
AISTats Submission Sept 26: up to "CBNs are causal theories"

1 Introduction

It is widely accepted that causal knowledge and statistical knowledge are distinct. At least two levels are common: statistics is concerned with *association* while causation is concerned with *consequences*; a distinction of this nature goes back at least to Hume [Morris and Brown, 2019], who is also noted for his argument that knowledge of the latter cannot be reduced to the former. Pearl [2009] has identified three level hierarchy of causal knowledge in contemporary work: first *association*, then *intervention* (analogous to Cartwright's *strategy*) and finally *counterfactuals*. Pearl argues that the types of things that can be known at higher levels subsumes what can be known at lower levels (e.g. all associational knowledge is a type of interventional knowledge), but lower levels do not subsume higher levels.

An apparently paradoxical feature of this three level hierarchy is that, though knowledge is claimed to flow only in one direction, we find that the first and third levels are both described by ordinary joint probability distributions. Counterfactual queries can be formulated as missing data problems, which are distinct from associational problems only due to the interpretations we assign to so-called *counterfactual random variables* or *potential outcomes*. Knowledge at the second level, on the other hand, is described by causal graphical models which are *not* joint probability distributions (in one treatment, they are introduced as indexed sets of joint probability distributions Pearl [2009]). Here is an apparently paradoxical feature of common approaches to causal inference: associational knowledge is distinguished from consequential knowledge in both interpretation and representation, while counterfactual knowledge – considered to subsume both – is distinguished from associational knowledge by interpretation only.

Statistical decision theory, introduced by Wald [1950], underpins much of modern machine learning. It introduced the fundamental notions of *loss* and *risk* to statistics and provided foundational theorems such as the *complete class theorem* which shows that all admissible decision rules are Bayesian decision rules for some prior. Key elements of statistical learning theory inherits heavily from statistical decision theory. While some descendants of statistical decision theory have grappled with the problem of causality [Lewis, 1981], SDT itself is regarded as a theory of statistical decision making and not of causality.

We show a surprising relationship between SDT and causal graphical models. We proceed in two steps: We note that a causal graphical model represents a relationship between probability measures on a given space and the consequences of a given set of actions. We then consider a modification of a standard statistical decision problem: suppose that, rather than being given a loss function that directly evaluates decisions, we are instead provided with a preference function over consequences of decisions that (following convention) we call a *utility*. The resulting problem is underspecified and provides no ordering over decisions. However, the type of relationship represented by a causal graphical model is then found to be precisely the type of object needed to fully specify the problem, and does so in a way that induces a regular statistical decision problem.

This motivates the definition of *causal statistical decision problems* (CSDPs). These relate to regular statistical decision problems (SDPs) in loose analogy with the way that model based reinforcement learning relates to model free reinforcement learning; while the former keeps track of both consequences and rewards/utilities of decisions, the latter “forgets about the consequences” and only works with reward/utility.

Is this true?
There are substantial similarities between SDT and SLT, but I haven't found direct evidence of lineage in e.g. a citation from Valiant

CSDPs introduce the notion of *causal theories*. Causal theories represent relationships between probability measures and consequences and are a generalisation of causal graphical models. In Pearl’s language, they represent the connection between associational knowledge and interventional knowledge; in Cartwright’s, they connect associational knowledge with the consequences of strategies.

Thanks to the clarity of our approach, we are able to shed light on the questions raised in the second paragraph: we require a causal theory to bring knowledge from levels 1 to 2 of Pearl’s hierarchy and we *also* require a causal theory to bring knowledge from level 3 to level 2. A joint distribution over counterfactuals can only answer interventional questions *given interventional assumptions* (we speculate that such assumptions may have been taken for granted). Associational knowledge is represented with probability distributions, knowledge of consequences with stochastic maps and relationships between the two with causal theories.

Choosing appropriate causal theories is a hard problem. Whether we build a causal theory with graphical models or Potential Outcomes (with additional assumptions), it is often the case that a nontrivial result rests on assumptions that are not obvious, generic or testable. Generic principles such as the bias-variance tradeoff have proved to be immensely powerful in the world of statistics, and we regard the question of whether there are generic principles that govern causal inference and what they may be to be one of the most important questions in the field.

We are primarily concerned with setting out a clear framework for reasoning about causal theories, and do not propose principles for constructing a causal theory in this paper. We are able to show a general negative result - causal theories that are symmetric over permutations of decisions cannot yield nontrivial decision rule orderings. We term this result “no causes in, no causes out” as it demonstrates that some causal knowledge is required at the outset if we hope for any nontrivial decision rules. Such asymmetric causal assumptions must be problem specific, so from the outset we cannot build causal theories on “problem neutral” assumptions alone.

There’s another half baked angle here, which is “what kinds of causal theories are represented by graphical models”? In particular, via the question of dominance we can consider causal theories to be related by three different types of randomisation. Also, if we examine marginal causal models, we note that they all represent causal theories that are related to a “nice” causal theory (in the sense that identification is straightforward) via two of these types of randomisation. It’s half baked because I can’t yet say a lot from there, save for the fact that the operation of randomisation seems more amenable to being generalised to a continuous version than DAGs do.

I could also include the “free” results from statistical decision theory somewhere - complete class theorem, purification

2 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 $l : \Theta \times D \rightarrow \mathbb{R}$. 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 $H : \theta \mapsto \mu_\theta$ a *statistical experiment*. Given a *decision strategy* $\pi : E \rightarrow \Delta(\mathcal{D})$, define the *risk* of π given state θ to be the expected loss of π in state θ . Specifically, $R : \Pi \times \Theta \rightarrow \mathbb{R}$ given by $R : (\pi, \theta) \mapsto \delta_\theta \curlyvee (H\pi \otimes \text{Id}_\Theta)l$, where we make use of the product notation and copy map for brevity.

Supposing some unknown true state θ^* , we would ideally find a strategy π that minimises the risk in θ^* . Unfortunately, most statistical decision problems do not admit such strategies. Two alternative decision rules are available:

Given a measure $\xi \in \Delta(\Theta)$ called a prior, ξ -*Bayes decision rule* is a decision rule π_{Ba}^* such that the *Bayes risk* $R_\xi : \pi \mapsto \xi \curlyvee (H\pi \otimes \text{Id}_\Theta)l$ is minimised:

$$\pi_{\text{Ba}}^* \in \arg \min_{\pi \in \Pi} R_\xi(\pi) \quad (1)$$

A *minimax* decision rule π_{MM}^* minimises the worst-case risk. Unlike a Bayes rule, it does not invoke a prior:

Need a canonical measure on Θ ; the coarsest measure rendering the evaluation maps measurable?

$$\pi_{\text{Mm}}^* \in \arg \min_{\pi \in \Pi} \max_{\theta \in \Theta} R(\theta, \pi) \quad (2)$$

We emphasise here that we regard the set Θ as a “state of nature” or a “theory of nature” and not a “parameter set” - it is possible that for some $\theta \neq \theta'$ we have $\mu_\theta = \mu_{\theta'}$, a possibility not supported by the interpretation of Θ as a set of distribution parameters. 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.

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 $\langle \Theta, E, D, H, l \rangle$ where Θ, E and D are measurable sets, H is a stochastic map $\Theta \rightarrow \Delta(\mathcal{E})$ and l a measurable function $E \rightarrow \mathbb{R}$. We leave implicit the set Π of decision strategies $E \rightarrow \Delta(\mathcal{D})$ and \mathbb{R} , the codomain of l .

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. In many areas 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 $u : F \rightarrow \mathbb{R}$; “utility” being a very conventional term for such a desirability function. Given such a u , the tuple $\langle \Theta, E, D, H, u \rangle$ is an ill-posed problem; we want to evaluate the desirability of decisions D (or decision strategies π), but we have no means of connecting decisions with consequences F . An obvious move is to introduce for each state of nature θ a *consequence map* $\kappa_\theta : D \rightarrow \Delta(\mathcal{F})$; let C be the map $\theta \mapsto \kappa_\theta$. We can then define the *causal risk* $S : \Pi \times \Theta \rightarrow \mathbb{R}$ by $S : (\pi, \theta) \mapsto -\delta_\theta \vee (H\pi \otimes ID\Theta)Cu$, and Bayes and minimax risks are defined by obvious analogy.

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

$$T_\theta := \begin{array}{c} \triangle \delta_\theta \\ \text{---} \quad \text{---} \end{array} \begin{array}{c} \boxed{H} \\ \boxed{C} \end{array} \begin{array}{c} \text{---} E \\ \text{---} F \end{array} \quad (3)$$

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

Given a CSDP $\alpha = \langle \Theta, E, F, D, T, u \rangle$ where T is a theory arising from some H and C as in Equation 3, we can recover $H = T(\text{Id}_E \otimes *_F)$ and $C = T(*_E \otimes \text{Id}_F)$. Given α and letting $l := Cu$ we induce the canonical SDP $\beta = \langle \Theta, E, D, H', l \rangle$ such that for any $\theta \in \Theta$, $\pi \in \Pi$, $R^{(\beta)}(\pi, \theta) = S^{(\alpha)}(\pi, \theta)$, and thus, if we accept that the risk functional is the only means of evaluating the desirability of a strategy (whether we choose Bayes, minimax or some other meta-rule to select a strategy), α and β will always produce identical recommendations.

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

Thus, in some sense every problem that can be represented as an SDP can be represented as a CSDP and vice-versa (this may not hold true if we invoke some decision rule that doesn't just depend on the risk functional).

We will finally note an alternative representation of a CSDP. Consider the set $\mathcal{T} = \{T_\theta | \theta \in \Theta\}$ and the evaluation map $\text{Ev}_{\mathcal{T}} : \mathcal{T} \times D \rightarrow \Delta(\mathcal{E} \otimes \mathcal{F})$ given by $(T_\theta, d) \mapsto T_\theta(d; \cdot)$. Then the problems $\alpha = \langle \Theta, E, F, D, T, u \rangle$ and $\alpha' = \langle \mathcal{T}, E, F, D, \text{Ev}_{\mathcal{T}}, u \rangle$ are related in the sense that $S^{(\alpha)}(\pi, \theta) = S^{(\alpha')}(\pi, T_\theta)$. Thus, unlike with regular SDPs where Θ is an arbitrary set, for CSDPs we can regard Θ as a subset of kernels $D \rightarrow \Delta(\mathcal{E} \otimes \mathcal{F})$ that can be written as in Eq. 3 with the

This is highly nonobvious, depends on the work of Jacobs, and is only known to be true for finite sets E and F . On the other hand, there's a very intuitive graphical proof.

this might be equivalent to the set of 1-combs, see Jacobs

canonical kernel $\text{Ev}_{\mathcal{T}}$. We call \mathcal{T} the *set causal theory* and, as there is a bijection between kernel theories T along with their domains Θ and set theories \mathcal{T} , we typically refer to either as simply a *causal theory*. We say that α is a CSDP in kernel form and α' is a CSDP in set form.

Finally, we note that causal theories can *also* be represented as a set of (distribution, consequence map) pairs.

Theorem 2.1 (Causal theories are sets of pairs). *There is a bijection between the set of causal theories and the power set $\mathcal{P}(\Delta(\mathcal{E}) \times \Delta(\mathcal{F})^D)$*

Proof. Sketch: Theories to pairs goes $\{(T_\theta(\text{Id}_E \otimes *_F), T_\theta(*_E \otimes \text{Id}_F)) | T_\theta \in \mathcal{T}\}$ and pairs to theories goes $\{\mu \otimes \kappa | (\mu, \kappa) \in \text{Pairs}\}$. \square

A *causal theory* - that is a “consequence-aware” analogue of a statistical experiment. It can be represented as a Markov kernel or a set of (observation, consequence) pairs. The kernel representation foregrounds the relationship between causal theories and statistical experiments, and we will exploit the latter to justify the following claim: *Causal Bayesian Networks are a subset of causal theories*.

In fact, this holds for the following generalisations of causal graphical models:

- Arbitrary sets of CBNs (e.g. ADMGs)
- Marginal models (e.g. mDAGs)
- CBNs with different intervention rules
- Cyclic SEMs (???)

don't know about this one

Show this for all the examples claimed

It is not the case, however, that *every* type of “causal graphical model” is a representation of a causal theory - SWIGs are an important exception. This is because SWIGs, like all counterfactual models, represent hypothesis classes (a hypothesis class is the range of a statistical experiment).

3 Causal Bayesian Networks

Suppose we have a set of “interventions” R which factorises as $R = \otimes_{i \in [n]} \{\#\} \cup X^i$ 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 $\{X^i | i \in \mathbb{N}\}$ such that $X^i : E \rightarrow X^i$. We denote an element $(x^0, \#, \dots, \#, x^n) \in R$, $x^0, x^n \neq \#$ by the notation $do(X^0 = x^0, X^n = x^n)$ where occurrences of the distinguished element $*$ are ommitted. Denote by $\#$ the element of R consisting entirely of $\#$ (equivalently, $do()$).

For $n \in \mathbb{N}$, 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 $\{X^i\}_{i \in [n]}$ of random variables. That is, we have bijective functions $f : V \rightarrow [n]$ and $g : \{X^i\}_{i \in [n]} \rightarrow [n]$ and we adopt the convention that $f(i) := V^i$ and $g(i) := X^i$. In addition, we will sometimes let a set $U \subset V$ or $a \subset [n]$ to denote a set of random variables rather than vertices or natural numbers; this is licenced by the bijections f and g . We will therefore overload notation and simply refer the nodes of \mathcal{G} as the random variables X^i .

We also suppose we have surjective $h : R \rightarrow \mathcal{P}([n])$ such that $h : (x^0, \dots, x^n) \mapsto \{i | x^i \neq *\}$. That is, h picks out the indices that aren't suppressed in the $do(\dots)$ notation for elements of V . Define $X^{i'} : R \rightarrow \{\#\} \cup X^i$ by the function returning the i -th element of r for $r \in R$. Again, we suppose we have a bijection between primed random variables and natural numbers and can therefore pick out corresponding sets of primed RVs and unprimed RVs or natural numbers.

Definition 3.1 (Causal Bayesian Network). Given R , E and $P_* : R \rightarrow \Delta(\mathcal{E})$ and $\{X^i\}_{i \in [n]}$, a Causal Bayesian Network (CBN) compatible with P_* is a directed acyclic graph (DAG) \mathcal{G} of degree n such that for all $r \in R$

1. P_r is compatible with \mathcal{G} (see Pearl [2009])

2. For all $i \in h(r)$, $P_r F_{X^i} = \delta_{X^{i'}(r)} F_{X^i}$
3. For all $i \notin h(r)$, $P_r|_{\text{Pa}_G(X^i)} F_{X^i} = P_{\#|_{\text{Pa}_G(X^i)}} F_{X^i}$, $P_{\#}$ -almost surely

This definition differs slightly from that given in Pearl [2009]; for example P_* is a map to $\Delta(\mathcal{E})$ rather than a set of labeled members of $\Delta(\mathcal{E})$, and we formulate it in directly in terms of measure theoretic probability rather than elementary probability. Nonetheless, we claim these choices don't meaningfully alter the standard definition, at least if we restrict E to be finite, and they make for a more convenient connection with CSDPs.

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

At least in the case of discrete E and $P_*(\#)$ positive definite, we have from this definition for any $r \in V$ the *truncated factorisation* property:

$$P_r F_X(A) = \prod_{i \in h(r)} \delta_{X^{i'}(r)}(X^i(A)) \sum_{a \in A} \prod_{i \notin h(r)} P_{\#|_{\text{Pa}_G(X^i)}} F_{X^i}(a; \{X^i(a)\}) \quad (4)$$

As a consequence of the existence of conditional probability, given \mathcal{G} and μ there exists a unique set of interventional maps P_* compatible with both \mathcal{G} and μ as above. This property licenses a typical use case of CBNs: $\mathcal{G}(\cdot)$ is treated as a *map* from the subset of $\Delta(\mathcal{E})$ compatible with \mathcal{G} to interventional maps $V \rightarrow \Delta(\mathcal{E})$. More generally, provided μ is compatible with \mathcal{G} we have that 4 exists, and so $\mathcal{G}(\mu)$ is non-empty.

Condition 3 presents some difficulties in the presence of measure 0 sets, as when a conditional probability such as $P_{\#|_{\text{Pa}_G(V^i)}}$ may be variously intended to mean a particular element of the class of conditional probabilities, any element or every element in the class class [Çınlar, 2011]; condition 3 will have different implications for these various interpretations.

Letting $\mathcal{H}^G \subset \Delta(\mathcal{E})$ be some *hypothesis class* of probability measures compatible with a causal graph \mathcal{G} , define the set of pairs $\mathcal{T}^G := \{(\mu, \kappa) | \mu \in \mathcal{H}, \kappa \in \mathcal{G}(\mu)\}$. Recall that a causal theory can be represented as a set of (observation, consequence map) pairs; i.e. \mathcal{T}^G is a causal theory. The map $\text{Th} : \mathcal{G} \mapsto \mathcal{T}^G$ is therefore a map from directed acyclic graphs to causal theories. Unlike the map from DAGs to sets of probability measures, the map from DAGs to causal theories is injective.

Theorem 3.2 (The map Th is injective). *For DAGs $\mathcal{G}, \mathcal{G}'$ on the same set of RV's $\{X^i\}_{[n]}$, $\mathcal{G} \neq \mathcal{G}' \implies \mathcal{T}^G \neq \mathcal{T}^{G'}$.*

Proof. Sketch: \mathcal{G} and \mathcal{G}' must disagree on at least one parental set. Choose some μ such that $P_{\#|_{\text{Pa}_G(X^i)}} F_{X^i} \neq P_{\#|_{\text{Pa}_{G'}(X^i)}} F_{X^i}$. Then take r, r' such that $h(r) = h(r') = \text{Pa}_G(X^i) \cup \text{Pa}_{G'}(X^i)$, $\text{Pa}_G(X^{i'}) (r) = \text{Pa}_G(X^{i'}) (r')$ but $r \neq r'$. Then $P_r F_{X^i} \neq P_{r'} F_{X^i}$ so $P_r \neq P_{r'}$. \square

Each DAG \mathcal{G} represents a causal theory \mathcal{T}^G . For every causal theory \mathcal{T} , either it is not represented by any graph or there is a unique graph \mathcal{G} such that $\mathcal{T} = \mathcal{T}^G$. It is in this sense that we claim Causal Bayesian Networks are a subset of causal theories.

4 Counterfactual models

A counterfactual model poses a number of ordinary random variables X, Y and a number of counterfactual random variables Y_x . It is not clear what the set of counterfactual indices should be in general; we will posit that it can be taken to be the set of decisions D . It is also not clear whether every random variable should have counterfactual versions; we will consider that this is so (so we have X_x , though this may be trivial).

Additionally, a counterfactual model poses certain restrictions on the values and joint distribution of random variables. Consistency is sometimes stronger than a restriction on the joint distribution:

$$\forall e \in E, X(e) = x \implies Y(e) = Y_x(e) \quad (5)$$

I haven't found a formulation of CBNs on infinite spaces, let alone continuous ones

General definition: $f(A)$ is the image of A under f and X as the copy-mapped tensor product of variables in set X

Surely Pearl or a student has dealt with this somewhere? Any element seems to be the most appropriate choice, but this renders CBNs useless for continuous spaces unless we place extra restrictions on P_*

4.1 Can we consider causal theories to be counterfactual models?

There is an obvious way to do this, but it fails to capture the assumption of consistency.

More generally we can employ the standard method of turning a SDP into a CSDP.

4.2 Can we consider counterfactual models to be causal theories?

Given a CSDP we can construct an SDP. Not a counterfactual SDP...but we can always turn an SDP into a censored version of an SDP with additional structure.

The backwards construction of interest: ill-posed counterfactual SDP to CSDP. “Can a counterfactual SE do the job of a CT”?

Yes, there is an obvious correspondence between a causal theory and a class of counterfactual models (injection $CT \rightarrow P(CF)?$). But: this correspondence is at odds with the way that people actually use counterfactual (see ATE (why take a difference?) and ETT (Heckman: incentives might mean ETT is appropriate rather than ATE)). Have people been using counterfactuals wrongly, or should we consider a richer set of $CF \rightarrow CT$ rules?

More importantly, **any $CF \rightarrow CT$ rule is itself a causal theory!**. We can’t avoid invoking a causal theory to go from ill-posed CF SDP to CSDP.

Proposition: Isomorphism between rules: ill-posed SDP to CSDP and causal theories.

Conclusion: contra three-level hierarchy, if we care about consequences we need a causal theory.

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