

Causal Inference

From policy questions to credible designs

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The course is organized around identification

PART I

Causal inference

- Potential outcomes and policy estimands
- Selection on observables and DML
- Experiments, IV, DiD, and RD

PART II

Supporting models

- Panel data, clustering, and GMM
- Limited outcomes and selection
- Duration, count, and choice models

The organizing question is always: which variation identifies which causal parameter?

A disciplined causal workflow



If one link is vague, the causal claim is vague.

01 Causal questions and potential outcomes

- Separate prediction from intervention
- Define the missing counterfactual
- Name the population whose effect matters

Prediction is not causality

A regression coefficient acquires causal meaning only through a model and an identification argument.

Prediction

- **Object:** $E[Y|X]$ or its best linear approximation
- **Variation:** all observed variation in X
- **Question:** what outcome is expected at $X=x$?

Causal effect

- **Object:** a contrast of potential outcomes
- **Variation:** a defended exogenous component of treatment
- **Question:** what changes under intervention?

Potential outcomes make the missing counterfactual explicit

Each unit has two conceptually well-defined outcomes; only one is observed.

$$Y_i = T_i Y_{1i} + (1 - T_i) Y_{0i}$$

- **Observed:** Y_{1i} for treated units and Y_{0i} for untreated units.
- **Missing:** the untreated outcome for the treated—and vice versa.
- **Identification:** construct a credible substitute for that missing outcome.

ATE, ATT, and ATU answer different policy questions

Estimand	Definition	Policy question
ATE	$E[Y_1 - Y_0]$	What if everyone in the population were treated?
ATT	$E[Y_1 - Y_0 \mid T=1]$	What did treatment do for participants?
ATU	$E[Y_1 - Y_0 \mid T=0]$	What would treatment do for nonparticipants?

The population index is part of the estimand—not a reporting detail.

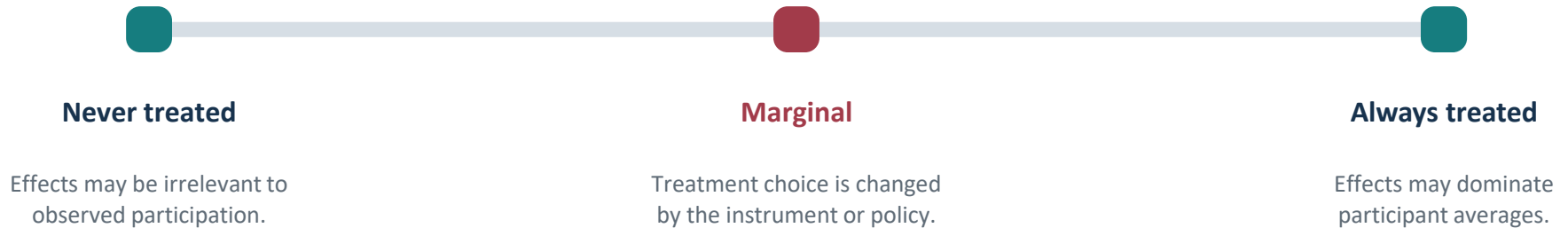
The raw treated–control gap mixes effect and selection

$$E[Y|T=1] - E[Y|T=0] = ATT + \{E[Y_0|T=1] - E[Y_0|T=0]\}$$

- **ATT:** the causal effect for those who selected into treatment.
- **Selection bias:** how treated and controls would differ even without treatment.
- **Design goal:** make the bracketed counterfactual difference zero or estimable.

Heterogeneous effects change what every estimator means

When $\beta_i = Y_{1i} - Y_{0i}$ varies, selection may occur on both levels and gains.



OLS, IV, ATT, and ATE generally weight the distribution of β_i differently.

CIA, SUTVA, and overlap carry the identification argument

Assumption	Operational meaning	Failure mode
CIA	$(Y_1, Y_0) \perp T \mid X$	Important unobservables still drive treatment
SUTVA	One treatment version; no interference	Spillovers or hidden versions of treatment
Overlap	$0 < P(T=1 \mid X) < 1$	No comparable treated/control units in some cells

Assumptions should be translated into features of the institutional setting.

02 Selection on observables

- Design comparisons inside common support
- Use the propensity score to balance covariates
- Diagnose failure before estimating effects

Unconfoundedness identifies effects within covariate cells

$$E[Y_1 - Y_0 \mid X=x] = E[Y \mid T=1, X=x] - E[Y \mid T=0, X=x]$$

- **Claim:** within X-cells, treatment assignment is as good as random.
- **Aggregation:** average conditional effects over the target population.
- **Risk:** functional-form extrapolation can hide lack of common support.

Matching is a design exercise before it is an estimator

1

Choose pre-treatment covariates. Include confounders; do not condition on post-treatment variables.

2

Enforce common support. Discard comparisons that require extrapolation.

3

Construct comparable sets. Match, stratify, or weight using a stated distance rule.

4

Inspect balance. Evaluate standardized differences and the full distributions.

5

Estimate and stress-test. Vary specifications and report sensitivity.

The propensity score compresses X while preserving balance

What it buys

- A scalar balancing score $p(X)=P(T=1|X)$
- A practical route to matching, stratification, and weighting
- A visible diagnostic for common support

What it does not buy

- It does not make unconfoundedness true
- It does not repair omitted pre-treatment confounders
- It does not justify extreme extrapolation

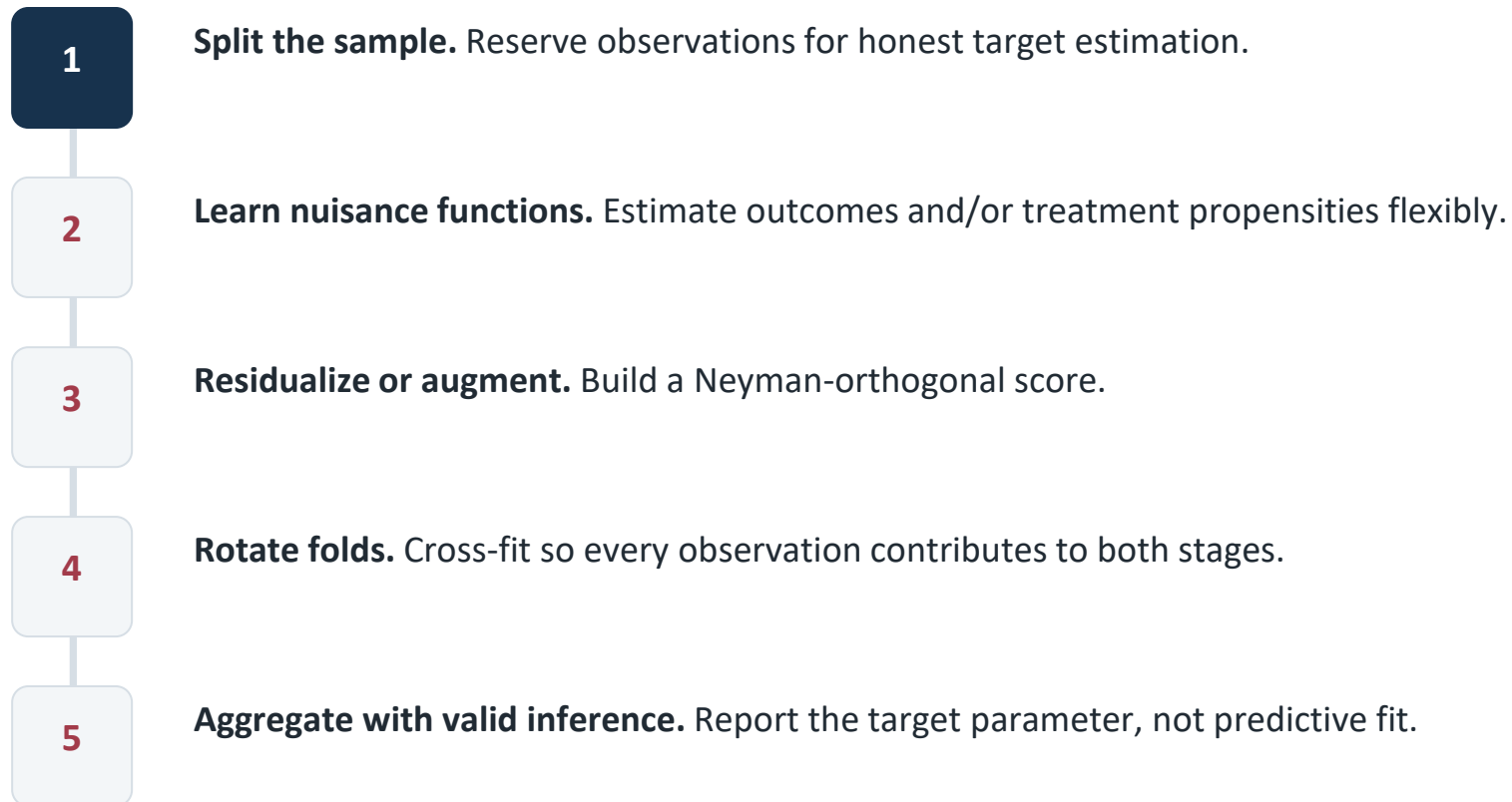
IPW reconstructs a target population

$$\text{ATE: } E[TY/p(X) - (1-T)Y/\{1-p(X)\}]$$

- **Treated units:** receive weight $1/p(X)$.
- **Control units:** receive weight $1/\{1-p(X)\}$.
- **Practical warning:** extreme propensities create unstable weights and high variance.

AIPW and DML protect the target moment from nuisance error

Orthogonal scores reduce first-order sensitivity to imperfect nuisance estimates; cross-fitting limits own-observation bias.



Diagnostics decide whether the design is credible

Estimation comes after the design passes observable stress tests.

- **Overlap:** plot propensity distributions; identify regions without comparisons.
- **Balance:** compare means and distributions after matching or weighting.
- **Placebos:** test for effects on lagged outcomes or predetermined covariates.
- **Sensitivity:** show how results move across design choices.

A precise standard error cannot rescue an implausible comparison.

03 Experiments, noncompliance, and IV

- Separate assignment from treatment received
- Interpret the Wald ratio through compliance types
- Match the local effect to the policy question

Random assignment identifies ITT even when treatment is not taken

Keep the two causal questions distinct: the effect of an offer and the effect of treatment received.

Assignment Z

- Randomized by design
- Often known for every unit
- Direct comparison identifies ITT

Treatment D

- May be chosen or imperfectly complied with
- Potentially endogenous
- Needs IV assumptions for a treatment effect

The Wald ratio rescales assignment effects by compliance

$$\text{LATE} = \{E[Y|Z=1] - E[Y|Z=0]\} / \{E[D|Z=1] - E[D|Z=0]\} = \text{ITT} / \text{first stage}$$

- **Numerator:** reduced-form effect of assignment on the outcome.
- **Denominator:** change in treatment take-up caused by assignment.
- **Interpretation:** average effect for units whose treatment is changed by Z.

Monotonicity partitions the population into four response types

Type	D(0)	D(1)	Response to encouragement
Never-taker	0	0	Never takes treatment
Complier	0	1	Follows the encouragement
Defier	1	0	Moves opposite to encouragement
Always-taker	1	1	Always takes treatment

Monotonicity rules out defiers; only compliers generate the first stage.

A valid instrument needs four linked assumptions

Requirement	Formal idea	Institutional question
Relevance	$\text{Cov}(Z, D) \neq 0$	Does assignment materially shift treatment?
Independence	$Z \perp \text{potential outcomes}$	Why is the instrument as-if random?
Exclusion	Z affects Y only through D	Could the instrument change outcomes directly?
Monotonicity	$D(1) \geq D(0)$	Could anyone systematically move the other way?

LATE is local to the compliers moved by this instrument

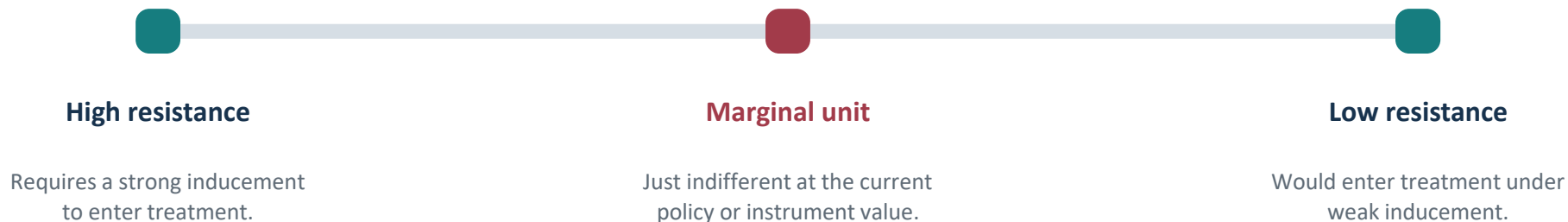
Different instruments can identify different averages—even for the same treatment and outcome.

- **Describe compliers:** use first-stage heterogeneity across observed characteristics.
- **Assess transport:** explain why complier effects should—or should not—generalize.
- **Align policy:** ask whether the planned intervention moves the same margin.

External validity begins with a clear description of the identifying margin.

MTE connects local margins to policy-relevant averages

$MTE(x, u_D) = E[\beta \mid X=x, U_D=u_D]$ is the effect for people at a treatment-choice margin.



ATE, ATT, LATE, and policy-relevant effects integrate the MTE with different weights.

Weak instruments demand identification-robust inference

Warning signs

- Small or unstable first-stage effects
- Large standard errors and sensitivity to controls
- 2SLS pulled toward OLS in finite samples

Responses

- Report first-stage diagnostics and reduced form
- Use Anderson–Rubin or related robust procedures
- Consider LIML/JIVE and simplify the instrument set

Shift-share IVs require a precise source-of-randomness story

$$Z_i = \sum_k \text{share}_{ik,0} \times \text{shift}_k$$

- **Shock-based design:** many independent, exogenous shifts drive identification.
- **Share-based design:** initial exposure shares are the defended exogenous objects.
- **Inference:** account for common exposure and concentration of identifying shocks.

04 Difference-in-differences

- Construct the missing trend, not just a before–after contrast
- Treat event-study comparisons as designed 2×2 experiments
- Make staggered-adoption controls explicit

The 2×2 DiD subtracts the counterfactual trend

$$ATT = (\bar{Y}_{T,post} - \bar{Y}_{T,pre}) - (\bar{Y}_{C,post} - \bar{Y}_{C,pre})$$

- **First difference:** removes group-specific time-invariant levels.
- **Second difference:** removes the common untreated time shock.
- **Target:** the treated group in the post-treatment period.

Parallel trends and no anticipation are the design assumptions

Parallel trends

- Untreated outcome changes would match across groups
- May hold only conditional on covariates
- Is about potential outcomes, not observed pre-trends alone

No anticipation

- Units do not respond before treatment starts
- Policy announcements can shift behavior early
- Leads can reveal—but not fully rule out—violations

Event studies reveal dynamics but do not prove identification

Event time traces effects relative to a stated reference period.

- **Pre-treatment coefficients:** diagnose gross differences in trends and anticipation.
- **Post-treatment coefficients:** show onset, persistence, and possible adaptation.
- **Inference:** account for multiple testing when reading an entire path.
- **Design:** define valid controls separately for each cohort and event time.

Staggered TWFE can compare treated units with already-treated units

Valid comparisons

- Newly treated versus never treated
- Newly treated versus not-yet treated
- Cohort-specific event-time contrasts

Contaminated comparisons

- Newly treated versus already treated
- Effects with opposite or opaque weights
- Compositional changes hidden by one coefficient

Modern DiD estimators make the control group explicit

Approach	Control group	Key trade-off
Sun–Abraham	Never treated	Stable controls; may be less comparable
Callaway–Sant’Anna	Never or not-yet treated	Flexible group-time ATT aggregation
Borusyak–Jaravel–Spiess	Untreated observations under stronger trend structure	Efficient imputation; stronger assumptions

Start from the group-time ATT, then aggregate transparently.

Covariate adjustment belongs inside the DiD design

1

Define cohort and event time. State the treatment timing and target ATT.

2

Choose valid controls. Never treated and/or not-yet treated.

3

Enforce overlap. Compare units with support in pre-treatment X.

4

Adjust flexibly. Use regression adjustment, IPW, AIPW, or DML.

5

Aggregate transparently. Show the weights across cohorts and event times.

Continuous treatment and spillovers change the estimand

Treatment intensity

- Parallel trends need not rule out selection on dose
- Define a causal response curve, not an opaque slope
- Generalized propensity scores can rebalance intensity

Interference

- Untreated units may receive indirect exposure
- Control outcomes depend on neighbors' treatment
- Specify an exposure mapping and a new estimand

05 Regression discontinuity

- Exploit a known treatment assignment threshold
- Estimate a local effect with transparent smoothing choices
- Use manipulation and balance checks as design evidence

RD compares units at a treatment threshold

If potential outcomes vary smoothly in the running variable, units just above and below the cutoff are comparable.

- **Running variable R:** determines treatment through a known cutoff c .
- **Locality:** the design identifies effects for units near c .
- **Continuity:** counterfactual outcomes should not jump at c absent treatment.

The cutoff generates the comparison; the regression only estimates it.

Sharp and fuzzy RD identify different local effects

Design	Treatment rule	Estimand
Sharp RD	$D = 1\{R \geq c\}$	Jump in $E[Y R]$ at c
Fuzzy RD	$P(D=1 R)$ jumps at c	Outcome jump / treatment jump: local Wald effect

Fuzzy RD is an IV design for compliers at the threshold.

Bandwidth choice trades variance against approximation bias

Local polynomial estimation uses observations within h of the cutoff.



Narrow h

Low approximation bias; few observations and high variance.



Balanced h

Data-driven bandwidth targets an explicit loss criterion.



Wide h

More precision; greater reliance on functional form.

Report robust bias-corrected inference and sensitivity to reasonable bandwidths.

Credible RD evidence includes density, balance, and sensitivity checks

- **Density at the cutoff:** look for sorting or manipulation of the running variable.
- **Predetermined covariates:** verify continuity at the threshold.
- **Alternative bandwidths/orders:** show the result is not a tuning artifact.
- **Placebo cutoffs:** check for jumps where no treatment change occurs.

A smooth fitted line is not evidence of a valid RD design.

06 Panel data and inference

- Use repeated observations to remove fixed heterogeneity
- Identify which units and periods generate the coefficient
- Cluster at the level of independent treatment variation

Fixed effects remove time-invariant omitted variables

$$y_{it} - \bar{y}_i = (x_{it} - \bar{x}_i)\beta + (\varepsilon_{it} - \bar{\varepsilon}_i)$$

- **Removed:** all unit-specific factors that are constant over time.
- **Retained:** within-unit changes in x and y.
- **Still required:** strict or appropriately sequential exogeneity of the within variation.

First differences reveal that movers identify the coefficient

$$\Delta y_{it} = \Delta x_{it} \beta + \Delta \varepsilon_{it}$$

- **Binary treatment:** units with $\Delta D=0$ do not identify β in the differenced equation.
- **Interpretation:** the effect is local to switchers or movers.
- **Threat:** switching may respond to transitory shocks in $\Delta \varepsilon$.

Random effects gain efficiency by adding an orthogonality restriction

Fixed effects

- Allows $\text{Cov}(a_i, x_{it}) \neq 0$
- Uses within-unit variation
- Cannot estimate time-invariant regressors directly

Random effects

- Requires $E[a_i | x_{i1}, \dots, x_{iT}] = 0$
- Combines within and between variation
- More efficient when the restriction is credible

Standard errors must follow the level of identifying variation

1

Locate the treatment variation. Individual, firm, region, cohort, or time?

2

Locate dependence. Which observations share shocks or assignment?

3

Choose clusters. Cluster at or above the independent assignment level.

4

Check cluster count. Use small-cluster corrections or randomization inference when needed.

5

Explain the design. A large row count does not create independent treatment variation.

Two-way fixed effects are a panel model—and a DiD estimator

Panel interpretation

- Unit FE absorb time-invariant levels
- Time FE absorb common shocks
- β comes from residual treatment changes

DiD interpretation

- With one adoption date, β reproduces a DiD contrast
- With staggered timing, β aggregates many 2×2 comparisons
- Heterogeneous effects can make that aggregation misleading

07

IV mechanics, GMM, and dynamic panels

- Project treatment onto defended instruments
- Treat moment conditions as economic assumptions
- Use internal instruments carefully in dynamic panels

2SLS uses only the component of treatment projected on instruments

$$\text{Stage 1: } D = Z\pi + X\gamma + v \quad \text{Stage 2: } Y = D\beta + X\theta + u$$

- **Relevance:** Z must explain D conditional on X.
- **Exogeneity:** Z must be orthogonal to the structural error.
- **One coefficient:** may still summarize heterogeneous effects with instrument-specific weights.

Overidentification tests are specification checks, not proof of validity

What rejection says

- At least one moment condition is inconsistent with the others
- The model or instrument set needs investigation
- Heterogeneous effects may also create unequal IV estimates

What non-rejection does not say

- It does not prove every instrument is exogenous
- Low power can conceal violations
- Common invalidity can pass undetected

Measurement error creates endogeneity

$$\mathbf{x}^* = \mathbf{x} + \mathbf{v} \Rightarrow \text{plim } \beta_{\text{OLS}} = \beta \cdot \text{Var}(\mathbf{x}) / \{\text{Var}(\mathbf{x}) + \text{Var}(\mathbf{v})\}$$

- **Classical error:** attenuates the slope toward zero.
- **Panel data:** differencing can amplify noise relative to signal.
- **Remedies:** validation data, repeated measures, reliability corrections, or valid IV.

GMM turns economic restrictions into sample moments

Choose θ to make $\bar{g}(\theta) = n^{-1} \sum_i g(W_i, \theta)$ close to zero

- **Moments:** encode orthogonality or other model restrictions.
- **Weighting matrix:** determines efficiency and the geometry of the fit.
- **Diagnostics:** use Hansen/Sargan tests cautiously and inspect instrument proliferation.

Arellano–Bond instruments differenced dynamics with lagged levels

1

Start with dynamics. $y_{it} = \rho y_{i,t-1} + x_{it}\beta + a_i + \varepsilon_{it}$.

2

Difference out a_i . The lagged dependent variable becomes endogenous.

3

Use deeper lags. Levels dated $t-2$ and earlier can instrument $\Delta y_{i,t-1}$ under serial-correlation restrictions.

4

Test the error structure. AR(1) is expected after differencing; AR(2) is a warning.

5

Limit instruments. Too many instruments overfit endogenous variables and weaken diagnostics.

08

Limited outcomes, selection, and duration

- Match the model to the support of the outcome
- Translate latent-index coefficients into observable effects
- Model selection when the observed sample is non-random

LPM, logit, and probit encode different functional-form commitments

Model	Conditional mean	Primary advantage
LPM	$P(Y=1 X)=X\beta$	Transparent slopes; easy FE/IV; robust interpretation
Logit	$\Lambda(X\beta)$	Probabilities in [0,1]; convenient odds structure
Probit	$\Phi(X\beta)$	Latent-normal framework; useful in selection models

Model choice should follow the estimand and identification problem—not habit.

Report marginal effects on probabilities, not latent-index coefficients

$$\partial P(Y=1|X)/\partial x_k = f(X\beta) \cdot \beta_k$$

- **Nonlinearity:** the effect varies with X even when β_k is constant.
- **Summary:** state whether you report average marginal effects or effects at chosen X.
- **Comparisons:** coefficient scales can differ across groups when latent variances differ.

Censoring and truncation discard different information

Censoring

- The unit remains in the sample
- The outcome is only known to lie beyond a threshold
- Likelihood combines mass at the limit with a density above it

Truncation

- Units outside the selection rule are absent
- Their covariates and outcomes may be unobserved
- Likelihood conditions on sample inclusion

Heckman correction models selection through an inverse Mills ratio

1

Selection equation. Estimate $P(S=1 | Z)$ with a probit model.

2

Compute λ . The inverse Mills ratio summarizes selection under joint normality.

3

Outcome equation. Regress Y on X and λ in the selected sample.

4

Identify carefully. A credible exclusion from the outcome equation is strongly preferred.

5

Interpret structurally. The correction is only as credible as the distribution and selection model.

Roy and switching models put comparative advantage at the center

Outcome states

- $Y_0 = X\theta_0 + U_0$
- $Y_1 = X\theta_1 + U_1$
- Individual gain $\beta_i = Y_1 - Y_0$

Selection rule

- Choose treatment when expected gain exceeds cost
- Selection may depend on both baseline outcomes and gains
- Observed groups are positively selected into their comparative advantage

Duration models separate survival from the instantaneous hazard

$$h(t|X) = \lim_{\Delta \rightarrow 0} P(t \leq T < t + \Delta \mid T \geq t, X) / \Delta$$

- **Survival $S(t)$:** probability the spell lasts beyond t .
- **Hazard $h(t)$:** instantaneous exit rate conditional on survival to t .
- **Censoring:** contributes exposure information without a completed duration.

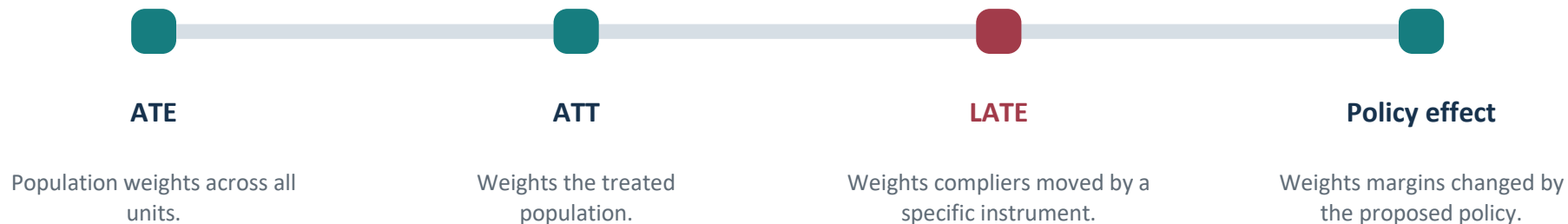
Count and multiple-choice models must match the outcome support

Outcome	Common model	Key diagnostic
Counts	Poisson QML / negative binomial	Mean specification, zeros, overdispersion
Unordered choices	Multinomial or nested logit	IIA and substitution patterns
Ordered choices	Ordered logit/probit	Threshold structure; marginal effects by category

Coefficient signs alone may not reveal probability effects in nonlinear multi-outcome models.

Estimands are weighted averages of heterogeneous effects

The question is not only whether effects differ—but how the design weights them.



Report the weighting population and the identifying margin alongside the estimate.

Choose the method from the source of identifying variation

Variation	Natural design	Typical estimand
As-if random assignment	RCT / IV	ITT or LATE
Selection on observed X	Matching / IPW / AIPW	ATE or ATT on overlap
Differential policy timing	DiD / event study	Group-time ATT
Threshold rule	Sharp or fuzzy RD	Local ATE or local LATE
Within-unit change	FE / first differences	Effect for movers under exogeneity

A credible empirical design survives four audits

- **Estimand audit:** whose effect, under which intervention, over what horizon?
- **Identification audit:** which variation is exogenous, and why?
- **Estimation audit:** what weights, functional forms, tuning choices, and diagnostics?
- **Inference audit:** what is the independent unit of variation, and are clusters sufficient?

A transparent limitation is stronger than an overbroad causal claim.

Close the loop: answer the policy question you actually identified

A strong empirical conclusion has three sentences.

- **Design:** “Variation in treatment comes from...”
- **Estimand:** “Under these assumptions, the estimate is the effect for...”
- **Policy meaning:** “This evidence supports—or does not support—extrapolation to...”

Credibility comes from alignment: question → estimand → design → estimator → inference.