University of Antwerp
Faculty of Social Sciences

DAGs and PP

or commonly know as:
"a bit more transparent way to state
your research assumptions and questions"

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(Josema, for the friends)

May 10, 2022

What are we going to talk about? I

1. About research
   - A typical scientific lab
   - Research hypothesis production
2. DAGs and PP
3. Example cases
   - Experimental design: the panacea
   - Simulation conventions
   - Fork bias: spurious relationships
   - Fork bias: masked relationships (a)
   - Fork bias: masked relationships (b)
   - Fork bias: multicollinearity

What are we going to talk about? II

- No more fork bias: neutral control
- Pipe bias: precision parasite
- Pipe bias: post-treatment
- Pipe bias: Simpson's paradox
- No pipe/fork bias: good controls
- Pipe/Fork bias: bias amplification
- Collider bias: Berkson's paradox
- Collider bias: M-bias
- XXX bias: sensitivity analysis
- XXX bias: post-stratification

What are we going to talk about? III

- Descendant fix: proxies (a)
- Descendant fix: proxies (b)
- Descendant bias: case control

4 Concluding remarks

5 Do you wanna know more???

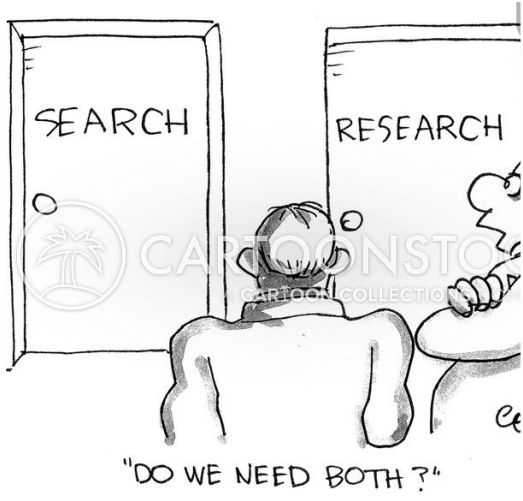
1. About research

A typical scientific lab

A typical scientific lab[1]

What is needed?

1. Quality of theory
2. Quality of data
3. Reliable procedures and code
4. Quality of data analysis
5. Documentation
6. Reporting



[1] McElreath [12], lecture 20 and McElreath [13], chapter 17

A typical scientific lab

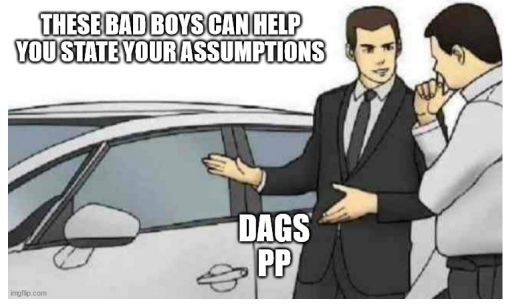
What we “normally” focus on?

1. Quality of theory
2. Quality of data
3. Reliable procedures and code
4. Quality of data analysis
5. Documentation
6. Reporting

A typical scientific lab

What can be improved?[a]
(with DAGs and PP)

1. Quality of theory
2. Quality of data
3. Reliable procedures and code
4. Quality of data analysis
5. Documentation
6. Reporting



[a] see Yarkoni [21] on a discussion on how the failure in alignment between verbal and statistical expressions is related to psychology's replication crisis.

1. About research

Research hypothesis production

Research hypothesis production

Well known challenges[a]

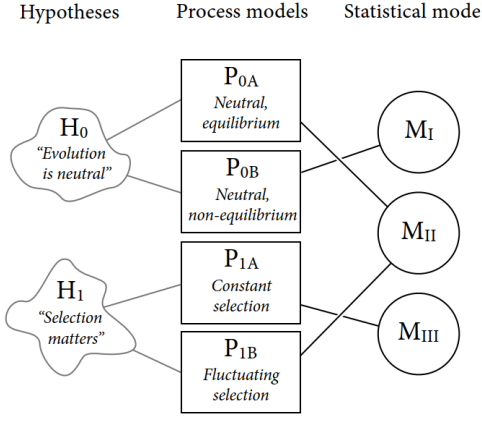
- Insufficient data
- Wrong population
- Measurement error
- Selection bias
- Confounding

[a] Hernán [8], lesson 4

Research hypothesis production

but we should not forget[a]

- No one-to-one relationship exists between our process models and statistical models,
- Nor between our hypothesis and a process models

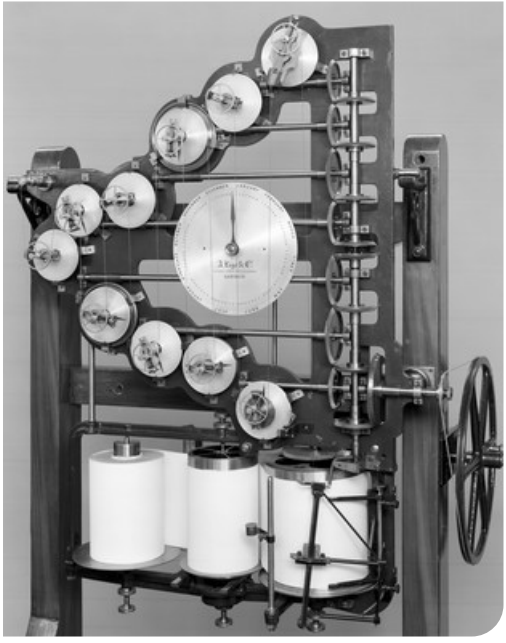


[a] Figure 1.2 reproduced from chapter 1 McElreath [13]

Research hypothesis production

and also

- statistical models are just "machines to find association", not a reliable reflection of the theory (I can prove it!!).



Research hypothesis schematics[2]

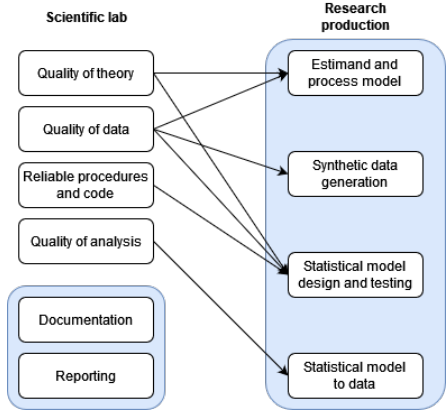
a. Estimand and process model
b. Synthetic data generation
c. Statistical model design and testing
d. Apply statistical model to data

[2] McElreath [13], lecture 20, Pearl [16]. Follow Fogarty et al. [6] on item (c).

Research hypothesis schematic

Where does it match with the previous?

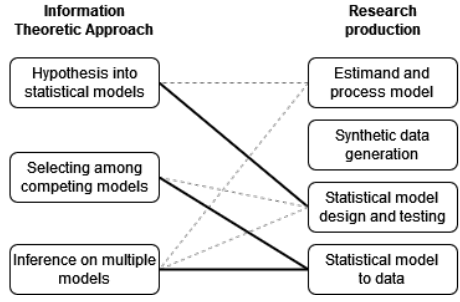
a. Estimand and process model maps 1 (theory) and 2 (data) to a heuristic model.
b. Synthetic data generation maps 2 (data) to an idealized data.
c. Statistical model design and testing maps 1 (theory), 2 (data), and 3 (reliable code) to an statistical model.
d. Apply statistical model to data maps 4 (analysis) onto a result.



Where does the ITA fit?

Information Theoretic Approach (ITA) is framework to select among competing models [1, 3]:

1. Hypothesis into statistical models,
   (how about a process model?)
2. Select among competing models,
   (do the code works as intended?)
3. Make inferences based on one or multiple models.
   (do the code works as intended?, are there variables that can bias our results?)



2. DAGs and PP

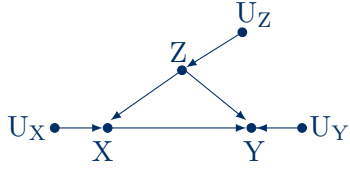
DAGs and PP

- Directed acyclic graphs (DAGs), are a type of structural causal model (SCM) [15, 4]
- DAGs can be represented by a structural model, and its associated causal diagram[a].
- we put distributional assumptions to the structural model through probabilistic programming (PP) [10]. (more in part 3)

[a] reproduced from Cinelli et al. [4].

$$M = \begin{cases} Z \leftarrow f_Z(U_z) \\ X \leftarrow f_X(Z, U_x) \\ Y \leftarrow f_Y(X, Z, U_y) \\ U \sim P(\mathbf{U}) \end{cases}$$

(a) structural model



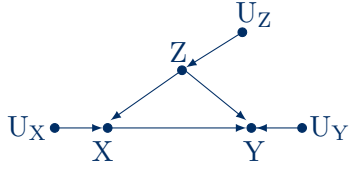
(b) causal diagram

DAGs and PP

- $\mathbf{V} = \{Z, X, Y\}$ are called endogenous variables.
- $\mathbf{U} = \{U_Z, U_X, U_Y\}$ are called exogenous variables.
  (drawn when strictly required)
- $\mathbf{F} = \{f_Z, f_X, f_Y\}$ are called structural equations.

$$M = \begin{cases} Z \leftarrow f_Z(U_Z) \\ X \leftarrow f_X(Z, U_X) \\ Y \leftarrow f_Y(X, Z, U_Y) \\ U \sim P(\mathbf{U}) \end{cases}$$

(a) structural model



(b) causal diagram

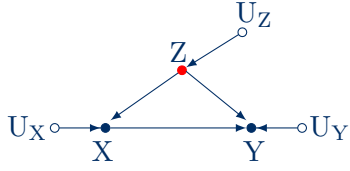
DAGs and PP

Causal diagram conventions [4],

- black nodes are observed variables.
- white nodes are unobserved variables.
- red nodes are variables for which we will decide its inclusion or not.

$$M = \begin{cases} Z \leftarrow f_Z(U_Z) \\ X \leftarrow f_X(Z, U_X) \\ Y \leftarrow f_Y(X, Z, U_Y) \\ U \sim P(\mathbf{U}) \end{cases}$$

(a) structural model



(b) causal diagram

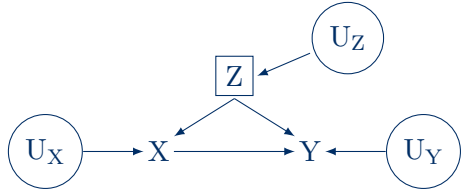
DAGs and PP

Other causal diagram conventions,

- no circle nodes are observed variables.
- circled nodes are unobserved variables.
- squared nodes are variables for which we will decide its inclusion or not.

$$M = \begin{cases} Z \leftarrow f_Z(U_Z) \\ X \leftarrow f_X(Z, U_X) \\ Y \leftarrow f_Y(X, Z, U_Y) \\ U \sim P(\mathbf{U}) \end{cases}$$

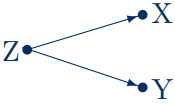
(a) structural model



(b) causal diagram

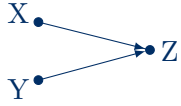
The benign case of DAG elementals

For everything can be depicted with them

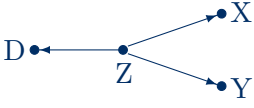


(a) fork

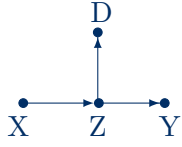
(b) pipe



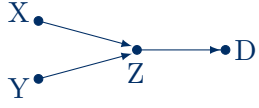
(c) collider



(d) descendant on fork



(e) descendant on pipe



(f) descendant on collider

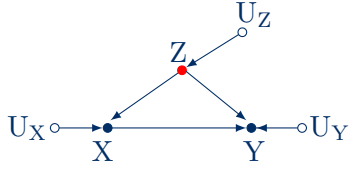
About D-separation

Causal graph theory [14, 15, 17, 18, 19],

1. descendant (child, grandchild), parent (grandparent).
   (path specific)
2. paths (directional, non-directional).
3. paths are blocked or open according to the D-separation rules.
   (also path specific)
4. there are only four (4) D-separation rules.

$$M = \begin{cases} Z \leftarrow f_Z(U_Z) \\ X \leftarrow f_X(Z, U_X) \\ Y \leftarrow f_Y(X, Z, U_Y) \\ U \sim P(\mathbf{U}) \end{cases}$$

(a) structural model



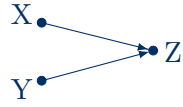
(b) causal diagram

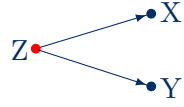
About D-separation

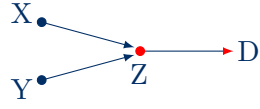
The D-separation (Directional) rules [8],

1. If no variables being conditioned on, a path is blocked if and only if, two arrowheads on the path collide at some variable on the path.
2. Any path that contains a noncollider that has been conditioned on, is blocked (backdoor path)[a].
3. A collider that has been conditioned on does not block a path.
4. A collider that has a descendant that has been conditioned on does not block a path.

[a] there is also a front-door path (if you wonder).



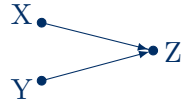


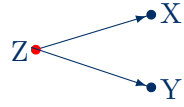


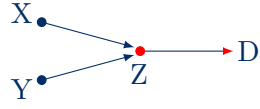
About D-separation

The D-separation rules implications,
(independent of distributional assumptions)

1. $X \perp\!\!\!\perp Y \Longrightarrow$
   $P(X, Y) = P(X) \cdot P(Y)$
2. $X \perp\!\!\!\perp Y|\ Z \Longrightarrow$
   $P(X, Y|Z) = P(X|Z) \cdot P(Y|Z)$
   (same for fork or pipe)
3. $X \not\perp\!\!\!\perp Y|\ Z \Longrightarrow$
   $P(X, Y|Z) \neq P(X|Z) \cdot P(Y|Z)$
4. $X \not\perp\!\!\!\perp Y|\ D \Longrightarrow$
   $P(X, Y|D) \neq P(X|D) \cdot P(Y|D)$

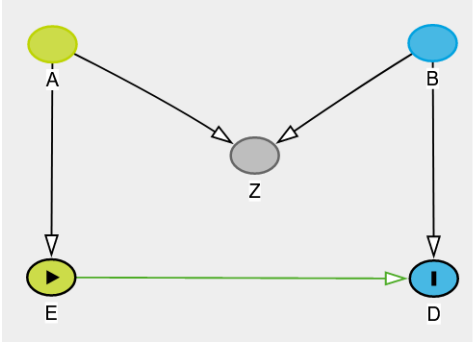






Oh DAGitty!! mijn vriendin

- browser (R package) environment for creating, editing, and analyzing causal diagrams [20].
- available online: http://dagitty.net
- But there are more fish in the sea: http://www.causalfusion.net [2] (b**** better have my $$$)



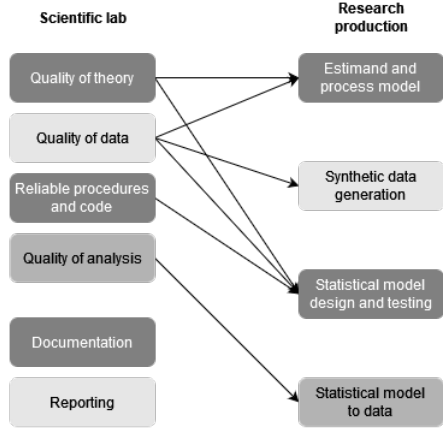
Where do DAGs and PP fit?

starts with:

- A clear definition of the estimand and process model (assumptions).
- An improved the reliability of your procedures.
- As a documentation procedure.

and leads to:

- A sound analysis, and result (even when we cannot have an answer to our question)
- An improved planning to get data.



3. Example cases

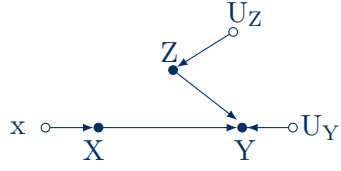
Experimental design: the panacea

Experimental design[3]

- Purpose: to control all factors responsible for the outcome's variation.
  (understand the system)
- It is modeled by modifying the structural model (and causal diagram).

$$M = \begin{cases} Z \leftarrow f_Z(U_Z) \\ X \leftarrow f_X(x) \\ Y \leftarrow f_Y(X, Z, U_Y) \\ U \sim P(\mathbf{U}) \end{cases}$$

(a) structural model



(b) causal diagram

[3] Cinelli et al. [4], appendix A (p. 15)

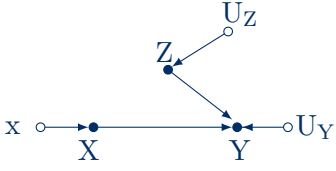
Experimental design

- intervention on X can be written in do-calculus[a] as: $P(\mathbf{V} \mid do(X = x))$.
- remember:
  $\mathbf{V} = \{Z, X, Y\}$,
  $\mathbf{U} = \{U_Z, U_X, U_Y\}$, and
  $\mathbf{F} = \{f_Z, f_X, f_Y\}$.

[a] an appropriate treatment can be found with the usual suspects [14, 15, 17, 18])

$$M = \begin{cases} Z \leftarrow f_Z(U_Z) \\ X \leftarrow f_X(x) \\ Y \leftarrow f_Y(X, Z, U_Y) \\ U \sim P(\mathbf{U}) \end{cases}$$

(a) structural model



(b) causal diagram

Effects of interest

two types of effects,

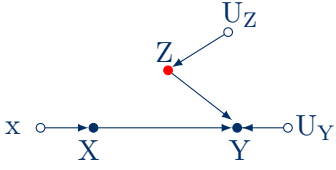
1. Average causal effect:
   $\text{ACE}(x) = \text{E}[Y|\text{do}(x+1)] - \text{E}[Y|\text{do}(x)]$
2. Controlled direct effect:
   $\text{CDE}(x, z) = \text{E}[Y|\text{do}(x+1), \text{do}(z)] - \text{E}[Y|\text{do}(x), \text{do}(z)]$

points to consider:

- CDE takes a particular relevance with observational data.
- There is also a distinction between total effect and direct effect.

$$M = \begin{cases} Z \leftarrow f_Z(U_Z) \\ X \leftarrow f_x(x) \\ Y \leftarrow f_Y(X, Z, U_Y) \\ U \sim P(\mathbf{U}) \end{cases}$$

(a) structural model



(b) causal diagram

3. Example cases

Simulation conventions

Simulation conventions

one way to defined it,

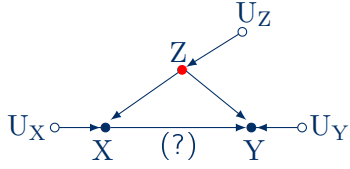
$$
\begin{aligned}
Z &= U_Z && ; U_Z \sim N(0, \sigma_Z) \\
X &= \beta_Z Z + U_X && ; U_X \sim N(0, \sigma_X) \\
Y &= \beta_Z Z + \beta_X X + U_Y && ; U_Y \sim N(0, \sigma_Y)
\end{aligned}
$$

a more succinct way,

$$
\begin{aligned}
Z &\sim N(0, \sigma_Z) \\
X &\sim N(\beta_Z Z, \sigma_X) \\
Y &\sim N(\beta_Z Z + \beta_X X, \sigma_Y)
\end{aligned}
$$

$$
M = \begin{cases}
Z \leftarrow f_Z(U_Z) \\
X \leftarrow f_X(Z, U_X) \\
Y \leftarrow f_Y(Z, X, U_Y) \\
U \sim P(\mathbf{U})
\end{cases}
$$

(a) structural model



(b) causal diagram

3. Example cases

Fork bias: spurious relationships

Spurious relationships[4]

also known as,

- spurious association
- confounder
- an instance of fork bias

research question,

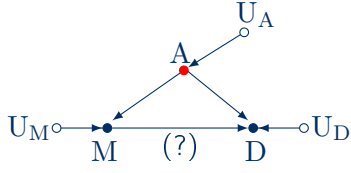
- Does M has a (direct) effect on D?

variables,

- A, median age at marriage
- M, marriage rate
- D, divorce rate

$$M = \begin{cases} A \leftarrow f_A(U_A) \\ M \leftarrow f_M(A, U_M) \\ D \leftarrow f_D(A, M, U_D) \\ U \sim P(\mathbf{U}) \end{cases}$$

(a) structural model



(b) causal diagram

[4] McElreath [12], chapter 5 (p. 125)

Simulation setting

```r
# sim
A = rnorm( 100 )
M = rnorm( 100 , mean=-1*A )
D = rnorm( 100 , mean=-1*A + 0*M )
d = data.frame(A=A,M=M,D=D)
```

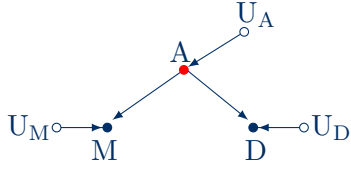
(c) R code

$$M = \begin{cases} A \leftarrow f_A(U_A) \\ M \leftarrow f_M(A, U_M) \\ D \leftarrow f_D(A, U_D) \\ U \sim P(\mathbf{U}) \end{cases}$$

(a) structural model

Implications,

- $M \not\perp\!\!\!\perp D$
- $M \perp\!\!\!\perp D \mid A$

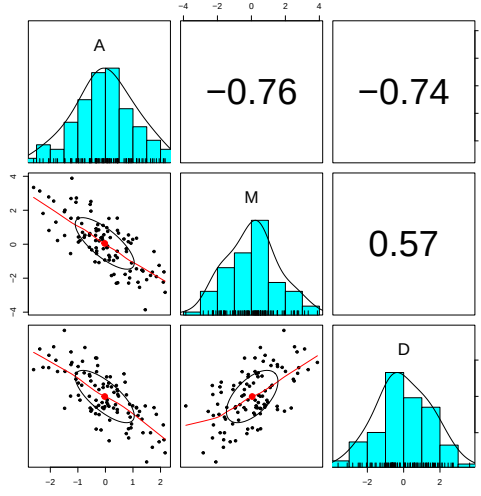


(b) causal diagram

“Eyeballing” analysis

based on correlation analysis,

- $\text{cor}(\text{A}, \text{D}) < 0$ and $\text{cor}(\text{M}, \text{D}) > 0$ goes in line of our “rudimentary” understanding of the data.
- why there is $\text{cor}(\text{M}, \text{D}) > 0$?
  (hint: univariate correlation)
- we include M as a covariate in our statistical model
  (is our research hypothesis)



Regression, regression!!

based on statistical analysis,

- we have two different stories, (which one is the “truth”?)

```
> summary(lm(D ~ M, data=d)) # spurious relation

Call:
lm(formula = D ~ M, data = d)

Residuals:
     Min       1Q   Median       3Q      Max
-2.80012 -0.90447 -0.03866  0.80220  2.82970

Coefficients:
            Estimate Std. Error t value Pr(>|t|)
(Intercept) -0.23298    0.12412  -1.877   0.0635 .
M            0.40233    0.08986   4.477 2.04e-05 ***
> summary(lm(D ~ A + M, data=d)) # controlled relation

Call:
lm(formula = D ~ A + M, data = d)

Residuals:
     Min       1Q   Median       3Q      Max
-2.27295 -0.68174  0.03781  0.78885  2.95320

Coefficients:
            Estimate Std. Error t value Pr(>|t|)
(Intercept) -0.18854    0.09871  -1.910   0.0591 .
A           -1.03121    0.13483  -7.648 1.49e-11 ***
M           -0.06134    0.09362  -0.655   0.5139
```

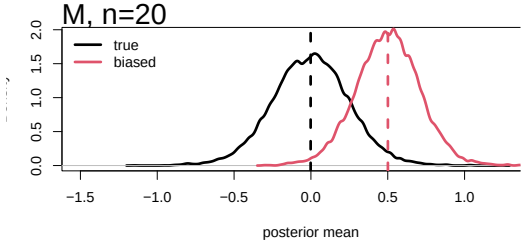
I'll get more data!!

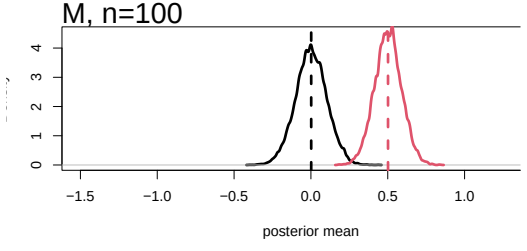
imagine we can continue sampling,

- top: $10,000$ samples $n = 20$
- bottom: $10,000$ samples $n = 100$

under the incorrect model,
the larger the sample size,

- the more certain you are about your biased estimates
  (the winner's curse)





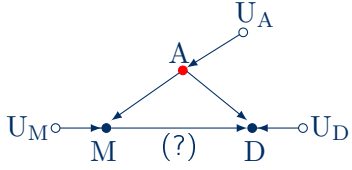
The dream team!!

based on DAG and statistical model,

- the 2nd D-separation rule requires you to control any noncollider to block the backdoor path, i.e. $M \perp\!\!\!\perp D \mid A$
- conditioning on A we can find, $E[D|do(m)] = E[\, E[D|M = m, A]\,]$ (law of total expectation)
- then we can find the $ACE(m) = E[D|do(m+1)] - E[D|do(m)]$ (Frisch-Waugh-Lovell theorem)

$$M = \begin{cases} A \leftarrow f_A(U_A) \\ M \leftarrow f_M(A, U_M) \\ D \leftarrow f_D(A, M, U_D) \\ U \sim P(\mathbf{U}) \end{cases}$$

(a) structural model



(b) causal diagram

the dream team!!

based on DAG and statistical analysis,

- the less biased model is the second, (assuming our DAG is true)

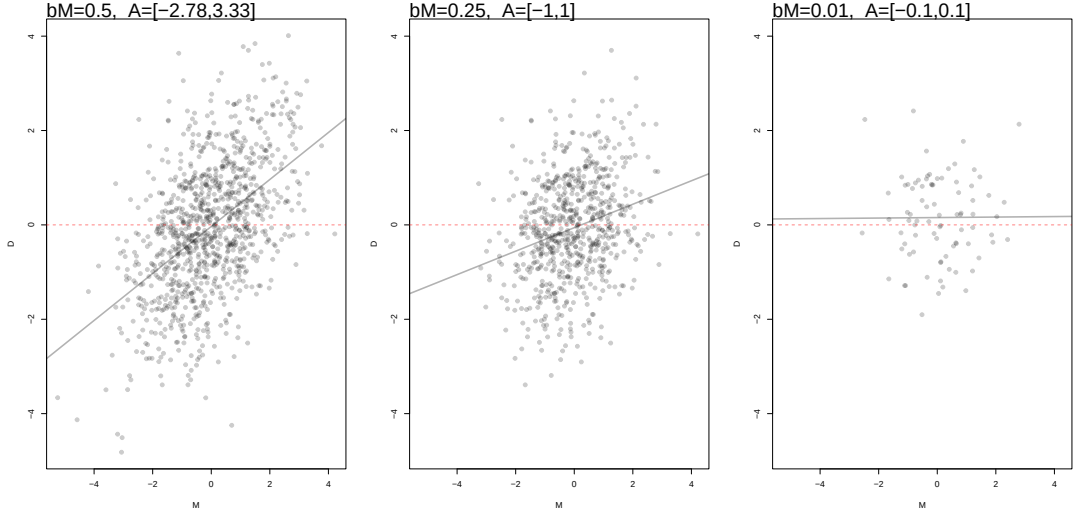
```
> summary(lm(D ~ A + M, data=d)) # controlled relation

Call:
lm(formula = D ~ A + M, data = d)

Residuals:
     Min       1Q   Median       3Q      Max
-2.27295 -0.68174  0.03781  0.78885  2.95320

Coefficients:
            Estimate Std. Error t value Pr(>|t|)
(Intercept) -0.18854    0.09871  -1.910   0.0591 .
A           -1.03121    0.13483  -7.648 1.49e-11 ***
M           -0.06134    0.09362  -0.655   0.5139
```

So, what is going on?



3. Example cases

Fork bias: masked relationships (a)

Masked relationships (a)[5]

also known as,

- omitted variable bias
- an instance of fork bias

research question,

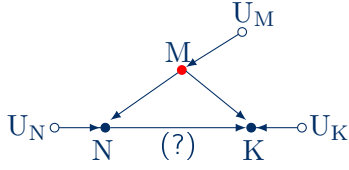
- Does N has a (direct) effect on K?

variables,

- M, mammal mass in kg.
- N, ratio neocortex over total brain mass
- K, Kcal. per gram of milk

$$\mathrm{M} = \begin{cases} \mathrm{M} \leftarrow \mathrm{f_M}(\mathrm{U_M}) \\ \mathrm{N} \leftarrow \mathrm{f_N}(\mathrm{M}, \mathrm{U_N}) \\ \mathrm{K} \leftarrow \mathrm{f_K}(\mathrm{M}, \mathrm{N}, \mathrm{U_K}) \\ \mathrm{U} \sim \mathrm{P}(\mathbf{U}) \end{cases}$$

(a) structural model



(b) causal diagram

[5] McElreath [12], chapter 5 (p. 144)

Simulation setting

```r
# sim
M = rnorm( 100 )
N = rnorm( 100 , 1*M )
K = rnorm( 100 , 1*N + -1*M )
d = data.frame(N=N,M=M,K=K)
```

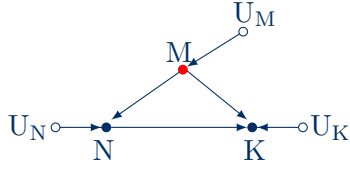
(c) R code

$$M = \begin{cases} M \leftarrow f_M(U_M) \\ N \leftarrow f_N(M, U_N) \\ K \leftarrow f_K(M, N, U_K) \\ U \sim P(\mathbf{U}) \end{cases}$$

(a) structural model

Implications,

- $N \not\perp K$
- $N \not\perp K \mid M$

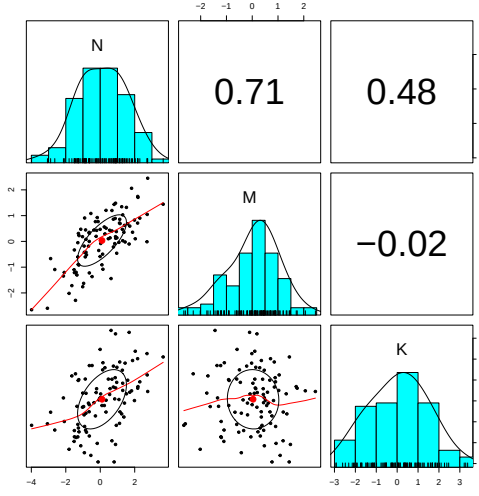


(b) causal diagram

“Eyeballing” analysis

based on correlation analysis,

- $\text{cor}(\text{N}, \text{K}) > 0$ goes in line of our “rudimentary” understanding of the data.
- but why there is $\text{cor}(\text{M}, \text{k}) \approx 0$? (hint: univariate correlation)
- we might not include M as a covariate in our statistical model



Regression, regression!!

based on statistical analysis,

- we have two different stories, (which one is the “truth”?)

```
> summary(lm(K ~ N, data=d)) # biased estimate

Call:
lm(formula = K ~ N, data = d)

Residuals:
    Min      1Q  Median      3Q     Max
-2.8355 -0.8110  0.0188  0.7897  3.4276

Coefficients:
            Estimate Std. Error t value Pr(>|t|)
(Intercept)  0.01401    0.12057   0.116    0.908
N            0.53002    0.09332   5.680 1.38e-07 ***
> summary(lm(K ~ N + M, data=d)) # less biased estima

Call:
lm(formula = K ~ N + M, data = d)

Residuals:
     Min       1Q   Median       3Q      Max
-2.50873 -0.72626 -0.01968  0.69016  2.93000

Coefficients:
            Estimate Std. Error t value Pr(>|t|)
(Intercept)  0.22096    0.09845   2.244   0.0271 *
N            0.95510    0.10089   9.466 1.91e-15 ***
M           -1.06246    0.15462  -6.871 6.14e-10 ***
```

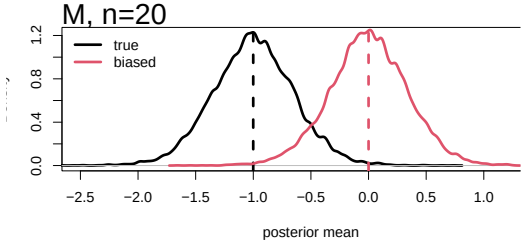
I'll get more data!!

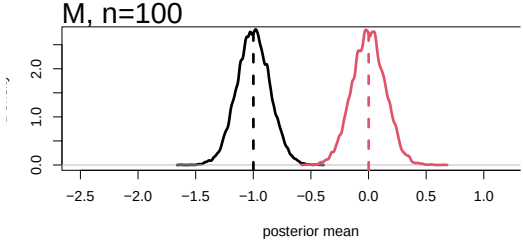
imagine we can continue sampling,

- top: $10,000$ samples $n = 20$
- bottom: $10,000$ samples $n = 100$

under the incorrect model,
the larger the sample size,

- the more certain you are about your biased estimates





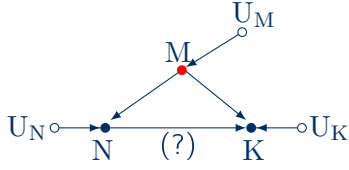
The dream team!!

based on DAG and statistical model,

- the 2nd D-separation rule requires you to control any noncollider to block the backdoor path, i.e. $N \not\perp\!\!\!\perp K \mid M$
- conditioning on M we can find, $E[K|do(n)] = E[\, E[K|N = n, M]\,]$ (law of total expectation)
- then we can find the $ACE(n) = E[D|do(n+1)] - E[D|do(n)]$ (Frisch-Waugh-Lovell theorem)

$$M = \begin{cases} M \leftarrow f_M(U_M) \\ N \leftarrow f_N(M, U_N) \\ K \leftarrow f_K(M, N, U_K) \\ U \sim P(\mathbf{U}) \end{cases}$$

(a) structural model



(b) causal diagram

the dream team!!

based on DAG and statistical analysis,

- the less biased model is the second, (assuming our DAG is true)

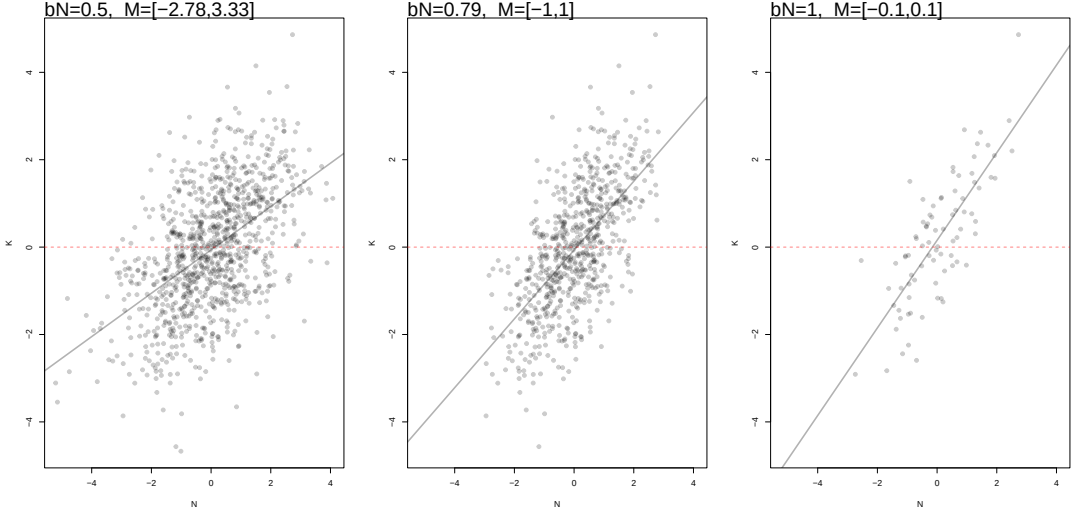
```
> summary(lm(K ~ N + M, data=d)) # less biased estima

Call:
lm(formula = K ~ N + M, data = d)

Residuals:
     Min       1Q   Median       3Q      Max
-2.50873 -0.72626 -0.01968  0.69016  2.93000

Coefficients:
            Estimate Std. Error t value Pr(>|t|)
(Intercept)  0.22096    0.09845   2.244   0.0271 *
N            0.95510    0.10089   9.466 1.91e-15 ***
M           -1.06246    0.15462  -6.871 6.14e-10 ***
```

So, what is going on?



3. Example cases

Fork bias: masked relationships (b)

Masked relationships (b)[6]

also known as,

- (unobserved) omitted variable bias
- an instance of fork bias

research question,

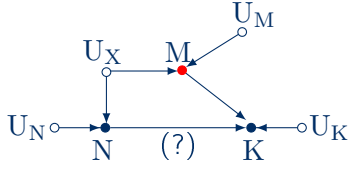
- Does N has a (direct) effect on K?

variables,

- U_X, unobservable (e.g. genetics)
- M, mammal mass in kg.
- N, neocortex over total brain mass
- K, Kcal. per gram of milk

$$M = \begin{cases} N \leftarrow f_N(U_N, U_X) \\ M \leftarrow f_M(U_M, U_X) \\ K \leftarrow f_K(M, N, U_K) \\ U \sim P(\mathbf{U}) \end{cases}$$

(a) structural model



(b) causal diagram

[6] McElreath [12], chapter 5 (p. 144)

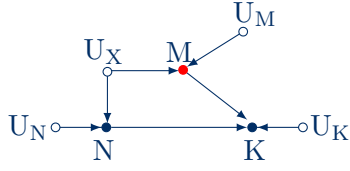
Simulation setting

```r
# sim
U = rnorm( 100 )
N = rnorm( 100 , 1*U )
M = rnorm( 100 , 1*U )
K = rnorm( 100 , 1*N + -1*M )
d = data.frame(U=U,N=N,M=M,K=K)
```

(c) R code

$$M = \begin{cases} N \leftarrow f_N(U_N, U_X) \\ M \leftarrow f_M(U_M, U_X) \\ K \leftarrow f_K(M, N, U_K) \\ U \sim P(\mathbf{U}) \end{cases}$$

(a) structural model



(b) causal diagram

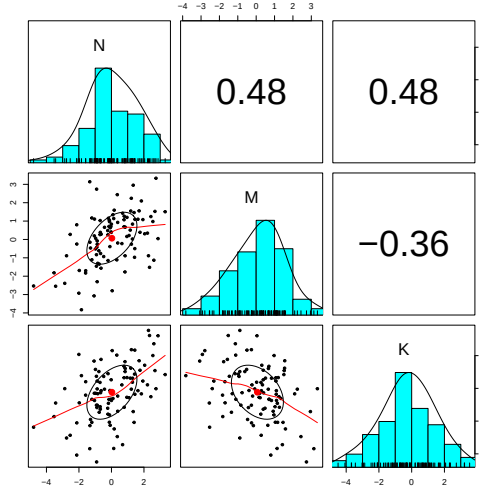
Implications,

- $N \not\perp K$
- $N \not\perp K \mid M$

“Eyeballing” analysis

based on correlation analysis,

- $\text{cor}(\text{N}, \text{K}) > 0$ goes in line of our “rudimentary” understanding of the data.
- $\text{cor}(\text{M}, \text{K}) < 0$ does NOT goes in line of our “rudimentary” understanding of the data.
  (hint: univariate correlation)
- we include M as a covariate in our statistical model
  (by chance?)



Regression, regression!!

based on statistical analysis,

- we have two different stories, (which one is the “truth”?)

```
> summary(lm(K ~ N, data=d)) # unobserved path still

Call:
lm(formula = K ~ N, data = d)

Residuals:
    Min      1Q  Median      3Q     Max
-3.7763 -0.8480  0.1497  0.9874  3.3530

Coefficients:
            Estimate Std. Error t value Pr(>|t|)
(Intercept) -0.24867    0.14573  -1.706   0.0911 .
N            0.51406    0.09502   5.410 4.46e-07 ***
> summary(lm(K ~ N + M, data=d)) # unobserved path cl

Call:
lm(formula = K ~ N + M, data = d)

Residuals:
     Min       1Q   Median       3Q      Max
-2.58218 -0.58434 -0.00579  0.72016  1.78724

Coefficients:
            Estimate Std. Error t value Pr(>|t|)
(Intercept) -0.19978    0.09375  -2.131   0.0356 *
N            0.90893    0.06958  13.064   <2e-16 ***
M           -0.89676    0.07572 -11.843   <2e-16 ***
```

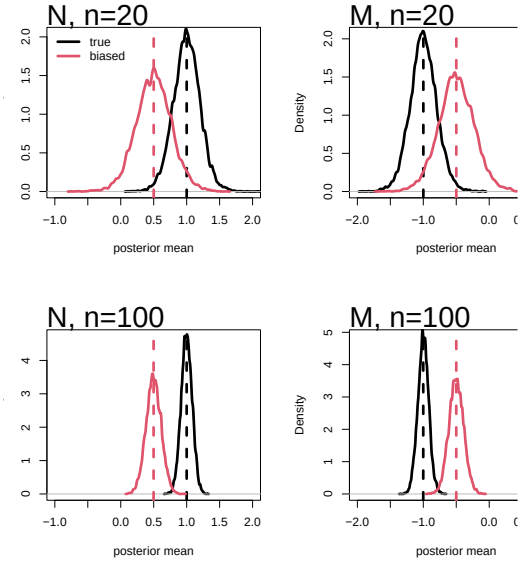
I'll get more data!!

imagine we can continue sampling,

- top: $10,000$ samples $n = 20$
- bottom: $10,000$ samples $n = 100$

under the incorrect model,
the larger the sample size,

- the more certain you are about your biased estimates



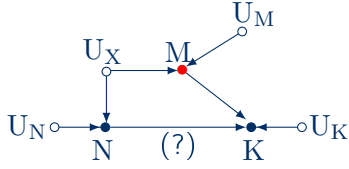
The dream team!!

based on DAG and statistical model,

- the 2nd D-separation rule requires control on any noncollider to block the backdoor path,
  i.e. $N \not\perp\!\!\!\perp K \mid U_X$
  (but it is unobservable)
- still we use the 2nd D-separation rule by controlling for M,
  i.e. $N \not\perp\!\!\!\perp K \mid M$
- conditioning on M we can still find,
  $E[K|do(n)] = E[\, E[K|N = n, M] \,]$
  (law of total expectation)
- then we can find the
  $ACE(n) = E[D|do(n+1)] - E[D|do(n)]$
  (Frisch-Waugh-Lovell theorem??)

$$M = \begin{cases} N \leftarrow f_N(U_N, U_X) \\ M \leftarrow f_M(U_M, U_X) \\ K \leftarrow f_K(M, N, U_K) \\ U \sim P(\mathbf{U}) \end{cases}$$

(a) structural model



(b) causal diagram

the dream team!!

based on DAG and statistical analysis,

- the less biased model is the second, (assuming our DAG is true)

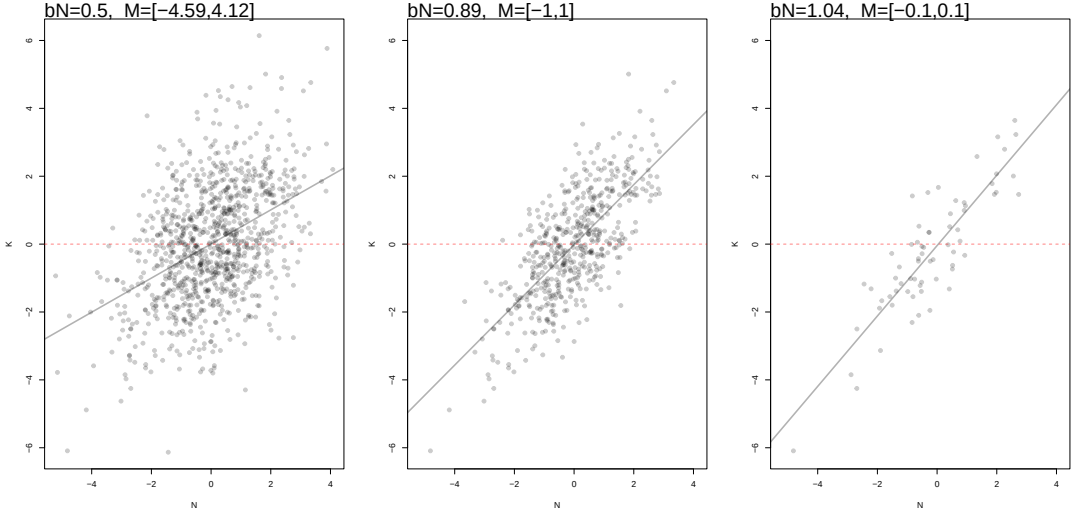
```
> summary(lm(K ~ N + M, data=d)) # unobserved path cl

Call:
lm(formula = K ~ N + M, data = d)

Residuals:
     Min       1Q   Median       3Q      Max
-2.58218 -0.58434 -0.00579  0.72016  1.78724

Coefficients:
            Estimate Std. Error t value Pr(>|t|)
(Intercept) -0.19978    0.09375  -2.131   0.0356 *
N            0.90893    0.06958  13.064   <2e-16 ***
M           -0.89676    0.07572 -11.843   <2e-16 ***
```

So, what is going on?



Similar scenario, unobserved masked[7]

research question,

- Does E has a (direct) effect on I?

variables,

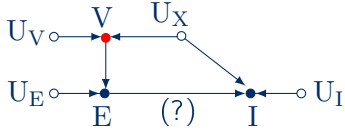
- U_X, unobservable (e.g. family context)
- V, personal values
- E, education
- I, income

then,

- we need to control by V to get an unbiased estimate of $E \to I$

$$M = \begin{cases} V \leftarrow f_M(U_V, U_X) \\ E \leftarrow f_E(V, U_E) \\ I \leftarrow f_I(E, U_X, U_I) \\ U \sim P(\mathbf{U}) \end{cases}$$

(a) structural model



(b) causal diagram

[7] Cinelli et al. [4] (p. 3), McElreath [13], lecture 6

3. Example cases

Fork bias: multicollinearity

Multicollinearity[8]

also known as,

- extreme case of masked relationships
- an instance of fork bias

research question,

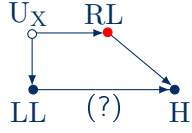
- Should we include RL in our model?

variables,

- U_X, unobservable (e.g. genetics and context)
- LL, individual's left leg
- RL, individual's right leg
- H, individual's height

$$M = \begin{cases} LL \leftarrow f_L(U_X) \\ RL \leftarrow f_L(U_X) \\ H \leftarrow f_K(RL, LL) \\ U \sim P(\mathbf{U}) \end{cases}$$

(a) structural model



(b) causal diagram

[8] McElreath [12], chapter 6 (p. 163)

Simulation setting

```r
# backward simulation
H = round( rnorm( 100 , 170, 2), 1)
Lp = runif( 100 , 0.5-0.05, 0.5+0.05)
LL = round( Lp*H + rnorm( 100 , 0, 1 ), 1)
RL = round( Lp*H + rnorm( 100 , 0, 1 ), 1)
d = data.frame(LL,RL,H)
```

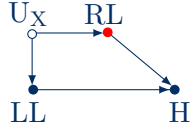
(c) R code

Implications,

- LL $\not\perp$ RL

$$M = \begin{cases} LL \leftarrow f_L(U_X) \\ RL \leftarrow f_L(U_X) \\ H \leftarrow f_K(RL, LL) \\ U \sim P(\mathbf{U}) \end{cases}$$

(a) structural model

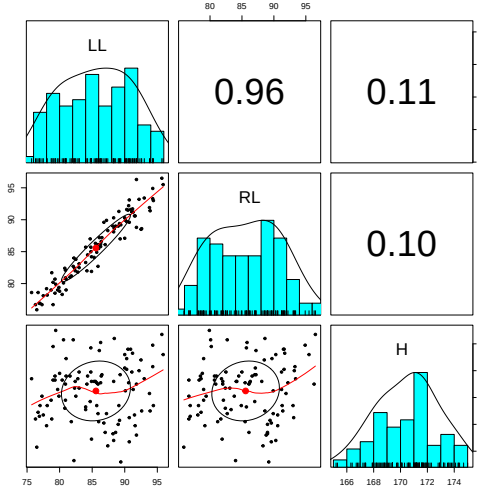


(b) causal diagram

“Eyeballing” analysis

based on correlation analysis,

- $\text{cor}(\text{LL}, \text{H}) > 0$, $\text{cor}(\text{RL}, \text{H}) > 0$ and $\text{cor}(\text{LL}, \text{RL}) > 0$ goes in line of our “understanding” of the data.
- we might not include RL as a covariate in our statistical model (based on univariate correlation)



Regression, regression!!

based on statistical analysis,

- the second regression show a smaller effect of LL,
- the second regression show way larger SE values
  (not rejecting the null)

```
> summary(lm(H ~ -1 + LL, data=d)) # unbiased,

Call:
lm(formula = H ~ -1 + LL, data = d)

Residuals:
     Min       1Q   Median       3Q      Max
-17.9704  -8.3662   0.7494  10.5256  21.1464

Coefficients:
   Estimate Std. Error t value Pr(>|t|)
LL  1.98486    0.01229   161.6   <2e-16 ***
> summary(lm(H ~ -1 + LL + RL, data=d)) # ine

Call:
lm(formula = H ~ -1 + LL + RL, data = d)

Residuals:
    Min      1Q  Median      3Q     Max
-16.780  -8.592   0.532  10.253  18.299

Coefficients:
   Estimate Std. Error t value Pr(>|t|)
LL   1.0094     0.6969   1.448    0.151
RL   0.9757     0.6970   1.400    0.165
```

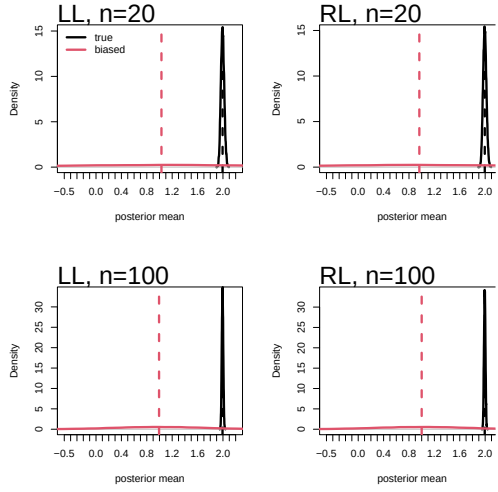
I'm sure data won't help

imagine we can continue sampling,

- top: $10,000$ samples $n = 20$
- bottom: $10,000$ samples $n = 100$

under the incorrect model,
the larger the sample size,

- the less certain are your biased estimates



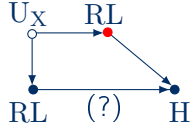
Not so great now??

based on DAG and statistical model,

- the 2nd D-separation rule requires control on any noncollider to block the backdoor path,
  i.e. $\mathrm{LL} \not\perp\!\!\!\perp \mathrm{H} \mid \mathrm{U_X}$
  (but it is unobservable)
- we still use the 2nd D-separation rule by controlling for RL, but still we have $\mathrm{LL} \not\perp\!\!\!\perp \mathrm{H} \mid \mathrm{RL}$
- issue goes beyond the backdoor path
  the issue is that RL and LL give the same information to the model,
  i.e. they form a singular matrix,
  (is like having a causal model like b)

$$\mathrm{M} = \begin{cases} \mathrm{LL} = \mathrm{RL} \\ \mathrm{RL} \leftarrow \mathrm{f_L}(\mathrm{U_X}) \\ \mathrm{H} \leftarrow \mathrm{f_K}(\mathrm{RL}) \\ \mathrm{U} \sim \mathrm{P}(\mathbf{U}) \end{cases}$$

(a) structural model

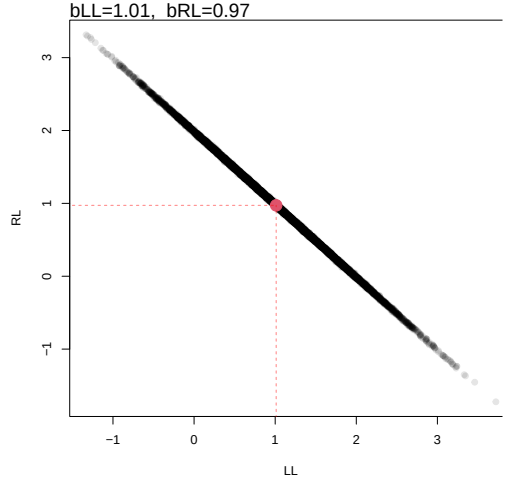


(b) causal diagram

So, what is going on then??

the estimated parameters under the second regression,

- are in a thin ridge
  $\text{Cor}(\text{bLL}, \text{bRL}) \approx -1$
- statistical model finds not only one solution (red dot), the thin ridge implies there are infinite solutions for the parameters,
  (related to the singular matrix thing)



Not so great now??

based on DAG and statistical analysis,

- the less biased and more precise model is the first,
  (assuming our DAG is true)

```
> summary(lm(H ~ -1 + LL, data=d)) # unbiased

Call:
lm(formula = H ~ -1 + LL, data = d)

Residuals:
     Min       1Q   Median       3Q      Max
-17.9704  -8.3662   0.7494  10.5256  21.1464

Coefficients:
   Estimate Std. Error t value Pr(>|t|)
LL  1.98486    0.01229   161.6   <2e-16 ***
```

3. Example cases

No more fork bias: neutral control

Neutral control[9]

also known as,

- precision "booster"
- similar to experimental design

research question,

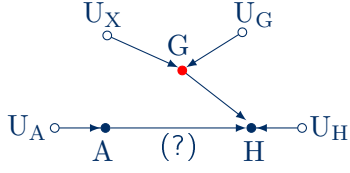
- Should we include G on our model?

variables,

- A, "hearing" age
- G, gender
- U_X, unobservable (e.g. no idea yet)
- H, inverse logit of entropy (approximate of speech intelligibility)

$$M = \begin{cases} G \leftarrow f_G(U_G, U_X) \\ A \leftarrow f_A(U_A) \\ H \leftarrow f_H(A, G, U_H) \\ U \sim P(\mathbf{U}) \end{cases}$$

(a) structural model



(b) causal diagram

[9] Cinelli et al. [4] (p. 4)

Simulation setting

```
# sim
G = sample( 0:1, 100 , replace=T )
A = rnorm( 100 )
H = rnorm( 100 , -1*A + -1*G )
d = data.frame(G=G,A=A,SI=SI)
```

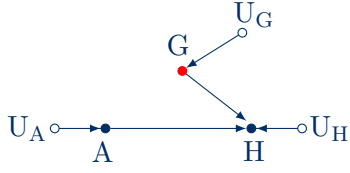
(c) R code

$$M = \begin{cases} G \leftarrow f_G(U_G) \\ A \leftarrow f_A(U_A) \\ H \leftarrow f_H(A, G, U_H) \\ U \sim P(\mathbf{U}) \end{cases}$$

(a) structural model

Implications,

- $A \perp\!\!\!\perp G$
- $A \not\perp\!\!\!\perp H$
- $G \not\perp\!\!\!\perp H$

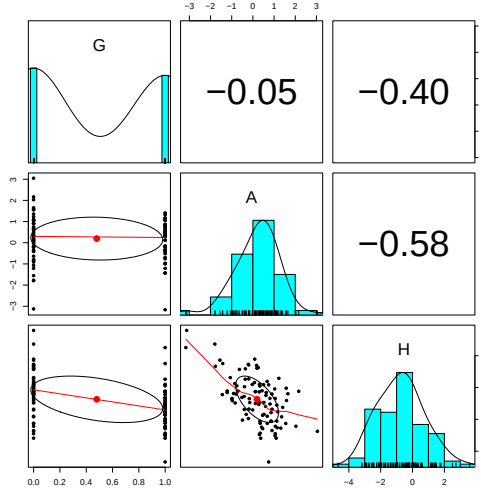


(b) causal diagram

“Eyeballing” analysis

based on correlation analysis,

- $\text{cor}(\text{G}, \text{H}) < 0$, $\text{cor}(\text{G}, \text{A}) \approx 0$ and $\text{cor}(\text{A}, \text{H}) < 0$ goes in line of our “rudimentary” understanding of the data.
- we include both as a covariate in our statistical model



Regression, regression!!

based on statistical analysis,

- almost no change on our estimates,
- lower SE for A when G is included (because we have explained some variability in H, not related to A)

```
> summary(lm(H ~ A, data=d)) # correct estimate

Call:
lm(formula = H ~ A, data = d)

Residuals:
    Min      1Q  Median      3Q     Max
-3.4714 -0.8797 -0.0633  0.8963  2.4346

Coefficients:
            Estimate Std. Error t value Pr(>|t|)
(Intercept)  -0.5770     0.1216  -4.746 7.07e-06 ***
A            -0.8410     0.1183  -7.108 1.92e-10 ***
> summary(lm(H ~ A + G, data=d)) # correct estimate,

Call:
lm(formula = H ~ A + G, data = d)

Residuals:
    Min      1Q  Median      3Q     Max
-2.7994 -0.6914  0.0579  0.7796  1.8274

Coefficients:
            Estimate Std. Error t value Pr(>|t|)
(Intercept)  0.03317    0.14312   0.232    0.817
A           -0.87360    0.10090  -8.658 1.05e-13 ***
G           -1.25786    0.20371  -6.175 1.55e-08 ***
```

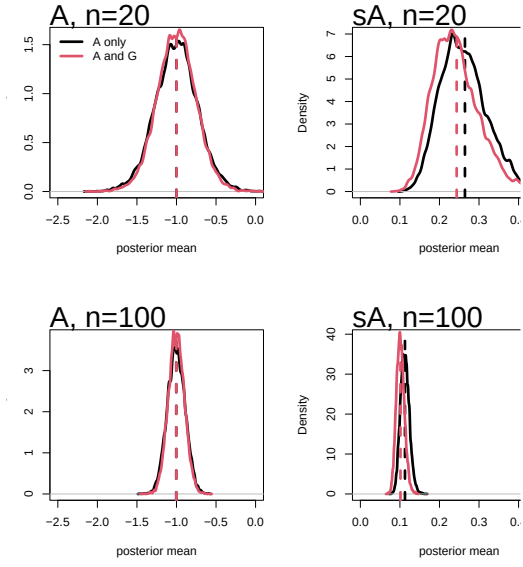
Does more data works in this case?

imagine we can continue sampling,

- top: $10,000$ samples $n = 20$
- bottom: $10,000$ samples $n = 100$

under the second model,
the larger the sample size,

- the more certain you are about your non-biased estimates (under the any model)
- this is how random effects work (in a way)



3. Example cases

Pipe bias: precision parasite

Precision parasite[10]

research question,

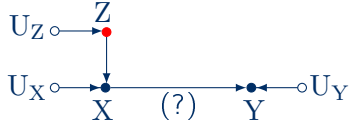
- What is the effect of X on Y?
- Should we include Z in the model?

variables,

- Z, "parent" of X
- X, exposure
- Y, outcome

$$M = \begin{cases} Z \leftarrow f_Z(U_Z) \\ X \leftarrow f_X(Z, U_X) \\ Y \leftarrow f_Y(X, U_Y) \\ U \sim P(\mathbf{U}) \end{cases}$$

(a) structural model



(b) causal diagram

[10] McElreath [13], lecture 6; Cinelli et al. [4] (p. 5)

Simulation setting

```r
# sim
Z = rnorm( 100 )
X = rnorm( 100 , 1*Z )
Y = rnorm( 100 , 1*X )
d = data.frame(Z,X,Y)
```

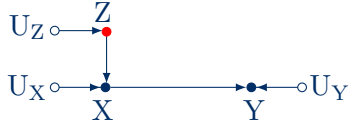
(c) R code

$$M = \begin{cases} Z \leftarrow f_Z(U_Z) \\ X \leftarrow f_X(Z, U_X) \\ Y \leftarrow f_Y(X, U_Y) \\ U \sim P(\mathbf{U}) \end{cases}$$

(a) structural model

Implications,

- $X \not\perp Y$
- $Z \not\perp Y \mid X$

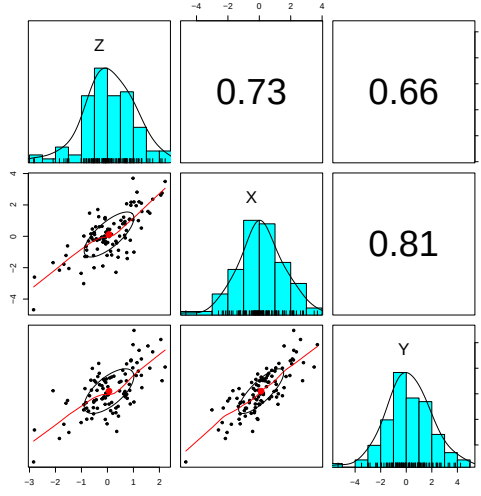


(b) causal diagram

“Eyeballing” analysis

based on correlation analysis,

- $\text{cor}(\text{Z}, \text{X}) > 0$ is not large enough to discard it as multicollinearity.
- $\text{cor}(\text{Z}, \text{Y}) > 0$ and $\text{cor}(\text{X}, \text{Y}) > 0$ indicate both should be in our model
  (it might be our research hypothesis)



Regression, regression!!

based on statistical analysis,

- no bias in parameter if Z is in,
- but we loose precision on X

```
> summary(lm(Y ~ X, data=d)) # unbiased effect, more

Call:
lm(formula = Y ~ X, data = d)

Residuals:
     Min       1Q   Median       3Q      Max
-2.41746 -0.73659 -0.09384  0.63812  2.10338

Coefficients:
            Estimate Std. Error t value Pr(>|t|)
(Intercept)  0.03433    0.10274   0.334    0.739
X            1.16908    0.06717  17.405   <2e-16 ***
> summary(lm(Y ~ X + Z, data=d)) # unbiased effects,

Call:
lm(formula = Y ~ X + Z, data = d)

Residuals:
     Min       1Q   Median       3Q      Max
-2.27019 -0.74072 -0.06355  0.66643  2.20770

Coefficients:
            Estimate Std. Error t value Pr(>|t|)
(Intercept)  0.04722    0.10350   0.456    0.649
X            1.08881    0.10358  10.512   <2e-16 ***
Z            0.15431    0.15159   1.018    0.311
```

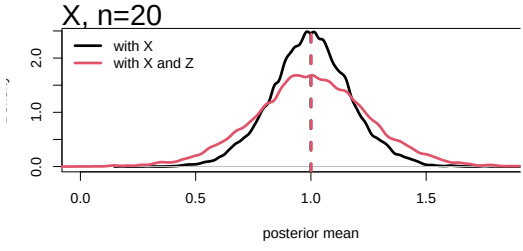
With more data??

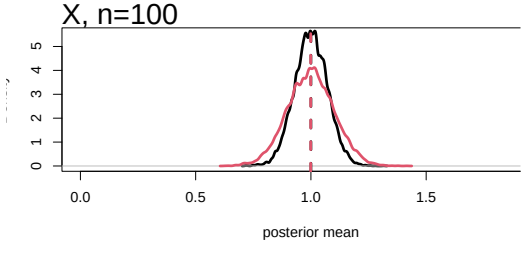
imagine we can continue sampling,

- top: $10,000$ samples $n = 20$
- bottom: $10,000$ samples $n = 100$

under the second model,
the larger the sample size,

- still less precise estimates
- more difficult to test hypothesis





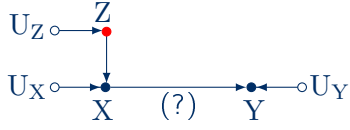
Now, what is going on here??

based on DAG and statistical model,

- conditioning on Z reduces variation on X, leaving less variability that can explain outcome Y

$$M = \begin{cases} Z \leftarrow f_Z(U_Z) \\ X \leftarrow f_X(Z, U_X) \\ Y \leftarrow f_Y(X, U_Y) \\ U \sim P(\mathbf{U}) \end{cases}$$

(a) structural model



(b) causal diagram

Now, what is going on here??

based on DAG and statistical analysis,

- the more appropriate model (for inference) is the first,
  (assuming our DAG is true)

```
> summary(lm(Y ~ X, data=d)) # unbiased effect, more

Call:
lm(formula = Y ~ X, data = d)

Residuals:
     Min       1Q   Median       3Q      Max
-2.41746 -0.73659 -0.09384  0.63812  2.10338

Coefficients:
            Estimate Std. Error t value Pr(>|t|)
(Intercept)  0.03433    0.10274   0.334    0.739
X            1.16908    0.06717  17.405   <2e-16 ***
```

The same, but not quite[11]

research question,

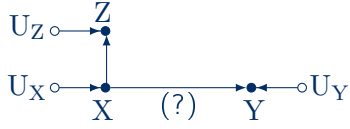
- What is the effect of X on Y?
- Should we include Z in the model?

variables,

- Z, "child" of X
- X, exposure
- Y, outcome

$$M = \begin{cases} X \leftarrow f_X(U_X) \\ Z \leftarrow f_Z(X, U_Z) \\ Y \leftarrow f_Y(X, U_Y) \\ U \sim P(\mathbf{U}) \end{cases}$$

(a) structural model



(b) causal diagram

[11] Cinelli et al. [4] (p. 7)

3. Example cases

Pipe bias: post-treatment

Post-treatment bias[12]

case of,

- full mediation

research question,

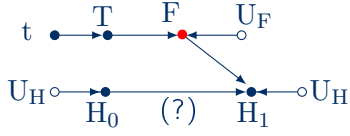
- Does the treatment T works?

variables,

- H_0, height of plant at $t = 0$
- T, antifungal treatment
- F, presence of fungus
- H_1, height of plant at $t = 1$

$$M = \begin{cases} H_0 \leftarrow f_H(U_H) \\ T \leftarrow f_T(t) \\ F \leftarrow f_F(T, U_F) \\ H_1 \leftarrow f_H(F, H_0, U_H) \\ U \sim P(\mathbf{U}) \end{cases}$$

(a) structural model



(b) causal diagram

[12] McElreath [12], chapter 6 (p. 170)

Simulation setting

```r
# sim
h0 = rnorm( 100 , 10, 2)
Tr = rep( 0:1 , each=100/2 )
Fu = rbinom( n , size=1 , prob=0.5 + -0.4*Tr )
h1 = h0 + rnorm( n , 5 + -3*Fu)
d = data.frame( h0=h0, h1=h1, Tr=Tr, Fu=Fu )
```

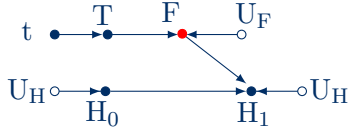
(c) R code

$$M = \begin{cases} H_0 \leftarrow f_H(U_H) \\ T \leftarrow f_T(t) \\ F \leftarrow f_F(T, U_F) \\ H_1 \leftarrow f_H(F, H_0, U_H) \\ U \sim P(\mathbf{U}) \end{cases}$$

(a) structural model

Implications,

- $T \perp\!\!\!\perp H_0$
- $T \not\perp\!\!\!\perp H_1$
- $T \not\perp\!\!\!\perp H_1 \mid F$



(b) causal diagram

Descriptive analysis

based on descriptive analysis,

- positive change in height with treatment.
- negative change in height with fungus.
- diluted relationship for T when both are in the model
  (hint: blocking path of information)

Tr	mean	sd	n	se
<fct>	<dbl>	<dbl>	<int>	<dbl>
0	2.96	1.75	50	0.248
1	4.53	1.45	50	0.205

Fu	mean	sd	n	se
<fct>	<dbl>	<dbl>	<int>	<dbl>
0	4.93	0.953	62	0.121
1	1.81	0.902	38	0.146

Tr	Fu	mean	sd	n	se
<fct>	<fct>	<dbl>	<dbl>	<int>	<dbl>
0	0	4.91	0.810	19	0.186
0	1	1.76	0.861	31	0.155
1	0	4.93	1.02	43	0.155
1	1	2.01	1.12	7	0.423

Again regression!!

based on statistical analysis we have two different stories (but not quite),

- treatment has a significant effect,
- but gets completely diluted when fungus is considered in the model

```
> summary(lm(h1-h0 ~ Tr, data=d)) # only treatment

Call:
lm(formula = h1 - h0 ~ Tr, data = d)

Residuals:
    Min      1Q  Median      3Q     Max
-4.1166 -1.0929  0.1755  1.2621  3.3990

Coefficients:
            Estimate Std. Error t value Pr(>|t|)
(Intercept)   2.9598     0.2273   13.02  < 2e-16 ***
Tr1           1.5656     0.3215    4.87 4.29e-06 ***
> summary(lm(h1-h0 ~ Tr + Fu, data=d)) # only fungus

Call:
lm(formula = h1 - h0 ~ Tr + Fu, data = d)

Residuals:
    Min      1Q  Median      3Q     Max
-2.1691 -0.4823  0.0963  0.5315  2.0357

Coefficients:
            Estimate Std. Error t value Pr(>|t|)
(Intercept)  4.86397    0.19127  25.430   <2e-16 ***
Tr1          0.09138    0.21579   0.423    0.673
Fu1         -3.07122    0.22229 -13.816   <2e-16 ***
```

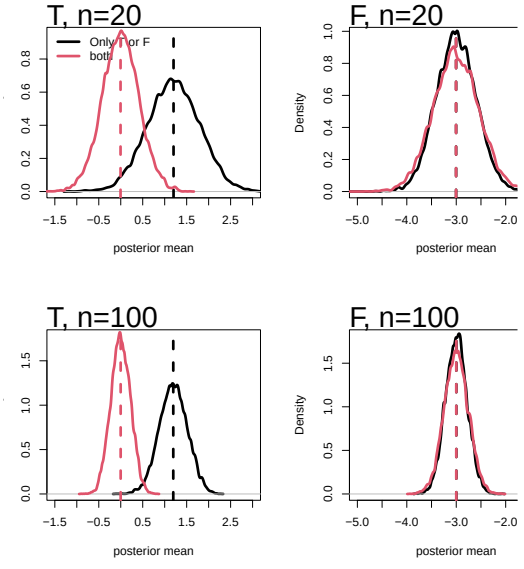
I can guess what happens with more data!!

imagine we can continue sampling,

- top: $10,000$ samples $n = 20$
- bottom: $10,000$ samples $n = 100$

under the "incorrect" model,
the larger the sample size,

- the more certain you are about your biased T estimates (not F) (this result is not wrong!)



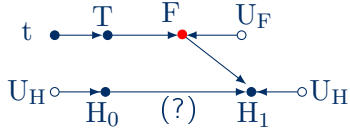
The dream team!!

based on DAG and statistical model,

- the 2nd D-separation states that if you to control any noncollider you block the backdoor path, i.e. $T \perp\!\!\!\perp H_1 \mid F$
- therefore if we want to find if $T = 1$ works, we should not stratify by F

$$M = \begin{cases} H_0 \leftarrow f_H(U_H) \\ T \leftarrow f_T(t) \\ F \leftarrow f_F(T, U_F) \\ H_1 \leftarrow f_H(F, H_0, U_H) \\ U \sim P(\mathbf{U}) \end{cases}$$

(a) structural model



(b) causal diagram

the dream team!!

based on DAG and statistical analysis,

- the model that answers our research question is the first one, (assuming our DAG is true)

```
> summary(lm(h1-h0 ~ Tr, data=d)) # only treatment

Call:
lm(formula = h1 - h0 ~ Tr, data = d)

Residuals:
     Min       1Q   Median       3Q      Max
-4.1166 -1.0929  0.1755  1.2621  3.3990

Coefficients:
            Estimate Std. Error t value Pr(>|t|)
(Intercept)   2.9598     0.2273   13.02  < 2e-16 ***
Tr1           1.5656     0.3215    4.87 4.29e-06 ***
```

3. Example cases

Pipe bias: Simpson's paradox

Masked relationships[13]

also known as,

- mediation
- masked relationships
- an instance of pipe bias

research question,

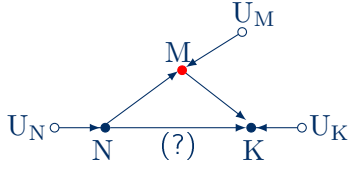
- Does N has a (direct) effect on K?

variables,

- M, mammal mass in kg.
- N, neocortex over total brain mass
- K, Kcal. per gram of milk

$$\mathrm{M} = \begin{cases} \mathrm{N} \leftarrow \mathrm{f_N}(\mathrm{U_N}) \\ \mathrm{M} \leftarrow \mathrm{f_M}(\mathrm{N}, \mathrm{U_M}) \\ \mathrm{K} \leftarrow \mathrm{f_K}(\mathrm{M}, \mathrm{N}, \mathrm{U_K}) \\ \mathrm{U} \sim \mathrm{P}(\mathbf{U}) \end{cases}$$

(a) structural model



(b) causal diagram

[13] McElreath [12], chapter 6 (p. 170)

Simulation setting

```r
# sim
N = rnorm( 100 )
M = rnorm( 100 , 1*N )
K = rnorm( 100 , 1*N + -1*M )
d = data.frame(N=N,M=M,K=K)
```

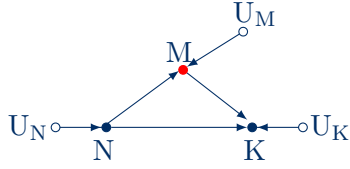
(c) R code

$$M = \begin{cases} N \leftarrow f_N(U_N) \\ M \leftarrow f_M(M, U_M) \\ K \leftarrow f_K(M, N, U_K) \\ U \sim P(\mathbf{U}) \end{cases}$$

(a) structural model

Implications,

- $N \not\perp K$
- $N \not\perp K \mid M$

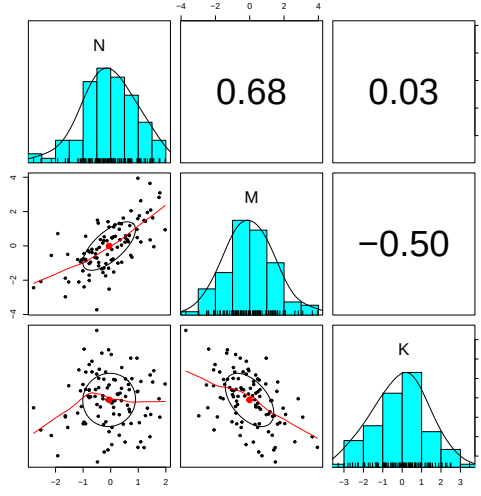


(b) causal diagram

“Eyeballing” analysis

based on correlation analysis,

- $\text{cor}(\text{M}, \text{K}) < 0$ does NOT goes in line of our “rudimentary” understanding of the data.
- and why there is $\text{cor}(\text{N}, \text{K}) \approx 0$? (hint: univariate correlation)
- we include N as a covariate in our statistical model (is our research hypothesis)



Regression, regression!!

based on statistical analysis,

- two regressions with two different results, which model is the “true”?

```
> summary(lm(K ~ N, data=d)) # biased estimate

Call:
lm(formula = K ~ N, data = d)

Residuals:
    Min      1Q  Median      3Q     Max
-3.1751 -0.9009  0.1519  0.8574  3.6041

Coefficients:
            Estimate Std. Error t value Pr(>|t|)
(Intercept) -0.10412    0.13808  -0.754    0.453
N            0.05005    0.14487   0.345    0.730
> summary(lm(K ~ N + M, data=d)) # less biased estimate

Call:
lm(formula = K ~ N + M, data = d)

Residuals:
    Min      1Q  Median      3Q     Max
-2.58484 -0.59175  0.04378  0.61175  2.43360

Coefficients:
            Estimate Std. Error t value Pr(>|t|)
(Intercept) -0.06181    0.09825  -0.629    0.531
N            0.98297    0.13994   7.024 2.98e-10 ***
M           -0.93107    0.09457  -9.846 2.89e-16 ***
```

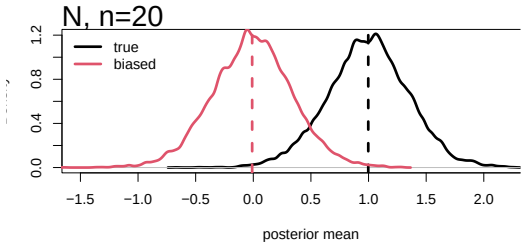
I'll get more data!!

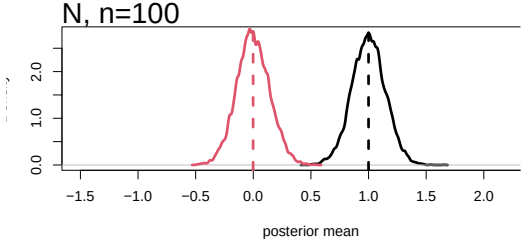
imagine we can continue sampling,

- top: $10,000$ samples $n = 20$
- bottom: $10,000$ samples $n = 100$

under the "incorrect" model,
the larger the sample size,

- the more certain you are about your biased estimates





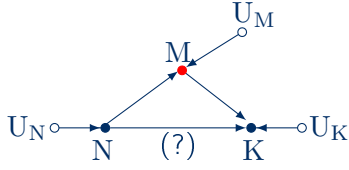
The dream team!!

based on DAG and statistical model,

- the 2nd D-separation rule requires you to control any noncollider to block the backdoor path, i.e. $N \not\perp K \mid M$
- conditioning on M we can find, $E[K|do(n)] = E[\, E[K|N = n, M]\,]$ (law of total expectation)
- then we can find the $ACE(n) = E[D|do(n+1)] - E[D|do(n)]$ (Frisch-Waugh-Lovell theorem)

$$M = \begin{cases} N \leftarrow f_N(U_N) \\ M \leftarrow f_M(M, U_M) \\ K \leftarrow f_K(M, N, U_K) \\ U \sim P(\mathbf{U}) \end{cases}$$

(a) structural model



(b) causal diagram

the dream team!!

based on DAG and statistical analysis,

- the less biased model is the second, (assuming our DAG is true)

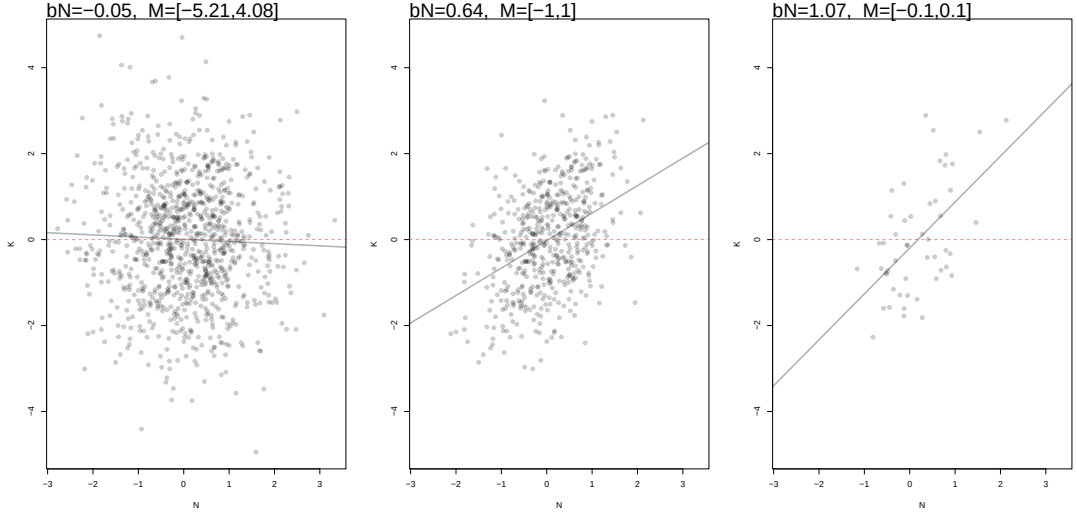
```
> summary(lm(K ~ N + M, data=d)) # less biased estimate

Call:
lm(formula = K ~ N + M, data = d)

Residuals:
     Min       1Q   Median       3Q      Max
-2.58484 -0.59175  0.04378  0.61175  2.43360

Coefficients:
            Estimate Std. Error t value Pr(>|t|)
(Intercept) -0.06181    0.09825  -0.629    0.531
N            0.98297    0.13994   7.024 2.98e-10 ***
M           -0.93107    0.09457  -9.846 2.89e-16 ***
```

So, what is going on?



Similar case, gender discrimination[14]

research question,

- Do females are discriminated in school admissions, i.e. does G → A?

variables,

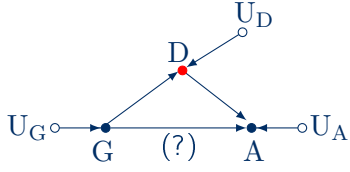
- G, gender
- D, department of application
- A, admission

then,

- stratification by D close mediation path (backdoor path on A)
- not stratifying by D finds the total effect of G on A (all paths) (e.g. "structural" discrimination)

$$M = \begin{cases} G \leftarrow f_G(U_G) \\ D \leftarrow f_D(G, U_D) \\ A \leftarrow f_A(D, G, U_A) \\ U \sim P(\mathbf{U}) \end{cases}$$

(a) structural model



(b) causal diagram

[14] McElreath [12], chapter 11 (p. 340)

Another similar case, children achievement[15]

research question,

- Does M has a (direct) effect on D?

variables,

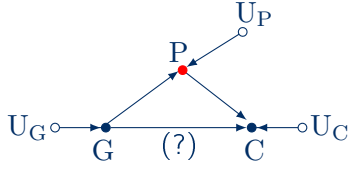
- G, grandparents' level of education
- P, parents' level of education
- C, children's educational achievement

then,

- we should stratify by P

$$\mathrm{M} = \begin{cases} \mathrm{G} \leftarrow \mathrm{f_G}(\mathrm{U_G}) \\ \mathrm{P} \leftarrow \mathrm{f_P}(\mathrm{G}, \mathrm{U_P}) \\ \mathrm{C} \leftarrow \mathrm{f_C}(\mathrm{G}, \mathrm{P}, \mathrm{U_C}) \\ \mathrm{U} \sim \mathrm{P}(\mathbf{U}) \end{cases}$$

(a) structural model



(b) causal diagram

[15] McElreath [12], chapter 6 (p. 180)

3. Example cases

No pipe/fork bias: good controls

Pipe/fork good controls[16]

research question,

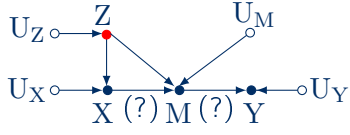
- What is the (total) effect of X on Y?
  (all directional paths from X to Y)
  (i.e. no mediators)

variables,

- Z, confounder
- X, exposure
- M, mediator
- Y, outcome

$$\mathrm{M} = \begin{cases} \mathrm{Z} \leftarrow \mathrm{f_Z}(\mathrm{U_Z}) \\ \mathrm{X} \leftarrow \mathrm{f_X}(\mathrm{Z}, \mathrm{U_X}) \\ \mathrm{M} \leftarrow \mathrm{f_M}(\mathrm{X}, \mathrm{Z}, \mathrm{U_M}) \\ \mathrm{Y} \leftarrow \mathrm{f_Y}(\mathrm{M}, \mathrm{U_Y}) \\ \mathrm{U} \sim \mathrm{P}(\mathbf{U}) \end{cases}$$

(a) structural model



(b) causal diagram

[16] Cinelli et al. [4] (p. 3)

Simulation setting

```r
# sim
Z = rnorm( 100 )
X = rnorm( 100 , 1*Z )
M = rnorm( 100 , 0*X + 1*Z )
Y = rnorm( 100 , 0.5*M )
d = data.frame(Z=Z,X=X,M=M,Y=Y)
```

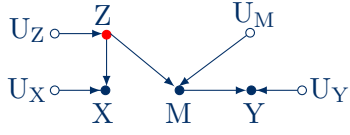
(c) R code

$$\mathrm{M} = \begin{cases} \mathrm{Z} \leftarrow \mathrm{f_Z}(\mathrm{U_Z}) \\ \mathrm{X} \leftarrow \mathrm{f_X}(\mathrm{Z}, \mathrm{U_X}) \\ \mathrm{M} \leftarrow \mathrm{f_M}(\mathrm{X}, \mathrm{Z}, \mathrm{U_M}) \\ \mathrm{Y} \leftarrow \mathrm{f_Y}(\mathrm{M}, \mathrm{U_Y}) \\ \mathrm{U} \sim \mathrm{P}(\mathbf{U}) \end{cases}$$

(a) structural model

Implications,

- $\mathrm{X} \not\perp\!\!\!\perp \mathrm{M} \mid \mathrm{Z}$
- $\mathrm{Z} \perp\!\!\!\perp \mathrm{Y}$

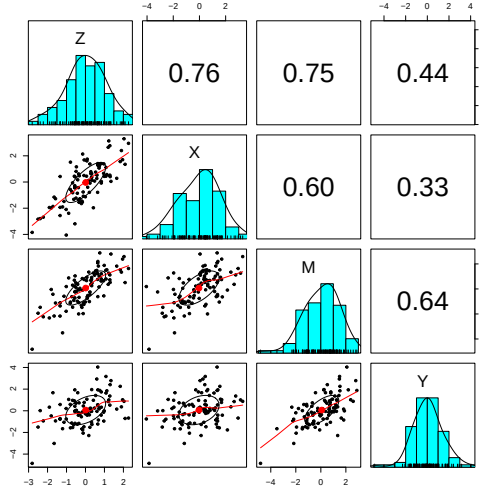


(b) causal diagram

“Eyeballing” analysis

based on correlation analysis,

- X and M should be in your model, (but we know a full mediator closes the backdoor path, and we do not want that: not our research question)
- while $\text{cor}(Z, M) \approx 0.8$ prevents from using Z as a covariate (possibly a multicollinearity problem)
- since we will not include M, now Z can be included



Regression, regression!!

based on statistical analysis,

- two different stories
- in one X has small but significant effect
- which model is the “truth”?

```
> summary(lm(Y ~ X, data=d)) # biased

Call:
lm(formula = Y ~ X, data = d)

Residuals:
    Min      1Q  Median      3Q     Max
-3.8115 -0.7762 -0.1034  0.7210  3.5707

Coefficients:
            Estimate Std. Error t value Pr(>|t|)
(Intercept)  0.07480    0.12867   0.581 0.562354
X            0.29099    0.08428   3.453 0.000821 ***
> summary(lm(Y ~ X + Z, data=d)) # unbiased

Call:
lm(formula = Y ~ X + Z, data = d)

Residuals:
    Min      1Q  Median      3Q     Max
-3.3281 -0.7697 -0.0931  0.8735  3.6160

Coefficients:
            Estimate Std. Error t value Pr(>|t|)
(Intercept)  0.057974   0.123169   0.471  0.63892
X           -0.005456   0.123103  -0.044  0.96474
Z            0.571826   0.179473   3.186  0.00194 **
```

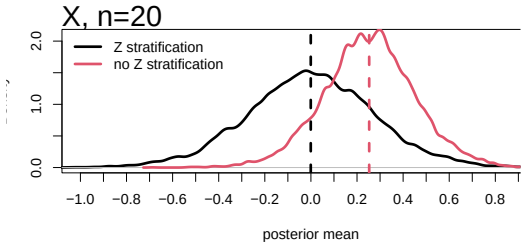
the telled tell of more data!!

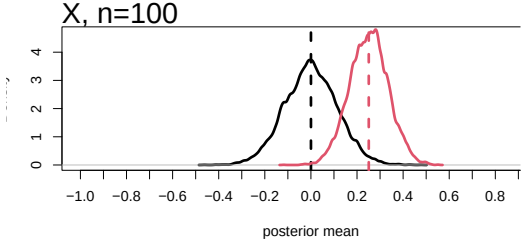
imagine we can continue sampling,

- top: $10,000$ samples $n = 20$
- bottom: $10,000$ samples $n = 100$

under the incorrect model,
the larger the sample size,

- the more certain you are about your biased estimates





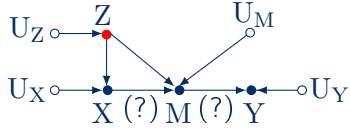
Yo, what is going on??

based on DAG and statistical model,

- there are two paths from X to Y
  $X \to M \to Y$
  $X \to Z \to M \to Y$
- stratifying by Z closes the second,
  (a confounder path)
- estimate bZ corresponds to the total effect of Z on Y,
  i.e. $Z \to M \to Y$
  (but this is not our main research interest)

$$M = \begin{cases} Z \leftarrow f_Z(U_Z) \\ X \leftarrow f_X(Z, U_X) \\ M \leftarrow f_M(X, Z, U_M) \\ Y \leftarrow f_Y(M, U_Y) \\ U \sim P(\mathbf{U}) \end{cases}$$

(a) structural model



(b) causal diagram

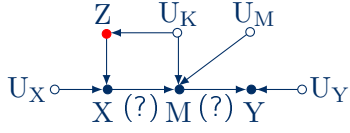
Similar cases[17]

research question,

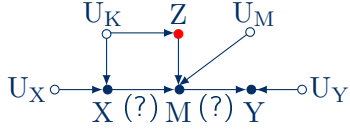
- What is the (total) effect of X on Y?
  (all directional paths from X to Y)
  (i.e. no mediators)

then,

- stratifying by Z is still a good idea
  (U_Z is not draw out of convenience)



(b) causal diagram



(b) causal diagram

[17] Cinelli et al. [4] (p. 3)

Careful what you control for (though)

```r
# sim
Z = rnorm( 100 )
X = rnorm( 100 , 1*Z )
M = rnorm( 100 , 0.5*X + 1*Z )
Y = rnorm( 100 , 0*M )
d = data.frame(Z=Z,X=X,M=M,Y=Y)
```

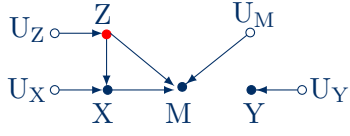
(c) R code

$$\mathrm{M} = \begin{cases} \mathrm{Z} \leftarrow \mathrm{f_Z}(\mathrm{U_Z}) \\ \mathrm{X} \leftarrow \mathrm{f_X}(\mathrm{Z}, \mathrm{U_X}) \\ \mathrm{M} \leftarrow \mathrm{f_M}(\mathrm{X}, \mathrm{Z}, \mathrm{U_M}) \\ \mathrm{Y} \leftarrow \mathrm{f_Y}(\mathrm{U_Y}) \\ \mathrm{U} \sim \mathrm{P}(\mathbf{U}) \end{cases}$$

(a) structural model

Implications,

- $\mathrm{X} \perp\!\!\!\perp \mathrm{Y}$
- $\mathrm{Z} \perp\!\!\!\perp \mathrm{Y}$
- $\mathrm{X} \not\perp\!\!\!\perp \mathrm{M} \mid \mathrm{Z}$

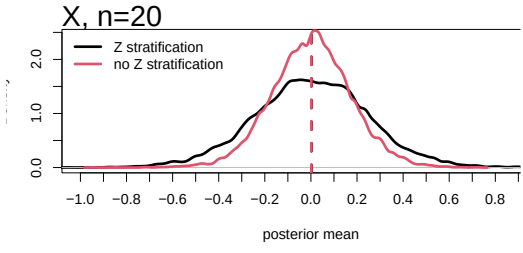


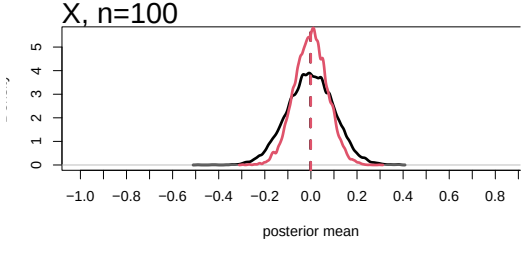
(b) causal diagram

Careful what you control for (though)

under the NO stratification model, the larger the sample size,

- the more certain you are about your correct estimates
- Z now works as a precision parasite i.e. conditioning on Z reduces variation on X, leaving less variability that can explain outcome Y





Not all is bad (though)

```r
# sim
Z = rnorm( 100 )
X = rnorm( 100 , 0*Z )
M = rnorm( 100 , 0.5*X + 1*Z )
Y = rnorm( 100 , 0.5*M )
d = data.frame(Z=Z,X=X,M=M,Y=Y)
```

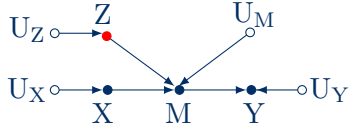
(c) R code

$$\mathrm{M} = \begin{cases} \mathrm{Z} \leftarrow \mathrm{f_Z}(\mathrm{U_Z}) \\ \mathrm{X} \leftarrow \mathrm{f_X}(\mathrm{U_X}) \\ \mathrm{M} \leftarrow \mathrm{f_M}(\mathrm{X}, \mathrm{Z}, \mathrm{U_M}) \\ \mathrm{Y} \leftarrow \mathrm{f_Y}(\mathrm{M}, \mathrm{U_Y}) \\ \mathrm{U} \sim \mathrm{P}(\mathbf{U}) \end{cases}$$

(a) structural model

Implications,

- $\mathrm{X} \not\perp\!\!\!\perp \mathrm{Y}$
- $\mathrm{X} \perp\!\!\!\perp \mathrm{Y}$
- $\mathrm{Z} \perp\!\!\!\perp \mathrm{Y}$

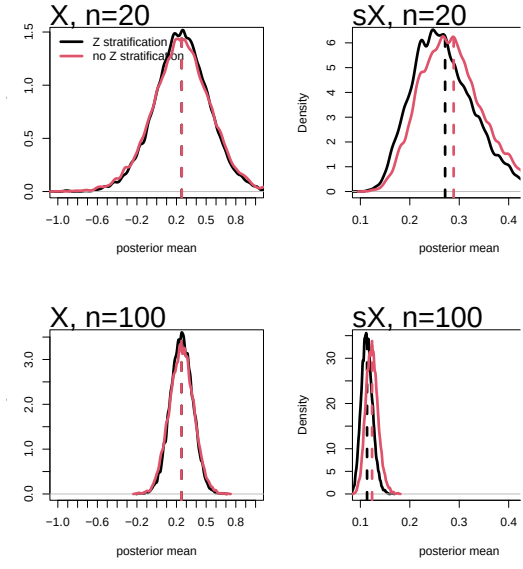


(b) causal diagram

Not all is bad (though)

under the stratification model,
the larger the sample size,

- the more certain you are about your correct estimates
- Z now works as a precision booster
  i.e. conditioning on Z reduces variation on Y (as Z is a cause of M, and M of Y), leaving less variability to be explained by X



3. Example cases

Pipe/Fork bias: bias amplification

Bias amplification[18]

also known as,

- (unobserved) omitted variable bias
- related to instrumental variables
- an instance of fork bias

research question,

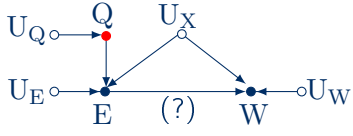
- Do E has a (direct) effect on W?

variables,

- Q, instrumental variable (e.g. quarter of the year)
- E, educational level
- U_X, unobservables (e.g. ability)
- W, future wages

$$M = \begin{cases} Q \leftarrow f_Q(U_Q) \\ E \leftarrow f_E(Q, U_X, U_E) \\ W \leftarrow f_W(E, U_X, U_W) \\ U \sim P(\mathbf{U}) \end{cases}$$

(a) structural model



(b) causal diagram

[18] McElreath [12], chapter 14 (p. 455), Cinelli et al. [4] (p. 5)

Simulation setting

```r
# sim
U = rnorm( 100 )
Q = sample( 1:4, 100, replace=T )
E = rnorm( 100 , 1*Q + 1*U )
W = rnorm( 100 , 0*E + 1*U )
d = data.frame(U=U,Q=Q,E=E,W=W)
```

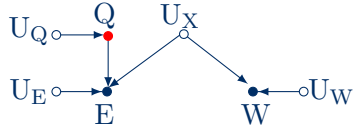
(c) R code

$$M = \begin{cases} Q \leftarrow f_Q(U_Q) \\ E \leftarrow f_E(Q, U_X, U_E) \\ W \leftarrow f_W(U_X, U_W) \\ U \sim P(\mathbf{U}) \end{cases}$$

(a) structural model

Implications,

- $E \not\perp\!\!\!\perp W$
- $E \perp\!\!\!\perp W \mid U_X$ (impossible)
- $Q \perp\!\!\!\perp U_X$ (cannot be tested)
- $Q \not\perp\!\!\!\perp E$
- $Q \perp\!\!\!\perp W \mid E$ (cannot be tested) (exclusion restriction)

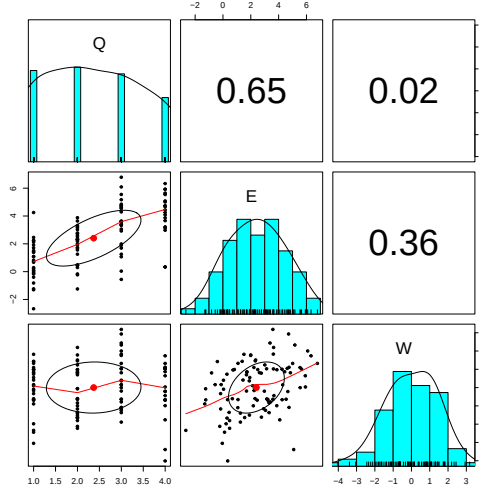


(b) causal diagram

“Eyeballing” analysis

based on correlation analysis,

- $\text{cor}(\text{Q}, \text{E}) > 0$ and $\text{cor}(\text{E}, \text{W}) > 0$ goes in line of our “rudimentary” understanding of the data.
- $\text{cor}(\text{Q}, \text{W}) > 0$ tells you about the exclusion restriction?
  (hint: No)
- we might NOT include Q as a covariate in our statistical model (but is the instrumental variable!!!)



Regression, regression!!

based on statistical analysis,

- two different stories
  (which model is the “truth”?)
- one is “worse”/“better” than the other?
- are both wrong?

```
> summary(lm(W ~ E, data=d)) # biased

Call:
lm(formula = W ~ E, data = d)

Residuals:
    Min      1Q  Median      3Q     Max
-4.0726 -0.9674  0.1771  0.9234  2.8787

Coefficients:
            Estimate Std. Error t value Pr(>|t|)
(Intercept) -0.60816    0.20506  -2.966 0.003793 **
E            0.25408    0.06559   3.873 0.000194 ***
> summary(lm(W ~ E + Q, data=d)) # more biased

Call:
lm(formula = W ~ E + Q, data = d)

Residuals:
    Min      1Q  Median      3Q     Max
-3.7405 -0.9774  0.0879  0.9162  2.9825

Coefficients:
            Estimate Std. Error t value Pr(>|t|)
(Intercept)  0.12229    0.30650   0.399  0.69078
E            0.42054    0.08262   5.090 1.75e-06 ***
Q           -0.47716    0.15361  -3.106  0.00249 **
```

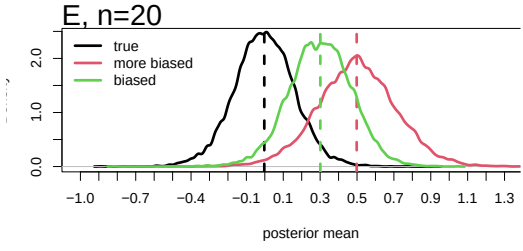
I'll get more data!!

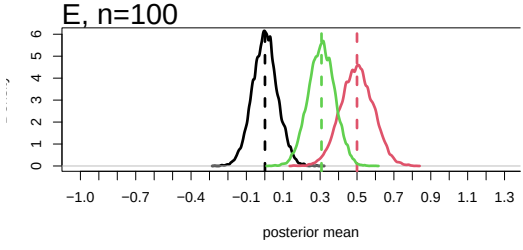
imagine we can continue sampling,

- top: $10,000$ samples $n = 20$
- bottom: $10,000$ samples $n = 100$

under the incorrect model,
the larger the sample size,

- the more certain you are about your biased estimates (under the any model!!)





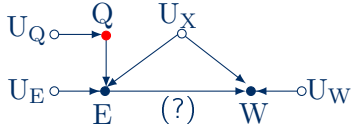
Yo, what is going on??

based on DAG and statistical model,

- the 2nd D-separation rule requires control on any noncollider to block the backdoor path,
  i.e. $E \perp\!\!\!\perp W \mid U_X$
  (but U_X is unobservable)
- if we use Q in the model, the 3rd D-separation rule kicks in:
  "A collider that has been conditioned on does not block a path."
  i.e. $Q \not\perp\!\!\!\perp U_X \mid E$
  (e.g. switch, electricity, and light bulb)

$$M = \begin{cases} Q \leftarrow f_Q(U_Q) \\ E \leftarrow f_E(Q, U_X, U_E) \\ W \leftarrow f_W(E, U_X, U_W) \\ U \sim P(\mathbf{U}) \end{cases}$$

(a) structural model



(b) causal diagram

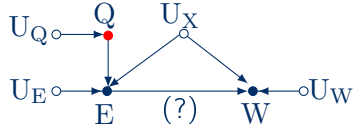
Yo, what is going on??

open paths?:

- $E \to W$
- $E \to U_x \to W$
- $E \to U_x \to Q \to E \to W$
- $E \to U_x \to Q \to E \to U_X \to W$

$$M = \begin{cases} Q \leftarrow f_Q(U_Q) \\ E \leftarrow f_E(Q, U_X, U_E) \\ W \leftarrow f_W(E, U_X, U_W) \\ U \sim P(\mathbf{U}) \end{cases}$$

(a) structural model



(b) causal diagram

What should I do then??

$$\begin{pmatrix} \mathrm{W} \\ \mathrm{E} \end{pmatrix} \sim \mathrm{MVN}\left[\begin{pmatrix} \mu_\mathrm{W} \\ \mu_\mathrm{E} \end{pmatrix}, \boldsymbol{\Sigma}\right]$$
$$\mu_\mathrm{W} = \alpha_\mathrm{W} + \beta_\mathrm{EW}\mathrm{E}$$
$$\mu_\mathrm{E} = \alpha_\mathrm{E} + \beta_\mathrm{QE}\mathrm{Q}$$

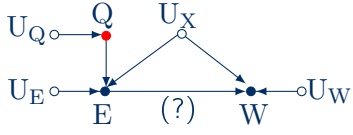
(c) probabilistic model

$$\mathrm{M} = \begin{cases} \mathrm{Q} \leftarrow \mathrm{f_Q}(\mathrm{U_Q}) \\ \mathrm{E} \leftarrow \mathrm{f_E}(\mathrm{Q}, \mathrm{U_X}, \mathrm{U_E}) \\ \mathrm{W} \leftarrow \mathrm{f_W}(\mathrm{E}, \mathrm{U_X}, \mathrm{U_W}) \\ \mathrm{U} \sim \mathrm{P}(\mathbf{U}) \end{cases}$$

(a) structural model

based on DAG and statistical model,
use the knowledge of the system

- one model for $\mathrm{Q} \to \mathrm{E}$
- one model for $\mathrm{E} \to \mathrm{W}$
- use the knowledge that $\mathrm{cov}(\mathrm{E}, \mathrm{W}) > 0$ due to unobserved confounder $\mathrm{U_X}$, (i.e. $\mathrm{cov}(\mathrm{E}, \mathrm{W}) = \boldsymbol{\Sigma} = \mathbf{SRS}$)



(b) causal diagram

did it worked???

based on DAG and bayesian statistical analysis,

- appropriate value estimated, (assuming our DAG is true)
- it picks up some of the unobserved correlation R[1, 2]

	mean	sd	5.5%	94.5%
aE	0.02	0.18	-0.26	0.30
aW	-0.14	0.16	-0.40	0.13
bQE	1.00	0.07	0.88	1.12
bEW	0.05	0.07	-0.06	0.16
R[1,1]	1.00	0.00	1.00	1.00
R[1,2]	0.33	0.11	0.15	0.50
R[2,1]	0.33	0.11	0.15	0.50
R[2,2]	1.00	0.00	1.00	1.00
S[1]	1.25	0.10	1.11	1.42
S[2]	1.39	0.10	1.24	1.56

did it worked???

frequentists guys apply
Two Stage Least Squares (2SLS)[a]:

- regress E ← Q,
- predict $\hat{E}$,
- regress W ← $\hat{E}$

```
s1 = lm( E ~ Q, data=d)
Ehat = s1$fitted.values
s2 = lm( W ~ Ehat, data=d)
# se not corrected

require(AER)
tsls = ivreg( W ~ E | Q, data=d)
# se corrected
```

```
> summary(s2)

Call:
lm(formula = W ~ Ehat, data = d)

Residuals:
    Min      1Q  Median      3Q     Max
-4.1911 -1.0670 -0.0643  1.3802  4.6813

Coefficients:
            Estimate Std. Error t value Pr(>|t|)
(Intercept)  -0.5992     0.3667  -1.634    0.106
Ehat          0.2093     0.1263   1.658    0.101
> summary(tsls)

Call:
ivreg(formula = W ~ E | Q, data = d)

Residuals:
    Min      1Q  Median      3Q     Max
-3.6571 -1.1852 -0.1819  1.0945  4.5328

Coefficients:
            Estimate Std. Error t value Pr(>|t|)
(Intercept)  -0.5992     0.3325  -1.802   0.0746 .
E             0.2093     0.1145   1.829   0.0705 .
```

[a] Hanck et al. [7], section 12.1,
See McElreath [12] chapter 14 (p. 460) for a discussion on the method.

Similar case, contextual confounds[19]

research question,

- Does W has an effect on L?
- should we include H in our model?

variables,

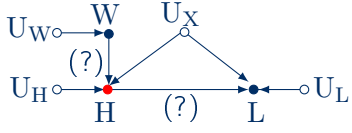
- W, win the lottery
- H, happiness
- U_X, contextual confound
- L, lifespan

Short answer;

- for total effects: No
  (two question marks together)
- for direct effect of H → L:
  will be always counfounded

$$M = \begin{cases} W \leftarrow f_W(U_W) \\ H \leftarrow f_H(W, U_X, U_H) \\ L \leftarrow f_L(H, U_X, U_L) \\ U \sim P(\mathbf{U}) \end{cases}$$

(a) structural model



(b) causal diagram

[19] McElreath [13], lecture 6

3. Example cases

Collider bias: Berkson's paradox

Berkson's paradox[20]

also known as,

- selection bias
- selection-distorsion effect
- "convenience" sample bias

research question,

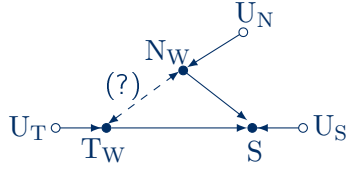
- Is there a true relationship between N_W and T_W in studies?

variables,

- N_W, "news-worthiness" of a study
- T_W, "trust-worthiness" of a study
- S, selected studies

$$M = \begin{cases} T_W \leftarrow f_T(U_T) \\ N_W \leftarrow f_N(U_N) \\ S \leftarrow f_S(T_W, N_W, U_S) \\ U \sim P(\mathbf{U}) \end{cases}$$

(a) structural model



(b) causal diagram

[20] McElreath [12], chapter 6 (p. 161); Cinelli et al. [4] (p. 8)

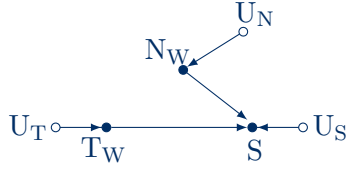
Simulation setting

```r
# sim
NW = rnorm( 100 ) # uncorrelated
TW = rnorm( 100 )
Sc = NW + TW  # total score
q = quantile( Sc , 1-0.1 ) # top 10% threshold
S = ifelse( Sc >= q , 1 , 0 ) # select top 10%
d = data.frame( NW=NW, TW=TW, S=S )
```

(c) R code

$$M = \begin{cases} T_W \leftarrow f_T(U_T) \\ N_W \leftarrow f_N(U_N) \\ S \leftarrow f_S(T_W, N_W, U_S) \\ U \sim P(\mathbf{U}) \end{cases}$$

(a) structural model



(b) causal diagram

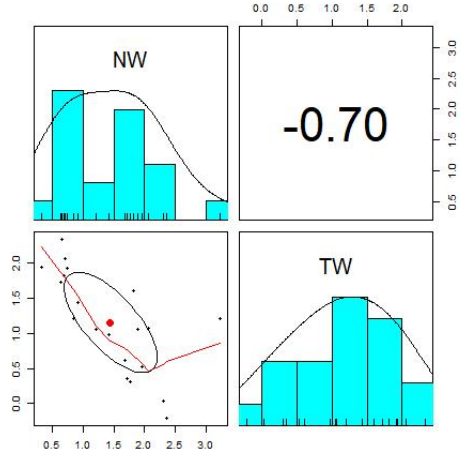
Implications,

- $T_W \perp\!\!\!\perp N_W$
- $T_W \not\perp\!\!\!\perp N_W \mid S$

Descriptive analysis

based on descriptive analysis,

- what we observe is $\text{Cor}(N_W, T_W \mid S)$,
  (data has been stratified on a score)
- $\text{Cor}(N_W, T_W \mid S)$ does NOT go in line with our "rudimentary" understanding of the data
  (N_W and T_W are related?)
- the issue is that we want to observe $\text{Cor}(N_W, T_W)$
  (unconditional correlation)



Regression does not solve anything!!

based on statistical analysis,

- T_W continues to "explain" N_W, (is the only model accessible)
- But is it correct though?

```
> summary(lm(NW ~ TW, data=d[d$S==1,])) # biased effe

Call:
lm(formula = NW ~ TW, data = d[d$S == 1, ])

Residuals:
    Min      1Q  Median      3Q     Max
-0.5955 -0.2437 -0.1069  0.1720  0.8788

Coefficients:
            Estimate Std. Error t value Pr(>|t|)
(Intercept)   2.3076     0.2176  10.605 3.59e-09 ***
TW           -0.7861     0.1402  -5.608 2.54e-05 ***
```

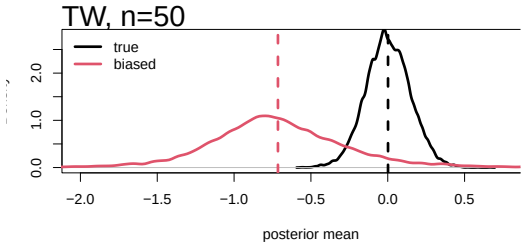
Let me guess?, more data, more problems

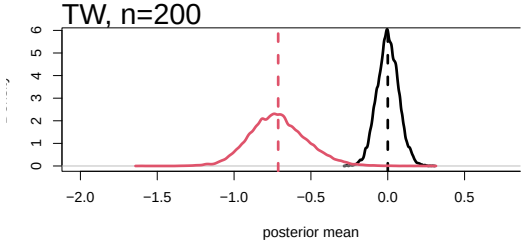
imagine we can continue sampling,

- top: $10,000$ samples $n = 20$
- bottom: $10,000$ samples $n = 100$

under the only available model,
the larger the sample size,

- the more certain you are about your biased estimates

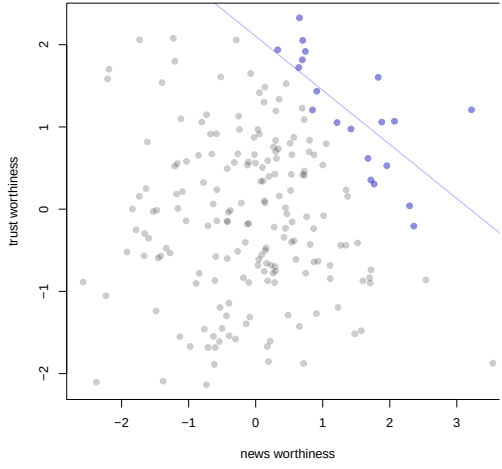




Ok, what is going on?

If we only observe data where $S = 1$,

- it means that even if your study had a low T_W, you know it necessarily has a high N_W to compensate and pass the threshold (e.g. switch, electricity, and light)



Similar case[21]

research question,

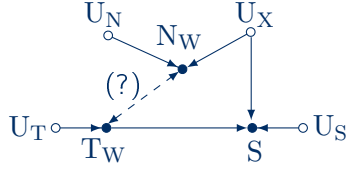
- Is there a true relationship between NW and TW in studies?

variables,

- NW, "news-worthiness" of a study
- TW, "trust-worthiness" of a study
- U_x, unobservable (e.g. no idea yet)
- S, selected studies

$$M = \begin{cases} T_W \leftarrow f_T(U_T) \\ N_W \leftarrow f_N(U_N) \\ \quad S \leftarrow f_S(T_W, N_W, U_S) \\ \quad U \sim P(\mathbf{U}) \end{cases}$$

(a) structural model



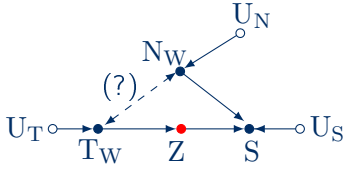
(b) causal diagram

[21] Cinelli et al. [4] (p. 8)

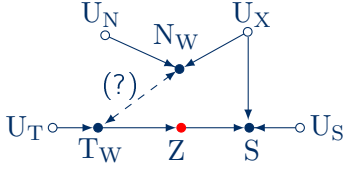
Is there a way to fix it?

a simple ways,

- use a variable that closes the selection path (2nd D-separation rule),
  i.e. stratify by a pipe to close the path
  $T_W \to Z \to S$ (see right)
  or
  $N_W \to Z \to S$ (not shown)
  $N_W \leftarrow U_X \to Z \to S$ (not shown)



(b1) causal diagram

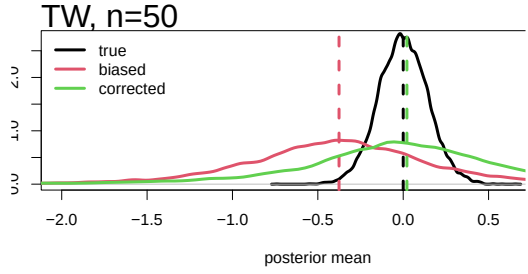


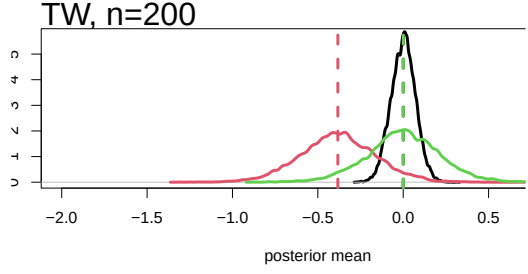
(b2) causal diagram

Does it work though?

based on the previous DAGs,
the statistical analysis reveals,

- stratifying by Z "corrects" the estimates,
  (but we still lose some precision)





Other ways to solve it?[22]

many other (much more complex) ways,

- sensitivity analysis
  (example here)
- post-stratification?
  (example on slide XX)
- matching?
- inverse-probability weighting?
- g-formula?
- g-estimations?

[22] most also apply for previous examples, but are beyond the scope of this document (for now)

3. Example cases

Collider bias: M-bias

M-bias[23]

case of,

- bias on pre-treatment variable

research question,

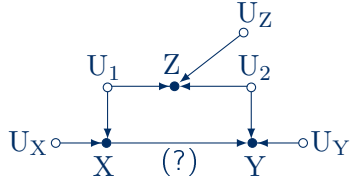
- Should we include Z in our model?

variables,

- Z, "health" quality of friends (defined as a continuum)
- X, health of individual 1
- U_1, hobbies of individual 1
- Y, health of individual 2
- U_2, hobbies of individual 2

$$\mathrm{M} = \begin{cases} \mathrm{X} \leftarrow \mathrm{f_X}(\mathrm{U_1}, \mathrm{U_X}) \\ \mathrm{Z} \leftarrow \mathrm{f_Z}(\mathrm{U_1}, \mathrm{U_2}, \mathrm{U_Z}) \\ \mathrm{Y} \leftarrow \mathrm{f_Y}(\mathrm{X}, \mathrm{U_2}, \mathrm{U_Y}) \\ \mathrm{U} \sim \mathrm{P}(\mathbf{U}) \end{cases}$$

(a) structural model



(b) causal diagram

[23] McElreath [13], lecture 6; Cinelli et al. [4] (p. 4)

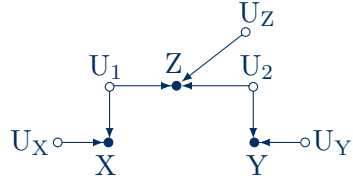
Simulation setting

```r
# sim
U1 = sample(1:5, 100 , replace=T)
U2 = sample(1:5, 100 , replace=T)
Z = rnorm( 100 , 0.5*U1 + 0.5*U2)
X = rnorm( 100 , 1*U1 + 0*Z)
Y = rnorm( 100 , 1*U2 + 0*X + 0*Z)
d = data.frame(U1,U2,Z,X,Y)
```

(c) R code

$$M = \begin{cases} X \leftarrow f_X(U_1, U_X) \\ Z \leftarrow f_Z(U_1, U_2, U_Z) \\ Y \leftarrow f_Y(X, U_2, U_Y) \\ U \sim P(\mathbf{U}) \end{cases}$$

(a) structural model



(b) causal diagram

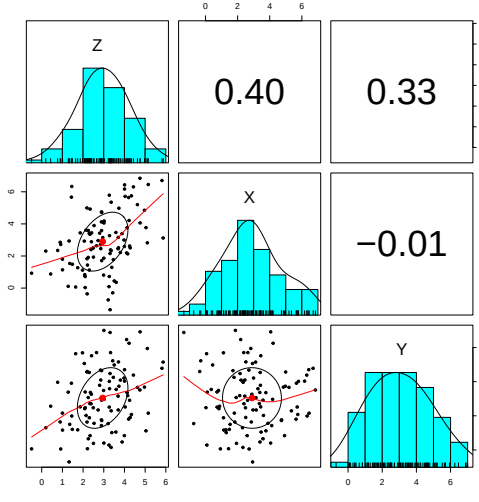
Implications,

- $X \perp\!\!\!\perp Y$
- $X \not\perp\!\!\!\perp Y \mid Z$

Descriptive analysis

based on descriptive analysis,

- $\text{Cor}(X, Y) \approx 0$, quite low
- larger $\text{Cor}(Z, Y)$, while $\text{Cor}(Z, X)$ is not high enough to discard it as a cause of multicollinearity,
- we might include Z rather than X (but the effect of X is our interest!!)
- then we include Z and X



Again regression!!

based on statistical analysis,

- X does not have an effect of Y, (in the first nor the second model)
- but X has an (non-negligible) effect when Z is in the model (but we do not reject the null)
- The increase of the X effect might lead you to think that with more data, we can reject the null (and you would be right!!)
- But is it correct to include Z?

```
> summary(lm(Y ~ X, data=d)) # unbiased effects (effic

Call:
lm(formula = Y ~ X, data = d)

Residuals:
    Min      1Q  Median      3Q     Max
-3.7452 -1.3300 -0.0253  1.2414  3.9479

Coefficients:
             Estimate Std. Error t value Pr(>|t|)
(Intercept)  3.067474   0.334586   9.168 7.74e-15 ***
X           -0.008255   0.097774  -0.084    0.933
> summary(lm(Y ~ X + Z, data=d)) # biased effects (ef

Call:
lm(formula = Y ~ X + Z, data = d)

Residuals:
    Min      1Q  Median      3Q     Max
-3.3756 -1.0898  0.0351  1.0540  3.7595

Coefficients:
            Estimate Std. Error t value Pr(>|t|)
(Intercept)  1.83324    0.45304   4.047 0.000105 ***
X           -0.15765    0.09991  -1.578 0.117835
Z            0.56590    0.14974   3.779 0.000272 ***
```

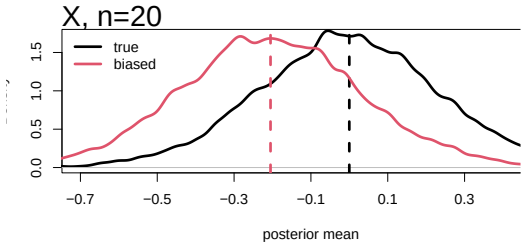
Ok, I get it!!, more data, more wrong!!

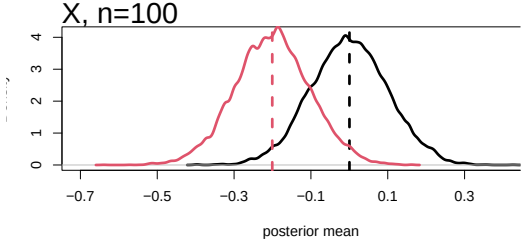
imagine we can continue sampling,

- top: $10,000$ samples $n = 20$
- bottom: $10,000$ samples $n = 100$

under the "incorrect" model,
the larger the sample size,

- the more certain you are about your biased estimates
  (with enough you could reject the null)





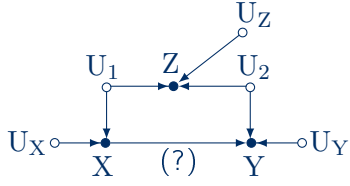
The dream team strikes back!!

based on DAG and statistical model,

- the 3rd D-separation states that a collider that has been conditioned on does not block a path,
  in this case: $X \to U_1 \to Z \to U_2 \to Y$
  i.e. $X \not\perp\!\!\!\perp Y \mid Z$
- therefore if we want to find the direct effect of $X \to Y$, we should not stratify by Z

$$M = \begin{cases} X \leftarrow f_X(U_1, U_X) \\ Z \leftarrow f_Z(U_1, U_2, U_Z) \\ Y \leftarrow f_Y(X, U_2, U_Y) \\ U \sim P(\mathbf{U}) \end{cases}$$

(a) structural model



(b) causal diagram

The dream team strikes back!!

based on DAG and statistical analysis,

- the model that answers our research question is the first one, (assuming our DAG is true)

```
> summary(lm(Y ~ X, data=d)) # unbiased effects (effic

Call:
lm(formula = Y ~ X, data = d)

Residuals:
    Min      1Q  Median      3Q     Max
-3.7452 -1.3300 -0.0253  1.2414  3.9479

Coefficients:
             Estimate Std. Error t value Pr(>|t|)
(Intercept)  3.067474   0.334586   9.168 7.74e-15 ***
X           -0.008255   0.097774  -0.084    0.933
```

3. Example cases

XXX bias: sensitivity analysis

3. Example cases

XXX bias: post-stratification

3. Example cases

Descendant fix: proxies (a)

Proxies (a)[24]

research question,

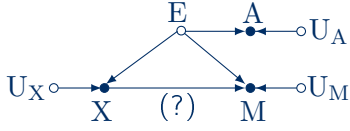
- Does X has a (direct) effect on M?

variables,

- X, teacher experience
- E, instruction type (old, new) (unobserved)
- A, proxy variable (e.g. age)
- M, teachers' score in mathematics (from a standardized evaluation)

$$M = \begin{cases} A \leftarrow f_A(E, U_A) \\ X \leftarrow f_X(E, U_X) \\ M \leftarrow f_M(E, X, U_M) \\ U \sim P(\mathbf{U}) \end{cases}$$

(a) structural model



(b) causal diagram

[24] McElreath [12], chapter 11 (p. 340); McElreath [13], lecture 10

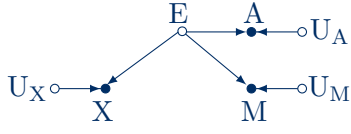
Simulation setting

```r
# sim
E = sample( 1:2, 100, replace=T)
A = round( rnorm( 100 , 35 + bEA*E))
X = rbinom( 100 , size=A , prob=0.5 + 0.2*E )
M = rnorm( n , 0*X + 2*E )
d = data.frame(X=X,A=A,E=E,M=M)
```

(c) R code

$$M = \begin{cases} A \leftarrow f_A(E, U_A) \\ X \leftarrow f_X(E, U_X) \\ M \leftarrow f_M(E, X, U_M) \\ U \sim P(\mathbf{U}) \end{cases}$$

(a) structural model



(b) causal diagram

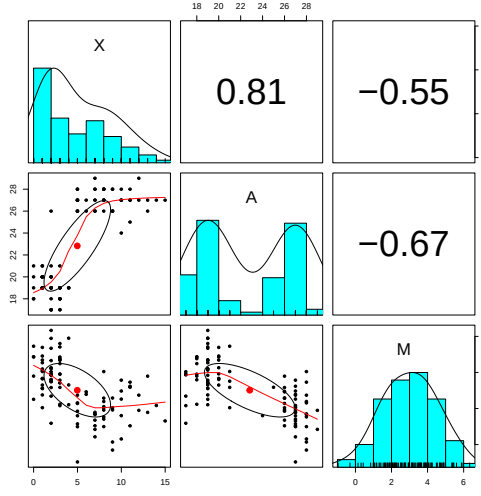
Implications,

- $A \not\perp M$
- $A \not\perp X$

“Eyeballing” analysis

based on correlation analysis,

- the task has become more complex
  (more variables to decide on)
- while $\mathrm{cor}(\mathrm{H}, \mathrm{I}) > 0$ indicate the more you work the more you gain
  (but is it the only way?)
- since $\mathrm{cor}(\mathrm{H}, \mathrm{I})$ is high we might include it as a covariate in our statistical model
  (to improve the precision?)



Regression, regression!!

based on statistical analysis,

- we now have two models with two different “levels” of effects
- which one is the “truth”?

descendant1_reg.pr

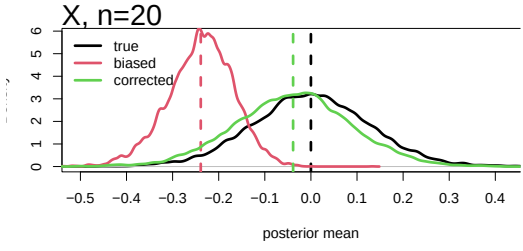
The data tell-tell story!!

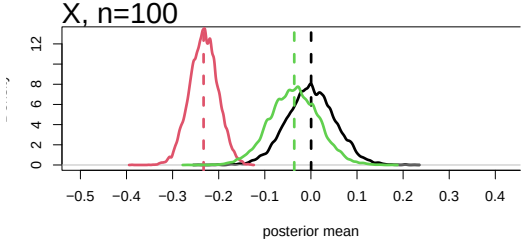
imagine we can continue sampling,

- top: $10,000$ samples $n = 20$
- bottom: $10,000$ samples $n = 100$

the larger the sample size,

- under the incorrect model, the more certain you are about your biased estimates
- under the corrected model, the more certain you are about your less biased estimates





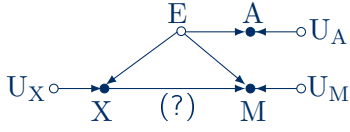
So, what is going on?

based on DAG and statistical model,

- stratifying on I explains variability in H (is his descendant)
  remaining variance is explained by E
  (big chunk is already explained)
- but there is more!!:
  H is now a collider in the path
  $E \rightarrow H \leftarrow U_H$ (virtual collider),
  then, stratifying by I opens that path
  (biasing the estimates).

$$M = \begin{cases} A \leftarrow f_A(E, U_A) \\ X \leftarrow f_X(E, U_X) \\ M \leftarrow f_M(E, X, U_M) \\ U \sim P(\mathbf{U}) \end{cases}$$

(a) structural model



(b) causal diagram

the dream team!!

based on DAG and statistical analysis,

- the less biased model is the first,
  (assuming our DAG is true)

3. Example cases

Descendant fix: proxies (b)

Proxies (b)[25]

research question,

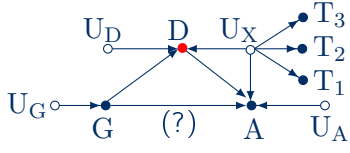
- Do females are discriminated in school admissions, i.e. does $G \to A$?

variables,

- G, gender
- D, department of application
- A, admission
- U_X, confound (e.g. ability) (unobserved)
- T_i, test scores, with $i = 1, 2, 3$ (U_{Ti} are reliabilities)

$$M = \begin{cases} G \leftarrow f_G(U_G) \\ D \leftarrow f_D(G, U_X, U_D) \\ A \leftarrow f_A(D, G, U_X, U_A) \\ T_i \leftarrow f_T(U_X, U_{Ti}) \\ U \sim P(\mathbf{U}) \end{cases}$$

(a) structural model



(b) causal diagram

[25] McElreath [12], chapter 11 (p. 340); McElreath [13], lecture 10

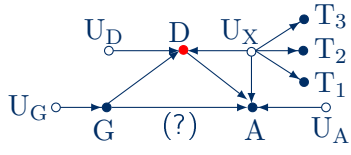
Simulation setting

```r
# sim
G = sample( 1:2, size=100, replace=T )
U = rbern(100, 0.1)
D = rbern( 100, ifelse( G==1, U*0.5, 0.8 ) ) + 1
ar[[1]] = matrix( c(0.1,0.1,0.1,0.3), nrow=2 )
ar[[2]] = matrix( c(0.2,0.3,0.2,0.5), nrow=2 )
T1 = rnorm(100, 1*U, 0.1)
T2 = rnorm(100, 1*U, 0.5)
T3 = rnorm(100, 1*U, 0.25)
p = sapply( 1:100 ,
            function(i){
              ifelse( U[i]==0,
                      ar[[1]][D[i],G[i]],
                      ar[[2]][D[i],G[i]] ) } )
A = rbern( 100 , p )
d = data.frame(U,T1,T2,T3,G=G,D=D,A)
```

(c) R code

$$M = \begin{cases} G \leftarrow f_G(U_G) \\ D \leftarrow f_D(G, U_X, U_D) \\ A \leftarrow f_A(D, G, U_X, U_A) \\ T_i \leftarrow f_T(U_X, U_T) \\ U \sim P(\mathbf{U}) \end{cases}$$

(a) structural model

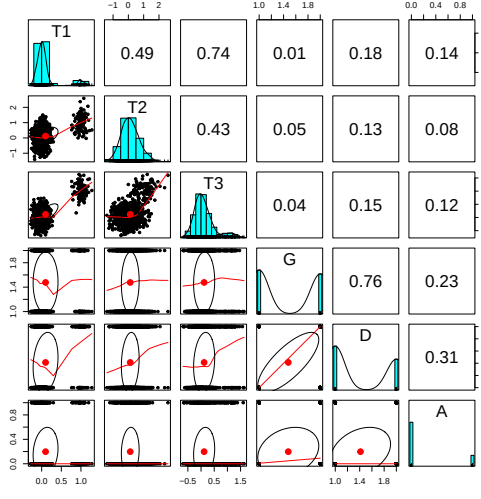


(b) causal diagram

“Eyeballing” analysis

based on correlation analysis,

- the task has become more complex
  (more variables to decide on)
- while $\mathrm{cor}(\mathrm{H}, \mathrm{I}) > 0$ indicate the more you work the more you gain
  (but is it the only way?)
- since $\mathrm{cor}(\mathrm{H}, \mathrm{I})$ is high we might include it as a covariate in our statistical model
  (to improve the precision?)



Regression, regression!!

based on statistical analysis,

- we now have two models with two different “levels” of effects
- which one is the “truth”?

descendant2_reg.pr

The data tell-tell story!!

imagine we can continue sampling,

- top: $10,000$ samples $n = 20$
- bottom: $10,000$ samples $n = 100$

under the incorrect model,
the larger the sample size,

- the more certain you are about your biased estimates

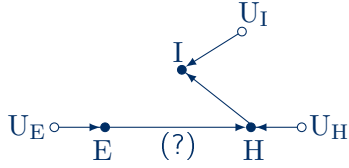
So, what is going on?

based on DAG and statistical model,

- stratifying on I explains variability in H (is his descendant)
  remaining variance is explained by E (big chunk is already explained)
- but there is more!!:
  H is now a collider in the path $E \rightarrow H \leftarrow U_H$ (virtual collider),
  then, stratifying by I opens that path (biasing the estimates).

$$M = \begin{cases} E \leftarrow f_E(U_E) \\ H \leftarrow f_H(E, U_H) \\ I \leftarrow f_I(H, U_I) \\ U \sim P(\mathbf{U}) \end{cases}$$

(a) structural model



(b) causal diagram

the dream team!!

based on DAG and statistical analysis,

- the less biased model is the first,
  (assuming our DAG is true)

descendant2_reg1.pr

3. Example cases

Descendant bias: case control

Case control[26]

also,

- virtual collider
- an instance of descendant bias

research question,

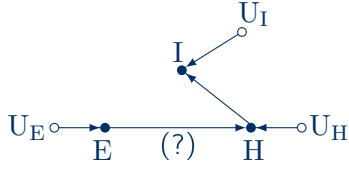
- Does E has a (direct) effect on H?
- Should we include I on our model?

variables,

- E, education
- H, hours in occupation (standardized)
- I, income

$$M = \begin{cases} E \leftarrow f_E(U_E) \\ H \leftarrow f_H(E, U_H) \\ I \leftarrow f_I(H, U_I) \\ U \sim P(\mathbf{U}) \end{cases}$$

(a) structural model



(b) causal diagram

[26] McElreath [13], lecture 6; Cinelli et al. [4] (p. 8, 19), Hernán [8], lesson 3

Simulation setting

```r
# sim
E = rnorm( 100 )
H = rnorm( 100 , -1*E )
I = rnorm( 100 , -1*H )
d = data.frame(E,H,I)
```

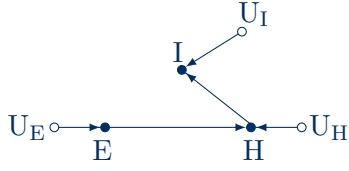
(c) R code

$$M = \begin{cases} E \leftarrow f_E(U_E) \\ H \leftarrow f_H(E, U_H) \\ I \leftarrow f_I(H, U_I) \\ U \sim P(\mathbf{U}) \end{cases}$$

(a) structural model

Implications,

- $E \not\perp\!\!\!\perp H$
- $E \perp\!\!\!\perp I \mid H$
- $E \not\perp\!\!\!\perp U_H \mid I$
  (virtual collider)

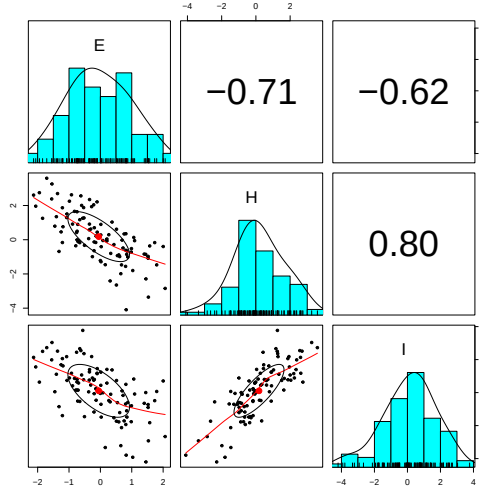


(b) causal diagram

“Eyeballing” analysis

based on correlation analysis,

- $\text{cor}(\text{E}, \text{I}) < 0$ does NOT goes in line of our “rudimentary” understanding of the data.
- while $\text{cor}(\text{H}, \text{I}) > 0$ indicate the more you work the more you gain (but is it the only way?)
- since $\text{cor}(\text{H}, \text{I})$ is high we might include it as a covariate in our statistical model (to improve the precision?)



Regression, regression!!

based on statistical analysis,

- we now have two models with two different “levels” of effects
- which one is the “truth”?

```
> summary(lm(H ~ E, data=d)) # unbiased effects

Call:
lm(formula = H ~ E, data = d)

Residuals:
     Min       1Q   Median       3Q      Max
-2.19319 -0.60621 -0.06694  0.55674  2.77776

Coefficients:
            Estimate Std. Error t value Pr(>|t|)
(Intercept)  0.04689    0.09534   0.492    0.624
E           -0.90223    0.08930 -10.104   <2e-16 ***
> summary(lm(H ~ E + I, data=d)) # biased effects

Call:
lm(formula = H ~ E + I, data = d)

Residuals:
     Min       1Q   Median       3Q      Max
-1.22991 -0.54882  0.01153  0.46886  1.79879

Coefficients:
            Estimate Std. Error t value Pr(>|t|)
(Intercept)  0.05036    0.06861   0.734    0.465
E           -0.45847    0.07915  -5.793 8.57e-08 ***
I            0.55017    0.05728   9.604 9.61e-16 ***
```

The data tell-tell story!!

imagine we can continue sampling,

- top: $10,000$ samples $n = 20$
- bottom: $10,000$ samples $n = 100$

under the incorrect model,
the larger the sample size,

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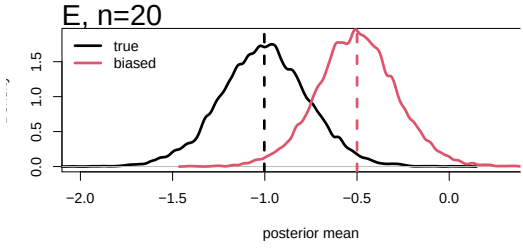

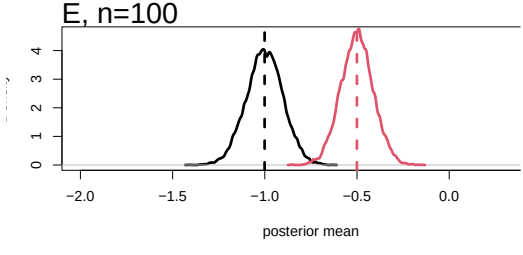

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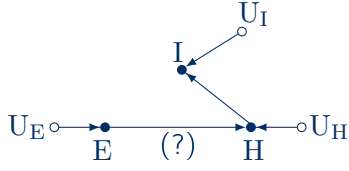
(a) structural model



(b) causal diagram

the dream team!!

based on DAG and statistical analysis,

- the less biased model is the first,
  (assuming our DAG is true)

```
> summary(lm(H ~ E, data=d)) # unbiased effects

Call:
lm(formula = H ~ E, data = d)

Residuals:
     Min       1Q   Median       3Q      Max
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Coefficients:
            Estimate Std. Error t value Pr(>|t|)
(Intercept)  0.04689    0.09534   0.492    0.624
E           -0.90223    0.08930 -10.104   <2e-16 ***
```

4. Concluding remarks

Concluding remarks

- Research is filled with challenges
  (you: duh!!)
- Statistical models are not theory
  (you: so obvious again!!)
- Don't trust your statistical model when no DAG is involved
  (me: how about that?!)
- For explanation, without a DAG the (sample) size does not matter
  (me: sorry?!)
- For prediction, sometimes a DAG can help
  (me: did you expect this one?!)

Concluding remarks

Additionally, simulation can serve as,

- a reflection of a hypothesis, and its research complexities
  (me: with DAGs baby!!)
- a place where you can reflect the status of a population
  (test "what happens if")
- a data where you can test your statistical model
  (parameter recovery, power)
- a tool for planning (all kinds of) experiments.

5. Do you wanna know more???

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[1] Anderson, D. [2008]. Model Based Inference in the Life Sciences: A Primer on Evidence, Springer.

[2] Bareinboim, E. and Pearl, J. [2016]. Causal inference and the data-fusion problem, Proceedings of the National Academy of Sciences 113(27): 7345–7352. doi: https://doi.org/10.1073/pnas.1510507113.

[3] Chamberlain, T. [1965]. The method of multiple working hypotheses, Science 148(3671): 754–759.
url: https://www.jstor.org/stable/1716334.

[4] Cinelli, C., Forney, A. and Pearl, J. [2021]. A crash course in good and bad controls, Technical report.

[5] Cunningham, S. [2022]. Causal inference: The mixtape.
url: https://mixtape.scunning.com/index.html.

[6] Fogarty, L., Madeleine, A., Holding, T., Powell, A. and Kandler, A. [2022]. Ten simple rules for principled simulation modelling, PLOS Computational Biology 18(3): 1–8.
doi: https://doi.org/10.1371/journal.pcbi.1009917.

[7] Hanck, C., Arnold, M., Gerber, A. and Schmelzer, M. [2021]. Introduction to econometrics with r.
url: https://www.econometrics-with-r.org/index.html.

[8] Hernán, M. [2020]. Causal diagrams: Draw your assumptions before your conclusions.
url: https://www.edx.org/course/causal-diagrams-draw-your-assumptions-before-your.

[9] Hernán, M. and Robins, J. [2020]. Causal Inference: What If, 1 edn, Chapman and Hall/CRC.
url: https://www.hsph.harvard.edu/miguel-hernan/causal-inference-book.

[10] Jaynes, E. [2003]. Probability Theory: The Logic of Science, Cambridge University Press.

[11] McElreath, R. [2019]. Statistical rethinking, 2019 course.
url: https://github.com/rmcelreath/statrethinking_winter2019.

[12] McElreath, R. [2020]. Statistical Rethinking: A Bayesian Course with Examples in R and STAN, Chapman and Hall/CRC.

[13] McElreath, R. [2022]. Statistical rethinking, 2022 course.
url: https://github.com/rmcelreath/stat_rethinking_2022.

[14] Pearl, J. [1988]. Probabilistic reasoning in intelligent systems: Networks of plausible inference, The Journal of Philosophy 88(8): 434–437.
doi: https://doi.org/10.2307/2026705.
url: https://www.jstor.org/stable/2026705.

[15] Pearl, J. [2009]. Causality: Models, Reasoning and Inference, Cambrige University Press.
[16] Pearl, J. [2019]. The seven tools of causal inference, with reflections on machine learning, Communications of the ACM 62(3): 54–60.
doi: https://doi.org/10.1177/0962280215586010.
[17] Pearl, J., Glymour, M. and Jewell, N. [2016]. Causal Inference in Statistics: A Primer, John Wiley Sons, Inc.
[18] Pearl, J. and Mackenzie, D. [2018]. The Book of Why: The New Science of Cause and Effect, 1st edn, Basic Books, Inc.
[19] Spirtes, P., Glymour, C. and Scheines, R. [1991]. From probability to causality, Philosophical Studies 64(1): 1–36.
url: https://www.jstor.org/stable/4320244.
[20] Textor, J., van der Zander, B., Gilthorpe, M., Liskiewicz, M. and Ellison, G. [2016]. Robust causal inference using directed acyclic graphs: the r package 'dagitty', Int J Epidemiol 45(6): 1887–1894.
doi: https://doi.org/10.1093/ije/dyw341.
[21] Yarkoni, T. [2020]. The generalizability crisis, The Behavioral and brain sciences 45(e1).
doi: https://doi.org/10.1017/S0140525X20001685.