Cognitive Modeling

Lecture 19: Causal Learning

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Reading: Tenenbaum and Griffiths (2001).

Note: Griffiths and Tenenbaum (2005) provides a much longer but easier to understand presentation, also with some additional material.

Causal Graphical Models

In the last lecture, we introduced causal graphical models:

- they are an extension of graphical models that can deal with interventions as well as observations;
- we saw that respecting the direction of causality results in efficient representation and inference;

Today, we'll look at modeling human learning of causal relationships using causal graphical models.

Rating Causality

Experiment: subjects are shown *contingency data* and must rate $P(C \rightarrow E)$, the probability that an event C causes outcome E.

Example: case studies with data from experiments in which rats are injected with a certain chemical and tested for expression of a certain gene.

- Case 1: 40 out of 100 injected rats express the gene, 0 out 100 uninjected rats express the gene (40/100, 0/100);
- Case 2: 7 out of 100 injected rats express the gene, 0 out 100 uninjected rats express the gene (7/100, 0/100);
- Case 3: 53 out of 100 injected rats express the gene, 46 out 100 uninjected rats express the gene (53/100, 46/100).

How do you rate $P(C \rightarrow E)$ in each case?

Rating Causality

Experimental results (ratings on a 0–20 scale):

	Case 1	Case 2	Case 3
Rating	14.9 ± 0.8	8.6 ± 0.9	4.9 ± 0.7
$P(e^+\|c^+)$	0.40	0.07	0.53

So clearly, subjects are not just using conditional probability: $P(C \to E) \neq P(e^+|c^+)$.

Two competing rational models have been proposed in the literature to explain these experimental results:

- ΔP model
- causal power model

ΔP

The ΔP model assumes people estimate $P(C \to E)$ as:

$$\Delta P = P(e^+|c^+) - P(e^+|c^-)$$

- $P(e^+|c^+)$ and $P(e^+|c^-)$ are computed as relative frequencies.
- Causality is indicated by a large difference in the probability of the effect when the cause is absent or present.
- Can be shown to be equivalent to evaluating the associative strength between cause and effect.

Causal Power

The causal power model assumes people estimate $P(C \to E)$ as:

$$power = \frac{\Delta P}{1 - P(e^+|c^-)}$$

- Based on axiomatic characterization of causality (Cheng 1997).
- Normalizes ΔP by cases where C could be observed to influence E.
  - $(36/60,\ 30/60)$: $\Delta P = 0.1$, power $= 0.2$.
  - $(60/60,\ 54/60)$: $\Delta P = 0.1$, power $= 1$.

ΔP vs. Causal Power

Both ΔP and causal power predict some trends in experimental data (more on this later), but don't fully account for the data.

	Case 1	Case 2	Case 3
Rating	14.9 ± 0.8	8.6 ± 0.9	4.9 ± 0.7
$P(e^+\|c^+)$	0.40	0.07	0.53
$P(e^+\|c^-)$	0	0	0.46
ΔP	0.40	0.07	0.07
power	0.40	0.07	0.13

Problematic Effects

1. Effect of $P(e^+|c^-)$ when $\Delta P = 0$:

- *Example:* (8/8, 8/8), (4/8, 4/8), (0/8, 0/8).
- Both ΔP and power predict $P(C \to E) = 0$ for all cases.
- But: subjects judge $P(C \to E)$ to decrease across these cases.
- Intuitive explanation: when $P(e^+|c^-)$ is lower, more opportuniy to observe C exert an effect, but still no effect.

Problematic Effects

2. Sample size effect:

- *Example:* (2/4, 0/4), (10/20, 0/20), (25/50, 0/50).
- Both ΔP and power predict $P(C \to E) = .5$ for all cases.
- But: subjects judge $P(C \to E)$ to increase across cases.
- Intuitive explanation: in small samples, effects could be just random noise.

Problematic Effects

3. Non-monotonic effects of changing $P(e^+|c^-)$:

- *Example:* (30/30, 18/30), (24/30, 12/30), (12/30, 0/30).
- ΔP predicts constant $P(C \to E)$, power predicts a decrease.
- But: subjects judge $P(C \to E)$ slightly lower for middle case.
- Previous researchers assumed this effect was just odd and ignored it.

Rethinking Causal Learning

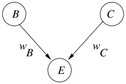
Using Bayes nets, Tenenbaum and Griffiths (2001) provide an explanation for the failures of ΔP and causal power and suggest an alternative model.

- Both ΔP and causal power can be viewed as *estimating parameters* of a particular causal graphical model.
- Tenenbaum and Griffiths (2001) suggest that subjects are actually performing *structure learning*: choosing between two different causal graphical models.

That is, previous models assumed people are judging the *strength* of causation, new model assumes they are judging the *existence* of causation.

Analyzing ΔP and Causal Power

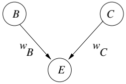
Given the following Bayes net:



C:	cause
E:	effect
B:	background (alternative cause/causes), with B=1 always.
w_B, w_C:	parameters (effect strengths) $P(E\|B)$, $P(E\|C)$.

We can analyze the ΔP and Causal Power models as two different *parameterizations* (i.e., ways of defining $P(E|B,C)$).

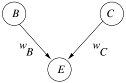
Parameterization



Linear parameterization: the effect strengths of B and C are additive.

$$\begin{aligned} P(e^+|c^-,b^+) &= w_B \\ P(e^+|c^+,b^+) &= w_B + w_C \end{aligned}$$

Parameterization



Noisy-OR parameterization: C and B act as independent causes.

$$\begin{aligned} P(e^+|c^-,b^+) &= w_B \\ P(e^+|c^+,b^+) &= w_B + w_C - w_B w_C \end{aligned}$$

Reduces to standard OR if $w_B = w_C = 1$.

Structure Learning

Tenenbaum and Griffiths (2001) show that:

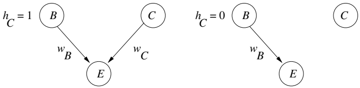
- ΔP corresponds Bayes net with linear parameterization;
- causal power corresponds to Bayes net with noisy-OR parameterization

where parameters w_B and w_C are estimated using maximum likelihood estimation.

Key insight: causal inference is a judgment of whether a causal link exists, not how strong the effect is. So, subjects are really doing *structure learning* for Bayes nets.

Structure Learning

Hypothesis: subjects are deciding between the following two Bayes nets:



Does cause C have an influence on effect E?

Tenenbaum and Griffiths (2001) use *Bayesian inference* over model structures to make this decision.

Causal Support

Tenenbaum and Griffiths's (2001) *Causal Support* model assumes:

- subjects' judgments correspond to inferences about the underlying causal structure, i.e. the probability that C is a direct cause of E;
- formally: decide between $h_C = 1$ (graph in which C is a parent of E) and $h_C = 0$ (graph in which C is not a parent of E);
- this amounts to estimating the *log posterior odds* of h_C:

$$support = \log \frac{P(h_C = 1|X)}{P(h_C = 0|X)}$$

Computing Causal Support

$$support = \log \frac{P(h_C = 1|X)}{P(h_C = 0|X)}$$

Assuming the prior probability of each graph is 0.5,

$$support = \log \frac{P(X|h_C = 1)}{P(X|h_C = 0)}$$

Compute $P(X|h_C = 1)$ by summing over possible parameter values (Bayesian inference):

$$P(X|h_C = 1) = \int_0^1 \int_0^1 P(X|w_B, w_C, h_C = 1)p(w_B, w_C|h_C = 1)dw_B\ dw_C$$

Similarly for $P(X|h_C = 0)$.

Computing Causal Support

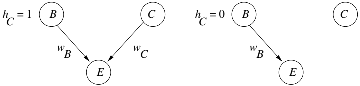
$$P(X|h_C = 1) = \int_0^1 \int_0^1 P(X|w_B, w_C, h_C = 1)p(w_B, w_C|h_C = 1)dw_B\ dw_C$$

- Assume $P(w_B, w_C|h_C = 1)$ is uniform (no particular prior knowledge about parameter values).
- Assume $P(X|w_B, w_C, h_C = 1)$ follows noisy-OR parameterization.
- Actual computation requires a computer program.

Can also compute other values from this model, e.g. $p(w_c|X)$.

- Causal Support is high when $p(w_c|X)$ has most of its mass on *non-zero values*.

Comparison of the Models



Comparison of the three models:

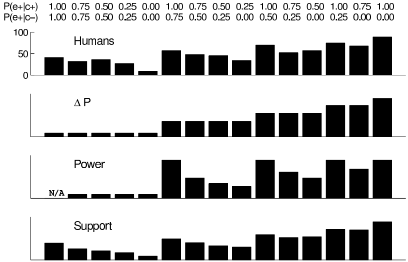
Model	Form of $P(E\|B, C)$	$P(C \to E)$
ΔP	Linear	w_C
Power	Noisy-OR	w_C
Support	Noisy-OR	$\log \frac{P(h_C=1)}{P(h_C=0)}$

Comparison with Experimental Data

Comparison of model performance with Buehner and Cheng's (1997) experimental data:

- subjects judged $P(C \to E)$ for hypothetical medical studies (similar to gene expression example);
- each subjects saw eight cases in which C occurred and eight cases in which C didn't occur;
- compare predictions of all three models to human judgments.

Comparison with Experimental Data

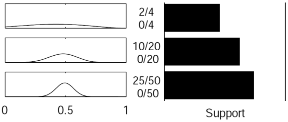


Comparison with Experimental Data

- $P(C \to E)$ increases as $P(e^+|c^-)$ decreases when $P(e^+|c^+) = 1$: captured by ΔP and Support, not Power (cols 1, 6, 11, 14, 16).
- $P(C \to E)$ decreases as $P(e^+|c^-)$ decreases (sometimes): captured by Power and Support, not ΔP (cols 6-10, 14-15).
- $P(C \to E)$ decreases as $P(e^+|c^-)$ decreases when $\Delta P = 0$: captured only by Causal Support (cols 1-5).
- Non-monotonic effect: captured only by Causal Support (cols 11-13).

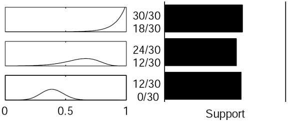
Overall, Causal Support has highest correlation with human data for this and other experimental data.

Sample Size Effect



- Left: $p(w_C|X)$. Right: Causal Support.
- More data $\Rightarrow$ more certainty in non-zero value of w_C.

Non-monotonic Effect



- Top: E occurs with C in all cases where it can $\Rightarrow$ high certainty in high value of w_C.
- Bottom: E never occurs without C $\Rightarrow$ lower value of w_C, but high certainty in non-zero value.
- Middle: Neither extreme $\Rightarrow$ most probable value of w_C is high, but lower certainty in non-zero value.

Discussion: results

- Causal Support correlates better with human data than previous models in a range of experiments.
- Captures several trends other models do not:
  - effects when $\Delta P = 0$;
  - non-monotonic effects;
  - sample size effects.
- Predictions stem from the assumption that humans are learning causal structure rather than estimating its strength.
- Also able to draw inferences based on very few observations (this was tested in subsequent experiments).

Discussion: methods

Causal Support model uses Bayesian inference to compare probabilities of different Bayes net structures.

- Previous models ask: what is the best (maximum-likelihood) estimate of w_C?
  Estimates further from zero $\Rightarrow$ greater $P(C \rightarrow E)$
- Causal Support asks: what is the most probable causal structure?
  More mass of w_C away from zero $\Rightarrow$ greater $P(C \rightarrow E)$

Summary

- Two standard models of causal inference exist:
  - ΔP: prob. of positive cause minus prob. of negative cause;
  - causal power: ΔP normalized by one minus probability of negative cause;
- these models can be analyzed as Bayes nets with linear parameterization and noisy-OR parameterization;
- but: more plausible to assume that the structure of the Bayes net is also learned;
- the causal support model achieves this by using Bayesian inference over the structure of the net;
- it accounts for patterns in the experimental data that other models fail to capture.

References

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