Intro to Causal Inference

Mees de Vries (BrainCreators)

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Overview

1. Motivation: why and what?
2. Causal models: the definitions and working with them.
3. Simpson's paradox: causal inference to the rescue!

Why Causal Reasoning?

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"Correlation is not causation!"

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Causality comes into play almost any time we want to take action based on statistical data. Consider:

- X: the frequency of high-sodium meals in my diet;
- Y: my blood pressure;
- Z: my headache frequency.

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- Y: my blood pressure;
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We might very well have that (X, Y) and (Z, Y) are identically distributed. But we do not treat hypertension with painkillers!

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- X: high-sodium meal count;
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If we want to model how a better diet would lower my blood pressure, we might want to estimate

$$\mathbb{P}(Y = y \mid X = x).$$

However, the quantity

$$\mathbb{P}(Y = y \mid Z = z)$$

gives us no such information. Why is this difference not in our mathematical language?

Do-calculus

Desideratum: an "operator for interventions". For X, Y random variables we want an expression

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For X, Y, Z as before we should have

$$\mathbb{P}(Y = y \mid \mathrm{do}(X = x)) = \mathbb{P}(Y = y \mid X = x),$$

yet

$$\mathbb{P}(Y = y \mid \mathrm{do}(Z = z)) = \mathbb{P}(Y = y).$$

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For our example:

$$X \longrightarrow Y \longrightarrow Z$$

This says: X is a direct cause of Y, and Y is a direct cause of Z. Implicit assumption: there are no relationships between the variables that are not in the model.

Definition

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A *causal model* consists of:

- A DAG $G = (V, E)$,
- independent random variables $(U_v)_{v \in V}$,
- functions $(f_v)_{v \in V}$ of the appropriate type.

The *value* X_v in a causal model (G, U, f) at a node v is defined recursively as

$$X_v = f_v(U_v, X_{\mathrm{par}(v)}).$$

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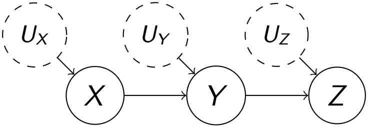
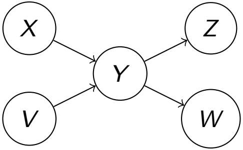
A *causal model* consists of $G = (V, E)$ with $(U_v)_{v \in V}$, $(f_v)_{v \in V}$.
The value in the causal model at v is $X_v = f_v(U_v, X_{\mathrm{par}(v)})$.

Causal model for hypertension:



From now on, we omit the exogenous variables (the U_v) from our diagrams.

Dependence and independence



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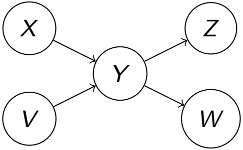

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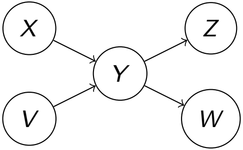

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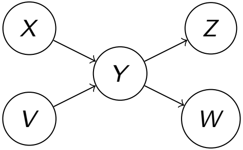

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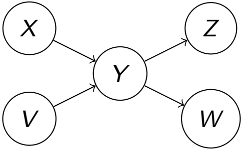

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- $X \not\perp W$
- $Z \not\perp W$
- $Z \perp W \mid Y$
- $X \not\perp V \mid Y$

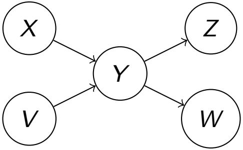
Dependence and independence

- A node v on an *undirected* path in a *directed* graph is a collider if both adjacent edges on the path go into v.
- Given $Z \subseteq V$, a node v on a path is Z-blocking if it either
  - is not a collider, and $v \in Z$, or
  - is a collider, and neither v nor any of its descendents are in Z.

Theorem

$X \perp Y \mid (Z_1, Z_2, \ldots Z_n)$ *if each undirected path* p *from* X *to* Y *has a* Z*-blocking node on it.*

Computations in Graphical Models



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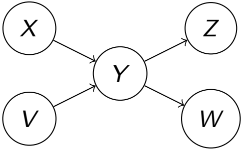

Graphical models allow us to simplify computations.

$$\mathbb{P}(z, w, y, x, v) = \mathbb{P}(z \mid w, y, x, v)\mathbb{P}(w \mid y, x, v)\mathbb{P}(y \mid x, v)\mathbb{P}(x \mid v)\mathbb{P}(x)$$

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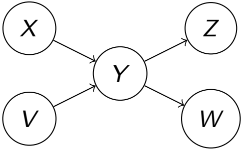

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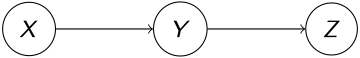
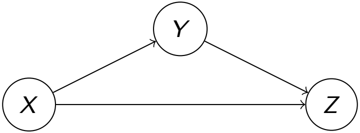
Caution: Intransitive

Note that models are not transitive! The following are different:





Causal Models and Bayesian Networks

So far, I have basically described Bayesian networks. What is the difference?

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In Bayesian networks, the following two are essentially the same model:

$$X \rightarrow Y \rightarrow Z$$

$$X \leftarrow Y \leftarrow Z$$

This is obviously not what we want for causal networks!

Interventions

Recall that causality came into play with interventions. What is an intervention in our model?

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Equations:

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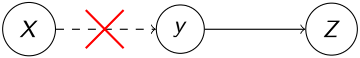

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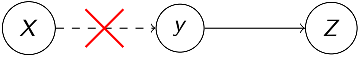

Equations:

$$
\begin{aligned}
X' &= f_X(U_X) \\
Y' &= y \\
Z' &= f_Z(U_Z, Y') = f_Z(U_Z, y)
\end{aligned}
$$

We use the $'$ to distinguish the random variables from the original and modified networks.

That do Operator

Definition

Let (G, U, f) be a causal model with nodes X, Y. Then

$$\mathbb{P}(Y = y \mid \text{do}(X = x))$$

is the probability that $Y' = y$ in the modified network with X set to x.

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Example: hypertension case.

Simpson's Paradox

There is a particular medication A on the market for disease X. Statisticians want to test the effect of the medication. They randomly sample people afflicted by X, and find:

Treatment	Recovery (male)	Recovery (female)	Recovery (total)
Medication A	$9/10 = 90\%$	$20/35 = 57\%$	$29/45 = 64\%$
None	$35/40 = 88\%$	$8/15 = 53\%$	$43/55 = 78\%$
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- but the medication has a good effect on both men and women!

Simpson's Paradox (alternative)

The statisticians decide to investigate further. They find 100 women who have the early stages of the disease for a double blind study. They additionally measure increase or decrease of a relevant protein P before and after treatment.

Treatment	Recovery (inc)	Recovery (dec)	Recovery (total)
Medication A	$1/10 = 10\%$	$28/40 = 70\%$	$29/50 = 58\%$
Placebo	$9/25 = 36\%$	$18/25 = 72\%$	$27/50 = 54\%$
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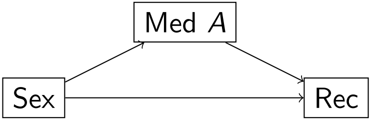
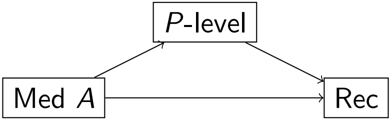
- The medication has a good effect on the whole population of women...
- but the medication has a bad effect on both people whose P-levels increase and decrease!

Causal Model for SP/SPa

The causal models are different for the two cases. Clearly, medication usage or recovery outcome cannot *cause* a difference in sex, so:

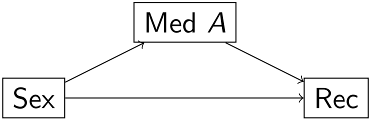


However, in our randomized trial, P-level cannot *cause* a difference in medication usage, so:



Interventions for SP

What if we make the intervention "take medication" in the original paradox?



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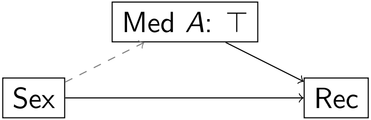

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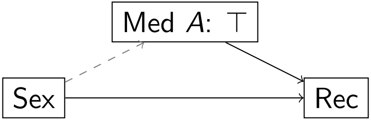

So we get:

$$
\begin{aligned}
\mathbb{P}(\text{Rec} = \top \mid \text{do}(A = \top)) &= \mathbb{P}(\text{Rec}' = \top) \\
&= \sum_{s \in \{M,F\}} \mathbb{P}(\text{Rec}' = \top \mid \text{Sex}' = s)\mathbb{P}(\text{Sex}' = s) \\
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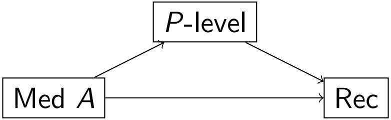

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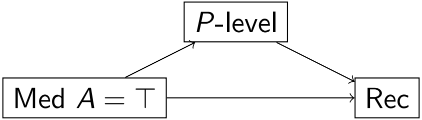

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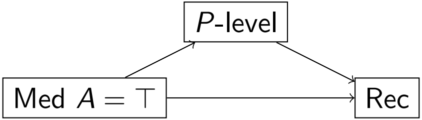

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So do not separately take the P-level cases into account!

Take-aways

- Basic causal modeling might not directly lead to new insight, but it helps us formalize our intuitions.
- Several counterintuitive problems become nicer in the context of causal modeling.
  - Simpson's paradox
  - Berkson's paradox
  - Monty Hall problem
- Given a causal model, you can often answer questions about interventions without making interventions.
- Major drawback: we require a DAG. Many real-world complex systems have feedback, and are thus not acyclic.