The Blessings of Multiple Causes

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Joint work with Yixin Wang

- The deconfounder
- Academic debate about the deconfounder
- Open research problems for the deconfounder

A frivolous causal inference problem

- Data about movies: casts and revenue
- Goal: Understand the **causal effect** of putting an actor in a movie
- Causal: “What will the revenue be if we make a movie with a particular cast?”

The naive solution

Title	Cast	Revenue
Avatar	{Sam Worthington, Zoe Saldana, Sigourney Weaver, Stephen Lang, … }	$2788M
Titanic	{Kate Winslet, Leonardo DiCaprio, Frances Fisher, Billy Zane, … }	$1845M
The Avengers	{Robert Downey Jr., Chris Evans, Mark Ruffalo, Chris Hemsworth, … }	$1520M
Jurassic World	{Chris Pratt, Bryce Dallas Howard, Irrfan Khan, Vincent D'Onofrio, … }	$1514M
Furious 7	{Vin Diesel, Paul Walker, Dwayne Johnson, Michelle Rodriguez, … }	$1506M
Avengers: Age of Ultron	{Robert Downey Jr., Chris Hemsworth, Mark Ruffalo, Chris Evans, … }	$1405M
Frozen	{Kristen Bell, Idina Menzel, Jonathan Groff, Josh Gad, … }	$1274M
Iron Man 3	{Robert Downey Jr., Gwyneth Paltrow, Don Cheadle, Guy Pearce, … }	$1215M
Minions	{Sandra Bullock, Jon Hamm, Michael Keaton, Allison Janney, … }	$1157M
Captain America: Civil War	{Chris Evans, Robert Downey Jr., Scarlett Johansson, Sebastian Stan, … }	$1153M
⋮	⋮	⋮

- Naive solution: Fit a regression (or use deep learning)
- Actors are features; revenue is the response
- Estimates revenue as a function of which actors are cast

The naive solution

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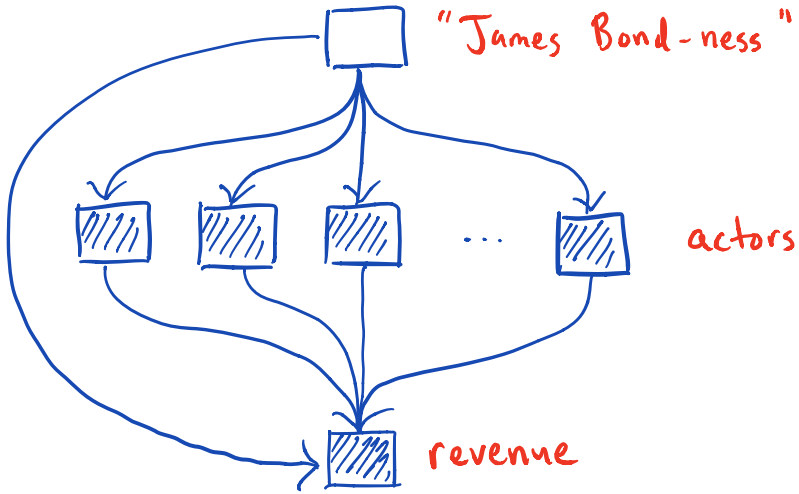
- But standard ML does not (necessarily) provide causal inferences
- Whether an *actor was cast* is different from *casting an actor*
- Causal inference is about **prediction under intervention**

[Hernan and Robins 2019; Imbens and Rubin 2015; Pearl 2009]


Metro Goldwyn Mayer
TRADE
MARK

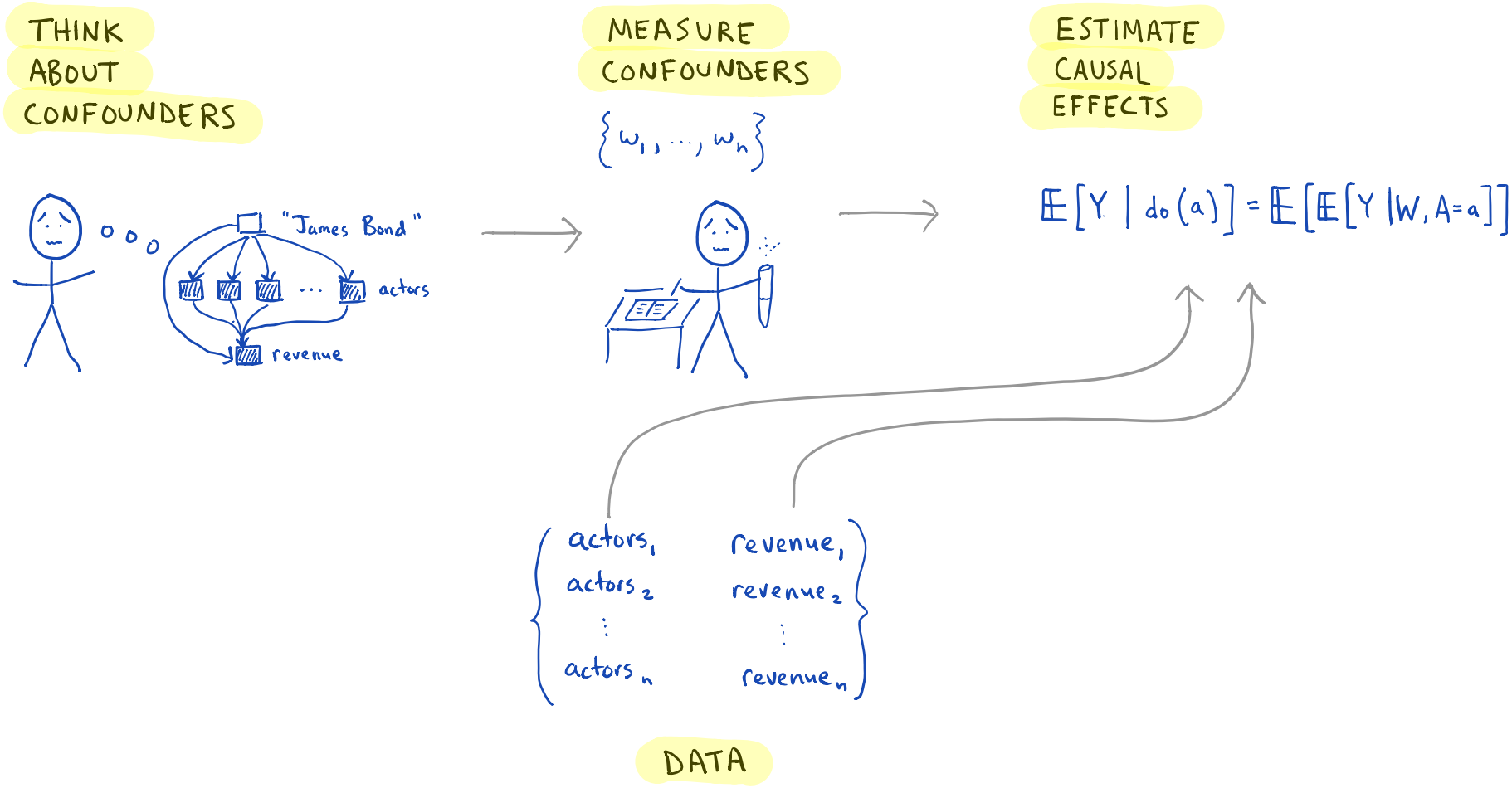

- James Bond movies are about James Bond, a British spy
- Cast James Bond, M, Q, Ms. Moneypenny
- M, Q, Ms Moneypenny only appear in Bond movies
- Bond movies always do well at the box office

The naive solution



- James Bond-ness is an **unobserved confounder**.
- Confounders affect both the cast (“causes”) and the revenue (“effect”)
- Confounders bias “passive ML,” when used to predict interventions.
  - Some actors overestimated; others are underestimated

The classical solution



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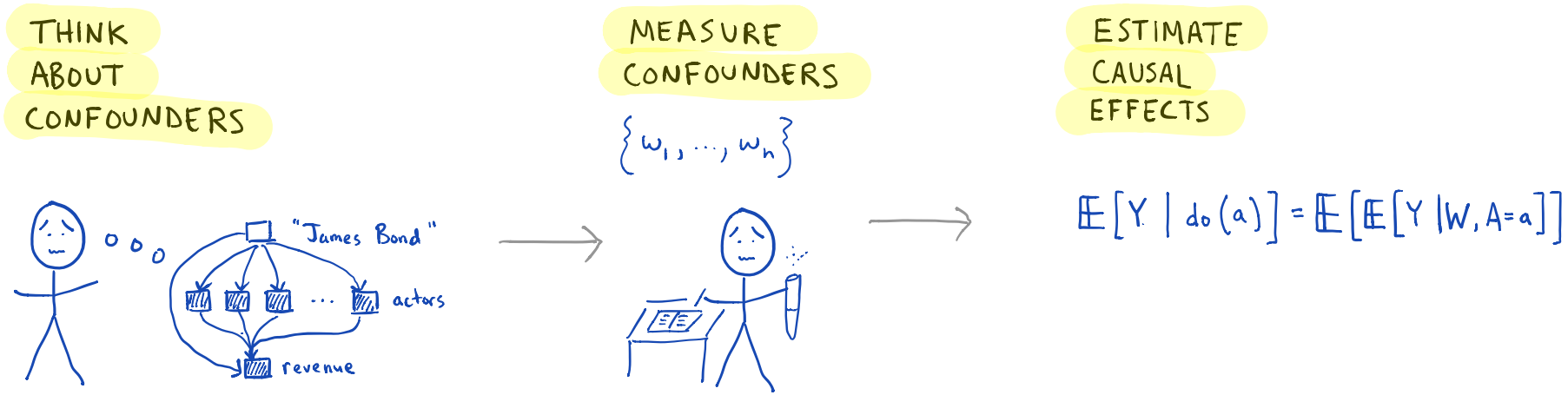

- This approach assumes that we measure **sufficient confounders**.
- But this assumption is **untestable**. [Imbens and Rubin 2015]

Multiple causal inference

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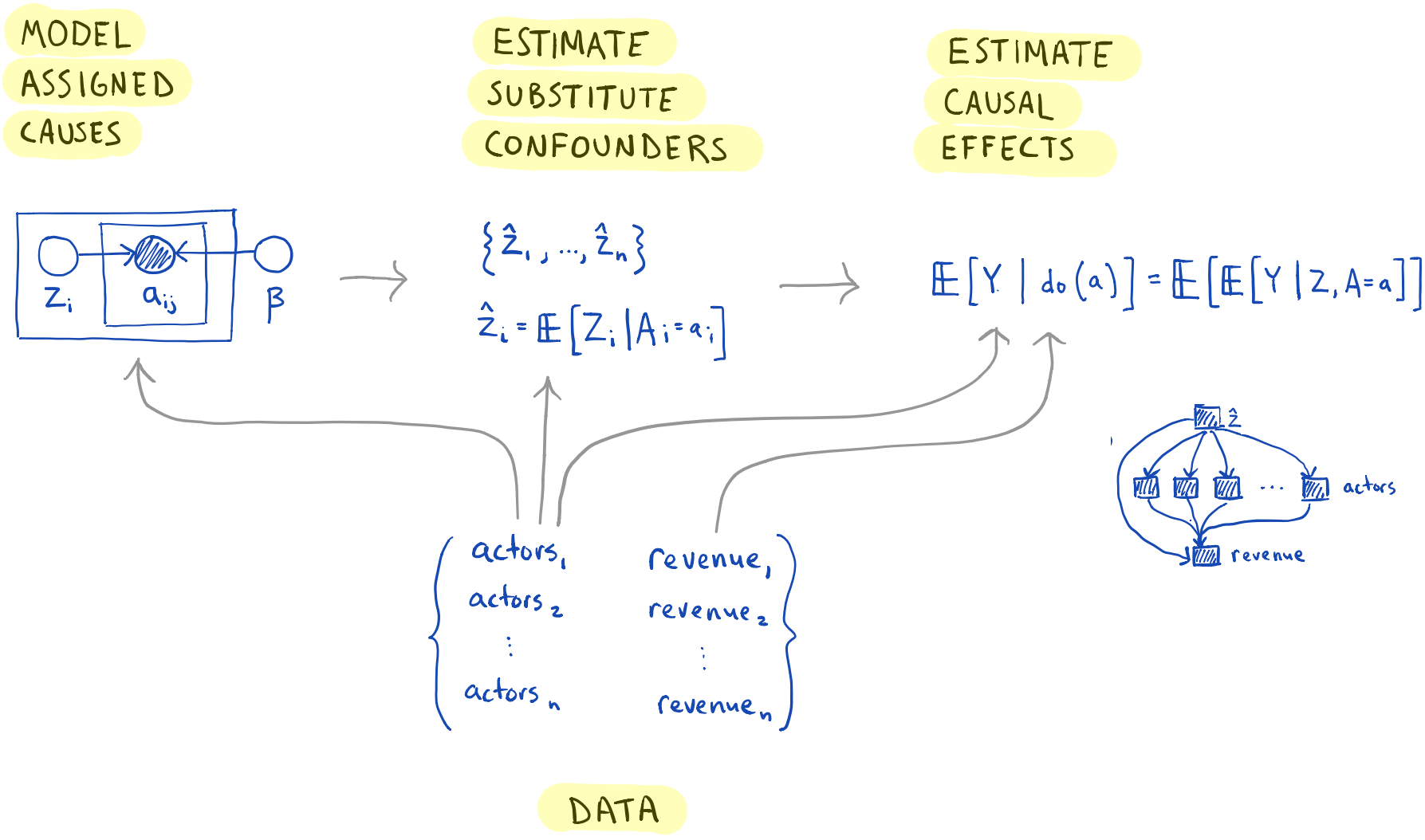
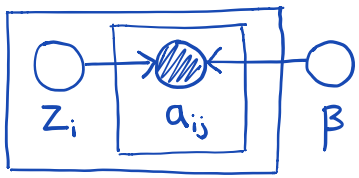
- But our problem is not classical.
- There are many causes (one per actor)—multiple causal inference
- **Multiple causes helps construct a variable that contains confounders.**

The deconfounder



The deconfounder

MODEL ASSIGNED CAUSES



ESTIMATE SUBSTITUTE CONFOUNDERS

$$\{\hat{z}_1, \ldots, \hat{z}_n\}$$

$$\hat{z}_i = \mathbb{E}[Z_i \mid A_i = a_i]$$

ESTIMATE CAUSAL EFFECTS

$$\mathbb{E}[Y \mid do(a)] = \mathbb{E}[\mathbb{E}[Y \mid Z, A = a]]$$

- Find, fit, and check a **factor model** of the assigned causes.
- Use the model to form **substitute confounders** for each individual.
- Use the substitute confounders in a **causal model** of the outcome.

(Note: There are still untestable assumptions!)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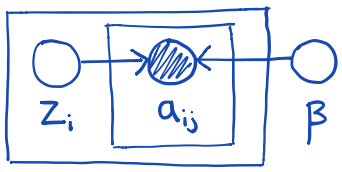
Intuition and assumptions

MODEL ASSIGNED CAUSES



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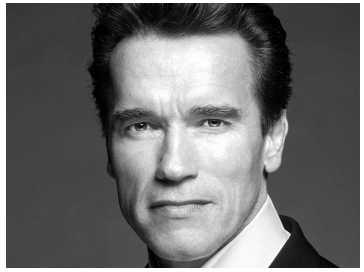
ESTIMATE CAUSAL EFFECTS

$$\mathbb{E}[Y \mid do(a)] = \mathbb{E}[\mathbb{E}[Y \mid Z, A = a]]$$

- Intuition: “Multi-cause confounders” induce dependence among the causes.
- That dependence is encoded in the data; we can capture it with a factor model
- Assume: No unobserved single-cause confounders. (Other assumptions too)

- “Overestimated”:



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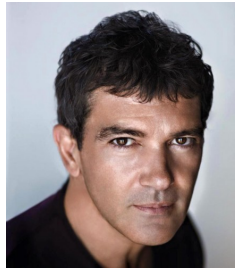
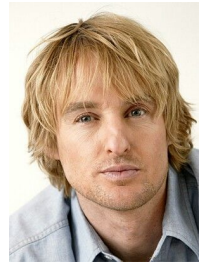
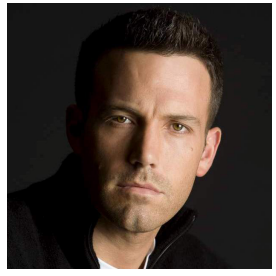
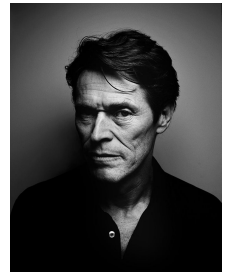


- Most “corrected”:







- Single cause confounding (Grimmer+ 2020):

Multiple causal inference (beyond James Bond)

- How do genes affect a trait?
- How do the players affect the game?
- How do prices of items affect how much money is spent?
- How do medicines affect lab measurements?
- How do neurons affect limb movement?

The deconfounder in more detail

Multiple causal inference

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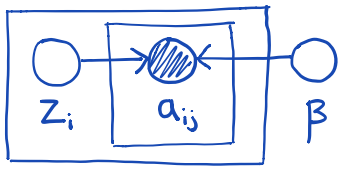
- Observed dataset $\mathscr{D} = \{(\mathbf{a}_1, y_1), \ldots, (\mathbf{a}_n, y_n)\}$
  - assigned causes $\mathbf{a}_i = \{a_{i1}, \ldots, a_{im}\}$
  - outcome y_i
- Goal: Do causal inference, $\mathbb{E}[Y; \mathrm{do}(\mathbf{a})]$
  - "The expectation of Y in the model where we intervened on $\mathbf{a}$."

The deconfounder

MODEL ASSIGNED CAUSES



ESTIMATE SUBSTITUTE CONFOUNDERS

$$\{\hat{z}_1, \ldots, \hat{z}_n\}$$

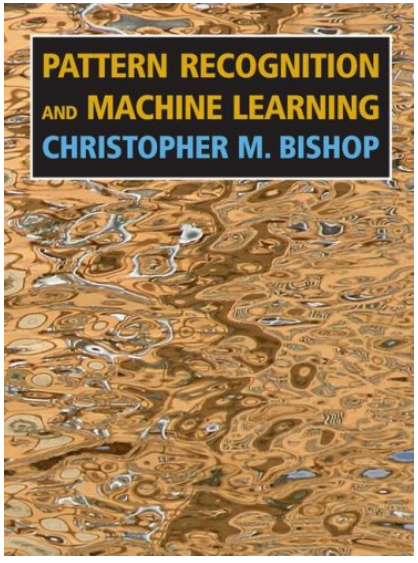
$$\hat{z}_i = \mathbb{E}[Z_i \mid A_i = a_i]$$

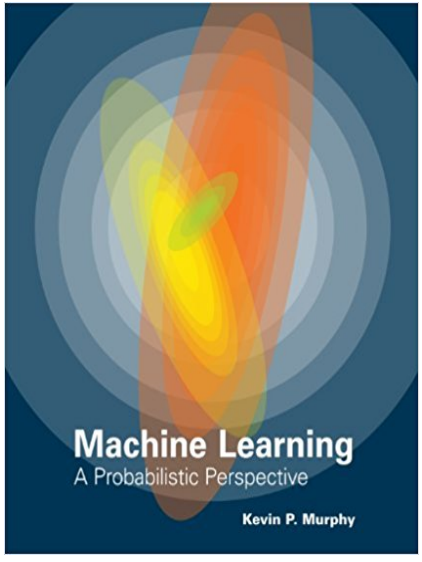
ESTIMATE CAUSAL EFFECTS

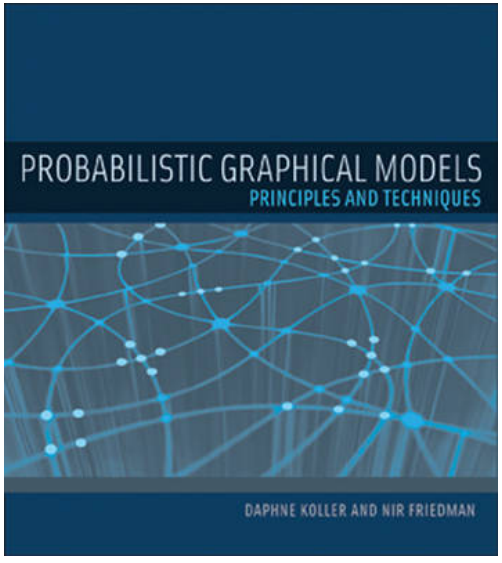
$$\mathbb{E}[Y \mid do(a)] = \mathbb{E}[\mathbb{E}[Y \mid Z, A = a]]$$

- Find, fit, and check a **factor model** of the movie casts.
- Use the factor model to form **substitute confounders** for each movie.
- Use the substitute confounders in a **causal model** of movie revenue.

Fit a probabilistic factor model





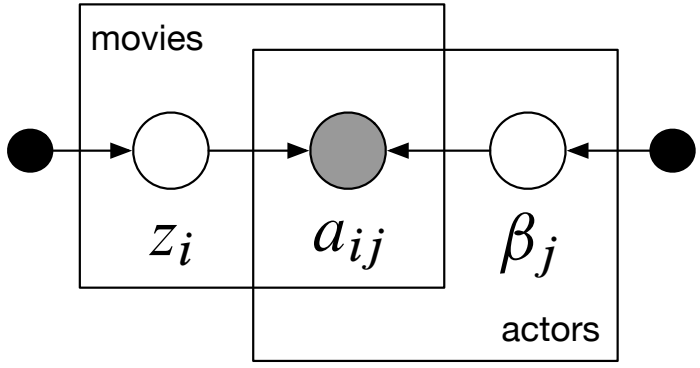


- A probabilistic factor model has the following form,

$$\begin{aligned} \beta_j &\sim \mathrm{p}(\beta_j) & j &= 1, \ldots, m \\ z_i &\sim \mathrm{p}(z_i) & i &= 1, \ldots, n \\ a_{ij} &\sim \mathrm{p}(a_{ij} \mid z_i, \beta_j). \end{aligned}$$

- E.g., mixtures, matrix factorization, deep generative models, topic models, ...

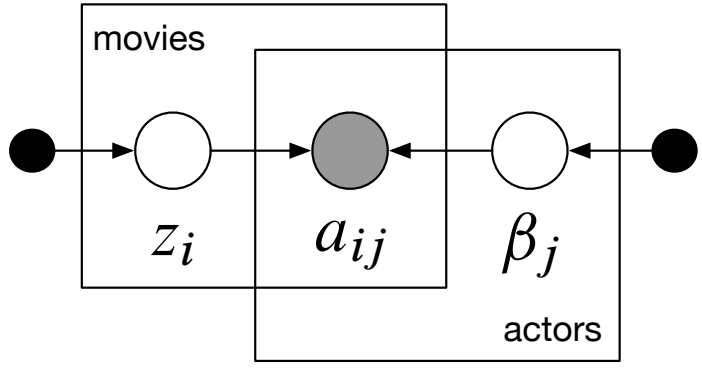
Poisson factorization [Gopalan+ 2015]



$$\beta_{jk} \sim \text{Gam}(a, b) \qquad i \in \{1, \ldots n\}$$
$$z_{ik} \sim \text{Gam}(a, b) \qquad j \in \{1, \ldots, m\}$$
$$a_{ij} \sim \text{Poi}(z_i^\top \beta_j) \qquad k \in \{1, \ldots, d\}$$

- Provides a generative model of the assigned causes a_{ij}.
- Can be approximated on large datasets with variational methods
- A Bayesian form of non-negative matrix factorization [Lee and Seung 1999]

Poisson factorization [Gopalan+ 2015]



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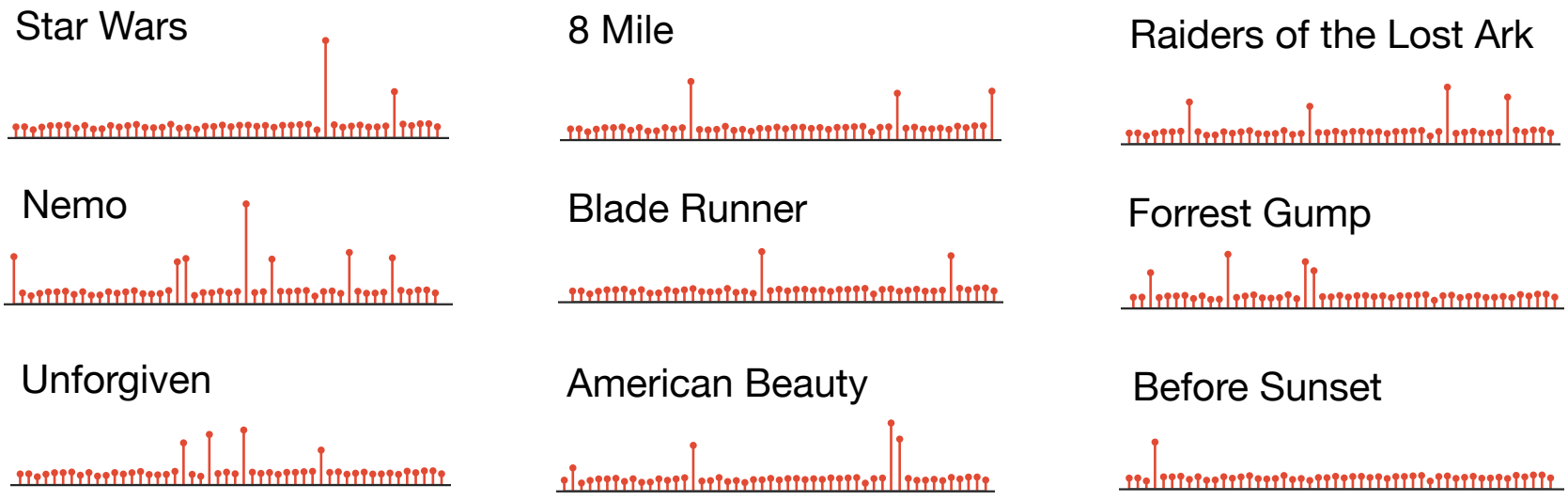
- Consider the dataset of casts $\mathbf{a}_{1:n}$.
- Approximate the posterior distribution $\text{p}(z_{1:n}, \beta_{1:m} \mid \mathbf{a}_{1:n})$.
- **We only model the actors $\mathbf{a}_i$; the outcome is not involved.**

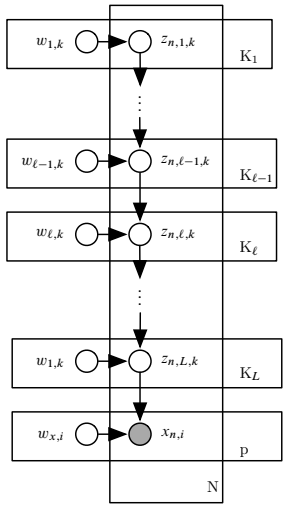
Check the factor model

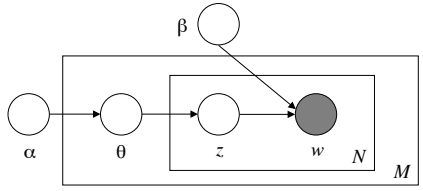


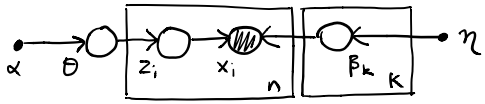
- We want the **learned representation** to capture the distribution of actors.
- Estimate $\hat{z}_i = \mathbb{E}_{\text{model}}[Z \mid \mathbf{a}_i, \boldsymbol{\beta}]$. (Approximate inference is OK.)
- Check how well $\hat{z}_i$ captures the true distribution of the actors.

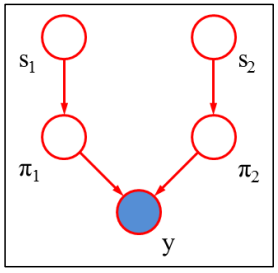
  [*Bayesian model criticism*: Rubin 1984; Gelfand+ 1992; Gelman+ 1996; ...]

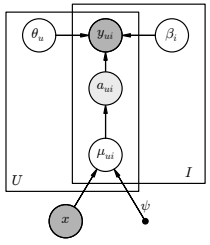
Check the factor model

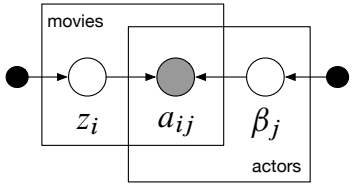






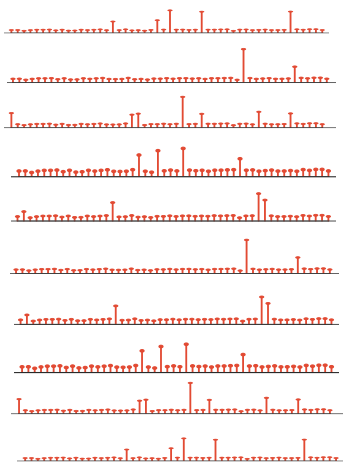






Model	Predictive score
Probabilistic PCA	0.14
Poisson factorization	0.16
Mixtures	0.01
Deep exponential families	0.19

Do causal inference



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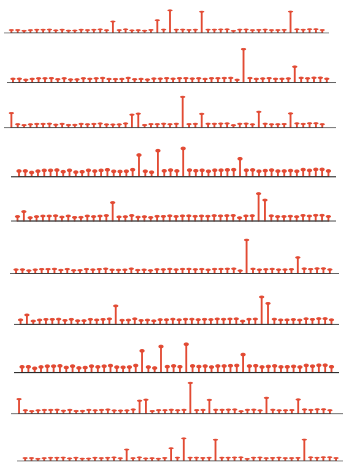
- The representations $\hat{z}_i$ are **substitute confounders**.
- They are latent attributes of movie casts that the factorization has uncovered.
- Form an **augmented dataset** of triplets $(\mathbf{a}_i, y_i, \hat{z}_i)$.

Do causal inference



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- Use the substitute confounders in a **causal inference**.
- E.g., fit regression from casts and confounders to revenue,

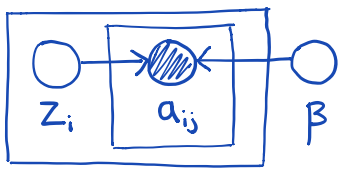
$$\mathbb{E}[Y \mid \mathbf{a}, \hat{z}] = \beta^\top \mathbf{a} + \eta^\top \hat{z}.$$

- Use adjustment to perform causal inference,

$$\mathbb{E}[Y \,;\, \mathrm{do}(\mathbf{a})] \approx \tfrac{1}{n} \textstyle\sum_{i=1}^{n} \mathbb{E}[Y \mid \mathbf{a}, \hat{z}_i].$$

What just happened?

MODEL ASSIGNED CAUSES



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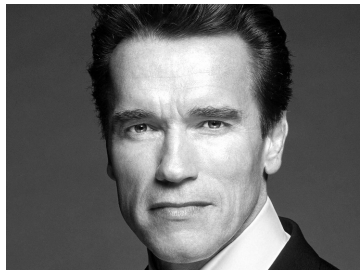
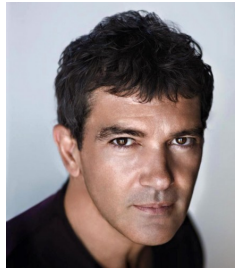
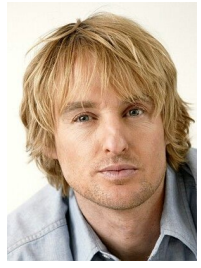
- We **modeled the causes** with a factor model.
- We used **learned representations** as substitutes for measured confounders.
- Idea: This exploratory method can correct for *some* unobserved confounding.

- “Overestimated”:



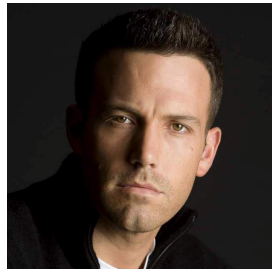
- “Underestimated”:

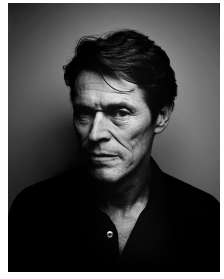


- Most “corrected”:





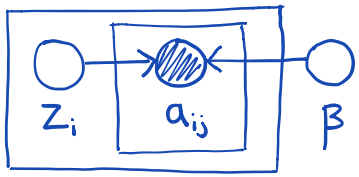


- Single cause confounding (Grimmer+ 2020):

A little theory

The deconfounder

MODEL ASSIGNED CAUSES



ESTIMATE SUBSTITUTE CONFOUNDERS

$$\{\hat{z}_1, \ldots, \hat{z}_n\}$$

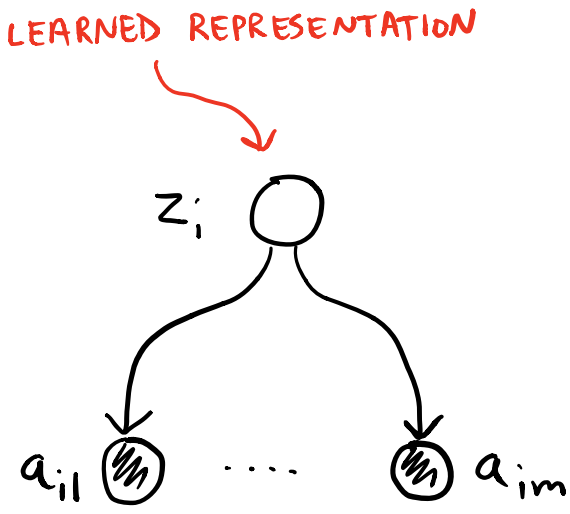
$$\hat{z}_i = \mathbb{E}[Z_i \mid A_i = a_i]$$

ESTIMATE CAUSAL EFFECTS

$$\mathbb{E}[Y \mid do(a)] = \mathbb{E}[\mathbb{E}[Y \mid Z, A = a]]$$

- Suppose we fit a **good factor model** of the assigned causes (the actors).
- Then its learned representation will contain **multi-cause confounders**.
- Main assumption: No unobserved single cause confounders.

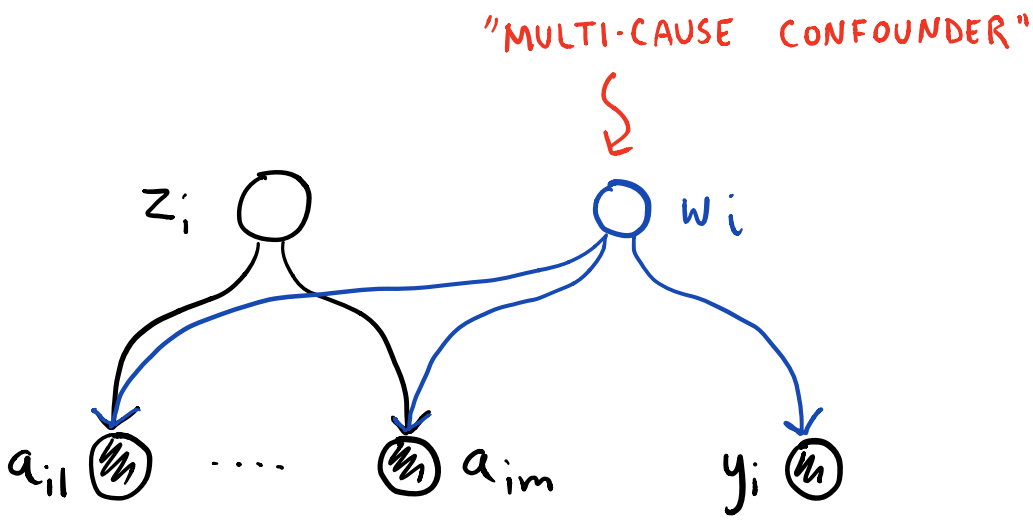
Intuition (through graphical models)



If we find a good factor model then

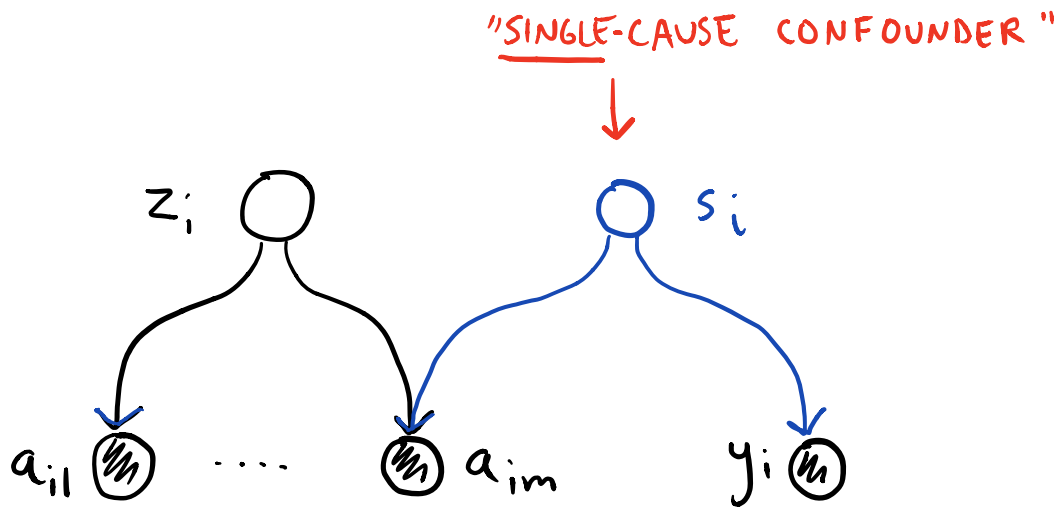
$$\mathrm{p}(a_{i1}, \ldots, a_{im} \,|\, z_i, \beta_{1:m}) = \prod_{j=1}^{m} \mathrm{p}(a_{ij} \,|\, z_i, \beta_j)$$

Intuition (through graphical models)



- There cannot be an additional unobserved **multi-cause confounder**.
- Contradiction: If one existed then the independence statement would not hold.

Intuition (through graphical models)



- There still might be a **single-cause confounder**.
- Reason: The conditional independence still holds.

THEOREM: THE DECONFOUNDER

Suppose $p_{\text{true}}(\mathbf{a})$ can be written $\int \mathrm{p}(z) \prod_j \mathrm{p}(a_j \mid z, \boldsymbol{\beta}) \, \mathrm{d}z$.

Further assume

1. No unobserved single-cause confounders X
2. The causes "pinpoint" the substitute $Z = f(\mathbf{a})$
3. Some other assumptions (see the paper)

Then

$$\mathbb{E}[Y \,;\, \mathrm{do}(\mathbf{a})] - \mathbb{E}\left[Y \,;\, \mathrm{do}(\mathbf{a}')\right] = \mathbb{E}_{Z,X}\left[\mathbb{E}_Y\left[Y \mid Z, X, \mathbf{a}\right] - \mathbb{E}_Y\left[Y \mid Z, X, \mathbf{a}'\right]\right].$$

(There has been further progress on identification; see references.)

The deconfounder

1. Find and fit a good probabilistic factor model of $\mathbf{A}_i$.
2. Use it to estimate Z_i, which renders the causes conditionally independent.
3. Use Z_i to help with causal inference.

- This theory motivates the algorithm.
- We assume that information about multi-cause confounders is embedded in the (observed) dependencies among the causes.
- The factor model extracts that information and uses it for causal inference.

The deconfounder

1. Find and fit a good probabilistic factor model of $\mathbf{A}_i$.
2. Use it to estimate Z_i, which renders the causes conditionally independent.
3. Use Z_i to help with causal inference.

- Genome-wide association studies: e.g., Pritchard+ 2000, Astle+ 2009, Yu+ 2006, Kang+ 2010, Song+ 2015, Haro+ 2015, Price+ 2006, Renaux+ 2020
- Econometrics in "factor-adjusted regression": e.g., Stock and Watson 2016, Gonclaves and Perron 2014, Cheng and Hansen 2015, Bai and Ng 2006
- Testing, covariance estimation, regression: e.g., Friguet+ 2009, Fan+ 2019, Shah and Meinshausen 2018, Cevid+ 2018, Guo+ 2020

The deconfounder

1. Find and fit a good probabilistic factor model of $\mathbf{A}_i$.
2. Use it to estimate Z_i, which renders the causes conditionally independent.
3. Use Z_i to help with causal inference.

How might the deconfounder go wrong?

- The factor model does not capture the distribution of causes. (It doesn't.)
- There is uncertainty about inference of z. (There is.)
- There is unmeasured single-cause confounding. (There probably is.)
- There is estimation variance. (Yes.)

The deconfounder

1. Find and fit a good probabilistic factor model of $\mathbf{A}_i$.
2. Use it to estimate Z_i, which renders the causes conditionally independent.
3. Use Z_i to help with causal inference.

My two cents

The deconfounder is an *exploratory method* that removes *some* sources of confounding bias. A better factor model captures more of the multi-cause confounding.

How to use a deconfounder: Condition on known confounders, both multi-cause and single-cause, and use domain knowledge to build a good factor model. Then use the deconfounder to explore hypothetical causal connections in your data.

Example: Genome-wide association studies (GWAS)

- GWAS is a problem of multiple causal inference
- How is genetic variation causally connected to a trait?
- For each individual: a trait and many measurements of the genome (SNPs).

Example: Genome-wide association studies (GWAS)

- Multiple-cause confounding is a problem.
- Non-causal SNPs may be highly correlated to causal SNPs
- Misestimates causal effects

Simulation study

ID (i)	SNP_1 ($a_{i,1}$)	SNP_2 ($a_{i,2}$)	SNP_3 ($a_{i,3}$)	SNP_4 ($a_{i,4}$)	SNP_5 ($a_{i,5}$)	SNP_6 ($a_{i,6}$)	SNP_7 ($a_{i,7}$)	SNP_8 ($a_{i,8}$)	SNP_9 ($a_{i,9}$)	$\cdots$	SNP_100K ($a_{i,100K}$)	Height (feet) (y_i)
1	1	0	0	1	0	0	1	2	0	$\cdots$	0	5.73
2	1	2	2	1	2	1	1	0	1	$\cdots$	2	5.26
3	2	0	1	1	0	1	0	1	1	$\cdots$	2	6.24
4	0	0	0	1	1	0	1	2	0	$\cdots$	0	5.78
5	1	2	1	1	1	0	1	0	0	$\cdots$	1	5.09
$\vdots$						$\vdots$						$\vdots$

- Generate SNPs a_{ij}, where each individual belongs to a latent group c_i.
- The true outcome is a trait y_i, drawn from

$$y_i = \sum_j \beta_j a_{ij} + \lambda_{c_i} + \varepsilon_i \quad \varepsilon_i \sim \mathcal{N}(0, \sigma_{c_i}),$$

  where many β_j are zero, i.e., non-causal SNPs.
- Confounded: the intercept λ_{c_i} and error ε_i are connected to the latent group.

Simulation study

	pred. score	**Real-valued outcome** RMSE$\times 10^2$	**Binary outcome** RMSE$\times 10^2$
No control	—		
Control for confounders*	—		
(G)LMM	—		
PPCA	0.14		
PF	0.15		
LFA	0.14		
Mixture	0.00		
DEF	0.20		

- We fit many factor models; none was the true model.
- Each provides different levels of predictive performance.
- All computation done in Edward [Tran+ 2018] .

Simulation study

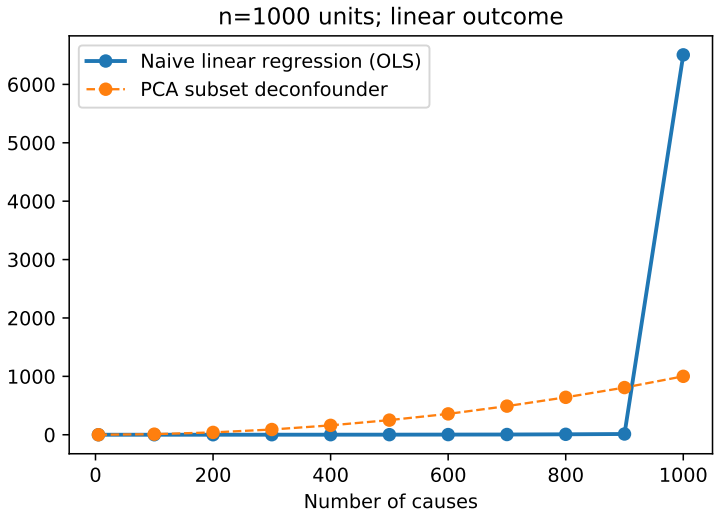
	pred. score	**Real-valued outcome** RMSE$\times 10^2$	**Binary outcome** RMSE$\times 10^2$
No control	—	58.82	29.50
Control for confounders*	—	25.32	25.77
(G)LMM	—	35.18	28.87
PPCA	0.14	33.32	26.70
PF	0.15	33.38	26.84
LFA	0.14	33.93	26.83
Mixture	0.00	57.59	29.96
DEF	0.20	**26.47**	**25.91**

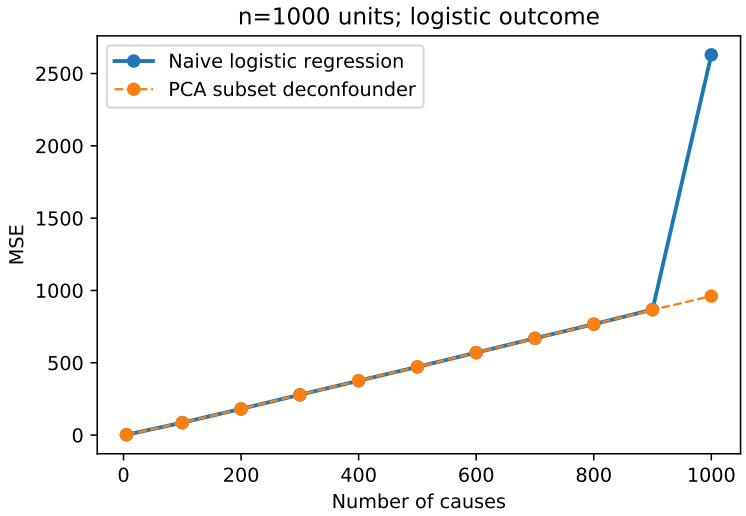
- Also fit outcome models with no control and with observed confounders
- The deconfounder provides good causal estimates.
- Predictive checks indicate downstream causal performance.

Is the theory correct?

- There has been some debate about the theory (Ogburn+ 2020, 2021).
- Key worry: By definition, we can *never* know anything about truly unobserved confounders from the observational data.
- The deconfounder does *not* challenge this indisputable fact.
- Rather, it finds a class of confounders that are *effectively observed*.
- Thus the deconfounder tries to *extract* effectively observed information.
- For a clarification of this theory, see Wang and Blei (2020).

Is the theory useful?





- Grimmer+ 2020 discusses assumptions required for identification.
- They argue that "pinpointability" $Z = f(\mathbf{A})$ is difficult to accept. (I agree.)
- They show that the assumption of pinpointability implies that simple regression also performs causal inference when $n \gg p$.
  (But see above when $n < p$.)
- They argue that single-cause confounding easily arises (e.g., Stan Lee).

Summary

- The deconfounder assumes there is information in the dependency among causes that is helpful for removing confounding bias.
- The algorithm tries to extract this information for causal inference. It uses unsupervised learning and Bayesian model criticism.

Caveat

- The deconfounder is not a turnkey solution to causal inference.
- It does not relieve the researcher from measuring confounders.
- It comes with uncheckable assumptions.

Ongoing collaboration with MSK

- What is the chemo-sensitivity of cells when we silence genes $a_1, \ldots, a_p$?
- Data: Individual cells
  - Causes: Gene expression level (counts)
  - Outcome: Chemo sensitivity (binary)
- Possible multi-cause confounding:
  - copy number
  - tumor microenvironment
  - batch effects
  - metabolic changes

Some ideas of further research about the deconfounder

- We need methods to **propagate uncertainty** through the deconfounder.
  (Uncertainty comes from factor modeling, no pinpointability, finite samples, ...)
- The field of **model criticism** needs attention.
  (It helps evaluate the substitute confounder.)
- The deconfounder does not easily handle **causally dependent causes**.
  (Can we adapt it to time series?)
- We don't yet understand the **bias-variance trade-off**.
  (The better the factor model, the more variance we incur in the estimator.)
- We don't have a complete picture of **causal identification**.
  (What can be identified? What assumptions can we relax?)

Theory:

- Y. Wang and D. Blei. The blessings of multiple causes. Journal of the American Statistical Association, 114(528):1574–1596, 2019.
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- Y. Wang and D. Blei. A proxy variable view of shared confounding. In International Conference on Machine Learning, 2021.

Applications:

- L. Zhang, Y. Wang, A. Ostropolets, J. Mulgrave, D. Blei, and G. Hripcsak. The medical deconfounder: Assessing treatment effects with electronic health records. In Machine Learning for Health Care, 2019.
- D. Sridhar, C. De Bacco, and D. Blei. Estimating social influence from observational data. In Causal Learning and Reasoning, 2022.

- Discussions and rejoinder of Wang and Blei (2019) in JASA
- M. Song, W. Hao, and J. Storey. Testing for genetic association in arbitrarily structured populations. Nature Genetics, 2015.
- R. Ranganath and A. Perotte. Multiple causal inference with latent confounding. arXiv:1805.08273, 2018.
- A. D'Amour. On multi-cause causal inference with unobserved confounding: Counterexamples, impossibility, and alternatives. In AISTATS, 2019.
- E. Ogburn, I. Shpitser, and E. T. Tchetgen. Counterexamples to "The blessings of multiple causes" by Wang and Blei. arXiv:2020.001, 2020.
- J. Grimmer, D. Knox, and B. Stewart. Naive regression requires weaker assumptions than factor models to adjust for multiple cause confounding. arXiv 2007.12702, 2020.
- A. Puli, A. Perotte, and R. Ranganath. Causal estimation with functional confounders. Neural Information Processing Systems, 2020.
- J. Zheng, A. D'Amour, and A. Franks. Copula-based sensitivity analysis for multi-treatment causal inference with unobserved confounding. arXiv:2102.09412, 2021.
- D. Kong, S. Yang, and L. Wang. Identifiability of causal effects with multiple causes and a binary outcome. Biometrika, to appear.
- W. Miao, W. Hu, E. Ogburn, and X. Zhou. Identifying effects of multiple treatments in the presence of unmeasured confounding. arXiv:2011.04504, 2021.
- A. Franks and J. Zheng. Bayesian inference for partial identification of multiple treatment effects. The Neglected Assumptions in Causal Inference, 2021.

Extra slides

On identification

- A causal quantity is identifiable if it can be written as a function of the observed variables.
- If the causal quantity changes, so does the distribution of the observed data.
- D'Amour (2019) gives two examples where $\mathbb{E}[Y; \mathrm{do}(\mathbf{a})]$ is not identifiable.
- These results help flesh out the theory of multiple causal inference.
- But identification is still possible (with assumptions).

On identification

- Assume we pinpoint a substitute confounder $\hat{z} = f(\mathbf{a})$, e.g., many causes.
- (Theorem) Differences of complete interventions are

$$\mathbb{E}[Y\,;\,\mathrm{do}(\mathbf{a})] - \mathbb{E}\big[Y\,;\,\mathrm{do}(\mathbf{a}')\big].$$

  They are nonparametrically identifiable when the outcome separates contributions from the unobserved confounders and causes.

- (Theorem) Consider a subset of causes B. The subset intervention is

$$\mathbb{E}[Y\,;\,\mathrm{do}(\mathbf{a}_B)].$$

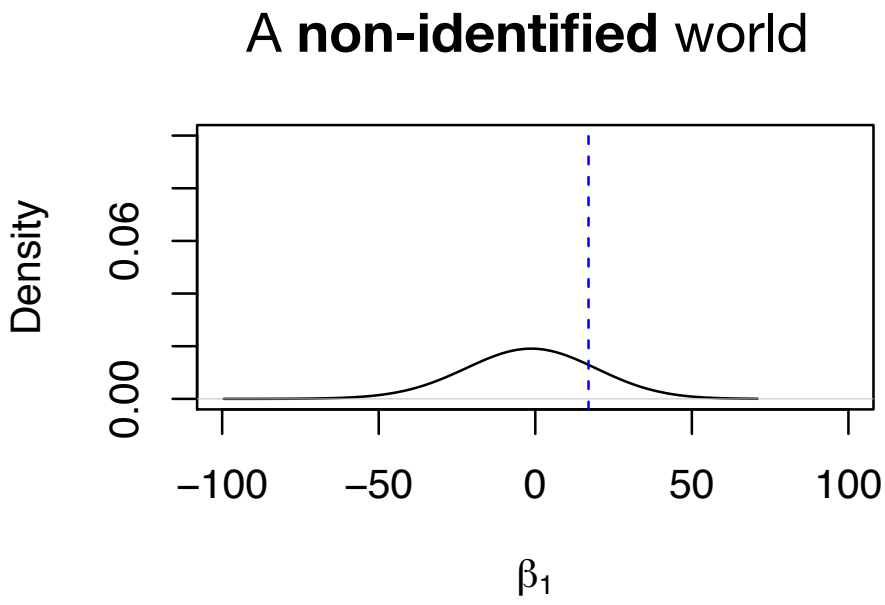
  It is identifiable with overlap on the subset, $\mathrm{p}(\mathbf{a}_B \,|\, z) > 0$.

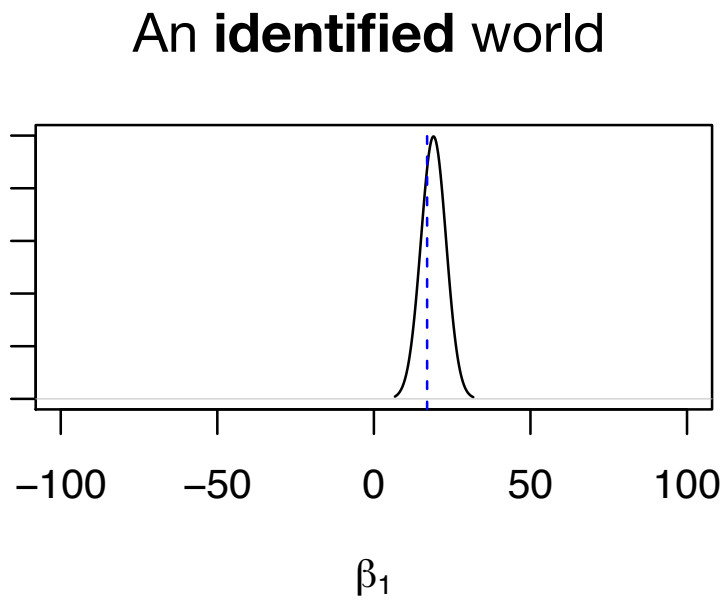
Causal quantity	Result	Condition	Source
$P(Y(\boldsymbol{a}))$	Non-ID	No conditions	D'Amour (2019)
$\mathbb{E}[Y(\boldsymbol{a})] - \mathbb{E}\left[Y(\boldsymbol{a}')\right]$	ID	Consistent substitute confounder; Categorical substitute confounder; No confounder/cause interaction; Differentiable relationships	Th. 6 (WB)
$\mathbb{E}_A\left[\mathbb{E}_Y\left[Y(a_{1:k}, A_{(k+1):m})\right]\right]$	ID	Consistent substitute confounder; $A_{1:k}$ satisfy overlap	Th. 7 (WB)
$\mathbb{E}\left[Y(\boldsymbol{a}') \mid \boldsymbol{A} = \boldsymbol{a}\right]$	ID	Consistent substitute confounder; $\boldsymbol{a}'$ and $\boldsymbol{a}$ map to same substitute	Th. 8 (WB)
$\mathbb{E}[Y(\boldsymbol{a})]$	ID	$\mathbb{E}[U \mid \mathbf{A}]$ nonlinear; $\mathbb{E}[Y \mid \mathbf{A}, U]$ linear	Sec 2.1 (IJ)
$\mathbb{E}[Y(\boldsymbol{a})]$	ID	Measure instrument W; Instrument W satisfies overlap	Sec 2.2 (IJ)
$\int Y(\boldsymbol{a}) q_1(\boldsymbol{a}) \mathrm{d}\boldsymbol{a}$ $- \int Y(\boldsymbol{a}) q_2(\boldsymbol{a}) \mathrm{d}\boldsymbol{a}$	ID	$p(\boldsymbol{a} \mid z) > 0$ when $q_1(\boldsymbol{a}), q_2(\boldsymbol{a}) > 0$	Sec 2.3 (IJ)

Theory *and* practice

- Scientist: That's complicated. Umm...Can I still use the deconfounder?
  Us: Yes, but please *assess its uncertainty*.
- Here is a Bayesian way. Draw samples of Z; then draw samples of outcome parameters conditional on Z; compute the causal quantity (e.g., ATE). We get many samples of the ATE, which lets us compute a credible interval.
- This uncertainty reflects estimation uncertainty and identification uncertainty. It tells us how the finite data informs us of the causal quantity of interest.
- In cases where a causal quantity is identified, this uncertainty only involves estimation uncertainty. When the causal quantity is not identifiable, it returns uninformative posterior (with uninformative priors).

Are we living in an identified world?





Predictive checks

- Goal: Make sure the factor model fits the data well.
- Our checks are similar to classical posterior predictive checks.
- Generate replicated datasets from the fitted factor model.
- Compare replicated data to real data

Predictive checks

- Statistic: Negative expected predictive log-likelihood entries

$$t(\mathbf{a}_{i,\text{heldout}}) = -\mathbb{E}[\log \mathrm{p}(\mathbf{a}_{i,\text{heldout}} \mid Z) \mid \mathbf{a}_{i,\text{obs}}]$$

  Larger values indicate more model misfit.

- Compute predictive scores

$$\text{predictive-score}_i = p\left(t(\mathbf{a}^{\text{rep}}_{i,\text{heldout}}) > t(\mathbf{a}_{i,\text{heldout}})\right),$$

  where the replicated data come from the predictive distribution.

- A factor model passes the check if the mean of the predictive scores > 0.1. (This is a subjective criterion.)

The bias-variance tradeoff

- The factor model should fit the assigned causes well. It makes sure the substitute confounder captures all multi-cause confounders; hence it leads to unbiased causal estimates.
- We lose efficiency in causal-effect estimation when the factor model fits the assigned causes too well, e.g., when the latent space has too many dimensions. (The resulting causal estimates are still unbiased.)
- The latent space can theoretically have more dimensions than the number of causes, but it might result in highly variable causal-effect estimates.
- For an optimal bias-variance tradeoff, we choose the smallest factor model that passes the predictive check.