

# Notes on Rubin 2005: Causal Inference Using Potential Outcomes: Design, Modeling, Decisions

Gray Stanton and Austin Ellingworth

October 20, 2020

## Discussion Questions

- Rubin points out that “causal inference is impossible without making assumptions.” As an researcher, how do you determine if your assumptions are too weak, too strong, or just right?
- By incorporating prior information and making stronger modeling assumptions, the Bayesian framework for causal inference that Rubin outlines seems to be able to go beyond just making assumptions on the assignment mechanism. What are the advantages and disadvantages of this approach?
- As Rubin points out in his example about concomitant variables, even very smart people can be misled by questions of causality. How does this manifest itself presently in research involving estimation of causal effects?

## 2. The Causal Estimand – ”The Science”

- Importance of understanding precisely the quantity you are interested in – the causal estimand.
- Basic example:  $N$  units (particular objects, treatment receivers), covariates  $X$  which cannot be affected by treatment. Potential outcomes:  $Y_i(1)$  and  $Y_i(0)$  which is the value for the outcome variable in the universe where the  $i$ th unit received the active treatment versus the control treatment.
- The ”Science” is contained in the  $X, Y(1), Y(0)$ .
- Unit level causal effects  $Y_i(1) - Y_i(0)$ .
- Fundamental problem of causal inference: Cannot observe both  $Y_i(1)$  and  $Y_i(0)$  because time cannot be unwound.
- Summarizing causal effects, e.g. mean unit level causal effect for some subset of the indices.
- Causal effect must be comparison of  $\{Y_i(1), i \in S\}$  with  $\{Y_i(0), i \in S\}$ . Can’t compare different sets of units.

- Important assumption: SUTVA (Stable Unit Treatment Value Assumption). Two parts: no interference between units i.e.  $Y_i(1)$  and  $Y_i(0)$  is not affected by the treatment assigned to the other units. No hidden treatment versions: active treatment on unit  $i$  always leads to  $Y_i(1)$
- Third underlying assumption: the science (both in covariates and outcomes) are not affected by how or whether the subjects try to learn about about it.
- "Causal inference is impossible without making assumptions, they are the strand which links statistics to science".

### 3. Fisher and Neyman on the Potential Outcomes Notation in Randomized Experiments and Beyond

- Importance of potential outcome notation.
- Average causal effect,  $\sum_{i=1}^N \frac{Y_i(1) - Y_i(0)}{n}$  is important causal estimand. Difference in observed treatment means is unbiased estimator of it and  $\frac{s_1^2}{n_1} + \frac{s_2^2}{n_2}$  is a positively biased estimator of its variance (Neyman).
- Fisher Sharp Null Hypothesis: For each unit, the treatment does exactly nothing. That is,  $Y_i(1) = Y_i(0)$  for all  $i$ . Sharp because under it, all potential outcomes are known for all units, regardless of the actual assignment.
- Fisher 1918 passage.
- Terminology alert: "Counterfactuals" versus "Potential Outcomes"

### 4. The Assignment Mechanism

- Assignment Mechanism: A method for assigning treatments to units, which creates missing potential outcomes.
- $W_i$  is the assignment for unit  $i$ . The only random quantity in this setup.
- Assignment mechanism is an assignment of the conditional probability  $P(W|X, Y(1), Y(0))$ .
- $E(\bar{y}_1 - \bar{y}_0 | X, Y(1), Y(0)) = \bar{Y}(1) - \bar{Y}(0)$  and  $V(\bar{y}_1 - \bar{y}_0) \geq E\left(\frac{s_1^2}{n_1} + \frac{s_2^2}{n_2} | X, Y(1), Y(0)\right)$  (Neyman).
- Random experiments are a subset of assignment mechanisms, and under RE, it can be that SUTVA is sufficient assumption for causal inference.
- Random experiments are an ignorable non-trivial assignment mechanisms: Non-trivial in that every unit has some possibility of being assigned each treatment  $0 < P(W_i = 1 | X, Y_{obs}) < 1$ . Ignorable in that potential outcomes don't affect assignment mechanism probabilities, just the observed outcomes:  $P(W|X, Y(0), Y(1)) = P(W|X, Y_{obs})$ .

- In sequential experiments, that's when dependence on  $Y_{obs}$  arises. Classical random experiments are unconfounded  $P(W|X) = P(W|X, Y_{obs})$ .
- Collapsing potential outcomes into just  $Y_{obs}$  mixes up science and what we to try and learn about science.
- Only a model on the assignment mechanism still allows for progress on statistical inference, even for observational studies (propensity scores).

## 5. Models on the Science

- However models on science can be have a critical role (Bayesian inference).
- Models only on assignment mechanism are more robust (but still require SUTVA), models on science allow for handling greater complexity and logical summaries of results.
- Using bayesian framework to create posterior predictive distribution for the missing half of the potential outcomes.
- Using  $P(X, Y(1), Y(0))$  model for the science +  $P(W|X, Y(0), Y(1))$  assignment mechanism, then can find PPD for  $P(Y_{mis}|X, Y_{obs}, W) \propto P(X, Y(1), Y(0))P(W|X, Y_{obs})$
- Can calculate distribution of any causal estimand, which is function of  $X, Y(1), Y(0)$ .
- Randomized Experiments have impact on bayesian inference: Any distribution on the science will be exchangeable. Ignorable means that second term is a constant.
- "Rubin's Causal Model" – Extends potential outcomes to all situations, explicitly includes assignment mechanisms with possible dependence on all potential outcomes, embeds assignment based + bayesian likelihood in common framework.

## 6. Decisions: Based on current knowledge of science and on costs of decisions

- Posterior distribution of causal estimands is "Summary of current knowledge of the science" from current data and past science (prior dist).
- Fisher perspective: unknown to what purpose discoveries will be put, so should not introduce cost functions.
- Likelihood function vs significance tests vs accept/reject tests.

## 7 Complex Experiments: "Direct" and "Indirect" Causal Effects

- Concomitant random variable: Outcome variable not of primary interest, but is on causal pathway from treatment to primary outcome. Not a covariate, but may want to adjust for it.
- Fisher suggestion: Analyze  $Y_{obs}$  via  $W$  and the concomitant  $C_{obs}$  with ANCOVA, but this is equivalent to regressing  $Y_{obs}$  on  $W, C_{obs}$ , which he called naive earlier. Compares  $Y_i(1)$  with  $Y_i(0)$  for those with common level of  $C_{obs}$  which is not a causal effect because  $C_{obs}$  is affected by treatment.
- Two thought experiments: When treatment impacts  $C$  but not  $Y$  i.e. no direct impact of treatment on outcome after adjusting for  $C$ , but if we condition on  $C_{obs}$ , actually appears that treatment plots do worse. Problem still arises even when there is direct treatment effect after adjusting for  $C$ .
- Controlling for  $C_{obs}$  essentially breaks ignorability of treatment assignments.
- Fisher's ANCOVA is predicated on ignorability of assignment bc assumes set of subjects with fixed  $C_{obs}$  are randomly assigned treatment/control, which is not the case.
- Rubin posits that combining  $Y$  and  $C$  into 1 variable (i.e.  $Y / C$ ) for treating  $(C, Y)$  as a bivariate outcome is the better route for co-contaminants.