Causality

Evgeny Burnaev
Rodrigo Rivera

Skoltech

References

The course is based on materials from

- Ilya Shpitser, course on Causal Inference, Johns Hopkins
- Kun Zhang, course on Causality and Machine Learning, Carnegie Mellon University
- Jonas Peters, course on Causlity, Univ. of Copenhagen

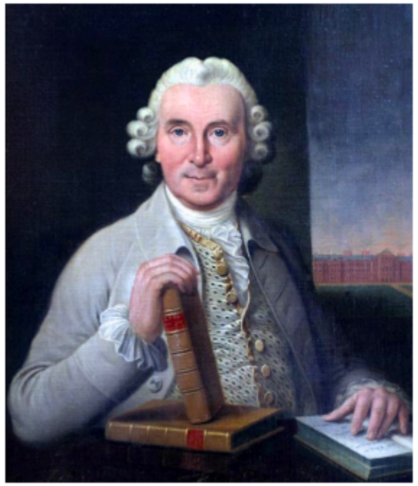
The main players are

- UCLA: Judea Pearl
- CMU: Peter Spirtes, Clark Glymour, Richard Scheines, Kun Zhang
- Harvard University: Donald Rubin, Jamie Robins
- ETH Zürich: Peter Bühlmann, Nicolai Meinshausen, Stefan Bauer
- Max-Planck-Institute Tübingen: Dominik Janzing, Bernhard Schölkopf, Mateo Rojas-Carulla
- University of Amsterdam: Joris Mooij

Two Quotes on Causality from the 1740s

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, ... where, if the first object had not been the second never had existed.

David Hume (1748)



Their cases were as similar as I could have them. They all in general had putrid gums, the spots and lassitude, with weakness of the knees. They lay together in one place, being a proper apartment for the sick in the forehold...

James Lind (1747)

Association vs. Causation

- Most scientific inquiry/data analyses have one of two goals:
    - Association/prediction, i.e., determine predictors or variables associated with the outcome of interest.
    - Causality, i.e., understand factors that cause or have an effect on the outcome of interest.
- We are often told that association is not causation.
- Spurious correlations are very common!

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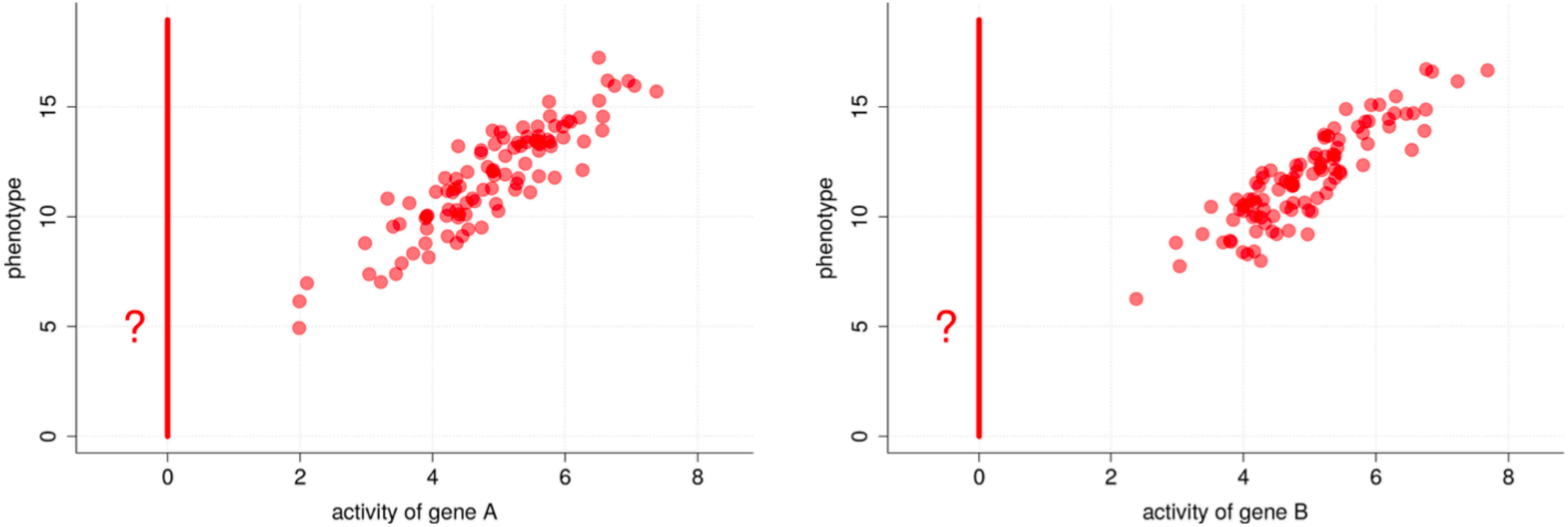
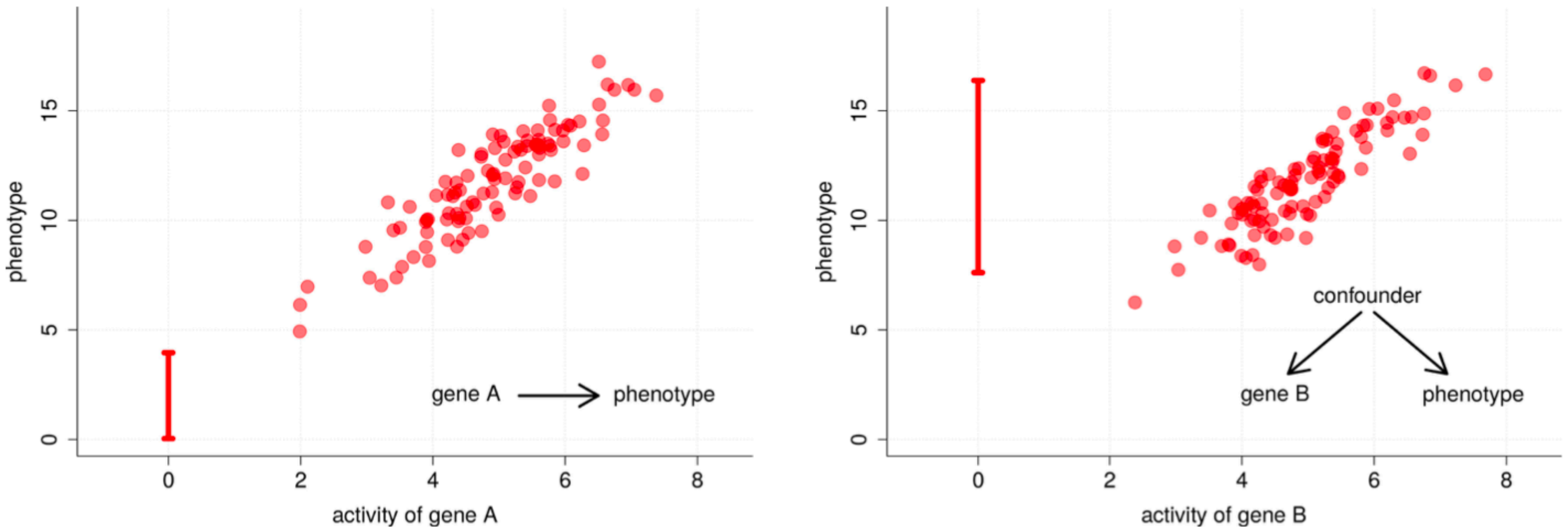
  - When can we claim an association is causation?
  - This is what causal inference is about.

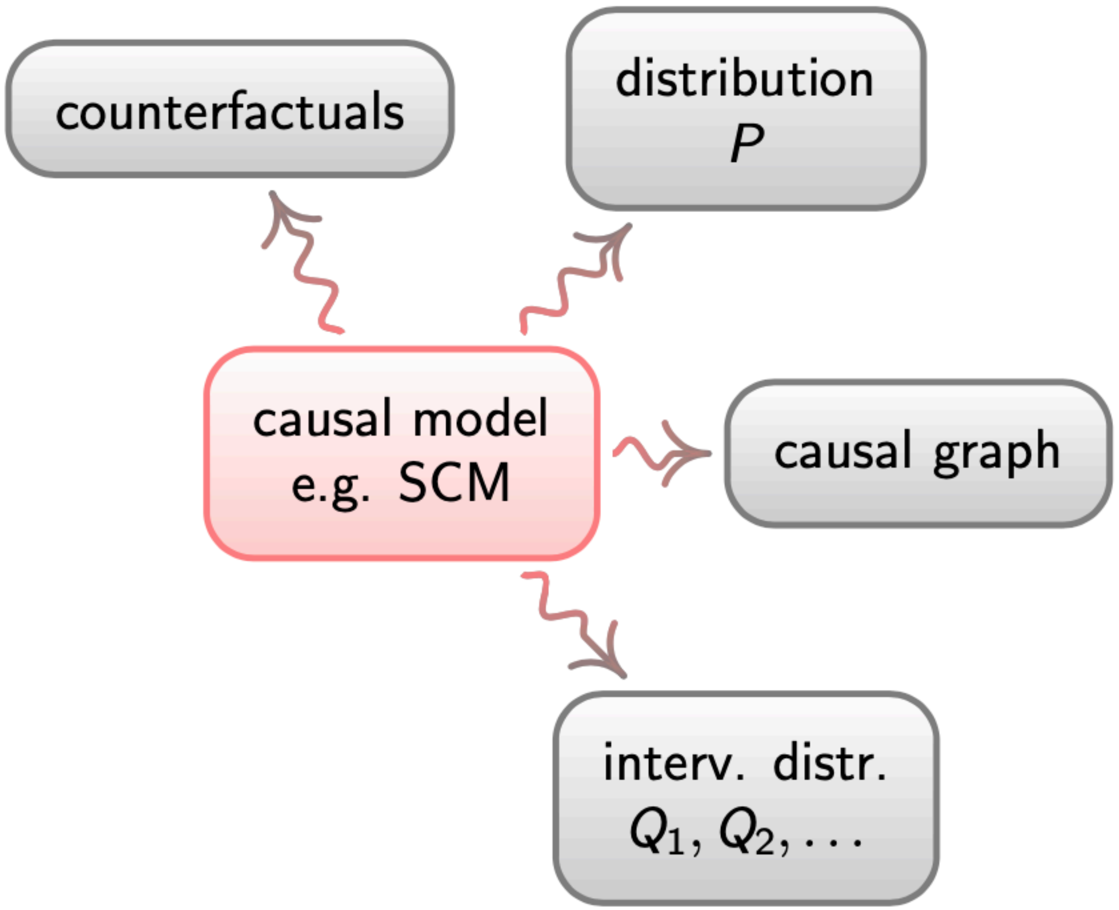
Step 1: Toy problem



Step 2: Causality matters!

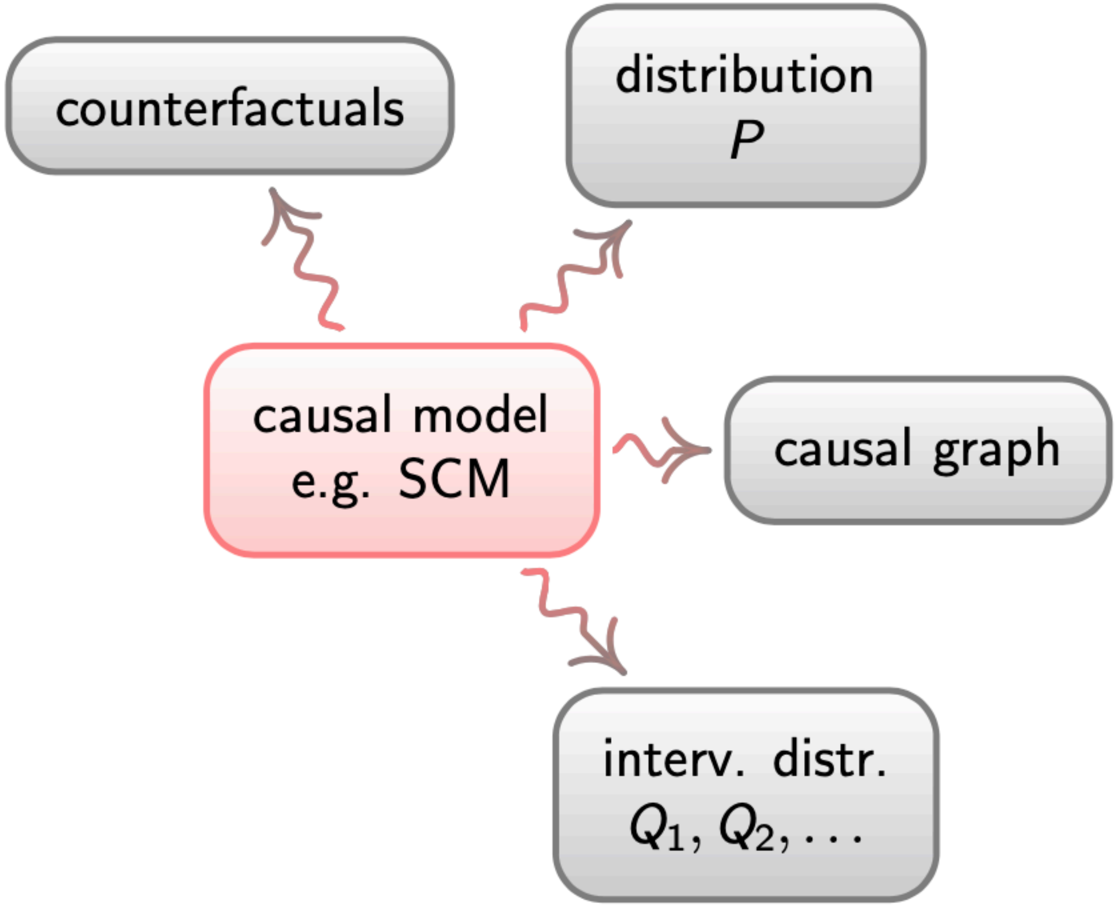


Step 3: What is a causal model?



Step 4: What questions are being asked?

- How does the model work?
- What if there are hidden variables or feedback?
- What are nice graphical representations?
- Can we test counterfactual statements?
- Can we infer the graph structure from data?
- Is causality useful, even in classical ML/statistics settings?



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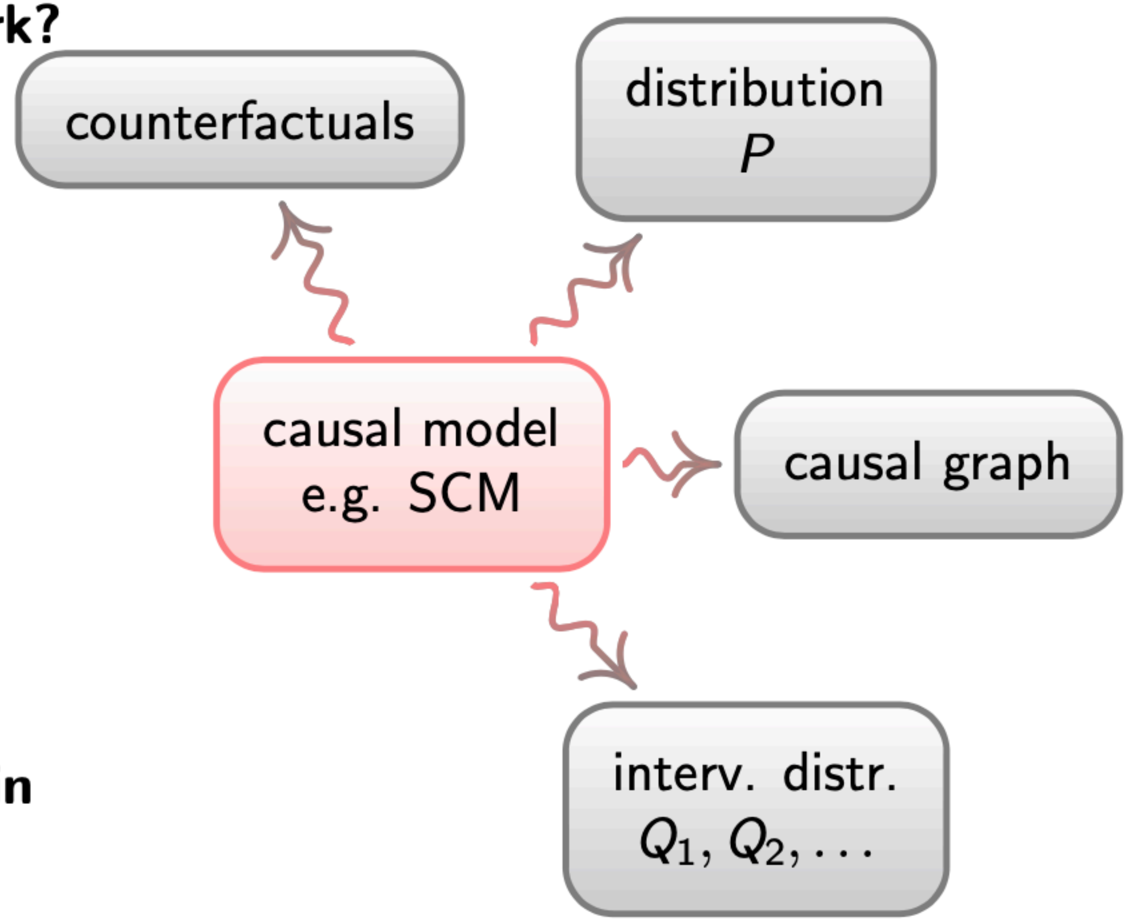
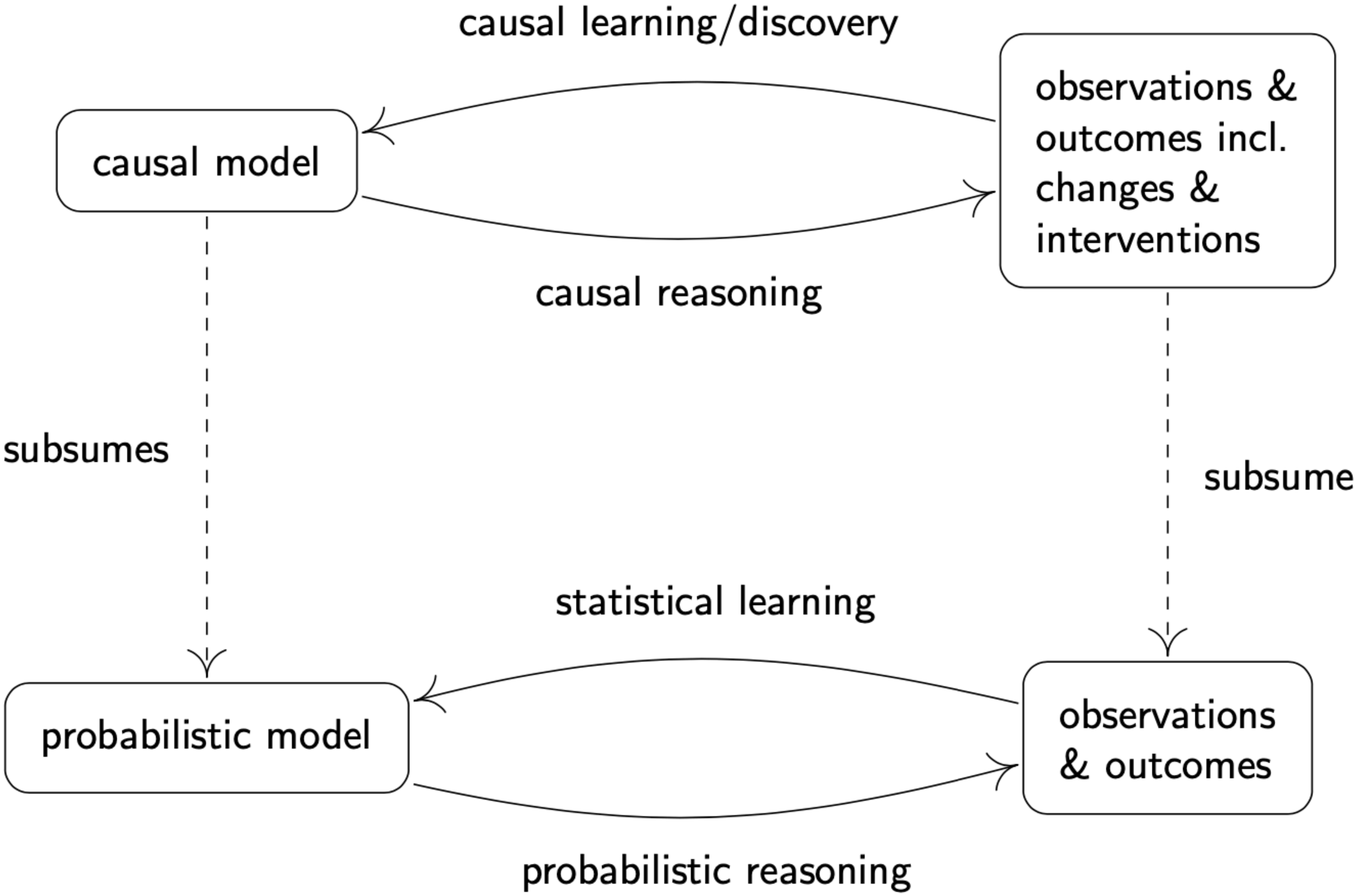
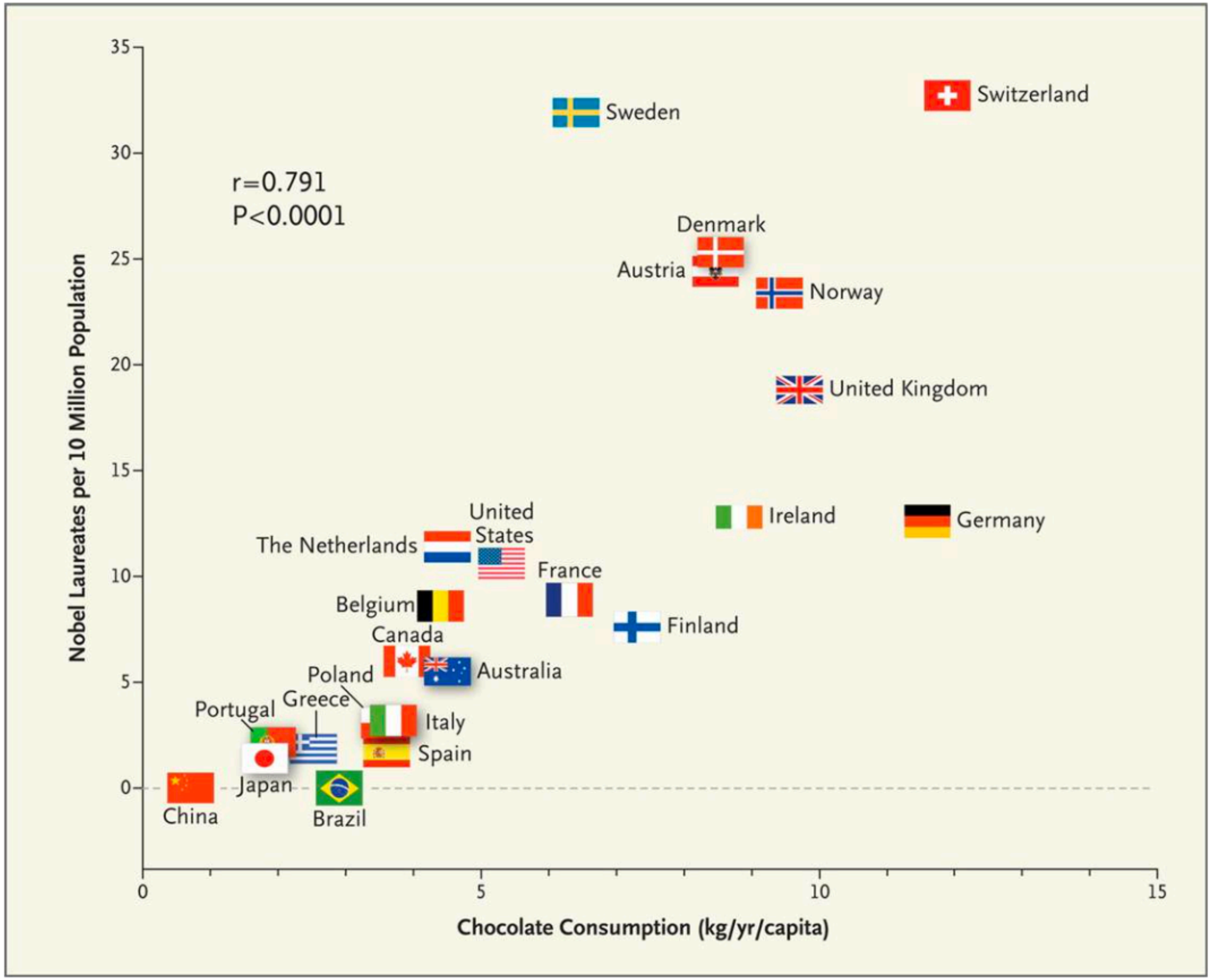



causal learning/discovery
causal model
observations & outcomes incl. changes & interventions
causal reasoning
subsumes
subsume
statistical learning
probabilistic model
observations & outcomes
probabilistic reasoning

Example: chocolate



F. H. Messerli: *Chocolate Consumption, Cognitive Function, and Nobel Laureates*, N Engl J Med 2012

Example: chocolate


Confectionery news.com

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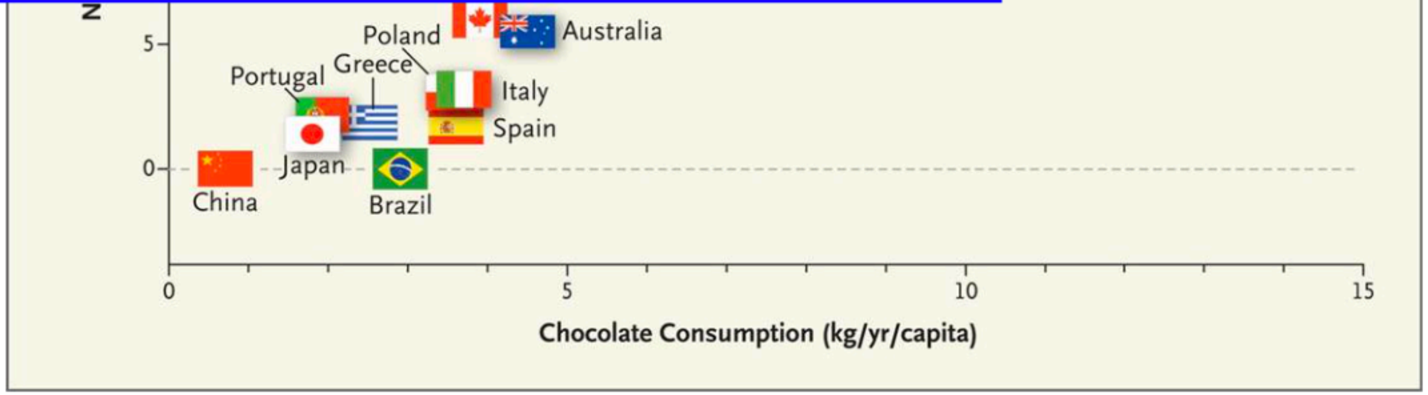
Eating chocolate produces Nobel prize winners, says study

By Oliver Nieburg, 11-Oct-2012

Related tags: noble prize, nobel laureate, Einstein, Marie Curie, chocolate, brain, Switzerland, Sweden, candy



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Example: chocolate

Example: smoking

BRITISH MEDICAL JOURNAL

LONDON SATURDAY SEPTEMBER 30 1950

SMOKING AND CARCINOMA OF THE LUNG

PRELIMINARY REPORT

BY

RICHARD DOLL, M.D., M.R.C.P.

Member of the Statistical Research Unit of the Medical Research Council

AND

A. BRADFORD HILL, Ph.D., D.Sc.

Professor of Medical Statistics, London School of Hygiene and Tropical Medicine; Honorary Director of the Statistical Research Unit of the Medical Research Council

In England and Wales the phenomenal increase in the number of deaths attributed to cancer of the lung provides one of the most striking changes in the pattern of mortality recorded by the Registrar-General. For example, in the quarter of a century between 1922 and 1947 the annual number of deaths recorded increased from 612 to

whole explanation, although no one would deny that it may well have been contributory. As a corollary, it is right and proper to seek for other causes.

Possible Causes of the Increase

Two main causes have from time to time been put for-

Example: smoking

BRITISH MEDICAL JOURNAL

TABLE VII.—*Estimate of Total Amount of Tobacco Ever Consumed by Smokers; Lung-carcinoma Patients and Control Patients with Diseases Other Than Cancer*

Disease Group	No. Who have Smoked Altogether					Probability Test
	365 Cigs.–	50,000 Cigs.–	150,000 Cigs.–	250,000 Cigs.–	500,000 Cigs.+	
Males:						
Lung-carcinoma patients (647)	19 (2·9%)	145 (22·4%)	183 (28·3%)	225 (34·8%)	75 (11·6%)	$\chi^2=30{\cdot}60$; n=4; P<0·001
Control patients with diseases other than cancer (622)..	36 (5·8%)	190 (30·5%)	182 (29·3%)	179 (28·9%)	35 (5·6%)	
Females:						
Lung-carcinoma patients (41)..	10 (24·4%)	19 (46·3%)	5 (12·2%)	7 (17·1%)	0 (0·0%)	$\chi^2=12{\cdot}97$; n=2; 0·001< P < 0·01 (Women smoking 15 or more cigarettes a day grouped together)
Control patients with diseases other than cancer (28) ..	19 (67·9%)	5 (17·9%)	3 (10·7%)	1 (3·6%)	0 (0·0%)	

UNG

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y Director of the Statistical

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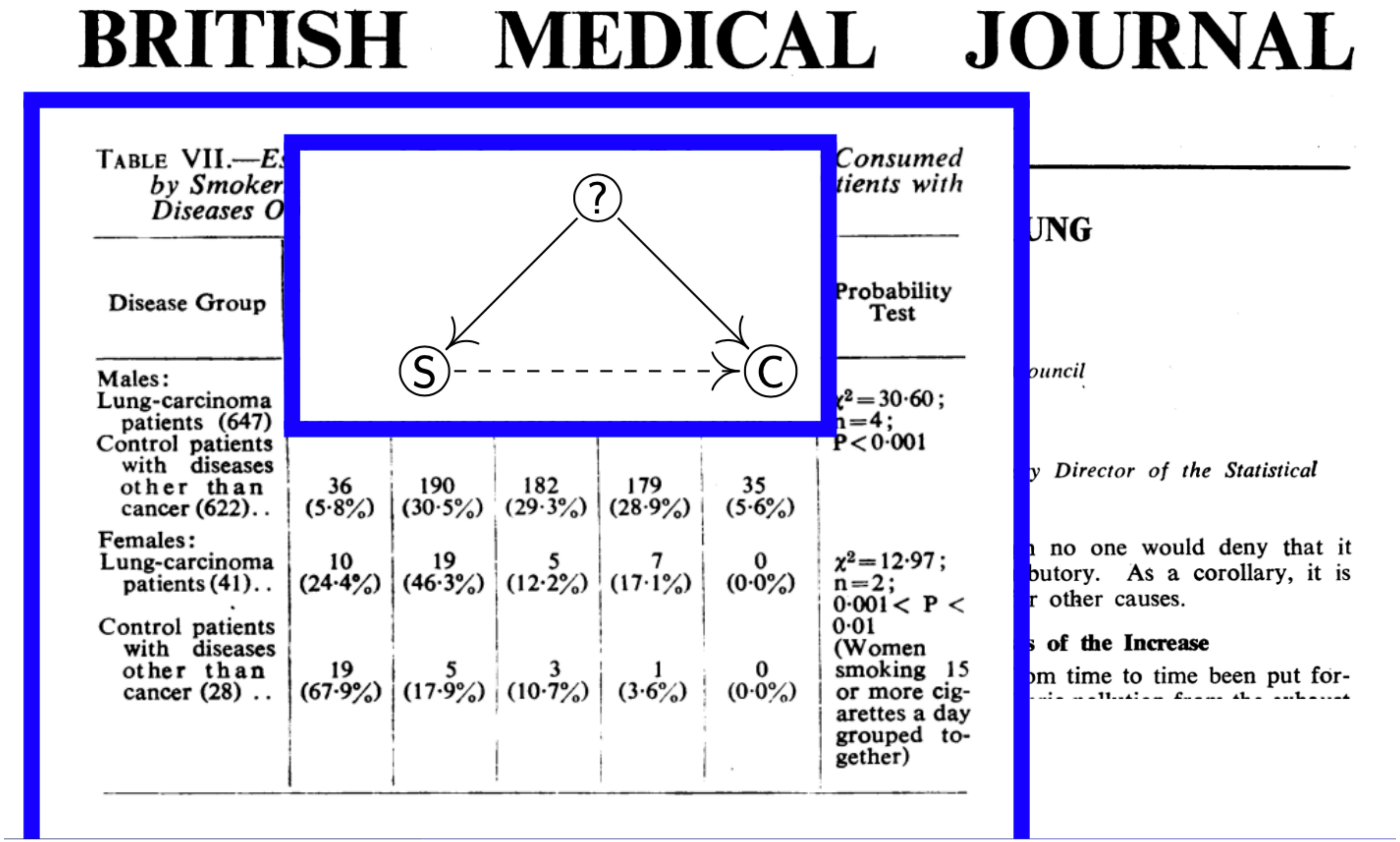
Example: smoking



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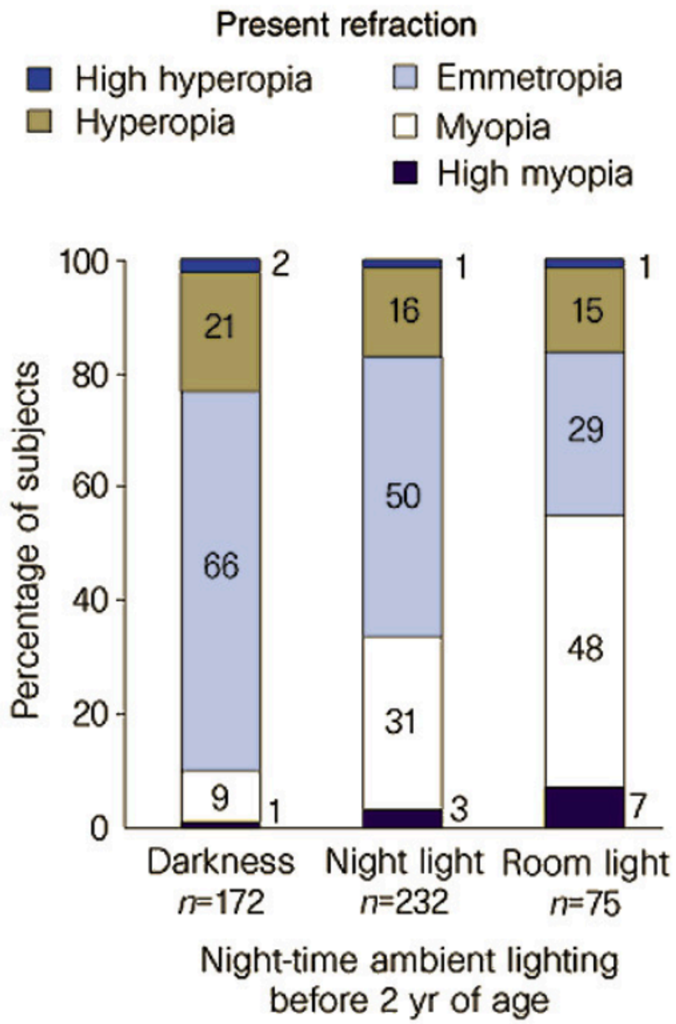
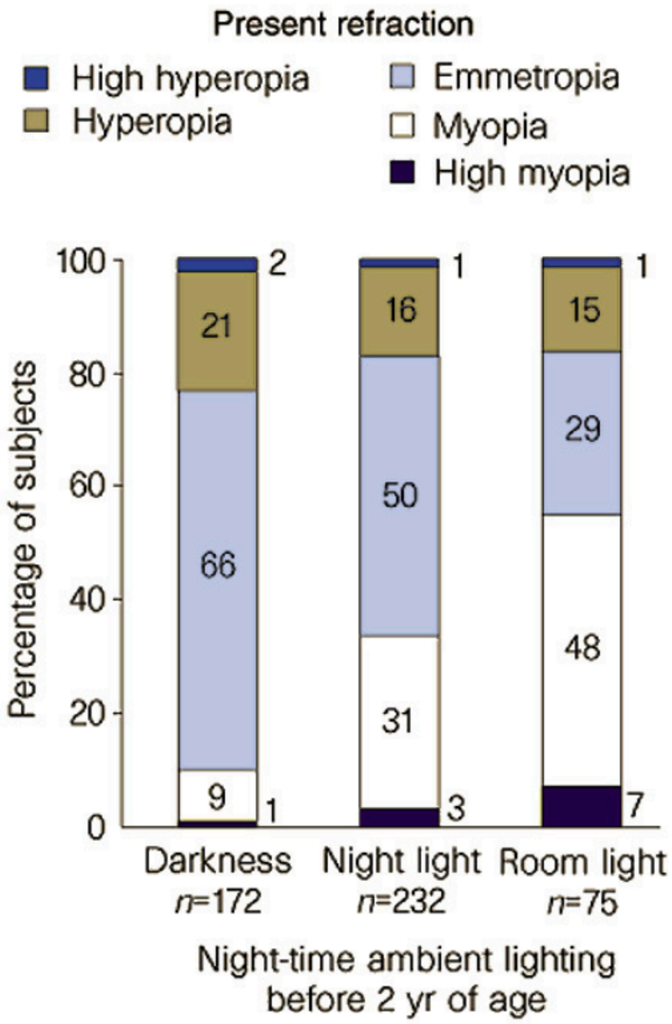
Example: myopia



Example: myopia



"the strength of the association . . . does suggest that the absence of a daily period of darkness during childhood is a potential precipitating factor in the development of myopia"

Quinn, Shin, Maguire, Stone: *Myopia and ambient lighting at night*, Nature 1999

Example: myopia

Patente

Night light with sleep timer

US 20050007889 A1

ZUSAMMENFASSUNG

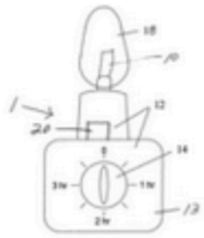
A timer a light and an optional music source is located on or in a housing of a nightlight assembly. When this assembly is plugged into a source of electric power, the timer is set to a selected time for the light and optional music to remain on. After this selected time has elapsed, the light and music automatically turns off, allowing for sleep in appropriate darkness and silence.

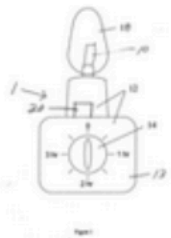
Veröffentlichungsnummer	US20050007889 A
Publikationstyp	Anmeldung
Anmeldenummer	US 10/614,245
Veröffentlichungsdatum	13. Jan. 2005
Eingetragen	8. Juli 2003
Prioritätsdatum ?	8. Juli 2003
Erfinder	Karin Peterson
Ursprünglich Bevollmächtigter	Peterson Karin Lyn
Zitat exportieren	BiBTeX, EndNote, R

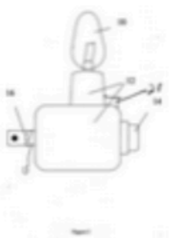
Klassifizierungen (4)

Externe Links: USPTO, USPTO-Zuordnung, Esp

BILDER (3)







BESCHREIBUNG

ANSPRÜCHE (18)

Example: myopia

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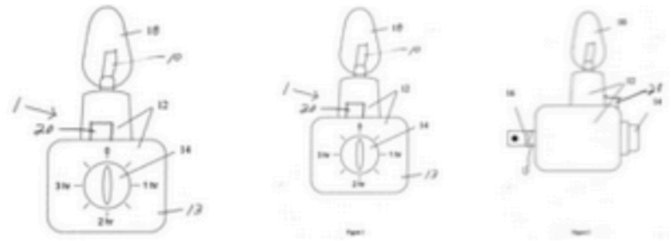

Question: Does the night light with sleep timer help?

BESCHREIBUNG

ANSPRÜCHE (18)

Example: kidney stones

	Treatment A	Treatment B
	$\frac{273}{350} = 0.78$	$\frac{289}{350} = 0.83$
	$\frac{562}{700} = 0.80$	

Charig et al.: *Comparison of treatment of renal calculi by open surgery, (...)* , British Medical Journal, 1986

Example: kidney stones

	Treatment A	Treatment B
Small Stones ($\frac{357}{700} = 0.51$)	$\frac{81}{87} = 0.93$	$\frac{234}{270} = 0.87$
Large Stones ($\frac{343}{700} = 0.49$)	$\frac{192}{263} = 0.73$	$\frac{55}{80} = 0.69$
	$\frac{273}{350} = 0.78$	$\frac{289}{350} = 0.83$
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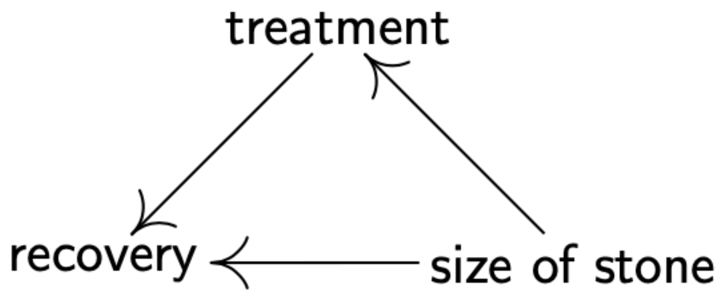
Charig et al.: *Comparison of treatment of renal calculi by open surgery, (...)* , British Medical Journal, 1986

Example: kidney stones

underlying ground truth:



Example: kidney stones

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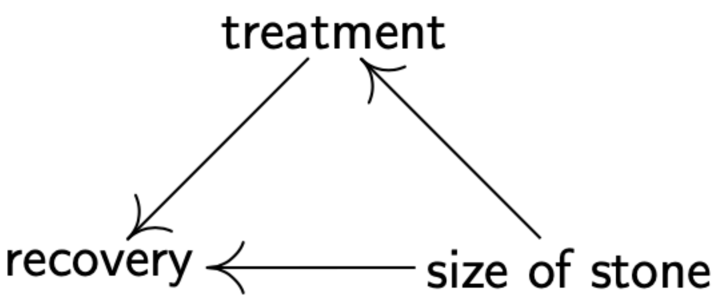
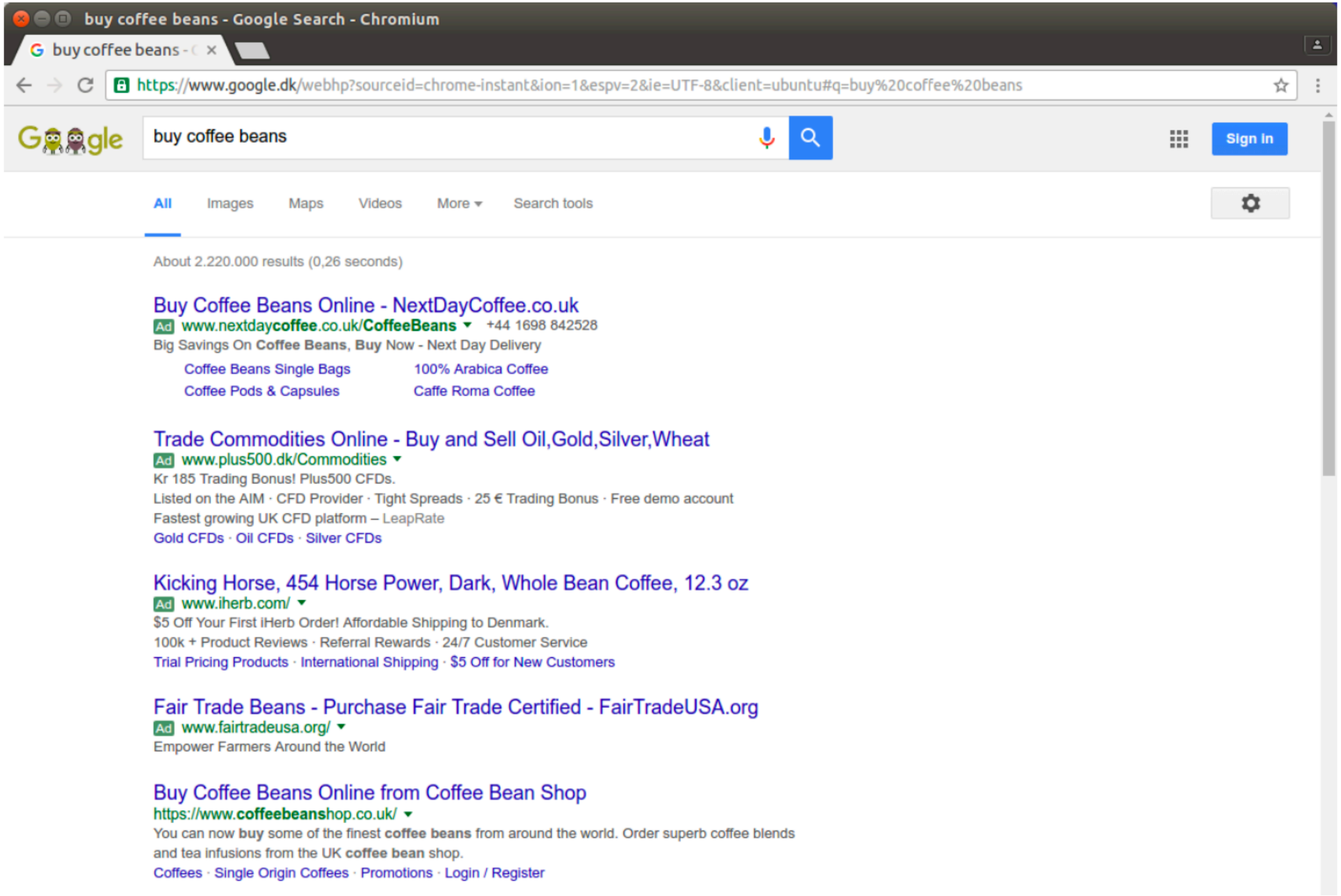


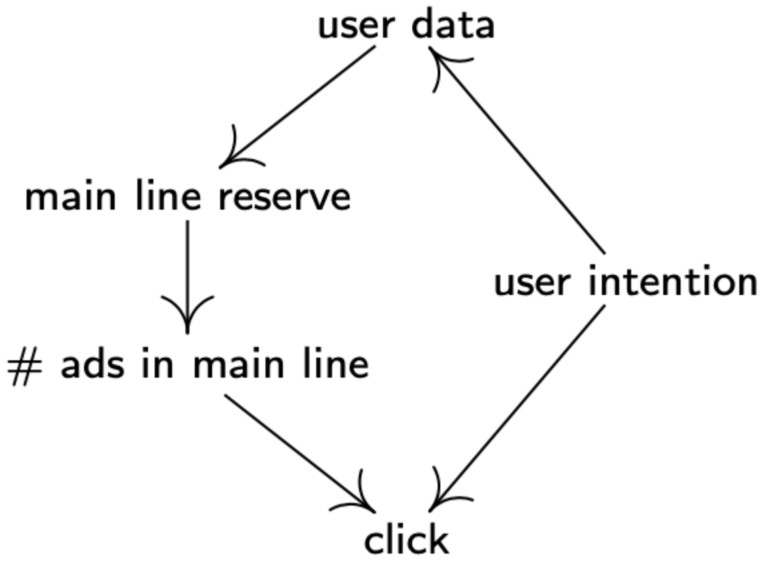
Question: What is the expected recovery if all get treatment B?

(Make treatment independent of size.)

Example: advertisement

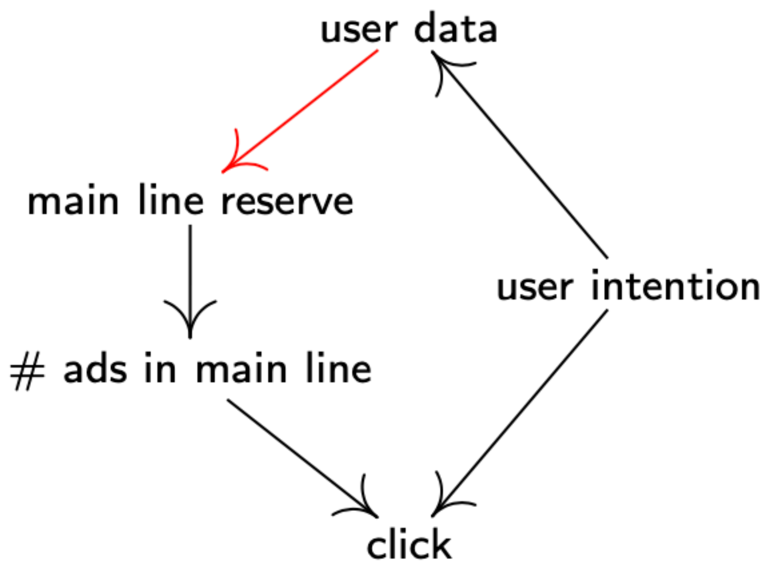


Example: advertisement



Bottou et al.: *Counterfactual Reasoning and Learning Systems: The Example of Computational Advertising*, JMLR 2013

Example: advertisement



Question: How do we choose an optimal main line reserve?

Bottou et al.: *Counterfactual Reasoning and Learning Systems: The Example of Computational Advertising*, JMLR 2013

Example: gene interactions

genetic perturbation experiments for yeast

- $p = 6170$ genes
- $n_{obs} = 160$ wild-types
- $n_{int} = 1479$ gene deletions (targets known)

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genetic perturbation experiments for yeast

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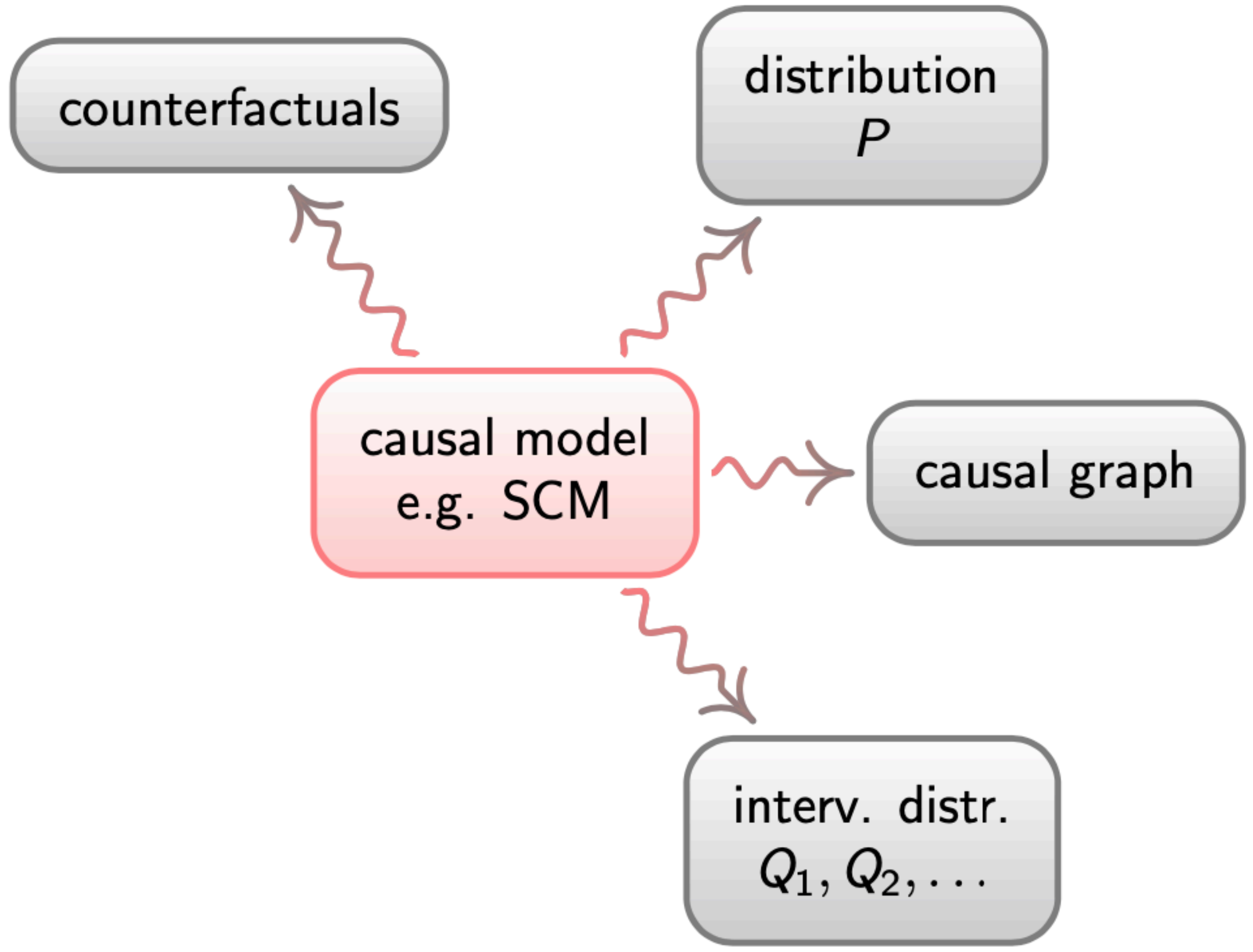
- Causal relationships are often stable!

Kemmeren et al.: Large-scale genetic perturbations reveal reg. networks and an abundance of gene-specific repressors. Cell, 2014

Part I: Causal Language and causal reasoning



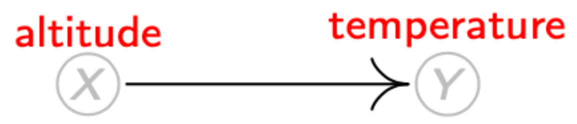
counterfactuals
distribution
P
causal model
e.g. SCM
causal graph
interv. distr.
$Q_1, Q_2, \ldots$

Example: Two variables

SCMs $(\mathbf{S}, P^{\mathbf{N}})$ model observational distributions.

$$X := N_X$$
$$Y := -6X + N_Y$$

$$N_X, N_Y \overset{\text{iid}}{\sim} \mathcal{N}(0,1)$$



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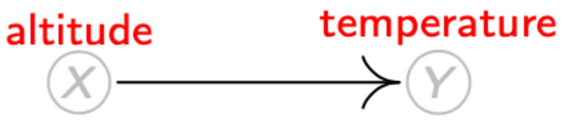


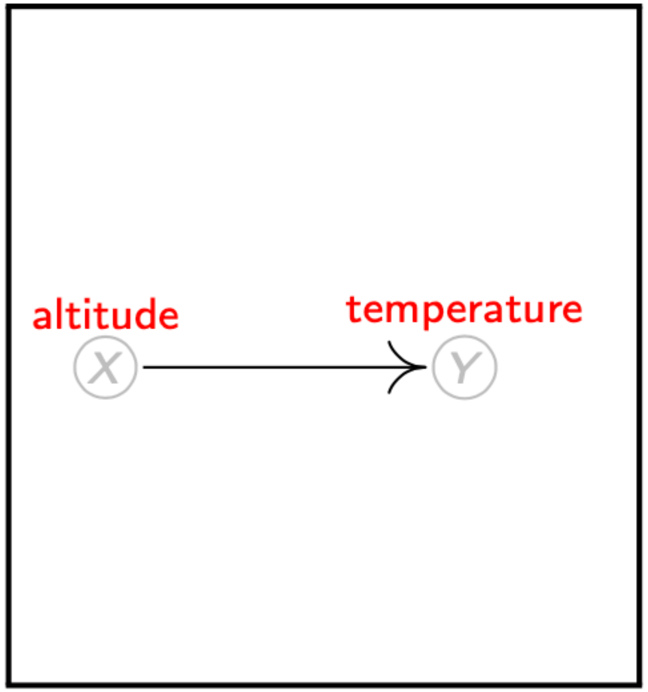
$$(X, Y) \sim \mathcal{N}\left(\begin{pmatrix} 0 \\ 0 \end{pmatrix}, \begin{pmatrix} 1 & -6 \\ -6 & 37 \end{pmatrix}\right)$$

Example: Two variables

SCMs $(\mathbf{S}, P^{\mathbf{N}})$ model interventions, too.

$$X := N_X \quad X := 3$$
$$Y := -6X + N_Y$$

$$N_X, N_Y \overset{\text{iid}}{\sim} \mathcal{N}(0,1)$$



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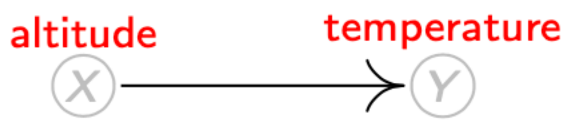

$$P(X = 3) = 1 \quad \text{and} \quad Y \sim \mathcal{N}(-18, 1)$$

Example: Two variables

SCMs $(\mathbf{S}, P^{\mathbf{N}})$ model interventions, too.

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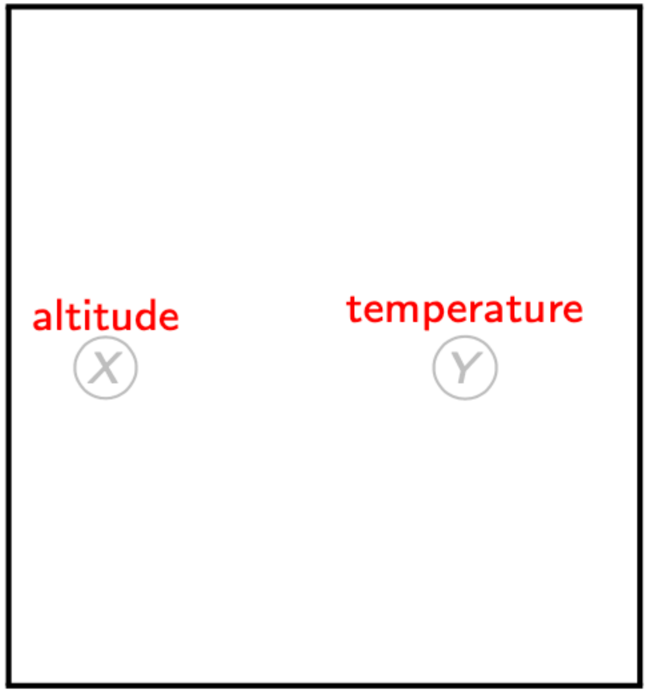
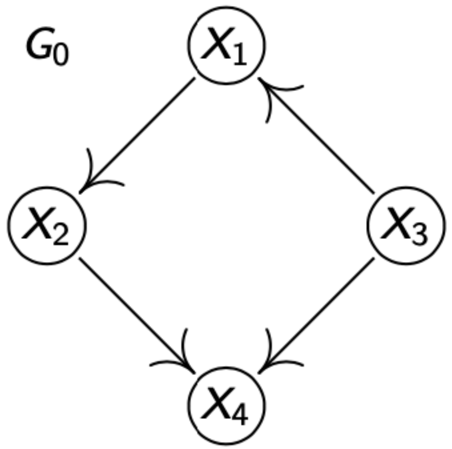


$$(X,Y) \sim \mathcal{N}\left(\begin{pmatrix} 0 \\ 2 \end{pmatrix}, \begin{pmatrix} 1 & 0 \\ 0 & 2 \end{pmatrix}\right)$$

SCMs $(\mathbf{S}, P^{\mathbf{N}})$: structural equations with noise distribution.

$$
\begin{aligned}
X_1 &:= f_1(X_3, N_1)\\
X_2 &:= f_2(X_1, N_2)\\
X_3 &:= f_3(N_3)\\
X_4 &:= f_4(X_2, X_3, N_4)
\end{aligned}
$$

- N_i jointly independent
- G_0 has no cycles



SCMs $(\mathbf{S}, P^{\mathbf{N}})$ model observational distributions over $X_1, \ldots, X_d$. Call it P.

$$
\begin{aligned}
X_1 &:= f_1(X_3, N_1) \\
X_2 &:= f_2(X_1, N_2) \\
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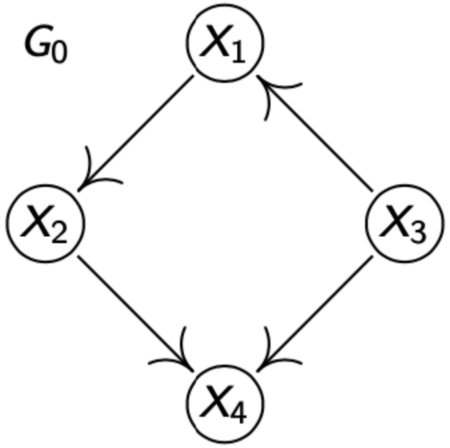

SCMs $(\mathbf{S}, P^{\mathbf{N}})$ model interventions, too. Call it: $P_{do(X_1:=0)}$.

$$\begin{aligned} X_1 &:= 0 \\ X_2 &:= f_2(X_1, N_2) \\ X_3 &:= f_3(N_3) \\ X_4 &:= f_4(X_2, X_3, N_4) \end{aligned}$$

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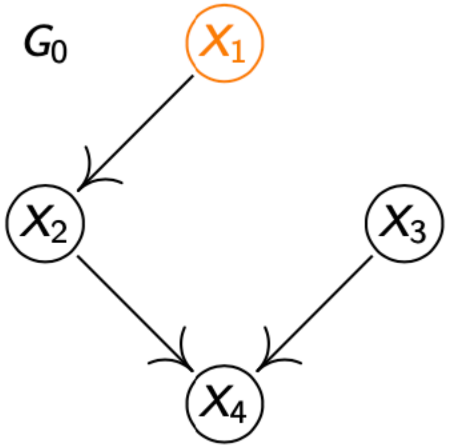

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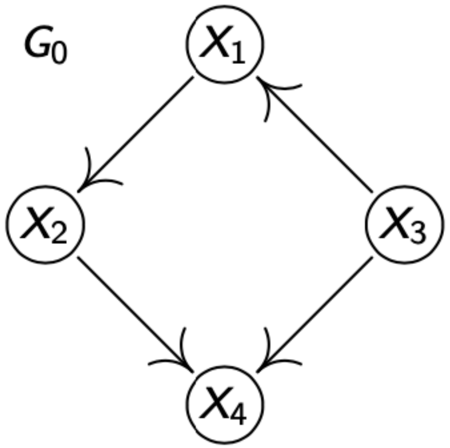

SCMs model interventions, too. Call it $P_{do(X_4:=13)} \neq P(.\,|\,X_4 = 13)$

$$
\begin{aligned}
X_1 &:= f_1(X_3, N_1)\\
X_2 &:= f_2(X_1, N_2)\\
X_3 &:= f_3(N_3)\\
X_4 &:= 13
\end{aligned}
$$

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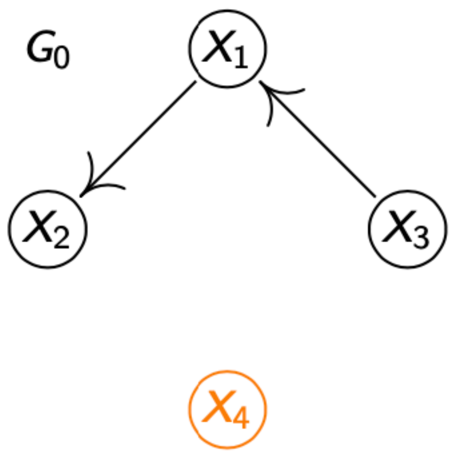
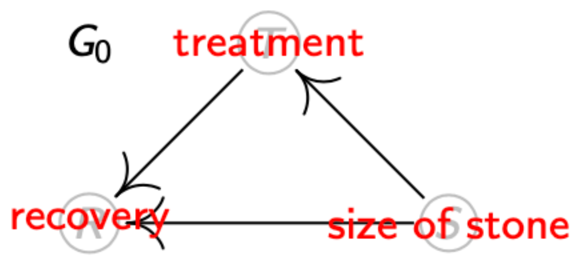


Example: kidney stones

Given: graph and P, i.e., only the structure, not the functions.

$$T := f_1(S, N_1)$$
$$R := f_2(T, S, N_2)$$
$$S := f_3(N_3)$$

- N_i jointly independent
- G_0 has no cycles



Example: kidney stones

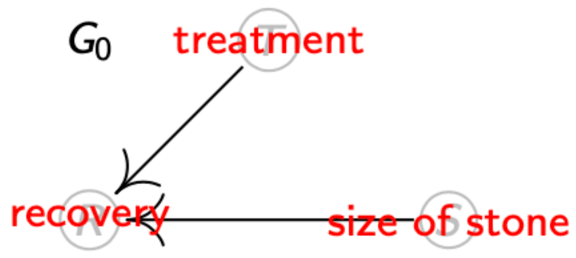
Given: graph and P. We want to compute $P_{\mathrm{do}(T:=A)}$.

$T := f_1(S, N_1)$ $T := A$

$R := f_2(T, S, N_2)$

$S := f_3(N_3)$

- N_i jointly independent
- G_0 has no cycles



IMPORTANT: modularity, autonomy: Aldrich 1989, Pearl 2009, Schölkopf et al. 2012, ...

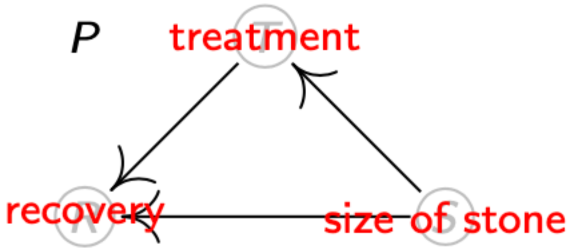
Most Useful Tautology Ever

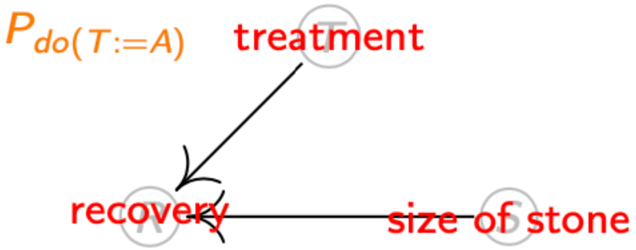
If you intervene only on X_j, you intervene only on X_j (MUTE).

Example: kidney stones

	Treatment A	Treatment B
Small Stones ($\frac{357}{700} = 0.51$)	$\frac{81}{87} = 0.93$	$\frac{234}{270} = 0.87$
Large Stones ($\frac{343}{700} = 0.49$)	$\frac{192}{263} = 0.73$	$\frac{55}{80} = 0.69$
	$\frac{273}{350} = 0.78$	$\frac{289}{350} = 0.83$
	$\frac{562}{700} = 0.80$	

Charig et al.: *Comparison of treatment of renal calculi by open surgery, (...)* , British Medical Journal, 1986





wanted: $P_{do(T:=A)}(R = 1)$

use: $P(R \mid S, T) \quad = \quad P_{do(T:=A)}(R \mid S, T)$

Example: kidney stones

$$
\begin{aligned}
E_{do(T:=A)}R &= P_{do(T:=A)}(R = 1) \\
&= \sum_s P_{do(T:=A)}(R = 1, S = s, T = A) \\
&= \sum_s P_{do(T:=A)}(R = 1 \mid S = s, T = A) P_{do(T:=A)}(S = s, T = A) \\
&= \sum_s P_{do(T:=A)}(R = 1 \mid S = s, T = A) P_{do(T:=A)}(S = s) \\
&= \sum_s P(R = 1 \mid S = s, T = A) P(S = s) \\
&= 0.832 \\
&> 0.782 \\
&= \dots \\
&= P_{do(T:=B)}(R = 1) = E_{do(T:=B)}R
\end{aligned}
$$

Example: kidney stones

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\begin{aligned}
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&= \sum_s P_{do(T:=A)}(R=1 \,|\, S=s, T=A) P_{do(T:=A)}(S=s) \\
&= \sum_s P(R=1 \,|\, S=s, T=A) P(S=s) \\
&= 0.832 \qquad \neq P(R=1 \,|\, T=A) \\
&> 0.782 \\
&= \ldots \\
&= P_{do(T:=B)}(R=1) = E_{do(T:=B)}R
\end{aligned}
$$

This idea holds more generally.

Definition

Given an SCM over $(X, Y, \mathbf{W})$. We call $\mathbf{Z} \subseteq \mathbf{W}$ a valid adjustment set for (X, Y) if

$$p_{do(X:=x)}(y) = \sum_{\mathbf{z}} p(y|x, \mathbf{z})p(\mathbf{z}) \neq p(y|x)$$

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Proposition (Parent Adjustment)

Assume $Y \notin PA(X)$. Then

$$PA(X) \text{ is a valid adjustment set for } (X, Y).$$

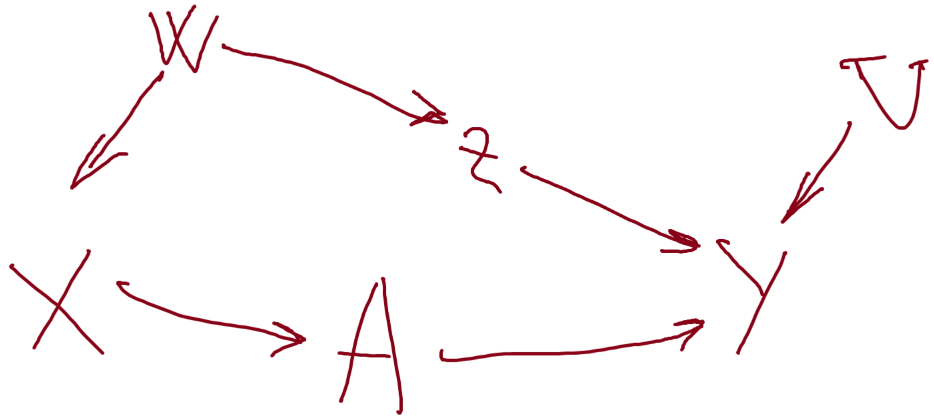
Adjusting in Linear Gaussian Models

Measure of variable influence

Compare $p(Y)$ and $P_{do(X=x)}(Y)$

$$C_{X\to Y} = \frac{\partial}{\partial x} \mathbb{E}_{do\,X=x}[Y]$$

Linear models



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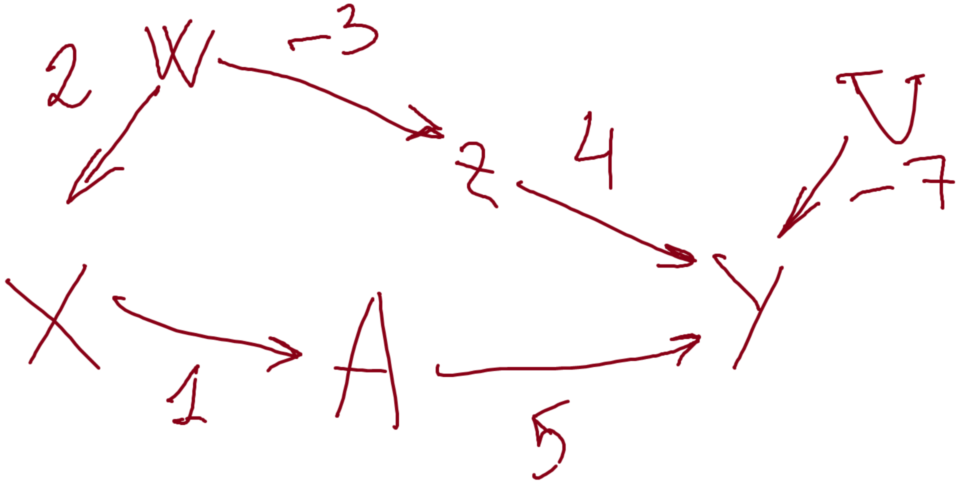

$$C_{X \to Y} = 1 * 5$$

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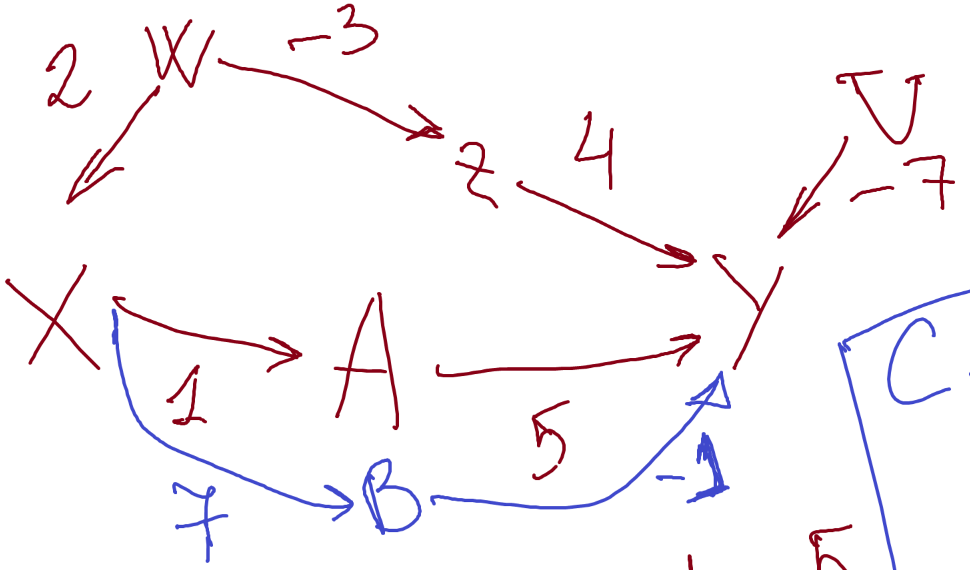


$$C_{X\to Y} = 1 * 5$$

$$\boxed{C_{X\to Y} = 1 * 5 + 7 * (-1) = -2}$$

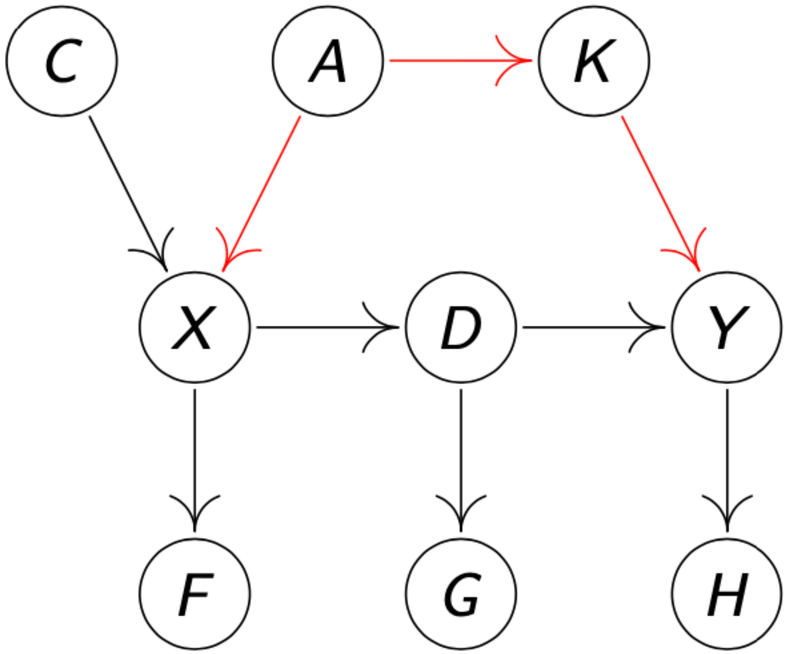
Adjustment and Linear models

If Z is a valid adjustment set

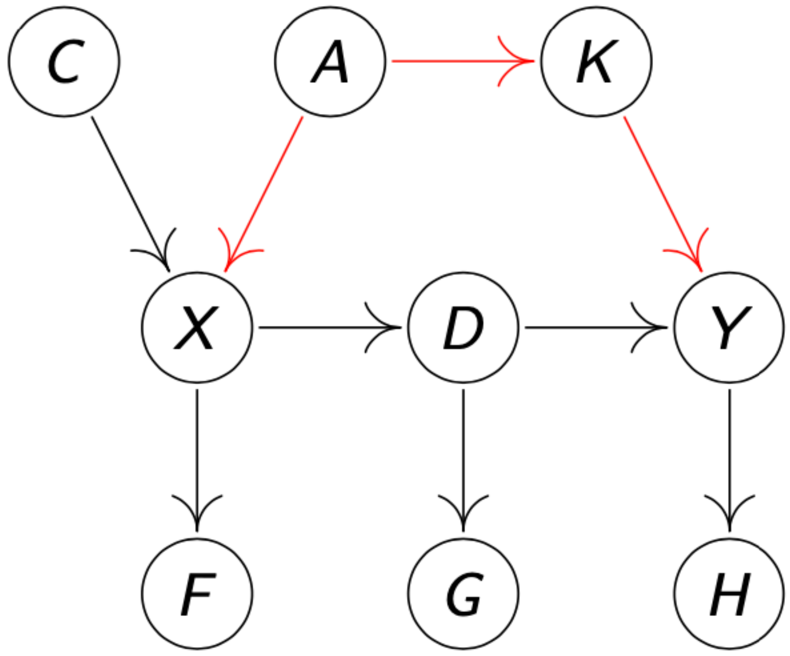
$C_{X \to Y}$ = regr. coefficient for X
in a linear model

$$Y \sim aX + bZ, \text{ i.e.}$$

$$C_{X \to Y} = a$$



$X \leftarrow A \rightarrow K \rightarrow Y$ is a “backdoor path” from X to Y.



$X \leftarrow A \rightarrow K \rightarrow Y$ is a "backdoor path" from X to Y.

$$\mathbf{Z} = \{C, A\},$$
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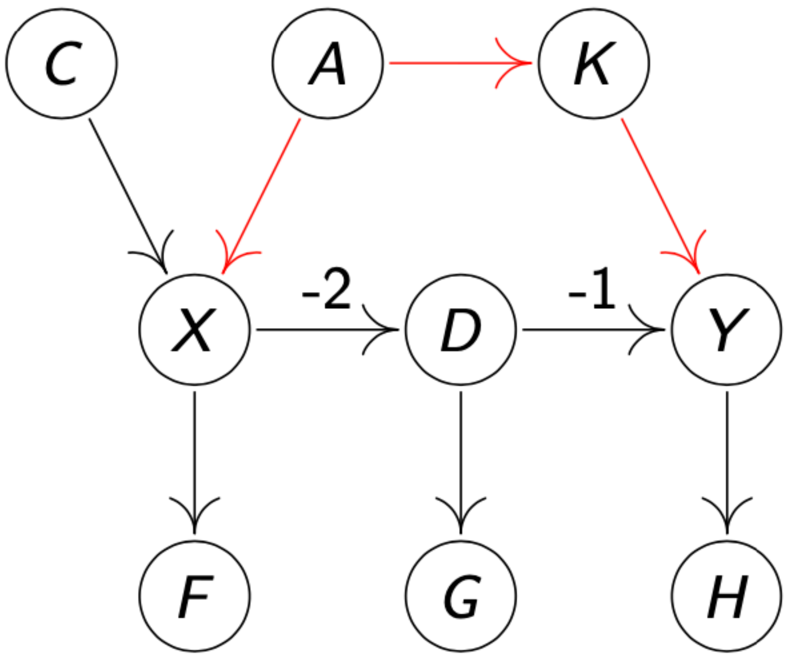
are valid adjustment sets for (X, Y) (no proof).



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```
n <- 500

# generate a sample from the distr. ent. by the SCM
set.seed(1)
C <- rnorm(n)
A <- 0.8*rnorm(n)
K <- A + 0.1*rnorm(n)
X <- C - 2*A + 0.2*rnorm(n)
F <- 3*X + 0.8*rnorm(n)
D <- -2*X + 0.5*rnorm(n)
G <- D + 0.5*rnorm(n)
Y <- 2*K - D + 0.2*rnorm(n)
H <- 0.5*Y + 0.1*rnorm(n)

lm(Y~X)$coefficients
lm(Y~X+K)$coefficients
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```

$Y \sim X \Rightarrow C_{X \to Y} \simeq \underline{1}.30$

$Y \sim X + K \Rightarrow C_{X \to Y} \simeq 2.0\underline{1}$

$Y \sim X + F + C + K \Rightarrow C_{X \to Y} \simeq 1.98$

$Y \sim X + F + C + K + H \Rightarrow C_{X \to Y} \simeq 0.11$

Example: smoking

BRITISH MEDICAL JOURNAL

LONDON SATURDAY SEPTEMBER 30 1950

SMOKING AND CARCINOMA OF THE LUNG

PRELIMINARY REPORT

BY

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Member of the Statistical Research Unit of the Medical Research Council

AND

A. BRADFORD HILL, Ph.D., D.Sc.

Professor of Medical Statistics, London School of Hygiene and Tropical Medicine; Honorary Director of the Statistical Research Unit of the Medical Research Council

In England and Wales the phenomenal increase in the number of deaths attributed to cancer of the lung provides one of the most striking changes in the pattern of mortality recorded by the Registrar-General. For example, in the quarter of a century between 1922 and 1947 the annual number of deaths recorded increased from 612 to

whole explanation, although no one would deny that it may well have been contributory. As a corollary, it is right and proper to seek for other causes.

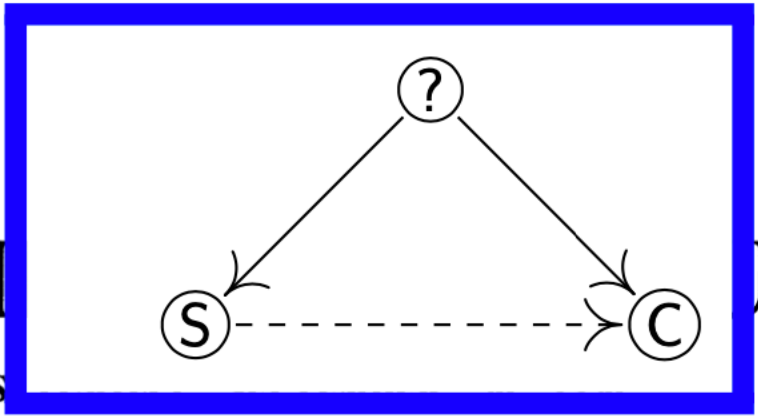
Possible Causes of the Increase

Two main causes have from time to time been put for-

Example: smoking

BRITISH M URNAL

LONDON S



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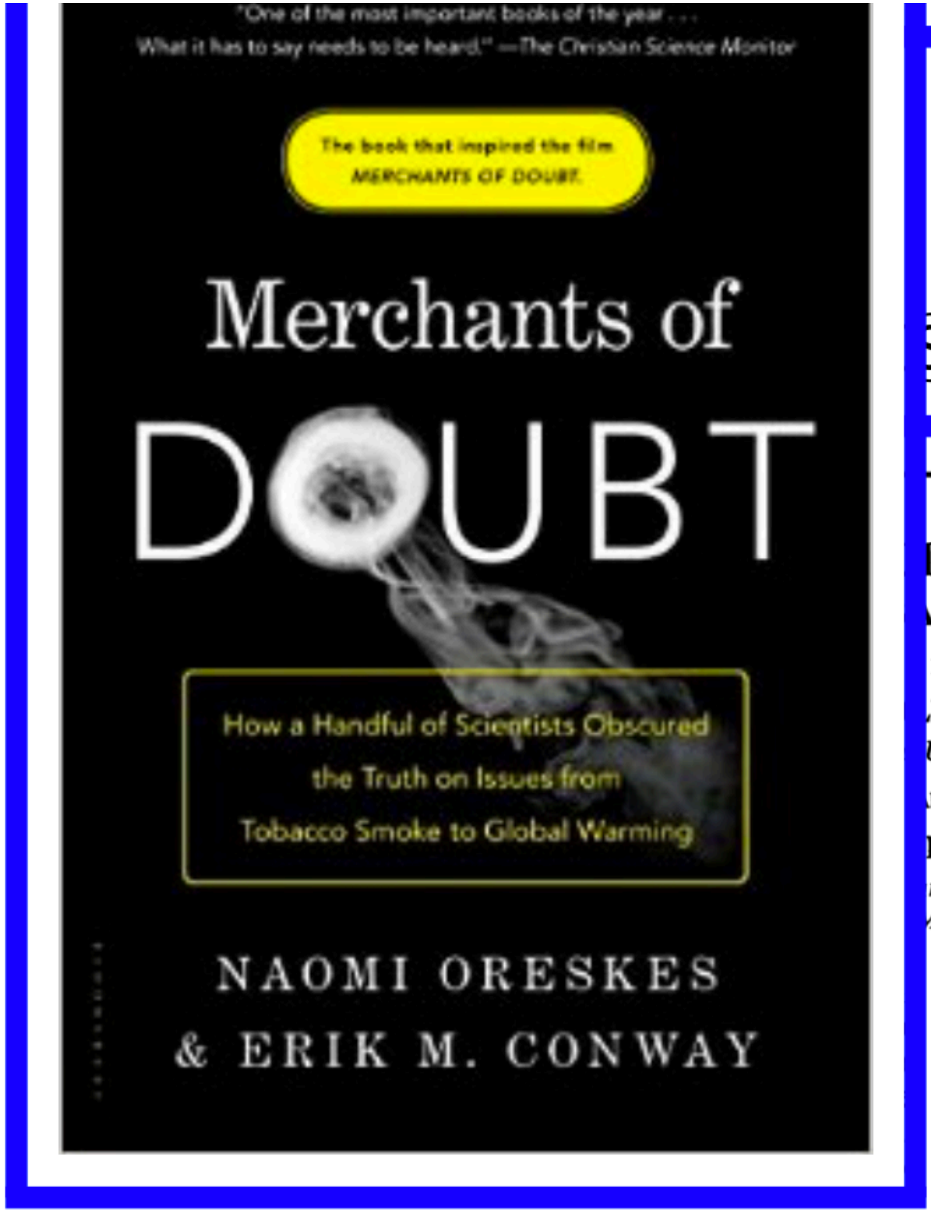

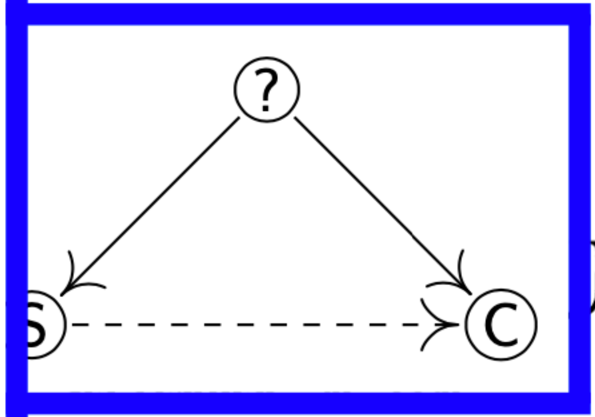

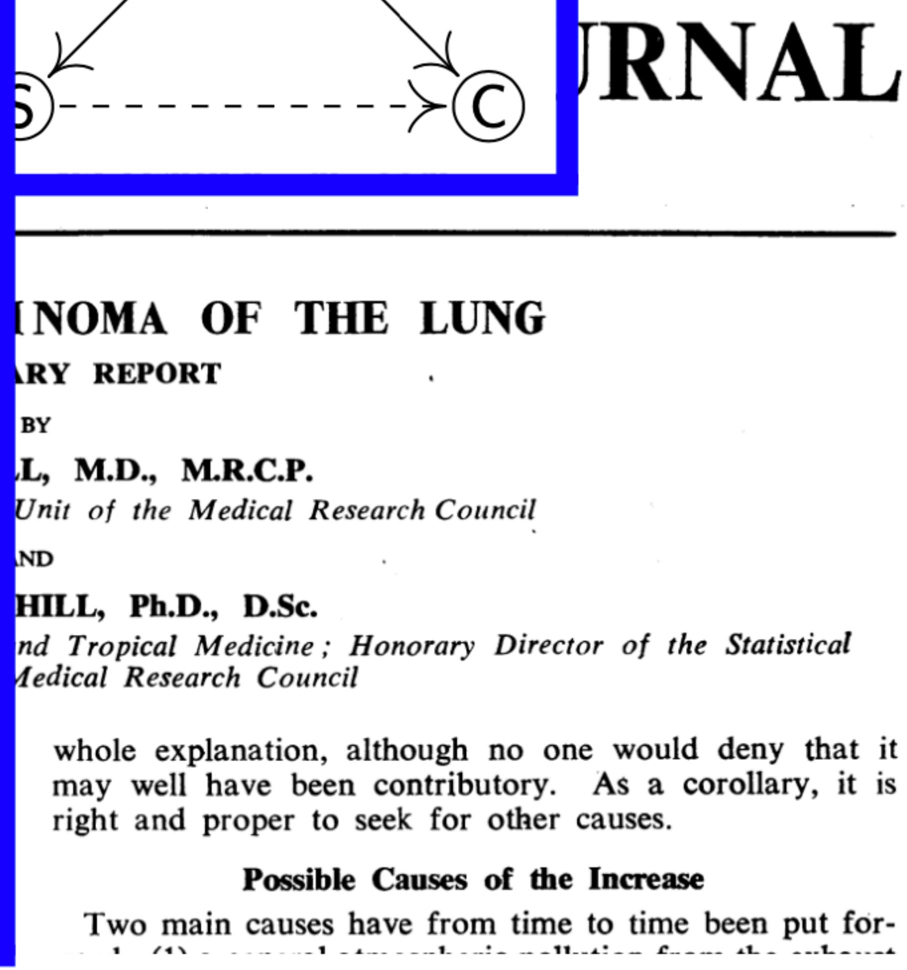

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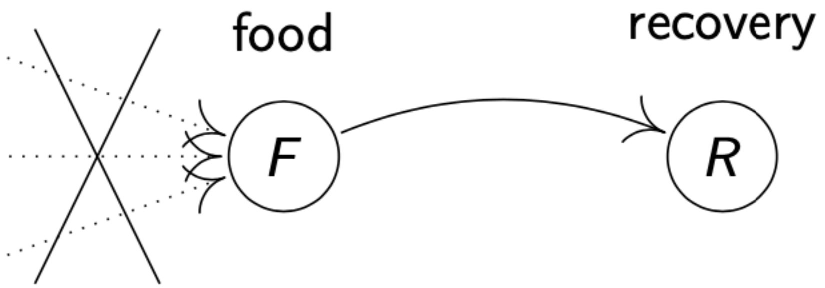

Then: $P_{do(F:=f)}(R) = P(R|F=f)$

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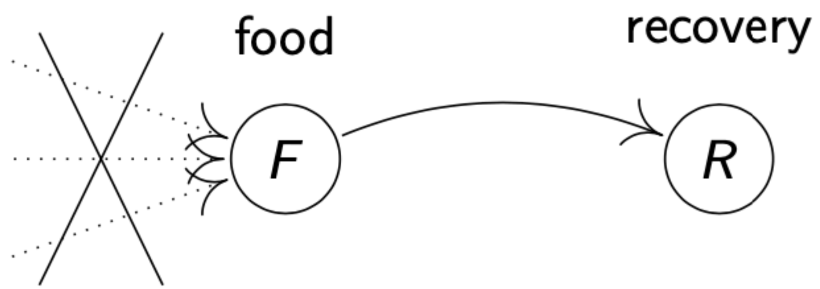

Then: $P_{do(F:=f)}(R) = P(R|F = f)$

"On the 20th of May 1747, I selected twelve patients in the scurvy, on board the Salisbury at sea. [...] Two were ordered each a quart of cyder a day. Two others took twenty-five drops of elixir vitriol three times a day [...] Two others took two spoonfuls of vinegar three times a day [...] Two of the worst patients were put on a course of sea-water [...] Two others had each two oranges and one lemon given them every day [...] The two remaining patients, took [...] an electary recommended by a hospital surgeon [...] The consequence was, that the most sudden and visible good effects were perceived from the use of oranges and lemons; one of those who had taken them, being at the end of six days fit for duty."

Definition (Equivalence of causal models)

Two models are called

$$\{\textbf{probabilistically} \,/\, \textbf{interventionally}\} \;\; \textbf{equivalent}$$

if they entail the same

$$\{\text{observational} \,/\, \text{observational \& interventional}\}$$

distributions. Here, it suffices to consider interventions that set a variable X_j to a fully supported $\tilde{N}_j$ ("randomized experiments").

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Given an SCM, there is a total causal effect from X to Y if one of the following equivalent statements is satisfied:

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(i) $X \not\perp\!\!\!\perp Y \quad$ in $P_{\mathrm{do}\,X:=\tilde{N}_X}$ for some random variable $\tilde{N}_X$.

(ii) There are $x^{\triangle}$ and $x^{\square}$, such that $P^{Y}_{\mathrm{do}\,X:=x^{\triangle}} \neq P^{Y}_{\mathrm{do}\,X:=x^{\square}}$.

(iii) There is $x^{\triangle}$, such that $P^{Y}_{\mathrm{do}\,X:=x^{\triangle}} \neq P^{Y}$.

(iv) $X \not\perp\!\!\!\perp Y$ in $P^{X,Y}_{\mathrm{do}\,X:=\tilde{N}_X}$ for any $\tilde{N}_X$ whose distribution has full support.

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Causal strength?

Instrumental Variables?

$$\begin{tikzcd}
& & H \arrow[dl, "\beta"'] \arrow[dr, "\delta"] & \\
I \arrow[r, "\delta"] & X \arrow[rr, "\alpha"] & & Y
\end{tikzcd}$$

H - not known factor

I doesn't cause Y

$$Y = \alpha X + \gamma H + N_Y$$

$$X = \beta H + \delta I + N_X$$

$$Y = (\alpha\beta + \gamma) H + \alpha\delta I + \alpha N_x + N_Y$$

Two stages least squares:

1) $X \sim \delta I$ 2) We get estimate of $\widehat{\delta I}$

3) $Y \sim \alpha * \widehat{\delta I}$

$$
\begin{tikzcd}
? \arrow[rr, dashed, squiggly] & & H \arrow[dl, "\beta"'] \arrow[dr, "\gamma"] & \\
I_1 \arrow[u, dash] \arrow[r, "\delta_1"] & X \arrow[rr, "\alpha"] & & Y \\
\vdots & & & \\
I_k \arrow[uur, "\delta_k"'] & & &
\end{tikzcd}
$$

$$\dim(I) = \dim(X)!$$

Counterfactuals

Given that Hilary Clinton did not win the 2016 presidential election, and given that she did not visit Michigan 3 days before the election, and given everything else we know about the circumstances of the election, what can we say about the probability of Hilary Clinton winning the election, had she visited Michigan 3 days before the election?

Let's try to unpack this. We are are interested in the probability that:

she hypothetically wins the election
conditionied on four sets of things:

she lost the election
she did not visit Michigan
any other relevant an observable facts
she hypothetically visits Michigan
It's a weird beast: you're simultaneously conditioning on her visiting Michigan and not visiting Michigan. And you're interested in the probability of her winning the election given that she did not. WHAT?

Why would quantifying this probability be useful? Mainly for credit assignment. We want to know why she lost the election, and to what degree the loss can be attributed to her failure to visit Michigan three days before the election. Quantifying this is useful, it can help political advisors make better decisions next time.

$SCM(\underline{S}, P^{\underline{N}})$

We have observed something

A counterfactual SCM is obtained from a given observation $\underline{X} = \underline{x}$ by conditioning the noise distribution

$$\widetilde{SCM}(S, P^{N|X=x})$$

What would have happened if ...?

"do" statements in this new SCM^{*}

Ex: $T := N_T$

$R := T \cdot N_R + (1-T)(1-N_R)$

$N_R \sim Ber(0.99)$

if treatment = yes, $N_R \stackrel{?}{=} 1 \Rightarrow R = 1$

Specific person: $N_T = 1$, $N_R = 0 \longrightarrow$ properties of the person

$(T=1, R=0) \Rightarrow$ the person now have a new

the person SCM: $T = 1$

$R = 1 - T$

$$P^{c \mid T=1, R=0}_{do(T:=0)}(R=1) = 1$$

Tom would have recovered had he received no treatment

Personal SCM:

$$T = 1$$
$$R = 1 - T$$

Shall the doctor pay? ;-)

Summary Part I:

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- But often, we are interested in a system's behaviour under intervention.
- SCMs entail graphs, obs. distr., interventions and counterfactuals.

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\begin{aligned}
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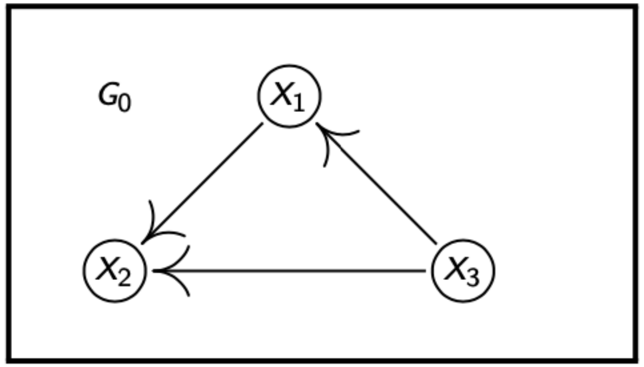
- N_i jointly independent
- G_0 has no cycles



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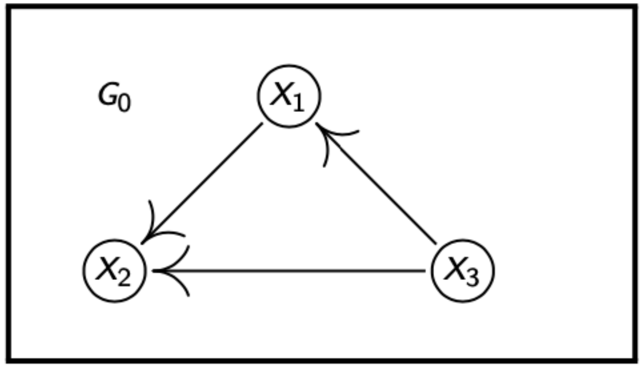

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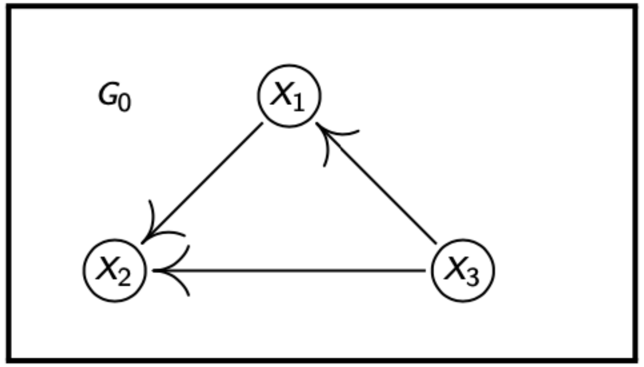

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- SCM + observational distribution $\rightsquigarrow$ counterfactuals
- Adjusting allows to compute interventions when there are (some) hidden variables