Introduction to Graphical Causal Modelling

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SIKS course on Causal Modelling – 30 May, 2023
Outline

1. Introduction to causality
2. Prediction vs. causation
3. Causal graphs and how to read them
4. Cause-effect estimation
5. The missing link & conclusion
Many important research questions are rooted in causality.

- Benefits of exercise and healthy nutrition
- Climate change
- Racial and gender bias in AI
- Covid vaccine efficacy
Many important research questions are rooted in causality.

- Answers typically involve ‘what causes it?’ and ‘how?’

“does human activity cause climate change?”
Many important research questions are rooted in causality

- ‘Finding a connection’ does not imply we know what causes what ...

“how are violent video games, ADHD, and aggression related?”
Many important research questions are rooted in causality

- Sometimes the difference between ‘connection’ and ‘causality’ is blurred ...

“can we infer functional brain connectivity from fMRI data?”
Many important research questions are rooted in causality

- Sometimes it is not even clear if the concept ‘cause’ makes sense

“can butterflies cause hurricanes?”
How do we recognize causality?
(apparently so simple we don’t teach this at school/university)
“Of course I know cause and effect!”
Causality: what is it?

How do we recognize causality?
(apparently so simple we don’t teach this at school/university)
“Of course I know cause and effect ...”

What exactly do we mean by ‘cause’ and ‘effect’?
Intuitively obvious, yet curiously hard to define. Often involves aspects of
- things that occur together
- things that follow each other in time
- things that are somehow necessary and/or sufficient to lead to another
- things that change the probability of something else happening
- things connected by a mechanistic chain of events, etc. etc.

Most definitions run into trouble somewhere ...
Main ‘cause’ behind a huge amount of philosophical controversy!
The subject of causality has a long history in philosophy. For example this is what Hume had to say about it:

“Thus we remember to have seen that species of object we call flame, and to have felt that species of sensation we call heat. We likewise call to mind their constant conjunction in all past instances. Without any farther ceremony, we call the one cause and the other effect, and infer the existence of the one from that of the other.”

David Hume, *Treatise of Human Nature* (1739)
Some philosophers even proposed to abandon the concept of causality altogether

“All philosophers, of every school, imagine that causation is one of the fundamental axioms or postulates of science, yet, oddly enough, in advanced sciences such as gravitational astronomy, the word ‘cause’ never occurs. The law of causality, I believe, like much that passes muster among philosophers, is a relic of a bygone age, surviving, like the monarchy, only because it is erroneously supposed to do no harm”.

Bertrand Russell, On the Notion of Cause (1913)
Karl Pearson (one of the founders of modern statistics, well-known from his work on the correlation coefficient) writes:

“Beyond such discarded fundamentals as ‘matter' and ‘force' lies still another fetish amidst the inscrutable arcana of even modern science, namely, the category of cause and effect.”

Karl Pearson, *The Grammar of Science* (1892)
Karl Pearson (one of the founders of modern statistics, well-known from his work on the correlation coefficient) writes:

“Beyond such discarded fundamentals as ‘matter' and ‘force' lies still another fetish amidst the inscrutable arcana of even modern science, namely, the category of cause and effect.”

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Since then, many statisticians tried to avoid causal reasoning:

- “Considerations of causality should be treated as they have always been in statistics: preferably not at all.” (Terry Speed, former president of the Biometric Society).
- “It would be very healthy if more researchers abandon thinking of and using terms such as cause and effect.” (Prominent social scientist).
Causality = ‘Effective manipulability’
- focus on relevant, measurable influence
- understand why things happen
- predict how things change if we intervene (effect computation)
- not about truth, but about validity (given assumptions)
- allows principled use of maths, statistics & logic on data and models
- verify by experiment (‘Randomized Controlled Trial’)

key risk factors in predicting car crashes ...
Effective manipulability

“does human activity cause climate change?”

“do butterflies cause hurricanes?”
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Example - Gene regulation

gene A → DNA → transcript T_A → mRNA → protein A → promote/inhibit T_B → gene B → protein B

central dogma of molecular biology
Example - Gene regulation

- Gene A
- Transcript $T_A$
- Protein A
- Gene B
- Transcript $T_B$
- Protein B

Promote / Inhibit

Microarray to measure transcription levels
Observed gene expression levels

transcription $T_B$

transcription $T_A$

$\text{gene A} \rightarrow \text{gene B}$
Predicting gene expression levels

\[ p(T_B | T_A = 0.8) \]
Predicting gene expression levels

\[ p(T_A | T_B = 0.8) \]
Observation vs. intervention: gene knock-out experiments

\[ p(T_B \mid do(T_A = 0.0)) \]
Observation vs. intervention: gene knock-out experiments

\[ p(T_A \mid do(T_B = 0.0)) \]
What if we do not know the model?

Q: Does X cause Y or does Y cause X? ... or “can’t tell”?
Causal direction from model simplicity

Easy to explain as
\[ Y = f(X) + \text{noise} \]

Difficult to explain as
\[ X = f(Y) + \text{noise} \]
Chocolate consumption and Nobel prizes...

Figure 1. Correlation between Countries' Annual Per Capita Chocolate Consumption and the Number of Nobel Laureates per 10 Million Population.

The principal finding of this study is a surprisingly powerful correlation between chocolate intake per capita and the number of Nobel laureates in various countries. Of course, a correlation between X and Y does not prove causation but indicates that either X influences Y, Y influences X, or X and Y are influenced by a common underlying mechanism. However, since chocolate consumption has been documented to improve cognitive function, it seems most likely that in a dose-dependent way, chocolate intake provides the abundant fertile ground needed for the sprouting of Nobel laureates. Obviously, these findings are hypothesis-generating only and will have to be tested in a prospective, randomized trial.

The only possible outlier in Figure 1 seems to be Sweden. Given its per capita chocolate consumption of 6.4 kg per year, we would predict that Sweden should have produced a total of about 14 Nobel laureates, yet we observe 32. Considering that in this instance the observed number exceeds the expected number by a factor of more than 2, one cannot quite escape the notion that either the Nobel Committee in Stockholm has some inherent patriotic bias when assessing the candidates for these awards or, perhaps, that the Swedes are particularly sensitive to chocolate, and even minuscule amounts greatly enhance their cognition. A second hypothesis, reverse causation — that is, that enhanced cognitive performance could stimulate countrywide chocolate consumption — must also be considered. It is conceivable that persons with superior cognitive function (i.e., the cognoscenti) are more aware of the health benefits of the flavanols in dark chocolate and are therefore prone to increasing their consumption. That receiving the Nobel Prize would in itself increase chocolate intake countrywide seems unlikely, although perhaps celebratory events associated with this unique occasion would lead to increased chocolate consumption.

Figure 1. Correlation between Countries’ Annual Per Capita Chocolate Consumption and the Number of Nobel Laureates per 10 Million Population.

Chocolate consumption and Nobel prizes...

Alternative explanation
- unobserved confounder
SMOKING AND CARCINOMA OF THE LUNG
PRELIMINARY REPORT

BY
RICHARD DOLL, M.D., M.R.C.P.
Member of the Statistical Research Unit of the Medical Research Council

Fig. 1.—Percentage of patients smoking different amounts of tobacco daily.
SMOKING AND CARCINOMA OF THE LUNG
PRELIMINARY REPORT

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Fig. 1.—Percentage of patients smoking different amounts of tobacco daily.

Fig. 2.—Death rate from cancer of the lung and rate of consumption of tobacco and cigarettes.
Question: What treatment would you prefer?

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## Treatment of kidney stones

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### Question: What treatment would you prefer now?
## Treatment of kidney stones

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**Question:** What treatment would you prefer now?

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"Simpson’s paradox"
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- **Stone size**: + +
- **Treatment A/B**: + -
- **Recovery**: +
### Treatment of kidney stones

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**Question:** How to compute the actual effect?
Computing causal effect sizes from observations

- split observed correlation in causal effect and confounding

\[ \rho_{xy} \]

observed correlation

combination of (possible) causal effect and (possible) confounding
Computing causal effect sizes from observations

- split observed correlation in causal effect and confounding

\[ \rho_{xy} \]

How to compute the causal effect?
- gold standard: randomized controlled trial!
- otherwise
  - adjustment formula to compensate for confounding (later this session)
  - more general: do-calculus \[ \text{[Pearl, Causality 2009]} \]
  - not always possible!
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1. Introduction to causality
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Key model assumption: causal DAG

- real world consists of networks of causally interacting variables,
- structure corresponds to a *directed acyclic graph* (DAG)
- arcs represent *direct* causes between variables in the system
• real world consists of networks of causally interacting variables, 
• structure corresponds to a *directed acyclic graph* (DAG) 
• arcs represent *direct* causes between variables in the system 
• subset of these variables observed in experiments

Key model assumption: causal DAG

*confounders*

*underlying causal DAG* $G$ 
(*Directed Acyclic Graph*)
Key model assumption: causal DAG

- real world consists of networks of causally interacting variables,
- structure corresponds to a *directed acyclic graph* (DAG)
- arcs represent *direct* causes between variables in the system
- subset of these variables observed in experiments

**underlying causal DAG** $G$
(Directed Acyclic Graph)

**equivalent ADMG** representation
(Ayclic Directed Mixed Graph)
Basic graphical model terminology

- nodes and edges

Diagram:

- vertex/node
- bi-directed edge
- arc
• path - sequence of (distinct) nodes $\pi = \langle X_1, X_2, \ldots, X_k \rangle$ where each successive pair of nodes along the path is adjacent (connected by an edge) in graph $G$
Collider and non-collider triples

- **collider** - triple of successive nodes \( \langle X,Y,Z \rangle \) along a path, where the edges from \( X \) and \( Z \) have an arrowhead ('collide') at \( Y \), e.g. \( X \leftrightarrow Y \leftrightarrow Z \)

- **non-collider** - any such triple that is not a collider, e.g. \( X \rightarrow Y \rightarrow Z \), \( X \leftarrow Y \leftarrow Z \), or \( X \leftarrow Y \rightarrow Z \)
Collider and non-collider triples

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Ancestral relations

- if \( X \rightarrow Y \) is in graph \( G \), then \( X \) is a parent of \( Y \), and \( Y \) is a child of \( X \)
- if \( X \leftrightarrow Y \) is in graph \( G \), then \( X \) is a spouse of \( Y \) (and v.v.)
- if there is a directed path \( X \rightarrow \ldots \rightarrow Y \) in \( G \), then \( X \) is ancestor of \( Y \), and \( Y \) is a descendant of \( X \)
Blocked and unblocked paths

- a path \( \pi = \langle X,..,Y \rangle \) is **unblocked** given set of nodes \( Z \) iff:
  - all non-colliders along \( \pi \) are *not* in \( Z \)
  - all colliders along \( \pi \) are in \( Z \) or are ancestor of some \( Z \in Z \)

otherwise the path is **blocked**

\[ \text{non-colliders} \]

*Path \( \langle A,B,F,G \rangle \) is unblocked given the empty set ...*
a path $\pi = \langle X, \ldots, Y \rangle$ is unblocked given set of nodes $Z$ iff:
- all non-colliders along $\pi$ are not in $Z$
- all colliders along $\pi$ are in $Z$ or are ancestor of some $Z \in Z$
otherwise the path is blocked

... path $\langle A, B, F, G \rangle$ is blocked given $F$ ...
Blocked and unblocked paths

- A path $\pi = \langle X,..,Y \rangle$ is **unblocked** given set of nodes $Z$ iff:
  - all non-colliders along $\pi$ are *not* in $Z$
  - all colliders along $\pi$ are in $Z$ or are ancestor of some $Z \in Z$
- otherwise the path is **blocked**

... but path $\langle A,B,D,G \rangle$ becomes **unblocked** given $F$ ...
Blocked and unblocked paths

- a path $\pi = \langle X,..,Y \rangle$ is unblocked given set of nodes $Z$ iff:
  - all non-colliders along $\pi$ are not in $Z$
  - all colliders along $\pi$ are in $Z$ or are ancestor of some $Z \in Z$
  otherwise the path is blocked

\[ ... \text{and path } \langle A, B, D, G \rangle \text{ is again blocked given } \{D,F\}. \]
in a graph $G$, nodes $X$ and $Y$ are \textit{d-separated} given $Z$, iff there are no unblocked paths in $G$ between $X$ and $Y$ given $Z$, otherwise they are \textit{d-connected}.
in a graph $G$, nodes $X$ and $Y$ are $d$-separated given $Z$, iff there are no unblocked paths in $G$ between $X$ and $Y$ given $Z$, otherwise they are $d$-connected.

$$d$$-separation

Nodes $A$ and $G$ are $d$-separated given $\{D,F\}$, but $d$-connected given $\{\}$, $D$, or $F$. 

Judea Pearl (Winner Turing Award 2012)
Exercise 1a – Paths and colliders

- **collider** - triple of successive nodes \( \langle X,Y,Z \rangle \) along a path, where the edges from \( X \) and \( Z \) have an arrowhead (‘collide’) at \( Y \), e.g. \( X \leftrightarrow Y \leftarrow Z \)

1. Is \( \langle A,C,B,A \rangle \) a path?
2. Is \( \langle A,C,E,D,B \rangle \) a (directed) path?
3. Is \( A \) an ancestor of \( D \)?
4. What are descendants of \( B \)?
5. Which nodes on the path \( \langle B,D,E,C,A \rangle \) are non-colliders?
6. A **v-structure** is a collider between non-adjacent nodes. How many v-structures are in the graph \( G \)?
Exercise 1a – Paths and colliders

- collider - triple of successive nodes \( \langle X,Y,Z \rangle \) along a path, where the edges from \( X \) and \( Z \) have an arrowhead (‘collide’) at \( Y \), e.g. \( X \leftrightarrow Y \leftrightarrow Z \)

1. Is \( \langle A,C,B,A \rangle \) a path? **No: A and B are not adjacent and A occurs twice.**
2. Is \( \langle A,C,E,D,B \rangle \) a (directed) path? **It is a path, but not a directed path.**
3. Is A an ancestor of D? **No: there is no directed path from A to D.**
4. What are descendants of B? **Nodes \{B,C,D\} (B is also its own descendant!)**
5. Which nodes on the path \( \langle B,D,E,C,A \rangle \) are non-colliders? **Nodes C and D.**
6. A **v-structure** is a collider between non-adjacent nodes. How many v-structures are in the graph \( G \)? **Two: A \( \rightarrow \) C \( \leftrightarrow \) B, and C \( \rightarrow \) E \( \leftrightarrow \) D.**
Exercise 1b – Blocked and unblocked paths

• a path \( \pi = \langle X,..,Y \rangle \) is unblocked given set of nodes \( Z \) iff:
  - all non-colliders along \( \pi \) are not in \( Z \)
  - all colliders along \( \pi \) are in \( Z \) or are ancestor of some \( Z \in Z \)
  otherwise the path is blocked

1. Is \( C \leftrightarrow B \rightarrow D \) blocked by (`given’) \( B \)?
2. Is \( A \rightarrow C \leftrightarrow B \) blocked given \( E \)?
3. Is \( A \rightarrow C \rightarrow E \leftarrow D \) blocked? (given empty set \( Z = \{\} \))
4. Is path \( \langle A,C,B,D \rangle \) blocked by \( \{C,E\} \)?
5. Which set(s) of nodes (if any) unblock a path from \( A \) to \( B \)?
6. Claim: ‘A path between two nodes can be blocked, iff they are non-adjacent’. True or false?
Exercise 1b – Blocked and unblocked paths

- a path $\pi = \langle X,..,Y \rangle$ is unblocked given set of nodes $Z$ iff:
  - all non-colliders along $\pi$ are not in $Z$
  - all colliders along $\pi$ are in $Z$ or are ancestor of some $Z \in Z$
  otherwise the path is blocked

1. Is $C \leftrightarrow B \rightarrow D$ blocked by (‘given’) $B$? Yes.
2. Is $A \rightarrow C \leftrightarrow B$ blocked given $E$? No.
3. Is $A \rightarrow C \rightarrow E \leftarrow D$ blocked? (given empty set $Z = \{\}$) Yes.
4. Is path $\langle A,C,B,D \rangle$ blocked by $\{C,E\}$? No.
5. Which set(s) of nodes (if any) unblock a path from $A$ to $B$? Any subset of $\{C,D,E\}$ containing at least one node from $\{C,E\}$.
6. Claim: ‘A path between two nodes can be blocked, iff they are non-adjacent’. True or false? False: reverse counter example $\langle B,D,E,C \rangle$. 
Exercise 1c – $d$-separation

- in a graph $G$, nodes $X$ and $Y$ are $d$-separated given $Z$, iff there are no unblocked paths in $G$ between $X$ and $Y$ given $Z$, otherwise they are $d$-connected

1. Are $A$ and $B$ $d$-separated? (given empty set $\{\}$)
2. Are $C$ and $D$ $d$-separated by $B$?
3. Are $A$ and $E$ $d$-separated by $C$?
4. Are $A$ and $D$ $d$-separated by $\{B, E\}$?
5. Which set(s) of nodes (if any) would $d$-separate $B$ and $E$?
6. True or false: ‘Two nodes can be $d$-separated, iff they are non-adjacent’?
Exercise 1c – $d$-separation

- in a graph $G$, nodes $X$ and $Y$ are $d$-separated given $Z$, iff there are no unblocked paths in $G$ between $X$ and $Y$ given $Z$, otherwise they are $d$-connected

1. Are $A$ and $B$ $d$-separated? (given empty set $\emptyset$) Yes.
2. Are $C$ and $D$ $d$-separated by $B$? Yes.
3. Are $A$ and $E$ $d$-separated by $C$? No: path $\langle A,C,B,D,E \rangle$ is unblocked by $C$.
5. Which set(s) of nodes (if any) would $d$-separate $B$ and $E$? $\{C,D\}, \{A,C,D\}$
6. True or false: ‘Two nodes can be $d$-separated, iff they are non-adjacent’? True for DAGs, but not for ADMGs in general!
graphical models offer an intuitive means to model causal interactions
so far we only considered the causal structure ...
... now we need to link the graphs to data
⇒ enter the Causal Bayesian Network!

![causal DAG G](image)
A **Bayesian Network** (BN) is a pair \((G, p)\), where

- \(G\) is a **directed acyclic graph** over variables \(X = \{X_1, X_2, \ldots, X_K\}\)
- \(p\) is a **joint probability distribution** over \(X\) that factorizes according to \(G\)

\[
p(X) = \prod_{k=1}^{K} p(X_k | pa(X_k))
\]

*causal DAG \(G\)  
*factorized joint probability distribution*
A **Bayesian Network** (BN) is a pair \((G, p)\), where

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\]

**factorized joint probability distribution**
A Bayesian Network \((G, p)\) is causal if

- all and only the directed edges in \(G\) correspond to direct causal relations,
- it satisfies the **Causal Markov condition**: 

> “In a causal DAG \(G\), every node is probabilistically independent of its non-descendants given its parents (direct causes) in \(G\).”

\[
X_i \perp nd(X_i) \mid pa(X_i)
\]
A Bayesian Network \((G, p)\) is causal if

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  “In a causal DAG \(G\), every node is probabilistically independent of its non-descendants given its parents (direct causes) in \(G\).”

---

**Causal DAG** \(G\)

\[
\begin{align*}
A & \indep B | - \\
B & \indep A | - \\
D & \indep A | B, C
\end{align*}
\]

**As a result**
- \(d\)-separation \(\Rightarrow\) probabilistic independence
Structural Causal Model

- each child-parent family in the causal DAG $G$ corresponds to a deterministic function

$$X_i = f_i(pa(X_i), \varepsilon_i)$$

with $\varepsilon_i$ representing all exogenous influences (noise) on $X_i$
- collection is a Structural Causal/Equation Model (SCM/SEM)
Structural Causal Model

- each child-parent family in the causal DAG G corresponds to a deterministic function
  \[ X_i = f_i(pa(X_i), \epsilon_i) \]
  with \( \epsilon_i \) representing all exogenous influences (noise) on \( X_i \)
- collection is a Structural Causal/Equation Model (SCM/SEM)

\[
A = \epsilon_A \\
B = \epsilon_B \\
C = \alpha A + \beta B + \epsilon_C \\
D = \gamma B + \delta D + \epsilon_D \\
\epsilon_i \sim N(0, \sigma_i)
\]

Example: multivariate Gaussian model
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Interventions

- intervening = actively changing the world

not this one ...

but this one
Interventions

• intervening = actively changing the world

Examples
• prescribing a treatment (or placebo) in an RCT
• gene knock-out experiment
• deciding to quit smoking
• governments changing laws / taxation levels,
• lowering home room temperatures
• adding a catalyst to a chemical reaction, etc.
Interventions

- intervening = actively changing the world

Examples
- prescribing a treatment (or placebo) in an RCT
- gene knock-out experiment
- deciding to quit smoking
- governments changing laws / taxation levels,
- lowering home room temperatures
- adding a catalyst to a chemical reaction, etc.

Common types of interventions
- **hard/soft** - (directly forcing a variable to a specific value vs. indirectly stimulating a variable to e.g. higher/lower values)
- **surgical/fat-hand** - (very precisely affecting only the target intervention variable vs. having possible unintended side-effects)
- **perfect interventions** = hard+surgical (Pearl’s do-operator, see next)
- **mechanism interventions** (acting on the functional form of the relations)
Perfect intervention in SCM

- externally force a node to a specific value: do($X_i = x_i$)
- replace structural equation $f_i(\ldots)$ with constant $x_i$
- corresponds to removing all incoming arcs to $X_i$ in causal DAG $G$

**causal DAG $G$ with intervention on $C$**

**intervention do($C = c$)**

\[
egin{align*}
A &= f_A(\epsilon_A) \\
B &= f_B(\epsilon_B) \\
C &= c \\
D &= f_D(B, C, \epsilon_D)
\end{align*}
\]
We can understand / predict the effect of an intervention if we can rewrite the (unknown) interventional distribution in terms of the known observed distribution.

$$p(A,B,C,D) = p(A)p(B)p(C|A,B)p(D|B,C)$$

original observed joint probability distribution

$$p(A,B,C,D|do(C = c)) = ...?$$

interventional distribution under do(C = c)
The difference between the expectation under pre- and post-interventional distribution then corresponds to the *causal effect*. 

Difficult to compute in general: Pearl’s *do-calculus*
• The **difference** between the expectation under pre- and post-interventional distribution then corresponds to the **causal effect**

• Difficult to compute in general: Pearl’s **do-calculus**

• Fortunately, for a large class of problems there exists a relatively straightforward procedure: ‘**adjusting** for the parents’

\[
p\left( Y = y \mid do(X = x) \right) = \sum_{Pa(X)} p\left( y \mid x, Pa(X) \right) p\left( Pa(X) \right)
\]

*adjustment formula for intervention on X*

**causal DAG G with intervention on C**
Theorem: A set of nodes $S$ is admissible for adjustment to find the causal effect of $X$ on $Y$, if:

- $X, Y \notin S$
- no element of $S$ is a descendant of $X$
- $S$ blocks all back-door paths $X \leftarrow \ldots Y$ (all paths between $X$ and $Y$ that start with an incoming arc on $X$)

$B$ is admissible for computing the causal effect of $C$ (or $A$) on $D$
• we can generalize adjustment to ‘admissible’ sets (instead of just parents)

**Theorem:** A set of nodes $S$ is *admissible for adjustment* to find the causal effect of $X$ on $Y$, if:
- $X, Y \notin S$
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General adjustment formula:

$$p(Y = y \mid do(X = x)) = \sum_s p(y \mid x, S = s) p(S = s)$$

$$= \int p(y \mid x, s) p(s) ds$$

$B$ is admissible for computing the causal effect of $C$ (or $A$) on $D$
if we can predict what happens on an intervention we can consider quantifying the causal impact of one variable on another

the **Average Causal Effect** (ACE) quantifies the causal effect of $X$ on $Y$ as the difference in expectation of $Y$ under different interventions on $X$

$$
ACE(X \rightarrow Y) = E[Y \mid do(X = 1)] - E[Y \mid do(X = 0)]
$$

$$
= \sum_{Y} Y \cdot p(Y \mid do(X = 1)) - \sum_{Y} Y \cdot p(Y \mid do(X = 0))
$$

ACE for causal effect of binary variable $X$ on ordinal variable $Y$
Exercise 2a – Admissible sets

A set of nodes $S$ is *admissible for adjustment* for the causal effect of $X$ on $Y$, if:

- $X, Y \notin S$
- no element of $S$ is a *descendant* of $X$
- $S$ blocks all *back-door paths* $X \leftarrow \ldots Y$ (all paths between $X$ and $Y$ that start with an incoming arc on $X$)

1. Is $B$ admissible for adjustment to find the causal effect of $D$ on $E$?
2. Is $\{\}$ admissible for the causal effect of $A$ on $E$?
3. Is $B$ admissible for the causal effect of $A$ on $E$?
4. Is $\{B, D\}$ admissible for the causal effect of $A$ on $E$?
5. Is $C$ admissible for the causal effect of $A$ on $E$?
6. Is $\{B, C\}$ admissible for the causal effect of $E$ on $A$?
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1. Is $B$ admissible for adjustment to find the causal effect of $D$ on $E$? **Yes.**
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3. Is $B$ admissible for the causal effect of $A$ on $E$? **Yes.**
4. Is $\{B,D\}$ admissible for the causal effect of $A$ on $E$? **Yes.**
5. Is $C$ admissible for the causal effect of $A$ on $E$? **No.**
6. Is $\{B,C\}$ admissible for the causal effect of $E$ on $A$? **Yes.**
Exercise 2b – Kidney stones revisited

1. Confirm that *Stone size* is a valid and necessary adjustment variable for the causal effect of *Treatment A/B* on *Recovery*.

2. Match the variables and values in the table above to the adjustment formula. In particular: what values need to be summed over?

3. Compute the causal effect of choosing *Treatment A* on *Recovery*.

4. Idem for the causal effect of *Treatment B*, and compare. What is the expected improvement (ACE) of choosing the optimal treatment?

<table>
<thead>
<tr>
<th></th>
<th>Treatment A</th>
<th>Treatment B</th>
</tr>
</thead>
<tbody>
<tr>
<td>Small stones</td>
<td>81/87 (93%)</td>
<td>234/270 (87%)</td>
</tr>
<tr>
<td>Large stones</td>
<td>192/263 (73%)</td>
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<tr>
<td>Total</td>
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<td>562/700 (80%)</td>
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\[
p(Y = y \mid do(X = x)) = \sum_{Pa(X)} p(y \mid x, Pa(X)) p(Pa(X))
\]

*adjustment formula for intervention on X*
Exercise 2b – Kidney stones revisited

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*adjustment formula for intervention on X*

Causal effect via adjustment

\[
p\left(\text{Recovery} \mid \hat{A}\right) = \sum_{S \in \{\text{small, large}\}} p\left(R \mid T = A, Size = S\right) p\left(S\right) = 0.93 \times 0.51 + 0.73 \times 0.49 = 0.832
\]

\[
p\left(\text{Recovery} \mid \hat{B}\right) = \sum_{S \in \{\text{small, large}\}} p\left(R \mid T = B, Size = S\right) p\left(S\right) = 0.87 \times 0.51 + 0.69 \times 0.49 = 0.782
\]

causal graph for kidney stone trial
Outline

1. Introduction to causality
2. Prediction vs. causation
3. Causal graphs and how to read them
4. Cause-effect estimation
5. The missing link & conclusion
Another experiment: preventing eclampsia

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- different labels, exact same numbers ... same conclusion?
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• different labels, exact same numbers ... same conclusion?

**Conclusion**

⇒ we need to know the true underlying causal graph to compute causal effects!
Take home messages so far ...

- causality is a very useful concept
- if we want to tap into its potential we can and should use methods that treat it in a principled manner (we aim for validity, not truth)
- key feature is distinguishing between association and causation
- not always easy, but often doable
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graphical causal models offer an intuitive way to model causal structure
we can link structure to data via structural equations / causal BNs
if we know the causal model we can use e.g. the *back-door criterion* and *adjustment* to compute/predict post-interventional distributions
leading to quantities of interest such as the *Average Causal Effect*

But much more to follow in the next two days!
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Thank you!