5. Challenges and Tools in Empirical Work

PhD Applied Methods

Duncan Webb
NovaSBE

Spring 2026

Empirical work in practice

- In the previous lectures, we focused on the **identification** of causal effects
  - Potential outcomes framework
  - Randomized controlled trials
  - Selection bias and how to avoid it

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- In the previous lectures, we focused on the **identification** of causal effects
  - Potential outcomes framework
  - Randomized controlled trials
  - Selection bias and how to avoid it
- But implementing empirical research involves many additional **practical challenges**
- Today: focus on a set of tools and techniques for dealing with these challenges

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- We'll use **randomized controlled trials** as our main example throughout
- **Why?** Because with RCTs we can abstract away from identification concerns
- This allows us to focus on the **empirical difficulties** that arise even when identification is clean
- **Important**: Most of these tools apply to other research designs too (DiD, IV, RDD, etc.)

Overview of topics

1. **Clustering**: When and how to randomize at the group level
2. **Power**: Designing experiments with sufficient statistical power
3. **Heterogeneous treatment effects**: How do effects vary across groups?
4. **Attrition**: What to do when participants drop out?
5. **Multiple hypothesis testing**: How to avoid false discoveries?
6. **Robustness and replication**: How reliable are empirical results?
7. **Standard errors**: How to correctly compute uncertainty?
8. **Spillovers**: What if SUTVA is violated?
9. **Measurement**: How to deal with measurement error?

Beyond individual randomization

- So far: randomization at the **individual level**
- Sometimes randomizing individuals is:
  - **Impractical**: Hard to treat some students in a school but not others
  - **Unethical**: Perceived as unfair within communities
  - **Contaminated**: Treatment spills over between individuals

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- **Solution**: Randomize at a higher level - **cluster randomization**
- Treat entire groups (clusters) as units: schools, villages, clinics, firms

Examples of clustered randomization

- **Education**: Randomize schools (not students)
  - Teacher training programs
  - School infrastructure improvements
- **Health**: Randomize clinics or villages
  - Deworming programs (Miguel & Kremer 2004)
  - Community health worker programs
- **Development**: Randomize villages or districts
  - Microfinance expansion
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Key insight: The unit of randomization $\neq$ unit of analysis

Spillovers and SUTVA violations

Recall our SUTVA assumption: Y_i depends only on own treatment D_i

With spillovers, potential outcomes become:

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Cluster randomization partially solves this:

- Captures within-cluster spillovers
- But still misses cross-cluster spillovers

Notation for clustered designs

Let $c = 1, ..., C$ index clusters, $i = 1, ..., N_c$ index individuals within cluster c

- $D_c \in \{0, 1\}$: treatment status of cluster c
- Y_{ic}: outcome for individual i in cluster c
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Observed outcome:

$$Y_{ic} = D_c Y_{ic}(1) + (1 - D_c) Y_{ic}(0) \tag{4}$$

Note: Everyone in the cluster has the same D_c!

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Intuition: 1000 students in 10 schools provides **less information** than 1000 randomly selected students

$\Rightarrow$ Use **cluster-robust standard errors**

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Implications:

- More clusters $>$ bigger clusters (for statistical power)
- If $\rho = 0.05$ and $n = 20$: need $\approx 2\times$ the sample size!
- Rules of thumb: Need at least 20-30 clusters for reliable inference

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Bottom line: Use clustered randomization when spillovers matter or individual randomization is infeasible

Outline

Designing an experiment

Two main questions when designing an experiment:

1. Who to randomize, how, etc.
2. Sample size (and share treated)

Experiments are an unusual case where you have great control over sample size

The last thing you want: go through the whole burden and have insignificant effects because you have high standard errors

Finite sample and inference

So far, we have always considered the asymptotic values of the estimator

For instance:

$$\mathbb{E}[Y_i|D_i = 1]$$

is the asymptotic value of:

$$\frac{1}{N_1} \sum_{i \in D_i = 1} y_i$$

which, inversely, is the empirical counterpart to $\mathbb{E}[Y_i|D_i = 1]$

This is because we have been interested in **identification** (what we would learn in infinite samples)

Finite sample and inference

Random experiment: T and C are similar for $N = \infty$

In finite samples, T and C always *somewhat* different, e.g. by chance my treatment group has slightly older students than the control group

This **imbalance** could be confounded with treatment effect

Inference is accounting for that:
With finite sample, can I consider that the difference T vs. C is high enough to indicate more than unavoidable imbalance?
Yes, if statistically "significant"

Imbalance is not a source of bias; the standard error is there to account for that

Reminder: significance tests

Estimator $\hat{\beta}$ asymptotically normal with mean β and variance $V(\hat{\beta}) = \sigma_\beta^2$

If $\beta = 0$, then, for a risk α (e.g. 5%) we can define $t_{\alpha/2}$ such that:

$$P\left(-t_{\alpha/2} < \frac{\hat{\beta}}{\sigma_\beta} < t_{\alpha/2}\right) = 1 - \alpha$$

Thus

$$2\Phi(t_{\alpha/2}) - 1 = 1 - \alpha$$

and we can read $t_{\alpha/2}$ for the normal distribution table

For $\alpha = 0.05$, $\Phi(1.96) = 0.975$

If $|\hat{\beta}/\sigma_\beta| > 1.96$, we can reject the null $\beta = 0$

Balance table

Table A3: *Baseline balance: covariates*

Variable	(1) Total Mean/(SD)	(2) Control Mean/(SD)	(3) Base + YGL Mean/(SD)	(4) Base Only Mean/(SD)	(3)-(2) Pairwise t-test P-value	(4)-(2) Pairwise t-test P-value
Girl's age (years)	14.000	13.741	14.104	14.033	0.292	0.483
	(6.798)	(6.904)	(6.535)	(7.356)		
Girl has a brother (=1)	0.548	0.574	0.556	0.508	0.512	0.033**
	(0.548)	(0.531)	(0.567)	(0.511)		
Mother passed away (=1)	0.049	0.040	0.050	0.053	0.331	0.282
	(0.210)	(0.183)	(0.223)	(0.209)		
Mother in household (=1)	0.816	0.835	0.805	0.820	0.208	0.595
	(0.450)	(0.469)	(0.439)	(0.459)		
Guardian knows how to read and write (=1)	0.828	0.829	0.836	0.810	0.799	0.474
	(0.485)	(0.479)	(0.514)	(0.428)		
Guardian has no education (=1)	0.095	0.085	0.096	0.102	0.465	0.310
	(0.365)	(0.255)	(0.418)	(0.342)		
Guardian attended secondary or higher education (=1)	0.303	0.308	0.293	0.318	0.648	0.794
	(0.648)	(0.681)	(0.623)	(0.685)		
Guardian occupation: Agriculture (=1)	0.773	0.768	0.781	0.762	0.696	0.899
	(0.666)	(0.716)	(0.632)	(0.697)		
Observations	2390	568	1216	606		
Schools	140	35	70	35		

Notes: Sample includes all girls in baseline. Columns (1)-(4) show means and standard deviations of covariates from the girls' baseline survey. Columns (5)-(6) show the p-value of a pairwise test comparing *Base Only* and *Base + YGL* with *control* , respectively. Standard errors cluster at the school level. $* p < 0.1$, $** p < 0.05$, $*** p < 0.01$.

Overall balance

Often, because we are doing **multiple hypothesis tests** we will get a few significant imbalances when looking across multiple outcomes

How can we test for **overall** imbalance?

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How can we test for **overall** imbalance?

1. Run regression:

$$\text{Treated}_i = \beta_0 + \beta_1 \text{Outcome1}_i + \beta_2 \text{Outcome2}_i + ... + \beta_K \text{OutcomeK}_i \quad (6)$$

2. Use an F-test (joint test) of $\beta_1 = \beta_2 = ... = \beta_K = 0$

The power of the experiment

If the policy has an impact, we want to be able to see it

Unless the effect is very small, we want to reject the null

But if there is a lot of imprecision (large estimator variance), we may fail to do so

Type II error is the probability of a **false negative**, i.e., $\beta > 0$, but we fail to reject the null ($\hat{\beta}/\sigma_\beta < 1.96$). In other words, we fail to detect an effect that is really there.

This will happen sometimes, for some samples

Power = $1 - P$(Type II error), i.e. the probability that we detect an effect if there really is one.

The power of the experiment

Usual approach: set an acceptable power (typically 80%), and then:

1. Set a reasonable β that you feel you should be able to "see" (the **minimum detectable effect** you want
2. And figure out the sample size that ensures that power for a true effect β

Computing the power

Let's calculate the power, where ($\hat{\beta}/\sigma_\beta < 1.96$) and β is random:

$$P\left(\frac{\hat{\beta}}{\sigma_\beta} > t_{\alpha/2}|\beta\right) = \kappa$$

where κ is the power.

$$P\left(\frac{\hat{\beta}-\beta}{\sigma_\beta} > t_{\alpha/2} - \frac{\beta}{\sigma_\beta}|\beta\right) = \kappa$$

$$\Phi\left(\frac{\beta}{\sigma_\beta} - t_{\alpha/2}\right) = \kappa$$

Thus:

$$\frac{\beta}{\sigma_\beta} - t_{\alpha/2} = t_{1-\kappa}$$

Minimum detectable effect

The β that will be "significant" 80% of the time (at 5% level) is such that:

$$\frac{\beta}{\sigma_\beta} - t_{\alpha/2} = t_{1-\kappa}$$

or

$$\beta = (t_{\alpha/2} + t_{1-\kappa})\sigma_\beta$$

with $t_{\alpha/2} = 1.96$ if $\alpha = 0.05$ and $t_{1-\kappa} = 0.84$ if $\kappa = 0.80$

$(t_{\alpha/2} + t_{1-\kappa})\sigma_\beta$ is the **minimum detectable effect** (MDE)

MDE and sample size

Consider the model:

$$y = c + \beta D_i + u$$

Remember that:

$$\sigma^2_\beta = \frac{1}{\bar{D}(1-\bar{D})}\frac{V(u)}{N}$$

Thus:

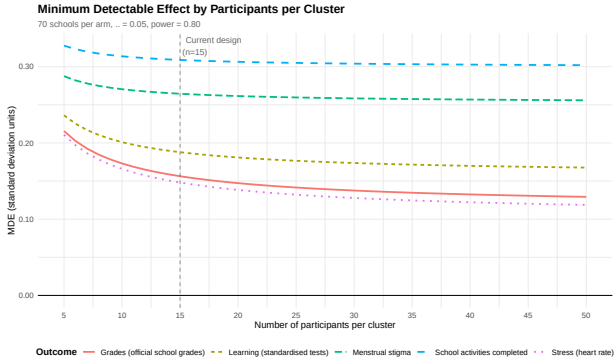
$$\text{MDE} = (t_{\alpha/2} + t_{1-\kappa})\sqrt{\frac{1}{\bar{D}(1-\bar{D})}\frac{V(u)}{N}}$$

Interpret each of those terms... (think in terms of finite sample imbalance)

How does MDE increase with sample size?

MDE and cluster size

Key insight: With clustered randomization, increasing cluster size doesn't decrease MDE much



What matters most is the **number of clusters**, not individuals per cluster

With instrumental variables

$$y = c + \beta T + u$$

where treatment T is instrumented by some random assignment D_i

Reminder:

$$V(\hat{\beta}_{IV}) = \frac{1}{\bar{D}(1-\bar{D})} \frac{V(u)}{N} \frac{1}{\pi_1^2}$$

The precision decreases linearly with the (net) take-up

So does the MDE

If take-up is 50%, implies more than doubling sample size.

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Beyond average treatment effects

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Conditional Average Treatment Effect (CATE):

$$\tau(X_i) = \mathbb{E}[Y_i(1) - Y_i(0)|X_i] \tag{8}$$

where X_i are observable characteristics

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Question: Why care about heterogeneity?

Why study heterogeneous effects?

1. **Policy targeting**
   - Which subgroups benefit most from the treatment?
   - Can improve cost-effectiveness

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- Which subgroups benefit most from the treatment?
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2. Understanding mechanisms

- How does the treatment work?
- Who is most affected and why?

3. External validity

- Will treatment work in different contexts?
- What characteristics predict larger effects?

Basic approach: Interaction terms

Simple regression with interactions:

$$Y_i = \alpha + \beta D_i + \gamma X_i + \delta(D_i \times X_i) + u_i \tag{9}$$

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Interpretation:

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Can test: $H_0 : \delta = 0$ (no heterogeneity)

Example: Cash transfers and suicide (Roth et al. 2019)

Setting: Rollout of unconditional cash transfer in Indonesia
Treatment: ˜10% of household expenditure
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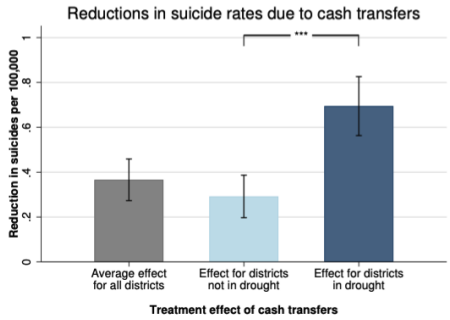
Heterogeneity:

- Larger reductions in areas with economic distress
- Larger effects in areas experiencing drought
- Benefits greatest among most vulnerable



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$\implies$ Need more disciplined, data-driven approach

Machine learning for CATEs

Goal: Estimate $\tau(x) = \mathbb{E}[Y_i(1) - Y_i(0)|X_i = x]$ in data-driven way

Two fundamental challenges:

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Two fundamental challenges:

1. The "ground truth" problem

- Standard ML: Cross-validate by comparing $\hat{Y}_i$ to observed Y_i
- Causal inference: We *never* observe $\tau_i = Y_i(1) - Y_i(0)$ for any unit
- $\implies$ Cannot directly cross-validate treatment effect predictions

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2. The "adaptive bias" problem

- Standard CART uses same data to: (1) find splits, (2) estimate effects
- You split *because* $\hat{\tau}$ looked large $\implies$ estimates biased upward
- Like picking the best-performing stock, then reporting its past returns as expected future returns

Causal trees (Athey & Imbens 2016)

Idea: Partition covariate space to maximize treatment effect heterogeneity

"Honest" estimation (key innovation):

1. Split sample in half: training + estimation
2. Use training data to build tree (decide where to split)
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Causal forests (Wager & Athey 2018): Average many honest trees for smoother estimates

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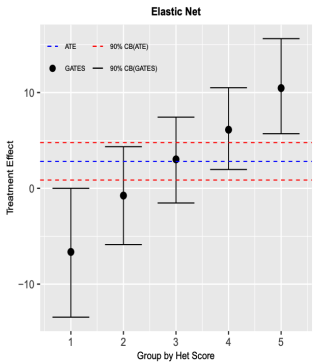
Interpretation: If ML detects real heterogeneity, Group 5 should have larger effects than Group 1



Upward slope $\Longrightarrow$ ML captures real heterogeneity

CLAN: Who are the high and low responders?

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Once GATES reveals heterogeneity, **who** are high vs. low responders?

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CLAN finding: Villages with low baseline rates benefited most $\implies$ target rollout there

TABLE 5. CLAN of Immunization Incentives

	Elastic Net		
	20% Most (δ_5)	20% Least (δ_1)	Difference ($\delta_5 - \delta_1$)
Number of vaccines to pregnant mother	2.161 (2.110,2.212) -	2.288 (2.237,2.337) -	-0.128 (-0.200,-0.055) [0.001]
Number of vaccines to child since birth	4.230 (4.100,4.369) -	4.714 (4.573,4.860) -	-0.513 (-0.710,-0.311) [0.000]
Fraction of children received polio drops	1.000 (1.000,1.000) -	1.000 (1.000,1.000) -	0.000 (0.000,0.000) [0.000]
Number of polio drops to child	2.964 (2.954,2.975) -	2.998 (2.987,3.007) -	-0.033 (-0.047,-0.019) [0.000]
Fraction of children received immunization card	0.899 (0.878,0.922) -	0.932 (0.908,0.956) -	-0.036 (-0.065,-0.004) [0.000]
Fraction of children received Measles vaccine by 15 months of age	0.127 (0.100,0.155) -	0.255 (0.230,0.282) -	-0.131 (-0.167,-0.094) [0.052]
Fraction of children received Measles at credible locations	0.290 (0.252,0.327) -	0.435 (0.400,0.470) -	-0.152 (-0.198,-0.097) [0.000]

Notes: Medians over 100 splits. 90% confidence interval in parenthesis.
Notes: P-values for the hypothesis that the parameter is equal to zero in brackets.

Top 20% vs. bottom 20% responders

Outline

The problem of attrition

Attrition: Participants drop out between treatment assignment and outcome measurement

Common in:

- Surveys with multiple waves
- RCTs
- Any study with follow-up measurement

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Why is this a problem?

Even with perfect randomization, attrition can create selection bias

Example: Job training RCT

Setup: Randomize 1,000 people to training vs. control

Measure employment 1 year later

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Issue: If attrition related to outcomes, estimates biased

- Maybe successful people in treatment don't respond (too busy working)
- Maybe unsuccessful people in control don't respond (discouraged)
- $\Longrightarrow$ Comparing non-random samples

Testing for differential attrition

First step: Test whether attrition differs by treatment status

Regress attrition indicator on treatment:

$$\text{Attrited}_i = \alpha + \beta D_i + u_i \tag{10}$$

Test $H_0 : \beta = 0$

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If reject H_0: **differential attrition**

Treatment affects who stays in sample

But: Even if no differential attrition, can still have bias if attrition related to potential outcomes

Testing attrition on baseline characteristics

Additional test: Compare baseline characteristics of attriters vs. non-attriters

For each baseline covariate X_i:

$$X_i = \alpha + \beta \text{Attrited}_i + \gamma D_i + \delta(\text{Attrited}_i \times D_i) + u_i \tag{11}$$

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Important: These are diagnostic tests, not solutions

Solutions to attrition

Three main approaches:

1. **Prevention** (best option)
   - Minimize attrition through study design
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   - Worst-case scenario analysis
   - No parametric assumptions

3. **Modeling** (e.g., inverse probability weighting)
   - Reweight to correct for selection
   - Requires strong assumptions

Lee bounds: Intuition

Idea (Lee 2009): Bound treatment effect without knowing why people attrite

Key assumption: Monotonicity

- Treatment affects attrition in only one direction
- E.g., treatment only increases attrition (or only decreases it)

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Logic:

- Suppose treatment group has 80% response, control has 100%
- 20% of treatment group are "extra attriters" caused by treatment
- Worst case: these 20% had highest (or lowest) outcomes
- $\Longrightarrow$ Trim top/bottom 20% of treatment group to get bounds

Lee bounds: Method

Setup: Let $S_i(d) \in \{0, 1\}$ indicate whether observed if assigned treatment d

Monotonicity assumption: $S_i(1) \geq S_i(0)$ for all i (or vice versa)

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Intuition: We don't know which 20% would have attrited, so consider worst cases

Manski bounds

Alternative approach (Manski 1990): Even weaker assumptions

For attriters, assume worst-case outcomes within support $[y_{\min}, y_{\max}]$:

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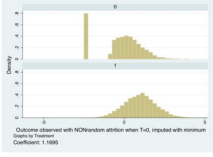

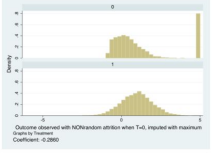

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Alternative approach: Model attrition probability

Basic intuition: Reweight observed sample to look like full sample (before attrition)

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$$Y_i = \alpha + \tau D_i + X_i' \beta + (X_i \times D_i)' \gamma + \varepsilon_i \tag{12}$$

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Assumption: Attrition independent of outcomes conditional on (X_i, D_i) — Much stronger than Lee bounds!

Outline

What is a p-value?

Question: What is the definition of a p-value?

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Standard hypothesis testing assumes: **We only do one test**

Problem: What if we run multiple tests?

The multiple testing problem

Thought experiment: Run 1,000 regressions on completely random data with $Y_i \perp X_i$

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$\Longrightarrow$ Our published results will be full of false positives!

Why this matters for empirical work

Multiple testing arises naturally in many contexts:

- Testing treatment effects on **many outcomes**
  - Health study: blood pressure, cholesterol, weight, ...
  - Education: test scores, attendance, graduation, ...

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  - Education: test scores, attendance, graduation, ...
- Testing for **heterogeneous effects**
  - By gender, age, income, region, ...
- Testing **many specifications**
  - Different control variables
  - Different samples
  - Different functional forms

Solutions: Pre-analysis plans

Pre-analysis plan (PAP): Document research design before seeing data

Specify in advance: primary outcomes, subgroup analyses, specifications

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Limitation: Doesn't solve multiple testing itself—still need to adjust p-values

Solutions: Adjust p-values

Idea: Adjust significance thresholds to account for multiple testing

Bonferroni correction (classic method):

- Testing m hypotheses
- Reject if $p < \alpha/m$
- Example: 20 tests, want FWER $\leq 0.05 \implies$ use threshold 0.05/20 = 0.0025

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Problem: Very conservative

- Low power to detect true effects
- Especially with many tests

Family-Wise Error Rate (FWER)

Definition: Probability of making *at least one* false rejection

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Issue: Very stringent criterion

- In exploratory research, may accept some false positives
- Want to balance false positives vs. false negatives

False Discovery Rate (FDR)

Definition: Expected proportion of false rejections among all rejections

$$\text{FDR} = \mathbb{E}\left[\frac{\text{False positives}}{\text{Total rejections}}\right] \tag{14}$$

Interpretation: Among all tests you reject, what fraction are false positives?

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Interpretation: Among all tests you reject, what fraction are false positives?

Difference from FWER:

- FWER: Probability of *any* false positive
- FDR: *Rate* of false positives among rejections
- FDR is less stringent $\implies$ more power

Benjamini-Hochberg procedure (1995)

Goal: Control FDR at level q (e.g., $q = 0.10$)

Procedure for m hypothesis tests:

1. **Sort p-values**: $p_{(1)} \leq p_{(2)} \leq \cdots \leq p_{(m)}$

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Result: FDR $\leq q$ under independence

Benjamini-Hochberg: Intuition

Key insight: Look for p-values that are “too small” to be false positives

Under null: p-values uniformly distributed on [0,1]

Under alternative: p-values concentrated near 0

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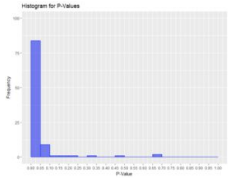
BH procedure finds cutoff where p-values deviate from uniform distribution

Benjamini-Hochberg: Visual intuition

True rejections: Concentrated near zero



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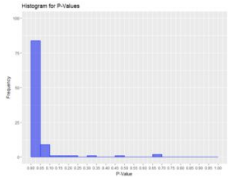
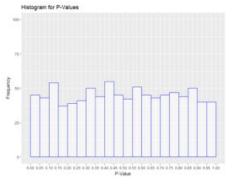


False rejections: Uniformly distributed



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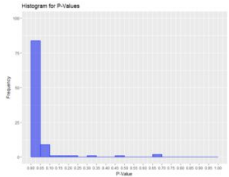

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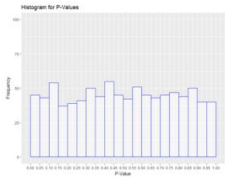
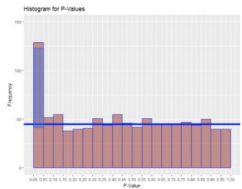


BH isolates p-values above uniform cutoff:



Example: Testing 10 hypotheses

Suppose we have 10 p-values, want FDR ≤ 0.10:

Rank k	$p_{(k)}$	$\frac{k}{m} \cdot q = \frac{k}{10} \cdot 0.10$	Reject?
1	0.001	0.010	✓
2	0.008	0.020	✓
3	0.015	0.030	✓
4	0.042	0.040	✓
5	0.056	0.050	×
6	0.120	0.060	×
⋮	⋮	⋮	×

Largest k with $p_{(k)} \leq k \cdot 0.01$ is $k = 4 \implies$ Reject first 4 hypotheses

Other approaches to reduce multiple testing

Beyond adjusting p-values:

1. Reduce number of tests / Use summary indices

- Pre-specify primary outcome(s)
- Use F-tests for joint hypotheses
- Combine outcomes into summary index (e.g., Anderson 2008)
  - Average standardized effects across related outcomes
  - Example: "Cognitive index" = average of test scores
  - Reduces m tests to 1 test

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2. Report all tests transparently

- Show results for all outcomes (not just significant ones)
- Let readers judge robustness

Practical recommendations

What should you do?

- **Pre-specify** main hypotheses when possible
- **Distinguish** confirmatory vs. exploratory analyses
- **Adjust p-values** when testing multiple outcomes
  - BH procedure for FDR control
  - Bonferroni for FWER control (if very conservative)
- **Report transparently**
  - Show all outcomes tested
  - Report both adjusted and unadjusted p-values
- **Implementation**: Available in R (`p.adjust()`) and Stata (`rwolf`, `wyoung`)

Outline

The replication crisis

Mounting evidence that empirical results are less robust than we thought

Problem 1: Low replication rates

- Psychology replication project: only 39% of studies replicate
- Economics: similar concerns emerging

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- Different choices $\implies$ different results
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Goal: Get to the truth! But what went wrong?

Evidence: Garden of forking paths

Study: Breznau, Rinke, Wuttke (PNAS 2022)

Design: 73 research teams test same hypothesis with same data

Does immigration reduce support for social policies?

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Does immigration reduce support for social policies?

Results: Massive variance in estimates

- Estimates ranged from strongly negative to strongly positive
- Driven by: controls, fixed effects, clustering, sample choices
- Much variance unexplained even controlling for observables

Evidence: Researcher-driven variation

Study: Huntington-Klein et al.

Design: 146 economist teams estimate same effect with progressive restrictions

1. No restrictions (same data)
2. Specify DiD design
3. Pre-cleaned data

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1. No restrictions (same data)
2. Specify DiD design
3. Pre-cleaned data

Results:

- IQR of 3-4pp (avg effect: 4pp) with freedom
- IQR still 2pp with pre-cleaned data
- Both data and analytical choices matter

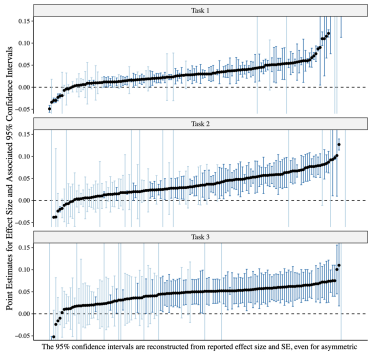

Figure 3: Specification Curve for All Reported Estimates by Task with Estimates Ordered From Smallest to Largest

What went wrong?

Three distinct but related issues:

1. Researcher degrees of freedom

- Many choices: sample definition, controls, specifications, outliers
- Different reasonable choices $\implies$ different results
- Creates flexibility to find "significant" results (even unintentionally)

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1. Researcher degrees of freedom

- Many choices: sample definition, controls, specifications, outliers
- Different reasonable choices $\implies$ different results
- Creates flexibility to find "significant" results (even unintentionally)

2. Publication bias (editorial decisions)

- Journals prefer novel, significant, "clean" results
- Null results, replications rarely published (file drawer problem)
- $\implies$ Published literature overrepresents significant findings

Solutions

How to improve reliability of empirical research?

1. Transparency

- Pre-register analysis plans (PAPs)
- Post data and code (replication packages)
- Report all specifications, not just significant ones

Solutions

How to improve reliability of empirical research?

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- Pre-register analysis plans (PAPs)
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- Report all specifications, not just significant ones

2. Robustness checks

- Show results not driven by one specific choice
- Vary sample, controls, functional forms
- Put extensive checks in appendix

Solutions

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1. Transparency

- Pre-register analysis plans (PAPs)
- Post data and code (replication packages)
- Report all specifications, not just significant ones

2. Robustness checks

- Show results not driven by one specific choice
- Vary sample, controls, functional forms
- Put extensive checks in appendix

3. Design for power

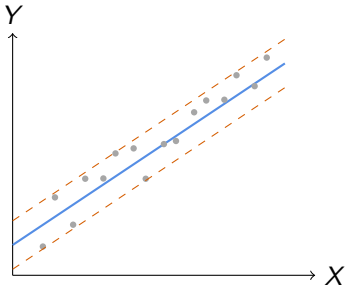
- Choose designs with high statistical power
- Reduces false negatives and winner's curse
- Better to detect true effects reliably

Outline

Homoskedasticity vs. Heteroskedasticity

Basic OLS assumption: Constant error variance (homoskedasticity)

Homoskedasticity



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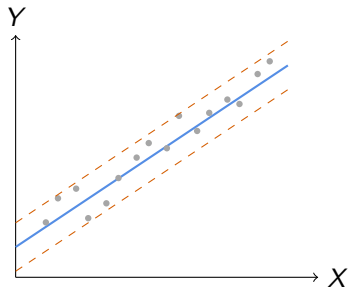

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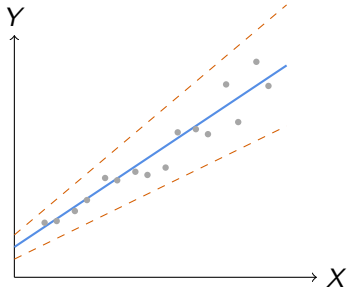

$V(\varepsilon_i|X_i) = \sigma_i^2$ (varies)

Variance of OLS estimator

Under homoskedasticity:

$$V(\hat{\beta}) = \sigma^2(X'X)^{-1} \tag{16}$$

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where $\Omega = \text{diag}(\sigma_1^2, \ldots, \sigma_n^2)$

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where $\Omega = \text{diag}(\sigma_1^2, \ldots, \sigma_n^2)$

Problem: If we use homoskedastic formula when heteroskedasticity present:

- Standard errors are wrong
- Confidence intervals and hypothesis tests invalid

Practical recommendation

Key point: There is almost never a good justification for assuming homoskedasticity

$\Longrightarrow$ **Always use robust (heteroskedasticity-consistent) standard errors**

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  - `reg y x, robust`

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$\Longrightarrow$ **Always use robust (heteroskedasticity-consistent) standard errors**

Implementation:

- **Stata**: Add `, robust` option to regression
  - `reg y x, robust`
- **R**: Use `vcovHC()` from `sandwich` package
  - `coeftest(model, vcov = vcovHC(model, type="HC1"))`

This should be your *default*, not an exception!

Clustering: Why does it matter?

Standard OLS assumption: Observations are independent (IID)

Problem: Often observations are correlated within groups

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Common examples:

- **Panel data**: Same individual over time
- **Schools/classrooms**: Students in same school
- **Geographic/spatial**: Units in same region
- **Families**: Siblings, households

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Key distinction: This is about *correlation in residuals*, not a SUTVA violation

Treatment of one unit doesn't affect others, but their errors are correlated

Clustered errors: Covariance structure

Example: 2 groups, 3 people each

Without clustering (IID)

$$\Omega = \begin{pmatrix} \sigma^2 & 0 & 0 & 0 & 0 & 0 \\ 0 & \sigma^2 & 0 & 0 & 0 & 0 \\ 0 & 0 & \sigma^2 & 0 & 0 & 0 \\ 0 & 0 & 0 & \sigma^2 & 0 & 0 \\ 0 & 0 & 0 & 0 & \sigma^2 & 0 \\ 0 & 0 & 0 & 0 & 0 & \sigma^2 \end{pmatrix}$$

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Diagonal: all errors independent

With clustering

$$\Omega = \begin{pmatrix} \sigma^2 & \sigma_{12} & \sigma_{13} & 0 & 0 & 0 \\ \sigma_{12} & \sigma^2 & \sigma_{23} & 0 & 0 & 0 \\ \sigma_{13} & \sigma_{23} & \sigma^2 & 0 & 0 & 0 \\ 0 & 0 & 0 & \sigma^2 & \sigma_{45} & \sigma_{46} \\ 0 & 0 & 0 & \sigma_{45} & \sigma^2 & \sigma_{56} \\ 0 & 0 & 0 & \sigma_{46} & \sigma_{56} & \sigma^2 \end{pmatrix}$$

Red: within-cluster correlations

Why clustering increases standard errors

Intuition: Correlation reduces effective sample size

Each observation provides *less independent information* when correlated

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If you ignore clustering:

- Standard errors too small
- Over-rejection of null hypotheses
- False confidence in results

Modeling cluster structure: Random effects

Approach 1: Assume random group effects (panel data structure)

Error decomposes: $\varepsilon_{gi} = u_g + v_{gi}$ where u_g is group shock, v_{gi} is idiosyncratic

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Then the covariance matrix Ω_g is:

$$\Omega_g = \begin{pmatrix} \sigma_u^2 + \sigma_v^2 & \sigma_u^2 & \cdots & \sigma_u^2 \\ \sigma_u^2 & \ddots & \ddots & \vdots \\ \vdots & \ddots & \ddots & \sigma_u^2 \\ \sigma_u^2 & \cdots & \sigma_u^2 & \sigma_u^2 + \sigma_v^2 \end{pmatrix}$$

with $V(u_g) = \sigma_u^2$ and $V(v_{gi}) = \sigma_v^2$

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with $V(u_g) = \sigma_u^2$ and $V(v_{gi}) = \sigma_v^2$

Note: $\forall i \neq j : \text{cov}(\varepsilon_{gi}, \varepsilon_{gj}) = \sigma_u^2$

Modeling cluster structure: Moulton factor

Approach 2: Constant within-group correlation (Moulton 1986)

Assumes within-group correlation is constant: $\text{cor}(\varepsilon_i, \varepsilon_j) = \rho$ if $G_i = G_j$, else 0

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Leading to covariance matrix: $\Omega_g = \sigma_e^2 \begin{pmatrix} 1 & \rho & \cdots & \rho \\ \rho & \ddots & \ddots & \vdots \\ \vdots & \ddots & \ddots & \rho \\ \rho & \cdots & \rho & 1 \end{pmatrix}$

Modeling cluster structure: No assumptions

Approach 3: Don't assume specific structure—just estimate

$$\widehat{\Omega}_g = \begin{bmatrix} \hat{u}_{g1}^2 & \hat{u}_{g1}\hat{u}_{g2} & \hat{u}_{g1}\hat{u}_{g3} \\ \hat{u}_{g2}\hat{u}_{g1} & \hat{u}_{g2}^2 & \hat{u}_{g2}\hat{u}_{g3} \\ \hat{u}_{g3}\hat{u}_{g1} & \hat{u}_{g3}\hat{u}_{g2} & \hat{u}_{g3}^2 \end{bmatrix}$$

This is what **cluster-robust standard errors** do

Most flexible approach—recommended!

Practical recommendations: Clustering

When to cluster?

Whenever observations might be correlated within groups

How to choose cluster level?

- Cluster at level where treatment is assigned (if applicable)
- Cluster at highest level of potential correlation
- When in doubt: cluster at higher level (more conservative)

Practical recommendations: Clustering

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- Cluster at level where treatment is assigned (if applicable)
- Cluster at highest level of potential correlation
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Implementation:

- **Stata**: `reg y x, cluster(groupvar)`
- **R**: `coeftest(model, vcov = vcovCL(model, cluster = ~groupvar))`

Result: Accounting for clustering *almost always* increases SEs

Outline

Spillovers as SUTVA violations

Recall SUTVA (Stable Unit Treatment Value Assumption):

Treatment of unit i doesn't affect outcomes of unit j

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Recall SUTVA (Stable Unit Treatment Value Assumption):

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Examples:

- Vaccines: My vaccination protects you (positive spillover)
- Deworming: Treating some kids reduces transmission to others
- Information: Treated individuals share knowledge with control group
- Market effects: Training increases labor supply, affects wages

Indirect treatment effect

Object of interest: How much does treatment of others affect me?

Indirect Treatment Effect (ITE): Compare untreated in treated clusters vs. untreated in control clusters

$$\text{ITE} = \mathbb{E}[Y_i(0, D_{-i} = 1) - Y_i(0, D_{-i} = 0) | D_i = 0] \tag{19}$$

where:

- $Y_i(0, D_{-i} = 1)$: Outcome for untreated i when others in cluster are treated
- $Y_i(0, D_{-i} = 0)$: Outcome for untreated i when others in cluster are untreated

If ITE $\neq 0$, SUTVA is violated

Measuring spillovers: Two-step clustered RCT

Design:

1. **Step 1**: Randomize which clusters get treatment (e.g., schools, villages)

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This allows estimation of:

- **Direct effect**: Treated vs. untreated in same cluster
- **Indirect effect**: Untreated in treated cluster vs. untreated in control cluster
- **Total effect**: Treated in treated cluster vs. untreated in control cluster

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- **Total effect**: Treated in treated cluster vs. untreated in control cluster

Extension: Can vary proportion treated in each cluster (e.g., {0%, 30%, 60%})

Estimates how spillovers change with treatment intensity (but requires high power)

Example: Miguel & Kremer (2004) deworming study

Setting: Deworming treatment in Kenyan schools

Design: Phased randomization of schools

- Group 1 schools treated in 1998
- Group 2 schools treated in 1999
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Implication: Standard RCT would *underestimate* total benefits

(Control group also benefits from nearby treatment)

Testing for spillovers: The naive approach

Idea: Test if treatment effects vary by exposure to treated individuals

Basic regression:

$$Y_i = \alpha + \beta D_i + \gamma \text{Exposure}_i + \varepsilon_i \tag{20}$$

where Exposure_i = number of treated individuals near i

Problem with naive approach

Issue: Exposure is not exogenous!

Example problems:

- Urban vs. rural: People in dense cities more exposed to treated units
- Social networks: Popular people more exposed
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$\Longrightarrow$ Exposure$_i$ correlated with unobservables (e.g., density, centrality)

$\Longrightarrow$ γ is **biased** (OVB problem)

Cannot distinguish true spillovers from confounding by exposure-related factors

Solution: Recentering approach (Borusyak & Hull 2023)

Key idea: Control for *expected* exposure under random assignment

Steps:

1. Simulate random assignments many times (e.g., 10,000)
2. Calculate expected exposure: $\mathbb{E}[\text{Exposure}_i]$ for each unit

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Steps:

1. Simulate random assignments many times (e.g., 10,000)
2. Calculate expected exposure: $\mathbb{E}[\text{Exposure}_i]$ for each unit
3. Regress outcome on actual exposure, controlling for expected exposure:

$$Y_i = \beta D_i + \gamma \text{Exposure}_i + \delta \mathbb{E}[\text{Exposure}_i] + \varepsilon_i \tag{21}$$

Recentering: Intuition

Why does this work?

- $\mathbb{E}[\text{Exposure}_i]$ captures confounders (density, centrality, etc.)

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- These confounders affect *expected* exposure, not random deviations
- Actual exposure $-$ expected exposure = exogenous variation from randomization
- γ identifies causal spillover effect from this exogenous variation

Summary: Spillovers

Key takeaways:

- Spillovers violate SUTVA—treatment of i affects j
- Can be positive or negative, and quantitatively important
- **In RCTs**: Use two-step clustered design to measure indirect effects
- **Testing for spillovers**:
  - Naive exposure regressions are biased
  - Use recentering approach (Borusyak & Hull 2023)
  - Control for expected exposure to remove confounding
- Applies beyond RCTs: Any setting with spatial/network structure
- **Practical advice**: Always consider whether spillovers plausible in your setting

Outline

Measurement matters

Often overlooked: Measurement is critical but undervalued in econometrics courses

Key insight: Regardless of causal identification, if we don't measure the right thing, we can't even know Y_i!

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Two types of measurement error:

1. **Classical measurement error**: Random noise, independent of true value
   - $X_i^{\text{observed}} = X_i^{\text{true}} + \varepsilon_i$ where $\mathbb{E}[\varepsilon_i] = 0$, $\text{Cov}(X_i^{\text{true}}, \varepsilon_i) = 0$

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2. **Non-classical measurement error**: Systematic, correlated with truth: $\text{Cov}(X_i^{\text{true}}, \varepsilon_i) \neq 0$
   - Example: Rich people overreport income, poor people underreport
   - Can bias estimates in *any* direction

Classical measurement error: Attenuation bias

Setup: True model: $Y_i = \alpha + \beta X_i^{\text{true}} + u_i$; observe $X_i^{\text{obs}} = X_i^{\text{true}} + \varepsilon_i$

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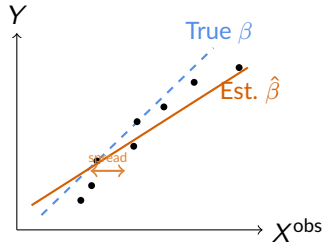
Regressing Y_i on X_i^{obs}:

$$\hat{\beta} = \frac{\text{Cov}(Y_i, X_i^{\text{obs}})}{\text{Var}(X_i^{\text{obs}})} = \frac{\beta \text{Var}(X_i^{\text{true}})}{\text{Var}(X_i^{\text{true}}) + \text{Var}(\varepsilon_i)} = \beta \cdot \underbrace{\frac{\text{Var}(X^{\text{true}})}{\text{Var}(X^{\text{true}}) + \text{Var}(\varepsilon)}}_{\text{attenuation factor } <1} \tag{22}$$

$\Longrightarrow$ **Attenuation bias**: Estimates biased toward zero

Visualizing measurement error

Noise in X (attenuation)



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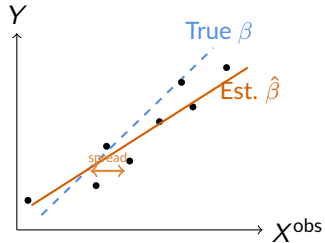

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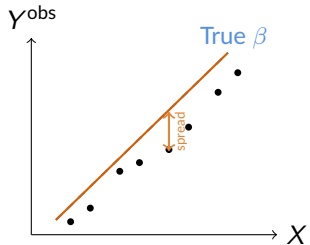
Noise in Y (no bias)



Noise **spreads out** Y **conditional on** X (vertically)
$\Longrightarrow$ Same slope, no bias, just less precision

Sources of bias: Self-reported data (Part 1)

Self-reports are common but problematic. Key sources of bias:

1. Recall bias

- People misremember past events/behaviors
- **Example**: Arthi et al. (2018, Tanzania): People report 4$\times$ more farm work hours when recalling vs. real-time measurement
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4. Framing effects

- Same information, different presentation $\implies$ different responses
- **Examples**:
  - "90% survival rate" vs. "10% mortality rate"
  - "Tax relief" vs. "tax cuts" vs. "tax decreases"

Sources of bias: Self-reported data (Part 3)

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5. Selection bias

- Who answers the survey?
- Non-response often non-random
- **Example**: US political polling often has $<$5% response rate (Pew)
- Those who respond differ systematically from non-responders

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6. Hypothetical vs. incentivized responses

- Hypothetical: "Would you buy this product at $10?"
- Incentivized: Actually buying with real money
- **Recommendation**: Use incentive-compatible elicitation when possible
- Revealed preferences $>$ stated preferences

Partial solution to classical error: Creating indices

Idea: Combine multiple noisy measures to reduce measurement error

Simple case: Average two measures X_1 and X_2 of same construct: $X^{\text{index}} = (X_1 + X_2)/2$

If both have independent noise: Var(noise in index) $= \frac{1}{2}$Var(noise in each)

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More generally: n measures $\implies$ noise variance reduced by factor of $1/n$

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If both have independent noise: Var(noise in index) $= \frac{1}{2}$Var(noise in each)

Derivation: Let $X_j = X^{\text{true}} + \varepsilon_j$ with $\varepsilon_1 \perp \varepsilon_2$. Then: $X^{\text{index}} = X^{\text{true}} + \frac{\varepsilon_1+\varepsilon_2}{2}$ and
$\text{Var}\left(\frac{\varepsilon_1+\varepsilon_2}{2}\right) = \frac{1}{4} \cdot 2\sigma_\varepsilon^2 = \frac{1}{2}\sigma_\varepsilon^2$

More generally: n measures $\implies$ noise variance reduced by factor of $1/n$

Key assumption: Measurement errors independent (if correlated, gains smaller)

Weighted indices

Can do better than simple averages by weighting measures differently:

1. **Sum of standardized scores** (Kling et al. 2007)
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3. **Inverse-covariance weighting**
   - Variables uncorrelated with others provide independent info $\implies$ weight more
   - Opposite logic to factor analysis—context determines which is better

Dealing with outliers and missing values

Outliers: Extreme values can drive results

Solutions:

- **Winsorization**: Cap extreme values at percentile (e.g., 1st/99th)
- **Trimming**: Drop extreme observations entirely
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- **Robust regression**: Median regression (LAD), Huber M-estimator

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Missing values: Often not missing at random

Solutions:

- **Imputation**: Replace with median, mean, or predicted values
- **Bounds / IPW**: As with attrition (Lee bounds, inverse probability weighting)
- **Missing indicator**: Include dummy for missing, set value to 0
- **Leave as missing**: Report sample size, acknowledge limitation

Best practices: No single “right” answer

Reality: For many measurement issues, no obviously “best” approach

The strategy:

1. **Be clear and justify your choices**
   - Explain why you chose specific method
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Remember: Good measurement is as important as good identification

Can have perfect RCT but meaningless results if measuring wrong thing

Key Takeaways

Main lessons from today:

1. **Heterogeneous effects**: Average effects may mask important variation. Use modern ML methods (GATES, causal forests) to study heterogeneity rigorously.

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3. **Multiple hypothesis testing**: Testing many hypotheses inflates false positives. Control FDR (Benjamini-Hochberg) or FWER (Bonferroni). Pre-specify analyses.
4. **Replication**: Results less robust than we thought. Researcher degrees of freedom, publication bias, and winner's curse all contribute. Transparency and robustness checks are essential.

Key Takeaways (cont.)

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Overall theme: Even with perfect identification, empirical work requires careful attention to practical challenges. Good research design anticipates and addresses these issues proactively.

GATES/CLAN: Why sample splitting matters

Overfitting problem: Using *same* data to train ML and estimate GATES $\implies$ biased estimates

Solution: Sample splitting

- **Auxiliary sample**: Train ML predictor
- **Main sample**: Estimate GATES/CLAN (not used to choose groups)

Aggregation: Results may depend on the particular random split

- Repeat with many splits, report **median** estimates
- More robust than any single split

Implementation: R package `GenericML`

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