MLSS 2018: Causality

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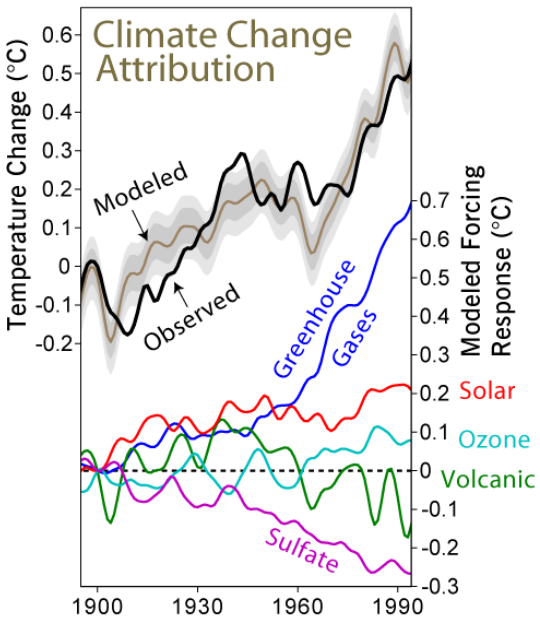
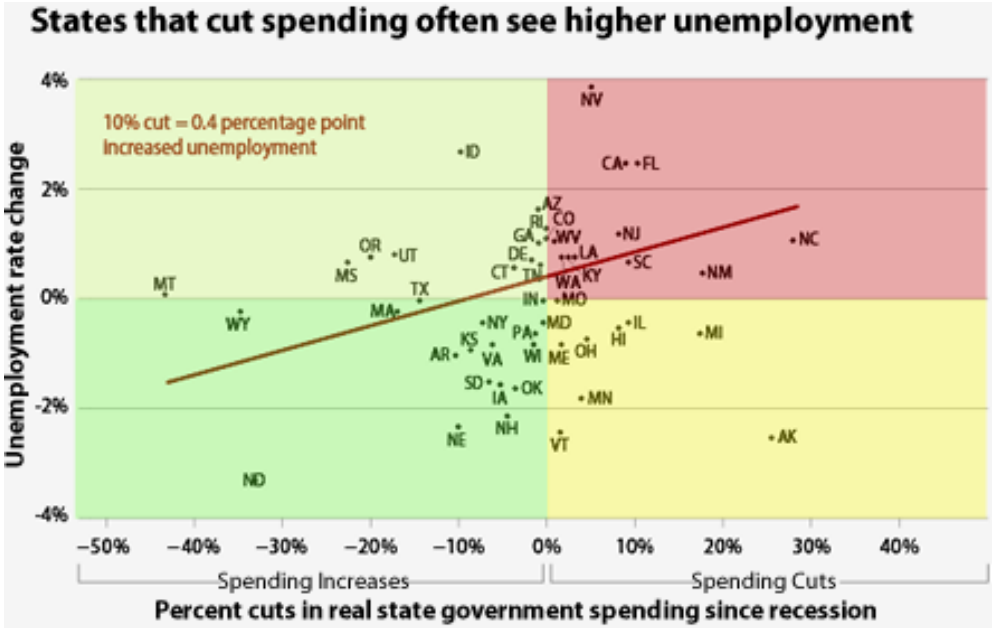
Many questions in science are *causal*

Climatology:

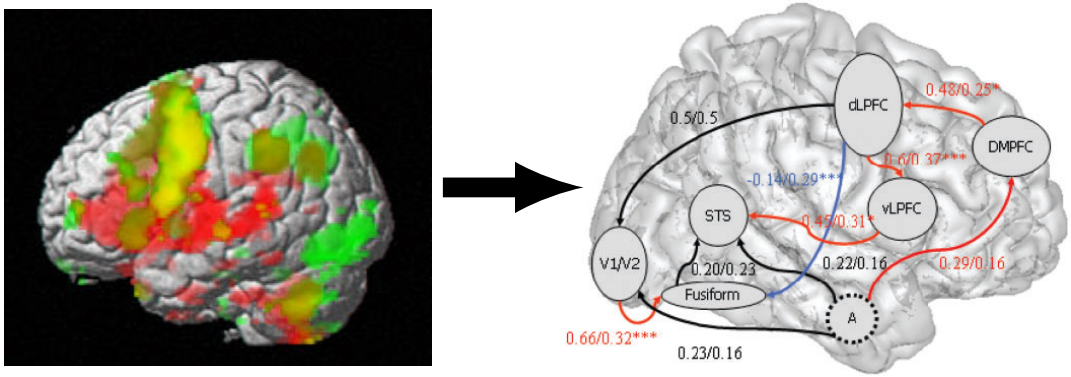


Economy:



Medicine:

Neuroscience:



Contents of this tutorial

Causality is clearly an important notion in daily life and in science.

- But how should we formalize the notion of causality?
- How to reason about causality?
- How can we discover causal relations from data?
- How to obtain causal predictions?
- How do they differ from ordinary predictions in ML?

That is what you will learn in this tutorial!

Probabilistic Inference vs. Causal Inference

Probabilistic Inference (traditional statistics / machine learning)

- Models the distribution of the data
- Focuses on predicting consequences of observations
- Useful e.g. in medical diagnosis: *given the symptoms of the patient, what is the most likely disease?*

Causal Inference

- Models the mechanism that generates the data
- Also allows to predict results of interventions
- Useful e.g. in medical treatment: *if we treat the patient with a drug, will it cure the disease?*

Causal reasoning is essential to answer questions of the type: *given the circumstances, what action should we take to achieve a certain goal?*

Contents

Causation $\neq$ Correlation

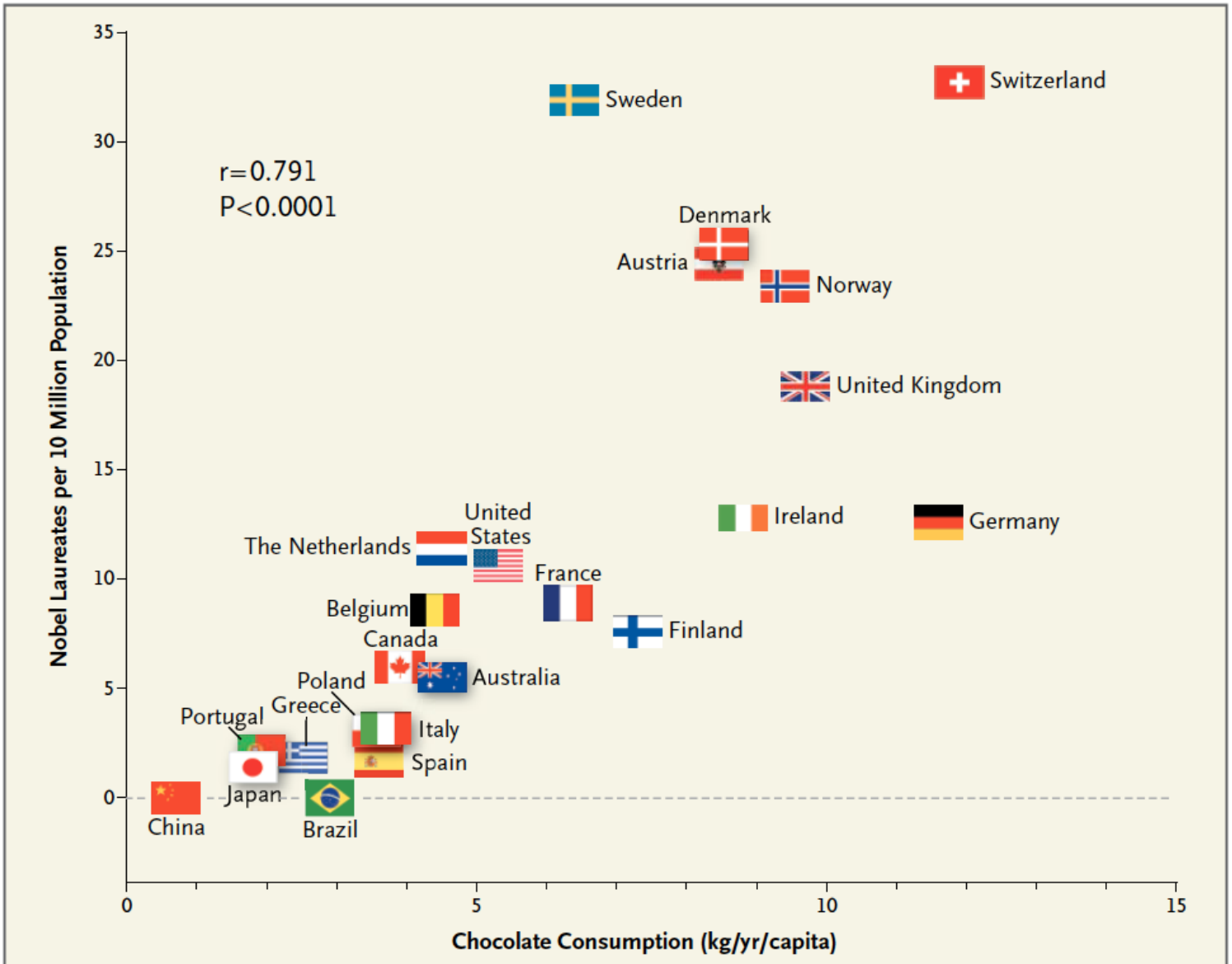


Figure 1. Correlation between Countries' Annual Per Capita Chocolate Consumption and the Number of Nobel Laureates per 10 Million Population.

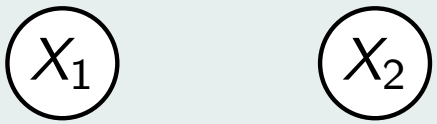
Causal relations

Definition (Informal)

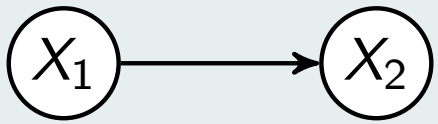
Let X and Y be two distinct variables of system. X causes Y if changing X (*intervening on* X) leads to a change of Y.

Causal graph represents causal relationships between variables graphically.

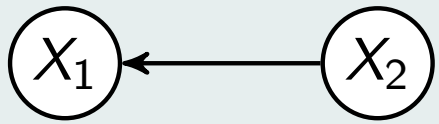
Example



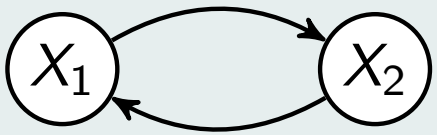
X_1 and X_2 are causally unrelated



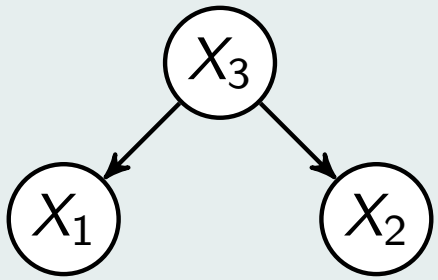
X_1 causes X_2



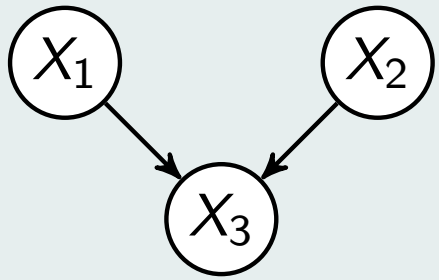
X_2 causes X_1



X_1 and X_2 cause each other



X_1 and X_2 have a common cause X_3



X_1 and X_2 have a common effect X_3

Direct causation

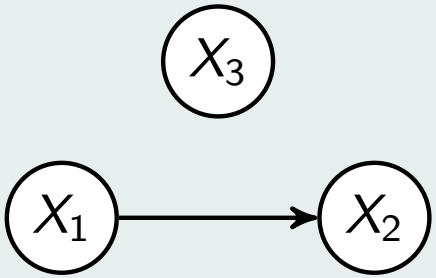
Let $\boldsymbol{V} = \{X_1, \ldots, X_N\}$ be a set of variables.

Definition

If X_i causes X_j even if all other variables $\boldsymbol{V} \setminus \{X_i, X_j\}$ are hold fixed at some values, then

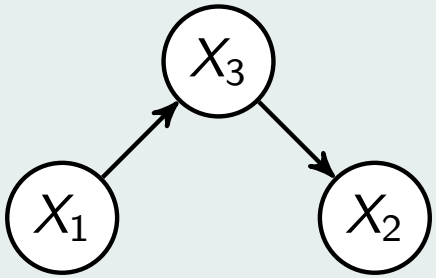
- we say that X_i causes X_j directly with respect to $\boldsymbol{V}$
- we indicate this in the causal graph on $\boldsymbol{V}$ by a directed edge $X_i \to X_j$

Example



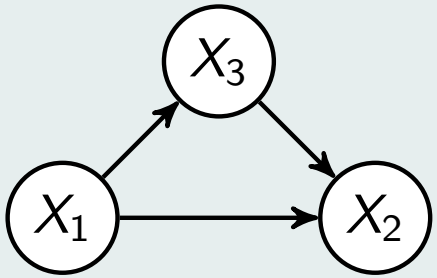
X_1 causes X_2;

X_1 causes X_2 directly w.r.t. $\{X_1, X_2, X_3\}$



X_1 causes X_2;

X_1 does not cause X_2 directly w.r.t. $\{X_1, X_2, X_3\}$

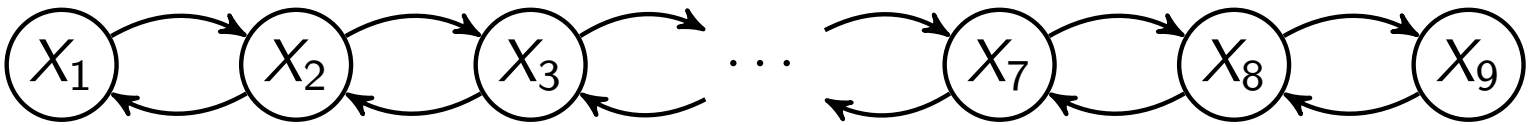


X_1 causes X_2;

X_1 causes X_2 directly w.r.t. $\{X_1, X_2, X_3\}$

Direct vs. indirect causation: Example

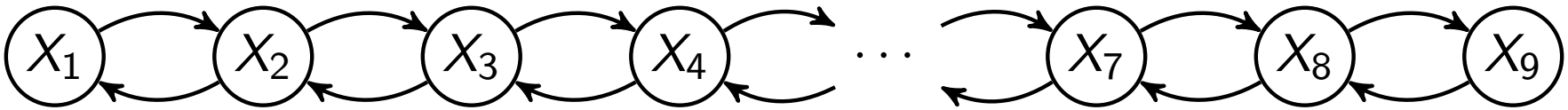
- Each stone causes *all* subsequent stones to topple.
- Each stone only directly causes the next neighboring stone to topple.
- Causal graph:



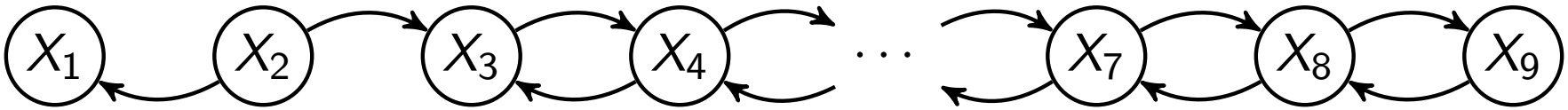
Perfect interventions: Example

Suppose we intervene by keeping the second stone fixed in an "upright" position (e.g. by glueing it to the floor), an operation that we denote by $\text{do}(X_2 = \text{upright})$.

Before the intervention, the causal graph is:



After the intervention $\text{do}(X_2 = \text{upright})$, the causal graph is:



If we keep the second stone fixed, it is no longer affected by the other stones.

Perfect interventions

Definition (Informal)

A perfect ("surgical", "atomic") intervention on a set of variables $\boldsymbol{X} \subseteq \boldsymbol{V}$, denoted $\mathrm{do}(\boldsymbol{X} = \boldsymbol{\xi})$, is an externally enforced change of the system that ensures that $\boldsymbol{X}$ takes on value $\boldsymbol{\xi}$ and leaves the rest of the system untouched.

The concept of perfect intervention assumes **modularity**: the causal system can be divided into two parts, $\boldsymbol{X}$ and $\boldsymbol{V} \setminus \boldsymbol{X}$, and we can make changes to one part while keeping the other part **invariant**.

Note

The intervention changes the causal graph by removing all edges that point towards variables in $\boldsymbol{X}$ (because none of the variables can now cause $\boldsymbol{X}$).

Confounders: Definition

Informally: a confounder is a latent common cause.

Definition

Consider three variables X, Y, H. H confounds X and Y if:

1. H causes X directly w.r.t. $\{X, Y, H\}$
2. H causes Y directly w.r.t. $\{X, Y, H\}$

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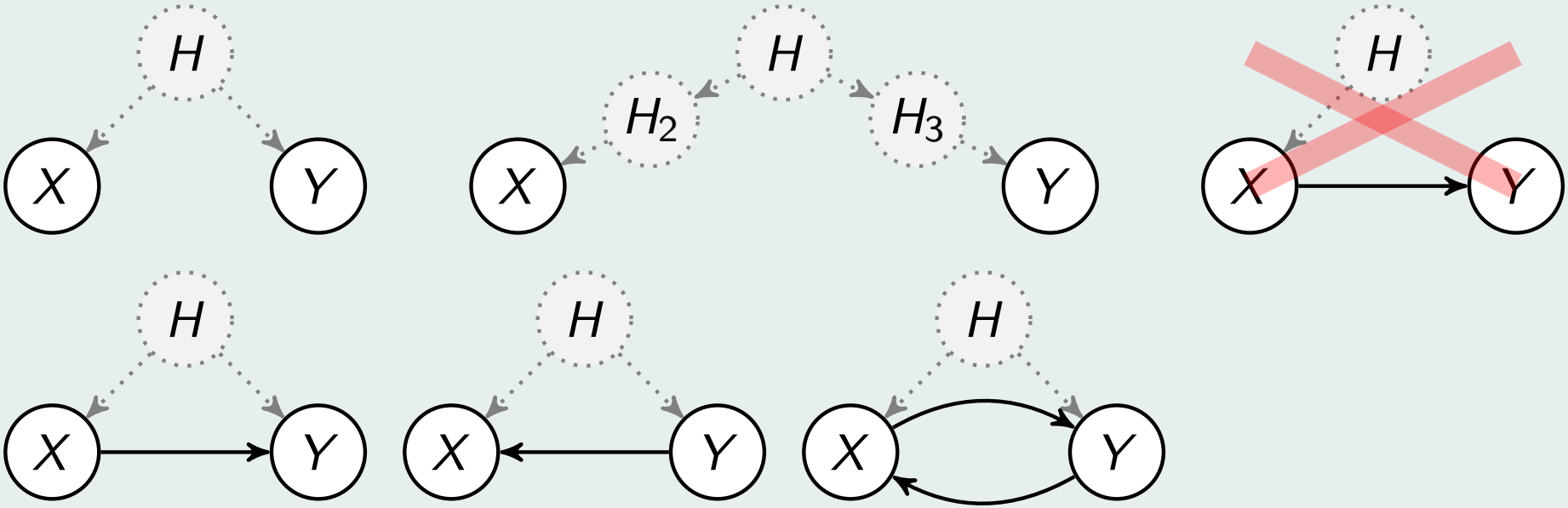
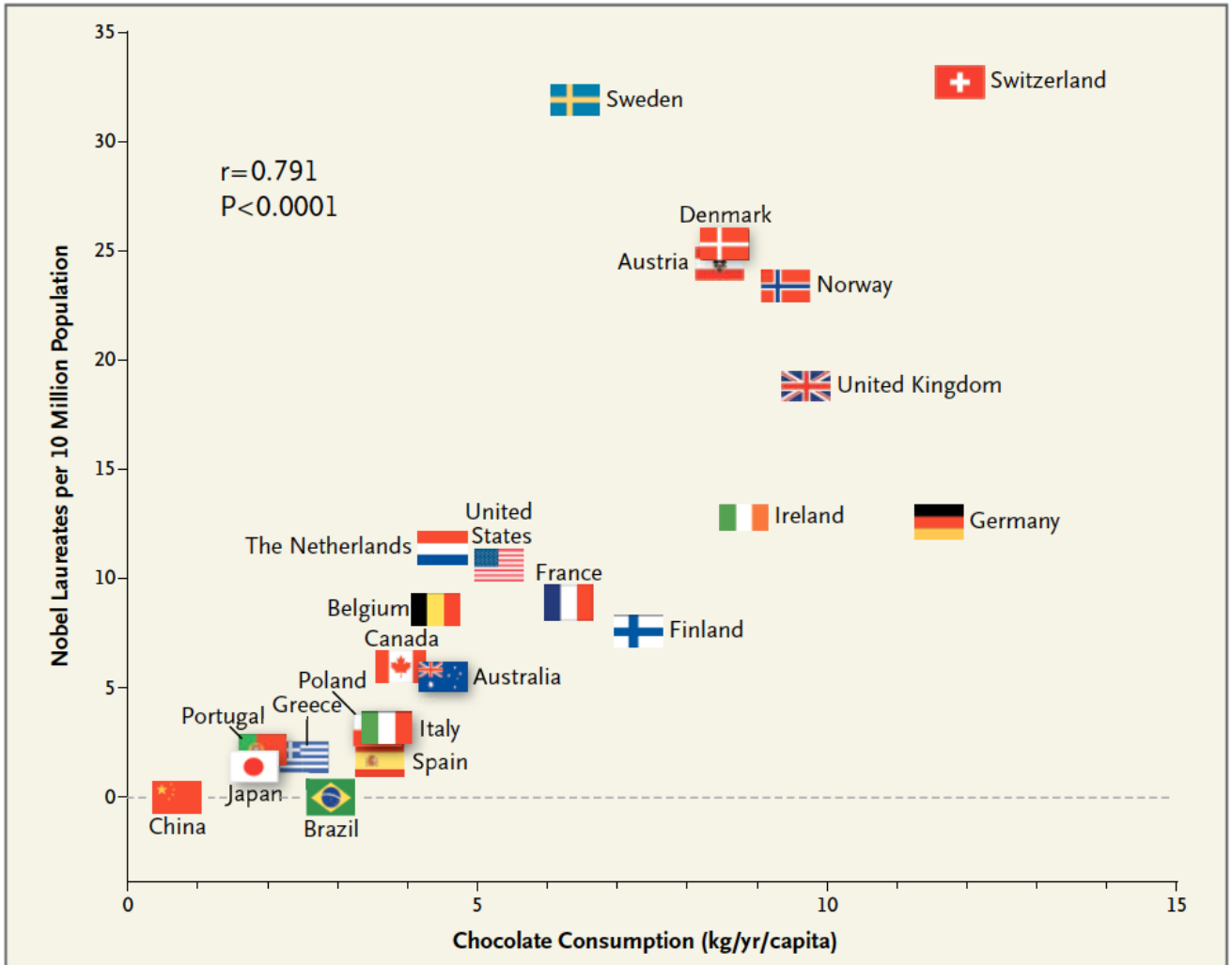


Confounders: Example

Wealth might confound chocolate consumption and Nobel prize winners.



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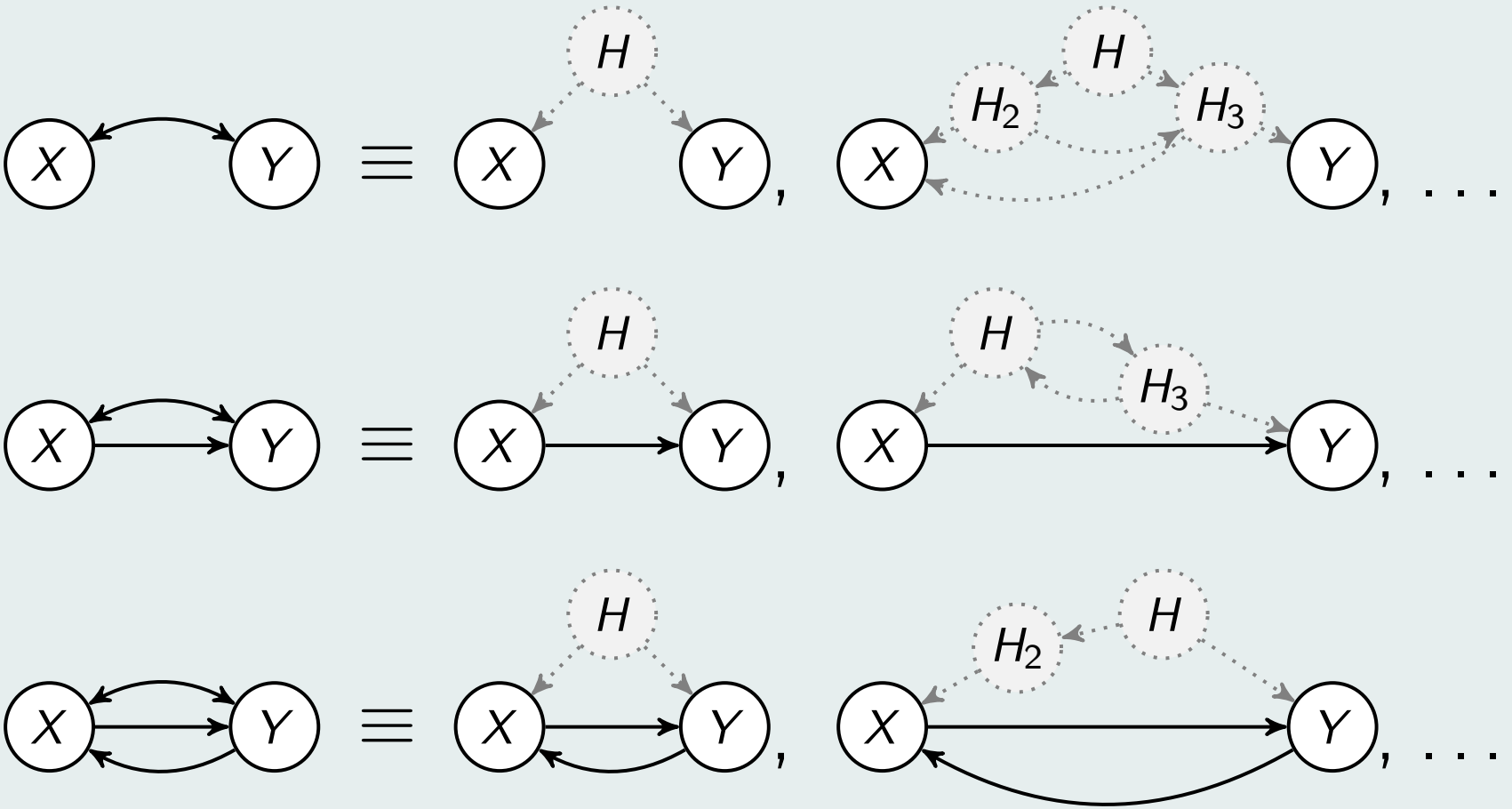
Confounders: Graphical notation

We denote latent confounders by bidirected edges in the causal graph:

Example



Causal Cycles: Definition and Example

Let X, Y be two variables in a system.

Definition

If X causes Y *and* X causes Y, then X and Y form a causal cycle.

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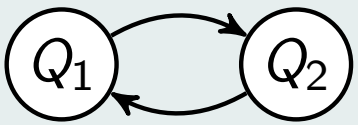
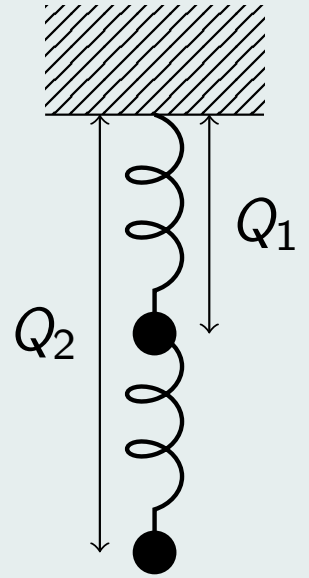
Example (Damped Coupled Harmonic Oscillators)

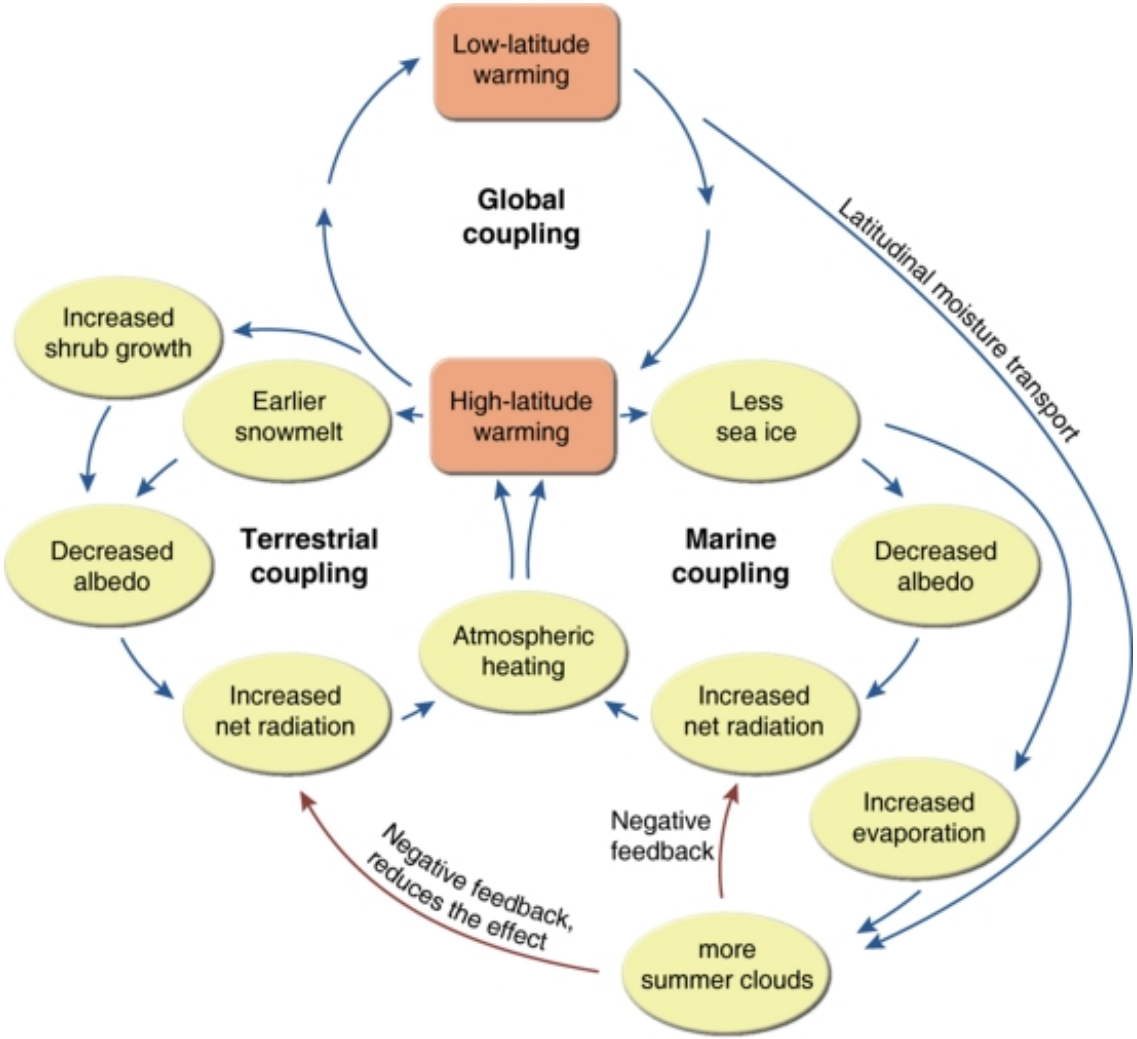
- Two masses, connected by a spring, suspended from the ceiling by another spring.
- Variables: vertical **equilibrium** positions Q_1 and Q_2.
- Q_1 causes Q_2.
- Q_2 causes Q_1.
- Causal graph:



- Cannot be modeled with acyclic causal model!

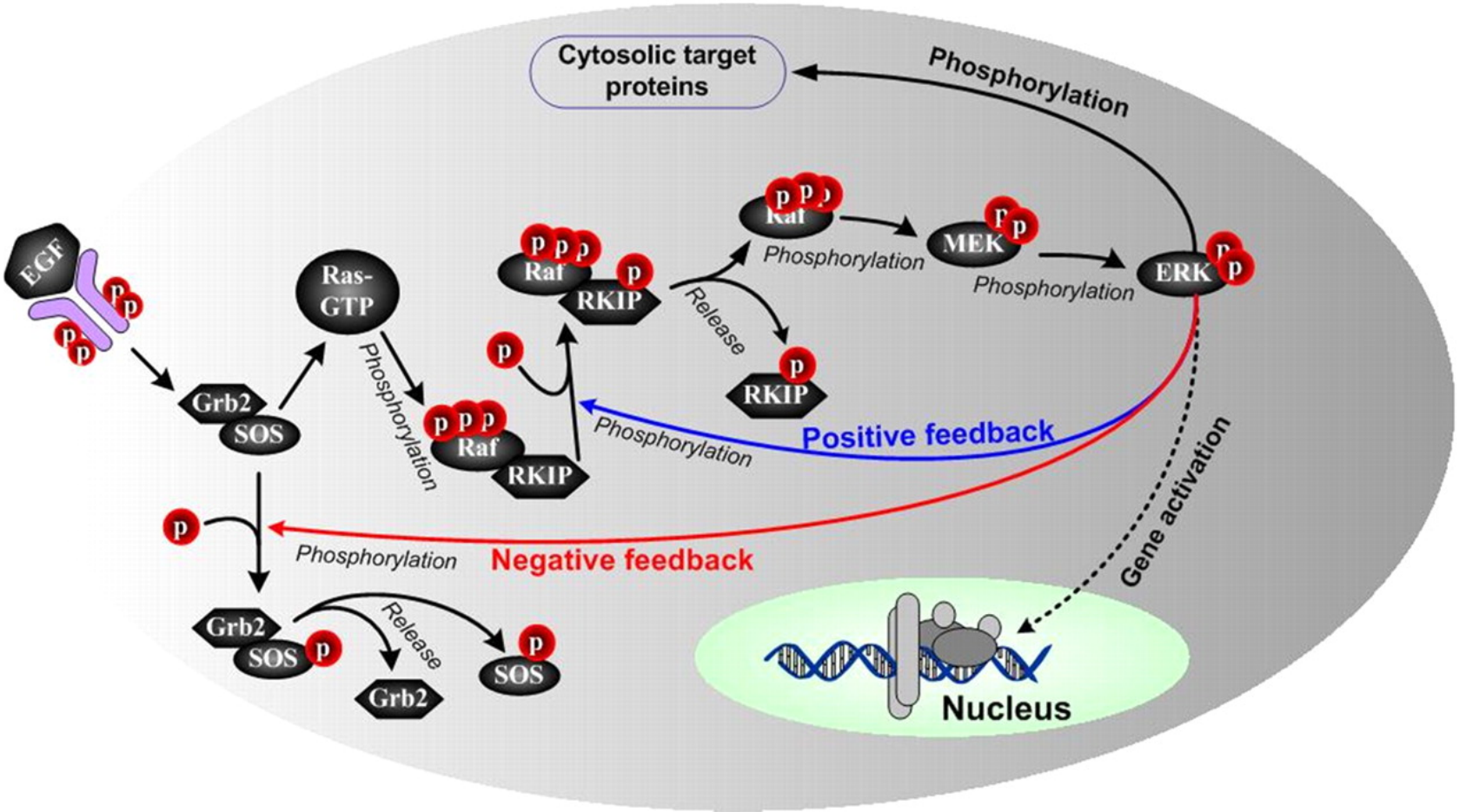


Cycles: Relevance in Climatology



"Part of the uncertainty around future climates relates to important feedbacks between different parts of the climate system: air temperatures, ice and snow albedo (reflection of the sun's rays), and clouds." [Ahlenius, 2007]

Cycles: Relevance in Biology

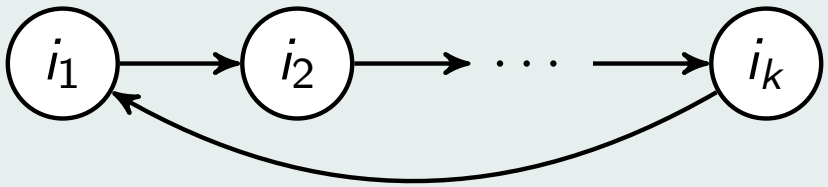


"Feedback mechanisms may be critical to allow cells to achieve the fine balance between dysregulated signaling and uncontrolled cell proliferation (a hallmark of cancer) as well as the capacity to switch pathways on or off when needed for physiologic purposes." [McArthur, 2014]

Graph Terminology

Definition

- A graph $\mathcal{G}$ that consists of directed and bidirected edges is called Directed Mixed Graph (DMG).
- If $i_1 \to i_2 \to \cdots \to i_k$ in $\mathcal{G}$ then i_1 is ancestor of i_k ($i_1 \in \mathrm{an}_{\mathcal{G}}(i_k)$).
- $\mathcal{G}$ is called cyclic if it contains a directed cycle:



- The strongly connected component of a node $i \in \mathcal{G}$ is the set of nodes $j \in \mathcal{G}$ such that i and j are each other's ancestors.
- If $\mathcal{G}$ does not contain such a directed cycle, it is called acyclic, and known as an Acyclic Directed Mixed Graph (ADMG).
- If, in addition, $\mathcal{G}$ does not contain any bidirected edges, it is called a Directed Acyclic Graph (DAG).

Contents

Defining Causality in terms of Probabilities?

When looking for a more quantitative treatment of causality, it is a natural idea to try to *define* causality in terms of probabilities.

A naïve example of such an attempt could be:

Attempt at a definition

Given two binary random variables A, B. If

- A precedes B in time, and
- $p(B = 1 \,|\, A = 1) > p(B = 1 \,|\, A = 0)$

then A causes B.

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then A causes B.

This does not work, as exemplified by *Simpson's paradox*.

Exercise

Please make Exercise 1.

Simpson's Paradox

Example (Simpson's paradox)

We collect electronic patient records to investigate the effectiveness of a new drug against a certain disease. We find that:

1. The probability of recovery is higher for patients that took the drug:

$$p(\text{recovery} \mid \text{drug}) > p(\text{recovery} \mid \text{no drug})$$

2. For **both male and female** patients, the relation is **opposite**:

$$p(\text{recovery} \mid \text{drug}, \text{male}) < p(\text{recovery} \mid \text{no drug}, \text{male})$$

$$p(\text{recovery} \mid \text{drug}, \text{female}) < p(\text{recovery} \mid \text{no drug}, \text{female})$$

Does the drug cause recovery? I.e., would *you* use this drug if you are ill?

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Does the drug cause recovery? I.e., would *you* use this drug if you are ill?

Note: Big data and deep learning **do not help** us here!

Quantitative Models of Causality

Problems like these have historically prevented statisticians from considering causality.

Nonetheless, different approaches have been proposed to model causality in a quantitative way:

- Potential outcome framework
- Causal Bayesian Networks
- **Structural Causal Models** (SCMs)

We will use SCMs, as they are arguably the most general of the three:

- SCMs can model cycles naturally (natural connection to ubiquitous ODE models)
- Acyclic SCMs are closed under marginalization (can efficiently handle latent variables)
- SCMs can model counterfactuals (provides alternative to potential outcome framework)
- SCMs generalize Causal Bayesian Networks

Structural Causal Models: Concepts

SCMs turn things upside down: rather than defining causality in terms of probabilities, probability distributions are defined by a causal model, thereby avoiding traps like Simpson's paradox.

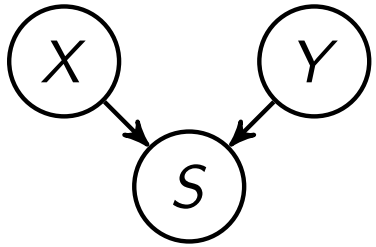
- The *system* we are modeling is described by endogenous variables; endogenous variables are:
  - observed,
  - modeled by structural equations.
- The *environment* of the system is described by exogenous variables; exogenous variables are:
  - latent (unobserved),
  - modeled by probability distributions,
  - *not caused* by endogenous variables,
  - provide the "source" of randomness.
- *Each endogenous variable has its own structural equation, which describes how this variable depends causally on other variables.*
- SCMs are equipped with a notion of perfect intervention, which gives them a *causal* semantics.

Structural Causal Models: Example

Endogenous variables (binary):

X: the battery is charged
Y: the start engine is operational
S: the car starts

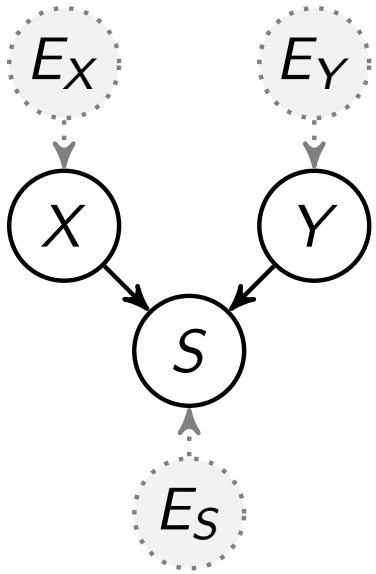
Causal graph:



Exogenous variables (latent, independent, binary):

$$E_X \sim \mathrm{Ber}(0.95)$$
$$E_Y \sim \mathrm{Ber}(0.99)$$
$$E_S \sim \mathrm{Ber}(0.999)$$

Augmented functional graph:



Structural equations (one per endogenous variable):

$$X = f_X(E_X) = E_X$$
$$Y = f_Y(E_Y) = E_Y$$
$$S = f_S(X, Y, E_S) = X \wedge Y \wedge E_S$$

Structural Causal Models: Formal Definition

Definition ([Wright, 1921, Pearl, 2000, Bongers et al., 2018])

A Structural Causal Model (SCM), also known as Structural Equation Model (SEM), is a tuple $\mathcal{M} = \langle \boldsymbol{\mathcal{X}}, \boldsymbol{\mathcal{E}}, \boldsymbol{f}, \mathbb{P}_{\boldsymbol{\mathcal{E}}} \rangle$ with:

1. a product of standard measurable spaces $\boldsymbol{\mathcal{X}} = \prod_{i \in \mathcal{I}} \mathcal{X}_i$ (domains of the endogenous variables)
2. a product of standard measurable spaces $\boldsymbol{\mathcal{E}} = \prod_{j \in \mathcal{J}} \mathcal{E}_j$ (domains of the exogenous variables)
3. a measurable mapping $\boldsymbol{f} : \boldsymbol{\mathcal{X}} \times \boldsymbol{\mathcal{E}} \to \boldsymbol{\mathcal{X}}$ (the causal mechanism)
4. a product probability measure $\mathbb{P}_{\boldsymbol{\mathcal{E}}} = \prod_{j \in \mathcal{J}} \mathbb{P}_{\mathcal{E}_j}$ on $\boldsymbol{\mathcal{E}}$ (the exogenous distribution)

Definition

A pair of random variables $(\boldsymbol{X}, \boldsymbol{E})$ is a solution of SCM $\mathcal{M}$ if $\mathbb{P}^{\boldsymbol{E}} = \mathbb{P}_{\boldsymbol{\mathcal{E}}}$ and the structural equations $\boldsymbol{X} = \boldsymbol{f}(\boldsymbol{X}, \boldsymbol{E})$ hold a.s..

Structural Causal Models: Example

Example

Structural Causal Model $\mathcal{M}$:

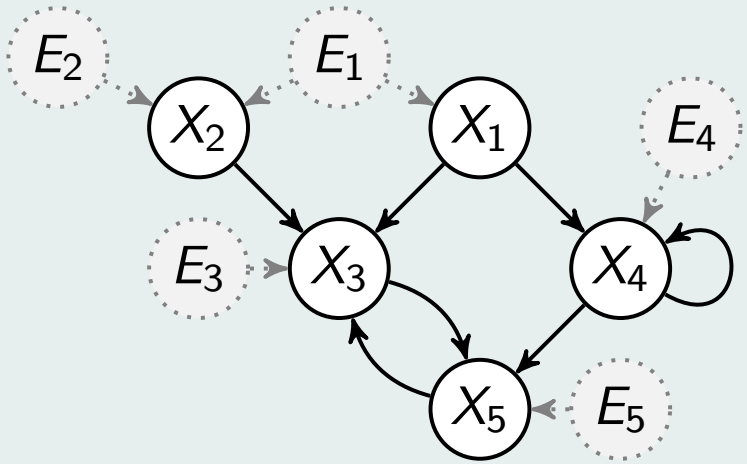
Formally:

$$(\boldsymbol{\mathcal{X}}, \boldsymbol{\mathcal{E}}, \boldsymbol{f}, \mathbb{P}_{\boldsymbol{\mathcal{E}}}) = \left(\prod_{i=1}^{5} \mathbb{R}, \prod_{j=1}^{5} \mathbb{R}, (f_1, \ldots, f_5), \prod_{j=1}^{5} \mathbb{P}_{\mathcal{E}_j}\right)$$

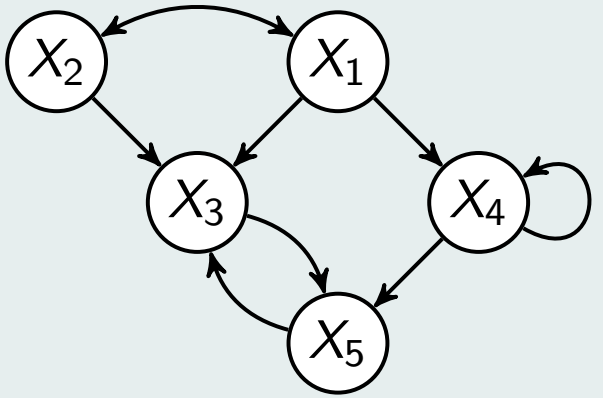
Informally:

$$\begin{aligned}
X_1 &= f_1(E_1) & \mathbb{P}^{E_1} &= \ldots \\
X_2 &= f_2(E_1, E_2) & \mathbb{P}^{E_2} &= \ldots \\
X_3 &= f_3(X_1, X_2, X_5, E_3) & \mathbb{P}^{E_3} &= \ldots \\
X_4 &= f_4(X_1, X_4, E_4) & \mathbb{P}^{E_4} &= \ldots \\
X_5 &= f_5(X_3, X_4, E_5) & \mathbb{P}^{E_5} &= \ldots
\end{aligned}$$

Augmented functional graph $\mathcal{G}^a(\mathcal{M})$:



Functional graph $\mathcal{G}(\mathcal{M})$:



(Augmented) Functional Graphs

Definition

The components of the causal mechanism usually do not depend on *all* variables: for $i \in \mathcal{I}$,

$$X_i = f_i(\boldsymbol{X}_{\mathrm{pa}_i^{\mathcal{I}}}, \boldsymbol{E}_{\mathrm{pa}_i^{\mathcal{J}}})$$

where f_i only depends on $\mathrm{pa}_i^{\mathcal{I}} \subseteq \mathcal{I}$ (the endogenous parents of i) and $\mathrm{pa}_i^{\mathcal{J}} \subseteq \mathcal{J}$ (the exogenous parents of i).

Definition

The augmented functional graph $\mathcal{G}^a(\mathcal{M})$ of SCM $\mathcal{M}$ is a directed graph with nodes $\mathcal{I} \dot{\cup} \mathcal{J}$ and an edge $k \to i$ iff $k \in \mathrm{pa}_i^{\mathcal{I}} \dot{\cup} \mathrm{pa}_i^{\mathcal{J}}$ is a parent of $i \in \mathcal{I}$.

Definition

The functional graph $\mathcal{G}(\mathcal{M})$ of SCM $\mathcal{M}$ is a DMG with nodes $\mathcal{I}$, directed edges $k \to i$ iff $k \in \mathrm{pa}_i^{\mathcal{I}}$, and bidirected edges $k \leftrightarrow i$ iff $\mathrm{pa}_i^{\mathcal{J}} \cap \mathrm{pa}_k^{\mathcal{J}} \neq \emptyset$.

Causal Graph

Definition

We say $\mathcal{M}$ has a self-loop at $i \in \mathcal{I}$ if $i \in \mathrm{pa}_i^{\mathcal{I}}$.

Proposition ([Bongers et al., 2018])

If $\mathcal{M}$ has no self-loops, the causal graph of $\mathcal{M}$ is a subgraph of the functional graph $\mathcal{G}(\mathcal{M})$.

In that case, generically:

- The directed edges in $\mathcal{G}(\mathcal{M})$ represent direct causal relations w.r.t. $\mathcal{I}$;
- The bidirected edges in $\mathcal{G}(\mathcal{M})$ may represent the existence of confounders w.r.t. $\mathcal{I}$.
- A direct causal relation $X_i \to X_j$ w.r.t. $\mathcal{I}$ can be detected experimentally by intervening on all variables $\boldsymbol{X}_{\mathcal{I}\setminus\{j\}}$ except X_j, and testing if the marginal distributions of the solutions on X_j depend on the value to which X_i is set.

Interventions

To interpret an SCM as a *causal* model, we also need to define its semantics under interventions.

Definition (Perfect Interventions, [Pearl, 2000])

- The perfect intervention $\mathrm{do}(\boldsymbol{X}_I = \boldsymbol{\xi}_I)$ enforces $\boldsymbol{X}_I$ to attain value $\boldsymbol{\xi}_I$.
- This changes the SCM $\mathcal{M} = \langle \boldsymbol{\mathcal{X}}, \boldsymbol{\mathcal{E}}, \boldsymbol{f}, \mathbb{P}_{\boldsymbol{\mathcal{E}}} \rangle$ into the intervened SCM $\mathcal{M}_{\mathrm{do}(\boldsymbol{X}_I = \boldsymbol{\xi}_I)} = \langle \boldsymbol{\mathcal{X}}, \boldsymbol{\mathcal{E}}, \tilde{\boldsymbol{f}}, \mathbb{P}_{\boldsymbol{\mathcal{E}}} \rangle$ where

$$\tilde{f}_i = \begin{cases} \xi_i & i \in I \\ f_i(\boldsymbol{X}_{\mathrm{pa}_i^{\mathcal{I}}}, \boldsymbol{E}_{\mathrm{pa}_i^{\mathcal{J}}}) & i \notin I. \end{cases}$$

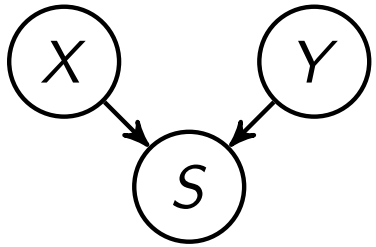
- Interpretation: overrides default causal mechanisms that normally would determine the values of the intervened variables.
- In the (augmented) functional graph, the intervention removes all incoming edges with an arrowhead at any intervened variable $i \in I$.

Interventions: Example

Endogenous variables (binary):

X: the battery is charged
Y: the start engine is operational
S: the car starts

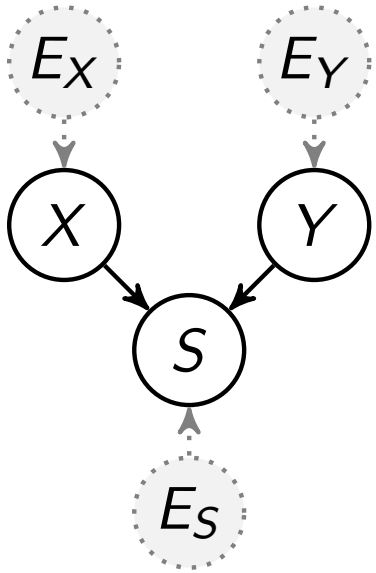
Causal graph:



Exogenous variables (latent, independent, binary):

$E_X \sim \mathrm{Ber}(0.95)$
$E_Y \sim \mathrm{Ber}(0.99)$
$E_Z \sim \mathrm{Ber}(0.999)$

Augmented functional graph:



Structural equations (one per endogenous variable):

$X = E_X$
$Y = E_Y$
$S = X \wedge Y \wedge E_S$

Interventions: Example

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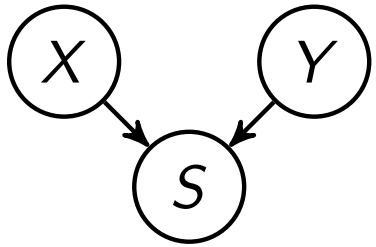

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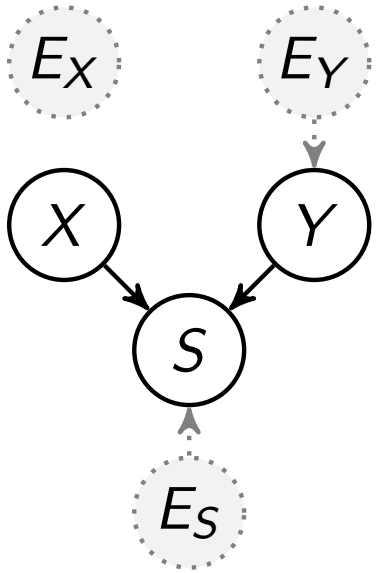


Structural equations (one per endogenous variable):
after charging the battery $\mathrm{do}(X = 1)$:

$X = 1$
$Y = E_Y$
$S = X \wedge Y \wedge E_S$

Interventions: Example

Endogenous variables (binary):

X: the battery is charged
Y: the start engine is operational
S: the car starts

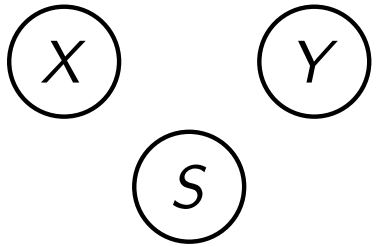
Exogenous variables (latent, independent, binary):

$E_X \sim \mathrm{Ber}(0.95)$
$E_Y \sim \mathrm{Ber}(0.99)$
$E_Z \sim \mathrm{Ber}(0.999)$

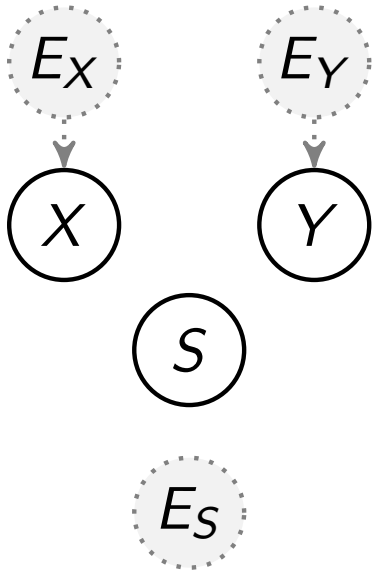
Structural equations (one per endogenous variable):
after loosing the key $\mathrm{do}(S = 0)$:

$X = E_X$
$Y = E_Y$
$S = 0$

Causal graph:



Augmented functional graph:



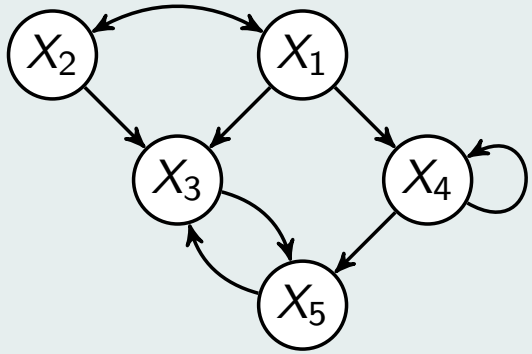
Example

Observational (no intervention):

Structural Causal Model $\mathcal{M}$:

$$
\begin{aligned}
X_1 &= f_1(E_1) & \mathbb{P}^{E_1} &= \dots \\
X_2 &= f_2(E_1, E_2) & \mathbb{P}^{E_2} &= \dots \\
X_3 &= f_3(X_1, X_2, X_5, E_3) & \mathbb{P}^{E_3} &= \dots \\
X_4 &= f_4(X_1, X_4, E_4) & \mathbb{P}^{E_4} &= \dots \\
X_5 &= f_5(X_3, X_4, E_5) & \mathbb{P}^{E_5} &= \dots
\end{aligned}
$$

Functional graph $\mathcal{G}(\mathcal{M})$:

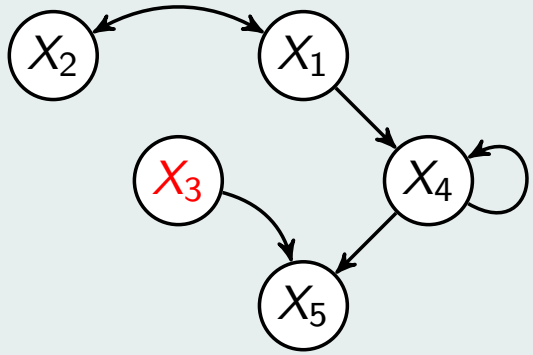


Intervention $\mathrm{do}(X_3 = \xi_3)$:

Structural Causal Model $\mathcal{M}_{\mathrm{do}(X_3=\xi_3)}$:

$$
\begin{aligned}
X_1 &= f_1(E_1) & \mathbb{P}^{E_1} &= \dots \\
X_2 &= f_2(E_1, E_2) & \mathbb{P}^{E_2} &= \dots \\
X_3 &= \xi_3 & \mathbb{P}^{E_3} &= \dots \\
X_4 &= f_4(X_1, X_4, E_4) & \mathbb{P}^{E_4} &= \dots \\
X_5 &= f_5(X_3, X_4, E_5) & \mathbb{P}^{E_5} &= \dots
\end{aligned}
$$

Functional graph $\mathcal{G}(\mathcal{M}_{\mathrm{do}(X_3=\xi_3)})$:



Distributions

Definition (Reminder)

A pair of random variables $(\boldsymbol{X}, \boldsymbol{E})$ is a solution of SCM $\mathcal{M}$ if $\mathbb{P}^{\boldsymbol{E}} = \mathbb{P}_{\boldsymbol{\mathcal{E}}}$ and the structural equations $\boldsymbol{X} = \boldsymbol{f}(\boldsymbol{X}, \boldsymbol{E})$ hold a.s..

Definition

We call the set of probability distributions of the solutions $\boldsymbol{X}$ of an SCM $\mathcal{M}$ the observational distributions of $\mathcal{M}$.

Distributions

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Definition

We call the set of probability distributions of the solutions $\boldsymbol{X}$ of an SCM $\mathcal{M}$ the observational distributions of $\mathcal{M}$.

A perfect intervention on $\mathcal{M}$ may change the distributions.

Definition

We call the family of sets of probability distributions of the solutions of $\mathcal{M}_{\mathrm{do}(I,\boldsymbol{\xi}_I)}$ (for $I \subseteq \mathcal{I}$, $\boldsymbol{\xi}_I \subseteq \boldsymbol{\mathcal{X}}_I$) the interventional distributions of $\mathcal{M}$.

Crucial difference with traditional probabilistic models: SCMs simultaneously model all distributions that are obtained under all perfect interventions on a system.

Acyclic SCMs vs. Causal Bayesian Networks

Definition

We call the SCM $\mathcal{M}$ acyclic if $\mathcal{G}(\mathcal{M})$ is acyclic.

Proposition

If $\mathcal{M}$ is acyclic, then:

- *its observational distribution exists and is unique.*
- *all its interventional distributions exist and are unique.*

In that case, we denote the observational density on $\boldsymbol{X}$ by $p_{\mathcal{M}}(\boldsymbol{x})$, and the interventional densities on $\boldsymbol{X}$ by $p_{\mathcal{M}}\big(\boldsymbol{x} \mid \mathrm{do}(\boldsymbol{X}_I = \boldsymbol{\xi}_I)\big)$, following the notation of [Pearl, 2000].

Proposition

If $\mathcal{G}(\mathcal{M})$ is acyclic and does not have bidirected edges, the SCM induces a Causal Bayesian Network. Vice versa, for every Causal Bayesian Network there exists an acyclic, causally sufficient SCM that induces it.

Marginalization (Example)

Can we "integrate out" the details of a subsystem?

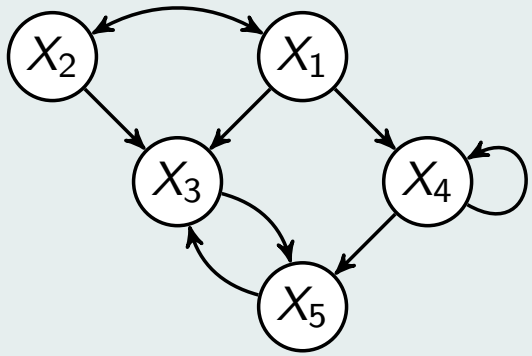
Example

SCM for complete system:

Structural Causal Model $\mathcal{M}$:

$$
\begin{aligned}
X_1 &= f_1(E_1) & \mathbb{P}^{E_1} &= \dots \\
X_2 &= f_2(E_1, E_2) & \mathbb{P}^{E_2} &= \dots \\
X_3 &= f_3(X_1, X_2, X_5, E_3) & \mathbb{P}^{E_3} &= \dots \\
X_4 &= f_4(X_1, X_4, E_4) & \mathbb{P}^{E_4} &= \dots \\
X_5 &= f_5(X_3, X_4, E_5) & \mathbb{P}^{E_5} &= \dots
\end{aligned}
$$

Functional graph $\mathcal{G}(\mathcal{M})$:

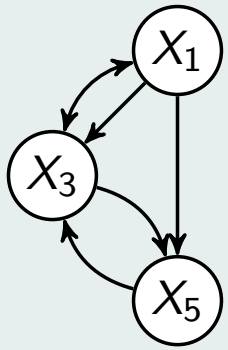


Marginalizing out X_2, X_4:

Marginalization $\mathcal{M}^{\setminus\{2,4\}}$:

$$
\begin{aligned}
X_1 &= f_1(E_1) & \mathbb{P}^{E_1} &= \dots \\
 & & \mathbb{P}^{E_2} &= \dots \\
X_3 &= f_3(X_1, g_2(E_1, E_2), X_5, E_3) & \mathbb{P}^{E_3} &= \dots \\
 & & \mathbb{P}^{E_4} &= \dots \\
X_5 &= f_5(X_3, g_4(X_1, E_4), E_5) & \mathbb{P}^{E_5} &= \dots
\end{aligned}
$$

Functional graph $\mathcal{G}(\mathcal{M}^{\setminus\{2,4\}})$:



Substituting equations

Given an SCM $\mathcal{M}$ and a subset of its endogenous variables $\mathcal{L} \subseteq \mathcal{I}$, with complement $\mathcal{O} := \mathcal{I} \setminus \mathcal{L}$. We can try to "substitute out" the structural equations for $\mathcal{L}$:

$$
\begin{aligned}
& \boldsymbol{X} = \boldsymbol{f}(\boldsymbol{X}, \boldsymbol{E}) \\
\iff & \begin{cases} \boldsymbol{X}_{\mathcal{L}} &= \boldsymbol{f}_{\mathcal{L}}(\boldsymbol{X}_{\mathcal{L}}, \boldsymbol{X}_{\mathcal{O}}, \boldsymbol{E}) \\ \boldsymbol{X}_{\mathcal{O}} &= \boldsymbol{f}_{\mathcal{O}}(\boldsymbol{X}_{\mathcal{L}}, \boldsymbol{X}_{\mathcal{O}}, \boldsymbol{E}) \end{cases} \\
\iff & \begin{cases} \boldsymbol{X}_{\mathcal{L}} &= \boldsymbol{g}_{\mathcal{L}}(\boldsymbol{X}_{\mathcal{O}}, \boldsymbol{E}) \\ \boldsymbol{X}_{\mathcal{O}} &= \boldsymbol{f}_{\mathcal{O}}(\boldsymbol{X}_{\mathcal{L}}, \boldsymbol{X}_{\mathcal{O}}, \boldsymbol{E}) \end{cases} \\
\iff & \begin{cases} \boldsymbol{X}_{\mathcal{L}} &= \boldsymbol{g}_{\mathcal{L}}(\boldsymbol{X}_{\mathcal{O}}, \boldsymbol{E}) \\ \boldsymbol{X}_{\mathcal{O}} &= \boldsymbol{f}_{\mathcal{O}}(\boldsymbol{g}_{\mathcal{L}}(\boldsymbol{X}_{\mathcal{O}}, \boldsymbol{E}), \boldsymbol{X}_{\mathcal{O}}, \boldsymbol{E}) \end{cases}
\end{aligned}
$$

This trick works if the structural equations for $\boldsymbol{X}_{\mathcal{L}}$ have a unique solution for $\boldsymbol{X}_{\mathcal{L}}$ in terms of $\boldsymbol{X}_{\mathcal{O}}$ and $\boldsymbol{E}$ (for acyclic SCMs, this always works).

Marginalization of an SCM

Definition ([Bongers et al., 2018])

If $\mathcal{M} = \langle \boldsymbol{\mathcal{X}}, \boldsymbol{\mathcal{E}}, \boldsymbol{f}, \mathbb{P}_{\boldsymbol{\mathcal{E}}} \rangle$ is uniquely solvable w.r.t. $\mathcal{L} \subseteq \mathcal{I}$, then it has a marginalization $\mathcal{M}^{\setminus \mathcal{L}} = \langle \boldsymbol{\mathcal{X}}_{\mathcal{I} \setminus \mathcal{L}}, \boldsymbol{\mathcal{E}}, \boldsymbol{f}^{\setminus \mathcal{L}}, \mathbb{P}_{\boldsymbol{\mathcal{E}}} \rangle$, where the marginal causal mechanism $\boldsymbol{f}^{\setminus \mathcal{L}}$ is obtained by substituting the solution function $\boldsymbol{g}_{\mathcal{L}}$ for $\boldsymbol{X}_{\mathcal{L}}$ in terms of $\boldsymbol{X}_{\mathcal{O}}$ (with $\mathcal{O} := \mathcal{I} \setminus \mathcal{L}$) and $\boldsymbol{E}$ into the causal mechanism $\boldsymbol{f}_{\mathcal{O}}$:

$$\boldsymbol{f}^{\setminus \mathcal{L}}(\boldsymbol{x}_{\mathcal{O}}, \boldsymbol{e}) := \boldsymbol{f}_{\mathcal{O}}\big(\boldsymbol{g}_{\mathcal{L}}(\boldsymbol{x}_{\mathrm{pa}(\mathcal{L}) \setminus \mathcal{L}}, \boldsymbol{e}_{\mathrm{pa}(\mathcal{L})}), \boldsymbol{x}_{\mathcal{O}}, \boldsymbol{e}\big).$$

The marginalization preserves the causal semantics (restricted to the remaining part of the system, $\mathcal{I} \setminus \mathcal{L}$):

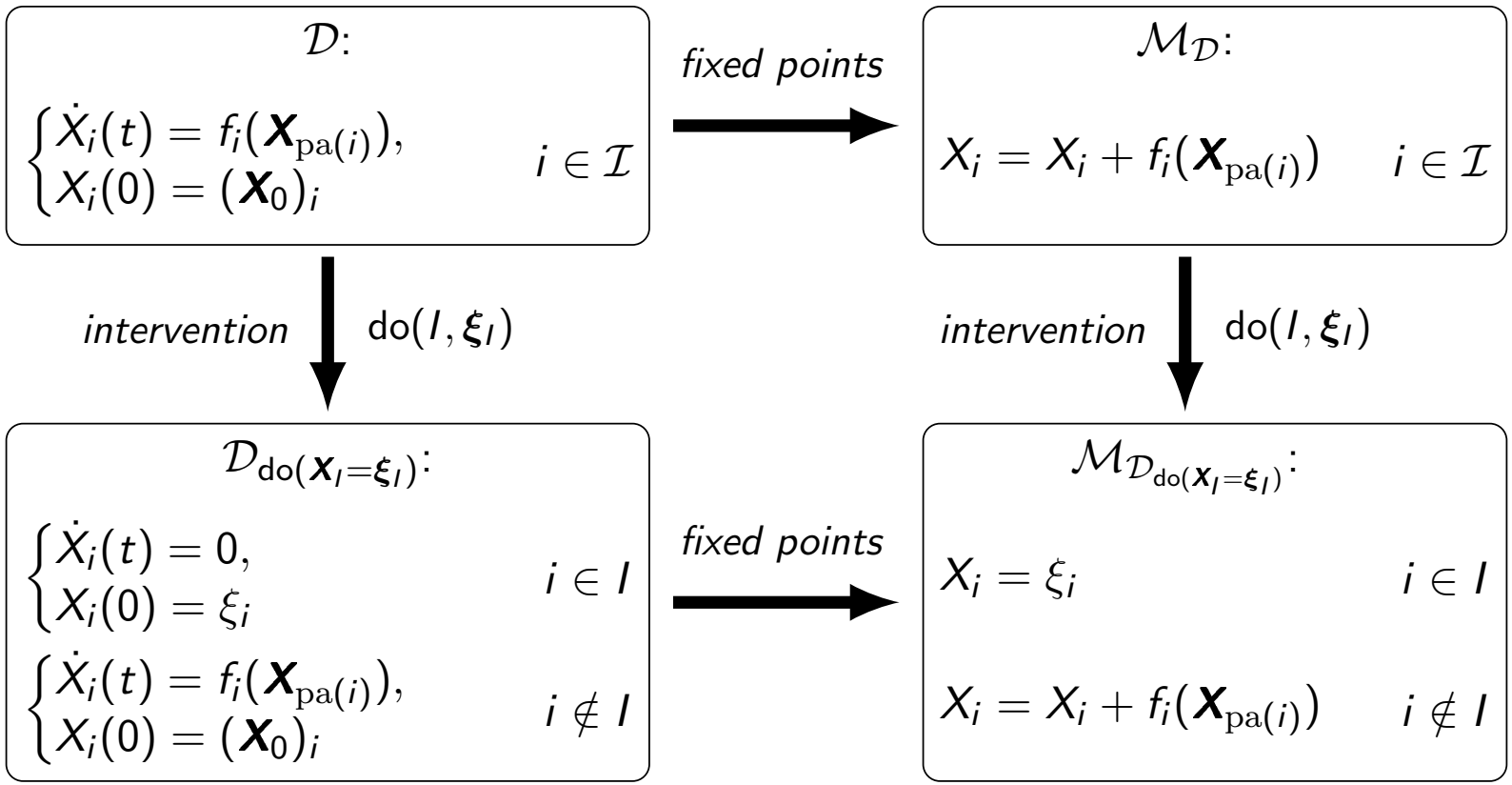
Theorem ([Bongers et al., 2018])

The marginalization $\mathcal{M}^{\setminus \mathcal{L}}$ is interventionally equivalent to $\mathcal{M}$ w.r.t. $\mathcal{I} \setminus \mathcal{L}$. In other words, for any perfect intervention on a subset of $\mathcal{I} \setminus \mathcal{L}$, $\mathcal{M}^{\setminus \mathcal{L}}$ and $\mathcal{M}$ admit the same solutions (marginalized onto $\boldsymbol{\mathcal{X}}_{\mathcal{I} \setminus \mathcal{L}}$).

Modeling (Random) ODE fixed points with an SCM

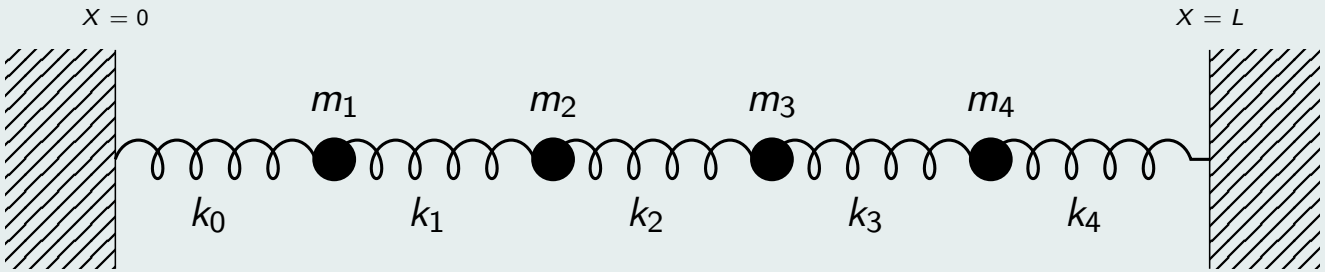
Theorem ([Mooij et al., 2013, Bongers and Mooij, 2018])

A random ODE describing a dynamical system induces an SCM that models its equilibrium states, and how these change under perfect interventions.



From ODE to SCM: Example 1

Example (Damped coupled harmonic oscillators)



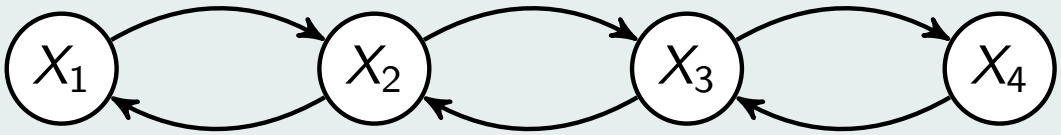
- ODE $\mathcal{D}$:

$$\ddot{X}_i = \frac{k_i}{m_i}(X_{i+1} - X_i - l_i) - \frac{k_{i-1}}{m_i}(X_i - X_{i-1} - l_{i-1}) - b_i \dot{X}_i$$

- Structural Equations of induced SCM $\mathcal{M}_{\mathcal{D}}$:

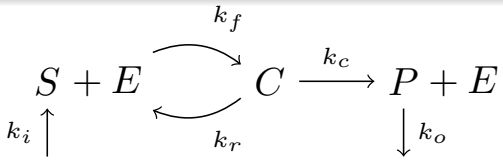
$$X_i = \frac{k_i(X_{i+1} - l_i) + k_{i-1}(X_{i-1} + l_{i-1})}{k_i + k_{i+1}}$$

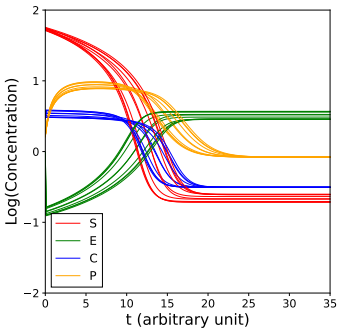
- Functional graph of induced SCM $\mathcal{G}(\mathcal{M}_{\mathcal{D}})$:



From ODE to SCM: Example 2

Enzyme reaction:





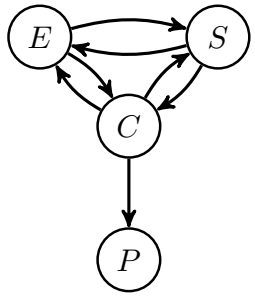
Random differential equations:

$$\frac{d}{dt}S = k_i - k_f ES + k_r C$$
$$\frac{d}{dt}E = -k_f ES + (k_r + k_c)C$$
$$\frac{d}{dt}C = k_f ES - (k_r + k_c)C$$
$$\frac{d}{dt}P = k_c C - k_o P$$

$\xrightarrow{t \to \infty}$

Structural causal model:

$$S = k_i k_f^{-1} E^{-1} - k_r k_f^{-1} E^{-1} C$$
$$E = k_f^{-1}(k_r + k_c) S^{-1} C$$
$$C = k_f (k_r + k_c)^{-1} ES$$
$$P = k_c k_o^{-1} C$$



$\downarrow \mathrm{do}(E = \eta)$

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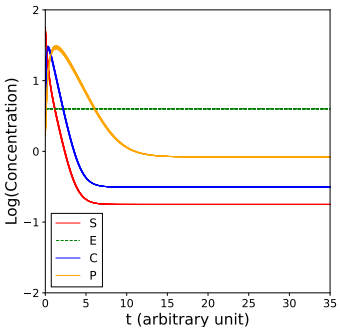


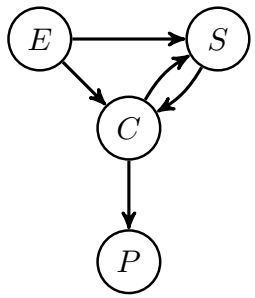
Intervened RDE:

$$\frac{d}{dt}S = k_i - k_f ES + k_r C$$
$$\frac{d}{dt}E = \eta$$
$$\frac{d}{dt}C = k_f ES - (k_r + k_c)C$$
$$\frac{d}{dt}P = k_c C - k_o P$$

$\xrightarrow{t \to \infty}$

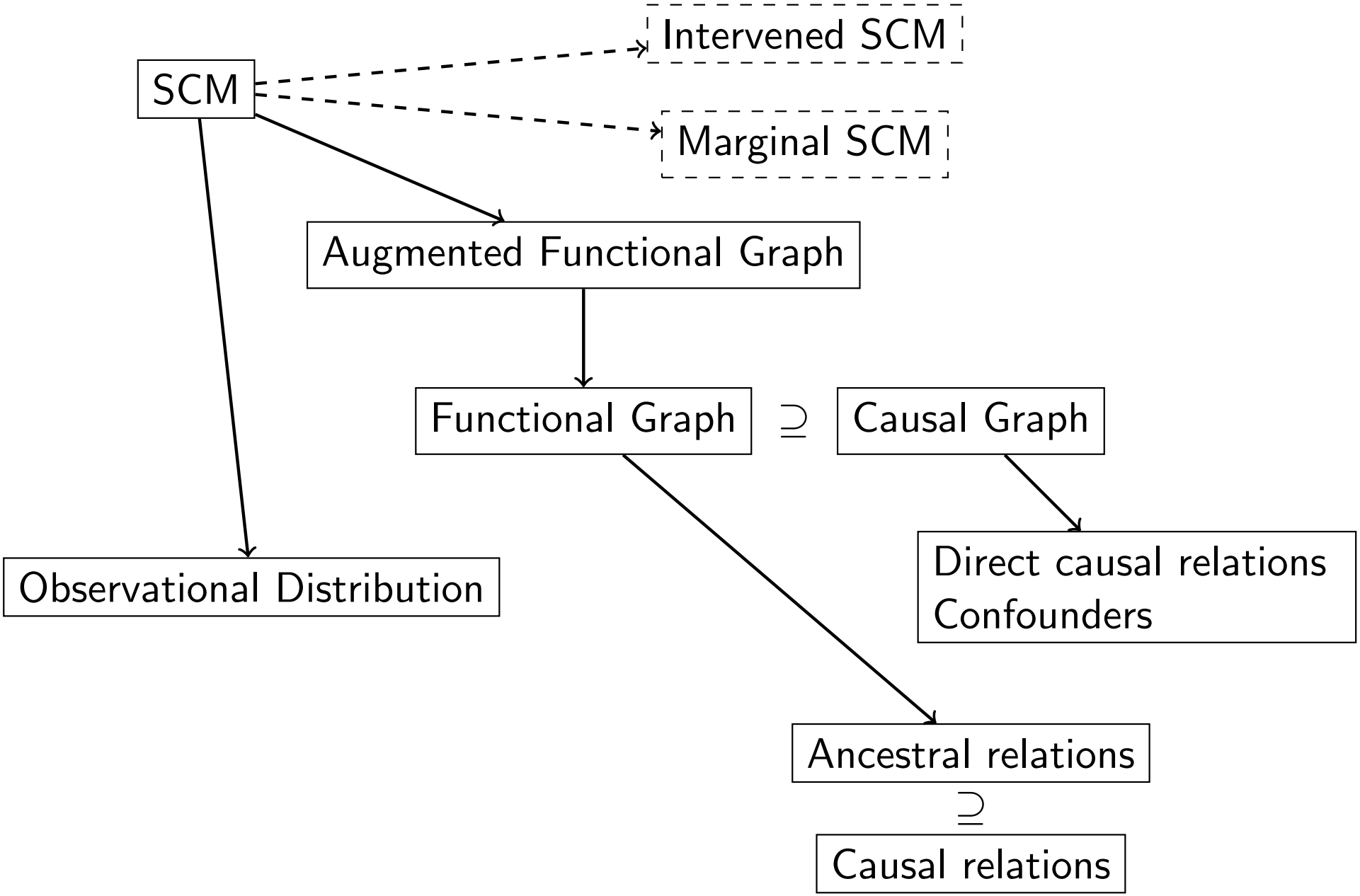
Intervened SCM:

$$S = k_i k_f^{-1} E^{-1} - k_r k_f^{-1} E^{-1} C$$
$$E = \eta$$
$$C = k_f (k_r + k_c)^{-1} ES$$
$$P = k_c k_o^{-1} C$$



More generally, any chemical reaction can be modeled as an SCM at equilibrium. (Note: the SCM is in general *underspecified*, i.e., it does not retain all information about the equilibrium states of the dynamical system [Blom & Mooij, 2018]).

Representations of (acyclic) SCMs [Bongers et al., 2018]



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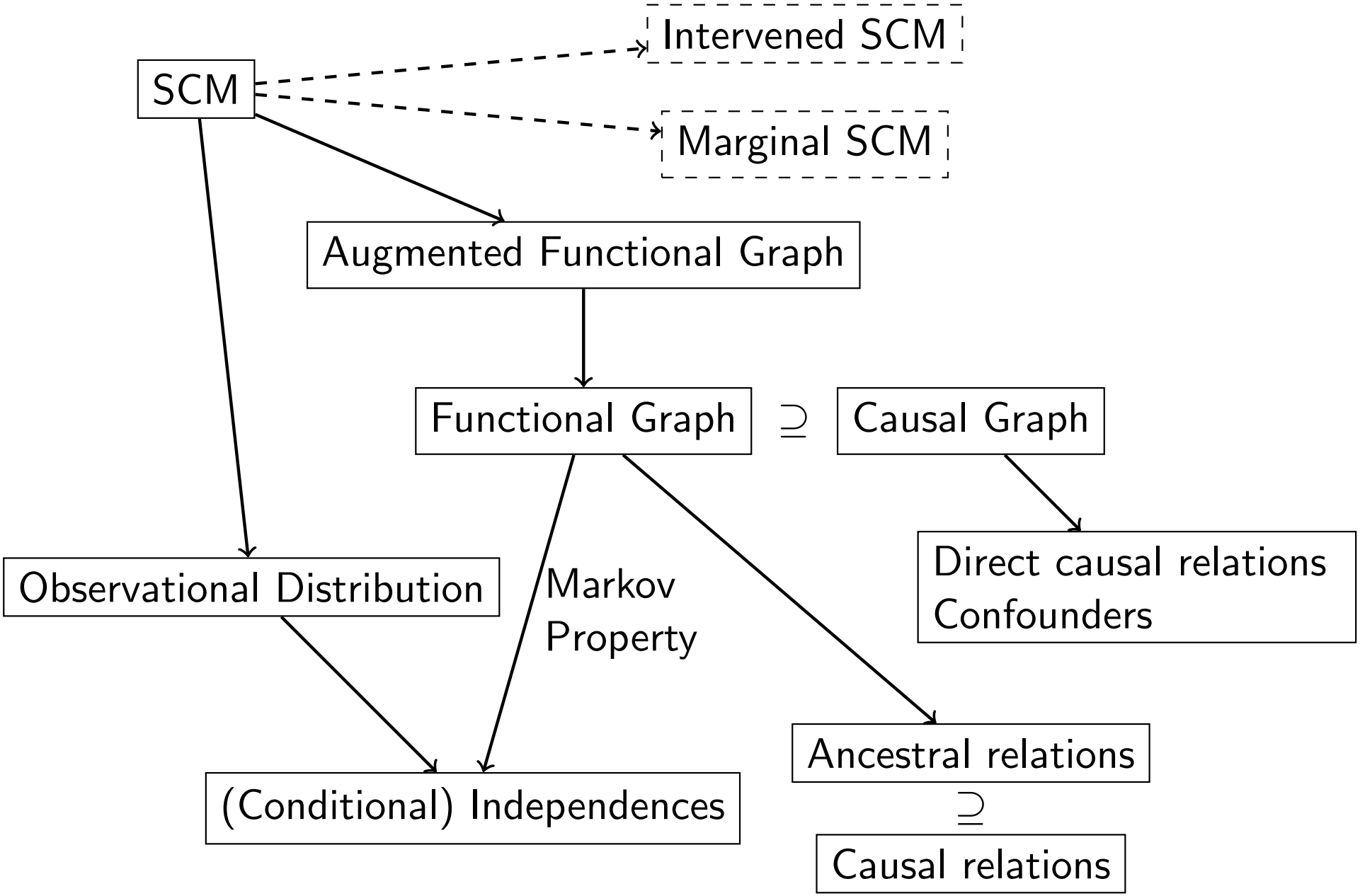

Contents

(Conditional) independences

Definition (Independence)

Given two random variables X, Y, we write $X \perp\!\!\!\perp Y$ and say that X is independent of Y if

$$p(x, y) = p(x)p(y).$$

Intuitively, X is independent of Y if we do not learn anything about X when told the value of Y (or vice versa).

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Intuitively, X is independent of Y if we do not learn anything about X when told the value of Y (or vice versa).

Definition (Conditional Independence)

Given a third random variable Z, we write $X \perp\!\!\!\perp Y \,|\, Z$ and say that X is (conditionally) independent from Y, given Z, if

$$p(x, y \,|\, Z = z) = p(x \,|\, Z = z)p(y \,|\, Z = z).$$

Intuitively, X is independent of Y if, given the value of Z, we do not learn anything new about X when told the value of Y.

(Directed) Paths

Definition (Paths, Ancestors)

Let $\mathcal{G}$ be a directed mixed graph.

- A path q is a sequence of adjacent edges in which no node occurs more than once.
- A directed path is of the form $i_1 \to i_2 \to \cdots \to i_k$.
- If there is a directed path from X to Y, X is called an ancestor of Y.
- The ancestors of Y are denoted $\mathrm{an}_{\mathcal{G}}(Y)$, and include Y.

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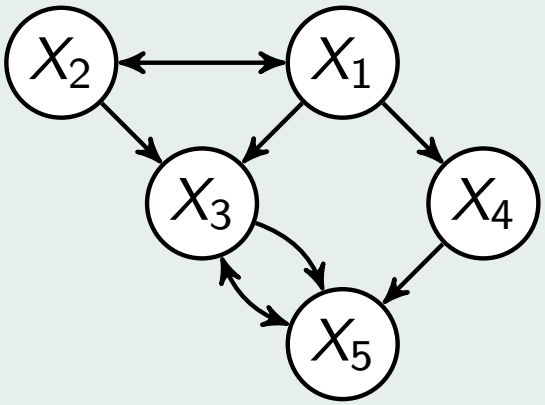
Example



$X_1 \to X_3 \leftarrow X_1$ is not a path.
$X_1 \leftrightarrow X_2 \to X_3$ is a path.

$X_1 \to X_4 \to X_5$ is a directed path.
$X_4 \to X_5 \leftarrow X_3$ is not a directed path.

The ancestors of X_3 are $\{X_1, X_2, X_3\}$.

Colliders and non-colliders

Definition (Colliders)

Let $\mathcal{G}$ be a directed mixed graph, and q a path on $\mathcal{G}$.

- A collider on q is a (non-endpoint) node X on q with precisely two arrowheads pointing towards X on the adjacent edges:

$$\rightarrow X \leftarrow, \quad \rightarrow X \leftrightarrow, \quad \leftrightarrow X \leftarrow, \quad \leftrightarrow X \leftrightarrow$$

- A non-collider on q is any node on the path which is not a collider.

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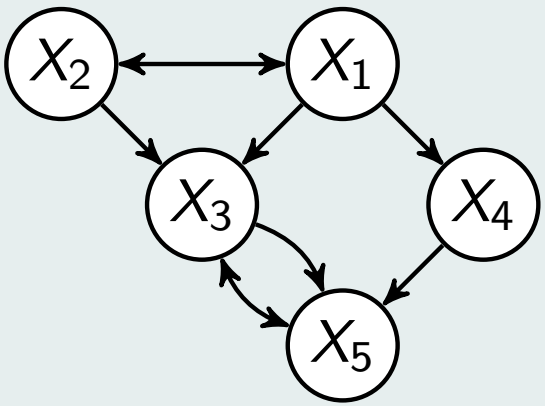
Example



The path $X_3 \rightarrow X_5 \leftarrow X_4$ contains a collider X_5.
The path $X_1 \leftrightarrow X_2 \rightarrow X_3$ contains no collider.
X_5 is a non-collider on $X_5 \leftrightarrow X_3 \leftarrow X_1$.

Blocked paths

Definition

Let $\mathcal{G}$ be a directed mixed graph. Given a path q on $\mathcal{G}$, and a set of nodes $\boldsymbol{S}$, we say that $\boldsymbol{S}$ blocks q if q contains

- a non-collider which is in $\boldsymbol{S}$, or
- a collider which is *not* an ancestor of $\boldsymbol{S}$.

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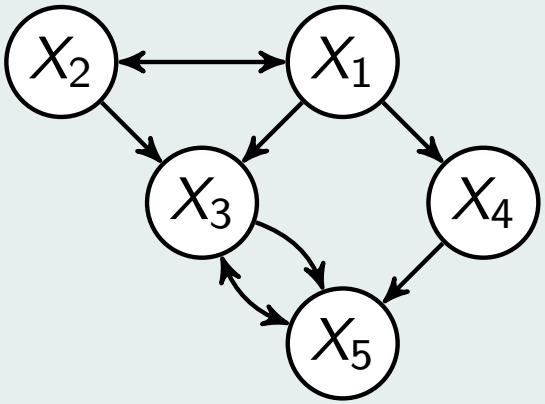
Example



$X_3 \to X_5 \leftarrow X_4$ is blocked by $\emptyset$.
$X_3 \to X_5 \leftarrow X_4$ is blocked by $\{X_1\}$.
$X_3 \to X_5 \leftarrow X_4$ is not blocked by $\{X_5\}$.

$X_3 \leftarrow X_2 \leftrightarrow X_1 \to X_4$ is blocked by $\{X_1\}$.
$X_3 \leftarrow X_2 \leftrightarrow X_1 \to X_4$ is not blocked by $\{X_5\}$.

d-separation

Definition (d-separation)

Let $\mathcal{G}$ be a directed mixed graph. For three sets $\boldsymbol{X}, \boldsymbol{Y}, \boldsymbol{Z}$ of nodes in $\mathcal{G}$, we say that $\boldsymbol{X}$ and $\boldsymbol{Y}$ are d-separated by $\boldsymbol{Z}$ iff all paths between a node in $\boldsymbol{X}$ and a node in $\boldsymbol{Y}$ are blocked by $\boldsymbol{Z}$, and write $\boldsymbol{X} \perp_{\mathcal{G}} \boldsymbol{Y} \mid \boldsymbol{Z}$.

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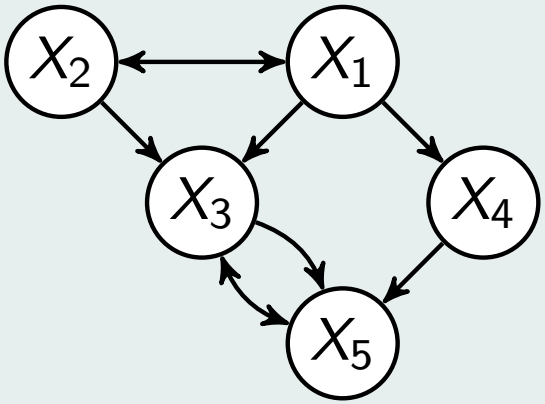
Example



X_3 and X_4 are d-separated by $\{X_1\}$.
X_3 and X_4 are d-separated by $\{X_1, X_2\}$.
X_3 and X_4 are not d-separated by $\emptyset$.
X_3 and X_4 are not d-separated by $\{X_1, X_5\}$.

Exercise 2

Please make Exercise 2

Acyclic Global Markov Property

Theorem

For an acyclic SCM, the following Global Markov Property holds:

$$\boldsymbol{X}, \boldsymbol{Y} \underset{\mathcal{G}(\mathcal{M})}{\perp} \boldsymbol{Z} \qquad \Longrightarrow \qquad \boldsymbol{X} \underset{p_{\mathcal{M}}}{\perp\!\!\!\perp} \boldsymbol{Y} \,|\, \boldsymbol{Z}$$

for all subsets $\boldsymbol{X}, \boldsymbol{Y}, \boldsymbol{Z}$ *of nodes.*

In words: every d-separation in the functional graph $\mathcal{G}(\mathcal{M})$ of $\mathcal{M}$ implies a (conditional) independence in the (unique) observational distribution associated to $\mathcal{M}$.

For *cyclic* SCMs, the notion of d-separation is too strong in general. A weaker notion called σ-separation has to be used instead [Forré and Mooij, 2017]. Under additional solvability conditions, a global Markov condition using σ-separation can be shown to hold.

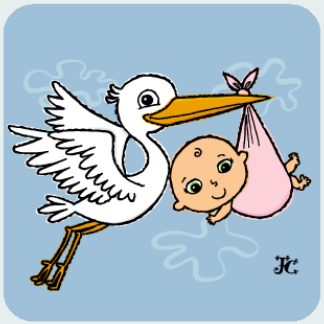
Reichenbach's Principle

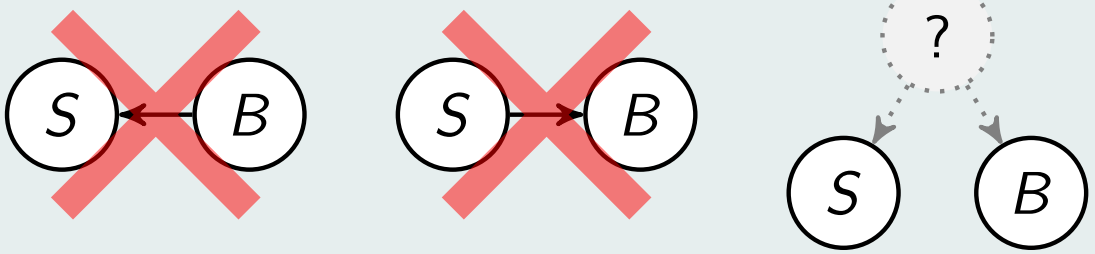
Reichenbach's Principle of Common Cause

The dependence $X \not\perp\!\!\!\perp Y$ implies that $X \to Y$, $Y \to X$, or $X \leftrightarrow Y$ (or any combination of these three).

Example

- Significant correlation ($p = 0.008$) between human birth rate and number of stork populations in European countries [Matthews, 2000]
- Most people nowadays do not believe that storks deliver babies (nor that babies deliver storks)
- There must be some confounder explaining the correlation

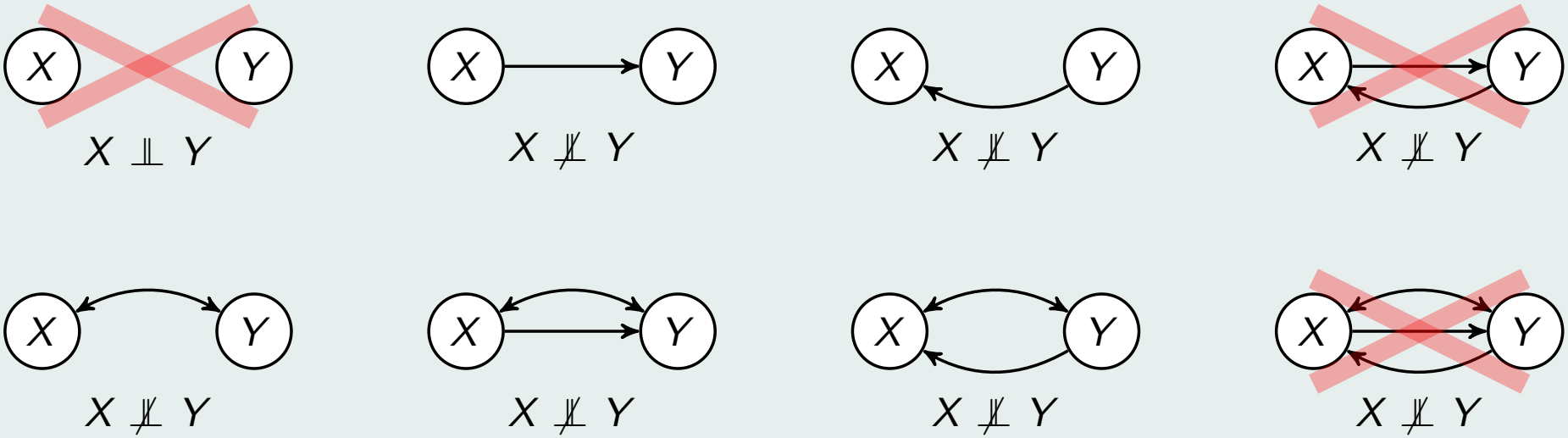




Proof of Reichenbach's Principle

Assuming that $p(X, Y)$ is generated by an acyclic SCM, we can easily prove Reichenbach's Principle by applying the Global Markov property:

Proof



(The proof can be extended to include the cyclic case)

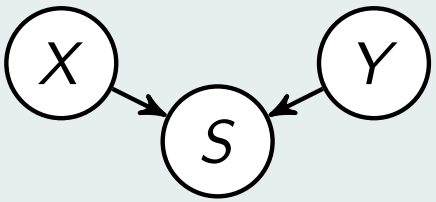
Selection Bias

Reichenbach's Principle may fail in case of *selection bias*.

Definition

If a data set is obtained by only including samples conditional on some event, selection bias may be introduced.

Example



X: the battery is charged
Y: the start engine is operational
S: the car starts

- A car mechanic (who only observes cars for which $S = 0$) will observe a dependence between X and Y: $X \not\perp\!\!\!\perp Y \mid S$.
- When the car mechanic invokes Reichenbach's Principle without realizing that he is selecting on the value of S (maybe S is a latent variable), a wrong conclusion will be drawn.

Faithfulness Assumption

Let $\mathcal{M}$ be an acyclic SCM.

We have seen that the *Global Markov Property* holds:

$$\boldsymbol{X}, \boldsymbol{Y} \underset{\mathcal{G}(\mathcal{M})}{\perp} \boldsymbol{Z} \qquad \Longrightarrow \qquad \boldsymbol{X} \underset{p_{\mathcal{M}}}{\perp\!\!\!\perp} \boldsymbol{Y} \,|\, \boldsymbol{Z}$$

for all subsets $\boldsymbol{X}, \boldsymbol{Y}, \boldsymbol{Z}$ of nodes.

Definition (Faithfulness Assumption)

For all subsets $\boldsymbol{X}, \boldsymbol{Y}, \boldsymbol{Z}$ of nodes,

$$\boldsymbol{X}, \boldsymbol{Y} \underset{\mathcal{G}(\mathcal{M})}{\perp} \boldsymbol{Z} \qquad \Longleftarrow \qquad \boldsymbol{X} \underset{p_{\mathcal{M}}}{\perp\!\!\!\perp} \boldsymbol{Y} \,|\, \boldsymbol{Z}$$

Note: Faithfulness holds generically, i.e., up to measure-zero sets of parameters [Meek, 1995]. In other words, SCM parameters need to be *carefully tuned* in order to violate the faithfulness assumption.

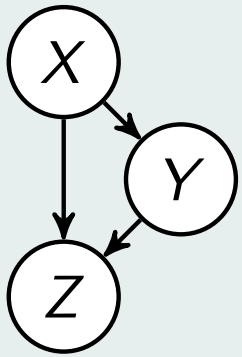
Faithfulness Violations

Faithfulness violations may occur e.g. in case of parameter cancellations or deterministic relations.

Example (Parameter cancellation)

Consider an SCM $\mathcal{M}$:

$$X = E_X$$
$$Y = X + E_Y$$
$$Z = X - Y + E_Z$$

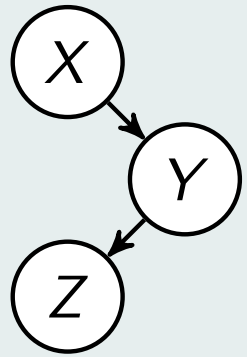


Then:
$Z \perp\!\!\!\perp_{p_{\mathcal{M}}} X$ but $Z \not\perp\!\!\!\perp_{\mathcal{G}(\mathcal{M})} X$.

Example (Deterministic relation)

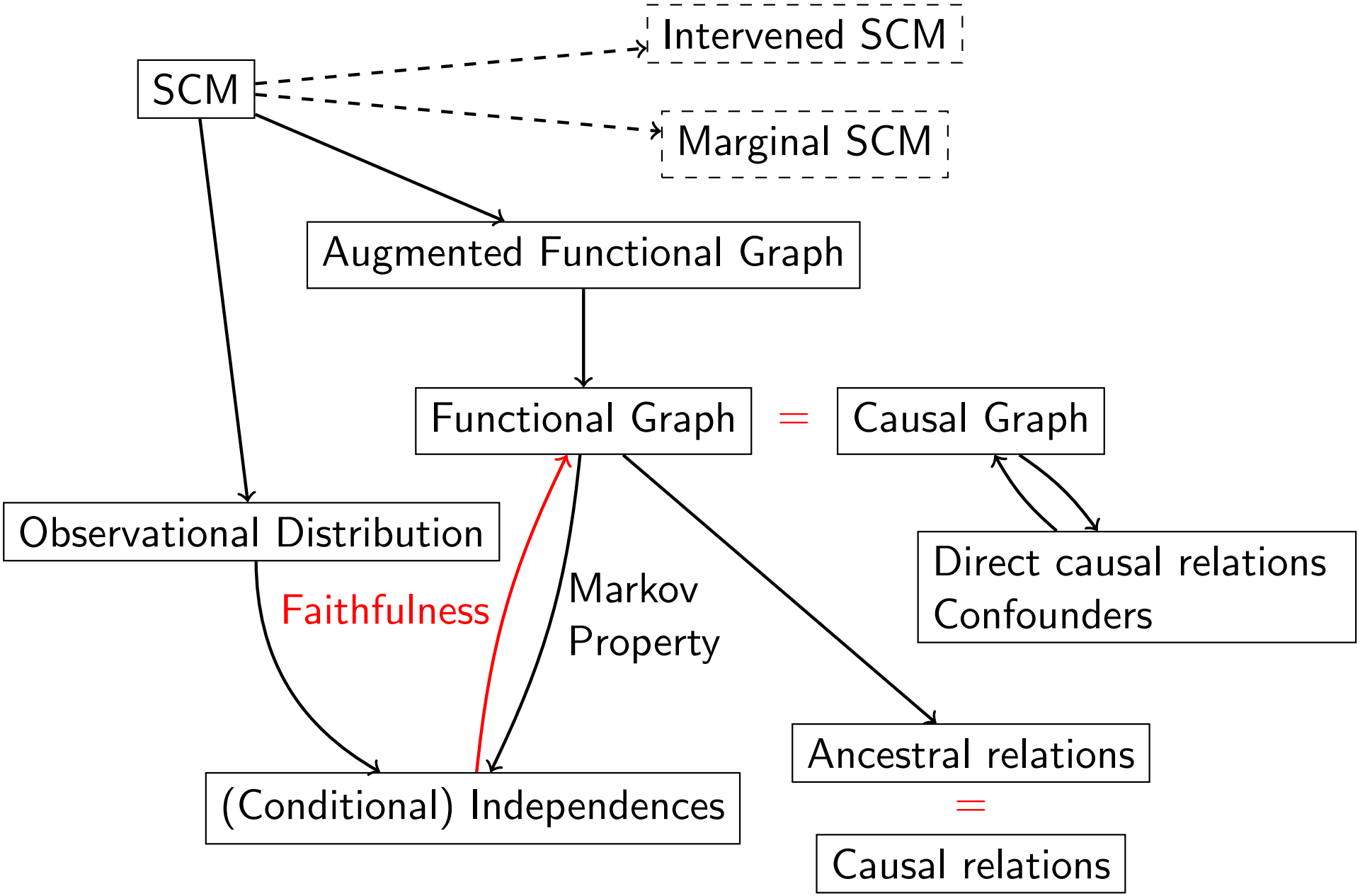
Consider an SCM $\mathcal{M}$:

$$X = E_X$$
$$Y = X$$
$$Z = Y + E_Z$$



Then:
$Z \perp\!\!\!\perp_{p_{\mathcal{M}}} Y \,|\, X$ but $Z \not\perp\!\!\!\perp_{\mathcal{G}(\mathcal{M})} Y \,|\, X$.

Representations of acyclic, faithful SCMs



Contents

Causal Inference: Predicting Causal Effects

One important task ("*causal inference*") is the prediction of causal effects.

Definition

The causal effect of X on Y is defined as $p\big(y \mid \mathrm{do}(X = x)\big)$.

Special cases:

- X binary: $\mathbb{E}\big(Y \mid \mathrm{do}(X = 1)\big) - \mathbb{E}\big(Y \mid \mathrm{do}(X = 0)\big)$
- X, Y linearly related: $\frac{\partial}{\partial x}\mathbb{E}\big(Y \mid \mathrm{do}(X = x)\big)$

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- X, Y linearly related: $\frac{\partial}{\partial x}\mathbb{E}\big(Y \mid \mathrm{do}(X = x)\big)$

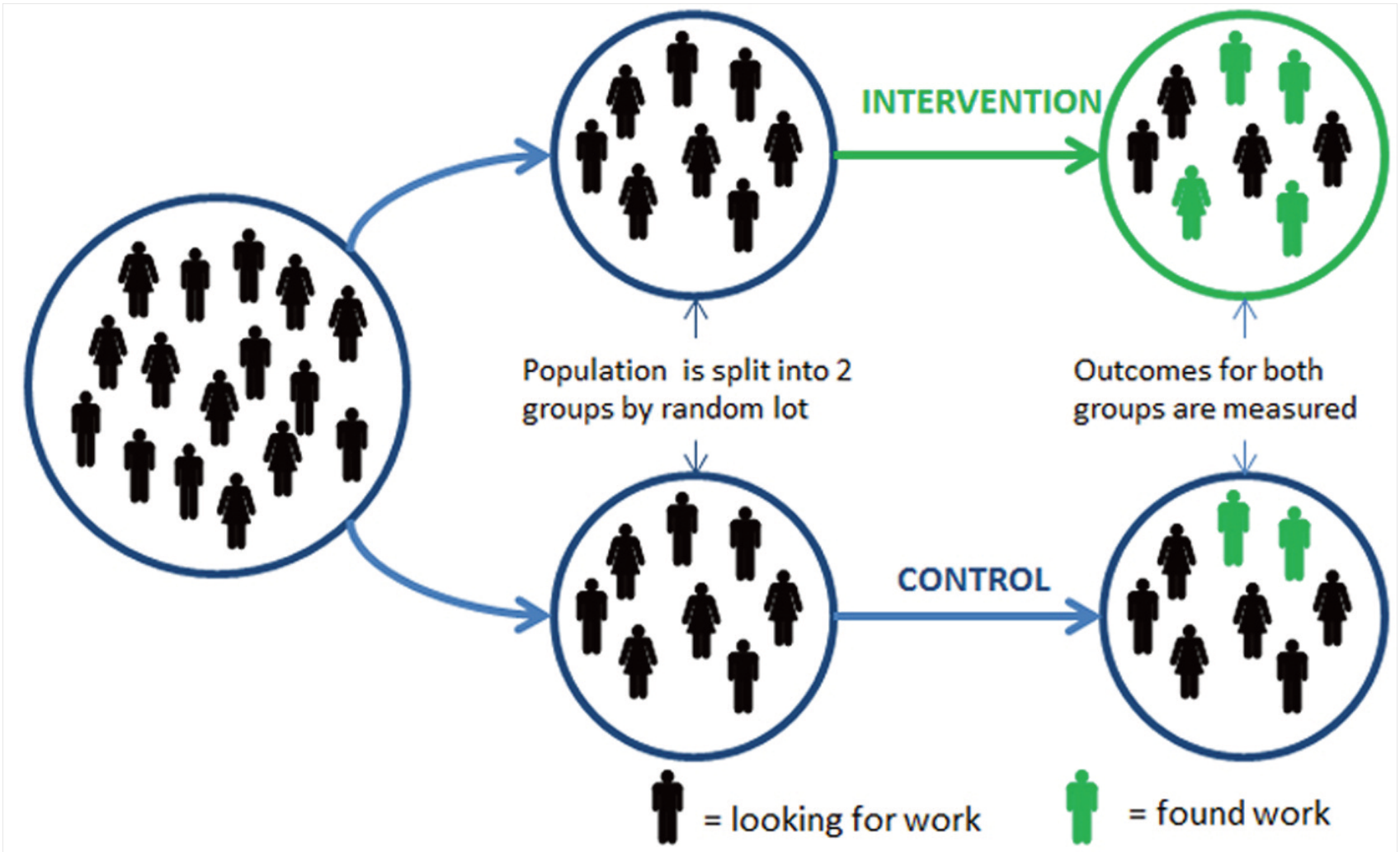
Note: In general, since $p\big(y \mid \mathrm{do}(X = x)\big) \neq p(y \mid X = x)$, we cannot use standard supervised learning (regression, classification) for this task.

Two approaches can be used:

- Experimentation (Randomized Controlled Trials, A/B-testing)
- Apply the Back-door Criterion (if causal graph is known)

Causal discovery by experimentation

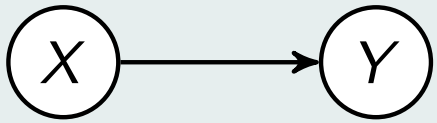
Experimentation (e.g., Randomized Controlled Trials, A/B-testing, . . .) provides the gold standard for causal effect estimation.



Identifiability: Example

If we cannot do experiments... Can we express $p\big(y \mid \mathrm{do}(X = x)\big)$ in terms of the observational distribution?

Example



$$p\big(y \mid \mathrm{do}(X = x)\big)$$

$$=$$

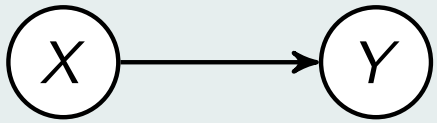
$$p(y \mid X = x)$$

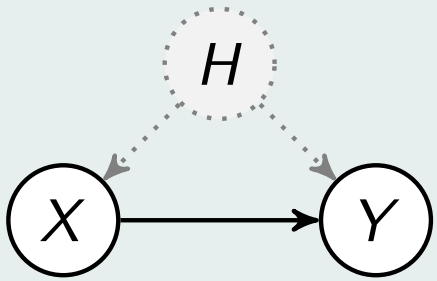
Yes!

Identifiability: Example

If we cannot do experiments... Can we express $p\big(y \mid \mathrm{do}(X = x)\big)$ in terms of the observational distribution?

Example





$$p\big(y \mid \mathrm{do}(X = x)\big) \qquad p\big(y \mid \mathrm{do}(X = x)\big) = \int p(h)p(y \mid x, h)\, dh$$

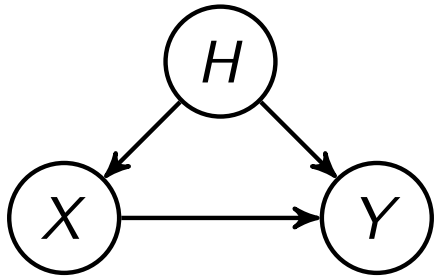
$$= \qquad \neq$$

$$p(y \mid X = x) \qquad p(y \mid X = x) = \int p(h \mid x)p(y \mid x, h)\, dh$$

Yes! No!

Adjustment for covariates

We have seen that for the following causal graph,



adjusting for the confounder H, yields the causal effect of X on Y:

$$\int p(h)p(y \mid x, h)\, dh = p\big(y \mid \mathrm{do}(X = x)\big)$$

More generally, given a causal graph: which covariates $\boldsymbol{H}$ could we adjust for in order to express the causal effect of X on Y in terms of the observational distribution?

A sufficient condition is given by the Back-door Criterion.

The Back-Door Criterion

Theorem (Back-Door Criterion [Pearl, 2000])

For an acyclic SCM, nodes X, Y and set of nodes $\boldsymbol{H}$: if

1. $X, Y \notin \boldsymbol{H}$;
2. X *is not an ancestor of any node in* $\boldsymbol{H}$ *in* $\mathcal{G}(\mathcal{M})$;
3. $\boldsymbol{H}$ *blocks all back-door paths* $X \leftarrow \ldots Y$ *and* $X \leftrightarrow \ldots Y$ *in* $\mathcal{G}(\mathcal{M})$ *(i.e., all paths between* X *and* Y *that start with an arrowhead at* X*).*

then the causal effect of X *on* Y *can be obtained by adjusting for* H*:*

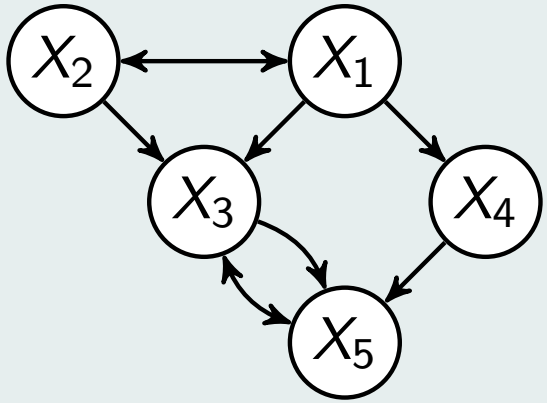
$$p\big(y \,|\, \mathrm{do}(X = x)\big) = \int p(y \,|\, x, \boldsymbol{h}) p(\boldsymbol{h}) \, d\boldsymbol{h} \left(= \sum_{\boldsymbol{h}} p(y \,|\, x, \boldsymbol{h}) p(\boldsymbol{h}) \right).$$

For the special case $\boldsymbol{H} = \emptyset$*, this should be read as:*

$$p\big(y \,|\, \mathrm{do}(X = x)\big) = p(y \,|\, x).$$

The Back-door Criterion: Example

Example



The sets of variables that are admissible for adjustment to get the causal effect of X_2 on X_5 are: $\{X_1\}$, $\{X_1, X_4\}$. Therefore:

$$p(x_5 \mid \mathrm{do}(X_2 = x_2)) = \int p(x_5 \mid x_1, x_2) p(x_1)\, dx_1$$
$$= \int p(x_5 \mid x_1, x_2, x_4) p(x_1, x_4)\, dx_1\, dx_4$$

Some sets of variables that are *not* admissible for adjustment to get the causal effect of X_2 on X_5 are: $\{X_3\}$, $\{X_1, X_3\}$.

Exercise 3

Please make Exercise 3

Simpson's Paradox

Remember Simpson's paradox:

Example (Simpson's paradox)

We collect electronic patient records to investigate the effectiveness of a new drug against a certain disease. We find that:

1. The probability of recovery is higher for patients that took the drug:

$$p(\text{recovery} \mid \text{drug}) > p(\text{recovery} \mid \text{no drug})$$

2. For **both male and female** patients, the relation is **opposite**:

$$p(\text{recovery} \mid \text{drug}, \text{male}) < p(\text{recovery} \mid \text{no drug}, \text{male})$$

$$p(\text{recovery} \mid \text{drug}, \text{female}) < p(\text{recovery} \mid \text{no drug}, \text{female})$$

Does the drug cause recovery? I.e., would *you* use this drug if you are ill?

The answer depends on the causal relationships between the variables!

Resolving Simpson's paradox

The crux to resolving Simpson's paradox is to realize:

Seeing $\neq$ doing

- $p(R = 1 \mid D = 1)$: the probability that somebody recovers, given the observation that the person took the drug.
- $p\big(R = 1 \mid \text{do}(D = 1)\big)$: the probability that somebody recovers, if we *force* the person to take the drug.

Simpson's paradox only manifests itself if we misinterpret correlation as causation by identifying $p(r \mid D = d)$ with $p\big(r \mid \text{do}(D = d)\big)$.

We should prescribe the drug if

$$p\big(R = 1 \mid \text{do}(D = 1)\big) > p\big(R = 1 \mid \text{do(D=0)}\big).$$

How to find the causal effect of the drug on recovery?

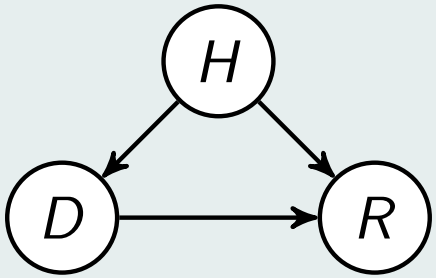
1. Randomized Controlled Trials
2. Back-Door Criterion (requires knowledge of causal graph)

Exercise 4

Please make Exercise 4

Back-Door Criterion for Simpson's paradox

Example (Scenario 1)



R: Recovery
D: Took drug
H: Gender

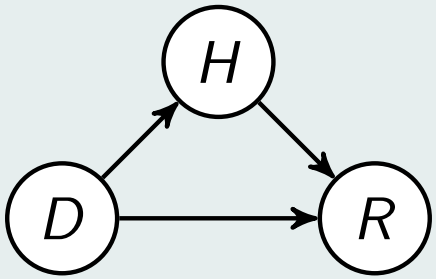
- There is one back-door path: $D \leftarrow H \rightarrow R$, which is blocked by $\{H\}$.
- D is not an ancestor of H.
- Therefore, adjust for $\{H\}$ to obtain causal effect of drug on recovery:

$$p\big(r \mid \mathrm{do}(D = d)\big) = \sum_h p(r \mid D = d, H = h)p(h)$$

- So in scenario I, you should **not** take the drug: for both males and females, taking the drug lowers the probability of recovery.

Back-Door Criterion for Simpson's paradox

Example (Scenario 2)



R: Recovery
D: Took drug
H: Gender

- There are no back-door paths.
- D is an ancestor of H.
- Do **not** adjust for $\{H\}$ to obtain causal effect of drug on recovery:

$$p\big(r \mid \mathrm{do}(D = d)\big) = p(r \mid D = d)$$

- So in scenario II, you **should** take the drug: in the general population, taking the drug increases the probability of recovery.

(If you think gender-changing drugs are unlikely, replace "gender" by "high/low blood pressure", for example).

Mutilated graphs

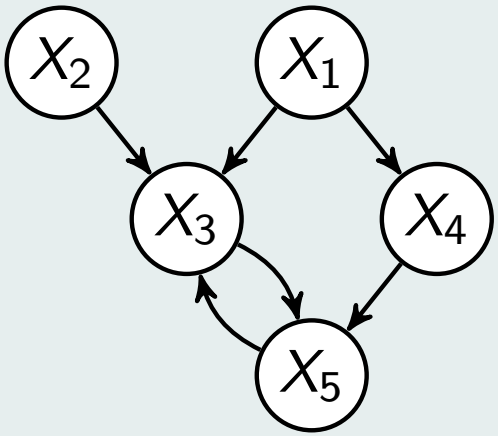
Definition

Given a DMG $\mathcal{G}$ and a subset $\boldsymbol{X}$ of nodes in $\mathcal{G}$, we define

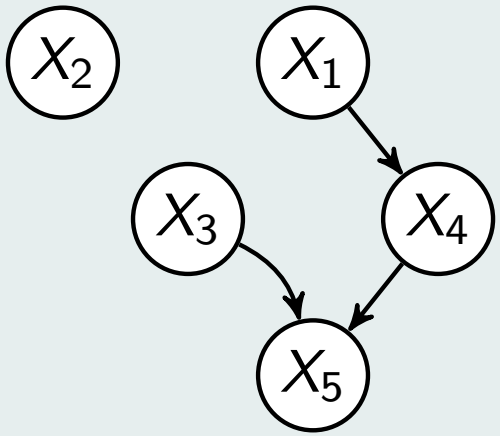
- $\mathcal{G}_{\overline{\boldsymbol{X}}}$ to be $\mathcal{G}$ without the incoming edges on nodes in $\boldsymbol{X}$;
- $\mathcal{G}_{\underline{\boldsymbol{X}}}$ to be $\mathcal{G}$ without the outgoing edges from nodes in $\boldsymbol{X}$.

Example

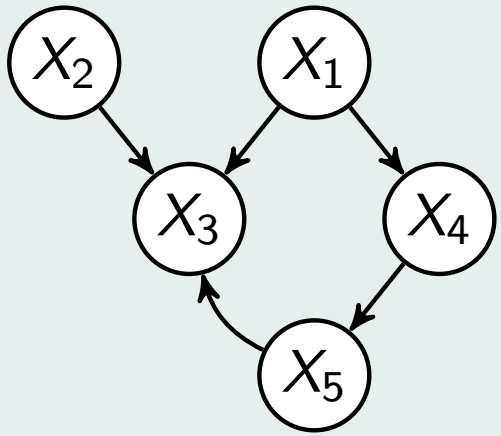
$\mathcal{G}$:



$\mathcal{G}_{\overline{X_3}}$:



$\mathcal{G}_{\underline{X_3}}$:



Do-calculus [Pearl, 2000]

Pearl formulated three rules (the "**do-calculus**") that can be used in addition to the usual rules for probabilistic reasoning:

1. **Ignoring observations**:

$$p(\boldsymbol{y} \mid \mathrm{do}(\boldsymbol{x}), \boldsymbol{w}, \boldsymbol{z}) = p(\boldsymbol{y} \mid \mathrm{do}(\boldsymbol{x}), \boldsymbol{w}) \qquad \text{if } \boldsymbol{Y} \underset{\mathcal{G}_{\overline{\boldsymbol{X}}}}{\perp} \boldsymbol{Z} \mid \boldsymbol{X}, \boldsymbol{W}$$

2. **Action/observation exchange**:

$$p(\boldsymbol{y} \mid \mathrm{do}(\boldsymbol{x}), \mathrm{do}(\boldsymbol{z}), \boldsymbol{w}) = p(\boldsymbol{y} \mid \mathrm{do}(\boldsymbol{x}), \boldsymbol{z}, \boldsymbol{w}) \qquad \text{if } \boldsymbol{Y} \underset{\mathcal{G}_{\overline{\boldsymbol{X}},\underline{\boldsymbol{Z}}}}{\perp} \boldsymbol{Z} \mid \boldsymbol{X}, \boldsymbol{W}$$

3. **Ignoring actions**:

$$p(\boldsymbol{y} \mid \mathrm{do}(\boldsymbol{x}), \mathrm{do}(\boldsymbol{z}), \boldsymbol{w}) = p(\boldsymbol{y} \mid \mathrm{do}(\boldsymbol{x}), \boldsymbol{w}) \qquad \text{if } \boldsymbol{Y} \underset{\mathcal{G}_{\overline{\boldsymbol{X}},\overline{\boldsymbol{Z(W)}}}}{\perp} \boldsymbol{Z} \mid \boldsymbol{X}, \boldsymbol{W}$$

   where $\boldsymbol{Z(W)} = \boldsymbol{Z} \setminus An_{\mathcal{G}_{\overline{\boldsymbol{X}}}}(\boldsymbol{W})$.

The do-calculus allows us to reason with (probabilistic) causal statements, given (partial) knowledge of the causal structure. These rules are more powerful than the Back-door Criterion for causal prediction purposes.

Contents

Causal Discovery

We have seen how to perform causal reasoning, given the causal model. But how do we get the causal model in the first place?

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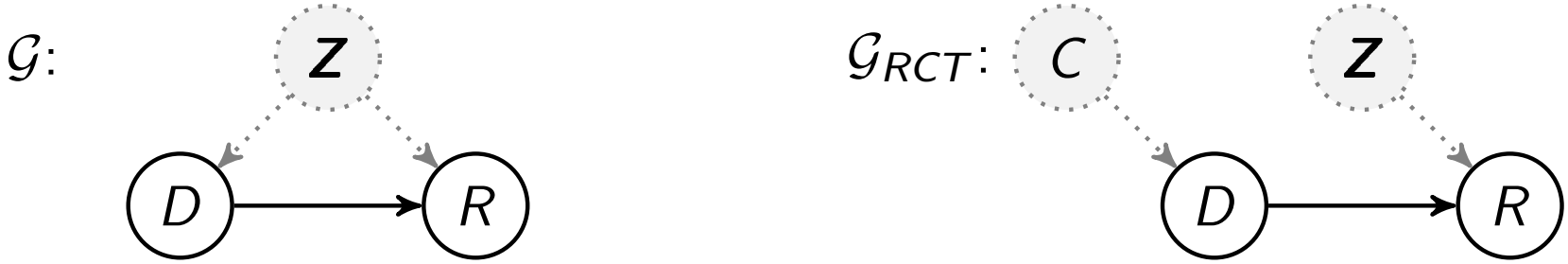
These ideas have inspired causal discovery methods that combine observational and interventional data in various ways.

Contents

Randomized Controlled Trials [Fisher, 1935]

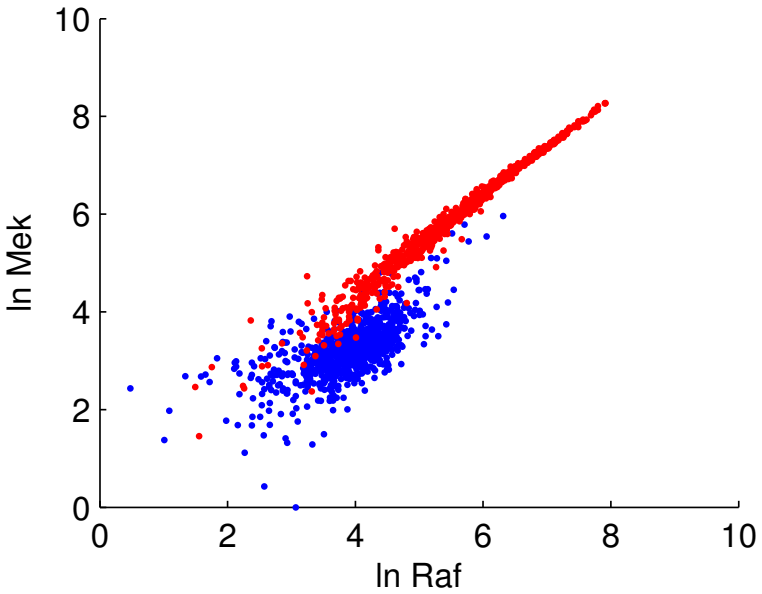


R: Recovery, D: Drug, $\boldsymbol{Z}$: latent confounders (e.g., genetics), C: coin flip.

- Divide patients into two groups: treatment and control randomly (e.g., by a coin flip).
- Patients in the treatment group are forced to take a drug, and patients in the control group are forced to not take the drug (but to take a placebo instead): $D = C$.
- Estimating the causal effect of the drug now becomes a standard statistical exercise, as $p(R \mid D = C) = p(R \mid \text{do}(D = C))$.
- The RCT intervention breaks any back-door paths, if existent.

All *evidence-based* medicine is based on this idea.

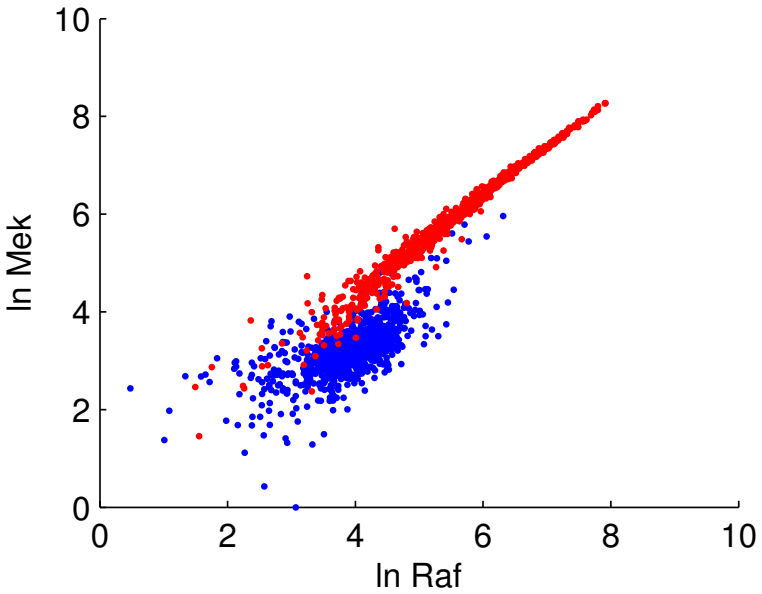
Causal Discovery by Experimentation: Example



- Each dot is a measurement in a single human immune system cell
- Raf: abundance of phosphorylized Raf
- Mek: abundance of phosphorylized Mek
- blue = baseline,
  red = reagent U0126 added

Question: What is the causal relation between Raf and Mek?

Causal Discovery by Experimentation: Example



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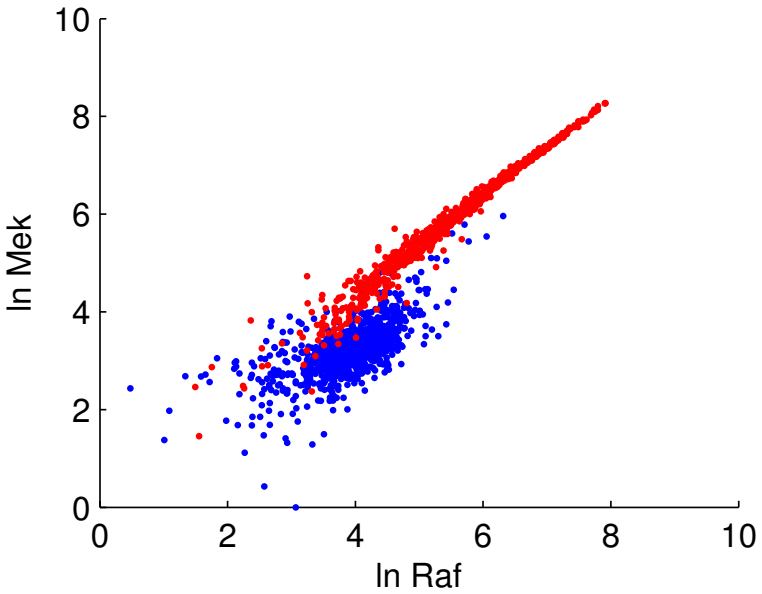
Hint: U0126 inhibits Mek.

Causal Discovery by Experimentation: Example



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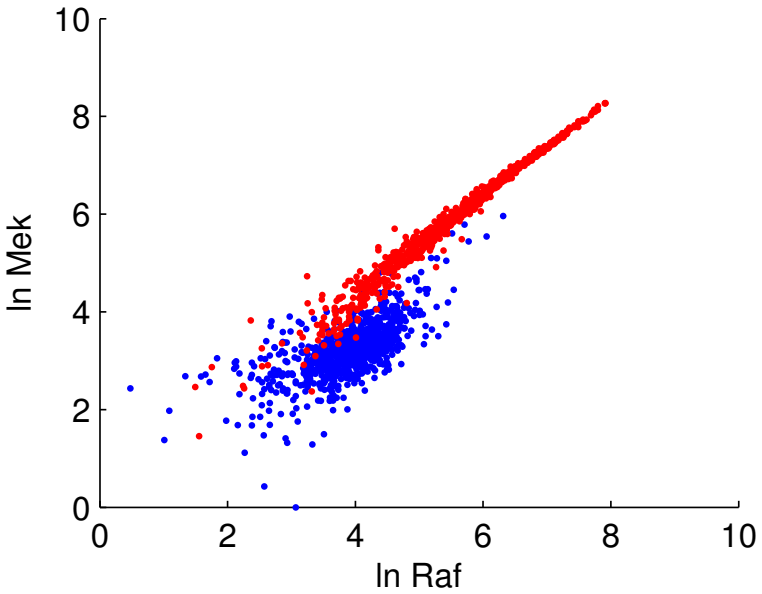
Question: What is the causal relation between Raf and Mek?
Hint: U0126 inhibits Mek.

Answer: Mek causes Raf
(Changing activity of Mek changes abundance of Raf.)

Causal Discovery by Experimentation: Example



- Each dot is a measurement in a single human immune system cell
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- blue = baseline,
  red = reagent U0126 added

Question: What is the causal relation between Raf and Mek?
Hint: U0126 inhibits Mek.

Answer: Mek causes Raf
(Changing activity of Mek changes abundance of Raf.)

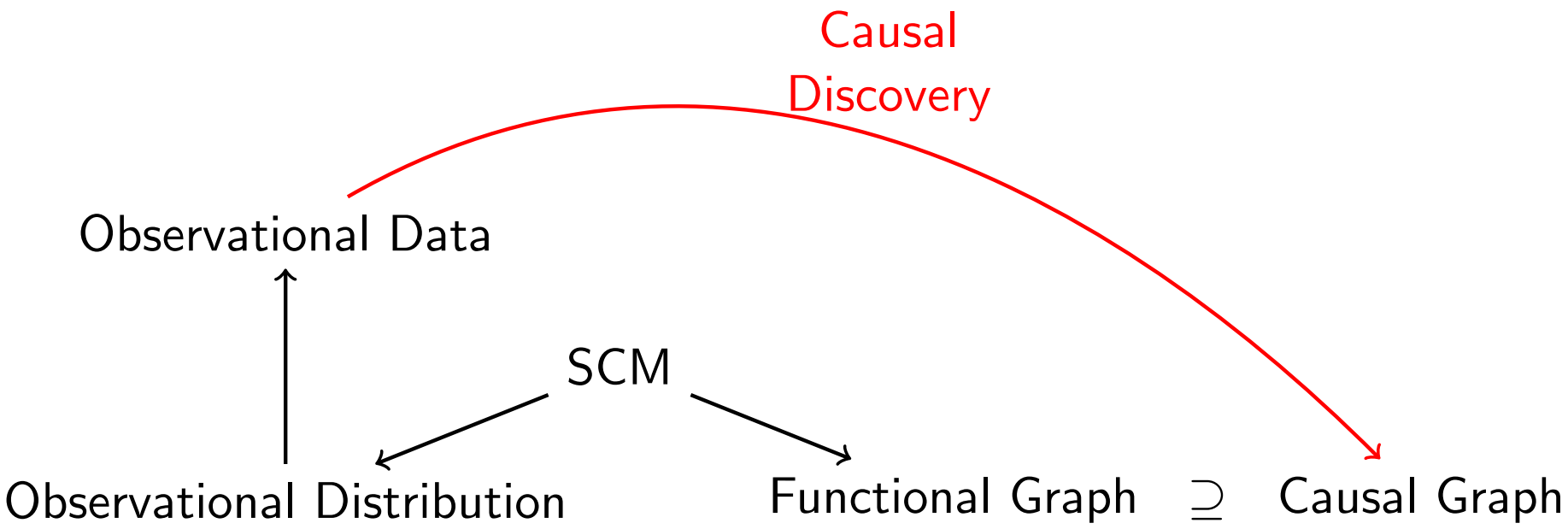
Note: How did we know that "U0126 inhibits Mek" in the first place?

Contents

Causal Discovery from Observational Data

Experiments can be expensive, time-consuming, unethical, impractical or even infeasible.

Intriguing alternative: causal discovery from purely observational data [Spirtes et al., 2000, Pearl, 2000]!



Disclaimer: Works only under strong assumptions and with (possibly very) large sample sizes.

Approaches to Causal Discovery from Observational Data I

Conditional-independence constraint-based

Independence patterns in the data constrain the possible causal graphs.

- **LCD (Cooper, 1997)**
- **Y-Structures (Mani & Cooper, 2004)**
- PC (Spirtes & Gleimour & Scheines, 2000), IC (Pearl, 2000)
- FCI (Spirtes & Meek & Richardson, 1995; Zhang, 2008)
- ...

General constraint-based

Similar, but exploiting more general types of constraints in the data.

- Verma constraints (Robins (1986), Verma & Pearl (1990), Tian & Pearl (2002))
- Nested Markov Models (Richardson, Evans, Robins, Shpitser (2017))
- Algebraic Constraints (Van Ommen & Mooij (2017))
- ...

Approaches to Causal Discovery from Observational Data II

Likelihood-based approaches

Score penalized likelihoods of possible causal graphs and select the best one(s).

- Bayesian Network Learning (Heckerman, Geiger, Chickering, 1995)
- Greedy Equivalence Search (Chickering, 2002)
- ...

Restrictions on functional causal relations and noise distributions

Minimize the "complexity" of causal models.

- LINGAM (Kano, Shimizu, 2003; Shimizu *et al.*, 2006)
- Additive Noise Models (Hoyer *et al.*, 2006)
- Post-Nonlinear Model (Zhang & Hyvärinen, 2009)
- ...

Constraint-based Causal Discovery

From the pattern of conditional independences in the data we can reconstruct a set of possible underlying causal graphs, even when allowing for latent confounders [Spirtes et al., 2000].

Data

X_1	X_2	X_3	X_4
2	0.1	0.2	0.5
2	0.13	0.21	0.49
2	0.23	0.21	0.51
5	0.5	0.19	0.52
5	0.6	0.18	0.51
2	0.2	0.22	0.92
2	0.23	0.21	0.99
5	0.53	1.2	0.95
5	0.55	1.19	0.97

CIs

$X_2 \not\perp\!\!\!\perp X_4$

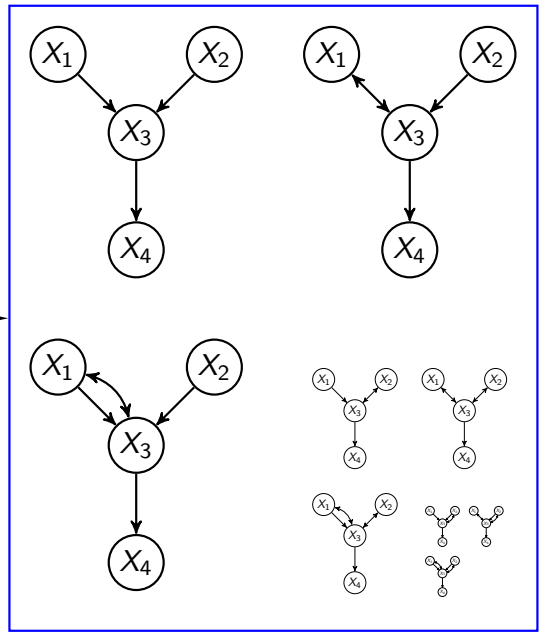
$X_2 \perp\!\!\!\perp X_4 \mid X_3$

$X_1 \perp\!\!\!\perp X_2$

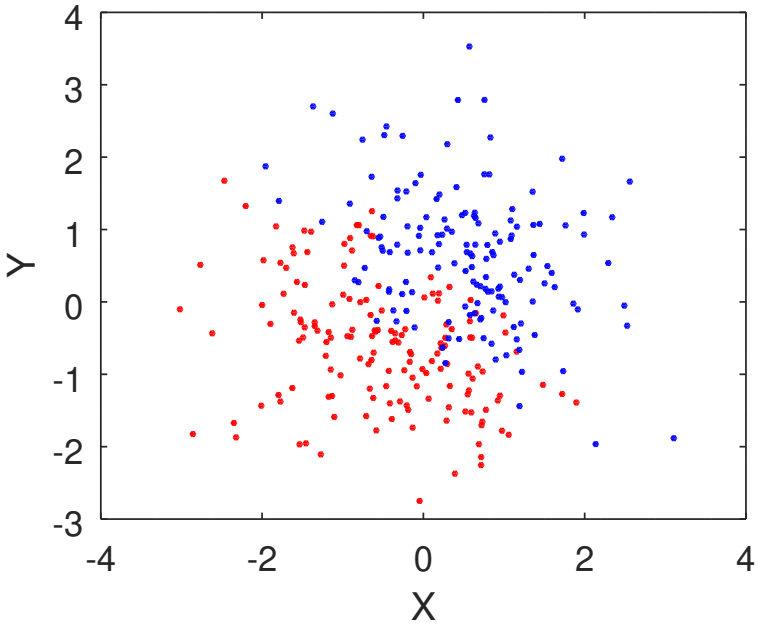
$X_1 \not\perp\!\!\!\perp X_2 \mid X_3$

$\ldots$

Possible Causal Graphs



Causal Discovery from Observational Data: V-Structure

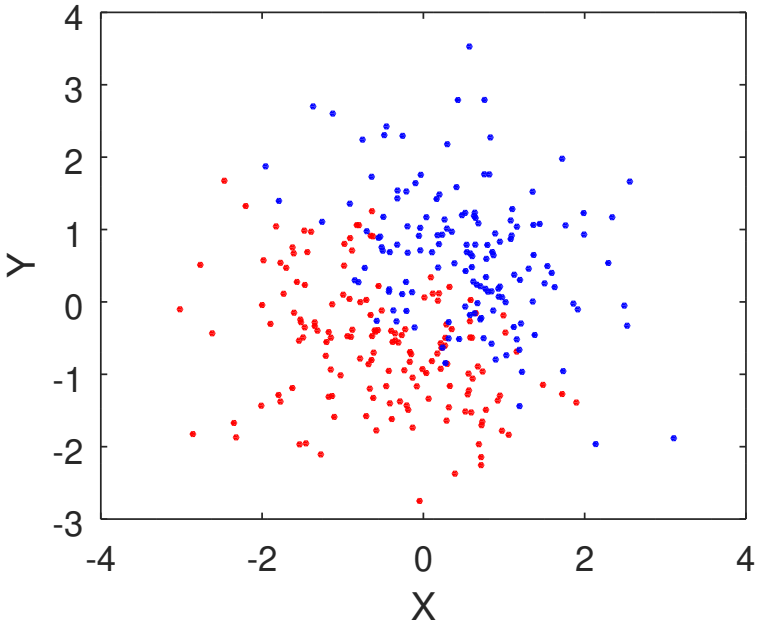


blue: $Z = 0$, red: $Z = 1$

$$X \perp\!\!\!\perp Y,\ X \not\perp\!\!\!\perp Y \mid Z,$$
$$X \not\perp\!\!\!\perp Z,\ X \not\perp\!\!\!\perp Z \mid Y,$$
$$Y \not\perp\!\!\!\perp Z,\ Y \not\perp\!\!\!\perp Z \mid X.$$

Question: What is the causal relation between X, Y and Z?

Causal Discovery from Observational Data: V-Structure



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$$X \perp\!\!\!\perp Y,\ X \not\perp\!\!\!\perp Y \mid Z,$$
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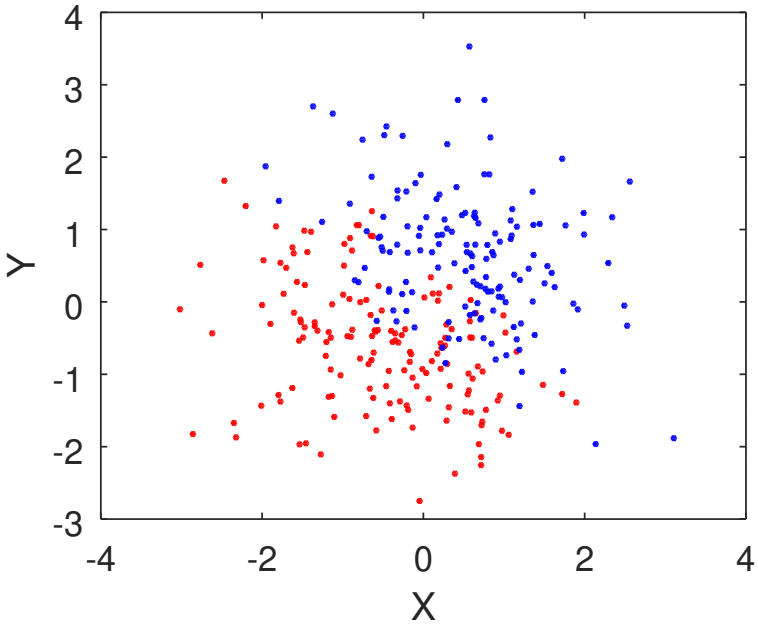
$$Y \not\perp\!\!\!\perp Z,\ Y \not\perp\!\!\!\perp Z \mid X.$$

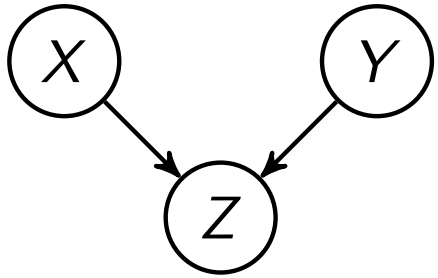
Question: What is the causal relation between X, Y and Z?

Hint: Assume an acyclic, faithful SCM without latent confounders generated the data, and assume no selection bias or measurement error

Causal Discovery from Observational Data: V-Structure



blue: $Z = 0$, red: $Z = 1$



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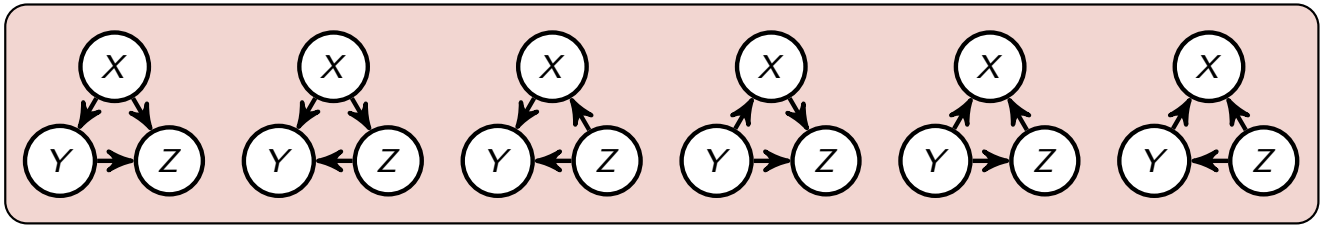
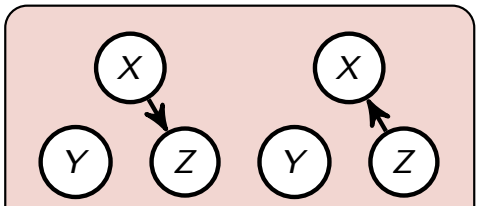
Question: What is the causal relation between X, Y and Z?
Hint: Assume an acyclic, faithful SCM without latent confounders generated the data, and assume no selection bias or measurement error

Answer: X causes Z; Y causes Z; X and Y causally unrelated

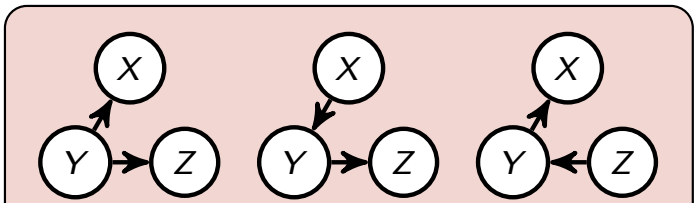
Note: Strong assumptions, but no experiments needed!

Markov equivalence classes for three variables

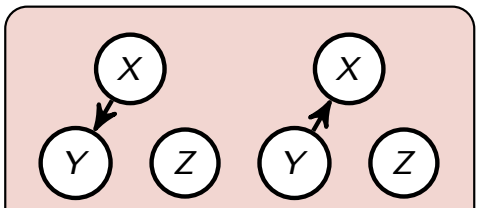




$Y \perp\!\!\!\perp X, Y \perp\!\!\!\perp X \mid Z$
$Y \perp\!\!\!\perp Z, Y \perp\!\!\!\perp Z \mid X$



$Z \perp\!\!\!\perp X \mid Y$



$Z \perp\!\!\!\perp X, Z \perp\!\!\!\perp X \mid Y$
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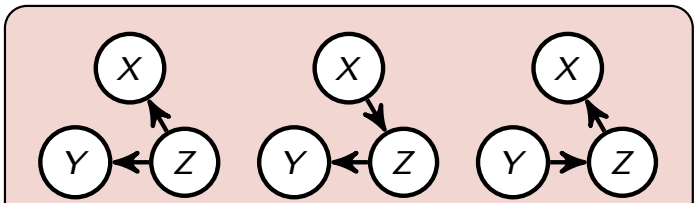
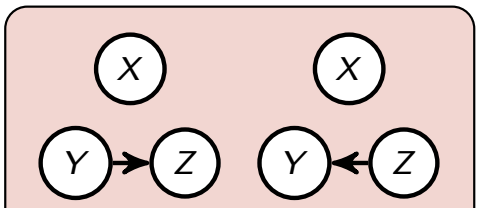


$X \perp\!\!\!\perp Y \mid Z$



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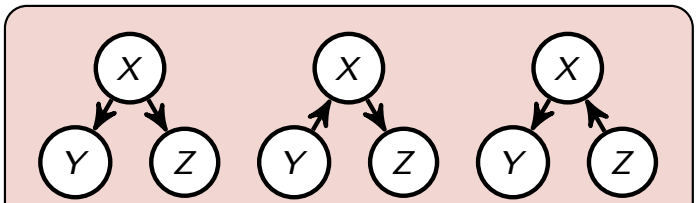
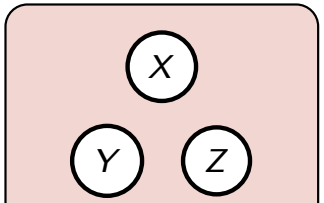
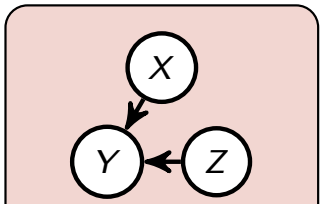


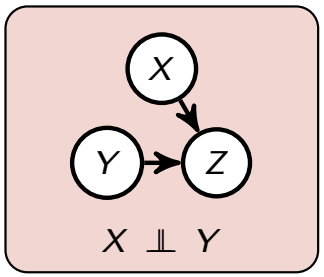
$Y \perp\!\!\!\perp Z \mid X$



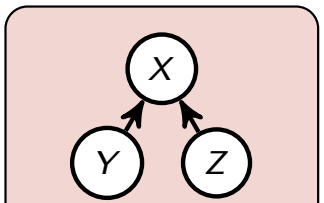
$X \perp\!\!\!\perp Y, X \perp\!\!\!\perp Y \mid Z$
$Y \perp\!\!\!\perp Z, Y \perp\!\!\!\perp Z \mid X$
$Z \perp\!\!\!\perp X, Z \perp\!\!\!\perp X \mid Y$



$Z \perp\!\!\!\perp X$



$X \perp\!\!\!\perp Y$

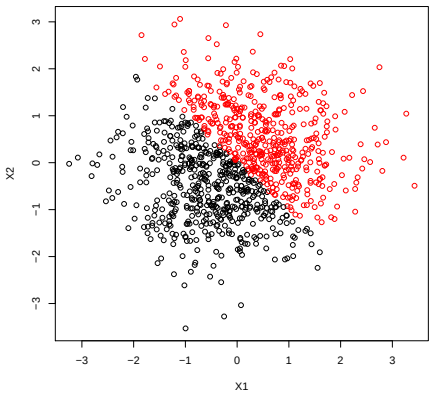


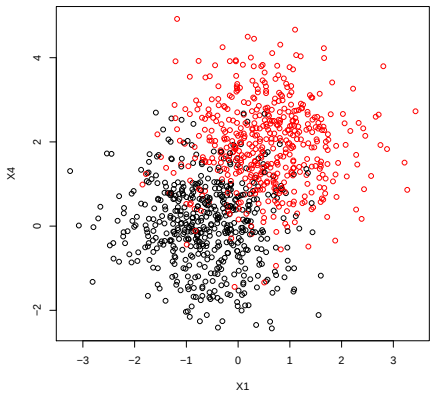
$Y \perp\!\!\!\perp Z$

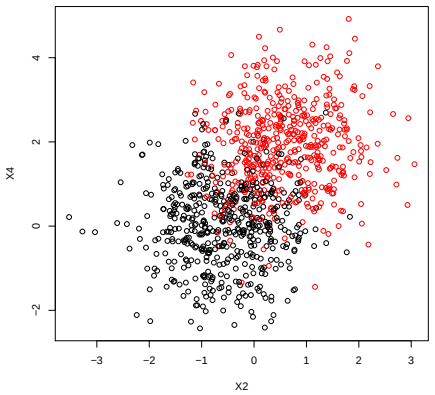
Exercise 5

Please make Exercise 5

Causal Discovery from Observational Data: Y-Structure







$X_1 \perp\!\!\!\perp X_2$
$X_1 \not\perp\!\!\!\perp X_2 \,|\, X_3$

$X_1 \not\perp\!\!\!\perp X_4$
$X_1 \perp\!\!\!\perp X_4 \,|\, X_3$

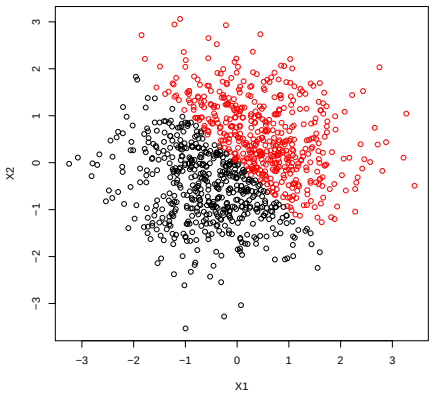
$X_2 \not\perp\!\!\!\perp X_4$
$X_2 \perp\!\!\!\perp X_4 \,|\, X_3$

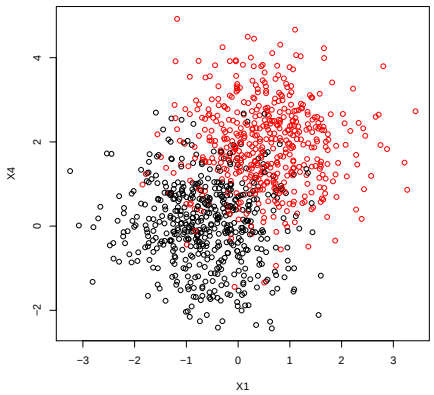
black: $X_3 = 0$, red: $X_3 = 1$

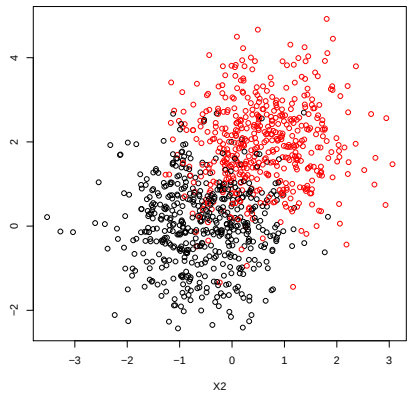
Question: What is the causal relation between X_3 and X_4?

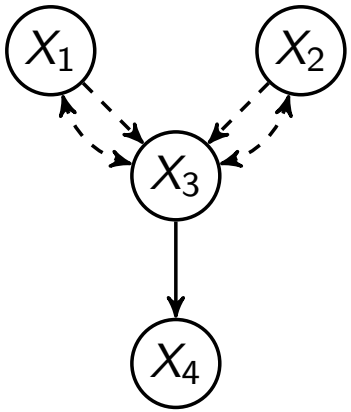
Hint: Assume an acyclic, faithful SCM generated the data, and assume no selection bias or measurement error.

Causal Discovery from Observational Data: Y-Structure









$X_1 \perp\!\!\!\perp X_2$
$X_1 \not\perp\!\!\!\perp X_2 \mid X_3$

$X_1 \not\perp\!\!\!\perp X_4$
$X_1 \perp\!\!\!\perp X_4 \mid X_3$

$X_2 \not\perp\!\!\!\perp X_4$
$X_2 \perp\!\!\!\perp X_4 \mid X_3$

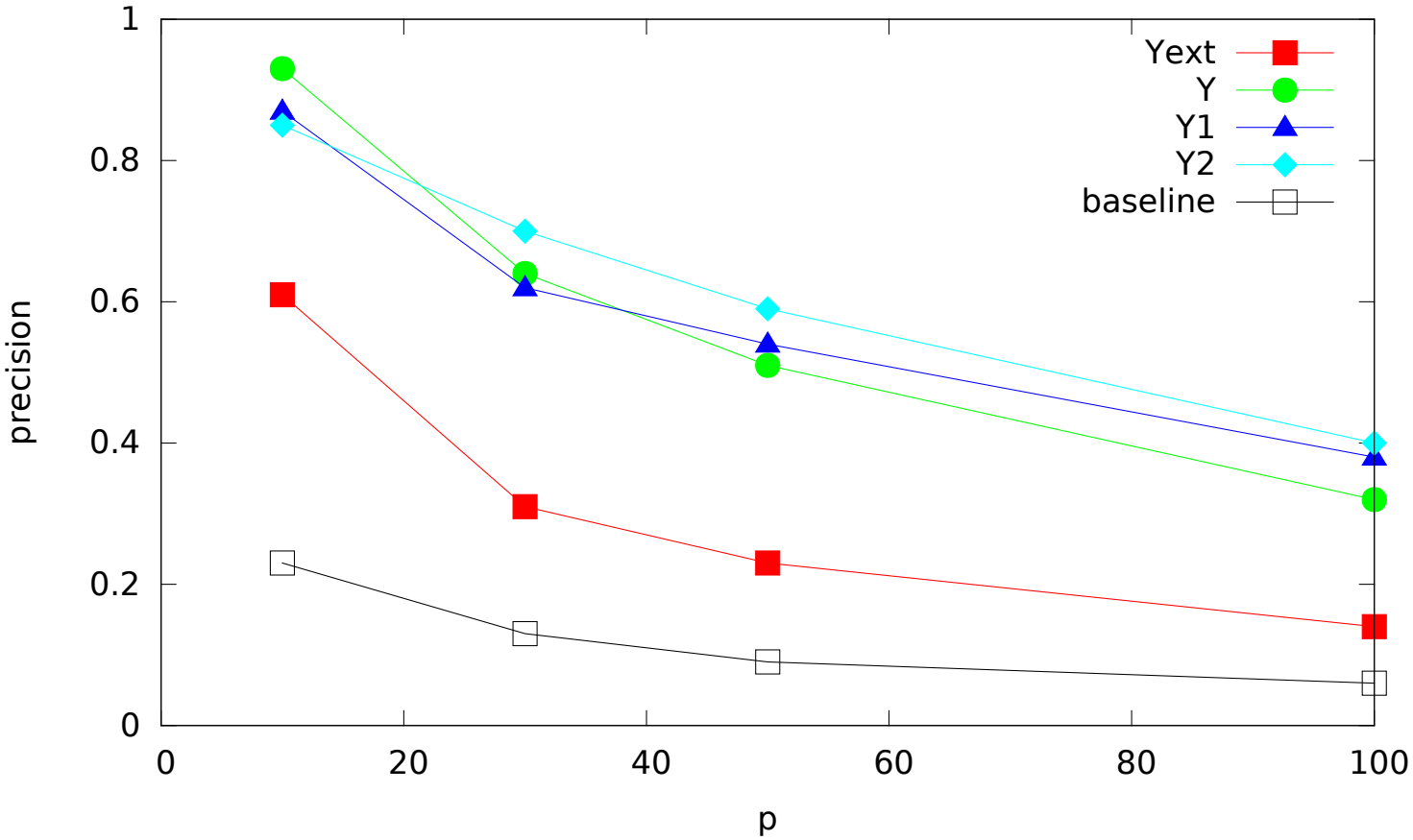
black: $X_3 = 0$, red: $X_3 = 1$

Question: What is the causal relation between X_3 and X_4?
Hint: Assume an acyclic, faithful SCM generated the data, and assume no selection bias or measurement error.

Answer: X_3 causes X_4 and they are not confounded. Hence, the causal effect of X_3 on X_4 satisfies $p(x_4 \mid \mathrm{do}(X_3 = x_3)) = p(x_4 \mid x_3)$.

Y-structures: Empirical Performance I

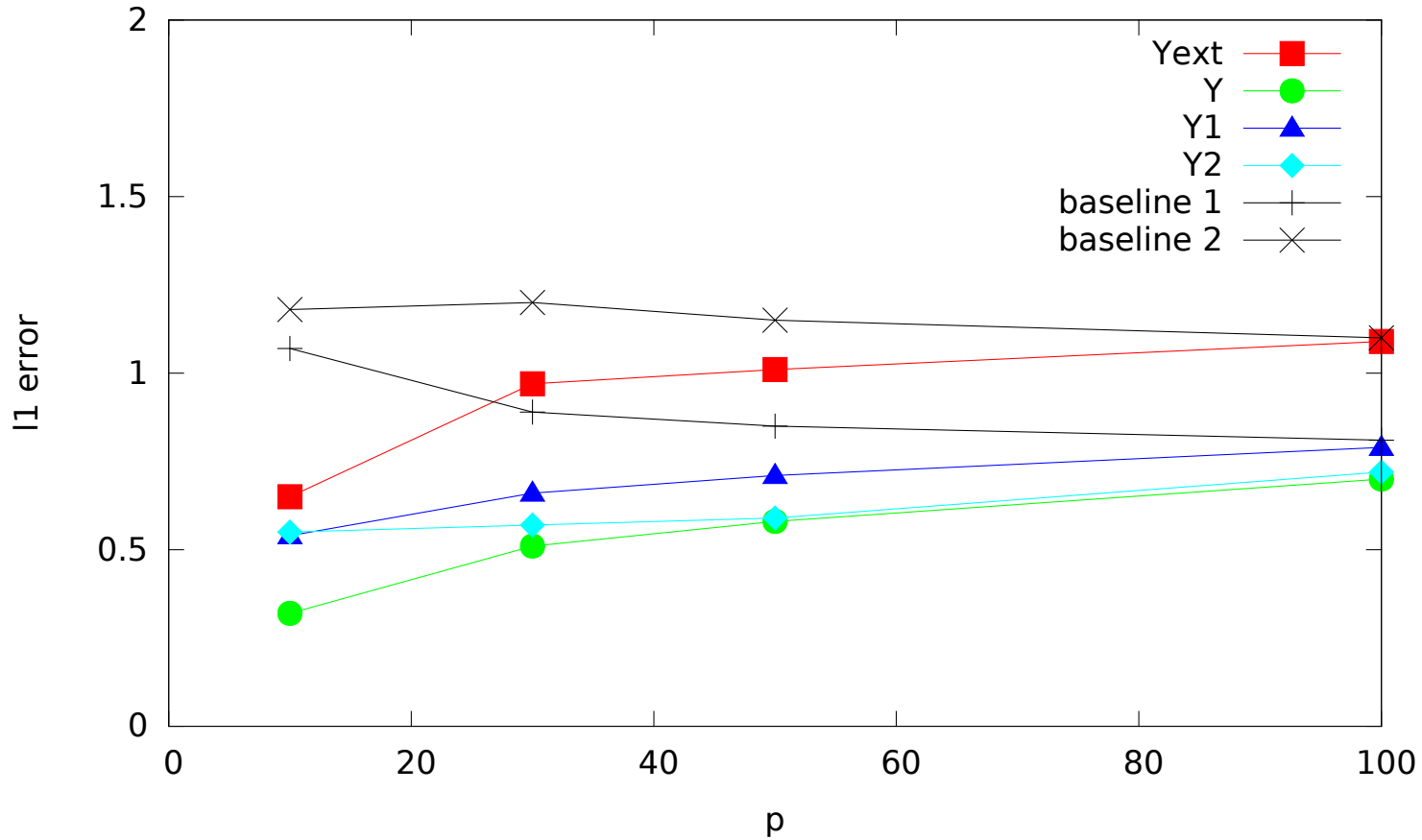
Precision of prediction X causes Y:



Baseline: random guessing

Y-structures: Empirical Performance II

Causal prediction error for $\mathbb{E}(Y \mid \mathrm{do}(X = x))$:



Baseline 1: $p(y \mid \mathrm{do}(X = x)) = p(y)$, Baseline 2: $p(y \mid \mathrm{do}(X = x)) = p(y|x)$

Hardness of Causal Discovery

d	Number of DAGs with d nodes
1	1
2	3
3	25
4	543
5	29281
6	3781503
7	1138779265
8	783702329343
9	1213442454842881
10	4175098976430598143
11	31603459396418917607425
12	521939651343829405020504063
13	18676600744432035186664816926721
14	1439428141044398334941790719839535103
15	237725265553410354992180218286376719253505
16	83756670773733320287699303047996412235223138303
17	62707921196923889899446452602494921906963551482675201
18	9942119532215951589522891459235452451655502687858830501478 3
19	33277190122710759173617757331126112588358307625842190258354677 3505

Table B.1: The number of DAGs depending on the number d of nodes, taken from `http://oeis.org/A003024` [OEIS Foundation Inc., 2017]. The length of the numbers grows faster than any linear term.

Source: [Peters et al., 2017]

(Augmented) FCI

[Spirtes *et al.*, 2000, Spirtes *et al.*, 1999, Ali *et al.*, 2005, Zhang, 2008]

$\mathcal{R}0a$ If $X \perp\!\!\!\perp Y \,|\, \mathbf{Z}$, then $X \not\!\!\perp\!\!\!\perp Y$, $Sep(X,Y) \leftarrow \mathbf{Z}$.

$\mathcal{R}0b$ If $X *\!\!-\!\!* Z \circ\!\!-\!\!* Y$ and $X \not\!\!\perp\!\!\!\perp Y$, then if $Z \notin Sep(X,Y)$, then $X *\!\!\rightarrow Z \leftarrow\!\!* Y$.

$\mathcal{R}1$ If $X *\!\!\rightarrow Z \circ\!\!-\!\!* Y$, and $X \not\!\!\perp\!\!\!\perp Y$, then $Z \longrightarrow Y$.

$\mathcal{R}2a$ If $Z \longrightarrow X *\!\!\rightarrow Y$ and $Z *\!\!-\!\!\circ Y$, then $Z *\!\!\rightarrow Y$.

$\mathcal{R}2b$ If $Z *\!\!\rightarrow X \longrightarrow Y$ and $Z *\!\!-\!\!\circ Y$, then $Z *\!\!\rightarrow Y$.

$\mathcal{R}3$ If $X *\!\!\rightarrow Z \leftarrow\!\!* Y$, $X *\!\!-\!\!\circ W \circ\!\!-\!\!* Y$, $X \not\!\!\perp\!\!\!\perp Y$, and $W *\!\!-\!\!\circ Z$, then $W *\!\!\rightarrow Z$.

$\mathcal{R}4a$ If $u = \langle X, .., Z_k, Z, Y \rangle$ is a discriminating path between X and Y for Z, and $Z \circ\!\!-\!\!* Y$, then if $Z \in Sep(X,Y)$, then $Z \longrightarrow Y$.

$\mathcal{R}4b$ Idem, if $Z \notin Sep(X,Y)$ then $Z_k \longleftrightarrow Z \longleftrightarrow Y$.

$\mathcal{R}5$ If $u = \langle Z, X, .., W, Y, Z, X \rangle$ is an uncov. circle path, then $Z \!-\!\!-\!\! Y$ (idem for all edges on u).

$\mathcal{R}6$ If $X \!-\!\!-\! Z \circ\!\!-\!\!* Y$, then orient as $Z \!-\!\!* Y$.

$\mathcal{R}7$ If $X \!-\!\!\circ Z \circ\!\!-\!\!* Y$, and $X \not\!\!\perp\!\!\!\perp Y$, then $Z \!-\!\!* Y$.

$\mathcal{R}8a$ If $Z \longrightarrow X \longrightarrow Y$ and $Z \circ\!\!\rightarrow Y$, then $Z \longrightarrow Y$.

$\mathcal{R}8b$ If $Z \!-\!\!\circ X \longrightarrow Y$ and $Z \circ\!\!\rightarrow Y$, then $Z \longrightarrow Y$.

$\mathcal{R}9$ If $Z \circ\!\!\rightarrow Y$, $u = \langle Z, X, W, .., Y \rangle$ is an uncov. p.d. path, and $X \not\!\!\perp\!\!\!\perp Y$, then $Z \longrightarrow Y$.

$\mathcal{R}10$ If $Z \circ\!\!\rightarrow Y$, $X \longrightarrow Y \longleftarrow W$, $u_1 = \langle Z, S, .., X \rangle$ and $u_2 = \langle Z, V, .., W \rangle$ are uncov. p.d. paths, (possibly with $S = X$ and/or $V = W$), then if $S \not\!\!\perp\!\!\!\perp V$, then $Z \longrightarrow Y$.

Input : independence oracle for $\mathbf{V}$
Output : complete PAG $\mathcal{P}$ over $\mathbf{V}$

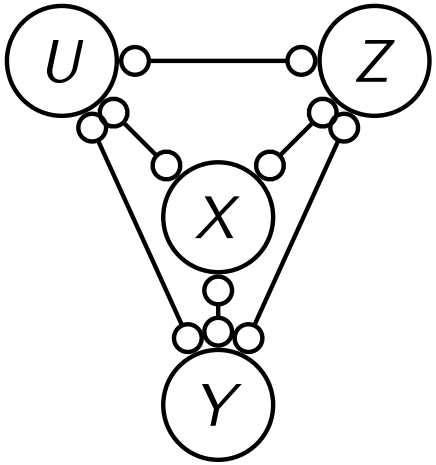
$\mathcal{P} \leftarrow$ fully $\circ\!\!-\!\!\circ$ connected graph over $\mathbf{V}$
for all $\{X, Y\} \in \mathbf{V}$ **do**
  search *in some clever way* for a $X \perp\!\!\!\perp Y \,|\, \mathbf{Z}$
    $\mathcal{P} \leftarrow \mathcal{R}0a$ (eliminate $X \not\!\!\perp\!\!\!\perp Y$)
    record $Sep(X,Y) \leftarrow \mathbf{Z}$
end for
$\mathcal{P} \leftarrow \mathcal{R}0b$ (unshielded colliders)
repeat $\mathcal{P} \leftarrow \mathcal{R}1 - \mathcal{R}4b$ **until** finished
$\mathcal{P} \leftarrow \mathcal{R}5$ (uncovered circle paths)
repeat $\mathcal{P} \leftarrow \mathcal{R}6 - \mathcal{R}7$ **until** finished
repeat $\mathcal{P} \leftarrow \mathcal{R}8a - \mathcal{R}10$ **until** finished

Algorithm 1: Augmented FCI algorithm

Source: [Claassen & Heskes, 2011]

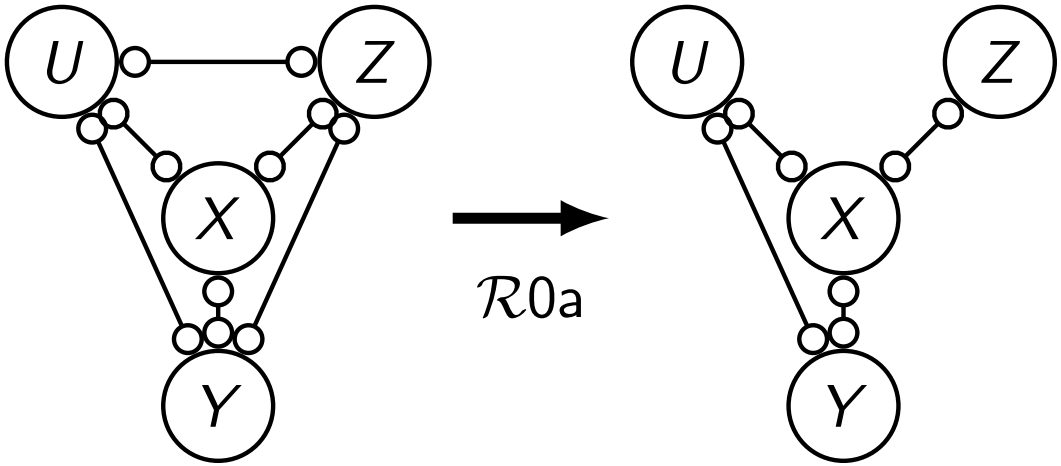
FCI: Example ("Extended Y-structure")

Independences: $Z \perp\!\!\!\perp U$, $Z \perp\!\!\!\perp Y \mid X$



FCI: Example ("Extended Y-structure")

Independences: $Z \perp\!\!\!\perp U,\ Z \perp\!\!\!\perp Y \mid X$



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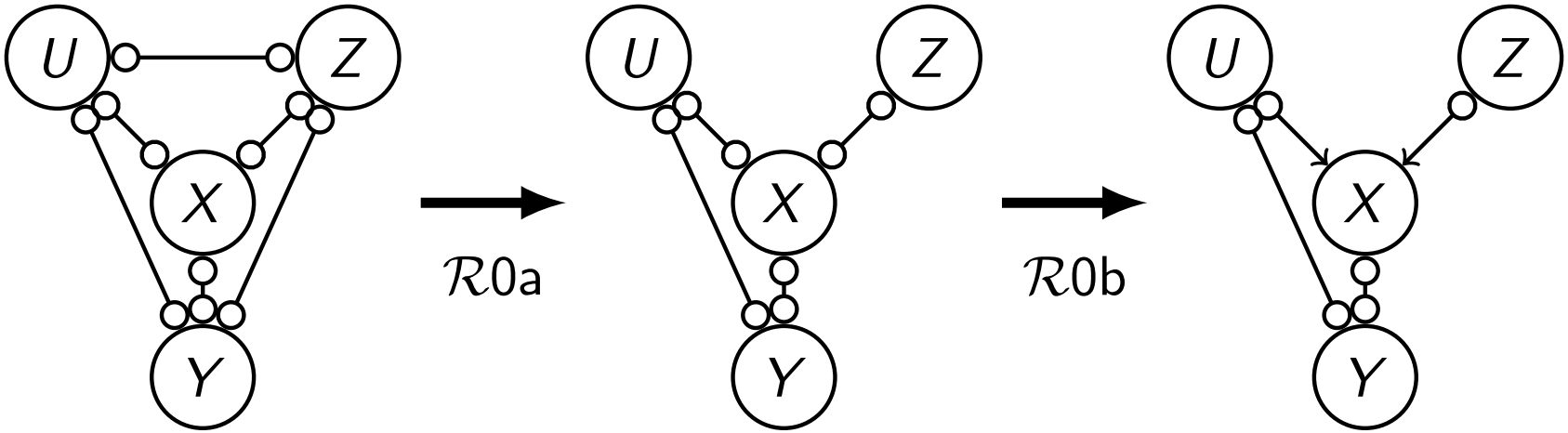

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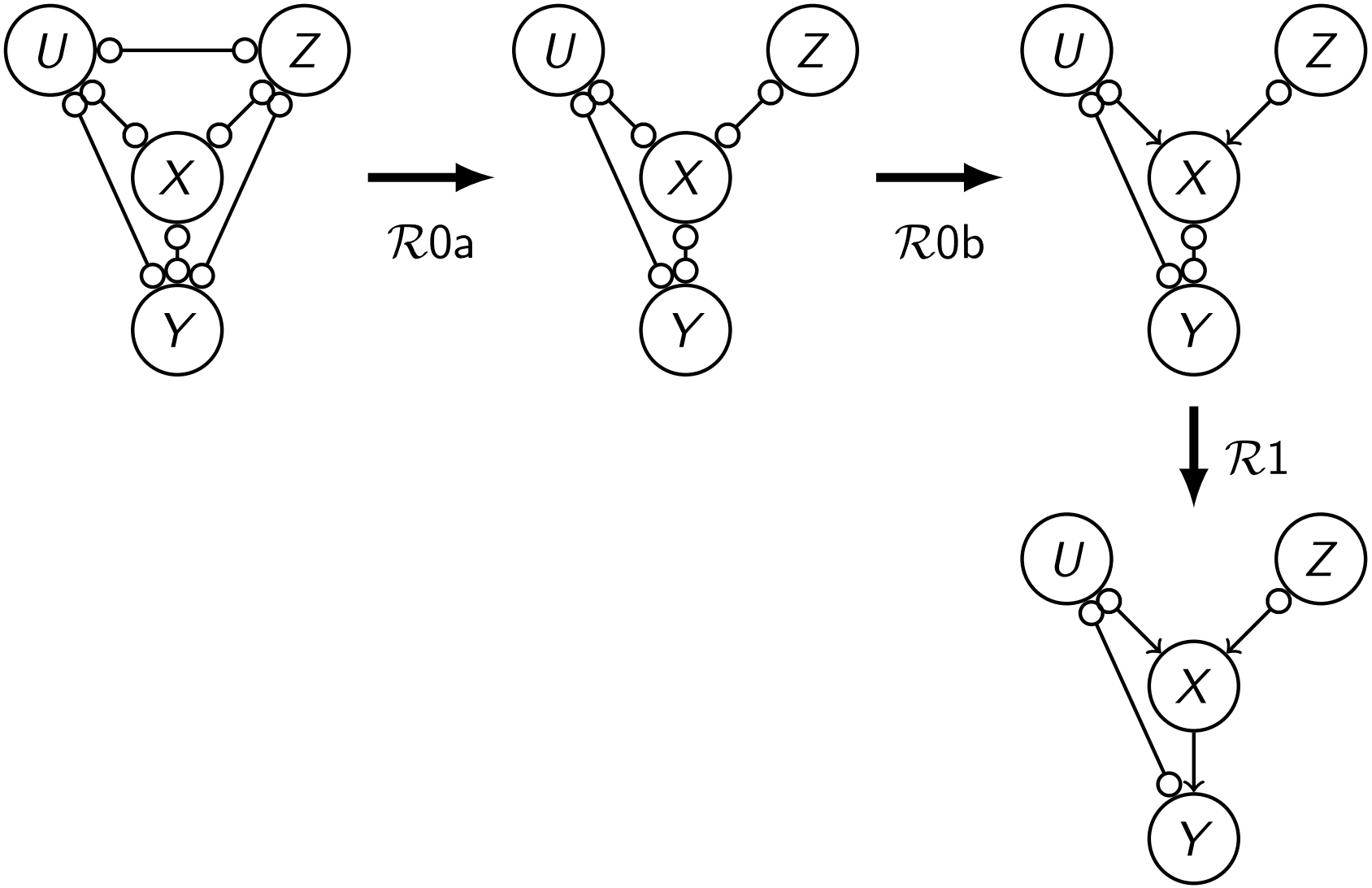

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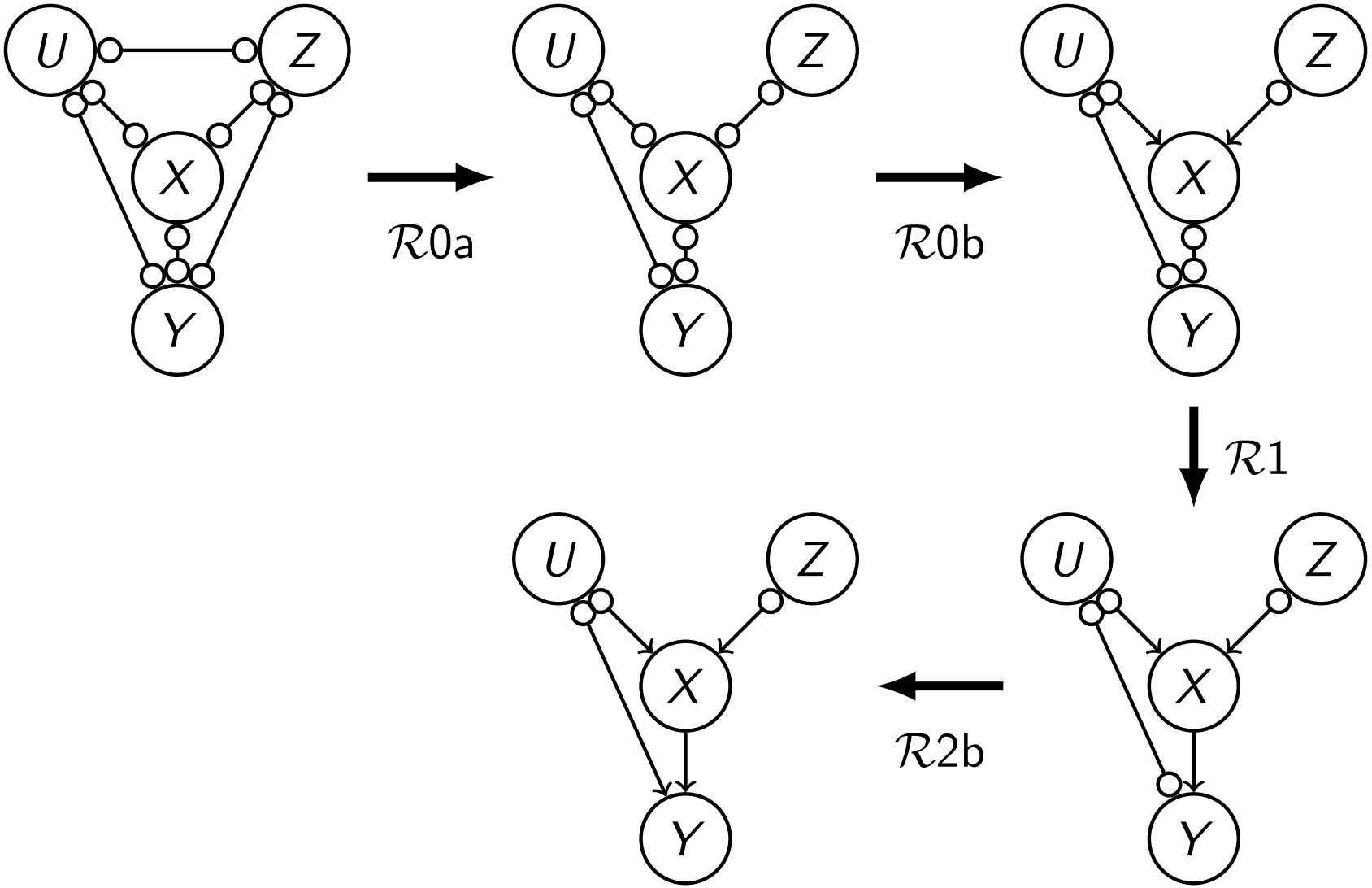

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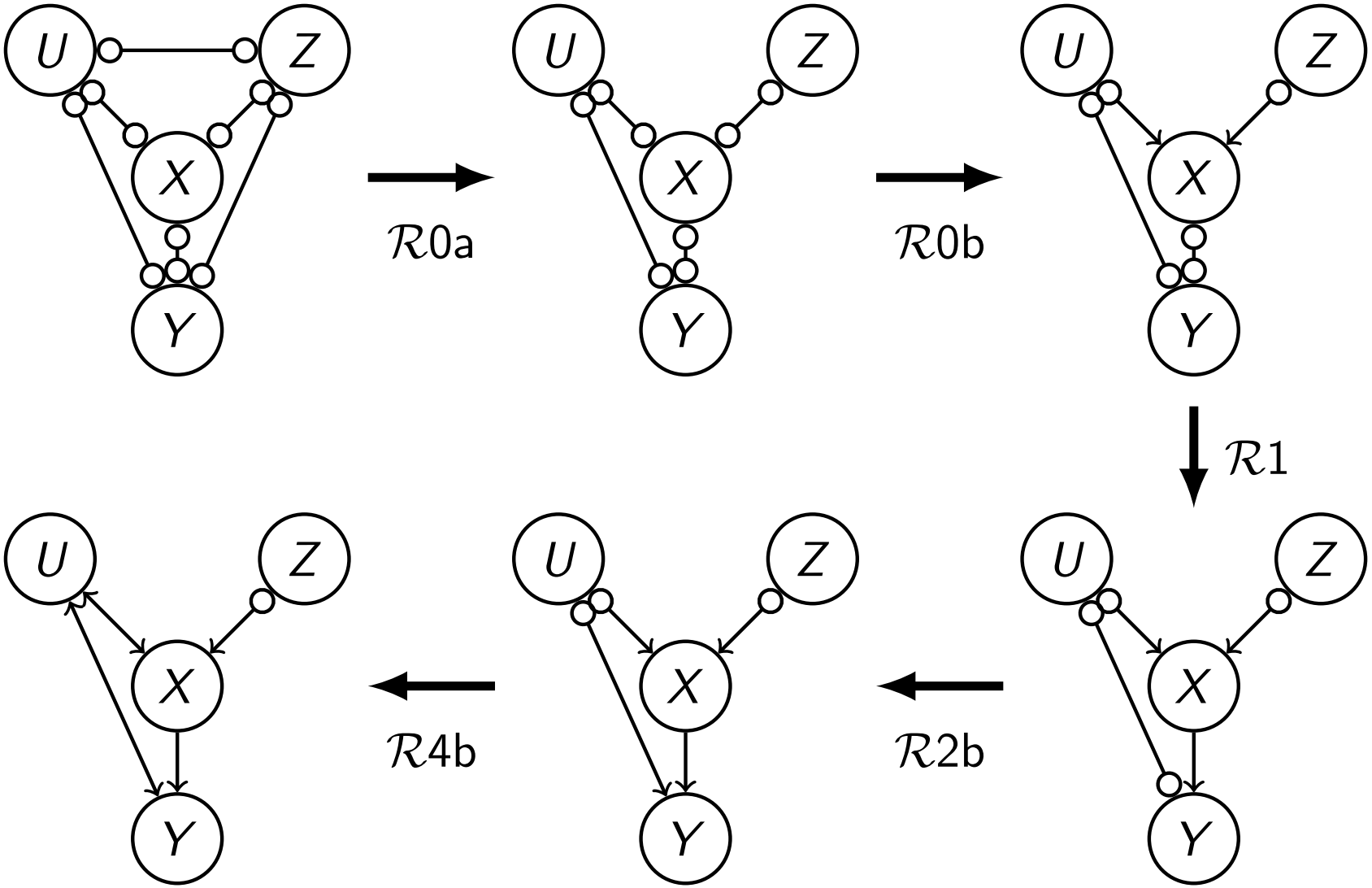


Local Causal Discovery (LCD)

Local Causal Discovery: simple causal discovery algorithm (Cooper, 1997).

Definition

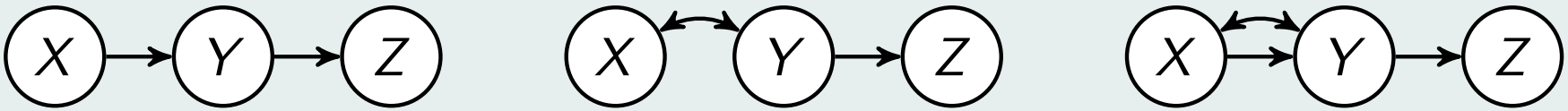
If for three variables X, Y, Z:

$$Y \notin \mathrm{an}(X) \ \wedge \ Z \notin \mathrm{an}(X) \ \wedge \ X \not\perp\!\!\!\perp Y \ \wedge \ Y \not\perp\!\!\!\perp Z \ \wedge \ X \perp\!\!\!\perp Z \,|\, Y,$$

then (X, Y, Z) is an LCD triplet.

Theorem

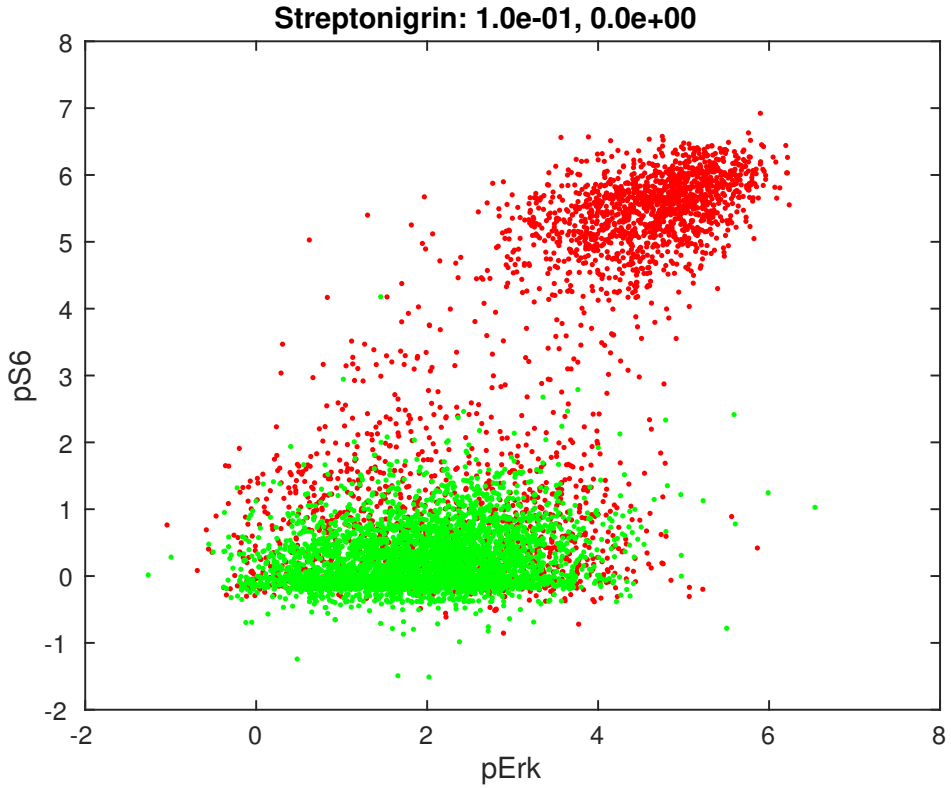
If an acyclic, faithful SCM generated the data without selection bias or measurement error, the only causal graphs that yield an LCD triplet are:



Therefore, $Y \in \mathrm{an}(Z)$ *and* $p(Z \,|\, \mathrm{do}(Y = y)) = p(Z \,|\, Y = y)$.

LCD: Example

- *pErk*: abundance of phosphorylized Erk in each cell
- *pS6*: abundance of phosphorylized S6 in cell
- *I*: green = baseline, red = PMA-IONO activator added



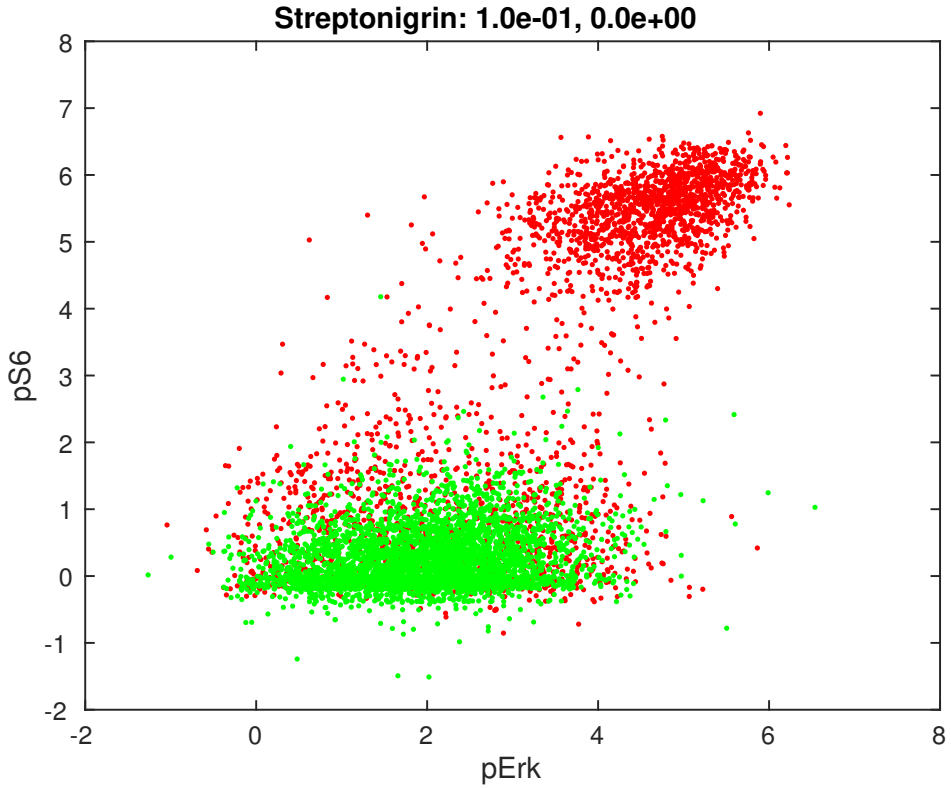
(X, Y, Z) is LCD triplet iff:

$$Y \notin \mathrm{an}(X)$$
$$Z \notin \mathrm{an}(X)$$
$$X \not\perp\!\!\!\perp Y$$
$$Y \not\perp\!\!\!\perp Z$$
$$X \perp\!\!\!\perp Z \mid Y$$

What is the causal relation?

LCD: Example

- *pErk*: abundance of phosphorylized Erk in each cell
- *pS6*: abundance of phosphorylized S6 in cell
- *I*: green = baseline, red = PMA-IONO activator added



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$$Z \notin \mathrm{an}(X)$$
$$X \not\perp\!\!\!\perp Y$$
$$Y \not\perp\!\!\!\perp Z$$
$$X \perp\!\!\!\perp Z \mid Y$$

What is the causal relation? LCD triplet $(I, pS6, pErk)$, so $pS6 \to pErk$.

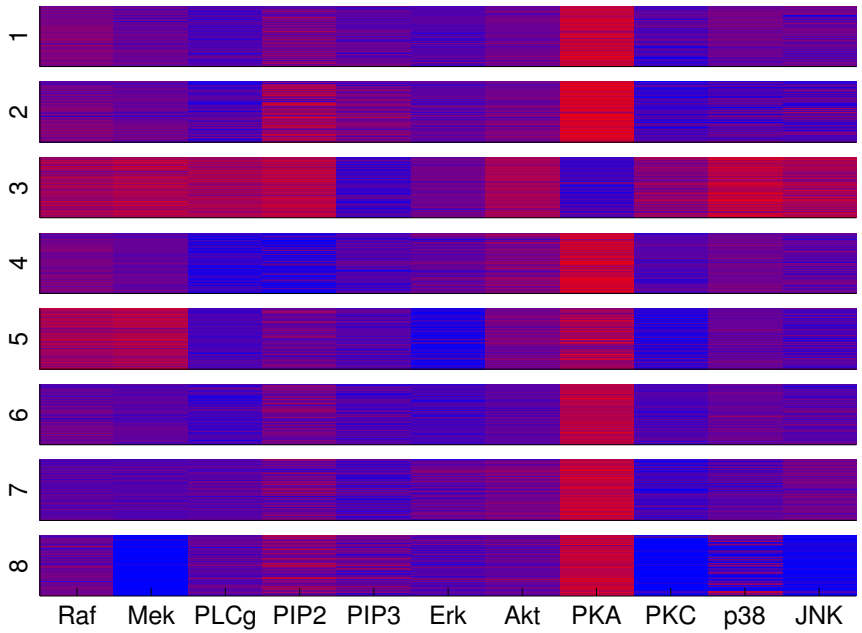
Note: no prior knowledge on the effects of PMA-IONO needed!

Contents

Causal Discovery: Example Application

Protein Abundance Data:

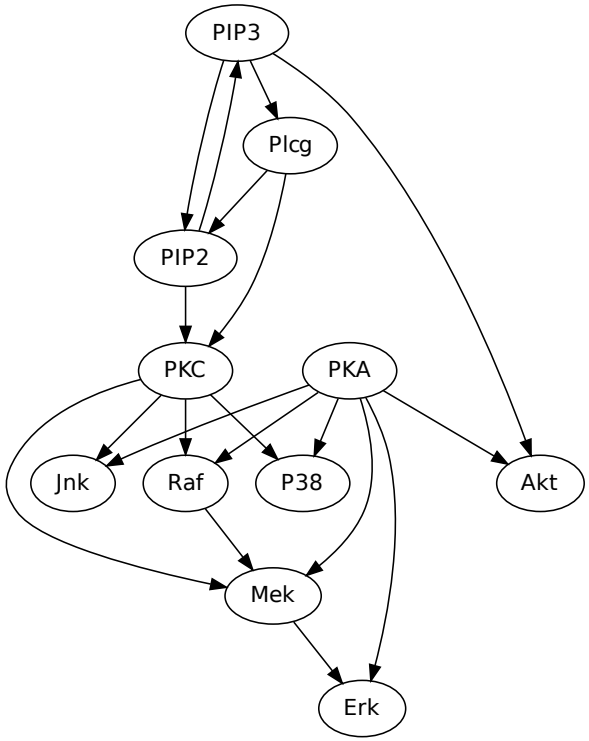
(Sachs *et al*, 2005)



Condition	Reagent	Intervention
1	-	observational
2	Akt-inhibitor	inhibits AKT activity
3	G0076	inhibits PKC activity
4	Psitectorigenin	inhibits PIP2 abundance
5	U0126	inhibits MEK activity
6	LY294002	inhibits PIP2/PIP3 activity
7	PMA	activates PKC + global
8	β2CAMP	activates PKA + global

Causal Graph:

("Signalling network")



(depicted here: "consensus" network)

Causal Discovery from Multiple Contexts

	Latent confounders	Nonlinear mechanisms	Cycles	Perfect interventions	Mechanism changes	Activity interventions	Side effects	Other context changes	Unknown intervention/context targets	Learns intervention/context targets	Multiple system variables	Different variables per context	Combination strategy
(Fisher, 1935)	+	+	+	+	+	+	+	+	+	+	-	-	b
(Cooper and Yoo, 1999)	-	+	-	+	-	-	-	-	-	-	+	-	b
(Tian and Pearl, 2001)	-	+	-	-	+	-	-	+	-	-	+	-	b
(Sachs et al., 2005)	-	+	-	+	-	-	-	-	-	-	+	-	b
(Eaton and Murphy, 2007)	-	+	-	+	+	+	+	+	+	+	+	-	b
(Chen et al., 2007)	+	+	+	+	+	+	+	+	+	+	+	-	b
(Claassen and Heskes, 2010)	+	+	-	-	+	+	+	+	+	-	+	+	a
(Tillman and Spirtes, 2011)	+	+	-	-	+	+	+	+	+	-	+	+	a
(Hauser and Bühlmann, 2012)	-	+	-	+	-	-	-	-	-	-	+	-	b
(Hyttinen et al., 2012)	+	-	+	+	-	-	-	-	-	-	+	-	a
(Mooij and Heskes, 2013)	-	±	±	+	+	+	-	+	-	-	+	-	b
(Hyttinen et al., 2014)	+	+	±	+	-	-	-	-	-	-	+	+	a
(Triantafillou and Tsamardinos, 2015)	+	+	-	+	-	-	-	-	-	-	+	+	a
(Rothenhäusler et al., 2015)	+	-	±	-	-	-	-	+	+	+	+	-	a
(Peters et al., 2016)	±	±	±	+	+	+	+	+	+	-	+	-	b
(Oates et al., 2016a)	-	-	-	-	-	-	-	+	-	-	+	-	b
(Zhang et al., 2017)	-	+	-	+	+	+	+	+	+	+	+	-	b
JCI	+	+	+	+	+	+	+	+	+	+	+	±	b
JCI-LCD (Cooper, 1997)	+	+	+	+	+	+	+	+	+	+	+	-	b
JCI-HEJ	+	+	±	+	+	+	+	+	+	+	+	-	b
JCI-FCI	+	+	-	+	+	+	+	+	+	+	+	-	b

JCI: Combining the best of two worlds

Question

Can we combine the ideas of the "classical" approach to causal discovery based on experimentation with the "modern" approach based on conditional independences?

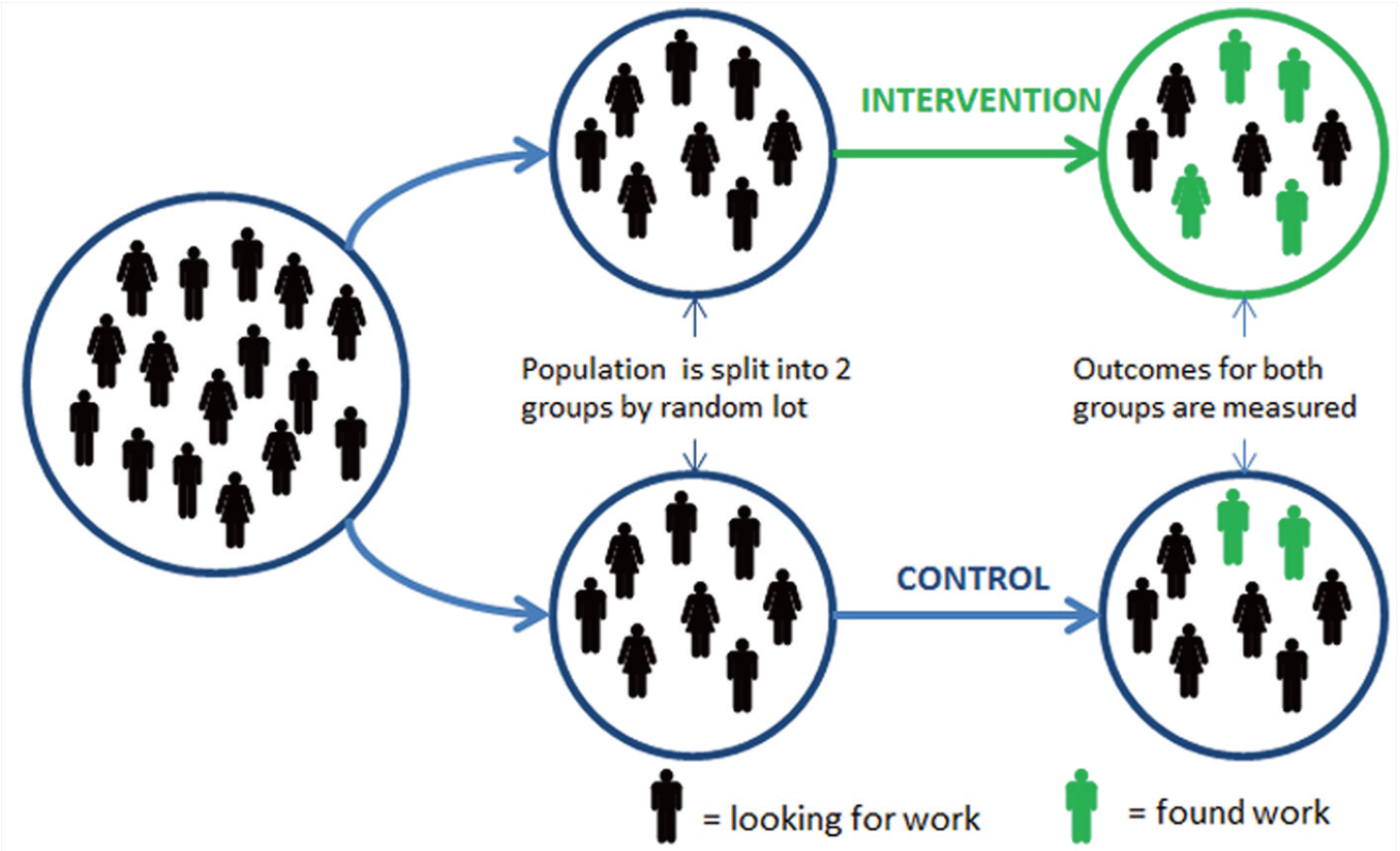
We hope to:

- obtain reliability of "classical" approach
- exploit conditional independences in the data to reduce the number of experiments necessary

Answer

We propose Joint Causal Inference, a framework for causal discovery, that achieves this.

Randomized Controlled Trials, or A/B-testing



C_1	X_1
0	1
0	0
0	1
0	0
0	0
0	0
0	0
0	0
1	0
1	0
1	1
1	1
1	0
1	1
1	0
1	1

Two variables: context variable C_1, system variable X_1

C_1: 0=control, 1=intervention

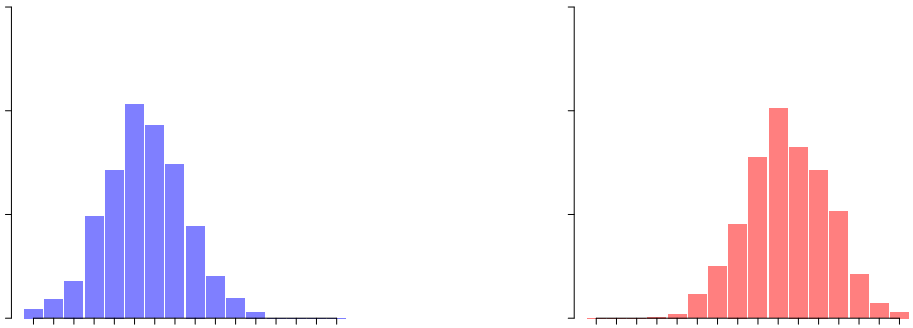
X_1: 0=looking for work, 1=found work

Two equivalent points of view

(a) Separate data sets

Placebo ($C = 0$):
X
-0.2
0.6
-1.7
...

Drug ($C = 1$):
X
-0.3
1.8
-0.1
...

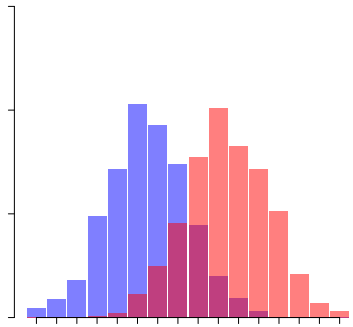


Two-sample test:

Is $p\big(x \mid \mathrm{do}(C = 0)\big) = p\big(x \mid \mathrm{do}(C = 1)\big)$?

(b) Pooled data

C	X
0	-0.2
0	0.6
0	-1.7
0	...
1	-0.3
1	1.8
1	-0.1
1	...



Independence test:

Is $X \perp\!\!\!\perp C$?

Causal Inference for Randomized Controlled Trial

Proposition

Suppose C (treatment) and X (outcome) can be modeled with a Structural Causal Model. The Randomized Controlled Trial assumptions

- X does not cause C (*because X happens after C*)
- X and C are unconfounded (*because of the randomization*)
- no selection bias (*measure and analyze all samples*)

imply that if $C \not\perp\!\!\!\perp X$, then C causes X (*correlation implies causation*).

Causal Inference for Randomized Controlled Trial

Proposition

Suppose C (treatment) and X (outcome) can be modeled with a Structural Causal Model. The Randomized Controlled Trial assumptions

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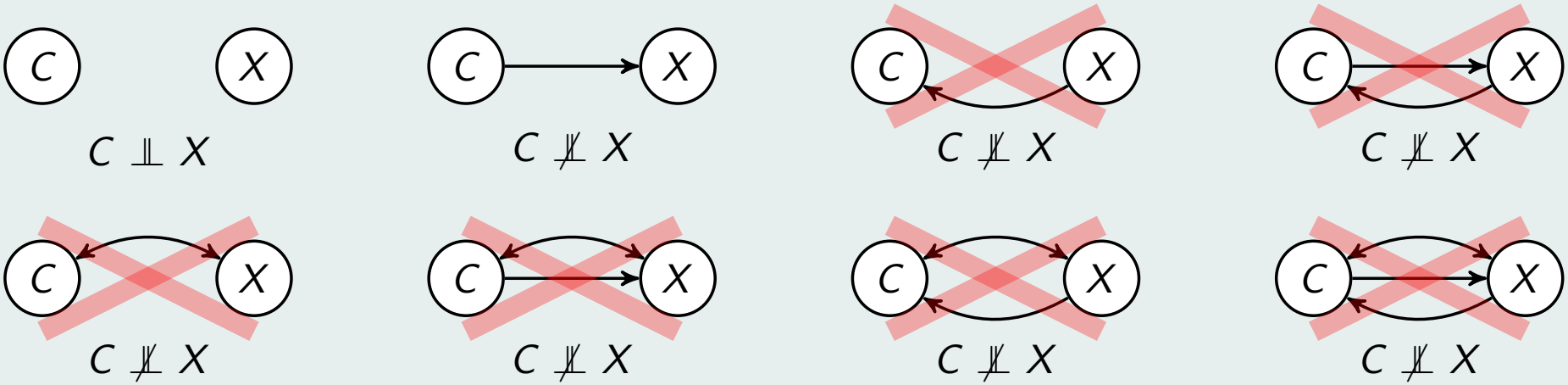
imply that if $C \not\perp\!\!\!\perp X$, then C causes X (*correlation implies causation*).

Proof



JCI: Two types of variables

Definition

JCI generalizes the idea of RCTs to multiple context and system variables.
Distinguish:

- **Context variables** $\{C_i\}_{i \in \mathcal{I}}$ that model the context of the system,
- **System** variables $\{X_j\}_{j \in \mathcal{J}}$ that model the system of interest.

Example

Data for 3 observed system variables in 4 experimental conditions:

System variables:
X_1: salary
X_2: drug abuse
X_3: depression

Context variables:
C_1: back-to-work program
C_2: psychotherapy

no interventions:

X_1	X_2	X_3
0.1	0.2	0.5
0.13	0.21	0.49
0.23	0.21	0.51

only psychotherapy:

X_1	X_2	X_3
0.5	0.19	0.52
0.6	0.18	0.51

only back-to-work program:

X_1	X_2	X_3
0.2	0.22	0.92
0.23	0.21	0.99

both interventions:

X_1	X_2	X_3
0.53	1.2	0.95
0.61	1.21	0.90
0.55	1.19	0.97

JCI: Pooling the data

After explicitly adding the context variables, we pool the data:

Example

no interventions:

X_1	X_2	X_3
0.1	0.2	0.5
0.13	0.21	0.49
0.23	0.21	0.51

only psychotherapy:

X_1	X_2	X_3
0.5	0.19	0.52
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only back-to-work program:

X_1	X_2	X_3
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X_1	X_2	X_3
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C_1	C_2	X_1	X_2	X_3
0	0	0.1	0.2	0.5
0	0	0.13	0.21	0.49
0	0	0.23	0.21	0.51
0	1	0.5	0.19	0.52
0	1	0.6	0.18	0.51
1	0	0.2	0.22	0.92
1	0	0.23	0.21	0.99
1	1	0.53	1.2	0.95
1	1	0.61	1.21	0.90
1	1	0.55	1.19	0.97

System variables:
X_1: salary
X_2: drug abuse
X_3: depression

Context variables:
C_1: back-to-work program
C_2: psychotherapy

JCI: Assumptions

JCI Assumptions (Intuitive formulation)

We are modelling a generic setting in which the experimenter decides on the performed interventions *before* the measurements are performed, and this decision does not depend on anything else that might affect the system of interest.

Formal JCI Assumptions

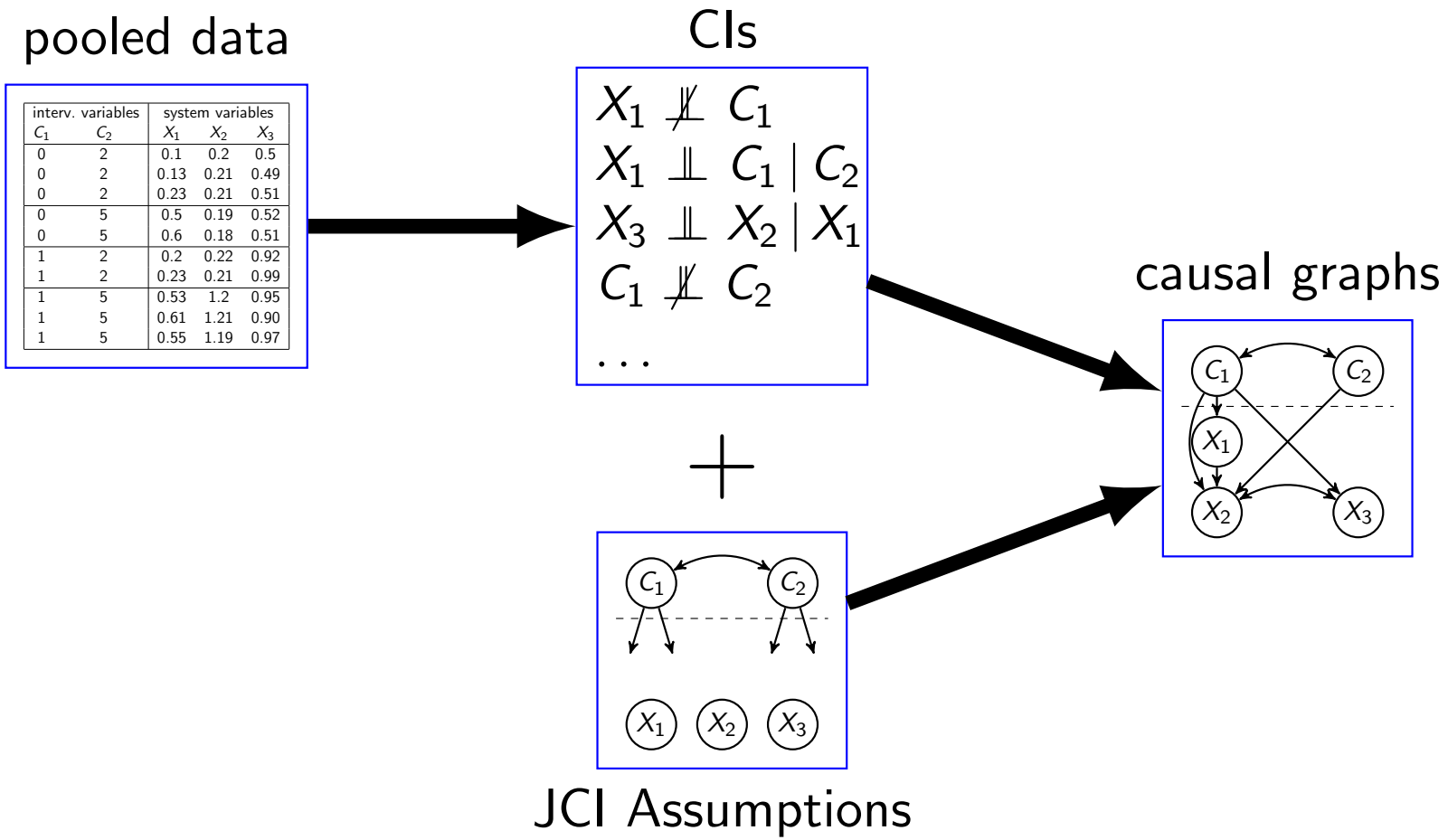
The causal graph $\mathcal{G}$ that includes both system variables $\{X_1, \ldots, X_p\}$ and context variables $\{C_1, \ldots, C_d\}$, which jointly models the experimental design and the system in *all* experimental conditions, satisfies:

- no variable directly causes any context variable C_i, and
- none of the pairs $\{X_k, C_i\}$ of system and context variables is confounded, and
- each pair of context variables $\{C_i, C_j\}$ is confounded.

Furthermore, we assume the absence of selection bias.

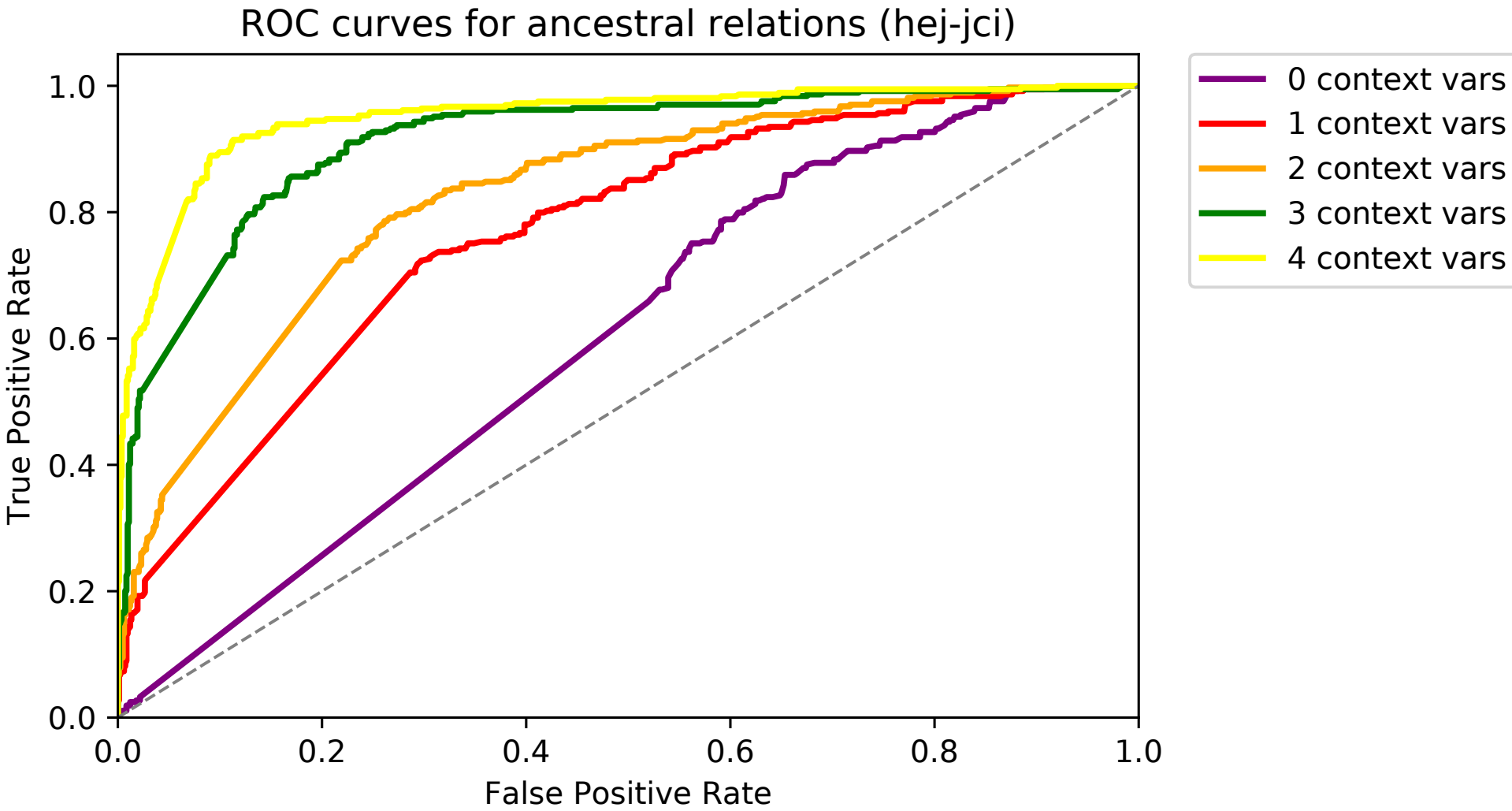
Joint Causal Inference

Question: How can we discover



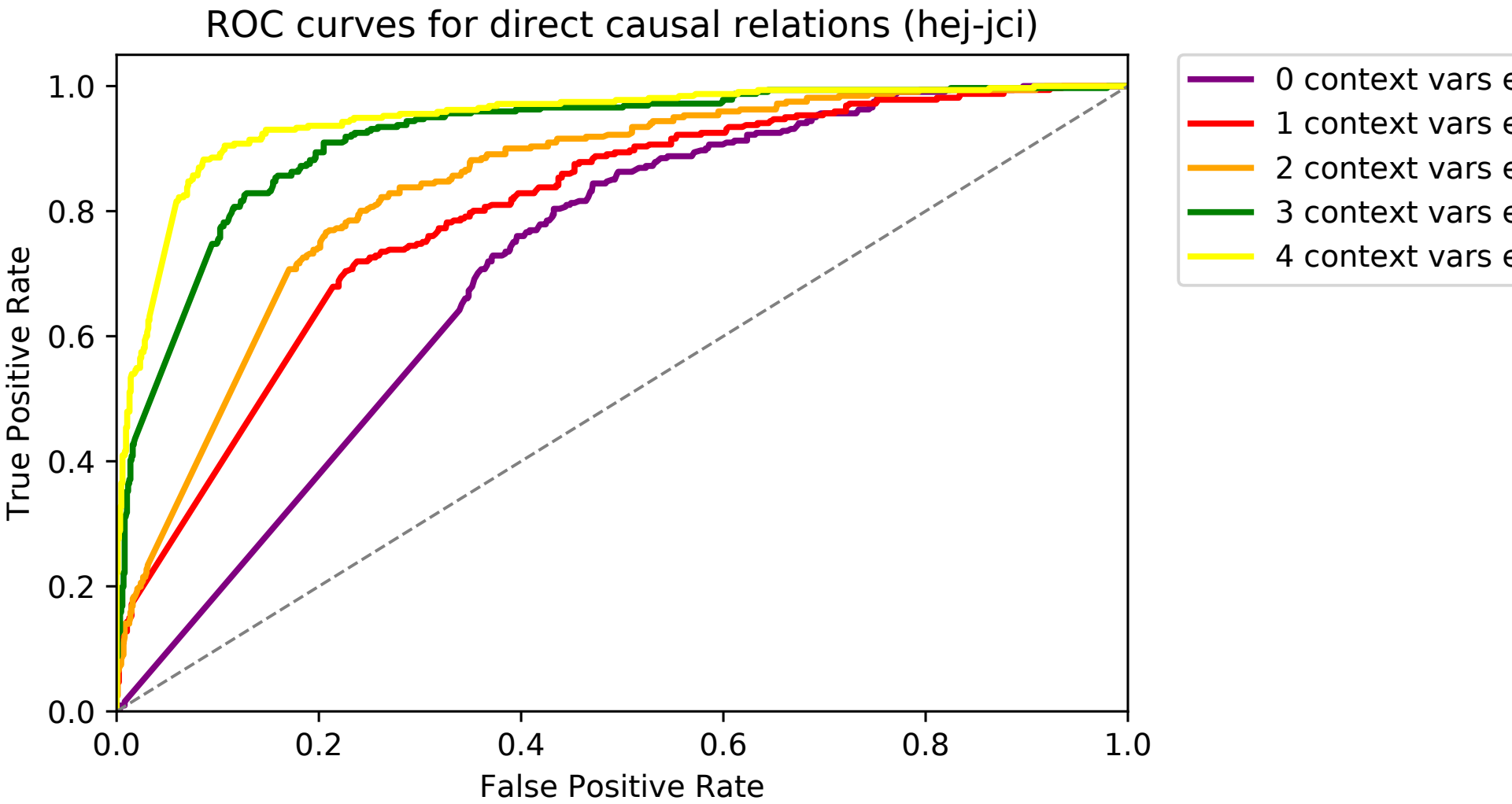
Answer: Simply apply a standard constraint-based causal discovery method (designed for purely observational data) on the *pooled* data, and incorporate the JCI assumptions as background knowledge.

Evaluation on simulated data I



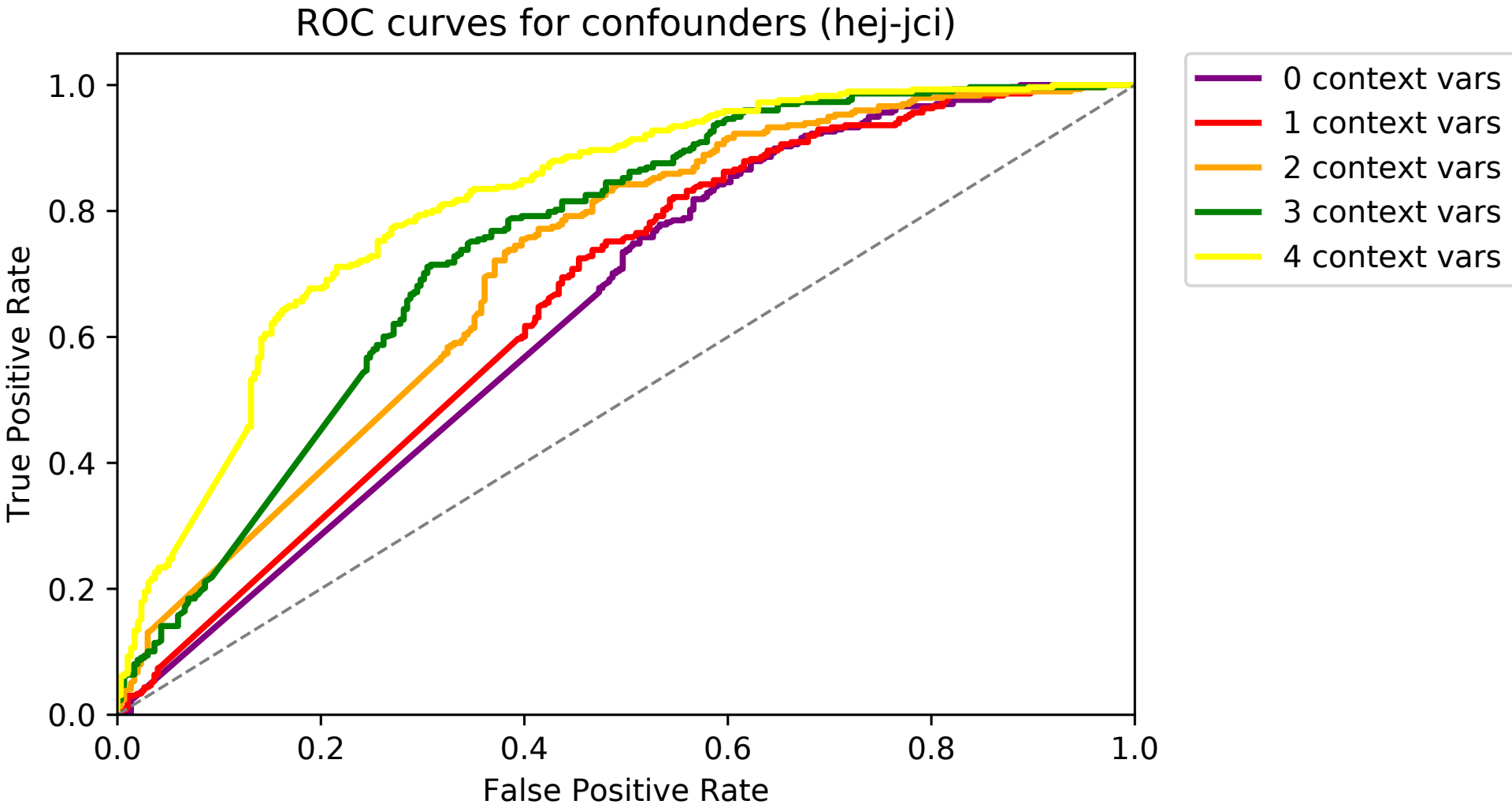
(4 system variables, 500 samples in each data set)

Evaluation on simulated data II



(4 system variables, 500 samples in each data set)

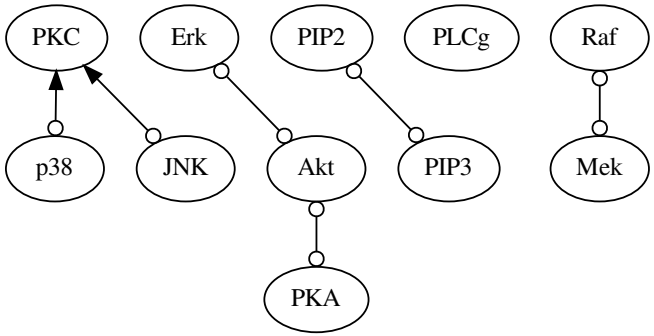
Evaluation on simulated data III



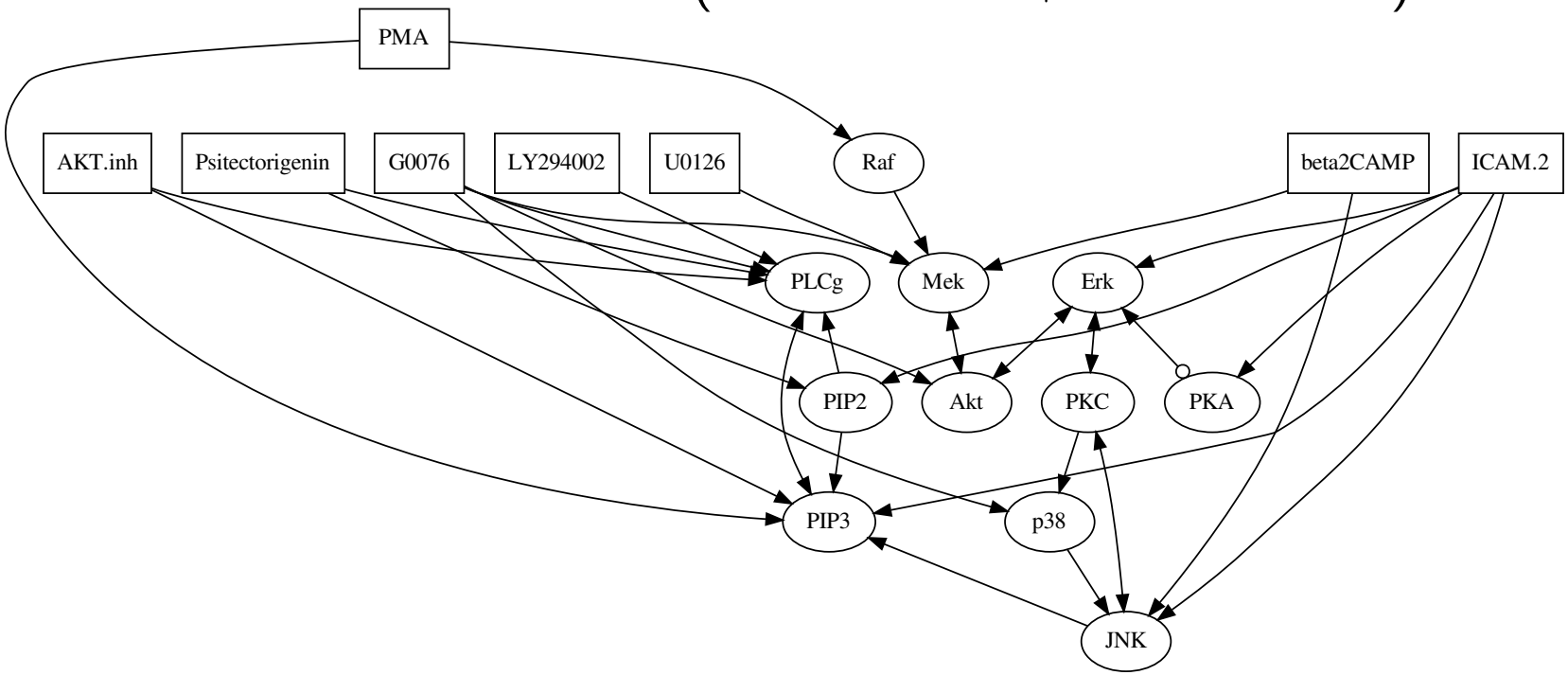
(4 system variables, 500 samples in each data set)

Evaluation on real-world flow cytometry data

Only observational data:



All (observational+interventional) data:



Contents

The generalized directed global Markov property

Given the importance of the Markov property, the first thing we need is a Markov property for cyclic SCMs.
We introduce a notion σ-separation that generalizes d-separation:

- σ-separation implies d-separation.
- For acyclic graph, σ-separation is equivalent to d-separation.

Inspired by ideas by [Spirtes, 1996], we show:

Theorem ([Forré and Mooij, 2017])

If an SCM $\mathcal{M}$ is uniquely solvable w.r.t. every strongly connected component in $\mathcal{G}(\mathcal{M})$, then the generalized directed global Markov property holds for any solution $\boldsymbol{X}$ of $\mathcal{M}$ with respect to the functional graph $\mathcal{G}(\mathcal{M})$:

$$A \overset{\sigma}{\underset{\mathcal{G}(\mathcal{M})}{\perp}} B \,|\, Z \implies \boldsymbol{X}_A \underset{\mathbb{P}^{\boldsymbol{X}}}{\perp\!\!\!\perp} \boldsymbol{X}_B \,|\, \boldsymbol{X}_Z \qquad A, B, Z \subseteq \mathcal{I}.$$

Markov properties: σ-separation

Definition (σ-separation, [Forré and Mooij, 2017])

In a DMG $\mathcal{G}$, a path

$$i_1 \; \begin{matrix} \leftarrow \\ \rightarrow \\ \leftrightarrow \end{matrix} \; \cdots \; \begin{matrix} \leftarrow \\ \rightarrow \\ \leftrightarrow \end{matrix} \; i_n$$

is called σ-blocked by a set of nodes Z iff

- one or both end nodes i_1, i_n are in Z, or
- it contains a collider $i_{k-1} \begin{matrix} \rightarrow \\ \leftrightarrow \end{matrix} i_k \begin{matrix} \leftarrow \\ \leftrightarrow \end{matrix} i_{k+1}$ with $i_k \notin \mathrm{an}_{\mathcal{G}}(Z)$, or
- it contains a non-collider with $i_k \in Z$:

$$i_{k-1} \begin{matrix} \rightarrow \\ \leftarrow \\ \leftrightarrow \end{matrix} i_k \rightarrow i_{k+1}, \quad i_{k-1} \leftarrow i_k \begin{matrix} \rightarrow \\ \leftarrow \\ \leftrightarrow \end{matrix} i_{k+1},$$

where the child i_{k+1} (resp. i_{k-1}) is not in $\mathrm{sc}_{\mathcal{G}}(i_k)$.

We say that A is σ-separated from B by Z, denoted $A \perp^{\sigma} B \mid Z$, if every path with one end node in A and one end node in B is σ-blocked by Z.

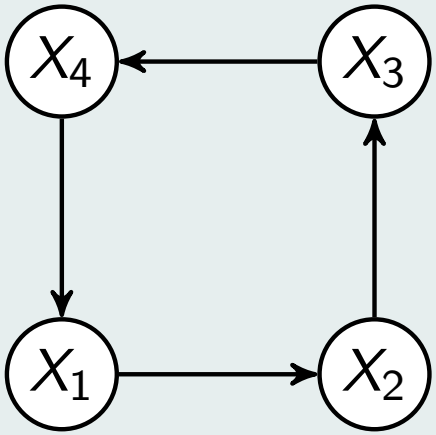
Markov properties: Example

Example

SCM $\mathcal{M}$:

$$
\begin{aligned}
X_1 &= f_1(X_4, E_1) = X_4 + E_1 \\
X_2 &= f_2(X_1, E_2) = X_1 \cdot E_2 \\
X_3 &= f_3(X_2, E_3) = X_2 + E_3 \\
X_4 &= f_4(X_3, E_4) = X_3 \cdot E_4
\end{aligned}
$$

Functional graph $\mathcal{G}(\mathcal{M})$:



$$X_1 \perp^d X_3 \,|\, X_2, X_4$$

but

$$X_1 \not\perp^\sigma X_3 \,|\, X_2, X_4$$

So for any solution $\boldsymbol{X}$ of the SCM $\mathcal{M}$, in general we do not have that $X_1 \perp\!\!\!\perp X_3 \,|\, X_2, X_4$.

In general: No σ-separations between nodes within the same strongly connected component.

Directed global Markov property

Stronger statements can be derived for special cases:

Theorem ([Forré and Mooij, 2017])

If an SCM $\mathcal{M}$ satisfies at least one of the following three conditions:

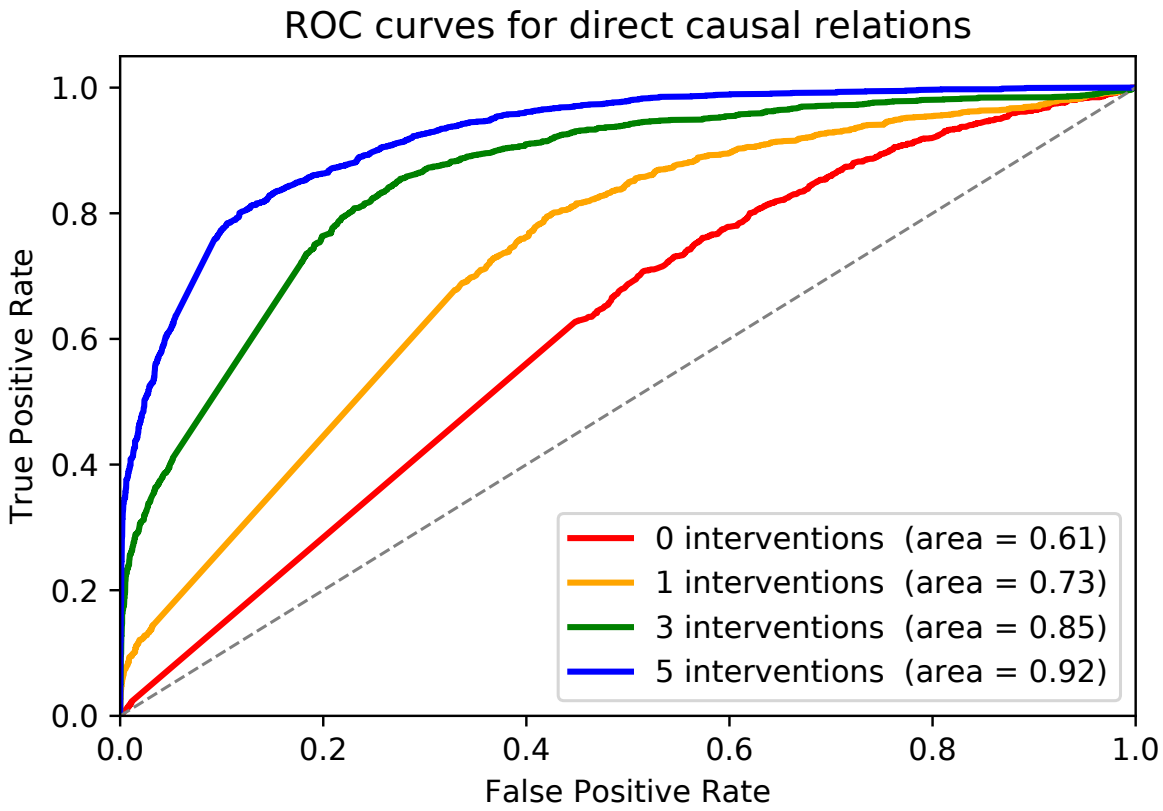
1. *$\mathcal{M}$ is linear, its exogenous variables have a density with respect to Lebesgue measure, and $\mathcal{M}$ is solvable w.r.t. $\mathcal{I}$;*
2. *all endogenous variables are discrete-valued, $\mathcal{M}$ is uniquely solvable w.r.t. each ancestral subgraph of $\mathcal{G}(\mathcal{M})$;*
3. *$\mathcal{M}$ is acyclic;*

then the directed global Markov property holds for any solution $\boldsymbol{X}$ of $\mathcal{M}$ with respect to the functional graph $\mathcal{G}(\mathcal{M})$:

$$A \overset{d}{\underset{\mathcal{G}(\mathcal{M})}{\perp}} B \,|\, Z \implies \boldsymbol{X}_A \underset{\mathbb{P}^{\boldsymbol{X}}}{\perp\!\!\!\perp} \boldsymbol{X}_B \,|\, \boldsymbol{X}_Z \qquad A, B, Z \subseteq \mathcal{I}.$$

Results on Synthetic Data [Forré and Mooij, 2018]

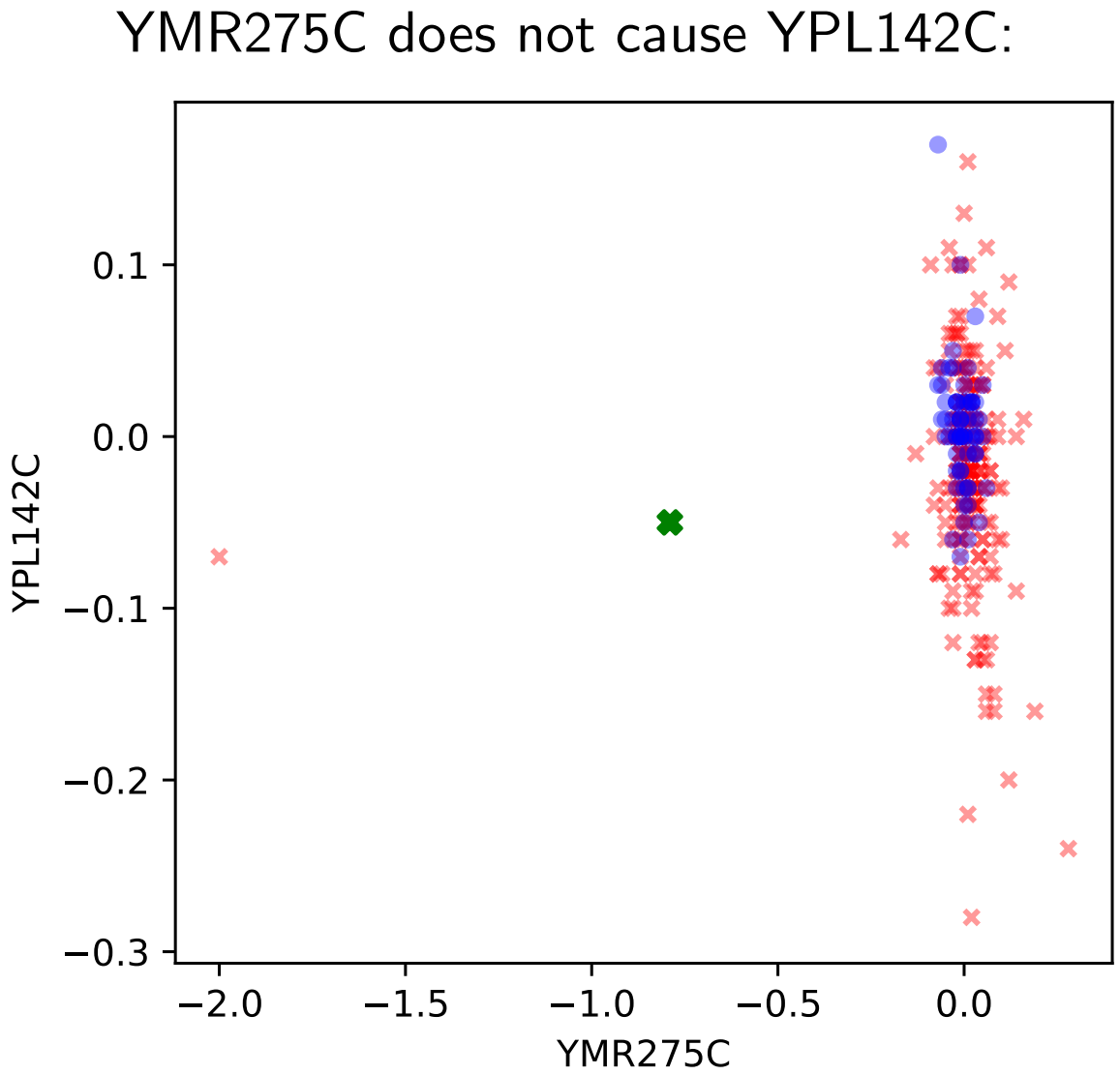
[Forré and Mooij, 2018]: the first causal discovery algorithm that can handle cycles, nonlinear relationships, latent confounding variables and data from different (interventional) contexts.



ROC curves for detecting direct causal relations from observational and interventional data, for varying numbers of interventional data sets.

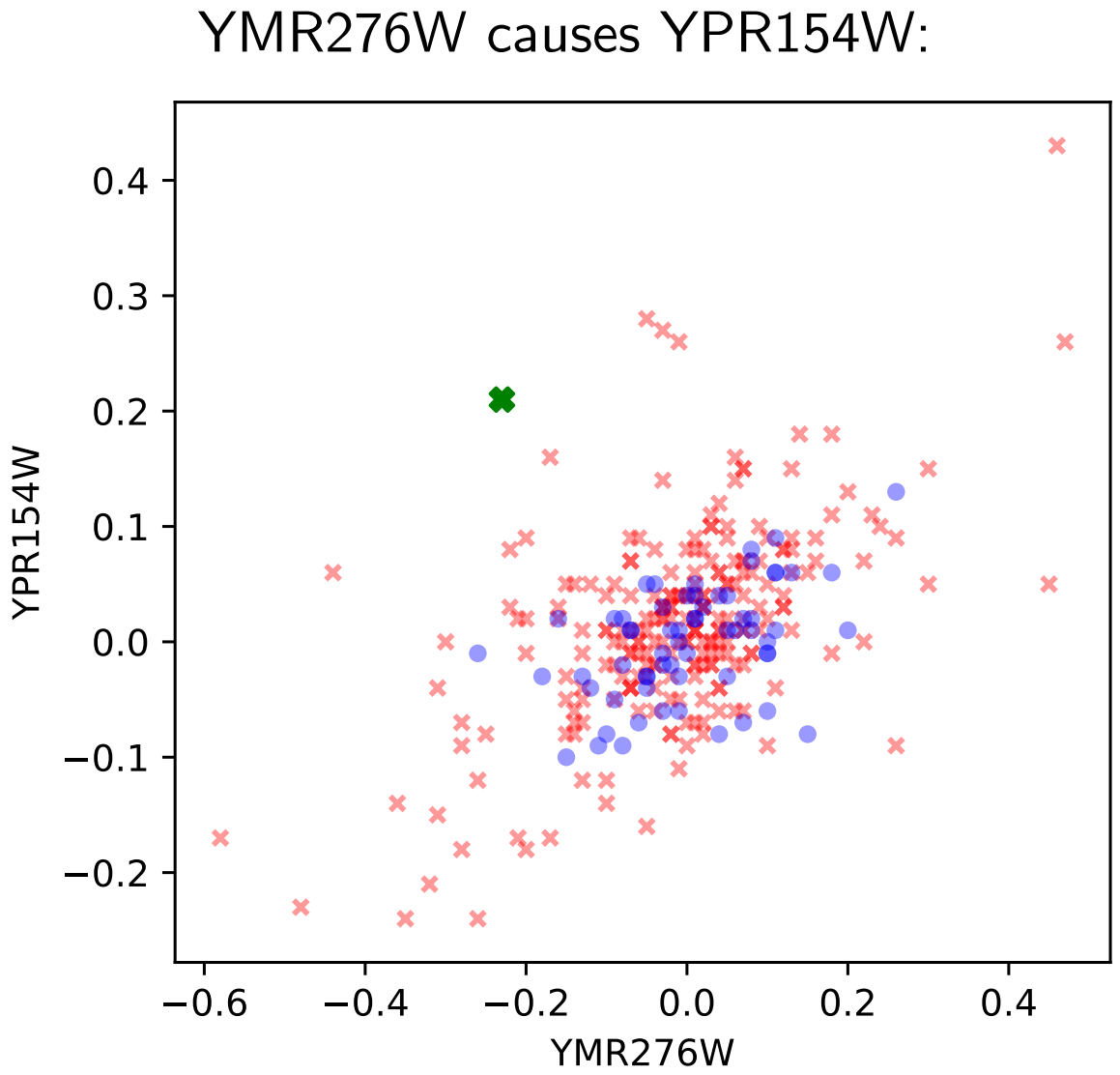
Contents

Example Gene Expression Data



Blue: observational; Red: interventional; Green: knockout of gene X.

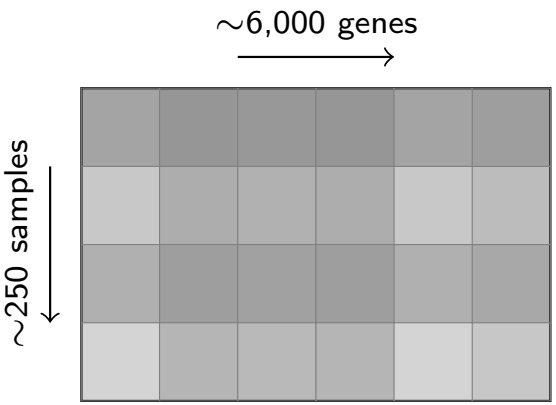
Example Gene Expression Data



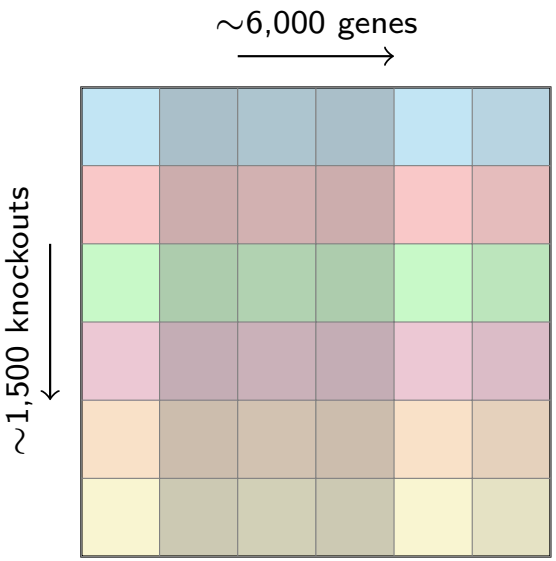
Blue: observational; Red: interventional; Green: knockout of gene X.

Causal Discovery from Large-Scale Micro-Array Data

Observational:



Interventional:



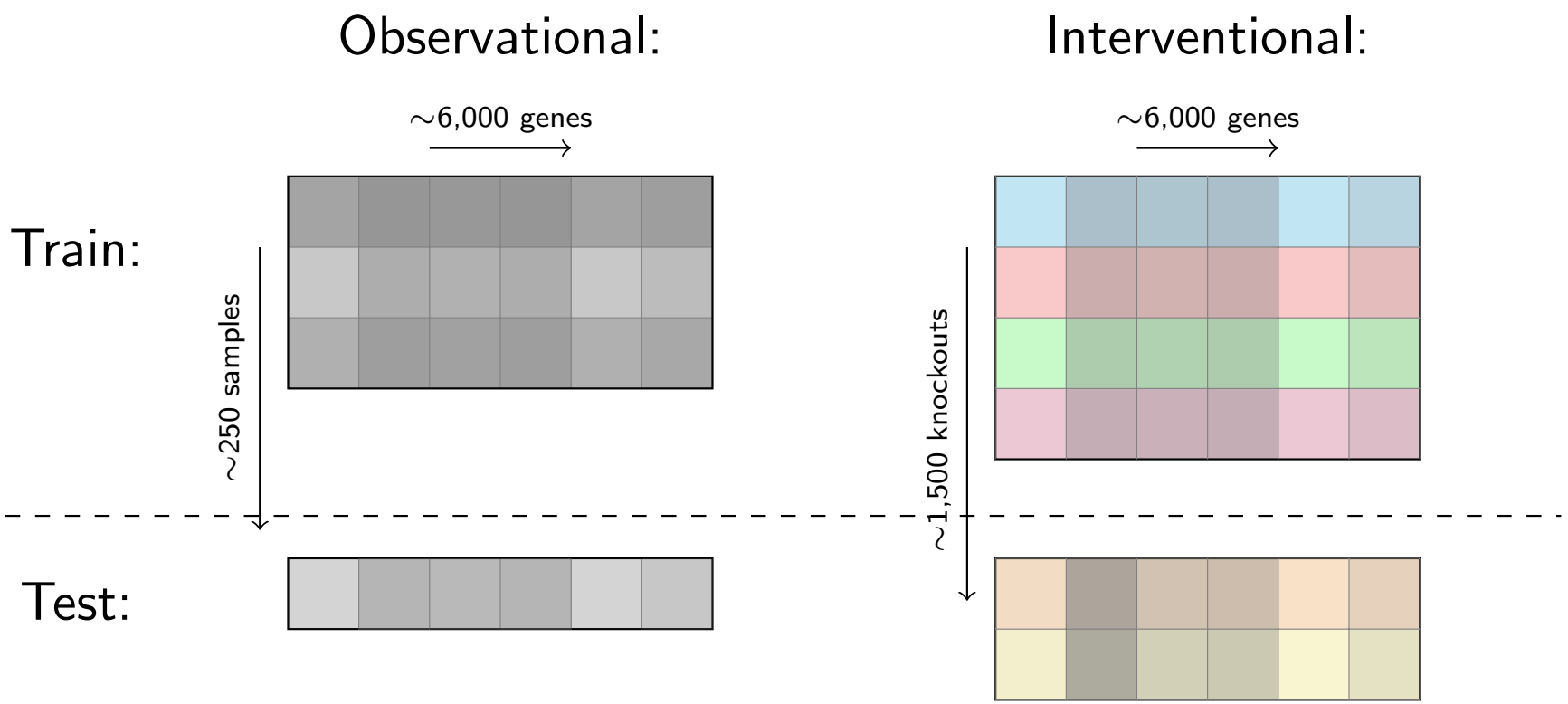
Large-scale Micro-Array Gene Expression Data (Kemmeren *et al.*, 2014):

- Variables $i \in \{1, \ldots, p\}$
  (*population gene expression levels*)
  $p = 6170$
- Observational samples X_{in}, $n = 1 \ldots N_{\text{obs}}$
  (*wild-type vs. wild-type*)
  $N_{\text{obs}} = 262$
- Interventional samples X_i^k, $k = 1 \ldots N_{\text{int}}$
  (*single-gene knockouts/knockdowns*)
  $N_{\text{int}} = 1462$
  one sample for every knocked out gene

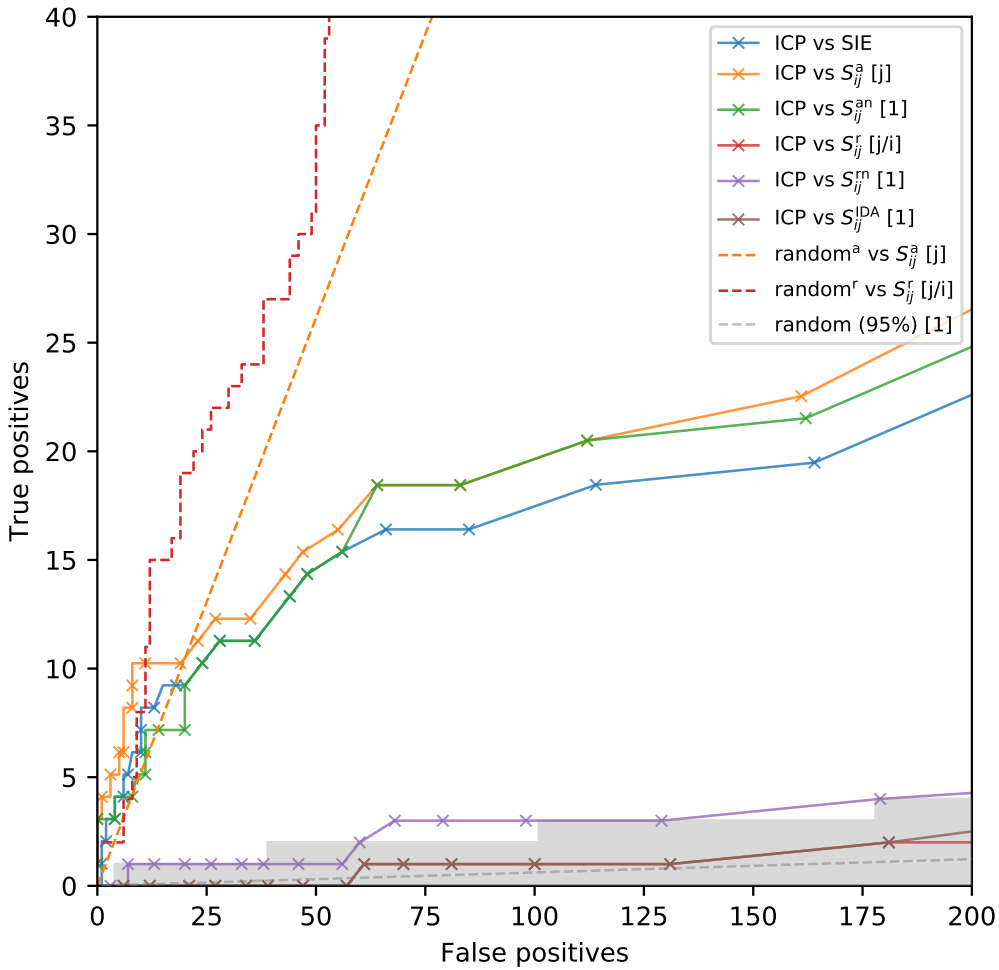
Task: Predict from the data which gene expression levels change when a certain gene is knocked out.

k-fold Cross-validation

Using 5-fold cross-validation, we split the data into a training set used to make predictions, and a test set used to define a ground truth for validating the predictions.



ICP Performance



ICP outperforms baselines (for the 0.61% strongest effects) for certain ground truth scores (absolute normalized, SIE)

Conclusion

Causality is clearly an important notion in daily life and in science, and yet underexplored in statistics and machine learning.

In this tutorial, you have learned how to:

- formalize the notion of causality;
- reason about causality;
- discover causal relations from data;
- make causal predictions
- that *seeing* is not the same as *doing*

This was just a sample of topics in an exciting research field. There is still much more to learn and to discover!

Further reading I

Bongers, S. and Mooij, J. M. (2018).

From random differential equations to structural causal models: the stochastic case.

arXiv.org preprint, arXiv:1803.08784 [cs.AI].

Bongers, S., Peters, J., Schölkopf, B., and Mooij, J. M. (2018).

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arXiv.org preprint, arXiv:1710.08775 [math.ST].

Further reading II

Forré, P. and Mooij, J. M. (2018).

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Mooij, J. M., Janzing, D., and Schölkopf, B. (2013).

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In Nicholson, A. and Smyth, P., editors, *Proceedings of the 29th Annual Conference on Uncertainty in Artificial Intelligence (UAI-13)*, pages 440–448. AUAI Press.

Pearl, J. (2000).

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Further reading III

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Wright, S. (1921).

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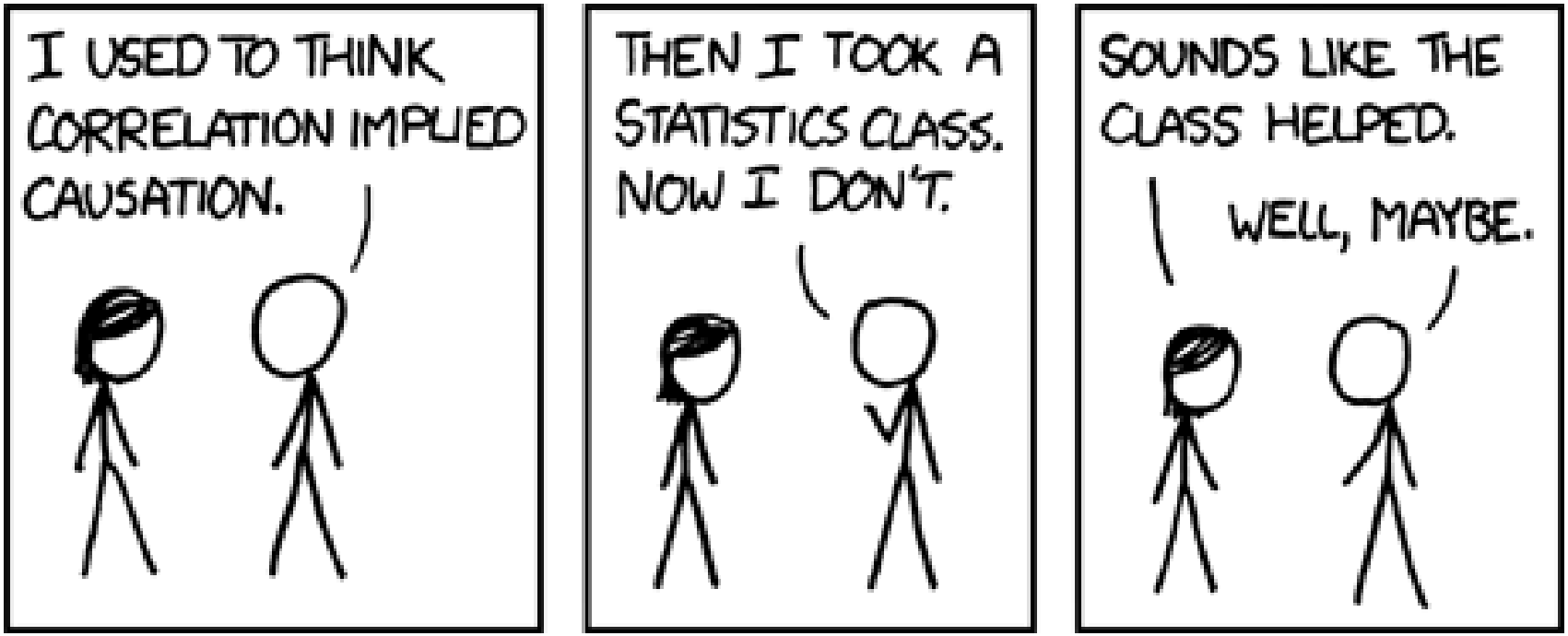
Thank you for your attention!



Randall Munroe, www.xkcd.org