theta$ there is an associated probability measure $\mu_\theta \in \Delta(\mathcal{E})$ where $(E, \mathcal{E})$ is some measurable space. Call the stochastic map $\mathbf{H} : \theta \mapsto \mu_\theta$ a *statistical experiment*. Given a *decision strategy* $\mathbf{J} : E \to \Delta(\mathcal{D})$, define the *risk* of $\mathbf{J}$ given state θ to be the expected loss of $\mathbf{J}$ in state θ. Specifically, $R : \Delta(\mathcal{D})^E \times \Theta \to \mathbb{R}$ given by $R : (\mathbf{J}, \theta) \mapsto \mathbf{H}_\theta \mathbf{J} l_\theta$, where we make use of the product notation for brevity.

We would ideally find a strategy $\mathbf{J}$ that minimises the risk in the "true state" θ^*. Unfortunately, we don't know the true state. If there were a decision strategy that minimised the loss in every state, such a strategy would clearly minimise the loss in the true state, but most statistical decision problems don't admit such a strategy. Two alternative decision rules are available:

Given a measure $\xi \in \Delta(\Theta)$ called a prior, *ξ-Bayes decision rule* is a decision rule $\mathbf{J}^*_{\text{Ba}}$ such that the *Bayes risk* $R_\xi : \mathbf{J} \mapsto \int_\Theta H_\theta \mathbf{J} l_\theta d\xi$ is minimised. A *minimax* decision rule $\mathbf{J}^*_{\text{MM}}$ minimises the worst-case risk: $\mathbf{J}^*_{\text{Mm}} \in \arg\min_{\mathbf{J}} \max_{\theta \in \Theta} R(\mathbf{J}, \theta)$ Unlike a Bayes rule, a minimax rule does not invoke a prior. In general, a decision rule is some rule that selects a decision on the basis of the risk functional $R(\mathbf{J}, \cdot)$.

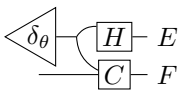
Our representation of statistical experiment is slightly different to, for example, Le Cam (1996), who introduces statistical experiments as an ordered collection

of probability measures. Both representations do the same job, and the representation as a map makes for a clearer connection with causal statistical decision problems.

Formally, we define an SDP as the tuple $\langle \Theta, E, D, \mathbf{H}, l \rangle$ where Θ, E and D are measurable sets, $\mathbf{H}$ is a stochastic map $\Theta \to \Delta(\mathcal{E})$ and l a measurable function $E \to \mathbb{R}$. We leave implicit the set of decision strategies $E \to \Delta(\mathcal{D})$. This is a very bare bones exposition of the theory of SDPs, and for more details we refer readers to Ferguson (1967).

Observe that a statistical decision problem supplies a loss l that tells us immediately how desirable a pair $(\theta, d) \in \Theta \times D$ is. It is more typical to talk about how desirable the *consequences* of a decision are than how desirable a (state, decision) pair is. If the set of possible consequences of a decision is denoted by a set F, let the desirability of an element $f \in F$ be given by a utility function $u : F \to \mathbb{R}$. Given such a u, the tuple $\langle \Theta, E, D, \mathbf{H}, u \rangle$ is an ill-posed problem: we want to evaluate the desirability of decision strategies $\mathbf{J}$, but we have no means of connecting decisions with consequences F. We introduce for each state of nature θ a *consequence map* $\mathbf{C}_\theta : D \to \Delta(\mathcal{F})$; let $\mathbf{C}$ be the Markov kernel $\theta \mapsto \kappa_\theta$. We can then define the *causal risk* $S : \Delta(\mathcal{D})^E \times \Theta \to \mathbb{R}$ by $S : (\mathbf{J}, \theta) \mapsto -H_\theta \mathbf{F} C_\theta u$, and Bayes and minimax risks are defined analogously.

For each state $\theta \in \Theta$, the Markov kernel

$\mathbf{T}_\theta :=$ 

(3)

Is sufficient to compute the causal risk. Thus we can replace $\mathbf{H}$ and $\mathbf{C}$ with the *causal theory* $\mathbf{T} : \Theta \times D \to \Delta(\mathcal{E} \otimes \mathcal{F})$ given by $(\theta, d) \mapsto \mathbf{T}_\theta(d; \cdot)$. A causal statistical decision problem (CSDP) is therefore a tuple $\langle \Theta, E, F, D, \mathbf{T}, u \rangle$.

Given a CSDP $\alpha = \langle \Theta, E, F, D, T, u \rangle$ where $\mathbf{T}$ is a theory arising from some $\mathbf{H}$ and $\mathbf{C}$ as in Equation 3, we can recover the original kernels by marginalisation: $\mathbf{H} = T(mathbf{Id} \otimes *)$ and $\mathbf{C} = \mathbf{T}(* \otimes \mathbf{Id})$. Given α and letting $l := \mathbf{C}u$ we induce the canonical SDP $\beta = \langle \Theta, E, D, \mathbf{H}', l \rangle$ such that for any $\theta \in \Theta$, $\mathbf{J}$, $R^{(\beta)}(\mathbf{J}, \theta) = S^{(\alpha)}(\mathbf{J}, \theta)$, and thus α and β will always produce identical recommendations.

It is also possible to induce a CSDP from an arbitrary SDP $\beta := \langle \Theta, E, D, \mathbf{H}, l \rangle$. First, define $F := \Theta \times D$ and then let $u := -l$. Define $\mathbf{C} : \Theta \to (D \to \Delta(\mathcal{F}))$ by $\mathbf{C} : \theta \mapsto (d \mapsto (\theta, d))$, and then construct $\mathbf{T}$ from $\Theta, \mathbf{H}$ and $\mathbf{C}$ as in 3. Then the CSDP $\alpha := \langle \Theta, E, F, D, \mathbf{T}, u \rangle$ has the property $S^{(\alpha)}(\mathbf{J}, \theta) = R^{(\beta)}(\mathbf{J}, \theta)$.

Causal theories are the central object of study here. They provide a bridge between the experiment $\mathbf{H}$ and the consequences $\mathbf{C}$ and allow us to use the former to make inferences about the latter.

4 Causal Bayesian Networks

We formulate causal Bayesian networks following Pearl (2009), relying on the source to define the key concept of *compatibility* as it is not a central concern here. We argue for the following claim: a causal Bayesian network represents a causal theory.

Suppose we have a set of "interventions" R which factorises as $R = \times_{i \in [n]} \{\#\} \cup X^i$ for some $n \in \mathbb{N}$, collection of sets $\{X^i\}_{i \in [n]}$ and distinguished element $\# \notin R^i$ for any i. Suppose we also have a measurable space E and set of random variables $\{\mathsf{X}^i | i \in \mathbb{N}\}$ such that $\mathsf{X}^i : E \to X^i$. To explain this setup, an element $(x^0, \#, ..., \#, \#) \in R$ identifies a do intervention where the only non-# component x^0 is the "active" element and the components taking the value # are "do-nothing" elements. Thus $(x^0, \#, ..., \#, \#)$ corresponds to $do(\mathsf{X}^0 = x^0)$ where occurrences of the passive elements are ommitted. Denote by $\underline{\#}$ the element of R consisting entirely of # (equivalently, $do()$).

For $n \in \mathbb{N}$, a directed acyclic graph (DAG) of degree n is a graph $\mathcal{G} = (V, A)$ where V is a set of vertices such that $|V| = n$ and $A \subset V \times V$ is a set of directed edges ("arrows") such that A induces no cycles (for a definition of cycles see Pearl (2009)).

Strictly, we are considering labeled graphs $\mathcal{G}$ and sets $\{\mathsf{X}^i\}_{[n]}$ of random variables. That is, we suppose there is a bijective correspondence between graph nodes and random variables in $\{\mathsf{X}^i\}_{[n]}$ and, leaning on this correspondence, we simply label nodes of $\mathcal{G}$ with the random variables.

We also suppose we have surjective $h : R \to \mathcal{P}([n])$ such that $h : (x^0, ..., x^n) \mapsto \{i | x^i \neq \#\}$. That is, h picks out the active components of r. Define $\mathsf{X}^{i\prime} : R \to \{\#\} \cup X^i$ by the function returning the i-th element of r for $r \in R$. We identify primed and unprimed random variables in the obvious way.

Let $\Pi_{\mathsf{X}^i} := \delta_{\pi_{\mathsf{X}^i}}$ be the Markov kernel that takes the marginal distribution of X^i, and denote by $\mathbf{P}_{r|A}\Pi_{\mathsf{X}^i} : E \to \Delta(\mathcal{X}^i)$ the conditional probability of X^i given the set of random variables A.

Definition 4.1 (Causal Bayesian Network)**.** Given discrete R, E, $\mathbf{P} : R \to \Delta(\mathcal{E})$ and $\{\mathsf{X}^i\}_{i \in [n]}$, a Causal Bayesian Network (CBN) compatible with $\mathbf{P}$ is a directed acyclic graph (DAG) $\mathcal{G}$ of degree n such that for all $r \in R$

1. $\mathbf{P}_r$ is compatible with $\mathcal{G}$ (see Pearl (2009))

2. For all $i \in h(r)$, $\mathbf{P}_r \Pi_{\mathsf{X}^i} = \delta_{\mathsf{X}^{i\prime}(r)}$
3. For all $i \notin h(r)$, $\mathbf{P}_{r|\mathrm{Pa}_{\mathcal{G}}(\mathsf{X}^i)} \Pi_{\mathsf{X}^i} = \mathbf{P}_{\underline{\#}|\mathrm{Pa}_{\mathcal{G}}(\mathsf{X}^i)} \Pi_{\mathsf{X}^i}$, $\mathbf{P}_{\underline{\#}}$-almost surely

This definition differs slightly from Pearl (2009) in that $\mathbf{P}$ is a Markov kernel rather than a set of labeled elements of $\Delta(\mathcal{E})$.

Given a graph $\mathcal{G}$ and a measure $\mu \in \Delta(\mathcal{E})$ compatible with $\mathcal{G}$ we can define a class of stochastic maps $\mathcal{K} \subset \Delta(\mathcal{E})^V$ such that every $\mathbf{P} \in \mathcal{K}$ is compatible with $\mathcal{G}$ and $\mathbf{P}(\#) = \mu$. Let the notation $\mathcal{G}(\mu)$ stand for the set $\mathcal{K}$ as defined here; note that $\mathcal{G}(\mu)$ is in general set-valued.

We have from this definition for any $r \in V$ the *truncated factorisation* property:

$$P_r F_{\mathbf{X}}(A) = \prod_{i \in h(r)} \delta_{\mathsf{X}^{i\prime}(r)}(\mathsf{X}^i(A)) \sum_{a \in A} \prod_{i \notin h(r)} \mathbf{P}_{\#|\mathrm{Pa}_{\mathcal{G}}(\mathsf{X}^i)} \Pi_{\mathsf{X}^i}(a; \{\mathsf{X}^i(a)\}) \tag{4}$$

As a consequence of the existence of conditional probability for standard measurable spaces, provided μ is compatible with $\mathcal{G}$ we have that the right hand side of4 exists, and so $\mathcal{G}(\mu)$ is non-empty. If μ is positive definite this relationship is functional; in such a case we could treat $\mathcal{G}(\mu)$ as a function from $\Delta(\mathcal{E})$ to interventional maps $\mathbf{P}$.

Suppose we define some arbitrary hypothesis class $\mathcal{H}^{\mathcal{G}} \subset \Delta(\mathcal{E})$ of possible observed distributions. Then let $\Theta := \{(\mu, \mathbf{P}) | \mu \in \mathcal{H}, \mathbf{P} \in \mathcal{G}(\mu)\}$ and define $\mathbf{T}^{\mathcal{G}} : \Theta \times R \to \Delta(\mathcal{E}^2)$ by $(\mu, \mathbf{P}, r) \mapsto \mu \otimes \mathbf{P}_r$. It is natural to consider $\mathbf{T}^{\mathcal{G}}$ the causal theory represented by $\mathcal{G}$ for two reasons: first, it is a natural construction from the definition of a CBN if we take the set of possible *do*-interventions to be the set of decisions for $\mathbf{T}$. Secondly, if we take $\mathcal{H} = \Delta(\mathcal{E})$ then the map from DAGs to causal theories is injective (this is in contrast to, for example, the map from DAGs to probability distributions as in ordinary Bayesian networks(Bishop, 2006)).

Theorem 4.2 (The map $\mathcal{G} \mapsto \mathbf{T}^{\mathcal{G}}$ is injective)**.** *For DAGs $\mathcal{G}$, $\mathcal{G}'$ on the same set of RV's $\{\mathsf{X}^i\}_{[n]}$, $\mathcal{G} \neq \mathcal{G}' \implies \mathcal{T}^{\mathcal{G}} \neq \mathcal{T}^{\mathcal{G}'}$ if these theories are induced by a complete hypothesis class.*

Proof. $\mathcal{G}$ and $\mathcal{G}'$ must disagree on at least one parental set. Suppose this is on the parents of X^i. Choose some μ such that $\mu_{|\mathrm{Pa}_{\mathcal{G}}(\mathsf{X}^i)} \Pi_{\mathsf{X}^i} \neq \mu_{|\mathrm{Pa}_{\mathcal{G}'}(\mathsf{X}^i)} \Pi_{\mathsf{X}^i}$. By the non-equality of these conditional probabilities there are some r, r' such that $h(r) = h(r') = \mathrm{Pa}_{\mathcal{G}}(\mathsf{X}^i) \cup \mathrm{Pa}_{\mathcal{G}'}(\mathsf{X}^i)$, $\mathrm{Pa}_{\mathcal{G}}(\mathsf{X}'^i)(r) = \mathrm{Pa}_{\mathcal{G}}(\mathsf{X}'^i)(r')$ but $\mathbf{P}_r^{\mathcal{G}'} \Pi_{\mathsf{X}^i} \neq \mathbf{P}_{r'}^{\mathcal{G}'} \Pi_{\mathsf{X}^i}$, but we also have $\mathbf{P}_r^{\mathcal{G}} \Pi_{\mathsf{X}^i} = \mathbf{P}_{r'}^{\mathcal{G}} \Pi_{\mathsf{X}^i}$ by equality of r and r' on $\mathrm{Pa}_{\mathcal{G}}(\mathsf{X}^i)$. Thus $\mathbf{T}^{\mathcal{G}} \neq \mathbf{T}^{\mathcal{G}}$. □

4.1 Causal Bayesian Networks Induce Rich Causal Theories

The causal theory $\mathbf{T}^{\mathcal{G}}$ associated with a CBN $\mathcal{G}$ features a large number of decisions. Given some utility function $u : \times_{i \in [n]} X^i \to \mathbb{R}$, there is always a decision that fixes the values of all X^i deterministically to a value maximising u, if such a maximum exists. Clearly, for a practical decision problem $\mathbf{T}^{\mathcal{G}}$ is inappropriate. There may be some cases where there is an more realistic theory $\mathbf{T}^{\mathcal{G}\prime}$ closely related to $\mathbf{T}^{\mathcal{G}}$ but that features, for example, decisions limited to interventions on particular variables. To actually solve a decision problem, it is necessary to move from the theory $\mathbf{T}^{\mathcal{G}}$ to such a modified theory. We call theories such as $\mathbf{T}^{\mathcal{G}}$ *rich theories* and their associated decision sets D *rich decision sets.*

5 Potential outcomes models

We present one formalisation of potential outcomes (we do not claim it is authoritative) based on Rubin (2005), and note any points of divergence.

We will eschew any discussion of sequences. Following the convention set out in the introduction, we will interchangeably use sans serif letters to refer to "random variables" and "particular strings in the string diagram". W is the treatment assignment taking values in $\{0, 1\}$, $\mathsf{Y}(0)$ $\mathsf{Y}(1)$ are the potential outcomes taking values in Y, Y is the observed outcome also taking values in Y and X is a "vector of background facts" taking values in X.

Given an underlying state space Θ, a potential outcomes model $\mathcal{O}$ consists of a set of Markov kernels $\langle \mathbf{P}, \mathbf{W}, \mathbf{Y} \rangle$ and a canonical composition that yields a statistical experiment $\mathbf{H} : \Theta \to \Delta(\mathcal{X} \otimes \mathcal{Y} \otimes \mathcal{W})$.

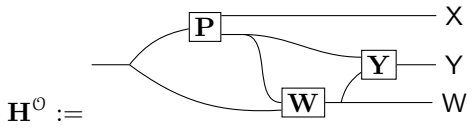
The kernels are:

- A "model of the science", $\mathbf{P} : \Theta \to \Delta(\mathcal{X} \otimes \mathcal{Y} \otimes \mathcal{Y})$ (In Rubin's notation, $\mathbf{P}$ is $\prod_i f(\mathsf{X}_i, \mathsf{Y}_i(0), \mathsf{Y}_i(1) | \theta)$, though as noted we do not consider sequences here)
- An "assignment mechanism", $\mathbf{W} : \Theta \times X \times Y^2 \to \Delta(\{0, 1\})$ (in Rubin's notation, $\mathbf{W}$ is $Pr(\mathsf{W} | X, Y(1), Y(0)))$
- An "observation model", $\mathbf{Y} : \{0, 1\} \times Y^2 \to \Delta(\mathcal{Y})$, defined explicitly as $\mathbf{Y} : (\mathbf{y}^0, \mathbf{y}^1, \mathbf{w}) \mapsto (1 - \mathbf{w}) \odot \delta_{\mathbf{y}^0} + \mathbf{w} \odot \delta_{\mathbf{y}^0}$ where $\odot$ is the elementwise product

We differ from Rubin by defining $\mathbf{Y}$ as a Markov kernel rather than a function. This approach means that we can at best assert $W = w \implies \mathsf{Y} = \mathsf{Y}(w)$ *almost*

surely with respect to some probability measure, as a Markov kernel cannot guarantee exact equality. We also differ from Rubin by including Θ in the domain of $\mathbf{W}$ as in our framework leaving this dependence out is equivalent to assuming that the treatment assignment mechanism is known *a priori* (as a result, our state Θ is larger than Rubin's).

We then define the *canonical experiment* $\mathbf{H}^{\mathcal{O}}$ by



(5)

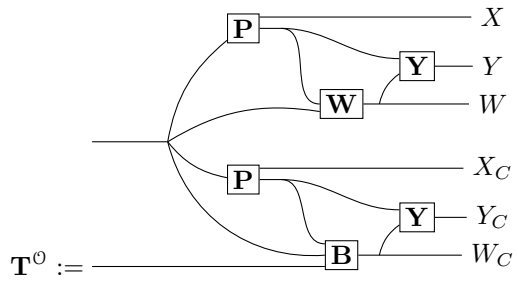
We have labeled the wires carrying the three observed random variables X, Y and W. The potential outcomes $[\mathsf{Y}(0), \mathsf{Y}(1)]$ are jointly carried by the lower wire exiting from $\mathbf{P}$ – drawing separate wires would make the diagram quite crowded. Apart from the connection between Θ and $\mathbf{W}$, this composition is consistent with Rubin, though the notation is substantially different.

5.1 Are potential outcomes models causal theories?

By assumption, a potential outcomes model induces a canonical statistical experiment. Given that potential outcomes models are a type of causal model, we can ask whether we can induce a canonical *causal theory*. It is not possible to do this as naturally as with causal Bayesian networks because potential outcomes models do not feature consequence maps. Nevertheless, a causal theory can be induced by considering the science $\mathbf{P}$ to be fixed and the assignment function $\mathbf{W}$ to be variable, which seems to us to be in line with the ideas underpinning the potential outcomes approach. In addition, the rule we obtain distinguishes every potential outcomes model with different science $\mathbf{P}$, which we consider a minimal requirement for any rule claiming to represent potential outcomes models as causal theories.

Suppose a potential outcomes model $\mathcal{O}$ is equipped with discrete spaces $\Theta, X, Y, \{0,1\}$ and define $D := [0,1]^{|\Theta|+2|Y|+|X|}$. There is a kernel $\mathbf{B} : D \times \Theta \times X \times Y^2 \to \Delta(\mathcal{W})$ such that for every Markov kernel $\mathbf{W}' : \Theta \times X \times Y^m \to \Delta(\{0,1\})$ there exists $d \in D$ with $\mathbf{W}' = \mathbf{B}_d$; that is, D indexes the set possible treatment assignment maps. The assumption of discrete spaces is to guarantee the existence of such a $\mathbf{B}$.

From $\mathcal{O}$ and $\mathbf{B}$ we define the *canonical theory* $\mathbf{T}^{\mathcal{O}}$:



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$\mathbf{T}^{\mathcal{O}}$ is two parallel copies of $\mathcal{H}^{\mathcal{O}}$ where $\mathbf{W}$ is replaced by $\mathbf{B}$ in the lower version.

Theorem 5.1. *Given potential outcomes models* $\mathcal{O} = \langle \mathbf{P}, \mathbf{W}, \mathbf{Y} \rangle$, $\mathcal{O}' = \langle \mathbf{P}', \mathbf{W}', \mathbf{Y} \rangle$ *sharing spaces* $\Theta, X, Y, [m]$, *then* $\mathbf{T}^{\mathcal{O}} = \mathbf{T}^{\mathcal{O}'}$ *if and only if* $\mathbf{P} = \mathbf{P}'$ *and* $\mathbf{H} = \mathbf{H}'$.

Proof. Let $\mathbf{T} := \mathbf{T}^{\mathcal{O}}$ and $\mathbf{T}' := \mathbf{T}^{\mathcal{O}'}$.

If $\mathbf{P} = \mathbf{P}'$ and $\mathbf{H} = \mathbf{H}'$ we clearly have $\mathbf{T}(* \otimes \mathrm{Id}) = \mathbf{T}'(* \otimes \mathrm{Id}) := \mathbf{C}$ as all kernels in the bottom half of 6 ($\mathbf{P}, \mathbf{B}$ and $\mathbf{Y}$) are the same by definition. But then $\mathbf{T} = \curlyvee(\mathbf{H} \otimes \mathbf{C}) = \mathbf{T}'$.

Suppose $\mathbf{T} = \mathbf{T}'$ and $\mathbf{P} \neq \mathbf{P}'$. Then there exists some $A \in \mathcal{X} \otimes \mathcal{Y}^2$, $\theta \in \Theta$ such that $\mathbf{P}_\theta(A) \neq \mathbf{P}'_\theta(A)$. Choose $d \in D$ such that $\mathbf{B}_{d,\theta,x,y_0,y_1} = \delta_0$ if $(x, y_0, y_1) \in A$ and $\mathbf{B}_{d,\theta,x,y_0,y_1} = \delta_1$ otherwise. Then $\mathbf{T}^{\mathcal{O}}_{\theta,d}\Pi_{\mathsf{W}}(\{0\}) = \mathbf{P}_\theta(A) \neq \mathbf{P}'_\theta(A) = \mathbf{T}^{\mathcal{O}'}_{\theta,d}\Pi_{\mathsf{W}}(\{0\})$, a contradiction. Thus $\mathbf{P} = \mathbf{P}'$. In addition, $\mathbf{H} = \mathbf{T}(\mathrm{Id} \otimes *) = \mathbf{T}'(\mathrm{Id} \otimes *) = \mathbf{H}'$. □

Note that the assignment $\mathbf{W}$ may differ between $\mathcal{O}$ and $\mathcal{O}'$. Suppose $X = \emptyset$, $Y = \{0,1\}$ and for some θ, $\mathbf{P}_\theta = \frac{1}{4}(\delta_{0,0} + \delta_{0,1} + \delta_{1,0} + \delta_{1,1})$. Then $\mathbf{W}_\theta : (y_0, y_1) \mapsto [\![y_0 = y_1]\!]\delta_0 + [\![y_0 \neq y_1]\!]\delta_1$ and $\mathbf{W}'_\theta := 1 - \mathbf{W}_\theta$ both yield the same observations $\mathbf{H}_\theta$. It may be desirable that a mapping from PO models to causal theories also distinguishes PO models that differ only on $\mathbf{W}$ - for example, we might want some decision $d \in D$ to always be interpretable as "in the state θ, raise the probability of treatment above the observed level iff $y_0 \neq y_1$", a decision which would yield different consequences given a theory based on $\mathbf{W}_\theta$ or $\mathbf{W}'_\theta$.

6 Potential Outcomes Models Induce Rich Causal Theories

Though the theory $\mathbf{T}^{\mathcal{O}}$ appears to be quite different in a number of ways from theories induced by a causal graph $\mathbf{T}^{\mathcal{G}}$, it also features a rich set of decisions. If it is possible to choose any assignment function from $\Theta \times X \times Y^2 \to \Delta(\{0,1\})$, then it is (for example)

possible to choose a functon that assigns treatment if and only if $y_1 > y_0$ independent of state θ.

As with causal Bayesian networks, for a real decision problem we would like to work with a realistic theory $\mathbf{T}^r$ on a set of decisions D^r that correspond to actions we believe we can actually take. However, we may be willing to accept that the realistic theory $\mathbf{T}^r$ corresponds closely to the rich theory $\mathbf{T}^{\mathcal{O}}$ with, for example, a limited set of decisions. To illustrate this, consider the problem of evaluating the "effect of assigning treatment" vs "the effect of receiving treatment". From Shrier et al. (2017):

> In public health, we are normally concerned with the effect of assigning a treatment. If we implement a prevention or treatment program that is efficacious only under strict research conditions but people in the real world would not receive it for any possible reason, the program will not be effective. This real-world context is best estimated by the intention-to-treat (ITT) analysis [...]
>
> There are 2 reasons why the average causal effect of receiving a treatment may be more important than the ITT for some people. First, investigators may want to know what the average causal effect of a treatment program would be if they could improve participation in the program. [...] Also, the average causal effect of receiving a treatment is of primary interest to a patient deciding whether or not to take the treatment as recommended.

Concretely, suppose we have two rich theories $\mathbf{T}^{\mathrm{ITT}}$ and $\mathbf{T}^{\mathrm{RT}}$ modelling effects of intention-to-treat and receiving treatment respectively, and we want realistic theories $\mathbf{T}^p, \mathbf{T}^t$ describing the effects of prescribing treatment and taking treatment respectively. Consider the first decision problem: we have two decisions $D^p = \{d_0, d_1\}$ where d_1 corresponds to "implement a treatment program" and d_0 corresponds to "do nothing". Under the intention to treat model $\mathbf{T}^{\mathrm{ITT}}$ it is plausible that these decisions correspond to determinisically setting $\mathsf{W} = 1$ and $\mathsf{W} = 0$ respectively. In detail, we suppose that the consequence of choosing d_1 in the pragmatic theory $\mathbf{T}^p$ is the same as the consequence of choosing the decision $e_1 \in D^{\mathrm{ITT}}$ such that for all θ, x, y_0, y_1, $\mathbf{B}^{\mathrm{ITT}}_{\theta,d,x,y_0,y_1} = \delta_1$ and analogously d_0 corresponds to the element $e_0 \in D^{\mathrm{ITT}}$ that sets W to 0.

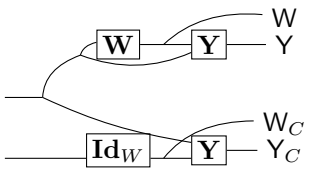
Consider the same problem – that of implementing a treatment program – for the rich theory of receiving treatment $\mathbf{T}^{\mathrm{RT}}$. Here, a correspondence between D^p and D^{RT} is less clear. We may accept that implementing a treatment program corresponds to *some* choice of treatment taking function $\mathbf{B}^{\mathrm{RT}}$, one that is perhaps more likely to result in treatment than that for doing nothing. However, without more information we have only that there is a correspondence between D^{RT} and D^p and not what this correspondence is. We could, for example, express our uncertainty with a set of possible correspondences or a probability measure over over this set and proceed from there.

Consider the third decision problem, where decisions correspond to taking the treatment, and in particular consider using the rich theory $\mathbf{T}^{\mathrm{ITT}}$. It is very likely that *no* choice of prescription function $\mathsf{W}^{\mathrm{ITT}}$ is consistent with the test subjects always taking the treatment. In this case we're not just uncertain about the correspondence between rich decisions D^{ITT} and realistic decisions D^t – we are are actually confident that there is no such correspondence.

7 Comparing Induced Causal Theories

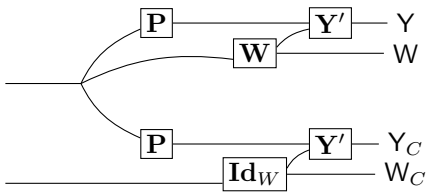
Causal theories induced by CBN and PO models are directly comparable, and the induces theories are typically rather different. For example, a CBN $\mathcal{G}$ defines an intervention operation for every random variable that has been represented as a node of $\mathcal{G}$, which is reflected in $\mathbf{T}^{\mathcal{G}}$, while an induced PO theory $\mathbf{T}^{\mathcal{O}}$ will typically not allow any decisions that deterministically set Y in all states, and no decisions may affect X at all. On the other hand, decisions in a theory $\mathbf{T}^{\mathcal{O}}$ allow W to depend arbitrarily on X and θ together, which is not possible for a theory induced by a CBN. Despite these differences, when we move from rich theories to realistic theories we may end up with the same result using either method.

Suppose we have a CBN $\mathcal{G} := W \twoheadrightarrow Y$, where W and Y are random variables taking values in some arbitrary spaces W and Y. Suppose also that we require a realistic theory $\mathbf{T}^{\mathcal{G}} : \Theta \times W \to \Delta([\mathcal{W} \otimes \mathcal{Y}]^2)$ where our decisions correspond only to "hard do interventions" $do(W = w)$ on W under the full CBN theory – that is, we have no decisions corresponding to do-interventions on Y or do-nothing. Then there exist Markov kernels $\mathbf{W} : \Theta \to \Delta(\mathcal{W})$, $\mathbf{Y} : \Theta \times W \to \Delta(\mathcal{Y})$ such that $\mathbf{T}^{\mathcal{G}}$ can be represented as in the diagram 7. Conversely, any causal theory that can be represented in this manner is a candidate for $\mathbf{T}^{\mathcal{G}}$ (see A)



(7)

Suppose we have a PO model $\mathcal{O} = \langle \mathbf{P}, \mathbf{W}, \mathbf{Y} \rangle$ on Θ, W and Y such that $\mathbf{W}$ depends only on Θ. Suppose also that we require a realistic theory where, similarly to the case above, decisions correspond only to "setting" W in the standard theory associated with $\mathcal{O}$. Then the resulting theory can be represented by the diagram 8. Conversely, there is a PO model for every causal theory with this representation.



(8)

In the lower diagram we can define $\mathbf{Y}^* := (\mathbf{P} \otimes \mathbf{Id})\mathbf{Y}'$ to produce a diagram that is equivalent to 7. While $\mathbf{P}$ and $\mathbf{W}$ are arbitrary, $\mathbf{Y}'$ has a particular form. It is still possible to express a general kernel $\mathbf{Y} : \Theta \times W \to \Delta(\mathcal{Y})$ in the form of $\mathbf{Y}^*$; let $\mathbf{P} : \theta \mapsto \mathbf{Y}_{\theta,0} \otimes \mathbf{Y}_{\theta,1}$. Then $\mathbf{Y}^* = \mathbf{Y}$. Thus under the restrictions given, the sets of viable realistic theories derived from the CBN and the PO models are exactly the same.

7.1 Coarsening and Reusable Inferences

We turn our attention in more detail to the operation of finding a "realistic" theory $\mathbf{T}$ that corresponds to a rich theory $\mathbf{T}^*$. As a motivation for this development, note that proponents of both approaches discussed here have advocated for the universality of the "causal effects" their models represent:

> The perspective that (1) the science exists independently of how we try to learn about it and that (2) if the model used for analysis of the resulting data is approximately correct, then the resulting posterior distribution will give a fair summary of the current state of knowledge of that science seems, at least to me, consistent with common views of the scientific enterprise [...] The potential outcomes, together with covariates, define the science in the sense that all causal estimands are functions of these values (Rubin, 2005)

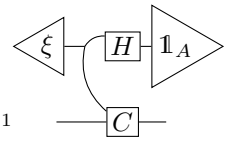
> By representing the domain in the form of an assembly of stable mechanisms, we have in fact created an oracle capable of answering queries about the effects of a huge set of actions and action combinations (Pearl, 2009)

We present here a somewhat speculative account of what both of these approaches are trying to achieve based on the notion of *coarsening*. The basic story is: suppose our job is to study the causal dynamics of some system, but we're not quite sure of who will put our results to use or what decisions they will have available. We adopt a rich theory $\mathbf{T}^*$ with the hope that our results can be useful to end users, even if they are operating with a more realistic theory $\mathbf{T}$. Here we show that if $\mathbf{T}$ is related to $\mathbf{T}^*$ via a coarsening, inference on $\mathbf{T}^*$ can be reused on $\mathbf{T}$.

Definition 7.1 (Coarsening)**.** A theory $\mathbf{T}^* : \Theta \times D \to \Delta(\mathcal{E} \otimes \mathcal{F})$ can be coarsened to a theory $\mathbf{T} : \Theta \times D' \to \Delta(\mathcal{E} \otimes \mathcal{F})$ if there exists $M : D' \to \Delta(\mathcal{D})$ such that $\mathbf{T} = (\mathbf{Id} \otimes M)\mathbf{T}^*$.

We recall the convention for denoting by $\mathbf{H}$ the statistical experiment associated with a causal theory $\mathbf{T}^*$. That is, $\mathbf{H} := \mathbf{T}(\mathbf{Id} \otimes *)$.

Given $\mathbf{T}^*$, an event $A \in \mathcal{E}$ with $\xi\mathbf{H}^*\mathbb{1}_A > 0$, write the theory conditioned on A as $\mathbf{T}_\xi|A : D \to \Delta(\mathcal{F})$, defined as



$$\mathbf{T}_\xi|A := (\xi\mathbf{H}(A))^{-1} \tag{9}$$

Note that $\mathbf{T}_\xi|A$ along with a strategy $\gamma \in \Delta(\mathcal{D})$ is the conditional probability of F by the elementary definition – for $B \in \mathcal{F}$, $\mathbf{T}_{\xi,\gamma}|A : B \mapsto \frac{(\xi \otimes \gamma)\mathbf{T}(A,B))}{\xi\mathbf{H}(A)}$.

Theorem 7.2. *Given* $\mathbf{T}^* : \Theta \times D^* \to \Delta(\mathcal{E} \otimes \mathcal{F})$ *and* $\mathbf{T} : \Theta \times D \to \Delta(\mathcal{E} \otimes \mathcal{F})$*, there exists* $\mathbf{M}$ *such that* $\mathbf{T}_{\gamma,\xi}|A = \gamma\mathbf{M}\mathbf{T}^*_\xi|A$ *for all* $\xi \in \Delta(\Theta)$*,* $\gamma \in \Delta(\mathcal{D})$ *and* $A \in \mathcal{E}$ *where* $\xi\mathbf{H}^*(A) > 0$ *if and only if* $\mathbf{T}$ *is a coarsening of* $\mathbf{T}^*$*.*

Proof. This follows from the fact that $\xi\mathbf{H}^*(A) > 0$ iff $\xi\mathbf{H}(A) > 0$ and that the two Markov kernels $\mathbf{T}$ and $(\mathbf{Id} \otimes M)\mathbf{T}^*$ are equal if and only if they are equal on all inputs and outputs. See B for a complete proof. □

In other words, if and only if $\mathbf{T}$ can be coarsened to $\mathbf{T}'$ then we can "save" the results of conditioning $\mathbf{T}^*_\xi$ on A via $\mathbf{T}^*_\xi|A$, which is a kernel $D \to \Delta(\mathcal{F})$. We can then "reuse" this kernel to determine the consequences of some mixed decision γ on $\mathbf{T}$ via $\gamma\mathbf{M}\mathbf{T}^*_\xi|A$.

Recall our discussion of the problems of determining the effects of a treatment program and determining the effects of taking the treatment. We had established that there was known correspondence between D^{ITT} and D^p – this correspondence was, precisely, a coarsening from $\mathbf{T}^{\mathrm{ITT}}$ to $\mathbf{T}^p$ (concretely, it proceeds via the kernel $\mathbf{M} : 1 \mapsto \delta_{e_1}$ and $0 \mapsto \delta_{e_0}$). On the other hand, while we didn't identify a known coarsening from $\mathbf{T}^{\mathrm{RT}}$ to $\mathbf{T}^p$, we were argued that such a coarsening likely existed. If we represented our uncertainty in this second case with

a Markov kernel – i.e. a mixture over correspondences $D^p \to D^{\mathrm{ITT}}$ then we would also have a coarsening. Finally, we rejected the possibility of a coarsening from $\mathbf{T}^{\mathrm{ITT}}$ to $\mathbf{T}^t$.

7.2 Limits of Coarsening

We postulate that at least one of the aims of the modelling approaches we study here is to yield rich causal theories can be coarsened to a wide variety of useful realistic theories. One way that such theories might be useful is as follows: suppose we do not know the appropriate theory $\mathbf{T}$, but we do believe that it should be a coarsening of $\mathbf{T}^*$. In that case, all we have to do is work out which decisions in D correspond to which mixtures of decisions in D^*, a task that may be substantially easier than determining the full causal theory $\mathbf{T}$; it is the difference between "what are the consequences of d_0" and "given that d_0 is a do-intervention, is it $do(X = x)$?"

There are limits on this enterprise, however. If a theory $\mathbf{T}^*$ can be coarsened to the entire set of viable realistic theories then there must be at least as many coarsenings as realistic theories. If factorisation into a rich theory and a coarsening helps with the problem of choosing a causal theory, it cannot be by reducing the number of options available to choose from.

8 Discussion

We have introduced an original approach to formulating questions of causal inference and analysing approaches to causal modelling. We take cues from statistical decision theory and make heavy use of the theory of Markov kernels for reasoning about causal theories, the central object of our approach. Our approach makes crystal clear the distinction between "statistical" and "causal" knowledge – the former is represented by a statistical experiment and the latter by a causal theory. Causal Bayesian networks and Potenial Outcomes models can both be used to generate causal theories.

While we do not address the unique questions that can be posed using counterfactual models (Pearl, 2009), our approach suggests an alternative view for the relationship between counterfactual and interventional causal models. Rather than occupying different levels of a hierarchy, each yields a different kind of rich causal theory. We might speculate that CBN theories are particularly suited to some domains and PO theories to others. Indeed, we see extensive discussion of counterfactual treatment effects in the econometrics literature, where decisions usually involve changing incentives which can plausibly be understood as altering the assignment function $\mathbf{W}$ in unpredictable ways (Angrist and Pischke, 2014; Carneiro et al., 2010; Imbens and Angrist, 1994). Causal Bayesian Networks, on the other hand, have found applications in the study of biological systems which typically feature large numbers of variables which permit a wide variety of targetted interventions (Sachs et al., 2005; Maathuis et al., 2009).

Though we develop CSDT in the context of "small world decision problems" (Joyce, 1999), we can apply causal theories to the study of questions beyond this context, such as the questions of coarsening and reusable inference discussed here. Theorem 7.2 is a means by which rich theories can inform decisions involving more realistic theories, though it is by no means the only one. It raises a number of questions: what other methods allow for inferences to be reused? Given that we know a coarsening exists, how much do we need to know about the realistic theory in order to determine what this coarsening is? More broadly, how can we formally pose the question "what makes a rich causal theory a 'good' one"?

A number of the results here are predicated on discrete spaces which allows us to disregard questions of measurability. A second important direction is extending this theory to continuous spaces and understanding what limitations this introduces. Relatedly, the notions of conditional probability, conditioning, independence and Bayesian inversion are well understood in the context of probability measures, including in their string diagrammatic treatment (Cho and Jacobs, 2019), but we are not aware of analogues of these notions for general Markov kernels. These basic notions would be invaluable tools in the analysis of causal theories.

The string diagram notation we use has a strong connection with the DAGs (Fong, 2013) used in causal graphical models as well as to influence diagrams(Dawid, 2002), as do Markov kernels themselves. It would not be surprising if there were a deep connection between the two.

Causal statistical decision theory is new, and many details are still being worked out. We have shown how CSDT can bring new understanding to existing approaches to causality by formalising fundamental but previously informal notions such as that of "stable causal models", and we believe that it is an approach that has a great deal of potential in furthering our understanding of causal inference.

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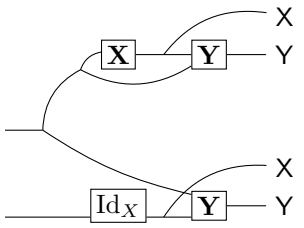
Appendix:

A CBN representation as a causal theory

Definition A.1 (Elementary Causal Bayesian Network)**.** Given D, E, Θ, random variables $\{\mathsf{X}^i\}_{i\in[n]}$ on E, a distinguished variable X^0 taking values in D and a causal theory $\mathbf{T} : \Theta \times D \to \Delta(\mathcal{E} \otimes \mathcal{E})$ with $\mathbf{H} := \mathbf{T}(\mathrm{Id}_E \otimes *_E)$ and $\mathbf{C} := \mathbf{T}(*_E \otimes \mathrm{Id}_E)$, an *elementary Causal Bayesian Network* (eCBN) compatible with $\mathbf{T}$ is a directed acyclic graph (DAG) $\mathcal{G}$ with nodes $\{\mathsf{X}^i\}_{i\in[n]}$ such that

1. $\mathbf{H}_\theta$ and $\mathbf{C}_{\theta,d}$ are compatible with $\mathcal{G}$ (see Pearl (2009))
2. $\mathbf{C}_{\theta,d}\Pi_{\mathsf{X}^i} = \delta_d$
3. For all $i \neq 0$, $\mathbf{C}_{\theta|\mathrm{Pa}_\mathcal{G}(\mathsf{X}^i)}\Pi_{\mathsf{X}^i} = \mathbf{H}_{\theta|\mathrm{Pa}_\mathcal{G}(\mathsf{X}^i)}\Pi_{\mathsf{X}^i}$, $\mathbf{H}_\theta$-almost surely

Suppose we have the EDAG $\mathcal{G} := X \twoheadrightarrow Y$, where X and Y are random variables taking values in some arbitrary spaces X and Y. Then $\mathcal{G}$ is compatible with a causal theory $\mathbf{T} : \Theta \times X \to \Delta([\mathcal{X} \otimes \mathcal{Y}]^2)$ if and only if there exist Markov kernels $\mathbf{X} : \Theta \to \Delta(\mathcal{X})$, $\mathbf{Y} : \Theta \times X \to \Delta(\mathcal{Y})$ such that



(10)

Here we represent the identity kernel explicitly to make clear that it replaces $\mathbf{X}$ in the lower part of the diagram. This fact is hidden by the usual convention of representing the identity by a bare wire.

Proof. Condition 1 is vacuous.

Condition 2 is equivalent to asserting that $C_\theta\Pi_{\mathsf{X}^i}$ is the identity map.

Suppose $\mathbf{T}$ is compatible with $\mathcal{G}$. Then for any θ, d, both $\mathbf{C}_{\theta,d|\{\mathsf{X}\}}\Pi_\mathsf{Y}$ and $\mathbf{H}_{\theta|\{\mathsf{X}\}}\Pi_\mathsf{Y}$ must be $\mathbf{H}_\theta$ almost surely equal, and both may be arbitrary on any set of measure 0. $X \times Y$ is a discrete space, so they may both in fact be arbitrary on every set of measure 0. Thus for all events they are either equal or jointly arbitrary, and so we can always choose them to be equal. Defining the resulting choice $\mathbf{Y}$, it is a property of conditional probability that the construction in 10 agrees with $\mathbf{T}$ for any θ, d, and hence the construction is equal to $\mathbf{T}$.

Suppose $\mathbf{T}$ is represented by 10. Then $\mathbf{C}_{\theta|\{\mathsf{X}\}}\Pi_\mathsf{Y}$ and $\mathbf{H}_{\theta|\{\mathsf{X}\}}\Pi_\mathsf{Y}$ are equal, so they are equal $\mathbf{H}_\theta$ almost surely. □

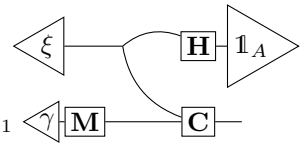
An interesting feature of this representation is the fact that the edge cutting behaviour, usually an implicit part of the definition of a CBN, is displayed explicitly by replacing $\mathbf{X}$ with the identity kernel. We also note that 10 has what looks like a backdoor path between $\mathbf{X}$ and $\mathbf{Y}$ which is not in $\mathcal{G}$. Though we do not know which if any correspondences between DAG and causal theory diagrams exist, we speculate that this backdoor is "blocked" by the fact that we invoke particular values of θ in the defintion of an eCBN.

B Reusable Inference

Theorem B.1. *Given* $\mathbf{T}^* : \Theta \times D^* \to \Delta(\mathcal{E} \otimes \mathcal{F})$ *and* $\mathbf{T} : \Theta \times D \to \Delta(\mathcal{E} \otimes \mathcal{F})$*, there exists* $\mathbf{M}$ *such that* $\mathbf{T}_{\gamma,\xi}|A = \gamma\mathbf{M}\mathbf{T}^*_\xi|A$ *for all* $\xi \in \Delta(\Theta)$*,* $\gamma \in \Delta(\mathcal{D})$ *and* $A \in \mathcal{E}$ *where* $\xi\mathbf{H}^*(A) > 0$ *if and only if* $\mathbf{T}$ *is a coarsening of* $\mathbf{T}^*$*.*

Proof. Let the coarsening from $\mathbf{T}^*$ to $\mathbf{T}$ be induced by $\mathbf{M} : D \to \Delta(\mathcal{D}^*)$. For arbitrary ξ, A such that $\xi\mathbf{H}^*(A) > 0$ and arbitrary γ:

$\gamma\mathbf{M}\mathbf{T}^*_\xi|A = (\xi\mathbf{H}^*(A))^{-1}$



(11)

$$= (\xi\mathbf{H}(A))^{-1}(\xi \otimes \gamma)\mathbf{T}(\mathbb{1}_A \otimes \mathrm{Id}_F) \quad (12)$$

$$= \mathbf{T}_{\gamma,\xi}|A \quad (13)$$

Where 12 follows from the fact that $\mathbf{T}$ is by assumption equal to the central subdiagram on line 11.

Suppose we have $\mathbf{M}$ such that $\mathbf{T}_{\gamma,\xi}|A = \gamma M\mathbf{T}^*_\xi|A$ for all $\xi \in \Delta(\Theta)$, $\gamma \in \Delta(\mathcal{D})$ and $A \in \mathcal{E}$ where $\xi\mathbf{H}^*(A) > 0$. Choose some arbitrary θ. Then for *all* $A \in \mathcal{E}$ either $\mathbf{H}^*_\theta(A) = 0$ in which case $\mathbf{M}\mathbf{T}^*_{\theta,d}(A \times B) = \mathbf{T}_{\theta,d}(A \times B) = 0$ for all $d \in D, B \in \mathcal{F}$ or $\mathbf{H}^*_\theta(A) > 0$ in which case for all d, B:

$$(\mathbf{H}^*_\theta(A))^{-1}(\delta_\theta \otimes \delta_d)\mathbf{T}(\mathbb{1}_A \otimes \mathbb{1}_B) \quad (14)$$

$$= (\mathbf{H}^*_\theta(A))^{-1}(\delta_\theta \otimes \delta_d\mathbf{M})\mathbf{T}^*(\mathbb{1}_A \otimes \mathbb{1}_B) \quad (15)$$

$$\therefore (\delta_\theta \otimes \delta_{d'})\mathbf{T}(\mathbb{1}_A \otimes \mathbb{1}_B) \quad (16)$$

$$= (\delta_\theta \otimes \delta_{d'}\mathbf{M})\mathbf{T}^*(\mathbb{1}_A \otimes \mathbb{1}_B) \quad (17)$$

Hence

$$(\mathbf{Id} \otimes \mathbf{M})\mathbf{T} = \mathbf{T}' \quad (18)$$

□