



MSc in Fundamental Principles of Data Science

3

Ethical Data Science

Bias and Discrimination II: Causality

Jordi Vitrià 2020-2021

Observational Data

Source: https://fairmlbook.org/

UC Berkeley	admissions a	data	from 1973.
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	Men		Women	
Departmen	nt Applie	ed Admitted ((%) Applied	Admitted (%)
A	825	62	108	82
В	520	60	25	68
С	325	37	593	34
D	417	33	375	35
E	191	28	393	24
F	373	6	341	7

Is this a fair admission process?

Observational Data

Source: https://fairmlbook.org/

	Men	Women	
Departr	nent Applie	d Admitted (%) Applied Admit	ted (%)
Α	825	62 108 82	
В	520	60 25 68	
С	325	593 34	
D	417	33 ← 375 35	
E	191	28 393 → 24	
F	373	6 341 7	

First Observation:

four of the six largest departments show a higher acceptance ratio among women, while two show a higher acceptance rate for men.

Observational Data

Source: https://fairmlbook.org/

		UC Berk	eley admissions	data from 1	973.	
	Men Women					
	Departmen	t Applie	ed Admitted (%	%) Applied	Admi	tted (%)
	A	825	62	108	82	
	В	520	60	25	68	
	С	325	37	593	34	
	D	417	33	375	35	
Second Observation: The acceptance rate	E	191	28	393	24	
across all six departments (aggregate admission decisions) for men is about	F	373	6	341	7	
44%, while it is only roughly 30% for women, a significant difference.			44% 1157/2	651	30%	556/1835

Such reversals are sometimes called **Simpson's paradox**

Simpson's paradox causes discomfort to some, because **intuition** suggests that a trend which holds for all subpopulations should also hold at the population level.

3rd Observation:

What is evident from the data is that **gender influences department choice**. Women and men appear to have different preferences for different fields of study.

Observational Data

Source: https://fairmlbook.org/

		UC Berke	ley admissions o	lata from 19	973.
		Men		Women	L
	Departmen	t Applied	d Admitted (%) Applied	Admitted (%)
Third Observation:	A	825	62 →	108	82
gender influences department choice	В	520	60	25	68
department choice	С	325 ←	37 →	593	34
	D	417	33	375	35
	E	191	28	393	24
	F	373	6	341	7

Simpson's paradox causes discomfort to some, because intuition suggests that a trend which holds for all subpopulations should also hold at the population level.

4th Observation:

Moreover, different departments have different admission criteria. Some have lower acceptance rates, some higher.

Observational Data

Source: https://fairmlbook.org/

	UC Berkeley admissions	s data from 19	73.
	Men	Women	
D	epartment Applied Admitted (%) Applied	Admitted (%)

Fourth Observation: Some Depts. have lower acceptance rates, some higher

	Men		Women	
Departme	nt Appli	ed Admitte	ed (%) Applied	Admitted (%)
A	825	62	108	82
В	520	60	25	68
С	325	37	593	34
D	417	33	375	35
E	191	28	393	24
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Observational Data

Source: https://fairmlbook.org/

	UC Berkeley admissi			lata from 1		
		Men		Women	l	
	Departmen	ıt Applie	d Admitted (%) Applied	Admitt	ed (%)
	Α	825	62	108	82	Less competitive
m) .	В	520	60	25	68	Less competitive
There is a pattern!	С	325 ←	37	593	34	
	D	417	33	375	35	More competitive
	E	191 ←	28	393	24	riore compensive
	F	373	6	341	7	<u> </u>

Therefore, one explanation for the data we see is that women chose to apply to more competitive departments (men chose to apply to less competitive departments), hence getting rejected (accepted) at a higher rate than men.

Indeed, this is the conclusion an original study (Bickel, Hammel, O'Connell, and others, "Sex Bias in Graduate Admissions.", 1975) drew:

The bias in the aggregated data stems not from any pattern of discrimination on the part of admissions committees, which seems quite fair on the whole, but apparently from prior screening at earlier levels of the educational system. Women are shunted by their socialization and education toward fields of graduate study that are generally more crowded, less productive of completed degrees, and less well funded, and that frequently offer poorer professional employment prospects.

In other words, the article concluded that the source of gender bias in admissions was a pipeline problem: Without any wrongdoing by the departments, women were "shunted by their socialization" that happened at an earlier stage in their lives.

We can ask why women applied to more competitive departments in the first place.

There are several possible reasons.

- Perhaps less competitive departments, such as engineering schools, were unwelcoming of women at the time. This may have been a general pattern at the time or specific to the university.
- Perhaps some departments had a track record of poor treatment of women that was known to the applicants.
- Perhaps the department advertised the program in a manner that discouraged women from applying.

We can ask why women applied to more competitive

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- Perhaps som treatment of
- Perhaps the chat discourant

There is no way of knowing what was the case from the data we have. We see that at best the original analysis leads to a number of follow-up questions.

At this point, we have two choices. One is to design a new study and collect more data in a manner that might lead to a more conclusive outcome. The other is to argue over which scenario is more likely based on our beliefs and plausible assumptions about the world.

Causal inference is helpful in either case.

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Causal Inference

"We can distinguish two kinds of intentionality. Primary intentionality is the repetition of causes that worked in the past. This is the intentionality of adaptation by natural selection and of conditioned reflexes. Past effects are anticipated to occur again. Secondary intentionality is choice of action after simulation of possible choices and their effects. Simulated effects are anticipated to occur when the action is performed. Secondary intentionality requires imagination, an ability to "hold in mind" and evaluate virtual outcomes. Primary intentionality is "primary" in the sense that anticipation evolved before imagination."

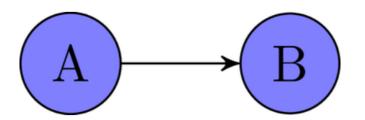
Fragment from: Haig, David; Dennett, Daniel C.;. "Selfish Genes, Social Selves, and the Meanings of Life".

Causality: Intuition

If I randomly picked a person from a population, and found out that she owns a Tesla, I'd find that she was more likely than the average person to have a college degree.

This means that owning a Tesla is **associated** with having a college degree, i.e. knowing if she has a college degree changes the likelihood that she also owns a Tesla.

Does this mean that owning a Tesla causes people to have a college degree?



Getting answers from data

OBSERVATIONAL (passive observation of the world) DATASET

	Sex	Race	Height	Income	Marital Status	Years of Educ.	Liberal- ness
R1001	M	1	70	50	1	12	1.73
R1002	M	2	72	100	2	20	4.53
R1003	F	1	55	250	1	16	2.99
R1004	М	2	65	20	2	16	1.13
R1005	F	1	60	10	3	12	3.81
R1006	M	1	68	30	1	9	4.76
R1007	F	5	66	25	2	21	2.01
R1008	F	4	61	43	1	18	1.27
R1009	М	1	69	67	1	12	3.25

Let's consider 3 different features in this dataset, (X, Y, Z).

Which can of questions can we answer from this dataset?

Q1: Which is the expected income Y that would have been observed if an individual had X = x and Z = z?

Association (or prediction) is using data to map some features of the world (the inputs) to other features of the world (the outputs). For example, $\mathbb{E}(Y|X,Z)$.

All we need to do prediction is a dataset sampled from p(X, Y, Z) and some inference tools (statistical inference & machine learning).

Mapping observed inputs to observed outputs is a natural candidate for automated data analysis because this task only requires 1) a large dataset with inputs and outputs, 2) an algorithm that establishes a mapping between inputs and outputs, and 3) a metric to assess the performance of the mapping, often based on a gold standard.

Q2: Estimate the mean income that would have been observed if all individuals had race=1 vs. if they had race=2 or race= 3.

Causal Inference is using data to predict certain features of the world **if the world had been different**. We cannot get these data by passive observation of the world! The world was different!

Answers to causal questions cannot be derived exclusively from p(X, Y, Z). Answering a causal question (yes, sometimes is possible!) typically requires a combination of data, analytics, and expert **causal knowledge**.

Let's say we have i.i.d. data sampled from some joint p(X, Y, Z).

Say we are ultimately interested in how variable Y behaves given X. At a high level, one can ask this question in two ways:

• observational, based on p(Y|X,Z):

What is the distribution of Y given that I observe variable X takes value x?

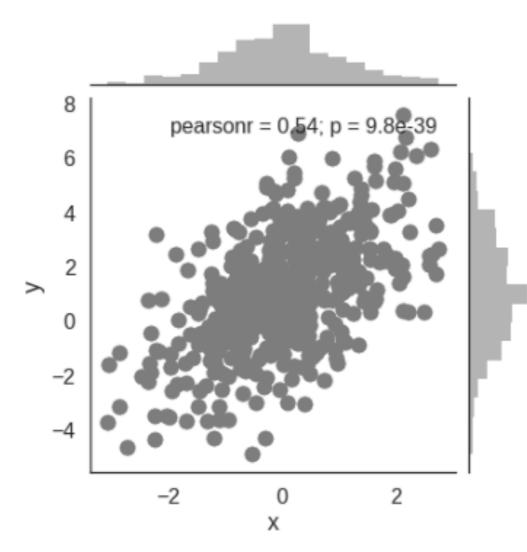
For example, estimate the income that would have been observed if all individuals had race=1 vs. if

they had race=2 or race= 3.

• **interventional**, based on $p(Y|do(X), Z) \neq p(Y|X, Z)$: What is the distribution of *Y* if I were to **set** the value of *X* to *x*.

This describes the distribution of Y I would observe if I **intervened** in the data generating process by artificially forcing the variable X to take value x, but otherwise **simulating the rest of the variables according to the original process that generated the data**.

In order to understand what is p(Y|do(X)), let's suppose I have observed p(X, Y).



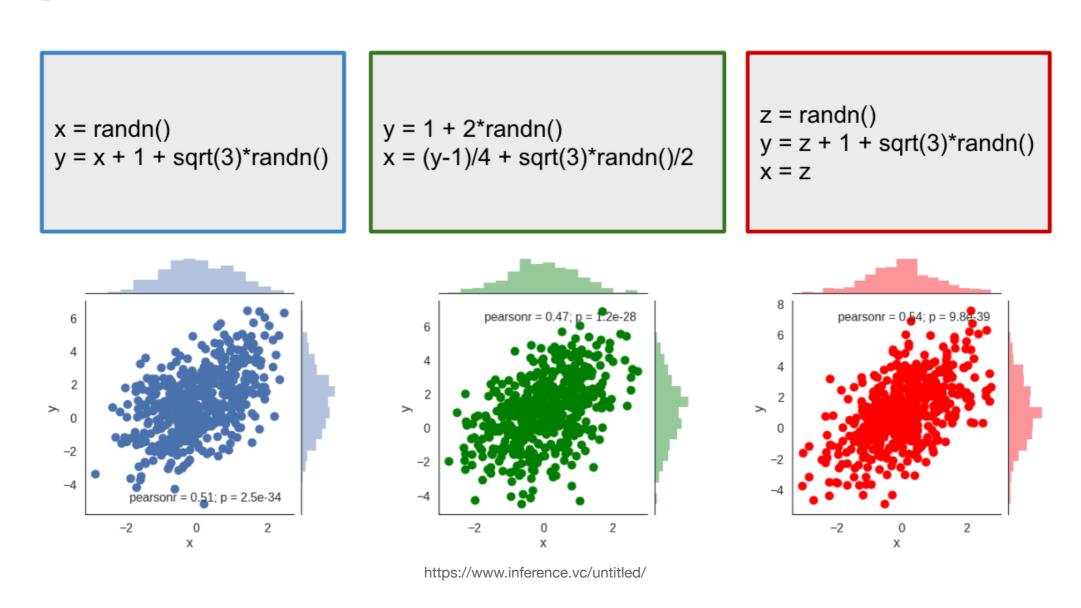
https://www.inference.vc/untitled/

This is all we need to compute p(Y|X). We can give an answer to any <u>associational question</u>.

For example:

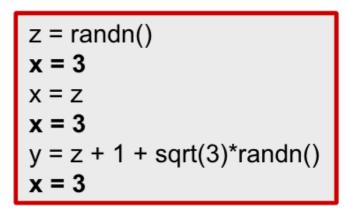
- What is the expected value of Y if we observe X = 3?
 (Regression)
- What is the expected MAX/ MIN/MEDIAN value of Y if we observe X = 3? (Quantile regression)

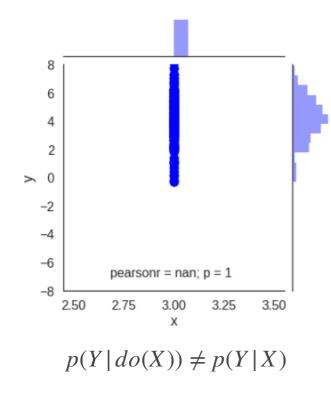
Given p(X, Y), there are several generative models that are compatible with p(X, Y):

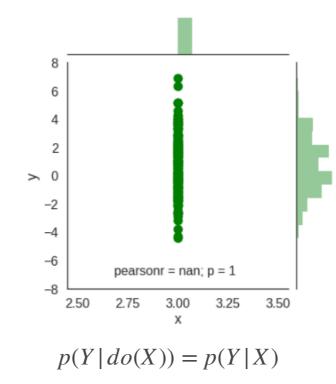


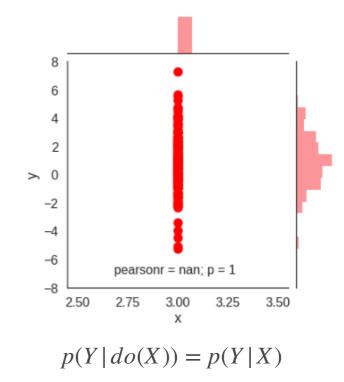
Based on the joint distribution the three scripts are indistinguishable.

Let's now consider an **intervention** p(Y | do(X = 3))





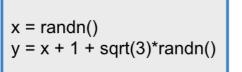




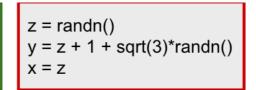
The joint distribution of data p(X, Y, Z) alone is insufficient to predict behavior under interventions.

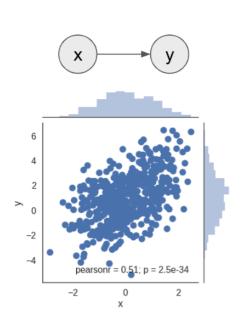
An intervention can be understood as a modification of the generative model of the data, producing a different probability distribution p(do(X), Y, Z).

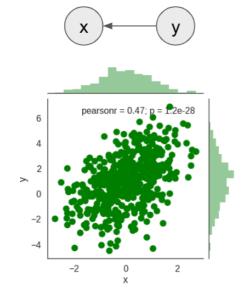
The resulting distribution depends on the original model:

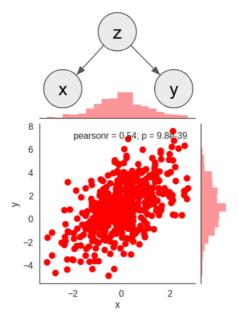


```
y = 1 + 2*randn()
x = (y-1)/4 + sqrt(3)*randn()/2
```









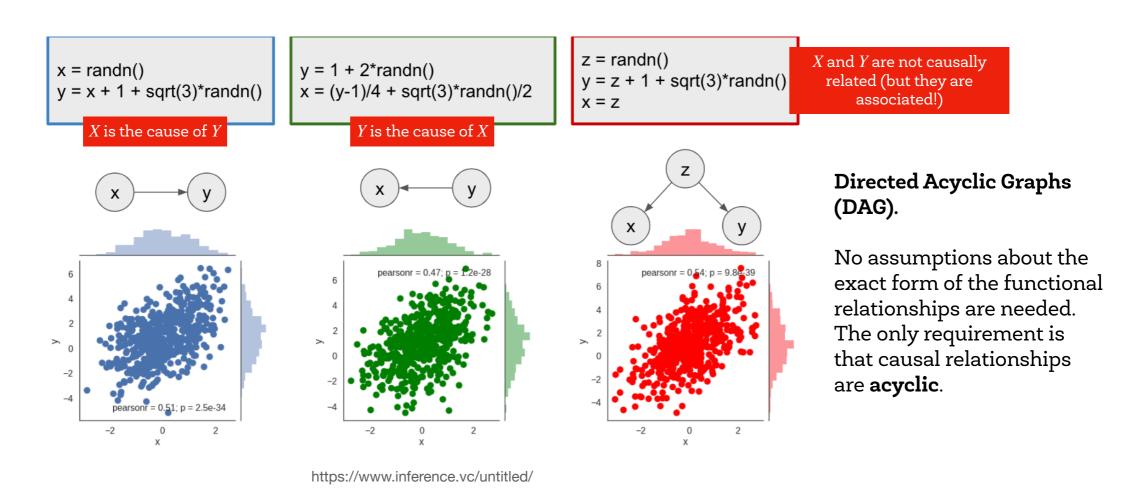
Directed Acyclic Graphs (DAG).

No assumptions about the exact form of the functional relationships are needed. The only requirement is that causal relationships are acyclic.

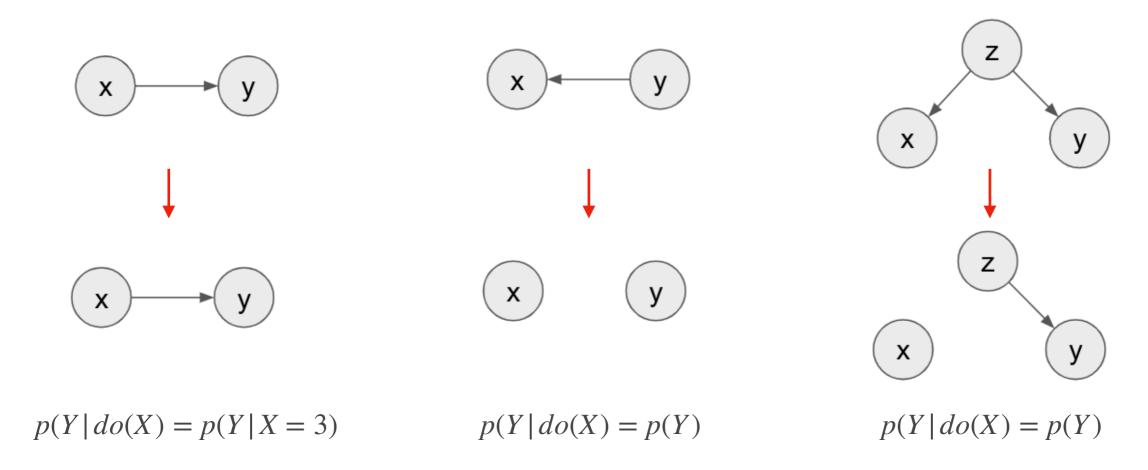
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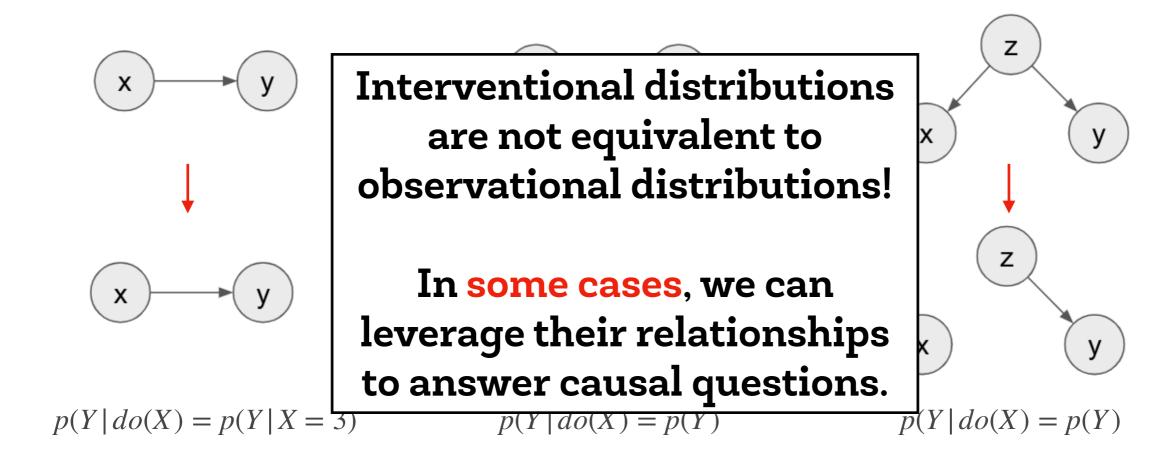


Graphically, to simulate the effect of an intervention, you mutilate the graph by removing all edges that point into the variable on which the intervention is applied, in this case X.



Just by only looking at the causal diagram, we are now able to predict how the scripts are going to behave under the intervention X=3.

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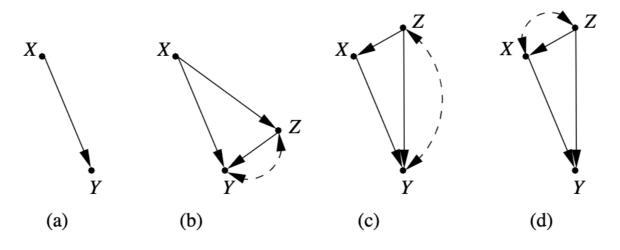


Just by only looking at the causal diagram, we are now able to predict how the scripts are going to behave under the intervention X=3.

An essential matter in causal inference is that of a query's identifiability.

Given a causal query (for example, p(Y|do(X=3))) for a certain DAG, we say it is **identifiable** if we can derive an statistical estimand (**only using observational terms**) for this query using the rules of **do-calculus**.

The do-calculus is an axiomatic system for replacing probability formulas containing the do operator with ordinary conditional probabilities. It consists of three axiom schemas that provide graphical criteria for when certain substitutions may be made.



Causal graphs where $P(y|do(\mathbf{x}))$ is identifiable

Source: Complete Identication Methods for Causal Inference, PhD Thesis, University of California. I.Shpitser https://ftp.cs.ucla.edu/pub/stat_ser/shpitser-thesis.pdf

There are <u>two ways</u> to assess the existence of a **causal** relationship between two variables, X and Y:

1. The easiest way is an **intervention** in the real world: You randomly force *X* to have different values and you measure *Y*.

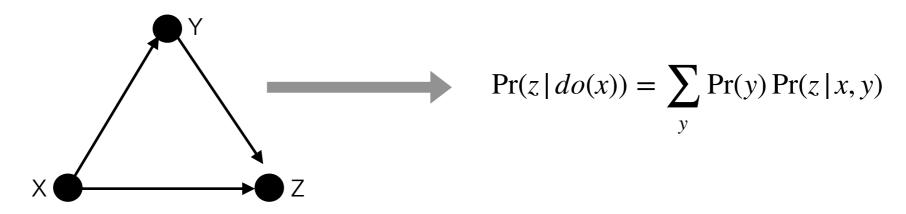
This is what we do in Randomized Clinical Trial (RCT) or in an A/B Test.

This is not always feasible (because of ethical or economical reasons)

There are <u>two ways</u> to assess the existence of a **causal** relationship between two variables, X and Y:

2. If the query is identifiable, **do-calculus** allows us to massage p(X, Y, Z) until we can express p(Y | do(X)) in terms of various marginals, conditionals and expectations under p(X, Y, Z)

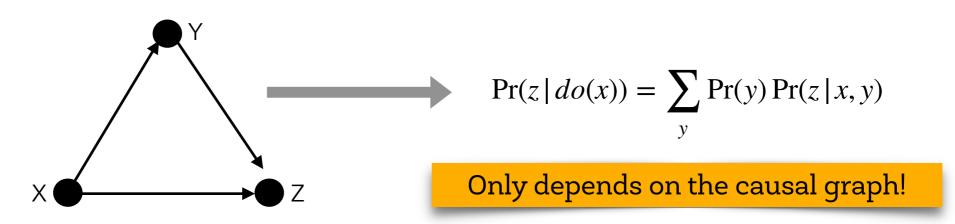
Example:

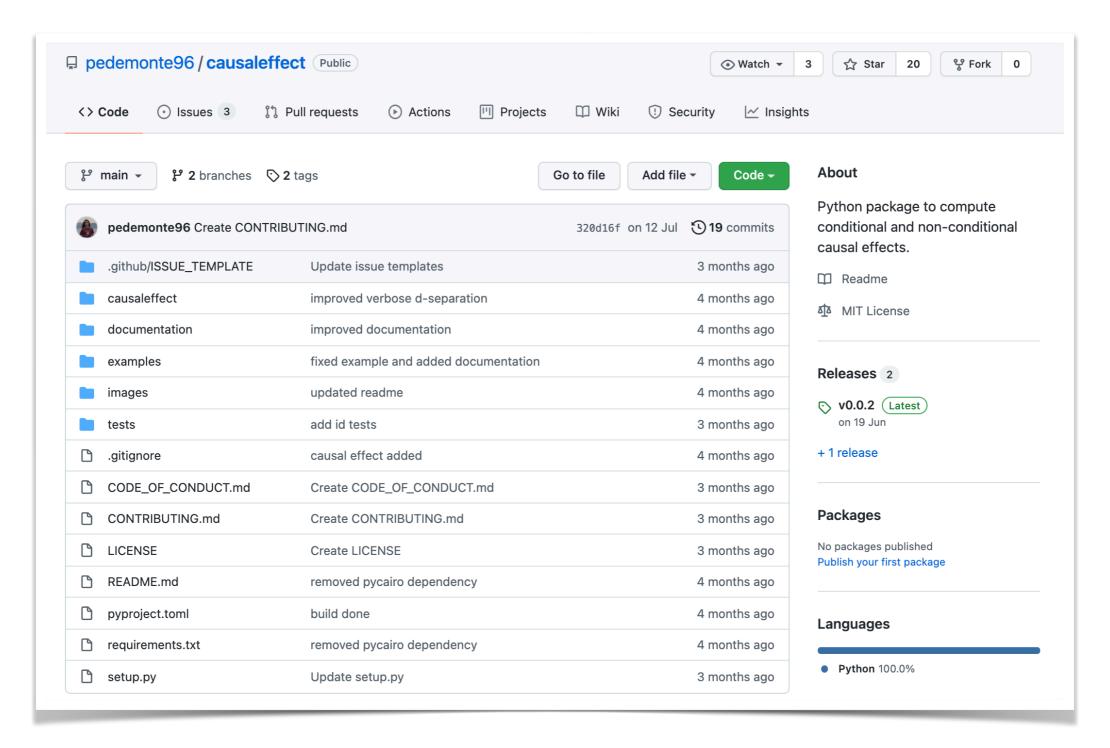


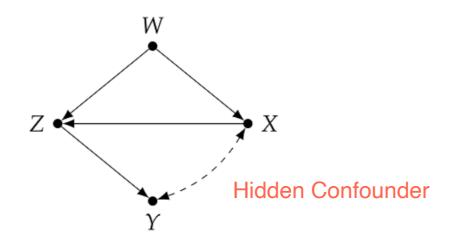
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Example:







```
import causaleffect

G = causaleffect.createGraph(['X<->Y', 'Z->Y', 'X->Z', 'W->X', 'W->Z'])
causaleffect.plotGraph(G)
```

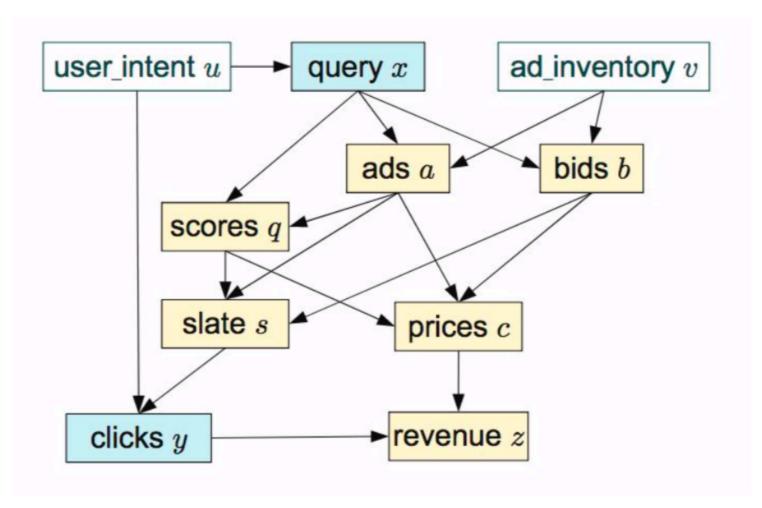
```
P = causaleffect.ID({'Y'}, {'X'}, G)
P.printLatex()
```

The code above computes the causal effect, and returns a string encoding the distribution in LaTeX notation:

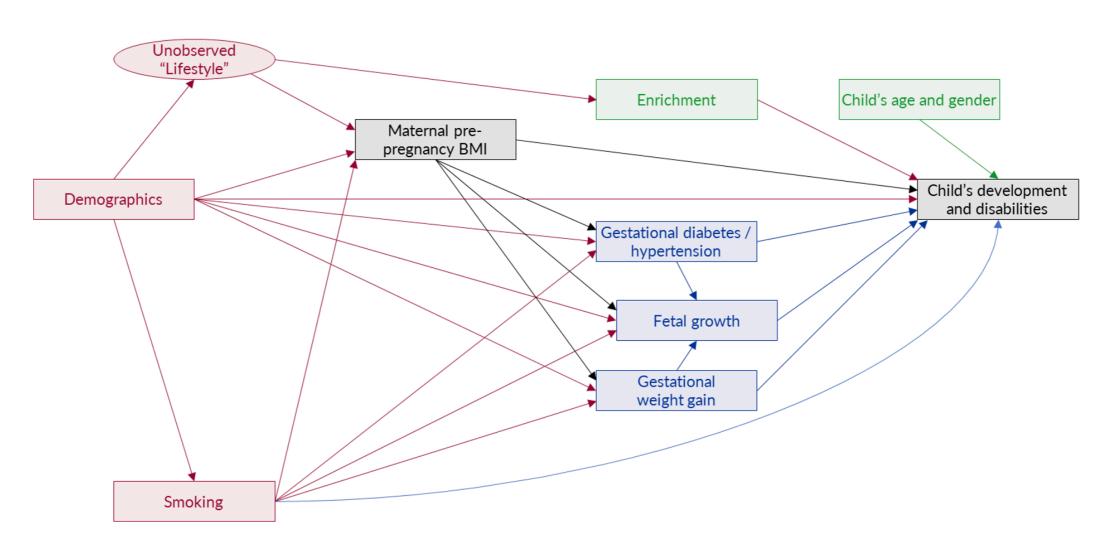
```
\label{eq:continuous} $$ \sum_{w, z}P(w)P(z|w, x)\left(\sum_{x}P(x|w)P(y|w, x, z)\right)' $$
```

This string, in LaTeX, is

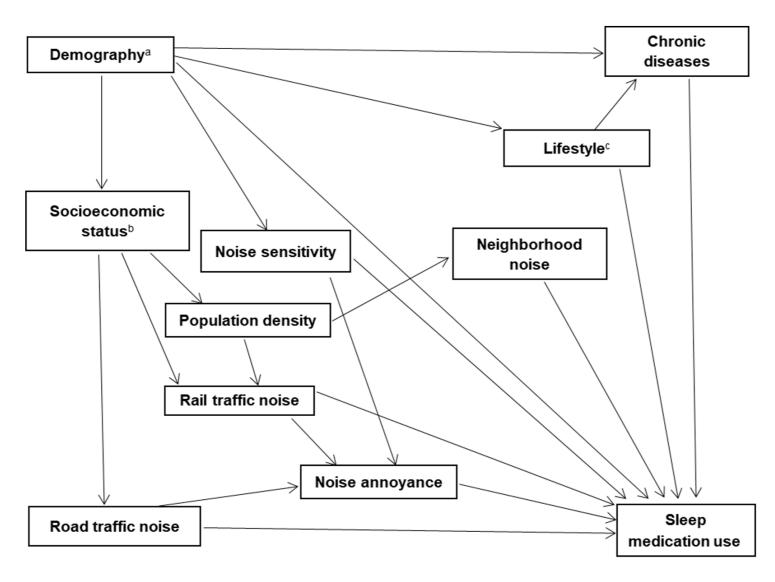
$$\sum_{w,z} P(w)P(z|w,x) \left(\sum_{x} P(x|w)P(y|w,x,z) \right)$$



Bottou, Léon, et al. "Counterfactual reasoning and learning systems: the example of computational advertising." *The Journal of Machine Learning Research* 14.1 (2013): 3207-3260.



ADAPTED FROM: Hinkle SN, Sharma AJ, Kim SY, Schieve LA. Maternal prepregnancy weight status and associations with children's development and disabilities at kindergarten. *Int J Obes (Lond)*. 2013;37(10):1344-51. DOI: 10.1038/ijo.2013.128 (Figure 1). Freely available at: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4407562



REPRODUCED UNDER CC-BY 4.0 LICENSE FROM: Evandt J, Oftedal B, Krog NH, et al. Road traffic noise and registry based use of sleep medication. *Environ Health.* 2017;16(1):110. DOI: 10.1186/s12940-017-0330-5 (Figure S1). Freely available at: https://www.doi.org/10.1186/s12940-017-0330-5

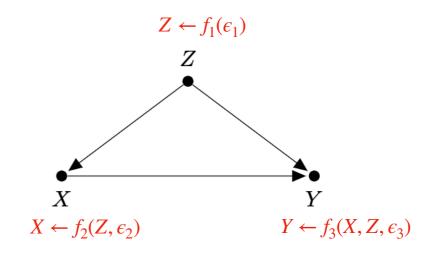
Causal Inference and SCM

The causal diagram can be seen as a representation of an underlying structural causal model (generative model):

Directed Acyclic Graphs (DAG):

No assumptions about the exact form of the functional relationships, as well as the distribution of background factors, are needed. The only requirement is that causal relationships are acyclic.

	Sex	Race	Height	Income	Marital Status	Years of Educ.	Liberal- ness
R1001	M	1	70	50	1	12	1.73
R1002	M	2	72	100	2	20	4.53
R1003	F	1	55	250	1	16	2.99
R1004	М	2	65	20	2	16	1.13
R1005	F	1	60	10	3	12	3.81
R1006	М	1	68	30	1	9	4.76
R1007	F	5	66	25	2	21	2.01
R1008	F	4	61	43	1	18	1.27
R1009	М	1	69	67	1	12	3.25



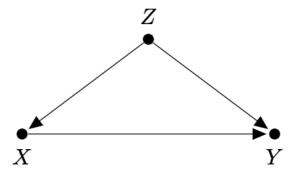
 ϵ_i are **exogenous background factors** represented by an arbitrary noise distribution.

They exert an influence on the endogenous variables in the model. Because they are <u>unobserved</u> from the standpoint of the analyst, this renders the model stochastic with a probability distribution over the set of **endogenous variables**.

Causal Inference and SCM

We can estimate f_i from data by using predictive methods.

Actions can now be defined as interventions on variables in the model.



$$Z \leftarrow f_1(\epsilon_1)$$

$$X \leftarrow f_2(Z, \epsilon_2)$$

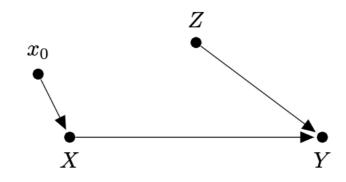
$$Y \leftarrow f_3(X, Z, \epsilon_3)$$

Structural Causal Model

For example, intervening on X amounts to deleting f_2 and setting X to a constant value x_0 .

$$Z \leftarrow f_1(\epsilon_1)$$
$$X \leftarrow x_0$$
$$Y \leftarrow f_3(X, Z, \epsilon_3)$$

Modified Structural Causal Model



This graph encodes the intervened distribution, from which we can **sample** and **compute causal queries** (if they are identifiable).

For example, compute **causal effects**:

$$p(Y | do(X = x_1) - p(Y | do(X = x_0))$$

Counterfactuals

Counterfactuals in our life:

Source: https://christophm.github.io/interpretable-ml-book/counterfactual.html

Let's support that we want to rent an apartment and we train a model with real data to predict a price.

After entering all the details about size, location, whether pets are allowed and so on, the model tells us that we can charge 900€.

How could we get (by doing an intervention) 1000€? We can play with the feature values of the apartment to see how we can improve the value of the apartment!

We find out that the apartment could be rented out for over 1000 Euro, if it were 15 m² larger. Interesting, but <u>non-actionable</u> knowledge, because we cannot enlarge the apartment.

Finally, by tweaking only the feature values under our control (built-in kitchen yes/no, pets allowed yes/no, type of floor, etc.), we find out that if we allow pets and install windows with better insulation, we can charge 1000€.

Counterfactuals

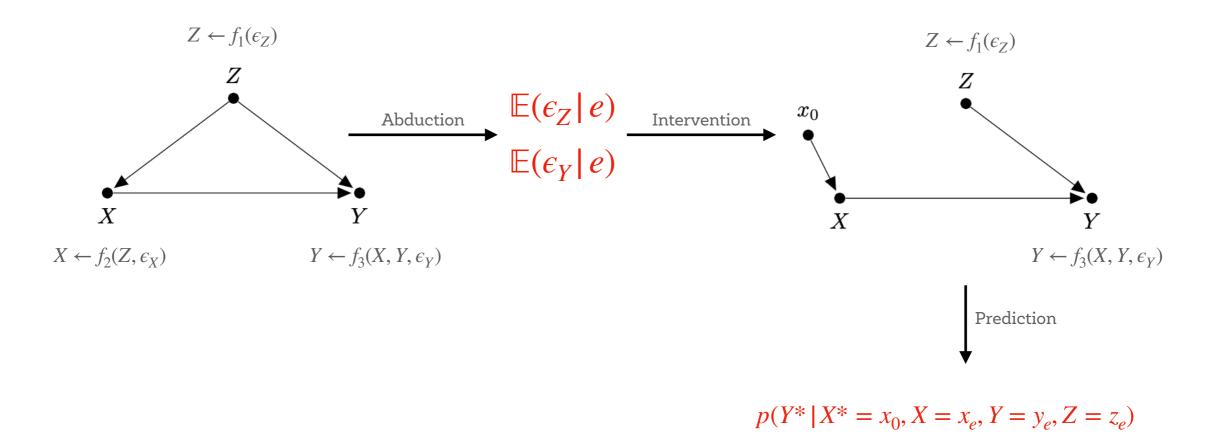
Given a **certain observational** sample $e = (x_e, y_e, z_e)$ and an intervention $do(X = x_q)$, a **counterfactual** is the result of an hypothetical experiment in the past, what would have happened to the value of variable Y had we intervened on X by assigning value x_q .

Identifiable counterfactuals can be computed as a three-step process:

- **Abduction**: compute the posterior distribution of ϵ conditioned on ϵ .
- **Intervention**: apply the desired intervention do(X = x)
- **Prediction**: compute the required prediction in the intervened distribution.

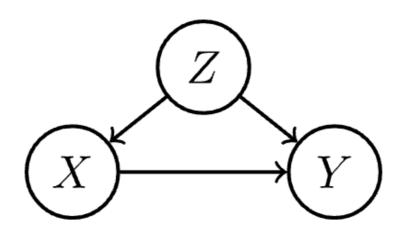
Counterfactuals

	R1001	M	1	70	50	1	12	1.73
<i>e</i>	R1002	М	2	72	100	2	20	4.53
	R1003		1	55	250	1	16	2.99
		M	2	65	20	2	16	1.13
	R1005	F	1	60	10	3	12	3.81
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	R1007	_	5	66	25	2	21	2.01

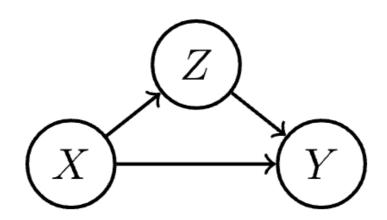


A **fork** is a node Z in a graph that has outgoing edges to two other variables X and Y.

Put differently, the node Z is a common cause of X and Y.

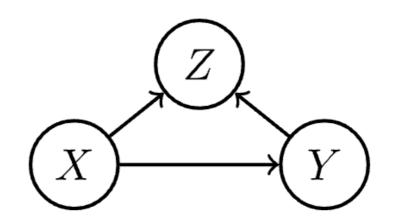


Z has a **confounding** effect: There is a positive association between X and Y (not due to $X \rightarrow Y$) because of Z.



In this case, the path $X \rightarrow Z \rightarrow Y$ contributes to the total effect of X on Y. It's a causal path and thus one of the ways in which X causally influences Y. That's why Z is not a confounder. We call Z a **mediator** instead.

The notion of a mediator is particularly relevant to the topic of discrimination analysis.



This is a **collider**. Colliders aren't confounders. In fact, in the above graph, X and Y are unconfounded, meaning that we can replace do-statements by conditional probabilities.

However, something interesting happens **when we condition** on a collider. The conditioning step can create association between X and Y, a phenomenon called *explaining* away.

When we speak of the **causal effect of a variable** X **on another variable** Y we refer to **all the ways** in which setting X to any possible value x affects the distribution of Y.

Often we think of X as a binary treatment variable and are interested in a quantity such as:

$$\mathbb{E}_{do(X=1)}[Y] - \mathbb{E}_{do(X=0)}[Y]$$

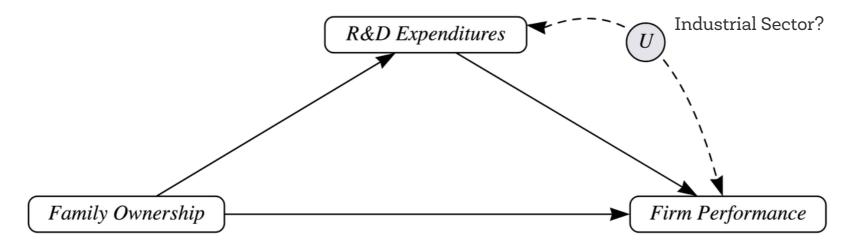
Causal effects are **population quantities**. They refer to effects averaged over the whole population. Often the effect of treatment varies greatly from one individual or group of individuals to another. Such treatment effects are called heterogeneous.

This quantity is called the average treatment effect.

Unobserved confounders

We have described causal inference with observational data under an assumption of no unobserved confounders.

This is not the case in most of the cases!



https://p-hunermund.com/2018/08/27/why-you-shouldnt-control-for-post-treatment-variables-in-your-regression/

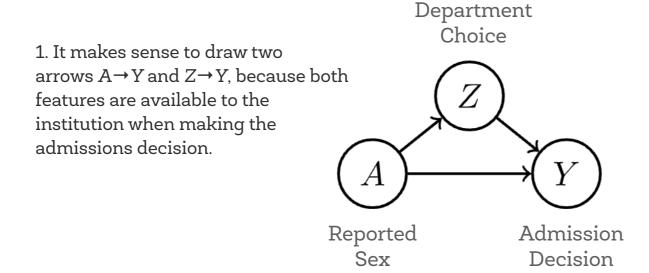
Do-calculus was extended to work with unobserved confounders. Identifiability issues are harder in the presence of unobserved confounders.



We now explore how we can bring causal graphs to bear on discussions of discrimination.

The first step is to come up with a plausible causal graph consistent with the data that we saw earlier.

BERKELEY ADMISSION

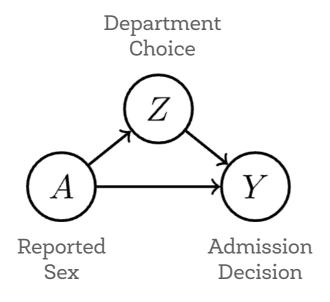




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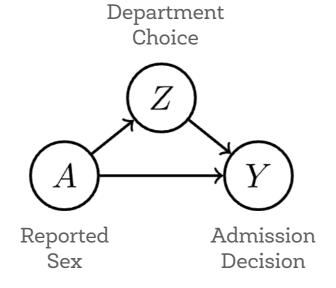


2. A and Z are not statistically independent. We can see from the table that several departments have a statistically significant gender bias among applicants. This means we need to include either the arrow $A \rightarrow Z$ or $Z \rightarrow A$. Which one?

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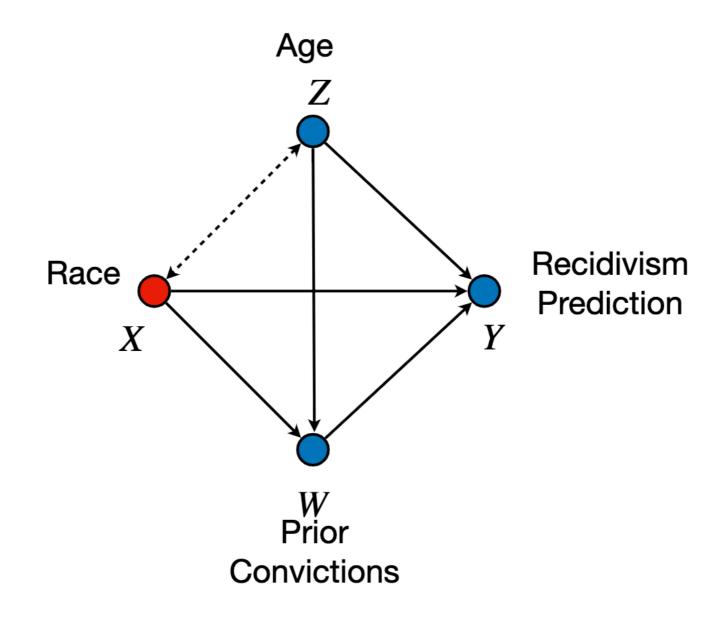
3. To align Bickel's story with our causal graph, we need the variable A to reference whatever ontological entity it is that through this "socialization process" influences intellectual and professional preferences, and hence, department choice.

It is difficult to maintain that this ontological entity coincides with sex as a biological trait. There is no scientific basis to support that the biological trait sex is what determines our intellectual

preferences.

https://fairmlbook.org/

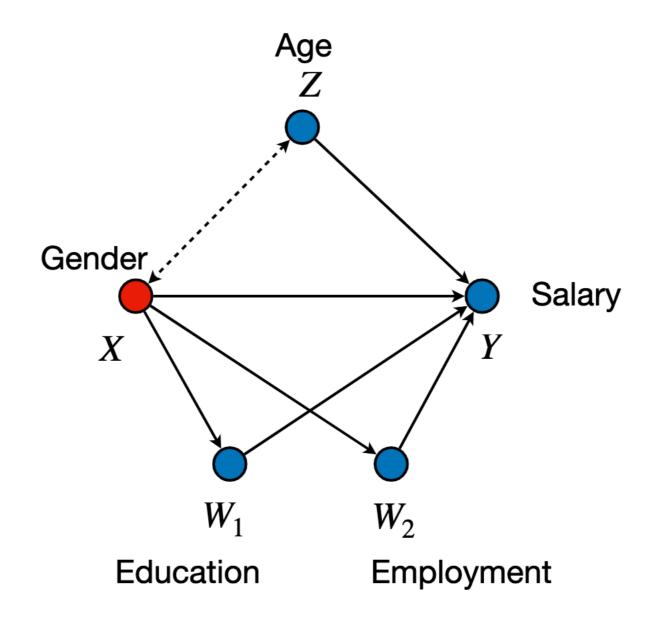
COMPASS PREDICTION



https://fairness.causalai.net/

UCI ADULT

The US census data records whether a person earns more than \$50,000/year (Y). The census also records age (Z), gender (X = 0 for male, X = 1 for female), education level (W_1) and employment status (W_2 with 10 job types).

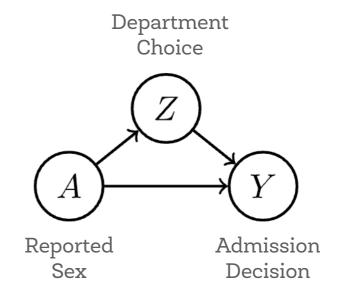


https://fairness.causalai.net/

In causal language, Bickel's argument about Berkeley admissions had two components:

- There is **no direct effect** of sex A on the admissions decision Y that favors men.
- The **indirect effect** of A on Y, that is mediated by Z, should not be counted as evidence of discrimination.

These are causal concepts that can be measured with data and a causal model!



Direct Effects

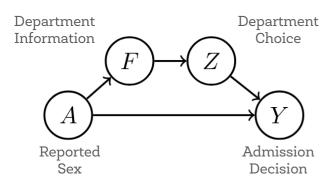
To measure the direct effect of A on Y we need to disable all paths between A and Y except for the direct link. In our model, we can accomplish this by holding department choice Z constant and evaluating the conditional distribution of Y given A.

$$\Pr(Y | do(A) = a) = \sum_{z} \Pr(Z = z) \Pr(Y | A = a, Z = z)$$

This is the right answer to this Simpson's paradox!

Indirect Effects

The direct effect of a protected variable on a decision is a poor measure of discrimination on its own as it cannot detect any form of proxy discrimination.



For example, the department may have advertised the program in a manner that strongly discouraged women from applying.

This indirect path encodes a pattern of discrimination.

We can think of the direct effect as whether or not the decision maker **explicitly** uses the attribute in its decision rule. Additionally, we have to carefully discuss what pathways we consider evidence for or against discrimination.

Causal statements can be easily translated into counterfactuals:

Causal Statement:

Was I not hired because I was black?

Counterfactual Statement:

Would I have been hired if I were non-black?

Then, alternatively, we can state some definitions of fairness in terms of counterfactuals: Individual Counterfactual Fairness, Counterfactual Parity, Conditional Counterfactual Parity.

For every individual i we only see Pr(Y|A = black) or $Pr(Y|A = non_black)$ (not both!), but we can consider its counterfactual.

Individual Counterfactual Fairness (ICF), for individual i

$$Pr(Y^*|A^* = non_black) = Pr(Y|A = black)$$

Would the hiring decision have been different if I were $A = non_black$ instead of A = black?

Counterfactual Parity (CP),

 $\mathbb{E}[Pr(Y^* | A^* = \text{non_black})] = \mathbb{E}[Pr(Y^* | A^* = \text{black})]$

Would the rates of hiring be different if everyone were black?

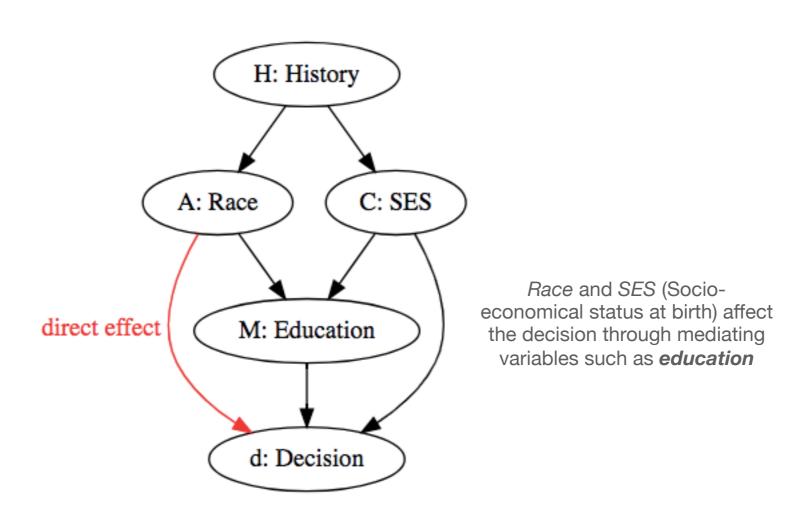
Conditional Counterfactual Parity (CCP),

 $\mathbb{E}[Pr(Y^* | A^* = \texttt{non_black}, X)] = \mathbb{E}[Pr(Y^* | A^* = \texttt{black}, X)]$

Would the rates of hiring be different if I everyone were black, **conditioned on education**?

These causal queries measure the total effect of race on the decision.

We can measure **non direct** causal pathways effects by defining different counterfactuals.



https://shiraamitchell.github.io/fairness/

Let $Pr(Y|A = non_black, M(black))$ be the decision if the applicant had not been black, but education remained the same.

No Direct Effect Fairness

$$\mathbb{E}[Pr(Y|A = black)] = \mathbb{E}[Pr(Y^*|A^* = non_black, M(black))]$$

This counterfactual assumes that there is no direct effect of race on hiring, but race is allowed to affect decision through education.