

# Inferring causality from observations

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# Online material

- Peters, Janzing, Schölkopf: *Elements of Causal Inference*, MIT Press 2017, free download as pdf at

<https://mitpress.mit.edu/books/elements-causal-inference>

- 5-day course at a Summer School 2014 in Finland:

<https://ei.is.tuebingen.mpg.de/publications/janzing14>

- 3 hours course (together with Bernhard Schölkopf) at the Machine Learning Summer School 2013

<http://mlss.tuebingen.mpg.de/2013/speakers.html>

- 4 lectures on causality from Jonas Peters

<https://stat.mit.edu/news/four-lectures-causality/>

# Outline

- ➊ **Motivation:** correlation versus causation
  - ➋ **Formalizing causality:** causal DAGs, functional causal models, Markov conditions, do-operator, potential outcomes
  - ➌ **Strong assumptions that enable causal discovery:** faithfulness, independence of mechanisms, additive noise, linear non-Gaussian models
  - ➍ **Macroscopic and microscopic causal models:** consistent coarse-graining of causal models
  - ➎ **Causal inference in time series:** Granger causality and its limitations
  - ➏ **Causal relations among individual objects:** algorithmic Markov conditions, analogy to probabilistic Markov conditions
- (some applications in neuroscience are spread over the sections)

## **1. Motivation:**

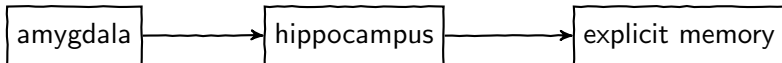
correlation versus causation



# Why Causality?

Check out discussion sections for causal terminology sneaking in ;-)

*Hippocampal activity in this study was correlated with amygdala activity, supporting the view that the amygdala **enhances** explicit memory by **modulating** activity in the hippocampus.*



# Drawing causal conclusions from statistical data

- challenging problem, ongoing research
- don't expect an algorithm to which you feed your data and the output is the causal structure
- applying existing algorithms in a sensible way requires deep understanding of the problems of causal inference
- this course will provide a basis for this

# Can we infer causal relations from passive observations?

## Study report less allergies for children who grew up without dishwasher

Hesselmar et al, Pediatrics March 2015, Vol135 / Issue 3



image source: Wikipedia 'Geschirrspülmaschine', author Christian Giersing

### Possible explanations:

- stronger exposure to microbes helps development of immune system
- families without dishwasher tend to have different life style also in other regards

⇒ Relation between statistical and causal dependences is tricky

# Statistical and causal statements...

...differ by **slight** rewording:

- “children growing up without dishwasher are less likely to have allergies”
- “children growing up without dishwasher are less likely to have allergies because of missing dishwasher”

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- **“children growing up without dishwasher are less likely to have allergies”**

**statistical statement:**

can be tested by standard statistical tools

- **“children growing up without dishwasher are less likely to have allergies because of the missing dishwasher”**

**causal statement:**

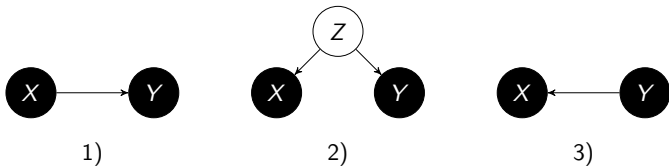
no standard methods available, the tutorial will give partial answers, don't expect simple ones!

...this raises the question...

does statistics tell us something about causality at all?

# Reichenbach's principle of common cause (1956)

If two variables  $X$  and  $Y$  are statistically dependent then either



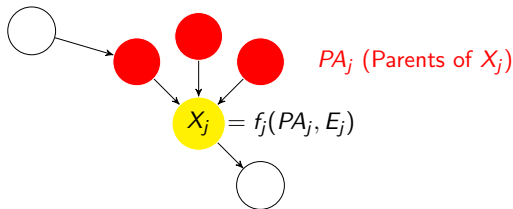
- every statistical dependence is due to a causal relation, we also call 2) “causal”.
- distinction between 3 cases is a key problem in scientific reasoning.
- cases 1-3 can also occur simultaneously

## 2. Formalizing causality:

causal DAGs, functional causal models, Markov conditions, do-operator, potential outcomes

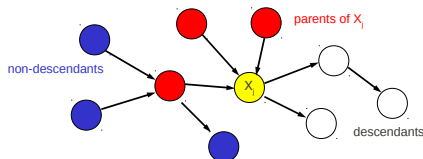


- every node  $X_j$  is a function of its parents  $PA_j$  and an unobserved noise term  $E_j$
- $f_j$  describes how  $X_j$  changes when parents are set to specific values



- all noise terms  $E_j$  are statistically independent (causal sufficiency)
- which properties of  $P(X_1, \dots, X_n)$  follow?

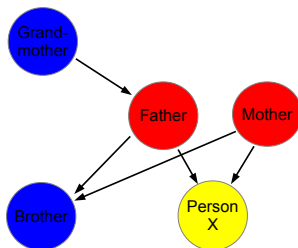
- **existence of a functional model**
- **local Markov condition:** every node is conditionally independent of its non-descendants, given its parents



(information exchange with non-descendants involves parents)

- **global Markov condition:** describes all ind. via d-separation
- **Factorization:**  $P(X_1, \dots, X_n) = \prod_j P(X_j | PA_j)$   
(every  $P(X_j | PA_j)$  describes a causal mechanism)

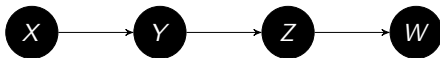
# Metaphor for local Markov condition



If someone knows the genes of  $X$ 's parents, neither the genes of the grandmother nor the genes of the brother contain additional information about  $X$

# Idea of the global Markov condition

conditional independences stated by the local Markov condition implies further conditional independences, e.g.



$$X \perp\!\!\!\perp W \mid Y$$

does not directly follow from the local Markov condition, although it's true

- intuitively reasonable: since the influence of  $X$  on  $W$  is intermediated by  $Y$ , the dependence disappears for fixed values of  $Y$
- there are mathematical rules about which conditional independences imply further independences

# Statistical independence vs. uncorrelatedness

- $X, Y$  **independent**: probabilities factorize, i.e.

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

(difficult to test)

- $X, Y$  **uncorrelated**: expectations factorize, i.e.

$$\mathbb{E}[X \cdot Y] = \mathbb{E}[X] \cdot \mathbb{E}[Y].$$

(easy to test: just compute empirical means)

independent implies uncorrelated but not vice versa

(note: physics literature is sometime sloppy about the difference)

# Reformulation of statistical independence

- **factorizing probabilities:**  $p(x, y) = p(x)p(y)$
- **knowing  $X$  does not change the distribution of  $Y$ :**

$$p(y|x) = p(y)$$

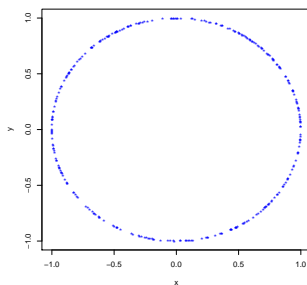
( $X$  contains no information about  $Y$  and vice versa)

- **functions of  $X$  and  $Y$  are uncorrelated:**

$$\mathbb{E}[f(X) \cdot g(Y)] = \mathbb{E}[f(X)] \cdot \mathbb{E}[g(Y)] \quad \forall f, g$$

# Dependence without correlation

Let  $P_{X,Y}$  be uniform distribution on a circle:



- uncorrelated because  $\mathbb{E}[XY] = 0$  and  $\mathbb{E}[X] = 0, \mathbb{E}[Y] = 0$  for symmetry reasons
- $X$  and  $Y$  are statistically dependent: knowing  $X$  reduces the possible values  $Y$  from  $[-1, 1]$  to just two options

*Path* = sequence of pairwise distinct nodes where consecutive ones are adjacent

A path  $q$  is said to be **blocked** by the set  $Z$  if

- $q$  contains a *chain*  $i \rightarrow m \rightarrow j$  or a *fork*  $i \leftarrow m \rightarrow j$  such that the middle node is in  $Z$ , or
- $q$  contains a *collider*  $i \rightarrow m \leftarrow j$  such that the middle node is not in  $Z$  and such that no descendant of  $m$  is in  $Z$ .

$Z$  is said to **d-separate**  $X$  and  $Y$  in the DAG  $G$ , formally

$$(X \perp\!\!\!\perp Y | Z)_G$$

if  $Z$  blocks every path from a node in  $X$  to a node in  $Y$ .

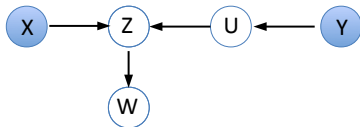


## Example (blocking of paths)



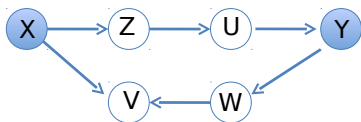
path from  $X$  to  $Y$  is blocked by conditioning on  $U$  or  $Z$  or both

## Example (unblocking of paths)



- path from  $X$  to  $Y$  is blocked by  $\emptyset$
- unblocked by conditioning on  $Z$  or  $W$  or both

## Example (blocking and unblocking of paths)



several options for blocking all paths between  $X$  and  $Y$ :

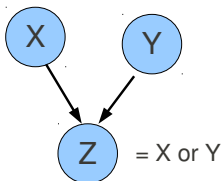
$$(X \perp\!\!\!\perp Y \mid ZW)_G$$

$$(X \perp\!\!\!\perp Y \mid ZUW)_G$$

$$(X \perp\!\!\!\perp Y \mid VZUW)_G$$

# Unblocking by conditioning on common effects

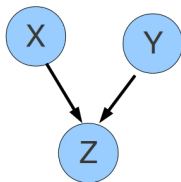
Berkson's paradox (1946), selection bias. Example:  $X, Y, Z$  binary



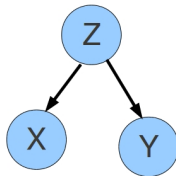
- assume language skills and science skills are independent a priori
- assume pupils go to high school if they have good skills in science or languages
- then there is a negative correlation between science skills and language skills in high school

# Asymmetry with respect to inverting arrows

Reichenbach: The direction of time (1956)



$$\begin{aligned} X &\perp\!\!\!\perp Y \\ X &\not\perp\!\!\!\perp Y|Z \end{aligned}$$



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## Formalizing the difference between seeing and doing

- **observational probabilities:**  $p(y|x)$  probability for  $Y = y$ , given that we observed  $X = x$
- **interventional probabilities:**  $p(y|do(x))$  probability for  $Y = y$ , given that we have set  $X$  to  $x$ .

confusing  $p(y|x)$  with  $p(y|do(x))$  is the reason for most of the common misconceptions about causality!

# Pearl's do operator

how to compute  $p(x_1, \dots, x_n | do(x'_i))$ :

- write  $p(x_1, \dots, x_n)$  as

$$\prod_{k=1}^n p(x_k | parents(x_k))$$

- replace  $p(x_i | parents(x_i))$  with  $\delta_{x_i, x'_i}$

$$p(x_1, \dots, x_n | do(x'_i)) = \prod_{k \neq i} p(x_k | parents(x_k)) \delta_{x_i, x'_i}$$

## How to compute $p(x_j|do(x_i))$

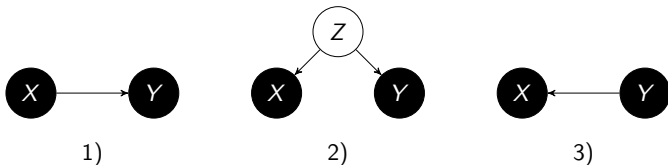
marginalize over all  $k \neq j$ :

$$\begin{aligned} p(x_j|do(x'_i)) &= \sum p(x_1, \dots, x_n|do(x'_i)) \\ &= \sum \prod_{k \neq i} p(x_k|parents(x_k)) \delta_{x_i, x'_i} \end{aligned}$$

(sum runs over all  $(x_1, \dots, x_{j-1}, x_{j+1}, \dots, x_n)$ )



## Simple examples



- ① interventional and observational probabilities coincide (seeing is the same as doing)

$$p(y|\text{do}(x)) = p(y|x)$$

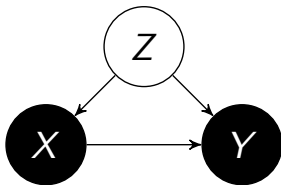
- ② intervening on  $x$  does not change  $y$

$$p(y|\text{do}(x)) = p(y) \neq p(y|x)$$

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## Most important case: confounder correction



$$p(y|do(x)) = \sum_z p(y|x, z)p(z) \neq \sum_z p(y|x, z)p(z|x) = p(y|x)$$

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(PW Holland, Statistics and Causal Inference. *Journal of the American Statistical Association*, 1986)

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e. g. survival times  $Y_t(u)$  and  $Y_c(u)$  of patient  $u$

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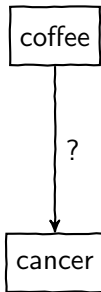
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- is equal to  $\mathbb{E}[Y_t]$  and  $\mathbb{E}[Y_c]$ .

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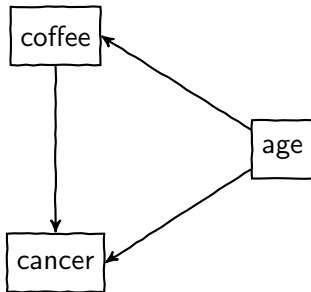
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$\Rightarrow \mathbb{E}[Y_{\blacksquare} | S = \blacksquare] - \mathbb{E}[Y_{\square} | S = \square]$  systematically overestimates the effect of cumulative coffee consumption on cancer

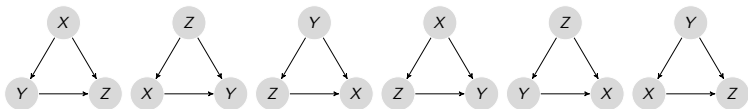
### **3. Strong assumptions that enable causal discovery:**

faithfulness, independence of mechanisms, additive noise, linear non-Gaussian models

# Causal discovery from observational data

Can we infer  $G$  from  $P(X_1, \dots, X_n)$ ?

- MC only describes which sets of DAGs are consistent with  $P$
- $n!$  many DAGs are consistent with any distribution



- reasonable rules for preferring **simple** DAGs required



# Independence of mechanisms (ICM)

The conditionals  $P(X_j|PA_j)$  in the causal factorization  $P(X_1, \dots, X_n) = \prod_{j=1}^n P(X_j|PA_j)$  represent independent mechanisms in nature

- **independent change:** they change independently across data sets
- **no information:** they contain no information about each other, formalization by algorithmic information theory: shortest description of  $P(X_1, \dots, X_n)$  is given by separate descriptions of  $P(X_j|PA_j)$

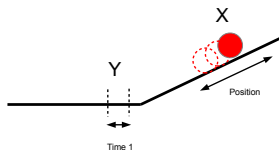
(see Peters, Janzing, Schölkopf: *Elements of Causal Inference* for historical overview)

## ICM for the bivariate case

- both  $P(\text{cause})$  and  $P(\text{effect}|\text{cause})$  may change across environments
- but they change independently
- knowing how  $P(\text{cause})$  has changed does not provide information about if and how  $P(\text{effect}|\text{cause})$  has changed
- knowing how  $P(\text{effect}|\text{cause})$  has changed does not provide information about if and how  $P(\text{cause})$  has changed

# Independent changes in the real world: ball track

relation between initial position (cause) and speed (effect)  
measured between two light barriers

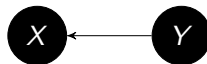


- $P(\text{cause})$  changes if another child plays
- $P(\text{effect}|\text{cause})$  changes if the light barriers are mounted at a different position
- hard to think of operations that change  $P(\text{effect})$  without affecting  $P(\text{cause}|\text{effect})$  or vice versa

# Implications of ICM for causal and anti-causal learning



**causal learning:**  
predict effect from cause



**anticausal learning:**  
predict cause from effect

- **Causal learning:**  
predict properties of a molecule from its structure
- **Anticausal learning:** tumor classification, image segmentation

Hypothesis: SSL only works for anticausal learning. Confirmed by screening performance studies in the literature.

# Anti-causal prediction: why unlabelled points may help

- let  $Y$  be some class label e.g.  $y \in \{male, female\}$
- Let  $X$  be a feature influenced by  $Y$ , e.g. height
- observe that  $P_X$  is bimodal



- probably the two modes correspond to the two classes (idea of cluster algorithms)



(can easily be confirmed by observing a *small* number of labeled points)

# Causal prediction: why unlabelled points don't help

- let  $Y$  be some class label of an effect  $y \in \{sick, healthy\}$
- Let  $X$  be a feature influencing  $Y$ , e.g. a risk factor like blood pressure
- observe that  $P_X$  is bimodal

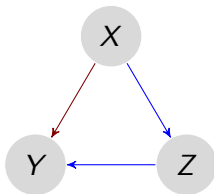


- no reasons to believe that the modes correspond to the two classes



Prefer those DAGs for which all observed conditional independences are implied by the Markov condition

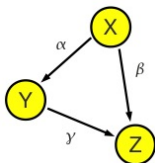
- **Idea:** generic choices of parameters yield faithful distributions
- **Example:** let  $X \perp\!\!\!\perp Y$  for the DAG



- not faithful, **direct** and **indirect** influence compensate

# Examples of unfaithful distributions

cancellation of direct and indirect influence in linear models



$$Y = \alpha X + N_Y$$

$$Z = \beta X + \gamma Y + N_Z$$

with independent  $X, N_Y, N_Z$

$X$  and  $Z$  are independent if  $\beta + \alpha\gamma = 0$



# Conditional-independence based causal inference

Spirtes, Glymour, Scheines and Pearl:

## **Causal Markov condition + Causal faithfulness:**

accept only those DAGs as causal hypotheses for which:

- **all** independences are true that are required by the Markov condition
- **only** those independences are true

identifies causal DAG up to Markov equivalence class  
(DAGs that imply the same conditional independences)

# Hidden Confounding and CI-based CI in Neuroimaging

(S Weichwald et al., *NeuroImage*, 2015; M Grosse-Wentrup et al., *NeuroImage*, 2016; S Weichwald et al., *IEEE ST SigProc*, 2016)

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$\implies$  Robust against hidden confounding

# Application: Neural Dynamics of Reward Prediction

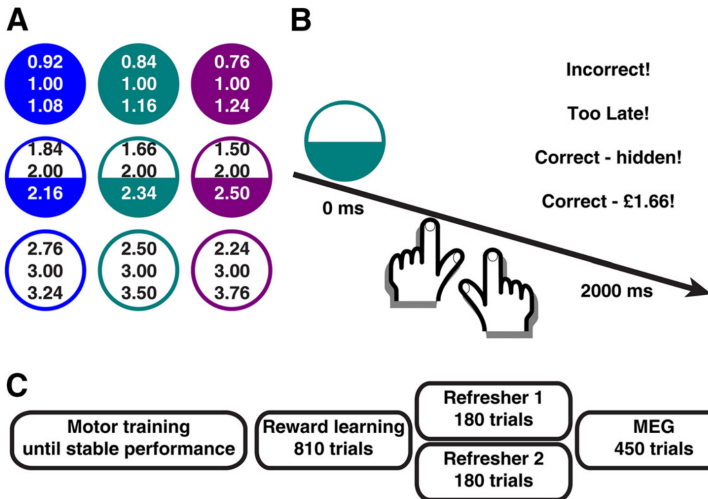
(Bach, Symmonds, Barnes, and Dolan. *Journal of Neuroscience*, 2017)



# Application: Neural Dynamics of Reward Prediction

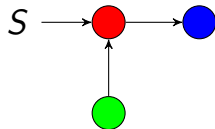
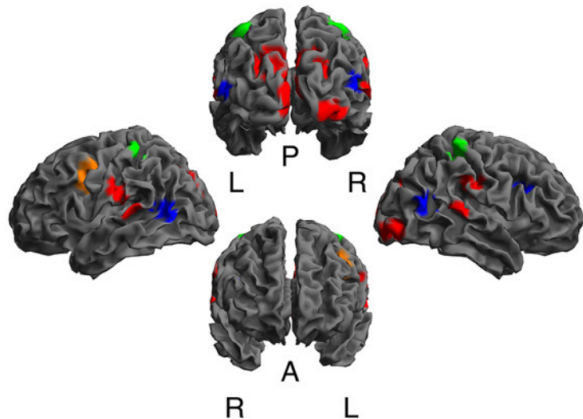
(Bach, Symmonds, Barnes, and Dolan. *Journal of Neuroscience*, 2017)

Bach et al. • Probabilistic Reward Prediction



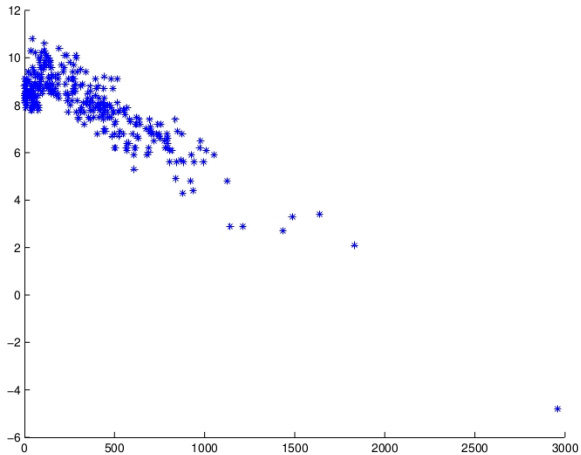
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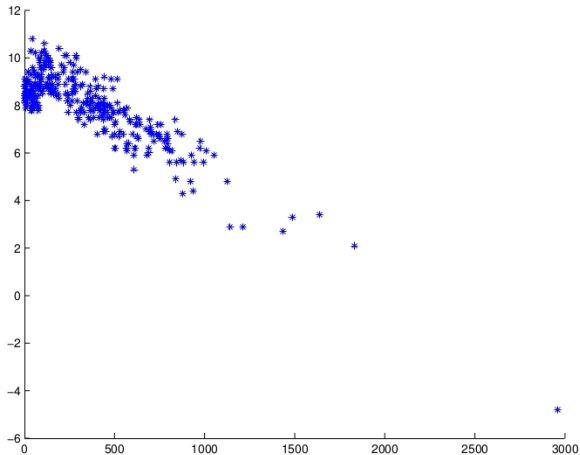


What can be said beyond Markov condition and faithfulness?

# What's the cause and what's the effect?

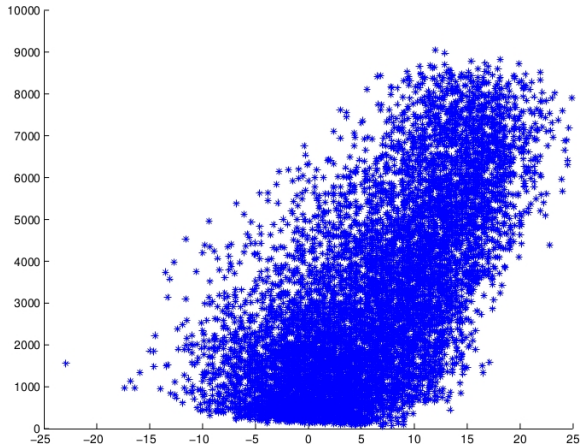


# What's the cause and what's the effect?

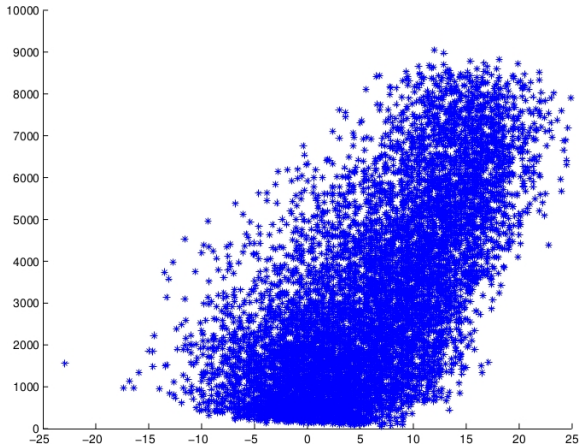


$X$  (Altitude)  $\rightarrow$   $Y$  (Temperature)

# What's the cause and what's the effect?

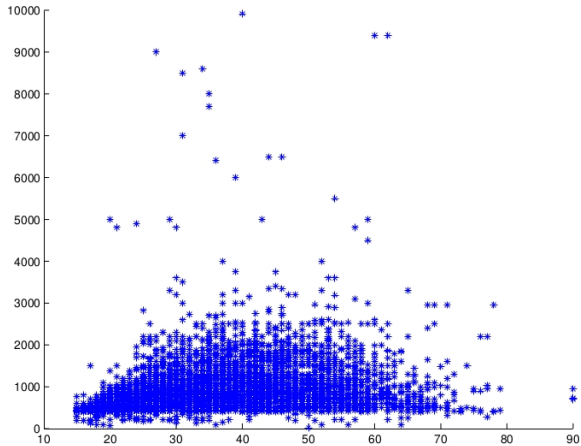


# What's the cause and what's the effect?



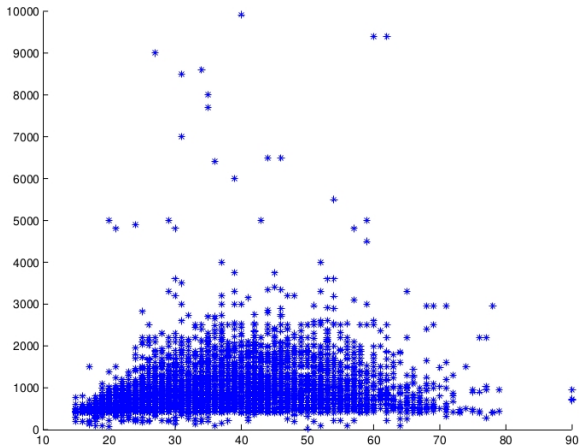
Y (Solar Radiation)  $\rightarrow$  X (Temperature)

# What's the cause and what's the effect?





# What's the cause and what's the effect?



$X$  (Age)  $\rightarrow$   $Y$  (Income)

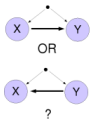
## Hence...

- there are asymmetries between cause and effect apart from those formalized by the causal Markov condition
- new methods that employ these asymmetries need to be developed

# Database with cause effect pairs



## Database with cause-effect pairs



This is a growing database with different data for testing causal detection algorithms. The goal here is to distinguish between cause and effect. We searched for data sets with known ground truth and how the data was derived.

Note that not always the first column is the cause and the second the effect. This is indicated in a [meta-data file](#). Please look at [README](#) for further explanations. We also suggest a way that is very similar if you want to calculate the overall performance.

To get all data files at once download [all data](#) as a zip file.

Note: pair0001 - pair0041 were taken from the UCI Machine Learning Repository, so if you use these data sets please refer to their [webpage](#). Here you will find their [citation policy](#).

If you have any comments, questions or suggestions for additional data sets, please contact [Dominik Janzing](#).

Data	Description	Scatter plot (PDF)
<a href="#">pair0001.txt</a>	<a href="#">pair0001_des.txt</a>	<a href="#">pair0001.pdf</a>
<a href="#">pair0002.txt</a>	<a href="#">pair0002_des.txt</a>	<a href="#">pair0002.pdf</a>
<a href="#">pair0003.txt</a>	<a href="#">pair0003_des.txt</a>	<a href="#">pair0003.pdf</a>
<a href="#">pair0004.txt</a>	<a href="#">pair0004_des.txt</a>	<a href="#">pair0004.pdf</a>
<a href="#">pair0005.txt</a>	<a href="#">pair0005_des.txt</a>	<a href="#">pair0005.pdf</a>
<a href="#">pair0006.txt</a>	<a href="#">pair0006_des.txt</a>	<a href="#">pair0006.pdf</a>
<a href="#">pair0007.txt</a>	<a href="#">pair0007_des.txt</a>	<a href="#">pair0007.pdf</a>

# Idea of the website

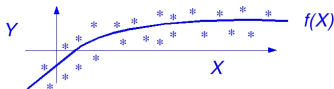
- to evaluate novel causal inference methods
- inspire development of novel methods
- provide data where ground truth is obvious to non-experts (as opposed to many data sets on economy, biology)
- should grow further (contains 105 pairs currently )
- ground truth discussed in: J. Mooij, J. Peters, D. Janzing, J. Zscheischler, B. Schölkopf: *Distinguishing cause from effect using observational data: methods and benchmarks*, Journal of Machine Learning Research, 2016.

# Non-linear additive noise based inference

Hoyer, Janzing, Mooij, Peters, Schölkopf, 2008

- Assume that the effect is a function of the cause up to an additive noise term that is statistically independent of the cause:

$$Y = f(X) + N_Y \quad \text{with} \quad N_Y \perp\!\!\!\perp X$$



- there will, in the generic case, be no model

$$X = g(Y) + N_X \quad \text{with} \quad N_X \perp\!\!\!\perp Y,$$

even if  $f$  is invertible! (proof is non-trivial)

## Note...

$$Y = f(X, N_Y) \quad \text{with} \quad N_Y \perp\!\!\!\perp X$$

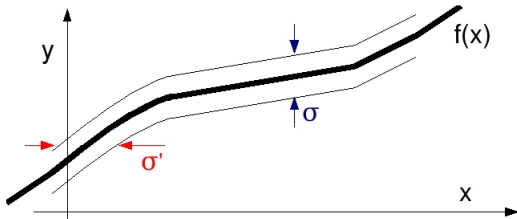
can model **any** conditional  $P_{Y|X}$

$$Y = f(X) + N_Y \quad \text{with} \quad N_Y \perp\!\!\!\perp X$$

restricts the class of  $P_{Y|X}$

# Intuition

- additive noise model from  $X$  to  $Y$  imposes that the width of noise is constant in  $x$ .
- for non-linear  $f$ , the width of noise won't be constant in  $y$  at the same time.



## Causal inference method:

**Prefer the causal direction that can better be fit with an additive noise model.**

Implementation:

- Compute a function  $f$  as non-linear regression of  $Y$  on  $X$ , i.e.,  $f(x) := \mathbb{E}[Y|x]$ .
- Compute the noise

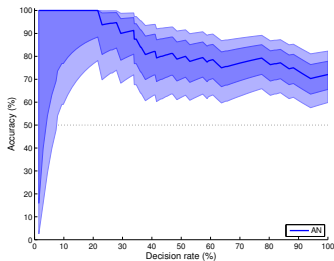
$$N_Y := Y - f(X)$$

- check whether  $N_Y$  and  $X$  are statistically independent (uncorrelated is not sufficient, method requires tests that are able to detect higher order dependences)
- performed better than chance on real data with known ground truth



# Extensive evaluation

Peters, Mooij, Janzing, Schölkopf: Causal Discovery with Continuous Additive Noise Models, JMLR 2014



- if the algorithm decides in all cases, about 75% decisions are right
- if it only decides in 'the most obvious' 20% of the cases, the fraction gets close to 100%

## Justification of the method

- we don't claim that every causal influence can be described by an additive noise model
- we only claim 'if there is an additive noise model from one direction but not the other the former is likely to be the causal direction'
- if nature chooses  $P_{\text{cause}}$  and  $P_{\text{effect}|\text{cause}}$  independently it is unlikely that the result is a joint distribution  $P_{\text{effect,cause}}$  that admits an additive noise model from effect to cause

## Some theoretical support

Assume  $Y = f(X) + N_Y$  with  $N_Y \perp\!\!\!\perp X$

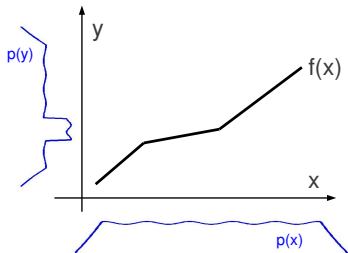
- Then  $P_Y$  and  $P_{X|Y}$  are related:

$$\frac{\partial^2}{\partial y^2} \log p(y) = -\frac{\partial^2}{\partial y^2} \log p(x|y) - \frac{1}{f'(x)} \frac{\partial^2}{\partial x \partial y} \log p(x|y).$$

$\Rightarrow \frac{\partial^2}{\partial y^2} \log p(y)$  can be computed from  $p(x|y)$  knowing  $f'(x_0)$  for one specific  $x_0$

- $P_{X|Y}$  almost determines  $P_Y$
- We reject  $Y \rightarrow X$  (provided that  $P_Y$  is complex) because we assume that nature chooses  $P_{\text{cause}}$  and  $P_{\text{effect}|\text{cause}}$  independently

- Problem: infer whether  $Y = f(X)$  or  $X = f^{-1}(Y)$  is the right causal model
- Idea: if  $X \rightarrow Y$  then  $f$  and the density  $p_X$  are chosen independently “by nature”
- Hence, peaks of  $p_X$  do not correlate with the slope of  $f$
- Then, **peaks of  $p_Y$**  correlate with the **slope of  $f^{-1}$**



# Inferring causal structure via ICA

(Shimizu et al. (2006))

A linear acyclic SCM

$$\begin{pmatrix} X_1 \\ X_2 \\ \vdots \\ X_d \end{pmatrix} = \begin{pmatrix} 0 & b_{12} & \dots & b_{1d} \\ 0 & 0 & \dots & b_{2d} \\ \vdots & \vdots & \ddots & \vdots \\ 0 & 0 & \dots & 0 \end{pmatrix} \begin{pmatrix} X_1 \\ X_2 \\ \vdots \\ X_d \end{pmatrix} + \begin{pmatrix} S_1 \\ S_2 \\ \vdots \\ S_d \end{pmatrix}$$

with mutually independent components  $S_1, \dots, S_d$

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$\iff$

$$(\text{Id} - \mathbf{B}) \cdot X = S$$



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with mutually independent components  $S_1, \dots, S_d$

is closely linked to ICA (Independent Component Analysis) as per

$$X = \mathbf{B} \cdot X + S$$

$$\iff (\text{Id} - \mathbf{B}) \cdot X = S$$

$$\iff X = (\text{Id} - \mathbf{B})^{-1} \cdot S$$

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(Shimizu et al. (2006))

LiNGAM: Linear Non-Gaussian Acyclic Model  $X = \mathbf{B}X + S$

Identify  $\mathbf{B}$  via two steps:

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*Non-Gaussianity!*

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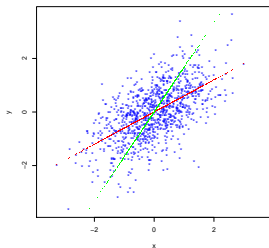
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*Non-Gaussianity!*

- 2 resolve scaling and permutation to obtain  $\mathbf{B}$

*Acyclicity!*

# Bivariate Gaussian and Indeterminacies of ICA



The same distribution can be described as

$$X = N_X$$

or

$$X = \beta \cdot Y + N_X$$

$$Y = \alpha \cdot X + N_Y$$

$$Y = N_Y$$

where  $N_X$  and  $N_Y$  are suitable independent Gaussian distributions

# Linear non-Gaussian models

Kano & Shimizu 2003

## Theorem

*Let  $X \not\perp\!\!\!\perp Y$ . Then  $P_{X,Y}$  admits linear models in both directions, i.e.,*

$$\begin{aligned} Y &= \alpha X + N_Y && \text{with } N_Y \perp\!\!\!\perp X \\ X &= \beta Y + N_X && \text{with } N_X \perp\!\!\!\perp Y, \end{aligned}$$

*if and only if  $P_{X,Y}$  is bivariate Gaussian*

- if  $P_{X,Y}$  is non-Gaussian, there can be a linear model in at most one direction.
- LINGAM: causal direction is the one that admits a linear model



# LiNGAM and confounding-robust ICA

(Pfister\*, Weichwald\*, et al. (2018) arXiv:1806.01094)

LiNGAM

$$X = \mathbf{B}X + S$$

where  $S$  has mutually independent components

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Confounded LiNGAM

$$X = \mathbf{B}X + S + H$$

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(Pfister\*, Weichwald\*, et al. (2018) arXiv:1806.01094)

$$\begin{aligned}\text{LiNGAM} \quad X &= \mathbf{B}X + S \\ \iff X &= (\text{Id} - \mathbf{B})^{-1}S\end{aligned}$$

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where  $S$  has mutually independent components  
and  $H$  is *group-wise stationary* confounding

$\rightsquigarrow$  corolCA allows to identify the confounded LiNGAM model  
and accounts for dependencies due to  $H$   
if  $H$  is group-wise stationary

## **4. Macroscopic and microscopic causal models:**

consistent coarse-graining of causal models

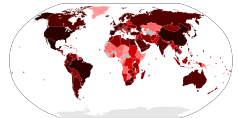
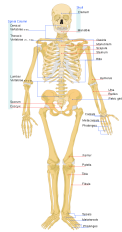
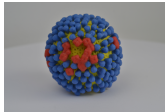
# Models at different levels

Fine-grained

Coarse-grained



Disease

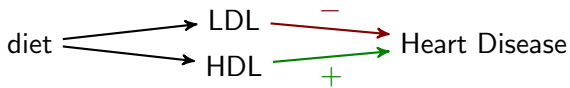


Traffic

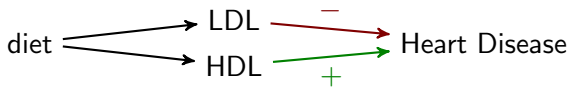
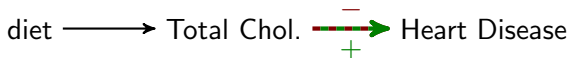




# What can go wrong? Cholesterol and Heart Disease



# What can go wrong? Cholesterol and Heart Disease



# What can go wrong? Cholesterol and Heart Disease

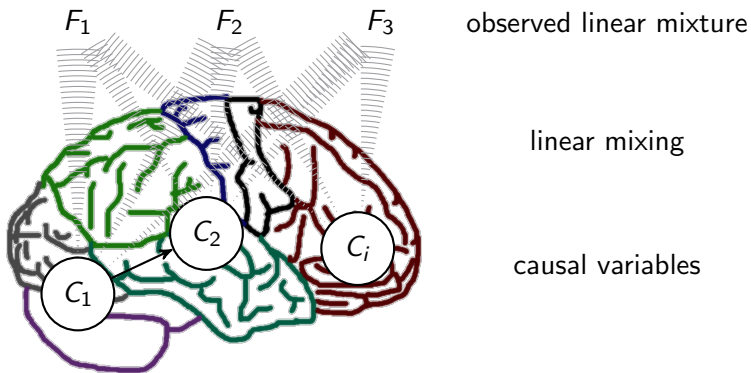
diet  $\longrightarrow$  Total Chol.  $\xrightarrow[-]{+}$  Heart Disease



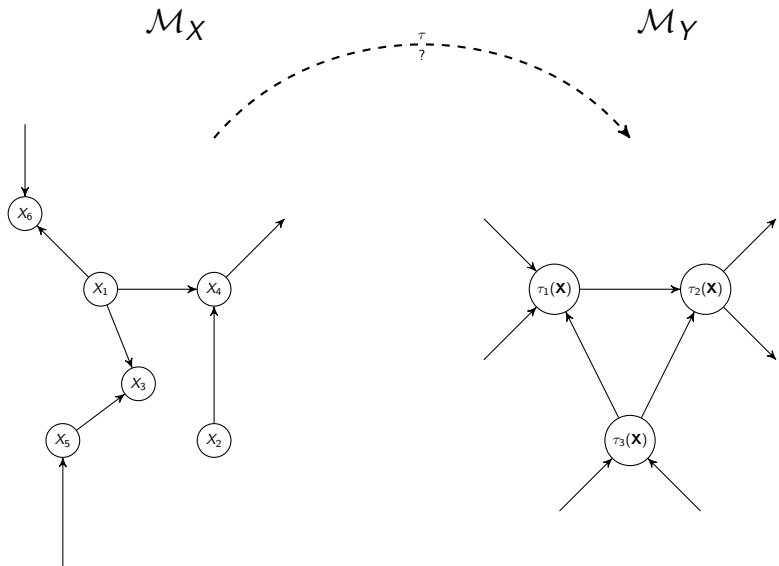
diet  $\begin{cases} \longrightarrow \text{LDL} \\ \longrightarrow \text{HDL} \end{cases}$   $\begin{cases} \xrightarrow{-} \\ \xrightarrow{+} \end{cases}$  Heart Disease

Incorrectly 'transforming' the model can lead to problems.

# Limited ability to observe breaks causal reasoning



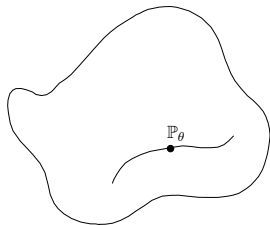
# Transformations of causal models



# Causal Models as Posets of Distributions

“Normal” Probabilistic Model:

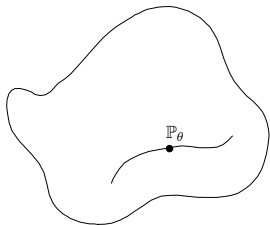
$$\mathcal{M}_X : \theta \mapsto \mathbb{P}_\theta$$



# Causal Models as Posets of Distributions

“Normal” Probabilistic Model:

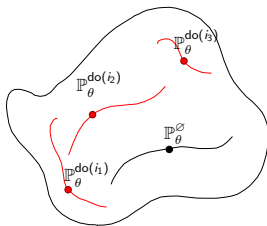
$$\mathcal{M}_X : \theta \mapsto \mathbb{P}_\theta$$



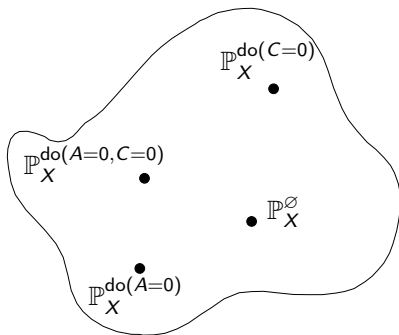
Causal Model:

$$\mathcal{M}_X : \theta \mapsto \{\mathbb{P}_\theta^{\text{do}(i)} : i \in \mathcal{I}_X\}$$

$\mathcal{I}_X$  is set of **interventions**.

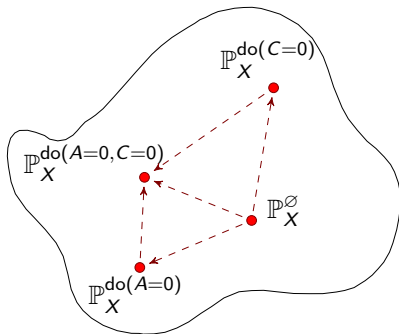


# Causal Models as Posets of Distributions



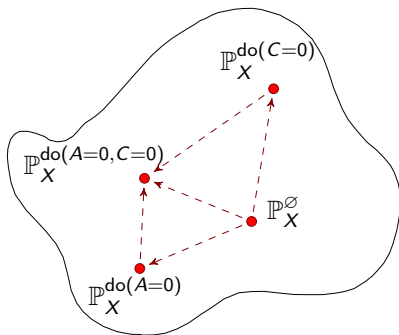


# Causal Models as Posets of Distributions



$\mathcal{I}_X$  has **partial ordering** structure

# Causal Models as Posets of Distributions



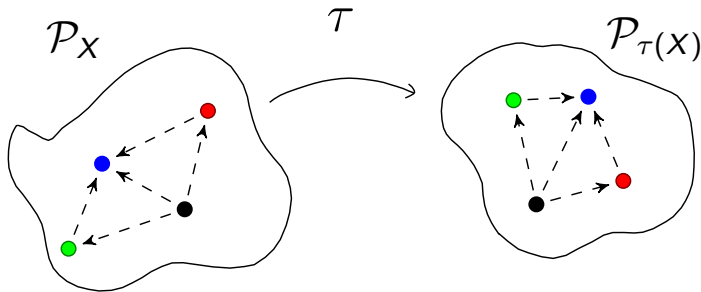
$\mathcal{I}_X$  has **partial ordering** structure

$\mathcal{M}_X$  implies the **poset of distributions**  $\mathcal{P}_X := \left( \left\{ \mathbb{P}_X^{\text{do}(i)} : i \in \mathcal{I}_X \right\}, \leq_X \right)$

# Transformations of Structural Equation Models

Suppose we are given  $\mathcal{M}_X$  and a 'measuring device'  $\tau : \mathcal{X} \rightarrow \mathcal{Y}$   
 $X \sim \mathbb{P}_X$  an r.v. in  $\mathcal{X} \implies \tau(X) \sim \mathbb{P}_{\tau(X)}$  is an r.v. in  $\mathcal{Y}$

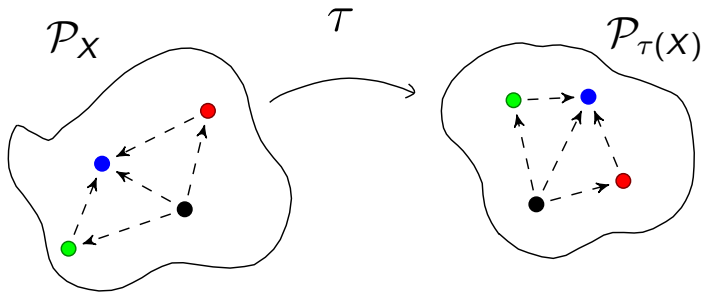
$$\tau : \mathcal{P}_X \rightarrow \mathcal{P}_{\tau(X)} = \left( \left\{ \mathbb{P}_{\tau(X)}^i : i \in \mathcal{I}_X \right\}, \leq_X \right)$$



# Transformations of Structural Equation Models

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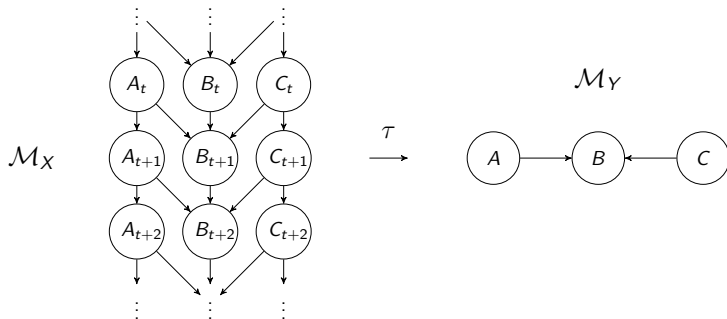
Does there exist an SEM  $\mathcal{M}_Y$  with  $\mathcal{P}_Y = \mathcal{P}_{\tau(X)}$ ?

If so, then  $\mathcal{M}_Y$  will agree with our observations of  $\mathcal{M}_X$  via  $\tau$ .

# In What Sense is Causal Reasoning Preserved?

Does there exist an SEM  $\mathcal{M}_Y$  with  $\mathcal{P}_Y = \mathcal{P}_{\tau(X)}$ ?

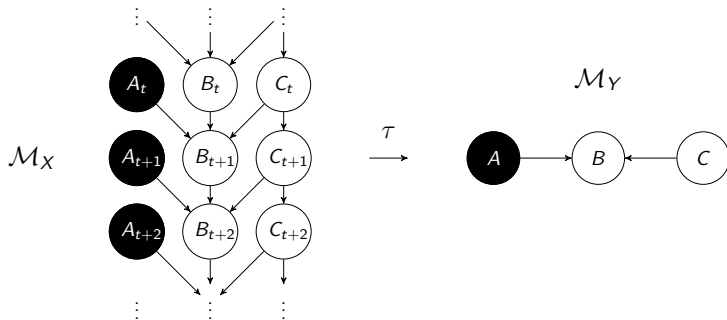
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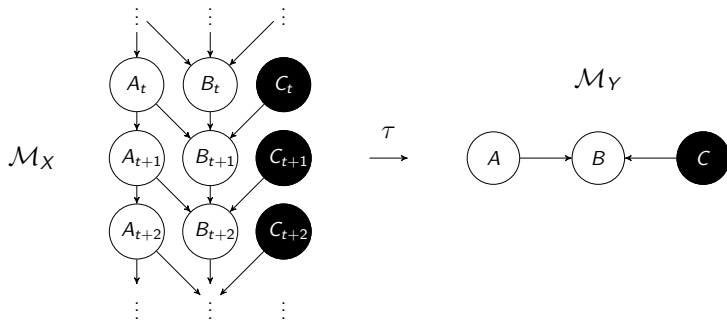
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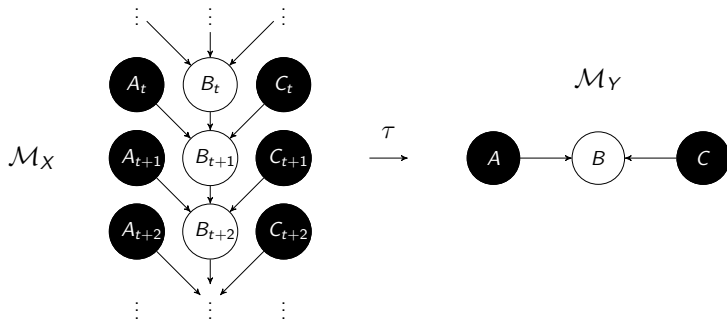
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If so, then  $\mathcal{M}_Y$  will agree with our observations of  $\mathcal{M}_X$  via  $\tau$



Compositions of interventions are preserved!



## Definition (Exact Transformations between SEMs)

Let  $\mathcal{M}_X$  and  $\mathcal{M}_Y$  be SEMs and  $\tau : \mathcal{X} \rightarrow \mathcal{Y}$  be a function. We say  $\mathcal{M}_Y$  is an *exact*  $\tau$ -transformation of  $\mathcal{M}_X$  if there exists a *surjective order-preserving* map  $\omega : \mathcal{I}_X \rightarrow \mathcal{I}_Y$  such that

$$\mathbb{P}_{\tau(X)}^i = \mathbb{P}_Y^{\text{do}(\omega(i))} \quad \forall i \in \mathcal{I}_X$$

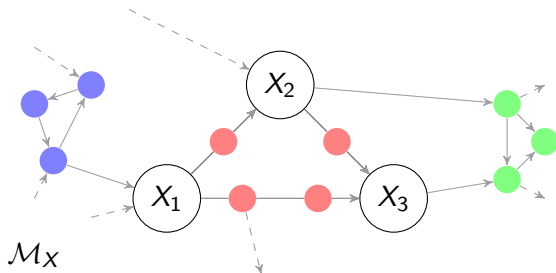
## Theorem

*The following diagram commutes:*

$$\begin{array}{ccccc} \mathbb{P}_X & \xrightarrow{\text{do}(i)} & \mathbb{P}_X^{\text{do}(i)} & \xrightarrow{\text{do}(j)} & \mathbb{P}_X^{\text{do}(j)} \\ \tau \downarrow & & \downarrow \tau & & \downarrow \tau \\ \mathbb{P}_Y & \xrightarrow{\text{do}(\omega(i))} & \mathbb{P}_Y^{\text{do}(\omega(i))} & \xrightarrow{\text{do}(\omega(j))} & \mathbb{P}_Y^{\text{do}(\omega(j))} \end{array}$$

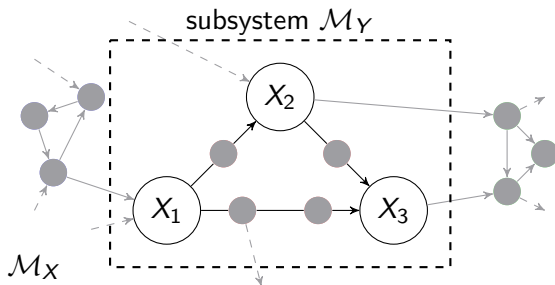
# Transformations for Pragmatic Causal Models

- Marginalisation of variables



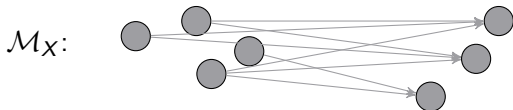
# Transformations for Pragmatic Causal Models

- Marginalisation of variables



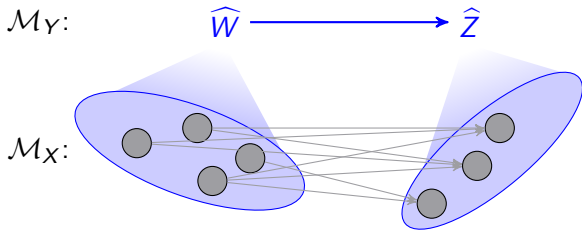
# Transformations for Pragmatic Causal Models

- Marginalisation of variables
- Micro- to macro-level and aggregate features



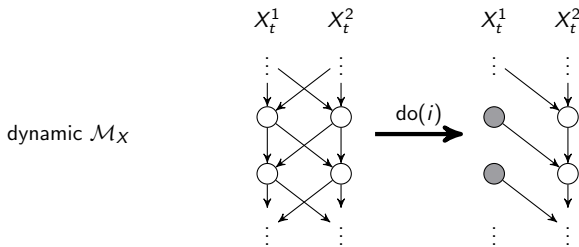
# Transformations for Pragmatic Causal Models

- Marginalisation of variables
- Micro- to macro-level and aggregate features



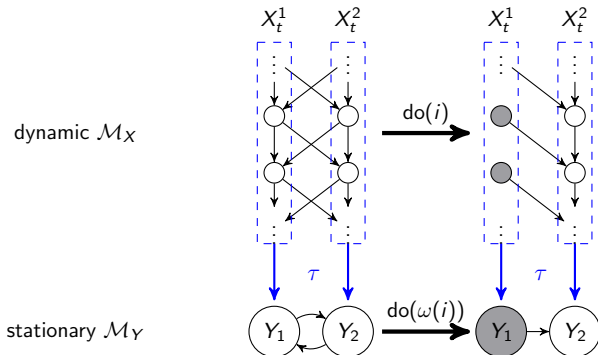
# Transformations for Pragmatic Causal Models

- Marginalisation of variables
- Micro- to macro-level and aggregate features
- Stationary behaviour of dynamical processes



# Transformations for Pragmatic Causal Models

- Marginalisation of variables
- Micro- to macro-level and aggregate features
- Stationary behaviour of dynamical processes



## **5. Causal inference in time series:**

Granger causality and its limitations



# Granger Causality

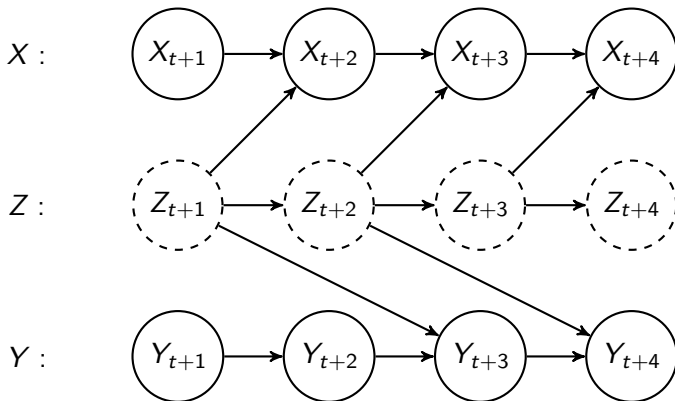
Simplified Definition: One stochastic process  $X$  is causal to a second  $Y$  if the autoregressive predictability of the second process at a given time point is improved by *including* measurements from the past of the first, i. e. if

$$\text{PredAcc}[Y_t | Y_{<t}] < \text{PredAcc}[Y_t | Y_{<t}, X_{<t}]$$

(not by C Granger)

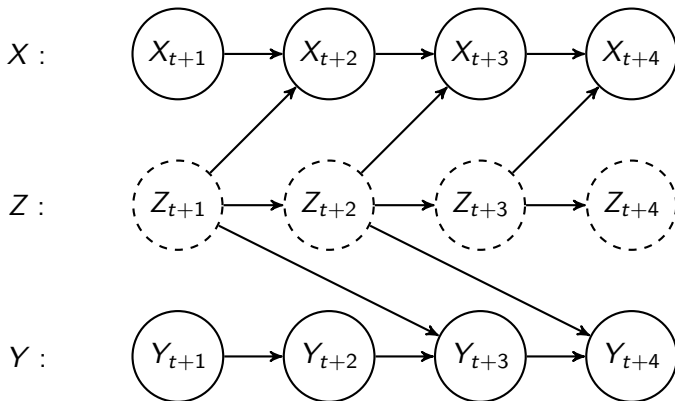
# Granger Causality

(J Peters et al. Causal discovery on time series using restricted structural equation models. *NIPS*, 2013)



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$$\text{PredAcc}[Y_t | Y_{<t}] < \text{PredAcc}[Y_t | Y_{<t}, X_{<t}]$$

Granger causality erroneously infers causal influence from  $X$  to  $Y$ !

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# Granger Causality

(CWJ Granger, Investigating Causal Relations by Econometric Models and Cross-spectral Methods. *Econometrica*, 1969)

Simplified Definition: One stochastic process  $X$  is causal to a second  $Y$  if the autoregressive predictability of the second process at a given time point is improved by *including* measurements from the past of the first, i. e. if

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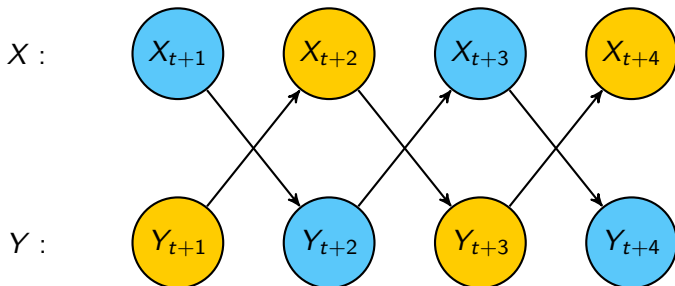
Granger's Definition: One stochastic process  $X$  is causal to a second  $Y$  if the predictability of the second process at a given time point is worsened by *removing* past measurements of the first from the universe's past, i. e. if

$$\text{PredAcc}[Y_t | \text{globe}_{<t}] > \text{PredAcc}[Y_t | \text{globe}_{<t} \setminus X_{<t}]$$

(by C Granger)

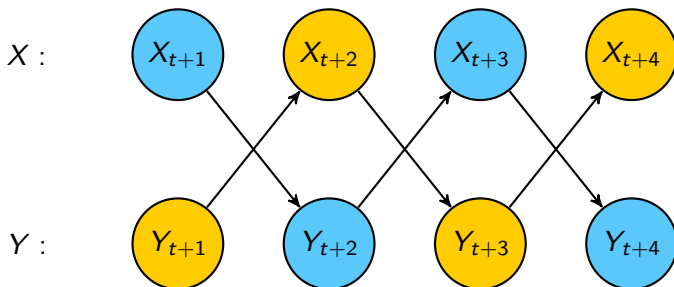
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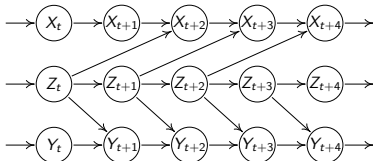
$$\text{PredAcc}[Y_t | \text{Globe}_{<t}] \not\approx \text{PredAcc}[Y_t | \text{Globe}_{<t} \setminus X_{<t}]$$

Granger causality fails to predict the effects of interventions!

# Granger works under Markov and faithfulness

Assumptions:

- no hidden common causes
- no instantaneous effects





## 6. Causal relations among individual objects

algorithmic Markov conditions, analogy to probabilistic Markov conditions

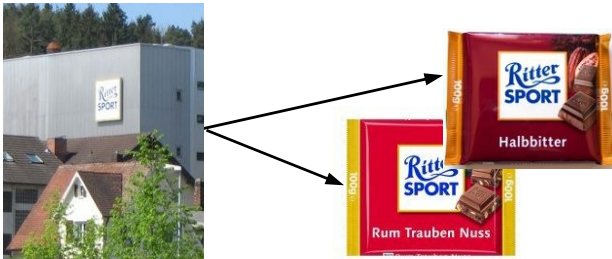
causal conclusions in real life are not always based on statistics!

these 2 objects are similar...



– *why* are they so similar?

# Conclusion: common history



similarities require an *explanation*

what kind of similarities require an explanation?



here we would *not* assume that anyone has copied the design...

..the pattern is too simple

- similarities require an explanation only if the pattern is sufficiently complex

## consider a binary sequence

### **Experiment:**

2 persons are instructed to write down a string with 1000 digits

### **Result:**

Both write 110010010000111110110101010001...

(all 1000 digits coincide)

the **naïve** statistician concludes



“There must be an agreement between the subjects”

correlation coefficient 1 (between digits) is highly significant for sample size 1000 !

- reject statistical independence
- infer the existence of a causal relation

## another mathematician recognizes...

$$11.0010010000111111011010101001... = \pi$$

- subjects may have come up with this number independently because it follows from a simple law
- superficially strong similarities are not necessarily significant if the pattern is too simple



How do we measure simplicity versus complexity of patterns / objects?

# Kolmogorov complexity

(Kolmogorov 1965, Chaitin 1966, Solomonoff 1964)  
of a binary string  $x$

- $K(x)$  = length of the shortest program with output  $x$  (on a Turing machine)
- interpretation: number of bits required to describe the rule that generates  $x$   
neglect string-independent additive constants; use  $\stackrel{+}{=}$  instead of  $=$
- strings  $x, y$  with low  $K(x)$ ,  $K(y)$  cannot have much in common
- $K(x)$  is uncomputable
- probability-free definition of information content

# Conditional Kolmogorov complexity

- $K(y|x)$ : length of the shortest program that generates  $y$  from the input  $x$ .
- number of bits required for describing  $y$  if  $x$  is given
- $K(y|x^*)$  length of the shortest program that generates  $y$  from  $x^*$ , i.e., the shortest compression  $x$ .
- subtle difference:  $x$  can be generated from  $x^*$  but not vice versa because there is no algorithmic way to find the shortest compression

# Algorithmic mutual information

Chaitin, Gacs

Information of  $x$  about  $y$  (and vice versa)

- $I(x : y) := K(x) + K(y) - K(x, y)$   
 $\quad \quad \quad \stackrel{\pm}{=} K(x) - K(x|y^*) \stackrel{\pm}{=} K(y) - K(y|x^*)$
- Interpretation: number of bits saved when compressing  $x, y$  jointly rather than compressing them independently

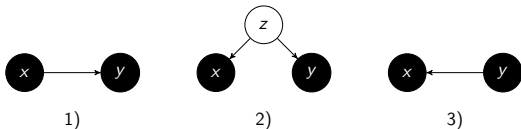
$$I(\star_{\bullet} : \star) = K(\star)$$

## Analogy to statistics:

- replace strings  $x, y$  (=objects) with random variables  $X, Y$
- replace Kolmogorov complexity with Shannon entropy
- replace algorithmic mutual information  $I(x : y)$  with statistical mutual information  $I(X; Y)$

# Causal Principle

If two strings  $x$  and  $y$  are algorithmically dependent then either



- every algorithmic dependence is due to a causal relation
- algorithmic analog to Reichenbach's principle of common cause
- distinction between 3 cases: use conditional independences on more than 2 objects

## conditional algorithmic mutual information

- $I(x : y|z) = K(x|z) + K(y|z) - K(x, y|z)$
- Information that  $x$  and  $y$  have in common when  $z$  is already given
- Formal analogy to statistical mutual information:

$$I(X : Y|Z) = S(X|Z) + S(Y|Z) - S(X, Y|Z)$$

- Define conditional independence:

$$I(x : y|z) \approx 0 :\Leftrightarrow x \perp\!\!\!\perp y|z$$



# Algorithmic Markov condition

Postulate [DJ & Schölkopf IEEE TIT 2010]

Let  $x_1, \dots, x_n$  be some observations (formalized as strings) and  $G$  describe their causal relations.

Then, every  $x_j$  is conditionally algorithmically independent of its non-descendants, given its parents, i.e.,

$$x_j \perp\!\!\!\perp nd_j \mid pa_j^*$$

# Equivalence of algorithmic Markov conditions

## Theorem

*For  $n$  strings  $x_1, \dots, x_n$  the following conditions are equivalent*

- **Local Markov condition:**

$$I(x_j : nd_j | pa_j^*) \stackrel{\pm}{=} 0$$

- **Global Markov condition:**

*$R$   $d$ -separates  $S$  and  $T$  implies  $I(S : T | R^*) \stackrel{\pm}{=} 0$*

- **Recursion formula for joint complexity**

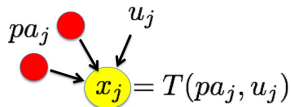
$$K(x_1, \dots, x_n) \stackrel{\pm}{=} \sum_{j=1}^n K(x_j | pa_j^*)$$

→ another analogy to statistical causal inference

# Algorithmic model of causality

Given  $n$  causality related strings  $x_1, \dots, x_n$

- each  $x_j$  is computed from its parents  $pa_j$  and an unobserved string  $u_j$  by a Turing machine  $T$



- all  $u_j$  are algorithmically independent
- each  $u_j$  describes the causal mechanism (the program) generating  $x_j$  from its parents
- $u_j$  is the analog of the noise term in the statistical functional model

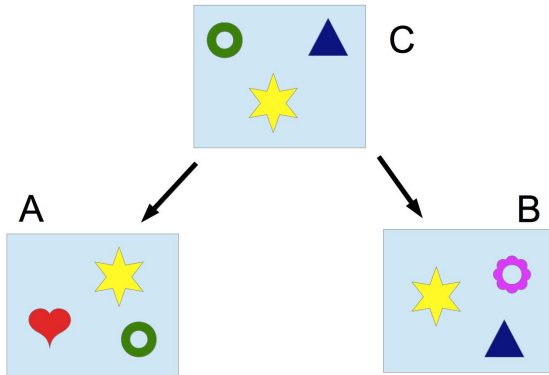
# Algorithmic model of causality implies Markov condition

## Theorem

*If  $x_1, \dots, x_n$  are generated by an algorithmic model of causality according to the DAG  $G$  then they satisfy the 3 equivalent algorithmic Markov conditions.*

# Causal inference for single objects

3 carpets



conditional independence  $A \perp\!\!\!\perp B \mid C$

## Take home messages

- Graphical causal models do not *solve* the hard causal problems, but they provide a clear framework to address them
- Subject to *strong* assumptions, causal structure can also be inferred from passive observation
- However, machine learning is used to rely on strong assumptions

# References

-  J. Pearl.  
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Thank you for your attention!

note also the following competition:  
<https://causeme.uv.es/neurips2019/>