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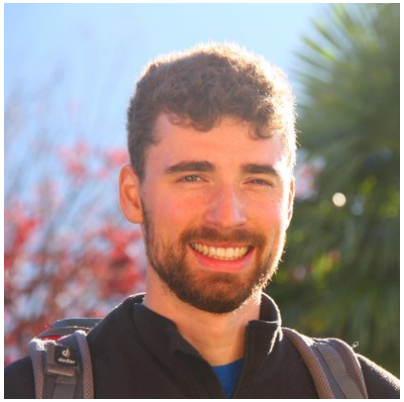
Causality and Propensity Score Methods

PyData Meetup Berlin, April 19th, 2017

Florian Wilhelm

About me



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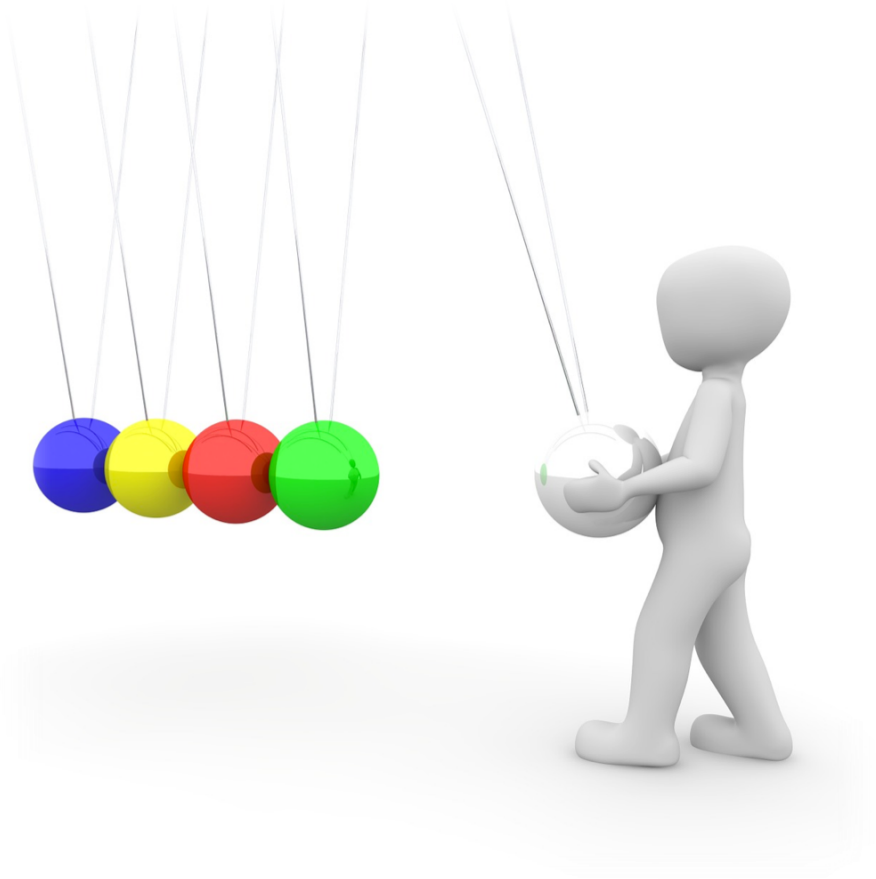
- Studies of mathematics and PhD at the Karlsruhe Institute of Technology about “Parallel Preconditioners for an Ocean Model in Climate Simulations“,
- Data scientist at Blue Yonder, leading provider of machine learning solutions for retail,
- Data scientist at inovex, an IT project house with focus on digital transformation. Currently working in the Data Team at mobile.de for more than one year.

Outline

1. Motivation
2. Theory
3. Examples
4. Q&A



Correlation vs. causality

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"Correlation trumps causation"

"…society will need to shed some of its obsession for causality in exchange for simple correlation: not knowing why but only what."

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Where does causality matter?

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Commercial features at mobile.de

Advertisement campaigns with coupons

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Causal effect

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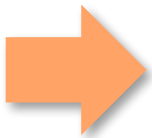
Feature 1	Feature 2	Feature 3	Do Feature
42	9	3	0
34.3	1729	4	1
23	69	5	0



Target
10
100
8

- Let y_{1i} be the target if the i-th sample had the do-feature set to 1,
- and let y_{0i} be the target if the do-feature was 0.
- The causal effect is the comparison of y_{1i} with y_{0i}, e.g. $\frac{y_{1i}}{y_{0i}}$ or $y_{1i} - y_{0i}$, average causal effect is $\mathrm{E}(y_1) - \mathrm{E}(y_0)$.
- $p(y|do(z))$ denotes the "causal effect" of Z on Y, i.e. the distribution of Y after setting variable Z to a constant $Z = z$ by external intervention.

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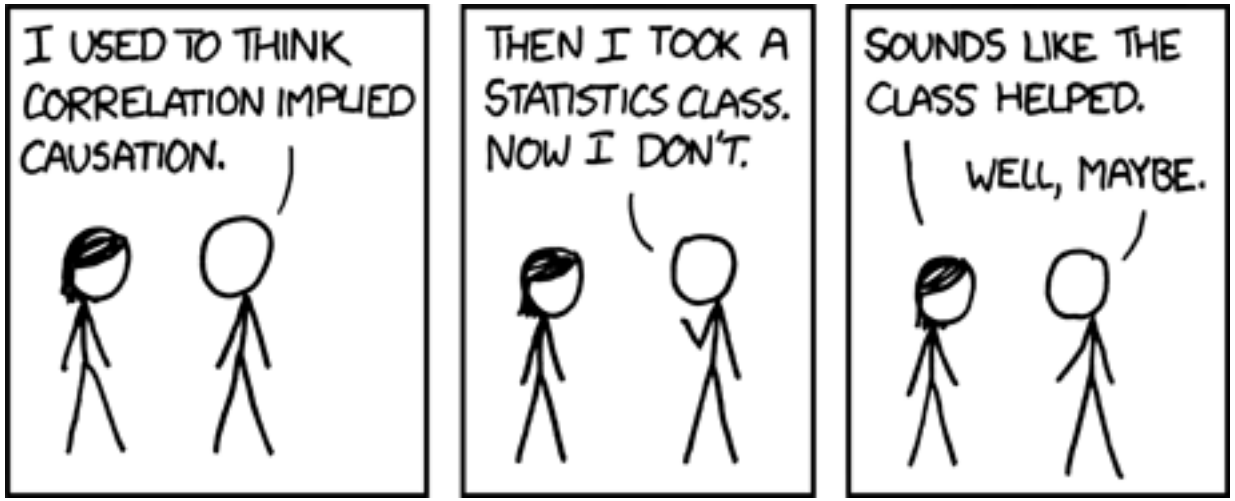
Fundamental problem

- A causal claim is a statement about what did not happen (counterfactuals, potential outcomes)
- Individual causal effects cannot be measured (fundamental problem)
- Correlation is not causation



Source: https://xkcd.com/552/

Clinical trials

Randomized trial:

- treatment assignment Z is random thus independent of features X and potential outcomes Y
- Z is *controllable*
- gold standard

Observational trial:

- treatment assignment Z depends on X
- Z is not *controllable*
- sometimes necessary for e.g. ethical reasons
- causal danger zone

Strongly ignorable & admissable

Let X denote the covariates, Y_0, Y_1 the potential outcomes for treated $Z = 1$ and control $Z = 0$ units.

Treatment assignment Z is *strongly ignorable* given X if

$$(Y_0, Y_1) \perp Z \mid X \quad \text{and } 0 < p(Z = 1|x) < 1.$$

Using this assumption for causal inference is equivalent to X being *admissable*, i.e.

$$p(y|\,\mathrm{do}(z)) = \sum_x p(y|x,z)p(x).$$

Causal effect in randomized trials

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Using machine learning to estimate individual causal effect:

1. train a model with covariates X and Z as feature and Y as target,
2. predict for a given x the response $\hat{y}_1$ with $Z = 1$ and $\hat{y}_0$ with $Z = 0$,
3. calculate the effect with $\hat{y}_1 - \hat{y}_0$ or $\frac{\hat{y}_1}{\hat{y}_0}$.

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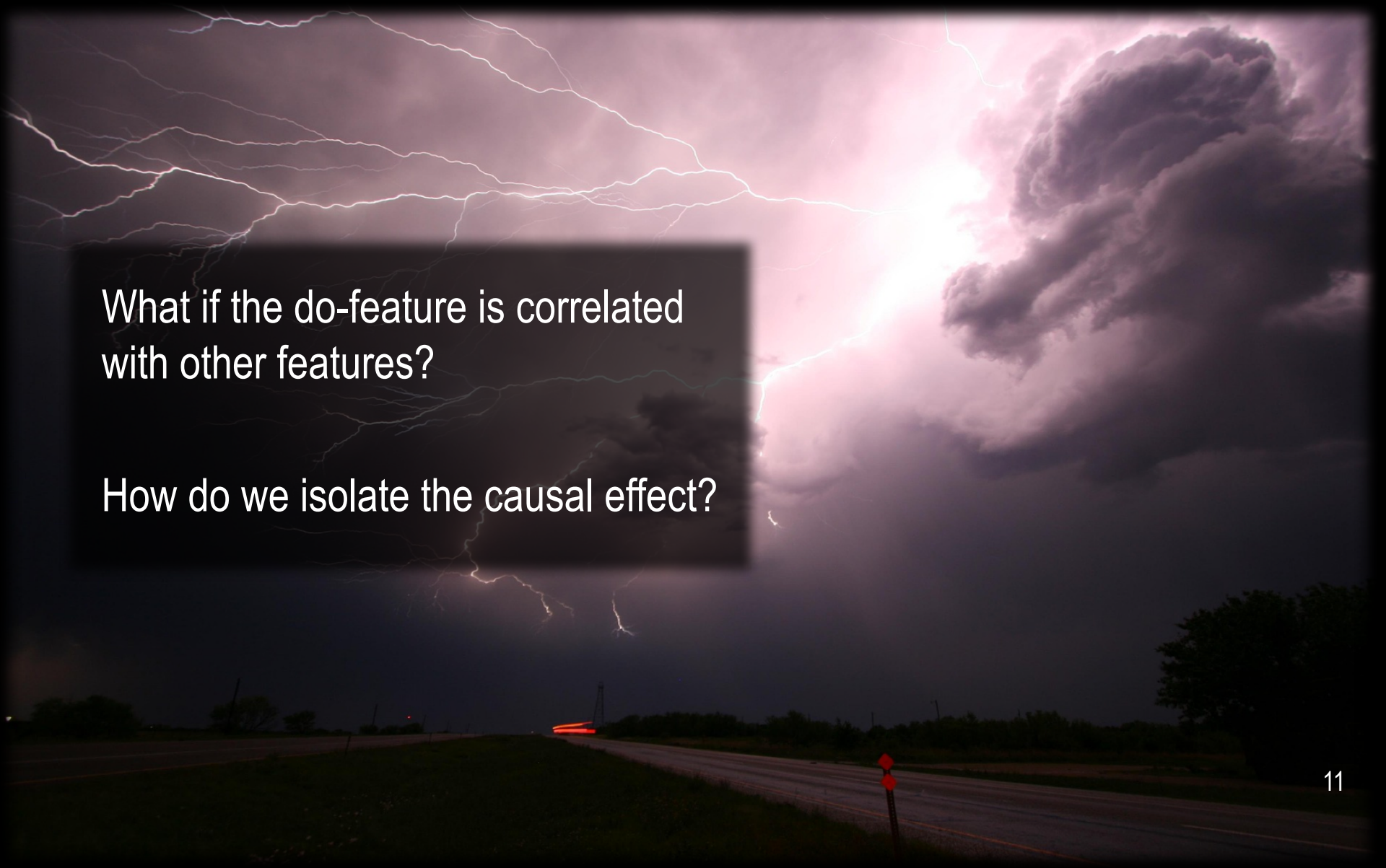
Causal effect in observational trials



What if the do-feature is correlated with other features?

How do we isolate the causal effect?

Propensity score

Propensity of receiving a treatment:

- propensity score e_i defined as $p(Z = 1|x_i)$,
- estimate with a classification method returning class probabilities.

Use e_i to define propensity weights w_i as

$$w_i := \frac{z_i}{e_i} + \frac{1 - z_i}{1 - e_i}.$$

Weight each sample i by its weight w_i in order to generate synthetic samples so that Z is no longer correlated to X. This is called *inverse probability of treatment weighting (IPTW).*

Causal effect in non-randomized trials

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Using machine learning and propensity:

1. train a model with covariates X in order to predict Z,
2. calculate the propensity scores e_i by applying the trained model to all x_i,
3. train a second model with covariates X and Z as features and response Y as target by using w_i as sample weight for the i-th observation,
4. use this model to predict the causal effect like in the approach of the randomized trial.

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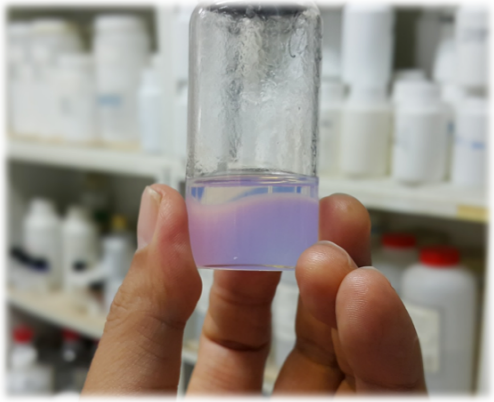
Synthetic example

Let the expected recovery time in days be Poisson distributed with

$$\mathrm{E}(t_{recovery}) = \exp(2 + 0.5 \cdot I_{male} + 0.03 \cdot age + 2 \cdot severity - 1 \cdot I_{medication}),$$

where I is an indicator function.

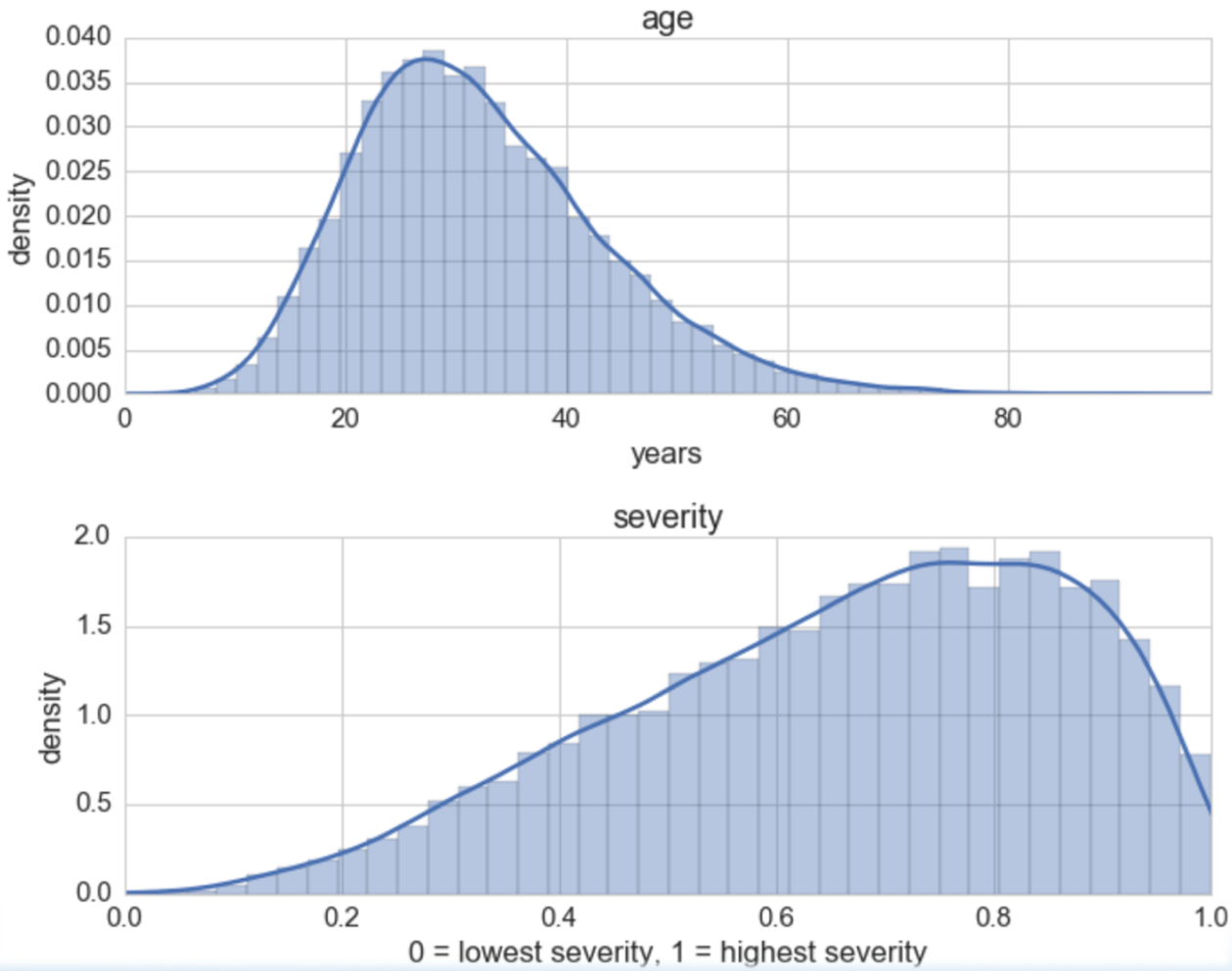


For the covariates we have:

- $sex \sim \mathcal{U}(\{0, 1\})$
- $age \sim \gamma(8,\ 4)$
- $severity \sim \beta(3,\ 1.5)$

Use this to generate 10,000 samples and note that $\exp(-1) \approx 0.37$.

Distribution of age and severity



Experimental setup

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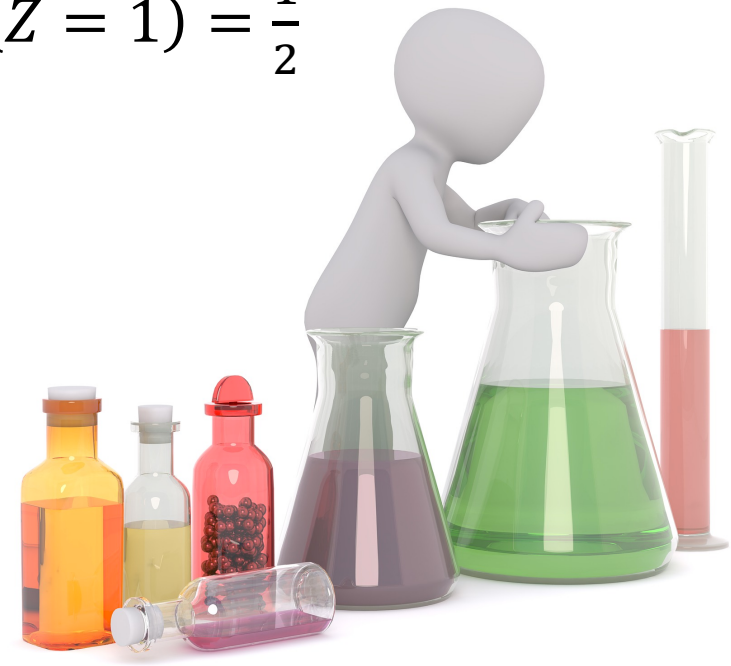
1. **Randomized trial**
   a) assign treatment randomly with $p(Z = 1) = \frac{1}{2}$
   b) use Poisson regression
   c) use Random forest

2. **Non-randomized trail**
   a) assign treatment based on X
   b) use Poisson regression
   c) use Random forest



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Correlation matrix in randomized trial

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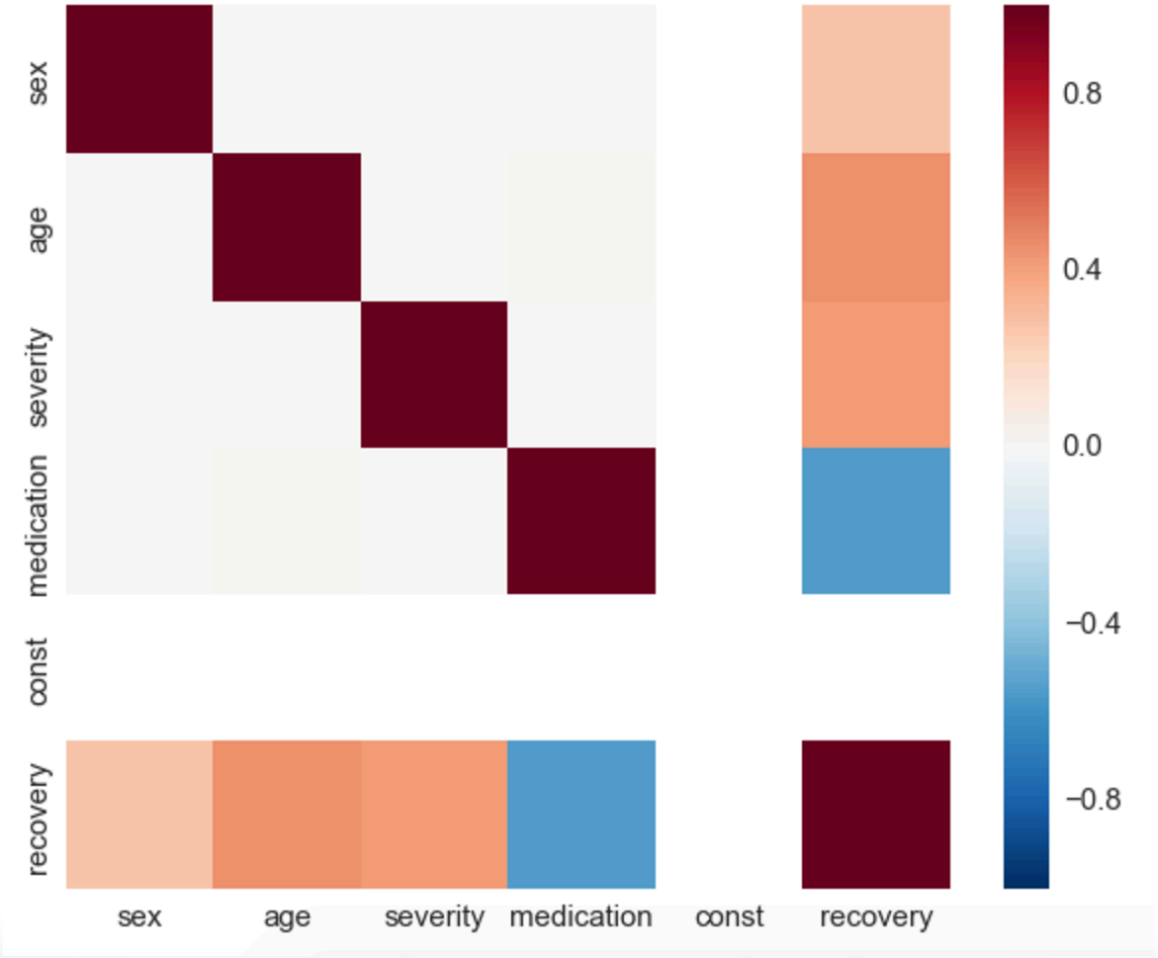
No correlation between X and Z



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Poisson regression in randomized trial

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Poisson regression correctly estimates the coefficients of the features

Dep. Variable:	recovery	**No. Observations:**	10000
Model:	GLM	**Df Residuals:**	9995
Model Family:	Poisson	**Df Model:**	4
Link Function:	log	**Scale:**	1.0
Method:	IRLS	**Log-Likelihood:**	-34429.
Date:	Fri, 31 Mar 2017	**Deviance:**	10080.
Time:	14:30:21	**Pearson chi2:**	1.00e+04
No. Iterations:	5		

	coef	**std err**	**z**	**P>|z|**	**[0.025**	**0.975]**
sex	0.4994	0.002	211.934	0.000	0.495	0.504
age	0.0301	8.95e-05	335.807	0.000	0.030	0.030
severity	2.0000	0.006	309.610	0.000	1.987	2.013
medication	-1.0024	0.003	-387.721	0.000	-1.007	-0.997
const	1.9990	0.006	326.234	0.000	1.987	2.011

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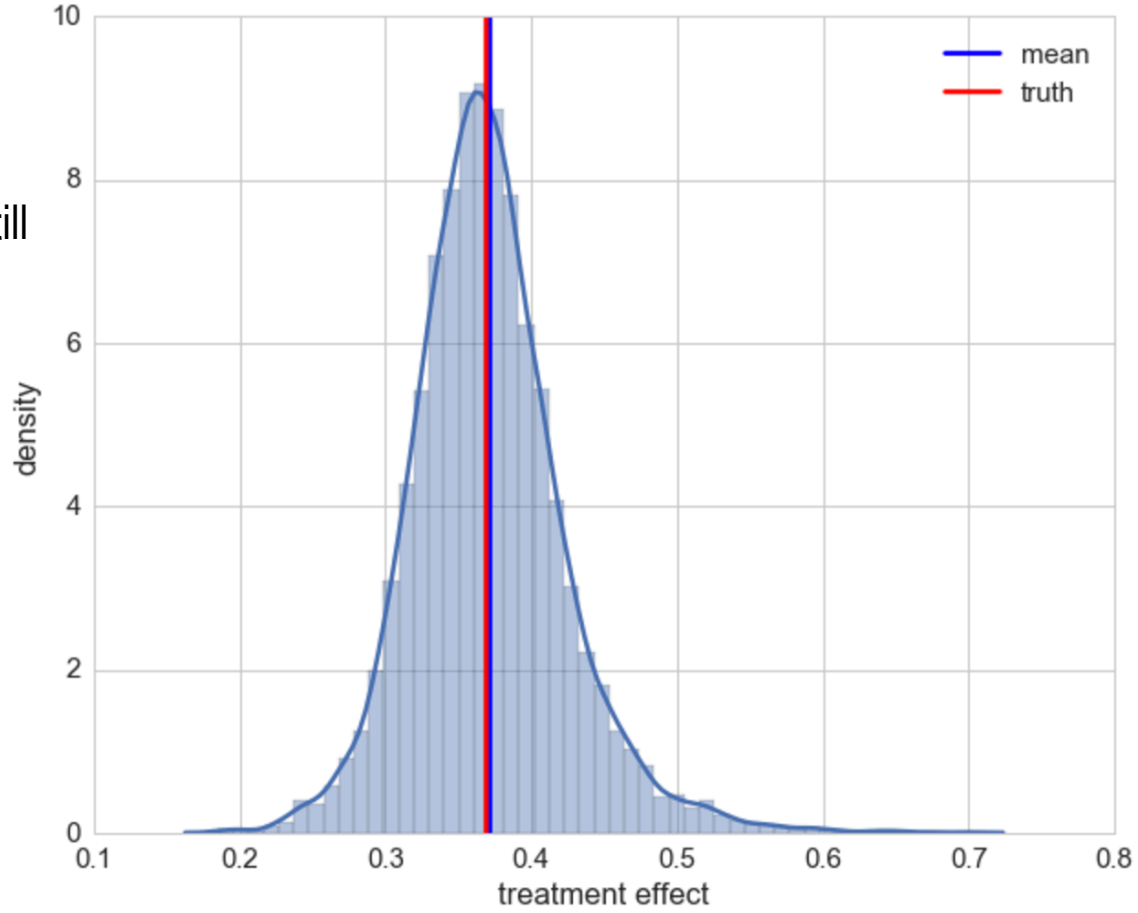
Random forest in randomized trial

- Estimation of the average effect quite accurate
- Estimation of individual effects still decent



Non-randomized Trial

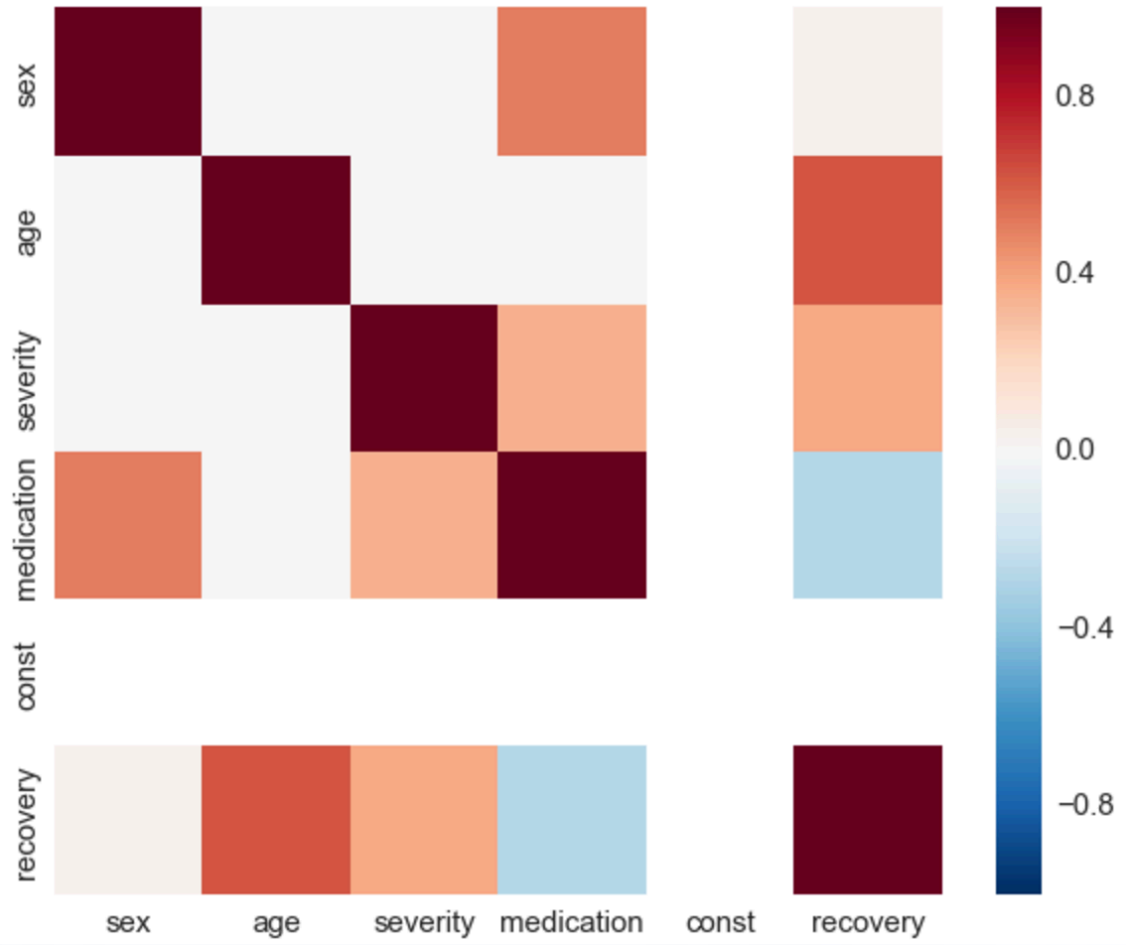
Assign treatment based on covariates, i.e.

$$Z = \begin{cases} 1, & \frac{1}{3} \cdot I_{male} + \frac{2}{3} \cdot severity + \epsilon > 0.8 \\ 0, & \text{otherwise} \end{cases},$$

where $\epsilon \sim \mathrm{N}(0,\ 0.15^2)$.

Correlation matrix in non-randomized trial

Feature *sex* and *severity* are highly correlated to *medication*



Poisson regression in non-randomized trial

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- Poisson regression still correctly estimates the coefficients
- Model dependence works in our favor

Dep. Variable:	recovery	**No. Observations:**	10000
Model:	GLM	**Df Residuals:**	9995
Model Family:	Poisson	**Df Model:**	4
Link Function:	log	**Scale:**	1.0
Method:	IRLS	**Log-Likelihood:**	-35645.
Date:	Fri, 31 Mar 2017	**Deviance:**	10018.
Time:	14:30:23	**Pearson chi2:**	9.98e+03
No. Iterations:	5		

	coef	std err	z	P>|z|	[0.025	0.975]
sex	0.5043	0.002	203.256	0.000	0.499	0.509
age	0.0299	8.58e-05	349.024	0.000	0.030	0.030
severity	1.9996	0.006	313.055	0.000	1.987	2.012
medication	-1.0063	0.003	-302.201	0.000	-1.013	-1.000
const	2.0013	0.006	340.305	0.000	1.990	2.013

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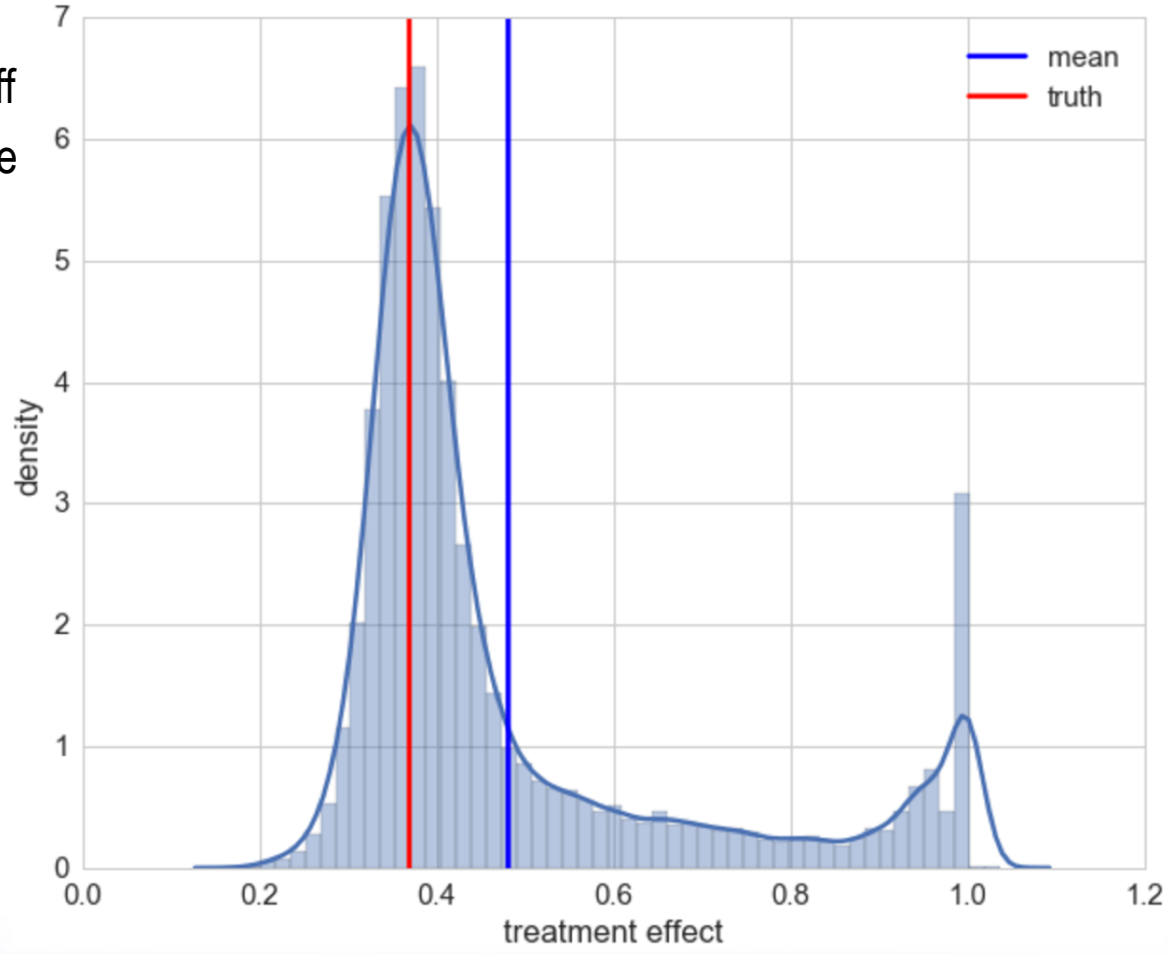
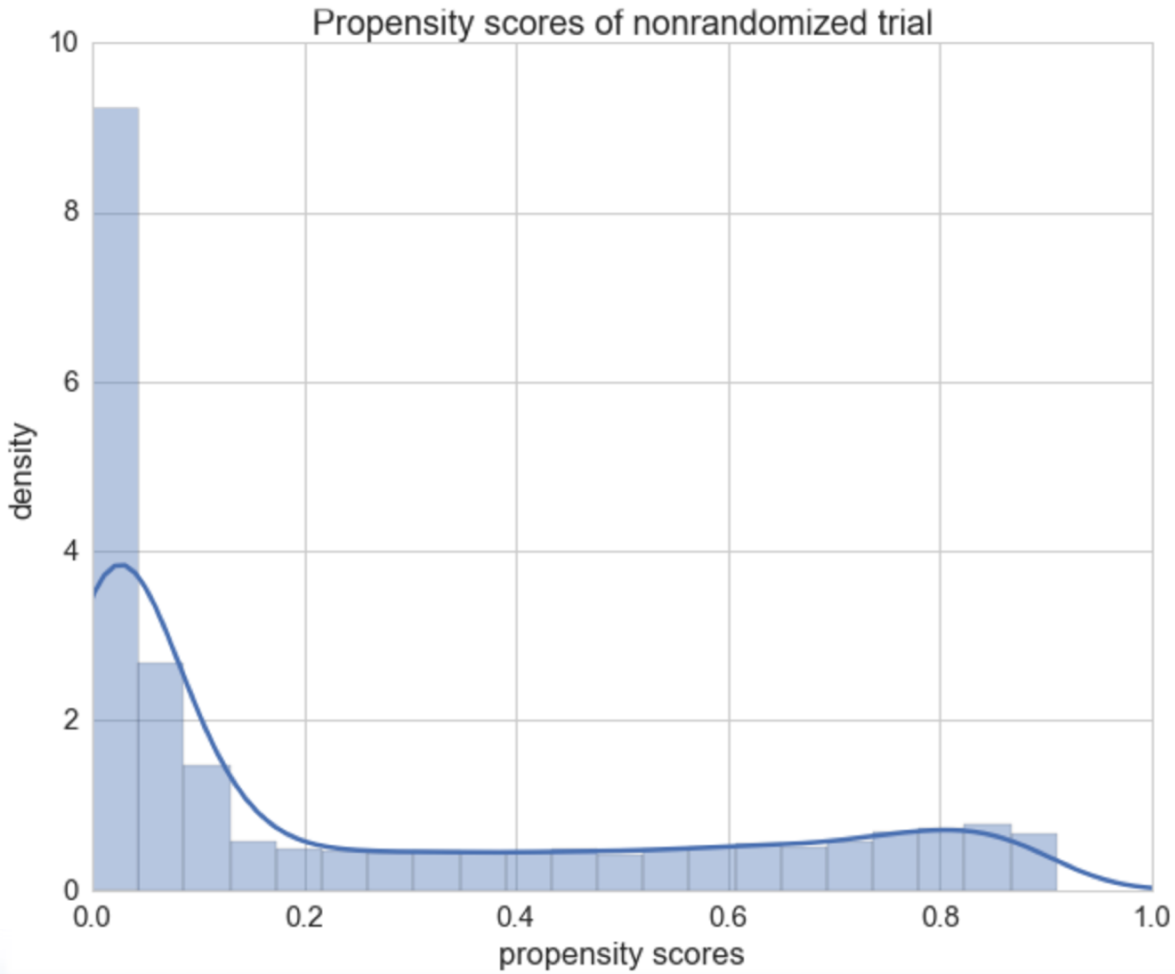
Random forest in non-randomized trial

- Average causal effect is quite off
- Quite many individual effects are estimated too high or the treatment effect too low resp.



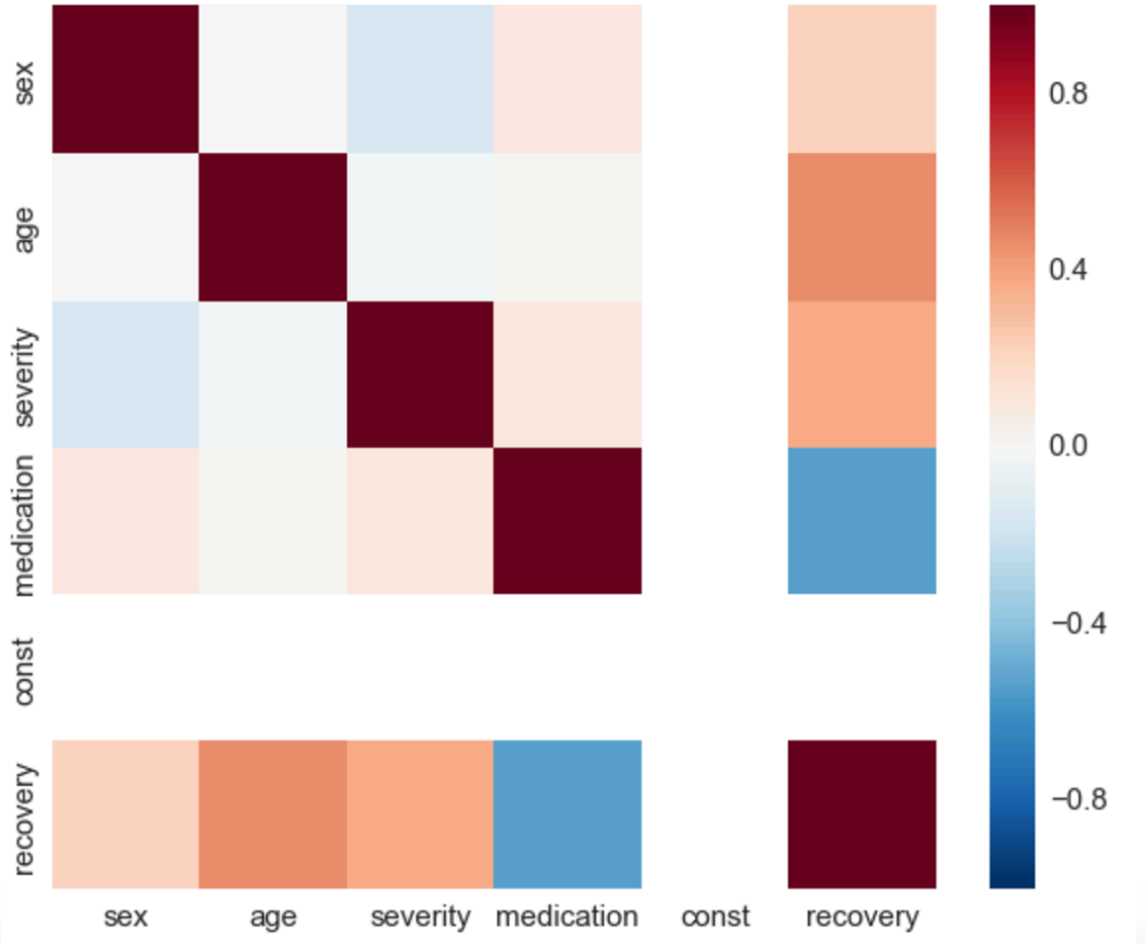
Propensity scores in non-randomized trial

Calculation of the propensity scores in order to get the weights for the samples.



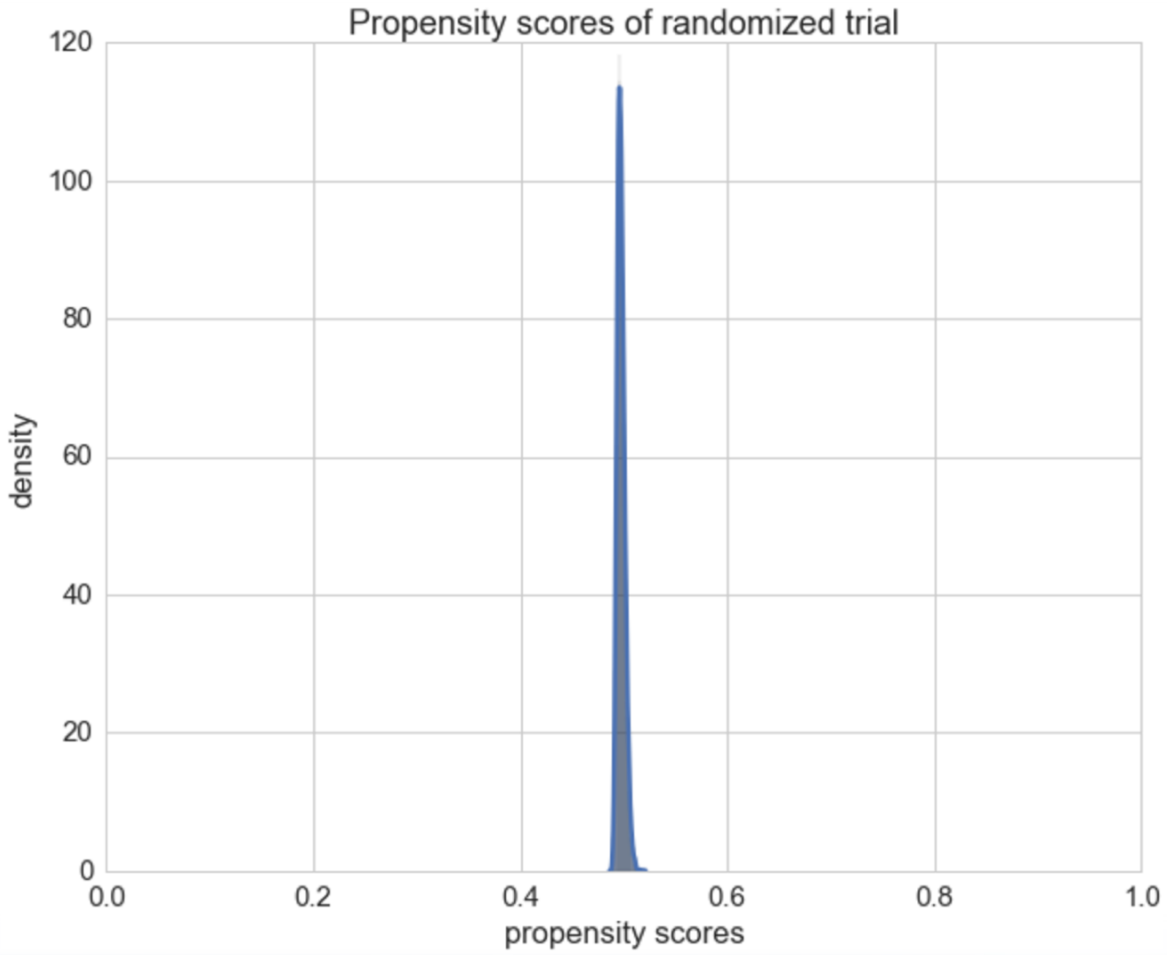
Correlation matrix weighted by propensity score

By using the propensity weights feature *sex* and *severity* are no longer as strongely correlated as before.



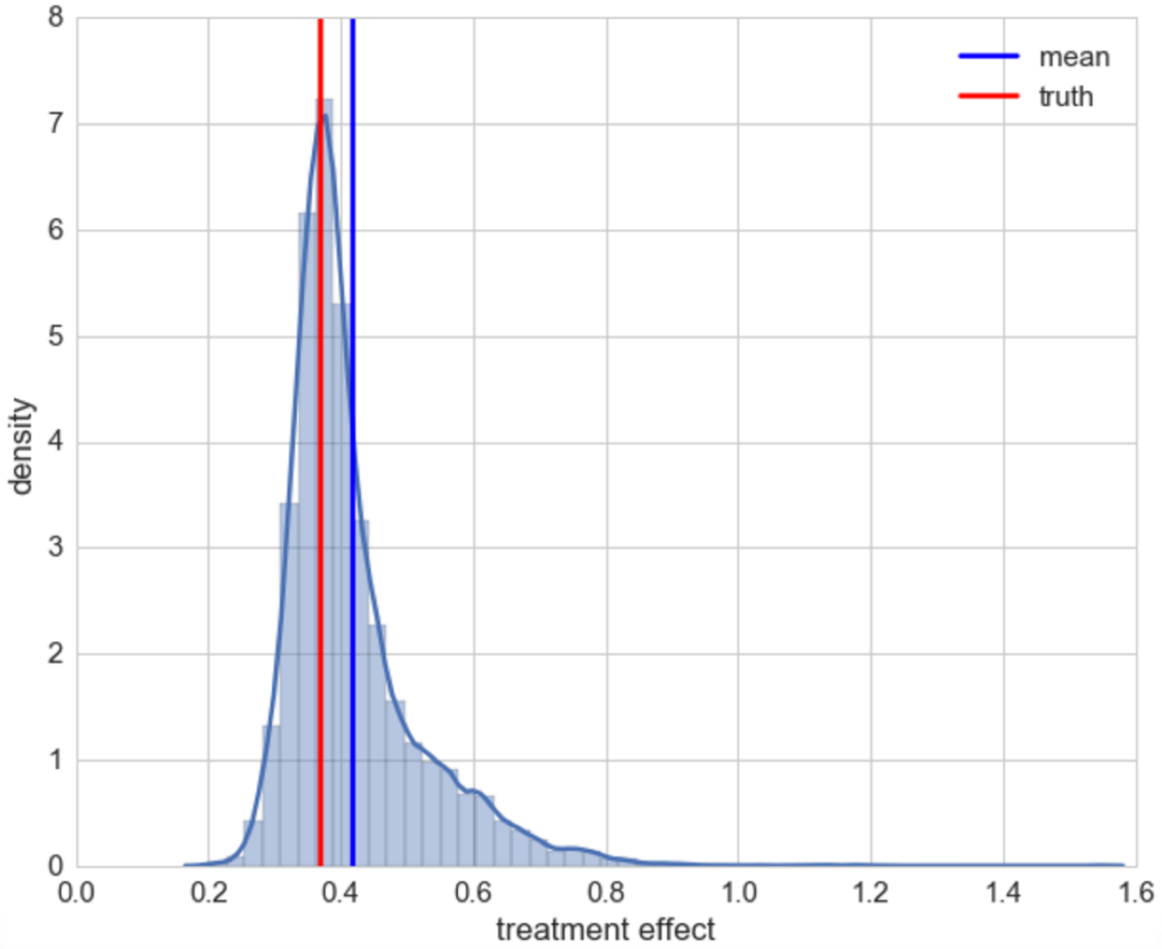
Propensity score in the randomized trial

- for comparison the propensity score in a randomized trial
- propensity score is $\frac{1}{2}$ as expected



Random forest with propensity

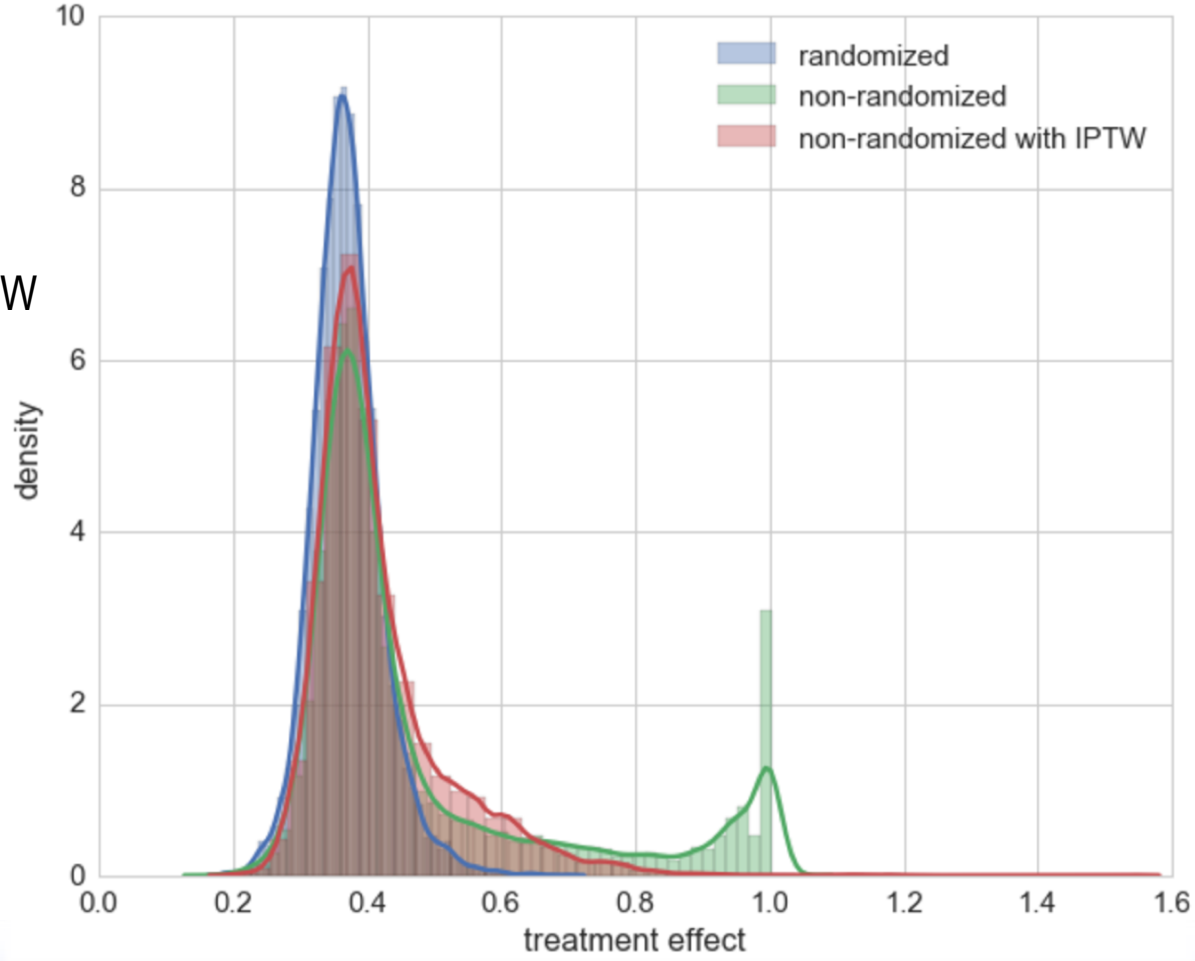
- estimation of the average causal effect improved
- estimation of individual causal effects improved in most cases but some outliers



Comparison

treatment effects for:

- randomized trial
- non-randomized trial
- non-randomized trial with IPTW



Conclusion

- IPTW improves estimating the causal effect in an observational trial compared to a naive approach.
- Postulating a model, e.g. Poisson regression, works for data from observational trials but is a **bold** assumption.
- Randomized trials remain gold standard, use it whenever possible.
- Further improvements can be accomplished by using the do feature only in a residual training.

References

- **E. Stuart**; The why, when, and how of propensity score methods for estimating causal effects; Johns Hopkins Bloomberg School of Public Health, 2011
- **Paul R. Rosenbaum, Donald B. Rubin**; “The Central Role of the Propensity Score in Observational Studies for Causal Effects”; Biometrika, Vol. 70, No. 1., Apr., 1983, pp. 41-55
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Questions?