Friedrich-Alexander-Universität
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Using Directed Acyclic Graphs (DAGs) to Advance Causal Inference with Observational Data

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Before we start…

I would like to know a bit about your background and knowledge:

- Master‘s / PhD student / Researcher ?
- Geography / Communication / Politics / Psychology / Social Policy / Sociology
- Regression / confounding / fundamental problem of causality / colliders

Outline

A) Counterfactuals, causal inference and observational data

- The counterfactual model of causality
- The fundamental problem of causal inference
- Assignment and the conditional independence assumption

B) Basics of directed acyclic graphs

- Terminology and core concepts
- Fundamentals of analyzing causal graphs
- Identification strategies

C) DAGs in action

- Exemplary application: weight and wages
- Analyzing causal graphs with the software DAGitty
- Short exercise

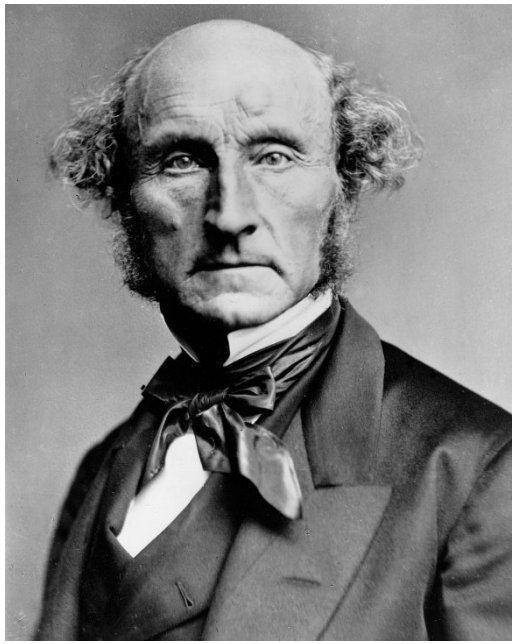
Part A
Counterfactuals, Causal Inference and Observational Data

Counterfactual causality

The basic idea can be already found in work by John Stuart Mill (1806-1873)

> "*Thus, if a person eats of a particular dish, and dies in consequence, that is, would not have died if he had not eaten of it, people would be apt to say that eating of that dish was the cause of his death.*"

Source: John Stuart Mill (2002). A System of Logic. Reprinted from the 1981 edition (first published 1843). Honolulu: UP of the Pacific. S. 214.



The counterfactual model

Building on early work by John Stuart Mill, Jerzy Neyman and others, Donald Rubin (1974, 1977, 1978) formalized these ideas

Journal of Educational Psychology
1974, Vol. 66, No. 5, 688–701

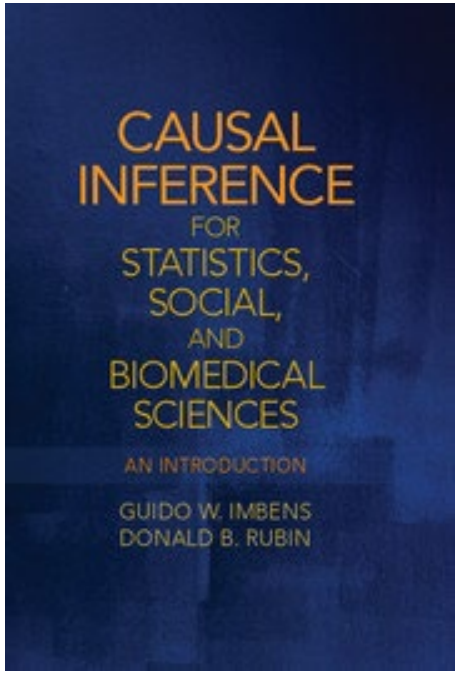
ESTIMATING CAUSAL EFFECTS OF TREATMENTS IN RANDOMIZED AND NONRANDOMIZED STUDIES[1]

DONALD B. RUBIN[2]
Educational Testing Service, Princeton, New Jersey

Starting point:

- Binary treatment
  - D = 1 treatment (treatment group)
  - D = 0 no treatment (control group)
- "Outcome" variable Y: continuous

At least theoretically, each unit could be observed in two states: → Potential outcomes $\left(Y_i^0, Y_i^1\right)$



Counterfactual model: individual treatment effect

Defining causal effects I: Individual treatment effect (ITE)

$$ITE = \delta_i = Y_i^1 - Y_i^0$$

→ the causal effect of treatment D on outcome Y for study unit i is defined as the difference between the two potential outcomes in the experimental and control condition

Counterfactual model: average treatment effect

Defining causal effects II: Average treatment effect (ATE)

$$ATE = \frac{\sum \delta_i}{n} = \frac{\sum_{i=1}^{n} Y_i^1 - Y_i^0}{n}$$

$$= E\left(Y_i^1 - Y_i^0\right)$$

$$= E\left[Y_i^1\right] - E\left[Y_i^0\right]$$

→ the average treatment effect (ATE) at group level is thus the mean of the individual treatment effects

Example: a hypothetical study on social media use

- Experimental group (D = 1): Individuals in the experimental condition committed to severely reducing their social media use for one month.
- Control group (D = 0): individuals in the control condition can use social media without restrictions.
- The ability to concentrate is measured by means of a validated test after one month and can have values from 0 (very poor) to 100 (very good).

→ There are thus two potential outcomes for each participant:

- the ability to concentrate after a one-month break Y^1
- the ability to concentrate without this intervention Y^0

Example: a hypothetical study on social media use

In reality, we would measure "ability to concentrate" from 0 (very poor) to 100 (very good) with the following observed outcomes (denoted by lower y)

		y^1	y^0	δ_i
$D = 1$	$i = 1$	90	missing	
	$i = 2$	60	missing	
	Ø $(D = 1)$	75	60	
$D = 0$	$i = 3$	Missing	80	
	$i = 4$	Missing	60	
	$i = 5$	Missing	60	
	Ø $(D = 0)$	75	66,7	

→ let's assume we are in a hypothetical world, in which we can observe all the potential outcomes (denoted by capital Y)

Example: a hypothetical study on social media use

The following table contains potential outcomes

		Y^1	Y^0	δ_i
$D = 1$	$i = 1$	90	80	
	$i = 2$	60	40	
	Ø $(D = 1)$	75	60	
$D = 0$	$i = 3$	80	80	
	$i = 4$	75	60	
	$i = 5$	70	60	
	Ø $(D = 0)$	75	66,7	

Example: a hypothetical study on social media use

		Y^1	Y^0	δ_i
$D = 1$	$i = 1$	90	80	10
	$i = 2$	60	40	20
	Ø $(D = 1)$	75	60	15
$D = 0$	$i = 3$	80	80	0
	$i = 4$	75	60	15
	$i = 5$	70	60	10
	Ø $(D = 0)$	75	66,7	8,3

individual treatment effects (ITE)

$$ITE = \delta_i = Y_i^1 - Y_i^0$$

i = 1: 90 – 80 = 10

….

average treatment effect (ATE)

$$ATE = \frac{\sum \delta_i}{n}$$

= (10 + 20 + 0 + 15 + 10) / 5

= 11

The fundamental problem of causal inference

- The individual causal effect is theoretically clearly defined, but we cannot observe the same unit of study in two different states at one point in time
- observed outcome (as opposed to potential outcome): only one potential outcome can be observed at a time (= factual state).
- the assignment mechanism D determines which one is observed

$$Y_i = D_i Y_i^1 + (1 - D_i) Y_i^0$$

→ $Y_i = Y^1$ für D=1

→ $Y_i = Y^0$ für D=0

To distinguish between potential and observed outcomes, observed outcomes are sometimes written as lower y (as compared to the potential outcome as capital Y)

The fundamental problem of causal inference

- Paul Holland (1986) called this the fundamental problem of causal inference

	Y^1	Y^0
D=1	factual (observed)	counterfactual (unobserved)
D=0	counterfactual (unobserved)	factual (observed)

The fundamental problem of causal inference

- We can re-write this as conditional expected values

	Y^1	Y^0
D=1	$E[Y^1 \mid D=1]$	$E[Y^0 \mid D=1]$
D=0	$E[Y^1 \mid D=0]$	$E[Y^0 \mid D=0]$

The fundamental problem of causal inference

As explained, one would calculate the average treatment effect (ATE) as follows:

$$ATE = \frac{\sum \delta_i}{n} = E(Y_i^1 - Y_i^0) = E[Y_i^1] - E[Y_i^0]$$

This can be re-written as:

$$ATE = \{\pi E[Y^1|D=1] + (1-\pi)E[Y^1|D=0]\} - \{\pi E[Y^0|D=1] + (1-\pi)E[Y^0|D=0]\}$$

with $\pi = E(D)$ as the relative size of the treatment group as compared to the overall sample size

→ if treatment and control group have equal size π=0.5

→ In our social media example, treatment group is π= 2/5 =0.4

The fundamental problem of causal inference

$$ATE = \{\pi E[Y^1|D=1] + (1-\pi)E[Y^1|D=0]\}$$
$$-\{\pi E[Y^0|D=1] + (1-\pi)E[Y^0|D=0]\}$$

→ Essential information is missing for calculating the ATE.

→ Causal inference as a missing data problem!

→ The same holds for the ITE.

→ As a consequence, one always has to make assumptions for identification. This illustrates the central role of assumptions and uncertainty in causal inference.

Observational, not experimental data

Very often we do not have experimental data.

For example, we might want to analyze data for

- Individuals i who use social media heavily (d_i=1)
- Individuals j who use social media less often (d_j=0)

Suppose we

- have a measure Y about their ability to concentrate (from a survey or test)
- decide to compare average Y for the two groups

Estimator: naïve average treatment effect (NATE)

With groups of treated and controls, one might try to estimate the ATE as follows:

$$\widehat{ATE} = E[y_i|d_i = 1] - E[y_j|d_j = 0]$$

→ This simple comparison of treated and controls rarely leads to the correct estimate (exception experiment). Some call it the **naïve average treatment effect (NATE)**

→ If we compare this with the correct equation, a core assumption becomes clear, namely that treatment and control group are comparable.

$$ATE = \{\pi E[Y^1|D = 1] + (1 - \pi)E[Y^1|D = 0]\}$$
$$-\{\pi E[Y^0|D = 1] + (1 - \pi)E[Y^0|D = 0]\}$$

We use one group to approximate the counterfactual value for the other group.

Estimator: naïve average treatment effect (NATE)

The NATE will provide bias in case of selection

$$NATE = E[Y^1|D=1] - E[Y^0|D=0]$$

$$= ATE + \underbrace{\underbrace{E[Y^0|D=1] - E[Y^0|D=0]}_{\text{baseline bias}} + (1-\pi)\underbrace{(ATT - ATC)}_{\text{treatm. effect bias}}}_{\text{selection bias}}$$

selection bias = baseline bias + $(1-\pi)$*treatment effect bias

In our example of social media use:

- Baseline bias: before the treatment both groups might differ in Y
- Treatment effect bias: social media use might affect Y differently for both groups

The conditional independence assumption

- The key requirement for unbiasedness is the **conditional independence assumption (CIA)**

$$\left(Y^0, Y^1\right) \perp D \middle| X$$

- If the CIA holds, the conditional NATE (that is the NATE after conditioning for all relevant Xs) provides an unbiased estimate of the ATE
- But there is no proof that the CIA actually holds

The assignment mechanism

- Since selection leads to biases, estimation of causal effects of a treatment (usually) starts with studying the selection process, also known as **assignment mechanism**

$$Y_i = D_i Y_i^1 + (1 - D_i) Y_i^0$$

- In our example:
  - Which type of individuals use social media how?
  - Which factors explain variation in social media use?
    - Age?
    - Education?
    - …

The conditional independence assumption

What does the CIA imply for working with observational data? $\left(Y^0, Y^1\right) \perp D \mid X$

→ Guided by intuition, theory, and previous reserach identify all Xs that are relevant

→ **But: not just include every X that comes to your mind, but draw a DAG!**

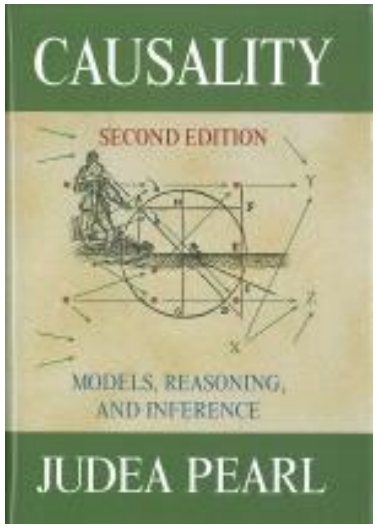
→ ex post adjustment for these X variables

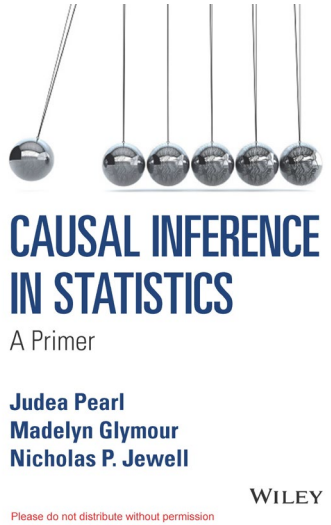
- Stratification (e.g., subgroup analyses, crosstables etc.)
- Regression (condition on X),
- Matching (Compare individuals with same/similar X values),
- Weighting (Weigh T and C in a way that X values are balanced)

→ conditional NATE

Part B
Basics of
Directed Acyclic Graphs

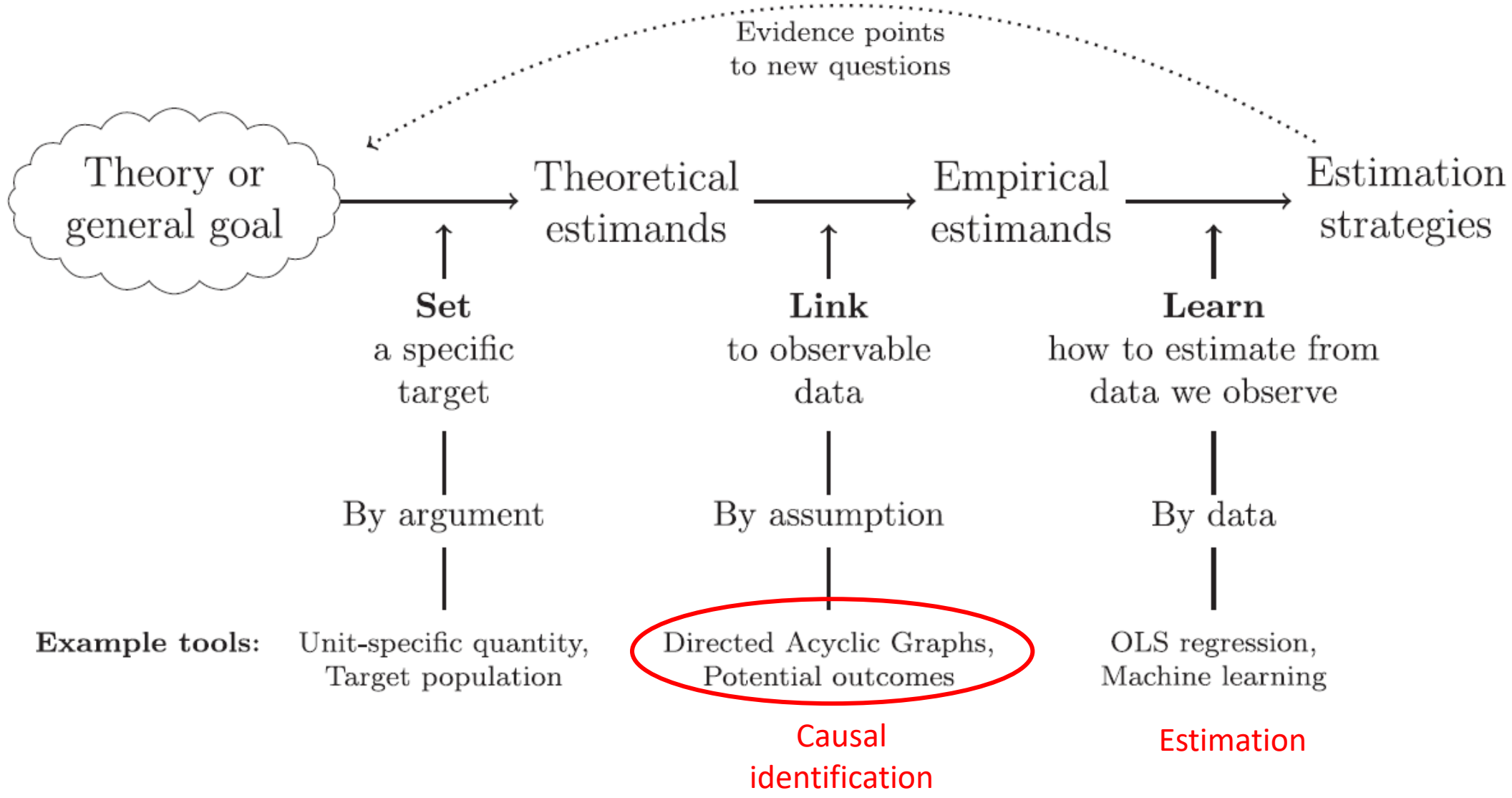
Directed acyclic graphs (DAGs)





- Popular framework in machine learning, statistics, and increasingly the social and behavioral sciences
- Graphical representation of causal relationships

  → visualizes your causal assumptions
- Proposed and advanced by Pearl (2000, 2009, 2019)
- At first glance similar to classical structural equation models, but in contrast to SEMs, no assumptions about functional form of relationships → non-parametric structural equation model (e.g. Y = f(Z))
- Focus on identification of causal effects treating estimation as a distinct step



Lundberg et al. 2021, p. 534

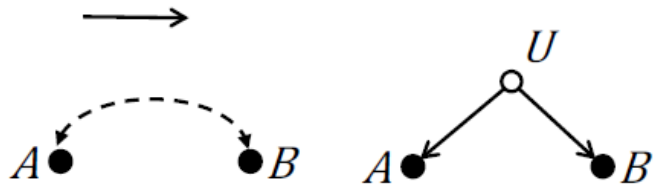
Concepts/terminology

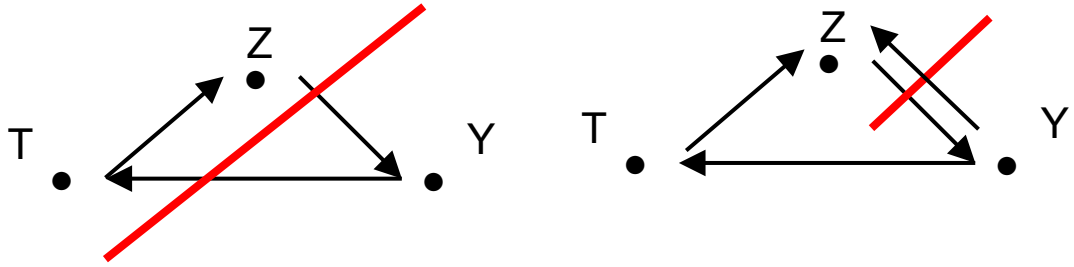
- Nods depict random variables.
- Variables can be observed ● or unobserved ○
- Directed edges (arrows) connect nods
- Bidirectional edges are a shorthand for

  mapping a common influence
- Graphs need to be acylic

→ No cycles

→ No mutual causality at T





→ Drawing a DAG is equivalent to specifying a joint probability distribution of the variables involved in the system (Bayesian network/Hidden Markov Model)

Open paths as a source of bias

A **path** is any connection between two nods via edges irrespective of their direction.

Paths can be:

- **direct** (X → Y) or **indirect** (X → M → Y).
- **causal** (X → M → Y) or **non-causal (**X ← Z → Y or X → Z ← Y).

  [non-causal if at least one arrow points toward T]
- **front door** path (X → M → Y; note: all arrows run towards Y) and **back door** path (**D←Z→Y, D←Z→V←Y,** note: at least one arrow does not run towards Y)

Front door paths and – open or closed – back door paths

Causality flows through **front door** paths:

X → Y

X → M → Y

Biases <u>potentially</u> flow through **back door** paths:

→ A back door path is a source of bias if it is **open**.

D←Z→Y or D←Z1→ Z2→ Y

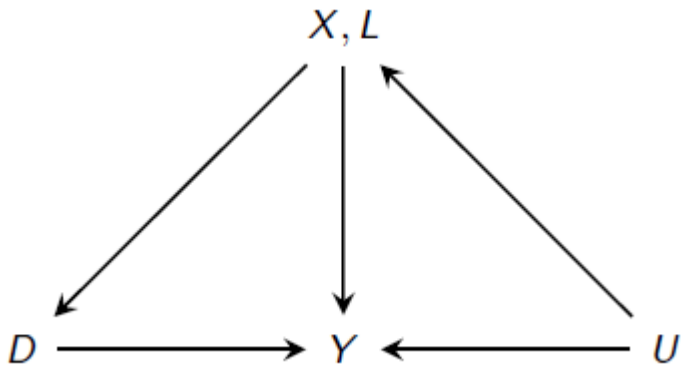
→ To eliminate bias, you should **close** open paths.

D←Z→Y → D←Z→Y

→ **Beware:** some back door paths are already closed, but you might open them

D→C← Y → D→C← Y

Reading Assumptions in a DAG



What are the assumptions in this DAG?

- No edge from U to D
- No edge from Y to U, D to X, D to U … (cycles)

Indeed, all variables except for D and U are already directly connected

Correlation and causality

- Starting point: a correlation between two variables implies that some causal process is going on
- … but not necessarily that one of the two variables causally affects the other
- In a DAG, a correlation implies that there is at least one **open path** between these two variables
- If there is no correlation, then there is either no path or all paths are **blocked**, this is called **d-separated**

  → testable implication of a DAG: zero correlation

Open and blocked paths

Obviously, a direct effect from A to B (A → B) causes an open path.

However, a path between A and B can also exist due to third variables

Structural positions of covariates

- Confounder
- Collider
- Mediator/intervening variable

→ Position affects whether a path is open / closed and hence how to deal with this X

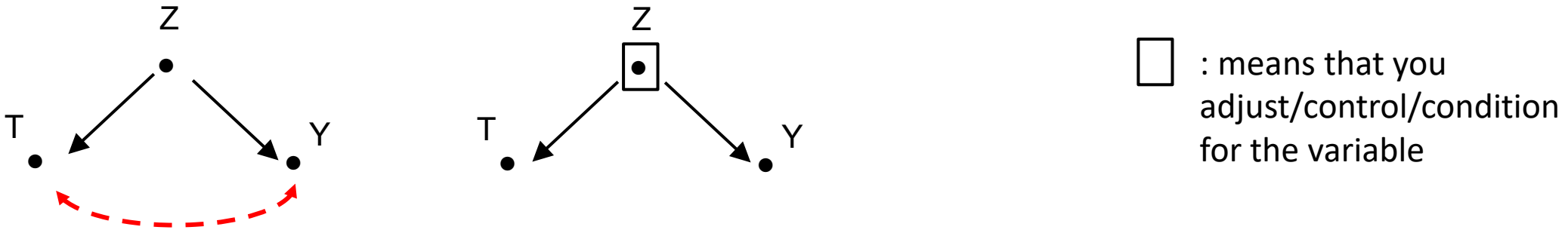
→ Unfortunately, in more complex graphs a variable can have different structural positions at the same time (which can complicate things quite a bit….)

Confounder: A well-known problem

- Common understanding: covariate is associated with treatment and outcome.
- Including this covariate in the model changes the causal effect of interest.

→ Definition too broad: Also includes covariates whose control leads to bias!

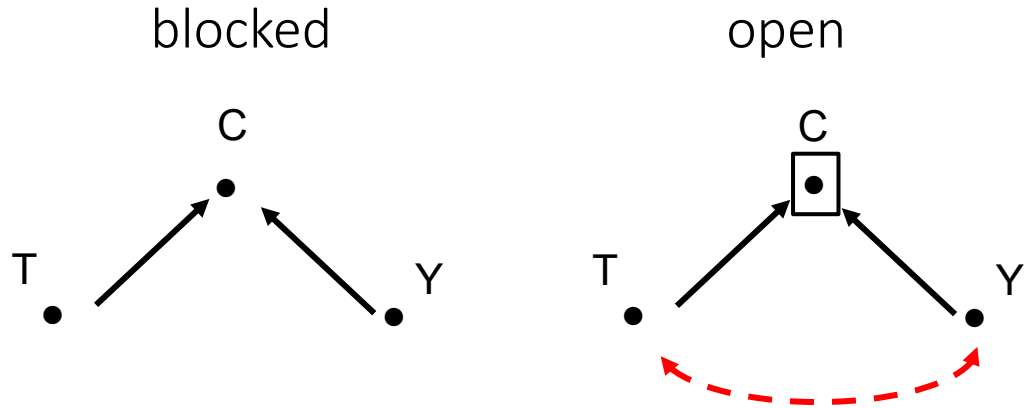
Pearl's concept of the back door path



Collider: A not so well-known problem

Collider: over-control, missing data and selection effects.

- Reverse scenario to the confounder



- well-intended covariate adjustment

  → leads to biased estimates

  → obviously, we need to move away from a "control everything that could potentially be relevant in some way" strategy

Closing open backdoor paths

When should I control for a variable on the path from X to Y?

→ Backdoor criterion: We need to block all open non-causal paths from treatment to outcome

What is a non-causal path?

Which ones are open? Which are already closed?

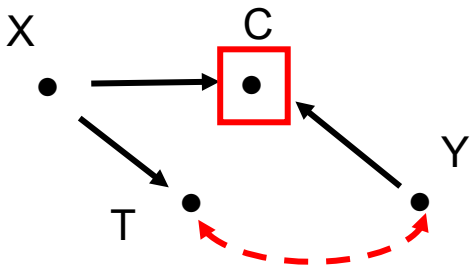
→ Confounder leads to open backdoor path → control for it

→ Collider leads to closed backdoor path → don't control for it

Collider: A not so well-known problem

Collider

- many distortions that have been treated rather disparately so far are special collider problems (Elwert/Winship 2013)



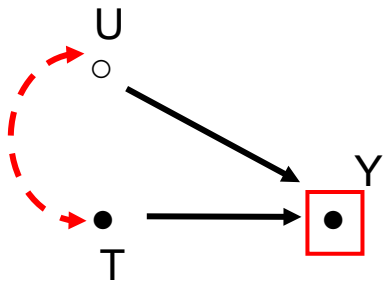
  - Truncation and censoring
  - Missing data (non-response depending on outcome or collider)
  - Selection bias in the Heckman sense
  - Panel attrition
  - Social contagion in networks

  → not only an exceptional case, but a widespread problem

Collider: A not so well-known problem

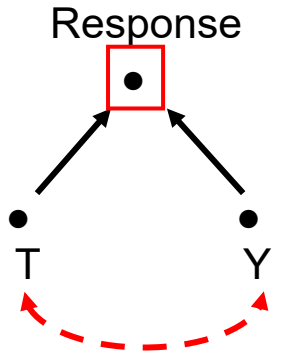
Sample selection on the dependent variable

- Actually an unproblematic model
- But if only a part of the value range of Y can enter the sample then a back-door path opens up
- Example: Truncation of income (no "high earners" in the sample)



Survey Non-Response

- Survey non-response can produce an apparent correlation if both T and Y influence the response
  E.g., education and income

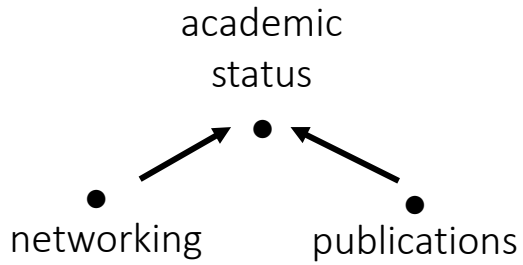


Source: Brüderl
Slides Causal Analysis

Collider: A not so well-known problem

Illustration: Berkson's paradox

- RQ: Does stronger networking of researchers increase publication output?
- Additional assumption: Both factors increase the chance of obtaining a professorship.
- Hypothetical causal structure:



Simulation: a) Networking & publications each exponentially distributed.

b) Networking & publications uncorrelated

c) Networking, publications & random factors influence p(professorship)(31.7%)

Collider: A not so well-known problem

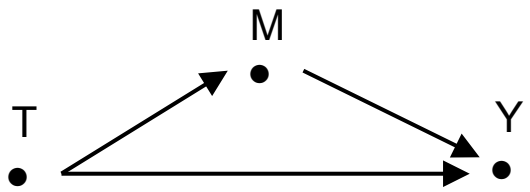
Illustration: Berkson's paradox

	Model 1			***Model 2***
DV: number of publications	***Whole population***	***Professor***	***No professor***	***Whole population***
Network size	0,0004 (-0,31)	-0,025*** (-8,91)	-0,090*** (-11,52)	-0,031*** (-12,48)
Academic status (1 = Professor)				4,858*** (-15,32)
Constant	9,029*** (-85,45)	13,070*** (-35,74)	9,619*** (-71,81)	8,911*** (-85,08)
N	10.000	3.168	6.832	10.000
R^2	0,00001	0,025	0,019	0,023
Adj. R^2	-0,00009	0,024	0,019	0,023

Mediator: well-known, but often mishandled

Mediators

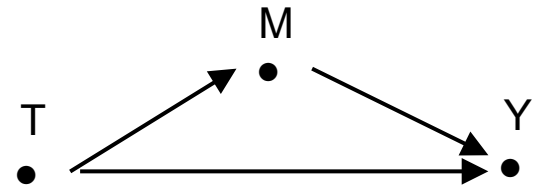
- Pearl: indirect effect via front-door pathway
- Example with direct and indirect effect



- Desirable because
  - deeper understanding of generative mechanisms (especially important for impact evaluation and transfer of measures)
  - Sometimes: only possibility to identify causal effects

Mediator: well-known, but often mishandled

Unfortunately, a too-common misunderstanding:



If there is an effect of T on Y, which is disappears after controlling for M, this does not mean that T has no effect on Y.

It means, T has no direct effect on Y and that all/most of the total effect flows through M (indirect effect).

Example: Physical appearance & job interview invites

Job vacancies

Editor for a Magazine in the Area of

[Automotive / Home Ideas & Decoration]

Our company is a renowned specialized publisher based in Nuremberg. In our house, a monthly magazine is released with a focus on [automotive / home ideas & decoration].

Tasks:

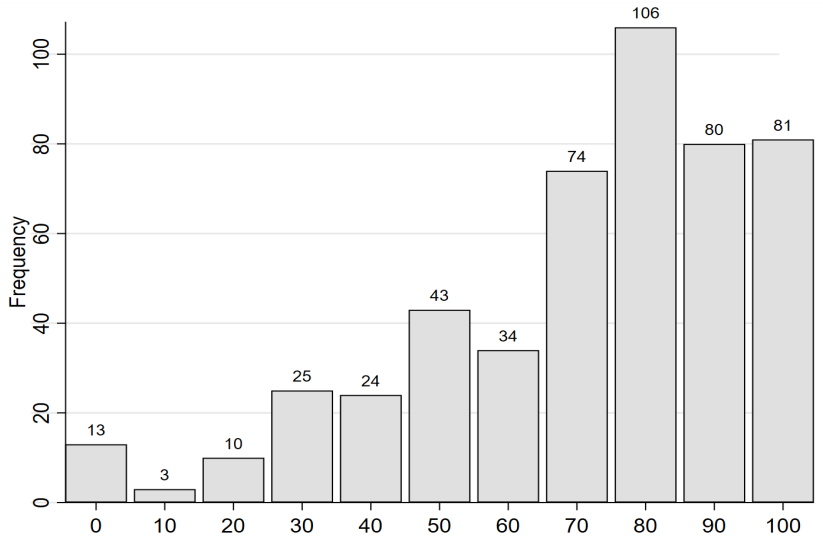
- Writing own articles and reports as well as proofreading contributions
- Idea generation and conception of relevant new topics
- Coordination within the editorial team

Required Qualifications:

- Several years of experience in the conceptual and practical production of magazines
- Extensive specialist knowledge
- High degree of autonomy
- Reliability
- Leadership and social skills

Deepfaked application videos

Probability (invite to interview)



Kühn & Wolbring, 2024

Example: Physical appearance & job interview invites

	(1) Treatment	(2) incl. Controls	(3) incl. Competence
Ref: Less attractive applicant			
More attractive applicant	5.717** [1.41,10.02]	5.797** [1.42,10.17]	0.345 [-2.98,3.67]
Ref: Male applicant			
Female applicant	-5.197* [-9.52,-0.87]	-5.400* [-9.77,-1.03]	-6.755*** [-10.08,-3.43]
Ref: Home Ideas & Deco			
Automobile	1.662 [-2.62,5.94]	1.849 [-2.45,6.15]	1.142 [-2.02,4.30]
More attractive applicant # Female applicant			
Index of Competence			14.774*** [12.97,16.58]
Controls: Respondent gender, age, hiring experience		included	included
Constant	70.056*** [65.61,74.51]	70.166*** [57.88,82.45]	-6.198 [-19.99,7.59]
Observations	493	493	493
R^2	0.027	0.036	0.454
Adjusted R^2	0.021	0.016	0.442

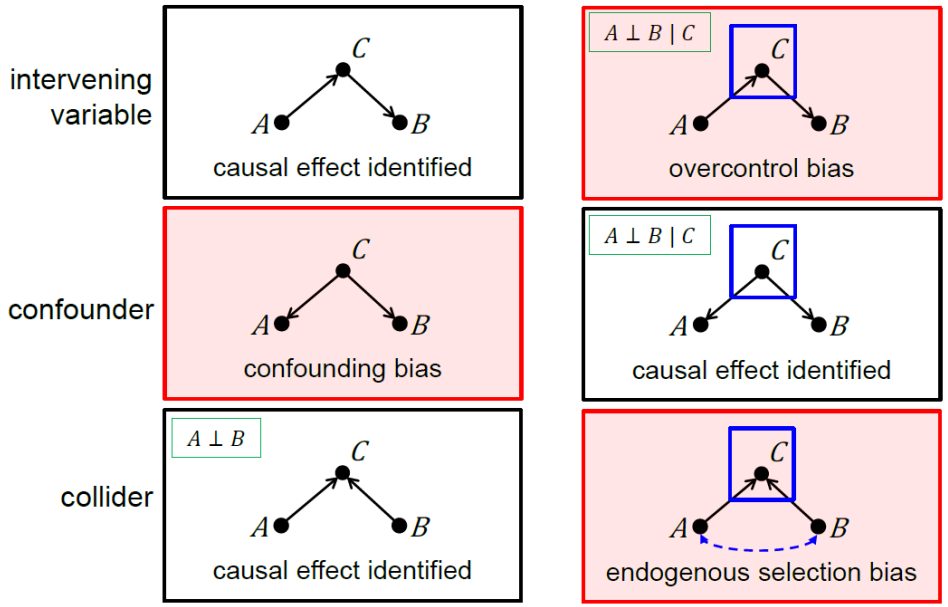
Kühn & Wolbring, 2024

Open and blocked paths

Structural positions of covariates

- Mediator → Risk of over-control (if interested in total effect)
- Confounder → Risk of under-control
- Collider → Risk of over-control

→ Affects how to deal with covariates



Different identification strategies

Identification by design: exogenous variation in the treatment

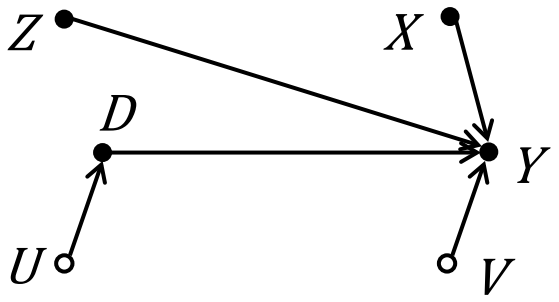
Identification by design with the prototype “experiments” with three features:

(1) At least one treatment and one control group

(2) Active intervention – researcher has control over treatment assignment

(3) Random assignment of treatment

→Treatment assignment by mechanism D is exogenous: $(Y^0, Y^1) \perp D$



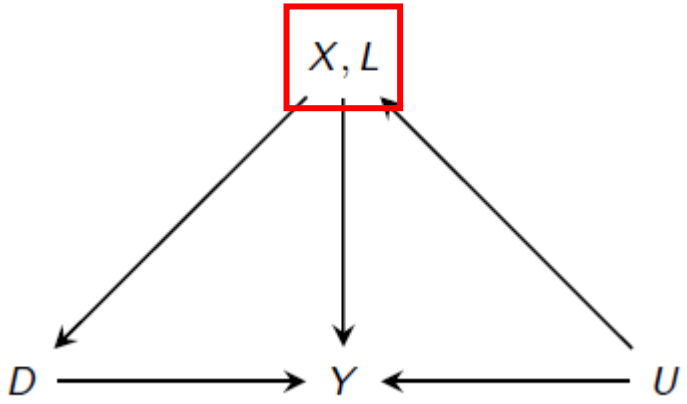
→Other identification strategies such as instrumental variables, natural experiments, and regression-discontinuity designs try to mimick the experimental approach

Identification by back door conditioning

Back door conditioning is most common with observational data.

The idea is to close all the open back door paths.

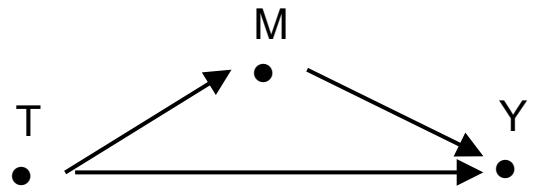
If all open back door paths closed, the CIA holds.



Identification via front door paths

Mediators

- Basic idea: 3-stage procedure
  1) Estimation of the effect T → M
  2) Estimation of the effect M → Y
  3) Combining estimates 1) and 2)



Important assumption: Isolability of mechanisms

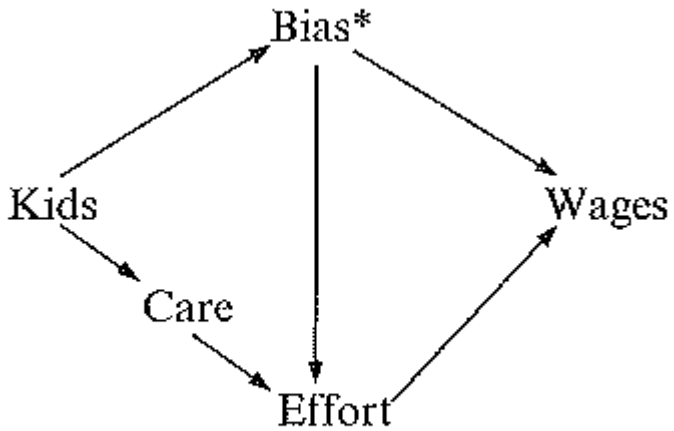
→ often unrealistic in practice (also in experiments)

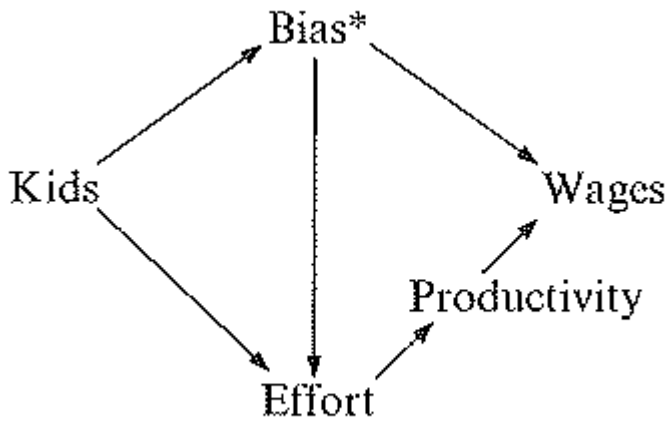
→ Current debate on appropriate methods and designs, mainly influenced by Pearl's perspective (e.g. in Psych. Methods; blog update by David A. Kenny in October 2013)

Identification via front door paths

Mediators: A more complex example

- Motherhood and Wages





Quelle: Knight/Winship 2013

Part C
DAGs in action

Application

Weight & Wages

Economics and Human Biology 9 (2011) 356–363

Contents lists available at ScienceDirect

Economics and Human Biology

journal homepage: http://www.elsevier.com/locate/ehb

Fat, muscles, and wages

Christiane Bozoyan *, Tobias Wolbring [1]

LMU Munich, Institute of Sociology, Konradstraße 6, 80801 Munich, Germany

Schmollers Jahrbuch 135 (2015), 83 – 96
Duncker & Humblot, Berlin

The Usefulness of Directed Acyclic Graphs: What Can DAGs Contribute to a Residual Approach to Weight-Related Income Discrimination?*

By Christiane Bozoyan and Tobias Wolbring

European Sociological Review, 2018, 1–14
doi: 10.1093/esr/jcy009
Original article

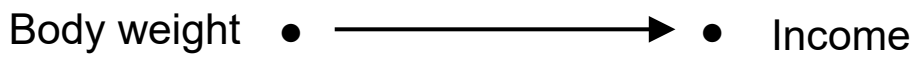
OXFORD

The Weight Wage Penalty: A Mechanism Approach to Discrimination

Christiane Bozoyan[1] and Tobias Wolbring[2,*]

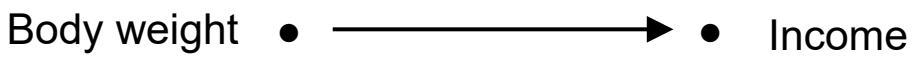
Weight and wages

Theoretical estimand



Weight and wages

Theoretical estimand



Causal Identification of empirical estimand

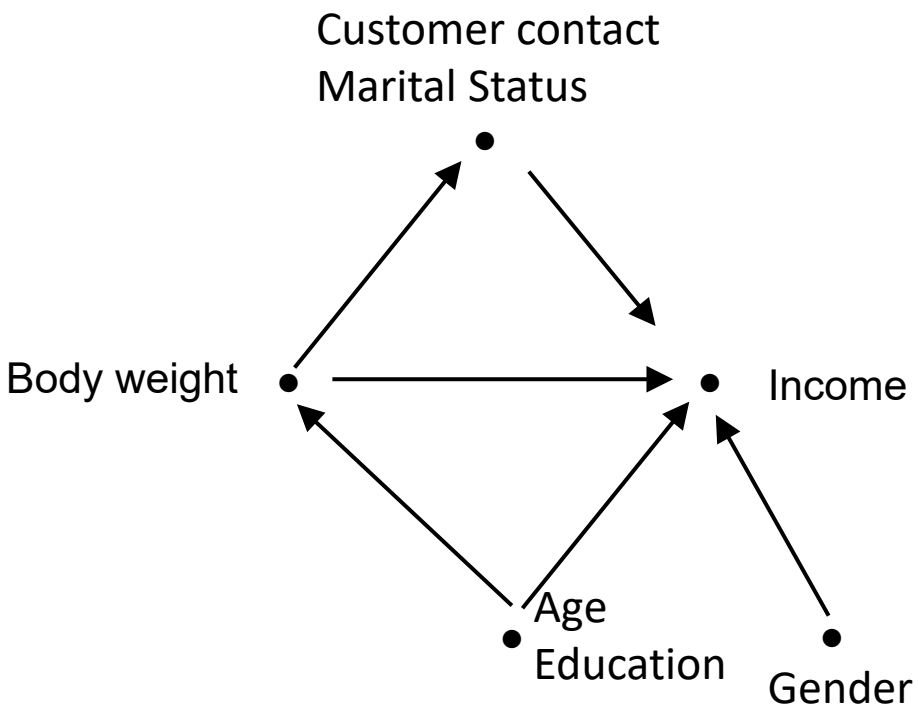
You consider to include the following variables in the model for causal identification:

- Gender (male/female/other)
- Age (in years)
- Customer contact of job (yes/no)
- High school education (yes/no)
- Marital status (married/ not married)

However, before you estimate the model, you want to draw a DAG for these variables to make sure you get the model right.

Weight and wages

Causal Identification of empirical estimand



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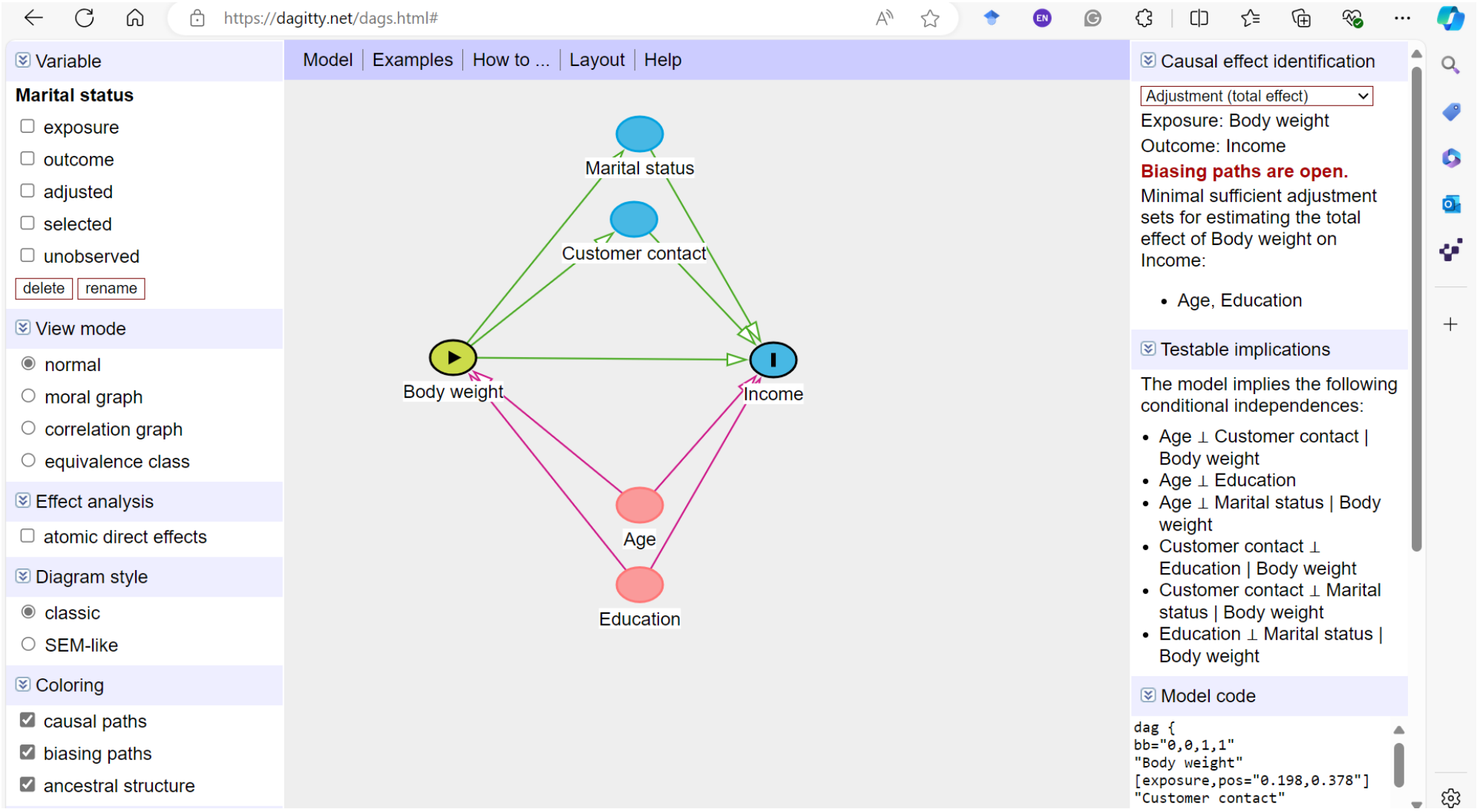
However, before you estimate the model, you want to draw a DAG for these variables to make sure you get the model right.

DAGitty — draw and analyze causal diagrams

Software to facilitate covariate selection

- www.dagitty.net (Knüppel/Stang 2010; Textor et al. 2011)
- all minimal subsets of covariates whose control is sufficient to identify the causal effect (minimal sufficient adjustment sets).

DAGitty — draw and analyze causal diagrams



Total effect/ direct effect/ …

Testable implication

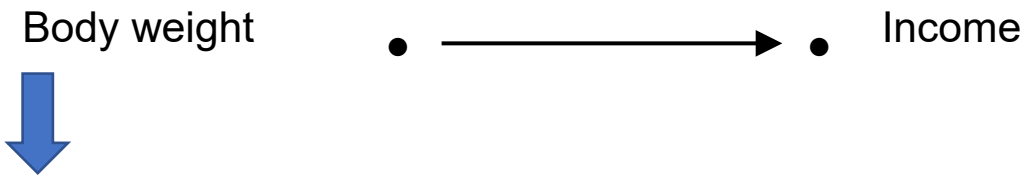
R code for reproducing the model

DAGitty — draw and analyze causal diagrams

```
dag {
bb="0,0,1,1"
"Body weight" [exposure,pos="0.198,0.378"]
"Customer contact" [pos="0.413,0.190"]
"Marital status" [pos="0.420,0.074"]
Age [pos="0.420,0.579"]
Education [pos="0.420,0.688"]
Income [outcome,pos="0.579,0.381"]
"Body weight" -> "Customer contact"
"Body weight" -> "Marital status"
"Body weight" -> Income
"Customer contact" -> Income
"Marital status" -> Income
Age -> "Body weight"
Age -> Income
Education -> "Body weight"
Education -> Income
}
```

Weight and wages

Theoretical estimand



Empirical estimand

You consider to include the following variables in the model for causal identification:

- Gender (male/female/other)
- …

→ Think about the causal structure, draw a causal graph and analyze it (e.g. with DAGitty).

Estimation

- Do you compare means? Or do you run a regression?
- How do you operationalize the variables? (e.g., dummy for obesity, hourly wage)
- Do you log Y? Do you use polynomes for age?

Time for
an exercise

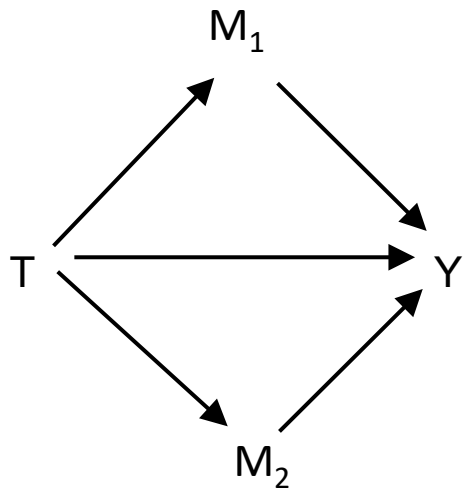
Exercise

Please analyze the following graph with your seat neighbour(s).

A1) What do you need to control for estimating the total causal effect of T on Y?

A2) What do you need to control for estimating the direct causal effect of T on Y?

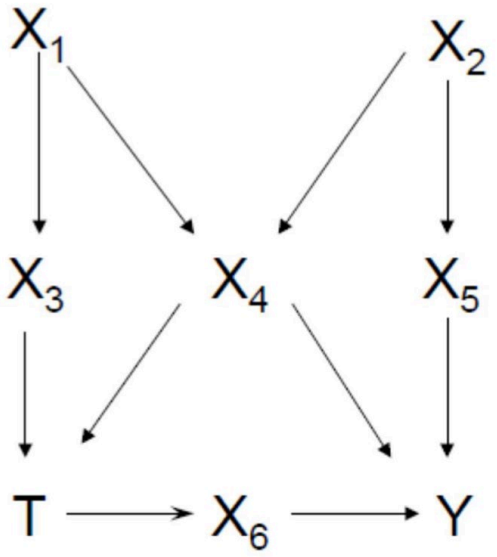
A3) There is a way to determine the indirect effect via M1. Any idea how you could do it?



Note: The example could be the effect of having kids on wages. The two mediators could be biases by employers and effort by employees.

Exercise

B1) Please analyze the following graph by visual inspection. Which variables would you control to estimate the effect of T on Y?



B2) Now draw the same graph in DAGitty and interpret the output of DAGitty with respect to potential control variables.

Graph for exercise B

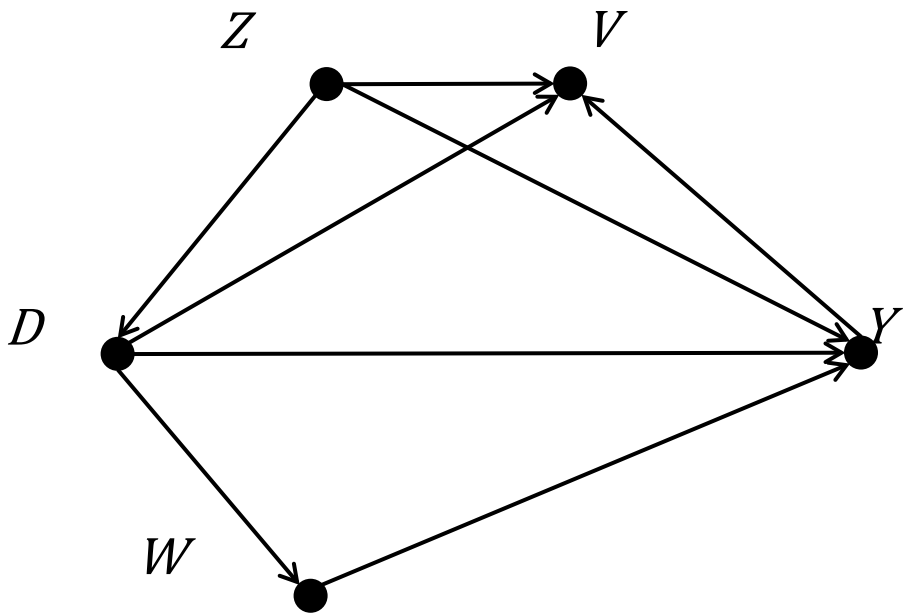
```
dag {
bb="0,0,1,1"
T [exposure,pos="0.212,0.477"]
X1 [pos="0.205,0.111"]
X2 [pos="0.496,0.099"]
X3 [pos="0.208,0.301"]
X4 [pos="0.374,0.298"]
X5 [pos="0.506,0.302"]
X6 [pos="0.368,0.482"]
Y [outcome,pos="0.518,0.480"]
T -> X6
X1 -> X3
X1 -> X4
X2 -> X4
X2 -> X5
X3 -> T
X4 -> T
X4 -> Y
X5 -> Y
X6 -> Y
}
```

Exercise

We want to identify the causal effect of D on Y

C1) Which paths are causal? Which paths are non-causal (backdoor paths)?

C2) Which backdoor paths are open? Which are closed?



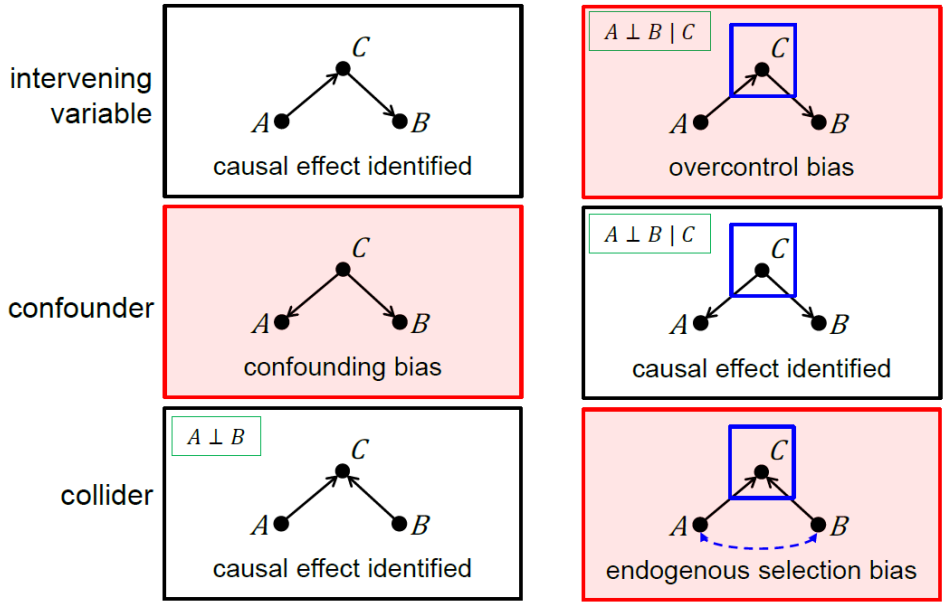
Graph for exercise C

```
dag {
bb="0,0,1,1"
D [exposure,pos="0.218,0.493"]
V [pos="0.496,0.266"]
W [pos="0.358,0.671"]
Y [outcome,pos="0.587,0.488"]
Z [pos="0.291,0.267"]
D -> V
D -> W
D -> Y
W -> Y
Y -> V
Z -> D
Z -> V
Z -> Y
}
```

Summary

- Graph methodology as a link between theory and statistical analysis
- Clear explication of the assumptions and the limits of identification
- Both risk of under-control and over-control

  → no "control everything" strategy, but need to think about causal structure



Recommended reading

Cinelli, C., Forney, A., & Pearl, J. (2024). A Crash Course in Good and Bad Controls. Sociological Methods & Research, 53(3), 1071-1104.

Elwert, Felix (2013): Graphical Causal Models. In: Morgan, Stephen L. (Hg.): Handbook of Causal Analysis for Social Research. Dordrecht: Springer, S. 245-273.

Elwert, Felix/Winship, Christopher (2014): Endogenous Selection Bias: The Problem of Conditioning on a Collider Variable. Annual Review of Sociology 2014 40:1, 31-53.

Morgan, Stephen L./Winship, Christopher (2015): Counterfactuals and Causal Inference. Methods and Principles for Social Research. 2. edition. Cambridge, MA: Cambridge University Press.

Schuessler, J., & Selb, P. (2023). Graphical Causal Models for Survey Inference. Sociological Methods & Research, 1-32. https://doi.org/10.1177/00491241231176851

Recommended reading: more comprehensive treatments

Hernán MA, Robins JM (2020). Causal Inference: What If. Boca Raton: Chapman & Hall/CRC."

Pearl, Judea (2009): Causality: Models, Reasoning, and Inference. Cambridge: Cambridge University Press (2. ed.).

Pearl, Judea, Glymour, Madelyn and Nicholas P. Jewell (2016) Causal Inference in Statistics: A Primer, Wiley.

Pearl, Judea and Dana Mackenzie (2018): The Book of Why: The New Science of Cause and Effect, New York: Basic Books.

VanderWeele. T.J. (2015). Explanation in Causal Inference: Methods for Mediation and Interaction. Oxford University Press.

Recommended reading: DAGitty

Knüppel Sven/Stang Andreas (2010): DAG Program: Identifying Minimal Sufficient Adjustment Sets. In: Epidemiology, 21(1), S. 159.

Textor, Johannes/Hardt,Juliane/Knüppel, Sven (2011): Letter to the Editor: DAGitty: A Graphical Tool for Analyzing Causal Diagrams. In: Epidemiology, 22(5), S. 745.

Textor, Johannes/ Liśkiewicz, Maciej (2011): Adjustment Criteria in Causal Diagrams: An Algorithmic Perspective. In: Proceedings of the 27th Conference on Uncertainty in Artificial Intelligence (UAI 2011), S. 681-688

Thank you
for your attention!

Feel free to contact me if you have questions: tobias.wolbring@fau.de