Causal Inference

Conditional Independences and Beyond

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Roadmap

- informal motivation
- functional causal models
- causal graphical models;
  d-separation, Markov conditions, faithfulness
- formalizing interventions
- causal inference...
  - using time order
  - using conditional independences
  - using restricted function classes
  - using "independence" of mechanisms
  - not using statistics

Dependence vs. Causation

Storks Deliver Babies (p= 0.008)

Robert Matthews

Article first published online: 25 DEC 2001

DOI: 10.1111/1467-9639.00013

Teaching Statistics Trust, 2000

Issue

Teaching Statisti
Volume 22, Issue
38, June 2000

Country	Area (km^2)	Storks (pairs)	Humans (10^6)	Birth rate (10^3/yr)
Albania	28,750	100	3.2	83
Austria	83,860	300	7.6	87
Belgium	30,520	1	9.9	118
Bulgaria	111,000	5000	9.0	117
Denmark	43,100	9	5.1	59
France	544,000	140	56	774
Germany	357,000	3300	78	901
Greece	132,000	2500	10	106
Holland	41,900	4	15	188
Hungary	93,000	5000	11	124
Italy	301,280	5	57	551
Poland	312,680	30,000	[illegible]	[illegible]
Portugal	92,390	1500	10	120
Romania	237,500	5000	23	367
Spain	504,750	8000	39	439
Switzerland	41,290	150	6.7	82
Turkey	779,450	25,000	56	1576

mailto:rajm@compuserve.com

Table 1. Geographic, human and stork data for 17 European countries

Chandoo.org

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Amazon's recommendation system – is it crazy?

Posted on January 12th, 2008 in business , Humor , technology , wonder why - 6 comments

We have a saying in *Telugu* that goes like this, "*thaadu vundhi kada ani eddu kontama?*" which means, "just because you have a rope you dont buy a bullock to tie". Amazon's recommendation system must have been coded by someone with a skewed view of reality. How else can you explain this?

Thanks to P. Laskov.

The NEW ENGLAND JOURNAL of MEDICINE

ORIGINAL ARTICLE

Association of Coffee Drinking with Total and Cause-Specific Mortality

Neal D. Freedman, Ph.D., Yikyung Park, Sc.D., Christian C. Abnet, Ph.D., Albert R. Hollenbeck, Ph.D., and Rashmi Sinha, Ph.D.

N Engl J Med 2012; 366:1891-1904 | May 17, 2012

BACKGROUND

Coffee is one of the most widely consumed beverages, but the association between coffee consumption and the risk of death remains unclear.

Full Text of Background...

METHODS

We examined the association of coffee drinking with subsequent total and cause-specific mortality among 229,119 men and 173,141 women in the National Institutes of Health–AARP Diet and Health Study who were 50 to 71 years of age at baseline. Participants with cancer, heart disease, and stroke were excluded. Coffee consumption was assessed once at baseline.

We present risk estimates separately for men and women. Multivariate models were adjusted for the following baseline factors: age; body-mass index (BMI); race or ethnic group; level of education; alcohol consumption; the number of cigarettes smoked per day, use or nonuse of pipes or cigars, and time of smoking cessation (<1 year, 1 to <5 years, 5 to <10 years, or ≥10 years before baseline); health status; presence or absence of diabetes; marital status; level of physical activity; total energy intake; consumption of fruits, vegetables, red meat, white meat, and saturated fat; and use of any vitamin supplement (yes vs. no). In addition, risk estimates for death from cancer were adjusted for history of cancer (other than nonmelanoma skin cancer) in a first-degree relative (yes vs. no). For women, status with respect to postmenopausal hormone therapy was also included in multivariate models. Less than 5% of the cohort lacked any single covariate; for each covariate, we

RESULTS

During 5,148,760 person-years of follow-up between 1995 and 2008, a total of 33,731 men and 18,784 women died. In age-adjusted models, the risk of death was increased among coffee drinkers. However, coffee drinkers were also more likely to smoke, and, after adjustment for tobacco-smoking status and other potential confounders, there was a significant inverse association between coffee consumption and mortality. Adjusted hazard ratios for death among men who drank

CONCLUSIONS

In this large prospective study, coffee consumption was inversely associated with total and cause-specific mortality. Whether this was a causal or associational finding cannot be determined from our data.

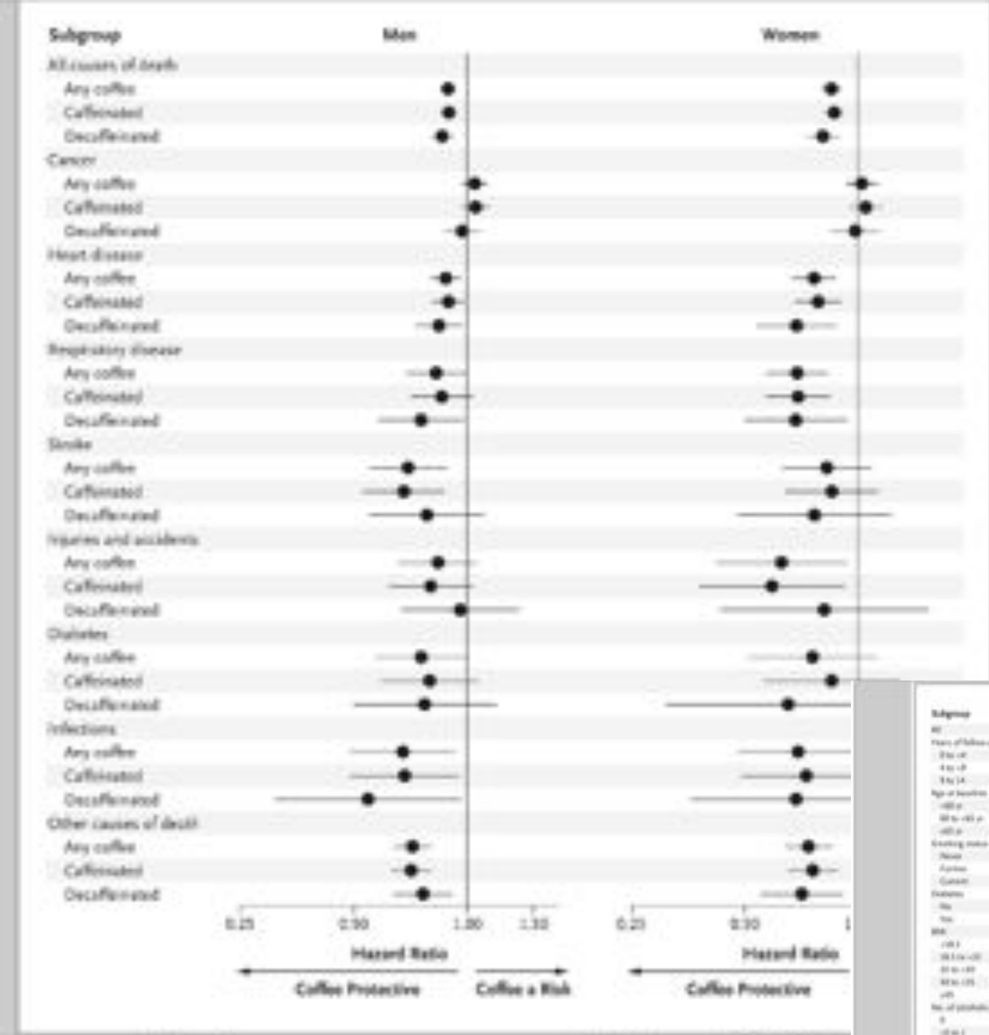

Figure 1. Subgroup Analysis of Associations between the Consumption of 4 or More Cups of Coffee per Day and Total and Cause-Specific Mortality.

Hazard ratios for death from all causes and from specific causes are for the comparison of men and women who drank 4 or more cups of coffee per day with those who did not drink coffee. Participants were classified as drinking caffeinated or decaffeinated coffee according to whether they reported drinking caffeinated or decaffeinated coffee more than half the time. Risk estimates for other categories of coffee consumption are shown in Tables 2 and 3 in the Supplementary Appendix. Risk estimates were adjusted for the following factors at baseline: age; body-mass index; race or ethnic group; level of education; alcohol consumption; the number of cigarettes smoked per day, use or nonuse of pipes or cigars, and time of smoking cessation (<1 year, 1 to <5

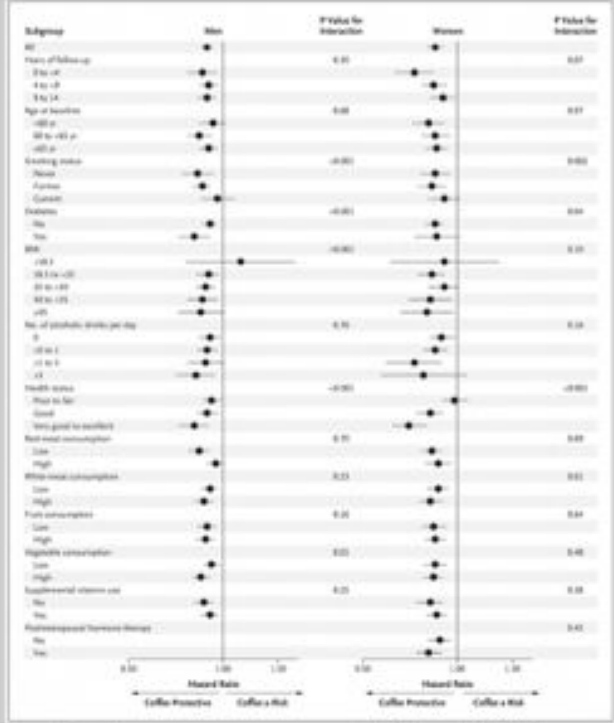

Figure 2. Subgroup Analysis of Associations between the Consumption of 4 or More Cups of Coffee per Day and Total Mortality.

Hazard ratios for death from any cause are for the comparison of men and women who drank 4 or more cups of coffee per day with those who did not drink coffee. The multivariate model was adjusted for the following factors at baseline: age; body-mass index (BMI; the weight in kilograms divided by the square of the height in meters); race or ethnic group; level of education; alcohol consumption; the number of cigarettes smoked per day, use or nonuse of pipes or cigars, and time of smoking cessation (<1 year, 1 to <5 years, 5 to <10 years, or 10 years before baseline); health status; diabetes (yes vs. no); marital status; physical activity; total energy intake; consumption of fruits, vegetables, red meat, white meat, and saturated fat; use or nonuse of vitamin supplements; and, in women, use or nonuse of postmenopausal hormone therapy. Risk estimates for other categories of coffee consumption are shown in Tables 4 and 5 in the Supplementary Appendix. High and low dietary-intake categories are split at the median. Horizontal lines represent 95% confidence intervals. P values for interactions were computed with the use of likelihood-ratio tests comparing Cox proportional-hazards models with and without cross-product terms for each level of baseline stratifying variables, with coffee consumption as an ordinal variable. P values for the years of follow-up were derived from testing the addition of a cross-product

MAX-PLANCK-GESELLSCHAFT

12.12.2007

Deutsches Kinderkrebsregister untersucht Häufigkeit von Krebserkrankungen bei Kindern in der Nähe von Kernkraftwerken

Neue Studie veröffentlicht

Immer wieder wird der Verdacht geäußert, dass Kinder in der Nähe von Kernkraftwerken häufiger an Krebs erkranken. Eine frühere Studie des Kinderkrebsregisters mit Kindern unter 15 Jahren schien darauf hinzudeuten, dass speziell in den ersten Lebensjahren das Leukämie-Risiko in den betreffenden Gegenden erhöht war.

In diesen Tagen erscheinen zwei wissenschaftliche Veröffentlichungen über eine neue Studie des Deutschen Kinderkrebsregisters in Mainz. Das Ergebnis: In Deutschland findet man einen Zusammenhang zwischen der Nähe der Wohnung zu einem Kernkraftwerk und der Häufigkeit, mit der Kinder vor ihrem fünften Geburtstag an Krebs und besonders an Leukämie erkranken. Allerdings erlaubt die Studie keine Aussage darüber, wodurch sich die beobachtete Erhöhung der Anzahl von Kinderkrebsfällen in der Umgebung deutscher Kernkraftwerke erklären lässt. So kommt nach dem heutigen Wissensstand Strahlung, die von Kernkraftwerken im Normalbetrieb ausgeht, als Ursache für die beobachtete Risikoerhöhung nicht in Betracht. Denkbar wäre, dass bis jetzt noch unbekannte Faktoren beteiligt sind oder dass es sich doch um Zufall handelt.

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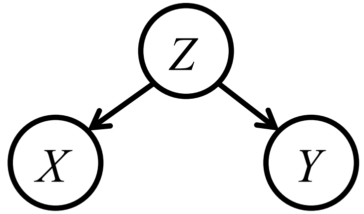
“Correlation does not tell us anything about causality”

- Better to talk of dependence than correlation
- Most statisticians would agree that causality does tell us something about dependence
- But dependence does tell us something about causality too:

Statistical Implications of Causality

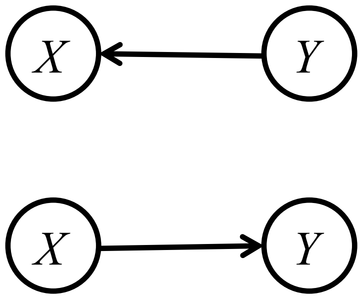
Reichenbach's
Common Cause Principle
links **causality** and **probability:**

(i) if X and Y are statistically dependent, then there is a Z causally influencing both;



special cases:

(ii) Z screens X and Y from each other (given Z, the observables X and Y become independent)



Notation

- A, B event
- X, Y, Z random variable
- x value of a random variable
- $\Pr$ probability measure
- P_X probability distribution of X
- p density
- p_X or $p(X)$ density of P_X
- $p(x)$ density of P_X evaluated at the point x
- always assume the existence of a joint density, w.r.t. a product measure

Independence

Two events A and B are called *independent* if

$$\Pr(A \cap B) = \Pr(A) \cdot \Pr(B).$$

$A_1, \dots, A_n$ are called *independent* if for every subset $S \subset \{1, \dots, n\}$ we have

$$\Pr\left(\bigcap_{i \in S} A_i\right) = \prod_{i \in S} \Pr(A_i).$$

Note: for $n \geq 3$, *pairwise independence* $\Pr(A_i \cap A_j) = \Pr(A_i) \cdot \Pr(A_j)$ for all i, j does not imply *independence*.

Independence of random variables

Two real-valued random variables X and Y are called *independent*,

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

if for every $a, b \in \mathbb{R}$, the events $\{X \leq a\}$ and $\{Y \leq b\}$ are independent.

Equivalently, in terms of densities: for all x, y,

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

Note:

If $X \perp\!\!\!\perp Y$, then $E[XY] = E[X]E[Y]$, and $\text{cov}[X, Y] = E[XY] - E[X]E[Y] = 0$.

The converse is not true: $\text{cov}[X, Y] = 0 \not\Rightarrow X \perp\!\!\!\perp Y$.

However, we have, for large $\mathcal{F}$: $(\forall f, g \in \mathcal{F} : \text{cov}[f(X), g(Y)] = 0) \Rightarrow X \perp\!\!\!\perp Y$

Conditional Independence of random variables

Two real-valued random variables X and Y are called *conditionally independent* given Z,

$$(X \perp\!\!\!\perp Y)\,|Z \quad \text{or} \quad X \perp\!\!\!\perp Y\,|Z \quad \text{or} \quad (X \perp\!\!\!\perp Y\,|Z)_p$$

if

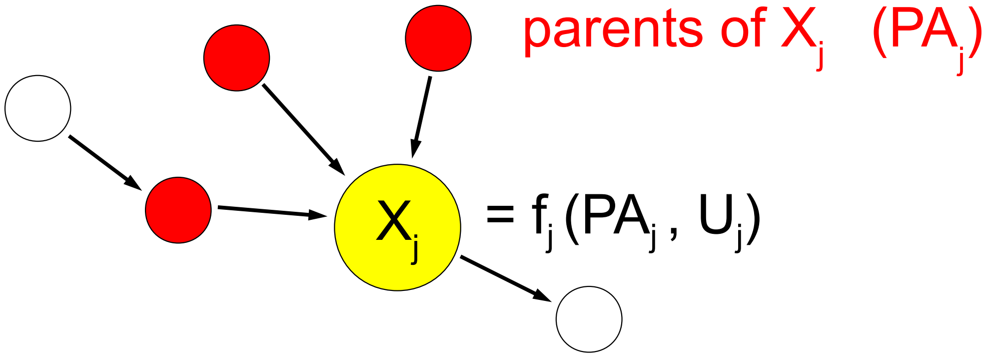
$$p(x, y|z) = p(x|z)p(y|z)$$

for all x, y, and for all z s.t. $p(z) > 0$.

Note: conditional independence neither implies nor is implied by independence.
I.e., there are X, Y, Z such that we have only independence or only conditional independence.

Functional Causal Model *(Pearl et al.)*

- Set of observables $X_1, \ldots, X_n$
- directed acyclic graph G with vertices $X_1, \ldots, X_n$
- Semantics: parents = direct causes
- $X_i = f_i(\text{ParentsOf}_i, \text{Noise}_i)$, with independent $\text{Noise}_1, \ldots, \text{Noise}_n$.
- "Noise" means "unexplained" (or "exogenous"), we use U_i
- Can add requirement that $f_1, \ldots, f_n, \text{Noise}_1, \ldots, \text{Noise}_n$ "independent" (cf. *Lemeire & Dirkx 2006, Janzing & Schölkopf 2010* — more below)

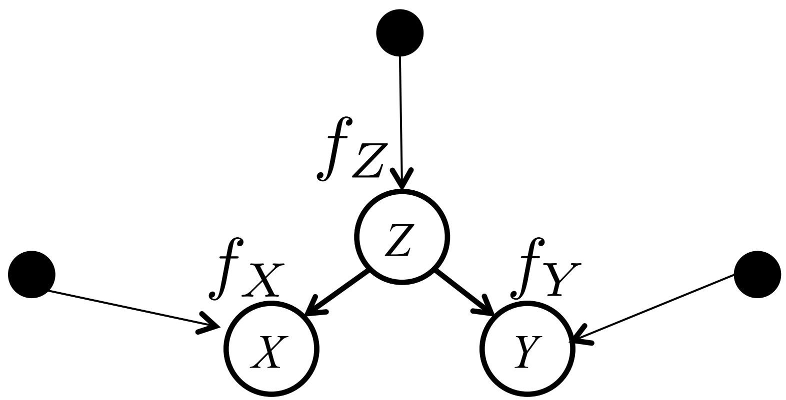


Functional Causal Model, ctd.

- this model can be shown to satisfy Reichenbach's principle:

  1. functions of independent variables are independent, hence dependence can only arise in two vertices that depend (partly) on the same noise term(s).

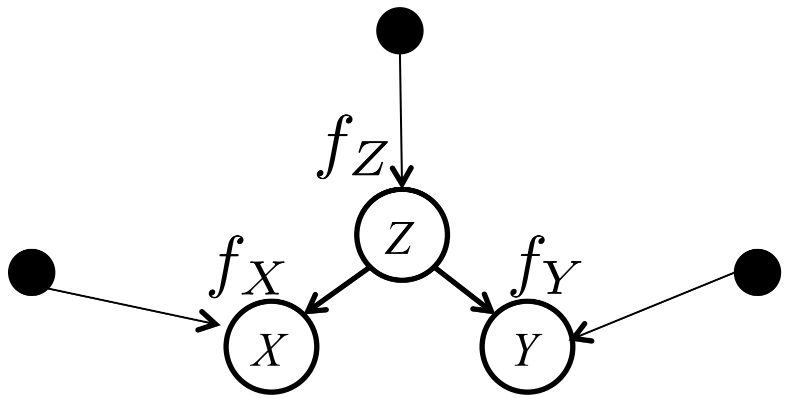
  2. if we condition on these noise terms, the variables become independent



Functional Causal Model, ctd.

- Independence of noises is a form of "causal sufficiency:" if the noises were dependent, then Reichenbach's principle would tell us the causal graph is incomplete

- Interventions are realized by replacing functions by values



- the model entails a joint distribution $p(X_1, \ldots, X_n)$. **Questions:**

  (1) What can we say about it?

  (2) Can we recover G from p?

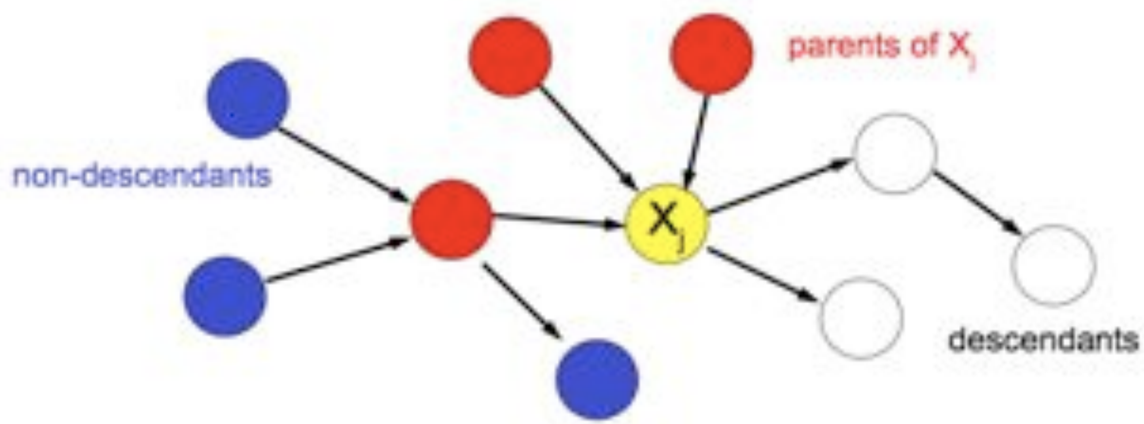
Functional Model and Markov conditions

(Lauritzen 1996, Pearl 2000)

Theorem: the following are equivalent:

- Existence of a functional causal model
- Local Causal Markov condition: X_j *statistically independent* of non-descendants, given parents (i.e.: every information exchange with its non-descendants involves its parents)
- Global Causal Markov condition: d-separation (characterizes the set of independences implied by local Markov condition)
- Factorization $p(X_1, \ldots, X_n) = \prod_j p(X_j \mid \text{Parents}_j)$ (conditionals as causal mechanisms generating statistical dependence)

(subject to technical conditions)



Counterfactuals and Interventions

- David Hume (1711–76): *"... we may define a cause to be an object, followed by another, and where all the objects similar to the first are followed by objects similar to the second. Or in other words where, if the first object had not been, the second never had existed."*

- Jerzy Neyman (1923): consider m plots of land and ν varieties of crop.

  Denote U_{ij} the crop yield that *would be* observed if variety $i = 1, \ldots, \nu$ were planted in plot $j = 1, \ldots, m$

  For each plot j, we can only experimentally determine *one* U_{ij} in each growing season.

  The others are called "counterfactuals".

- this leads to the view of causal inference as a missing data problem — the "potential outcomes" framework (Rubin, 1974)

- in $X_i = f_i(\mathrm{ParentsOf}_i, \mathrm{Noise}_i)$, the equality sign is interpreted as an assignment ":=" — interventions can only take place on the right hand side

From Ordinary Differential Equations to Structural Causal Models: the deterministic case

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[stat.OT] 30 Apr 2013

Abstract

We show how, and under which conditions, the equilibrium states of a first-order Ordinary Differential Equation (ODE) system can be described with a deterministic Structural Causal Model (SCM). Our exposition sheds more light on the concept of causality as expressed within the framework of Structural Causal Models, especially for cyclic models.

algorithms (starting from different assumptions) have been proposed for inferring cyclic causal models from observational data (Richardson, 1996; Lacerda et al., 2008; Schmidt and Murphy, 2009; Itani et al., 2010; Mooij et al., 2011).

The most straightforward extension to the cyclic case seems to be offered by the structural causal model framework. Indeed, the formalism stays intact when one simply drops the acyclicity constraint. However, the question then arises how to interpret cyclic structural equations. One option is to assume an under-

UAI 2013

Pearl's do-calculus

- Motivation: goal of causality is to infer the effect of interventions

- distribution of Y given that X is set to x:

$$p(Y|do\,X=x) \quad \text{or} \quad p(Y|do\,x)$$

- don't confuse it with $P(Y|x)$

- can be computed from p and G

Computing $p(X_1, \ldots, X_n | do\, x_i)$

from $p(X_1, \ldots, X_n)$ and G

- Start with causal factorization

$$p(X_1, \ldots, X_n) = \prod_{j=1}^{n} p(X_j | PA_j)$$

- Replace $p(X_i | PA_i)$ with $\delta_{X_i x_i}$

$$p(X_1, \ldots, X_n | do\, x_i) := \prod_{j \neq i} p(X_j | PA_j) \delta_{X_i x_i}$$

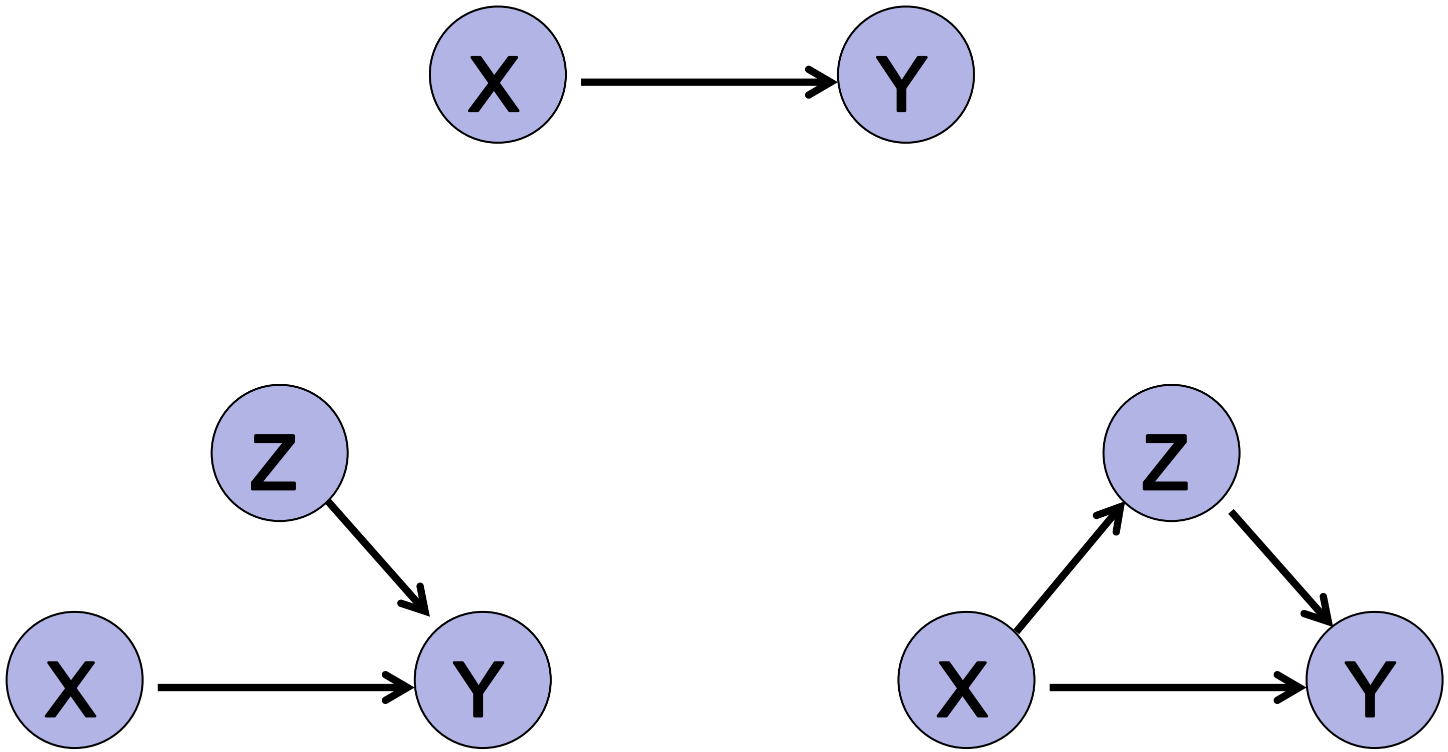
Computing $p(X_k|do\,x_i)$

summation over x_i yields

$$p(X_1,\ldots,X_{i-1},X_{i+1},\ldots,X_n|do\,x_i) = \prod_{j\neq i} p(X_j|PA_j(x_i))\,.$$

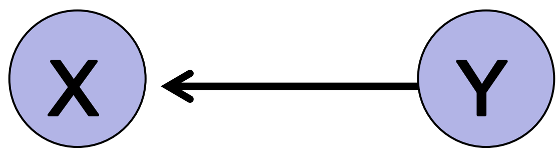
- distribution of X_j with $j \neq i$ is given by dropping $p(X_i|PA_i)$ and substituting x_i into PA_j to get $PA_j(x_i)$.
- obtain $p(X_k|do\,x_i)$ by marginalization

Examples for $p(.|do\, x) = p(.|x)$

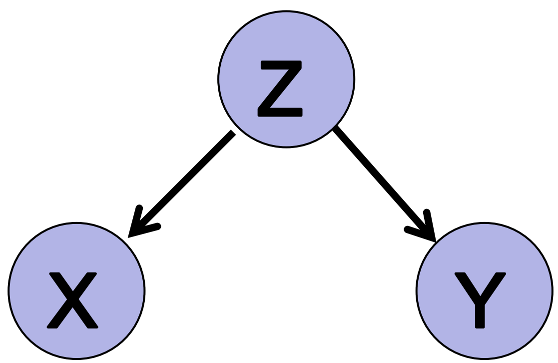


Examples for $p(.|do\, x) \neq p(.|x)$

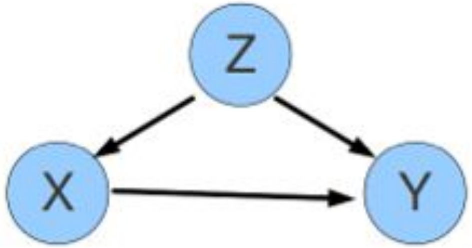
- $p(Y|do\, x) = P(Y) \neq P(Y|x)$



- $p(Y|do\, x) = P(Y) \neq P(Y|x)$



Example: controlling for confounding



$X \not\perp\!\!\!\perp Y$ partly due to the confounder Z and partly due to $X \to Y$

- causal factorization

$$p(X, Y, Z) = p(Z)p(X|Z)p(Y|X, Z)$$

- replace $P(X|Z)$ with δ_{Xx}

$$p(Y, Z|do\, x) = p(Z)\ \delta_{Xx}\ p(Y|X, Z)$$

- marginalize

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

Identifiability problem

e.g. Tian & Pearl (2002)

- given the causal DAG G and two nodes X_i, X_j
- which nodes need to be observed to compute $p(X_i | do\, x_j)$?

Inferring the DAG

- Key postulate: Causal Markov condition

- Essential mathematical concept: d-separation
  (describes the conditional independences required by a causal DAG)

d-separation (Pearl 1988)

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

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

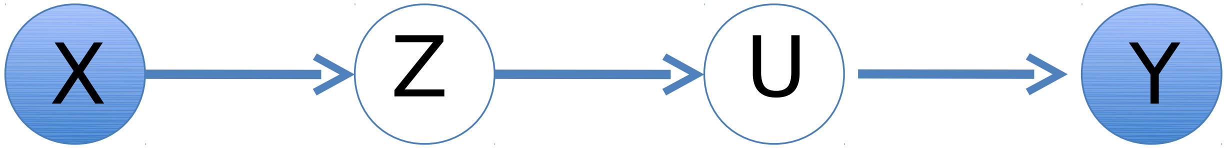
- q contains a *chain* $i \rightarrow m \rightarrow j$ or a *fork* $i \leftarrow m \rightarrow j$ such that the middle node is in Z, or
- q contains a *collider* $i \rightarrow m \leftarrow j$ such that the middle node is not in Z and such that no descendant of m is in Z.

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

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

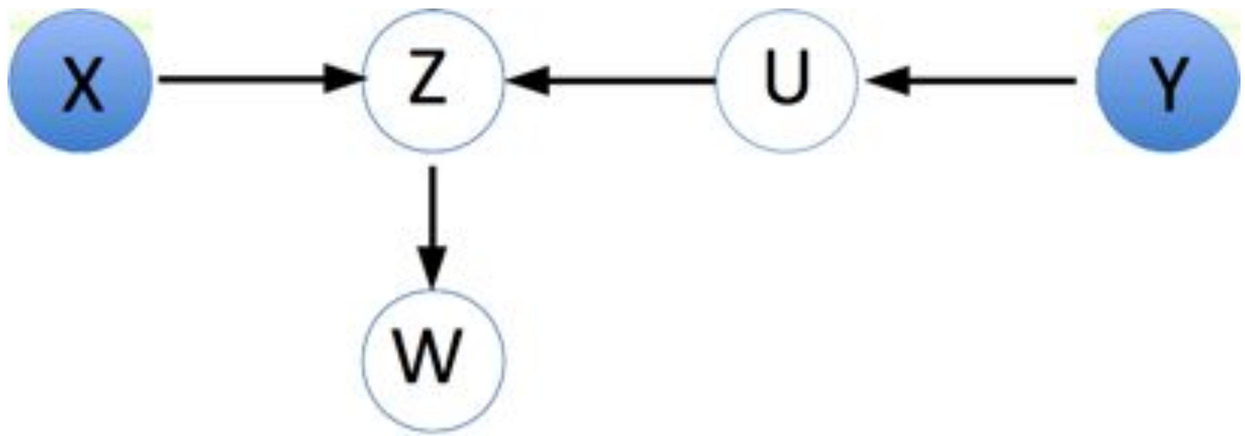
if Z blocks every path from a node in X to a node in Y.

Example (blocking of paths)



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

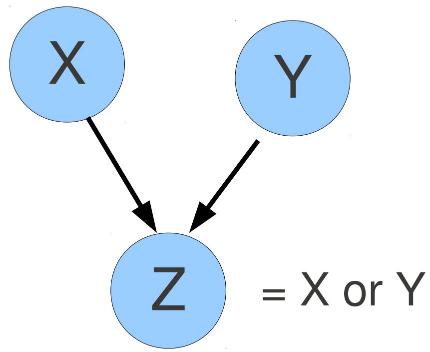
Example (unblocking of paths)



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

Unblocking by conditioning on common effects

Berkson's paradox (1946)
Example: X, Y, Z binary

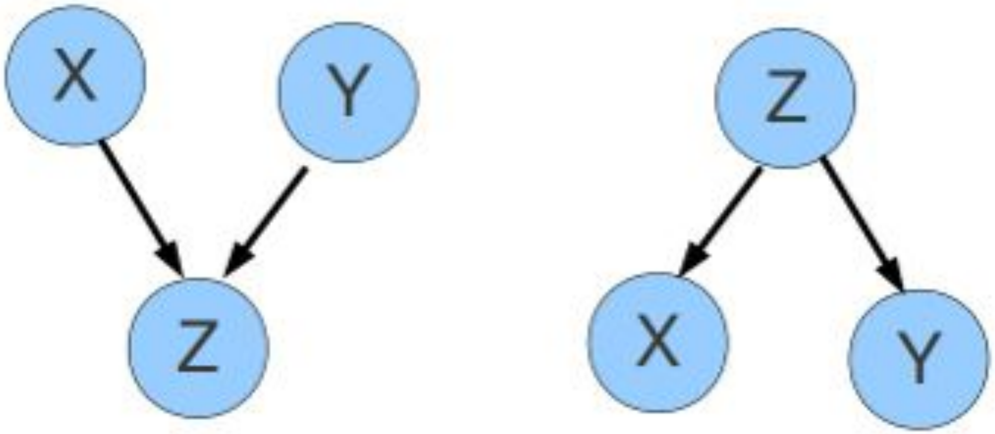


$$X \perp\!\!\!\perp Y \quad \text{but} \quad X \not\perp\!\!\!\perp Y \,|Z$$

- assume: for politicians there is no correlation between being a good speaker and being intelligent
- politician is successful if (s)he is a good speaker or intelligent
- among the successful politicians, being intelligent is negatively correlated with being a good speaker

Asymmetry under inverting arrows

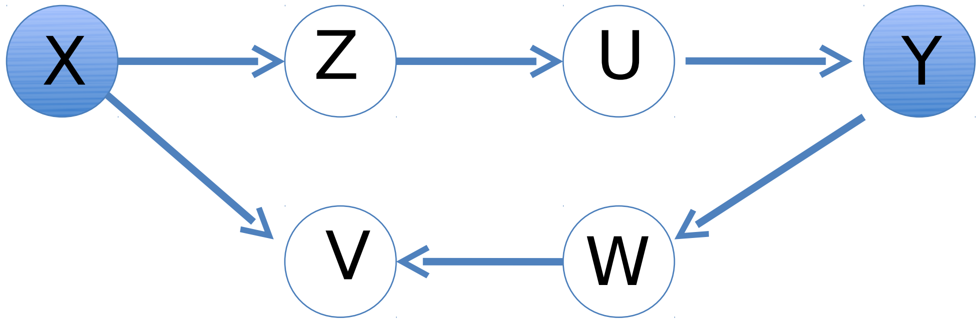
(Reichenbach 1956)



$$X \perp\!\!\!\perp Y \qquad\qquad X \not\!\perp\!\!\!\perp Y$$

$$X \not\!\perp\!\!\!\perp Y \,|Z \qquad\qquad X \perp\!\!\!\perp Y \,|Z$$

Examples (d-separation)

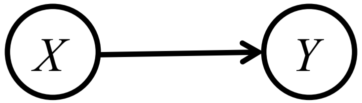


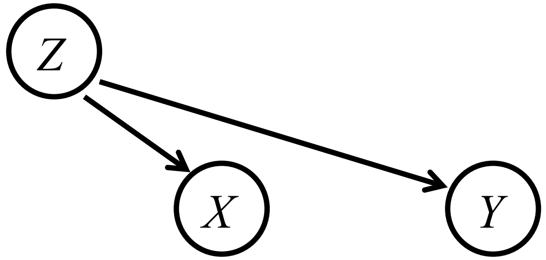
$$(X \perp\!\!\!\perp Y \,|ZW)_G$$
$$(X \perp\!\!\!\perp Y \,|ZUW)_G$$
$$(X \perp\!\!\!\perp Y \,|VZUW)_G$$

$$(X \not\!\perp\!\!\!\perp Y \,|VZU)_G$$

Causal inference for time-ordered variables

assume $X \not\perp\!\!\!\perp Y$ and X earlier. Then $X \leftarrow Y$ excluded, but still two options:





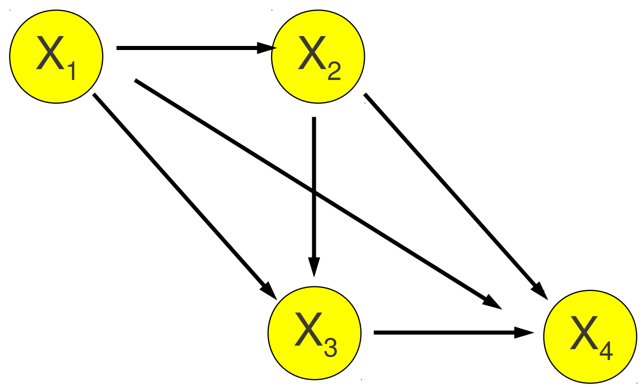
Example (Fukumizu 2007): barometer falls before it rains, but it does not cause the rain

Conclusion: time order makes causal problem (slightly?) easier but does not solve it

Causal inference for time-ordered variables

assume $X_1, \ldots, X_n$ are time-ordered and **causally sufficient**

- start with complete DAG



- remove as many parents as possible:

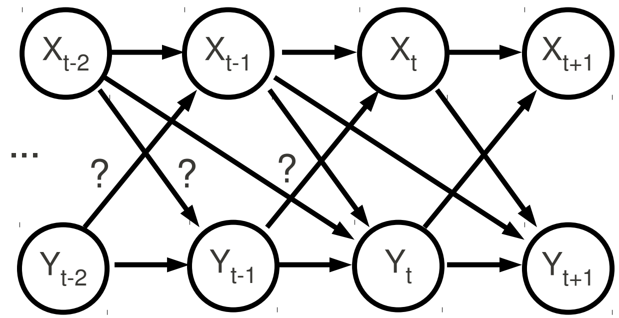
  $p \in PA_j$ can be removed if

$$X_j \perp\!\!\!\perp p \,|\, PA_j \setminus p$$

(going from potential arrows to true arrows "only" requires statistical testing)

Time series and Granger causality

Does X cause Y and/or Y cause X?



exclude instantaeous effects and common causes

- if

$$Y_{present} \not\perp\!\!\!\perp X_{past} \,|Y_{past}$$

  there must be arrows from X to Y (otherwise d-separation)

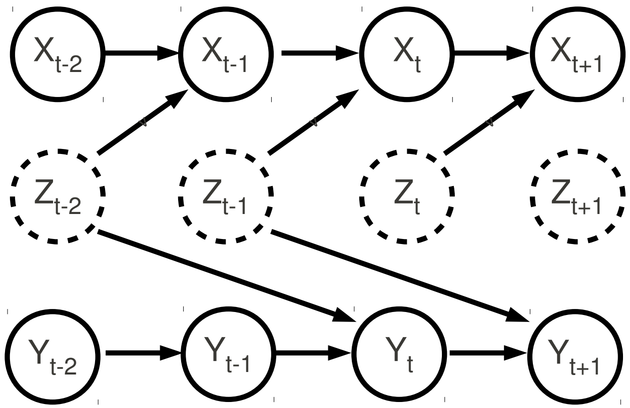
- Granger (1969): the past of X helps when predicting Y_t from its past

- strength of causal influence often measured by transfer entropy

$$I(Y_{present}; X_{past} \,|Y_{past})$$

Confounded Granger

Hidden common cause Z relates X and Y



due to different time delays we have

$$Y_{present} \not\perp\!\!\!\perp X_{past} \,|Y_{past}$$

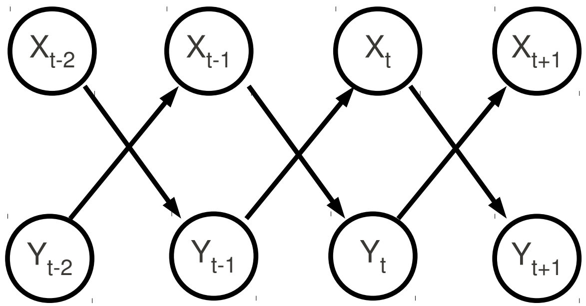
but

$$X_{present} \perp\!\!\!\perp Y_{past} \,|X_{past}$$

Granger infers $X \to Y$

Why transfer entropy does not quantify causal strength (Ay & Polani, 2008)

deterministic mutual influence between X and Y



- although the influence is strong

$$I(Y_{present}; X_{past} \,|Y_{past}) = 0\,,$$

  because the past of Y already determines its present

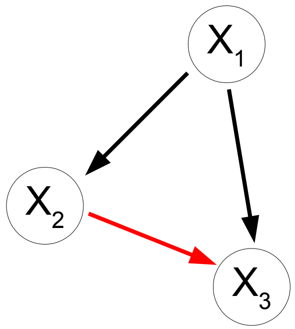
- quantitatively still wrong for non-deterministic relation

- recent paper on definitions of causal strength: Janzing, Balduzzi, Grosse-Wentrup, Schölkopf, *Annals of Statistics* 2013

Quantifying causal influence for general DAGs

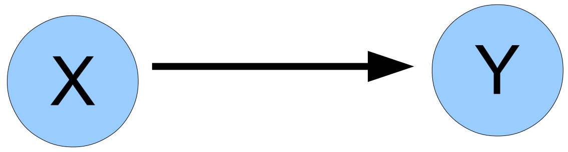
Given:
causally sufficient set of variables $X_1, \ldots, X_n$ with

- known causal DAG G
- known joint distribution $P(X_1, \ldots, X_n)$



Goal:
construct a measure that quantifies the strength of $X_i \rightarrow X_j$ with the following properties:

Postulate 1: (mutual information)



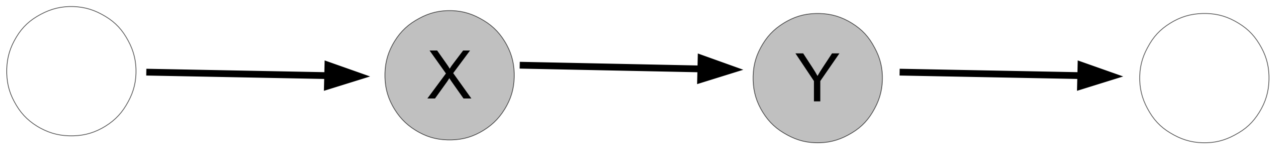
For this simple DAG we postulate

$$c_{X \to Y} = I(X;Y)$$

(no other path from X to Y, hence the dependence is caused by the arrow $X \to Y$)

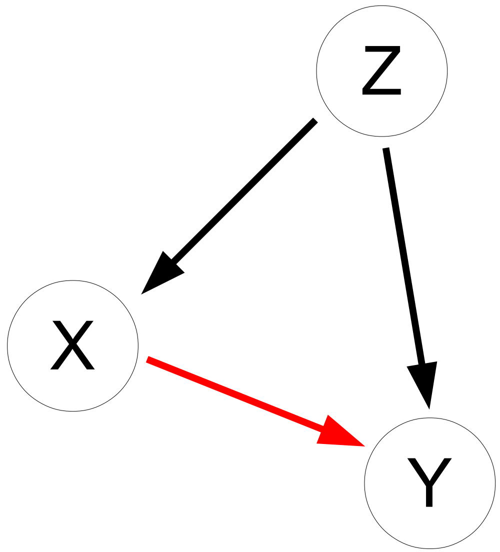
Postulate 2: (localility)

causes of causes and effects of effects don't matter



here we also postulate $c_{X\to Y} = I(X;Y)$

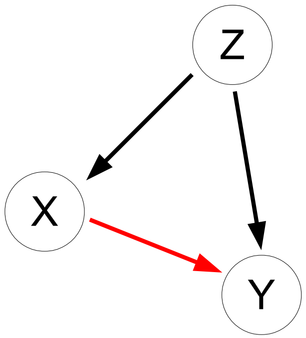
Postulate 3: (strength majorizes conditional dependence, given the other parents)



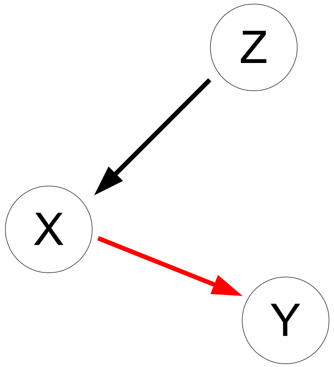
$$c_{X \to Y} \geq I(X;Y\,|Z)$$

(without $X \to Y$ the Markov condition would imply $I(X;Y\,|Z) = 0$)

Why $c_{X\to Y} = I(X;Y\,|Z)$ is a bad idea



contains



as a limiting case (weak influence $Z \to Y$),

where we postulated $c_{X\to Y} = I(X;Y)$ instead of $I(X;Y\,|Z)$

Our approach: "edge deletion"

- define a new distribution

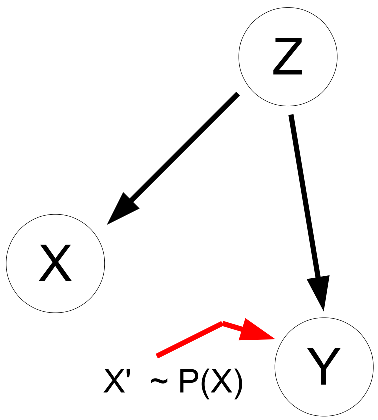
$$P_{X\to Y}(x,y,z) = P(z)P(x|z)\sum_{x'} P(y|x',z)P(x')$$

- define causal strength by the 'impact of edge deletion'

$$c_{X\to Y} := D(P\|P_{X\to Y})$$

- intuition of edge deletion:

  cut the wire between devices and feed the open end with an iid copy of the original signal



related work:
Ay & Krakauer (2007)

Properties of our measure

- strength also defined for set of edges
- satisfies all our postulates
- also applicable to time series
- conceptually more reasonable than Granger causality and transfer entropy

Inferring the causal DAG without time information

- Setting: given observed n-tuples drawn from $p(X_1, \ldots, X_n)$, infer G
- Key postulates: Causal Markov condition and causal faithfulness

Causal faithfulness

Spirtes, Glymour, Scheines

p is called faithful relative to G if only those independences hold true that are implied by the Markov condition, i.e.,

$$(X \perp\!\!\!\perp Y \,|Z)_G \quad \Leftarrow \quad (X \perp\!\!\!\perp Y \,|Z)_p$$

Recall: Markov condition reads

$$(X \perp\!\!\!\perp Y \,|Z)_G \quad \Rightarrow \quad (X \perp\!\!\!\perp Y \,|Z)_p$$

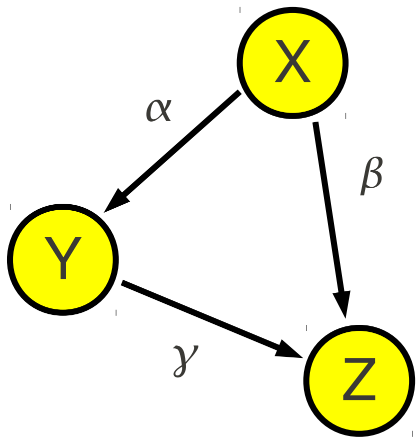
Examples of unfaithful distributions (1)

Cancellation of direct and indirect influence in linear models

$$\begin{aligned} X &= U_X \\ Y &= \alpha X + U_Y \\ Z &= \beta X + \gamma Z + U_Z \end{aligned}$$

with independent noise terms U_X, U_Y, U_Z

$$\beta + \alpha\gamma = 0 \quad \Rightarrow \quad X \perp\!\!\!\perp Z$$

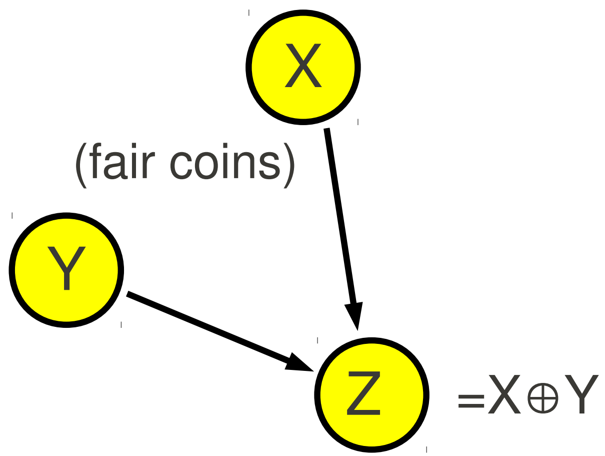


Examples of unfaithful distributions (2)

binary causes with XOR as effect

- for $p(X), p(Y)$ uniform: $X \perp\!\!\!\perp Z$, $Y \perp\!\!\!\perp Z$.
  i.e., unfaithful (since X, Z and Y, Z are connected in the graph).

- for $p(X), p(Y)$ non-uniform: $X \not\perp\!\!\!\perp Z$, $Y \not\perp\!\!\!\perp Z$.
  i.e., faithful



unfaithfulness considered unlikely because it only occurs for non-generic parameter values

Conditional-independence based causal inference

Spirtes, Glymour, Scheines and Pearl

Causal Markov condition + Causal faithfulness:

- accept only those DAGs G as causal hypotheses for which

$$(X \perp\!\!\!\perp Y \,|Z)_G \quad \Leftrightarrow \quad (X \perp\!\!\!\perp Y \,|Z)_p \,.$$

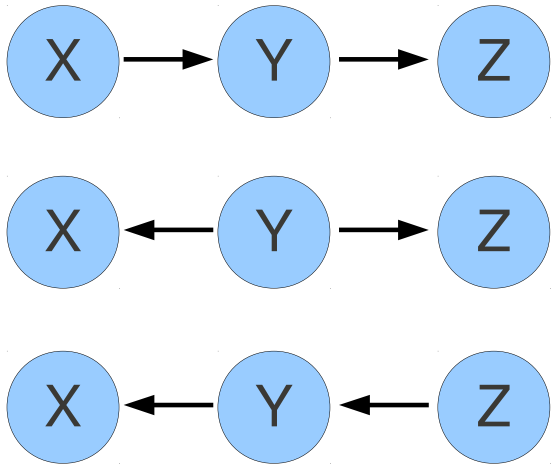
- identifies causal DAG up to Markov equivalence class
  (DAGs that imply the same conditional independences)

Markov equivalence class

Theorem (Verma and Pearl, 1990): two DAGs are Markov equivalent iff they have the same skeleton and the same v-structures.

skeleton: corresponding undirected graph
v-structure: substructure $X \to Y \leftarrow Z$ with no edge between X and Z

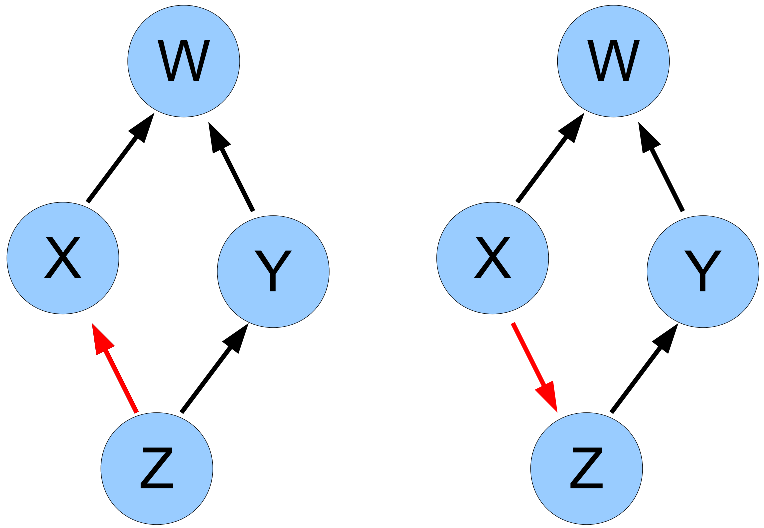
Markov equivalent DAGs



same skeleton, no v-structure

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

Markov equivalent DAGs



same skeleton, same v-structure at W

Algorithmic construction of causal hypotheses

IC algorithm by Verma & Pearl (1990) to reconstruct DAG from p

idea:

1. Construct skeleton
2. Find v-structures
3. direct further edges that follow from
   - graph is acyclic
   - all v-structures have been found in 2)

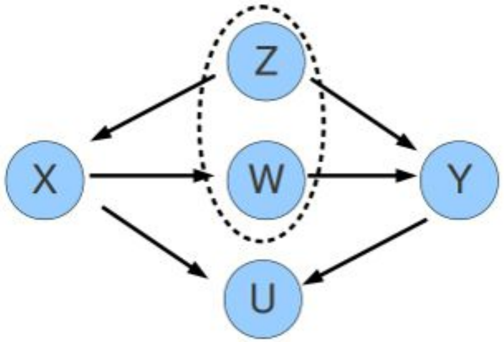
Construct skeleton

Theorem: X and Y are linked by an edge iff there is no set S_{XY} such that

$$(X \perp\!\!\!\perp Y \,|\, S_{XY} \,.$$

(assuming Markov condition and Faithfulness)

Explanation: dependence mediated by other variables can be screened off by conditioning on an appropriate set



$$X \perp\!\!\!\perp Y \,|\, \{Z, W\}$$

...but not by conditioning on all other variables!

S_{XY} is called a Sepset for (X, Y)

Efficient construction of skeleton

PC algorithm by Spirtes & Glymour (1991)

iteration over size of Sepset

1. remove all edges $X - Y$ with $X \perp\!\!\!\perp Y$
2. remove all edges $X - Y$ for which there is a neighbor $Z \neq Y$ of X with $X \perp\!\!\!\perp Y \,|Z$
3. remove all edges $X - Y$ for which there are two neighbors $Z_1, Z_2 \neq Y$ of X with $X \perp\!\!\!\perp Y \,|Z_1, Z_2$
4. ...

Advantages

- many edges can be removed already for small sets
- testing all sets S_{XY} containing the adjacencies of X is sufficient
- depending on sparseness, algorithm only requires independence tests with small conditioning tests
- polynomial for graphs of bounded degree

Find v-structures

- given $X - Y - Z$ with X and Y non-adjacent
- given S_{XY} with $X \perp\!\!\!\perp Y \,|S_{XY}$

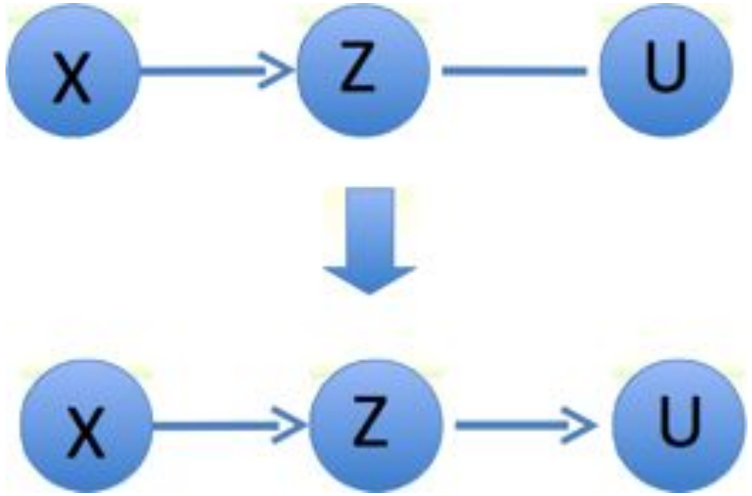
a priori, there are 4 possible orientations:

$$\left.\begin{array}{l} X \rightarrow Z \rightarrow Y \\ X \leftarrow Z \rightarrow Y \\ X \leftarrow Z \leftarrow Y \end{array}\right\} \qquad\qquad Z \in S_{XY}$$

$$X \rightarrow Z \leftarrow Y \qquad\qquad\qquad Z \notin S_{XY}$$

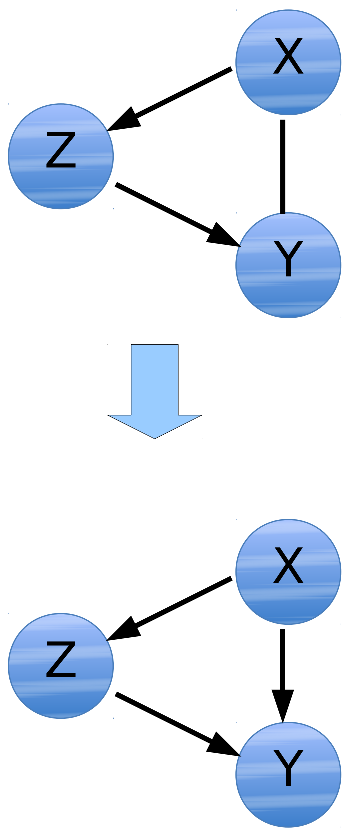
Orientation rule: create v-structure if $Z \notin S_{XY}$

Direct further edges (Rule 1)



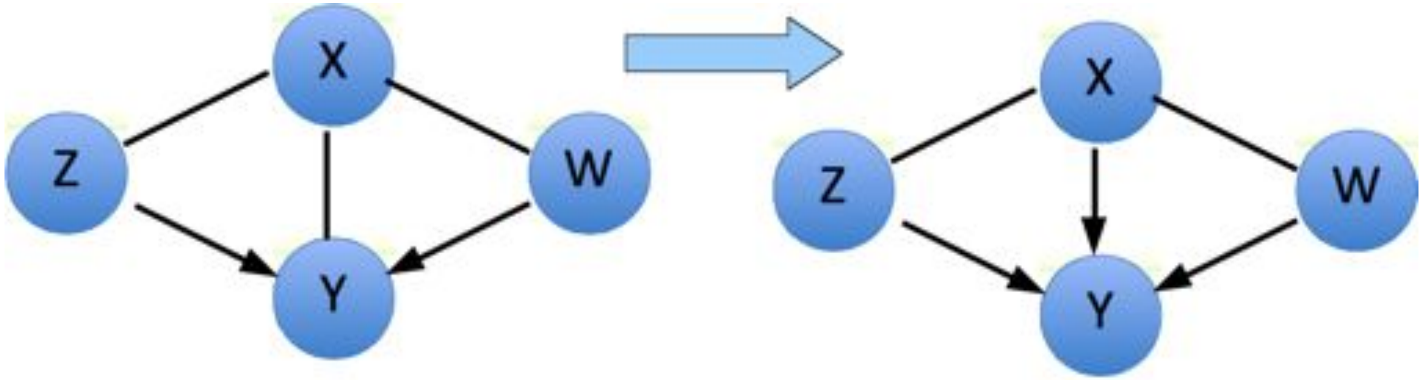
(otherwise we get a new v-structure)

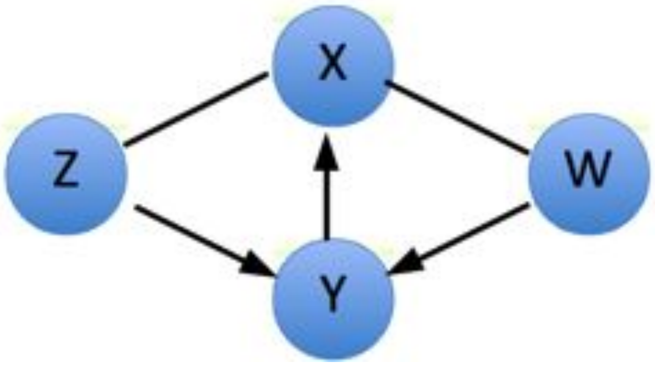
Direct further edges (Rule 2)



(otherwise one gets a cycle)

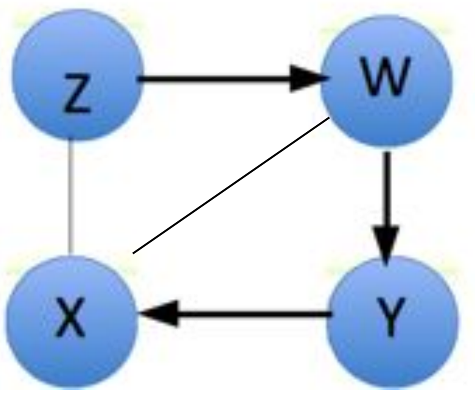
Direct further edges (Rule 3)





could not be completed
without creating a cycle
or a new v-structure

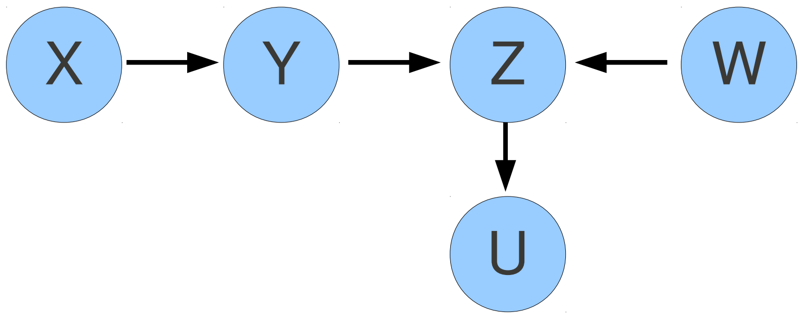
Direct further edges (Rule 4)



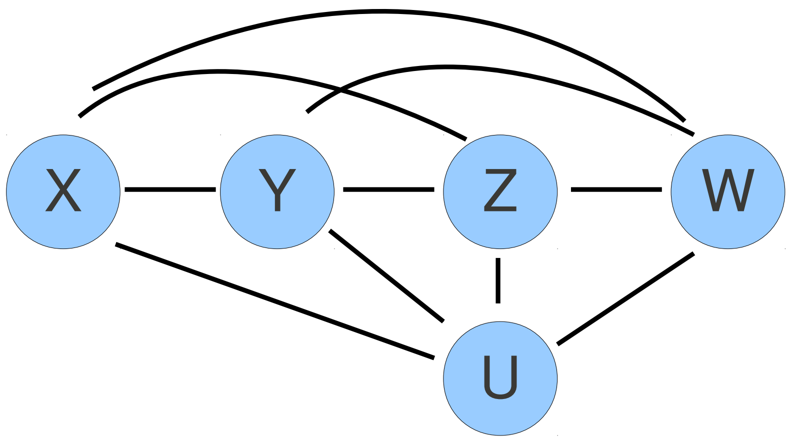
could not be completed
without creating a cycle
or a new v-structure

Examples

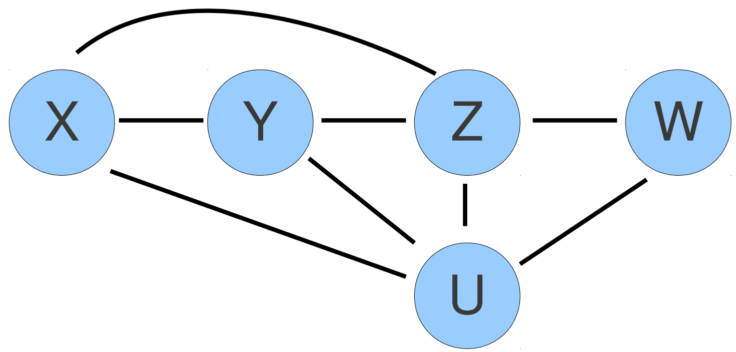
(taken from Spirtes et al, 2010)
true DAG



start with fully connected undirected graph

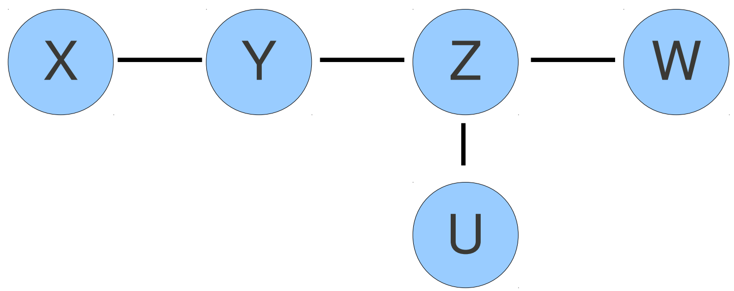


remove all edges $X - Y$ with $X \perp\!\!\!\perp Y \,|\emptyset$



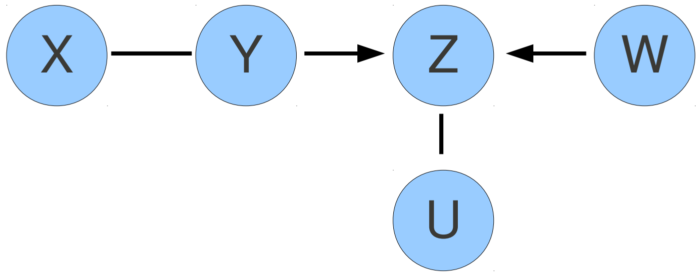
$X \perp\!\!\!\perp W \quad Y \perp\!\!\!\perp W$

remove all edges having Sepset of size 1



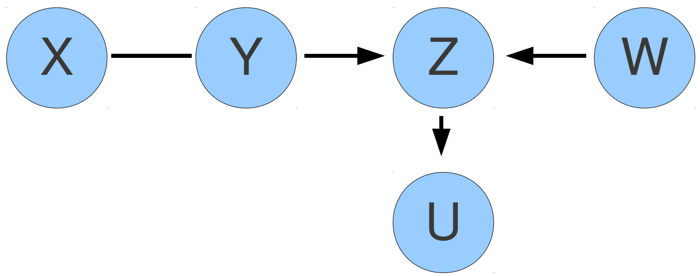
$X \perp\!\!\!\perp Z \,|Y \quad X \perp\!\!\!\perp U \,|Y \quad Y \perp\!\!\!\perp U \,|Z \quad W \perp\!\!\!\perp U \,|Z$

find v-structure



$$Z \notin S_{YW}$$

orient further edges (no further v-structure)



edge $X - Y$ remains undirected

Conditional independence tests

- **discrete case:** contingency tables
- **multi-variate gaussian case:**
  covariance matrix

non-Gaussian continuous case: challenging, recent progress via reproducing kernel Hilbert spaces (Fukumizu...Zhang...)

Improvements

- CPC (conservative PC) by Ramsey, Zhang, Spirtes (1995) uses weaker form of faithfulness
- FCI (fast causal inference) by Spirtes, Glymour, Scheines (1993) and Spirtes, Meek, Richardson (1999) infers causal links in the presence of latent common causes
- for implementations of the algorithms see homepage of the TETRAD project at Carnegie Mellon University Pittsburgh

Bayesian approach e.g. Cooper, Heckerman, Meek (1997), Stegle, Janzing, Zhang, Schölkopf (2010)

idea:

- define prior over possible DAGs
- the conditionals $p(X_j|PA_j)$ are free parameters in the factorization

$$p(X_1,\ldots,X_n) = \prod_{j=1}^{n} p(X_j|PA_j)$$

- define priors on the parameter space of each DAG
- compute posterior probabilities of DAGs

implicit preference of faithful DAGs

Note: whether Markov equivalent DAGs obtain the same posterior probability depends on the prior

Large scale evaluation of PC-related approach

Maathuis, Colombo, Kalisch & Bühlmann (2007)

Given

- **Observational data:** expression profiles of 5,361 genes of yeast (wild type)
- **Interventional data:** expression profiles of 5,361 genes for interventions on 234 genes

Evaluation:

- use observational data to select the genes that are most influenced by the interventions

  (new method: compute lower bound on the effect over all equivalent DAGs)
- compare with those selected from interventional data

success rates clearly significant: e.g. 33 true positive instead of 5

Saccharomyces
57 full-genome
yeast deletion
data), together
ression profiles
ments (observa-
under the same
data cleaning
), the interven-
ession measure-
234 single-gene
and the obser-
xpression mea-
61 genes for 63

onal data as the
ating the total
eleted genes on
is, 234 × 5,360
Methods). We
ntage of these
as our target set
DA could iden-
observational
argest predicted
= 50, 250, 1,000

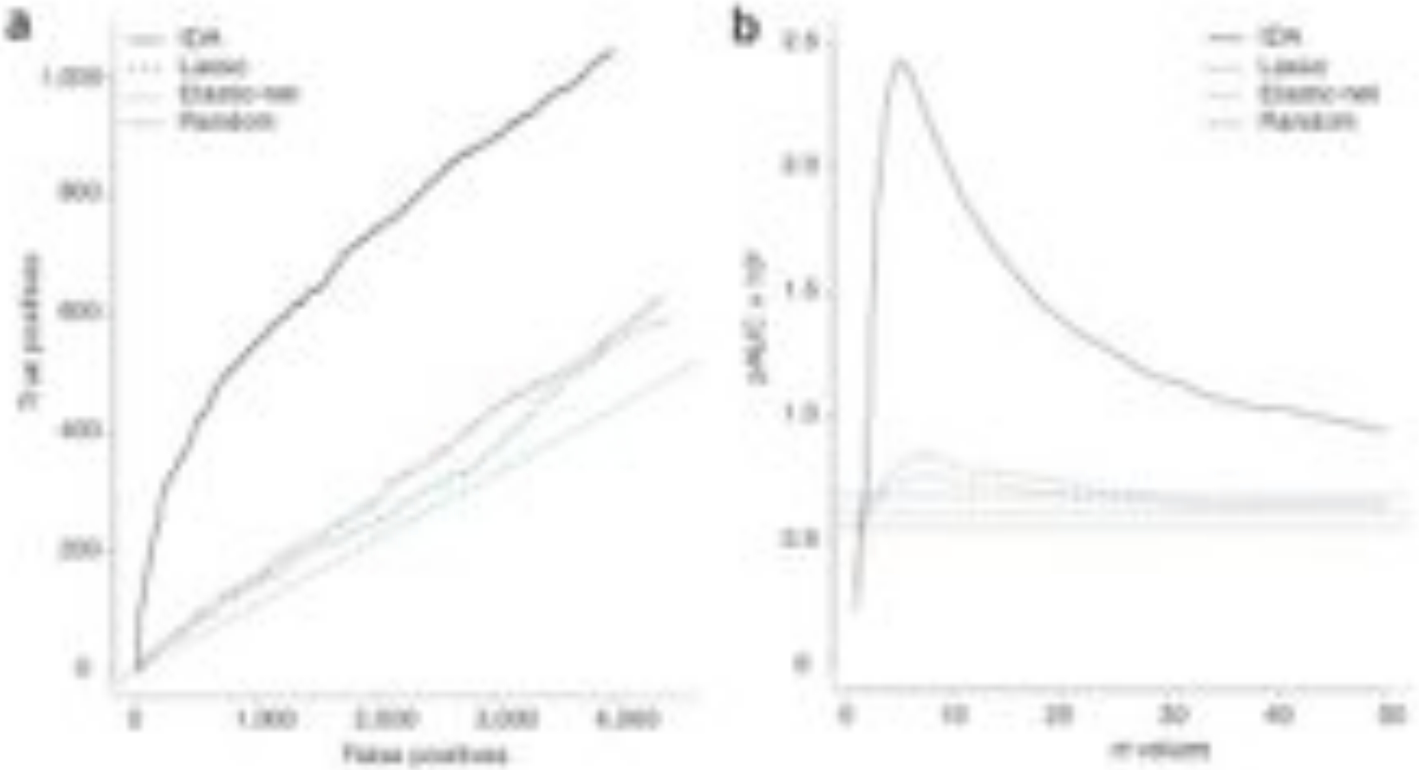


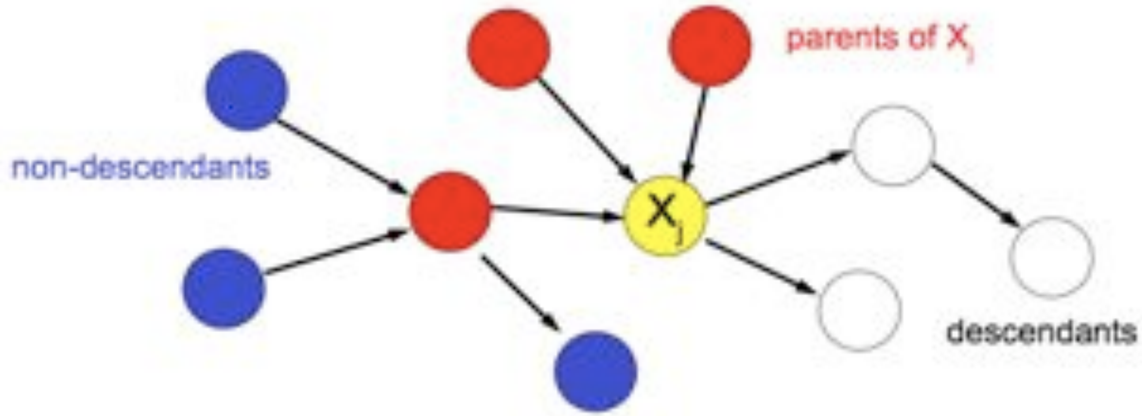
Figure 1 | Predicting causal effects from observational data (data are from ref. 1). (**a**) The number of true positives versus the number of false positives are plotted for the indicated methods, for the top 5,000 predicted effects from the observational data. The target set is the top 10% of the effects as computed from the interventional data. (**b**) The partial area under the receiver operating characteristic curve (pAUC) is plotted versus *m* values, when the target set is the top *m* percentage of the effects as computed from the interventional data. The pAUC was computed up to the false-positive rate determined by the top 5,000 effects from IDA for *m* = 10. The three horizontal lines for random guessing correspond to the 2.5th, 50th and 97.5th percentiles of a simulated distribution based on random orderings of effects.

Equivalence of Markov conditions

Theorem: the following are equivalent:

- Existence of a functional causal model
- Local Causal Markov condition: X_j *statistically independent* of non-descendants, given parents
- Global Causal Markov condition: d-separation
- Factorization $p(X_1, \ldots, X_n) = \prod_j p(X_j \mid PA_j)$

(subject to technical conditions)



Local Markov $\Rightarrow$ factorization *(Lauritzen 1996)*

- Assume X_n is a terminal node, i.e., it has no descendants, then $ND_n = \{X_1, \dots, X_{n-1}\}$. Thus the local Markov condition implies

$$X_n \perp\!\!\!\perp \{X_1, \dots, X_{n-1}\} \,|PA_n \,.$$

- Hence the general decomposition

$$p(x_1, \dots, x_n) = p(x_n | x_1, \dots, x_{n-1}) p(x_1, \dots, x_{n-1})$$

becomes

$$p(x_1, \dots, x_n) = p(x_n | pa_n) p(x_1, \dots, x_{n-1}) \,.$$

- Induction over n yields

$$p(x_1, \dots, x_n) = \prod_{j=1}^{n} p(x_j | pa_j) \,.$$

Factorization $\Rightarrow$ global Markov

(Lauritzen 1996)

Need to prove $(X \perp\!\!\!\perp Y \,|Z)_G \Rightarrow (X \perp\!\!\!\perp Y \,|Z)_p$.

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

- define the smallest subgraph G' containing X, Y, Z and all their ancestors
- consider moral graph G'^m (undirected graph containing the edges of G' and links between all parents)
- use results that relate factorization of probabilities with separation in undirected graphs

Global Markov ⇒ local Markov

Know that if Z d-separates X, Y, then $X \perp\!\!\!\perp Y \,|Z$.
Need to show that $X_j \perp\!\!\!\perp ND_j \,|PA_j$.

Simply need to show that the parents PA_j d-separate X_j from its non-descendants ND_j:

All paths connecting X_j and ND_j include a $P \in PA_j$, but never as a collider

$$\cdot \to P \leftarrow X_j$$

Hence all paths are chains

$$\cdot \to P \to X_j$$

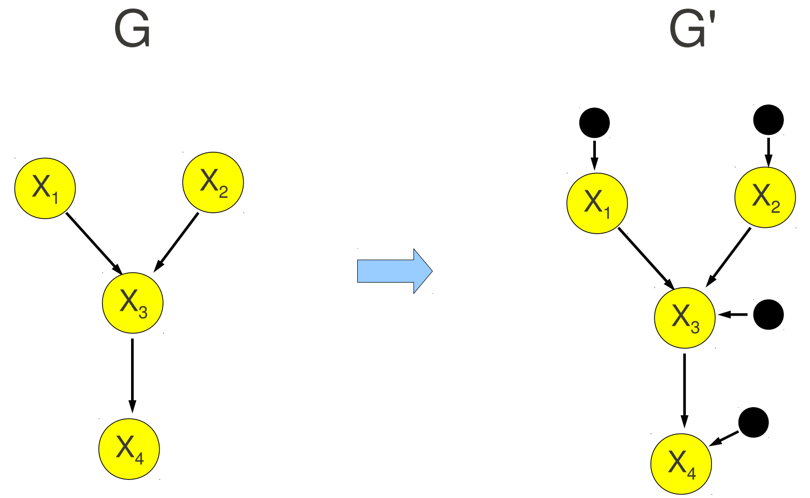
or forks

$$\cdot \leftarrow P \to X_j$$

Therefore, the parents block every path between X_j and ND_j.

functional model $\Rightarrow$ local Markov condition

(Pearl 2000)



- augmented DAG G' contains unobserved noise
- local Markov-condition holds for G':

  (i): the unexplained noise terms U_j are jointly independent, and thus (unconditionally) independent of their non-descendants

  (ii): for the X_j, we have

  $$X_j \perp\!\!\!\perp ND'_j \,|PA'_j$$

  because X_j is a (deterministic) function of PA'_j.

- local Markov in G' implies global Markov in G'
- global Markov in G' implies local Markov in G (proof as last slide)

factorization $\Rightarrow$ functional model

generate each $p(X_j|PA_j)$ in

$$p(X_1, \ldots, X_n) = \prod_{j=1}^{n} p(X_j|PA_j)$$

by a deterministic function:

- define a vector valued noise variable U_j
- each component $U_j[pa_j]$ corresponds to a possible value pa_j of PA_j
- define structural equation

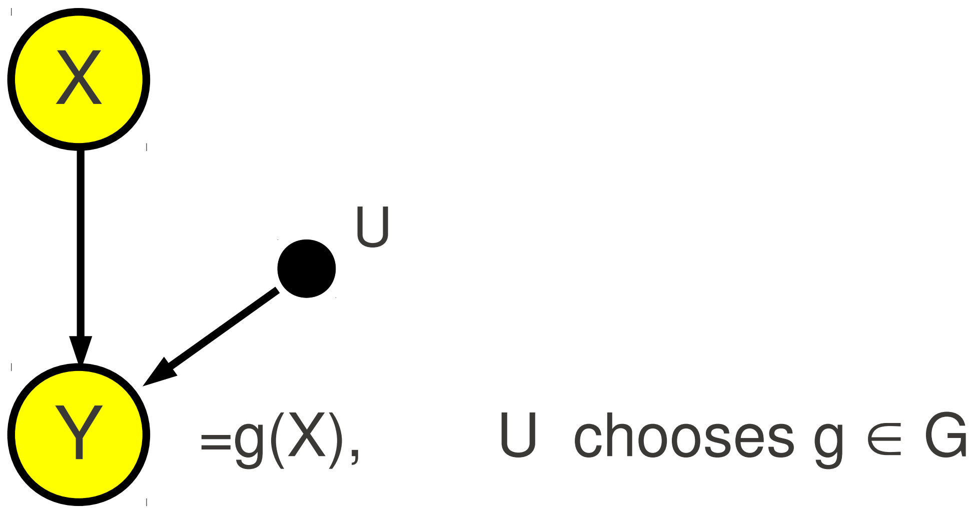
$$x_j = f_j(pa_j, u_j) := u_j[pa_j]\,.$$

- let component $U_j[pa_j]$ be distributed according to $p(X_j|pa_j)$.

Note: joint distribution of all $U_j[pa_j]$ is irrelevant, only marginals matter

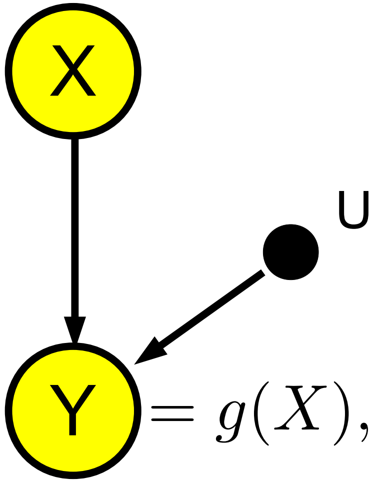
MAX-PLANCK-GESELLSCHAFT

different point of view



- G denotes set of deterministic mechanisms
- U randomly chooses a mechanism

Example: X, Y binary



$$= g(X), \quad U \text{ chooses } g \in \{ID, NOT, 1, 0\}$$

the same $p(X, Y)$ can be induced by different distributions on G:

- model 1 (no causal link from X to Y)

$$P(g = 0) = 1/2, \quad P(g = 1) = 1/2$$

- model 2 (random switching between ID and NOT)

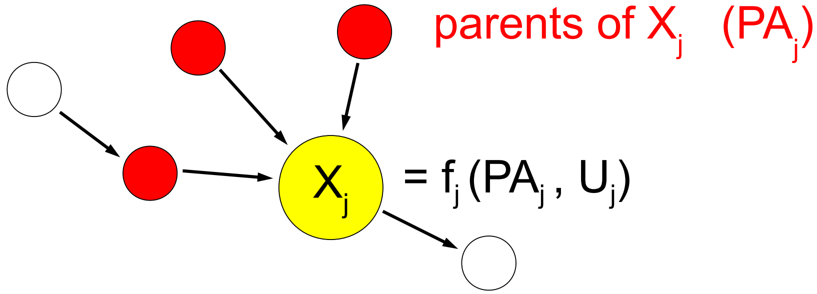
$$P(g = ID) = 1/2, \quad P(g = NOT) = 1/2$$

both induce the uniform distribution for Y, *independent* of X

INTERVAL

Recap: Functional Causal Model

- $X_i = f_i(\mathrm{ParentsOf}_i, \mathrm{Noise}_i)$, with jointly independent $\mathrm{Noise}_1, \ldots, \mathrm{Noise}_n$.



- entails $p(X_1, \ldots, X_n)$ with particular conditional independence structure

  Under certain assumptions, given p, can recover an equivalence class containing the correct graph using conditional independence testing.

  Problems:

  1. does not work for graphs with only 2 vertices (even with infinite data)
  2. if we don't have infinite data, conditional independence testing can be arbitrarily hard

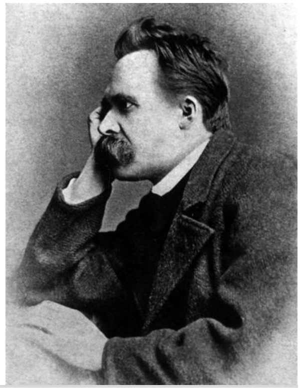
  Hypothesis:

  Both issues can be resolved by making assumptions on function classes.

Friedrich Nietzsche's

TWILIGHT OF THE IDOLS

or How to Philosophize with a Hammer



Götzen-Dämmerung

oder

Wie man mit dem Hammer philosophirt.

Von

Friedrich Nietzsche.

LEIPZIG.

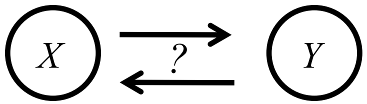
Translated, with commentary, by R.J. Hollingdale

[. . .]

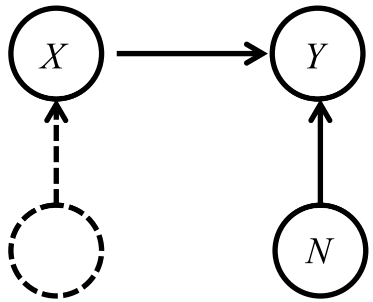
The Four Great Errors

1

The error of mistaking cause for consequence. - There is no more dangerous error than that of *mistaking the consequence for the cause*: I call it reason's intrinsic form of corruption. Nonetheless, this error is among the most ancient and most recent



Restricting the Functional Model



$X \perp\!\!\!\perp N$

- consider the graph $X \to Y$
- general functional model

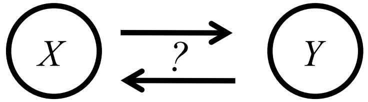
$$Y = f(X, N)$$

Note: if N can take d different values, it could switch randomly between mechanisms $f^1(X), \ldots, f^d(X)$

- additive noise model

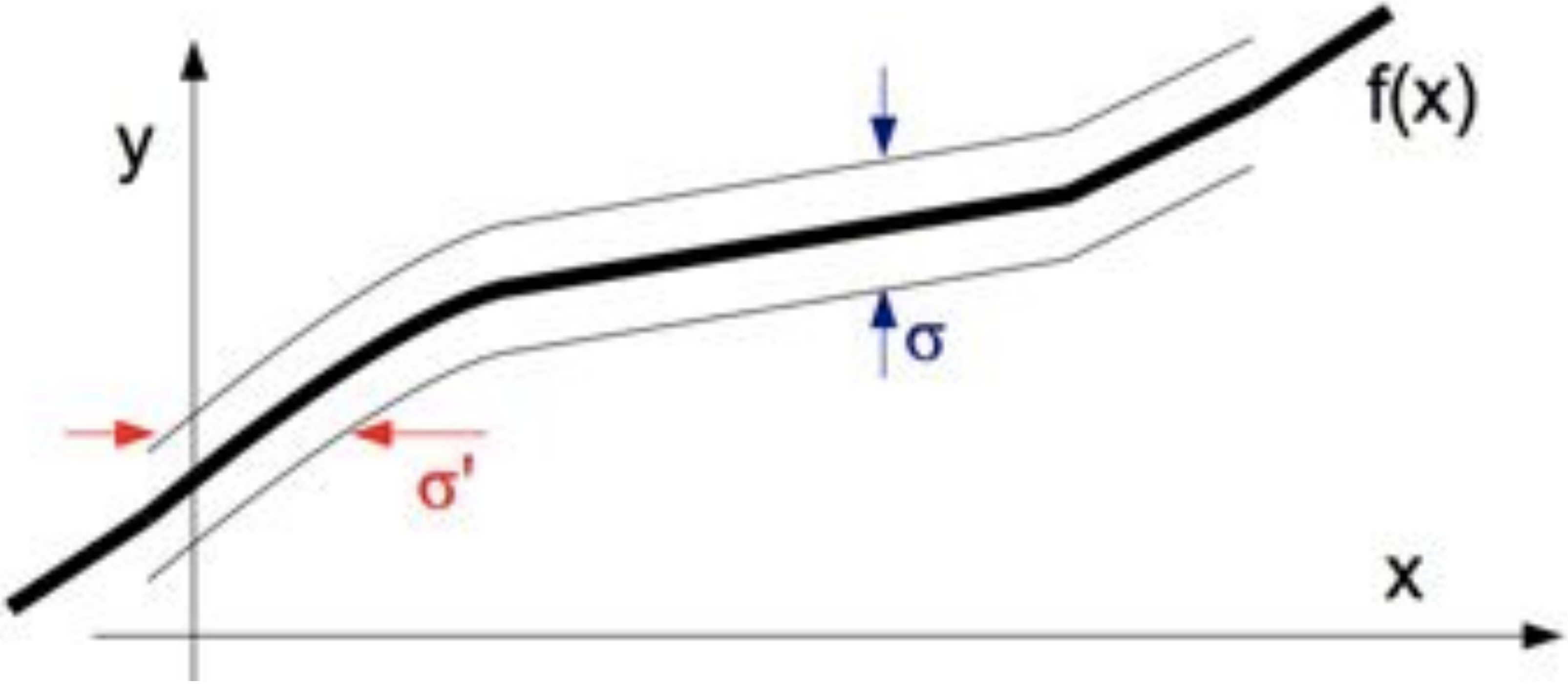
$$Y = f(X) + N$$

Causal Inference with Additive Noise, 2-Variable Case



Forward model:
$y := f(x) + n$, with $x \perp\!\!\!\perp n$

Identifiability: when is there a backward model of the same form?



Hoyer et al.: Nonlinear causal discovery with additive noise models. *NIPS* 21, 2009
Peters et al.: Detecting the Direction of Causal Time Series. *ICML* 2009

Identifiability Result *(Hoyer, Janzing, Mooij, Peters, Schölkopf, 2008)*

Theorem 1 *Let the joint probability density of x and y be given by*

$$p(x,y) = p_n(y - f(x))p_x(x)\,, \tag{1}$$

where p_n, p_x are positive probability densities on $\mathbb{R}$. If there is a backward model

$$p(x,y) = p_{\tilde{n}}(x - g(y))p_y(y)\,, \tag{2}$$

then, denoting $\nu := \log p_n$ and $\xi := \log p_x$ and assuming sufficient differentiability, the triple (f, p_x, p_n) must satisfy the following differential equation for all x, y with $\nu''(y - f(x))f'(x) \neq 0$:

$$\xi''' = \xi''\left(-\frac{\nu''' f'}{\nu''} + \frac{f''}{f'}\right) - 2\nu'' f'' f' + \nu' f''' + \frac{\nu'\nu''' f'' f'}{\nu''} - \frac{\nu'(f'')^2}{f'}\,, \tag{3}$$

where we have skipped the arguments $y - f(x)$, x, and x for ν, ξ, and f and their derivatives, respectively. Moreover, if for a fixed pair (f, ν) there exists $y \in \mathbb{R}$ such that $\nu''(y - f(x))f'(x) \neq 0$ for all but a countable set of points $x \in \mathbb{R}$, the set of all p_x for which p has a backward model is contained in a 3-dimensional affine space.

Corollary 1 *Assume that $\nu''' = \xi''' = 0$ everywhere. If a backward model exists, then f is linear.*

Idea of the proof

If $p(x, y)$ admits an additive noise model

$$Y = f(X) + E$$

we have

$$p(x, y) = q(x)r(y - f(x)).$$

It then satisfies the differential equation

$$\frac{\partial}{\partial x}\left(\frac{\partial^2 \log p(x, y)/\partial x^2}{\partial^2 \log p(x, y)/\partial x \partial y}\right) = 0.$$

If it also holds with exchanging x and y, only specific cases remain.

Alternative View *(cf. Zhang & Hyvärinen, 2009)*

H differential entropy

I mutual information

$n_y = y - f(x), n_x = x - g(y)$ residual noises

Lemma: For arbitrary joint distribution of x, y and functions f, $g : \mathbf{R} \to \mathbf{R}$, we have:

$$H(x, y) = H(x) + H(n_y) - I(n_y, x) = H(y) + H(n_x) - I(n_x, y).$$

If x causes y, we can find f such that $n_y \perp\!\!\!\perp x$, while "almost all" g lead to $n_x \not\perp\!\!\!\perp y$, i.e.:

$$I(n_y, x) = 0 \text{ and } I(n_x, y) > 0$$

Thus

$$H(x, y) = H(x) + H(n_y) \leq H(y) + H(n_x).$$

Causal Inference Method

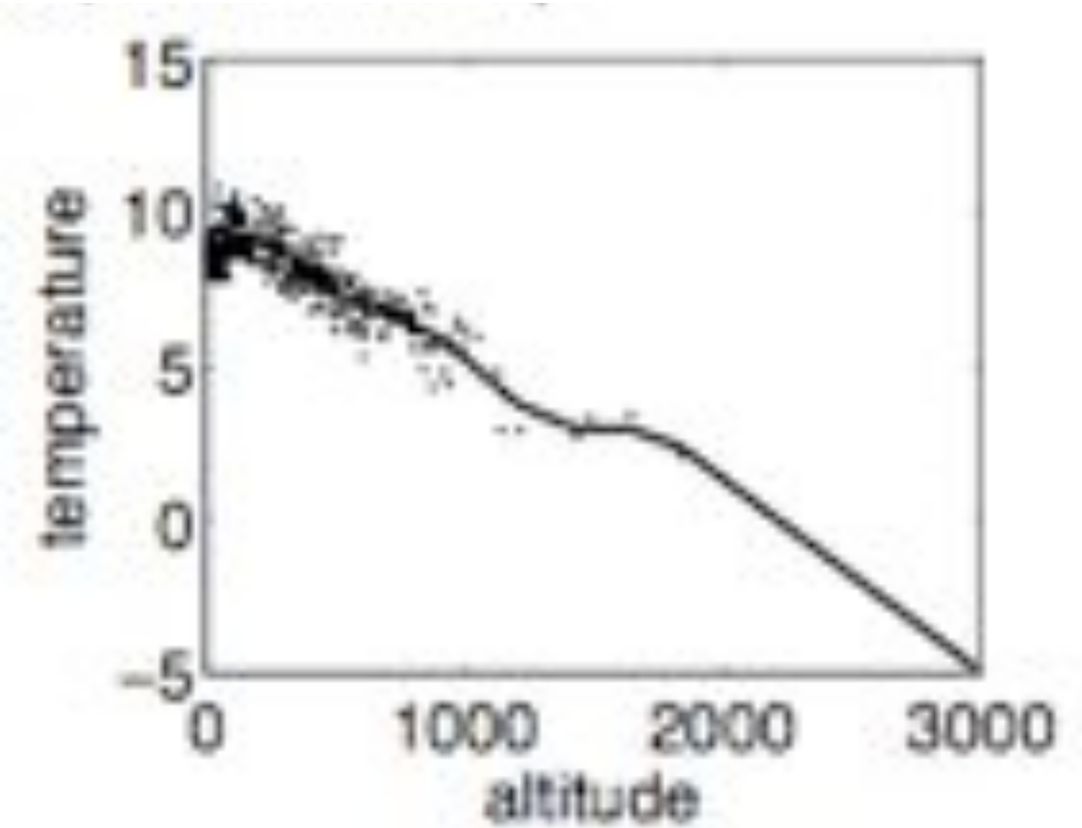
Prefer the causal direction that can better be fit with an additive noise model.

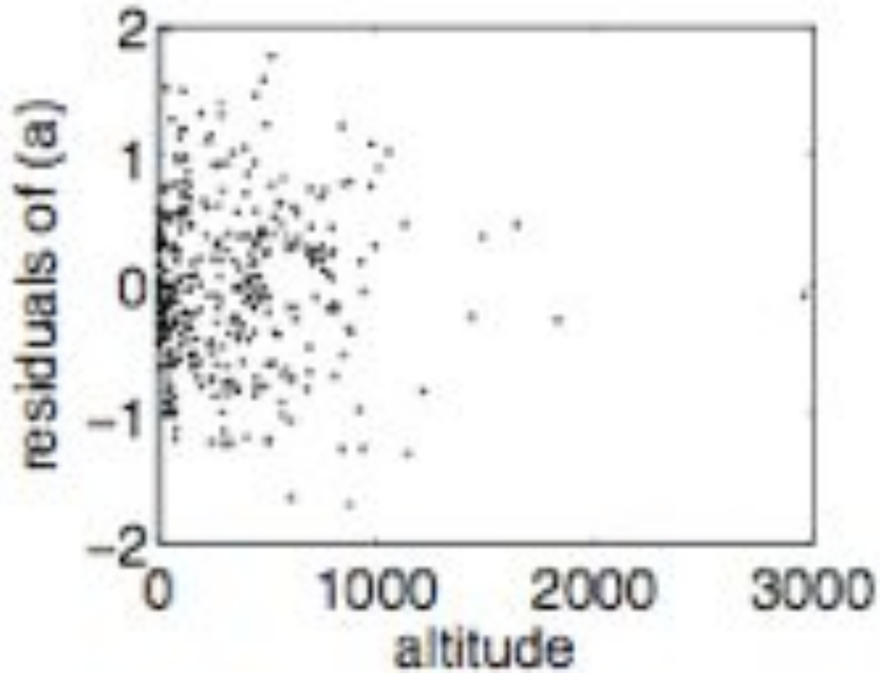
Implementation:

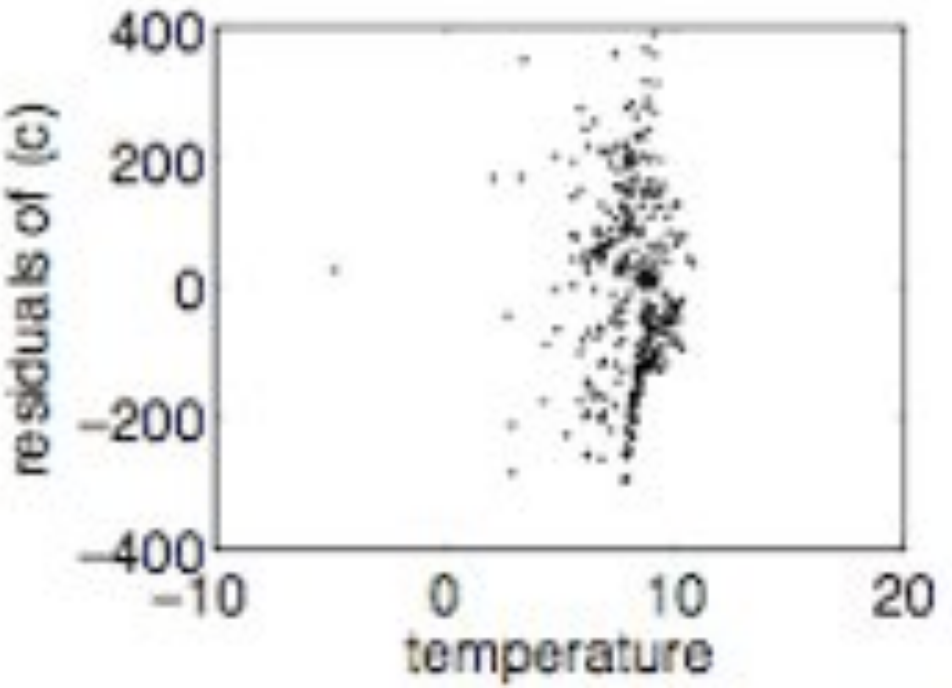
- Compute a function f as non-linear regression of X on Y
- Compute the residual
$$E := Y - f(X)$$
- check whether E and X are statistically independent (uncorrelated is not enough)

Experiments

Relation between altitude (cause) and average temperature (effect) of places in Germany







Our independence tests detect strong dependence.
Hence the method prefers the correct direction

altitude → temperature

- Generalization to post-nonlinear additive noise models: Zhang & Hyvärinen: *On the Identifiability of the Post-Nonlinear Causal Model*, UAI 2009

- Generalization to graphs with more than two vertices: Peters, Mooij, Janzing, Schölkopf: *Identifiability of Causal Graphs using Functional Models*, UAI 2011

- Generalization to two-vertex-graphs with loops: Mooij, Janzing, Heskes, Schölkopf: *Causal discovery with Cyclic additive noise models*, NIPS 2011

Independence-based Regression *(Mooij et al., 2009)*

- Problem: many regression methods assume a particular noise distribution; if this is incorrect, the residuals may become dependent
- Solution: minimize dependence of residuals rather than maximizing likelihood of data in regression objective
- Use RKHS distance between kernel mean embeddings/Hilbert-Schmidt-norm of cross-covariance operator between two RKHSes as a dependence measure

Mooij, Janzing, Peters, Schölkopf: Regression by dependence minimization and its application to causal inference. *ICML* 2009.
Yamada & Sugiyama: Dependence Minimizing Regression with Model Selection for Non-Linear Causal Inference under Non-Gaussian Noise. *AAAI* 2010.

Kernel Independence Testing *(Gretton et al., 2007)*

k bounded p.d. kernel; P Borel probability measure

Define the *kernel mean map*

$$\mu : P \mapsto \mathbf{E}_{x \sim P}[k(x, .)].$$

Theorem: If k is universal, μ is injective.

Discussion: a measure can be represented as an element of the RKHS associated with k without loss of information.

Let's represent $p(X, Y)$ and $p(X)p(Y)$ — they will only map to the same element if they are equal, i.e., if $X \perp\!\!\!\perp Y$.

Proposition 1 *Assume that k is strictly pd, and for all i, j, $x_i \neq x_j$, and $y_i \neq y_j$. If for some $\alpha_i, \beta_j \in \mathbb{R} - \{0\}$, we have*

$$\sum_{i=1}^{m} \alpha_i k(x_i, .) = \sum_{j=1}^{n} \beta_j k(y_j, .), \tag{1}$$

then $X = Y$.

Proof (by contradiction): W.l.o.g., assume that $x_1 \notin Y$. Subtract $\sum_{j=1}^{n} \beta_j k(y_j, .)$ from (1), and make it a sum over distinct points, to get

$$0 = \sum_i \gamma_i k(z_i, .),$$

where $z_1 = x_1, \gamma_1 = \alpha_1 \neq 0$, and $z_2, \dots \in X \cup Y - \{x_1\}$, $\gamma_2, \dots \in \mathbb{R}$.
Take the dot product with $\sum_j \gamma_j k(z_j, .)$, using $\langle k(z_i, .), k(z_j, .)\rangle = k(z_i, z_j)$, to get

$$0 = \sum_{ij} \gamma_i \gamma_j k(z_i, z_j),$$

with $\gamma \neq 0$, hence k cannot be strictly pd.

Kernel Independence Testing: HSIC

Corollary: $x \perp\!\!\!\perp y \Longleftrightarrow \Delta := \|\mu(p_{xy}) - \mu(p_x \times p_y)\| = 0.$

- For $k((x,y),(x',y')) = k_x(x,x')k_y(y,y')$:

  Δ^2 = HS-norm of cross-covariance operator between the two RKHSes (HSIC, Gretton et al., 2005)

- empirical estimator $\frac{1}{n^2}\mathrm{tr}[K_x K_y]$ (ignoring centering)

- Why does this characterize independence: $x \perp\!\!\!\perp y$ iff

$$\sup_{f,g \,\in\, \text{RHKS unit balls}} \mathrm{cov}(f(x), g(y)) = 0$$

(cf. Kernel ICA, *Bach & Jordan, 2002)*

Hilbert-Schmidt Normalized Independence Criterion
(Fukumizu et al., 2007)

- normalize out variance of X and Y to get HSNIC; can be shown to equal the *mean squared contingency*

$$\int \left(\frac{p(x,y)}{p(x)p(y)} - 1 \right) dp(x,y)$$

  independent of the (characteristic/universal) kernel

- can be shown to be upper bounded by the mutual information,

$$\text{HSNIC}(X,Y) \leq \text{MI}(X,Y) = \int \log \left(\frac{p(x,y)}{p(x)p(y)} \right) dp(x,y)$$

Approximating the null distribution

- to construct a test, need to compute the *null distribution* of our test statistic (HSIC): how is the empirical HSIC distributed if $X \perp\!\!\!\perp Y$?

- can use a (complicated) asymptotic expression for HSIC (Gretton et al., 2008), but there's an easy practical method to generate samples consistent with the null hypothesis (independence), and the original marginals $p(X), p(Y)$:

- given a permutation σ, turn $(x_1, y_1), \ldots, (x_n, y_n)$ into $(x_1, y_{\sigma(1)}), \ldots, (x_n, y_{\sigma(n)})$

- the case of conditional independence is harder: given $(x_1, y_1, z_1), \ldots, (x_n, y_n, z_n)$, need to generate samples consistent with $X \perp\!\!\!\perp Y \,|Z$, and original $p(X|Z), p(Y|Z)$.

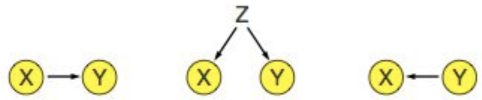
- if z only takes few values, can permute within groups having the same value of z (Fukumizu et al., 2007)

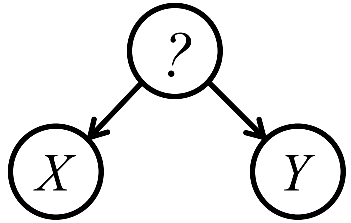
- general case is an open problem, but see e.g. Zhang et al., UAI 2011

Detection of Confounders

Given $p(X, Y)$, infer whether

- $X \to Y$
- $Y \to X$
- $X \leftarrow T \to Y$ for some (possibly) unobserved variable T





- Confounded additive noise (CAN) models

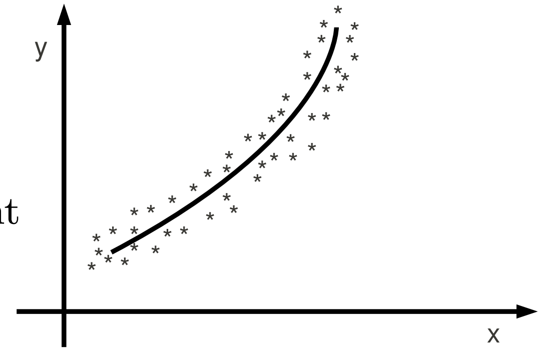
$$X = f_X(T) + U_X$$
$$Y = f_Y(T) + U_Y$$

with functions f_X, f_Y and U_X, U_Y, T jointly independent

Note: includes the case

$$Y = f(X) + U$$

by setting $f_X = id$ and $U_X = 0$.



- Estimate $(f_X(T), f_Y(T))$ using dimensionality reduction
- If U_X or U_Y is close to zero, output 'no confounder'
- Identifiability result for small noise

Janzing, Peters, Mooij, Schölkopf: Identifying latent confounders using additive noise models. *UAI 2009*

Identifying discrete confounders by independence-based clustering

Identifying Finite Mixtures of Nonparametric Product Distributions and Causal Inference of Confounders

Eleni Sgouritsa[1], Dominik Janzing[1], Jonas Peters[1,2], Bernhard Schölkopf[1]

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$$P(X_1,\ldots,X_d)=\sum_{i=1}^{m}P(z^{(i)})\prod_{j=1}^{d}P(X_j|z^{(i)})$$

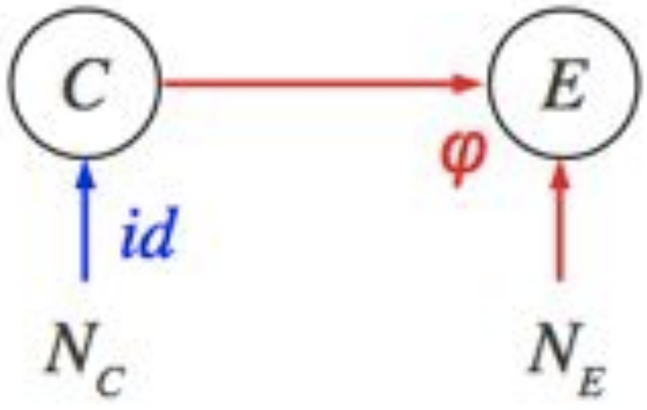
However, employing properties of the noise is not the only way of inferring cause and effect.

What about the noiseless case?

Independence of input and mechanism

Causal structure:

C cause
E effect
N noise
φ mechanism

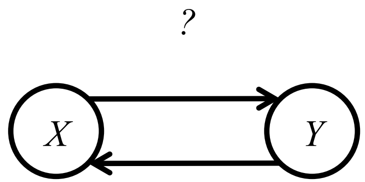


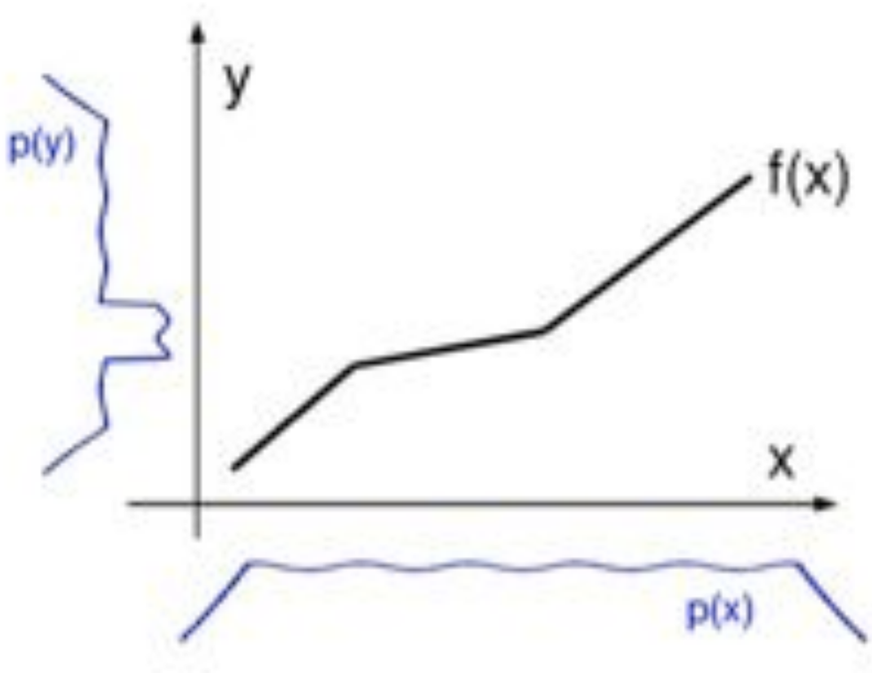
Assumption:
$p(C)$ and $p(E|C)$ are "independent"

Janzing & Schölkopf, IEEE Trans. Inf. Theory, 2010; cf. also Lemeire & Dirkx, 2007

Inferring deterministic causal relations

- Does not require noise
- Assumption: $y = f(x)$ with invertible f





Daniusis, Janzing, Mooij, Zscheischler, Steudel, Zhang, Schölkopf: Inferring deterministic causal relations, *UAI* 2010

Causal independence implies anticausal dependence

Assume that f is a monotonously increasing bijection of $[0,1]$.
View p_x and $\log f'$ as RVs on the prob. space $[0,1]$ w. Lebesgue measure.

Postulate (independence of mechanism and input):

$$\mathrm{Cov}\,(\log f', p_x) = 0$$

Note: this is equivalent to

$$\int_0^1 \log f'(x)p(x)dx = \int_0^1 \log f'(x)dx,$$

since

$$\mathrm{Cov}\,(\log f', p_x) = E\,[\log f' \cdot p_x] - E\,[\log f']\,E\,[p_x] = E\,[\log f' \cdot p_x] - E\,[\log f'].$$

Proposition:

$$\mathrm{Cov}\,(\log f^{-1\prime}, p_y) \geq 0$$

with equality iff $f = Id$.

u_x, u_y uniform densities for x, y

v_x, v_y densities for x, y induced by transforming u_y, u_x via f^{-1} and f

Equivalent formulations of the postulate:

Additivity of Entropy:

$$S(p_y) - S(p_x) = S(v_y) - S(u_x)$$

Orthogonality (information geometric):

$$D(p_x \| v_x) = D(p_x \| u_x) + D(u_x \| v_x)$$

which can be rewritten as

$$D(p_y \| u_y) = D(p_x \| u_x) + D(v_y \| u_y)$$

Interpretation:

irregularity of p_y = irregularity of p_x + irregularity introduced by f

Slope-Based Estimator

Slope-based IGCI: infer $X \to Y$ whenever

$$\int_0^1 \log|f'(x)|\,P(x)\,dx < \int_0^1 \log|g'(y)|\,P(y)\,dx.$$

We introduce the following estimator:

$$\hat{C}_{X\to Y} := \int \log|f'(x)|\,P(x)\,dx \approx \frac{1}{m-1}\sum_{i=1}^{m-1}\log\left|\frac{y_{i+1}-y_i}{x_{i+1}-x_i}\right|$$

where the x_i values are ordered.

- infer $X \to Y$ whenever

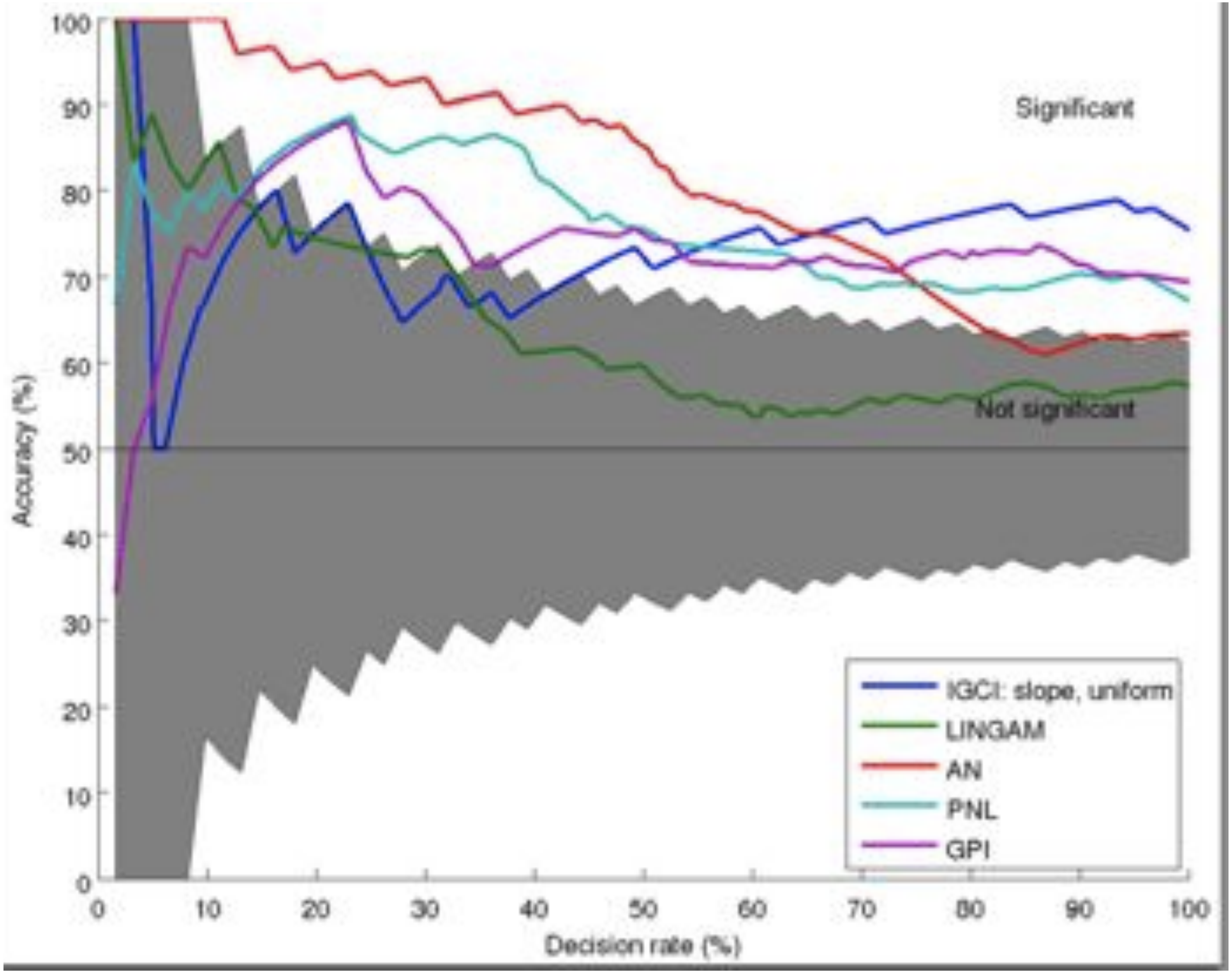
$$\hat{C}_{X\to Y} < \hat{C}_{Y\to X}\,.$$

80 Cause-Effect Pairs

80 Cause-Effect Pairs − Examples

	var 1	var 2	dataset	ground truth
pair0001	Altitude	Temperature	DWD	→
pair0005	Age (Rings)	Length	Abalone	→
pair0012	Age	Wage per hour	census income	→
pair0025	cement	compressive strength	concrete_data	→
pair0033	daily alcohol consumption	mcv mean corpuscular volume	liver disorders	→
pair0040	Age	diastolic blood pressure	pima indian	→
pair0042	day	temperature	B. Janzing	→
pair0047	#cars/24h	specific days	traffic	←
pair0064	drinking water access	infant mortality rate	UNdata	→
pair0068	bytes sent	open http connections	P. Daniusis	←
pair0069	inside room temperature	outside temperature	J. M. Mooij	←
pair0070	parameter	sex	Bülthoff	→
pair0072	sunspot area	global mean temperature	sunspot data	→
pair0074	GNI per capita	life expectancy at birth	UNdata	→
pair0078	PPFD (Photosynth. Photon Flux)	NEP (Net Ecosystem Productivity)	Moffat A. M.	→

http://webdav.tuebingen.mpg.de/cause-effect/



IGCI:
Deterministic
Method

LINGAM:
Shimizu et al.,
2006

AN:
Additive Noise
Model (nonlinear)

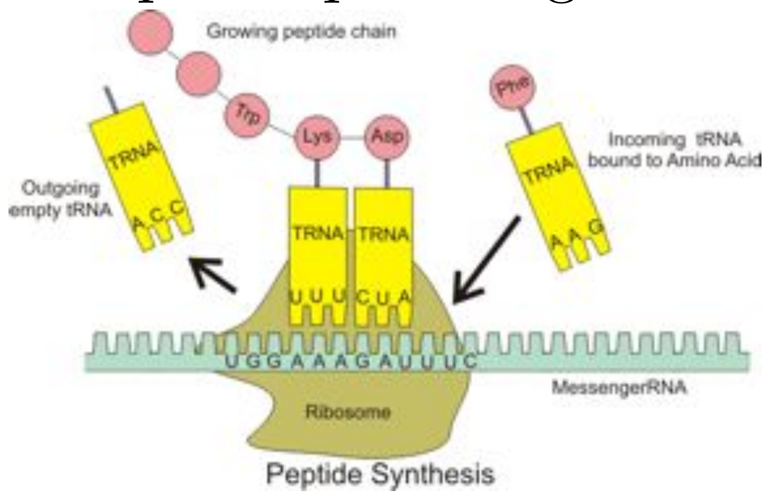
PNL:
AN with post-
nonlinearity

GPI:
Mooij et al.,
2010

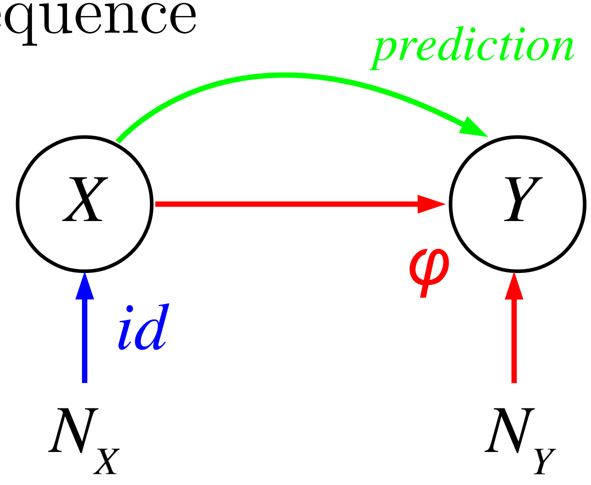
Causal Learning and Anticausal Learning

Schölkopf, Janzing, Peters, Sgouritsa, Zhang, Mooij, *ICML* 2012

- example 1: predict gene from mRNA sequence

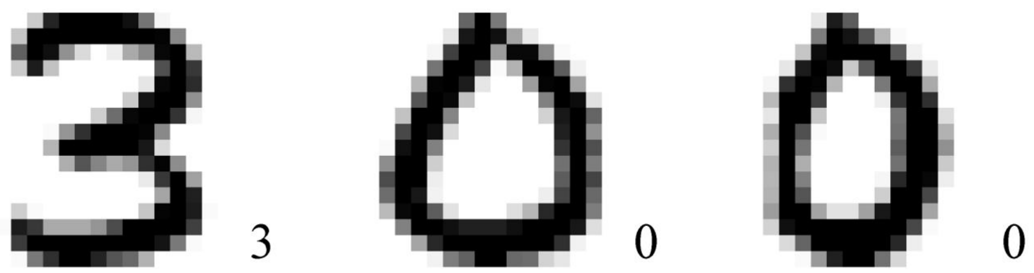


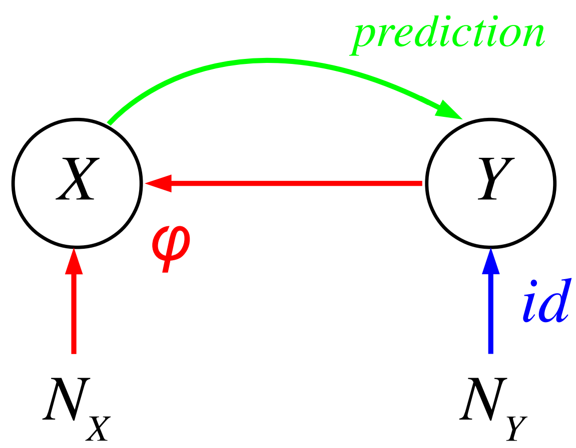
Source: http://commons.wikimedia.org/wiki/File:Peptide_syn.png



causal mechanism φ

- example 2: predict class membership from handwritten digit



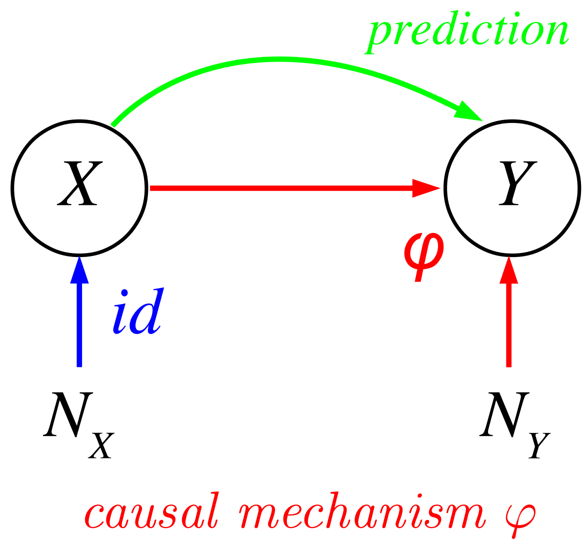


Covariate Shift and Semi-Supervised Learning

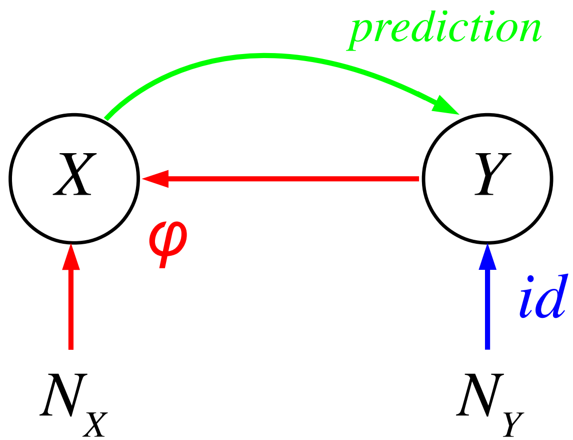
Assumption: $p(C)$ and mechanism $p(E|C)$ are "independent"

Goal: learn $X \mapsto Y$, i.e., estimate (properties of) $p(Y|X)$

- covariate shift (i.e., $p(X)$ changes): mechanism $p(Y|X)$ is unaffected by assumption
- semi-supervised learning: impossible, since $p(X)$ contains no information about $p(Y|X)$
- transfer learning (N_X, N_Y change, φ not): could be done by additive noise model with conditionally independent noise



- $p(X)$ **changes**: need to decide if change is due to mechanism $p(X|Y)$ or cause distribution $p(Y)$ (sometimes: by deconvolution)
- **semi-supervised learning**: possible, since $p(X)$ contains information about $p(Y|X)$ — e.g., cluster assumption.
- transfer learning: as above



(cf. Storkey, 2009)

Semi-Supervised Learning *(Schölkopf et al., ICML 2012)*

- Known SSL assumptions link $p(X)$ to $p(Y|X)$:
  - *Cluster assumption*: points in same cluster of $p(X)$ have the same Y
  - *Low density separation assumption*: $p(Y|X)$ should cross 0.5 in an area where $p(X)$ is small
  - *Semi-supervised smoothness assumption*: $E(Y|X)$ should be smooth where $p(X)$ is large

- Next slides: experimental analysis

SSL Book Benchmark Datasets – Chapelle et al. (2006)

Table 1. Categorization of eight benchmark datasets as Anticausal/Confounded, Causal or Unclear

Category	Dataset
Anticausal/ Confounded	g241c: the class causes the 241 features.
	g241d: the class (binary) and the features are confounded by a variable with 4 states.
	Digit1: the positive or negative angle and the features are confounded by the variable of continuous angle.
	USPS: the class and the features are confounded by the 10-state variable of all digits.
	COIL: the six-state class and the features are confounded by the 24-state variable of all objects.
Causal	SecStr: the amino acid is the cause of the secondary structure.
Unclear	BCI, Text: Unclear which is the cause and which the effect.

UCI Datasets used in SSL benchmark – Guo et al., 2010

Table 2. Categorization of 26 UCI datasets as Anticausal/Confounded, Causal or Unclear

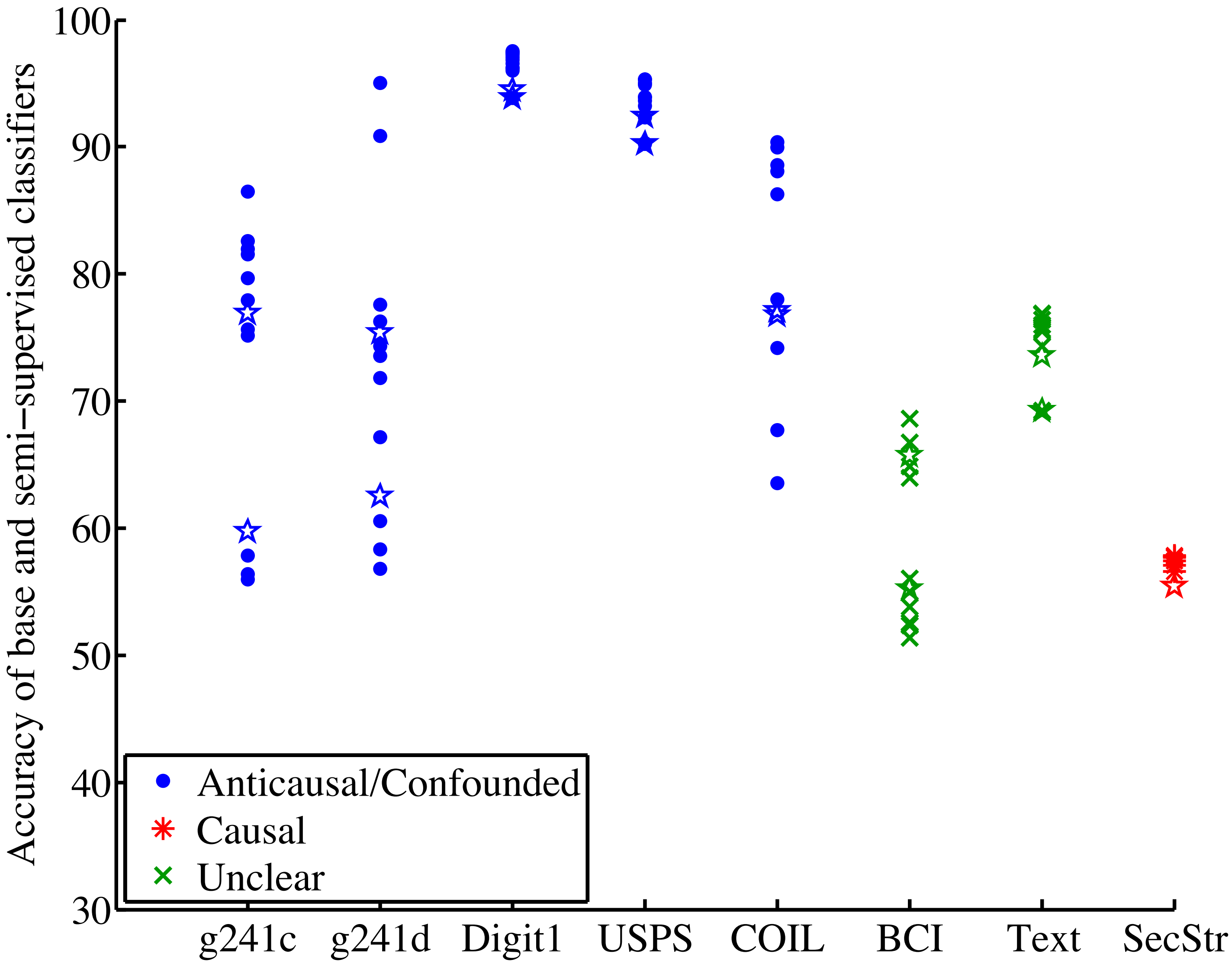
Categ.	Dataset
Anticausal/Confounded	Breast Cancer Wisconsin: the class of the tumor (benign or malignant) causes some of the features of the tumor (e.g., thickness, size, shape etc.).
	Diabetes: whether or not a person has diabetes affects some of the features (e.g., glucose concentration, blood pressure), but also is an effect of some others (e.g. age, number of times pregnant).
	Hepatitis: the class (die or survive) and many of the features (e.g., fatigue, anorexia, liver big) are confounded by the presence or absence of hepatitis. Some of the features, however, may also cause death.
	Iris: the size of the plant is an effect of the category it belongs to.
	Labor: cyclic causal relationships: good or bad labor relations can cause or be caused by many features (e.g., wage increase, number of working hours per week, number of paid vacation days, employer's help during employee 's long term disability). Moreover, the features and the class may be confounded by elements of the character of the employer and the employee (e.g., ability to cooperate).
	Letter: the class (letter) is a cause of the produced image of the letter.
	Mushroom: the attributes of the mushroom (shape, size) and the class (edible or poisonous) are confounded by the taxonomy of the mushroom (23 species).
	Image Segmentation: the class of the image is the cause of the features of the image.
	Sonar, Mines vs. Rocks: the class (Mine or Rock) causes the sonar signals.
	Vehicle: the class of the vehicle causes the features of its silhouette.
	Vote: this dataset may contain causal, anticausal, confounded and cyclic causal relations. E.g., having handicapped infants or being part of religious groups in school can cause one's vote, being democrat or republican can causally influence whether one supports Nicaraguan contras, immigration may have a cyclic causal relation with the class. Crime and the class may be confounded, e.g., by the environment in which one grew up.
	Vowel: the class (vowel) causes the features.
	Wave: the class of the wave causes its attributes.
Causal	Balance Scale: the features (weight and distance) cause the class.
	Chess (King-Rook vs. King-Pawn): the board-description causally influences whether white will win.
	Splice: the DNA sequence causes the splice sites.
Unclear	Breast-C, Colic, Sick, Ionosphere, Heart, Credit Approval were unclear to us. In some of the datasets, it is unclear whether the class label may have been generated or defined based on the features (e.g., Ionoshpere, Credit Approval, Sick).

Datasets, co-regularized LS regression – Brefeld et al., 2006

Table 3. Categorization of 31 datasets (described in the paragraph "Semi-supervised regression") as Anticausal/Confounded, Causal or Unclear

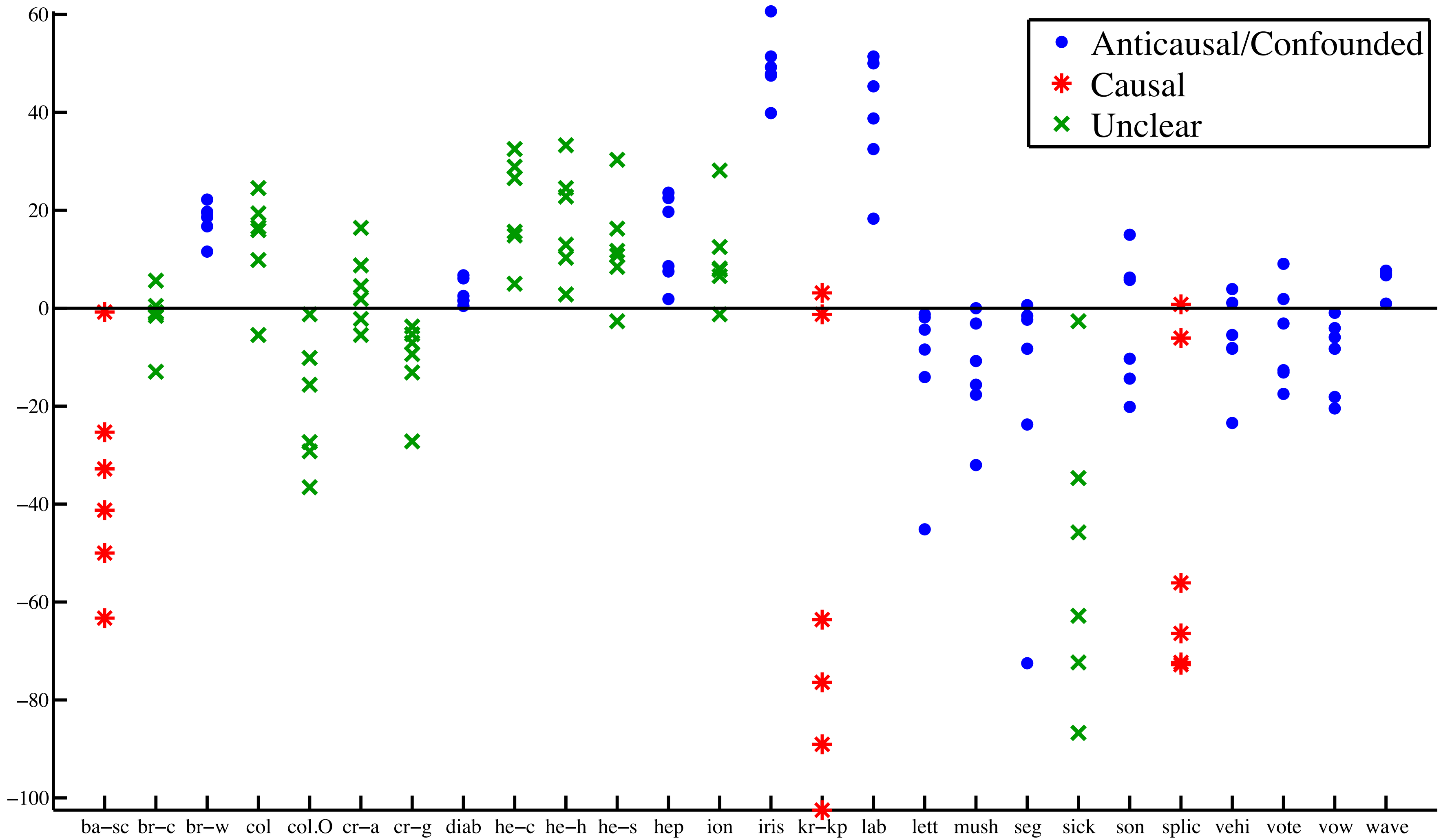
Categ.	Dataset	Target variable	Remark
Anticausal/Confounded	breastTumor	tumor size	causing predictors such as inv-nodes and deg-malig
	cholesterol	cholesterol	causing predictors such as resting blood pressure and fasting blood sugar
	cleveland	presence of heart disease in the patient	causing predictors such as chest pain type, resting blood pressure, and fasting blood sugar
	lowbwt	birth weight	causing the predictor indicating low birth weight
	pbc	histologic stage of disease	causing predictors such as Serum bilirubin, Prothrombin time, and Albumin
	pollution	age-adjusted mortality rate per 100,000	causing the predictor number of 1960 SMSA population aged 65 or older
	wisconsin	time to recur of breast cancer	causing predictors such as perimeter, smoothness, and concavity
Causal	autoMpg	city-cycle fuel consumption in miles per gallon	caused by predictors such as horsepower and weight
	cpu	cpu relative performance	caused by predictors such as machine cycle time, maximum main memory, and cache memory
	fishcatch	fish weight	caused by predictors such as fish length and fish width
	housing	housing values in suburbs of Boston	caused by predictors such as pupil-teacher ratio and nitric oxides concentration
	machine_cpu	cpu relative performance	see remark on "cpu"
	meta	normalized prediction error	caused by predictors such as number of examples, number of attributes, and entropy of classes
	pwLinear	value of piecewise linear function	caused by all 10 involved predictors
	sensory	wine quality	caused by predictors such as trellis
	servo	rise time of a servomechanism	caused by predictors such as gain settings and choices of mechanical linkages
Unclear	auto93 (target: midrange price of cars); bodyfat (target: percentage of body fat); autoHorse (target: price of cars); autoPrice (target: price of cars); baskball (target: points scored per minute); cloud (target: period rainfalls in the east target); echoMonths (target: number of months patient survived); fruitfly (target: longevity of mail fruitflies); pharynx (target: patient survival); pyrim (quantitative structure activity relationships); sleep (target: total sleep in hours per day); stock (target: price of one particular stock); strike (target: strike volume); triazines (target: activity); veteran (survival in days)		

Benchmark Datasets of *Chapelle et al. (2006)*



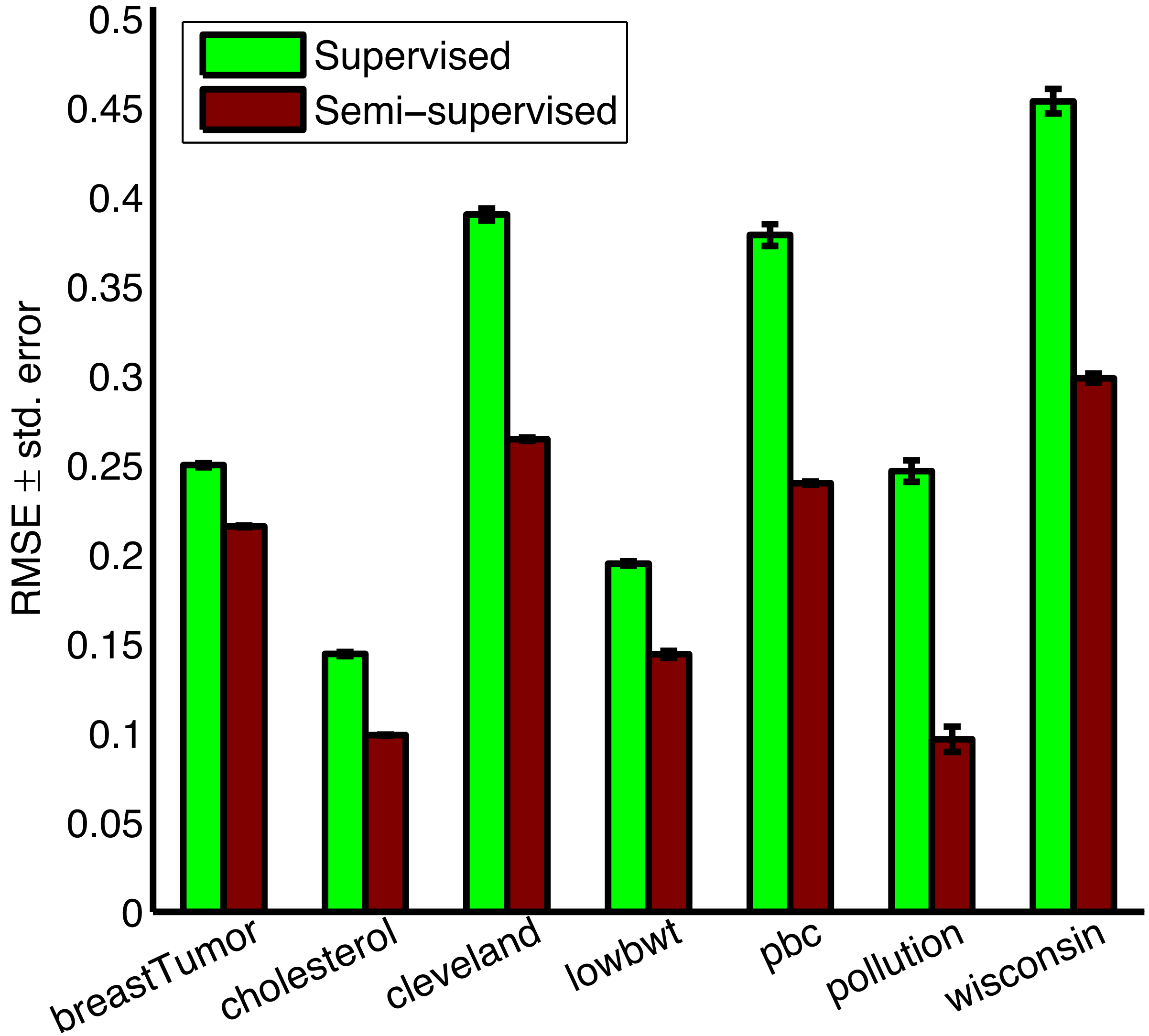
Asterisk = 1-NN, SVM

Self-training does not help for causal problems (cf. *Guo et al., 2010)*

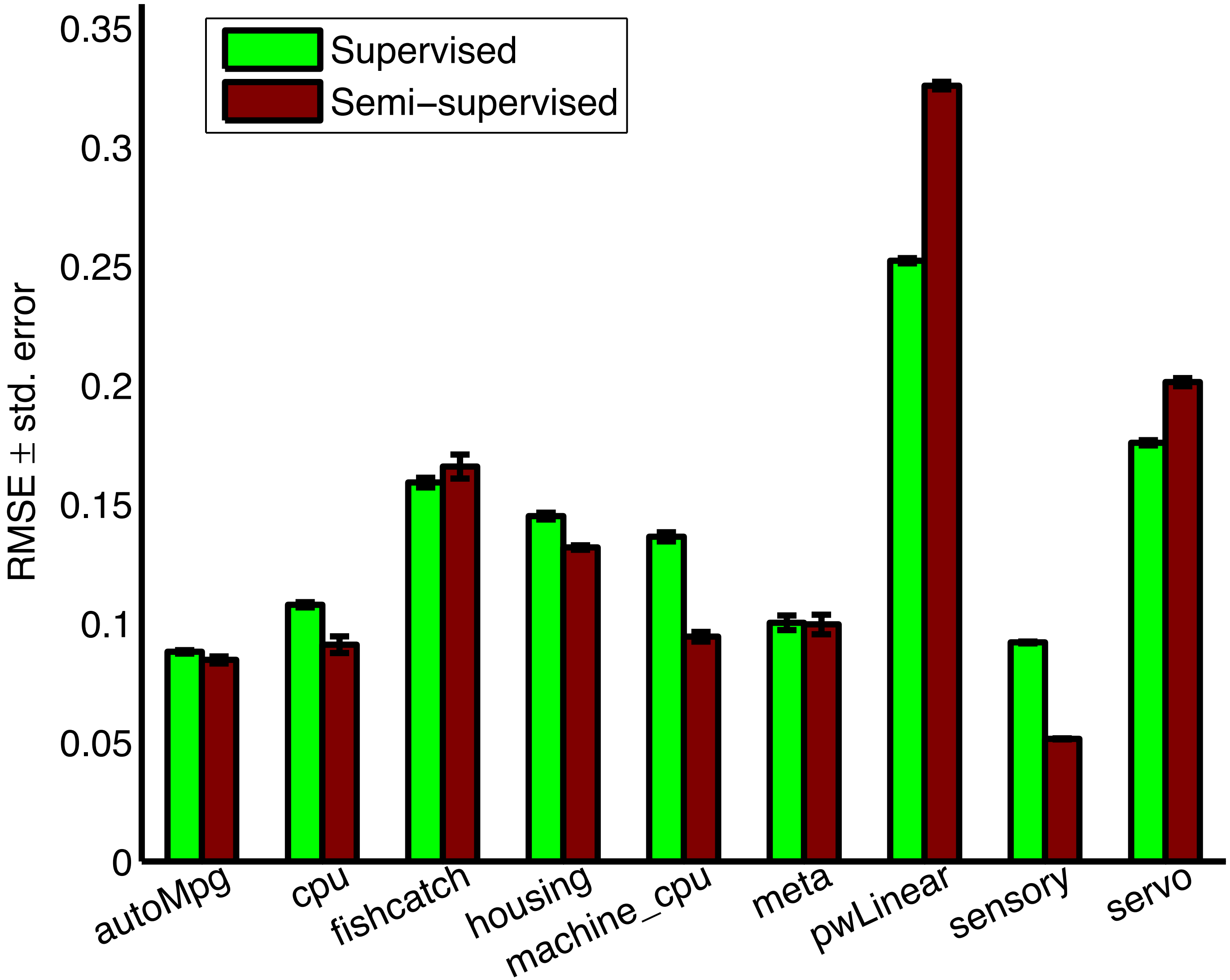


Relative error decrease = (error(base) –error(self-train)) / error(base)

Co-regularization helps for the **anticausal** problems of *Brefeld et al., 2006*



Co-regularizarion hardly helps for the **causal** problems of *Brefeld et al., 2006*



Causal Inference for Individual Objects *(Janzing & Schölkopf, 2010)*

Similarities between single objects also indicate causal relations:

However, if similarities are too simple there need not be a common cause:

Causal Markov Conditions

- Recall the **(Local) Causal Markov condition**:
  An observable is statistically independent of its non-descendants, given parents

- Reformulation:
  Given all direct causes of an observable, its non-effects provide no additional *statistical* information on it

Causal Markov Conditions

- Generalization:
  Given all direct causes of an observable, its non-effects provide no additional ~~*statistical*~~ information on it

- **Algorithmic Causal Markov Condition**:
  Given all direct causes of an object, its non-effects provide no additional *algorithmic* information on it

Kolmogorov complexity

(Kolmogorov 1965, Chaitin 1966, Solmonoff 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
- equality "=" is always understood up to string-independent additive constants (often denoted by $\stackrel{+}{=}$, but we drop the "+")

- $K(x)$ is uncomputable
- probability-free definition of information content

Conditional Kolmogorov complexity

- $K(y \,|\, x^*)$: length of the shortest program that generates y from the shortest description of the input x. For simplicity, we write $K(y \,|\, x)$.
- number of bits required for describing y if the shortest description of x is given
- note: x can be generated from its shortest description 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

- $$\begin{aligned} I\,(x:y) &:= K(x)+K(y)-K(x,y) \\ &= K(x)-K(x\,|\,y)=K(y)-K(y\,|\,x) \end{aligned}$$
- Interpretation: number of bits saved when compressing x, y jointly rather than independently
- Algorithmic independence $x \perp\!\!\!\perp y$: $\iff$ $I\,(x:y)=0$

Conditional algorithmic mutual information

Information that x has on y (and vice versa) when z is given

- $I\,(x : y \,|\, z) := K\,(x \,|\, z) + K\,(y \,|\, z) - K\,(x, y \,|\, z)$
- Analogy to statistical mutual information:

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

- Conditional algor. independence $x \perp\!\!\!\perp y \,|\, z :\Longleftrightarrow I\,(x : y \,|\, z) = 0$

MAX-PLANCK-GESELLSCHAFT

Algorithmic mutual information: example

Postulate: Local Algorithmic Markov Condition

Let $x_1, \ldots, x_n$ be observations (formalized as strings). Given its direct causes pa_j, every x_j is conditionally algorithmically independent of its non-effects nd_j

$$x_j \perp\!\!\!\perp nd_j \,|\, pa_j$$

Equivalence of Algorithmic Markov Conditions

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

- Local Markov condition

$$I\left(x_j \,:\, nd_j \,|\, pa_j\right) = 0$$

- Global Markov condition:

  If R d-separates S and T then $I\left(S : T \,|\, R\right) = 0$

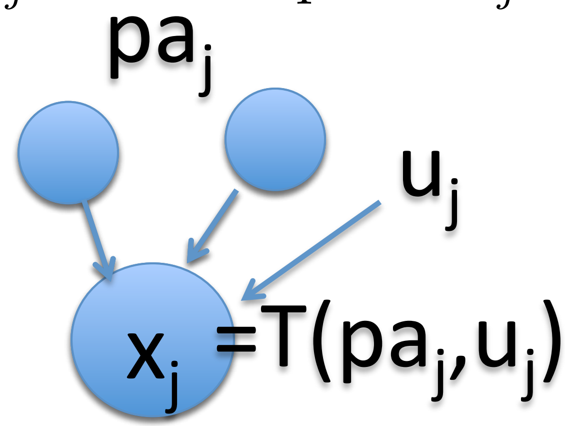
- Recursion formula for joint complexity

$$K(x_1, \ldots, x_n) = \sum_{j=1}^{n} K(x_j \,|\, pa_j)$$

Janzing & Schölkopf, IEEE Trans. Information Theory, 2010

Algorithmic model of causality

- for every node x_j there exists a program u_j that computes x_j from its parents pa_j



- all u_j are jointly independent
- the program u_j represents the causal mechanism that generates the effect from its causes
- u_j are the analog of the unobserved noise terms in the statistical functional model

Theorem: this model implies the algorithmic Markov condition

“Independent”= algorithmically independent?

Postulate (Janzing & Schölkopf, 2010, inspired by Lemeire & Dirkx, 2006):
The causal conditionals $p(X_j|PA_j)$ are algorithmically independent

- special case: $p(X)$ and $p(Y|X)$ are alg. independent for $X \to Y$
- can be used as justification for novel inference rules (e.g., for additive noise models: Steudel & Janzing 2010)
- excludes many, but not all violations of faithfulness (Lemeire & Janzing, 2012)

Generalized independences *Steudel, Janzing, Schölkopf (2010)*

Given n objects $\mathcal{O} := \{x_1, \dots, x_n\}$

Observation: if a function $R : 2^{\mathcal{O}} \to \mathbb{R}_0^+$ is submodular, i.e.,

$$R(S) + R(T) \geq R(S \cup T) + R(S \cap T) \qquad \forall S, T \subset \mathcal{O}$$

then

$$I(A; B\,|C) := R(A \cup C) + R(B \cup C) - R(A \cup B \cup C) - R(C) \geq 0$$

for all disjoint sets $A, B, C \subset \mathcal{O}$

Interpretation: I measures conditional dependence
(replace R with Shannon entropy to obtain usual mutual information)

Generalized Markov condition

Theorem: the following conditions are equivalent for a DAG G

- local Markov condition

$$x_j \perp\!\!\!\perp nd_j \,|pa_j$$

- global Markov condition: d-separation implies independence

- sum rule

$$R(A) = \sum_{j \in A} R(x_j|pa_j)\,,$$

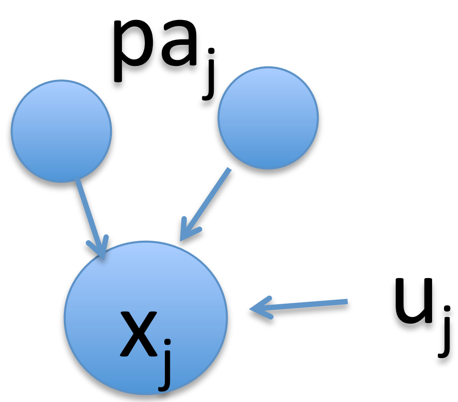
  for every ancestral set A of nodes.

–but can we postulate that the conditions hold w.r.t. to the true DAG?

Generalized functional model

Theorem:

- assume there are unobserved objects $u_1, \dots, u_n$



- assume

$$R(x_j, pa_j, u_j) = R(pa_j, u_j)$$

(x_j contains only information that is already contained in its parents + noise object)

then $x_1, \dots, x_n$ satisfy the Markov conditions

$\Rightarrow$ causal Markov condition is justified provided that mechanisms fit to information measure

Generalized PC

PC algorithm also works with generalized conditional independence

Examples:

1. $R :=$ number of different words in a text
2. $R :=$ compression length (e.g. Lempel Ziv is approximately submodular)
3. $R :=$ logarithm of period length of a periodic function

example 2 yielded reasonable results on simple real texts (different versions of a paper abstract)

Summary

- conventional causal inference algorithms use conditional statistical dependences
- more recent approaches also use other properties of the joint distribution
- non-statistical dependences also tell us something about causal directions

Selection within Markov equivalence classes

different approaches

- some "independence" condition between $p(X_j|PA_j)$

  Information-geometric method, Trace Method

- restricting conditionals/functional models to subsets

  Additive-noise models, post-nonlinear model

- define priors on $p(X_j|PA_j)$ that can yield different posteriors for equivalent DAGs

  Gaussian process based prior by Mooij, Stegle, Janzing, Schölkopf (2010)

- ?

Thank you for your attention





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