Week 2: Randomized Experiments

PLSC 30600 - Causal Inference

Identifying causal effects

Last week

- We defined fundamental causal quantities in terms of *potential outcomes*
  - The individual treatment effect $\tau_i = Y_i(1) - Y_i(0)$
- Why can we not identify individual treatment effects (w/o strong assumptions)?
  - Fundamental problem of causal inference!
  - Observed data only reveal $Y_i(1)$ or $Y_i(0)$ (under consistency)

This week

- What assumptions do we need to identify treatment effects from the data?
  - Consistency + positivity aren't enough!
- We need some statement about how the treatment was assigned.
  - Ignorability/exogeneity: $\{Y_i(1), Y_i(0)\} \perp\!\!\!\perp D_i$
- Why does a randomized experiment get us this assumption by design?

Identifying the ATT

- Suppose we want to identify the Average Treatment Effect on the Treated (ATT)

$$\tau_{\text{ATT}} = E[Y_i(1) - Y_i(0) \mid D_i = 1]$$

- Let's see what our consistency/SUTVA assumption gets us!
- First, let's use linearity:

$$\tau_{\text{ATT}} = E[Y_i(1) \mid D_i = 1] - E[Y_i(0) \mid D_i = 1]$$

- Next, consistency

$$\tau_{\text{ATT}} = E[Y_i \mid D_i = 1] - E[Y_i(0) \mid D_i = 1]$$

Identifying the ATT

- Still not enough though. We have an *unobserved* term $E[Y_i(0) | D_i = 1]$. Why can't we observe this directly?

$$\tau_{\text{ATT}} = E[Y_i | D_i = 1] - E[Y_i(0) | D_i = 1]$$

- Let's see what the difference would be between the ATT and the simple difference-in-means $E[Y_i | D_i = 1] - E[Y_i | D_i = 0]$. Add and subtract $E[Y_i | D_i = 0]$

$$\tau_{\text{ATT}} = E[Y_i | D_i = 1] - E[Y_i(0) | D_i = 1] - E[Y_i | D_i = 0] + E[Y_i | D_i = 0]$$

- Rearranging terms

$$\tau_{\text{ATT}} = \Big(E[Y_i | D_i = 1] - E[Y_i | D_i = 0]\Big) - \Big(E[Y_i(0) | D_i = 1] - E[Y_i | D_i = 0]\Big)$$

Identifying the ATT

- Now we have an expression for the ATT in terms of the difference-in-means and a bias term

$$\tau_{\text{ATT}} = \underbrace{\Big(E[Y_i \mid D_i = 1] - E[Y_i \mid D_i = 0]\Big)}_{\text{Difference-in-means}} - \underbrace{\Big(E[Y_i(0) \mid D_i = 1] - E[Y_i(0) \mid D_i = 0]\Big)}_{\text{Selection-into-treatment bias}}$$

- What does this bias term represent? How can we interpret it?
  - How much higher are the potential outcomes under control for units that receive treatment vs. those that receive control.
  - Sometimes called a **selection-into-treatment** problem -- units that choose treatment may have higher or lower potential outcomes than those that choose control.

Identifying the ATC

- We can use the same exact approach for the ATC and we get a similar expression with a slightly different selection-into-treatment bias term ($Y_i(1)$ instead of $Y_i(0)$)

$$\tau_{\text{ATC}} = \underbrace{\Big(E[Y_i \mid D_i = 1] - E[Y_i \mid D_i = 0]\Big)}_{\text{Difference-in-means}} - \underbrace{\Big(E[Y_i(1) \mid D_i = 1] - E[Y_i(1) \mid D_i = 0]\Big)}_{\text{Selection-into-treatment bias}}$$

- By law of total expectation, we could get the bias for the ATE (this is on your problem set!)

$$\underbrace{E[Y_i(1) - Y_i(0)]}_{\text{ATE}} = \underbrace{E[Y_i(1) - Y_i(0) \mid D_i = 1]}_{\text{ATT}}Pr(D_i = 1) + \underbrace{E[Y_i(1) - Y_i(0) \mid D_i = 0]}_{\text{ATC}}Pr(D_i = 0)$$

Selection-into-treatment bias

- Can use theory to "sign the bias" of the difference-in-means.
  - Suppose Y_i was an indicator of whether someone voted in an election and D_i was an indicator for whether they received a political mailer.
  - Consider a world where the mailer was sent out non-randomly to everyone who had signed up for a politician's mailing list.
  - If we took the difference in turnout rates between voters who received the mailer and voters who did not receive the mailer, would we be over-estimating or under-estimating the effect of treatment? Why?

Ignorability/Unconfoundedness

- What assumption can we make for the difference-in-means to identify the ATE?
- The selection-into-treatment bias for both the ATT and the ATC is 0

$$E[Y_i(0) \mid D_i = 1] = E[Y_i(0) \mid D_i = 0]$$

$$E[Y_i(1) \mid D_i = 1] = E[Y_i(1) \mid D_i = 0]$$

- This will be true under an assumption that treatment is assigned **independent** of the potential outcomes.

$$\{Y_i(1), Y_i(0)\} \perp\!\!\!\perp D_i$$

- Common names for this assumption: **exogeneity**, **unconfoundedness**, **ignorability**
    - In simple terms: Treatment is not systematically more/less likely to be assigned to units that have higher/lower potential outcomes.

Ignorability/Unconfoundedness

- What does ignorability give us?
- By independence

$$E[Y_i(1) | D_i = 1] = E[Y_i(1)]$$

$$E[Y_i(0) | D_i = 0] = E[Y_i(0)]$$

  - Technically we only need the above ("mean ignorability") and not full ignorability but there are few cases where we can justify former but not latter.

- Combined with consistency, we get:

$$E[Y_i | D_i = 1] = E[Y_i(1)]$$

$$E[Y_i | D_i = 0] = E[Y_i(0)]$$

- The observed data identify the ATE!

Ignorability/Unconfoundedness

- To summarize:

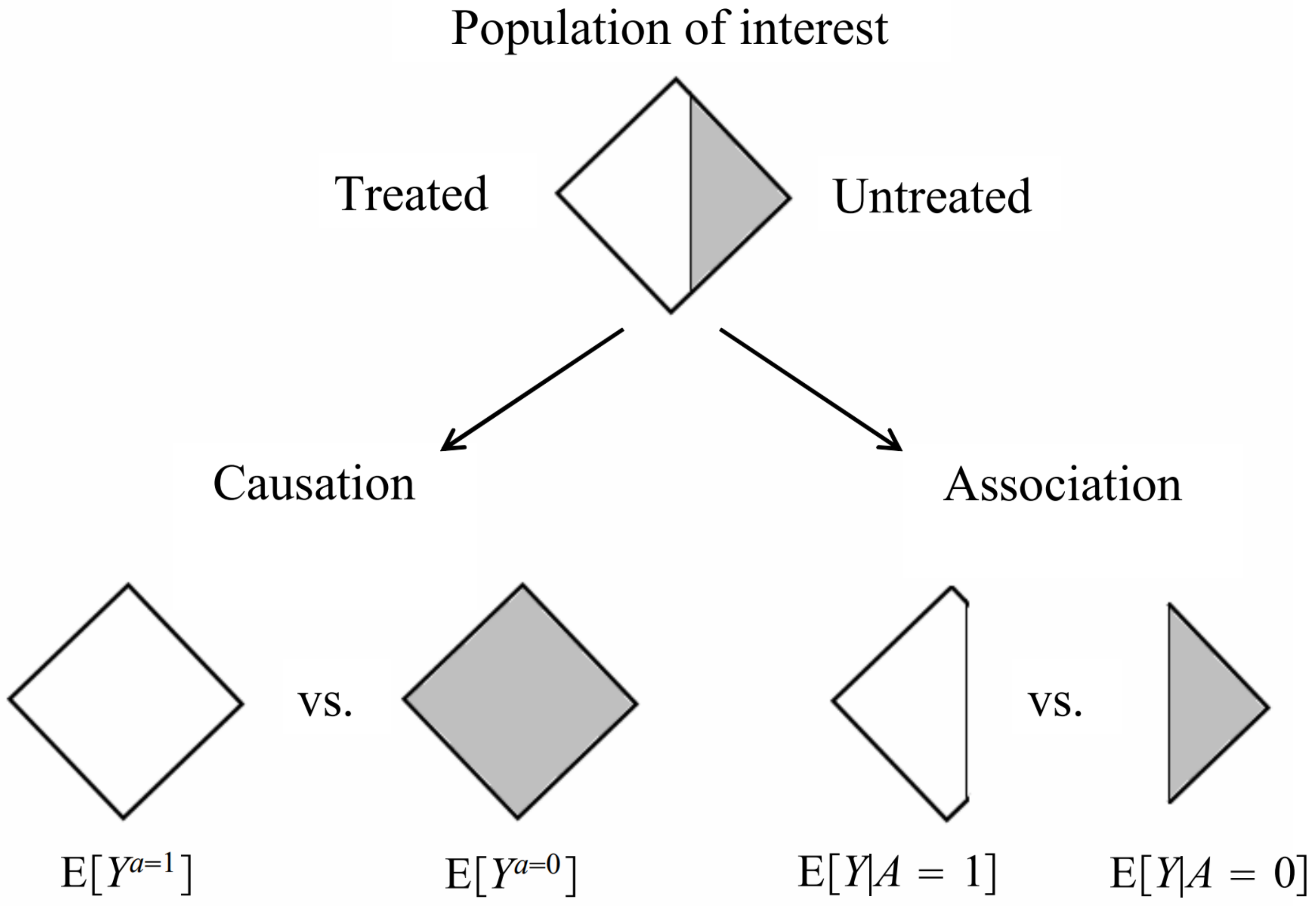
$$E[Y_i | D_i = 1] - E[Y_i | D_i = 0]$$

$$E[Y_i(1) | D_i = 1] - E[Y_i(0) | D_i = 0]$$

$$E[Y_i(1)] - E[Y_i(0)]$$

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

Ignorability/Unconfoundedness



Experiments

Randomized experiments

- What sort of research design justifies ignorability?
  - One design is a randomized experiment!
- An experiment is any study where a researcher knows and controls the treatment assignment probability $Pr(D_i = 1)$
- A randomized experiment is an experiment that satisfies:
  - Positivity: $0 < Pr(D_i = 1) < 1$ for all units
  - Ignorability: $Pr(D_i = 1 \mid \mathbf{Y}(1), \mathbf{Y}(0)) = Pr(D_i = 1)$
    - Another implication of $\mathbf{Y}(1), \mathbf{Y}(0) \perp\!\!\!\perp D_i$
    - Treatment assignment probabilities do not depend on the potential outcomes.

Types of experiments

- Lots of ways in which we could design a randomized experiment where ignorability holds:
- Let N_t be the number of treated units, N_c number of controls
- Bernoulli randomization:
  - Independent coin flips for each D_i. $Pr(D_i = 1) = p$
  - $D_i \perp\!\!\!\perp D_j$ for all i, j.
  - N_t, N_c are random variables
- Complete randomization
  - Fix N_t and N_c in advance. Randomly select N_t units to be treated.
  - Each unit has an equal probability to be treated.
  - Each assignment with N_t treated units is equally likely to occur
  - D_i is independent of potential outcomes, but treatment assignment is slightly dependent across units.

Types of experiments

- Stratified randomization
  - Using covariates X_i, form J total blocks or strata of units with similar or identical covariate values.
  - Completely randomize *within* each of the J blocks
  - If treatment probabilities are identical within each block, can analyze as though completely random.
- Cluster randomization
  - Each unit i belongs to some larger cluster. $C_i = \{1, 2, \ldots, C\}$, $C < N$.
  - Treatment is assigned at the cluster level -- randomly Select some number of clusters to be treated, remainder control.
  - If units share cluster membership, they get the same treatment ($C_i = C_j \twoheadrightarrow D_i = D_j$)

Complete randomization

- How do we do estimation and inference under complete randomization?
- We'll start with the finite-sample setting and illustrate the Neyman (1923; 1990) approach to inference for the SATE.

- Define our quantity of interest, the sample average treatment effect

$$\tau_{\text{SATE}} = \frac{1}{N}\sum_{i=1}^{N} Y_i(1) - Y_i(0)$$

- Our estimator is the sample difference-in-means.

$$\hat{\tau} = \frac{1}{N_t}\sum_{i=1}^{N} Y_i D_i - \frac{1}{N_c}\sum_{i=1}^{N} Y_i(1 - D_i)$$

Finite sample inference

- Why is $\hat{\tau}$ a random variable even when we don't assume sampling from a population?
  - Because the treatment is randomized!
  - Different randomizations would lead to different realizations of $\hat{\tau}$.
- Consider a study with $N_t = 3$, $N_c = 3$ and suppose we could see the true "table of science"
- Under one realization of the treatment $\mathbf{D}$, we have:

Unit i	Treatment D_i	$Y_i(1)$	$Y_i(0)$	Observed Y_i
1	1	5	0	5
2	0	2	-3	-3
3	1	9	4	9
4	0	4	-1	-1
5	0	1	-4	-4
6	1	2	-3	2

- For this randomization, our realization of $\hat{\tau}$ (our estimate) would be:

$$\frac{1}{3}(5+9+2) - \frac{1}{3}(-3-1-4) = 8$$

Finite sample inference

- How about another, equally likely realization?

Unit i	Treatment D_i	$Y_i(1)$	$Y_i(0)$	Observed Y_i
1	1	5	0	5
2	1	2	-3	2
3	0	9	4	4
4	0	4	-1	-1
5	0	1	-4	-4
6	1	2	-3	2

- For this randomization, our realization of $\hat{\tau}$ would be:

$$\frac{1}{3}(5+2+2) - \frac{1}{3}(4-1-4) = \frac{10}{3}$$

Finite sample inference

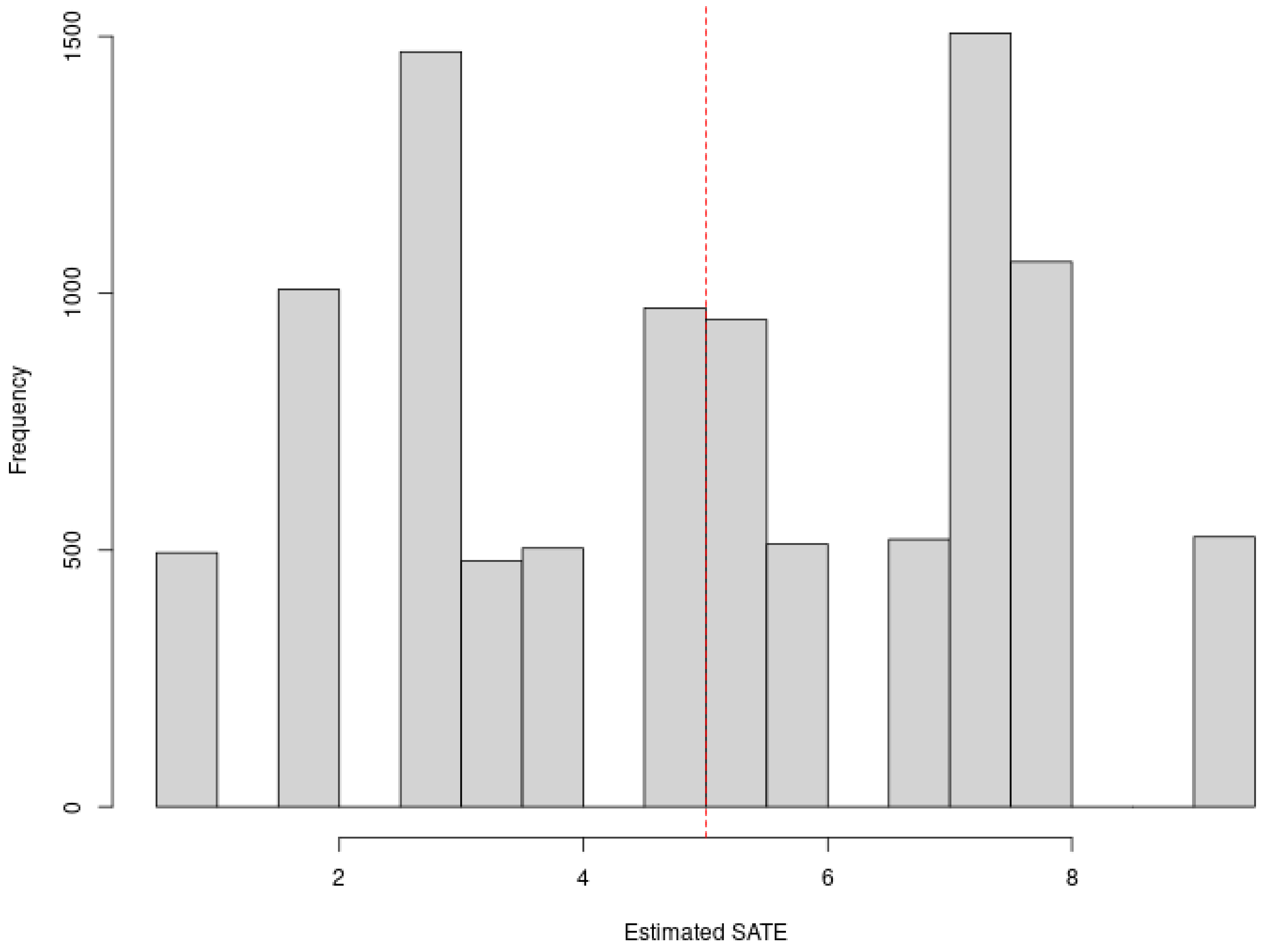
- Overall all possible randomizations, what is the distribution?
- We can run a quick simulation and find out

```
### Define the data frame
data <- data.frame(Y_1 = c(5,2,9,4,1,2),
                   Y_0 = c(0, -3, 4, -1, -4, -3))

## Simulate the sampling distribution
nIter = 10000
sate_est = rep(NA, nIter)
set.seed(60637)

for(i in 1:nIter){
  data$D = sample(rep(c(0,1), each=3))
  data$Y = data$D*data$Y_1 + (1-data$D)*data$Y_0
  sate_est[i] = mean(data$Y[data$D==1]) - mean(data$Y[data$D==0])
}
```

Finite sample inference



Finite sample inference

- Of course, in real data, we only get one estimate. Need to rely on theory to understand the *distribution* that estimate came from in order to do inference
    - Is $\hat{\tau} \mid \mathrm{Y}(1), \mathrm{Y}(0)$ unbiased for the SATE?
    - What is the sampling variance $Var(\hat{\tau})$ under a finite sample (fixed $\mathrm{Y}(1), \mathrm{Y}(0)$)?
    - What should our estimator of the sampling variance $\widehat{Var}(\hat{\tau})$ be?

Unbiasedness

- We can show that conditional on the potential outcomes $\hat{\tau}$ is unbiased for the SATE. First, by linearity of expectations

$$E[\hat{\tau} \mid \mathrm{Y}(1), \mathrm{Y}(0)] = \frac{1}{N_t}\sum_{i=1}^{N} E\left[Y_i D_i \,\middle|\, \mathrm{Y}(1), \mathrm{Y}(0)\right] - \frac{1}{N_c}\sum_{i=1}^{N} E\left[Y_i(1 - D_i) \,\middle|\, \mathrm{Y}(1), \mathrm{Y}(0)\right]$$

- By consistency $Y_i D_i = Y_i(1) D_i$ and $Y_i(1 - D_i) = Y_i(0)(1 - D_i)$

$$E[\hat{\tau} \mid \mathrm{Y}(1), \mathrm{Y}(0)] = \frac{1}{N_t}\sum_{i=1}^{N} E\left[Y_i(1) D_i \,\middle|\, \mathrm{Y}(1), \mathrm{Y}(0)\right] - \frac{1}{N_c}\sum_{i=1}^{N} E\left[Y_i(0)(1 - D_i) \,\middle|\, \mathrm{Y}(1), \mathrm{Y}(0)\right]$$

- Conditional on the potential outcomes, $Y_i(1)$ and $Y_i(0)$ are constants

$$E[\hat{\tau} \mid \mathrm{Y}(1), \mathrm{Y}(0)] = \frac{1}{N_t}\sum_{i=1}^{N} Y_i(1) E\left[D_i \,\middle|\, \mathrm{Y}(1), \mathrm{Y}(0)\right] - \frac{1}{N_c}\sum_{i=1}^{N} Y_i(0) E\left[(1 - D_i) \,\middle|\, \mathrm{Y}(1), \mathrm{Y}(0)\right]$$

Unbiasedness

- D_i has a known distribution under complete randomization and its expectation is $Pr(D_i = 1)$, which is just N_t/N

$$E[\hat{\tau} \mid \mathrm{Y}(1), \mathrm{Y}(0)] = \frac{1}{N_t}\sum_{i=1}^{N} Y_i(1)\frac{N_t}{N} - \frac{1}{N_c}\sum_{i=1}^{N} Y_i(0)\frac{N_c}{N}$$

- Pulling out the constants

$$E[\hat{\tau} \mid \mathrm{Y}(1), \mathrm{Y}(0)] = \frac{1}{N}\sum_{i=1}^{N} Y_i(1) - \frac{1}{N}\sum_{i=1}^{N} Y_i(0)$$

- Combining the sums, we have the SATE. Therefore the difference-in-means under complete randomization is unbiased for the SATE!

$$E[\hat{\tau} \mid \mathrm{Y}(1), \mathrm{Y}(0)] = \frac{1}{N}\sum_{i=1}^{N} Y_i(1) - Y_i(0)$$

Sampling variance

- What's the variance of $\hat{\tau}$ going to be (conditional on the sample)? Slightly tricky since D_i is not independent of D_j.

$$Var(\hat{\tau} \,|\, \mathrm{Y}(1), \mathrm{Y}(0)) = \frac{S_t^2}{N_t} + \frac{S_c^2}{N_c} - \frac{S_{\tau_i}^2}{N}$$

where

$$S_t^2 = \frac{1}{N-1} \sum_{i=1}^{N} \left(Y_i(1) - \overline{Y(1)} \right)^2$$

$$S_c^2 = \frac{1}{N-1} \sum_{i=1}^{N} \left(Y_i(0) - \overline{Y(0)} \right)^2$$

and

$$S_{\tau_i}^2 = \frac{1}{N-1} \sum_{i=1}^{N} \left((Y_i(1) - Y_i(0)) - (\overline{Y(1)} - \overline{Y(0)}) \right)^2$$

Sampling variance

- Can we estimate the sampling variance? Well S_t^2 and S_c^2 can be estimated from their sample analogues (just the sample variances within treated/control groups)

$$s_t^2 = \frac{1}{N_t - 1} \sum_{i:D_i=1} \left(Y_i(1) - \overline{Y}_t^{\text{obs}}\right)^2$$

$$s_c^2 = \frac{1}{N_c - 1} \sum_{i:D_i=0} \left(Y_i(0) - \overline{Y}_c^{\text{obs}}\right)^2$$

- However the $S_{\tau_i}^2$ term can't be estimated directly from the sample. Why?
  - The fundamental problem of causal inference! Can't observe individual treatment effects.

Neyman variance

- Neyman suggested just ignoring that third term and using our familiar estimator

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

- What are its properties?
  - We know it's **conservative** for the true variance since $S^2_{\tau_i} \geq 0$.
  - If treatment effects are constant, it's unbiased!
  - Confidence intervals using the Neyman standard error $\sqrt{\hat{\mathrm{V}}_{\text{Neyman}}}$ will be no smaller than they should be.

Population inference

- Suppose we don't condition on $\mathrm{Y}(1)$, $\mathrm{Y}(0)$ and are interested in the PATE.
  - The difference-in-means is still unbiased (under random sampling)!
- What happens to the true variance?
  - The $S^2_{\tau_i}$ term drops out of the variance of $\hat{\tau}$
  - Intuition: With random sampling from a target population, can think of treated group and control group as two separate N_t and N_c size samples from the population $Y_i(1)$ and $Y_i(0)$ respectively.
  - Neyman variance is unbiased for the sampling variance of the ATE in this setting (that third term goes away)

Illustration: Gerber, Green and Larimer (2008)

Social Pressure and Voter Turnout: Evidence from a Large-Scale Field Experiment

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Voter turnout theories based on rational self-interested behavior generally fail to predict significant turnout unless they account for the utility that citizens receive from performing their civic duty. We distinguish between two aspects of this type of utility, intrinsic satisfaction from behaving in accordance with a norm and extrinsic incentives to comply, and test the effects of priming intrinsic motives and applying varying degrees of extrinsic pressure. A large-scale field experiment involving several hundred thousand registered voters used a series of mailings to gauge these effects. Substantially higher turnout was observed among those who received mailings promising to publicize their turnout to their household or their neighbors. These findings demonstrate the profound importance of social pressure as an inducement to political participation.

Illustration: Gerber, Green and Larimer (2008)

- Gerber, Green and Larimer (2008) want to know what causes people to vote.
  - What sorts of encouragements will get people to turn out more or less?
- Five treatment conditions in a randomized GOTV mailer experiment:
  - No mailer (0)
  - "Researchers will be studying your turnout" mailer (Hawthorne) (1)
  - "Voting is a civic duty" mailer (Civic Duty) (2)
  - "Your and your neighbors' voting history" mailer (Neighbors) (3)
  - "Your turnout history" mailer (Self) (4)
- Gerber, Green and Larimer first analyze households. Why?
  - Is $Y_i(d)$ well-defined for an individual? Somewhat tricky - likely spillovers across household members.
  - Treatment is randomized by household.

Illustration: Gerber, Green and Larimer (2008)

```
# Load the data
data <- read_dta('assets/data/ggr_2008_individual.dta')

# Aggregate to the household level
data_hh <- data %>% group_by(hh_id) %>% summarize(treatment = treatment[1], voted = mean(voted)

# For each treatment condition, calculate N and share voting
kable(data_hh %>% group_by(treatment) %>% summarize(N = n(), voted = mean(voted)))
```

treatment	N	voted
0	99999	0.304
1	20002	0.332
2	20001	0.325
3	20000	0.389
4	20000	0.357

Illustration: Gerber, Green and Larimer (2008)

- Let's estimate the ATE of the "Neighbors" treatment relative to control

```r
# Estimated ATE of Neighbors (3) vs. Control (0)
ate_est_neighbors = mean(data_hh$voted[data_hh$treatment == 3]) -
  mean(data_hh$voted[data_hh$treatment == 0])

ate_est_neighbors
```

```
## [1] 0.0848
```

- Now let's use our Neyman variance estimator

```r
# Estimate the sampling variance
var_ate = var(data_hh$voted[data_hh$treatment == 3])/sum(data_hh$treatment == 3) +
  var(data_hh$voted[data_hh$treatment == 0])/sum(data_hh$treatment == 0)

# Square root to get estimated SE
sqrt(var_ate)
```

```
## [1] 0.0034
```

Illustration: Gerber, Green and Larimer (2008)

- 95% asymptotic confidence interval and p-value against null of no ATE.

```
# Confidence interval (assuming asymptotic normality)
ate_95CI = c(ate_est_neighbors - qnorm(.975)*sqrt(var_ate),
  ate_est_neighbors + qnorm(.975)*sqrt(var_ate))

ate_95CI
```

```
## [1] 0.0781 0.0915
```

```
# P-value H_0: \tau = 0, H_a: \tau \neq 0
p_val = 2*pnorm(-abs(ate_est_neighbors/sqrt(var_ate)))

p_val
```

```
## [1] 3.64e-137
```

Illustration: Gerber, Green and Larimer (2008)

- Fun fact: You can get this via regression! Samii and Aronow (2012) show that the Neyman variance is equal to the heteroskedasticity-robust regression w/ HC2 correction, which is the default used in the `lm_robust` routine in the `estimatr` package

```
lm_robust(voted ~ I(treatment==3), data=data_hh %>% filter(treatment == 3|treatment == 0))
```

```
##                        Estimate Std. Error t value   Pr(>|t|) CI Lower CI Upper
## (Intercept)              0.3043    0.00132   230.2  0.00e+00   0.3017   0.3069
## I(treatment == 3)TRUE    0.0848    0.00340    24.9 8.13e-137   0.0781   0.0915
##                            DF
## (Intercept)            119997
## I(treatment == 3)TRUE 119997
```

Randomization tests

Randomization inference

- Neyman framework:
  - **Estimand**: Average Treatment Effect: $\tau = E[Y_i(1) - Y_i(0)]$
  - Difference-in-means estimator: Known expectation and sampling variance
  - **Hypothesis test.** Null of no ATE $H_0 : \tau = 0$
  - Large sample/asymptotic theory to get the distribution on $\hat{\tau}$ to calculate p-values
- Fisher randomization:
  - Can we get a p-value under some null hypothesis without the large-sample assumptions?
  - What does randomization alone justify?
  - Exact p-values under a *sharp* null of no individual treatment effect $Y_i(1) = Y_i(0)$ for all i.
  - "Randomization test" or "permutation test" approach: a flexible framework for inference under any known randomization scheme

Running example - Exercise incentives

- Charness and Gneezy (Econometrica, 2008): Does paying people to go to the gym cause them to go more once the incentives run out?
- **Randomized Experiment**: 120 UChicago Undergraduates -- 40 received no incentives, 80 were given 25 dollars if they went to the gym once in the following week, 40 of those received 100 dollars if they went 8 times in the next 4 weeks
  - 3 treatment conditions; complete randomization: (Control, Small incentives, Intensive incentives)
- **Outcome**: Average weekly gym attendance after incentives ran out (6-12 weeks after start)
- Question: Did incentives affect gym attendance even after they ran out? Can incentives create habits?

Running example - Exercise incentives

- Treatment is a multi-level indicator $D_i \in \{\text{Control}, \text{Low}, \text{High}\}$

```r
# Load in exercise dataset
exercise <- read_csv("assets/data/exercise.csv")

# Make a 3-level treatment variable
exercise <- exercise %>% mutate(treatment = case_when(Control == 1 ~ "Control",
                                                       High == 1 ~ "High",
                                                       TRUE ~ "Low"))

# Summarize the data
kable(exercise %>% group_by(treatment) %>% summarize(N=n(), outcome = mean(After)))
```

treatment	N	outcome
Control	40	0.564
High	40	1.243
Low	40	0.757

Running example - Exercise incentives

- On average, we see that students receiving the "High" incentives treatment work out about .7 more times per week in that 7-week post-incentives period.
- We probably could do normal Neyman large-sample inference here and our normal approximation would be fine.
- But can we justify a p-value purely on the basis of randomization?
- Randomization testing = p-values on the basis of knowing the distribution of possible treatment assignments $\mathbf{D} = \{D_1, D_2, \ldots, D_N\}$
- Benefits:
    - Inferences are **exact** (or exact up to monte carlo simulation error)
    - Inferences are free of distributional assumptions/asymptotic theory
- Drawbacks:
    - Null hypothesis that we'll use is a bit odd: **sharp** null of no individual effects.

Hypothesis testing review

- Four steps to conducting a hypothesis test
  - Define the null hypothesis.
    - Neyman framework: $H_0 : \tau = 0$
    - Need a baseline null in order to calculate a probability for the data/test statistic
    - The probability of observing a particular value of the test statistic depends on what is "true" about the underlying parameter.
    - **Our thought experiment**: *If* the null were true, *How* likely would we see what we observe (or more extreme).
  - Choose a test statistic
    - In classical hypothesis testing, we pick something that has useful statistical properties:

$$T = \hat{\tau} / \sqrt{\widehat{Var}(\hat{\tau})}$$

Hypothesis testing review

- Determine the distribution of the test statistic under the null
  - In classical testing, in large samples, $T \sim \mathcal{N}(0, 1)$
  - In smaller samples, you may have made further assumptions (e.g. outcome is normally distributed) to show that T follows a $t-$ distribution
  - We need a distribution to get probabilities!
- What is the probability of observing the test statistic T that you observe in-sample (or a more extreme value) given the known distribution under the null?
  - That's a **p-value**!

What's different about randomization testing?

- Randomization tests are a kind of hypothesis test but with 2 main differences from our usual null hypothesis testing for the ATE.

  1. Different null hypothesis
     - We'll assume a sharp null of no individual effect
     - Different from null of no ATE
  2. No assumptions/asymptotics to derive the distribution of T
     - We can instead literally just calculate the value under each possible realization of $\mathbf{D}$
     - And we know the distribution of $\mathbf{D}$ because we control it in an experiment.
     - In practice, since there are often a lot of possible realizations of $\mathbf{D}$, we'll approximate by monte carlo sampling.

Sharp null of no effect.

- The sharp null hypothesis states:

$$H_0 : \tau_i = Y_i(1) - Y_i(0) = 0 \ \forall i$$

- Sharp null implies no ATE
  - But no ATE does not imply no sharp null
- Why do we make this assumption?
  - Because now the observed data tells us **everything** we need to know about the potential outcomes

The sharp null

- Remember our table of science? For a single realization of $\mathbf{D}$ we only observe half the potential outcomes?

Unit i	Treatment D_i	$Y_i(1)$	$Y_i(0)$	Observed Y_i
1	1	5	?	5
2	0	?	-3	-3
3	1	9	?	9
4	0	?	-1	-1
5	0	?	-4	-4
6	1	2	?	2

- But what does the sharp null imply about the unobserved potential outcomes? Can we fill in those question marks?

The sharp null

- Yes! Under the sharp null, $Y_i(1) = Y_i(0)$

Unit i	Treatment D_i	$Y_i(1)$	$Y_i(0)$	Observed Y_i
1	1	5	5	5
2	0	-3	-3	-3
3	1	9	9	9
4	0	-1	-1	-1
5	0	-4	-4	-4
6	1	2	2	2

- Why is this useful?
- Because now we can calculate the value of our test statistic *not only* under the observed $\mathbf{D}$ but under all other possible realizations of $\mathbf{D}$!

The sharp null

- Consider this alternate randomization. Under the null, the observed Y_i remain the same.

Unit i	Treatment D_i	$Y_i(1)$	$Y_i(0)$	Observed Y_i
1	1	5	5	5
2	1	-3	-3	-3
3	1	9	9	9
4	0	-1	-1	-1
5	0	-4	-4	-4
6	0	2	2	2

- But our test statistic (e.g. a difference-in-means) will change because of the new assignment.

The test statistic

- Our test statistic is any function of treatment assignments $\mathbf{D}$ and observed outcomes $\mathbf{Y}$.
- Lots of choices with different degrees of **power** for different kinds of treatment effects.
- We want to pick a test statistic that will return large values when the null is false and small values when it is true.

- One good option: (absolute) difference-in-means

$$t(\mathbf{D}, \mathbf{Y}) = \left| \frac{1}{N_t} \sum_{i=1}^{N} Y_i D_i - \frac{1}{N_c} \sum_{i=1}^{N} Y_i (1 - D_i) \right|$$

  - Well-powered when treatment effects are (roughly) constant

- What sorts of alternatives might this be bad for?
  - Offsetting positive and negative effects will return small values of $t(\mathbf{D}, \mathbf{Y})$ as would no effects.
- We might pick a different test statistic in this case!

The randomization distribution

- Under the sharp null, we can calculate $t(\mathbf{D}, \mathbf{Y})$ for every possible realization of $\mathbf{D}$.
  - Why? Because under the sharp null, observed Y_i is unaffected by treatment assignment.
- Then to get a distribution for $t(\mathbf{D}, \mathbf{Y})$, we just need to know the distribution of $\mathbf{D}$. We know this by designing the experiment!
- Complete randomization
  - Every possible realization of $\mathbf{D}$ with N_t treated units and N_c control units
- Bernoulli
  - Every possible realization of $\mathbf{D}$ is equally likely
  - Wait though! The test statistic is undefined when $N_c = 0$ or $N_t = 0$. We implicitly condition on observing at least one treated/control unit even under bernoulli randomization which places some restrictions on $\mathbf{D}$ (see Branson and Bind, 2019 in Statistical Methods in Medical Research)
  - In practice, we'll usually analyze bernoulli trial experiments and do inference *conditional* on the observed N_t and N_c which is a complete randomization design. In large samples, the difference doesn't matter.
- Stratified or Cluster randomized
  - Stratification/clustering can be thought of as placing further restrictions on possible values of $\mathbf{D}$
    - Only assignments where strata are perfectly balanced
    - Only assignments where units sharing a cluster share the same treatment.

Calculating p-values

- We get our p-value by comparing the observed test statistic for our particular sample t^* to the distribution of $t(\mathbf{D}, \mathbf{Y})$

- For complete randomization, each of the K possible realizations of $\mathbf{D}$ is equally likely, so we just enumerate all possible assignments $\mathbf{d} \in \Omega$ and calculate the share that are greater than our observed test statistic.

$$Pr(t^* \geq t(\mathbf{D}, \mathbf{Y})) = \frac{\sum_{\mathbf{d} \in \Omega} \mathrm{I}(t(\mathbf{d}, \mathbf{Y}) \geq t^*)}{K}$$

- This is our p-value, which we compare to some threshold level α and reject the null when it's below that level.

Monte carlo approximation

- For small samples, we could enumerate every possible treatment vector and actually just calculate $t(\mathbf{D}, \mathbf{Y})$.
- In large samples, that's tedious!
  - For the exercise example, just comparing High vs. Control incentives would involve $\binom{80}{40} \approx 1.07 \times 10^{23}$ unique assignments!
- We'll typically use a monte carlo approximation to the exact p-value.
  - This is also easier for more complicated randomization schemes.
- **Procedure:**
  - For K iterations:
    1. Draw a realization of the treatment vector $\mathbf{d}_k$ from the known distribution of $\mathbf{D}$.
    2. Calculate the test statistic $t_k = t(\mathbf{d}_k, \mathbf{Y})$
  - Our p-value is the share of these K test statistics that are greater than the observed t^*
- Think of the monte carlo procedure as generating an arbitrarily large number of samples from the sampling distribution of the test statistic under the null.

Putting it all together

- To do randomization inference under the sharp null

    1. Choose a test statistic
    2. Calculate the observed test statistic in your sample $t^* = t(\mathbf{D}, \mathbf{Y})$
    3. Draw another treatment vector $\mathbf{d}_1$ from the known distribution of $\mathbf{D}$
    4. Calculate $t_1 = t(\mathbf{d}_1, \mathbf{Y})$
    5. Repeat 3 and 4 as long as you want to get K samples from the distribution of the test statistic under the null
    6. Calculate $p = \frac{1}{K}\sum_{i=1}^{K} \mathrm{I}(t_k \geq t^*)$

Application: Incentives to exercise

- Let's look at a test for the effect of high vs. control incentives in the Charness and Gneezy (2008) experiment.

```
# Subset to high vs. control
exercise2 <- exercise %>% filter(treatment != "Low")

# Calculate the observed test stat (absolute diff-in-means)
t_observed <- abs(mean(exercise2$After[exercise2$treatment == "High"]) -
  mean(exercise2$After[exercise2$treatment == "Control"]))

t_observed
```

```
## [1] 0.679
```

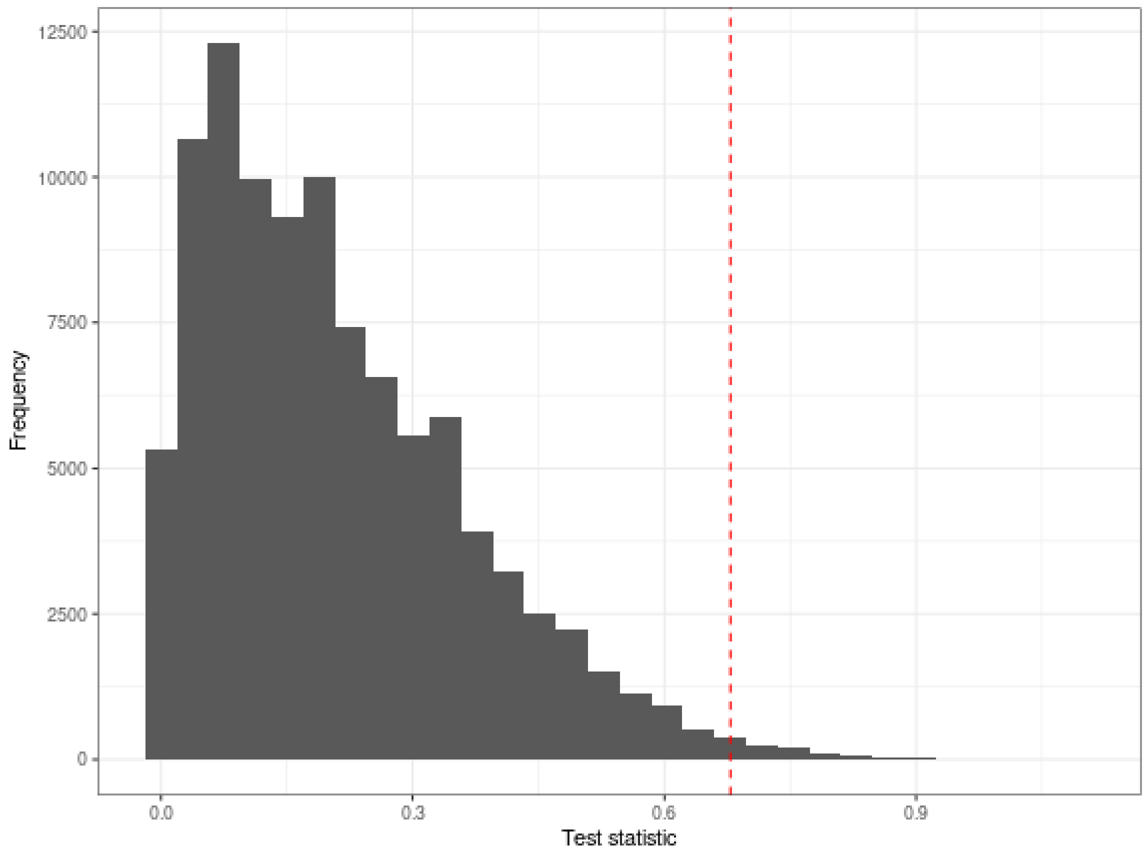
Application: Incentives to exercise

```
# For each iteration, sample a new treatment vector
nIter <- 1e5
set.seed(60637) # Set a random seed
t_stat <- rep(NA, nIter)
for (k in 1:nIter){
  # Permute treatment (complete randomization)
  exercise2$treatmentPerm = sample(exercise2$treatment)
  # Calculate test-stat
  t_stat[k] <- abs(mean(exercise2$After[exercise2$treatmentPerm == "High"]) -
  mean(exercise2$After[exercise2$treatmentPerm == "Control"]))
}

# P-value
mean(t_stat >= t_observed)
```

```
## [1] 0.00878
```

Application: Incentives to exercise



Alternate test statistics

- Depending on the distribution of treatment effects, we might be interested in using different test statistics.
- For example, a difference in quantiles

```
# Calculate the observed test stat (absolute diff in 75th quantile)
t_obs_quant <- abs(quantile(exercise2$After[exercise2$treatment == "High"], .75) -
  quantile(exercise2$After[exercise2$treatment == "Control"], .75))

t_obs_quant
```

```
##  75%
## 1.43
```

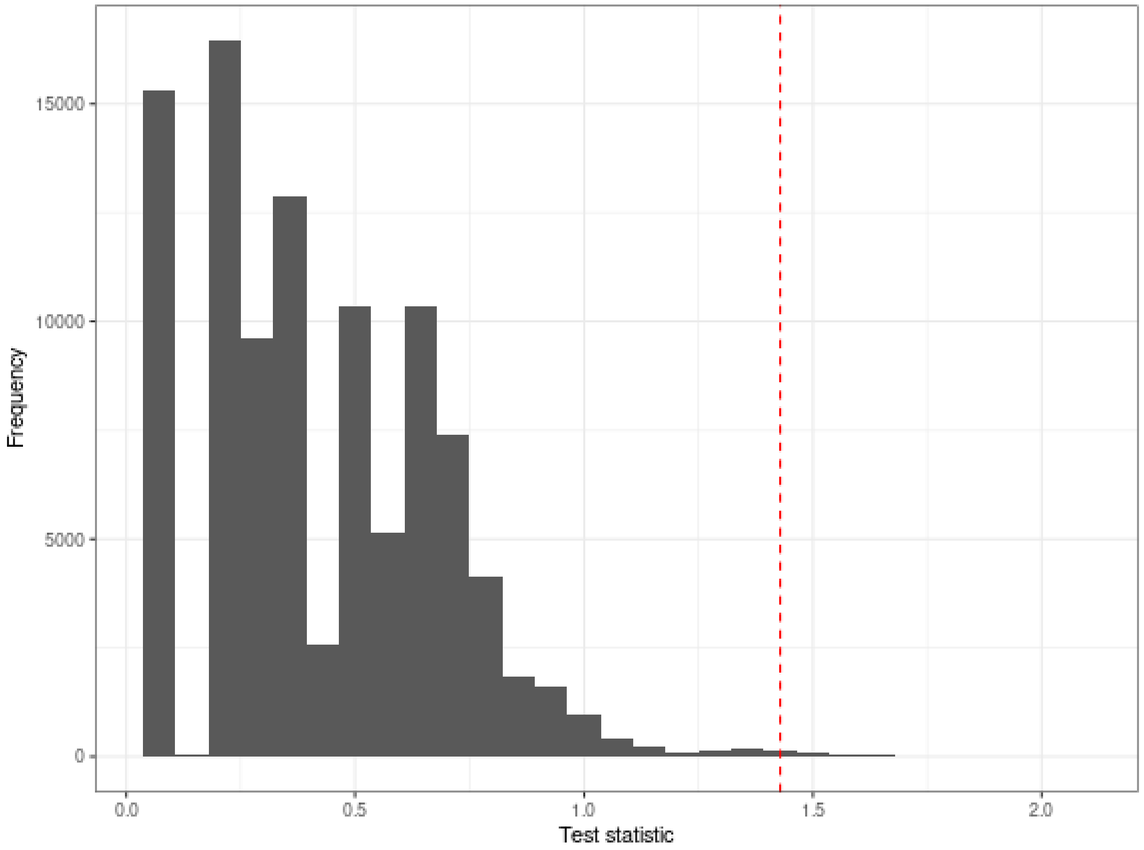
Quantile test statistic

```r
# For each iteration, sample a new treatment vector
nIter <- 1e5
set.seed(60637) # Set a random seed
t_stat_quant <- rep(NA, nIter)
for (k in 1:nIter){
  # Permute treatment (complete randomization)
  exercise2$treatmentPerm = sample(exercise2$treatment)
  # Calculate test-stat (75% quantile difference)
  t_stat_quant[k] <- abs(quantile(exercise2$After[exercise2$treatmentPerm == "High"], .75) -
  quantile(exercise2$After[exercise2$treatmentPerm == "Control"], .75))
}

# P-value
mean(t_stat_quant >= t_obs_quant)
```

```
## [1] 0.00318
```

Quantile test statistic



Difference in distributions

- We've focused on differences in *mean* outcomes between groups, but what if means remain the same but the distributions change?
- How do we compare distributions of treated/control outcomes? We look at their empirical cumulative distribution functions (eCDFs)

$$\hat{F}_c(y) = \frac{1}{N_c} \sum_{i:D_i=0} \mathrm{I}(Y_i \leq y)$$

$$\hat{F}_t(y) = \frac{1}{N_t} \sum_{i:D_i=1} \mathrm{I}(Y_i \leq y)$$

- But how do we summarize the difference between two eCDFs? A common method is to take the absolute maximum difference between the two functions across all possible values of y. The *Kolmogorov-Smirnov* statistic is

$$t_{\mathrm{KS}} = \sup_y \, |\hat{F}_t(y) - \hat{F}_c(y)|$$

Difference in distributions

- Let's just visualize first the difference in the eCDFs

```r
# ecdf() function will create a function given the inputs
ecdf_high <- ecdf(exercise2$After[exercise2$treatment == "High"])
ecdf_control <- ecdf(exercise2$After[exercise2$treatment == "Control"])

# They're functions -- you give them a value of y, they'll give you a probability
ecdf_high(1)
```

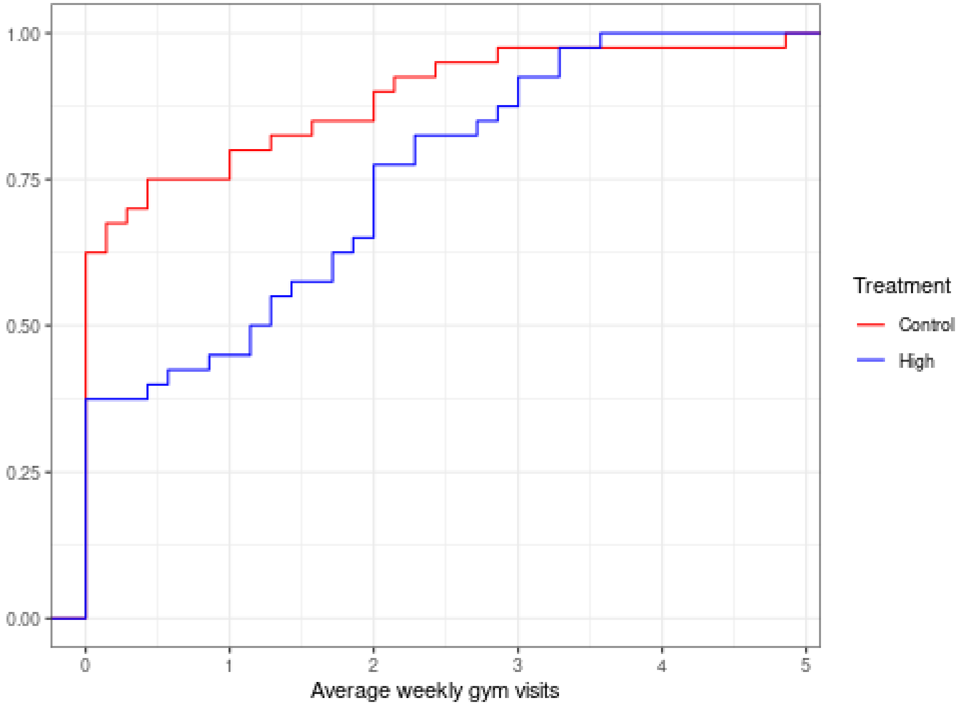
```
## [1] 0.45
```

```r
ecdf_high(median(exercise2$After[exercise2$treatment == "High"]))
```

```
## [1] 0.5
```

Difference in distributions

```
# ggplot has a function for plotting ECDFs
exercise2 %>% ggplot(aes(x=After, colour=treatment)) + stat_ecdf(geom="step") +
  scale_colour_manual("Treatment", values=c("Red", "Blue")) + xlab("Average weekly gym visits")
  ylab("") + theme_bw()
```



Randomization test with a KS test statistic

```r
# Calculate the observed test stat (KS-stat)
t_obs_ks <- ks.test(exercise2$After[exercise2$treatment == "High"],
  exercise2$After[exercise2$treatment == "Control"])$statistic

t_obs_ks
```

```
##    D
## 0.35
```

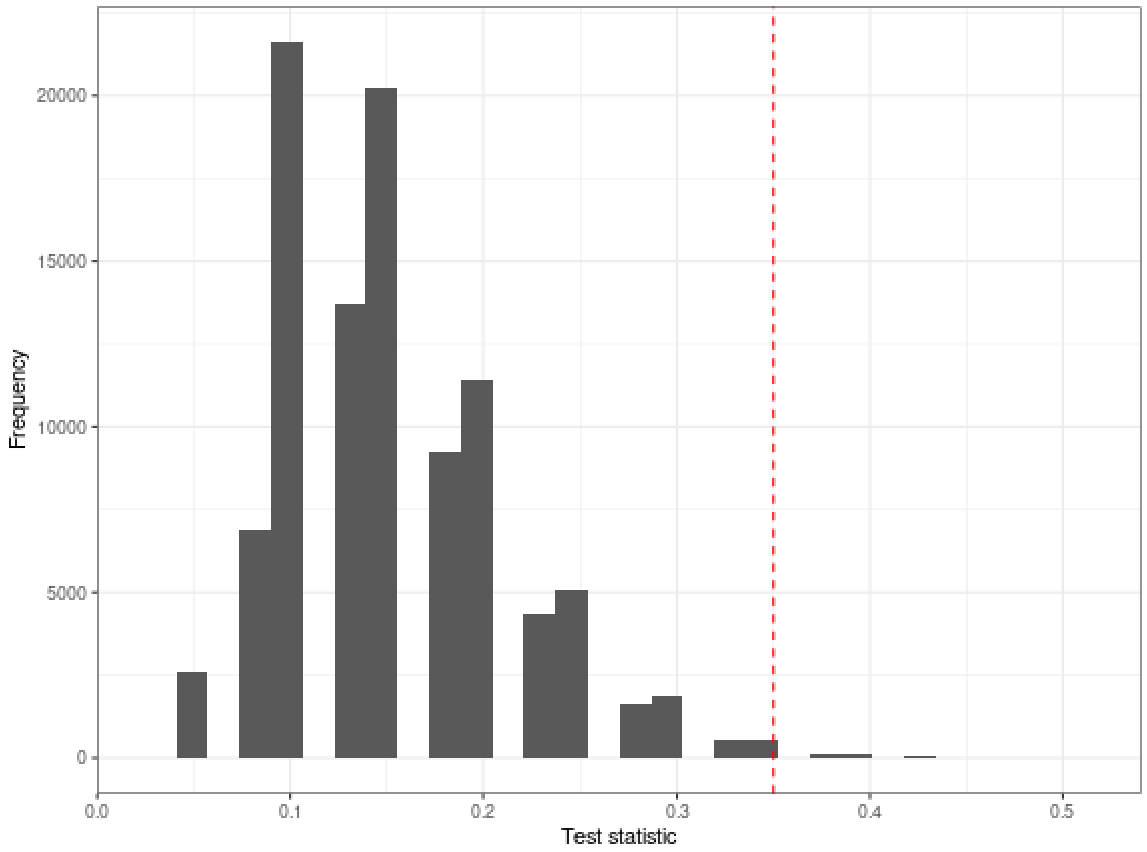
Randomization test with a KS test statistic

```
# For each iteration, sample a new treatment vector
nIter <- 1e5
set.seed(60637) # Set a random seed
t_stat_ks <- rep(NA, nIter)
for (k in 1:nIter){
  # Permute treatment (complete randomization)
  exercise2$treatmentPerm = sample(exercise2$treatment)
  # Calculate test-stat (KS-statistic)
  t_stat_ks[k] <- ks.test(exercise2$After[exercise2$treatmentPerm == "High"],
  exercise2$After[exercise2$treatmentPerm == "Control"])$statistic
}

# P-value
mean(t_stat_ks >= t_obs_ks)
```

```
## [1] 0.00871
```

Randomization test with a KS test statistic



Inverting tests to get confidence intervals

- Randomization tests alone give us p-values but no confidence intervals.
- One approach: "invert" the test -- for what values of a "treatment effect" would we fail to reject the null
  - A $100(1-\alpha)\%$ confidence interval contains the set of parameter values for which an α-level hypothesis test would fail to reject the null.

- Slight complication: We now need to actually define a "treatment effect" parameter:

  - For example, assume a *constant additive effect* for all units

$$Y_i(1) - Y_i(0) = \tau_0$$

  - Our confidence set would be all of the values of τ_0 for which we'd fail to reject the null
  - Calculate via a grid search through possible values of τ_0.

Randomization tests in practice

RESEARCH ARTICLE

Social influence and political mobilization: Further evidence from a randomized experiment in the 2012 U.S. presidential election

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Abstract

A large-scale experiment during the 2010 U.S. Congressional Election demonstrated a positive effect of an online get-out-the-vote message on real world voting behavior. Here, we report results from a replication of the experiment conducted during the U.S. Presidential Election in 2012. In spite of the fact that get-out-the-vote messages typically yield smaller effects during high-stakes elections due to saturation of mobilization efforts from many sources, a significant increase in voting was again observed. Voting also increased significantly among the close friends of those who received the message to go to the polls, and the total effect on the friends was likely larger than the direct effect, suggesting that understanding social influence effects is potentially even more important than understanding the direct effects of messaging. These results replicate earlier work and they add to growing evidence that online social networks can be instrumental for spreading offline behaviors.

Randomization tests in practice

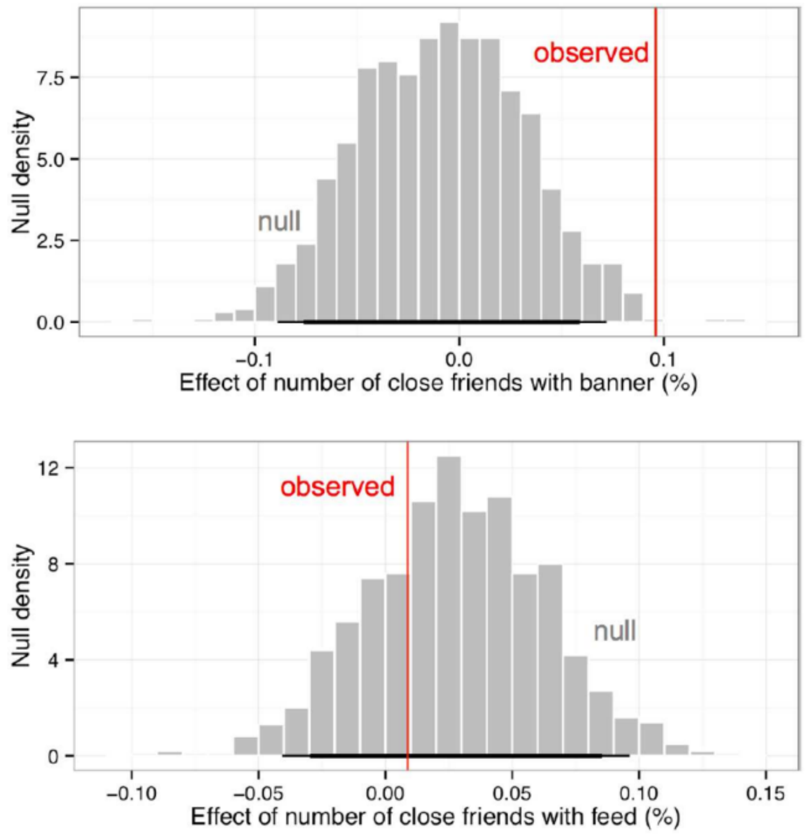

Fig 3. Observed increase in the probability of voting caused by each additional close friend in the banner (top) and feed (bottom) treatments are shown in red. A null distribution of possible outcomes when the network structure is fixed but the treatments are randomly permuted is shown in gray. The results suggest that the banner treatment was effective in spreading behavior to friends (a 0.1% increase in voting likelihood for each close friend treated), but the feed treatment was not. Stratified statistical analyses also replicate these results.

https://doi.org/10.1371/journal.pone.0173851.g003

Conclusion

- We've learned two frameworks for estimating and conducting inference for treatment effects in randomized experiments
- Neyman
  - Define an estimand, the ATE: $\tau = E[Y_i(1) - Y_i(0)]$
  - Choose an estimator, the difference-in-means
  - Estimator has known mean and variance, do hypothesis tests under large-sample assumptions on its distribution (normality)
- Fisher
  - Forget the estimation process, let's just do an (exact) test.
  - Sharp null of no effect ($Y_i(1) = Y_i(0) \; \forall i$)
  - No large-sample approximations -- justified by randomization alone
- Usually we want to actually get an estimate of some well-defined quantity (like an ATE or conditional ATE) so we'll mostly work in the Neyman setting, but Fisher randomization tests can be useful in complex assignment settings.