



```
course = "Estimating the credibility of past research"
```

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lesson_iteration = 10
lesson_title = "Explanations, causes, and claims"
```

```
auth = "Ian Hussey & Malte Elson"
dept = "Psychology of Digitalisation"
```

It's not just you

Causal inference

What is psychology's goal?

aut = "Ian Hussey";

dept = "Psychology of Digitalisation || Digitalisation of Psychology"

Causal inference

What is psychology's goal?

- Explain
- Predict
- Intervene
 - Via manipulable causes

Explanandum vs. explanans

Scientific explanations require a separation of:

- The thing to be explained (explanandum)
- The thing that explains (explanans)

Explanandum vs. explanans

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- The thing that explains (explanans)



Rocket's acceleration

Chemical reaction in engine

Explanandum vs. explanans

Psychologists often confuse them!

- The thing to be explained (explanandum)
- The thing that explains (explanans)

Which one is attention?

Explanandum vs. explanans

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Which one is attention?

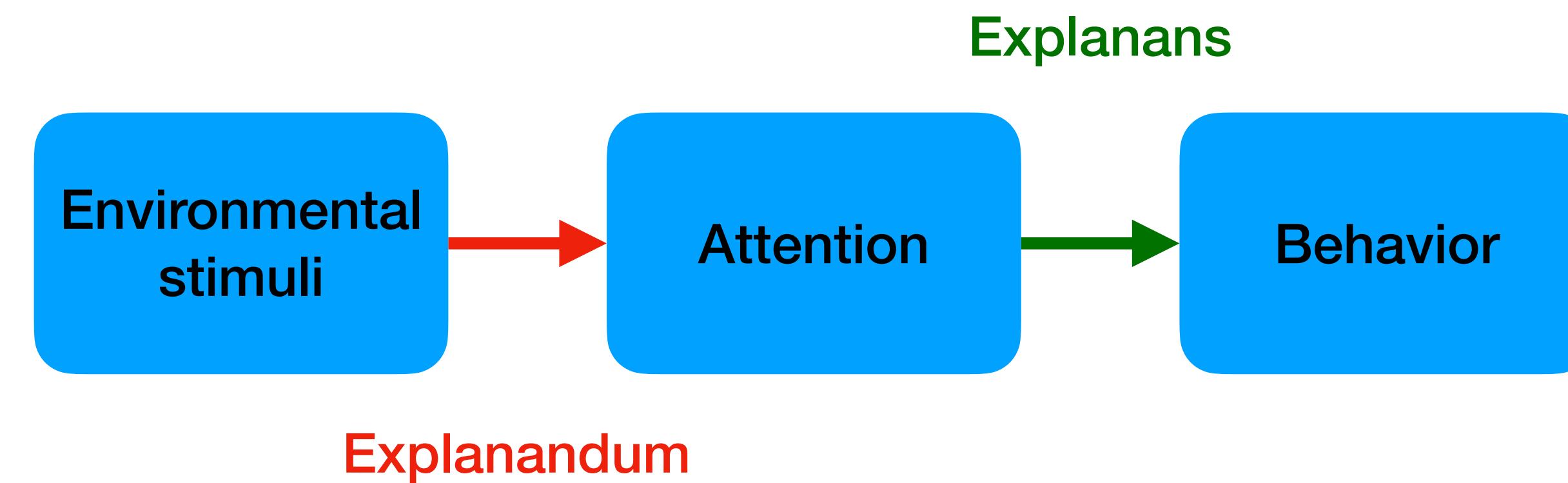


Explanandum vs. explanans

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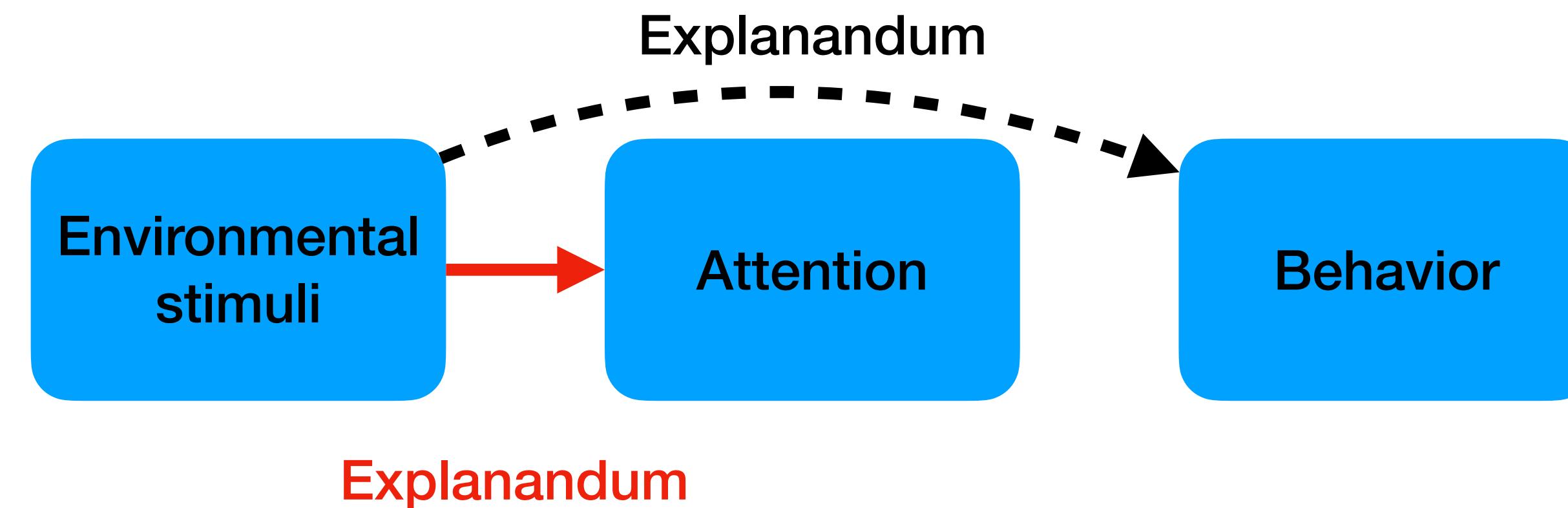
Hommel et al. (2019) "No one knows what attention is"

Explanandum vs. explanans

Psychologists often confuse them!

- The thing to be explained (explanandum)
- The thing that explains (explanans)

Which one is attention?



Wu (2023) "We know what attention is!"

- Explanandum only!

We establish causality by:

- Randomisation
 - Experiments/RCTs
 - What would have happened if you didn't intervene
- Causal inference from non-experimental studies
 - With lots of assumptions

We establish causality by:

- Randomisation
 - Experiments/RCTs
 - What would have happened if you didn't intervene
- Causal inference from non-experimental studies
 - With lots of assumptions



Causal inference

We are often *ambiguous* about causality

We train psychologists to be *vague*

“Correlation is not causation”



aut = "Ian Hussey";



ogy of Digitalisation || Digitalisation of Psychology"

Causal language

Poll

Instructions

Go to

www.menti.com

Enter the code

6948 3951



Or use QR code

Simon Says

A game for kids!



Causality



A game for academics!

If you don't use the magic word,
you don't have to provide evidence for it

drives

underpins

guides

moderates

improves

determines

impacts

~~‘causes’~~

mediates

shapes

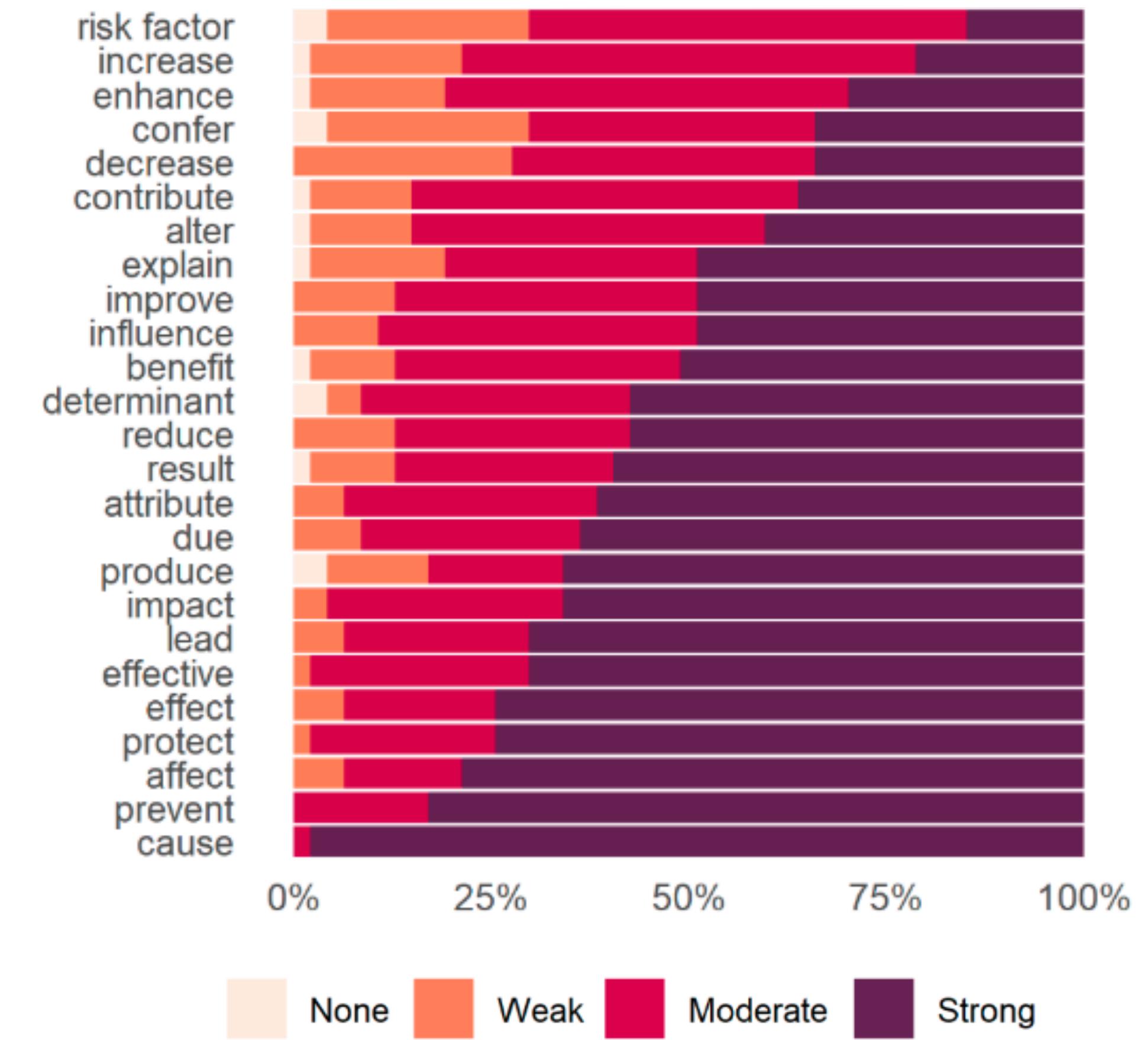
triggers

prevents

Readers' causal inferences

Even if you don't use 'cause',
readers often infer that's what you mean.

(Haber et al., 2021)



Case study 1

Explanans/explanandum & causal inference

Is it a causal claim?

Abstract

sleep → brain

The question of how much sleep is best for the brain attracts scientific and public interest, and there
is concern that insufficient sleep leads to poorer brain health. However, it is unknown how much
sleep is sufficient and how much is too much. We analyzed 51,295 brain magnetic resonance
images from 47,039 participants, and calculated the self-reported sleep duration associated with the

Case study 1

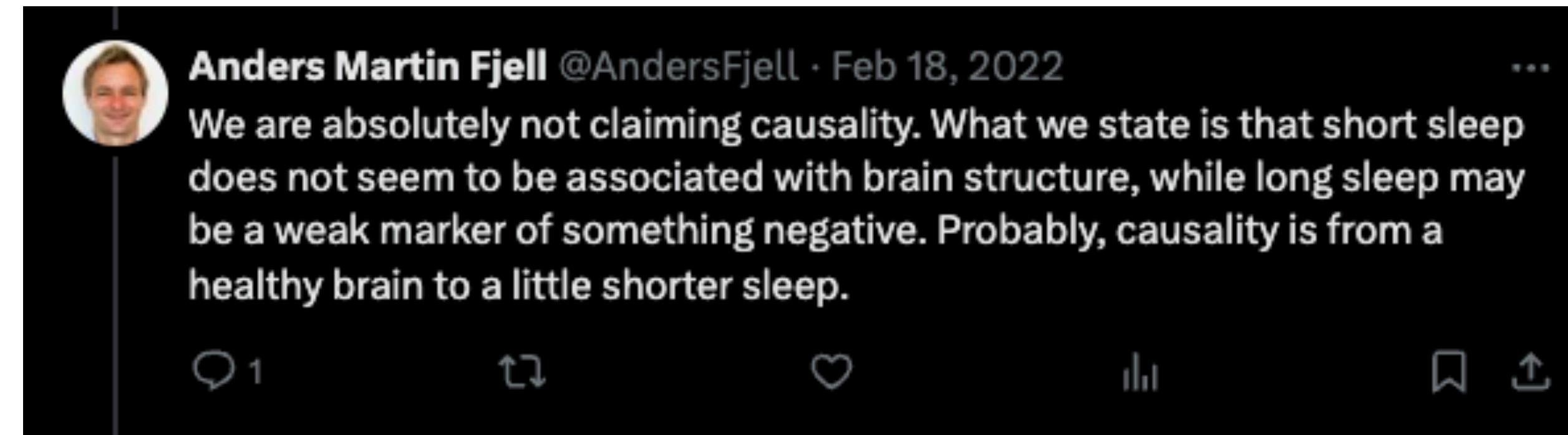
Explanans/explanandum & causal inference

Is it a causal claim?

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The question of how much sleep is best for the brain attracts scientific and public interest, and there is concern that insufficient sleep leads to poorer brain health. However, it is unknown how much sleep is sufficient and how much is too much. We analyzed 51,295 brain magnetic resonance images from 47,039 participants, and calculated the self-reported sleep duration associated with the



Anders Martin Fjell @AndersFjell · Feb 18, 2022

We are absolutely not claiming causality. What we state is that short sleep does not seem to be associated with brain structure, while long sleep may be a weak marker of something negative. Probably, causality is from a healthy brain to a little shorter sleep.

1 11 11 11 11

Case study 2

Explanans/explanandum & causal inference

Leveraging neuroscience for climate change research

Received: 27 February 2023

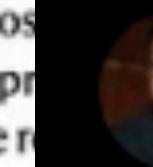
Accepted: 20 September 2023

Published online: 13 November 2023

 Check for updates

Kimberly C. Doell   , Marc G. Berman   , Gregory N. Bratman⁵, Brian Knutson   , Simone Kühn⁷, Claus Lamm   , Sabine Pahl   , Nik Sawe   , Jay J. Van Bavel   , Mathew P. White    & Tobias Brosch   

Anthropogenic climate change poses a threat to our planet and its inhabitants. Here, we argue that neuroscience can contribute to the fight against climate change and propose a research agenda to organize and prioritize neuroscience research. We show how neuroscience can be used to: (1) investigate the impact of climate change on the human brain; (2) identify the neural substrates of environmental decisions and outcomes; and (3) create neuroscience-based communication and intervention strategies that aim to reduce climate change denial. This paper is also a call to action for neuroscientists to tackle the existential environmental threat posed by climate change.



Jay Van Bavel, PhD 
@jayvanbavel

Our new paper outlines how neuroscience can be used to:

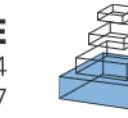
- (1) study the impact of climate change on the brain
- (2) identify ways to adapt
- (3) study the neural substrates of environmental decisions & outcomes
- (4) create better communication & interventions

Title: brain → climate change behavior

Content: climate change (behaviors) → brain

Case study 3

Explanans/explanandum & causal inference



Cognitive control in the self-regulation of physical activity and sedentary behavior

Jude Buckley¹, Jason D. Cohen², Arthur F. Kramer^{2,3}, Edward McAuley^{2,3} and Sean P. Mullen^{2,3*}

¹ School of Psychology, University of Auckland, Auckland, New Zealand

² Department of Kinesiology and Community Health, University of Illinois at Urbana-Champaign, Urbana, IL, USA

³ Beckman Institute for Advanced Science and Technology, Urbana, IL, USA

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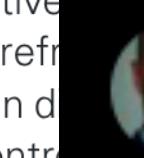
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Cognitive control of physical activity and sedentary behavior is receiving increased attention in the neuroscientific and behavioral medicine literature as a means of better understanding and improving the self-regulation of physical activity. Enhancing individuals' cognitive control capacities may provide a means to increase physical activity and reduce sedentary behavior. First, this paper reviews emerging evidence of the antecedence of cognitive control abilities in successful self-regulation of physical activity, and in precipitating self-regulation failure that predisposes to sedentary behavior. We then highlight the brain networks that may underpin the cognitive control of physical activity, including the default mode network, prefrontal brain regions and pathways associated with reward. We then describe training interventions that document improved cognitive control of influencing physical activity regulation. Key cognitive training the most effective at improving self-regulation are also highlighted, along with suggestions for future research.

Keywords: cognitive control, self-regulation, executive functioning, physical ac-



Dr. Sean Mullen 
@drseanmullen

I'm going to come back to this after I publish a paper. Then we can argue whether or not cognitive neuroscience is critical in health behavior change & maintenance. I'll claim it's integral to exercise adherence. Exercise is part of a broader healthier lifestyle we could promote.

...

Argument: cognition → exercise

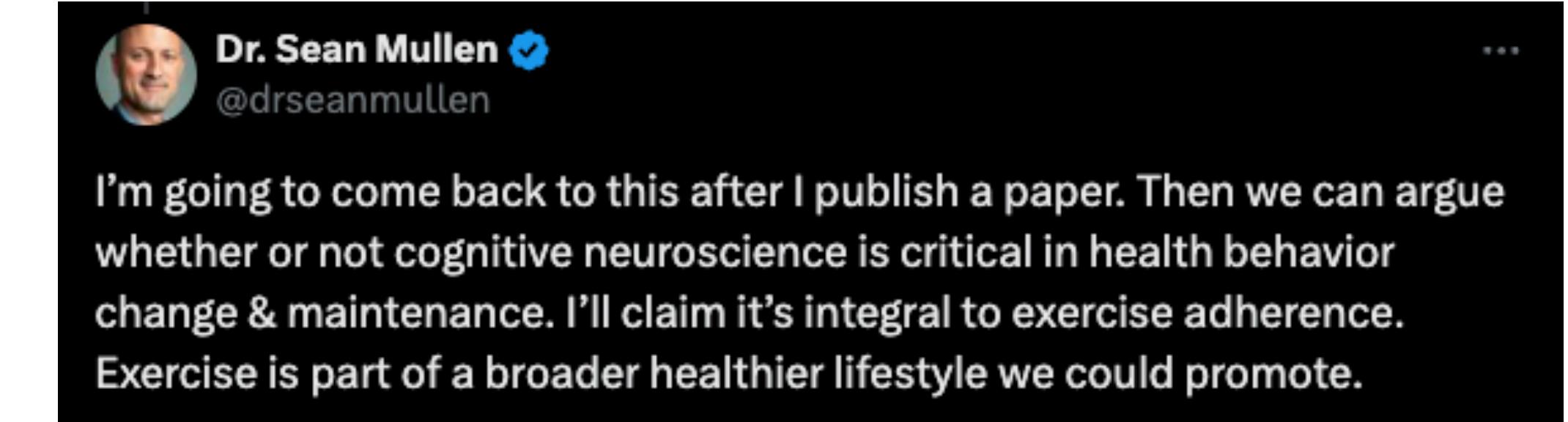
Evidence: exercise → cognition

Case study 3

Explanans/explanandum & causal inference

“To date, no research has examined whether cognitive training may have transfer effects on the self-regulation of physical activity behavior.”

- Buckley, Cohen, Kramer, McAuley & [Mullen \(2014\)](#)



Argument: cognition → exercise

Evidence: exercise → cognition

Case Study 4

Explanans/explanandum & causal inference

ORIGINAL ARTICLE

Subcortical brain alterations in major depressive disorder: findings from the ENIGMA Major Depressive Disorder working group

L Schmaal¹, DJ Veltman¹, TGM van Erp², PG Sämann³, T Frodl^{4,5}, N Jahanshad⁶, E Loehrer⁷, H Tiemeier^{7,8}, A Hofman⁷, WJ Niessen^{9,10},

The pattern of structural brain alterations associated with major depressive disorder (MDD) remains unresolved. This is in part due to small sample sizes of neuroimaging studies resulting in limited statistical power, disease heterogeneity and the complex interactions between clinical characteristics and brain morphology. To address this, we meta-analyzed three-dimensional brain magnetic resonance imaging data from 1728 MDD patients and 7199 controls from 15 research samples worldwide, to identify subcortical brain volumes that robustly discriminate MDD patients from healthy controls. Relative to controls, patients had significantly lower hippocampal volumes (Cohen's $d = -0.14$, % difference = -1.24). This effect was driven by patients with recurrent MDD (Cohen's $d = -0.17$, % difference = -1.44), and we detected no differences between first episode patients and controls. Age of onset ≤ 21 was associated with a smaller hippocampus (Cohen's $d = -0.20$, % difference = -1.85) and a trend toward smaller amygdala (Cohen's $d = -0.11$, % difference = -1.23) and larger lateral ventricles (Cohen's $d = 0.12$, % difference = 5.11). Symptom severity at study inclusion was not associated with any regional brain volumes. Sample characteristics such as mean age, proportion of antidepressant users and proportion of remitted patients, and methodological characteristics did not significantly moderate alterations in brain volumes in MDD. Samples with a higher proportion of antipsychotic medication users showed larger caudate volumes in MDD patients compared with controls. This currently largest worldwide effort to identify subcortical brain alterations showed robust smaller hippocampal volumes in MDD patients, moderated by age of onset and first episode versus recurrent episode status.

Molecular Psychiatry (2016) **21**, 806–812; doi:10.1038/mp.2015.69; published online 30 June 2015

Case Study 4

Explanans/explanandum & causal inference

Influence of recurrence status on brain volume. We examined how the current stage of a depressed patient may relate to brain volumes by splitting the sample into first episode ($n=583$) and recurrent episode patients ($n=1119$) and compared to healthy controls (Figure 2a). We did not detect any significant differences between first episode patients and healthy controls (all P -values >0.3). Recurrent episode patients showed lower mean hippocampal volume than controls ($d=-0.17$ (-0.25 , -0.10); P -value = 1.12×10^{-5} , % difference = -1.44). Full meta-analyzed recurrence status differences are listed in Supplementary Table S5 and Supplementary Table S6. Relative to the full MDD sample, recurrent patients showed larger effect sizes compared to controls. However, no significant differences were detected between recurrent and first episode patients (Supplementary Table S7).

Results

Influence of age of onset of depression on brain volume. We examined how the age of onset modulates volumetric brain changes (Figure 2b). Patients with an early age of onset (≤ 21 years; $n=541$) showed significantly lower mean hippocampal volumes than controls ($d=-0.20$ (-0.31 , -0.10); P -value = 2.31×10^{-4} , % difference = -1.85). In addition, we found lower amygdala volume ($d=-0.12$ (-0.23 , -0.01); P -value = 0.033 , % difference = -1.23) and higher mean lateral ventricle volume ($d=0.14$ (0.04 , 0.25); P -value = 0.009 , % difference = 5.11) in early onset patients compared to controls, but neither survived correction for multiple comparisons. Although sample sizes were too small to split first and recurrent episode patients into early and late onset groups, only about half (57%) of the early onset MDD patients had a recurrent episode, and the percentage of recurrent episode patients did not moderate the result of smaller hippocampal volumes in early onset MDD patients ($P=0.54$), suggesting that this effect is at least partly independent of recurrence status. Patients with a late age of onset (> 21 years; $n=997$) showed no detectable brain volumetric differences compared to controls. Full age of onset effect sizes are listed in Supplementary Table S8 and Supplementary Table S9. The effect sizes in early episode patients are larger than those obtained when considering the full

Case study 4

Explanans/explanandum & causal inference

Discussion

Our finding of smaller hippocampal volume in MDD is in line with previous retrospective meta-analyses of aggregated data.^{3–6} This robust finding of smaller hippocampal volume is often linked to the ‘neurotrophic hypothesis of depression’. This proposes that elevated glucocorticoid levels associated with chronic hyperactivity of the hypothalamic–pituitary–adrenal axis in MDD may induce brain atrophy via remodeling and downregulation of growth factors including brain-derived neurotrophic factor.¹⁹

Case study 4

Explanans/explanandum & causal inference

Coverage

“I think this resolves for good the issue that persistent experiences of depression hurts the brain”

- Prof Ian Hickie [coauthor]

<https://theconversation.com/depression-damages-parts-of-the-brain-research-concludes-43915>

Case study 4

Explanans/explanandum & causal inference

Coverage

“Those who have only ever had one episode do not have a smaller hippocampus, so it’s not a predisposing factor but a consequence of the illness state.”

- Prof Ian Hickie [coauthor]

<https://theconversation.com/depression-damages-parts-of-the-brain-research-concludes-43915>

Case study 4

Explanans/explanandum & causal inference

Response

Schaal et al (2016b)

“In our original paper, we did not claim that depression causes structural changes”

Case study 4

Explanans/explanandum & causal inference

Critique

Fried & Kievit (2016)

“to identify subcortical brain volumes that robustly discriminate MDD patients from healthy controls.”

What is Schaal’s goal?

- To use depression status to predict brain volume?
 - hippocampal_volume ~ depression_status
- To use brain volume to predict who has depression?
 - depression_status ~ hippocampal_volume

Case study 4

Explanans/explanandum & causal inference

Critique

Fried & Kievit (2016)

“to identify subcortical brain volumes that robustly discriminate MDD patients from healthy controls.”

What is Schaal’s goal? **Unclear**

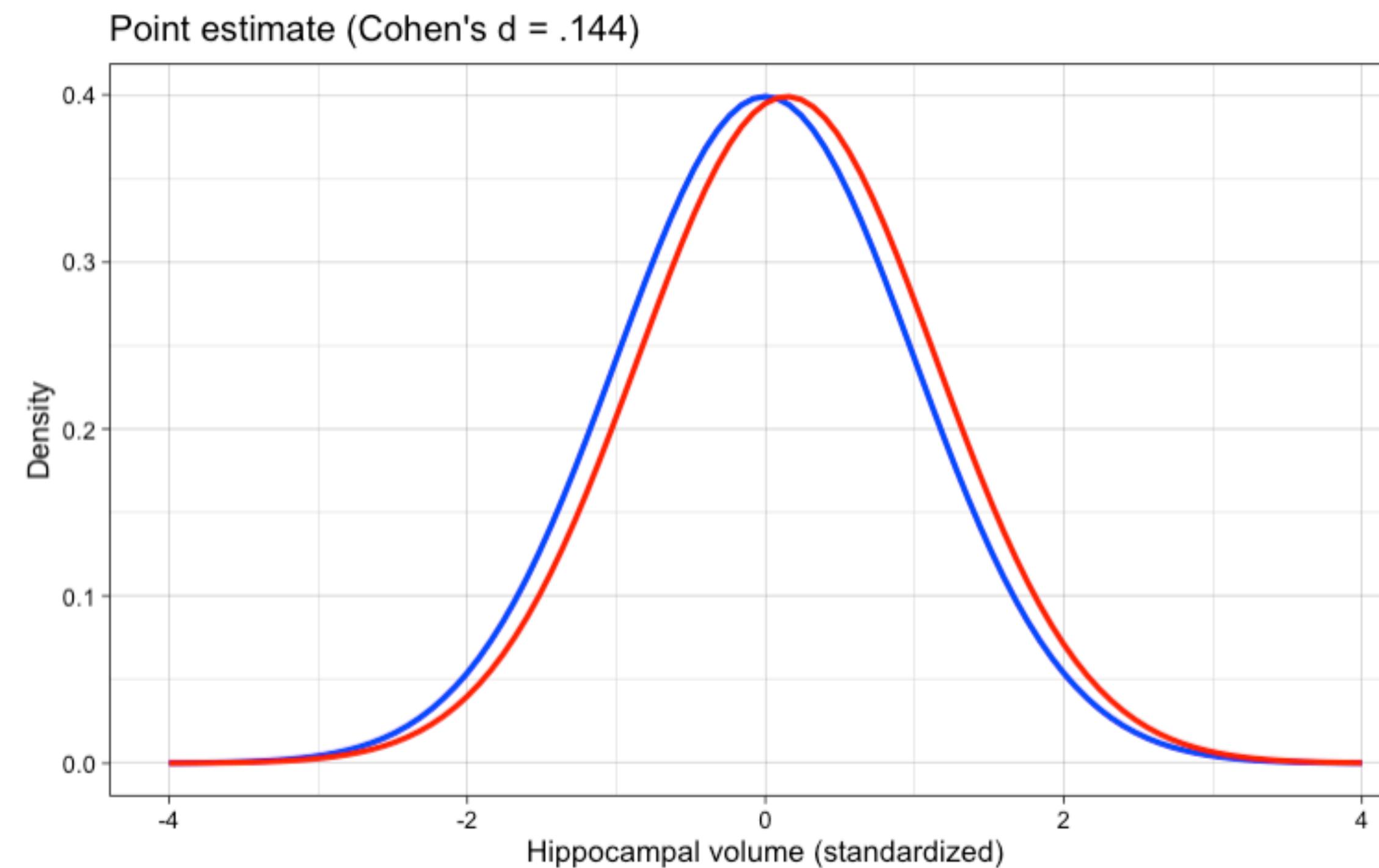
- To use depression status to predict brain volume?
 - hippocampal_volume ~ depression_status ← **What original did**
- To use brain volume to predict who has depression?
 - depression_status ~ hippocampal_volume

Critique

Fried & Kievit (2016)

“subcortical brain volumes that robustly discriminate MDD patients from healthy controls.”

Case study 4
Explanans/explanandum & causal inference



Case study 4

Explanans/explanandum & causal inference

Critique

Fried & Kievit (2016)

“subcortical brain volumes that robustly discriminate MDD patients from healthy controls.”

(simulated)

Classification accuracy

51.7%

95% CI [50.8, 52.9]

Case study 4

Explanans/explanandum & causal inference

Critique

Fried & Kievit (2016)

“subcortical brain volumes that robustly discriminate MDD patients from healthy controls.”

- Not specific to depression
- Alternative causes
 - Genetics
 - Exercise
- Could be a statistical artefact

Case study 4

Explanans/explanandum & causal inference

Response

Schaal et al (2016b)

“In our original paper, we did not claim that depression causes structural changes”

Case study 4

Explanans/explanandum & causal inference

Response

Schaal et al (2016b)

“We speculated that hippocampal volume reductions may be promoted by a chronic hyperactivity of the hypothalamic – pituitary – adrenal axis via remodelling and downregulation of growth factors including brain-derived neurotrophic factor, associated with (chronic) stress. Stressors include multiple episodes of depression, early-life stress and a family history of depression, which are all linked to early-onset depression, higher risk for recurrent depression, an overactive hypothalamic – pituitary – adrenal axis and smaller hippocampal volume.”

Case study 4

Explanans/explanandum & causal inference

Response

Schaal et al (2016b)

“We speculated that hippocampal volume reductions may be promoted by ... stress.

Stressors include ... smaller hippocampal volume.”

This is circular.

Consequence of unclear explanans vs explanandum.

Causal assumptions

in regression and SEM

Regressions assume causality

Psychologists talk about ‘assumptions’ as if they are personal beliefs.

Assumptions are mathematical assumptions of the test, which can change conclusions if violated.

Causal assumptions

in regression and SEM

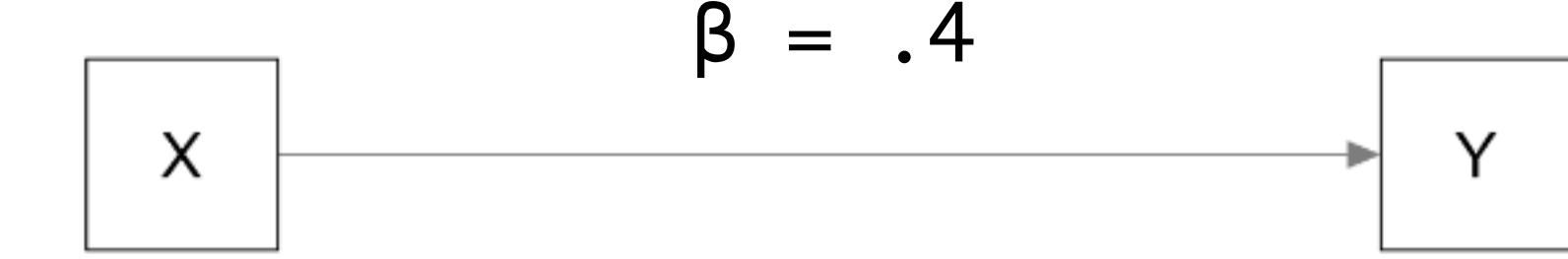
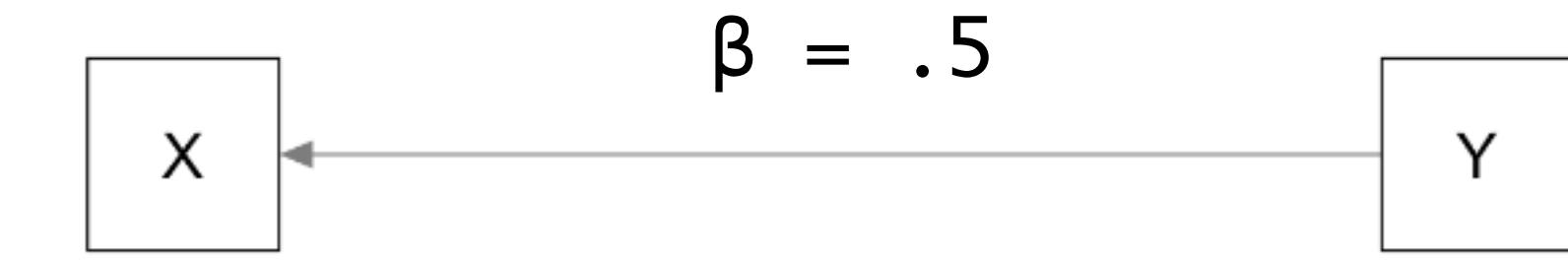
True population: Y causes X

Analysis: “How much does X cause Y?”

Results: $\beta = .40, p < .05$

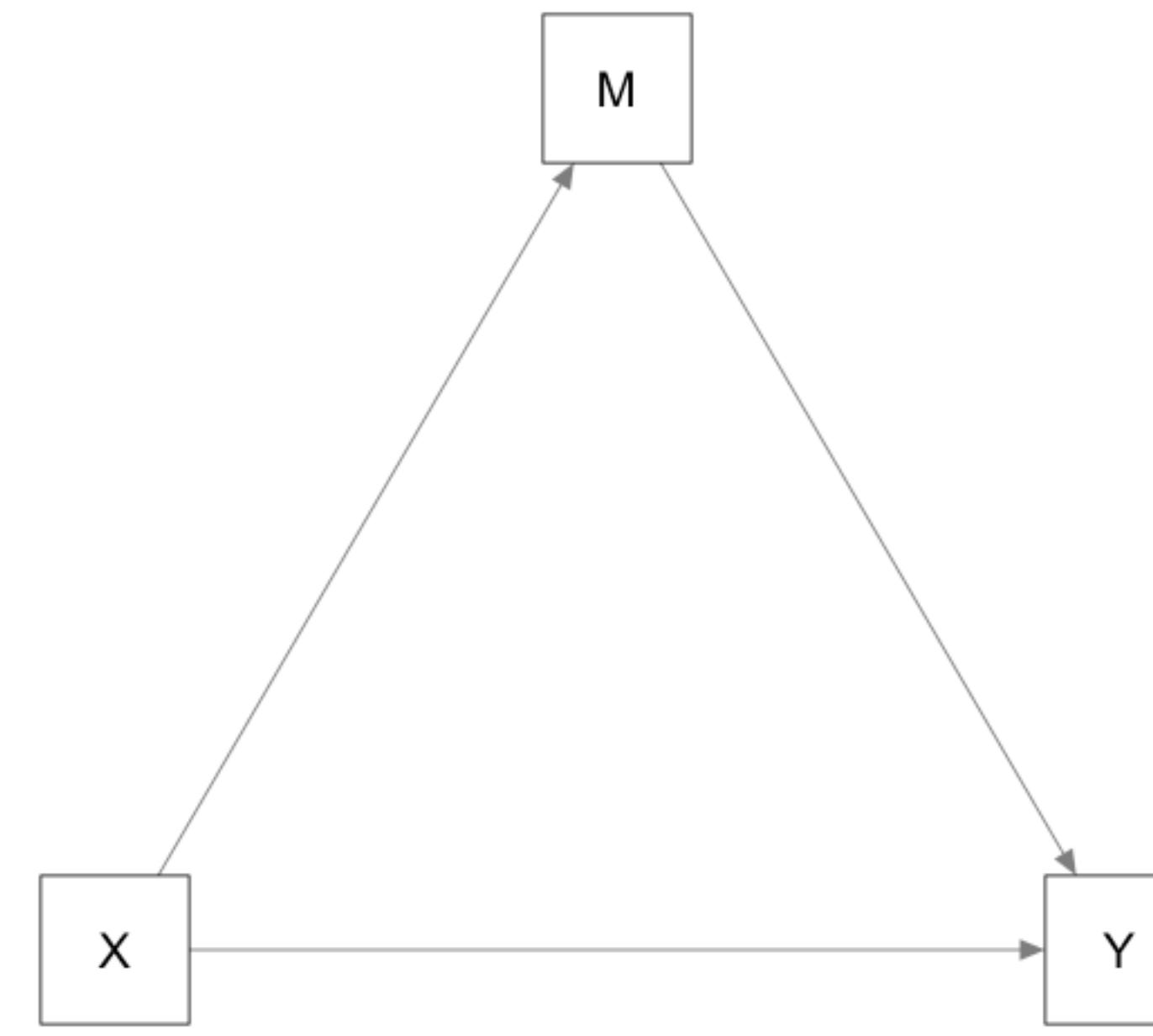
They do not test causality

Regressions assume causality



Causal assumptions

in regression and SEM

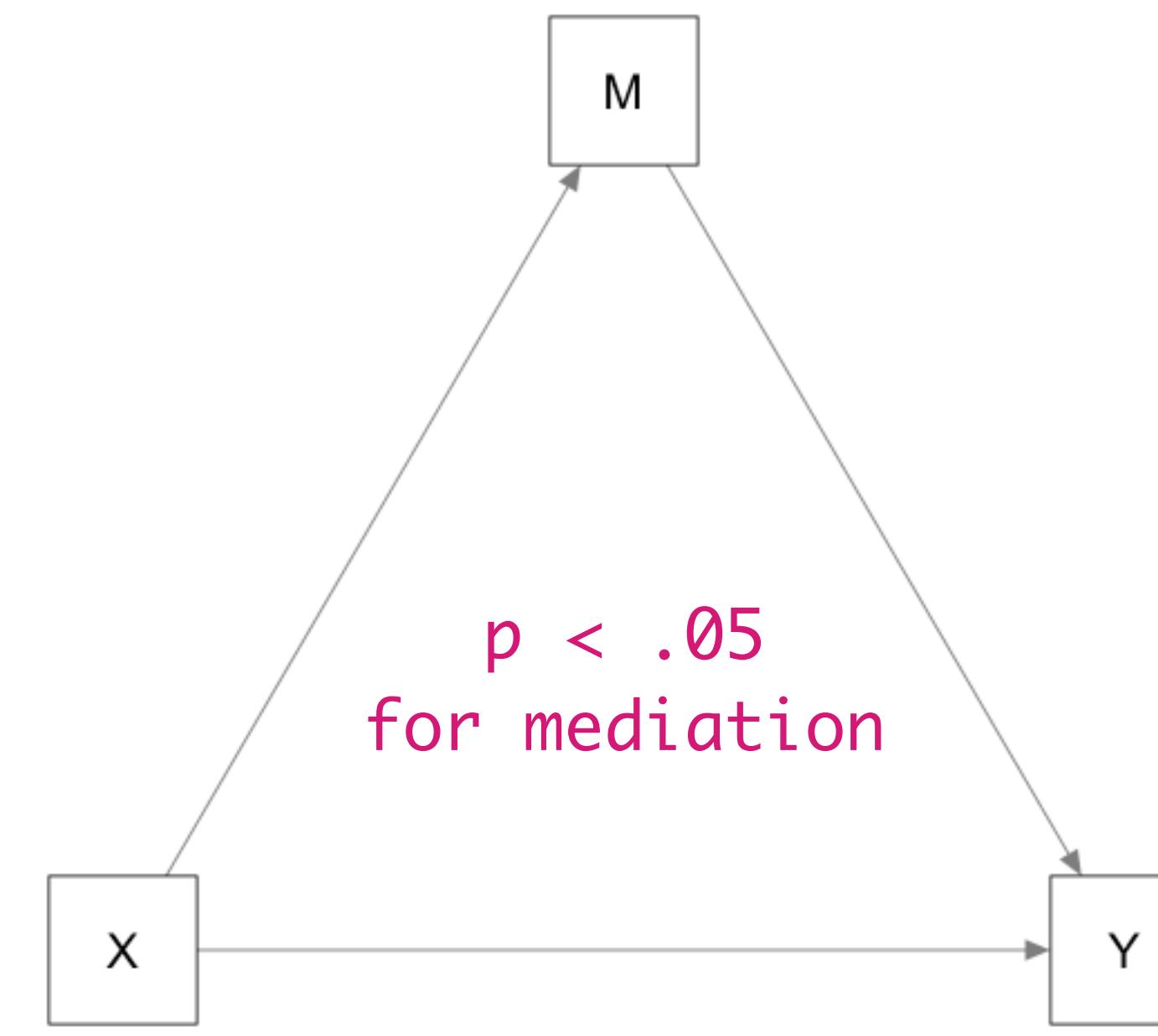


O'Connor's (2011) 'Integrated Motivational-Volitional Model of Suicidal Behavior'

Defeat (X) causes suicidality (Y)
Both directly and mediated via
entrapment (M)

Causal assumptions

in regression and SEM

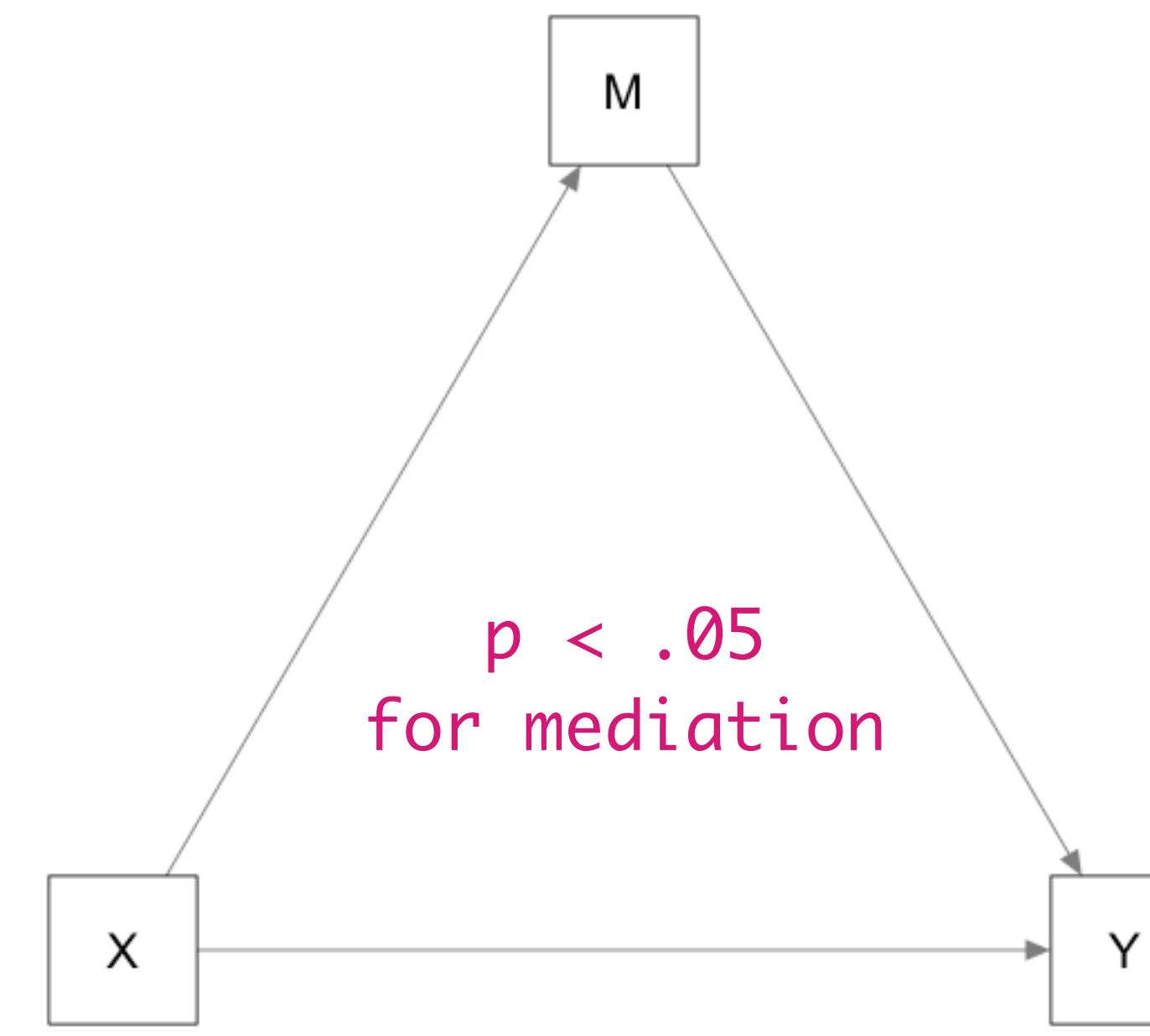


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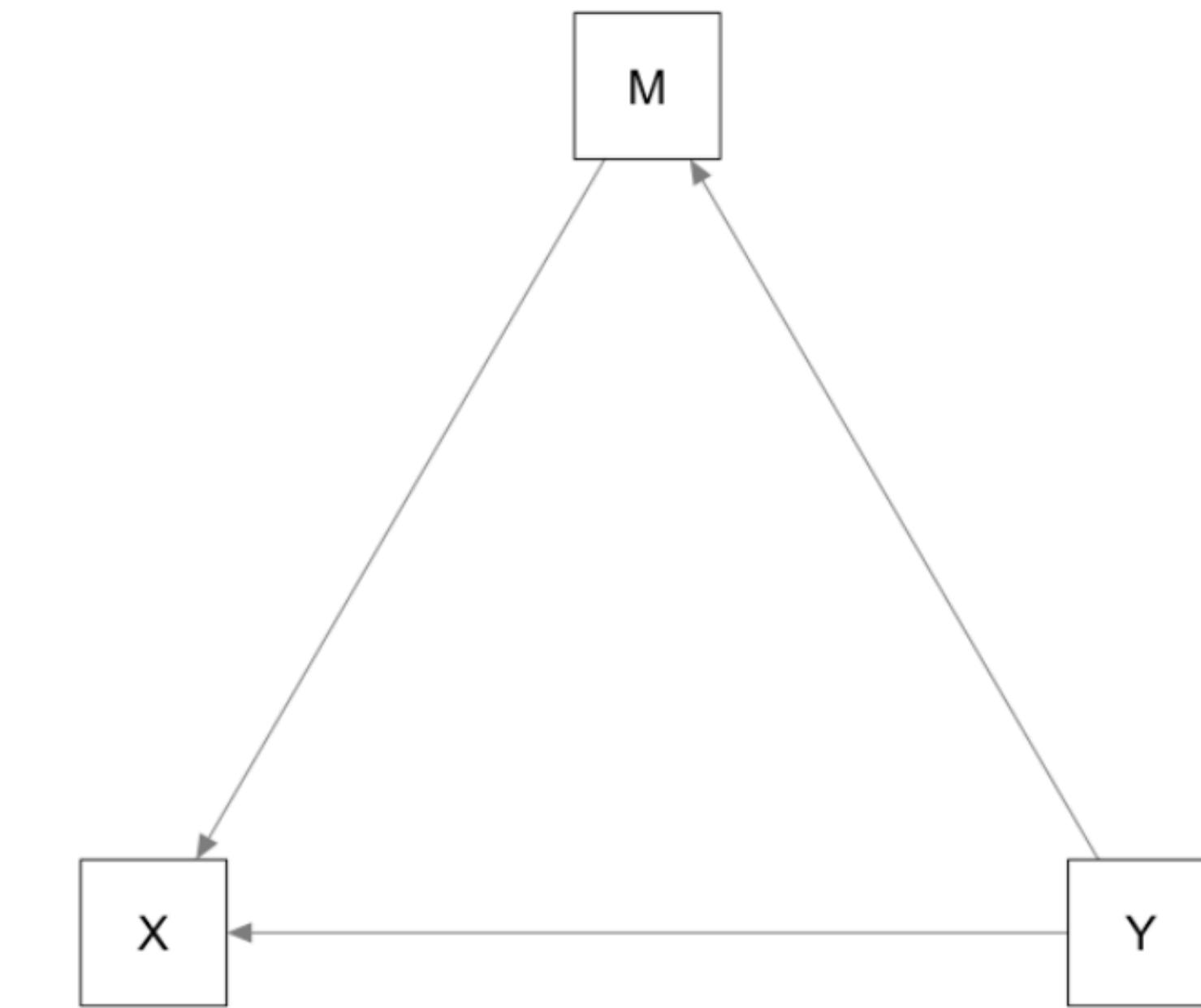
Causal assumptions

in regression and SEM



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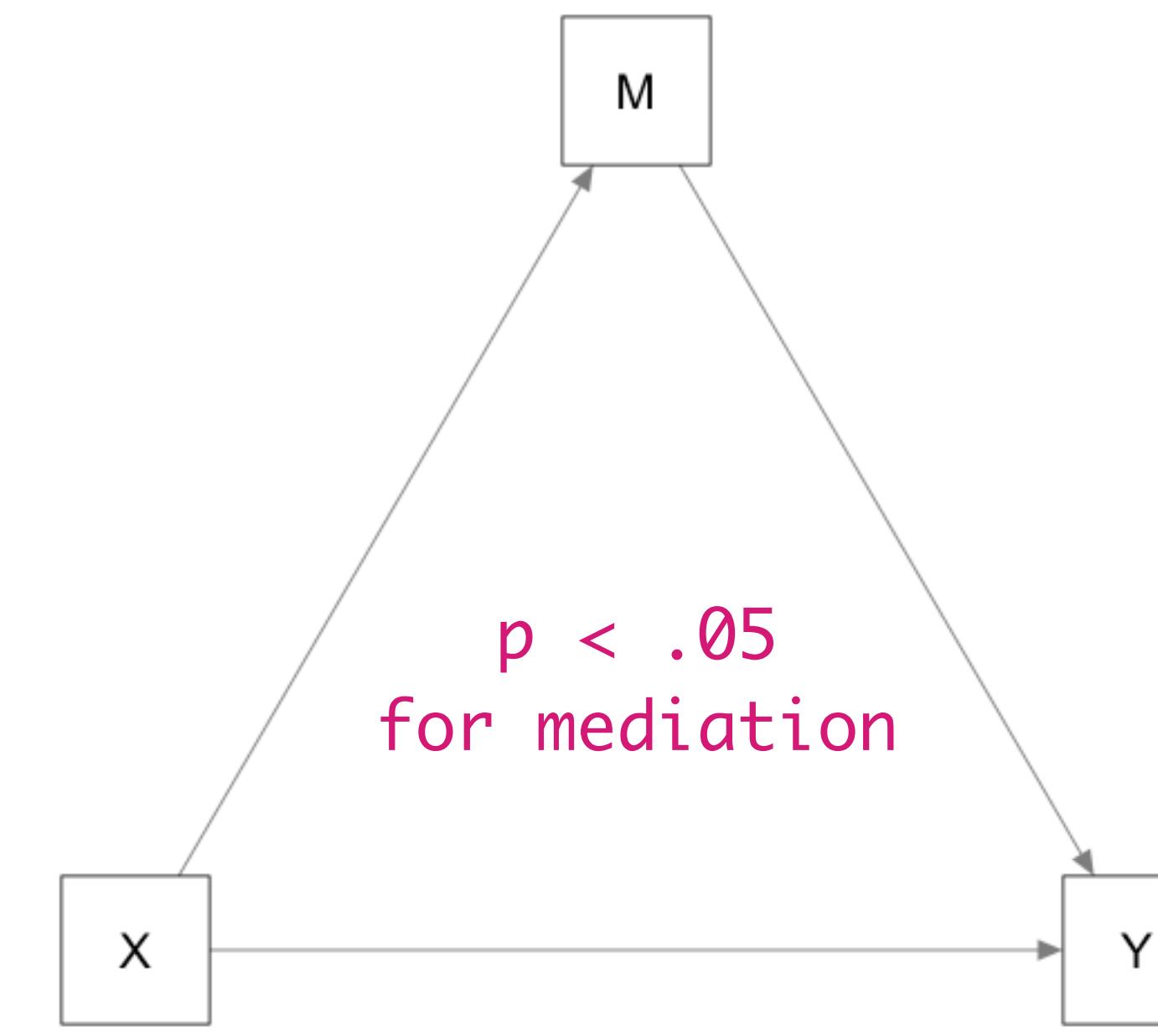


Analysis with the wrong model

Suicidal thoughts (Y) cause defeat (X)
Both directly and mediated via entrapment (M)

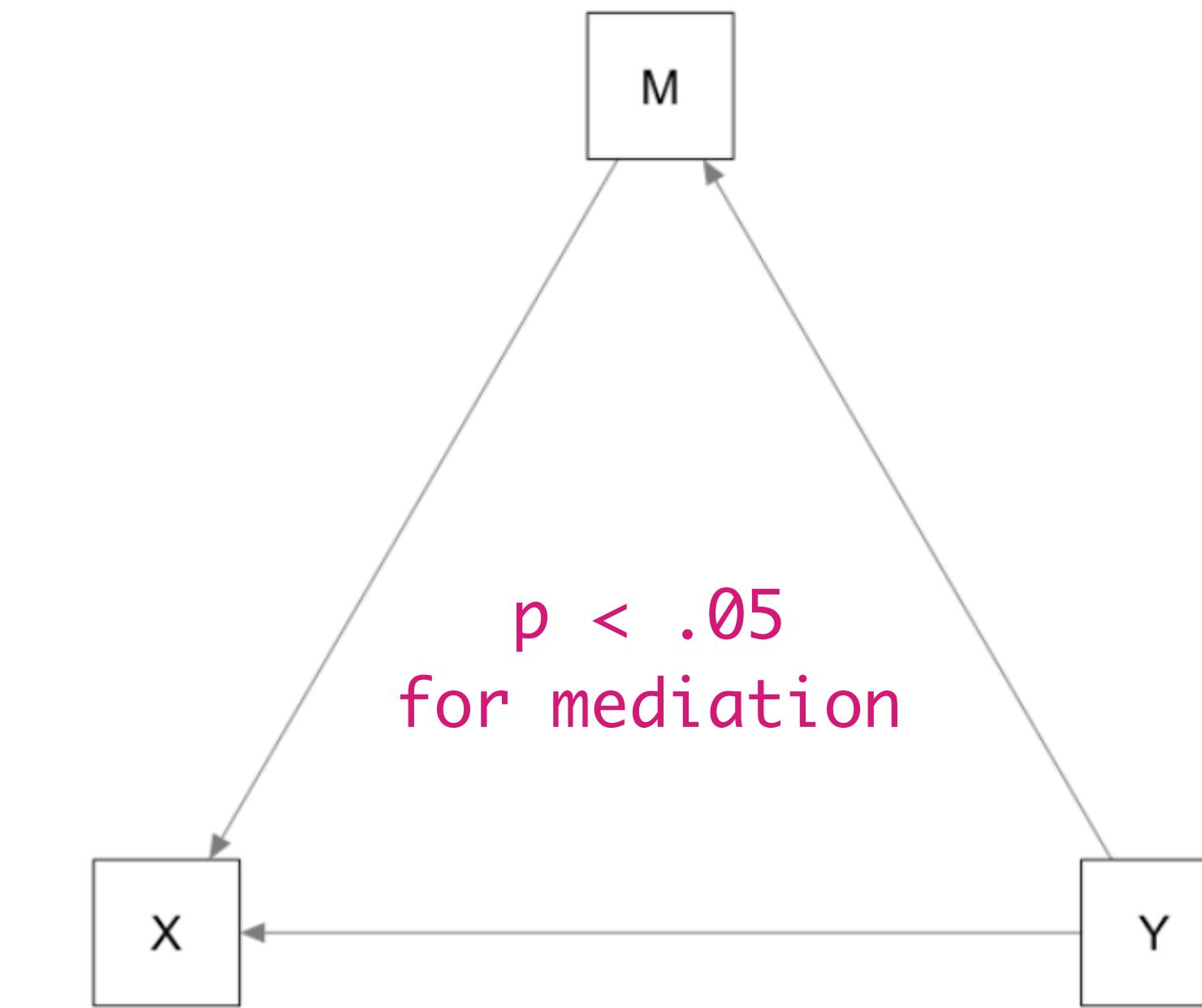
Causal assumptions

in regression and SEM



O'Connor's (2011) 'Integrated Motivational-Volitional Model of Suicidal Behavior'

Defeat (X) causes suicidality (Y)
Both directly and mediated via
entrapment (M)



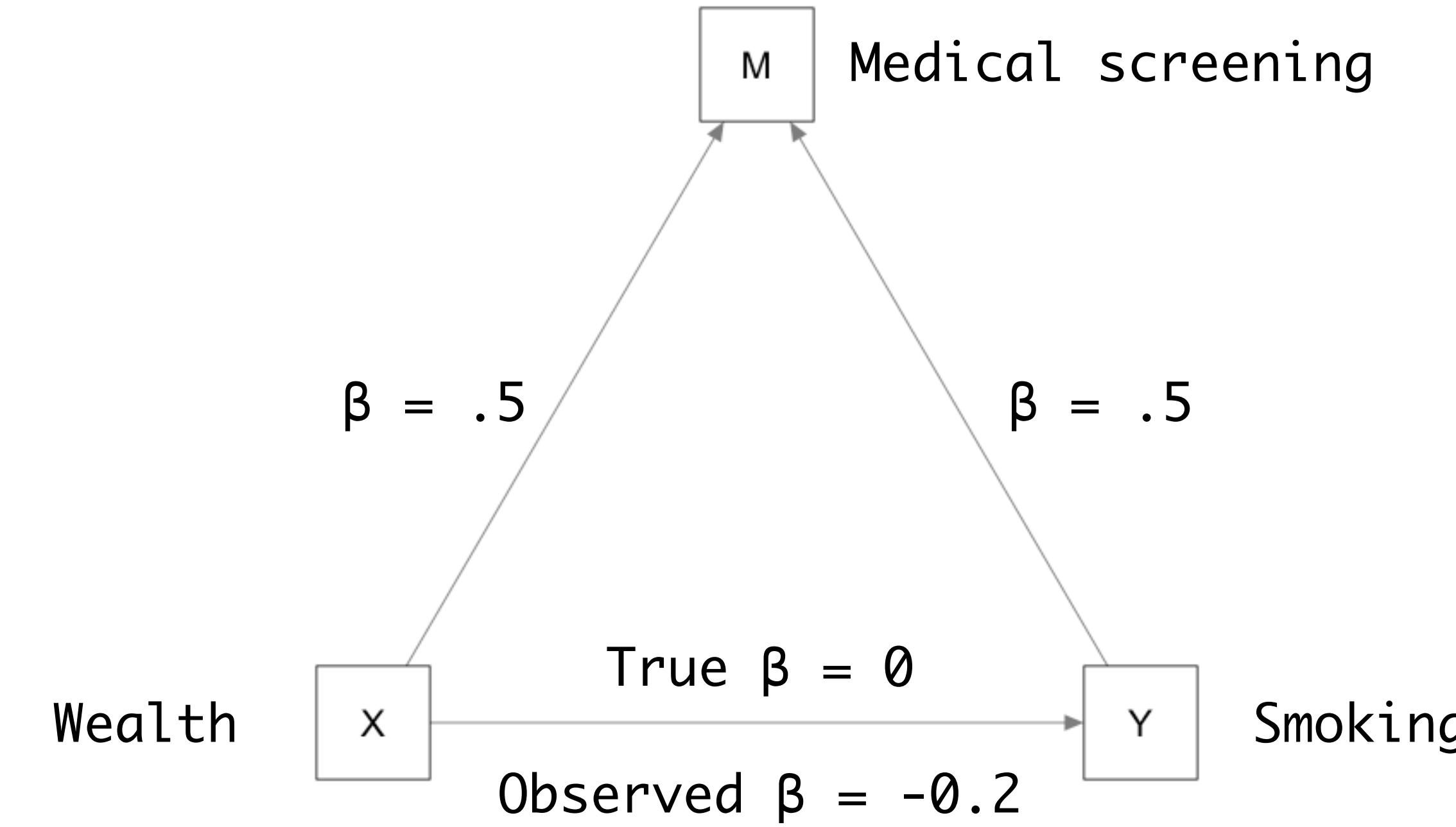
Analysis with the wrong model

Suicidal thoughts (Y) cause defeat (X)
Both directly and mediated via
entrapment (M)

Causal assumptions

in regression and SEM

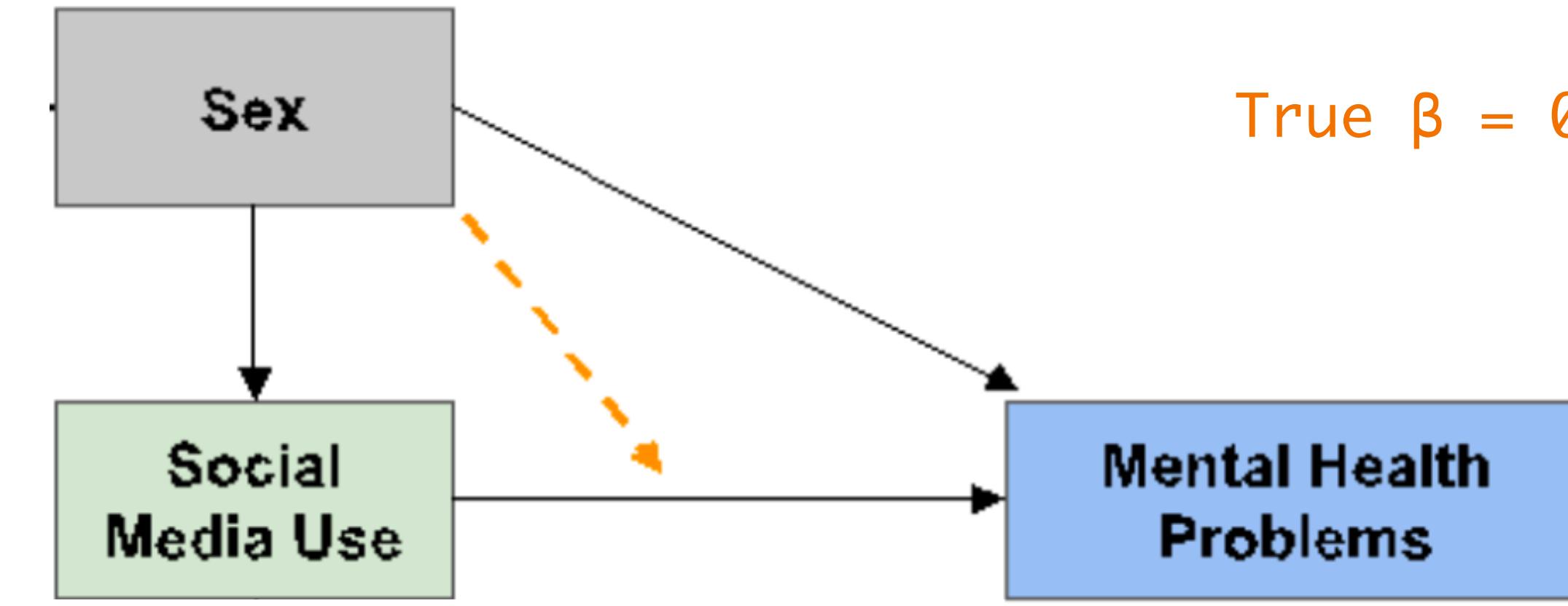
Data produced by a ‘Collider’
but analysed using other models (eg mediation)
produce spurious associations



“Wealthy people more likely to quit smoking!”

Causal assumptions

in regression and SEM



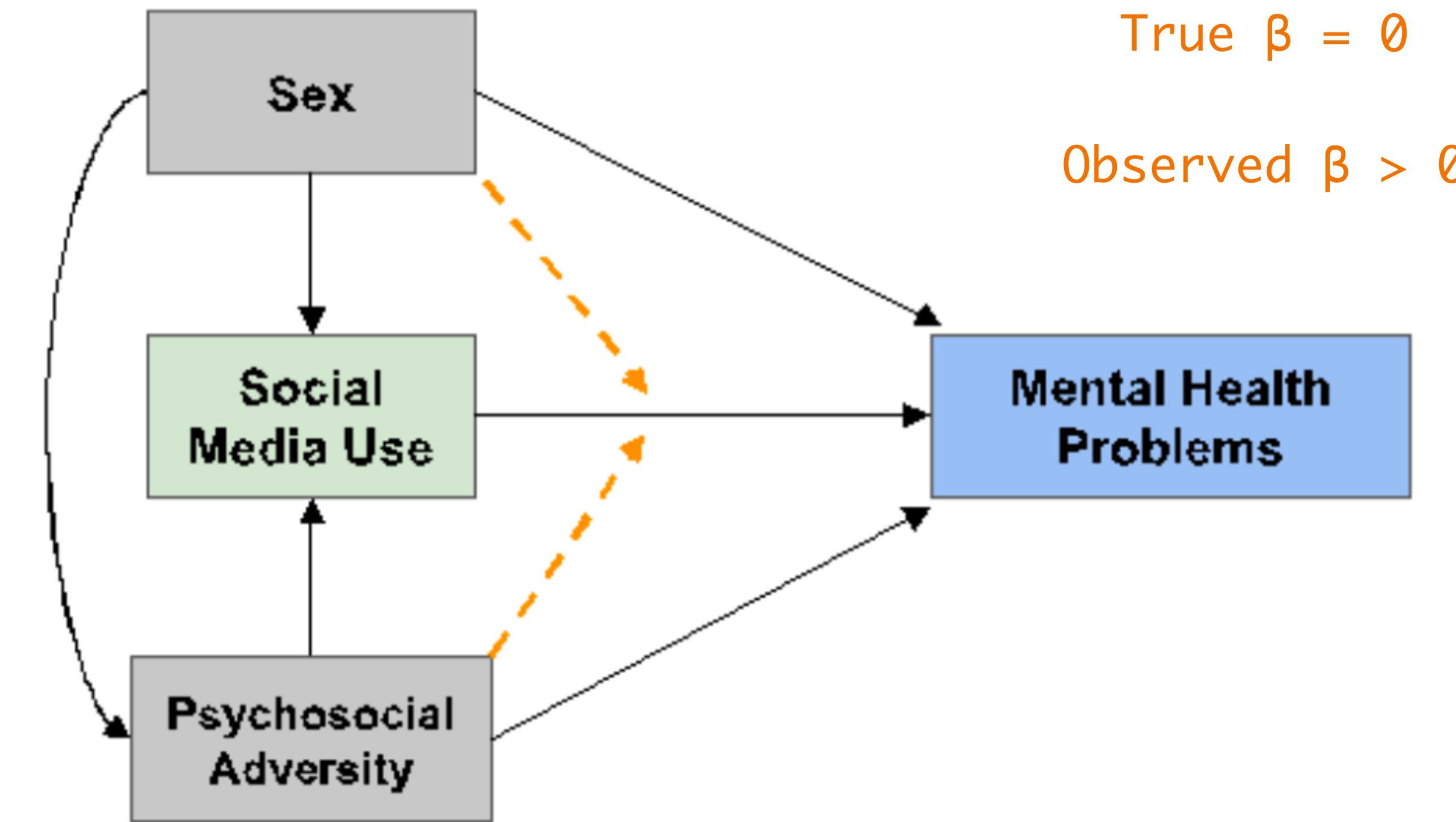
“Social media hurts adolescent girls’ mental health”

Causal assumptions

in regression and SEM

Omitted variables in larger causal models
Often give rise to statistical artefacts

These effects are replicable but untrue!



“Social media hurts adolescent girls’ mental health”

Take-home points:

1. Research that confuses these will go in circles
 - What is to be explained (explanandum)
 - What does the explaining (explanans)
2. Your analyses often make causal assumptions
3. Your readers will make causal interpretations
4. Make causal assumptions explicit