

Data Science Lab - 5

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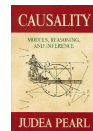
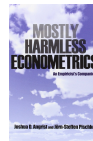
Causal Inference - Introduction

Why Causal Inference?

- Correlation is not causation: simple correlations can lead to misguided policies
- Among many different options, important to choose the *most effective* intervention
- Accurate cost-benefit analysis

Causality Frameworks

- Rubin Causal Model (Imbens & Rubin, 2015)
- Angrist & Pischke (2009)
- Pearl (2000)



RCM (1980): Potential Outcomes Framework



RCM (1980): Set Up

- Rubin's potential outcome framework (1974):
 - Given a set of N units, indexed by $i = 1, \dots, N$. Let W_i be the binary indicator of the reception of the treatment:

$$W_i \in \{0, 1\}$$

- Given this notation and SUTVA we can postulate the existence of a pair of potential outcomes for each unit:

$$Y_i^{obs} = Y_i(W_i) = \begin{cases} Y_i(0) & \text{if } W_i = 0 \\ Y_i(1) & \text{if } W_i = 1 \end{cases}$$

- We can define the Causal Effect as a simple difference between the potential outcome under treatment and under control:

$$\tau_i = Y_i(1) - Y_i(0)$$

RCM (1974): Science World

- Imagine that we want to assess the effect (*causal effect*) of a job training (*treatment*) on a pool of students (*units*)

ID	Education X_i	Treated W_i	No job training $Y_i(0)$	Job training $Y_i(1)$	Treatment effect $\tau_i = Y_i(1) - Y_i(0)$
1	High school	0	0	1	1
2	High school	1	0	1	1
3	High school	1	1	1	0
4	College	1	1	1	0
5	College	0	1	1	0
6	College	0	0	1	1

- Average Treatment Effect (ATE):

$$\begin{aligned}
 \bar{\tau} &= \bar{Y}(1) - \bar{Y}(0) \\
 &= 1 - 0.5 \\
 &= 0.5
 \end{aligned}$$

RCM (1974): Real World

ID	Education X_i	Treated W_i	No job training $Y_i(0)$	Job training $Y_i(1)$	Treatment effect $\tau_i = Y_i(1) - Y_i(0)$
1	High school	0	0	?	?
2	High school	1	?	1	?
3	High school	1	?	1	?
4	College	1	?	1	?
5	College	0	1	?	?
6	College	0	0	?	?

- Average Treatment Effect:

$$\bar{\tau} = 0.66$$



32% bigger: why this bias?

Selection Bias (intuition)

- People do not randomly select into various programs which we would like to evaluate

ID	Education X_i	Treated W_i	No job training $Y_i(0)$	Job training $Y_i(1)$	Treatment effect $\tau_i = Y_i(1) - Y_i(0)$
1	High school	0	0	?	?
2	High school	1	?	1	?
3	High school	1	?	1	?
4	College	1	?	1	?
5	College	0	1	?	?
6	College	0	0	?	?



Higher treatment rate & higher treatment effects: $W_i \not\perp Y_i(0), Y_i(1)$

Selection Bias (mathematical intuition)

- As noted above, simply comparing those who are and are not treated may provide a misleading estimate of a treatment effect
- This problem can be efficiently described by using mathematical expectation notation to denote population averages:

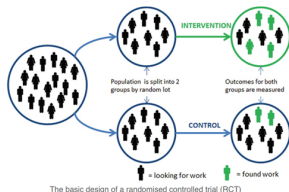
$$\begin{aligned}\bar{\tau} &= \mathbb{E}[Y_i(1)|W_i = 1] - \mathbb{E}[Y_i(0)|W_i = 0] \\ &= \underbrace{\mathbb{E}[Y_i(1) - Y_i(0)|W_i = 1]}_{\text{Average Treatment Effect on the Treated}} + \underbrace{[\mathbb{E}[Y_i(0)|W_i = 1] - \mathbb{E}[Y_i(0)|W_i = 0]]}_{\text{Selection bias}}\end{aligned}$$

- Thus, the naive contrast can be written as the sum of two components, ATET, plus Selection Bias
- Average earnings of non-trainees, $\mathbb{E}[Y_i(0)|W_i = 0]$, may not be a good standing for the earnings of trainees had they not been trained, $\mathbb{E}[Y_i(0)|W_i = 1]$

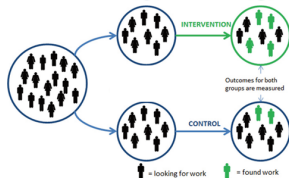
Possible solutions

- The problem of selection bias motivates the use of:

- 1 Random assignment (ex-ante) → experimental set-up



- 2 Unconfoundedness (ex-post) → observational studies



- 3 Instrumental variable (ex-post) → observational studies

Random Assignment

- Random assignment ensures that the potential earnings of trainees had they not been trained are well-represented by the randomly selected control group

- Formally, when W_i is randomly assigned, then:

$$\mathbb{E}[Y_i|W_i = 1] - \mathbb{E}[Y_i|W_i = 0] = [Y_i(1) - Y_i(0)|W_i = 1] = E[Y_i(1) - Y_i(0)]$$

- Replacing $E[Y_i|W_i = 1]$ and $E[Y_i|W_i = 0]$ with the corresponding sample analog provides a consistent estimate of ATE

Unconfoundedness (or CIA)

- The Unconfoundedness assumption states that conditional on observed characteristics, the selection bias disappears
- Formally, we overcome the problem that we have seen at slide 9, because: $W_i \perp\!\!\!\perp Y_i(0), Y_i(1) | X_i$
This holds true even if conditioning just on:

$$e(x) = P(W = 1 | X_i = x)$$

- Given unconfoundedness, comparison of average effects of job training have a **causal** interpretation:

$$\bar{\tau} = \mathbb{E}[Y_i(1) | W_i = 1, X_i] - \mathbb{E}[Y_i(0) | W_i = 0, X_i] = \mathbb{E}[Y_i(1) - Y_i(0) | X_i]$$

- This can be generalized to the case of a continuous treatment variable (i.e effects of education on employment): $s_i \perp\!\!\!\perp Y_{s_i} | X_i$
- Conditional on X_i , what is the average causal effect of a one-year increase in collage attendance?

$$\mathbb{E}[Y_i | s_i = s, X_i] - \mathbb{E}[Y_i | s_i = s - 1, X_i] = \mathbb{E}[f_i(s) - f_i(s - 1) | X_i]$$

Using regression to summarize

- Since s_i takes on many values, there are as many average causal effects as the possible increments in s_i
- Regression is a weighted average of the individual specific difference: $f_i(s) - f_i(s - 1)$
- Then if we assume that:

$$f_i(s) = Y_i = \underbrace{\beta_0 + \beta_1 s + \eta_i}_{\eta_i = X_i^T \gamma + \epsilon_i \text{ where } \mathbb{E}[\eta_i] = X_i^T \gamma}$$

- If unconfoundedness holds:

$$\mathbb{E}[f_i(s)|X_i, s_i] = \mathbb{E}[f(s_i)|X_i] = \beta_0 + \beta_1 s + \mathbb{E}[\eta_i] = \beta_0 + \beta_1 s + X_i^T \gamma$$

- If we assume ϵ_i to be uncorrelated with s_i and X_i then β_1 is the *causal effect*
- Yet, note that we assume that observable characteristics X_i are the only reason why s_i and η_i are correlated

Omitted Variable Bias

- Let $Y_i = \beta_0 + \beta_1 s_i + A_i^T \gamma + e_i$ where the vector A_i^T stands for *ability* (factors such as intelligence, motivation etc)
- If unconfoundedness holds given A_i then β_1 is the coefficient of a linear causal model
- But what happens if we cannot measure A_i i.e. we leave it out of the regression?

$$\frac{\text{Cov}(Y_i, s_i)}{\text{Var}(s_i)} = \frac{\text{Cov}([\beta_0 + \beta_1 s_i + A_i^T \gamma + e_i], s_i)}{\text{Var}(s_i)} = \underbrace{\beta_1}_{\text{causal effect}} + \underbrace{\gamma^T \delta_{A_s}}_{\text{OV bias}}$$

where δ_{A_s} is the vector of coefficients from the regression of A_i on s_i

- Take home message: the more regressor you include (longer regression), the less your causal estimand is biased

Machine Learning and Causality

Using CART to estimate heterogenous causal effect

Machine Learning and Causality

- Econometrics/ Statistics/ Social Science
 - Formal theory of causality
 - Potential outcomes methods (Rubin) maps onto economic approaches
 - Well-developed and widely used tools for estimation and inference of causal effect in experimental and observational studies
 - Used by social science, policy-makers, development organizations, medicine, business, experimentation
 - Weaknesses
 - Non-parametric approaches fail with many covariates
 - Model selection unprincipled

Motivations

- Experiments and Data-Mining
 - Concerns about ex-post “data-mining”
 - In medicine, scholars are required to pre-specify analysis plan (similar in economic field experiments)
- How is it possible to deal with sets of treatment effects among subsets of the entire population?
- Estimate of treatment effect heterogeneity needed for optimal decision-making

Definition 1 (Athey and Imbens, 2015; 2016)

- 1 Estimating heterogeneity by features in causal effects in experimental or observational studies
- 2 Conduct inference about the magnitude of the differences in the treatment effects across subsets of the population

Causal Inference Framework

- Causal inference in observational studies:
 - As we saw previously, assuming unconfoundedness to hold, we can treat observations as having come from a randomized experiment
 - Therefore we can define the conditional average treatment effect (CATE) as follows:

$$\tau(x) = E[Y_i(1) - Y_i(0) | X_i = x]$$

- The population average treatment effect then is:

$$\tau^p = E[Y_i(1) - Y_i(0)] = E[\tau(X_i)]$$

Why is CATE important?

- There are a variety of reasons that researchers wish to conduct estimation and inference on $\tau(x)$:
 - ④ It may be used to assign future units to their optimal treatment (in presence of different levels of the treatment):

$$W_i^{opt} = \max \tau(X_i)$$

- ② If we don't pre-specify the sub-populations it can be the case that the overall effect is negative, but it can be positive on subpopulations, then:

$$W_i^{PTE} = \mathbf{1}_{\tau(X_i) \geq 0}$$

e.g.: treatment is a drug \rightarrow prescribe it just to those who benefit from it

Using Trees to Estimate Causal Effects

Athey and Imbens (2015; 2016) propose 3 different approaches:

- Approach I: Analyze two groups separately:
 - Estimate $\hat{\mu}(1, x)$ using dataset where $W_i=1$
 - Estimate $\hat{\mu}(0, x)$ using dataset where $W_i=0$
 - Perform within group cross-validation to choose tuning parameters
 - Predict $\hat{\tau} = \hat{\mu}(1, x) - \hat{\mu}(0, x)$
- Approach II: Estimate $\mu(w, x)$ using just one tree:
 - Estimate $\hat{\mu}(1, x)$ and $\hat{\mu}(0, x)$ using just one tree
 - Perform within tree cross-validation to choose tuning parameters
 - Predict $\hat{\tau} = \hat{\mu}(1, x) - \hat{\mu}(0, x)$
 - Estimate is zero for x where tree does not split on w

The CATE Transformation of the Outcome

- 1 The authors' goal is to develop an algorithm that generally leads to an accurate approximation of $\hat{\tau}$ the Conditional Average Treatment Effect.
 - 1 Ideally we would measure the quality of the approximation in terms of goodness of fit using the MSE:

$$Q^{infeas} = \frac{1}{N} \sum_{i=1}^N (Y_i(1) - Y_i(0) - \hat{\tau}(X_i))^2$$

- 2 We can address this problem of infeasibility by transforming the outcome using the treatment indicator W_i and $e(X)$:

$$Y_i^* = Y_i^{obs} \cdot \frac{W_i - e(X_i)}{(1 - e(X_i)) \cdot e(X_i)}$$

- 3 Then:

$$E[Y_i^* | X_I = x] = \tau(x)$$

How to estimate the In-Sample Goodness of fit?

- The ideal goodness of fit measure would be:

$$Q^{infeas}(\hat{\tau}) = \mathbb{E}[(\tau_i - \hat{\tau}(X_i))^2].$$

- A useful proxy that can be used for the goodness of fit measure is:

$$\mathbb{E}[\tau_i^2 | X_i \in S_j] = \frac{1}{N} \sum_i \hat{\tau}(x_i)^2.$$

This leads to our In-sample goodness of fit function:

$$Q^{is} = -\frac{1}{N} \sum_i \hat{\tau}(x_i)^2.$$

Transformed Outcome Tree Model

• Approach 3:

① Model and Estimation

- Model Type: Tree structure
- Estimator $\hat{\tau}_i^{TOT}$: sample average treatment effect within leaf

② Criterion function (for fixed tuning parameter λ)

- In-sample Goodness-of-fit function:

$$Q^{is} = -MSE = -\frac{1}{N} \sum_{i=1}^N (\hat{\tau}_i^{TOT})^2$$

- Structure and use of criterion:

$$Q^{crit} = Q^{is} - \lambda \times \text{leaves}$$

- Select member of set of candidate estimators that maximizes Q^{crit} , given λ

③ Cross-validation approach

- Out-of-Sample Goodness-of-fit function:

$$Q^{oos} = -MSE = -\frac{1}{N} \sum_{i=1}^N (\hat{\tau}_i^{TOT} - Y_i^*)^2$$

- Approach: select tuning parameter λ with highest Q^{os}

Critique to the TOT approach

- Transformation of the Outcome in a randomized set-up:

$$Y_i^* = Y_i^{obs} \cdot \frac{W_i - p}{(1 - p) \cdot p} = \begin{cases} \frac{1}{p} \cdot Y_i^{obs} & \text{if } W_i = 1 \\ -\frac{1}{1 - p} \cdot Y_i^{obs} & \text{if } W_i = 0 \end{cases}$$

- Within a leaf the sample average of Y_i^* is not the most efficient estimator of treatment effect
- The proportion of treated units within the leaf is not the same as the overall sample proportion
- We use a weighted estimator similar to the Hirano, Imbens and Ridder (2003) estimator

Causal Tree Approach

- In details the Treatment Effect in a generic leaf \mathbb{X}_j is:

$$\tau^{CT}(X_i) = \frac{\sum_{j: X_j \in \mathbb{X}_j} Y_i^{obs} \cdot \frac{W_i}{\hat{e}(X_i)}}{\sum_{j: X_j \in \mathbb{X}_j} \frac{W_i}{\hat{e}(X_i)}} - \frac{\sum_{j: X_j \in \mathbb{X}_j} Y_i^{obs} \cdot \frac{(1-W_i)}{(1-\hat{e}(X_i))}}{\sum_{j: X_j \in \mathbb{X}_j} \frac{(1-W_i)}{(1-\hat{e}(X_i))}}$$

- This estimator is a consistent estimator of:

$$\tau_{\mathbb{X}_j} = \mathbb{E}[Y_i(1) - Y_i(0) | X_i \in \mathbb{X}_j]$$

- The variance can be estimated the Neyman estimator:

$$\hat{\mathbb{V}}_{Neyman} = \frac{s_t^2}{N_t} + \frac{s_c^2}{N_c}$$

These two quantities can be estimated as:

$$s_{t,j}^{te,2} = \frac{1}{N_t - 1} \sum_{i: W_i=1} (Y_i(1) - \bar{Y}_t^{obs})^2 = \frac{1}{N_t - 1} \sum_{i: W_i=1} (Y_i - \bar{Y}_t^{obs})^2$$

$$s_{c,j}^{te,2} = \frac{1}{N_c - 1} \sum_{i: W_i=0} (Y_i(0) - \bar{Y}_c^{obs})^2 = \frac{1}{N_c - 1} \sum_{i: W_i=0} (Y_i - \bar{Y}_c^{obs})^2$$

Attractive features of Causal trees

- 1 Can easily separate tree construction from treatment effect estimation
- 2 Tree constructed on training sample is independent of sampling variation in the test sample
- 3 Holding tree from training sample fixed, can use standard methods to conduct inference within each leaf of the tree on test sample
- 4 Can use any valid method for treatment effect estimation, not just the methods used in training
- 5 Simulations run by the authors show that the Causal Tree Algorithm outperforms the ST, TT and TOT approaches

Case Study

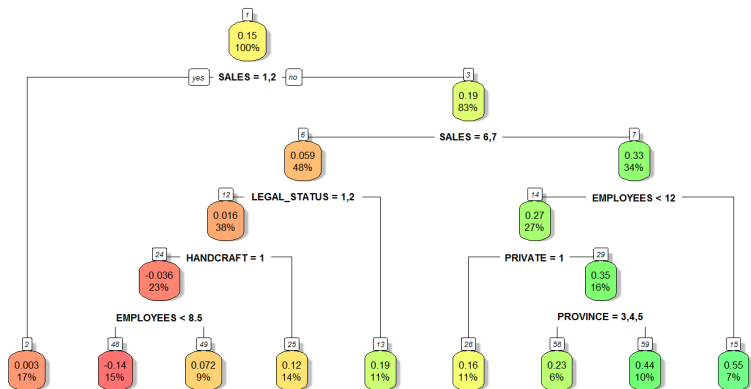


Figure: Bargagli-Stoffi & Gnecco (2020)

Causal Forests

An individual tree can be *noisy* as we saw in the last lecture → instead, fit a causal forest

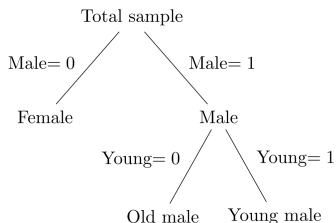
- 1 Draw a sample of size s
- 2 Split into a \mathcal{D} and \mathcal{I} sample
- 3 Grow a tree on \mathcal{D}
- 4 Estimate the effects on \mathcal{I}

Repeat many times

- Pros:
 - 1 Consistency for true $t(x)$
 - 2 Asymptotic normality
 - 3 Asymptotic variance is estimable
- Cons:
 - 1 Require sample splitting
 - 2 Large samples for asymptotic properties
 - 3 Not interpretable

Causal rules and interpretability

- In a causal scenario, interpretability can be defined as the ability of the algorithm to identify the subgroups where the effects are heterogeneous
- Decision rules are simple *if-then* statements regarding several conditions
- Rule-based learning improves interpretability

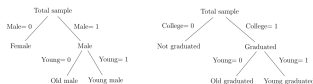


- Causal Rule Ensemble (CRE) algorithm (Lee, Bargagli-Stoffi and Dominici, 2020)

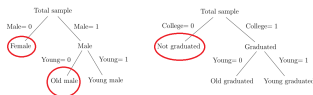
Intuition on CRE

Intuition on the CRE algorithm (5 steps):

- 1 Divide the overall sample into a *discovery* and *estimation* sample
- 2 Estimate the unit-level treatment effect $\tau^d(x)$ (where $X_i = x$)
- 3 On the *discovery* build a series of causal rules by regressing $\tau^d(x)$ on X_i using random forest (Breiman, 2001) and gradient trees (Friedman, 2001)



- 4 Select the *most important* rules using stability selection (Meinshausen and Bühlmann, 2010)



- 5 On the *estimation* sample estimate the treatment effects by regressing the estimated unit level treatment effects $\tau^e(x)$ on the selected rules

Conclusions

- ① The main problem to face is the absence of a *ground truth* when we deal with causal inference problems
- ② The approaches developed are strongly data-driven: selection of subpopulation is optimized by the algorithm
- ③ Work well with randomized experiments and some techniques (i.e., BCF, CRE) control for potential confounding bias
- ④ The approaches are tailored for applications where:
 - ① there may be many attribute relative to the number of units observed (*fat-data*)
 - ② the functional form of the relationship between treatment effects and the attributes of units is not known

Further Readings



S.Athey, G.Imbens *Machine learning methods for estimating heterogeneous causal effects*, 2015



S.Athey, S.Wager *Estimation and Inference of Heterogeneous Treatment Effects using Random Forest*, 2015



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T.J. Hastie, R.J. Tibshirani, J.H. Friedman. *The Elements of Statistical Learning*. Springer, New York, 2009



K.P. Murphy. *Machine Learning. A Probabilistic Perspective*. The MIT Press, Cambridge, Massachusetts, 2012