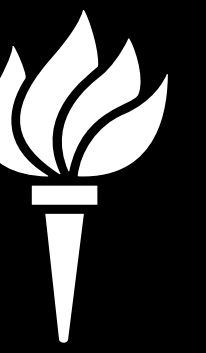




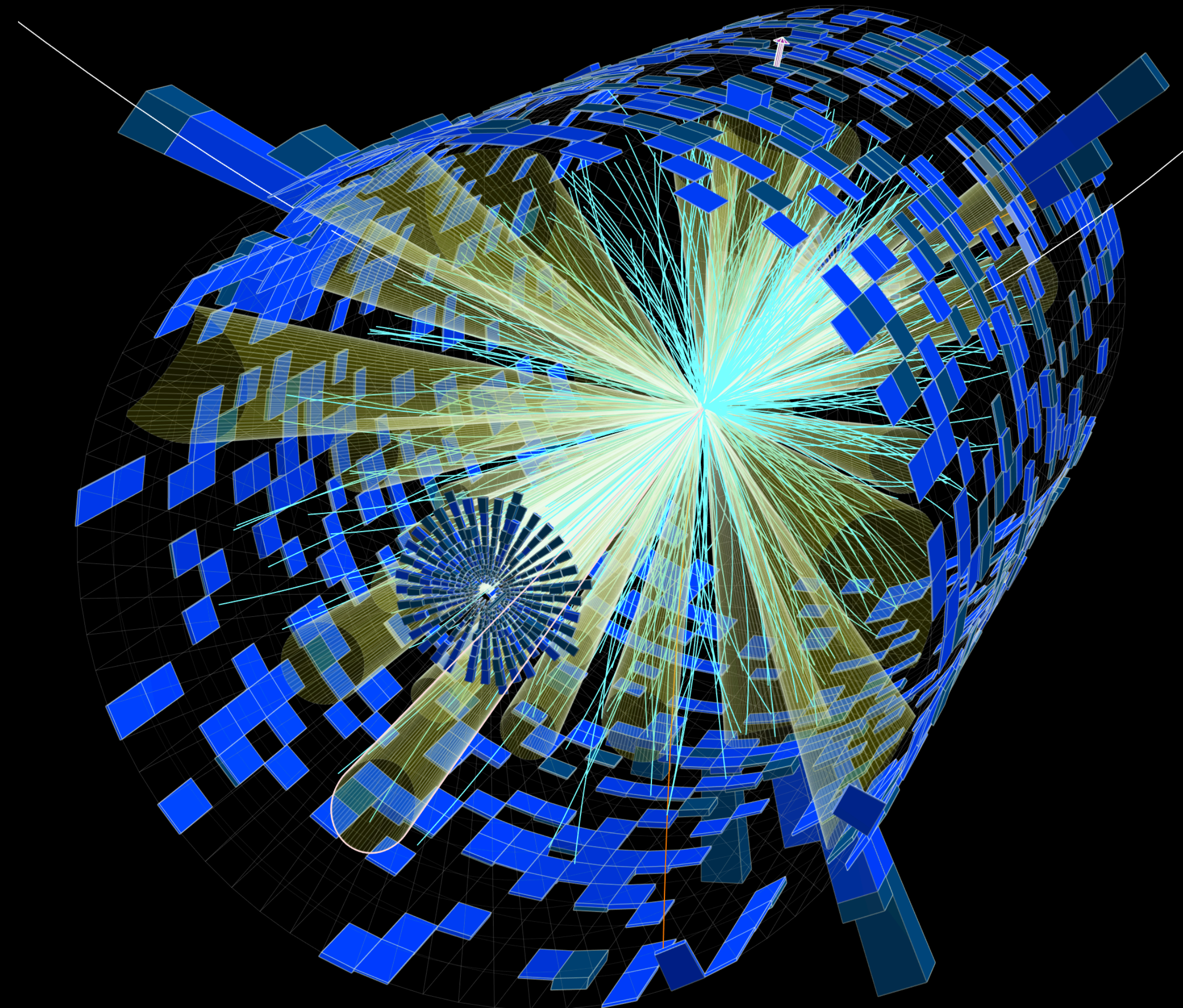
NYU CENTER FOR  
DATA SCIENCE

CENTER FOR  
COSMOLOGY AND  
PARTICLE PHYSICS



A CONVERSATION WITH

# NIHAT AY



**@KyleCranmer**  
New York University  
Department of Physics  
Center for Data Science  
CILVR Lab



# Yesterday's talk

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Recording

## Outline

- 1. The Structure of Causal Networks*
- 2. Markov Properties & Markov Equivalence*
- 3. Intervention & Causal Effects*
- 4. Identifiability of Causal Effects*
- 5. Application to Embodied Intelligence*
- 6. The Common Cause Principle*
- 7. Knockout Interventions for System Identification*

Ty Kamp

Kyle Cranmer

Priyamvada Natarajan

Nihat Ay

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Understanding  
the Nature of Inference:  
Correlation  
and Causation

A Multi-disciplinary  
Exploration  
Colloquium  
Series

THE FRANKE PROGRAM IN SCIENCE AND THE HUMANITIES  
AND THE JOHN TEMPLETON FOUNDATION

## On Experiments for Causal Inference and System Identification

A TALK BY

**Professor Nihat Ay**

Max Planck Institute for Mathematics in the Sciences

March 17, 2021 (for the talk)

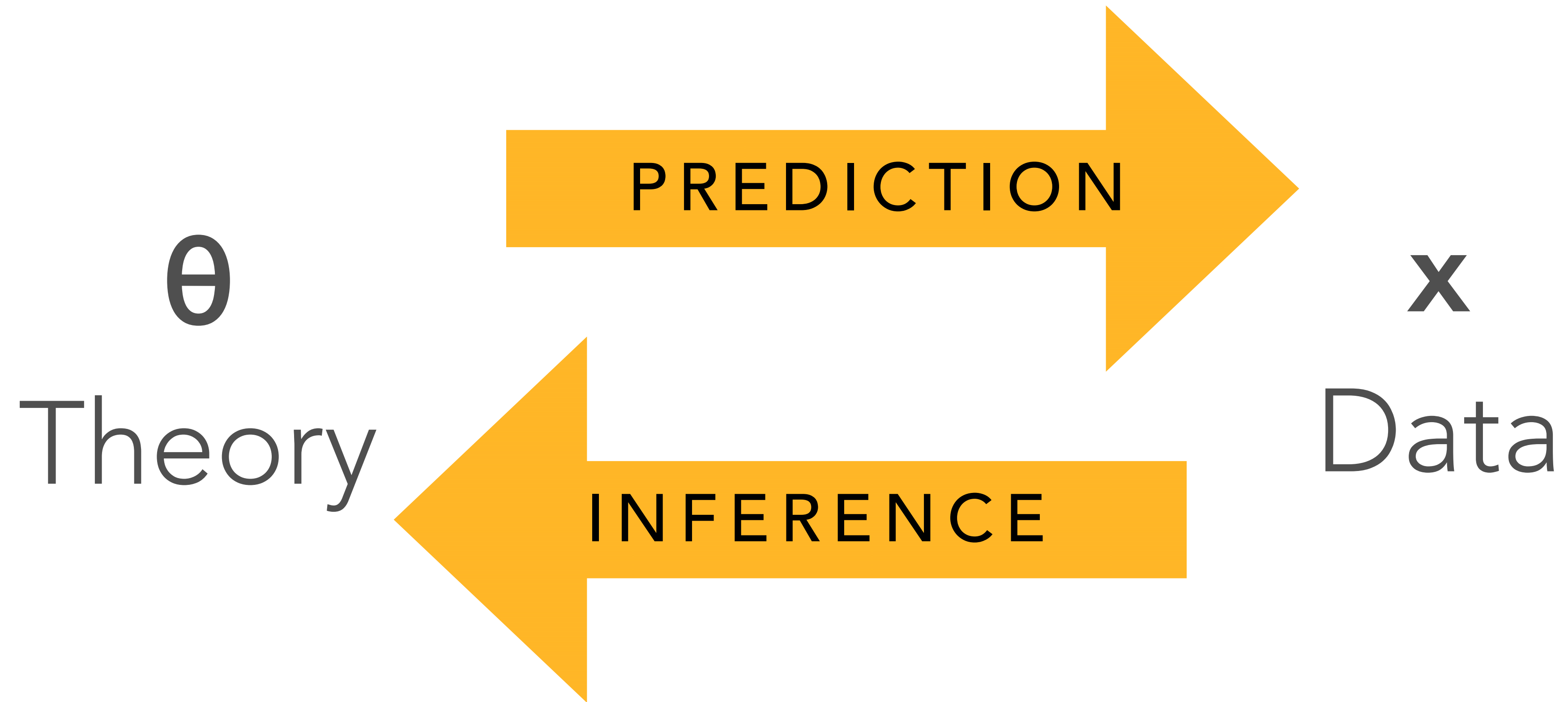
March 18, 2021 (moderated post-talk)

3:00 p.m.

Zoom details at

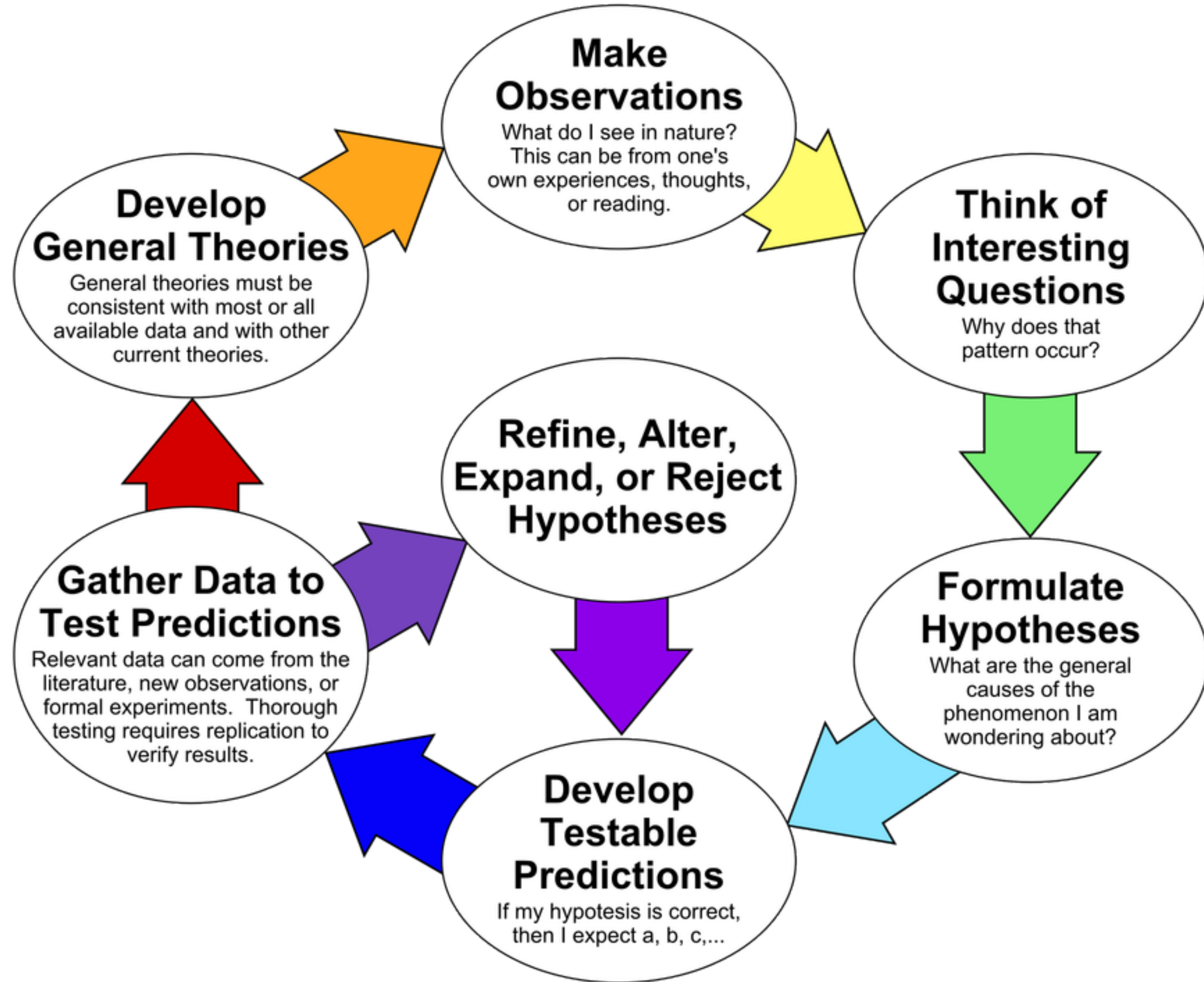
[frankeprogram.yale.edu](https://frankeprogram.yale.edu)







# The Scientific Method as an Ongoing Process





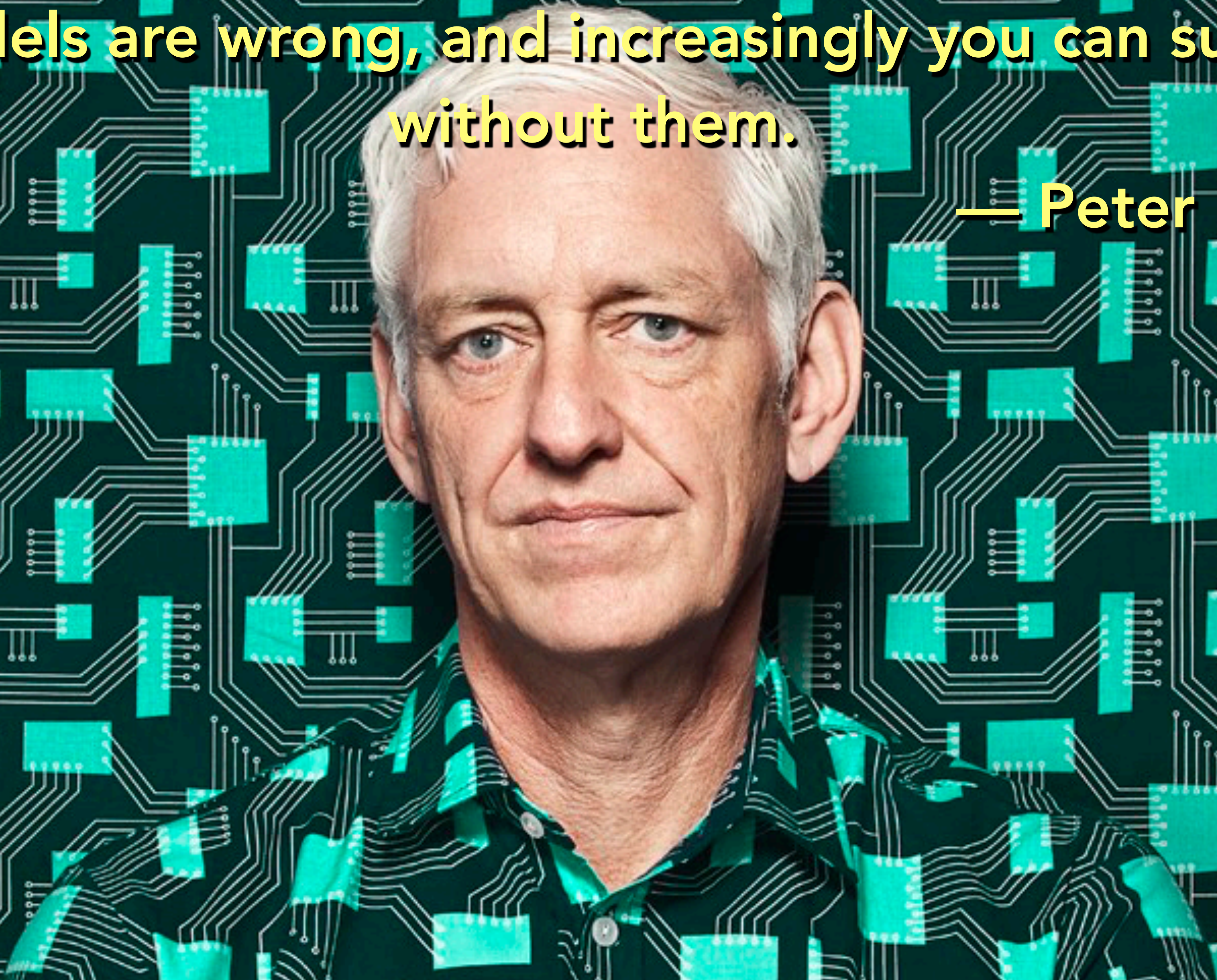
# THE END OF THEORY: THE DATA DELUGE MAKES THE SCIENTIFIC METHOD OBSOLETE





**All models are wrong, and increasingly you can succeed without them.**

**— Peter Norvig**





# The Ladder of Causation

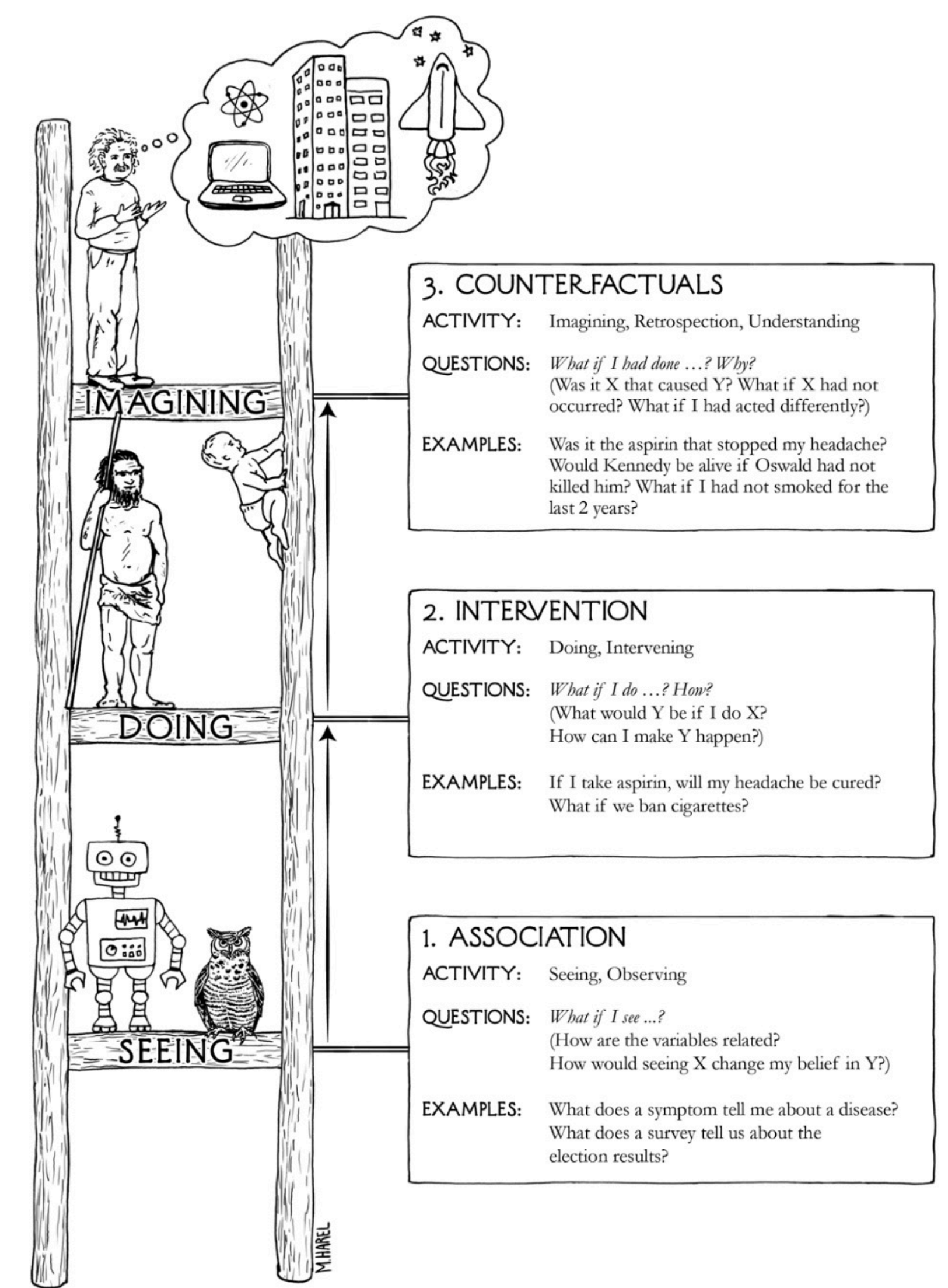


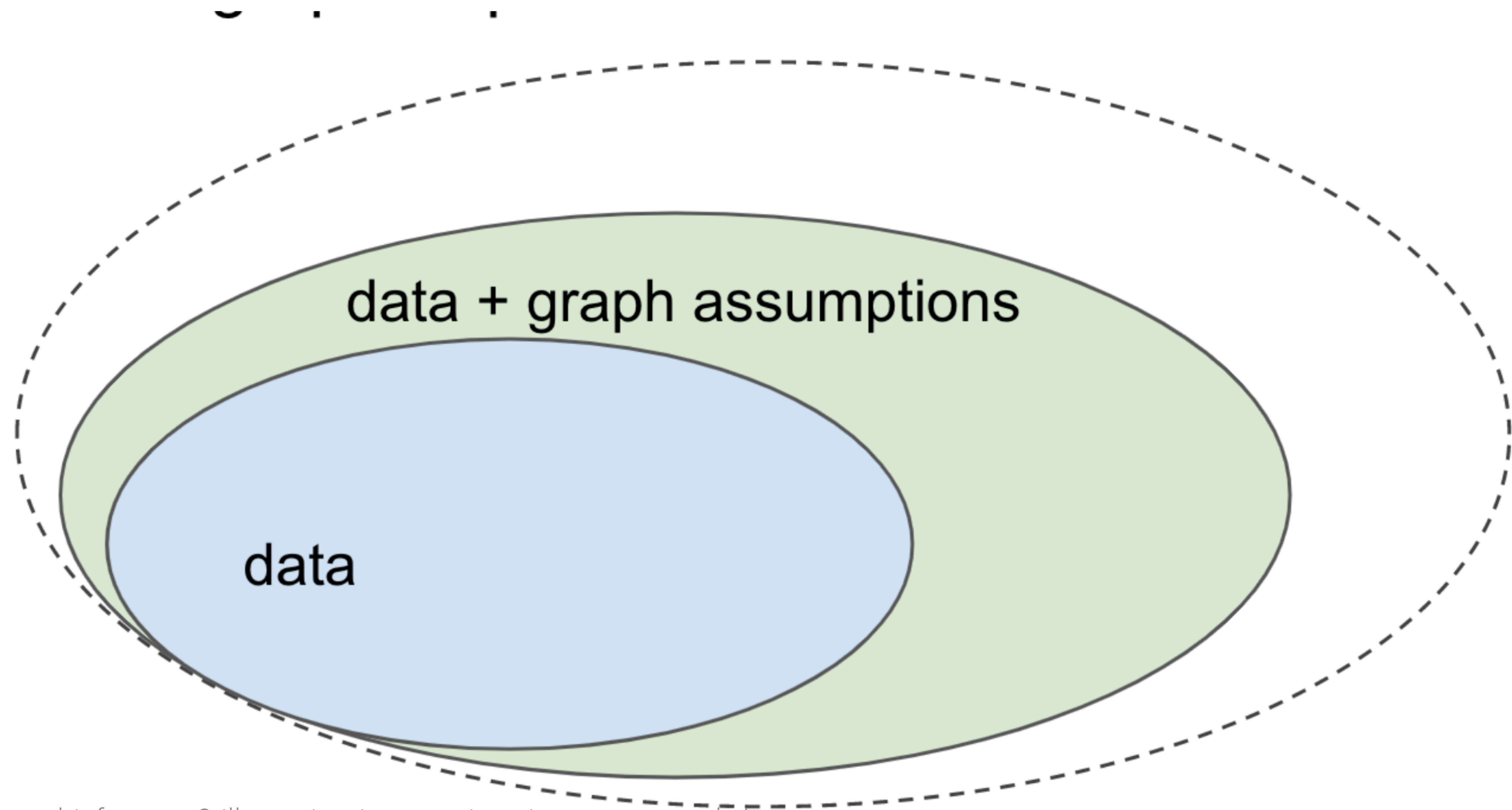
FIGURE 1.2. The Ladder of Causation, with representative organisms at each level. Most animals, as well as present-day learning machines, are on the first rung, learning from association. Tool users, such as early humans, are on the second rung if they act by planning and not merely by imitation. We can also use experiments to learn the effects of interventions, and presumably this is how babies acquire much of their causal knowledge. Counterfactual learners, on the top rung, can imagine worlds that do not exist and infer reasons for observed phenomena. (Source: Drawing by Maayan Harel.)





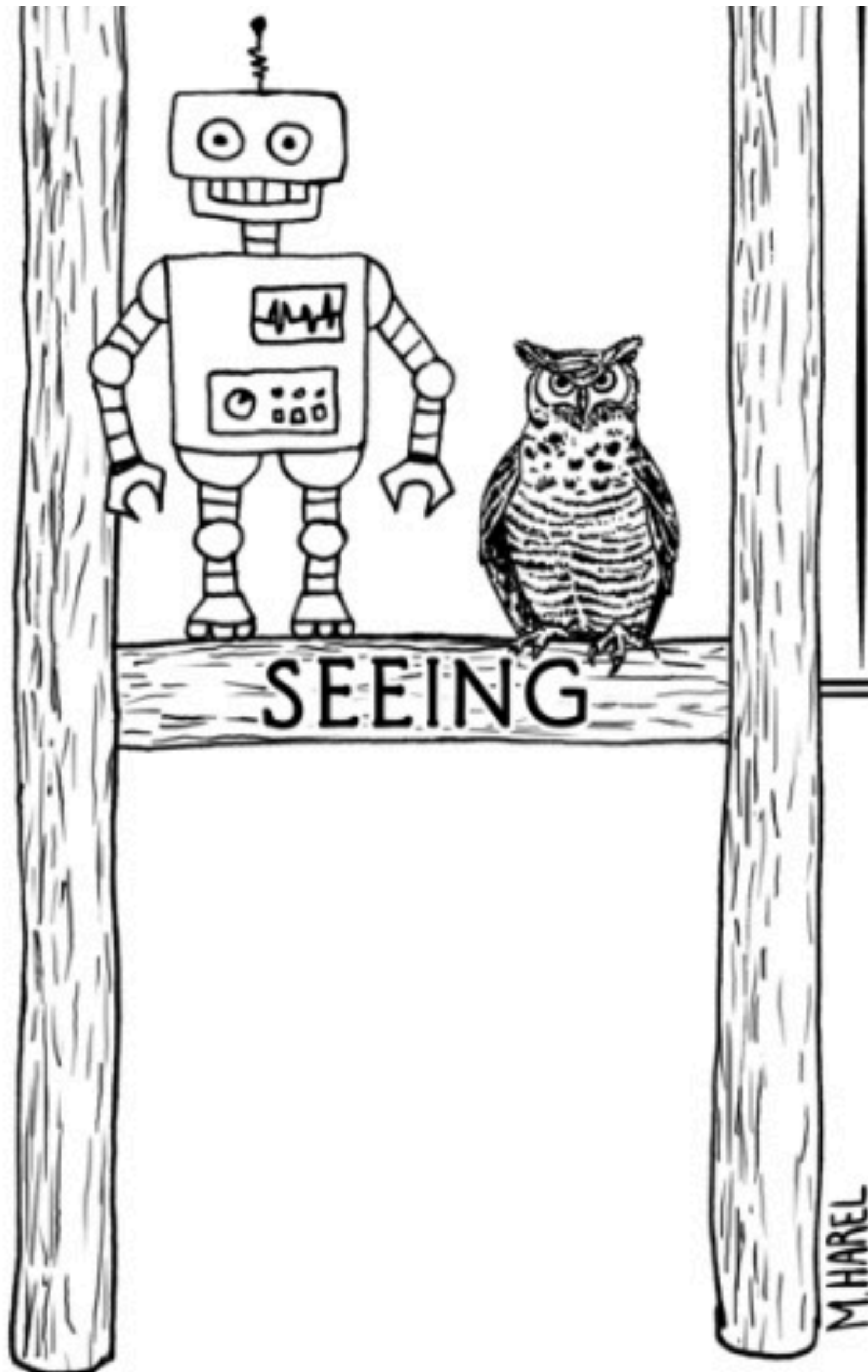
## The ~~morale~~ of the story

The ~~morale~~ of this story is summed up in the following picture:





# Association



## 1. ASSOCIATION

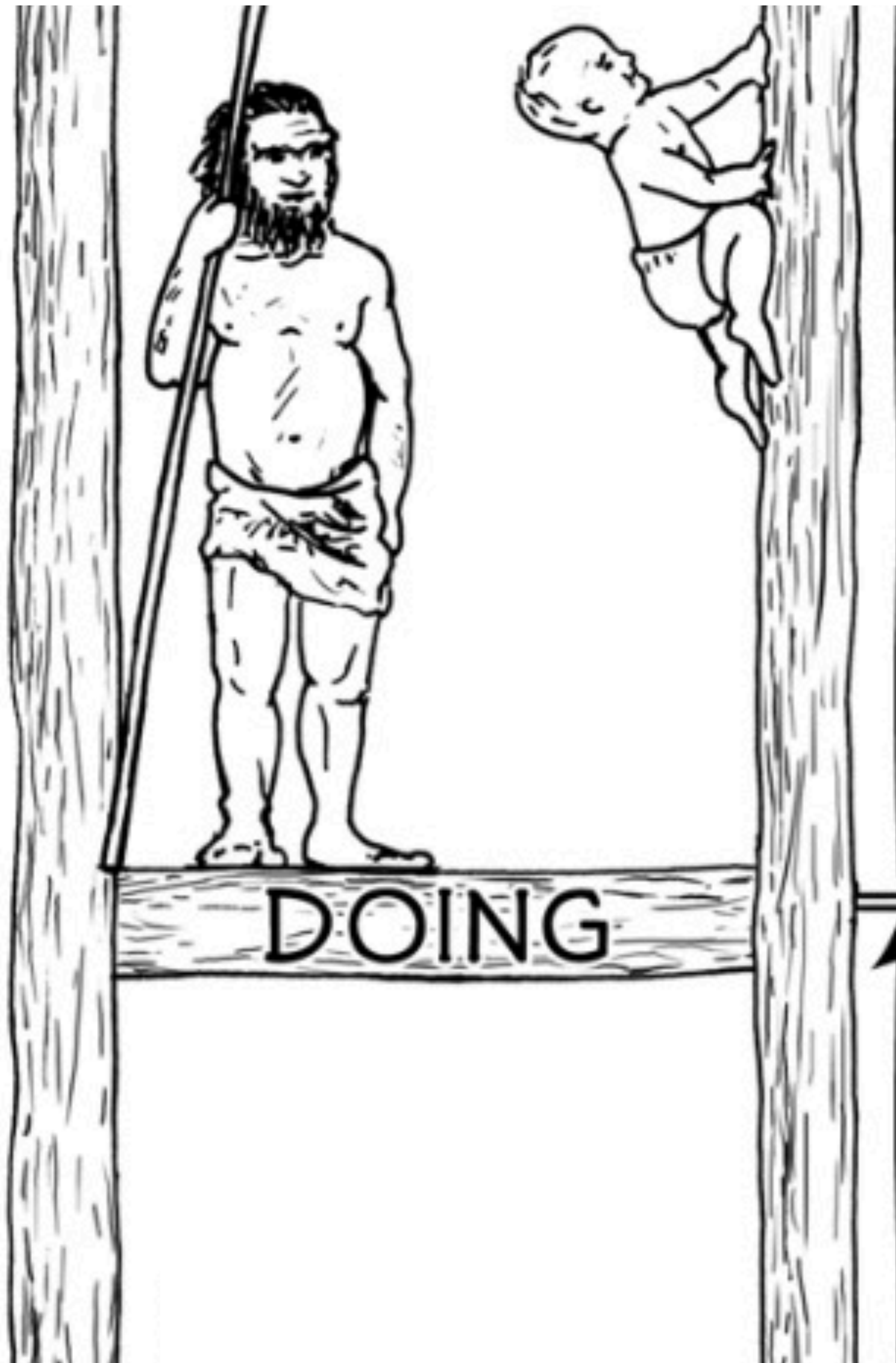
**ACTIVITY:** Seeing, Observing

**QUESTIONS:** *What if I see ...?*  
(How are the variables related?  
How would seeing X change my belief in Y?)

**EXAMPLES:** What does a symptom tell me about a disease?  
What does a survey tell us about the  
election results?



# Intervention



## 2. INTERVENTION

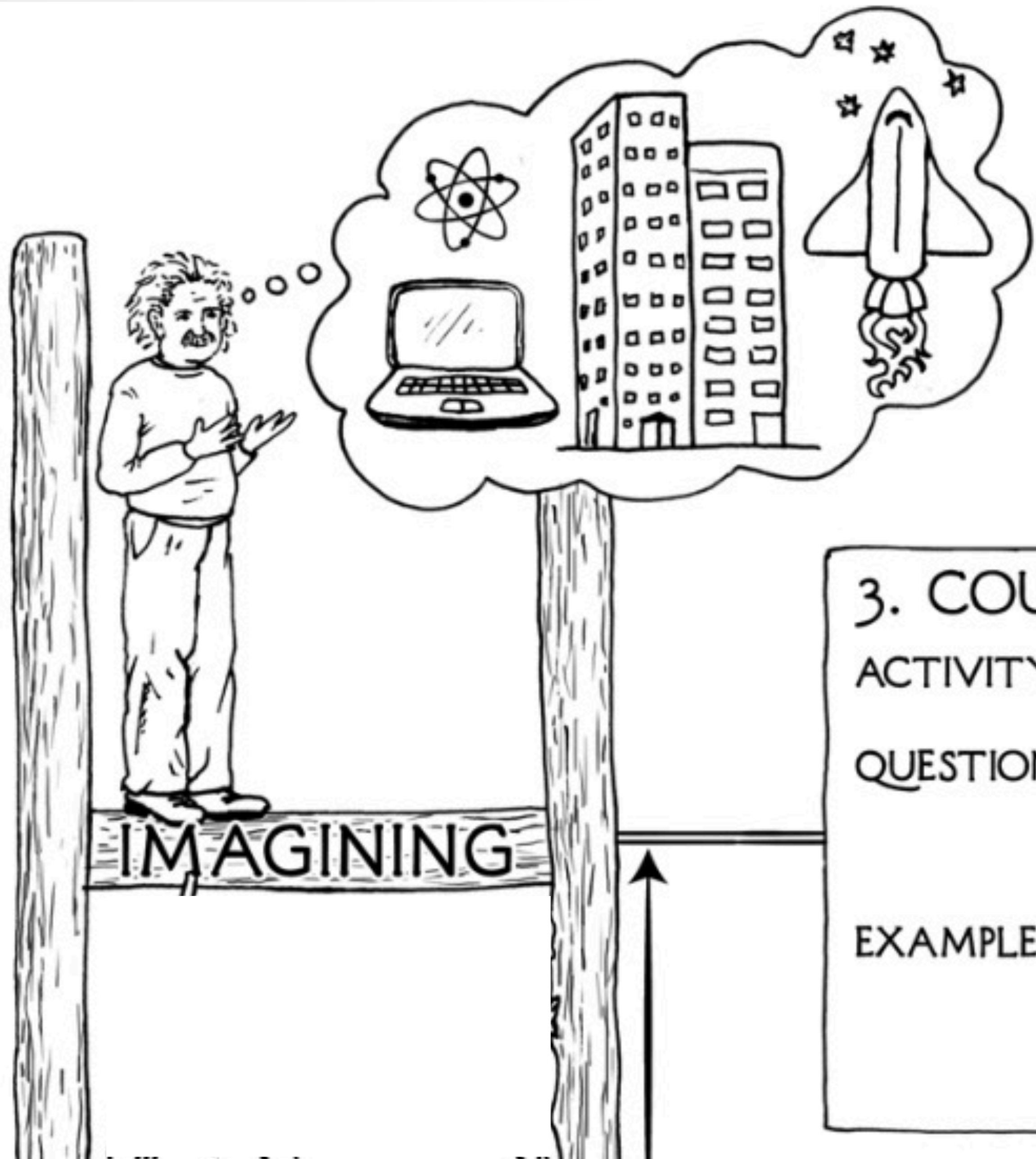
**ACTIVITY:** Doing, Intervening

**QUESTIONS:** *What if I do ...? How?*  
(What would Y be if I do X?  
How can I make Y happen?)

**EXAMPLES:** If I take aspirin, will my headache be cured?  
What if we ban cigarettes?



# Counterfactuals



## 3. COUNTERFACTUALS

**ACTIVITY:** Imagining, Retrospection, Understanding

**QUESTIONS:** *What if I had done ...? Why?*  
(Was it X that caused Y? What if X had not occurred? What if I had acted differently?)

**EXAMPLES:** Was it the aspirin that stopped my headache?  
Would Kennedy be alive if Oswald had not killed him? What if I had not smoked for the last 2 years?



What if we had the right causal structure?

Under the hypothesis of independent mechanisms and small changes across different distributions: smaller sample complexity to recover from a distribution change

for transfer learning, agent learning, domain adaptation,

What if we had the right causal structure?

CLAIM: Under the hypothesis of independent mechanisms and small changes across different distributions:

–smaller sample complexity to recover from a distribution change

- E.g. for transfer learning, agent learning, domain adaptation, etc.

Yoshua Bengio on [arXiv:1901.10912] and public FB discussion



**Max Welling** Isn't this what Bernhard Schoelkopf has been saying for a while?

Like · Reply · 6w



**Yann LeCun** ...and Leon Bottou ?

Like · Reply · 6w



**Leon Bottou** Yoshua's paper says: if you observe a distribution change that comes from a causal effect, then you'll adapt faster if your generative model matches the causal model.

Another way of seeing it is : the right causal graph suggests a particular factorization of the joint distribution (a directed bayesian network). A causal intervention means that you only change one of these factors (or a few factors) while leaving the other ones unchanged. Therefore if your generative model is the right causal model, meaning that it factorizes the joint in the same way, it will be easy to adapt it to the change because only a few parameters need changing (those associated with the factors that actually changed).

Said like this, it feels pretty trivial. Yoshua proposes to use this to infer the right causal model from a plurality of observed distributions.



**Dan Roy** **Max Welling** yes. He's been arguing for generative models with causal structure for years as the way to extract information for rich environments. So not this



**Max Welling** **Dan Roy** I am, and I think most of us, are keenly aware that Josh has been the big proponent of this view. And I think most people agree with him on this view. Integrating this view with deep learning for more narrowly defined tasks seems to me an interesting intellectual pursuit though. I think that's what's happening here but I was not at the talk 😊



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## Causally interpreted Bayesian networks

**Definition:** A DAG  $G = (V, E)$  together with a family  $\kappa_v : \mathbb{X}_{pa(v)} \times \mathbb{X}_v \rightarrow [0, 1]$ ,  $v \in V$ , of Markov kernels is called *Bayesian network*. The interpretation of the Markov kernels  $\kappa_v$  as mechanisms of the nodes implies a causal nature of the Bayesian network.

Consider two distinct nodes  $v, w \in V$ .

- If  $v \rightarrow w$  we call  $v$  a (pot.) *direct cause* of  $w$  and  $w$  a (pot.) *direct effect* of  $v$ .
- If  $v \rightsquigarrow w$  we call  $v$  a (pot.) *cause* of  $w$  and  $w$  an (pot.) *effect* of  $v$ .

```

graph TD
    X1["X1 season"] -- κ1 --> X3["X3 sprinkler"]
    X1 -- κ2 --> X2["X2 rain"]
    X3 -- κ3 --> X4["X4 wet"]
    X2 -- κ4 --> X4
    X4 -- κ5 --> X5["X5 slippery"]
        
```

```

graph TD
    U["φU genotype"] -- α --> A["A smoking"]
    U -- γ --> C["C cancer"]
    A -- β --> B["B tar"]
    B -- γ --> C
        
```

• J. Pearl. *Causality: Models, Reasoning, and Inference*. Cambridge University Press 2000, 2009.

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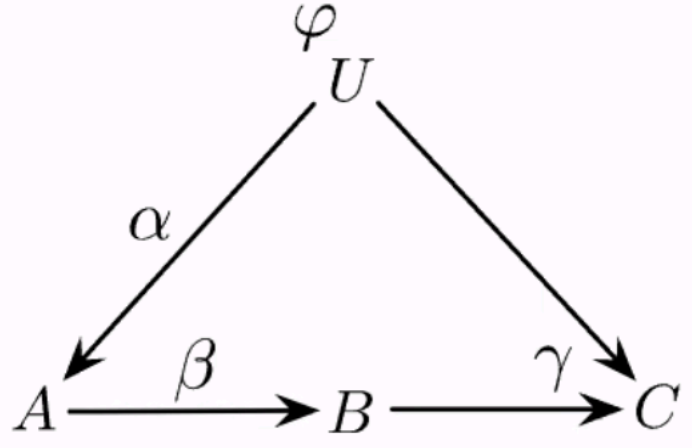
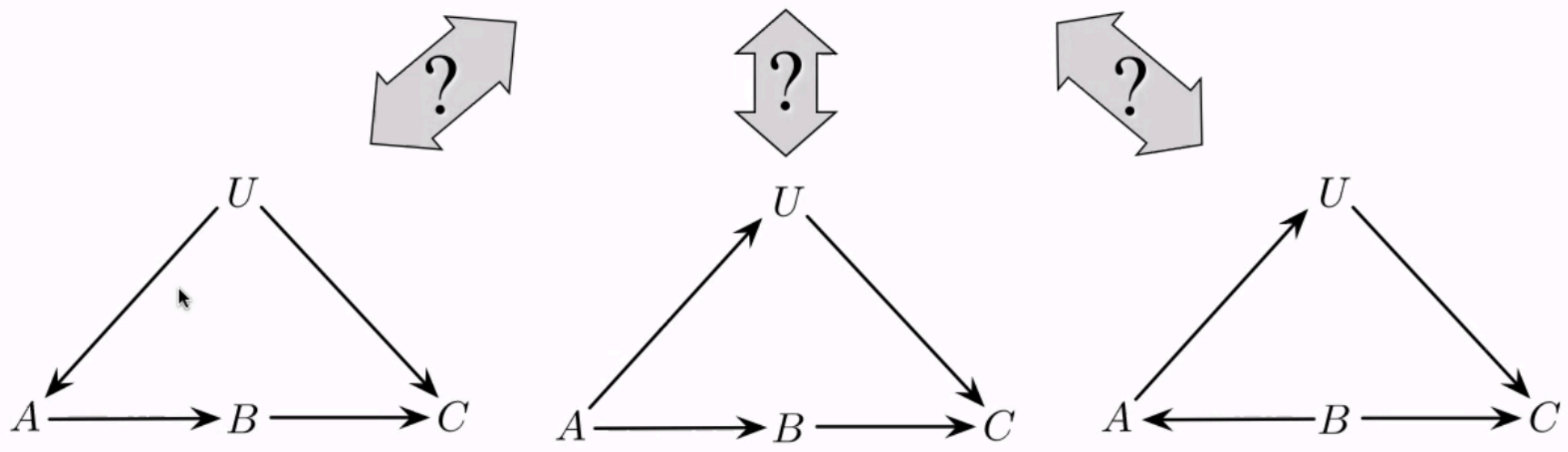
# Markov Equivalence

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## The data generating process


$$p(u, a, b, c) = \varphi(u) \alpha(u; a) \beta(a; b) \gamma(u, b; c)$$
$$B \perp\!\!\!\perp U \mid A, \quad C \perp\!\!\!\perp A \mid B, U$$


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# A toy example

Ferenc Huszár

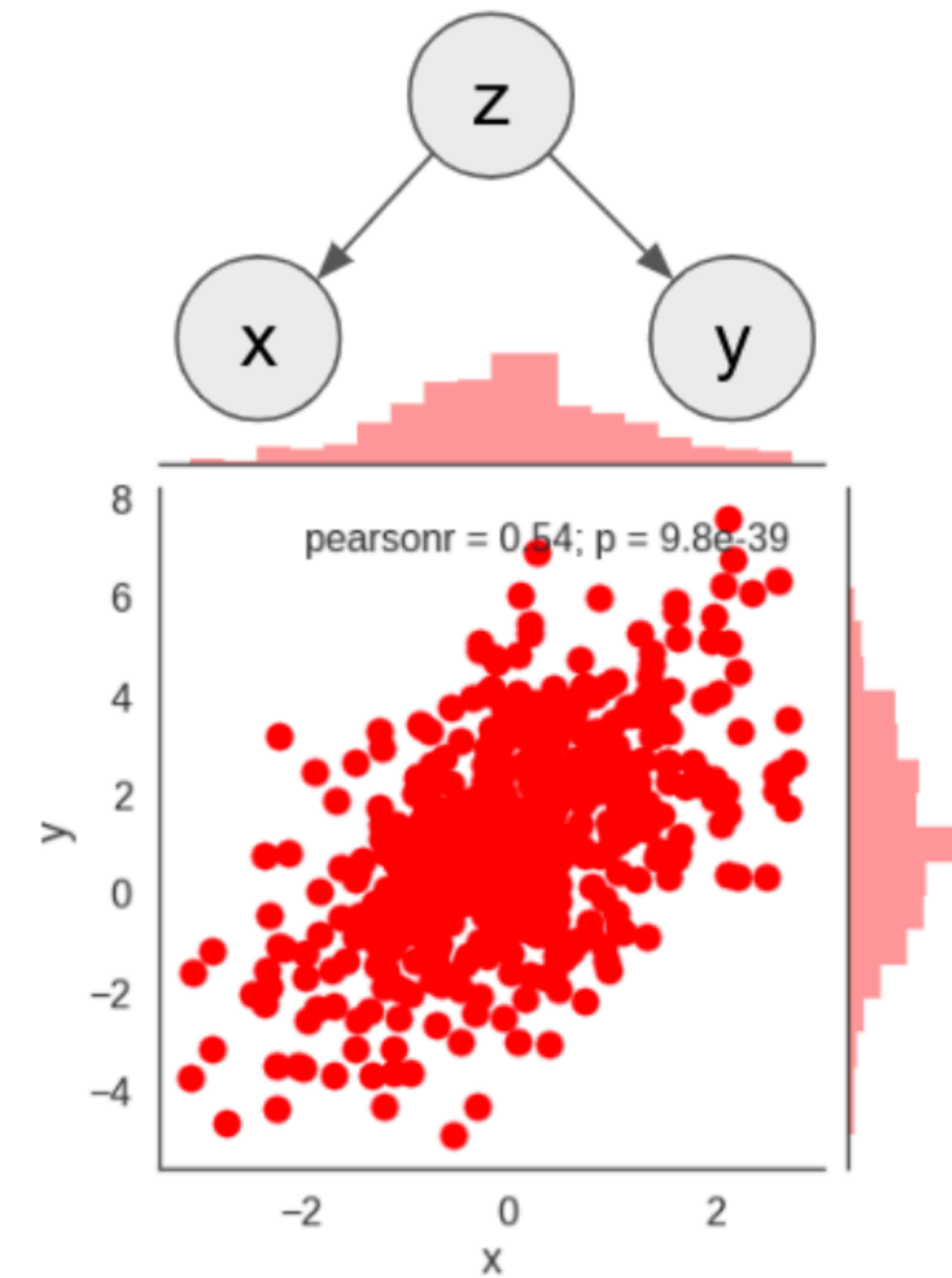
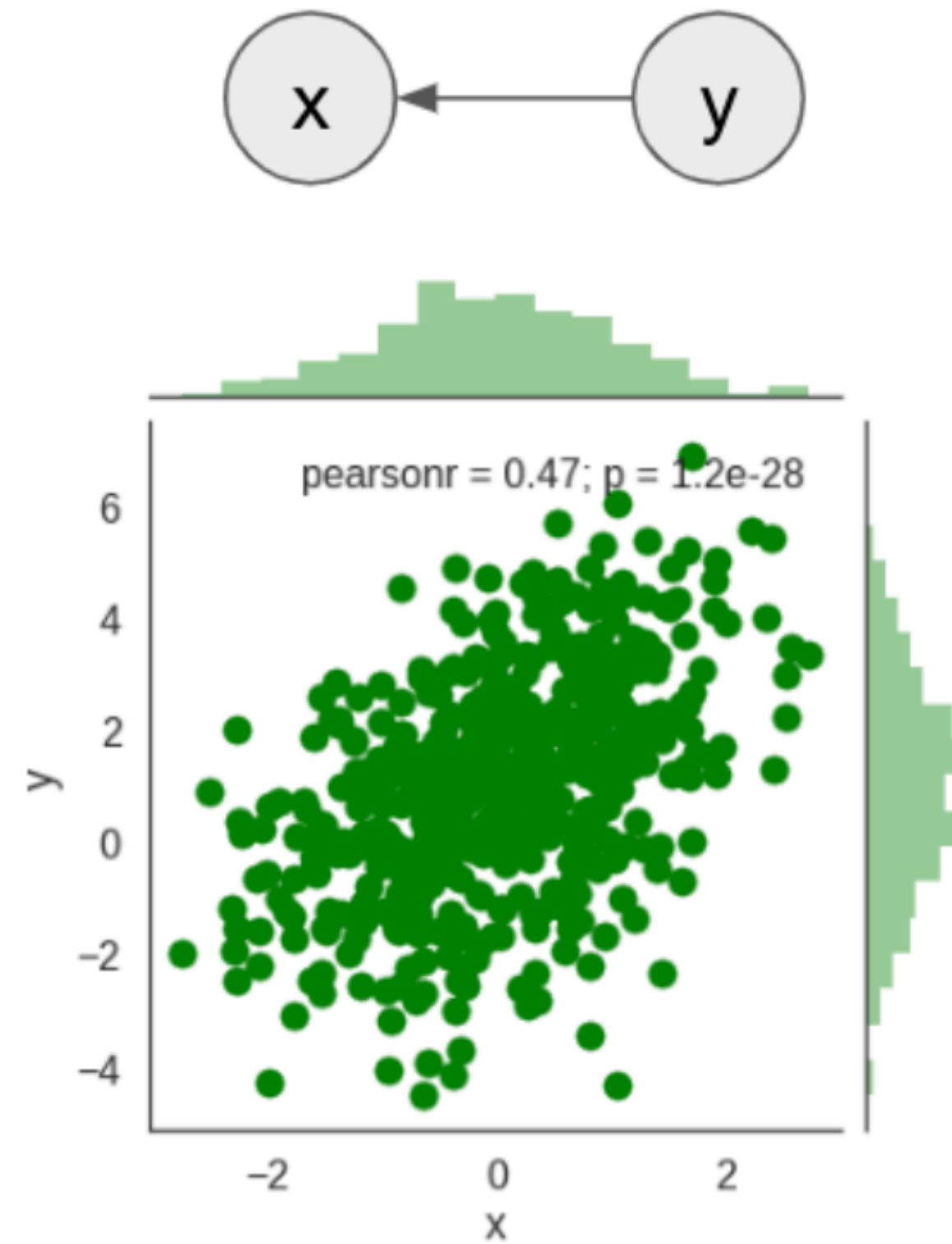
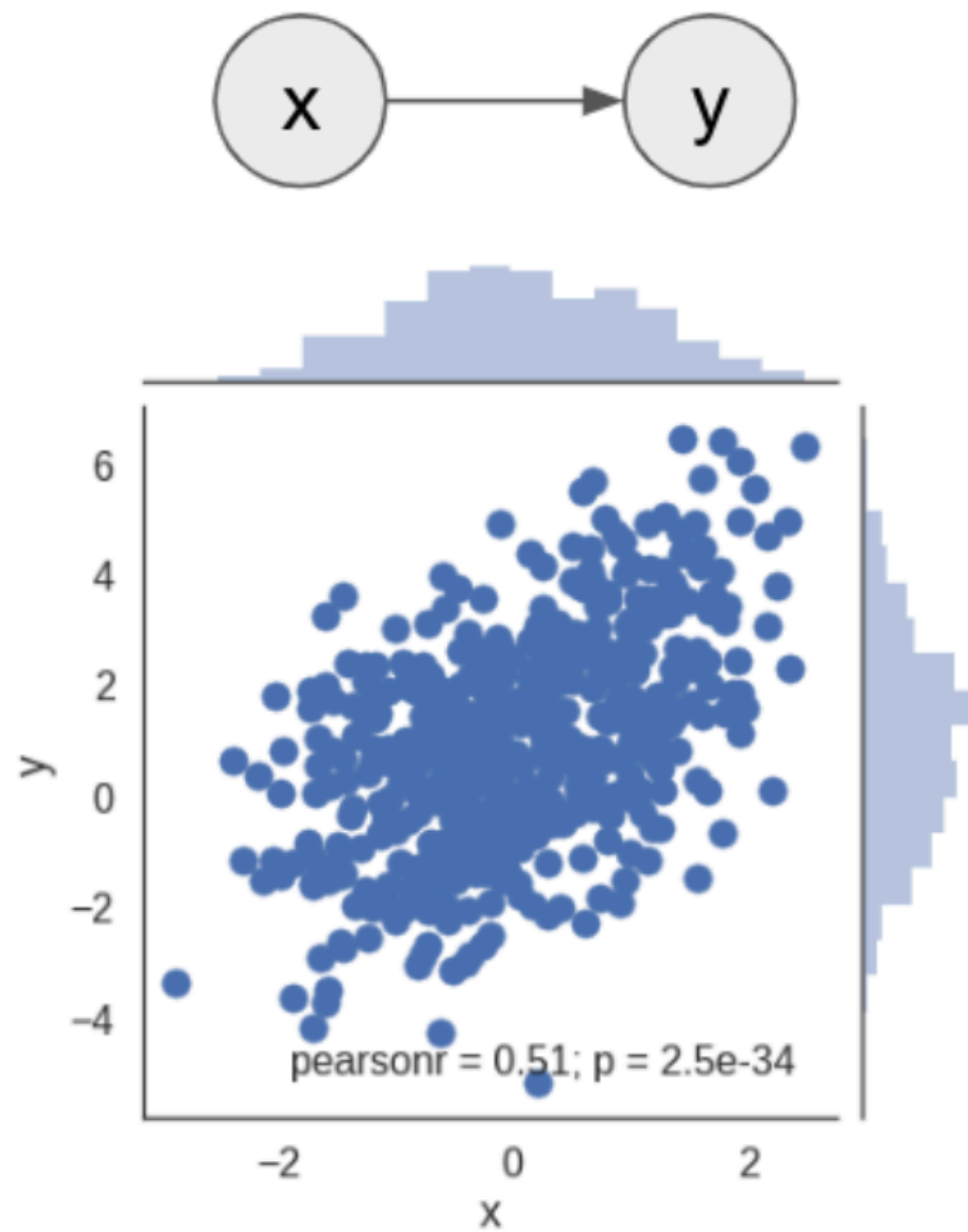


inFERENCE

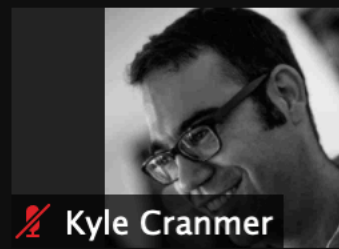
```
x = randn()  
y = x + 1 + sqrt(3)*randn()
```

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

```
z = randn()  
y = z + 1 + sqrt(3)*randn()  
x = z
```







Priyamvada N...



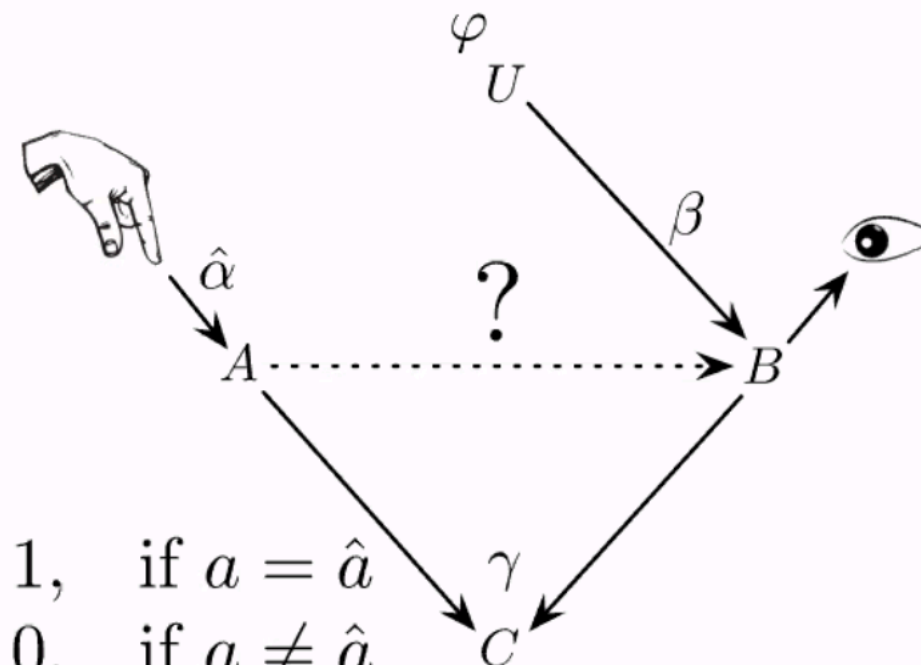
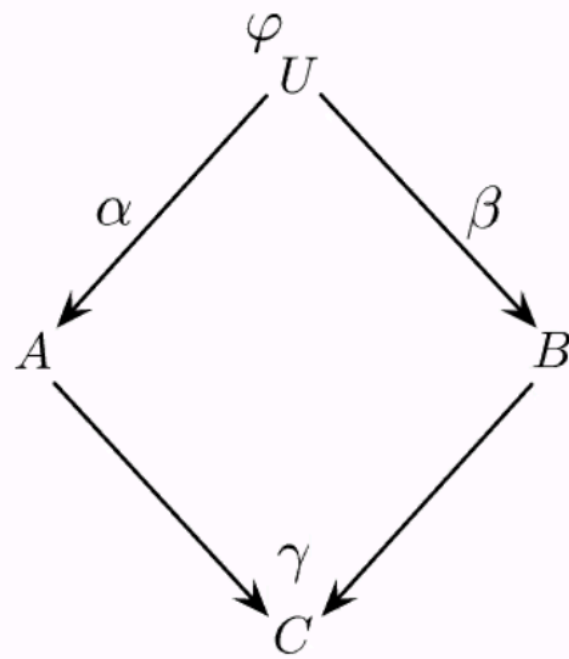
Ty Kamp



View

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# Intervention and the *do*-operation



$$\hat{\alpha}(a) := \begin{cases} 1, & \text{if } a = \hat{a} \\ 0, & \text{if } a \neq \hat{a} \end{cases}$$

$$p(u, a, b, c) = \varphi(u) \alpha(u; a) \beta(u; b) \gamma(a, b; c)$$

$$p(u, a, b, c | do(\hat{a})) := \varphi(u) \hat{\alpha}(a) \beta(u; b) \gamma(a, b; c)$$

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## Intervention and the *do*-operation

$$\begin{aligned}
 p(b | do(\hat{a})) &= \sum_{u,a,c} p(u, a, b, c | do(\hat{a})) \\
 &= \sum_{u,a,c} \varphi(u) \hat{\alpha}(a) \beta(u; b) \gamma(a, b; c) \\
 &= \sum_{u,c} \varphi(u) \beta(u; b) \gamma(\hat{a}, b; c) \\
 &= \sum_u \varphi(u) \beta(u; b) \sum_c \gamma(\hat{a}, b; c) \\
 &= p(b) \neq p(b | \hat{a})
 \end{aligned}$$

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## Intervention and the *do*-operation

$$\begin{aligned}
 p(c | do(\hat{u})) &= \sum_{u,a,b} p(u, a, b, c | do(\hat{u})) \\
 &= \sum_{u,a,b} \hat{\varphi}(u) \alpha(u; a) \beta(u; b) \gamma(a, b; c) \\
 &= \sum_{a,b} \alpha(\hat{u}; a) \beta(\hat{u}; b) \gamma(a, b; c) \\
 &= \frac{\sum_{a,b} \varphi(\hat{u}) \alpha(\hat{u}; a) \beta(\hat{u}; b) \gamma(a, b; c)}{\varphi(\hat{u})} \\
 &= \frac{\sum_{a,b} p(\hat{u}, a, b, c)}{p(\hat{u})} \\
 &= p(c | \hat{u})
 \end{aligned}$$

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## When do they coincide?

**Theorem:** Let  $G = (V, E)$  be a DAG, and let  $A$  and  $B$  be two disjoint subsets of  $V$ . Then the following two statements are equivalent:

- (1) For every Bayesian network  $\mathfrak{B}$  with underlying DAG  $G$  we have
 
$$p(x_B | do(x_A)) = p(x_B | x_A).$$
- (2)  $B$  is not a cause of  $A$  and there is no common cause of  $A$  and  $B$ .

$$\begin{aligned}
 p(b | do(a)) &= p(b | a) \\
 p(c | do(b)) &\neq p(c | b) \\
 p(c | do(a)) &\neq p(c | a)
 \end{aligned}$$

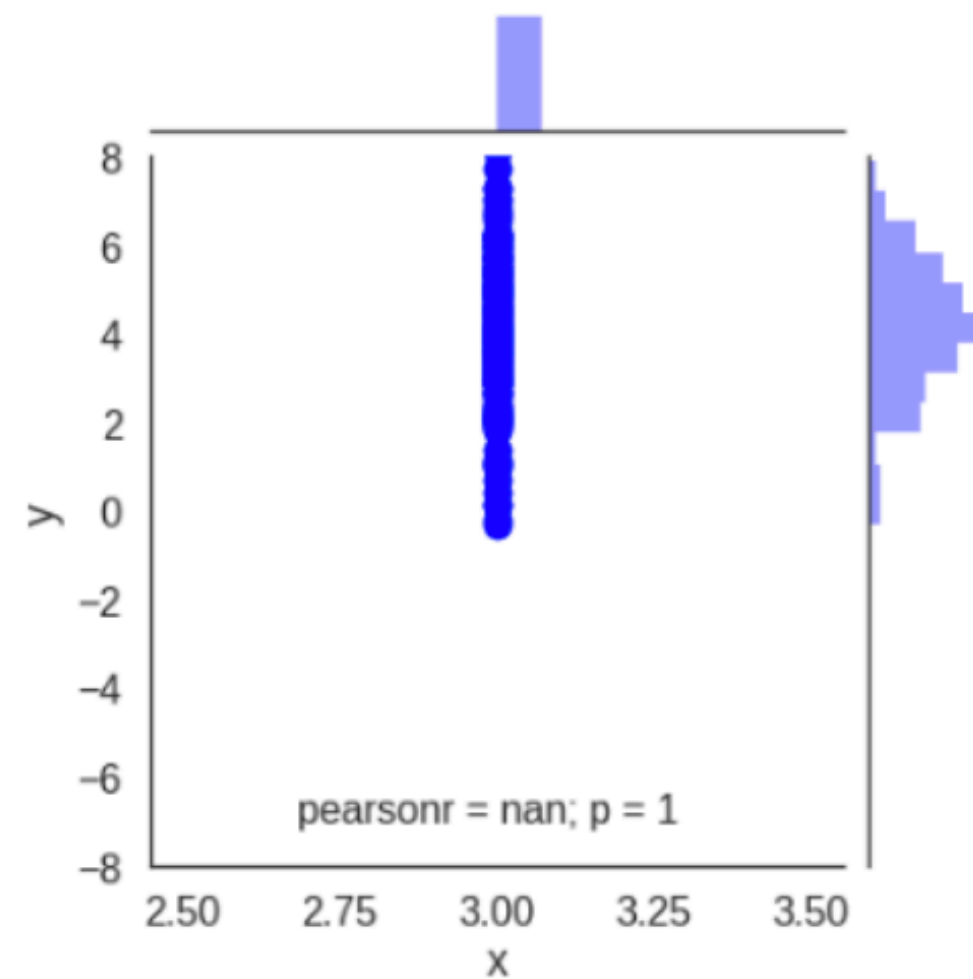


# A toy example



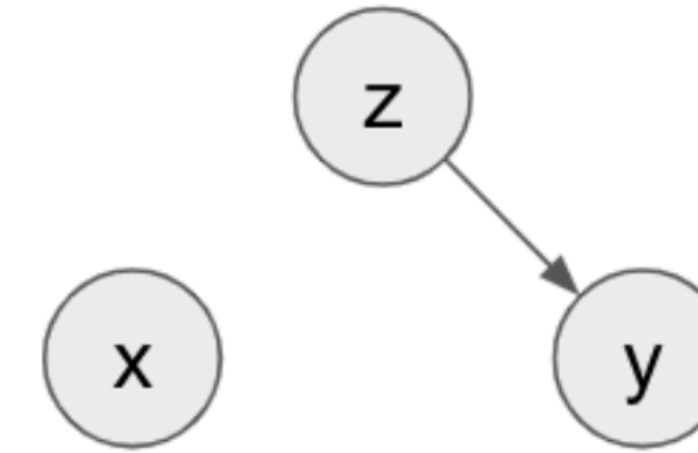
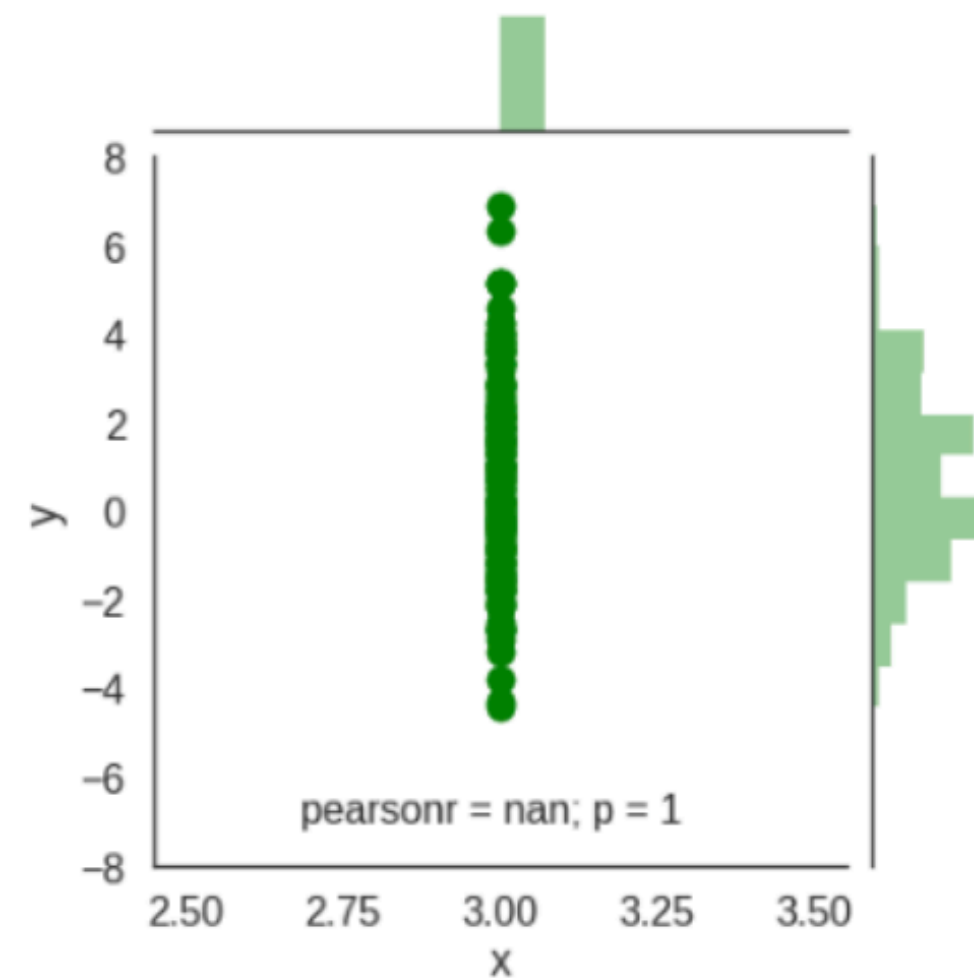
$$P(y|do(X)) = p(y|x)$$

```
x = randn()
x = 3
y = x + 1 + sqrt(3)*randn()
x = 3
```



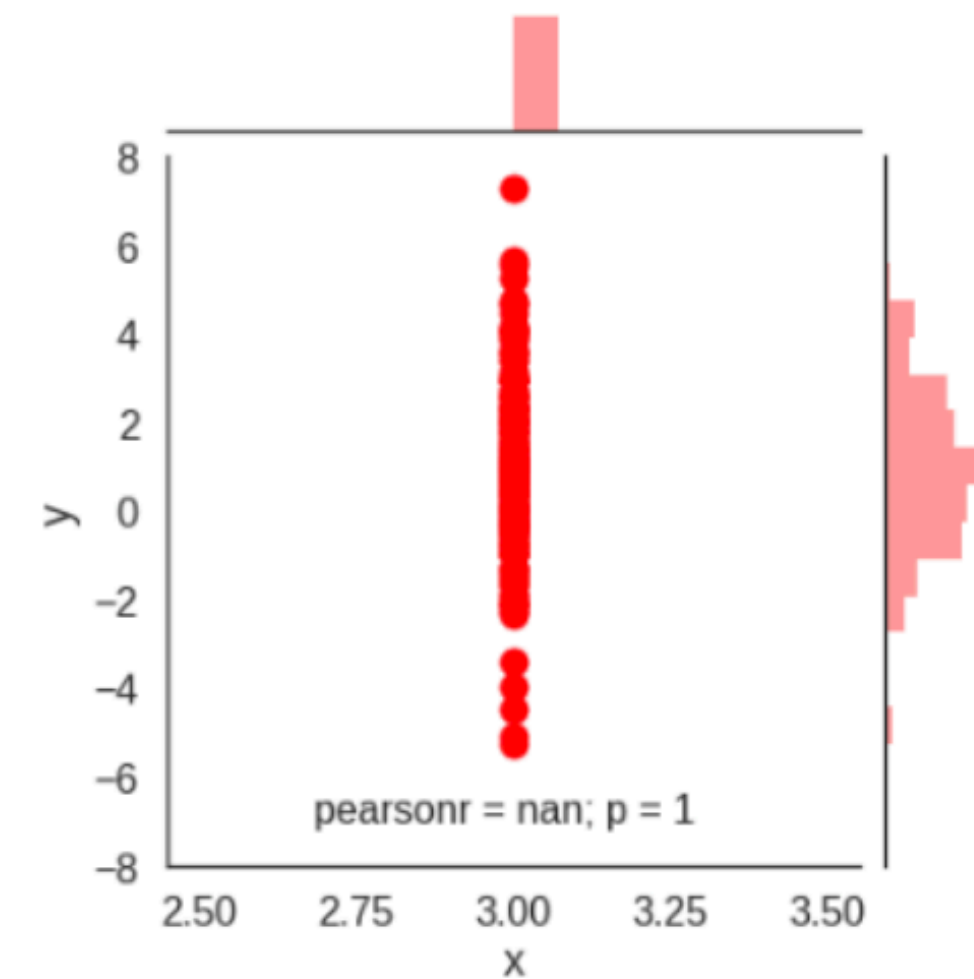
$$P(y|do(X)) = p(y)$$

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



$$P(y|do(X)) = p(y)$$

```
z = randn()
x = 3
x = z
x = 3
y = z + 1 + sqrt(3)*randn()
x = 3
```





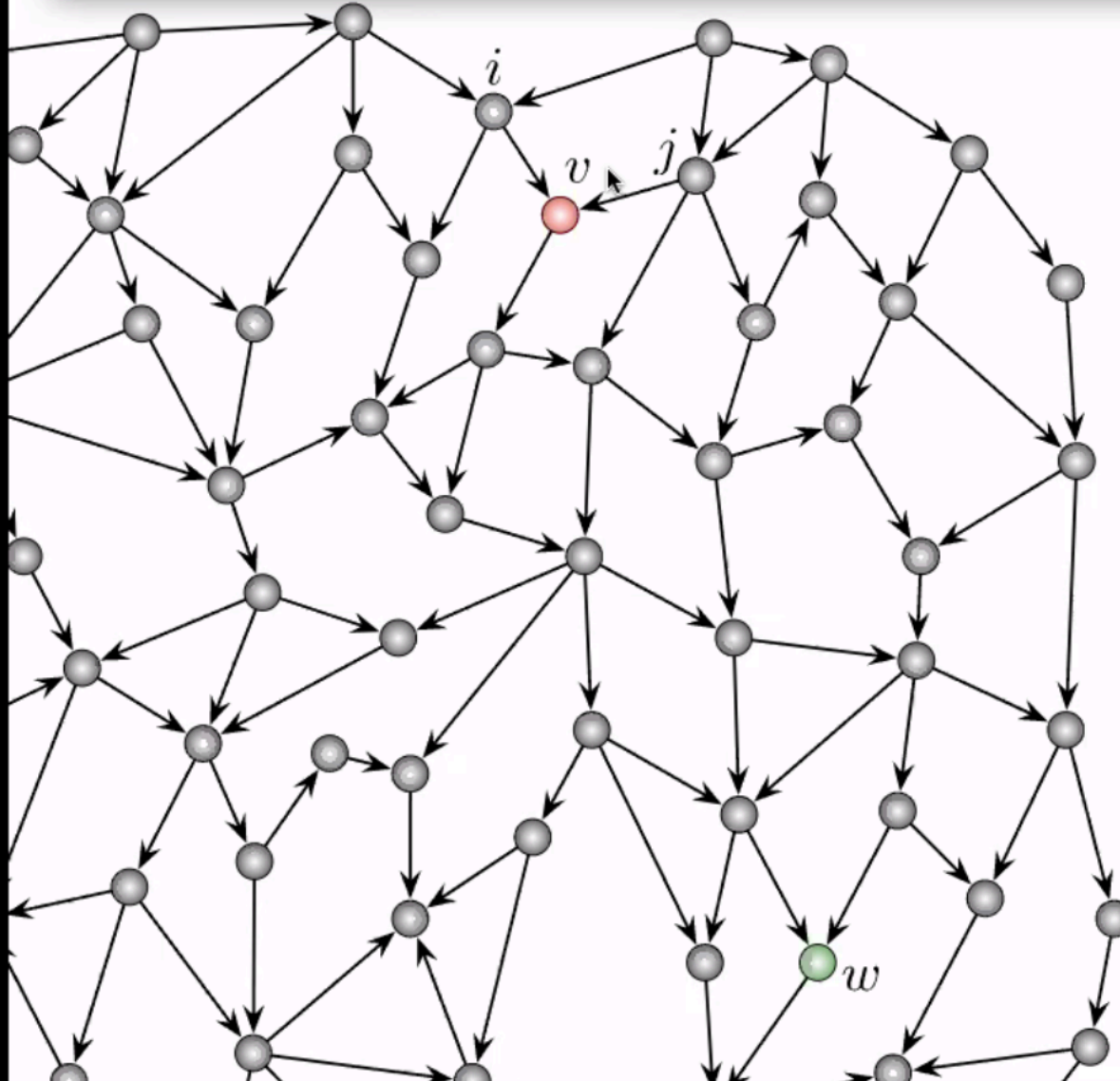
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# Identifiability of causal effects

**Theorem:** Let  $\mathcal{B}$  be a Bayesian network with DAG  $G = (V, E)$ . For two distinct nodes  $v$  and  $w$  the following holds:

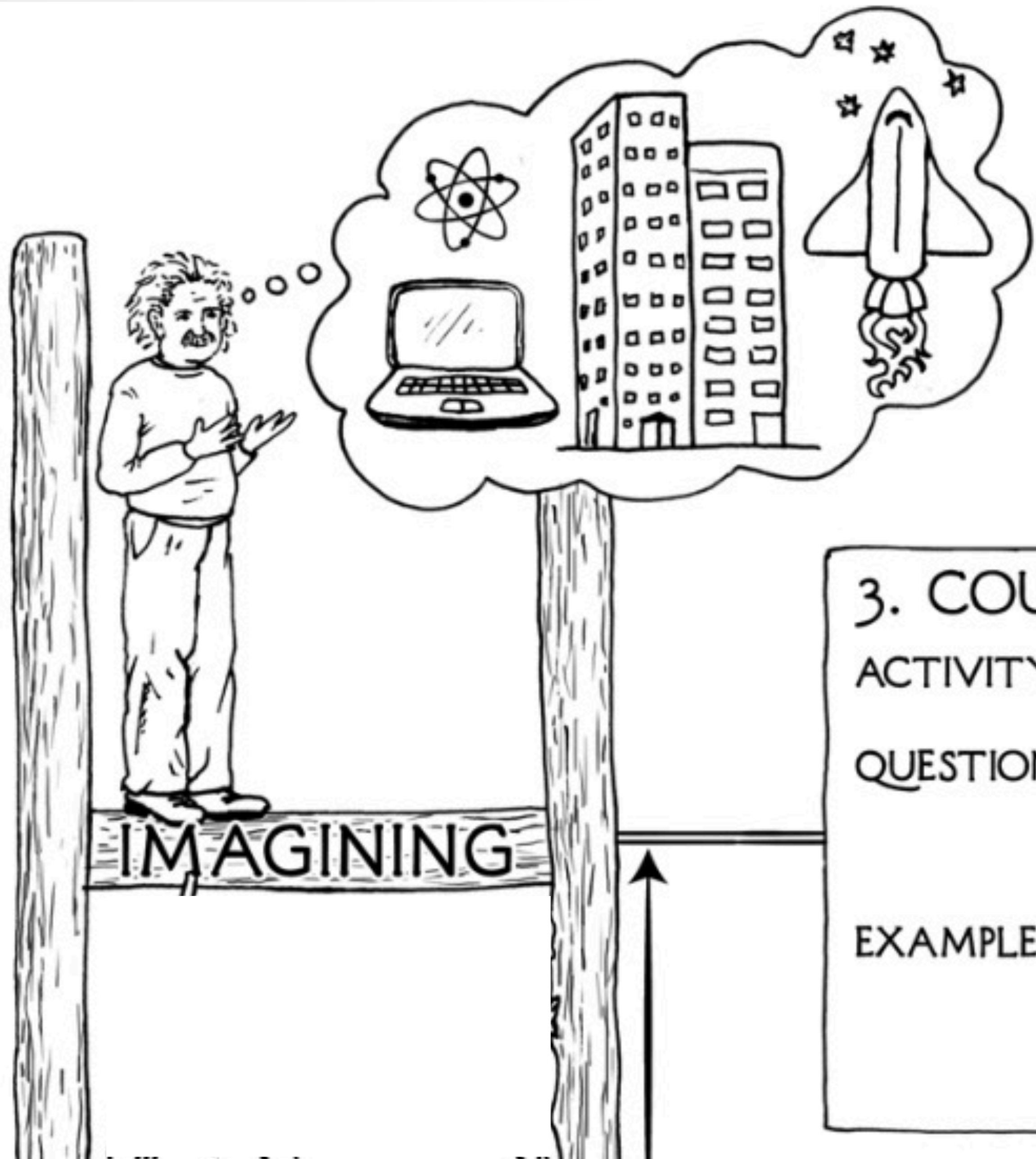
$$p(x_w | do(x_v)) = \sum_{x_{pa(v)}} p(x_{pa(v)}) p(x_w | x_v, x_{pa(v)}).$$


$$p(x_w | do(x_v)) = \sum_{x_i, x_j} p(x_i, x_j) p(x_w | x_v, x_i, x_j)$$

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# Counterfactuals



## 3. COUNTERFACTUALS

**ACTIVITY:** Imagining, Retrospection, Understanding

**QUESTIONS:** *What if I had done ...? Why?*  
(Was it X that caused Y? What if X had not occurred? What if I had acted differently?)

**EXAMPLES:** Was it the aspirin that stopped my headache?  
Would Kennedy be alive if Oswald had not killed him? What if I had not smoked for the last 2 years?



# Counterfactuals

## Example 1: David Blei's election example

This is an example David brought up during the **Causality Panel** and I referred back to this in my talk. I'm including it here for the benefit of those who attended my MLSS talk:

*Given that Hilary Clinton did not win the 2016 presidential election, and given that she did not visit Michigan 3 days before the election, and given everything else we know about the circumstances of the election, what can we say about the probability of Hilary Clinton winning the election, had she visited Michigan 3 days before the election?*

Let's try to unpack this. We are interested in the probability that:

- she *hypothetically* wins the election

conditioned on four sets of things:

- she lost the election
- she did not visit Michigan
- any other relevant an observable facts
- she *hypothetically* visits Michigan

It's a weird beast: you're simultaneously conditioning on her visiting Michigan and not visiting Michigan. And you're interested in the probability of her winning the election given that she did not. WHAT?

Why would quantifying this probability be useful? Mainly for credit assignment. We want to know why she lost the election, and to what degree the loss can be attributed to her failure to visit Michigan three days before the election. Quantifying this is useful, it can help political advisors make better decisions next time.



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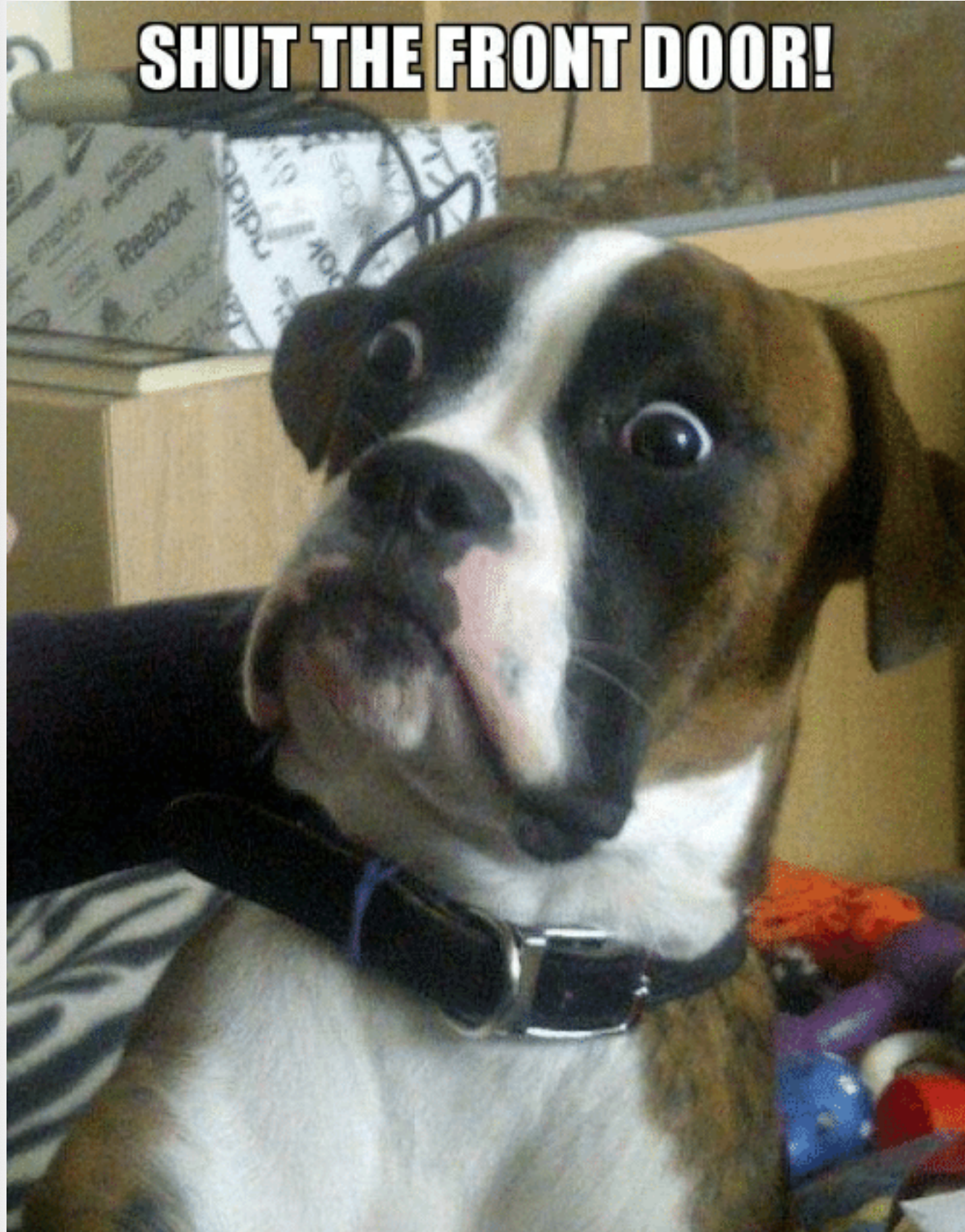
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# Identifiability of causal effects (front-door example)

$$\begin{aligned}
 p(c | do(a)) &= \sum_{u,b} \varphi(u) \beta(a; b) \gamma(u, b; c) \\
 &= \sum_{u,b} p(u) p(b | a) p(c | u, b) \\
 &= \sum_{u,b} \left( \sum_{a'} p(a') p(u | a') \right) p(b | a) p(c | u, b) \\
 &= \sum_b p(b | a) \sum_{a'} p(a') \sum_u p(u | a') p(c | u, b) \\
 &= \sum_b p(b | a) \sum_{a'} p(a') \sum_u p(u | a', b) p(c | u, a', b) \\
 &= \sum_b p(b | a) \sum_{a'} p(a') p(c | a', b)
 \end{aligned}$$

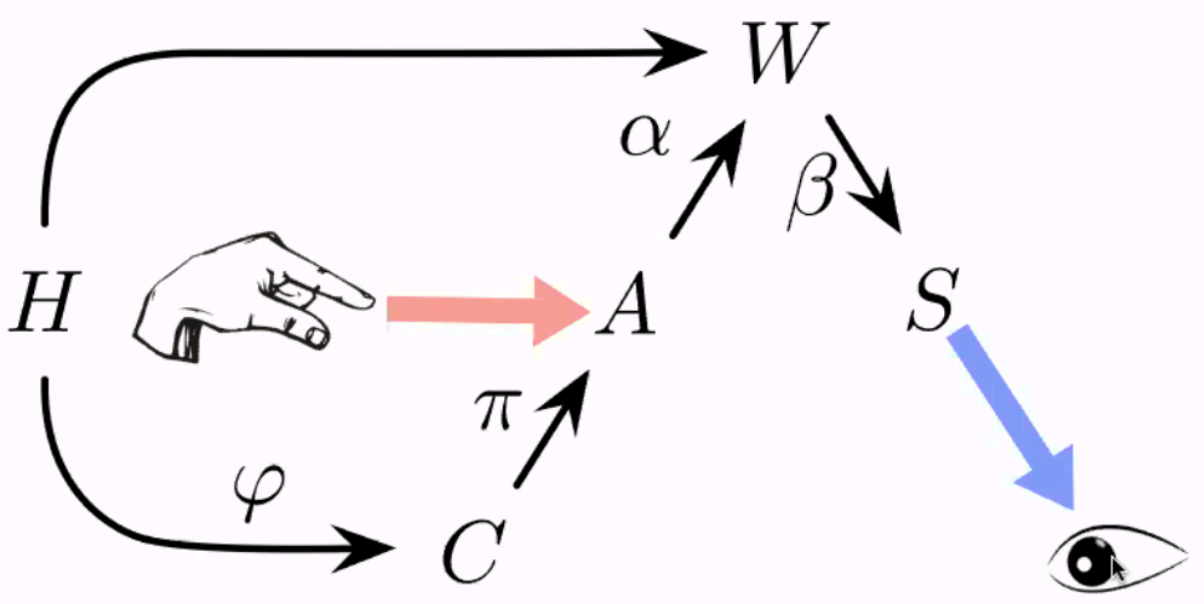
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# In robotics

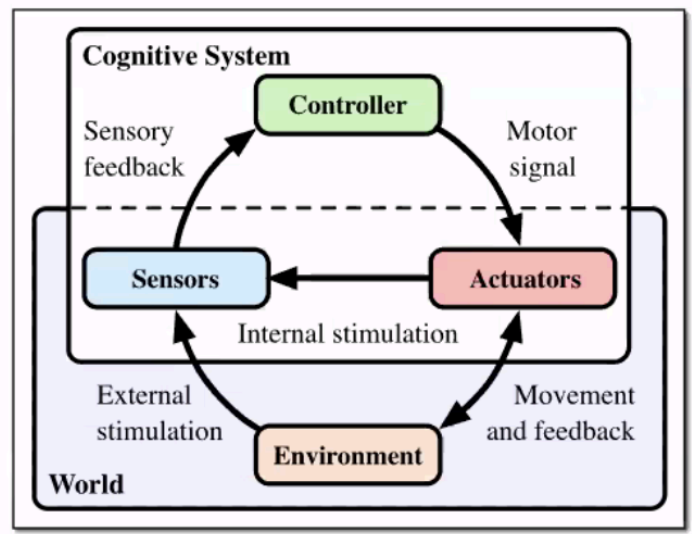
# Causal effects in the sensorimotor loop



$$p(s | do(a)) = \sum_c p(s | c, a) p(c)$$

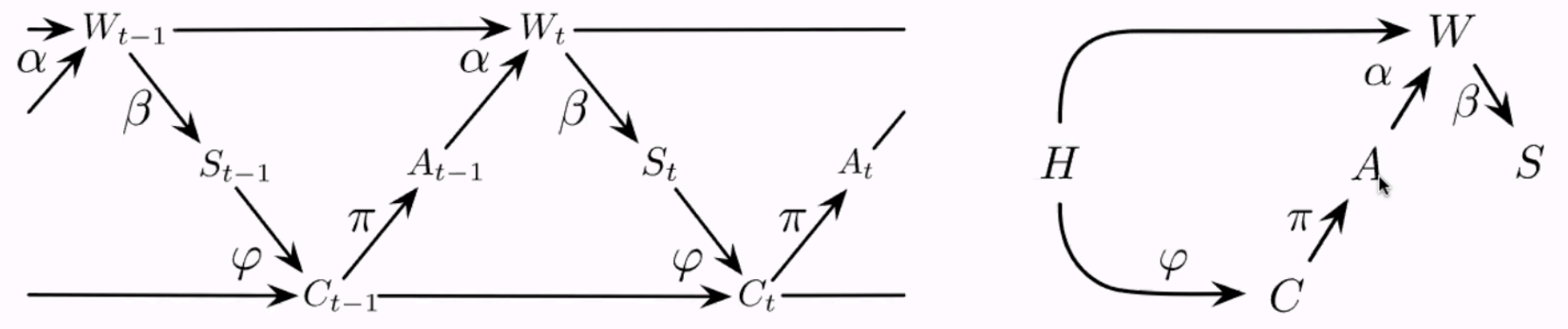
- N. Ay, K. Zahedi. *On the Causal Structure of the Sensorimotor Loop*. GSO 2014.
- N. Ay, K. Zahedi. *An Information-Theoretic Approach to Prediction and Deliberative Decision Making of Embodied Systems*. Proc. of ICCN 2011. Advances in Cognitive Neurodynamics, Springer 2012.

## The sensorimotor loop



by Keyan-Ghazi Zahedi

• R. Pfeifer, M. Lungarella, F. Iida. *Self-Organization, Embodiment, and Biologically Inspired Robotics*. Science 2007.



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## Causally Correct Partial Models for Reinforcement Learning

Danilo J. Rezende<sup>\*1</sup> Ivo Danihelka<sup>\*1,2</sup> George Papamakarios<sup>1</sup> Nan Rosemary Ke<sup>3</sup> Ray Jiang<sup>1</sup>  
 Theophane Weber<sup>1</sup> Karol Gregor<sup>1</sup> Hamza Merzic<sup>1</sup> Fabio Viola<sup>1</sup> Jane Wang<sup>1</sup> Jovana Mitrovic<sup>1</sup>  
 Frederic Besse<sup>1</sup> Ioannis Antonoglou<sup>1,2</sup> Lars Buesing<sup>1</sup>

### Causally Correct Partial Models

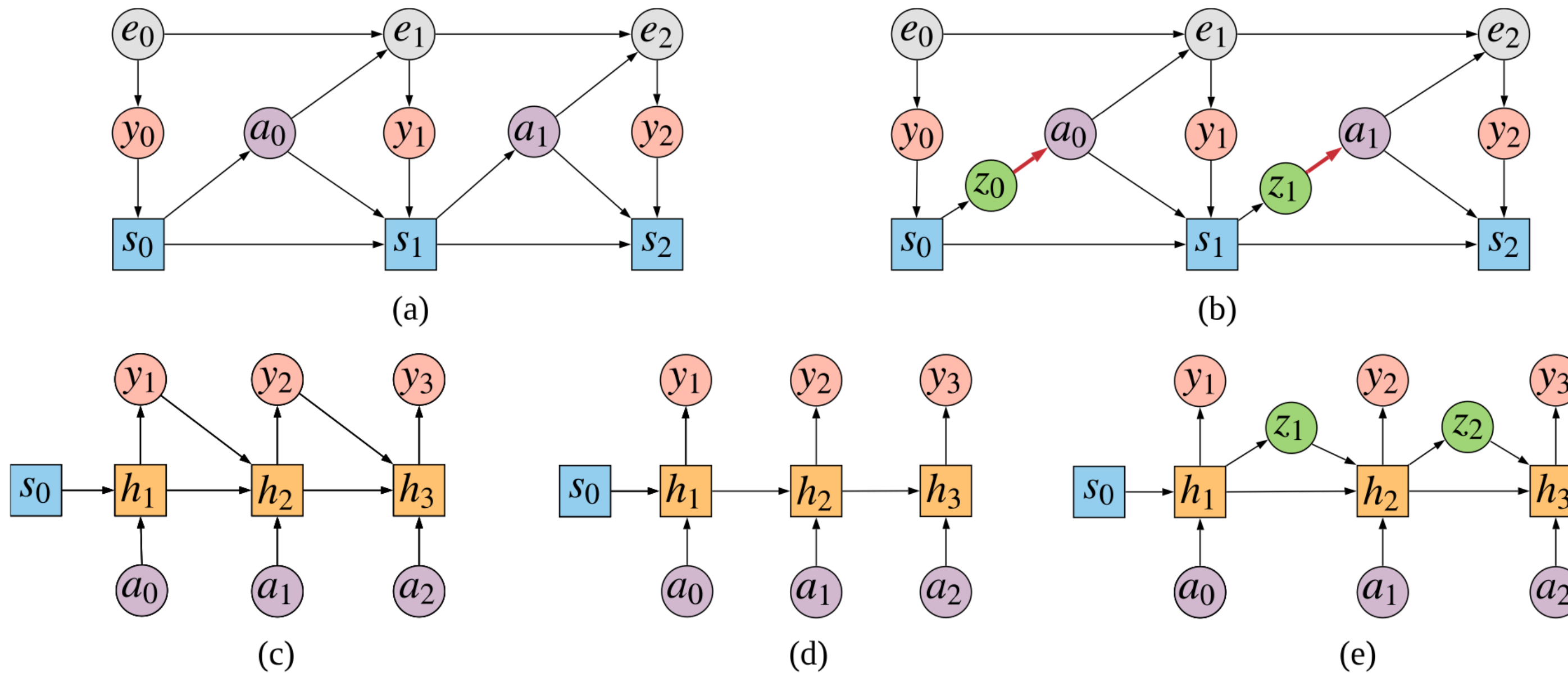


Figure 3. Graphical representations of the environment, the agent, and the various models. Circles are stochastic nodes, rectangles are deterministic nodes. (a) Agent interacting with the environment, generating a trajectory  $\{y_t, a_t\}_{t=0}^T$ . These trajectories are the training data for the models. (b) Same as (a) but also including the backdoor  $z_t$  in the generated trajectory. The red arrows indicate the locations of the interventions. (c) Standard autoregressive generative model of observations. The model predicts the observation  $y_t$  which it then feeds into  $h_{t+1}$ . (d) Example of a Non-Causal Partial Model (NCPM) that predicts the observation  $y_t$  without feeding it into  $h_{t+1}$ . (e) Proposed Causal Partial Model (CPM), with a backdoor  $z_t$  for the actions.