

Causality

A brief introduction to causal inference



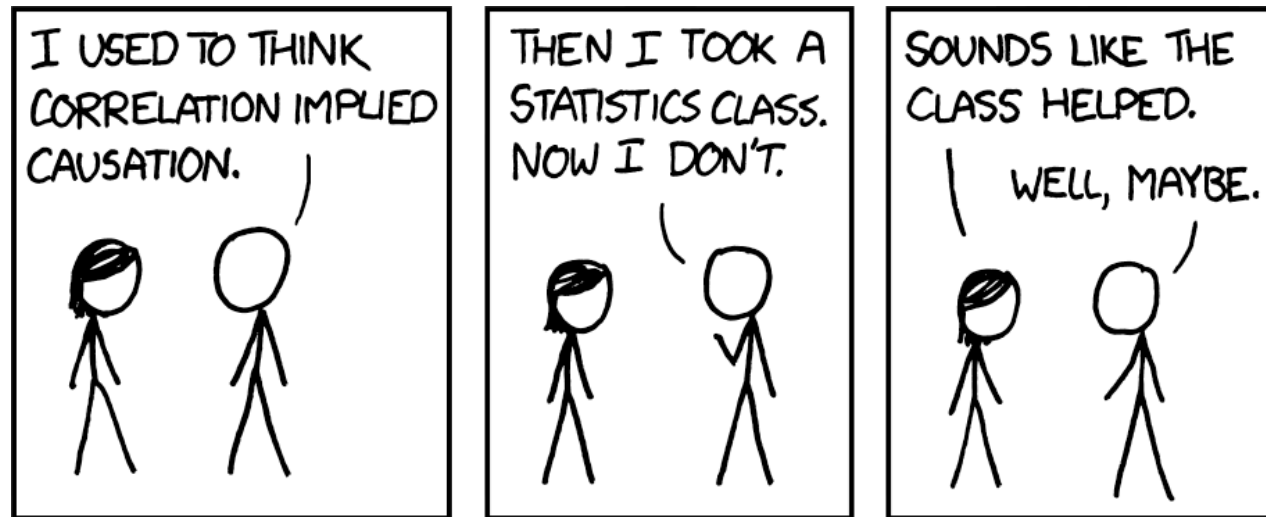
Neill D F Campbell

5th Aug 2026

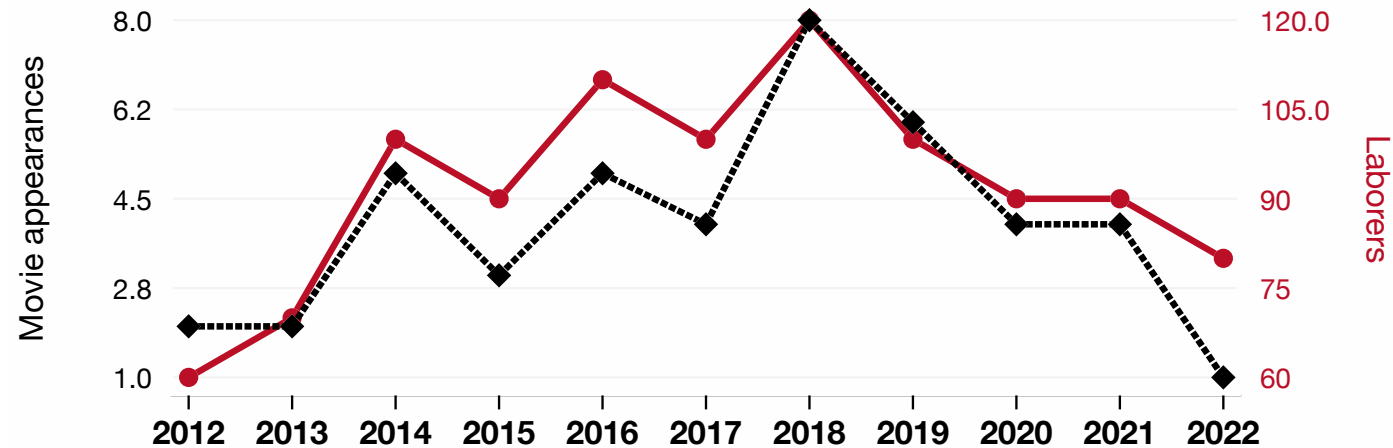
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Correlation vs Causation



The number of movies Nicolas Cage appeared in correlates with The number of MRI technicians in North Dakota



- ◆ The number of movies Nicolas Cage appeared in · Source: The Movie DB
- BLS estimate of magnetic resonance imaging technologists in North Dakota · Source: Bureau of Labor Statistics

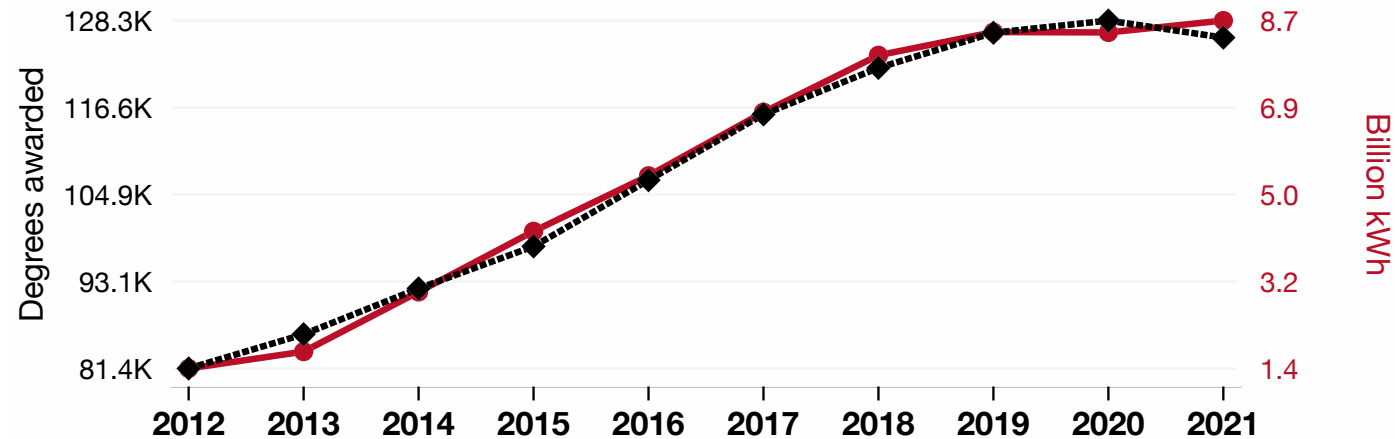
2012-2022, $r=0.871$, $r^2=0.759$, $p<0.01$ · tylervigen.com/spurious/correlation/6002

<https://tylervigen.com/spurious-correlations>

Bachelor's degrees awarded in Engineering

correlates with

Electricity generation in Cambodia



◆ Bachelor's degrees conferred by postsecondary institutions, in field of study: Engineering · Source: National Center for Education Statistics

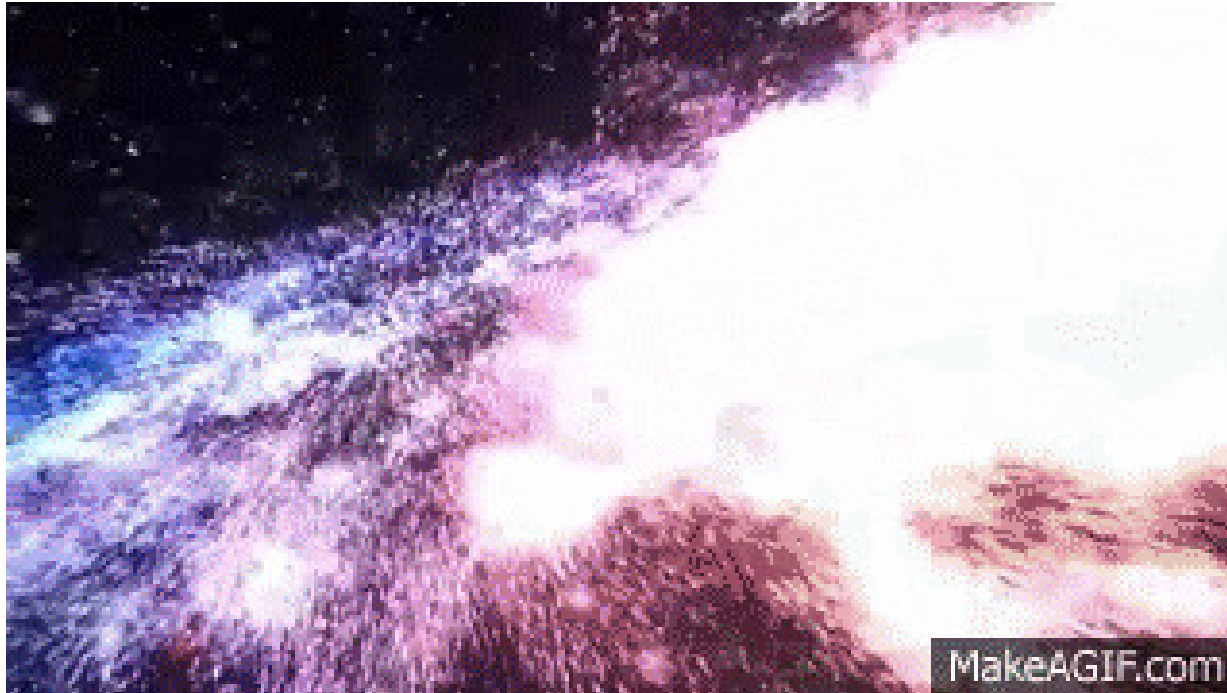
● Total electricity generation in Cambodia in billion kWh · Source: Energy Information Administration

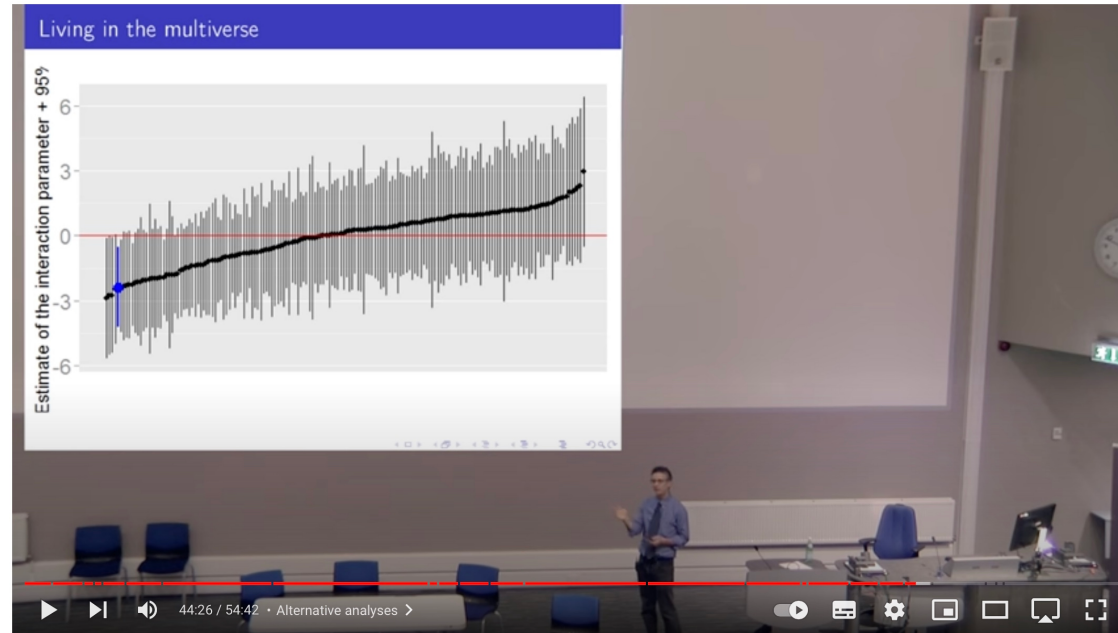
2012-2021, $r=0.997$, $r^2=0.994$, $p<0.01$ · tylervigen.com/spurious/correlation/2716

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- Do we need causation?
- Is science not just correlation?

Importance simultaneously undervalued and overestimated?





Crimes against data, Professor Andrew Gelman



National Centre for Research Methods (NCRM)

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Professor Andrew Gelman presented at the 7th ESRC Research Methods Festival, 5-7 July 2016, University of Bath. The Festival is organised every two years by the National Centre for Research Methods www.ncrm.ac.uk

Andrew Gelman: “Crimes against Data”

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 - e.g. atmospheric pressure and the barometer needle reading

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An object is the cause of another ...

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David Hume, *Enquiry Concerning Human Understanding*, 1748

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- Introduces the idea of a **counterfactual**

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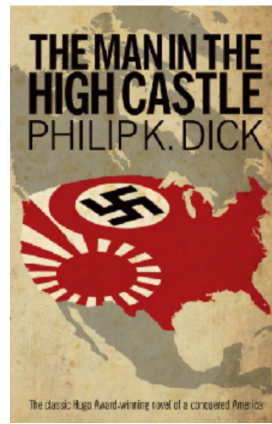
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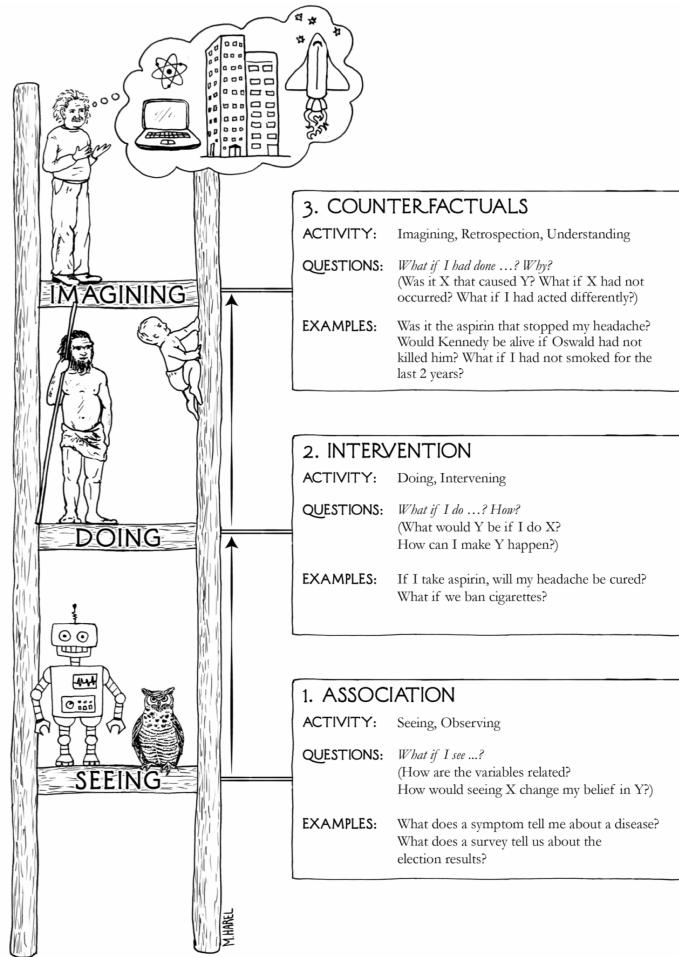
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- **Exciting question: what if we can't do an RCT?**
 - Can we use ML to estimate the counterfactuals? **Possibly!**



- Pearl proposes a “ladder of causation”
- Only the bottom rung can be answered by data alone
- Higher levels:
 - ▶ Require additional assumptions
 - ▶ Cannot be estimated from data alone...
 - ▶ **Need a model as well!**

[Pearl and Mackenzie 2017]

Bayesian Networks

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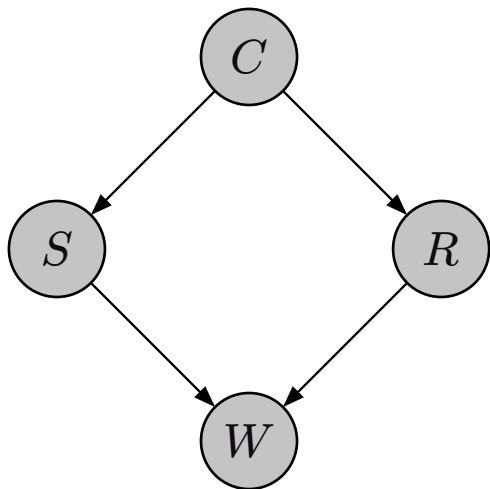
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- So a Bayesian network encodes a factorisation of the joint distribution.

The absence of edges is the important part!

Consider four binary variables: whether it is **cloudy** (C), whether the **sprinkler** is on (S), whether it **rains** (R), and whether the **grass is wet** (W).



The graph encodes the factorisation

$$p(C, S, R, W) = p(C) p(S \mid C) p(R \mid C) p(W \mid S, R)$$

Instead of the $2^4 - 1 = 15$ free parameters of a generic joint distribution over four binary variables, this factorisation needs only $1 + 2 + 2 + 4 = 9$.

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So what extra ingredients turns a DAG into a causal graph?

Two disease treatments (surgical / non-surgical) for kidney stones

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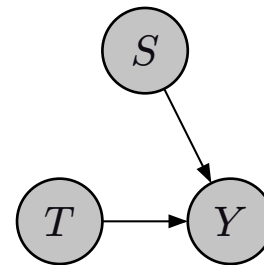
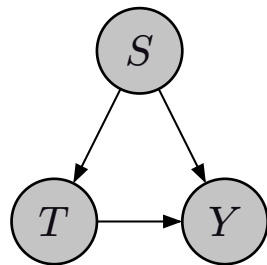
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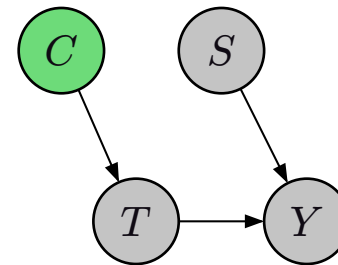
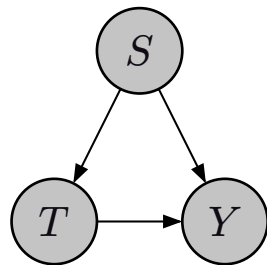
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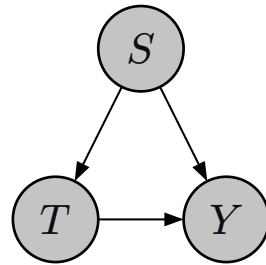
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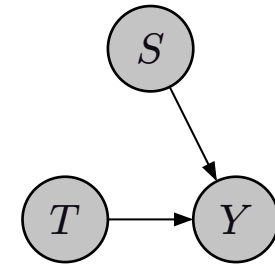


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$p(Y \mid T)$ means “probability of Y given T by observation alone”

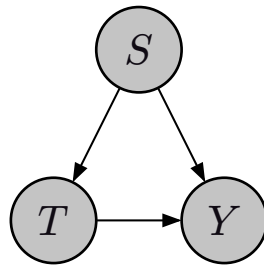
$$p(Y \mid T) = \sum_s p(Y \mid S, T) p(T \mid S) p(S) / p(T)$$

Probability of outcome Y given **intervening with treatment T** is $p(Y \mid \text{do}(T))$.

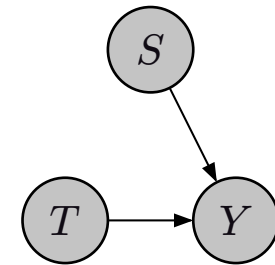
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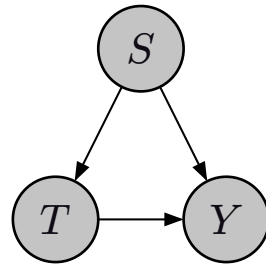
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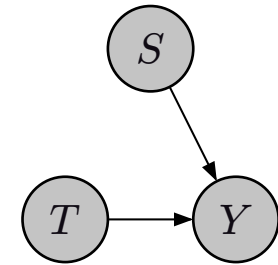
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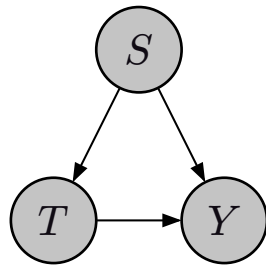


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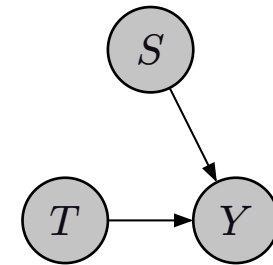
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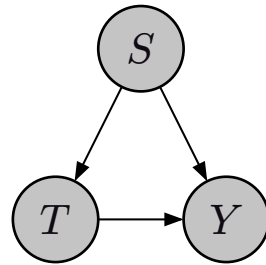
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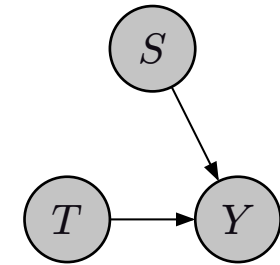
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Adjusting for the confounder recovers the correct causal ranking of the treatments!

- Statistical / probabilistic reasoning alone cannot support causal inference
- Determining the joint probability distribution of variables says nothing about causation
- **Causal Inference** promises to determine the necessary set of (non-data) assumptions sufficient to make a causal conclusion

Causal Graphs

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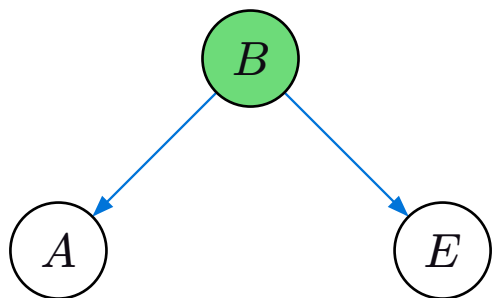
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- Causal graphs encode assumptions about the **data-generating process** itself, not just how a distribution happens to factorise
- This extra assumption is what lets us answer **interventional** questions, like $p(Y \mid \text{do}(T))$ from the kidney-stone example, that a Bayesian network alone cannot

The structure of the paths between two variables determines whether they are dependent.

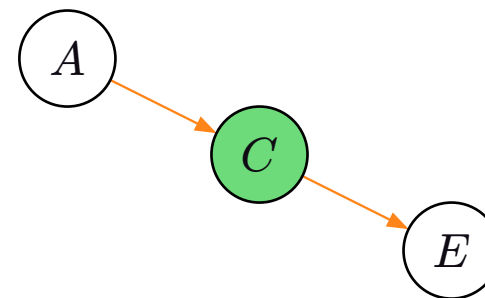
Fork (common cause)



B is a common cause of A and E .
Conditioning on B **blocks** the path:

$$A \perp E \mid B$$

Chain (mediation)



C mediates between A and E . Conditioning
on C **blocks** the path:

$$A \perp E \mid C$$

In both cases, **without** conditioning on the middle node information flows freely: $A \not\perp E$.

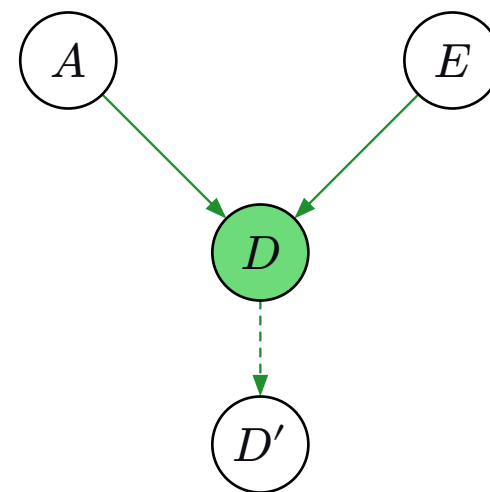
In a **collider** $A \rightarrow D \leftarrow E$, information flow is **blocked by default**:

$$A \perp E$$

Counterintuitively, conditioning on D (or on a **descendant** D' of D) **opens** the path:

$$A \not\perp E \mid D \quad A \not\perp E \mid D'$$

This is sometimes called **explaining away**; learning that D occurred, having ruled out A as the cause, makes E more probable as the explanation (and vice versa).



Do-Calculus



Do-calculus evaluates properties on a **modified graph** with certain edges removed:

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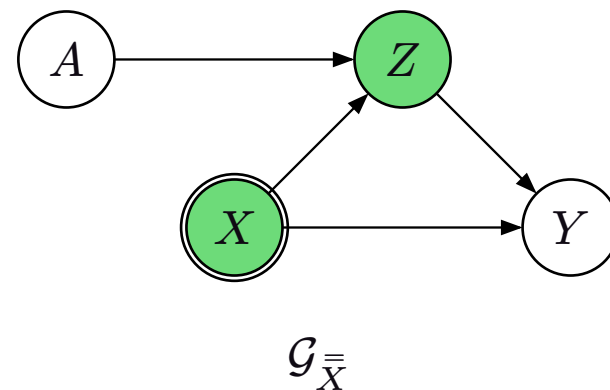
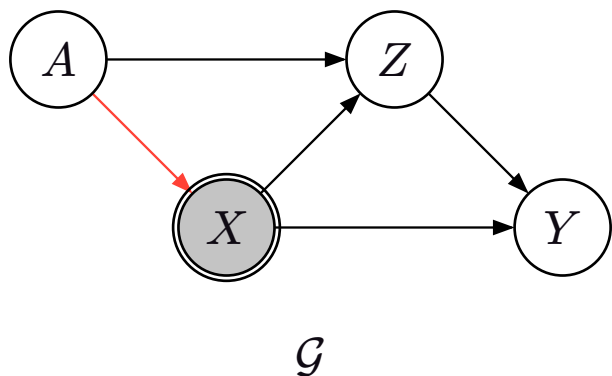
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Three transformation rules let us convert between **interventional** and **observational** distributions.

Insertion / deletion of an observation. If $Y \perp A \mid \{X, Z\}$ in $\mathcal{G}_{\bar{X}}$, we may add or remove the observation A from a conditional distribution:

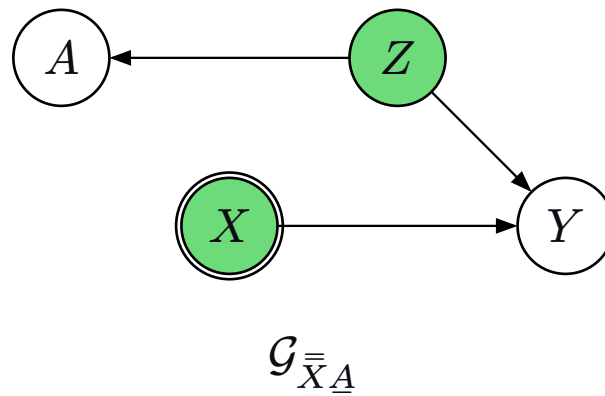
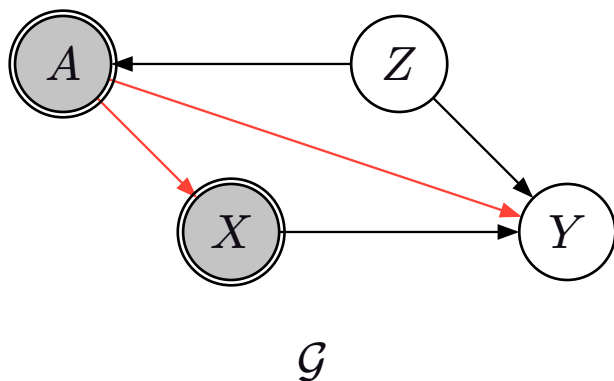
$$p(Y \mid Z, A, \text{do}(X)) \longleftrightarrow p(Y \mid Z, \text{do}(X))$$



Exchanging an intervention and an observation. If $Y \perp A \mid \{X, Z\}$ in $\mathcal{G}_{\bar{X}\underline{A}}$, we may swap an intervention on A for an observation of A :

$$p(Y \mid Z, \text{do}(A), \text{do}(X)) \longleftrightarrow p(Y \mid Z, A, \text{do}(X))$$

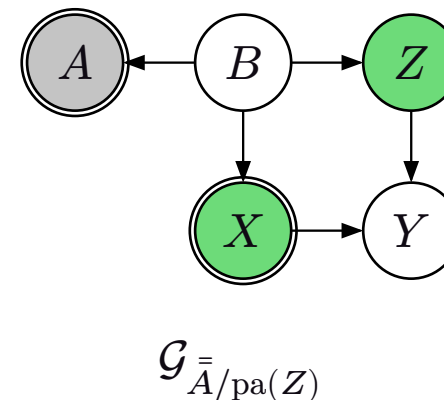
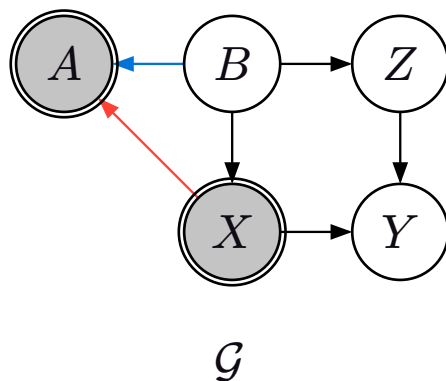
This is the **most important rule**: it lets us infer the effect of an intervention from passive observation, whenever A “behaves as if randomised” once we condition on Z .



Insertion / deletion of an intervention. If $Y \perp A \mid \{X, Z\}$ in $\mathcal{G}_{\bar{A}/\text{pa}(Z)}$, removing all edges into A **except** those from ancestors of Z , we may add or remove the intervention on A :

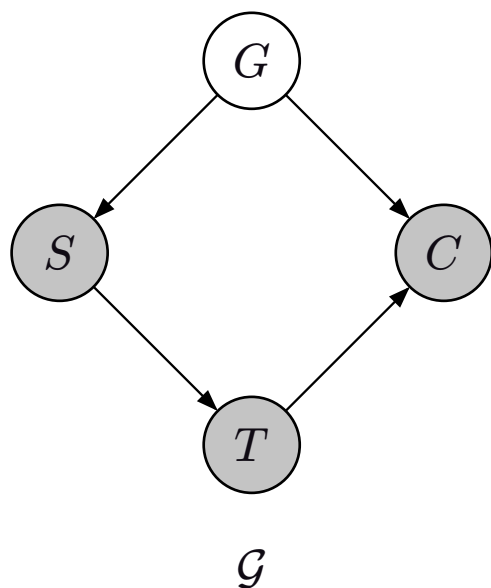
$$p(Y \mid Z, \text{do}(A), \text{do}(X)) \longleftrightarrow p(Y \mid Z, \text{do}(X))$$

This tells us A has **no causal effect** on Y in this context, so the intervention can simply be dropped.



Worked Example

We wish to infer the causal effect of smoking on lung cancer complicated by a possible genetic predisposition that affects both smoking and cancer risk. Measuring tar buildup (a mediator) will let us block this confounding path.



- S : does the individual smoke? (observed)
- T : is there a tar buildup in the lungs? (observed, mediator)
- C : does the individual have lung cancer? (observed)
- G : genetic predisposition. (latent, confounds S and C)

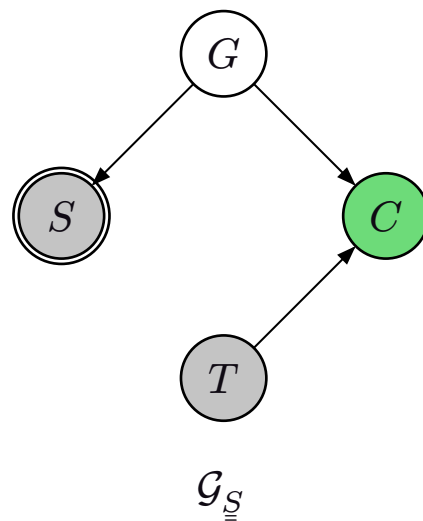
We need the interventional distribution $p(C \mid \text{do}(S))$ expressed purely in terms of **observational quantities**.

$$p(C \mid \text{do}(S)) = \sum_T p(C, T \mid \text{do}(S)) = \sum_T p(C \mid T, \text{do}(S)) p(T \mid \text{do}(S)) \quad (\text{Marginalisation})$$

Applying Rule 2 to swap the intervention on S for an observation:

$$p(T \mid \text{do}(S)) = p(T \mid S) \quad (\text{Rule 2: } T \perp S \text{ in } \mathcal{G}_{\underline{\underline{S}}}, \text{ blocked by } C)$$

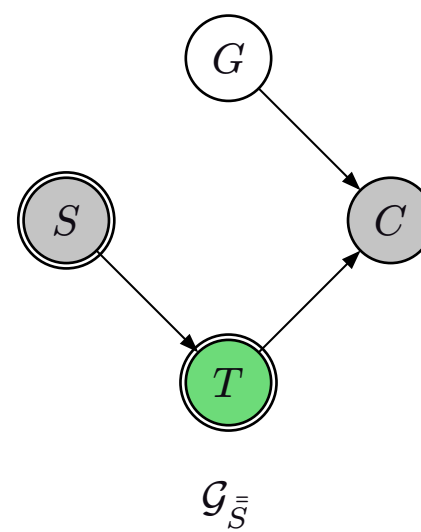
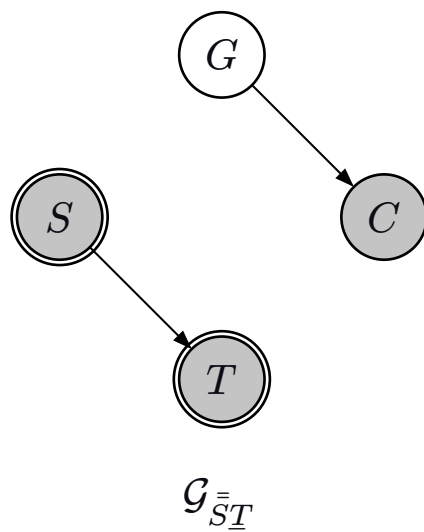
$$\Rightarrow p(C \mid \text{do}(S)) = \sum_T p(C \mid T, \text{do}(S)) p(T \mid S)$$



We cannot yet swap $p(C \mid T, \text{do}(S))$ directly: G still confounds S and C . Instead we use the mediator T to block that back-door path:

$$\begin{aligned} p(C \mid T, \text{do}(S)) &= p(C \mid \text{do}(S), \text{do}(T)) \quad (\text{Rule 2: } C \perp T \text{ in } \mathcal{G}_{\bar{S}\underline{\underline{T}}}) \\ &= p(C \mid \text{do}(T)) \quad (\text{Rule 3: } C \perp S \mid T \text{ in } \mathcal{G}_{\bar{S}}) \end{aligned}$$

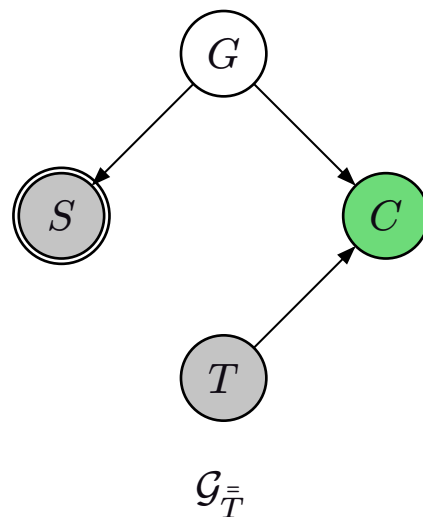
$$\Rightarrow p(C \mid \text{do}(S)) = \sum_T p(C \mid \text{do}(T)) p(T \mid S)$$



$$p(C \mid \text{do}(T)) = \sum_{S'} p(C, S' \mid \text{do}(T)) = \sum_{S'} p(C \mid S', \text{do}(T)) p(S' \mid \text{do}(T)) \quad (\text{Marginalisation})$$

Applying Rule 3 to drop the (now irrelevant) intervention on T :

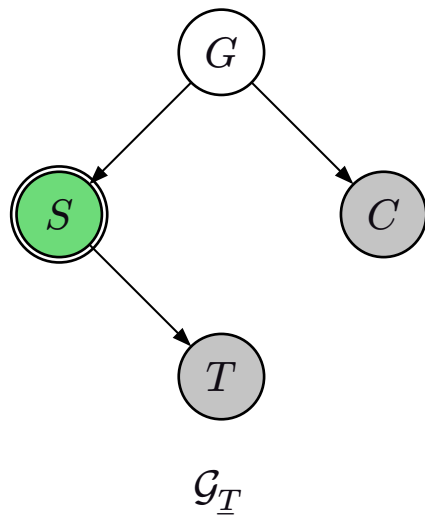
$$p(S \mid \text{do}(T)) = p(S) \quad (\text{Rule 3: } S \perp T \text{ in } \mathcal{G}_{\bar{T}}, \text{ blocked by } C)$$



For the remaining term, Rule 2 lets us swap the intervention on T once we condition on S :

$$p(C \mid S, \text{do}(T)) = p(C \mid S, T) \quad (\text{Rule 2: } C \perp T \mid S \text{ in } \underline{\underline{\mathcal{G}_T}})$$

$$\Rightarrow p(C \mid \text{do}(T)) = \sum_{S'} p(C \mid S', T) p(S')$$



Overall, the interventional distribution reduces to purely observational terms:

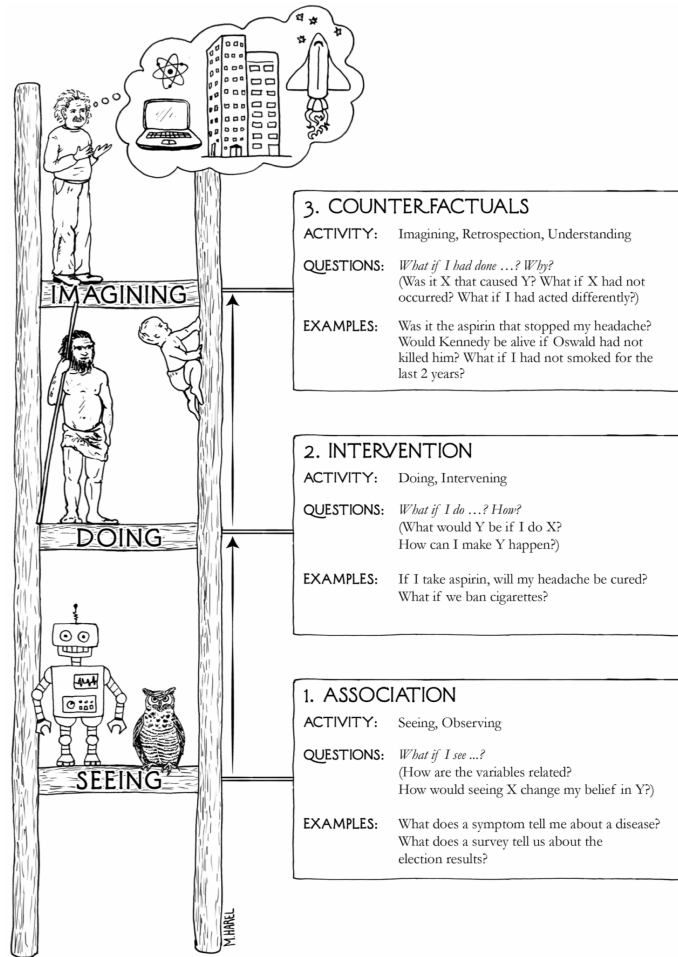
$$p(C \mid \text{do}(S)) = \sum_T p(C \mid \text{do}(T)) p(T \mid S) = \sum_T \left(\sum_{S'} p(C \mid S', T) p(S') \right) p(T \mid S)$$

Structural Causal Models

Causal knowledge cannot be extracted from data alone — you need a model that encodes assumptions about the world's mechanisms. **Structural causal models** make those assumptions explicit, testable, and mathematically tractable.

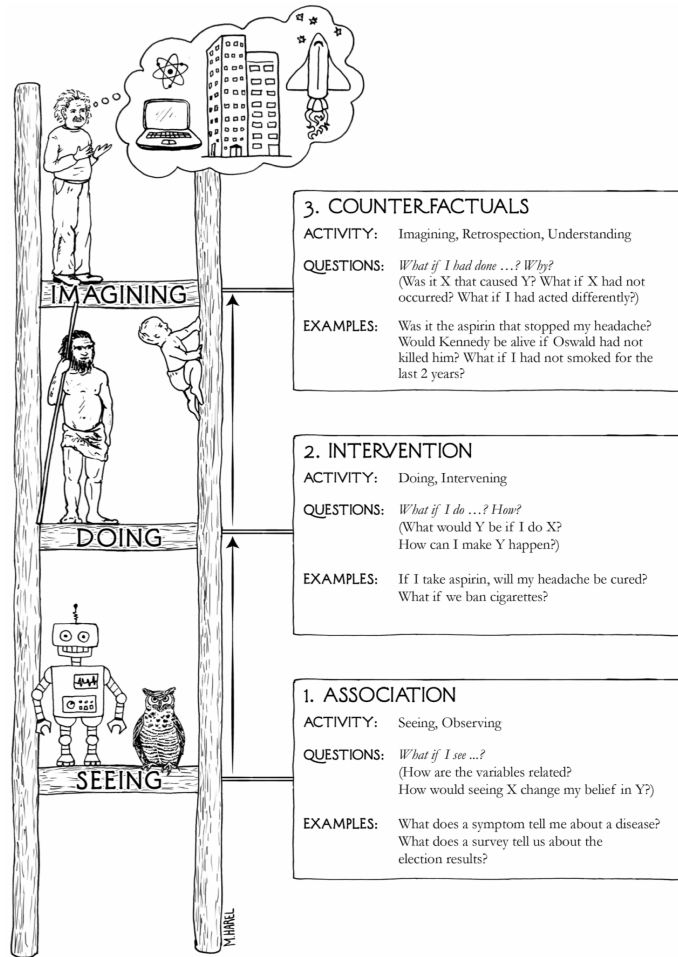
They provide a unified framework for:

- Causal inference from observational data
- Experimental design — what to randomise or control
- Mediation analysis — direct vs. indirect effects
- Policy evaluation — what happens if we intervene?
- Fairness in ML — defining and detecting discriminatory causation
- Explainable AI — counterfactual explanations



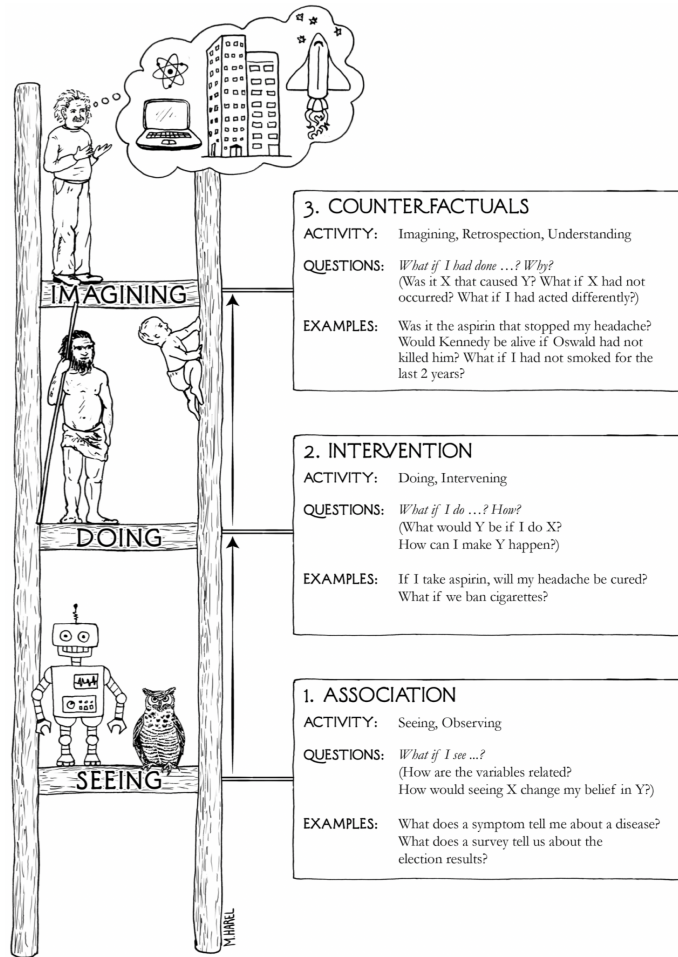
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[Pearl and Mackenzie 2017]



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 - ▶ Make causal claims at population level from observational data
 - ▶ Concerned with the effects of interventions (e.g., “what happens if we do X?”)
 - ▶ Models concerned with identifiability of causal effects from observational data

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- **Level 3: Counterfactuals: “what if?”, “why?”, fairness**
 - ▶ Make causal claims for individuals (from observational data)
 - ▶ Can we impute what would have happened to a *specific individual* if they had received a different treatment?

- J. Pearl (2009). *Causality: Models, Reasoning, and Inference* (2nd ed.).
- J. Pearl and D. Mackenzie (2017). *The Book of Why: The New Science of Cause and Effect*.
- Thanks to Julian Faraway for the correlation/causation illustrations used earlier in this talk.

Questions?