Week 3: Designing Experiments

PLSC 30600 - Causal Inference

Last week

- Why randomized experiments work
  - Guarantee treatment D_i is independent of potential outcomes $\{Y_i(1), Y_i(0)\}$

- Inference for treatment effects

  - **Fisher**: Can get exact p-values under the sharp null just knowing the distribution of treatment assignments.
  - **Neyman**: Know the variance for the difference-in-means

$$\widehat{Var(\hat{\tau})} = \frac{S_t^2}{N_t} + \frac{S_c^2}{N_c}$$

This week

- When and why do we use covariates in designing/analyzing an experiment
  - Balance checks
  - Stratification/blocking -- improving precision
  - Conditional ATEs -- treatment effect heterogeneity
- When should we *not* condition on covariates?
  - When they're **post-treatment**!

Balance checking

Balance tests

- One reason to use covariates even in completely randomized designs is to check whether the experiment **actually** did what it was supposed to do.
- Under any valid randomization scheme:

$$X_i \perp\!\!\!\perp D_i$$

$$E[X_i | D_i = 1] = E[X_i | D_i = 0]$$

- If we correctly randomized treatment, then the expectation (and the distribution) of covariates should be the same in treatment and control.
- But in any given sample, we'll observe a difference just by chance -- how do we know if this is a problem?

Against balance tests

- One view in some experimentally-focused fields is that you should **never** waste time checking balance

- Senn (1994) *Statistics in Medicine*

  1. Randomization ensures balance over all randomizations.
  2. Observing any particular imbalance in our sample doesn't disprove 1

- But what if we did screw up? What if the randomization software had a bug? What if there was some implementation issue?

- But how to interpret a "failed" balance test?
  - A reason to go back and check the randomization process -- if we think that it *did* actually work as intended, we may just have gotten unlucky.
  - $p < .05$ probably shouldn't be the threshold if we really believe treatment was randomized.

Against balance tests

- Another more nuanced argument is that you shouldn't use balance tests to decide whether to include or not include covariates.

- **Mutz, Pemantle and Pham (2019)**

  - Often researchers run a lot of univariate balance tests in an experiment.
  - If a test for some covariate fails, they include that covariate in adjustment.
  - This process risks raising false-positive rates through researcher "degrees-of-freedom"

- This is correct, but is less an argument against *balance testing* per-se and more against ad-hoc or data-dependent covariate choices.

- Choose your covariates to adjust for **ex-ante** (even if you're not explicitly blocking) and don't choose them based on the results of balance tests
  - Also, balance tests don't make sense as a criteria for covariate inclusion since if we do believe randomization, we've addressed confounding.
  - We want covariates that are predictive of Y

Guidelines for balance testing

- Testing for balance to assess whether randomization occurred as intended: Good
  - Careful with multiple testing/false positives.
  - $p < .05$ is probably too high of a threshold, but you should probably be concerned if $p < 1 \times 10^{-6}$
  - What do do if a balance check fails? Check your experiment!
- Testing for balance to pick which covariates to adjust for: Bad
  - "Garden of forking paths"
  - You should choose covariates ex-ante (even if not blocking)
  - Pick covariates that predict Y -- balance checks are the wrong criteria.

Stratification/blocking in experiments

Using covariates in experiments

- Sometimes we have covariate information X_i that predicts Y_i. Should we use this?
  - Yes: If X_i predicts Y_i well, we can get a more precise ATE estimator.
  - Covariates aren't relevant for **bias** in an experiment, but they can help with **variance**.
- **Stratification/Block-randomization**:
  - Consider a partition of our sample that puts our N units into G mutually-exclusive strata.
  - With discrete covariates, can think of each unique combination of the covariates.
  - With continuous covariates we coarsen them into bins.
  - Each stratum g has N_g units.
  - Treatment is completely randomized within each stratum: $N_{t,g}$ units receive treatment, $N_{c,g} = N_g - N_{t,g}$ receive control.

Using covariates in experiments

- Goal of stratification:
    - Get rid of possible randomizations where covariates are **imbalanced** between treated and control
    - Reduces the **variance** of our ATE estimator.
- "Pruning" the space of randomizations. Consider $N = 100$, $N_t = 50$ with two blocks.
    - Under complete randomization there are $\binom{100}{50} \approx 1.01 \times 10^{29}$ possible assignments
    - Under blocking: $\binom{50}{25} + \binom{50}{25} \approx 2.53 \times 10^{14}$.

Estimation under block-randomization

- Our estimator for the stratum-specific treatment effect $\hat{\tau}_g$ is just the difference-in-means

$$\hat{\tau}_g = \underbrace{\bar{Y}_{t,g}}_{\text{treated mean in stratum g}} - \underbrace{\bar{Y}_{c,g}}_{\text{control mean in stratum g}}$$

- And we can estimate the sampling variance using the within-stratum Neyman variance estimator

$$\widehat{Var(\hat{\tau}_g)} = \frac{S_{t,g}^2}{N_{t,g}} + \frac{S_{c,g}^2}{N_{c,g}}$$

where $S_{t,g}^2$, $S_{c,g}^2$ are the sample variance of Y within the treated group and control group respectively in stratum g.

- Imagine: We ran G independent mini-experiments and analyzed them separately. Each is unbiased for the conditional ATE (CATE) and has its own standard error.

Estimation under block-randomization

- How do we aggregate to get an estimate of the SATE? Take a weighted average by stratum size

$$\hat{\tau} = \sum_{g=1}^{G} \hat{\tau}_g \times \frac{N_g}{N}$$

- And the sampling variance?

$$\widehat{Var_{\text{strat}}}(\hat{\tau}) = \sum_{g=1}^{G} \widehat{Var(\hat{\tau}_g)} \times \left(\frac{N_g}{N}\right)^2$$

- When is the variance of the blocked design going to be lower than the variance under complete randomization?
  - When the strata explain some of the variance in Y (the population $\sigma^2_{t,g} < \sigma^2_t$ and $\sigma^2_{c,g} < \sigma^2_c$)

Can blocking hurt?

- Long debate over where it's possible to go wrong blocking. **Athey and Imbens (2017)** argue no downside to blocking.
- This answer depends on the framework for inference.
  - **Pashley and Miratrix (2021)** give an extensive review under alternative sampling/inference schemes.
- **Athey and Imbens** result holds under stratified random sampling from the population and equal treatment probability w/in strata.
  - Intuition: In the worst case scenario, stratification is just a two-stage randomization process equivalent to complete randomization.
- This also does not guarantee that the *estimated* standard error will be smaller -- with an irrelevant covariate, we will have fewer degrees of freedom as we are estimating multiple parameters. Estimated SEs under stratification might be higher.
- Athey and Imbens suggest falling back on the conservative complete randomization SE, but this will only work if treatment probabilities are constant across strata
  - With imbalanced treatment probabilties across strata, we only have ignorability **conditional** on stratum.

Post-stratification

- Suppose we didn't stratify ex-ante but have some covariates that we observe? Can we analyze *as-though* we had stratified on these?
  - Yes: **Post-stratification**
- Key difference from stratification: Number of treated/control w/in stratum is random and not fixed. Stratum sizes also not fixed.
- **Miratrix, Sekhon and Yu (2013)**
  - Usually not a problem -- relative to blocking ex-ante, the differences in variances are small.
  - Problems with many strata + poorly predictive strata.
  - Unlike the Athey and Imbens setting, benefits not guaranteed, but often doesn't hurt with good covariate choice.

Post-stratifying on a relevant covariate

- Let's go back to our UChicago Exercise experiment. We observe exercise in the pre-treatment period, let's stratify on that!

```
# What cutpoints to pick?
quantile(exercise2$Before, c(0, .25, .5, .75))
```

```
##   0%  25%  50%  75% 
## 0.00 0.00 0.00 1.16
```

```
# Okay, about 50% are 0 let's just stratify on an indicator of *any* exercise!
exercise2$AnyBefore <- as.integer(exercise2$Before != 0)

# Our conditional ATEs
ate_noexercise <- mean(exercise2$After[exercise2$treatment == "High"&exercise2$AnyBefore == 0])
  mean(exercise2$After[exercise2$treatment == "Control"&exercise2$AnyBefore == 0])
ate_someexercise <- mean(exercise2$After[exercise2$treatment == "High"&exercise2$AnyBefore == 1
  mean(exercise2$After[exercise2$treatment == "Control"&exercise2$AnyBefore == 1])

ate_strat <- ate_noexercise*mean(exercise2$AnyBefore == 0) +
  ate_someexercise*mean(exercise2$AnyBefore == 1)
ate_strat
```

```
## [1] 0.581
```

Post-stratifying on a relevant covariate

- Now to estimate the sampling variance

```
var_noexercise <- var(exercise2$After[exercise2$treatment == "High"&exercise2$AnyBefore == 0])/
  var(exercise2$After[exercise2$treatment == "Control"&exercise2$AnyBefore == 0])/sum(exercise2

var_someexercise <- var(exercise2$After[exercise2$treatment == "High"&exercise2$AnyBefore == 1]
  var(exercise2$After[exercise2$treatment == "Control"&exercise2$AnyBefore == 1])/sum(exercise2

var_strat <- var_noexercise*mean(exercise2$AnyBefore == 0)^2 + var_someexercise*mean(exercise2$

sqrt(var_strat)
```

```
## [1] 0.237
```

```
# How does it compare to unadjusted?
lm_robust(After ~ I(treatment == "High"), data=exercise2)
```

```
##                             Estimate Std. Error t value Pr(>|t|) CI Lower
## (Intercept)                    0.564      0.168    3.36  0.00123    0.229
## I(treatment == "High")TRUE     0.679      0.252    2.69  0.00871    0.177
##                             CI Upper DF
## (Intercept)                    0.899 78
## I(treatment == "High")TRUE     1.181 78
```

Post-stratifying on a relevant covariate

- Incidentally, we can get the stratified difference-in-means by interacting treatment with the de-meaned stratum indicators!
    - **Lin (2013)** estimator - we'll cover this more when we talk about regression adjustment in general.

```
tidy(lm_robust(After ~ I(treatment == "High")*I(AnyBefore - mean(AnyBefore)), data= exercise2))
```

```
##                                                         term estimate std.error
## 1                                               (Intercept)    0.639     0.142
## 2                                I(treatment == "High")TRUE    0.581     0.237
## 3                            I(AnyBefore - mean(AnyBefore))    1.490     0.333
## 4 I(treatment == "High")TRUE:I(AnyBefore - mean(AnyBefore))   -1.021     0.499
```

An irrelevant covariate

```
# Unadjusted
tidy(lm_robust(After ~ I(treatment == "High"), data=exercise2)) %>% select(term, estimate, std.
```

```
##                          term estimate std.error
## 1                 (Intercept)    0.564     0.168
## 2 I(treatment == "High")TRUE    0.679     0.252
```

```
# Post-stratifying on gender
tidy(lm_robust(After ~ I(treatment == "High")*I(Male - mean(Male)), data=exercise2)) %>% select
```

```
##                                              term estimate std.error
## 1                                     (Intercept)    0.572     0.174
## 2                      I(treatment == "High")TRUE    0.675     0.258
## 3                               I(Male - mean(Male))   -0.300     0.347
## 4 I(treatment == "High")TRUE:I(Male - mean(Male))    0.453     0.517
```

Summary

- When should you use **pre-treatment** covariates in an experiment?
  - When the covariates predict the outcome
- Ideally, you should block
  - This avoids finite-sample imbalance in treatment across groups.
- But adjusting ex-post isn't too bad
  - Especially when covariates strongly correlated with Y.
- Should try to fix your covariate choices in advance!
  - Avoid p-hacking/"researcher degrees of freedom" problems.

Conditional and Joint Treatment Effects

Conditional average treatment effects

- Sometimes we might be interested in treatment effects among different sub-groups in the sample
  - Effects of treatment are rarely homogeneous: Republicans respond to cues from Trump differently than Democrats!
  - The ATE is still a well-defined quantity under effect heterogeneity, but we might be explicitly interested in sub-group effects.

- The Conditional Average Treatment Effect (CATE)

$$\tau(x) = \underbrace{E[Y_i(1)|X_i = x]}_{\text{Mean P.O. under treatment among units with} X_i = x} - \underbrace{E[Y_i(0)|X_i = x]}_{\text{Mean P.O. under control among units with} X_i = x}$$

- Estimate using the standard difference-in-means *within* each sub-group. Conventional inference using the Neyman variance (though be careful on asymptotics when sub-groups are small).

Example: Exercise Experiment

- Again, let's look at our U Chicago exercise experiment! We might be interested in knowing whether the incentives mattered more for undergraduates who never exercised prior to the experiment compared to those who did.

```
# Difference among Before == 0
no_prior  <- lm_robust(After ~ I(treatment == "High"), data=exercise2 %>% filter(Before == 0))
tidy(no_prior) %>% filter(grepl("treatment", term)) %>% select(term, estimate, std.error, stati
```

```
##                          term estimate std.error statistic  p.value
## 1 I(treatment == "High")TRUE     1.01     0.273      3.72 0.000566
```

```
# Difference among Before != 0
some_prior <- lm_robust(After ~ I(treatment == "High"), data=exercise2 %>% filter(Before != 0))
tidy(some_prior) %>% filter(grepl("treatment", term)) %>% select(term, estimate, std.error, sta
```

```
##                          term estimate std.error statistic p.value
## 1 I(treatment == "High")TRUE -0.00652     0.417   -0.0156   0.988
```

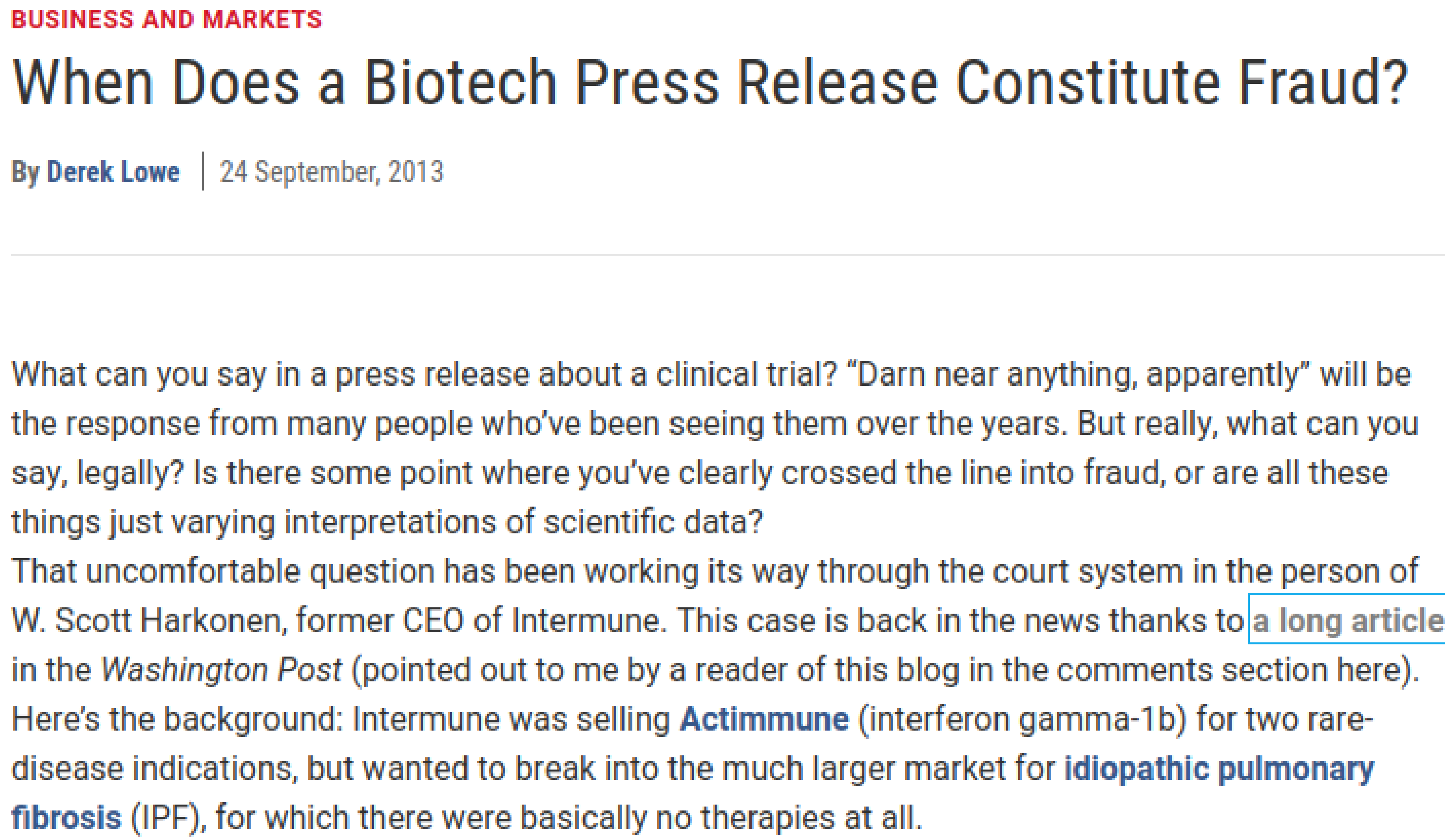
Example: Exercise Experiment

- Is there a difference between the two groups. Remember that the difference between a statistically significant and a statistically insignificant estimate is **not guaranteed** to be statistically significant.
- We need to explicitly test for the difference

```r
# Difference among Before == 0
cate_reg <- lm_robust(After ~ I(treatment == "High")*I(Before != 0), data=exercise2)
tidy(cate_reg)  %>% select(term, estimate, std.error, statistic, p.value)
```

```
##                                              term estimate std.error statistic
## 1                                     (Intercept)  0.00571   0.00571      1.00
## 2                      I(treatment == "High")TRUE  1.01469   0.27295      3.72
## 3                              I(Before != 0)TRUE  1.48952   0.33315      4.47
## 4 I(treatment == "High")TRUE:I(Before != 0)TRUE -1.02121   0.49863     -2.05
##    p.value
## 1 3.20e-01
## 2 3.83e-04
## 3 2.68e-05
## 4 4.40e-02
```

Careful with subgroup effects



BUSINESS AND MARKETS

When Does a Biotech Press Release Constitute Fraud?

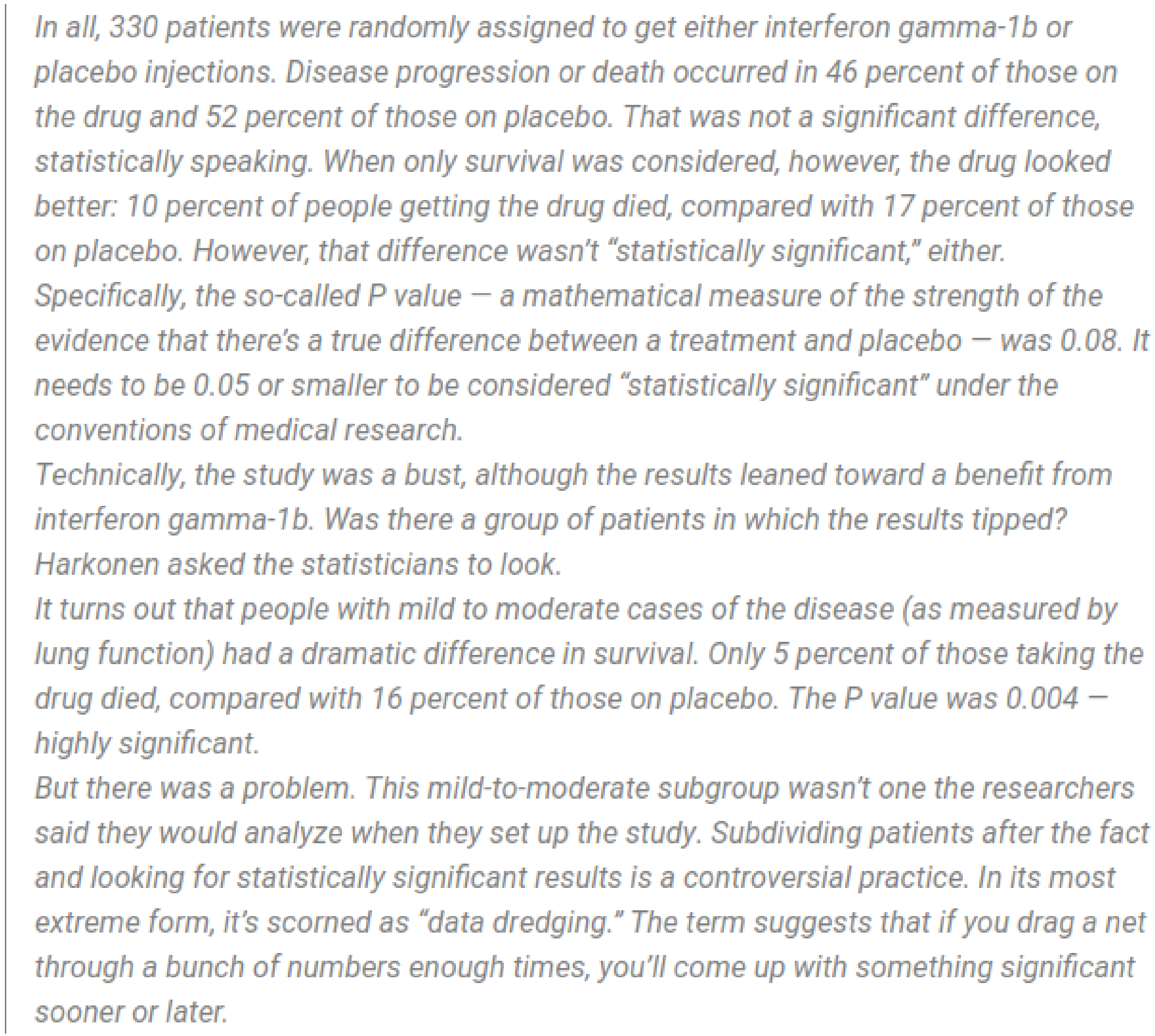
By Derek Lowe | 24 September, 2013

What can you say in a press release about a clinical trial? "Darn near anything, apparently" will be the response from many people who've been seeing them over the years. But really, what can you say, legally? Is there some point where you've clearly crossed the line into fraud, or are all these things just varying interpretations of scientific data?

That uncomfortable question has been working its way through the court system in the person of W. Scott Harkonen, former CEO of Intermune. This case is back in the news thanks to a long article in the *Washington Post* (pointed out to me by a reader of this blog in the comments section here).

Here's the background: Intermune was selling **Actimmune** (interferon gamma-1b) for two rare-disease indications, but wanted to break into the much larger market for **idiopathic pulmonary fibrosis** (IPF), for which there were basically no therapies at all.

Beware of p-hacking



> In all, 330 patients were randomly assigned to get either interferon gamma-1b or placebo injections. Disease progression or death occurred in 46 percent of those on the drug and 52 percent of those on placebo. That was not a significant difference, statistically speaking. When only survival was considered, however, the drug looked better: 10 percent of people getting the drug died, compared with 17 percent of those on placebo. However, that difference wasn't "statistically significant," either.
> Specifically, the so-called P value — a mathematical measure of the strength of the evidence that there's a true difference between a treatment and placebo — was 0.08. It needs to be 0.05 or smaller to be considered "statistically significant" under the conventions of medical research.
> Technically, the study was a bust, although the results leaned toward a benefit from interferon gamma-1b. Was there a group of patients in which the results tipped? Harkonen asked the statisticians to look.
> It turns out that people with mild to moderate cases of the disease (as measured by lung function) had a dramatic difference in survival. Only 5 percent of those taking the drug died, compared with 16 percent of those on placebo. The P value was 0.004 — highly significant.
> But there was a problem. This mild-to-moderate subgroup wasn't one the researchers said they would analyze when they set up the study. Subdividing patients after the fact and looking for statistically significant results is a controversial practice. In its most extreme form, it's scorned as "data dredging." The term suggests that if you drag a net through a bunch of numbers enough times, you'll come up with something significant sooner or later.

Effect modification v. Interaction

- Differences in CATEs assign no causal interpretation to X_i
  - Comparing the effect of a treatment among college gradautes vs. non-college tells us nothing about what would happen to the effect if we *assigned* college education.
  - Some X_i may be non-manipulable
- **Effect modification** -- the effect of an intervention varies across stata of a covariate X_i
- **Interaction** -- the effect of an intervention changes when **another** intervention is assigned.
- To think about interactions between treatments, we need to define potential outcomes with respect to **multiple treatments**
- Consider two treatments, $D_{1,i}$ and $D_{2,i}$
  - Define a joint potential outcome $Y_i(d_1, d_2)$.
  - Under consistency, $Y_i(d_1, d_2) = Y_i$ when $D_{1,i} = d_1$, $D_{2,i} = d_2$
- Treatment effects are defined in terms of differences in joint potential outcomes

$$\tau_{1,2} = E[Y_i(1,1) - Y_i(0,0)]$$

Joint treatment effects

- Lots more causal quantities of interest -- can define an "ATE" for any unique combination of treatments:
- Controlled Direct Effect -- what's the effect of one treatment fixing the other treatment to a particular level.

$$\text{CDE}_1(1) = E[Y_i(1,1) - Y_i(0,1)]$$

- Differences in CDEs in an experiment correspond to "interactions" -- how does *assigning* one level of treatment influence the effect of another.

Joint treatment effects

- Main effect -- what's the average effect of one treatment marginalizing over the distribution of the other treatment(s).

$$\tau_1 = \sum_{d_2 \in \mathcal{D}_2} \underbrace{E[Y_i(1, d_2) - Y_i(0, d_2)]}_{\text{CDE at } d_2} \times \Pr(D_{2,i} = d_2)$$

- Note that your main (or marginalized) effects depend on the distribution of the other treatment. This can cause external validity issues in large factorial experiments (e.g. conjoints) - See de la Cuesta, Egami, Imai (2022)

Post-treatment bias

Post-treatment covariates

- When talking about stratification/conditional average treatment effects, we've emphasized that X_i must be **pre-treatment**
- What happens when we condition on some post-treatment variable (call it M_i).
- **Intuition**: M_i is post-treatment. It has potential outcomes: $\{M_i(1), M_i(0)\}$
    - But we can't condition on the *latent* potential outcomes, we only condition on the observed M_i
    - This induces a form of "selection bias"
- Many settings in political science
    - Experiments with non-compliance
    - Attention checks in survey experiments.
    - Administrative data (police interactions are only recorded if a stop occurs)
    - Missingness caused by treatment (e.g. court proceedings that settle)

Post-treatment bias

- Let M_i denote the post-treatment covariate. Since it's post-treatment, it has potential outcomes $\{M_i(1), M_i(0)\}$ as though it were any other outcome.
- By randomization

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

- What happens if we take the difference-in-means conditional on $M_i = 1$

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

- By consistency:

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

Post-treatment bias

- Ignorability gets us

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

- Is this the ATE?
  - No! $M_i(1) = 1$ and $M_i(0) = 1$ define *two different subsets of the sample*
- Under what assumptions would we get the ATE?
- Either:
  1. No individual effect of treatment on M_i: $M_i(1) = M_i(0) \; \forall i$
  2. $\{M_i(1), M_i(0)\} \perp\!\!\!\perp \{Y_i(1), Y_i(0\}$
- Neither of these assumptions is guaranteed by an experiment since we don't randomize M_i
- Therefore, conditioning on a post-treatment quantity "breaks" the experiment -- now it's an observational study.

Example: Exercise experiment

- In the exercise experiment, we observe exercise/compliance with the incentives (once in week 1, 8 times in 2-5)
- What if we tried to estimate the "effect" conditional on having successfully complied with the incentives treatment

```
exercise2 <- exercise2 %>% mutate(reachedGoal = case_when(After1 > 0&(After2 + After3 + After4
                                                          TRUE ~ 0))

# Difference among those who did not
no_goal  <- lm_robust(After ~ I(treatment == "High"), data=exercise2 %>% filter(reachedGoal ==
tidy(no_goal) %>% filter(grepl("treatment", term)) %>% select(term, estimate, std.error, statis
```

```
##                          term estimate std.error statistic p.value
## 1 I(treatment == "High")TRUE    0.116     0.276     0.421   0.676
```

```
# Difference among those who reached goal
goal <- lm_robust(After ~ I(treatment == "High"), data=exercise2 %>% filter(reachedGoal != 0))
tidy(goal) %>% filter(grepl("treatment", term)) %>% select(term, estimate, std.error, statistic
```

```
##                          term estimate std.error statistic p.value
## 1 I(treatment == "High")TRUE    -1.13      0.53     -2.14   0.039
```

Example: Exercise experiment

- What's going on - why did we suddenly find a *negative* effect of treatment?
- Consider the treated and control groups among the incentives-completing subset
    - Treated group: People who worked out at the intense level having received the incentive
    - Control group: People who **still** worked out at the intense level **despite** not getting the incentive

Example: Non-compliance

- Consider the setting where the taking of treatment is subject to non-compliance.
    - Not everyone who is assigned treatment takes it and not everyone who is assigned to control stays in control.
    - Basically always happens in field experiments (in one direction at least)
- Let Z_i denote the *assigned* treatment and D_i denote the treatment that is actually taken
    - Z_i is randomized but D_i is not.
    - What happens when we analyze the difference between units with observed treatment $D_i = 1$ and observed treatment $D_i = 0$
    - D_i is post-treatment. It has potential outcomes $D_i(z)$. What are those groups?

Principal strata

- We can think of the combination of Z_i and D_i as defining a "sub-group" of units -- these are referred to as "principal strata"

Stratum	$D_i(1)$	$D_i(0)$
"Always-takers"	1	1
"Never-takers"	0	0
"Compliers"	1	0
"Defiers"	0	1

- Units with $D_i = 1$ could be any three of these strata. Even observing Z_i narrows it down to only two - we can't observe the strata directly.
- Strata aren't necessarily independent of potential outcomes $Y_i(d)$!

Treatment non-compliance

- In all cases of treatment non-compliance, we can always just take the difference between those *assigned* treatment and *assigned* control irrespective of their actual uptake. This is unbiased for the "intent-to-treat effect"
    - Challenge with interpreting the ITT -- it's strength depends on the *actual* effect of the treatment and the compliance rate.
    - Is a null ITT because our treatment doesn't work or because our encouragement doesn't work?
- When we get to instrumental variables, we'll talk about how -- in the treatment non-compliance setting -- we can obtain an estimate of the effect *among the compliers*
    - Unfortunately doesn't solve the problem of poor encouragements
    - Weak encouragement = biased/high-variance IV estimates.

Example: Administrative data

- Knox, Lowe and Mummolo (2020) consider the problem of estimating the effect of civilian race on police use of force.
  - Typically, past studies would use administrative data from police departments on stops
  - Compare police use of force among Black civilians who are stopped and white civilians who are stopped.
  - **Problem**: Stops are post-treatment!- Define D_i as the treatment (race of civilian), S_i is an indicator for whether a stop occurs, Y_i is severe use of force
- The difference-in-means does not identify the treatment effect - instead, we get:

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

- Let's think through the bias story. What's the substantive difference between these two sub-groups? Let's assume monotonicity in the effect of D_i on S_i
  - What strata are in the $S_i(1) = 1$ group?: $\{S_i(1) = 1, S_i(0) = 0\}$ and $\{S_i(1) = 1, S_i(0) = 1\}$
  - What strata are in the $S_i(1) = 0$ group?: $\{S_i(1) = 1, S_i(0) = 1\}$

Overview

- Take care when conditioning on variables that are affected by treatment!
- Results in a form of "selection bias" (often called 'collider bias' - we'll explain why with DAGs next week)
    - The treated group with observed mediator $M_i = 1$ is substantively different from the control group with
- We could define valid causal quantities conditional on the principal strata $\{M_i(1), M_i(0)\}$
    - But we can't observe them directly!
    - Can (with assumptions) observe them partially - we'll talk more about this in IV.
- Easy to identify "post-treatment" variables in an experiment. Trickier with poorly-defined interventions.
    - But be careful with variables that you might be implicitly conditioning on (e.g. analyzing only observed data)

Conclusion

- Three main reasons to care about covariates in an experiment
  1. **Improve precision**
  2. **Diagnose our experiment**
  3. **Examine sub-group effects** (careful to avoid p-hacking)
- Be careful with bad controls
  - We want to control for **pre-treatment** covariates and don't want to control for **post-treatment** covariates
  - Conditioning on **post-treatment** quantities breaks randomization
- Next week, we'll look at a fourth -- and most important -- reason for using covariates in *observational* designs: **adjusting for confounding**
  - Not a problem in experiments since we randomize treatment -- no observed or unobserved confounding
  - Big problem in observational designs since we don't randomize