# *Causal inference and discovery*

*Pierre Gentine + Adam Massmann + Jakob Runge*

# Causal Effects

# Introduction

Causal inference:

- Going beyond correlation, but at cause and effects
- Introduction to directed graphs
- Do calculus
- Transportability

# Introduction

Causality in many fields

- Economics
- Science
- Politics
- Medicine

# Introduction

## Causality in economics


There is a gender gap in earnings for the alumni at every top university, although the size of the difference varies greatly.Credit Matt Rourke/Associated Press

Gaps in Earnings Stand Out in Release of College Data

SEPT. 13, 2015

nytimes.com | Sept. 13, 2015

David Card. The causal effect of education on earnings (1999)

Conley and Heerwig. The Long-Term Effects of Military Conscription on Mortality: Estimates From the Vietnam-Era Draft Lottery (2012)

# Introduction

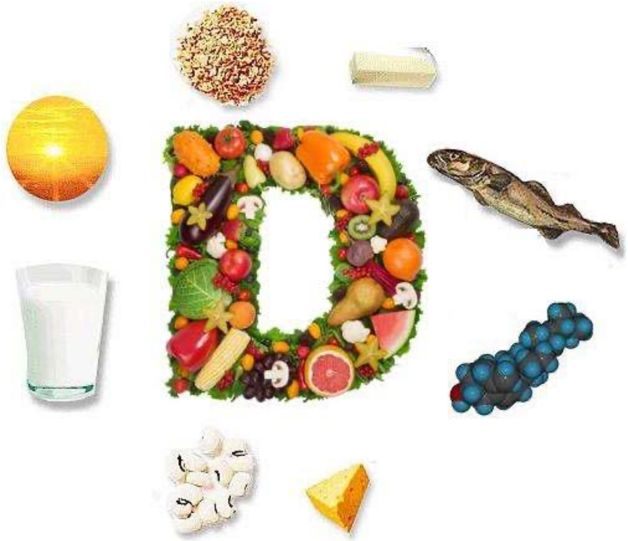
## Causality in human behavior

Thistlewaithe and Campbell. Effect of public recognition of scholastic achievement (1960)

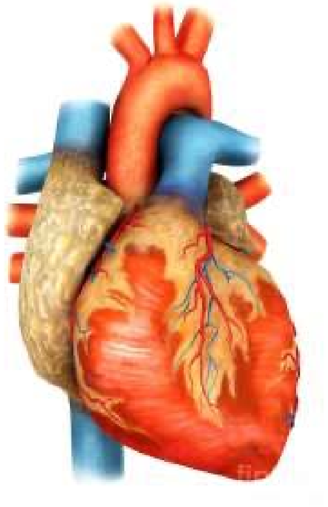
Christakis and Fowler. The collective dynamics of smoking in a large social network (2008)

# Introduction

## Causality in human behavior



Effect of Vitamin D deficiency on colon cancer



Effect of heart attack surgery on long-term health of patient

# Counterfactual reasoning

**Correlation question:** How well can X predict Y?

- Machine learning, Statistical estimation.

**Interventionist question:** If X is changed to X', what will be the value of Y?

- Experiments, Reinforcement learning, Contextual bandits.

**Counterfactual question:** If X would have been X', what would be the value of Y?

- Today's focus.

# Counterfactual reasoning

## Why is causal inference hard?

- Simpson's paradox

## The language of graphical models

- Backdoor criterion
- Frontdoor criterion

## Common approaches for causal inference

- Conditioning
- Mechanism-based
- Natural Experiments

## Example: Estimating causal impact of recommender systems

# Simpson's paradox

**Setup:**

There is a drug, which we call *drug 1*, that is supposed to lower the risk of having a heart attack. The following table summarizes data comparing a group of subjects that have and that have not taken the drug.[1]

| | Control Group (No Drug) | | Treatment Group (Took Drug) | |
|---|---|---|---|---|
| | Heart attack | No heart attack | Heart attack | No heart attack |
| Female | 1 | 19 | 3 | 37 |
| Male | 12 | 28 | 8 | 12 |
| Total | 13 | 47 | 11 | 49 |

**Question:**

Is it a good drug? That is, should one take this drug to reduce the risk of heart attack?

[1] Example taken from *J. Pearl, D. Mackenzie: The Book of Why. The New Science of Cause and Effect, Penguin Books (2018)*.

# Simpson's paradox

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**Risk of having a heart attack among female subjects**:

Risk in control group: $0.04 = \frac{1}{1+19}$

Risk in treatment group: $0.075 = \frac{3}{3+37}$

$\Rightarrow$ **Drug is bad for females**

# Simpson's paradox

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**Risk of having a heart attack among male subjects**:

Risk in control group: $0.30 = \frac{12}{12+28}$

Risk in treatment group: $0.40 = \frac{8}{8+12}$

$\Rightarrow$ **Drug is bad for males**

# Simpson's paradox

| | Control Group (No Drug) | | Treatment Group (Took Drug) | |
|---|---|---|---|---|
| | Heart attack | No heart attack | Heart attack | No heart attack |
| Female | 1 | 19 | 3 | 37 |
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**Risk of having a heart attack among all subjects**:

Risk in control group: $0.22 \simeq \frac{13}{13+47}$

Risk in treatment group: $0.18 \simeq \frac{11}{11+49}$

$\Rightarrow$ **Drug is good for people?**

# Simpson's paradox

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**Paradoxical finding**:

The drug is bad for females, bad for males, but good for people.

**This is clearly wrong!**

**What do you think? Is the drug good or bad?**

**Answer:**

**The drug is bad!**

# Simpson's paradox

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**How the numbers work out**:

- Males have a higher risk of heart attack.
- The proportion of males is higher in the control than in the treatment group

**But still: Why is the drug bad?**

The drug does not influence gender, but gender influences whether the drug is taken. This leads to a spurious, non-causal dependence, such that the effect has to be analyzed separately for female and male subjects.

# Simpson's paradox

**Setup:**

Now consider a second drug, *drug 2*. It is supposed to reduce the risk of heart attacks by lowering blood pressure. High blood pressure is a known cause of heart attacks.

| | Control Group (No Drug) | | Treatment Group (Took Drug) | |
|---|---|---|---|---|
| Blood pressure | Heart attack | No heart attack | Heart attack | No heart attack |
| Low | 1 | 19 | 3 | 37 |
| High | 12 | 28 | 8 | 12 |
| Total | 13 | 47 | 11 | 49 |

**Question:**

Is this a good drug, i.e., should one take this drug to reduce the risk of heart attack?

**Answer:**

**The drug is good!** Since the drug influences blood pressure we have to assess the drug based on all subjects.

# Simpson's paradox

- Questions about *effects* and *influences* cannot be answered from the data alone, hence these are not statistical questions.
- Such questions can only be answered when making additional assumptions about the data generating process, which are of causal nature.

General rule:

- There is a sharp division between statistical and causal questions.
- Answering causal questions always requires some causal assumptions.

# Simpson's paradox

Can we argue more conceptually why the data has to be analyzed differently for the two drugs?

**Drug 1: Analyze in subpopulations**

| | Control Group (No Drug) | | Treatment Group (Took Drug) | |
|---|---|---|---|---|
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| Female | 1 | 19 | 3 | 37 |
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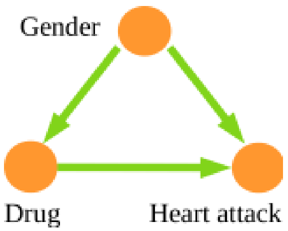
Direction of causal influence:




Must condition on *Gender* to **control for confounding** of *Drug* and *Heart attack*

# Simpson's paradox

Can we argue more conceptually why the data has to be analyzed differently for the two drugs?

**Drug 2: Analyze in full population**

| | Control Group (No Drug) | | Treatment Group (Took Drug) | |
|---|---|---|---|---|
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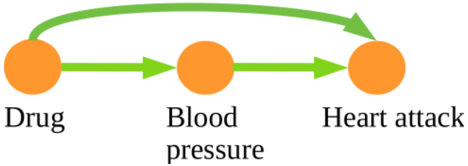
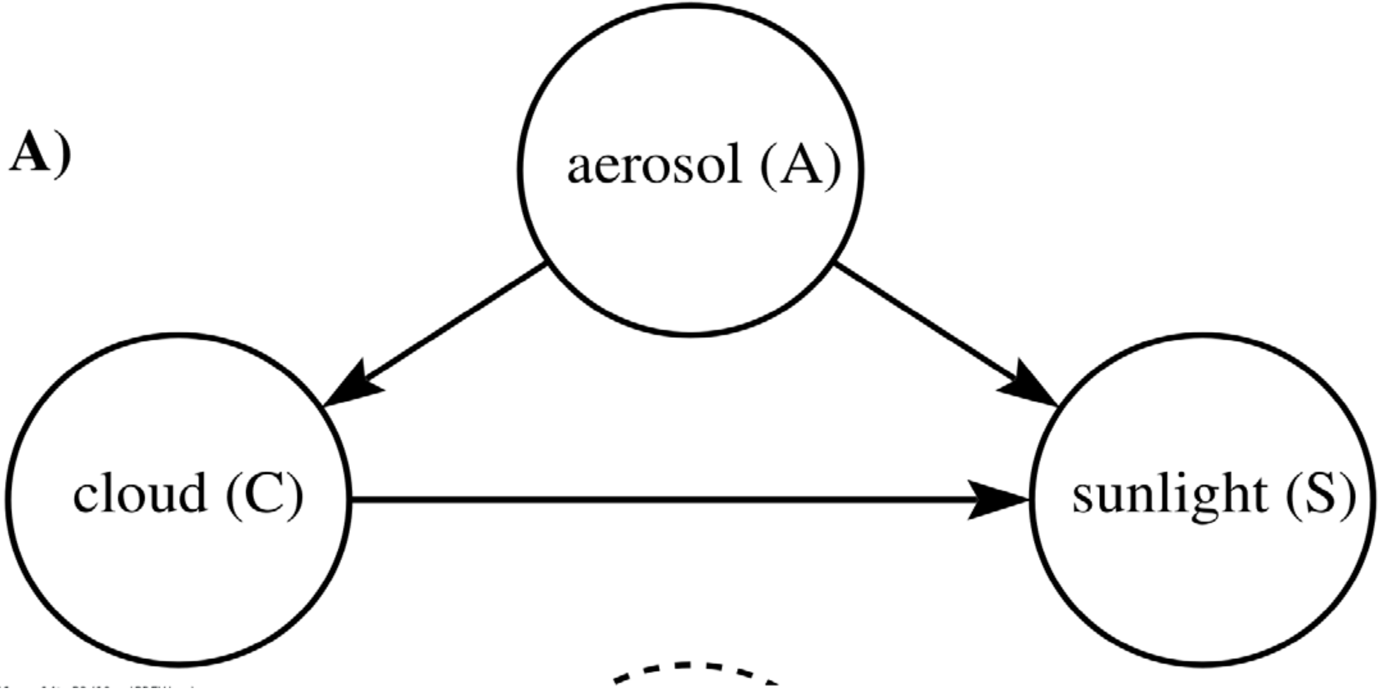



Must not condition on the *Blood pressure*, which **mediates** (part of) the effect of *Drug* on *Heart attack*.

# Graphs



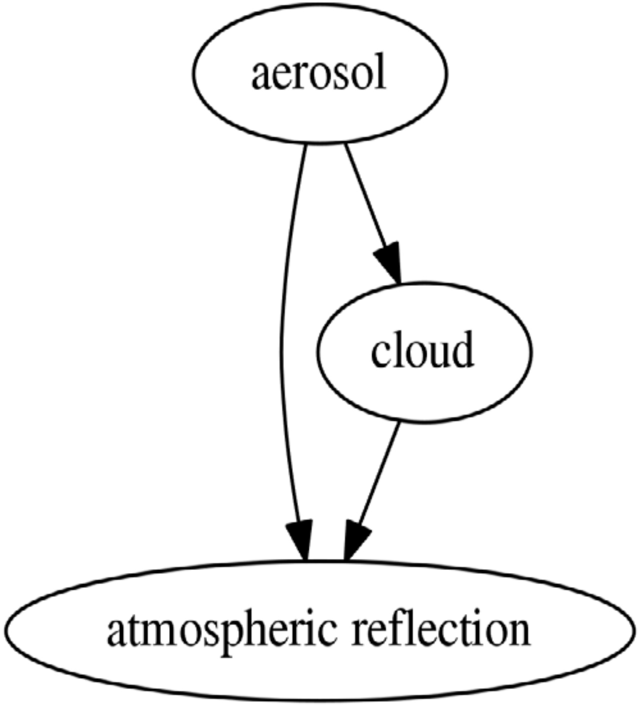

Graphs encode assumptions about causal dependencies in the system

# Probabilistic Graphs

Probabilistic graphical models: assumptions and communication

Probabilistic graphical models are **directed acyclic graphs (DAGs)** encoding assumptions about independence and dependence

Graphs are nice because they communicate a general meaning and a technical meaning:



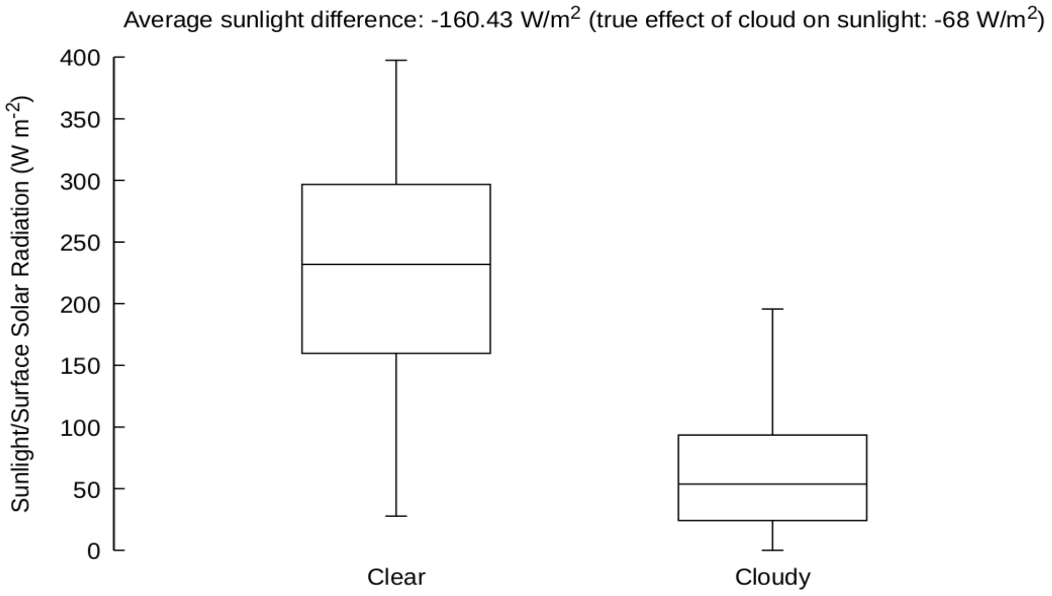

$$p(cloud,\ aerosol,\ reflection) = p(reflection \mid cloud,\ aerosol)\ p(cloud \mid aerosol)\ p(aerosol)$$

# Causal effects

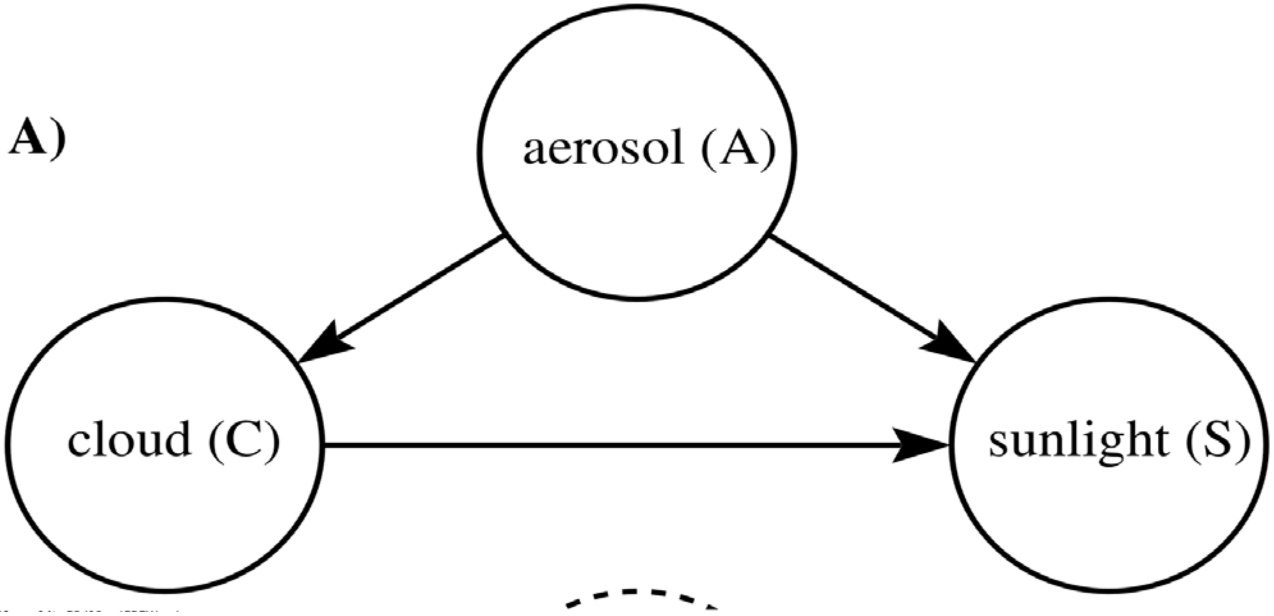
How would one estimate a causal effect of clouds on surface downwelling solar radiation?

# Causal effects

Naive approach (and/or when aerosol data are not available): bin observations by cloudy and clear day and compare the values of sunlight

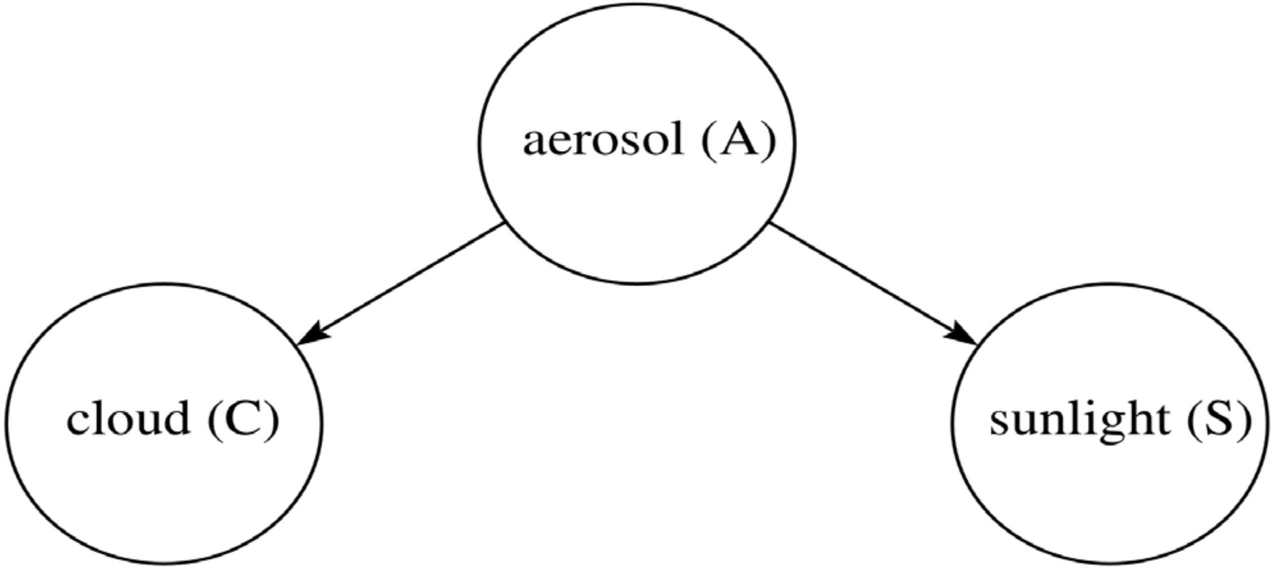


# What went wrong?



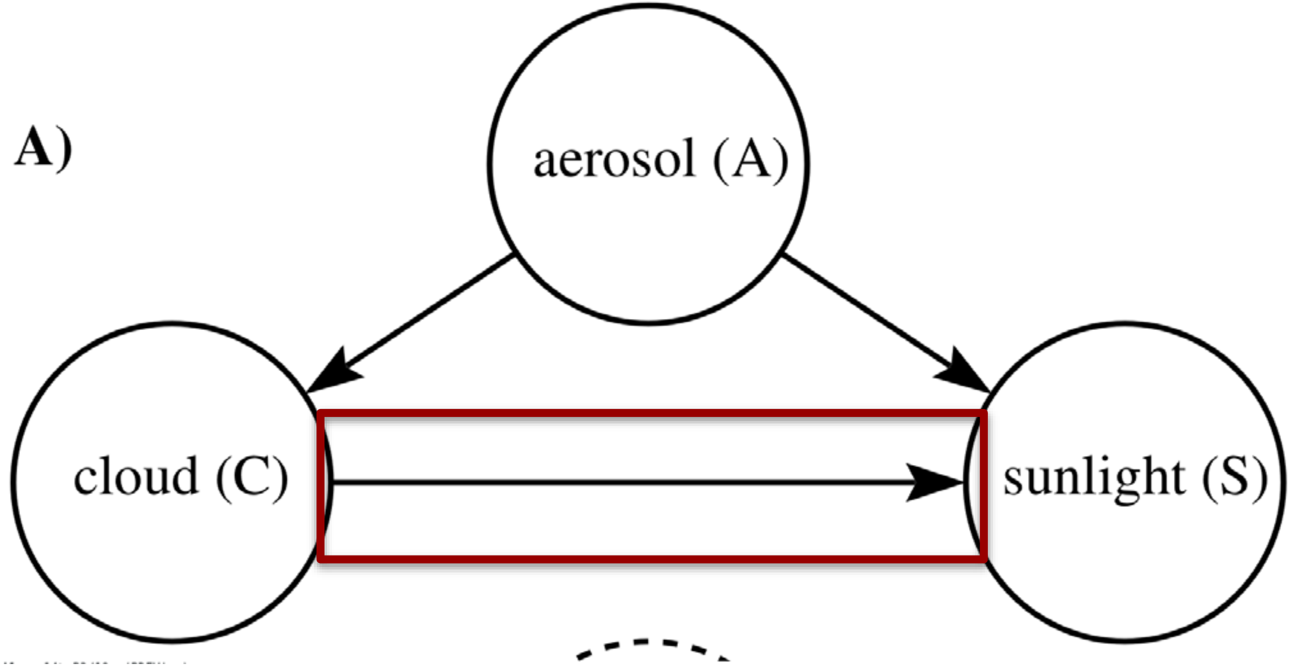
# Causal effects

Aerosol induces covariability in cloud and sunlight that has nothing to do with the causal link from cloud to aerosol!



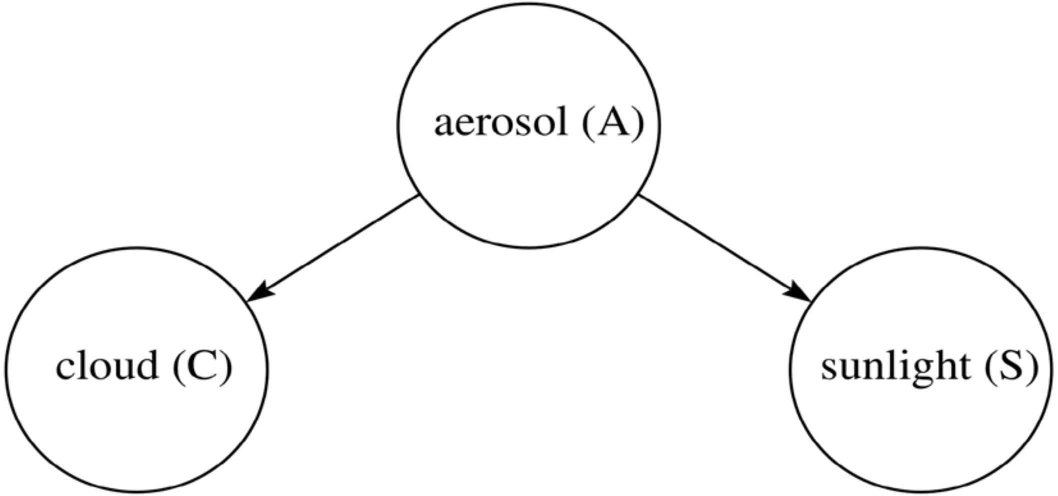
# Causal effects

Goal: we want to isolate the variability in aerosol due to the causal link from cloud



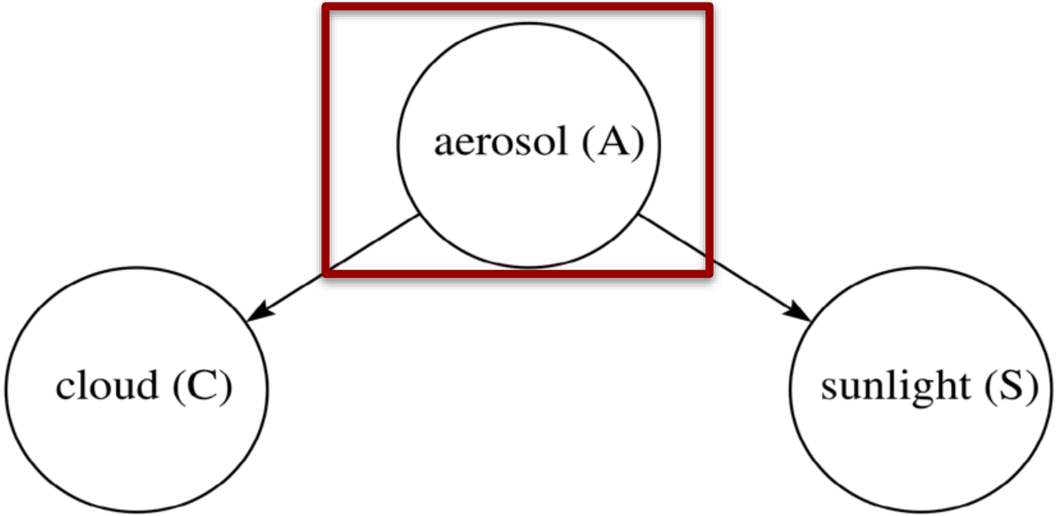
# Introduction

Intuitive graphical approach: remove causal paths from the cause to the children of causes and ask, are the cause and effect independent in the "mutilated" graph?



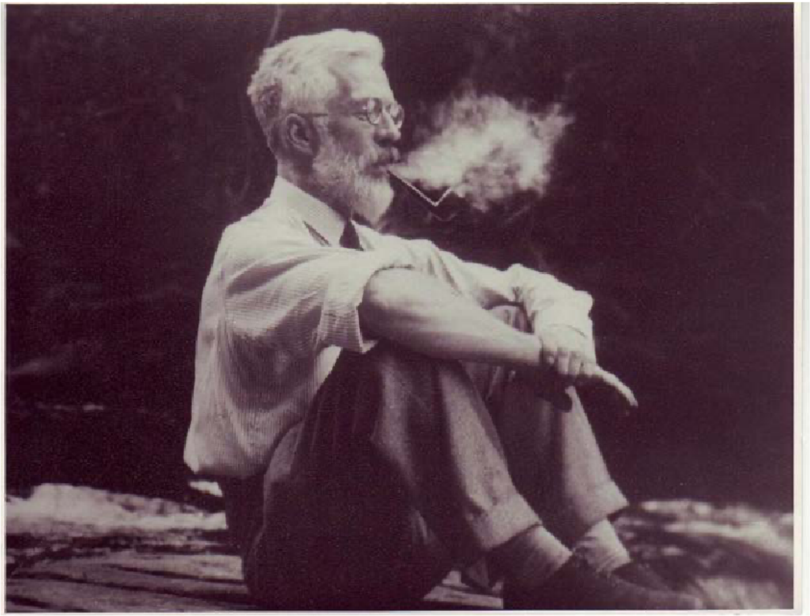
# Causal effects

If cause and effect are not independent, observe variables such that they are independent



# Causal effects

Does smoking cause lung cancer?



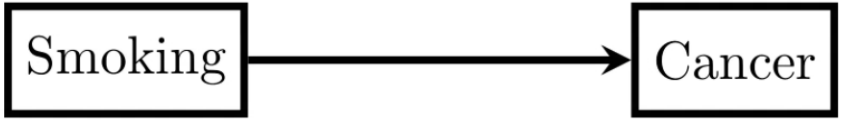
**Cancer and Smoking**

The curious associations with lung cancer found in relation to smoking habits do not, in the minds of some of us, lend themselves easily to the simple conclusion that the products of combustion reaching the surface of the bronchus induce, though after a long interval, the development of a cancer.

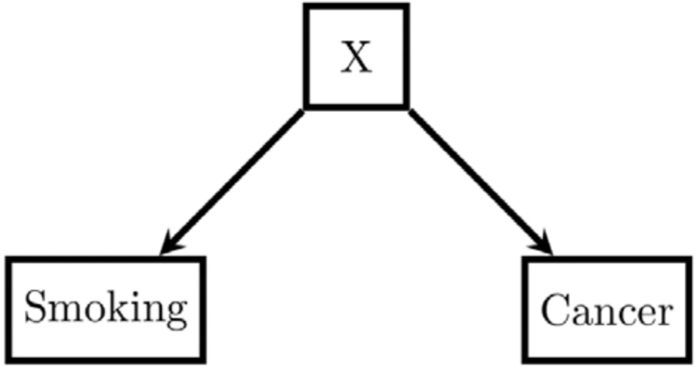
*Ronald A. Fisher*
*Nature 1958;182(4635):596.*

**Directed Acyclic Graph (DAG) to express causal relationships**

Smoking causes cancer




Patient characteristic X causes both smoking and cancer



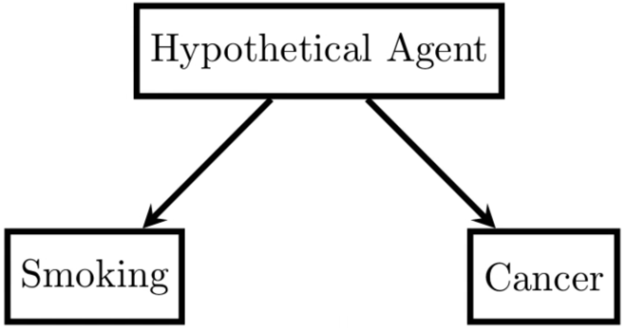
# Causal effects

## Smoking and lung cancer: recent evidence and a discussion of some questions*

Jerome Cornfield,[1] William Haenszel,[2] E. Cuyler Hammond,[3] Abraham M. Lilienfeld,[4] Michael B. Shimkin[5] and Ernst L. Wynder[6]

*J. Nat. Cancer Inst.* **22:**173–203, 1959

“The magnitude of the excess lung-cancer risk among cigarette smokers is so great that the results can not be interpreted as arising from an indirect association of cigarette smoking with some other agent or characteristic, since this hypothetical agent would have to be at least as strongly associated with lung cancer as cigarette use; no such agent has been found or suggested.“



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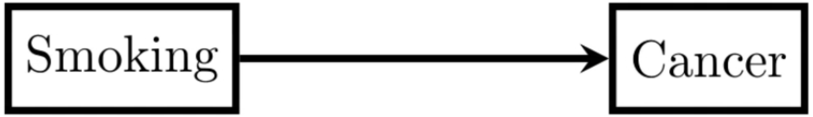
"The consistency of all the epidemiologic and experimental evidence also supports the conclusion of a causal relationship with cigarette smoking, while there are serious inconsistencies in reconciling the evidence with other hypotheses which have been advanced."



# Causal effects

Association, Prediction, Causality

- Carrying a lighter is strongly associated with lung cancer
- Whether or not somebody carries a lighter is predictive of lung cancer
- But this is not a causal relationship!

In some settings, having a good predictive model may be sufficient.

In others, causality is of main interest

# Randomized Control Trials

**Question:**

How can one, in general, determine whether a certain drug is helpful in reducing the risk of developing a certain desease?

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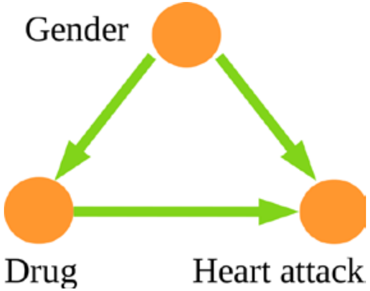

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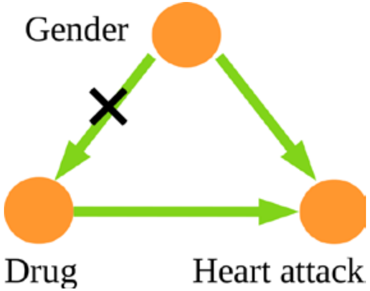

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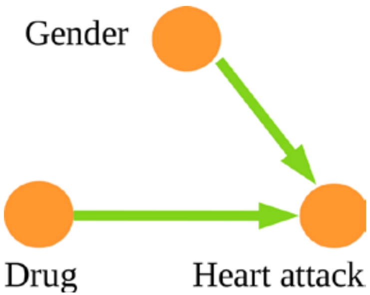


The gender of a subject does not longer influence whether a subject belongs to the treatment or control group.

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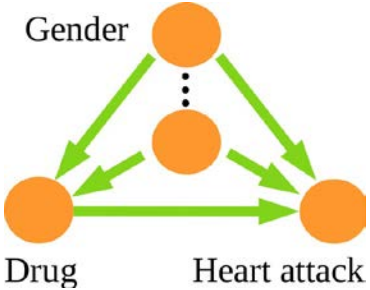

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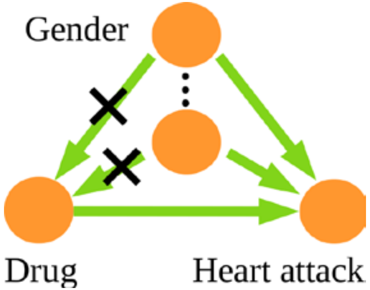

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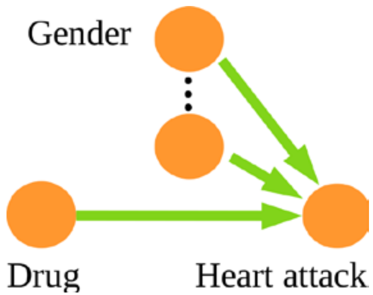


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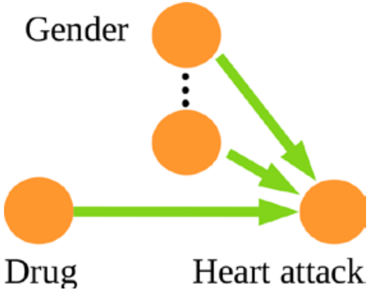


In fact, all confounders are eliminated! Therefore, **in RCT data, causal effects can be assessed by statistical proportions.**

# Experimentation

**Abstraction of the question:**
Consider two events or variables $X$ and $Y$. How can we determine whether $X$ causes $Y$?

**Experimentation:**
Experimentally manipulate $X$ while keeping all other conditions exactly the same. If this results in a change of $Y$, $X$ causes $Y$.

Such an idealization of experimental manipulation is referred to as an ***intervention on*** $X$.

# Experimentation

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Consider two events or variables $X$ and $Y$ . How can we determine whether $X$ causes $Y$ ?

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**Example: Lawn withsprinkler**

- Turning the sprinkler on makes the lawn wet.
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**Example: Lawn withsprinkler**

- Turning the sprinkler on makes the lawn wet.
- Making the lawn wet does not turn on the sprinkler.

We need to imagine the lawn being made wet without changing something else, in particular without turning the sprinkler on

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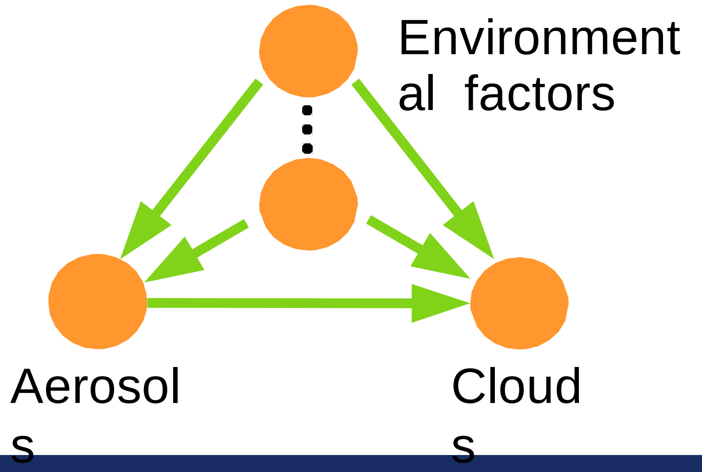

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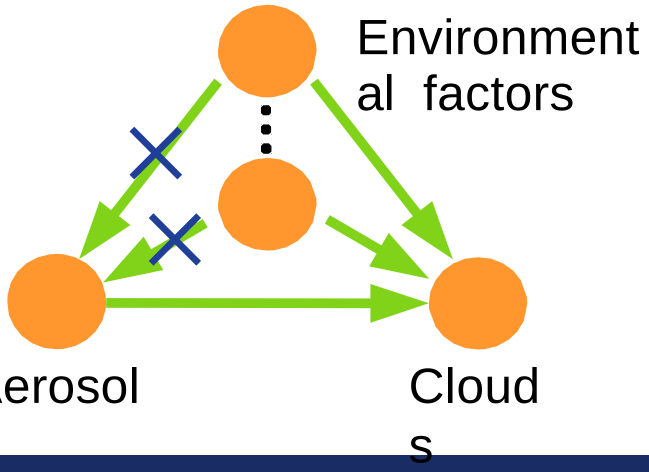

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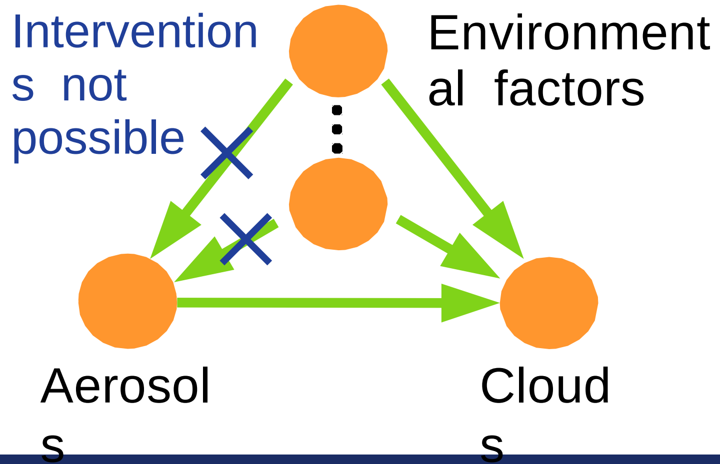

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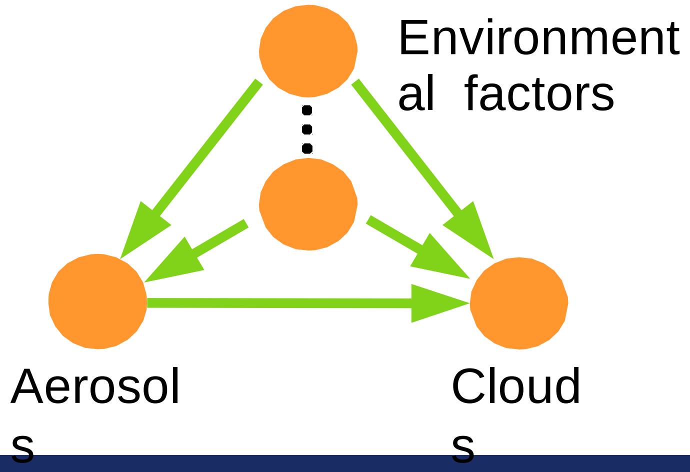

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Causal relationships are mostly known among microscopic physical variables, but usually less so among macroscopic variables.

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Causal relationships are mostly known among microscopic physical variables, but usually less so among macroscopic variables.

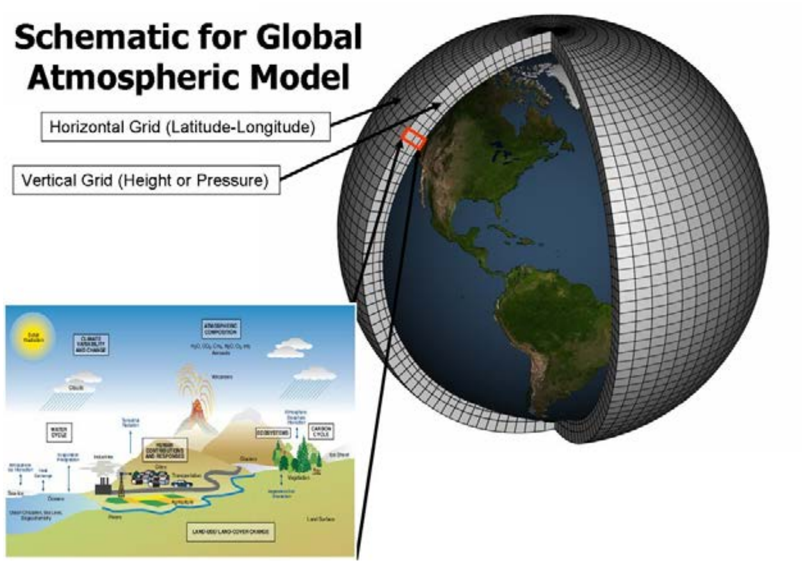
For example:

- Is there a causal relationship between arctic sea ice extend in winter and mid latitude weather in summer?
- Is there a causal relationship between ENSO and a certain extreme weather event somewhere on Earth?

# Two Approaches to Address These Challenges

**Simulation:**

Experimentation in climate models: Fix (intervene on) certain parameters and analyze the resulting effect on other parameters.



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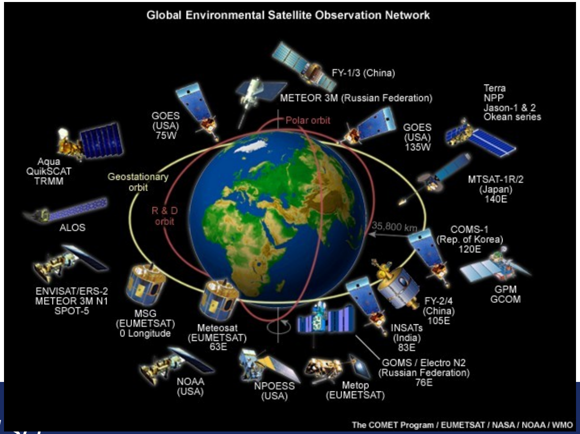

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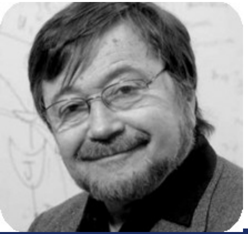
Seminal work by Judea Pearl, Peter Spirtes, Clark Glymour, Richard Scheines.

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*To mathematically treat causal inference, we need to formalize the idea of an underlying* **structural causal model (SCM)**, *define hypothetical* **interventions**, *and establish* **identifiability** *criteria to decide if and how causal effects can be estimated from data alone (without interventions).*

# Structural Causal Models

**Intuition:**

A structural causal model (SCM) specifies the functional causal relationships between a set of random variables.

**Example:**

$$X_{\text{aerosols}} = f_{\text{aerosols}}(X_{\text{env. facs.}}, \eta_{\text{aerosols}})$$

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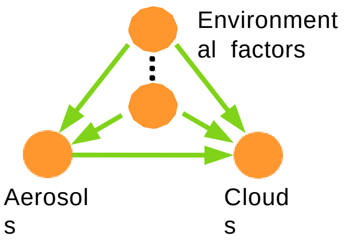

# Structural Causal Models

**Intuition:**

A structural causal model (SCM) specifies the functional causal relationships between a set of random variables.

**Example:**

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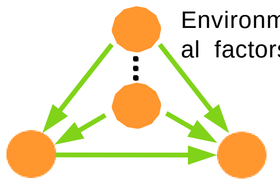


**Claims of an SCM:**

- Specifies the *direct causes*, also called *parents*, of each variable
- The functions $f_i$ are *independent mechanisms* by which nature determines the values of each variable based on the values of its direct causes plus random fluctuations



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# Structural Causal Models

**Definition:**

A structural causal model over the set of random variables $\mathbf{X} = \{X_1, \ldots, X_n\}$ consists of $n$ structural assignments

$$X_1 := f_1(PA_1, \eta_1)$$

$$\vdots$$

$$X_n := f_n(PA_n, \eta_n)$$

together with a specification of the probability distributions of the jointly independent 'noise' variables $\eta_i$. Here

$$PA_i \subset \{X_1, \ldots, X_{i-1}, X_{i+1}, X_n\} = \mathbf{X} \setminus \{X_i\}$$

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**Interpretation of noise variables:**

- Summarize all background factors outside causal model, i.e., all factors apart from $X_1, \ldots, X_n$
- Their joint independence means the model is sufficient to describe the causal relationship among the variables $X_1, \ldots, X_n$

# Causal Graphs

**Definition:**

Consider an SCM over the variables $\mathbf{X} = \{X_1, ..., X_n\}$. Its causal graph $G$ is the directed graph with

1. a vertex (node) for each variable $X_i$
2. for all $i$ a directed edge from each variable $X_j \in PA_i$ to $X_i$

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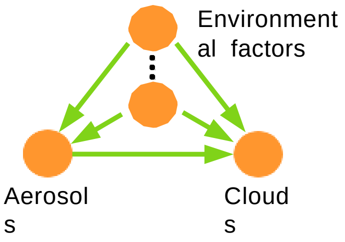

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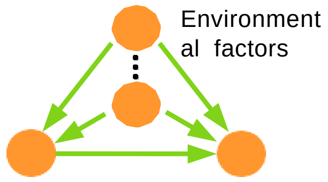


**Interpretation:**

- Nodes represent variables
- Edges represent causal influences

# Interventions

**Definition:**

Consider SCM over the variables $\mathbf{X} = \{X_1, \dots, X_n\}$.

An *intervention on* $X_i$, denoted as $do(X_i = x_{i,0})$, defines a modified SCM obtained by replacing the old assignment of $X_i$ with $X_i = x_{i,0}$ where $x_{i,0}$ is some value in the range of $X_i$:

$$
\begin{aligned}
X_1 &= f_1(PA_1, \eta_1) \\
&\vdots \\
X_i &= x_{i,0} \\
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\end{aligned}
$$

**Interpretation:**

- An intervention $do(X_i := x_{i,0})$ inactivates the natural mechanism $X_i = f_i(PA_i, \eta_i)$ by forcing $X_i$ to take the value $x_{i,0}$
- All other mechanisms remain unmodified
- Interventions can also be *more complex functions* than just $x_{i,0}$

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**Interventions model the notion of 'experimentally manipulate $X_i$ while keeping all other conditions exactly the same'**

**Effect on causal graphs:**

The causal graph of the intervened SCM is obtained from the causal graph of the original SCM by deleting all arrows pointing into $X_i$.

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Original SCM prior to intervention:

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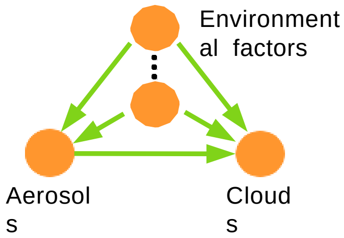

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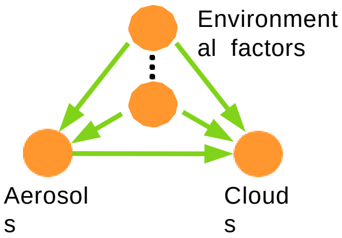


After intervention $do(X_{\text{aerosols}})$ := a certain aerosol concentration:

$X_{\text{aerosols}}$ = *a certain aerosol concentration*

$X_{\text{clouds}} = f_{\text{clouds}}(X_{\text{aerosols}}, X_{\text{env. facs.}}, \eta_{\text{clouds}})$

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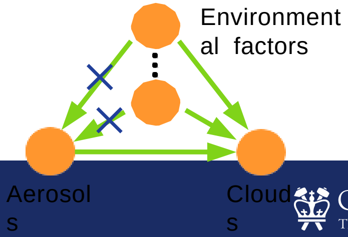

# Identification of Causal Effects

**General formalism:**

- Formally define the notion of *causal effect*.
- Based on the causal graph, decide whether the causal effect of $X$ on $Y$ can be identified from the observational data.
- If the effect can be identified, express causal effect in terms of observational data

# Identification of Causal Effects

**Example 1: No confounding**

Consider the linear SCM:

$$Z = \eta_Z,$$

$$X = \eta_X,$$

$$Y = a \cdot X + b \cdot Z + \eta_Y,$$

$$\eta_Z \sim N(0, \sigma_Z^2)$$

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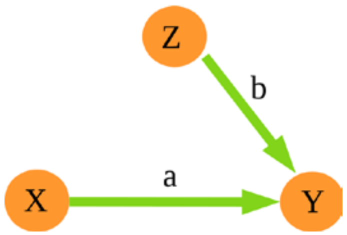

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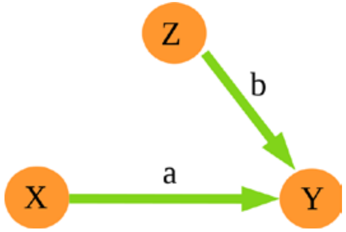


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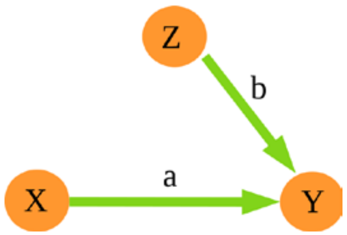


Goal: Determine $a$, the causal effect of $X$ on $Y$

Idea: Use covariance of $Y$ on $X$

$$
\begin{aligned}
Cov(Y, X) &= Cov(a \cdot \eta_X + b \cdot \eta_Z + \eta_Y, \eta_X,) \\
&= a \cdot Cov(\eta_X, \eta_X) + b \cdot Cov(\eta_Z, \eta_X) + Cov(\eta_Y, \eta_Z) \\
&= a \cdot Var(X) + b \cdot 0 + 0
\end{aligned}
$$

$$
\Rightarrow a = \frac{Cov(Y, X)}{Var(X)} = \rho(Y, X) \cdot \sqrt{\frac{Var(Y)}{Var(X)}}
$$

**Example 2: Observed confounding**

Consider the linear SCM:

$$Z = \eta_Z, \qquad \eta_Z \sim N(0, \sigma_Z^2)$$

$$X = c \cdot Z + \eta_X, \qquad \eta_X \sim N(0, \sigma_X^2)$$

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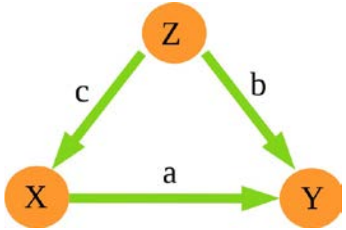

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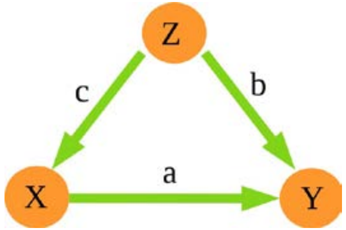


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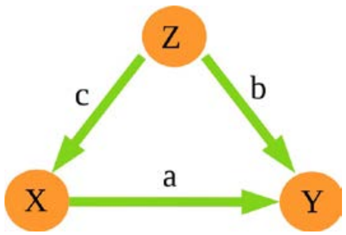



Goal: Determine $a$, the causal effect of $X$ on $Y$

First attempt: Linearly regress of $Y$ on $X$

$$\begin{aligned} Cov(Y, X) &= Cov((a \cdot c + b) \cdot \eta_Z + a \cdot \eta_X + \eta_Y, c \cdot \eta_Z + \eta_X) \\ &= c \cdot (a \cdot c + b) \cdot Cov(\eta_Z, \eta_Z) + a \cdot Cov(\eta_X, \eta_X) \end{aligned}$$

$$\begin{aligned} Var(X) &= Cov(c \cdot \eta_Z + \eta_X, c \cdot \eta_Z + \eta_X) \\ &= c^2 \cdot Cov(\eta_Z, \eta_Z) + Cov(\eta_X, \eta_X) \end{aligned}$$

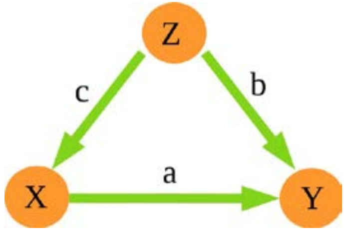
$$\Rightarrow a \neq \frac{Cov(Y, X)}{Var(X)}$$

# Identification of Causal Effects

**Example 2: Observed confounding**

Second attempt:

- Control for $Z$ : Regress out influence of $Z$ on $X$ and $Y$ to obtain residuals $\Delta X$ and $\Delta Y$
- Linearly regress $\Delta Y$ on $\Delta X$



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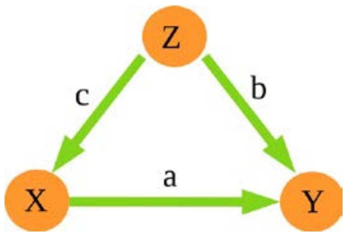


$$\begin{aligned}\Delta X &= X - \frac{Cov(X, Z)}{Var(Z)} \cdot Z \\ &= X - \frac{c \cdot Cov(\eta_Z, \eta_Z)}{Cov(\eta_Z, \eta_Z)} \cdot Z \\ &= X - c \cdot Z \\ &= \eta_X\end{aligned}$$

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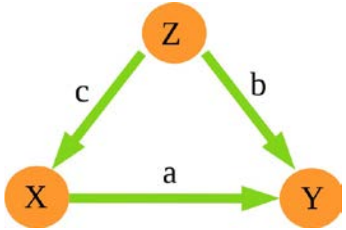


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$$\begin{aligned}\Delta Y &= Y - \frac{Cov(Y,Z)}{Var(Z)} \cdot Z \\ &= Y - \frac{(a \cdot c + b) Cov(\eta_Z, \eta_Z)}{Cov(\eta_Z, \eta_Z)} \cdot Z \\ &= X - (a \cdot c + b) \cdot Z \\ &= a \cdot \eta_X + \eta_Y\end{aligned}$$

**Example 3: Partial mediation**

Consider the linear SCM:

$$X = \eta_X, \qquad \eta_X \sim N(0, \sigma_X^2)$$

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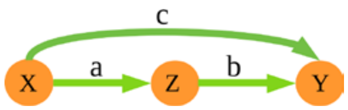

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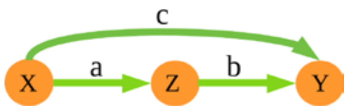



Total causal effect of $X$ on $Y$: $(a \cdot b + c)$

- Linearly regress $Y$ on $X$

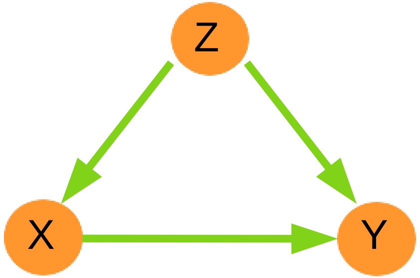
Direct causal effect of $X$ on $Y$: $c$

- Control for $Z$ by regressing out influence of $Z$ on $X$ and $Y$ to obtain residuals $\Delta X$ and $\Delta Y$
- Linearly regress $\Delta Y$ on $\Delta X$
- Same as *multivariate regression* of $Y$ on its parents $(X, Z)$

# Conceptual Findings

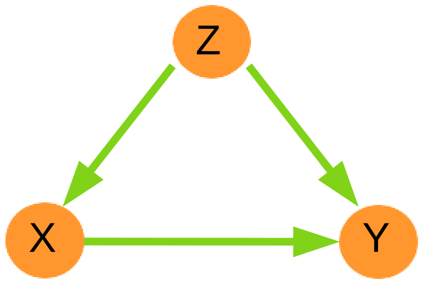
- Interventions model the notion of ‘experimentally manipulate $X_i$ while keeping all other conditions exactly the same’.
- The causal graph allows us to decide whether a certain causal effect is identifiable.
- If the causal effect is identifiable, the causal graph further allows us to estimate it from observational data.

## Example: Observed confounder



# Overview: Identification of Causal Effect

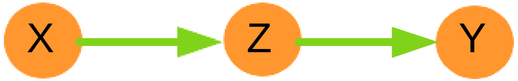
**Example: Observed confounder**




**Control for confounder** $Z$

# Overview: Identification of Causal Effect

## Example: Mediation



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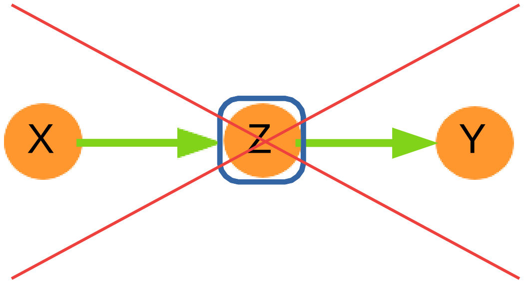
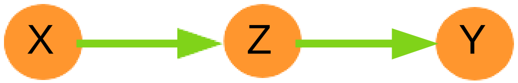


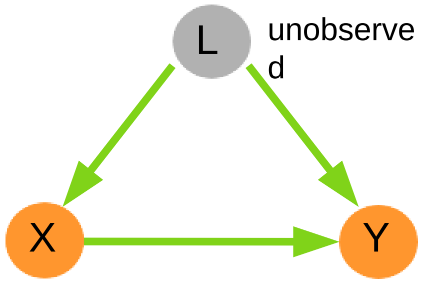
**Example:**
**Mediation**




**Do not control for mediator *Z***

# Overview: Identification of Causal Effect

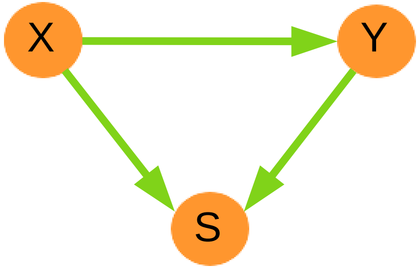
**Example: Unobserved confounder**




**Causal effect cannot be identified**

# Overview: Identification of Causal Effect

## Example: Common effect



# Overview: Identification of Causal Effect

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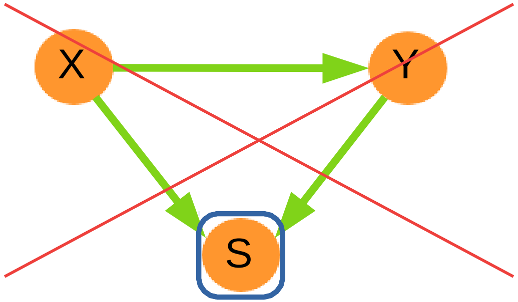
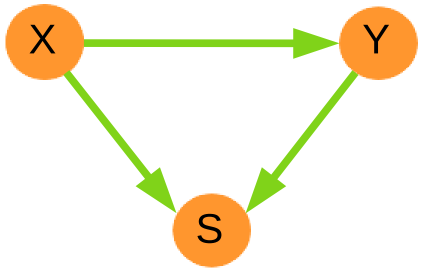
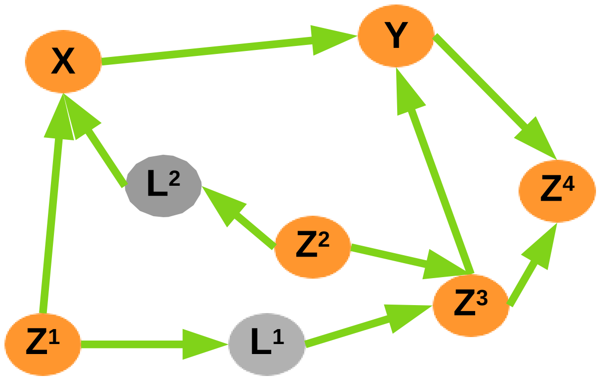


**Example: Common effect**




**Do not control for common effects of $X$ and $Y$, this would introduce bias.**
**However, sometimes the available dataset is already biased, e.g., if samples are missing (selection bias).**

**Example: Complicated case**




**Back-door criterion:**

**Block all non-causal dependence by controlling for an appropriate set of variables**

# Overview: Identification of Causal Effect

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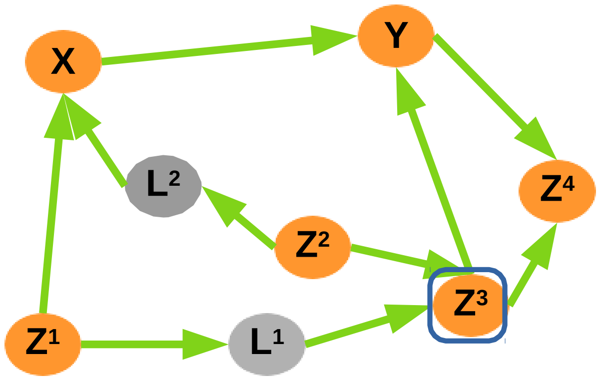


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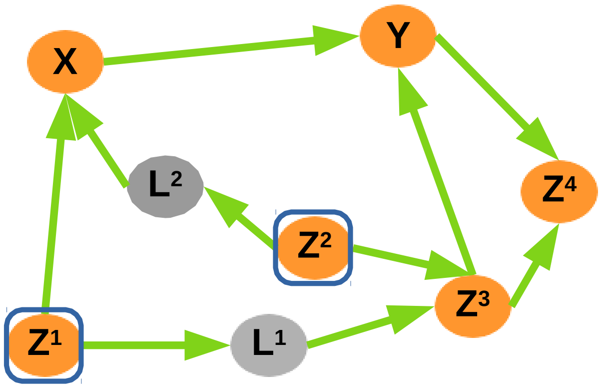
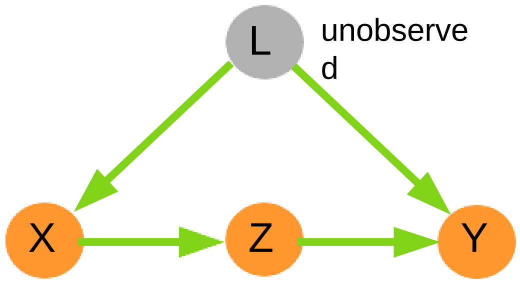



**Back-door criterion:**

**Block all non-causal dependence by controlling for an appropriate set of variables**

**Example: Unobserved confounder plus mediation**




**Front-door criterion:**
**Allows to identify causal effect, but not as simple as controlling for a certain set of variables**

# Observational Causal Discovery

# How to Get Causal Graphs

**Relevance: Result of previous section**

Knowing the causal graph of the data generating process, we can determine whether and how a causal effect can be identified from observational data

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**How to get the causal graph?**

**Option 1: Scientific knowledge**

Talk to domain experts, general reasoning

**Option 2: Observational causal discovery**

Learn from observational data

# Causal Graphs and (Conditional) Independencies

**Fact:**

The structure of the causal graph often has observable implications in terms of (conditional) independencies in the observed data.

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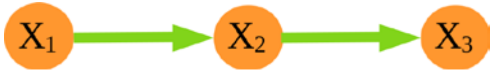


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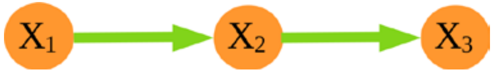


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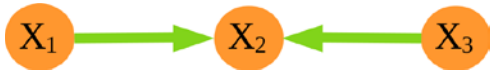


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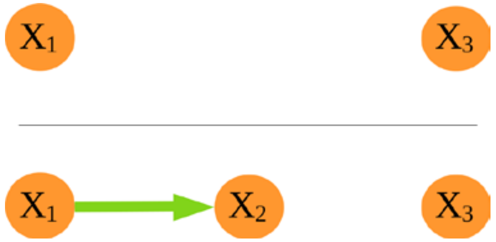
# Causal Graphs and (Conditional) Independencies

## Example 4: Disconnected variables

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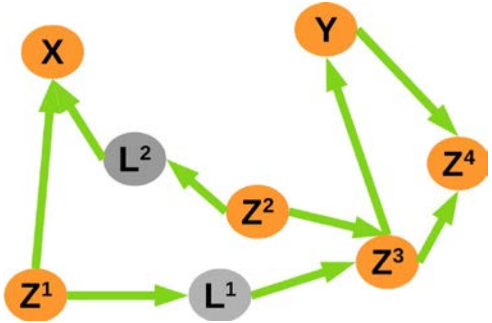
# Causal Graphs and (Conditional) Independencies

**General rule: d-separation**

The graphical criterion of *d-separation* allows to read off all (conditional) independencies implied by the structure of a particular causal graph.

*d-separation:* Whether a set X of variables is independent of another set Y, given a third set Z. The idea is to associate "dependence" with "connectedness" (i.e., the existence of a connecting path) and "independence" with "unconnected-ness" or "separation".

**Example 5: Complicated case**




Some independencies implied by d-separation

$$X \perp\!\!\!\perp Y | Z^1, Z^2$$

$$X \perp\!\!\!\perp Y | Z^3$$

# Constraint Based Causal Discovery

**Idea:**

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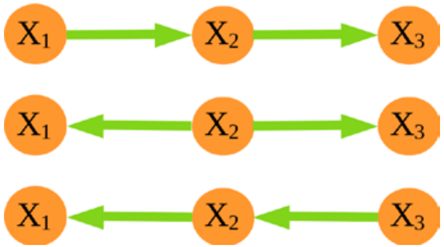

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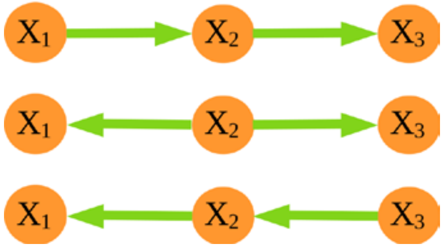


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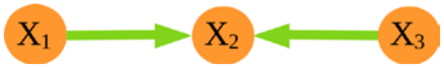


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Causal questions cannot be answered from observational data without making any additional assumptions.

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The data generating process can be described as an SCM. This implis the so called *causal Markov condition*:

d-separation in causal graph ⇒ (conditional) independence

**Assumption 2: Faithfulness**

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Assumption 3 can be dropped (but much more complicated)

Assumption 2 can be relaxed

# Some Famous Algorithms

**SGS-Algorithm:**
Tests all possible (conditional) independence statements, use results to constrain the causal graph as discussed in the examples.

*SGS* stands for *S* pirtes, *G* lymour, and *S* cheines.

**PC-Algorithm:**
Based on SGS-Algorithm, but using much fewer conditional independence tests.

*PC* stands for *P*eter Spirtes and *C*lark Glymour.

**FCI-Algorithm:**
Similar to the PC-Algorithm, but without requiring the assumption of causal sufficiency, i.e., allowing for hidden confounders and selection variabes.

*FCI* stands for *f* ast causal *i* nference.

# Two Main Approaches of Causal Discovery

**Constraint-based causal discovery:**
Constrain causal graph by using results of conditional independence tests in observational data.

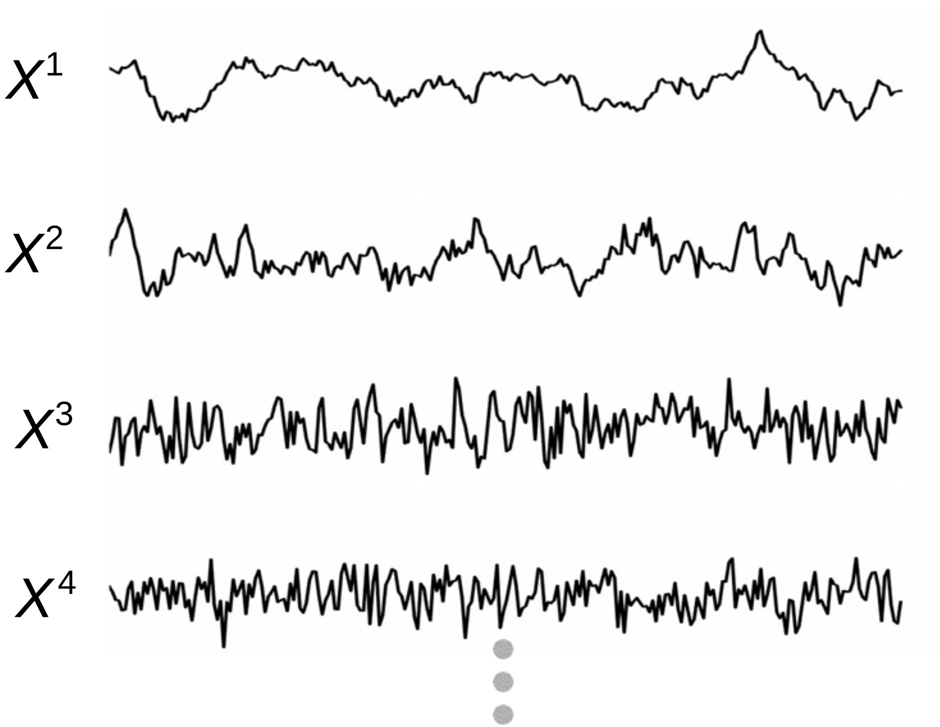
⇒ discussed

**SCM-based causal discovery:**
Make assumption on functional causal relationships (e.g., linear or non-linear) and noise distributions (e.g., Gaussian or non-Gaussian) of data generating SCM.

Generically, model can fit in one direction only. This allows to identify direction of causal influence.

⇒ not discussed

# Causal Discovery in Time Series



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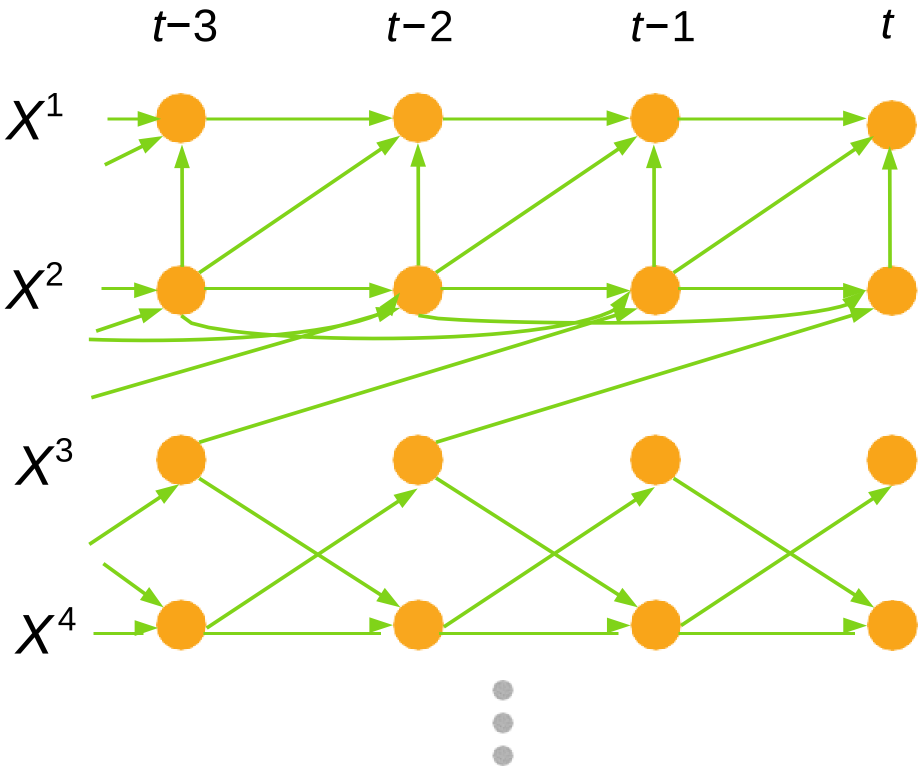


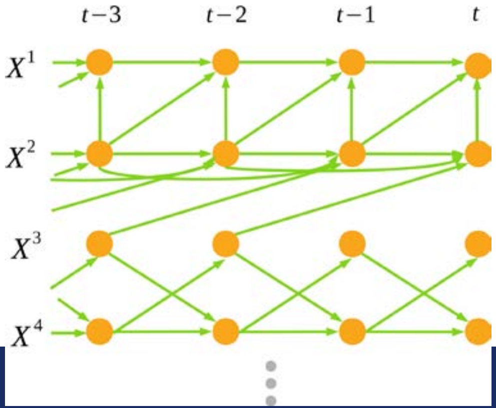
# Causal Discovery in Time Series

**Observations in time make things easier:**

- Additional constraint: Causation cannot go back in time.

**Observations in time make things harder:**

- High dimensionality: Resolving in time increases the number of variables
- Statistical issues: Autocorrelation makes conditional independence tests statistically harder



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**PCMCI-Algorithm:**

Adaption of the PC (Peters/Clark)-algorithm to time series in which these challenges are addressed.

# Alternative Concept: Granger Causality

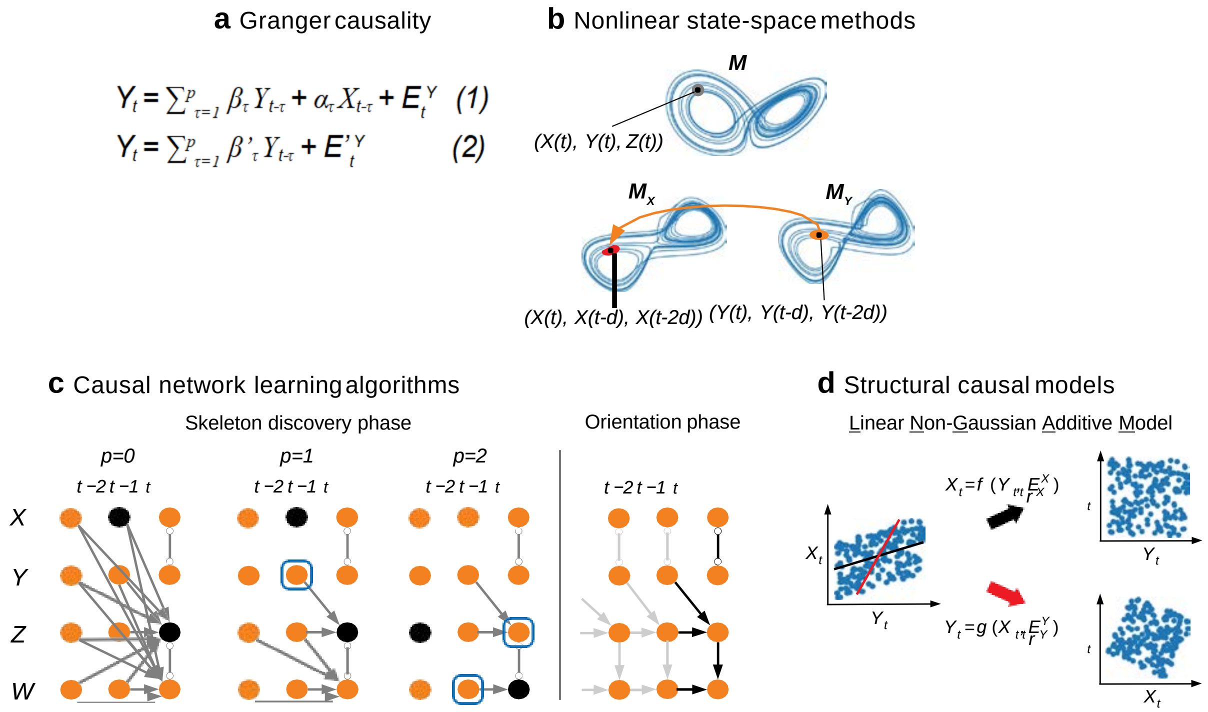
**Idea:**

$X$ causes $Y$ if the past of $X$ helps in predicting the $Y$ from its own past.

**Limitations:**

- Requires causal sufficiency
- Does not allow contemporaneous interactions

# State of the art: see Runge et al. NatComm 2019

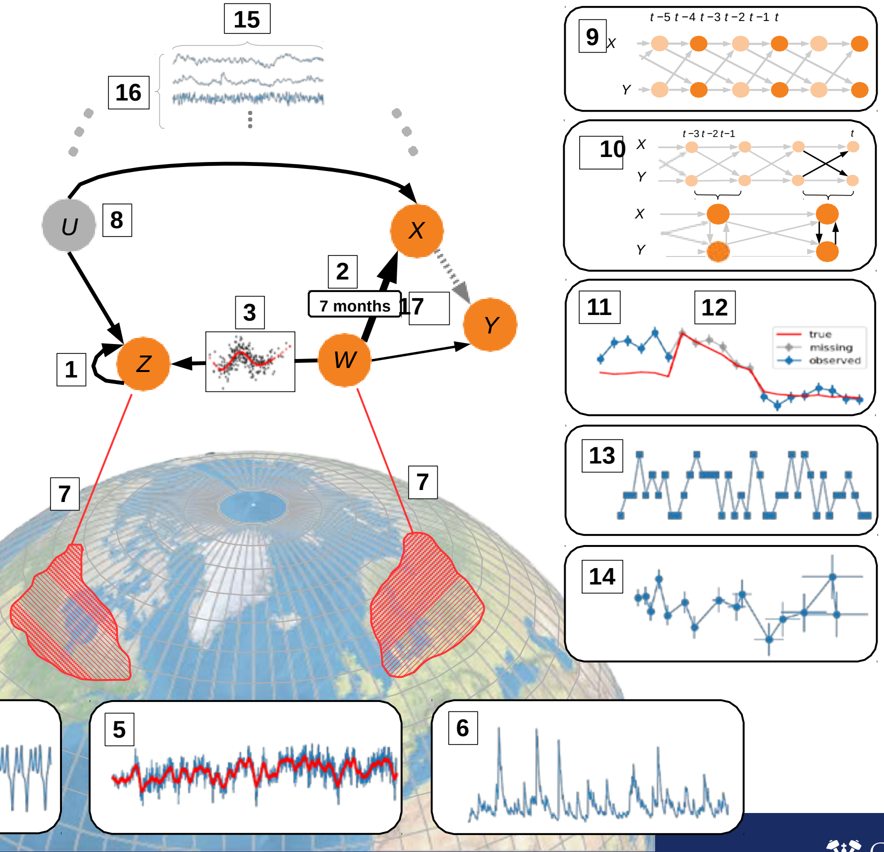


# Real world challenges: see Runge et al. NatComm 2019

**Challeng**
**es**
**Proces**
**s:** 1 Autocorrelation
2 Time delays
3 Nonlinear dependencies
4 Chaotic state-dependence
5 Different time scales
6 Noise distributions

**Data:**
7 Variable extraction
8 Unobserved variables
9 Time subsampling
10 Time aggregation
11 Measurement errors
12 Selection bias
13 Discrete data
14 Dating uncertainties

**Computational / statistical:**
15 Sample size
16 High dimensionality
17 Uncertainty estimation



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