Causal Inference & Paradoxes

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Outline of the talk

- Bayesian networks: From probability to causality
- Manipulation theorem to estimate the **effect of external interventions**
- **Confounding**: fundamental impediments to the elucidation of causal inferences from observational data
- Elucidation of some well-known controversies :
  - The selection bias or **Berkson's paradox** (1946),
  - The **birth-weight paradox** (1967)
  - The **Simpson's paradox** (1899)
  - The old debate on the relation between **smoking and lung cancer** (1964),
  - **Sex discrimination**: The « reverse regression controversy » between sex and salary which occupied the social science in the 1970s
- Rules of « **do calculus** »
- Case study: **effect of the pesticides on agricultural yields**
- Unbiased estimates despite **selection bias** and **missing data**

Cause-effect relationships

- The central aim of many studies in the physical, behavioral, social, and biological sciences is the **elucidation of cause-effect relationships** among variables or events, e.g., risk factor exposure on disease occurrence, advertising campaign on benefits, treatment on recovery rate, etc.

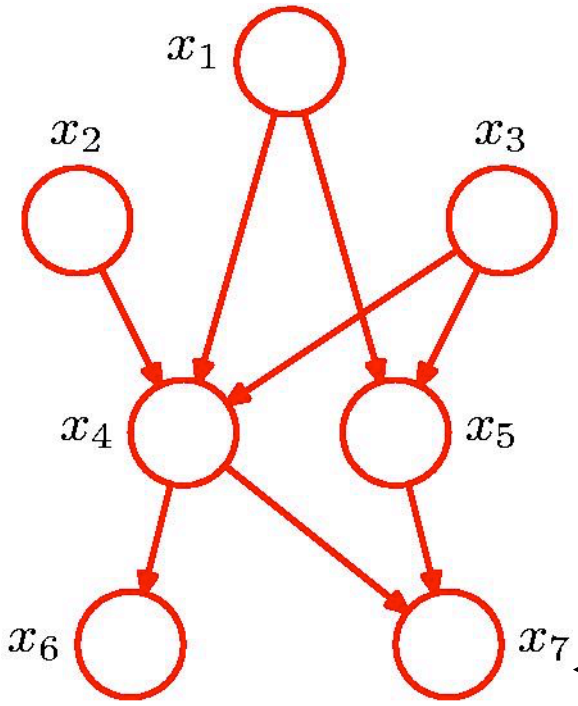
- However, the appropriate **methodology for extracting such relationships** from data has been fiercely debated.

- **Graphical models** provide clear semantics for causal claims, and non-trivial causal phenomena, **paradoxes and controversies** in causal analysis that long were regarded as **metaphysical** can now be understood, exemplified, analyzed and solved using **elementary mathematics**.

- Most of the material presented here is borrowed from **Judea Pearl'**s books and papers.

Bayesian Networks

$$p(x_1,\ldots,x_7) = p(x_1)p(x_2)p(x_3)p(x_4|x_1,x_2,x_3)$$
$$p(x_5|x_1,x_3)p(x_6|x_4)p(x_7|x_4,x_5)$$



General Factorization : $p(\mathbf{x}) = \prod_{k=1}^{K} p(x_k|\mathrm{pa}_k)$

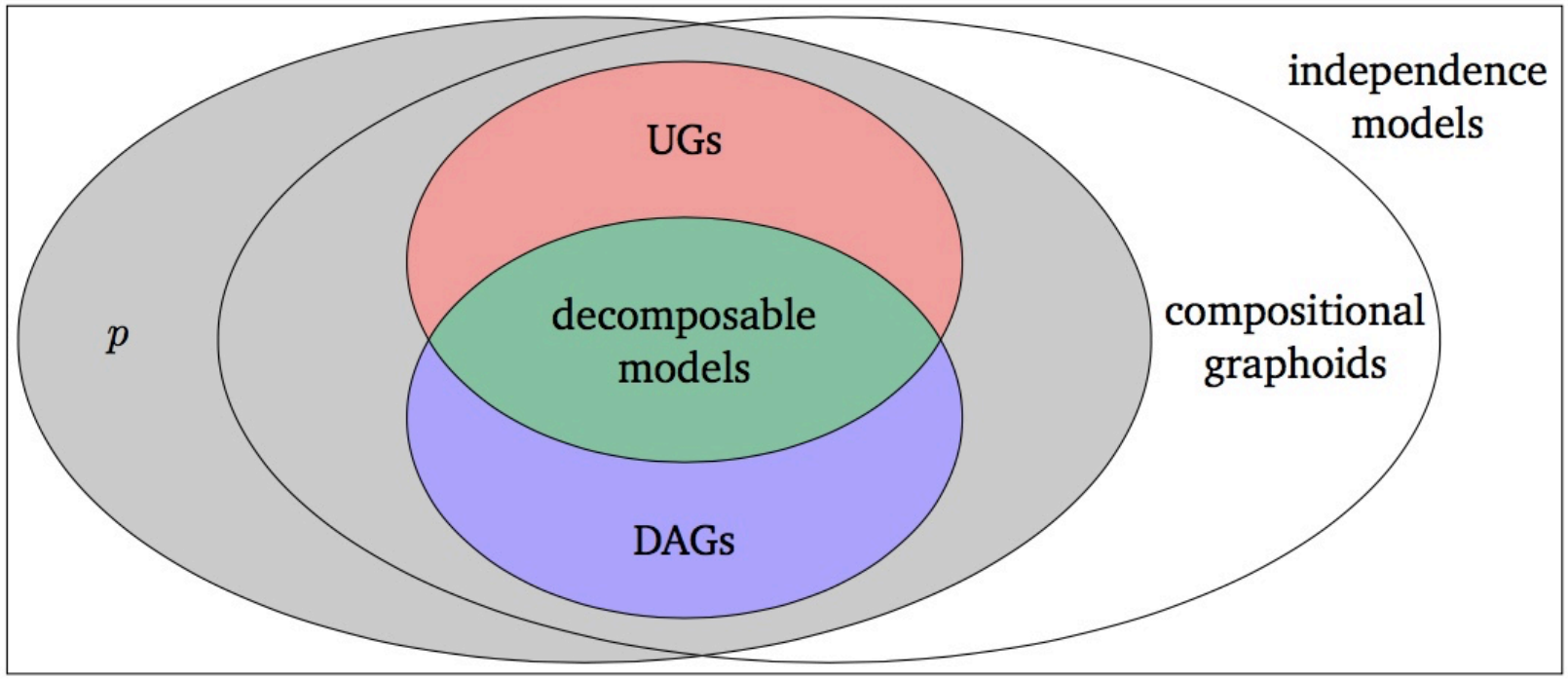
Conditional distribution for (binary) node x_7 :

x_4	x_5	1	0
0	0	0.4	0.6
0	1	0.1	0.9
1	0	0.7	0.3
1	1	0.6	0.4

Corollary (**Markov condition)** : every node given its parents is independent on its non-descendants nodes. Other independencies are entailed (**d-separation** criterion).

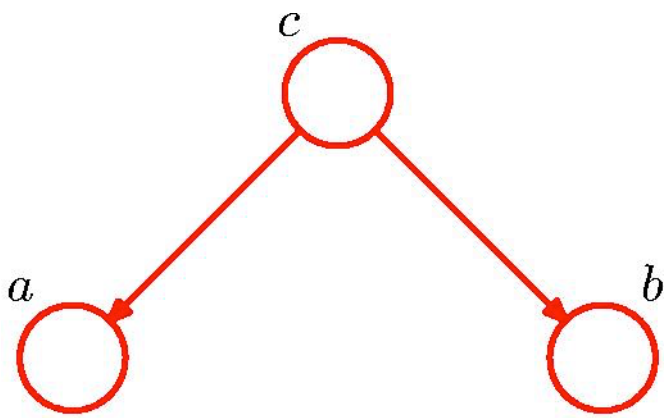
Illustration from **Christopher Bishop's** book : "*Pattern recognition* and *machine learning*".

Independence models



Overlapping between probabilistic independence models (p), independence models based on u-separation (UG-faithful), and d-separation (DAG-faithful).

Conditional Independence: Example 1

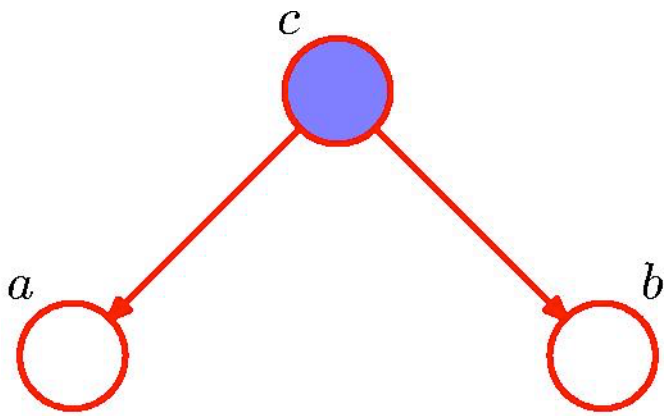


$$p(a, b, c) = p(a|c)p(b|c)p(c)$$

$$p(a, b) = \sum_c p(a|c)p(b|c)p(c)$$

$$a \not\perp\!\!\!\perp b \mid \emptyset$$

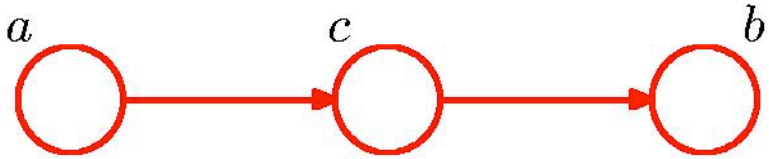
Conditional Independence: Example 1



$$p(a,b|c) = \frac{p(a,b,c)}{p(c)} = p(a|c)p(b|c)$$

$$a \perp\!\!\!\perp b \mid c$$

Conditional Independence: Example 2

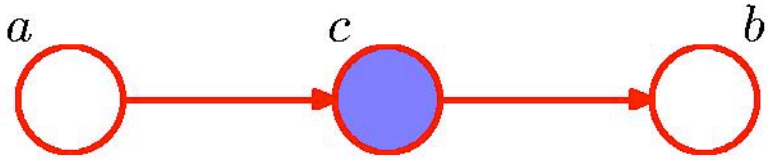


$$p(a, b, c) = p(a)p(c|a)p(b|c)$$

$$p(a, b) = p(a) \sum_c p(c|a)p(b|c) = p(a)p(b|a)$$

$$a \not\perp\!\!\!\perp b \mid \emptyset$$

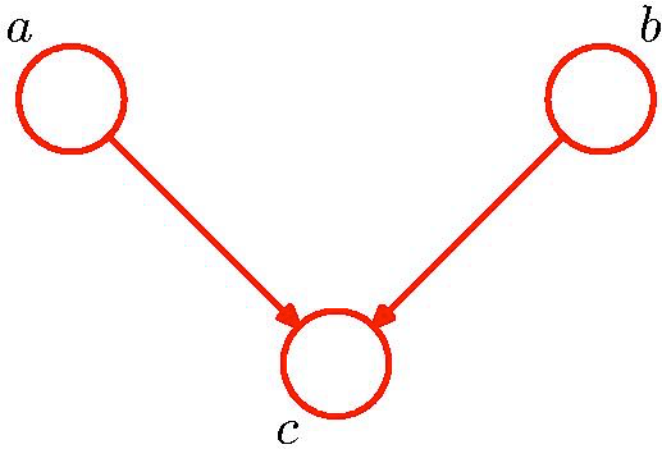
Conditional Independence: Example 2



$$
\begin{aligned}
p(a,b|c) &= \frac{p(a,b,c)}{p(c)} \\
&= \frac{p(a)p(c|a)p(b|c)}{p(c)} \\
&= p(a|c)p(b|c)
\end{aligned}
$$

$$a \perp\!\!\!\perp b \mid c$$

Conditional Independence: Example 3



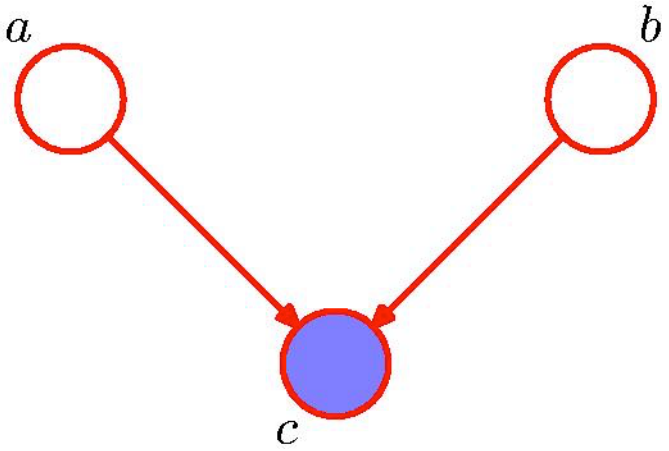
$$p(a, b, c) = p(a)p(b)p(c|a, b)$$

$$p(a, b) = p(a)p(b)$$

$$a \perp\!\!\!\perp b \mid \emptyset$$

Note: this is the opposite of Example 1, with c unobserved.

Conditional Independence: Example 3



$$p(a,b|c) = \frac{p(a,b,c)}{p(c)}$$

$$= \frac{p(a)p(b)p(c|a,b)}{p(c)}$$

$$a \not\perp\!\!\!\perp b \mid c$$

Compared to the previous examples, the opposite is observed:
Two **independent variables become dependent** given a third variable!

Limits of Bayesian Networks

- Two given DAGs are **observationally equivalent** if *every* probability distribution that is compatible (or faithful) with one of the DAGs is also compatible with the other (same conditional independences encoded).

- **Theorem**: Two DAGs are observationally equivalent if and only if they have the same skeletons and the **same sets of v-structures**, that is, two converging arrows whose tails are not connected by an arrow.

- Observational equivalence places a limit on our ability to infer directionality from probabilities alone.

- Networks that are observationally equivalent **cannot be distinguished without** resorting to **manipulative experimentation** or **human knowledge**

Causal Bayesian Networks

Graphs as Models of Interventions

- **Causal models**, unlike probabilistic models, can serve to **predict the effect of interventions**. This added feature requires that the joint distribution *P* be supplemented with a **causal diagram** - that is, a DAG that identifies causal connections.
- The causal diagram may represent the i**nvestigator's understanding** of the major causal influences among measurable quantities in the domain.

- Each child-parent family in a DAG *G* represents a deterministic function:

$$x_i = f_i(pa_i, \epsilon_i), \quad i = 1, \ldots, n$$

  where ***pa***$_i$ are the parents of variable $\mathbf{x}_i$ in *G*; the ϵ_i (*i=1,…,n*) are mutually **independent**, arbitrarily distributed random disturbances.
- The equality signs in structural equations convey the **asymmetrical relation** of "is determined by“.

Causal Bayesian Networks

$$p(x_1, \ldots, x_7) = p(x_1)p(x_2)p(x_3)p(x_4|x_1, x_2, x_3)$$
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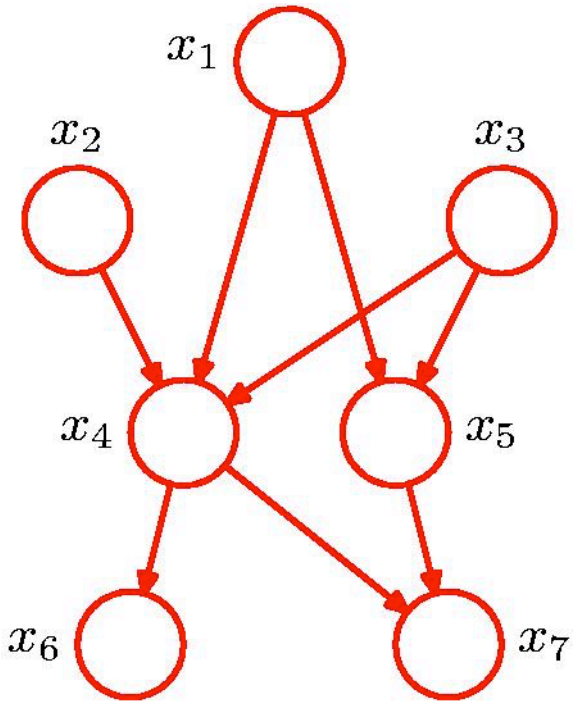


General Factorization

$$p(\mathbf{x}) = \prod_{k=1}^{K} p(x_k|\mathrm{pa}_k)$$

Now **supplemented** with causal assumptions

$$x_i = f_i(pa_i, \epsilon_i), \quad i = 1, \ldots, n$$

Finding causal relationships

- For finding causal relationships, the gold standard are **randomized controlled trials** initially developed in the context of agricultural research (Fisher, 1926).
- Problem: Not always feasible for ethical, financial or other reasons.

We are left with two problems:

- **Problem 1 (Causal Structure)**: Given observational data, find the DAG representing the causal structure, or, if this is not possible, give a class of DAGs to which the true DAG belongs.

- **Problem 2 (Interventional Distribution)**: Given observational data, find the interventional distribution of a random variable *Y* after some other random variable *X* was set to a certain value by external intervention to make quantitative predictions on the effect of interventions.

Manipulation theorem

- The **manipulation theorem** (Spirtes et al. 1993) states that given an external intervention on a variable *X* in a causal graph, we can derive the posterior probability distribution over the entire graph by simply modifying the conditional probability distribution of *X*.

- Intervention amounts to **removing all edges that are coming into** *X*. Nothing else in the graph needs to be modified, as the causal structure of the system remains unchanged.

- Thus, intervention can be expressed in a **simple truncated factorization** formula.

The do(.) operator

- Interventions are defined through a new mathematical operator called **do(*X*=*x*)**, which simulates physical interventions by deleting the probability factor corresponding to variable *X* in the joint factorization, while keeping the rest unchanged elsewhere with *X* fixed to *x*.

- The causal effect of *X* on *Y* is denoted *P*(*y*|**do(*X*=*x*)**). It is termed an ***interventional*** **distribution** and should not be confused from the ***observational*** **distribution** *P*(*y*|*x*).

- Interventions can be expressed as a simple ***truncated*** **factorization** formula:

$$P(x_1, \ldots, x_n \mid \mathbf{do}(x_i = x_i')) = \begin{cases} \prod_{j \neq i} P(x_j \mid \mathbf{pa_j}) & \text{if } x_i = x_i' \\ 0 & \text{if } x_i \neq x_i' \end{cases}$$

The do(.) operator

$$P(x_1, \ldots, x_n \mid \mathbf{do}(x_i = x_i')) = \begin{cases} \prod_{j \neq i} P(x_j \mid \mathbf{pa_j}) & \text{if } x_i = x_i' \\ 0 & \text{if } x_i \neq x_i' \end{cases}$$

Can be rewritten as:

$$P(x_1, \ldots, x_n \mid \mathbf{do}(x_i = x_i')) = \begin{cases} P(x_1, \ldots, x_n \mid x_i, \mathbf{pa}_j) P(\mathbf{pa}_j) & \text{if } x_i = x_i' \\ 0 & \text{if } x_i \neq x_i' \end{cases}$$

Summing over all variables except x_i and y leads to the result called **adjustment for direct causes:**

$$P(y \mid \mathbf{do}(x_i = x_i')) = \sum_{\mathbf{pa}_i} P(y \mid x_i', \mathbf{pa}_i) P(\mathbf{pa}_i)$$

In compact form:

$$P(y \mid \mathbf{do}(x)) = \sum_{\mathbf{pa}_x} P(y \mid x, \mathbf{pa}_x) P(\mathbf{pa}_x)$$

Controlling confounding biais

$$P(y \mid \mathbf{do}(x)) = \sum_{\mathbf{pa}_x} P(y \mid x, \mathbf{pa}_x) P(\mathbf{pa}_x)$$

- We ***adjust*** our measurements for possible variations of the **parents of** *X* in the causal DAG *G*, they are acting as "covariates" or « **confounders** ».
- **Adjustment for the direct parents** amounts to **partitioning the population** into groups that are homogeneous relative to $\boldsymbol{pa}_x$ assessing the effect of *X* on *Y* in each homogeneous group, and then averaging the results.
- This expression requires all the parents to be *observed.* Are other **variables appropriate for adjustment**?
- What criterion should one use to decide **which variables are appropriate for adjustment**?

Back-Door adjustment

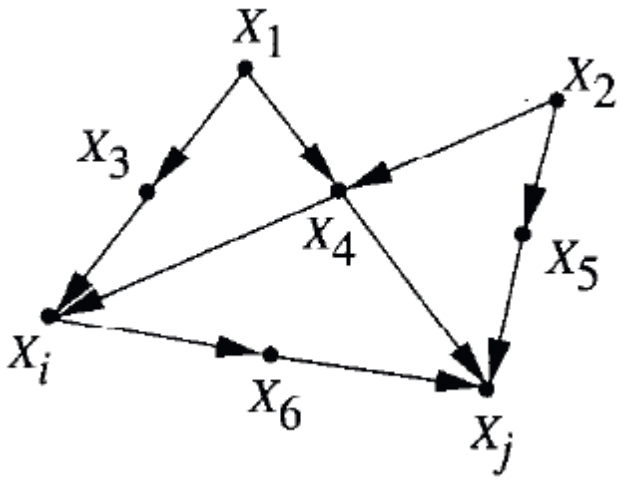
More generally, a set of variables ***Z*** satisfies the **back-door criterion** relative to (*X*,*Y*) in a DAG G iff,

- No node in **Z** is a descendant of X, and
- **Z** blocks every path between X and Y that contains an arrow into X.

Theorem – *If a set of variables **Z** satisfies the back-door criterion relative to (X,Y), then the causal effect of X on Y is **identifiable** and is given by the formula*,

$$P(y \mid do(x)) = \sum_{\mathbf{z}} P(y \mid x, \mathbf{z}) P(\mathbf{z})$$

Example:



- The sets **Z** =$\{X_3,X_4\}$ and **Z** =$\{X_4,X_5\}$ meet the back-door criterion relative to **(**X_i,X_j**)**

- But **Z** =$\{X_4\}$ does not !

Paradoxes & Controversies

Berkson’s paradox

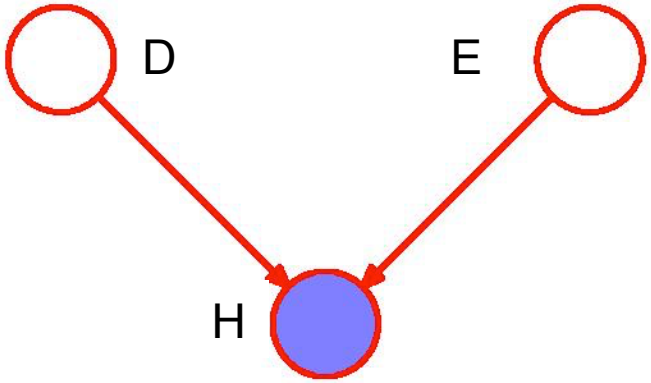
- Berkson's paradox is a result in **conditional probability** (not related de causality) which is counterintuitive for some people: given **two independent events**, if you only consider **outcomes where at least one event occurs, then they become negatively dependent.**

- **Example**: Berkson's original illustration involves a retrospective study examining a risk factor for a disease in a statistical sample. Because samples are taken from a hospital in-patient population, rather than from the general public, this can result in a spurious negative association between the disease and the risk factor

Berkson, Joseph. "Limitations of the Application of Fourfold Table Analysis to Hospital Data". Biometrics Bulletin. 1946

Berkson's paradox

	E^+		E^-	
	D^+	D^-	D^+	D^-
H^+	800	600	400	200
H^-	200	400	600	800



- The prevalence of the disease (***D***) is 50% among exposed (***E***) and unexposed.
- 70% are hospitalized (***H***) among exposed patients (30% among non exposed)
- 60% are hospitalized among diseased patients (40% among non diseased).

- **Within those hospitalized**, the prevalence of the disease is 57% among exposed and 66% among unexposed patients.

Birth weight paradox

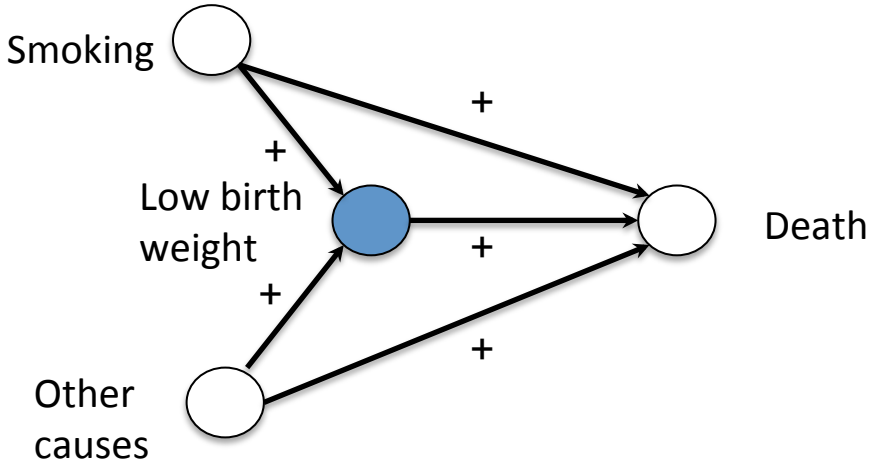
- The birth-weight paradox concerns the relationship between the birth weight and mortality. **Children of smoking mothers are more likely to be of low birth weight** and **low birth weight children have a significantly higher mortality rate than others** (it is in fact 100-fold higher)

- **Contrary to expectations**, low birth weight babies of smoking mothers have a lower child mortality than low birth weight babies of nonsmokers. **Having a smoking mother might be beneficial to one's health!**

- Like the Berkson's paradox, it is counterintuitive as it involves two independent events that become negatively dependent, having observed a third event.

Hernández-Díaz et al. "The birth weight paradox uncovered?" Am J Epidemiol 2006
Wilcox A. "On the importance—and the unimportance—of birth weight". Int J Epidemiol 2001.

Birth weight paradox



- Smoking may be harmful in that it contributes to low birth weight, but other causes (not measured) of low birth weight are generally more harmful.
- Consider a low weight baby, **finding that the mother smokes reduces the likelihood that those other causes are present**.

Simpson's paradox

C : taking a certain drug or treatment
E : recovery
F : gender

Under a causal interpretation the drug seems to be **harmful** to both males and females yet **beneficial** to the population as a whole !

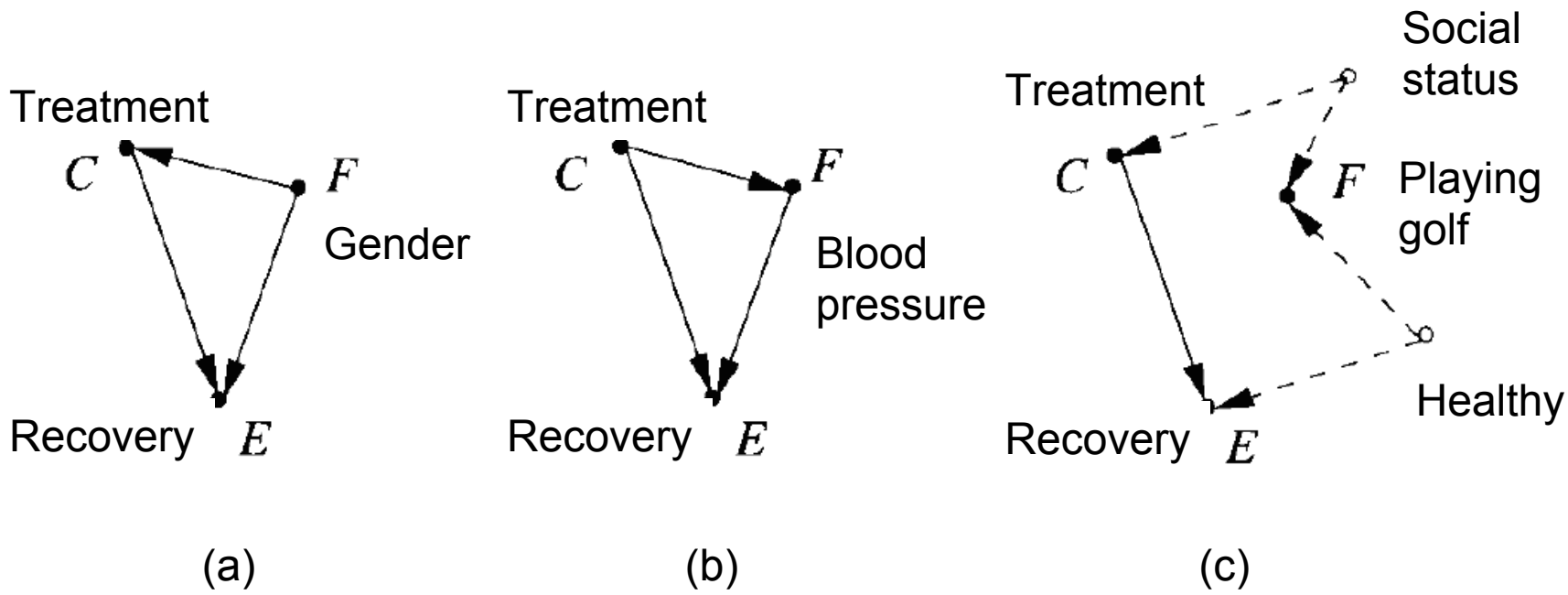
Males	E	$\bar{E}$	Tot.	Recovery rate
Drug (C)	18	12	30	60%
No Drug ($\bar{C}$)	7	3	10	70%
	25	15	40	

Females	E	$\bar{E}$	Tot.	Recovery rate
Drug (C)	2	8	10	20%
No Drug ($\bar{C}$)	9	21	30	30%
	11	29	40	

Combined	E	$\bar{E}$	Tot.	Recovery rate
Drug (C)	20	20	40	50%
No Drug ($\bar{C}$)	16	24	40	40%
	36	44	80	

Simpson's paradox

Three causal models capable of generating the data Model
(a) dictates use of the **gender-specific tables**,
whereas (b) and (c) dictates use of the **combined table.**



Simpson's paradox

As **F** connotes gender, the correct answer is the gender specific table, i.e.

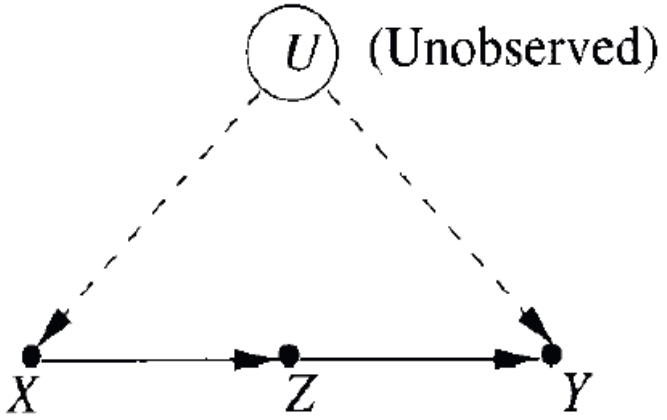
$$P(y|do(x)) = \sum_z P(y|x,z)\, P(z)$$

- **Conclusion**: every question related to the effect of actions must be decided by causal considerations; statistical information alone is insufficient.
- The question of choosing the correct table on which to base our decision is a special case of the **covariate selection problem.**

Front-Door adjustment

A set of variables *Z* is said to satisfy the **front-door criterion** relative to (*X*, *Y*) if

- Z intercepts all directed paths from *X* to *Y*;
- there is no back-door path from *X* to *Z*;
- all back-door paths from *Z* to *Y* are blocked by *X*.



Theorem : If *Z* satisfies the front-door criterion relative to *(X , Y)* and if *P(x,z)* > 0, then the causal effect of X on Y is **identifiable** and is given by the formula:

$$P(y|do(x)) = \sum_{z} P(z|x) \sum_{x'} P(y|z,x')\,P(x')$$

If *Z* were ***not*** **observed**, the causal effect of *X* on *Y* would ***not*** **be identifiable!**

Smoking and Lung Cancer

	Group Type	$P(x, z)$ Group Size (% of Population)	$P(Y = 1 \mid x, z)$ % of Cancer Cases in Group
$X = 0, Z = 0$	Nonsmokers, No Tar	47.5	10
$X = 1, Z = 0$	Smokers, No Tar	2.5	90
$X = 0, Z = 1$	Nonsmokers, Tar	2.5	5
$X = 1, Z = 1$	Smokers, Tar	47.5	85

- Old debate on the relation between **smoking**, X, and **lung cancer**, Y**.**
- If we ban smoking, will the rate of cancer cases be roughly the same as the one we find today among non smokers in the population ?
- **Controlled experiments** could answer the question but they are **illegal** to conduct.

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The tobacco industry has managed to forestall antismoking legislation (1964) by arguing that the observed correlation between smoking and lung cancer could be explained by some sort of **carcinogenic genotype**, *U* (unknown), that involves **inborn craving for nicotine**.

Smoking and Cancer

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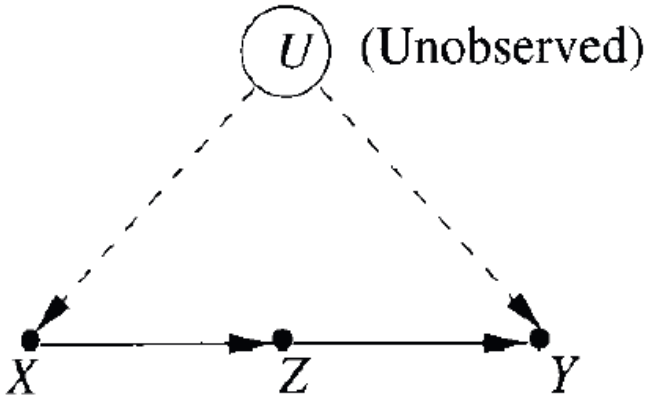


$$P(y|do(x)) = \sum_{z} P(z|x) \sum_{x'} P(y|z,x')\, P(x')$$

Numerical application

- **Crude analysis**:

$$P(X=1)=0.5; P(Z=1)=0.5; P(Y=1)=0.475$$
$$P(Y=1 \mid X=0)=(0.1\times 0.475+0.05\times 0.025)/0.5=0.0975$$
$$P(Y=1 \mid X=1)=(0.9\times 0.025+0.85\times 0.475)/0.5=0.8525$$

- These results seem to prove that **smoking is a major contributor to lung cancer.**
- However, the tobacco industry might argue that the table tells a different story - that smoking actually decreases one's risk of lung cancer…

Numerical application

$$\boldsymbol{P(y|do(x)) = \sum_{z} P(z|x) \sum_{x'} P(y|z,x')\, P(x')}$$

$$\begin{aligned}
P(Y=1 \mid do(X=1) &= 0.05 \times (0.1 \times 0.5 + 0.9 \times 0.5) \\
&\quad +0.95 \times (0.05 \times 0.5 + 0.85 \times 0.5) \\
&= 0.4525 \\
P(Y=1 \mid do(X=0) &= 0.95 \times (0.1 \times 0.5 + 0.9 \times 0.5) \\
&\quad +0.05 \times (0.05 \times 0.5 + 0.85 \times 0.5) \\
&= 0.4975
\end{aligned}$$

Contrary to expectation, the data prove **smoking** to be somewhat **beneficial to one's health** !

Discrimination controversy

- Another example involves a controversy called « reverse regression », which occupied the social science literature in the 1970s.
- Should we**,** in **salary discrimination cases**, compare **salaries of equally qualified men and women** or instead compare **qualifications of equally paid men and women**?
- Remarkably, the **two choices may lead to opposite conclusions**. It turns out that men earns a higher salary than equally qualified women and, ***simultaneously,*** men are more qualified than equally paid women.
- The moral is that all conclusions are extremely sensitive to which variables we choose to hold constant when we are comparing groups.

Discrimination controversy

- **Men earns a higher salary than equally qualified women** reads:

$$\sum_Q P(S|Male, Q)P(Q) > \sum_Q P(S|Female, Q)P(Q)$$

- **Men are more qualified than equally paid women** reads:

$$\sum_S P(Q|Male, S)P(S) > \sum_S P(Q|Female, S)P(S)$$

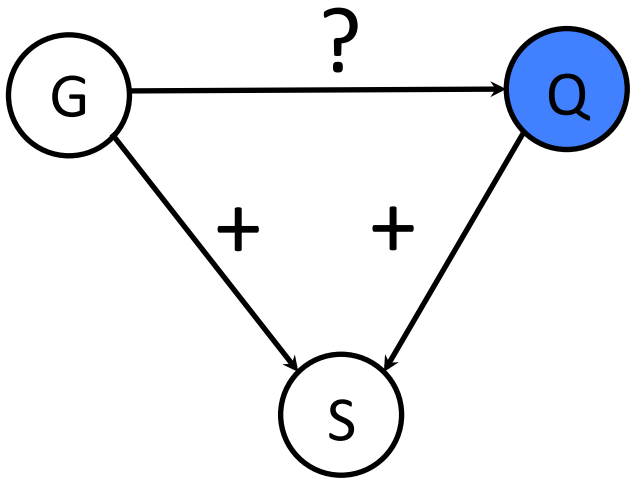
- The question we seek to answer: **does sex *directly* influence salary ?** Which is the court definition of discrimination, and reads:

$$P(S|\mathbf{do}(Male)) > P(S|\mathbf{do}(Female))$$

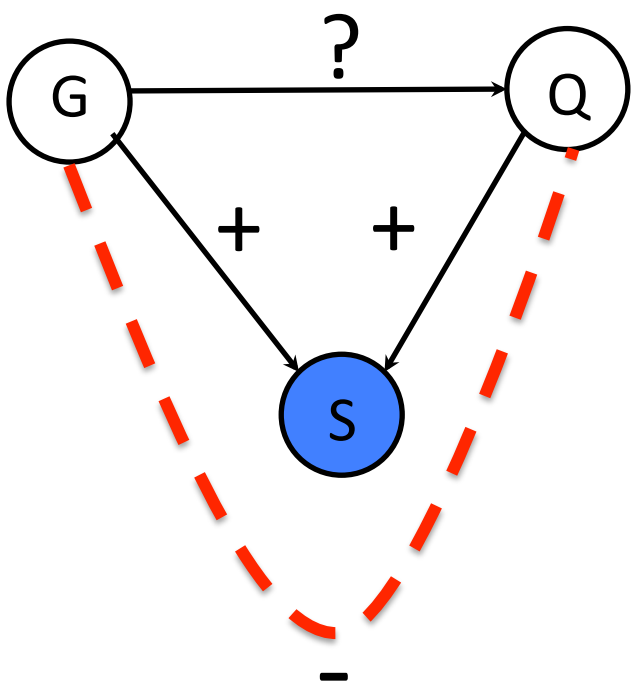
Discrimination controversy

Let G=1 for men, G=1 and S=1 for high qualification and salary. Suppose two direct effects are positive (hence sex discrimination on salary). Conditioned on *S*, *G* and *Q* become negatively correlated via the open path in dotted lines.

Men earns a higher salary than equally qualified women



Men are more qualified than equally paid women



The Rules of do-calculus

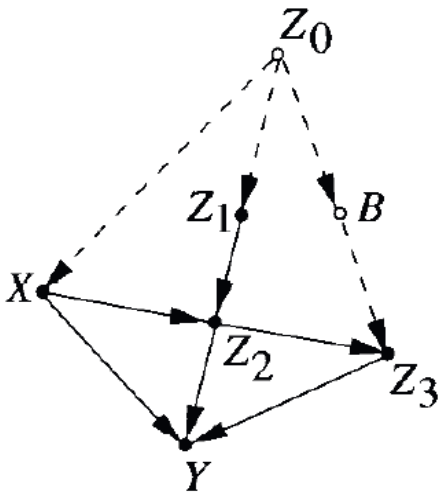
- When a query is given in the form of a do-expression, for example *P(y|**do(x)**,z),* its **identifiability** can be decided systematically using an **algebraic procedure** known as the do-calculus.

- The **do-calculus** was developed by **J. Pearl in 1995** to facilitate the identification of causal effects in non-parametric models.

- It consists of **three inference rules** that permits to map interventional and observational distributions whenever certain conditions hold in the causal diagram G.

- The do-calculus was shown to be *complete* (Tian and Pearl 2002a; Huang and Valtorta 2006; Shpitser and Pearl 2006; Bareinboim and Pearl 2012a).

Causal graphs: illustration

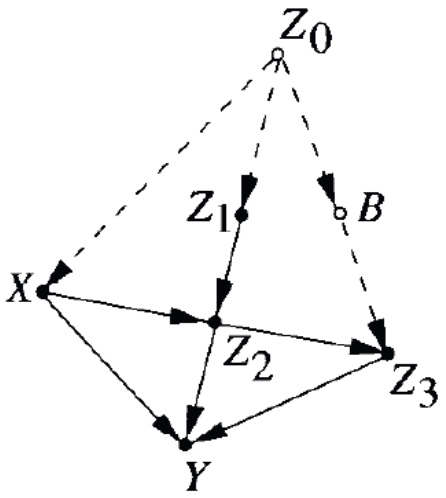


- We wish to assess the **total effect of the fumigants *X* on yields *Y*.**
- The causal diagram represents the investigator's understanding of the major causal influences among measurable quantities in the domain.

- Z_1, Z_2, Z_3 represent the **eelworm population** *before* treatment, *after* treatment, and *at the end* of the season, respectively.
- Z_0 represents *last year's* eelworm population.
- *B* is the population of birds and other predators.

Unmeasured quantities are designated by hollow circles and dashed lines.

The Rules of do-calculus



- **Using the do-calculus,** one can establish that the total effect of *X* on *Y* can be estimated consistently from the observed distribution of *X*, Z_1, Z_2, Z_3, and *Y*.
- These conclusions are obtained by **performing a sequence of symbolic derivations** (the 3 inference rules).

$$P(y \mid \mathbf{do}(x)) = \sum_{z_1,z_2,z_3} P(y \mid z_2, z_3, x) P(z_2 \mid z_1, x)$$

$$\times \sum_{x'} P(z_3 \mid z_1, z_2, x') P(z_1, x')$$

Confounding & Selection bias

Confounding & Selection bias

- The biases arising from confounding and selection are fundamentally different, though both constitute threats to the validity of causal inferences.
  - The **confounding bias** is the result of treatment *X* and outcome *Y* being affected by common ancestral variables,
  - The **selection bias** is due to treatment *X* or outcome *Y* (or ancestors) affecting the inclusion of the subject in the sample.

- In both cases, we have extraneous "flow" of information between treatment and outcome, which falls under the rubric of "spurious correlation," since it is not what we seek to estimate.

- What are the conditions for **recoverability of interventional distributions** for when selection and confounding biases are both present?

Confounding with latent variables

- Some relevant confounders are difficult to measure in many real-world applications (e.g., intention, mood, DNA mutation), which leads to the need of modelling explicitly **latent variables** that affect more than one observed variable in the system (Semi- Markovian models).

- In such models, identifiability is *not always achievable*.

- **Causal Effects Identifiability**: Let be ***V*** the set of *observable* variables, ***U*** is the set of *unobservable* variables. The causal effect of an action, **do**$(X = x)$ is said to be identifiable from P in G if $P(y|\mathbf{do}(x))$ is uniquely computable from $P(v)$.

- The evaluation of identifiability goes through a **non-trivial algebraic process**, namely the *do-calculus*.

Confounding : risks and pitfalls

- Researchers must weigh the benefit of reducing confounding bias carried by those covariates against the risk of **amplifying residual bias** carried by **unmeasured confounders.**

- According to Judea Pearl, epidemiologists often adjust for wrong sets of covariate (usually *Sex* and *Age* but other covariates are missing*)* .

- Is the prevailing practice in epidemiology misguided?

Selection bias

- Another major challenge that needs to be addressed when evaluating the effect of interventions is the problem of selection bias, caused by **preferential exclusion of samples** from the data.

- Selection bias is a **major obstacle to valid causal and statistical inferences**; it can hardly be detected in either experimental or observational studies.

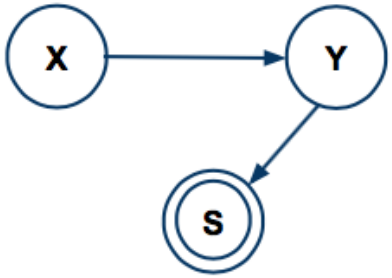
- **Example**: in a typical study of the effect of training program on earnings, subjects achieving higher incomes tend to report their earnings more frequently than those who earn less.

Selection bias

●To illuminate the nature of this bias, consider a variable *S* affected by both *X* (treatment) and *Y* (outcome), indicating entry into the data pool.

●Such preferential selection to the pool **amounts to conditioning on** *S*, which creates spurious association between *X* and *Y*.

●Our assumption about the selection mechanism are embodied in an **augmented causal graph G_s**.



●**Illustration** : Effect of training program on earnings

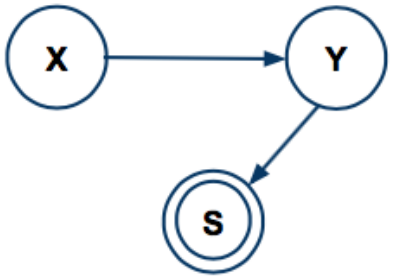
●S represents the selection mechanism. *S=1* indicates presence in the sample, and *S=0* exclusion.

Recoverability

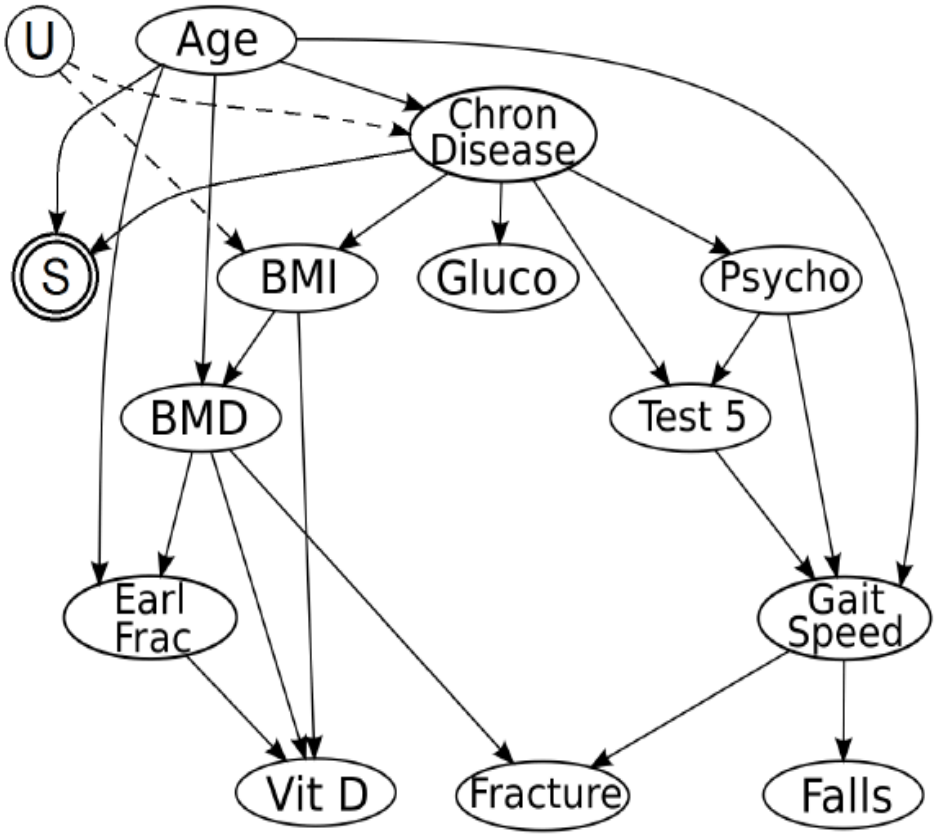
- Under what conditions $P(y|\mathbf{do}(x))$ can be recovered from data drawn from $P(y, x|S = 1)$?

- **Recoverability from Selection Bias:** Given a causal graph G_s augmented with S, $P(y|\mathbf{do}(x))$ is said to be recoverable from selection biased data in G_s if $P(y|\mathbf{do}(x))$ is expressible in terms of the distribution under selection bias $P(v|S = 1)$.

- In this example, $P(y|\mathbf{do}(x))$ is *not* recoverable



Osteoporotic fracture risk assessment



- Prospective cohort study with 7500 elderly osteoporotic women followed-up during 4 years.
- A *plausible* causal BN was learned from a combination of **non-experimental data** and qualitative assumptions that are deemed likely by health experts.
- Inclusion of a **selection mechanism** and an **unobserved confounder.**

- We seek to estimate the strength of the **causal effect of psychotropic drugs** on the risk of **hip fracture:**

$$P(Fracture \mid \mathbf{do}(Psycho)) = ?$$

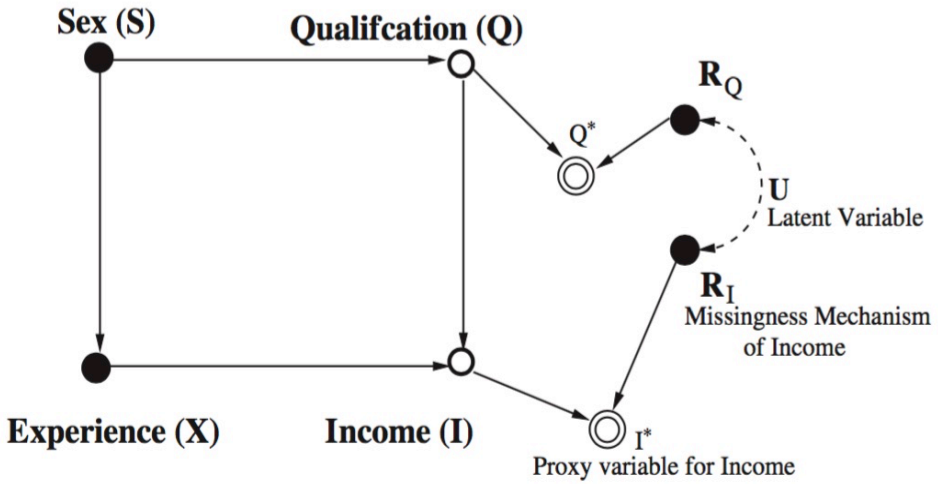
A. Aussem et al. “Analysis of risk factors of hip fracture with causal Bayesian networks”. IWBBIO 2014.
P.Caillet et al. “Hip fracture in the elderly: a re-analysis of the EPIDOS Study with causal Bayesian Networks”, Plos One, 2015

Missing data

Missing data

- All branches of experimental science are plagued by missing data
- The “missing data” problem arises when values for one or more variables are missing from recorded observations
- Occurs often in social science, epidemiology, biology and survival data analysis etc.
- Caused by varied factors such as high cost involved in measuring variables, failure of sensors, reluctance of respondents in answering certain questions
- **Improper handling of missing data** can bias outcomes and potentially **distort the conclusions** drawn from a study.

Misingness mechanism : *m*-graph

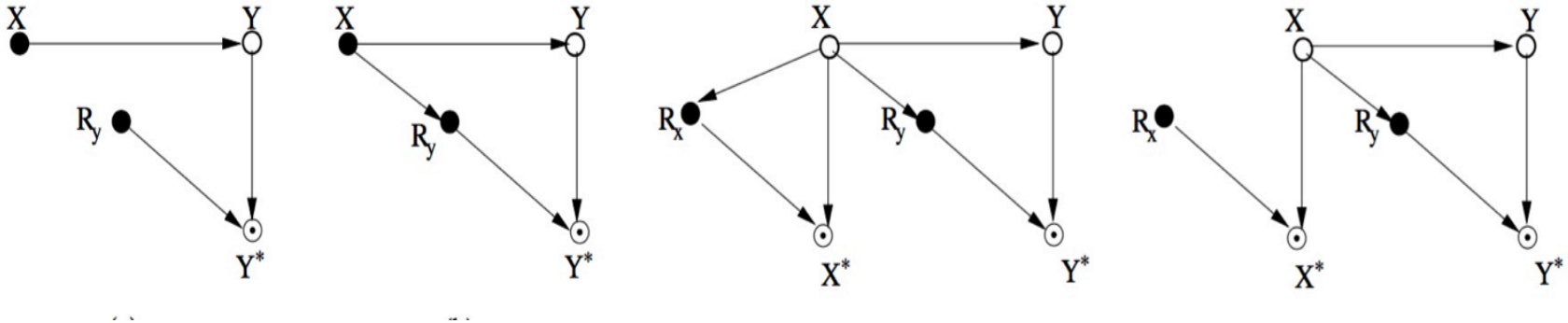


- Associated with every partially observed variable $V_j \in V_{miss}$ are **two other variables** R_j and V_j^*
- V_j^* is a proxy variable that is actually observed.
- R_j represents the status of the causal mechanism responsible for the missingness of V_j*

$$v_i^* = f(r_{v_i}, v_i) = \begin{cases} v_i & \text{if } r_{v_i} = 0 \\ m & \text{if } r_{v_i} = 1 \end{cases}$$

Observed and partially missing variables are represented by full and hollow circles respectively.

Missingness mechanisms



- V_j^* is a proxy variable that is actually observed, and R_i represents the status of the **causal mechanism responsible for the missingness** of V_j.

- Data that are: (a) MCAR, (b) MAR, (c) & (d) MNAR. Hollow and solid circles denote partially and fully observed variables respectively

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Mohan K. et al. *"Missing Data as a Causal Inference Problem".* NIPS, 2013

Recoverability with missing data

Let V_{obs}, V_{miss} be the set of observed and missing variables

- **Recoverability from Data Missingness Bias:** Given a causal graph *G* augmented with the missingness variables *R*, $P(y|\mathbf{do}(x))$ is said to be recoverable in *G* if $P(y|\mathbf{do}(x))$ is expressible in terms of the distributions $P(V_{obs}, V_{miss}|R = 0)$.

Conclusions

- Testing for cause and effect is difficult, discovering cause effect is even more difficult.
- But, once the **causal diagram** is provided (both from *expert knowledge* and data), identification of causal effects is straightforward using the *do-calculus* rules.
- Many **paradoxes** and **controversies** in social and medical sciences can be illustrated and understood by simple graphical means.
- The **data missingness** and **selection mechanisms** can easily be represented in the diagram for **bias correction** purposes.
- **Inference of causal relationships from massive data sets** is still a challenge but may eventually lead to new discoveries (e.g. cancer)

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Thank you for your attention, any question ?