



Causal Inference in Observational Studies

Theory, Methods and Presentation

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*Disclaimer: sections and lines in brown correspond to content which is **very much** ‘under construction’.*

1 Definitions

1.1 Research design and identification strategy

Research design = working from the research question, the overall manner in which data will be gathered, assembled and assessed in order to draw conclusions.

In the applied economics literature, and only in the context of (1) *observational* studies that aim to (2) identify a *causal* effect, a subordinate notion is the “identification strategy”¹.

If one’s research goal is to identify the causal effects of a specific event or situation (“treatment”), and sets internal validity as the priority², then one wants a research design that may credibly identify causal effects. That ideal research design is a **randomized trial**: an experiment which randomly assigns the participants to either a treatment or a control group.

This experiment can be considered as the “gold standard” against which to judge other research designs. Therefore, in an observational study, one attempts to approximate the force of evidence generated by such an experiment. A key aspect of the research design is hence the identification strategy:

Identification (causal inference) strategy = how *observational* data are used to approximate a real experiment. It is the set of assumptions that will *identify* the causal effect of interest, including:

- = $\left\{ \begin{array}{l} - \text{a clear source of identifying variation in a causal variable,} \\ - \text{the use of a particular econometric technique to exploit this information.} \end{array} \right.$

1.2 Experiments, natural experiments, quasi-experiments

A true experiment is a study in which the researcher manipulates the level of a *treatment* (the independent variable of interest) and measures the outcome (the dependent variable of interest). All the important factors that might affect the phenomena of interest are controlled.

A natural experiment is an observational study in which a *randomization* of a treatment X or instrument Z has occurred naturally – mimicking the exogeneity of a randomized experiment. Researchers do not create natural experiments – they find them.

Ex: weather

A quasi experiment is a study of intentional treatment, that resembles a randomized field experiment but lacks full random assignment. Participants are *not* randomly assigned to the treatment or control group. The groups therefore differ in often unobservable ways, so one must control for as many of these differences as possible. The control group is rather called a “comparison” group.

Ex: In the 1990s, the U.S. Department of Housing and Urban Development (HUD) implemented a grant program to encourage resident management of low-income public housing projects. Housing projects were *selected* in 11 cities nationwide, so the treatment (the award of HUD funding) was not randomly assigned. But “similar” housing projects in the same cities provided a reasonably valid comparison, so the HUD was able to evaluate the program.

¹Angrist and Pischke (2010) use the notions of research design and identification strategy interchangeably.

²True experimental designs may be the “gold standard” of scientific research when considering only internal validity. However, the very methods used to increase internal validity may also limit the generalizability or external validity of the findings. Ex: a zoo is a controllable setting amenable to drawing causal inferences about the behavior of animals, but these inferences may not generalize to the behavior of animals in the wild.

2 Theory

2.1 The original selection bias problem and the CIA

We have a treatment of interest $D_i \in \{0, 1\}$ whose causal effect we want to estimate. Let Y_i be the realized outcome, Y_{0i}, Y_{1i} the potential outcomes. The potential outcomes framework³ allows us to define:

– Treatment effect (TE)	$Y_{1i} - Y_{0i}$	<i>what we want to estimate</i>
– Average treatment effect (ATE)	$\mathbb{E}[Y_{1i} - Y_{0i}]$	<i>what we ideally want to compute</i>
– Average treatment effect on the treated (ATET)	$\mathbb{E}[Y_{1i} - Y_{0i} D_i=1]$	<i>what we reasonably want to compute</i>
– Difference in average observed outcomes	$\mathbb{E}[Y_i D_i=1] - \mathbb{E}[Y_i D_i=0]$	<i>what we can compute</i>
– Difference in average observed outcomes for same X_i	$\mathbb{E}[Y_i D_i=1, X_i] - \mathbb{E}[Y_i D_i=0, X_i]$	<i>what we can compute</i>

The focus on identification is due to the **original selection bias problem**:

- To measure $TE = Y_{1i} - Y_{0i}$, we need to see the same individual with and without treatment.
- This is impossible, as each individual has one existence. We cannot observe the counterfactual⁴. We can only compute the difference in average observed outcomes:

$$\mathbb{E}[Y_i | D_i=1] - \mathbb{E}[Y_i | D_i=0] = \dots = \underbrace{\mathbb{E}[Y_{1i} - Y_{0i} | D_i=1]}_{\text{ATET}} + \underbrace{\mathbb{E}[Y_{0i} | D_i=1] - \mathbb{E}[Y_{0i} | D_i=0]}_{\text{selection bias}}$$

The “selection bias” corresponds to the difference in Y_{0i} between the treated and untreated⁵.

- If treatment is randomly assigned, then potential outcomes are independent of the treatment ($Y_{0i}, Y_{1i} \perp\!\!\!\perp D_i$), so there is no selection bias. The *independence assumption* identifies the TE.
- In observational studies, $(Y_{0i}, Y_{1i}) \not\perp\!\!\!\perp D_i$. However, if we *match* treated and control individuals to be proper counterfactuals, i.e., if the potential outcomes are *conditionally* independent of the treatment ($Y_{0i}, Y_{1i} \perp\!\!\!\perp D_i | X_i$), then we again eliminate selection bias. The difference in average observed outcomes for treated and control individuals that share the same confounding covariate X_i equals the ATET: the *conditional independence assumption*⁶ (CIA) identifies the TE.

What makes our regression estimates causal is our **identifying assumption**:

- if we have the IA $(Y_{0i}, Y_{1i}) \perp\!\!\!\perp D_i \implies$ we can compute the ATE.
- if ~~IA~~ but we have the CIA $(Y_{0i}, Y_{1i}) \perp\!\!\!\perp D_i | X_i \implies$ we can compute the ATET.
- if ~~CIA~~ but \exists a relevant instrument Z_i that is an exogenous source of variation in D_i :

$\left\{ \begin{array}{l} \text{independent (new CIA)} \\ \text{relevant} \end{array} \right.$	$(Y_{0i}, Y_{1i}) \perp\!\!\!\perp Z_i X_i$	$Z_i \not\perp\!\!\!\perp D_i X_i$	\implies we can compute a LATE.
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So we need an **identification strategy** that convinces us that an IA holds.

³A strong assumption of the potential outcomes representation is that the treatment effect on one individual is independent of the treatment received by others. This excludes feedback effects and strategic interactions among agents.

⁴This is the ‘fundamental problem of causal inference’. Its implication: we *never* observe causal effects.

⁵For example: if individuals with low Y_{0i} choose treatment more frequently, then treated individuals would have been lower in the counterfactual than the untreated we observe: $\mathbb{E}[Y_{0i} | D_i=1] < \mathbb{E}[Y_{0i} | D_i=0]$. Comparing Y between treated and untreated underestimates the TE. Say we look at the effect of hospitalization; sick individuals go to the hospital (get treated) more often than healthy individuals. But they would also have been less healthy had they stayed at home.

⁶The CIA is also called “selection on observables”: conditioning on observed characteristics X_i erases selection bias.

Elaborate econometric techniques or regression controls won't bring causality. Reciprocally, with a good identification strategy, one need simply compare means – no need for controls.

2.2 Expressing TE as a linear regression

The relation between observed outcomes and potential outcomes (how we estimate our TE) can be written as a linear regression on the treatment:

$$\begin{aligned} Y_i &= Y_{0i} + (Y_{1i} - Y_{0i})D_i \\ &= \mathbb{E}[Y_{0i}] + (Y_{1i} - Y_{0i})D_i + (Y_{0i} - \mathbb{E}[Y_{0i}]) \\ &= \alpha + \beta_i D_i + U_i \end{aligned}$$

Consider the simple linear regression $Y_i = \alpha + \beta D_i + \varepsilon_i$. The OLS slope estimate $\widehat{\beta}_{OLS}$ is equal to $\frac{cov[Y_i, D_i]}{Var[D_i]}$, which simplifies to $\mathbb{E}[Y_i|D_i=1] - \mathbb{E}[Y_i|D_i=0]$: the difference in average observed outcomes.

2.3 Why might the IA/CIA not hold? Endogeneity

In the simple (linear & univariate) regression model $y_i = \alpha + \beta x_i + \varepsilon_i$, the variable x_i is

- **endogenous** if it is correlated with the error term: $cov[x_i, \varepsilon_i] \neq 0$.
- **exogenous** if it is uncorrelated with the error term: $cov[x_i, \varepsilon_i] = 0$.

If x is endogenous, the OLS slope estimator of β will comprise not only the partial derivative w.r.t. x (what we want) but also an indirect effect through ε : $\beta_{OLS} = \frac{dy(x, \varepsilon)}{dx} = \frac{\partial y}{\partial x} + \frac{\partial y}{\partial \varepsilon} \frac{\partial \varepsilon}{\partial x} = \beta + \frac{\partial \varepsilon}{\partial x} \neq \beta$. The OLS estimator is therefore biased and inconsistent for β .

In our case of interest, if the treatment D_i is endogenous, i.e., $cov[D_i, \varepsilon_i] \neq 0$, it means there is an imbalance in potential outcomes across the treatment groups. The CIA doesn't hold. Our estimate will be biased.

Sources of endogeneity

- reverse causality or simultaneity: If y also affects D , this is captured by the error term ε , making ε correlated with D .
- non-random (correlated to y) measurement error in D
- omitted variable bias (OVB): All omitted variables⁷ are captured by ε . Therefore, if an omitted variable w is correlated with D , ε is correlated with D . w is a “confounding variable”.

→ In observational studies,
 – excluding a confounding variable creates bias, so we must adjust for all *confounders*.
 – with all confounders assumed to be measured, we estimate an unbiased causal effect.
 – because we can rarely be certain that we have measured all confounders⁸, we turn to alternative causal inference or “**identification**” strategies, that rely on other assumptions.

⁷An omitted variable is an explanatory variable not included in the regression but which is a determinant of y .

⁸For instance, in cross-sectional approaches, we worry about time-invariant omitted variables. As a cross-section offers only inter-individual (across) variation, if y is affected by unobservable variables that systematically vary across groups, our estimate will be biased. With panel data, we have across variation and intra-individual (within) variation. Using fixed effects, we can focus on within variation only, which greatly reduces the threat of OVB.

3 Applied Identification Methods

3.1 Hierarchy of common identification methods

A contestable hierarchy of the most common identification methods in the ‘randomista’ toolkit⁹, based on their capacity to mimic random assignment, is as follows:

0. Randomized experiment (RCT)
1. Instrumental Variables (IV) and regression discontinuity (RD)
If we fear that there is selection into treatment based on unobservables, we use an instrument that induces quasi-experimental variation in treatment status.
2. Difference-in-differences (DiD) and event-studies
If we have repeated observations data and want to estimate the effect of an event, we use research designs that rely on the assumptions of parallel trends and time-invariant differences.
3. Matching estimators
Strategies based solely on matching are considered much less credible – in terms of making us believe in the CIA, and thus their ability to recover a causal effect – than strategies based on some exogenous variation. However, matching is a type of procedure that can complement a natural experiment design. It is addressed in section 4.

The sections below present, for each method, in the canonical setup, 1. the (assumed) data generating process (DGP), 2. the identifying assumptions, 3. the estimand of interest, 4. the estimator used, and 5. some best practices, and strengths and weaknesses.

Importantly, the **relation between the actual observed outcomes Y_i (or Y_{it}) and the conceptual potential outcomes Y_{0i}, Y_{1i}** is made explicit. This relation underlies how our estimation recovers a causal treatment effect.

For simplification purposes, all methods are presented without the inclusion of exogenous controls X_i . However, the relationships hold when they are all conditional on covariates X_i .

⁹Term shamelessly copied from Gibson (2019)

3.2 IV

Context (DGP) $Y_i = \alpha + \beta D_i + u_i$, $cov[D_i, u_i] \neq 0$: D_i is endogenous
But \exists a binary instrument Z_i that constitutes a source of variation in D_i , it “assigns treatment”¹⁰:

$$\begin{array}{ccccc}
 Z & \xrightarrow{\gamma} & D & \xrightarrow{\beta} & Y \\
 & & \uparrow & \nearrow & \\
 & & u & & \\
 D_i = \delta + \gamma Z_i + v_i & & & & \\
 Y_i = \alpha + \beta D_i + u_i, & cov[D_i, u_i] \neq 0 & & &
 \end{array}$$

In terms of potential outcomes:

We define the treatment assignment $Z_i \in \{0, 1\}$ and the treatment realization $D_i \in \{0, 1\}$.

$Z_i = 0$ induces the potential treatment status D_{0i} , which will be realized as 0 if individuals comply, 1 if not. $Z_i = 1$ induces D_{1i} , realized as 1 if they comply, 0 if not. The compliance behavior defines 4 categories of participants – which the researcher *cannot* observe; they can only observe the assignment Z_i and the realization D_i .

	D_{0i}	D_{1i}
compliers	0	1
always-takers	1	1
never-takers	0	0
defiers	1	0

Identifying assumptions

- (A1) independence w.r.t. the potential outcomes, i.e., $cov[Z_i, v_i] = 0$
- (A2) exclusion restriction: $cov[Z_i, u_i] = 0$
- (A3) relevance: $cov[Z_i, D_i] \neq 0$
- (A4) monotonicity: the instrument does not discourage treatment (no defiers). This assumption is weaker (and therefore more realistic) than the assumption of homogenous effects.

Estimand We define the IV estimand: $\beta_{IV} \equiv \frac{cov[Y_i, Z_i]}{cov[D_i, Z_i]} = \dots = \frac{\mathbb{E}[Y_i|Z_i=1] - \mathbb{E}[Y_i|Z_i=0]}{\mathbb{E}[D_i|Z_i=1] - \mathbb{E}[D_i|Z_i=0]}$

Note that:

- The slope estimate $\widehat{\gamma}_{LS} = \frac{\widehat{cov}[D_i, Z_i]}{\widehat{V}[Z_i]}$ from regressing D on Z consistently estimates $\gamma = \frac{cov[D_i, Z_i]}{V[Z_i]}$
 - The slope estimate $\widehat{\gamma \cdot \beta}_{LS} = \frac{\widehat{cov}[Y_i, Z_i]}{\widehat{V}[Z_i]}$ from regressing Y on Z consistently estimates $\gamma \cdot \beta = \frac{cov[Y_i, Z_i]}{V[Z_i]}$
- \implies Their ratio $\widehat{\beta}_{IV} \equiv \frac{\widehat{\gamma \cdot \beta}_{LS}}{\widehat{\gamma}_{LS}} = \frac{\widehat{cov}[Y_i, Z_i]}{\widehat{cov}[D_i, Z_i]} = \dots = \beta + \frac{\widehat{cov}[u_i, Z_i]}{\widehat{cov}[D_i, Z_i]}$: is consistent but has a bias, which \searrow with instrument strength.

The identifying assumptions reduce¹¹ $\frac{\mathbb{E}[Y_i|Z_i=1] - \mathbb{E}[Y_i|Z_i=0]}{\mathbb{E}[D_i|Z_i=1] - \mathbb{E}[D_i|Z_i=0]}$ to $\underbrace{\mathbb{E}[Y_{1i} - Y_{0i}|D_{0i}=0, D_{1i}=1]}_{\text{LATE on the compliers}}$

¹⁰For more complicated treatment variables, we will need more complicated instruments. To identify *several* treatment variables, we will need at least as many instruments. To identify a *continuous* treatment, we can't use a binary instrument.

¹¹Indeed:

Estimator Our natural choice of estimator is the sample analog $\widehat{\beta}_{IV} = \frac{\widehat{cov}[Y_i, Z_i]}{\widehat{cov}[D_i, Z_i]}$. It turns out to be numerically equivalent to the 2SLS estimator $\widehat{\beta}_{2SLS}$ obtained through the two-step procedure¹²:

$$\begin{aligned} \text{1st stage: } D_i &= \delta + \gamma \cdot Z_i + u_i \implies \widehat{D}_i = \mathbb{E}[D_i | Z_i] \\ \text{2nd stage: } Y_i &= \alpha + \beta \cdot \widehat{D}_i + \varepsilon_i \end{aligned}$$

Best practices

- Support the relevance assumption by showing that the F-statistic of the 1st stage is large (rule of thumb: $F > 10$). The bigger F , the “stronger” the instrument. Or run a test such as the Stock and Yogo test.

- Characterize the compliers to get more out of the LATE:

Compliers are rarely representative, due to selective uptake. While we cannot identify individual compliers in the data, we can estimate the size of the complier group: $P[D_{i1} > D_{i0}] = \mathbb{E}[D_{i1} - D_{i0}] = \mathbb{E}[D_i | Z_i = 1] - \mathbb{E}[D_i | Z_i = 0]$, and characterize them in terms of their distribution of observed covariates (see Kowalski (2018), Abadie (2003)).

Different valid instruments will yield different estimates because they correspond to different estimands, as each selected a specific set of compliers. Think of the group of compliers selected, to make sure the instrument is relevant w.r.t. the policy of interest.

Strengths & weaknesses

- + compelling identification strategy
- strong assumptions
- The IV estimator will be less efficient than the OLS estimator if the instrument is weak.

$$\begin{aligned} \text{Numerator: } & \mathbb{E}[Y_i | Z_i = 1] - \mathbb{E}[Y_i | Z_i = 0] = \dots \\ &= \mathbb{E}[Y_{1i} - Y_{0i} | D_{0i}=0, D_{1i}=1]P(D_{0i}=0, D_{1i}=1) - \mathbb{E}[Y_{1i} - Y_{0i} | D_{0i}=1, D_{1i}=0]P(D_{0i}=1, D_{1i}=0) \\ &= \mathbb{E}[Y_{1i} - Y_{0i} | D_{0i}=0, D_{1i}=1]P(D_{0i}=0, D_{1i}=1) \text{ as the probability of defiers is 0} \\ \text{Denominator: } & \mathbb{E}[D_i | Z_i = 1] - \mathbb{E}[D_i | Z_i = 0] = \dots \\ &= \mathbb{E}[D_{1i} - D_{0i}] \\ &= P(D_{1i} - D_{0i} = 1) \times 1 + P(D_{1i} - D_{0i} = 0) \times 0 + P(D_{1i} - D_{0i} = -1) \times (-1) \\ &= P(D_{0i}=0, D_{1i}=1) \text{ as the probability of defiers is 0} \end{aligned}$$

¹²The point estimates are equivalent, however the SEs of the 2nd stage would not give the correct SEs, as we need to adjust for the two stages of estimation. We must account for the estimation uncertainty from the first-stage (the first-stage is based on a sample, not the population, making \widehat{D}_i a random variable, instead of the usual fixed variable). Most 2SLS packages do the adjustment automatically – otherwise one can simply bootstrap the SEs manually.

3.3 RD (sharp)

Known assignment mechanism but no overlap

Context (DGP) Treatment D_i is not randomly assigned, it is assigned deterministically but *discontinuously* along Z_i , s.t. there is “local randomization” around a cutoff c . Units to the right of c receive treatment, those to the left don’t ($D_i = \mathbb{1}\{Z_i \geq c\}$). The only confounder is the assignment variable Z_i .

$$Y_i = \alpha + \beta \cdot D_i + k(Z_i) + u_i \quad ^{13}$$

Identifying assumptions

(A1) *local* independence: $Y_{1i}, Y_{0i} \perp D_i | Z_i$ for $Z_i \in [c-a, c+a]$, i.e., no confounders vary discontinuously across the threshold. \implies The average outcome of those right below the cutoff (who are denied the treatment) are a valid counterfactual for those right above (who receive the treatment).

(A2) relevance: discontinuity in the dependence of D_i on Z_i : $D_i = \mathbb{1}\{Z_i \geq c\}$

I.e., if there appears to be no reason, other than D_i , for Y_i to be a discontinuous function of Z_i , we can reasonably attribute a discontinuous jump in Y_i at c to the causal effect of D_i .

$$\textbf{Estimand} \quad \beta_{\text{RD}} = \lim_{z \rightarrow c^+} \mathbb{E}[Y_i | Z_i = z] - \lim_{z \rightarrow c^-} \mathbb{E}[Y_i | Z_i = z] = \dots = \underbrace{\mathbb{E}[Y_{1i} - Y_{0i} | Z_i = c]}_{\text{LATE at the cutoff}}$$

Estimator We could estimate β by running two separate regressions on each side of c , after transforming Z to $Z-c$, s.t. the intercepts correspond to the values of the regression functions at c .

We can estimate β (and its SE) *directly* by running a pooled regression – including interaction terms between D and Z to let the regression function differ on both sides of the cutoff:

$$\left. \begin{array}{l} Y_i = \alpha_l + k_l(Z_i - c) + v_i \\ Y_i = \alpha_r + k_r(Z_i - c) + w_i \end{array} \right\} \longrightarrow \begin{array}{lcl} Y_i & = & \alpha_l + (\alpha_r - \alpha_l) \mathbb{1}\{\text{right}\} + k(Z_i - c) + \varepsilon_i \\ & = & \alpha_l + \beta D_i + k(Z_i - c) + \varepsilon_i \end{array}$$

where $k() = k_l() + [k_r() - k_l()]D_i$

Best practices

- Choice of $k()$ and non-parametric estimation:
 - Option #1: local linear regression, with choice of the bandwidth h : $Y_i = \alpha_l + \beta D_i + \gamma_l(Z - c) + (\gamma_r - \gamma_l)(Z - c)D + \varepsilon_i$ with $c - h \leq X \leq c + h$. A larger h increases precision but also bias¹⁴.
 - Option #2: local polynomial regression model with choice of the polynomial’s order.
- In both cases, report the results of several specifications to assess the sensitivity to $k()$.

¹³The function $k()$, and therefore the true functional form of the DGP, is unknown. This is a problem, as misspecification of the functional form typically biases the estimate. Estimation in RDDs is therefore generally done non-parametrically (using polynomial regression models, or local linear regression models).

¹⁴Choose the optimal h by estimating the model’s predictive accuracy for different values of h , for example using leave-one-out cross-validation: iteratively for each observation i , the model is fit using only the observations $Z_i - h \leq \mathbf{Z} < Z_i < c$ when $Z_i < c$, and only the observations $c < Z_i < \mathbf{Z} \leq Z_i + h$ when $Z_i \geq c$.

Strengths & weaknesses

- + RDDs are similar to a local randomized experiment, and thereby require weak assumptions.
- + They have much potential in economic applications, as geographic boundaries or administrative or organizational rules (e.g., program eligibility thresholds) often create usable discontinuities.
- They risk being underpowered.
- The parameter estimates are “very local”, it may be hard to generalize from such a local result.

3.4 DiD

Context (DGP) We want to estimate the causal effect of *an event*, which occurs at time t_0 and affects a group of individuals in the population, on some outcome Y .

Treatment assignment is a function of 2 dimensions: group (treatment/control) and period (pre/post event). We define the binary variables $G_i \equiv \mathbb{1}\{i \in \text{treatment group}\}$ and $P_t \equiv \mathbb{1}\{t \in \text{post period}\}$.

Identifying assumptions

(A1) Same counterfactual trends across groups, i.e., comparable outcomes in the absence of treatment:

$$\mathbb{E}[Y_{0i}|G_i=1, P_t=1] - \mathbb{E}[Y_{0i}|G_i=1, P_t=0] = \mathbb{E}[Y_{0i}|G_i=0, P_t=1] - \mathbb{E}[Y_{0i}|G_i=0, P_t=0]$$

(A2) The sample composition does not vary over time.

Estimand

$$\begin{aligned} \beta_{\text{DiD}} &\equiv (\bar{Y}_{G_1 P_1} - \bar{Y}_{G_1 P_0}) - (\bar{Y}_{G_0 P_1} - \bar{Y}_{G_0 P_0}) \\ &\equiv \left(\mathbb{E}[Y_{it} | G_i=1, P_t=1] - \mathbb{E}[Y_{it} | G_i=1, P_t=0] \right) - \left(\mathbb{E}[Y_{it} | G_i=0, P_t=1] - \mathbb{E}[Y_{it} | G_i=0, P_t=0] \right) \\ &= \left(\mathbb{E}[Y_{1i} | G_i=1, P_t=1] - \mathbb{E}[Y_{1i} | G_i=1, P_t=0] \right) - \left(\mathbb{E}[Y_{0i} | G_i=0, P_t=1] - \mathbb{E}[Y_{0i} | G_i=0, P_t=0] \right) \\ &= \dots \\ &= \underbrace{\mathbb{E}[Y_{1i} - Y_{0i} | G_i=1, P_t=1]}_{\text{ATET}} \end{aligned}$$

Estimator The OLS estimator $\hat{\beta}_{\text{OLS}}$ of the following regression consistently estimates β_{DiD} :

$$\begin{aligned} Y_{it} &= \alpha + \beta_G G_i + \beta_P P_t + \beta G_i P_t + \varepsilon_{it} \\ &= \alpha + \lambda_G + \lambda_P + \beta G_i P_t + \varepsilon_{it} \end{aligned}$$

If we have panel data (instead of merely repeated cross-sections), we can estimate this in a more direct way. As $\beta = \mathbb{E}[Y_{1i} - Y_{0i} | G_i = 1] - \mathbb{E}[Y_{1i} - Y_{0i} | G_i = 0]$, we can simply regress the change for each unit $Y_{1i} - Y_{0i}$ on G_i : a first difference approach.

Best practices

- Support the assumption of parallel counterfactual trends by showing that pre-treatment trends coincide (if we have data for multiple pre-periods). Estimate the following regression model by OLS, and check that the coefficients β_τ where $\tau < t_0 - 1$ are 0:

$$y_{it} = \sum_{\tau \neq t_0 - 1} \beta_\tau G_i \mathbb{1}\{t = \tau\} + \lambda_i + \lambda_t + \varepsilon_{it}$$

- The regression above also enables us to look at whether the effect of treatment actually *accumulates* over time: $\beta_{\tau, \tau \geq t_0} \uparrow$ in τ .
- If the composition of the groups changes over time, interact covariates with P_t .

Strengths & weaknesses

- + DiD is a way of ruling out unobserved time-invariant confounders, by using repeated observations, thus creating comparable groups
- + Pre-trends aren't a problem (unlike in event-studies) as long as that of the two groups are *parallel*.
- + Identification only requires repeated observations, so repeated cross-sectional data suffice, as long as the sample composition does not vary over time. Panel data satisfy this condition by construction.

3.5 DiDiD

Context (DGP) The treatment varies along a 3rd dimension or “subgroup” (in addition to time and place), such as gender, season... We define the binary variable $S_i \equiv \mathbb{1}\{i \in \text{treatment subgroup}\}$.

Identifying assumptions

(A1) Same counterfactual trends across ~~groups~~ subgroups:

$$\begin{aligned} & (\mathbb{E}[Y_{0i}|G_1, S_1, P_1] - \mathbb{E}[Y_{0i}|G_1, S_1, P_0]) - (\mathbb{E}[Y_{0i}|G_1, S_0, P_1] - \mathbb{E}[Y_{0i}|G_1, S_0, P_0]) \\ &= (\mathbb{E}[Y_{0i}|G_1, S_1, P_1] - \mathbb{E}[Y_{0i}|G_1, S_1, P_0]) - (\mathbb{E}[Y_{0i}|G_1, S_0, P_1] - \mathbb{E}[Y_{0i}|G_1, S_0, P_0]) \end{aligned}$$

(A2) The sample composition does not vary over time.

Estimand

$$\begin{aligned} \beta_{\text{DiDiD}} &\equiv \left[(\bar{Y}_{G_1 S_1 P_1} - \bar{Y}_{G_1 S_1 P_0}) - (\bar{Y}_{G_0 S_1 P_1} - \bar{Y}_{G_0 S_1 P_0}) \right] - \left[(\bar{Y}_{G_1 S_0 P_1} - \bar{Y}_{G_1 S_0 P_0}) - (\bar{Y}_{G_0 S_0 P_1} - \bar{Y}_{G_0 S_0 P_0}) \right] \\ &= \dots \\ &= \underbrace{\mathbb{E}[Y_{1i} - Y_{0i}|G_i=1, S_i=1, P_t=1]}_{\text{ATET}} \end{aligned}$$

Estimator The OLS estimator $\widehat{\beta}_{\text{OLS}}$ of the following regression consistently estimates β_{DiDiD} :

$$\begin{aligned} Y_{it} &= \alpha + \beta_G G_i + \beta_S S_i + \beta_P P_t + \beta_{GS} G_i S_i + \beta_{GP} G_i P_t + \beta_{PS} P_t S_i + \beta G_i S_i P_t + \varepsilon_{it} \\ &= \beta G_i S_i P_t + \lambda_{GS} + \lambda_{GP} + \lambda_{PS} + \varepsilon_{it} \end{aligned}$$

Best practices

- A triple differences makes for a very specific control group. Before doing an DiDiD, one must be able to answer why a double differences wasn't satisfactory (why the control group in double differences isn't good enough), and even the first differences.

Strengths & weaknesses

- + A triple difference allows to difference out more confounding elements, it therefore gets harder to find a confounder.
- It requires more data, more variation.

3.6 Event study

Context (DGP) We want to estimate the causal effect of *an event*¹⁵, which occurs at different times t_{0i} for each unit i (“staggered adoption”) and affects *all units* in the population, on some outcome Y . Treatment assignment is a function of the period (pre/post event). We define the binary variable $P_t \equiv \mathbb{1}\{t \in \text{post period}\}$.

Identifying assumptions

- (A1) exogeneity: the event is unpredictable, and not a result of the outcome Y .
Random timing of the event implies that there cannot be any pre-trends, s.t. we can reasonably use a unit’s past value to construct its counterfactual post-event value.

Estimand $\beta_{\text{ES}} \equiv \mathbb{E}[Y_{it}|t=t_{0i}] - \mathbb{E}[Y_{it}|t=t_{0i}-1] = \dots = \underbrace{\mathbb{E}[Y_{1i} - Y_{0i}|P_t=1]}_{\text{ATET}}$

Estimator The OLS estimator $\hat{\beta}_{\text{OLS}}$ of the following regression consistently estimates β_{ES} :

$$Y_{it} = \sum_{\tau=-K}^{t_0-1} [\beta_{\tau} \mathbb{1}\{t = \tau\}] + \beta \mathbb{1}\{t = t_0\} + \sum_{\tau=t_0+1}^L [\beta_{\tau} \mathbb{1}\{t = \tau\}] + \lambda_i + \varepsilon_{it}$$

Best practices

- Report all β_{τ} s, to check that they are not increasing up to the event. An increase would suggest the presence of pre-trends... which are a sign of endogeneity of the treatment variable, making it hard to interpret the event (unless there is a trend discontinuity). Provide a plot of pre-trends.

Strengths & weaknesses

- It is difficult to rule out other things changing at the same time, i.e., unobserved confounders.

¹⁵Like in DiD, we are estimating the causal effect of an event, thanks to observing units repeatedly over time. We need a model to estimate the counterfactual value (if the event had not occurred), s.t. the difference from the counterfactual is the causal effect. DiD and event studies are simply different models of the counterfactual. We use DiD when there are control units, that we can use to remove trends in the outcome of interest.

Summary of common identification methods

	Source of identification & identifying assumptions	Estimand β & corresponding TE	Chosen estimator $\widehat{\beta}$	Strengths / Weaknesses
RCT	(A) independence	$\beta_{\text{RCT}} \equiv \mathbb{E}[Y_i D_i=1] - \mathbb{E}[Y_i D_i=0] = \underbrace{\mathbb{E}[Y_{1i} - Y_{0i}]}_{\text{ATE}}$	$\widehat{\beta}_{\text{OLS}}$ of the regression $Y_{it} = \alpha + \beta D_i + \varepsilon_{it}$. Is consistent and unbiased .	+ Random assignment structurally guarantees (A) \implies RCT = “gold standard”
IV	Id. from the exogenous variation in D induced by Z . (A1) independence (A2) exclusion restriction (A3) relevance (A4) monotonicity	$\beta_{\text{IV}} \equiv \frac{\text{cov}[Y_i, Z_i]}{\text{cov}[D_i, Z_i]} = \dots$ $= \frac{\mathbb{E}[Y_i Z_i=1] - \mathbb{E}[Y_i Z_i=0]}{\mathbb{E}[D_i Z_i=1] - \mathbb{E}[D_i Z_i=0]} : \text{“Wald estimand”}$ $= \dots$ $= \underbrace{\mathbb{E}[Y_{1i} - Y_{0i} D_{1i}=1, D_{0i}=0]}_{\text{LATE, compliers}}$	$\widehat{\beta}_{\text{IV}} \equiv \frac{\widehat{\text{cov}}[Y_i, Z_i]}{\widehat{\text{cov}}[D_i, Z_i]} = \dots = \text{numerically equivalent to } \widehat{\beta}_{\text{2SLS}}$ Is: consistent, biased , but bias \downarrow with strength of Z_i .	+ compelling identification strategy – strong assumptions – less efficient than $\widehat{\beta}_{\text{OLS}}$ if instrument is weak
sharp RD	Id. from a discontinuous treatment assignment based on a cutoff in Z . (A1) independence (A2) relevance	$\beta_{\text{RD}} \equiv \lim_{z \rightarrow c^+} \mathbb{E}[Y_i Z_i = z] - \lim_{z \rightarrow c^-} \mathbb{E}[Y_i Z_i = z]$ $= \dots$ $= \underbrace{\mathbb{E}[Y_{1i} - Y_{0i} Z_i = c]}_{\text{LATE, at the cutoff}}$	Estimate non-parametric regression functions: $Y_i = \alpha_l + \beta D_i + k(Z_i - c) + \varepsilon_i$ – local linear regression, bandwidth h – polynomial regression Is: consistent, biased , bias \uparrow with h .	+ akin to a local randomized experiment + weak & testable assumption – risk being underpowered – “very local” estimates, hard to generalize
DiD	(A1) same counterfactual trends across groups (A2) same group composition over time	$\beta_{\text{DiD}} \equiv \left(\mathbb{E}[Y_{it} G_i=1, P_t=1] - \mathbb{E}[Y_{it} G_i=1, P_t=0] \right) - \left(\mathbb{E}[Y_{it} G_i=0, P_t=1] - \mathbb{E}[Y_{it} G_i=0, P_t=0] \right)$ $= \dots$ $= \underbrace{\mathbb{E}[Y_{1i} - Y_{0i} G_i=1, P_t=1]}_{\text{ATET}}$	$\widehat{\beta}_{\text{OLS}}$ of the regression $Y_{it} = \alpha + \beta G_i P_t + \lambda_G + \lambda_P + \varepsilon_{it}$ Is consistent .	+ rules out unobserved time-invariant confounders
DiDiD	(A1) same counterfactual trends across subgroups (A2) same subgroup composition over time	$\beta_{\text{DiDiD}} \equiv \left[\left(\bar{Y}_{G_1 S_1 P_1} - \bar{Y}_{G_1 S_1 P_0} \right) - \left(\bar{Y}_{G_0 S_1 P_1} - \bar{Y}_{G_0 S_1 P_0} \right) \right] - \left[\left(\bar{Y}_{G_1 S_0 P_1} - \bar{Y}_{G_1 S_0 P_0} \right) - \left(\bar{Y}_{G_0 S_0 P_1} - \bar{Y}_{G_0 S_0 P_0} \right) \right]$ $= \dots$ $= \underbrace{\mathbb{E}[Y_{1i} - Y_{0i} G_i=1, S_i=1, P_t=1]}_{\text{ATET}}$	$\widehat{\beta}_{\text{OLS}}$ of the regression $Y_{it} = \alpha + \beta G_i S_i P_t + \lambda_{GS} + \lambda_{GP} + \lambda_{PS} + \varepsilon_{it}$ Is consistent .	+ differences out more confounding elements than in DiD, so harder to find a confounder – requires more data & variation
Event-study	(A) random timing of the event	$\beta_{\text{ES}} \equiv \mathbb{E}[Y_{it} t=t_0] - \mathbb{E}[Y_{it} t=t_0-1]$ $= \dots$ $= \underbrace{\mathbb{E}[Y_{1i} - Y_{0i} P_t=1]}_{\text{ATET}}$	$\widehat{\beta}_{\text{OLS}}$ of the regression $Y_{it} = \beta \mathbb{1}\{t = t_{0i}\} + \sum_{\tau \neq \{t_{0i}-1, t_{0i}\}} [\beta_\tau \mathbb{1}\{t = \tau\}] + \lambda_i + \varepsilon_{it}$ Is consistent .	– difficult to rule out unobserved confounders

4 Improving our causal inference

4.1 *Pre-estimation = improving design* – Matching

Matching refers to procedures that restructure the original sample prior to statistical analysis. The goal of this restructuring in a causal inference setting is to create a sample analogous to one created from a randomized experiment, i.e., we want the matched groups to be balanced and overlap¹⁶ w.r.t. the pre-treatment variables that we consider to be confounders X . This may involve 1:1 matching, weighting, or subclassification.

What does matching provide? Recall that if the independence assumption is satisfied conditional on the *confounding*¹⁷ covariates: $(Y_0, Y_1) \perp D|X$, and if we can achieve balance and overlap w.r.t. them, then the observed difference in means across treatment and control groups $\mathbb{E}[Y_1|D=1] - \mathbb{E}[Y_1|D=0]$ will be an *unbiased* estimate of the true treatment effect (TE).

Matching should not be thought of as an alternative to a *research design*-based method¹⁸, but as a first step in the causal analysis that can reduce the reliance on parametric assumptions of the model fitted to estimate the TE. Just as with a randomized experiment, the intuition is that if the treatment and control groups have sufficient overlap and balance, then, even if we misspecify the model, we should still get a reasonable estimate of the treatment effect (Gelman et al., 2020).

For matching to have the ability to capture by itself a *causal* effect, the assumption of selection on observables would have to be satisfied, i.e., that all the difference between the groups is captured by X . This is a very (too) strong assumption, that is not testable.

Therefore a *matching estimator* by itself, i.e., an identification strategy based solely on matching, is considered much less credible than one based on some exogenous variation. We need exogenous variation to believe the CIA. Matching does not bring causality; nor do regression controls. Both are only adjustment strategies.

Common matching methods

- Propensity score matching (PSM)
Units are matched based on their “propensity score”: their predicted probability of getting treated. A logistic regression of D_i on X_i produces predicted probabilities of getting treated \hat{p}_i , which are then used to match each treated unit to non-treated units.
 \triangle PSM ensures $\hat{p}(X^t) = \hat{p}(X^c)$, but not $X^t = X^c$.
Methods that stratify the data should be preferred.
- Mahalanobis Distance Matching
(Approximates Fully Blocked Experiment)
Matching is based on some distance metric, which can incorporate multiple dimensions of “closeness” between observations (whereas PSM necessarily reduces the dimensionality to 1):

¹⁶The treatment and control groups can be different in two ways:

- Imbalance: the distributions of the confounders differ across the groups. \rightarrow The simple difference of group averages is not a good estimate of the ATE. We must adjust for pre-treatment differences, e.g., by matching or weighting.
- Incomplete overlap: the support (range of x) differs across the two groups. We have no empirical counterfactuals for some treatment/control observations. The model will create counterfactual predictions by extrapolating over portions of the space where there is no data to support them.

¹⁷The pre-treatment variables that predict (= are associated with) both treatment assignment and the outcome.

¹⁸Methods in which a feature in the setting approximates a randomized experiment, and we fit a model that adjusts for potential confounders: RDs, IVs... (the methods described in the previous section).

$$\text{Distance}(X^c, X^t) = \sqrt{(X^c - X^t)' S^{-1} (X^c - X^t)}$$

Each treated unit is matched to its nearest control unit. Control units are not reused, and those unused are dropped.

Mahalanobis gets as close as possible to: $X^c = X^t \implies \hat{p}(X^T) = \hat{p}(X^C)$

4.2 Estimation – Good/bad regression controls

Separately from the identification strategy, controlling for balance covariates that are unrelated to D_i (and influence y) can increase the efficiency of the estimate (reduce the residual variation); whereas covariates that are correlated with D_i will introduce bias.

In addition to adding the X as predictors, Gelman et al. (2020) recommends considering adding interactions of the treatment D_i with those X that have large estimated effects, to look at how the treatment effect varies with the level of X .

Finally, if we center X , the treatment coefficient represents the treatment effect for individuals with the mean X score for the sample.

4.3 Post-estimation = checking assumptions – Falsification Tests

In empirical studies concerned with causal inference, one can never directly *test* the **identification assumptions**, but one can do falsification analyzes to *support* their validity – and thus the **internal validity** of the study.

They do not *prove* that the assumptions hold, instead their results either reject the claim, or increase confidence in it.

They are done almost automatically in RCTs – though rarely identified as such. They are virtually as easy to do in observational studies; the general approach is to estimate an alternative specification, which, if the identifying assumption holds, should not find an effect. Finding an effect $\neq 0$ will suggest the identifying assumption is violated.

Approach

- **RCT** The identifying assumption is random assignment (of each individual into the treatment vs control group). If that's true, then the sample means of explanatory variables should be the same across groups. A “balance test” table of sample means of the X s by group is therefore a falsification test of an RCT's central assumption¹⁹²⁰.
- **IV** The two main identifying assumptions can be tested:
 - relevance (Z_i is strongly related to sorting into treatment D_i): directly observable in the 1st stage;

¹⁹ Δ One should show balance tables (not t-tests!) of baseline observables. T-tests in this context are conceptually unsound: they amount to assessing the probability of an event (a difference in averages) having occurred by chance, when we already know that it could only occur by chance, as the allocation between treatment arms was carried out randomly. Hayes and Moulton (2017) explain that “the point of displaying between-arm comparisons is not to carry out a significance test, but to describe in quantitative terms how large any differences were, so that the investigator and reader can consider how much effect this may have had on the trial findings.” T-tests are only sound in the sense of wanting to test empirically whether the randomization was carried out correctly.

²⁰ Doug Almond's advice: even in observational settings, *always* show a balance test table. Andrew Gelman's advice: to show (im)balance in averages of X across groups, plot the standardized differences in mean values for the X s.

- exclusion restriction (Z_i isn't correlated with Y_i through some pathway other than D_i). The ideal falsification test is to estimate the reduced form effect of Z_i on Y_i in a situation where Z_i can't affect D_i . Finding an effect means Z_i affects Y_i through an other channel than D_i , falsifying the exclusion restriction. One can use an alternative population or an alternative outcome, that can't be affected by the treatment but would be by potential confounders (unobserved characteristics correlated with Z_i and Y_i).
- **RD** The two main identifying assumptions can be tested:
 - continuity or “local randomization” (all other factors determining Y_i evolve “smoothly” w.r.t. Z_i). Test: do other covariates jump at the cutoff c ? Estimate the same model, but using covariates instead of Y , and plot the observations and the fitted curves. If none do, we can assume the unobservables don't either.
 - relevance (discontinuity in the dependence of D_i on Z_i : $D_i = \mathbb{1}\{Z_i \geq c\}$). Test: do jumps occur at placebo cutoffs \tilde{c} ?
- **DiD** The two main identifying assumptions can be tested:
 - same counterfactual trends across groups. Tests: compare trends in the pre-period; use an alternative outcome that shouldn't be affected by the treatment; use an alternative control group (the estimated effect should be the same); move the event to points earlier in time (falsely assume that the onset of treatment occurs before it actually does), if the estimated treatment effect is no longer be statistically significant (i.e., is statistically indistinguishable from 0.), suggests that the observed change is more likely due to the treatment (event) than to some alternative force.
 - same group composition over time. Panel data satisfies this assumption by definition, but if we have instead repeated cross-sectional data, we can estimate covariate balance regressions.

Examples

DiD Linden and Rockoff (2008): *What is the hedonic price function for the local disamenity of crime risk (i.e., individuals' valuation of crime risk)?* Y_i = property value, D_i = a registered sex offender moves in nearby.

Falsification test of the “same counterfactual trends” assumption (if the prices of houses in offender areas are trending over time differently than the other houses in their neighborhood, they would estimate a spurious negative “impact” of the offender's arrival): the authors estimate the DiD model using false arrival dates (2-3 years prior to an offender's actual arrival), and find no effect.

Note: falsification tests are different from robustness checks, which consist in estimating alternative specifications that test the same hypothesis.

4.4 Which uncertainty matters? – Assignment inference (AI)

“In randomization-based inference, uncertainty arises naturally from the random assignment of the treatments, rather than from the hypothesized sampling from a large population.” (Athey and Imbens, 2017)

The inference techniques we commonly use in regression analysis correspond to “sampling-based” inference: they are based on variation in sampling. The uncertainty about population parameters is induced by random sampling from the population. They ask: *What would have occurred under a different random sample than the one sampled? Would the results hold?*

In causal inference studies, there is also another type of variation at play: variation in *assignment of treatment*. There is “design-based” uncertainty induced from not knowing what the regression outcome would

have been under alternative randomizations of treatment assignment. In “Randomization Inference”, introduced by Fisher (1935), the basis for inference is the distribution induced by the randomization of the treatment allocation. One takes “*a design-based perspective where the stochastic nature and properties of the estimators arises from the stochastic nature of the assignment of the treatments, rather than a sampling-based perspective where the uncertainty arises from the random sampling of units from a large population*” (Athey and Imbens, 2018). One asks: *What would have occurred under a different random assignment of treatment among units than the assignment observed? Would the results hold?*

The term “randomization inference” is somewhat confusing. Both approaches assume – and build inference from – the randomization of something: in the traditional approach, of the *sample*; in the treatment approach, of the *treatment assignment*. The rest of this section refers therefore to “assignment inference” (AI), which refers directly to the foundation of this alternative approach.

Why choose assignment-based inference instead of sampling-based inference?

- In some cases, there is no true sampling variation, making sampling-based p-values meaningless. Suppose we observed the universe of y outcomes; then there is no sampling from a large population. *Is what matters that we observe the entire population, or that we observe the entire possible range of values?*
- AI allows us to make inferences about causal effects without having to appeal to large samples, i.e., without relying on asymptotics, as it is based on the act of physical randomization alone.
- It is useful with some particular clustered data structures. Ex: rainfall – and other weather variables – are often used as a strategy for causal inference, as rainfall shocks are as-if randomly assigned. However, the assignment of rainfall is highly correlated across space, but the assignment clustering isn’t within well-defined boundaries, s.t. we can’t rely on common methods: we can’t cluster; sandwich-type spatially-robust covariance matrix estimators require modeling assumptions to specify the matrix structure... How to estimate SEs? Cooperman (2017) applies randomization inference with historical weather patterns as potential randomizations. This technique allows to remain agnostic about complex clustering across units by using national draws, that will preserve patterns of spatial dependence²¹.

In practice: simulation We repeat a large number of times (e.g., 1000) the following procedure²²:

1. Randomly re-assign treatment (keeping the structure of the original randomization, e.g., within strata), i.e., simulate the assignment mechanism, thus generating fake treatment statuses;
2. Estimate the regression model using these fake treatments, and store the $\hat{\beta}$ s.

We obtain a distribution for the $\hat{\beta}$ s.

Hypothesis testing and p-value Recall the approach to hypothesis testing: we formulate a null hypothesis that represents a fact about the data we’ll try to refute. In causal inference, it will generally be a hypothesis of no effect. And we derive a test statistic T s.t. when H_0 is true, T has a specific distribution. We then see where the value of T *for our observed data* lies within that distribution; the furthest in the tails, the less likely these observed data were under the null hypothesis, therefore the higher the confidence against it.

²¹Note that the use of historical data is disputable if climate change changes the distribution across years.

²²RI is a simulation approach, like Bootstrap. However RI is based on variation from treatment assignment, whereas Bootstrap still considers variation from sampling. A Bootstrap procedure resamples observations from our actual sample (which is fair, as we assumed it was representative of the population), with replacement, to simulate how *sampling* variation would affect our results.

Both “sampling-based” and “design-based” inference proceed along these steps. For a given H_0 and a given test statistic T , noting \hat{T}_{obs} the *observed* value of T :

Sampling-based inference	Assignment inference
H_0	
No average effect: $\mathbb{E}[Y_{1i}] - \mathbb{E}[Y_{0i}] = 0$	“Sharp” no effect: $Y_{1i} - Y_{0i} = 0, \forall i$
Distribution of \hat{T} under H_0	
Under H_0 , the distribution of \hat{T} across all random samples converges (as $n \rightarrow \infty$) to a known distribution: usually t or normal. → We compute the parameters of this distribution. → The asymptotic distribution of \hat{T} (across random samples) = the “sampling distribution”.	Under H_0 , however the treatment was assigned wouldn’t change the observed outcomes; but it would change the value of \hat{T} . → We compute \hat{T} for many simulated assignments. → The distribution of \hat{T} (across treatment assignments) = the “randomization distribution”.
$2\text{-sided } p\text{-value} = \Pr [\text{observing a } \hat{T} > \hat{T}_{\text{obs}}] \text{ under } H_0$ $= \text{share of the distribution that is } > \hat{T}_{\text{obs}}$	
$= \Pr[\text{the observed difference between groups would have been observed}]$ if they had been drawn from underlying sampling frames with no mean difference.	$= \Pr[\text{the observed difference between groups would have been observed}]$ if they had received a different treatment assignment.
\implies Given e.g. a rejection threshold $\alpha = 0.05$, the test will erroneously reject $H_0 < 5\%$ of the time	

5 Presentation

5.1 Characterizing the empirical strategy

The empirical strategy for any econometric analysis aiming for causal inference should contain – to some degree, explicitly – the following items:

1. Research question – *What causal effect of interest are we trying to estimate?*
2. Ideal experiment – *What ideal experiment would capture the causal effect?*
3. Identification strategy – *How are the observational data at hand used to make comparisons that approximate such an experiment? Specifying notably: the identifying assumptions, what makes them satisfied, the specific effect estimated (ATET, LATE...).*
 - Show a balance test table (table of sample means of the covariates X by treatment and control group), to check that the X are balanced across the treatment and control group, i.e. that X uncorrelated with treatment
4. Estimation method
5. Falsification tests that bring confidence in the identifying assumptions.

All these items can be characterized before opening the dataset.

5.2 Putting the paper in perspective

In addition to the paper's empirical strategy, one may want to discuss:

- Contributions to the literature on the topic or research question
- Methodological contributions
- Internal validity of the statistical analysis
 - Are the identifying assumptions plausible (are there stories under which the assumptions would not hold?) Could there be measurement error? Are there unexplained results?*
- External validity of the statistical analysis
 - w.r.t. policy: is there a gap between policy questions and the analyses performed?
 - w.r.t. the literature: how does the paper account for its results compared to other results in the literature?
 - w.r.t. other settings: are the results generalizable to other populations and settings?

Validity of a statistical analysis

- **Internal validity** = the extent to which the causal effect *in the population being studied* is properly identified. It is determined by how well the study can rule out alternative explanations for its findings.
- **External validity** = the extent to which the study's inferences can be generalized to other populations and settings.
 - ⚠ Even in randomized trials, the experimental sample often differs from the population of interest. If participation decisions are explained by observed variables, such differences can be overcome by reweighting. But participation may depend on unobserved variables...

6 Other branches of causal modelling

6.1 Structural Equation Models (SEMs)

Structural Equation Models are probabilistic models that unite multiple predictor and response variables in a single causal network.

SEMs²³ are often represented using path diagrams, a.k.a. directed acyclic graphs (DAG), where arrows indicate directional relationships between observed variables.

Implicit assumptions – what separate SEMs from traditional modelling approaches:

1. Implicitly assumes that the relationships among variables (paths) are causal.
A big leap from the traditional statistics’ “correlation does not imply causation”. By using pre-existing knowledge of the system, one can make an informed hypothesis about the causal structure of the variables, and SEM explicitly tests this supposed causal structure²⁴.
2. Variables can be both predictors and responses. → SEM is useful for testing and quantifying indirect (cascading) effects – that would otherwise go unrecognized by any single model.

Traditional SEM

Estimation: Coeffs are estimated simultaneously in a single variance-covariance matrix of all variables; typically by MLE.

Goodness-of-fit: = discrepancy between the observed and predicted covariance matrices. χ^2 test: the χ^2 statistic describes the agreement between the 2 matrices.

Assumptions

- Independent errors (data has no underlying structure)
- Normal errors (model fit to normal distrib)

Limits

- *Assumptions often violated in ecological research: e not independent (spatial or temporal correlation in observational studies), distribution not normal (count data \sim Poisson)...*
- computationally intensive (depending on the sizes of the v-cov matrix)
- if variables are nested, then the sample size is limited to the use of variables at the highest level of the hierarchy. Can shrink our sample and reduce the power of the analysis. . .

Piecewise SEM

Estimation: Decompose the network and estimate each relationship separately. Piece the m paths together after for inferences about the entire SEM. (we estimate m separate vcov matrices.)

⇒ Much easier to estimate than a single vcov matrix → can estimate large networks

⇒ Flexible: can incorporate many model structures, distributions... using extensions of linear reg (random effects, hierarchical models, non-normal responses, spatial correlation...)

Goodness-of-fit: No formal χ^2 test. Instead: “tests of directed separation”: are any paths missing from the model?

The ‘basis set’ = all k pair relationships unspecified in the model (i.e., independence claims). Test whether are indeed not significant (controlling for variables on which these paths are conditional), keep the p-value. From the k p-values, calculate Fisher’s C statistic $C = -2 \sum_{i=1}^k \ln(p_i) \sim \chi^2(2k)$. If C’s p-value > 0.05, accept the model. This approach is vulnerable to model misspecification.

Rmk: we can compute an AIC score for the SEM, for model comparisons: $AIC = C + 2k \frac{n}{n-k-1}$

²³SEMs are a fast growing statistical technique in ecological research. A new way to study ecological systems.

²⁴Causality is central: SEM is designed to test competing hypotheses about complex relationships. On causality in SEMs: Judea Pearl (2012) and Kenneth Bollen & Judea Pearl (2013).

6.2 Graphical causal modeling

Keep reading: <https://mixtape.scunning.com/dag.html#a-simple-dag>

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