

Causal Thinking

Using scientific knowledge to create probabilistic models

Diogo Melo

What are models for?

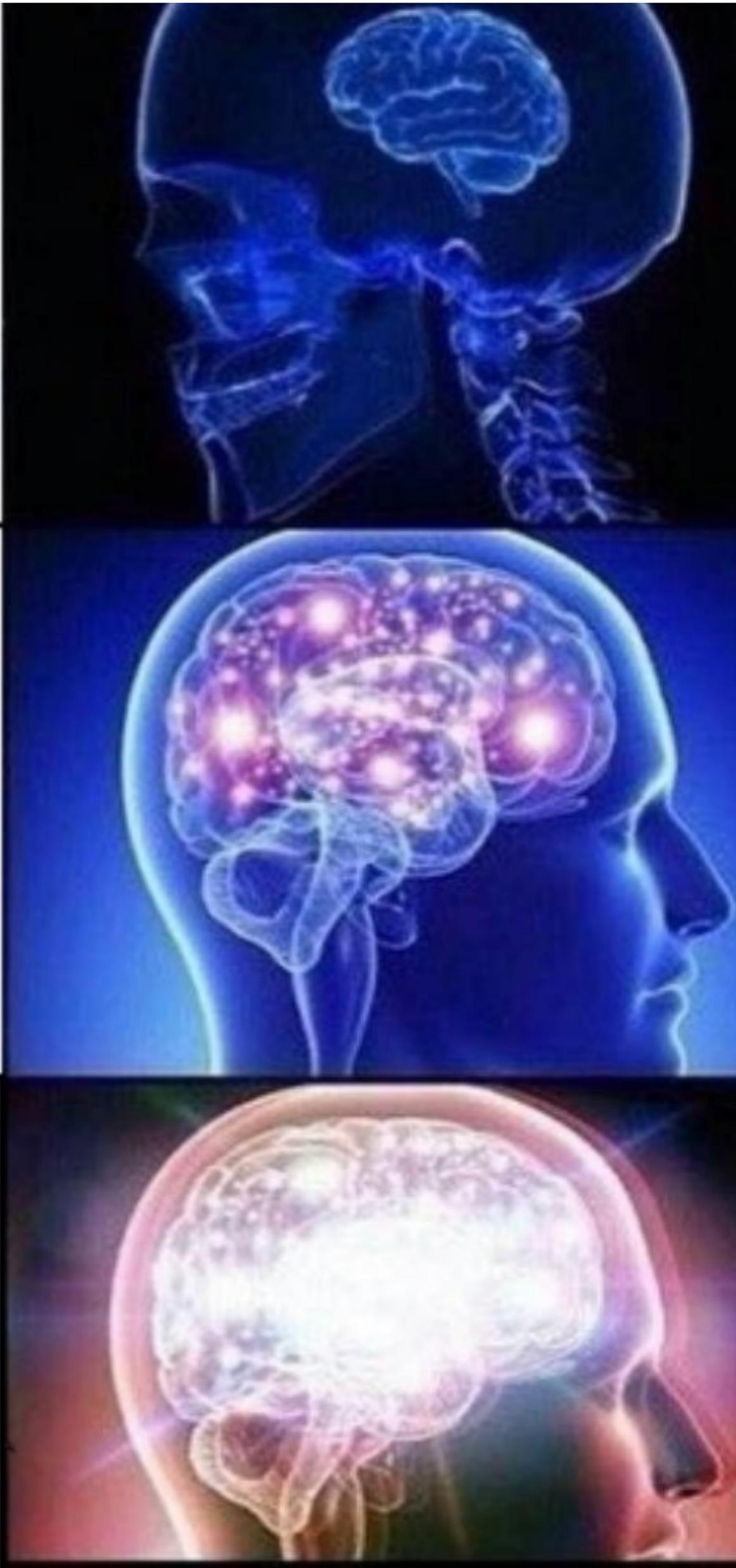
Prediction vs. Causal Inference

Correlation does not imply causation
But why?!

**CORRELATION
IMPLIES
CAUSATION**

**CORRELATION
DOES NOT
IMPLY CAUSATION**

**CAUSATION
DOES NOT
IMPLY CORRELATION**



Graph model representation

- We can use graphs to represent our putative causal model.
- An arrow between variables represents a potential causal effect.



This is a Directed Acyclic Graph, a **DAG**

Elemental triads

- All DAGs can be decomposed into a set of 3 elemental motifs:
- The pipe, the fork and the collider
- We can use these to structure our thinking about our models and decide what variable to include or exclude

The pipe:



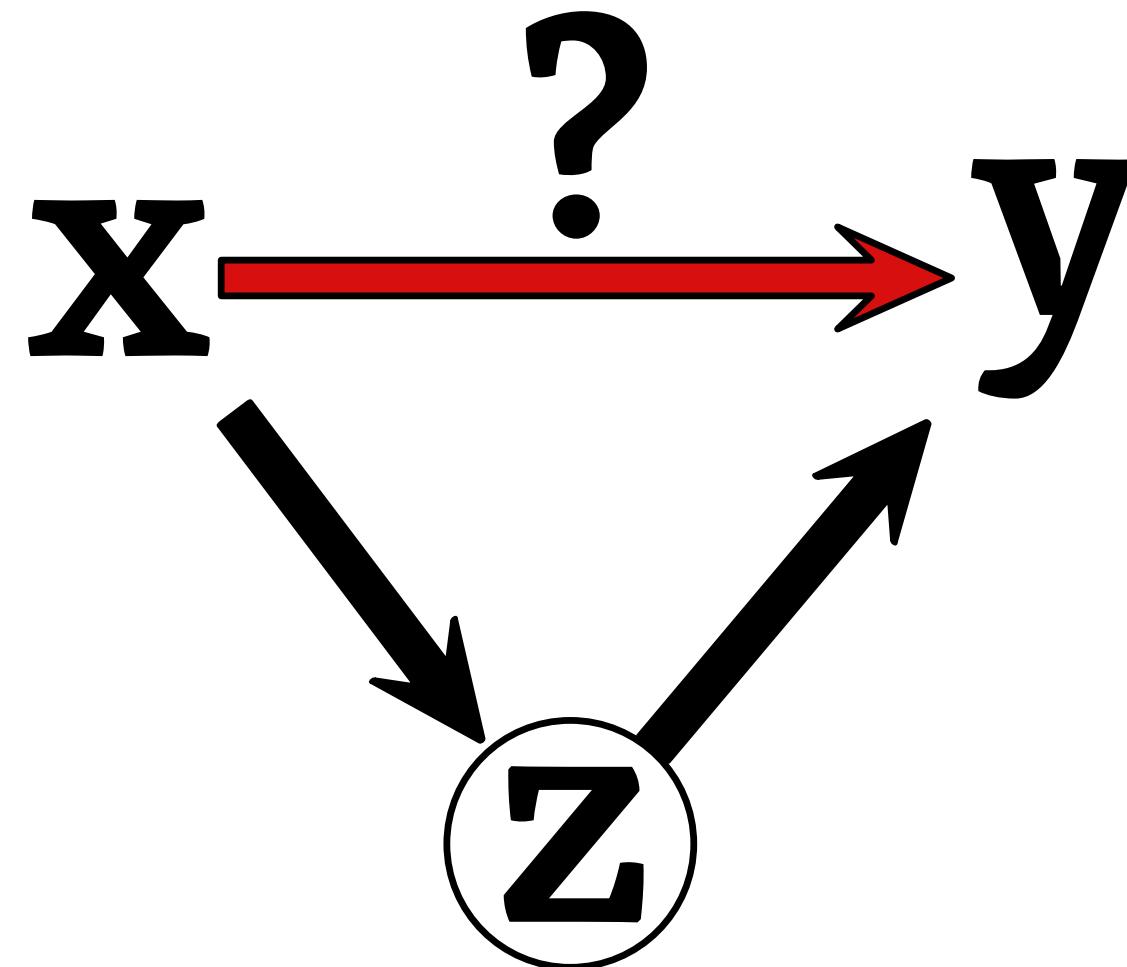
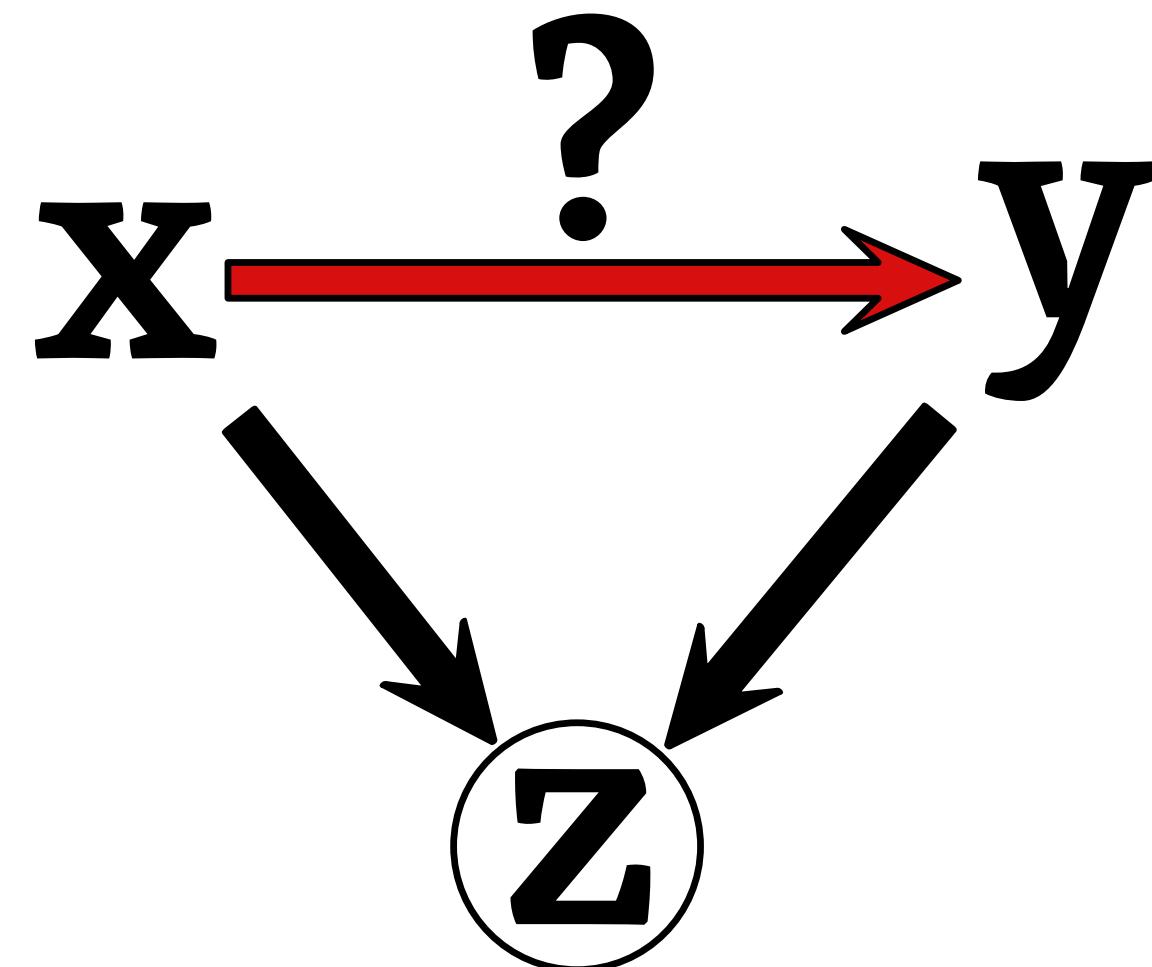
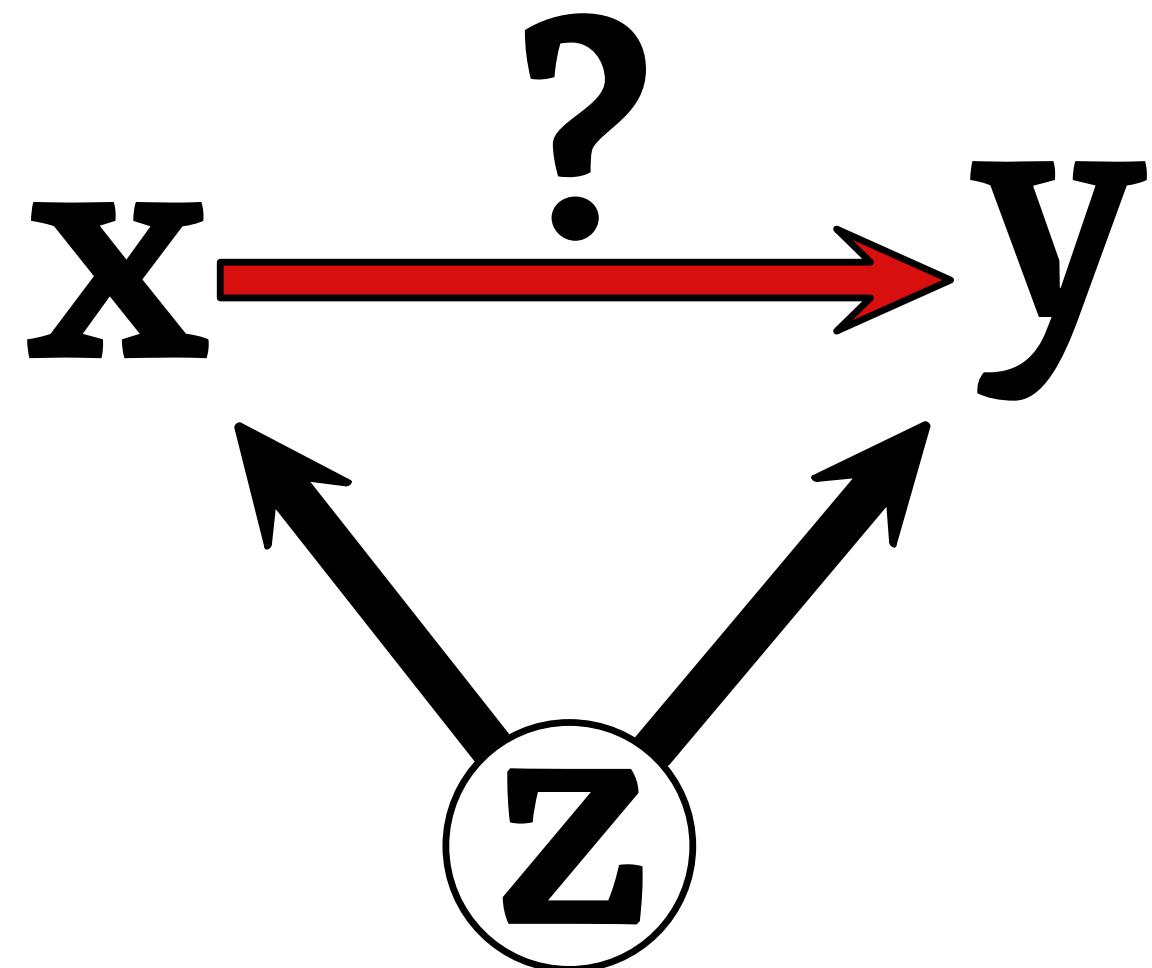
The fork:



The collider:



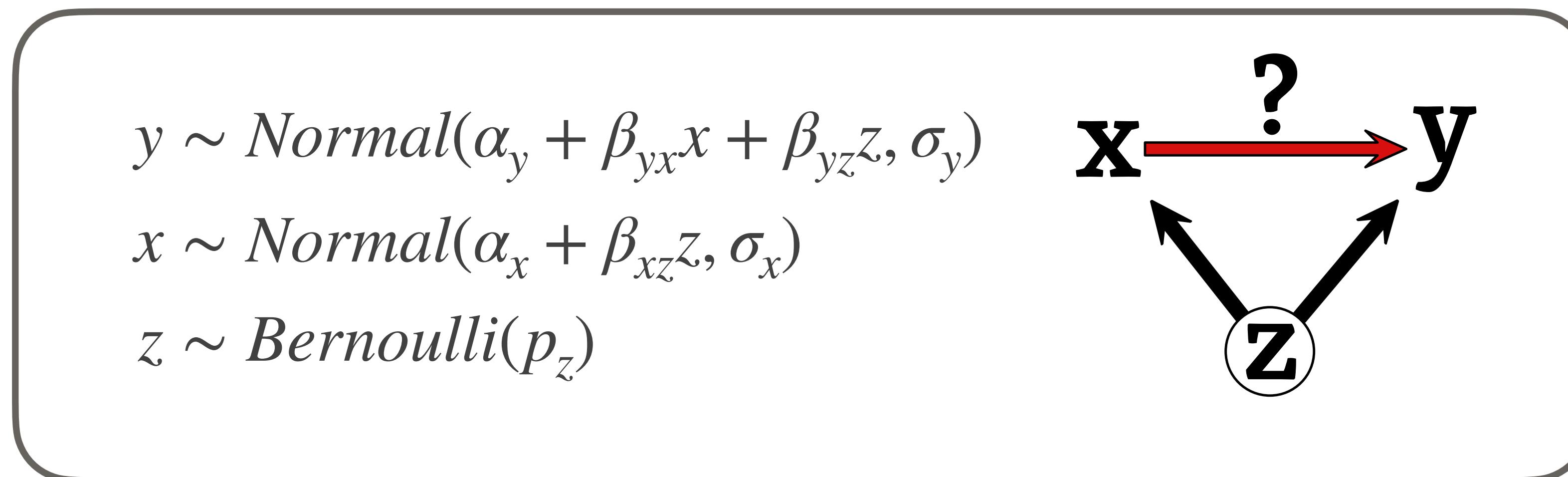
How does a confounder affect our estimate of the effect of x on y ?



The fork

Simulating a shared cause

- Every DAG implies a causal relation between variables.
- We can use distributions to simulate the generative model implied by this DAG:



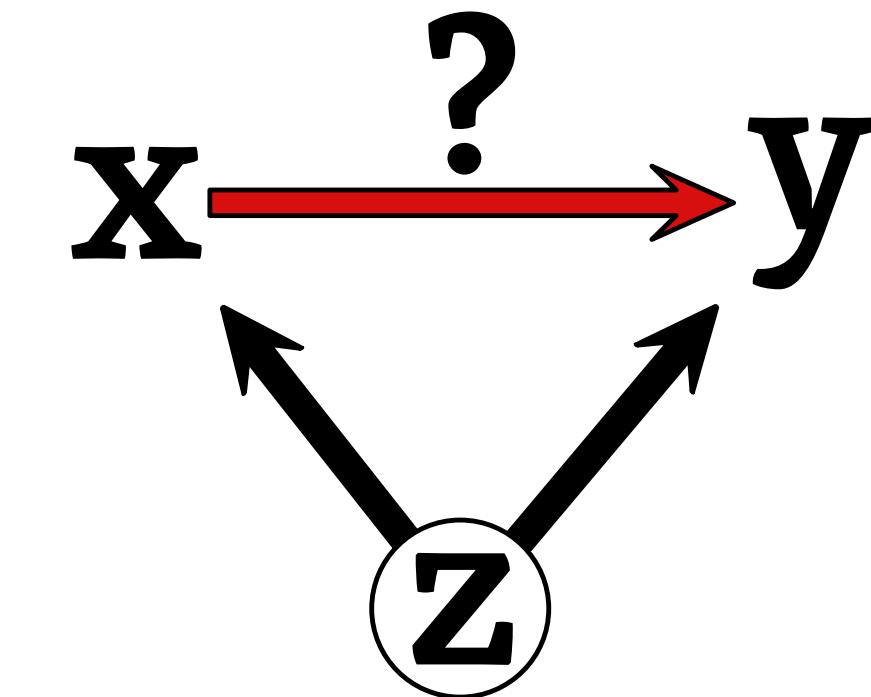
Simulating a shared cause

Math

$$y \sim Normal(\mu = 1 + 0.5x + 2z, \sigma = 1)$$

$$x \sim Normal(\mu = 1 + z, \sigma = 1)$$

$$z \sim Bernoulli(p = 0.5)$$



R Code

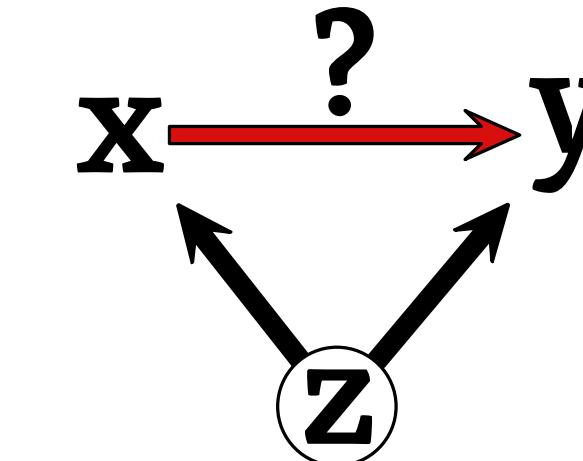
```
N = 200
z = rbinom(N, 1, 0.5)          # z ~ bernoulli(0.5)
x = rnorm(N, 1 + z)            # x ~ normal(1 + z, 1)
y = rnorm(N, 1 + 0.5*x + 2*z) # y ~ normal(1 + 0.5x + 2z, 1)
```

Statistical model without the confounder z

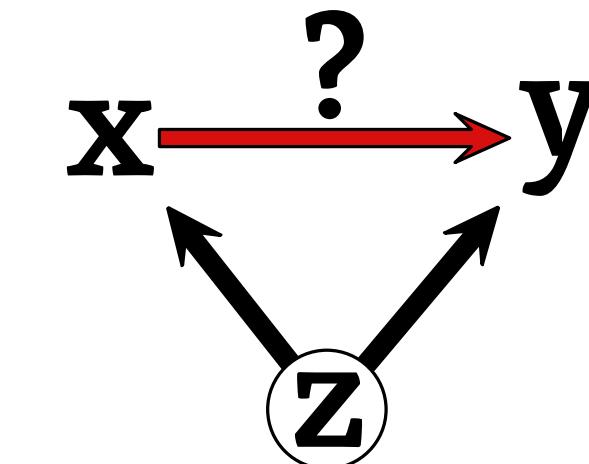
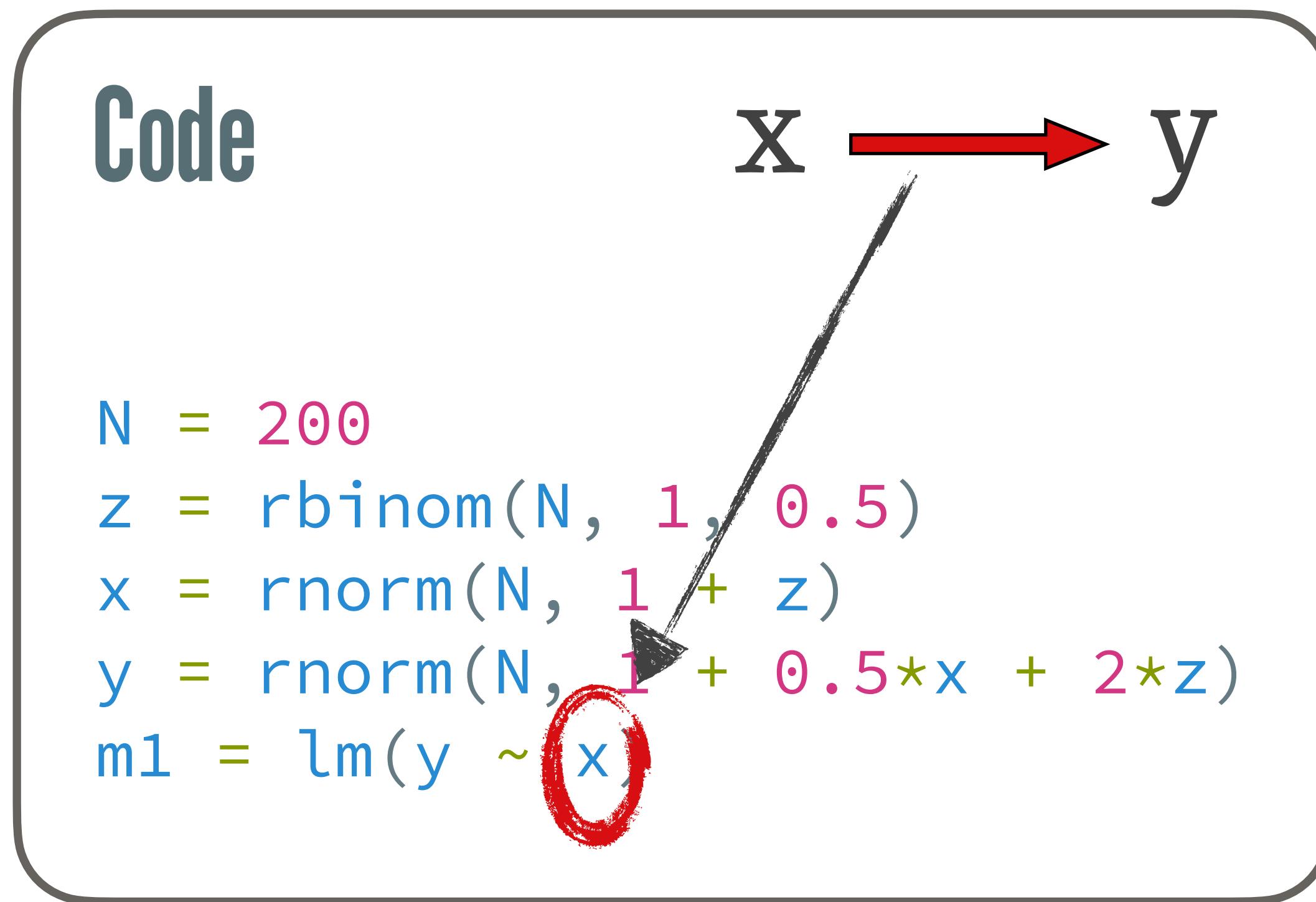
Code

$x \rightarrow y$

```
N = 200
z = rbinom(N, 1, 0.5)
x = rnorm(N, 1 + z)
y = rnorm(N, 1 + 0.5*x + 2*z)
m1 = lm(y ~ x)
```



Statistical model without the confounder z



Model estimates without the confounder

```
> (pm1 = precis(m1))
      mean   sd 5.5% 94.5%
(Intercept) 1.18 0.16 0.93  1.43
x            0.94 0.08 0.82  1.06
```

$$x \xrightarrow{\text{red arrow}} y$$

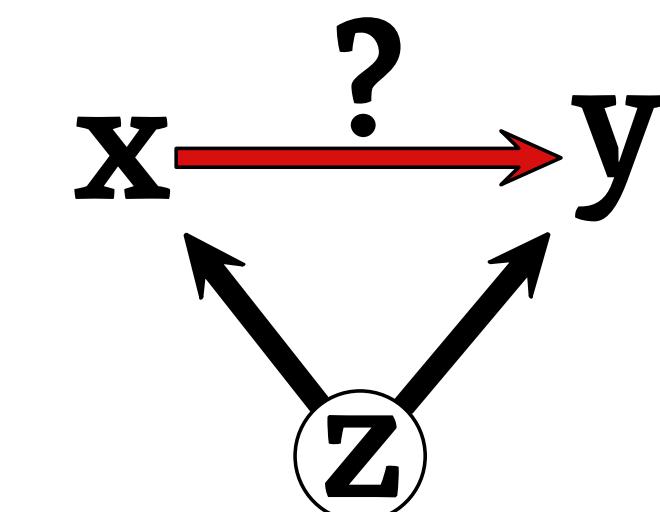
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Simulation R code

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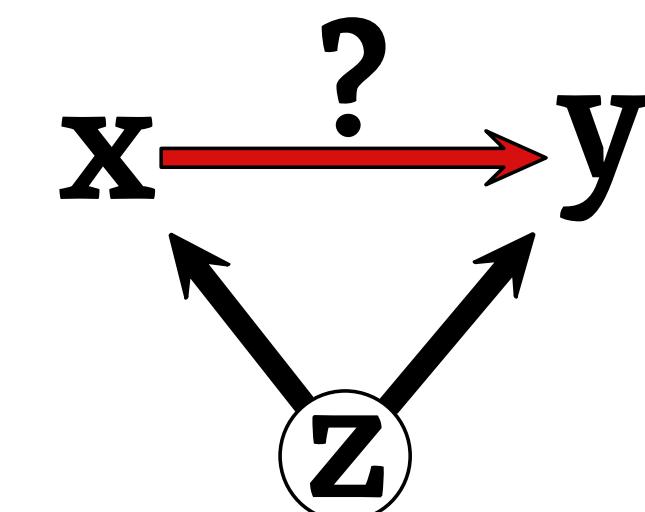
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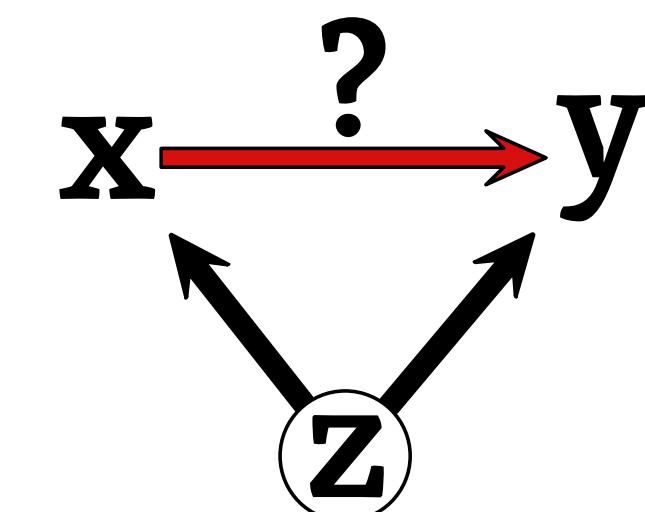
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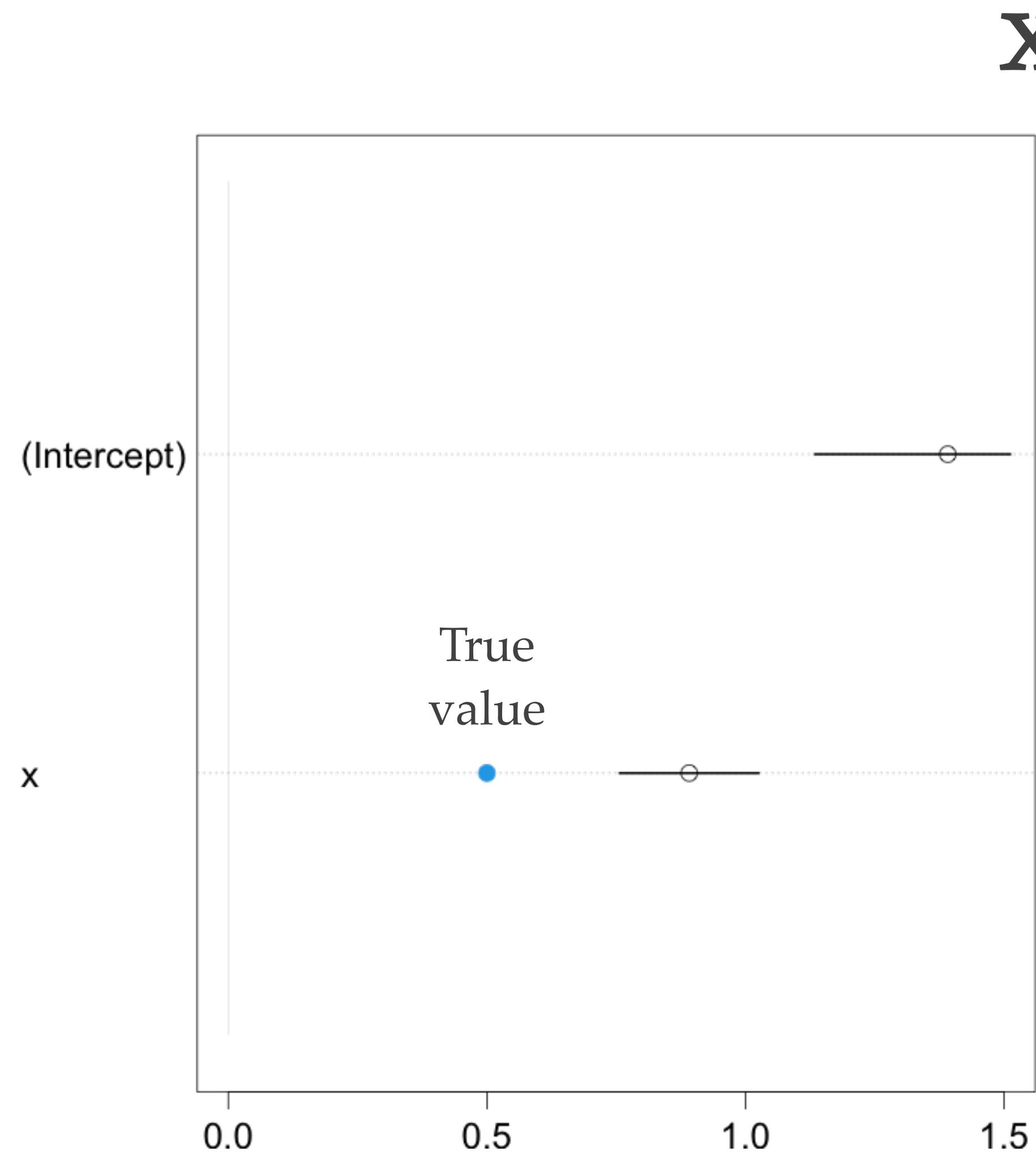


Simulation R code

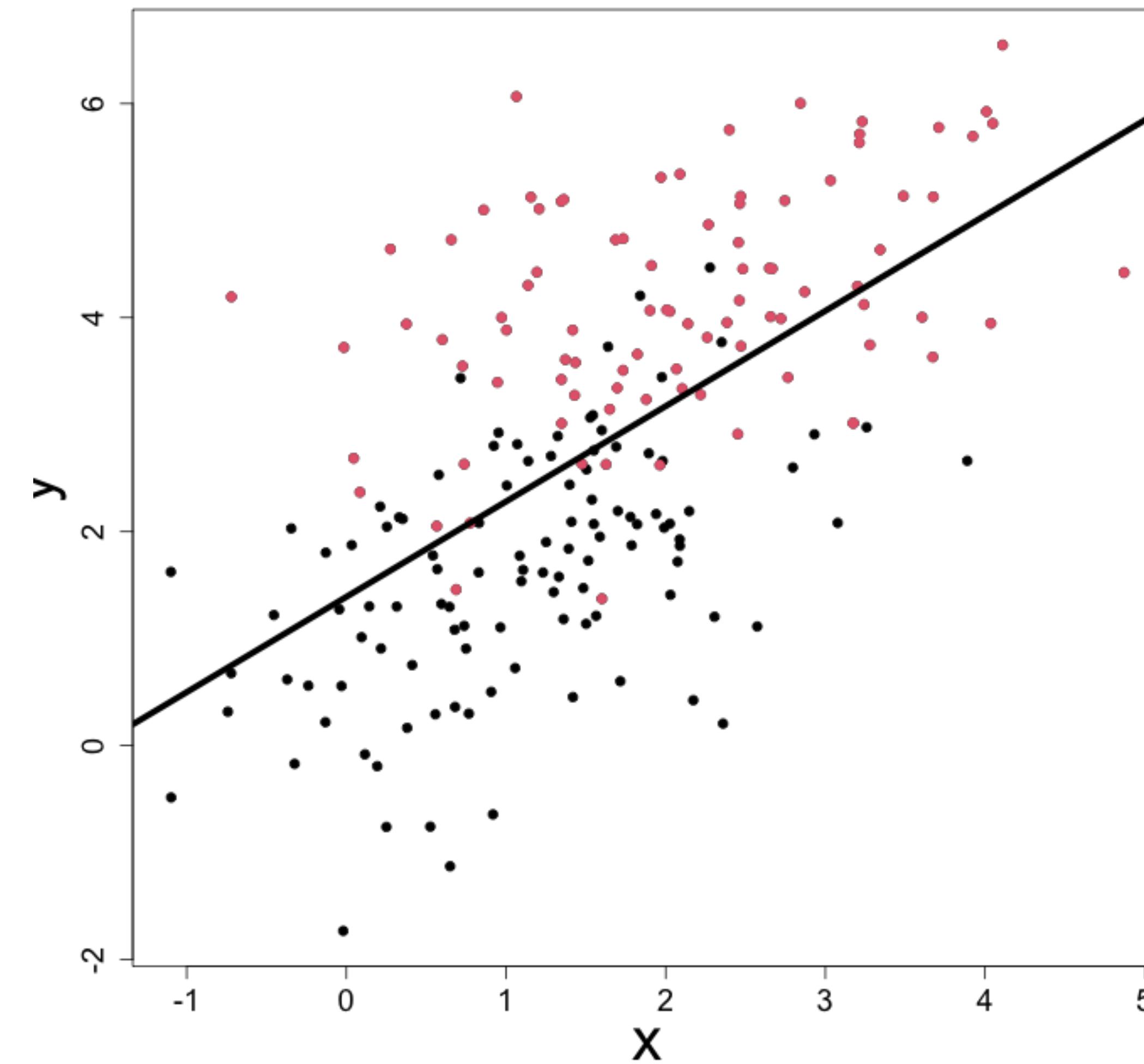
```
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```



Estimate of the effect of x on y without the confounder



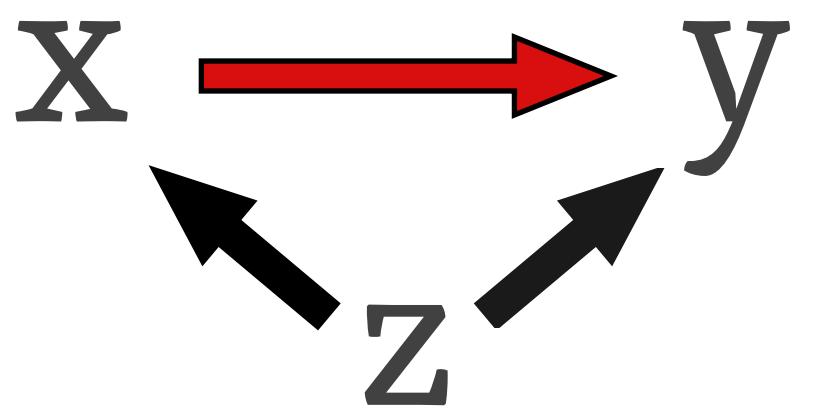
$x \rightarrow y$



Including the confounder

Statistically stratifying by z

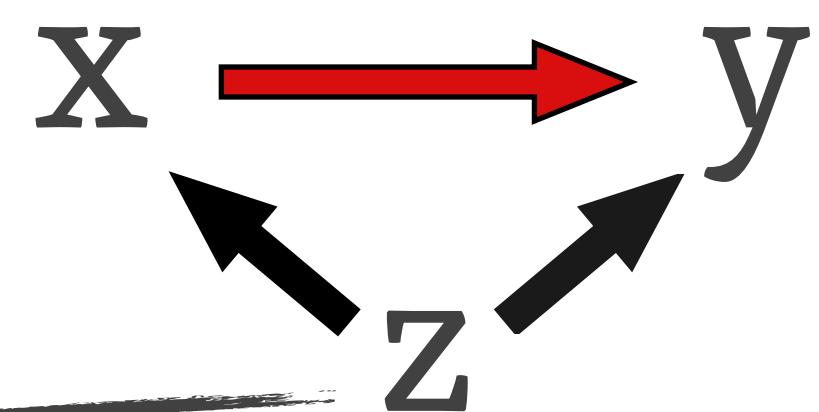
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(Intercept) 0.92  0.12  0.73  1.12
x            0.48  0.07  0.37  0.60
z            2.03  0.17  1.75  2.31
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Including the confounder

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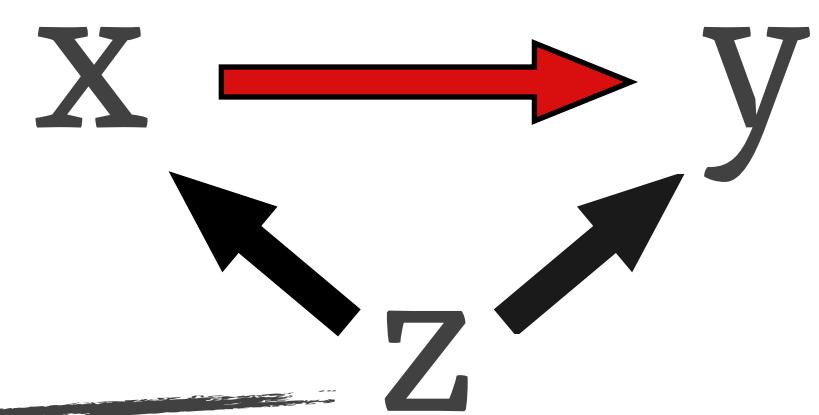
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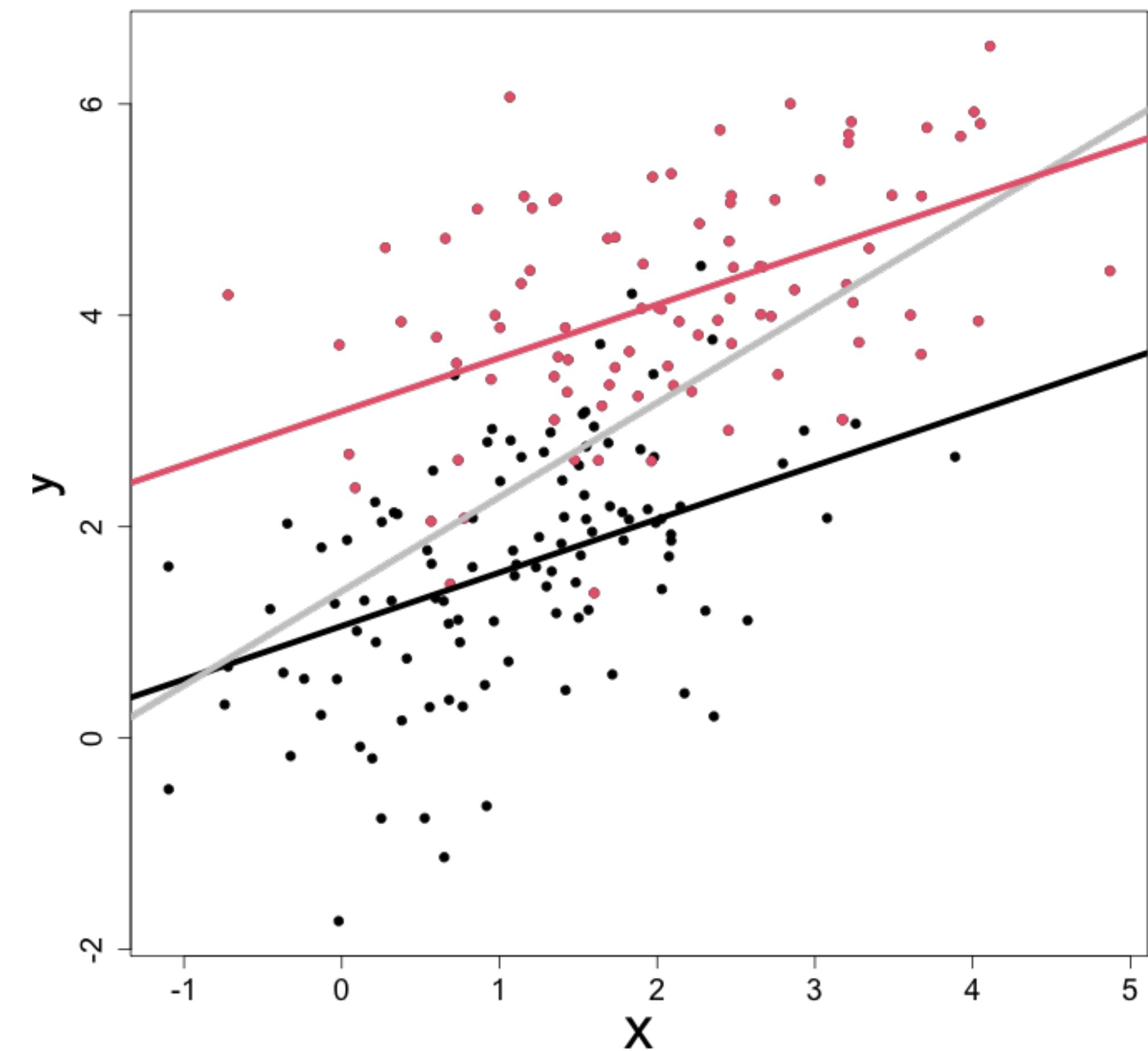
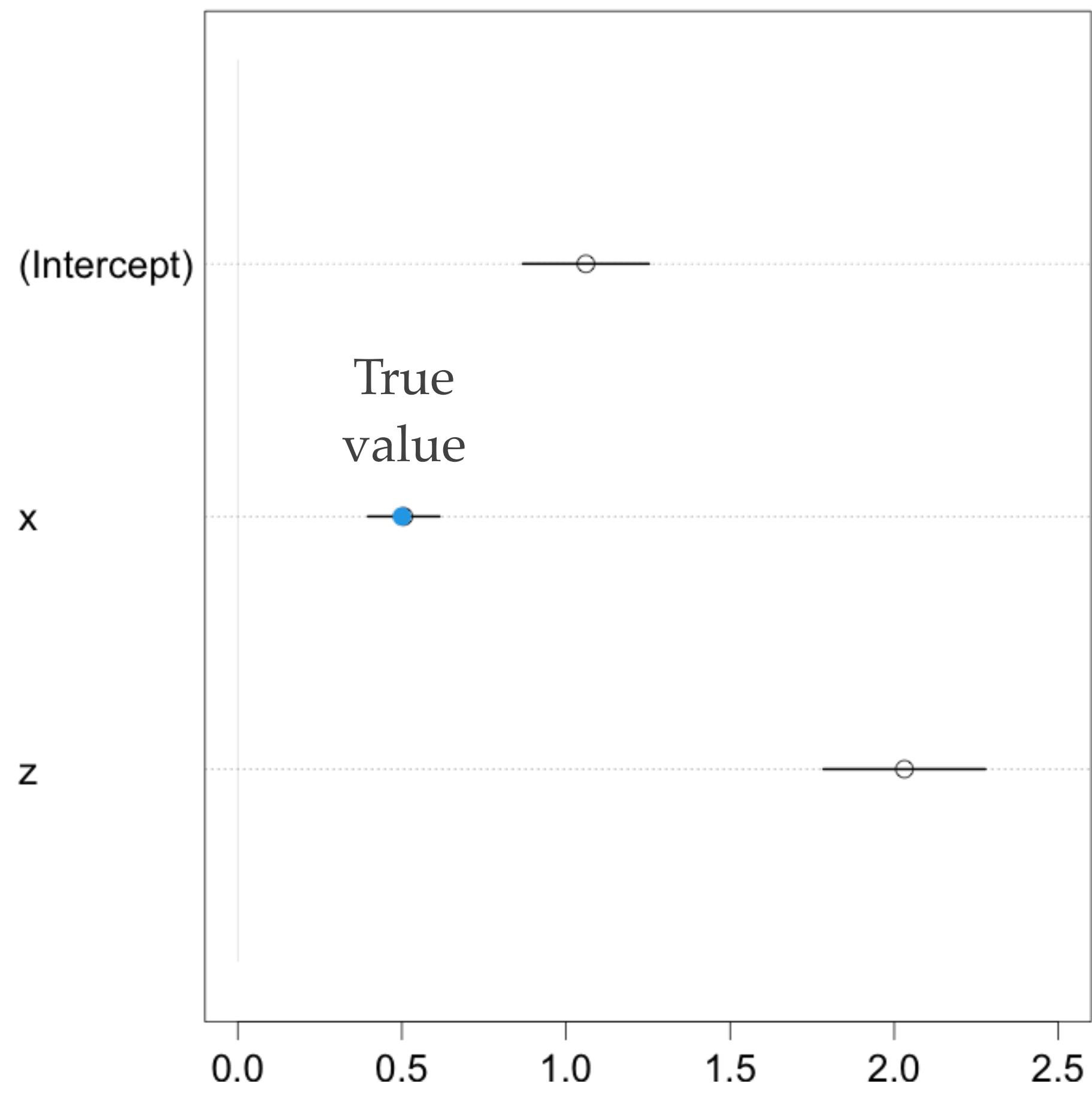
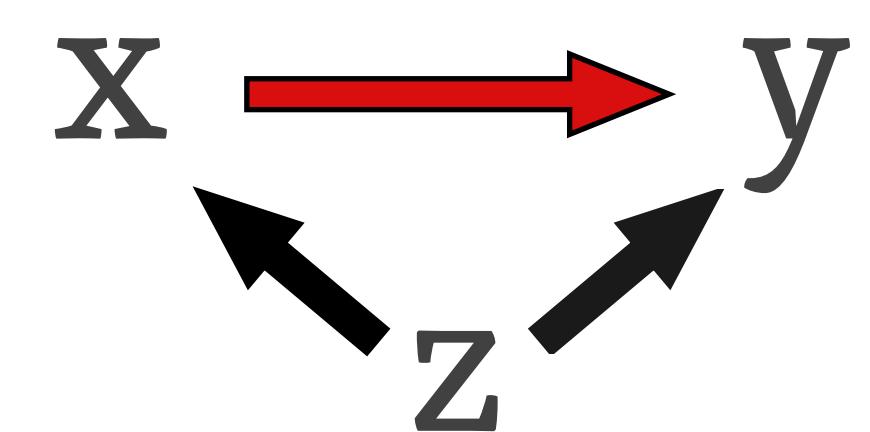
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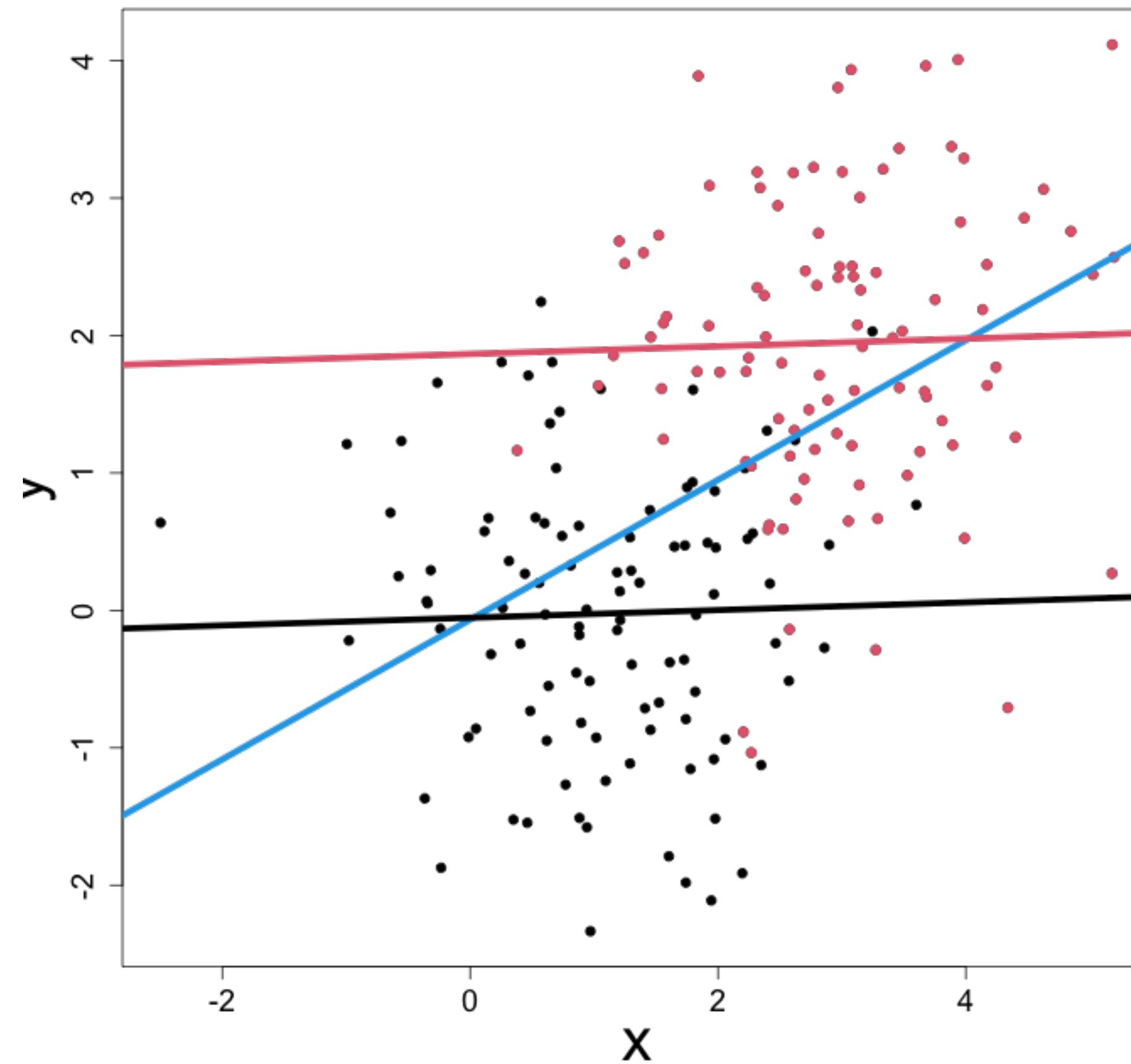


Including the confounder

Statistically stratifying by z

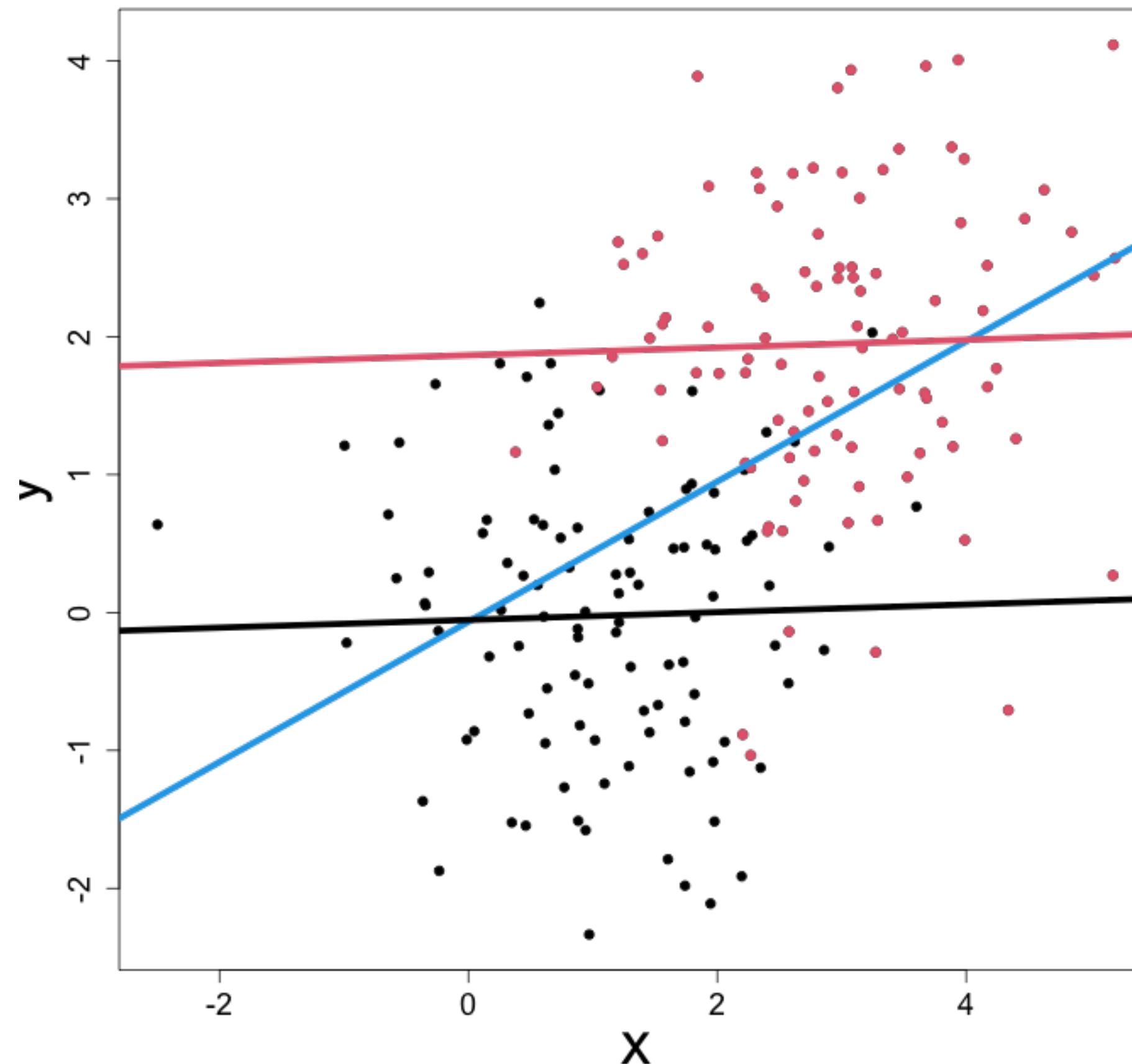


Confounders effect on the slope β_{xy}

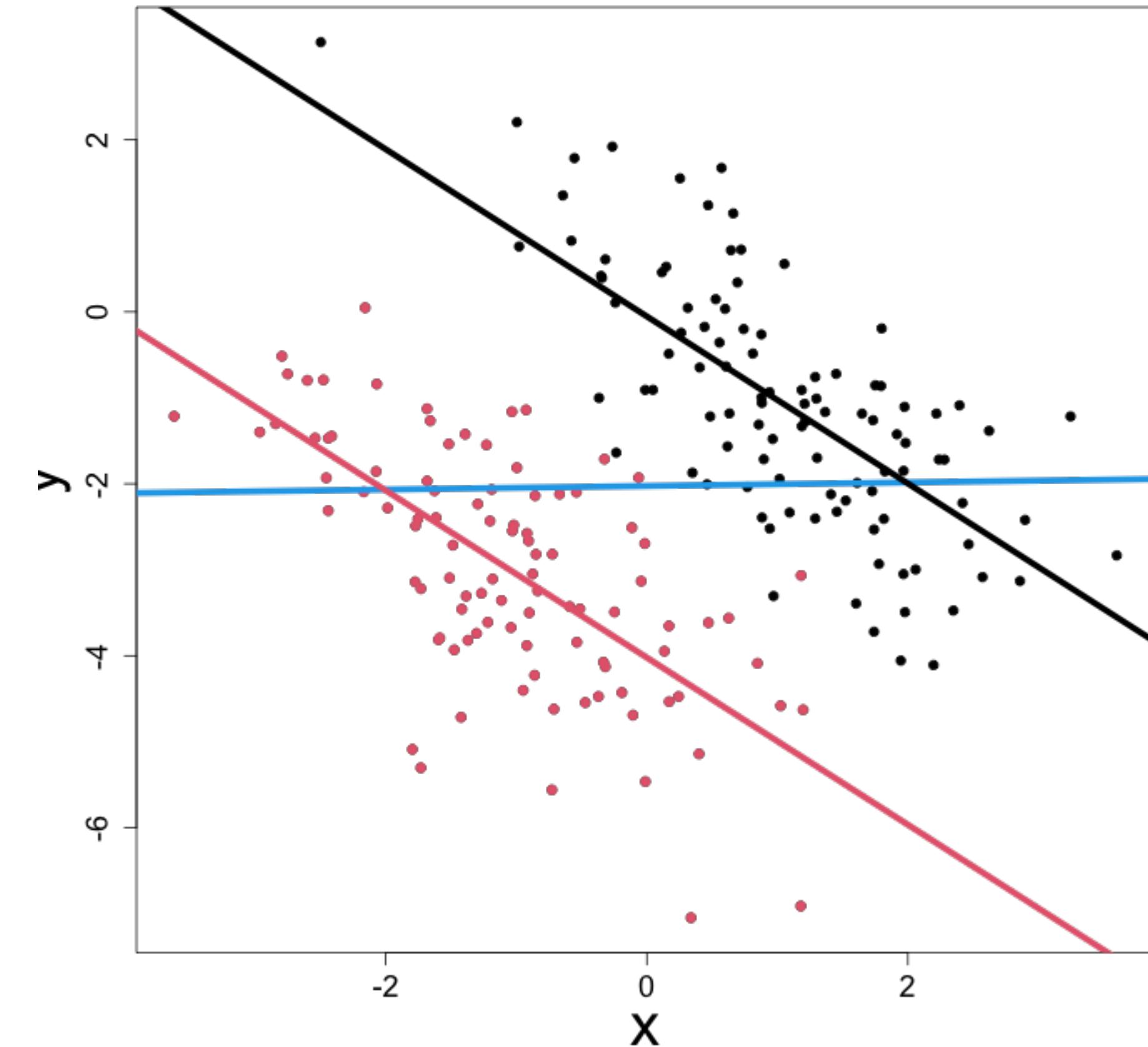


Confounders effect on the slope β_{xy}

Effect on y is created by z

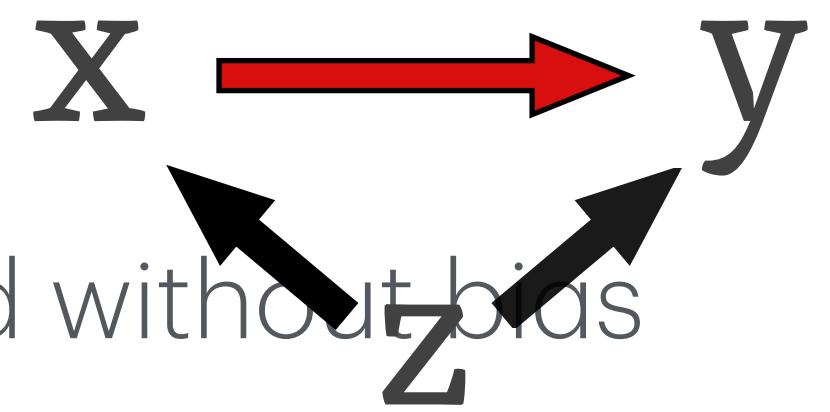


Effect on y is hidden by z



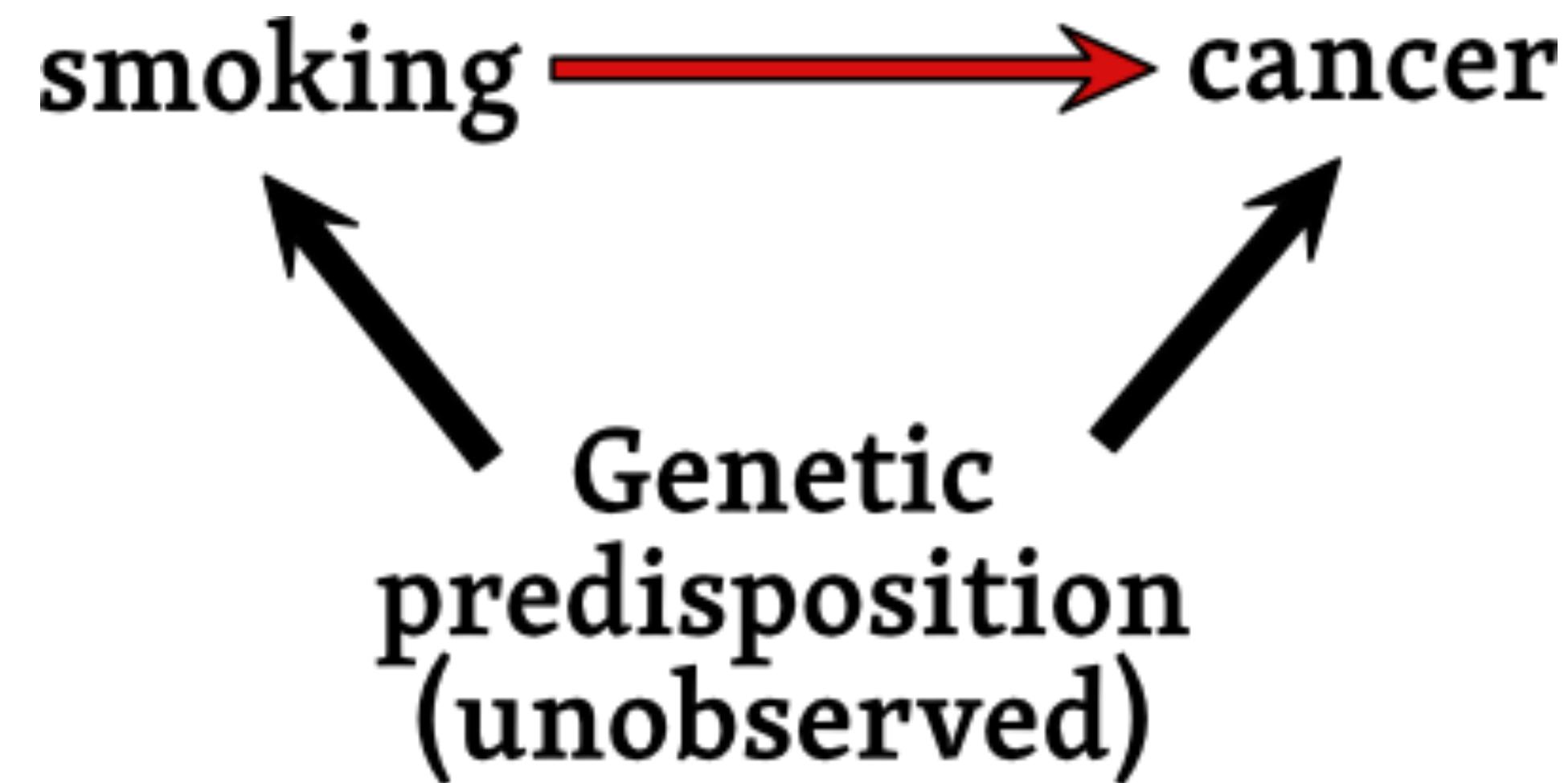
Examples of forks or confounders

- This is the quintessential “control” variable.
- Most variables are included in the model under the assumption that they are confounders and need have their effects taken into consideration.
- Conditioning on z, the causal effect of x on y can be estimated without bias



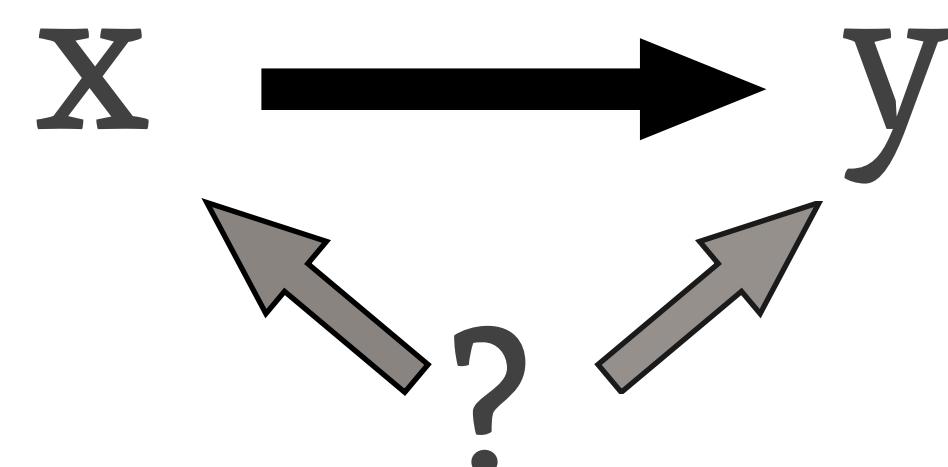
Smoking and Cancer

Famously, R. A. Fisher was not convinced that smoking caused cancer, and proposed that an unobserved propensity variable caused both cancer and smoking



Quantifying Omitted Variable bias

- Even without knowing the omitted variables, we can estimate how intense their effect would need to be in order to mask/cause the observed effect
- We can compare this estimated effect with known effects
 - **Ex:** an omitted variable would have to be twice as strong as smoking to explain the observed effect



Making sense of sensitivity: extending omitted variable bias

Carlos Cinelli and Chad Hazlett

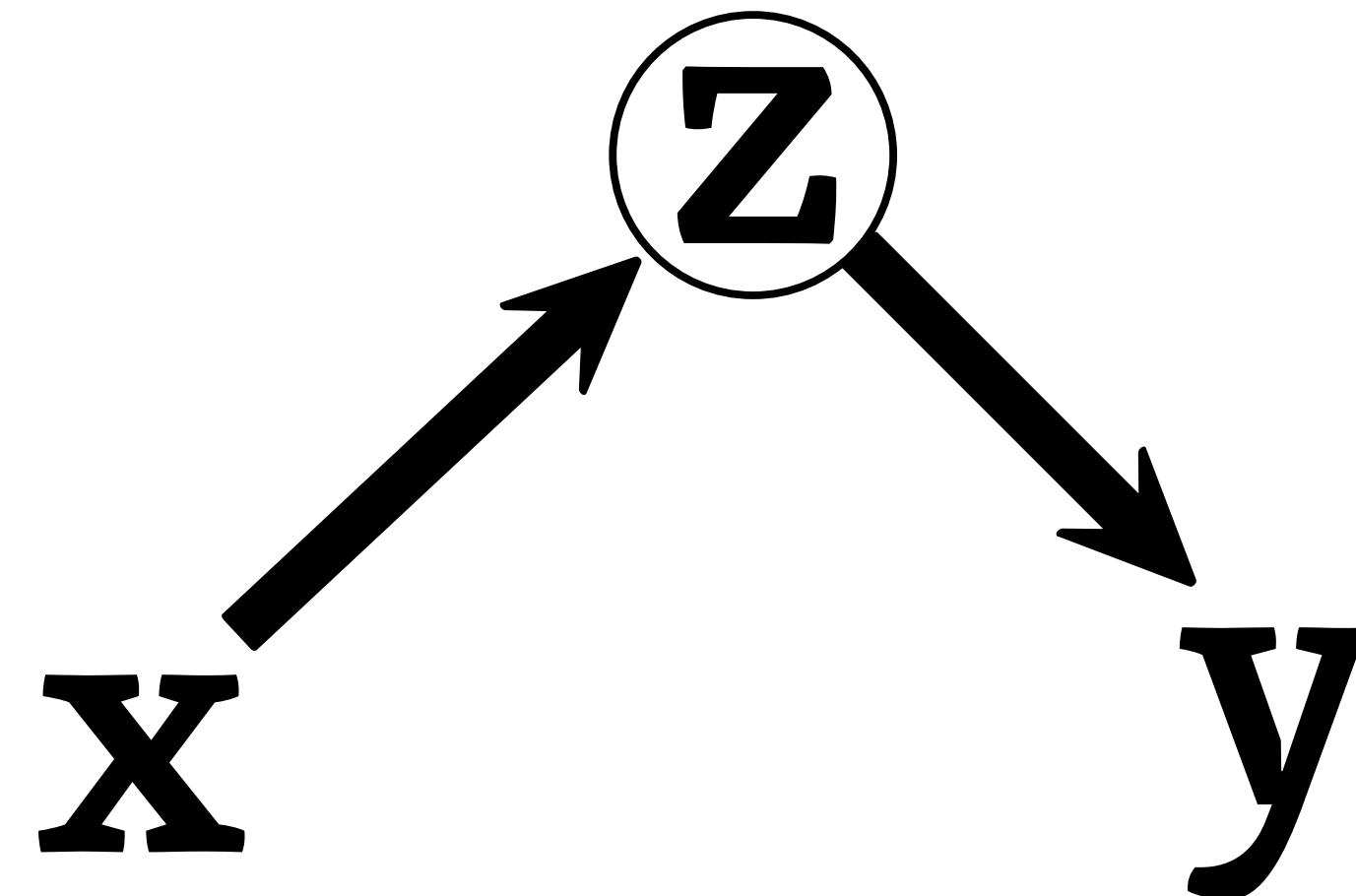
University of California, Los Angeles, USA

[Received August 2018. Final revision October 2019]

Summary. We extend the omitted variable bias framework with a suite of tools for sensitivity analysis in regression models that does not require assumptions on the functional form of the treatment assignment mechanism nor on the distribution of the unobserved confounders, naturally handles multiple confounders, possibly acting non-linearly, exploits expert knowledge to bound sensitivity parameters and can be easily computed by using only standard regression results. In particular, we introduce two novel sensitivity measures suited for routine reporting. The robustness value describes the minimum strength of association that unobserved confounding would need to have, both with the treatment and with the outcome, to change the research conclusions. The partial R^2 of the treatment with the outcome shows how strongly confounders explaining all the residual outcome variation would have to be associated with the treatment to eliminate the estimated effect. Next, we offer graphical tools for elaborating on problematic confounders, examining the sensitivity of point estimates and t -values, as well as ‘extreme scenarios’. Finally, we describe problems with a common ‘benchmarking’ practice and introduce a novel procedure to bound the strength of confounders formally on the basis of a comparison with observed covariates. We apply these methods to a running example that estimates the effect of exposure to violence on attitudes toward peace.

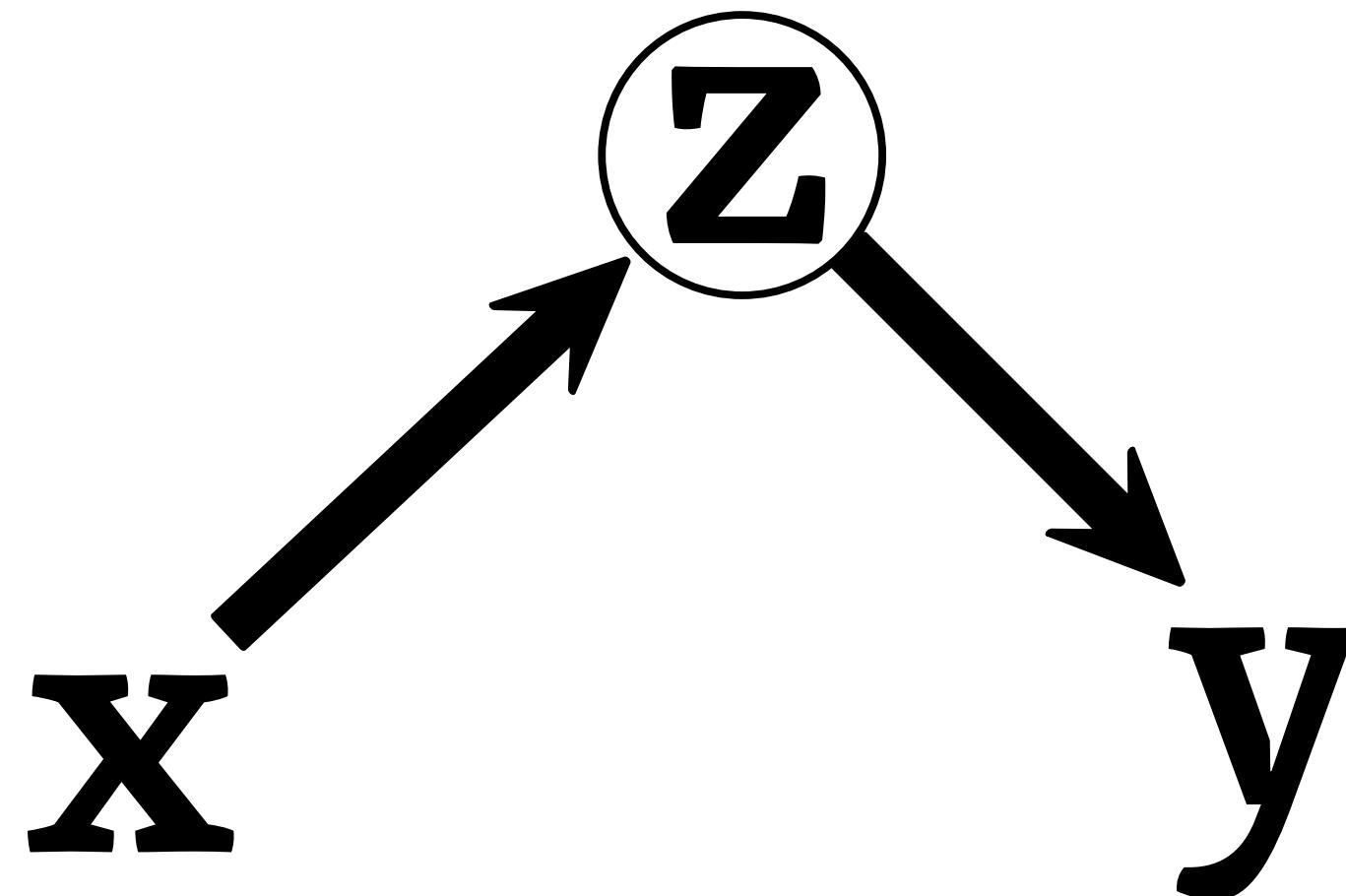
The pipe

All of the effect is mediated by z



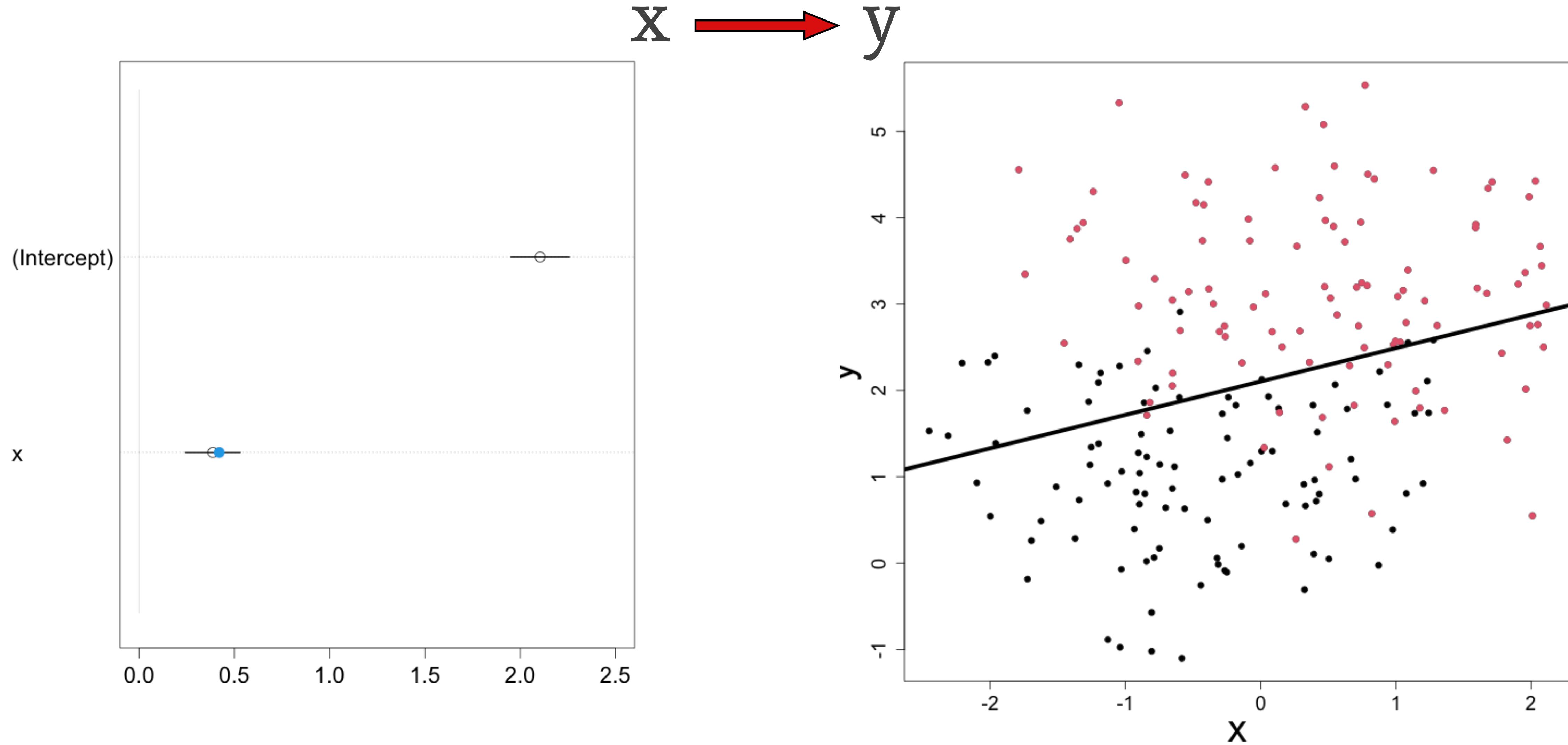
```
N = 200
x = rnorm(N)                      # x ~ normal(0, 1)
z = rbinom(N, 1, inv_logit(x))    # z ~ bernoulli(invlogit(x))
y = rnorm(N, 1 + 2*z)              # y ~ normal(1 + 2z, 1)
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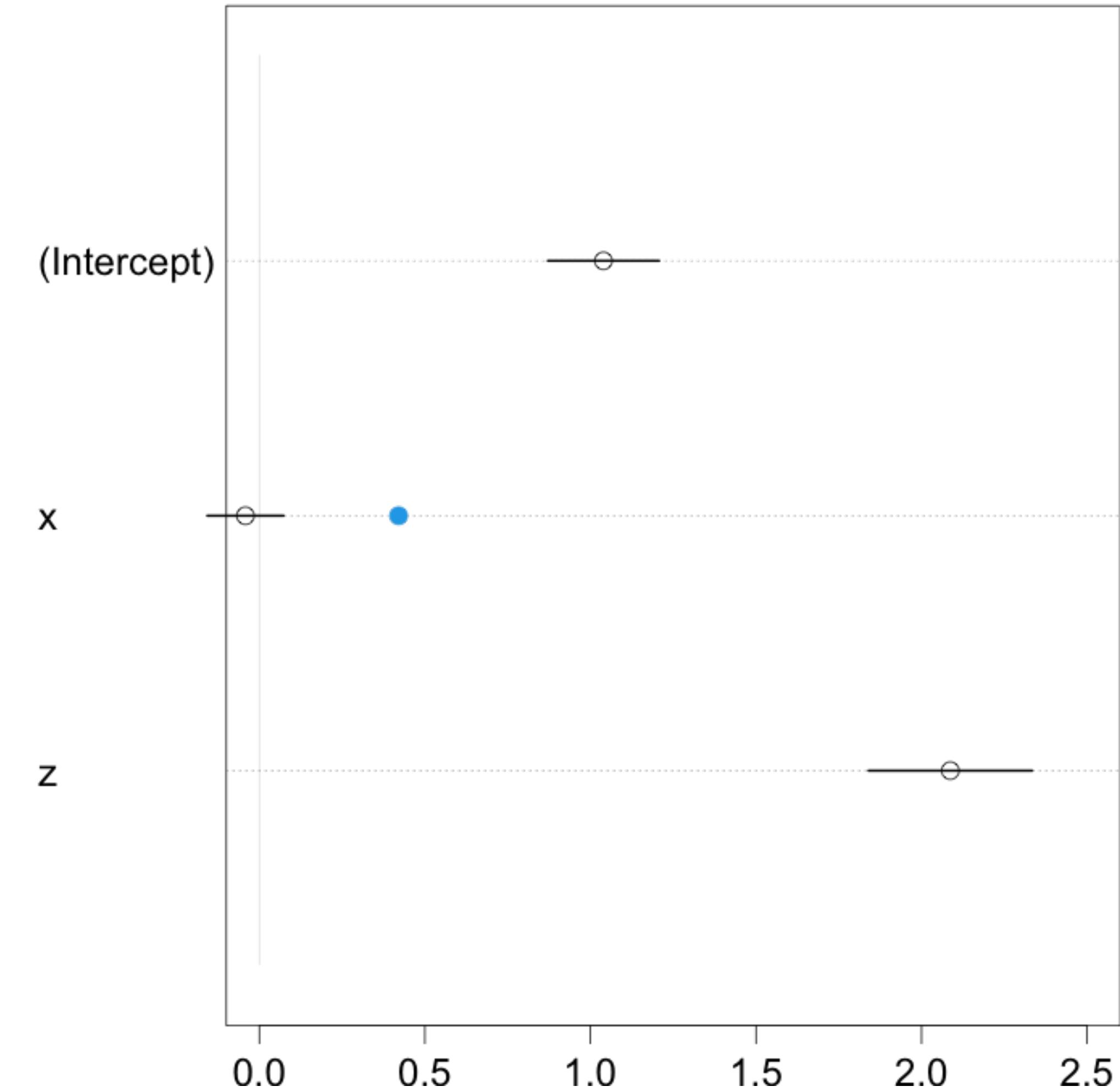
Model without the mediator



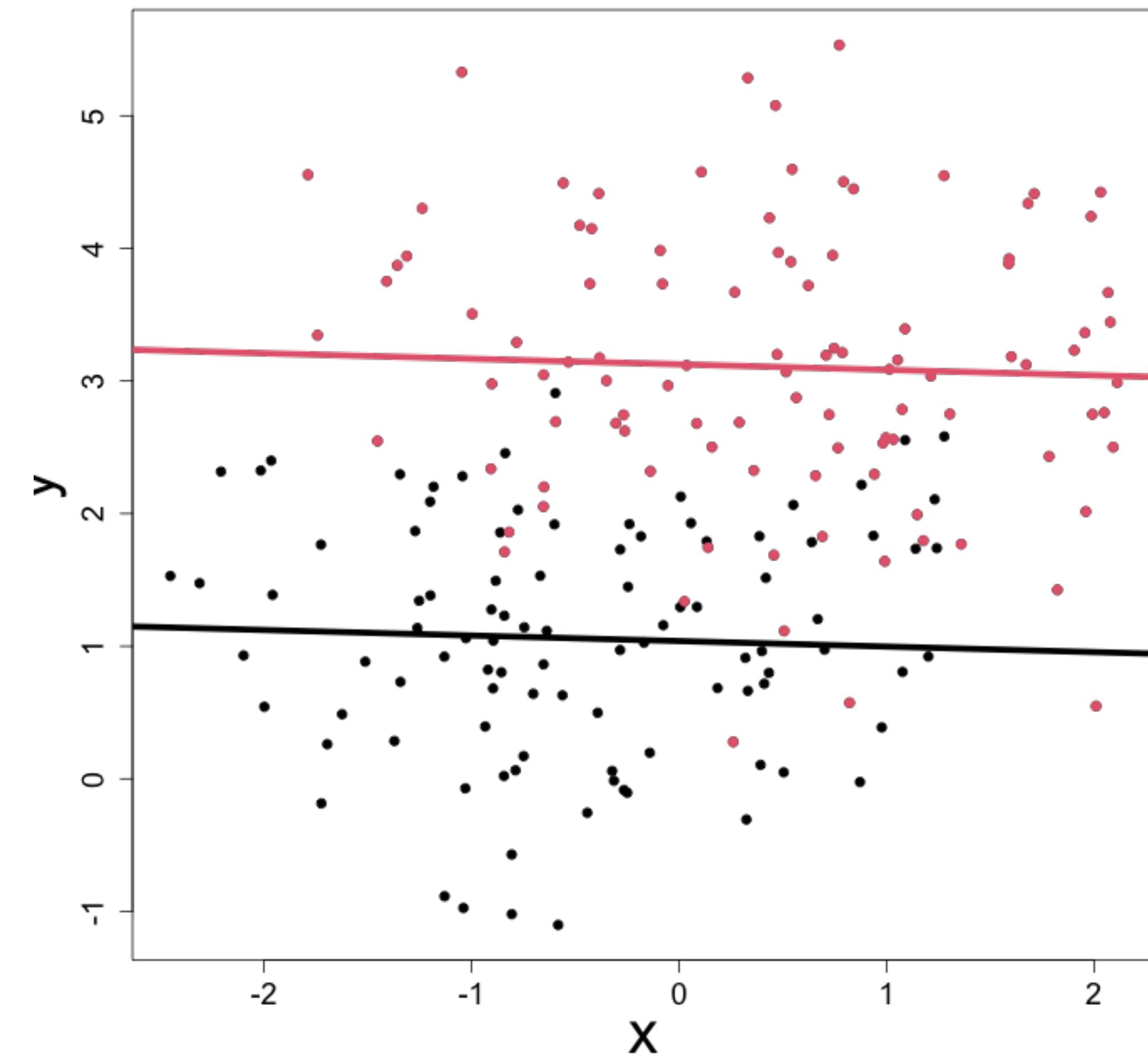
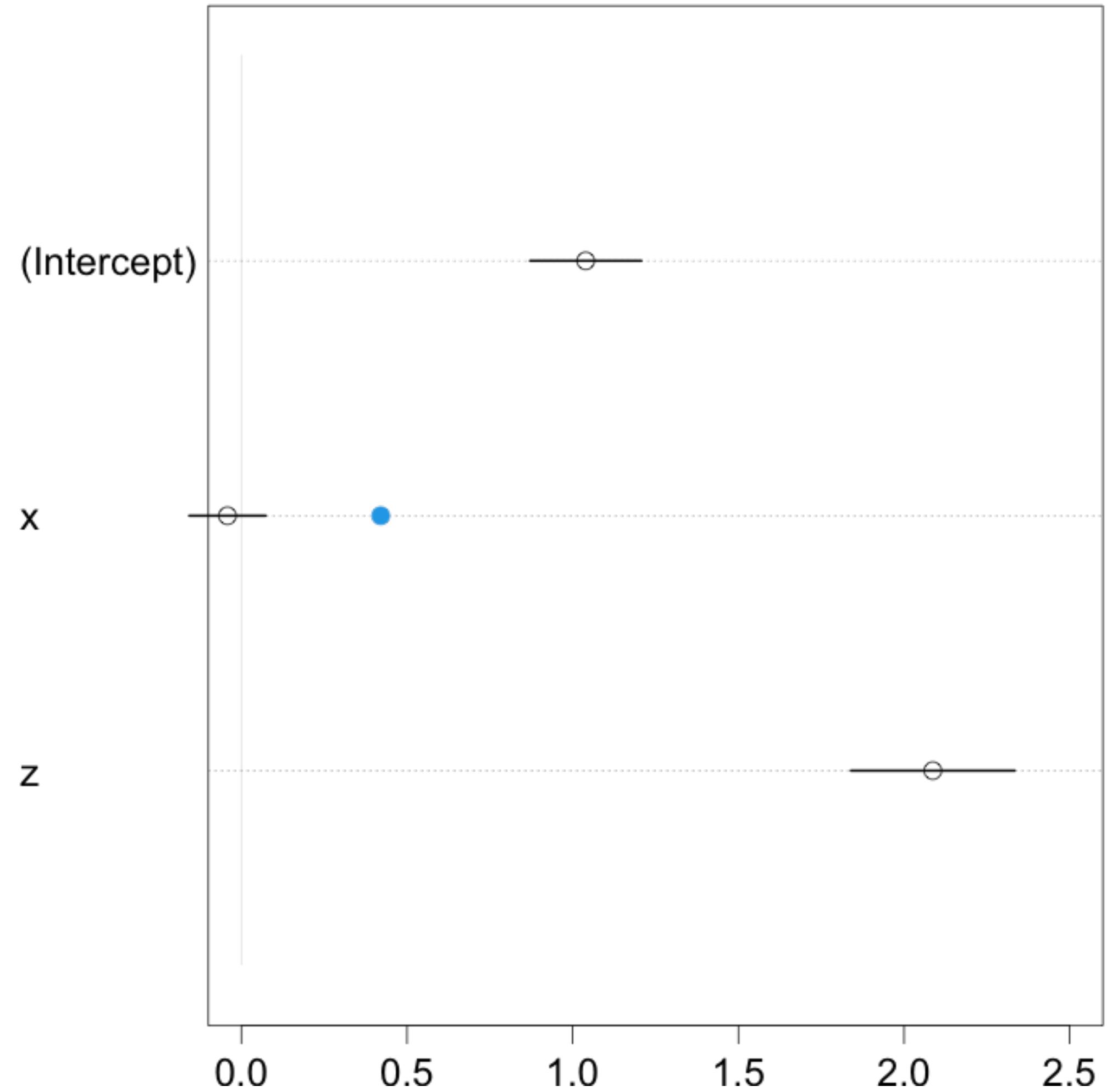
Including the mediador

```
> m2 = lm(y ~ x + z)
> (pm2 = precis(m2))

            mean      sd    5.5% 94.5%
(Intercept) 1.04 0.11  0.87  1.21
x           -0.04 0.07 -0.16  0.07
z            2.09 0.15  1.84  2.33
```

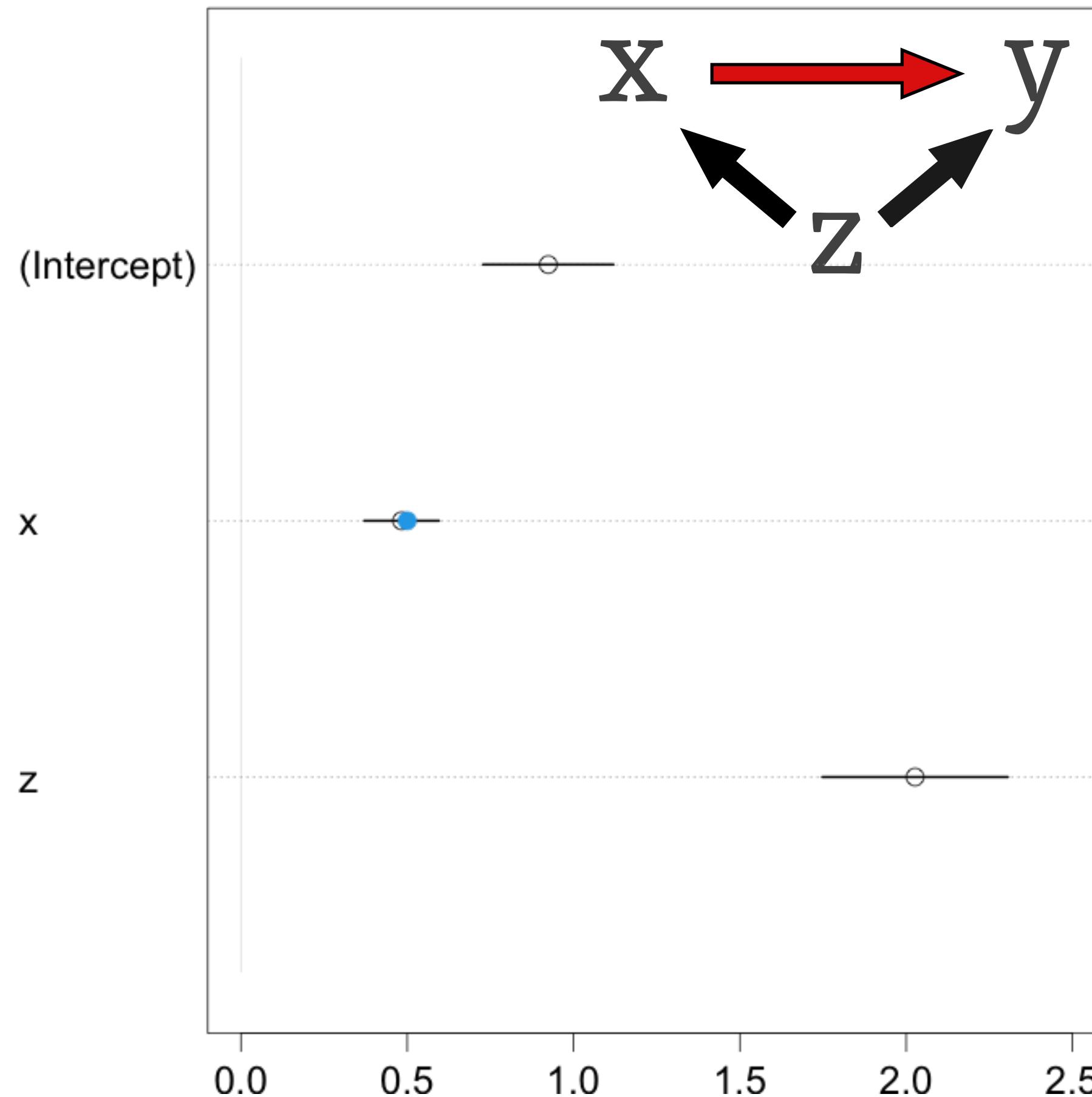


Including the mediador

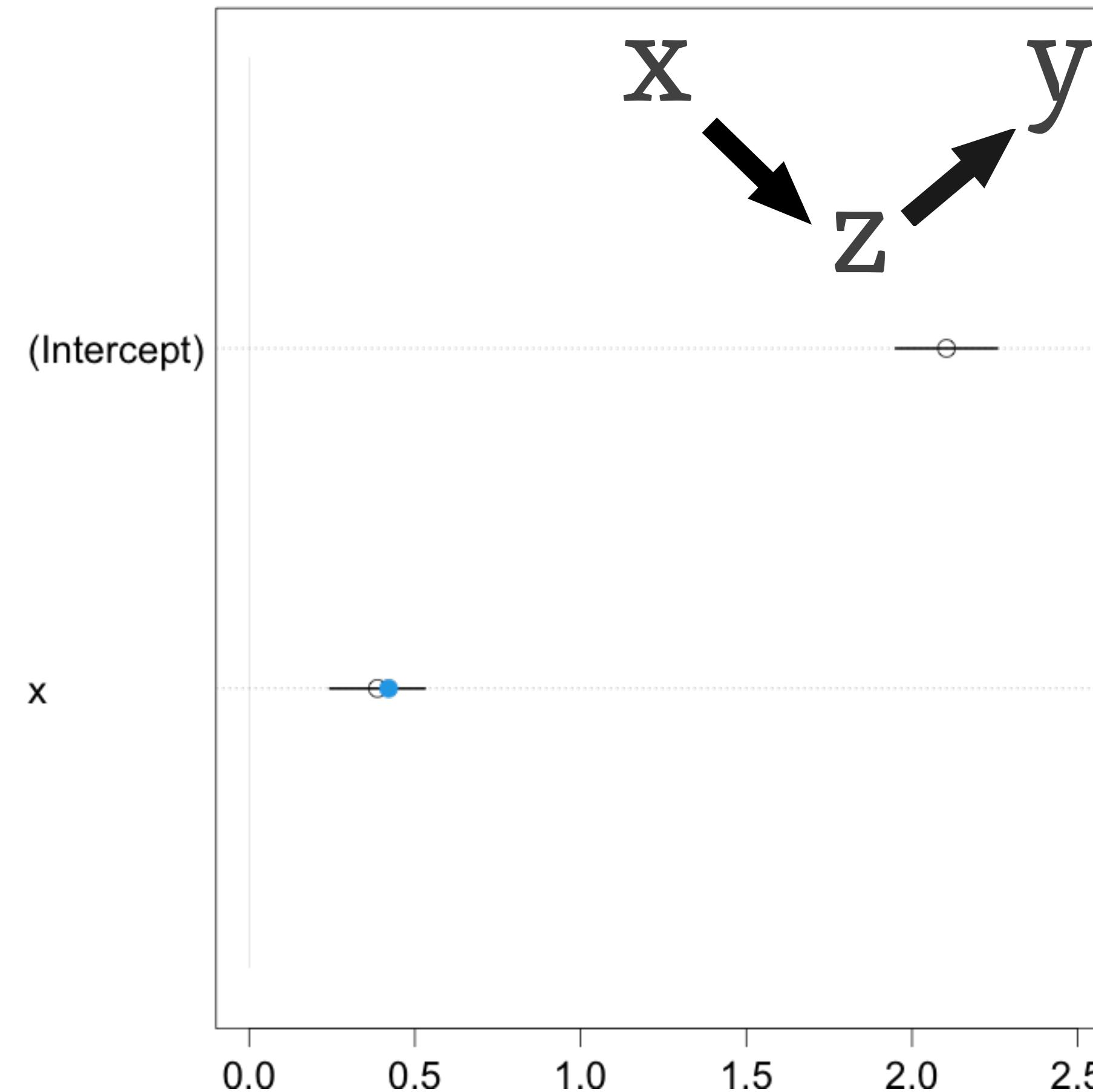


Fork vs. Pipe

Correct estimate with z



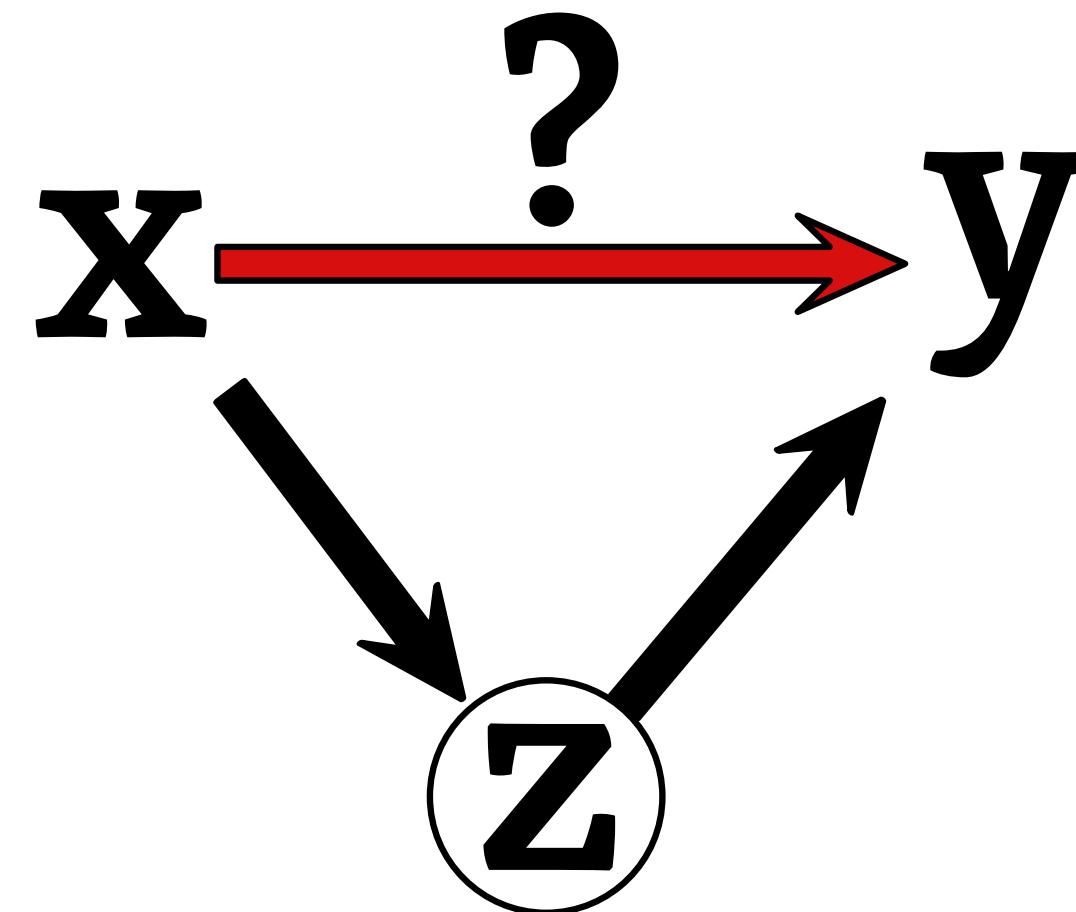
Correct estimate without z



Examples of pipes or mediators

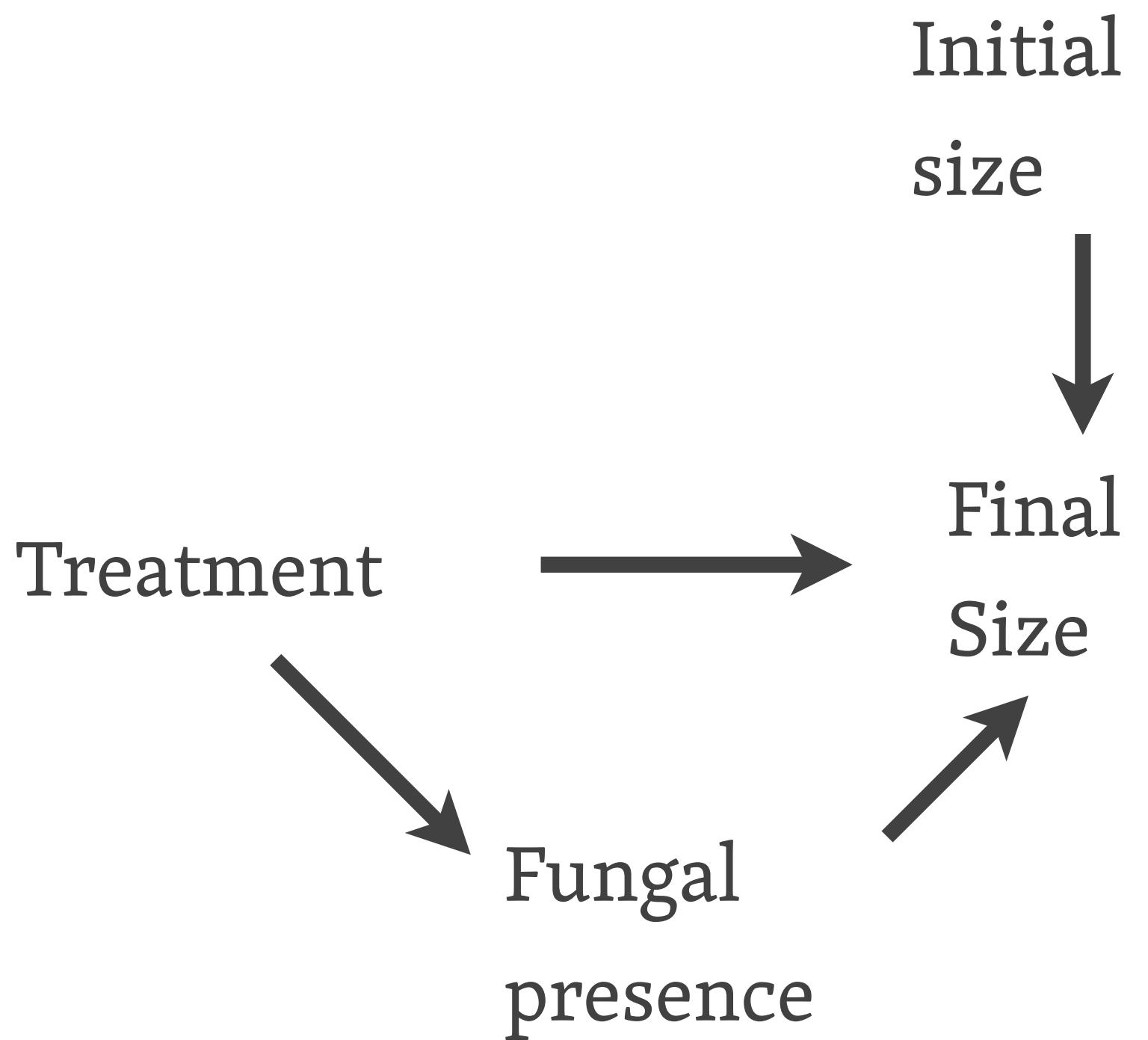
Including a mediator in our models can have catastrophic effects

A common mistake is to include post-treatment variables



Post-treatment variables

For example, in an experiment which attempts to estimate the effect of a treatment on the final size of a plant



Post-treatment variables



How Conditioning on Posttreatment Variables Can Ruin Your Experiment and What to Do about It



Jacob M. Montgomery Washington University in St. Louis

Brendan Nyhan Dartmouth College

Michelle Torres Washington University in St. Louis

Abstract: *In principle, experiments offer a straightforward method for social scientists to accurately estimate causal effects. However, scholars often unwittingly distort treatment effect estimates by conditioning on variables that could be affected by their experimental manipulation. Typical examples include controlling for posttreatment variables in statistical models, eliminating observations based on posttreatment criteria, or subsetting the data based on posttreatment variables. Though these modeling choices are intended to address common problems encountered when conducting experiments, they can bias estimates of causal effects. Moreover, problems associated with conditioning on posttreatment variables remain largely unrecognized in the field, which we show frequently publishes experimental studies using these practices in our discipline's most prestigious journals. We demonstrate the severity of experimental posttreatment bias analytically and document the magnitude of the potential distortions it induces using visualizations and reanalyses of real-world data. We conclude by providing applied researchers with recommendations for best practice.*

Post-treatment variables

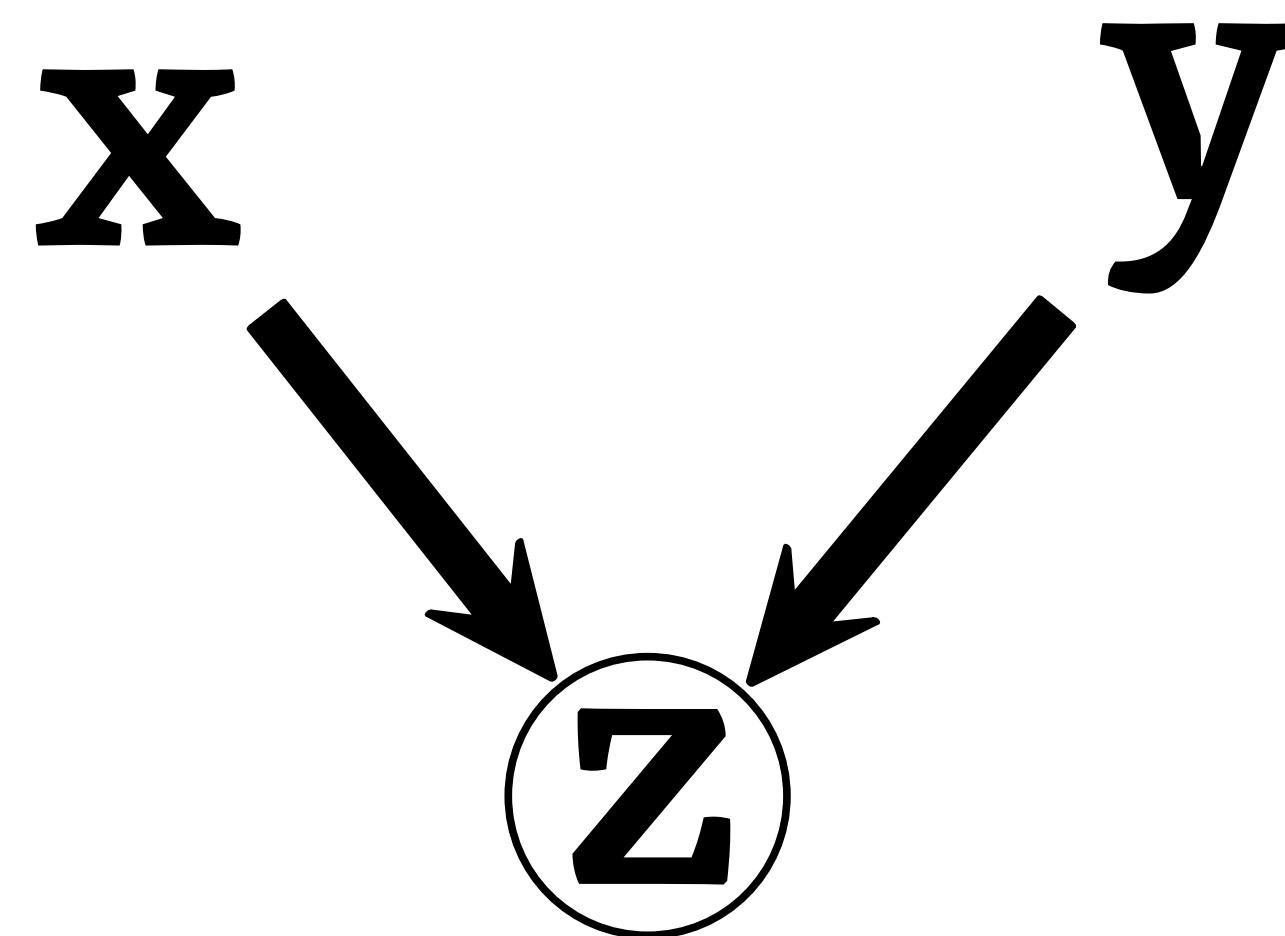
Table 1. Posttreatment Conditioning in Experimental Studies

Category	Prevalence
Engages in posttreatment conditioning	46.7%
Controls for/interacts with a posttreatment variable	21.3%
Drops cases based on posttreatment criteria	14.7%
Both types of posttreatment conditioning present	10.7%
No conditioning on posttreatment variables	52.0%
Insufficient information to code	1.3%

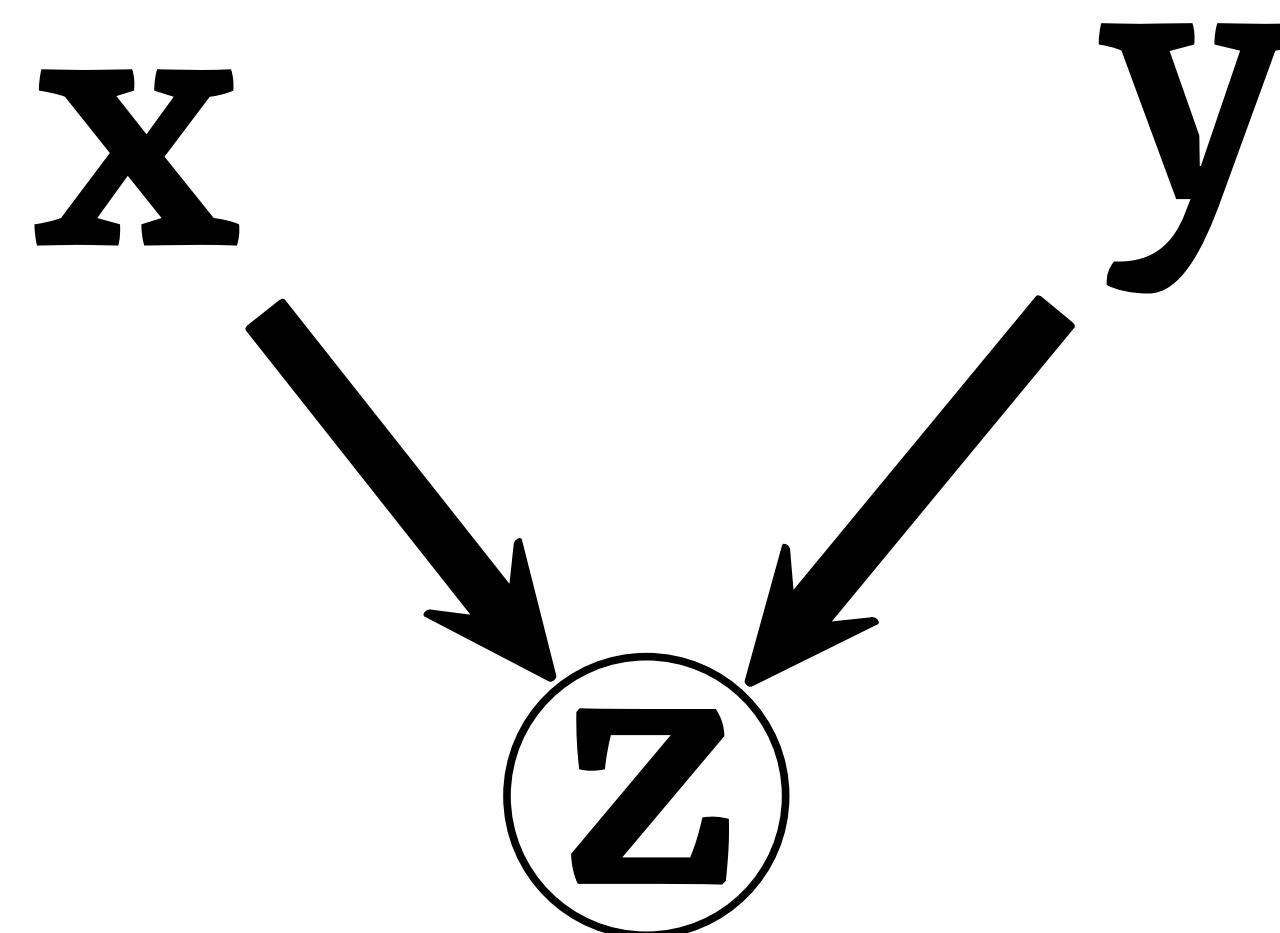
Note: The sample consists of 2012–14 articles in the *American Political Science Review*, the *American Journal of Political Science*, and the *Journal of Politics* including a survey, field, laboratory, or lab-in-the-field experiment (n = 75).

Collider

No effect of x on y, but both affect z



No effect of x on y , but both affect z



Math

$$y \sim \text{Normal}(0, 1)$$

$$x \sim \text{Normal}(0, 1)$$

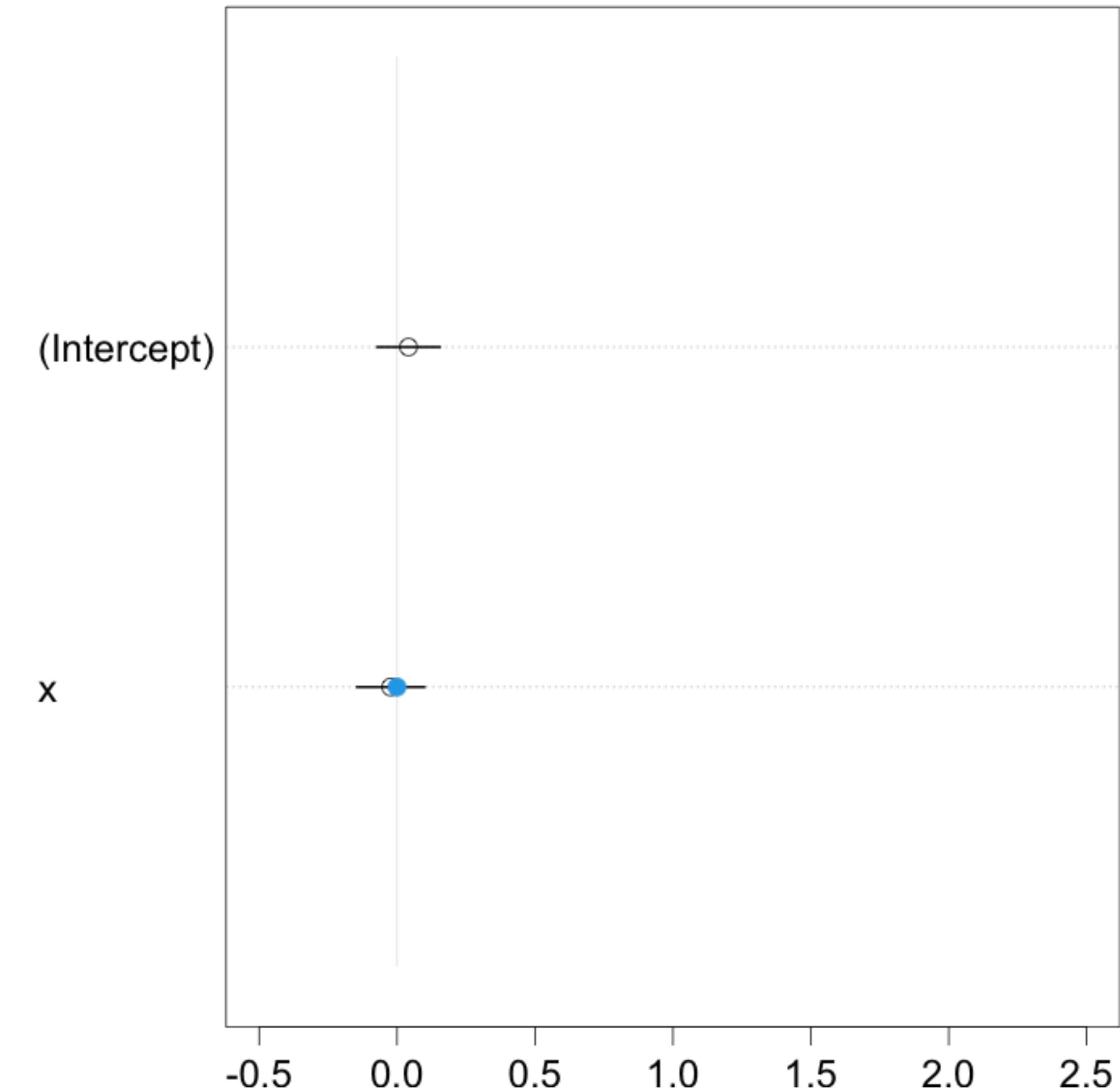
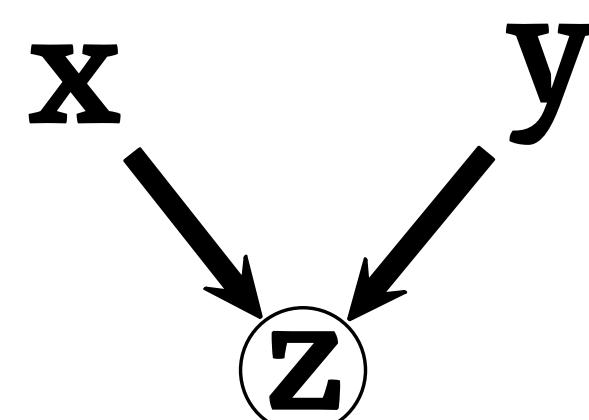
$$z \sim \text{Bernoulli}(\text{logit}^{-1}(2x + 2y - 2))$$

No effect of x on y, but both affect z

```
set.seed(1)
N = 200
x = rnorm(N)
y = rnorm(N)
z = rbinom(N, 1, inv_logit(2*x + 2*y - 2))

m1 = lm(y ~ x)

> (pm1 = precis(m1))
      mean    sd  5.5% 94.5%
(Intercept) 0.04  0.07 -0.07  0.16
x           -0.02  0.08 -0.15  0.10
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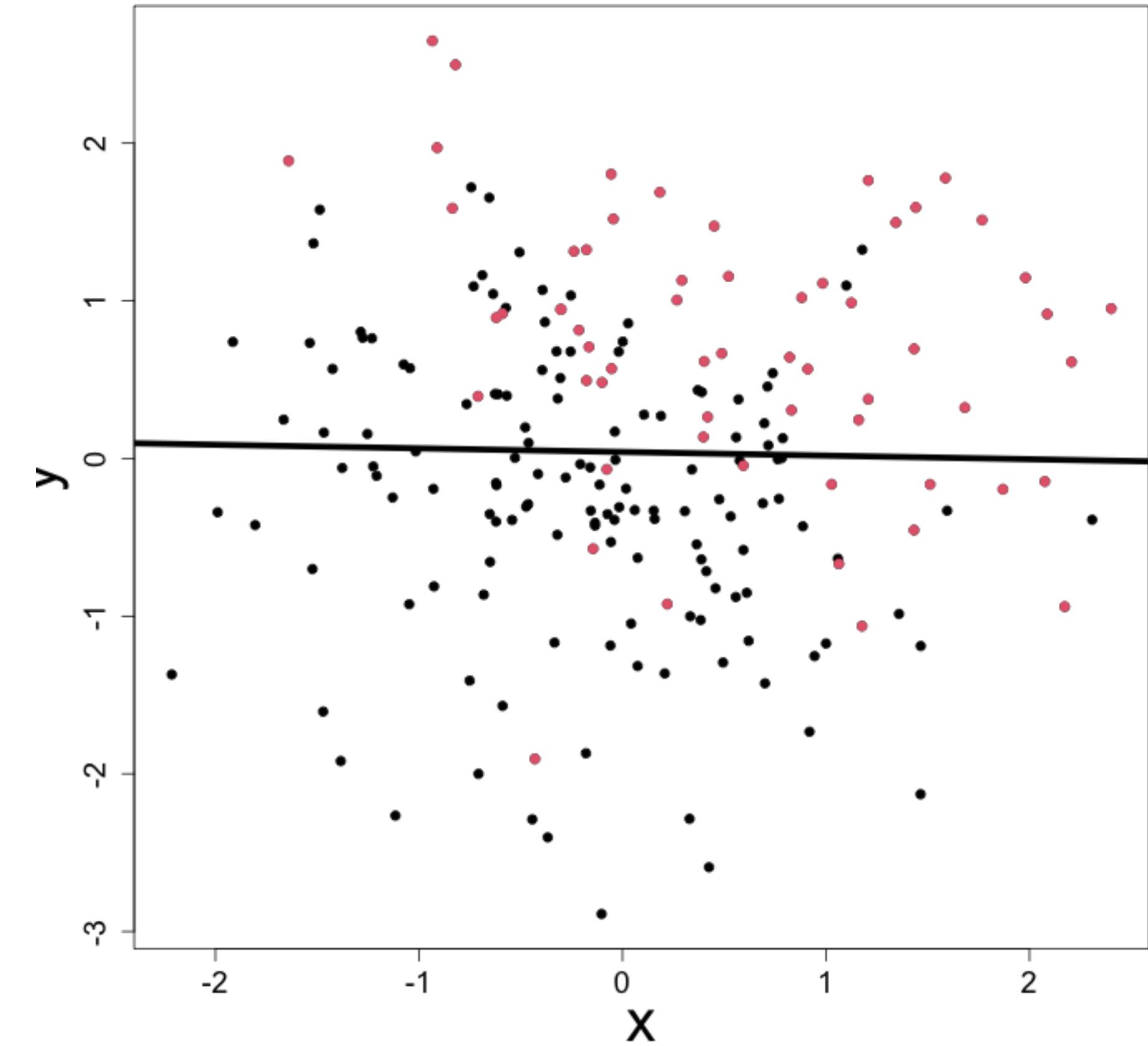
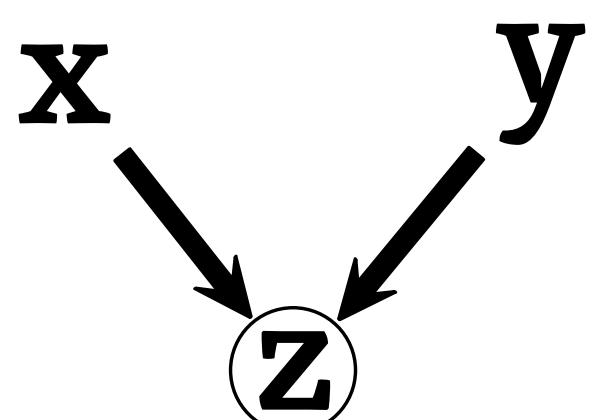


No effect of x on y, but both affect z

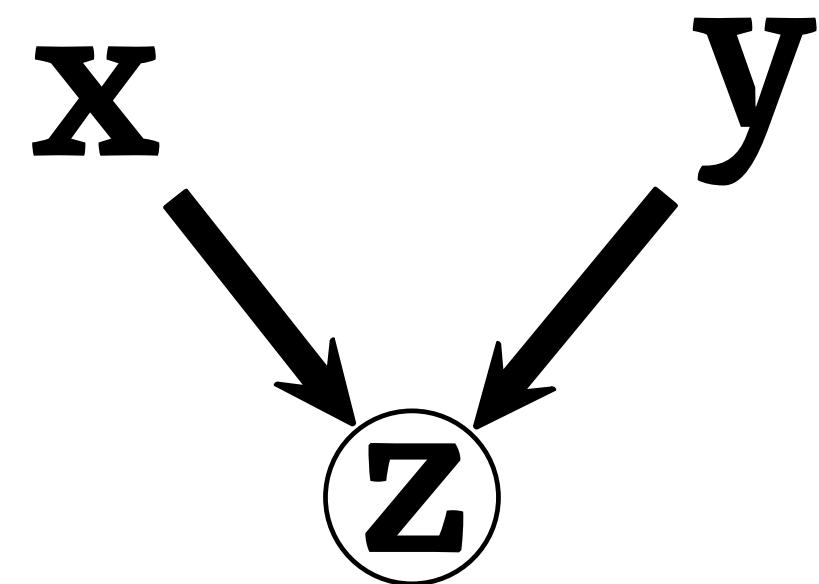
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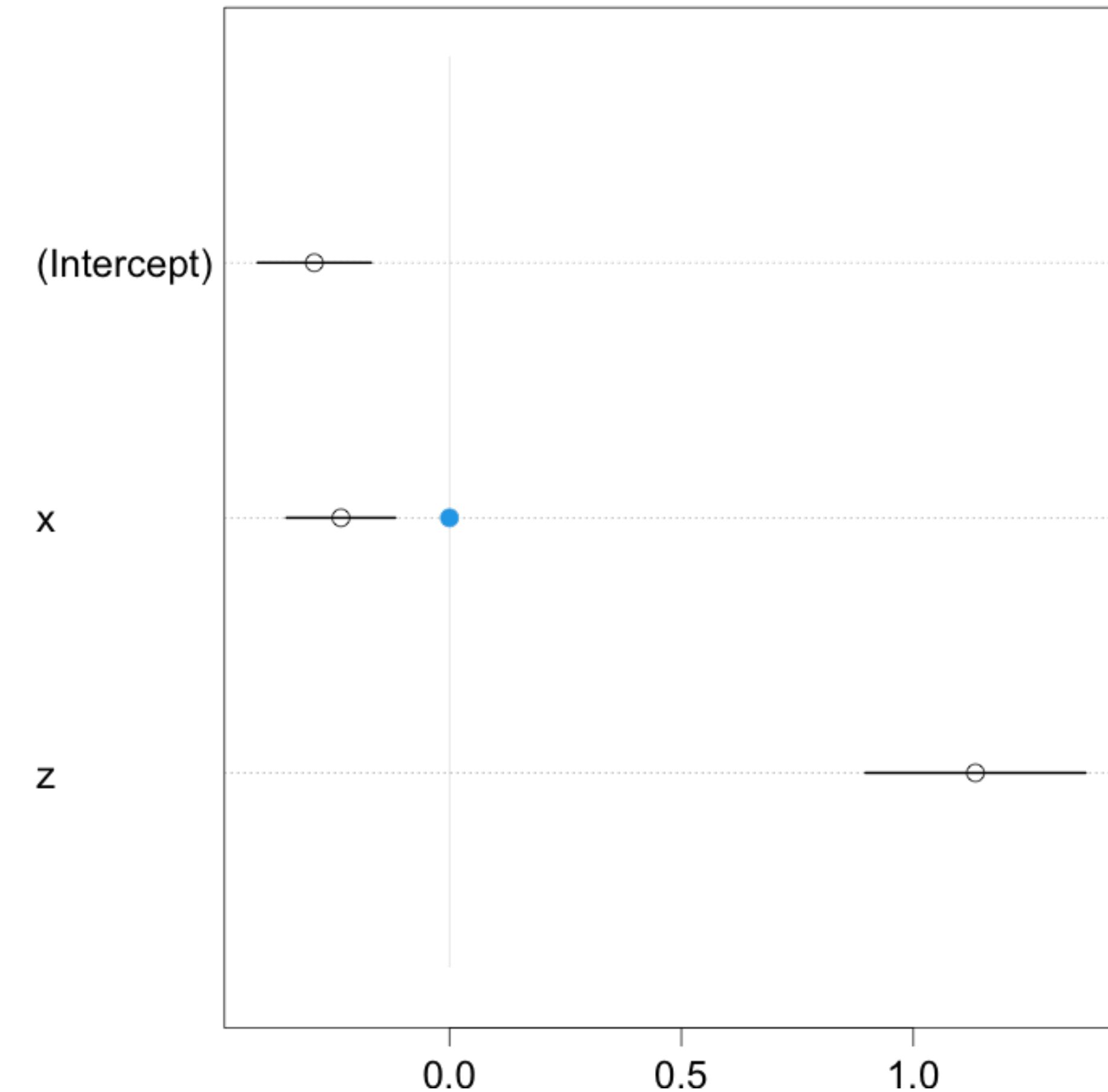


Now, the model with the collider z

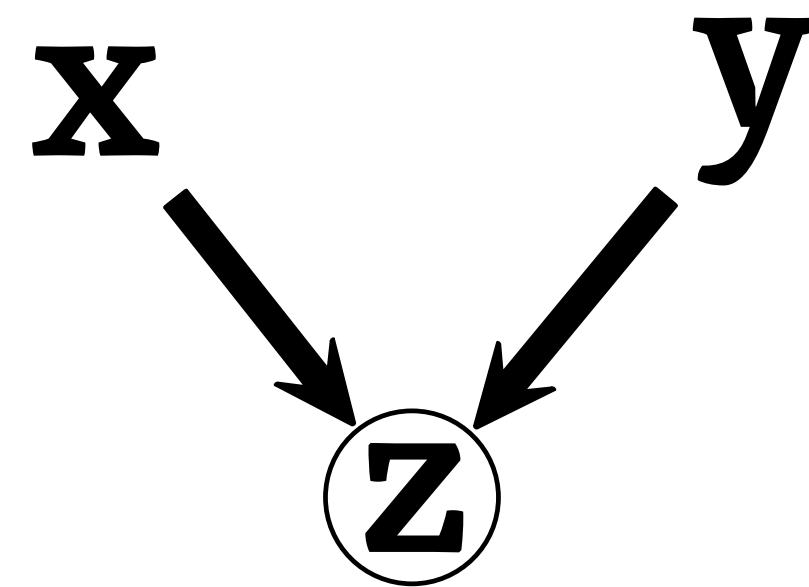


```
> m2 = lm(y ~ x + z)

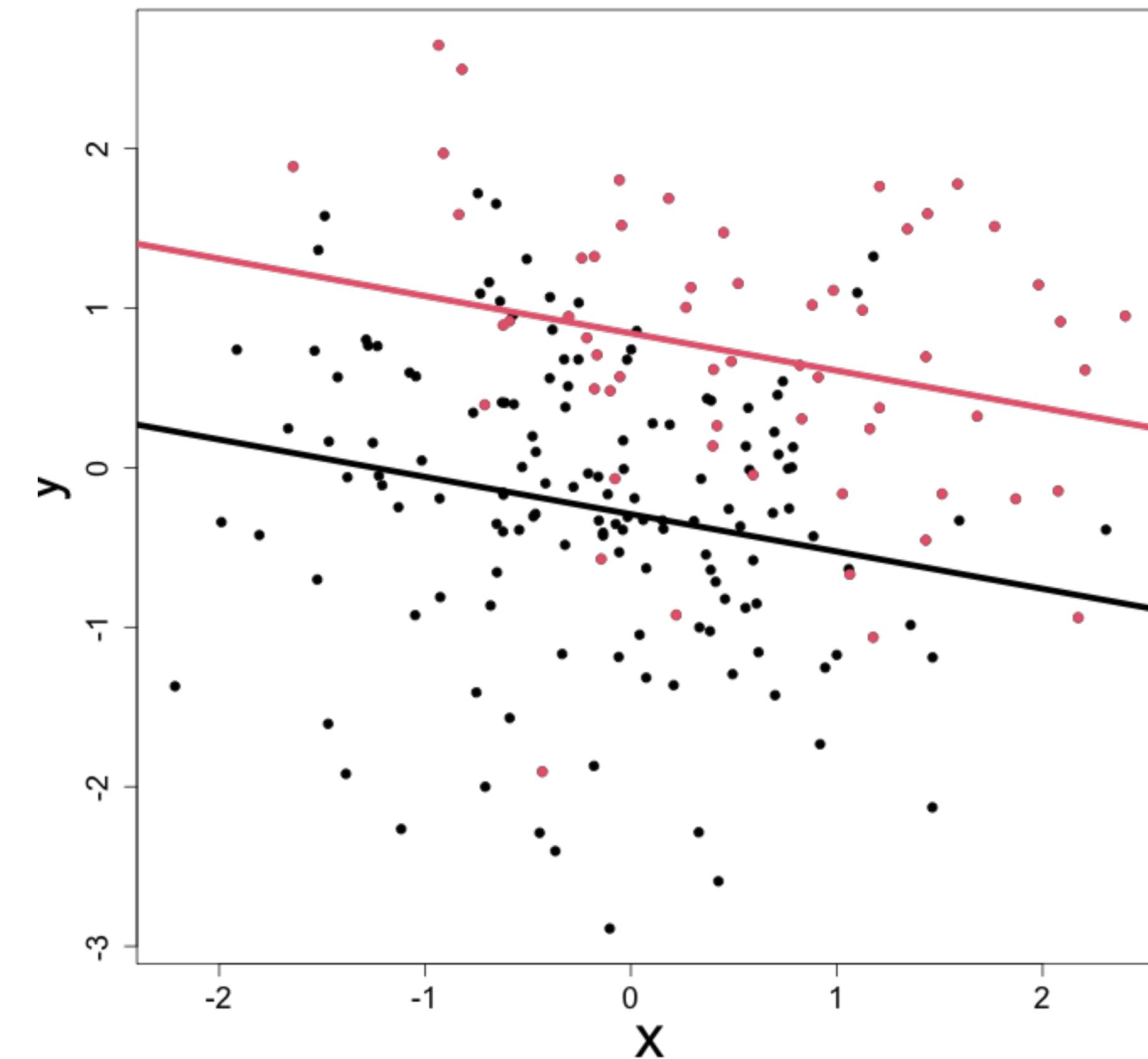
> (pm2 = precis(m2))
      mean     sd   5.5% 94.5%
(Intercept) -0.29  0.08 -0.41 -0.17
x            -0.23  0.07 -0.35 -0.12
z            1.13  0.15  0.90  1.37
```



Now, the model with the collider z



```
> m2 = lm(y ~ x + z)  
  
> (pm2 = precis(m2))  
      mean    sd   5.5% 94.5%  
(Intercept) -0.29  0.08 -0.41 -0.17  
x            -0.23  0.07 -0.35 -0.12  
z            1.13  0.15  0.90  1.37
```



What about the p-value?!?

```
> summary(m1)
```

Call:

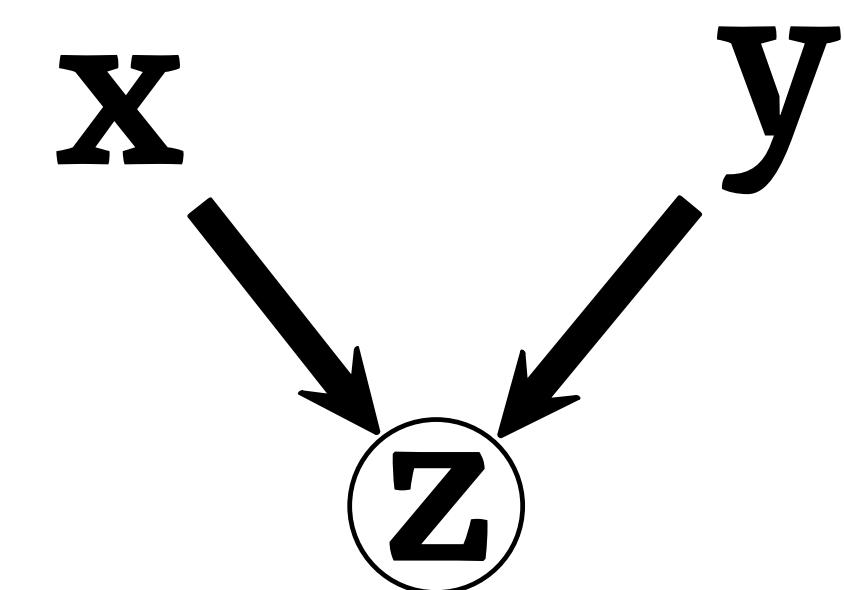
```
lm(formula = y ~ x)
```

Residuals:

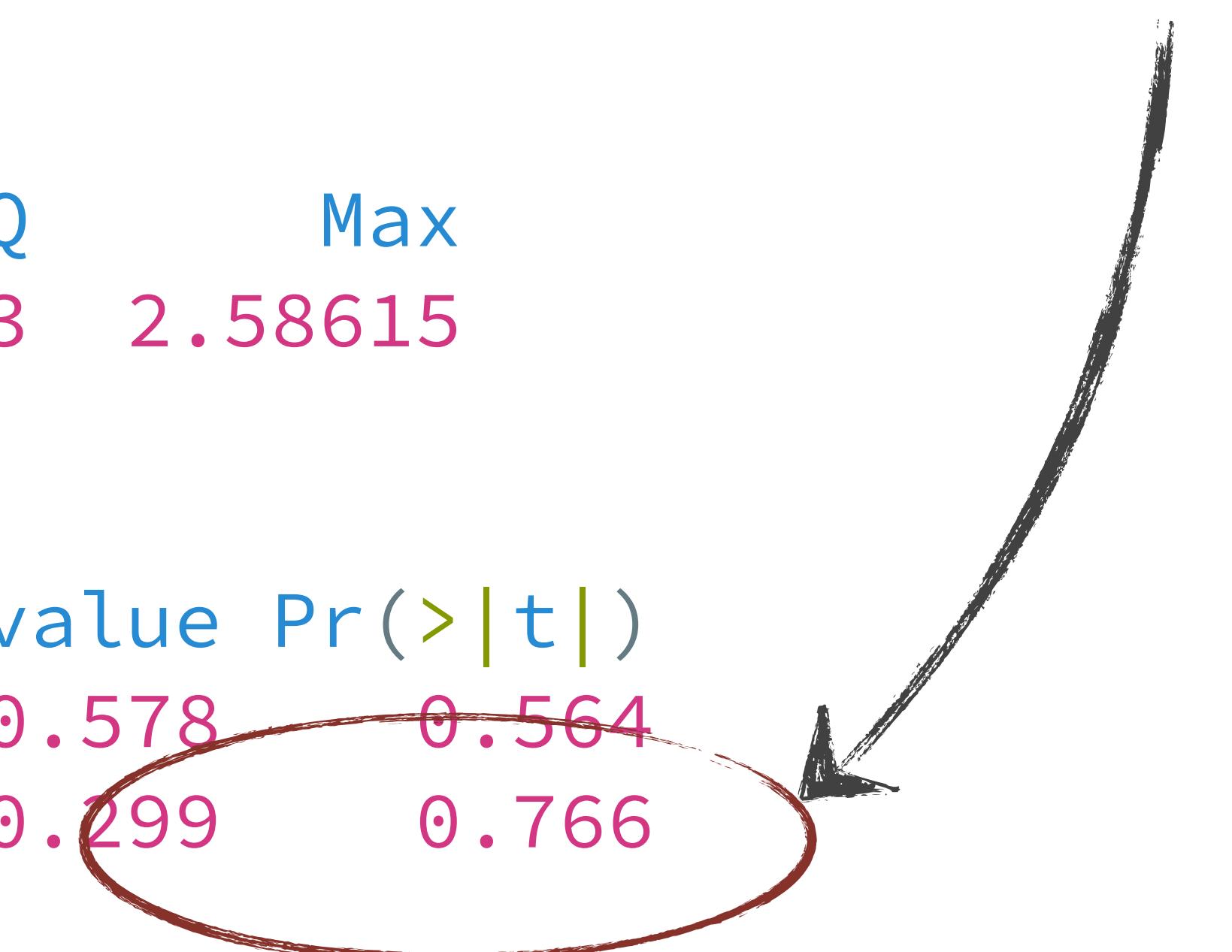
Min	1Q	Median	3Q	Max
-2.93275	-0.54273	-0.02523	0.66833	2.58615

Coefficients:

	Estimate	Std. Error	t value	Pr(> t)
(Intercept)	0.04146	0.07168	0.578	0.564
x	-0.02308	0.07729	-0.299	0.766



In the biased model, both effects are significant!



Maybe model comparison will save us?

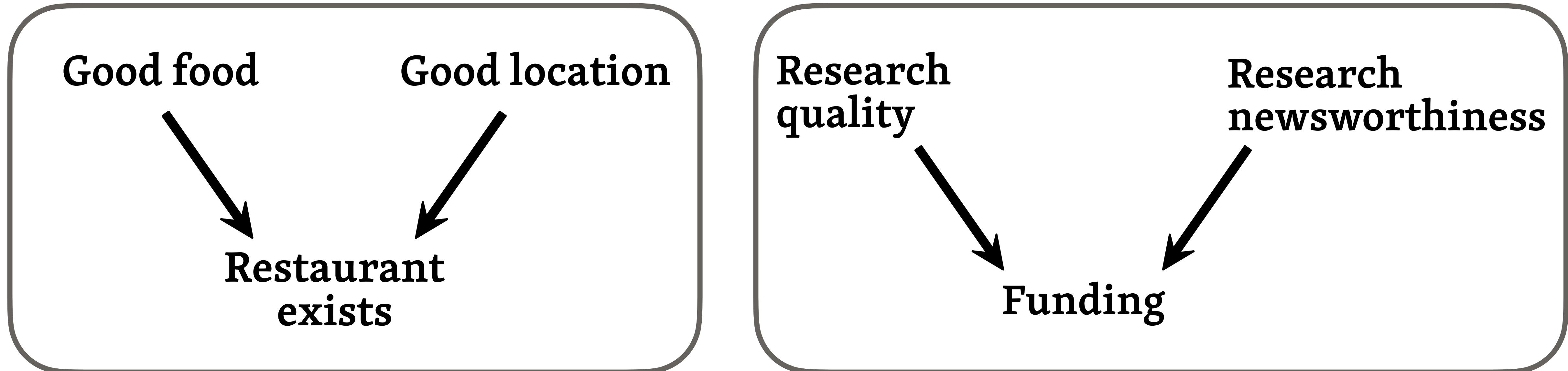
```
> AIC(m1, m2)
   df      AIC
m1  3  576.7421
m2  4  526.9382
```

$$m1 : y \sim x$$

$$m2 : y \sim x + z$$

The biased model has a lower AIC!

Natural colliders happen all the time



What now?!

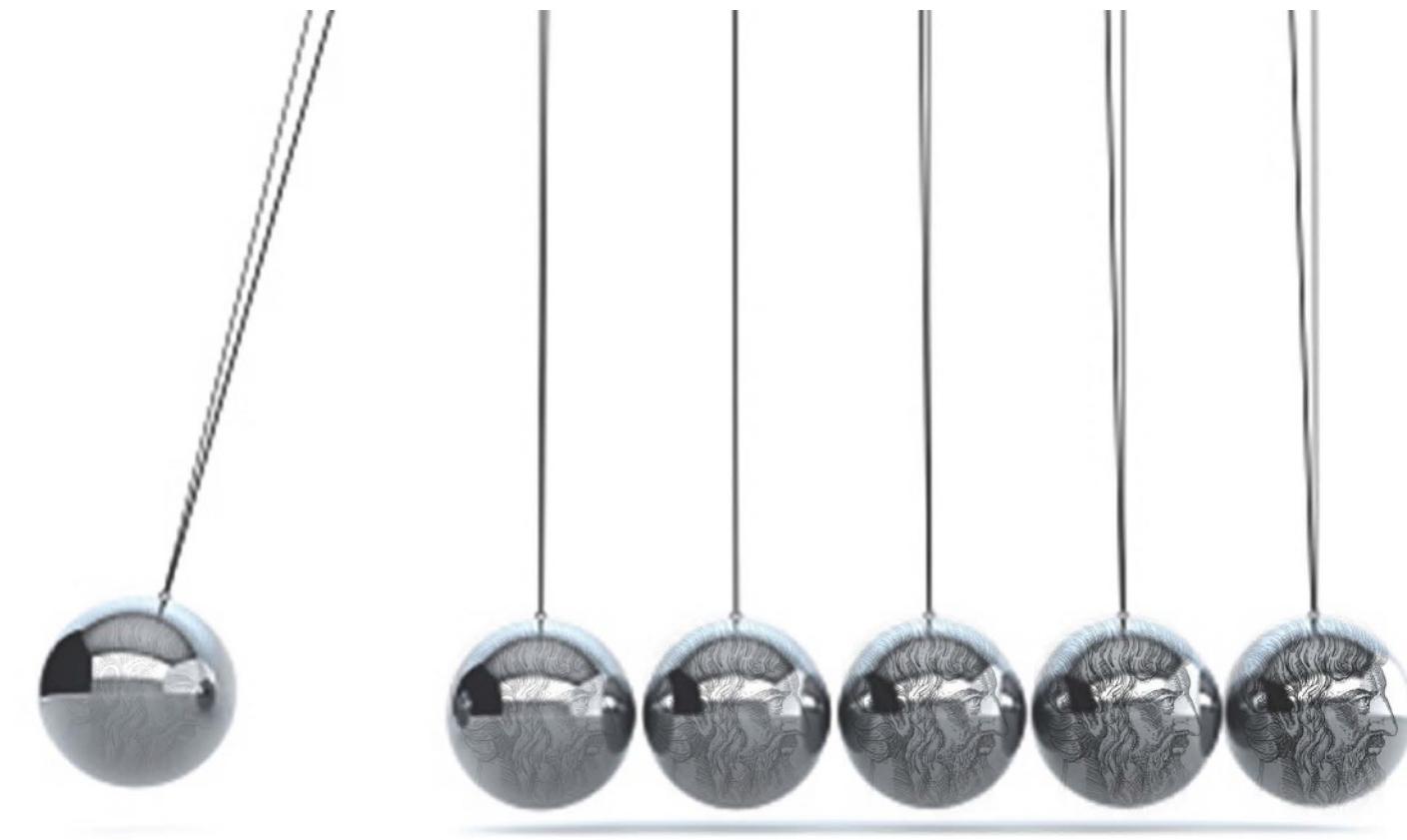
Using DAGs to build models

- If we represent our putative causal relations using DAGs, we have a set of rules that tells us what variables we need to include in the model in order to calculate a particular effect, or if the effect can be estimated at all

- This is a formalism called **do-calculus**
- There are also more complicated methods, like Structural Equation Modeling or **Full-Luxury**

Bayesian Inference:

- Regression, Fire, and Dangerous Things (1/3)



CAUSAL INFERENCE IN STATISTICS

A Primer

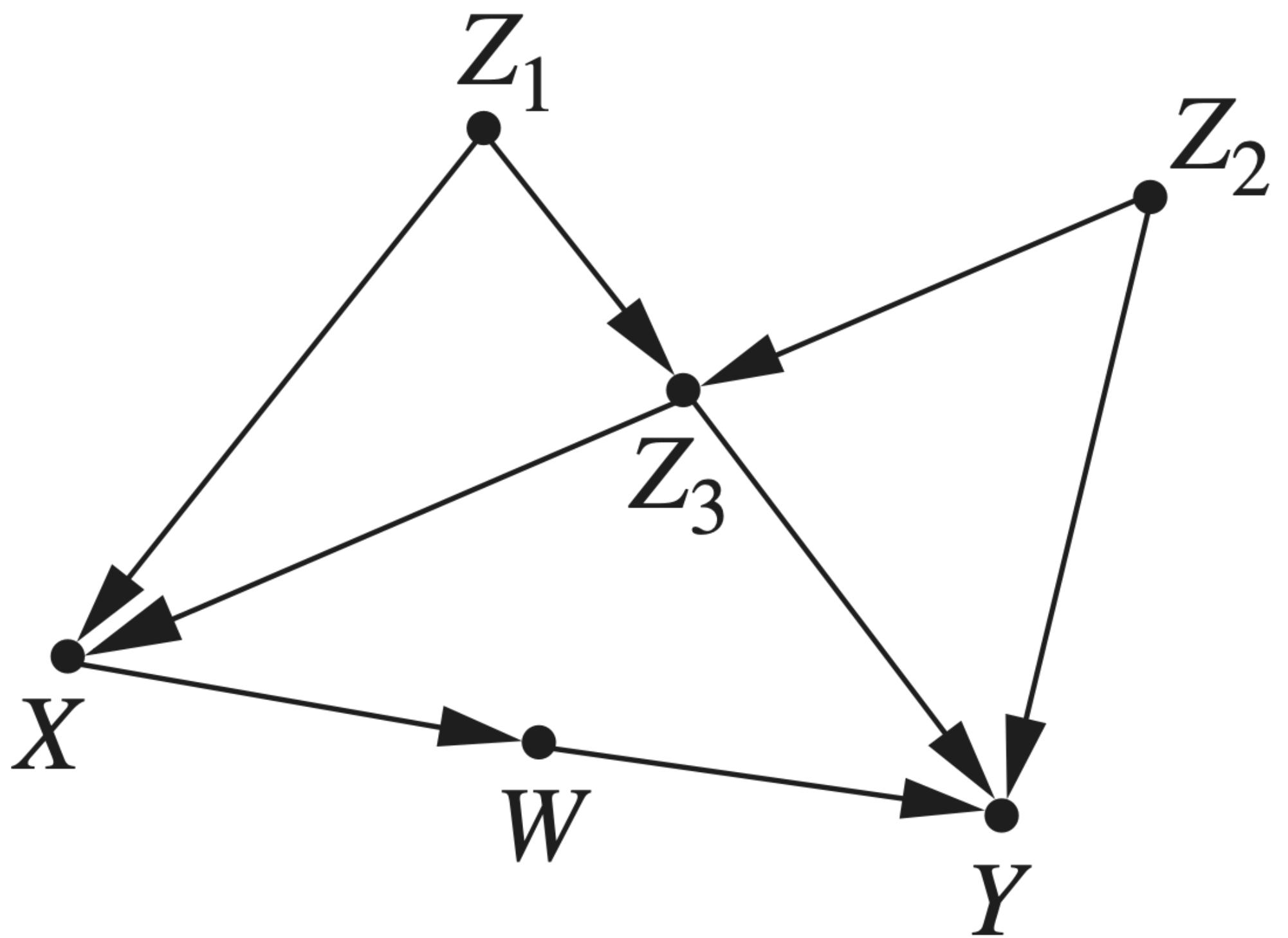
Judea Pearl
Madelyn Glymour
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Open and closed paths

- Paths containing uncontrolled pipes and forks are open
- Paths containing colliders are closed by default, but open if we condition on the collider
- To estimate the true causal effect of x on y , we need all non-causal paths from x to y to be closed in our model

Identify all the open paths from X to Y



Back door criterion

To estimate the causal effect of X on Y , identify a set of control variables such that no descendants of X are in the control set, and all paths between X and Y that contain an arrow into X are blocked.

Not all estimates are causal

The Table 2 Fallacy: Presenting and Interpreting Confounder and Modifier Coefficients FREE

Daniel Westreich , Sander Greenland  Author Notes

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Abstract

It is common to present multiple adjusted effect estimates from a single model in a single table. For example, a table might show odds ratios for one or more exposures and also for several confounders from a single logistic regression. This can lead to mistaken interpretations of these estimates. We use causal diagrams to display the sources of the problems. Presentation of exposure and confounder effect estimates from a single model may lead to several interpretative difficulties, inviting confusion of direct-effect estimates with total-effect estimates for covariates in the model. These effect estimates may also be confounded even though the effect estimate for the main exposure is not confounded. Interpretation of these effect estimates is further complicated by heterogeneity (variation, modification) of the exposure effect measure across covariate levels. We offer suggestions to limit potential misunderstandings when multiple effect estimates are presented, including precise distinction between total and direct effect measures from a single model, and use of multiple models tailored to yield total-effect estimates for covariates.

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Table 1. Characteristics of the Patients at Baseline.*

Characteristic	Ivermectin (N=679)	Placebo (N=679)	Total (N=1358)
Age			
Median (IQR) — yr	49 (39–57)	49 (37–56)	49 (38–57)
Distribution — no. (%)			
≤50 yr	359 (52.9)	372 (54.8)	731 (53.8)
>50 yr	320 (47.1)	307 (45.2)	627 (46.2)
Female sex — no. (%)	383 (56.4)	408 (60.1)	791 (58.2)
Race — no. (%)†			
Mixed race	648 (95.4)	645 (95.0)	1293 (95.2)
White	6 (0.9)	6 (0.9)	12 (0.9)
Black	7 (1.0)	5 (0.7)	12 (0.9)
Other	1 (0.1)	0	1 (0.1)
Unknown	17 (2.5)	23 (3.4)	40 (2.9)
Body-mass index — no. (%)			
<30	347 (51.1)	336 (49.5)	683 (50.3)
≥30	332 (48.9)	343 (50.5)	675 (49.7)
Time since onset of symptoms — no. (%)			
0–3 days	302 (44.5)	295 (43.4)	597 (44.0)
4–7 days	377 (55.5)	384 (56.6)	761 (56.0)
Risk factors — no. (%)			
Chronic cardiac disease	14 (2.1)	10 (1.5)	24 (1.8)
Uncontrolled hypertension	55 (8.1)	59 (8.7)	114 (8.4)
Chronic pulmonary disease	18 (2.7)	23 (3.4)	41 (3.0)
Asthma	54 (8.0)	60 (8.8)	114 (8.4)
Chronic kidney disease	2 (0.3)	5 (0.7)	7 (0.5)
Type 1 diabetes mellitus	3 (0.4)	9 (1.3)	12 (0.9)
Type 2 diabetes mellitus	79 (12)	89 (13)	168 (12)
Autoimmune disease	2 (0.3)	2 (0.3)	4 (0.3)
Any other risk factor or coexisting condition	22 (3.2)	19 (2.8)	41 (3.0)

* Missingness in covariate data was handled with multiple imputation by chained equations.¹⁶ IQR denotes interquartile range.

† Race was reported by the patient.

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Independent variable	General Linear Model				Ordinary Least Squares	
	df	MS	F	p	β^a	SE
Age	1	44.7	1.8	.175	-.04	.024
Gender (male)	1	294.7	12.1	.001	.10	.391
Education	1	35.2	1.4	.229	.04	.052
Financial strain	1	687.9	28.3	.000	.14	.206
Volunteer work	1	95.9	3.9	.047	.05	.409
Social support	1	95.6	3.9	.048	.05	.021
Religious participation	1	264.4	10.9	.001	-.09	.168
Cognitive deficit	1	202.1	8.3	.004	.08	.074
Stressful life events	1	591.3	24.3	.000	-.13	.082
Health status	1	1145.1	47.1	.000	-.21	.103
Daily activity limitations	1	1508.2	62.1	.000	-.24	.045
Vision	3	66.5	2.74	.021	-.11	.175
Hearing	3	2.2	1.0	.965	-.04	.169
Vision \times Hearing	9	12.1	0.5	.876	.01	.160
Corrected model	26	577.9	23.8	.000		
R^2 (adjusted)					.376	

^aStandardized regression coefficients.

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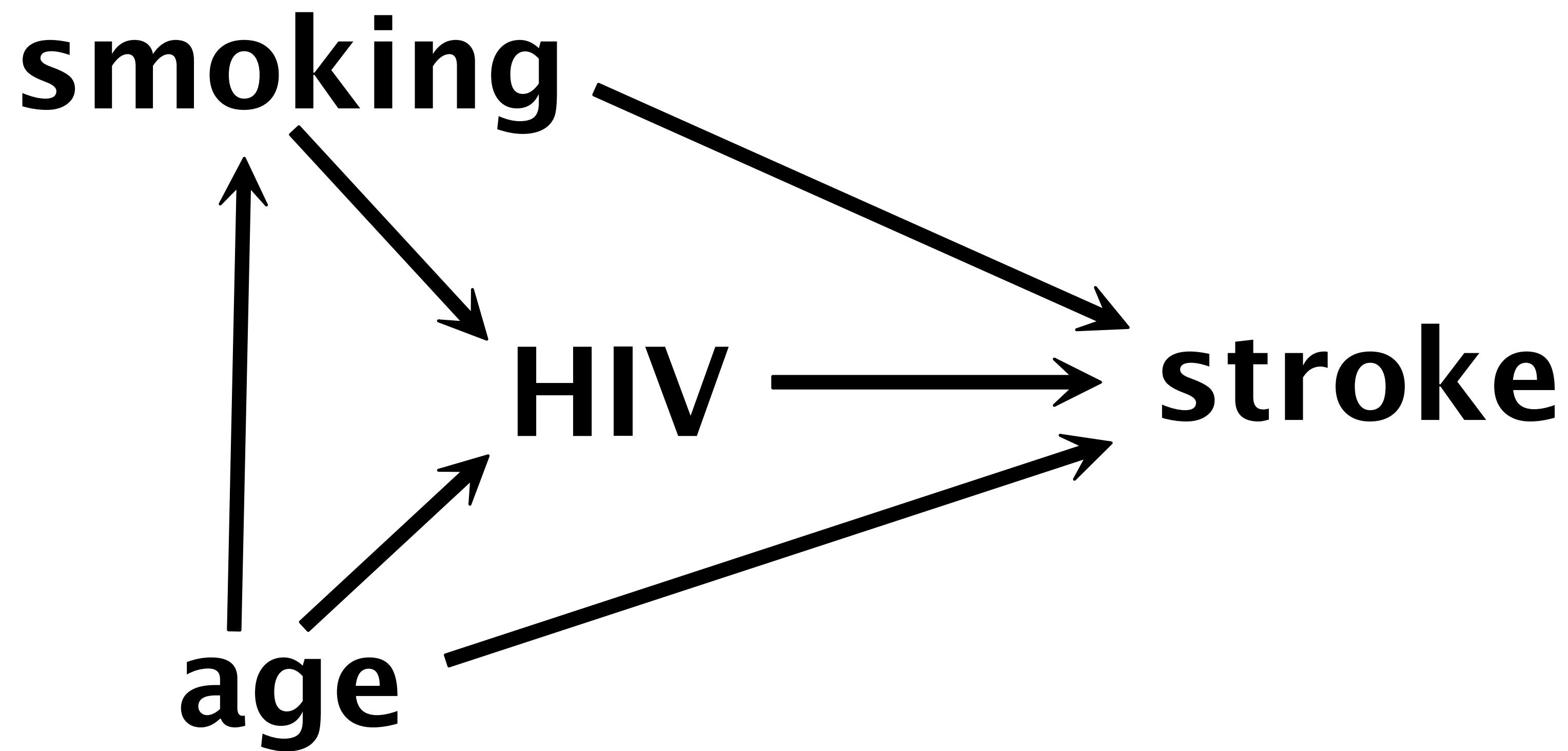
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HIV and Stroke



Good and bad controls

Good controls

- Block non-causal paths
- Improve precision
- Allow inference of causal effects

Bad controls

- Block causal paths (blocking pipes)
- Open non-causal paths (opening colliders)
- Reduce precision
- Prevents causal inference