Causal design and urban policy evaluation: A very brief introduction

Nick Dorward

University of Bristol

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nicholas.dorward@bristol.ac.uk

The case for causality

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  - Health - Do "clean air zones" reduce asthma incidence?
  - Inequality - Does improving neighbourhood quality change health and economic outcomes?

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What is causality?

- “We may define a cause to be an object, followed by another, and where all the objects similar to the first are followed by objects similar to the second. **Or in other words where, if the first object had not been, the second never had existed**." (David Hume, 1748)

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- **Key idea -** ***the counterfactual***. Alternative possibilities that we imagine in thought experiments to unpick causality.

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- Not a prediction of the effect that a **specific** choice or decision will have on an outcome

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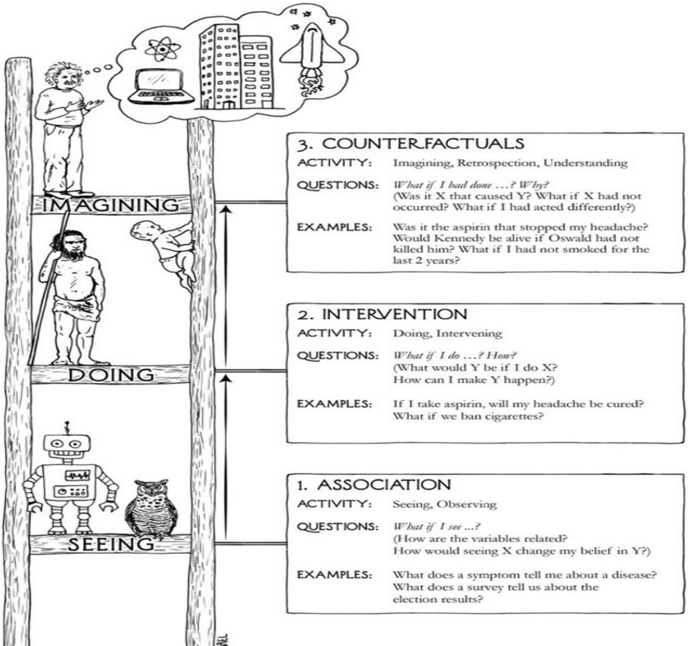
- Causal inference takes a predicted counterfactual and constructs a causal effect which, we hope, tells us something about the state of the future world in the event we make a specific choice.
- Key for policy applications. We know not only the past, but the future

The ladder of causation



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- **Heuristic:** Counterfactual thinking to formalise assumptions about processes. If this theory were true, what would prove me wrong?
- **Analytic:** Counterfactuals to make causal inferences. Estimate the true *effect* of an intervention on an outcome/process.
- **Hybrid:** Counterfactuals as a system of thinking, design, and analysis to make *more credible* claims about causal relationships.

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  - One in which a unit, i, receives an intervention - the actual state, let's call this Y_i^1.
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  - One in which the unit does not receive the intervention - the counterfactual state, Y_i^0.
- The individual causal (or treatment) effect of the intervention is the simple difference in outcomes (SDO) between the world in which the intervention occurs compared to the one where it does not:

$$\delta_i = Y_i^1 - Y_i^0 \tag{1}$$

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- Causal inference is a missing data problem where we need to make predictions, not of the present or future, but of a missing past.

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- If $D_i = 1$, then $Y_i = Y_i^1$ because the second term in (2) zeroes out. And if $D_i = 0$, the first term zeroes and $Y_i = Y_i^0$.

Potential outcomes in action

- But we have a distribution of both y_i^1 and y_i^0 in the population. So, we can estimate 'average treatment effects' (ATE) across the population by comparing outcomes for 'treatment' (those with y_i^1) and 'control' (those with y_i^0) groups.

- Average treatment effects are *unknowable* because, according to the switching equation, we don't have both potential outcomes for each observation. But it can be *estimated* from samples of data.

- The simple difference in means between the treatment and control groups will give us the average treatment effect from across the population.

Potential outcomes in action (cont'd)

$$
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SDO &= \frac{1}{N_T}\sum_{i=1}^{N}(y_i|d_i=1) - \frac{1}{N_C}\sum_{i=1}^{N}(y_i|d_i=0) \\
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&+ \underbrace{E[Y_i^0|D_i=1] - E[Y_i^0|D_i=0]}_{\text{Selection bias}}.
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Selection bias: the difference between treatment and control groups with no intervention.

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- Independence of Y_i^0 and D_i allows us to swap in $E[Y_i^1|D_i=1]$ in for $E[Y_0^1|D_i=1]$ in line 2 because because potential outcomes for Y_i^0 and Y_i^1 are the same.

Important assumptions

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- Stable Unit Treatment Value Assumption (SUTVA):
  - Homogeneous treatment - the level (or dosage) of the treatment is homogeneous across groups.
  - Non-interference - no externalities or spillover from treatment. Treatment status of unit i does not affect potential outcomes of unit j (e.g., (a)spatial networks).

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- But selection bias reflects endogenous sorting into treatment and control.
- Randomisation solves the selection problem (under certain assumptions)
- To make causal inferences we need random assignment of interventions or to be able to simulate randomness in some plausible way.

RCTs and (quasi)experiments

- **Experiments and RCTs** - explicitly randomise a policy intervention across treatment and control groups - 'balanced' on unobservables.

- **Natural experiments** - leverage arbitrary divergences in laws, policies, or practices to analyse the effects of an intervention on a population as is if they had been part of an experiment. Looks at differences across treatment and control groups 'as if' intervention was randomly assigned - regression discontinuity.

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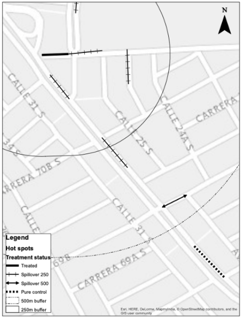
- Limitations include: non-compliance (50%), disruption of moving, non-random selection into destination neighbourhoods.

Policing in Bogata (Blattman et al., 2021)

- City randomly reallocated existing police and municipal resources across experimental street blocks.

Figure 2: An example of assignment to the four treatment conditions



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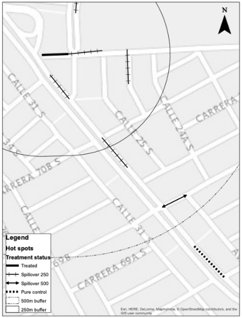

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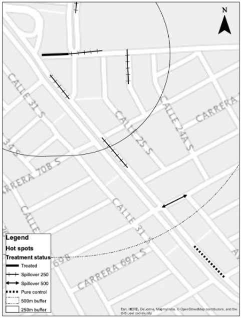

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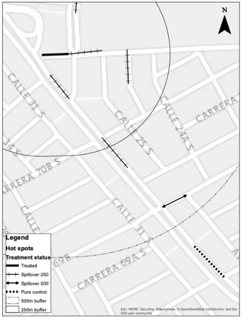

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- No overall decline in crime from policing or services, differences across violent and property crimes, policing treatment displaced crime into non-treatment streets.
- Many results not statistically significant, but may be substantively meaningful to policy makers.

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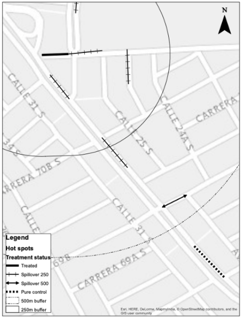

Part 2. The causal inference 'tool box'

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- Toolbox of post-assignment corrections that leverage 'as if' random variation in interventions to recover causal parameters:
  - Controls, matching, & fixed-effects
  - Difference-in-differences
  - Regression discontinuity
  - Instrumental variables

Backdoor criterion

$$\begin{tikzcd}
& U \arrow[dl, dashed] \arrow[dr, dashed] & \\
T \arrow[rr] & & Y
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Figure: Unobservable U

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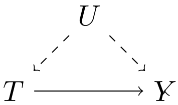

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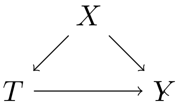

Figure: Observable X

- Consider the standard regression equation:

$$y_i = T_i\beta_i + X_i\delta_i + U_i + \epsilon_i$$

- where $T_i\beta_i$ is the treatment, $X_i\delta_i$ are observed controls, and U_i are unobserved confounders.

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  - Precision - Even if not related to the assignment probability, including controls that are related to the outcome will reduce residual variance increasing precision of estimates.

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- Constructs comparison groups that are similar along a set of *observed* matching variables using weights.
- In many ways similar to regression.
- Uses a different set of assumptions and is less model dependent than regression.
- Suffers from the same fatal flaw - at least when it comes to estimating causal effects - of assuming that our set of observed variables are enough to close all back doors.

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- Typically used in context of cross-sectional time-series data.
  - Removing variation between units focusing upon within unit variation over time.
- Can be extended to multiple fixed effects and time as well as geography (TWFE).

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- Removes time-invariant components of unobservables, U, that are common to treatment and control groups.
- Primary identifying assumption - parallel trends.
  - No time-varying differences in unobservables between treatment and control groups.
- Commonly applied to natural experiments where some areas receive intervention by chance.

John Snow's cholera study (1855)

John Snow's cholera study (cont'd)

- Years of studying cholera epidemics in the 19th C led Snow to question the prevailing miasma hypothesis

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- Proved that both companies served similar households within the same neighbourhoods (i.e., balance on covariates).
- Interpret effect of clean water while holding confounders - hygiene, poverty, neighbourhood - constant.

How diff-in-diff works

Table: Snow's data

Company name	**1849**	**1854**
Southwark and Vauxhall	135	147
Lambeth	85	19

1) First difference - difference in Lambeth and S&V outcomes in 1854.

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How diff-in-diff works

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   - Selection bias

2) Second difference - compare Lambeth before and after intervention.
   - Time trends.

3) Diff-in-diff - combine differences to eliminate selection bias and time trend
   - Parallel trends - difference between treated and untreated units the same pre- and post-treatment without intervention.

How diff-in-diff works (cont'd)

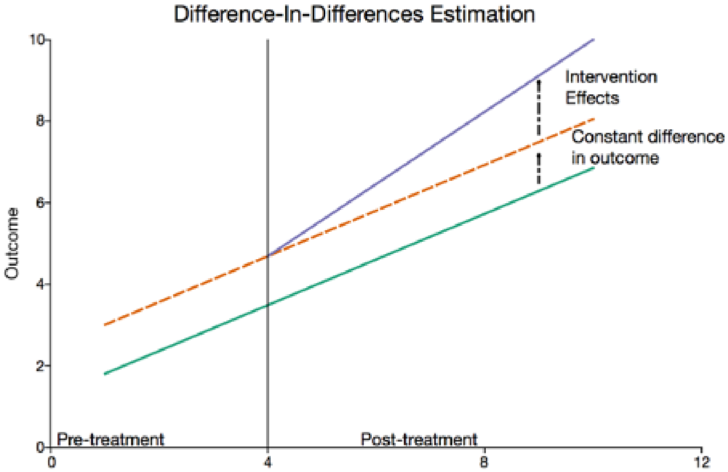

Figure: difference-in-differences estimator

How diff-in-diff works (cont'd)

- Diff-in-diff estimator:

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- This generalises to:
  - Multiple cross-sectional units
  - Multiple temporal units
  - Treatment in multiple periods

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- Compare changes in outcomes pre- and post-intervention using a naturally occurring control and treatment group.
- Difference out unobervables across units and time.
- Key assumption - parallel trends. Based on a counterfactual that cannot be empirically validated.
- John Snow example shows these assumptions rest on deep empirical and contextual knowledge of the problem.

Regression Discontinuity Design

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- We can estimate the causal effect of the intervention by comparing the sub-population of units around the threshold.
- Seen by many as the 'gold standard' in causal inference with observational data.

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- **Cutoff/threshold** - the specific value long the running variable at which treatment is assigned.
- **Bandwidth** - Everything is related to everything else but those things closer to the cutoff are more similar than things farther from the cutoff. The bandwidth determines how close to the cutoff we look to make our comparison.

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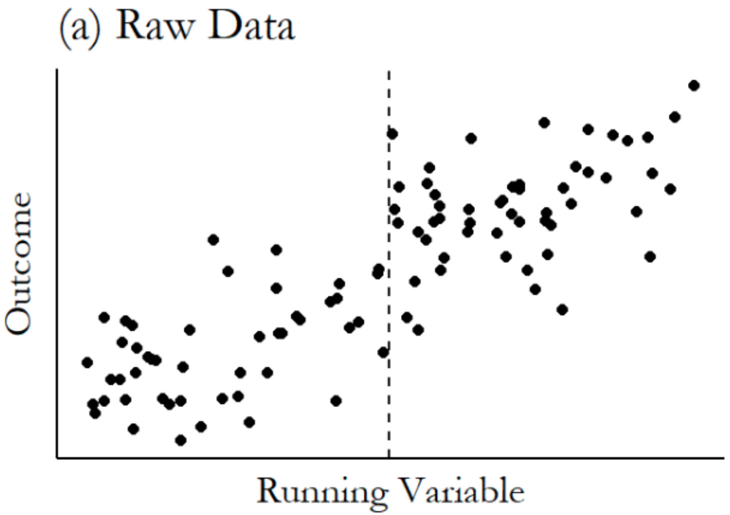
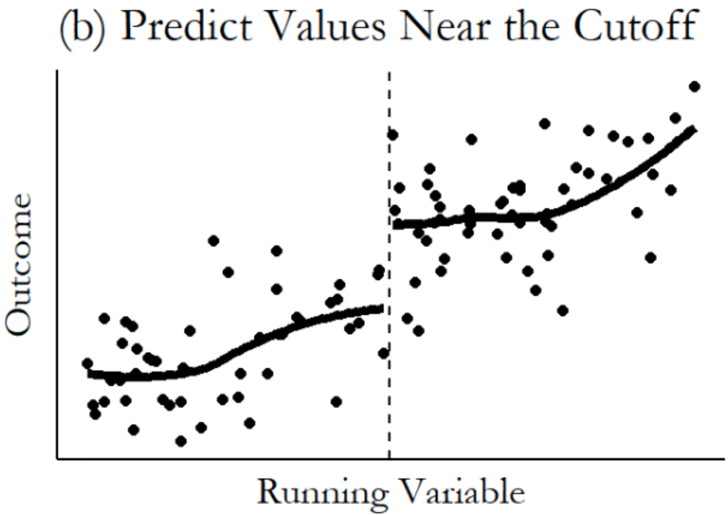
- Focus upon observations around the cutoff within the bandwidth.
- Compare the just-barely treated units against the just-barely untreated units.

How RDD works



How RDD works (cont'd)



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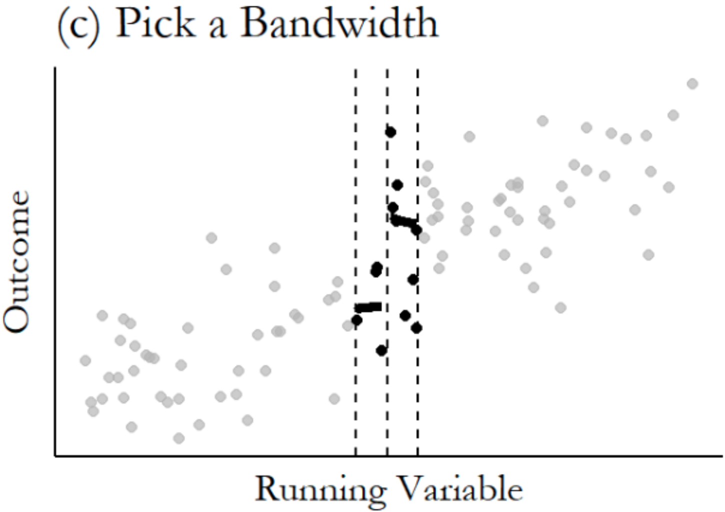

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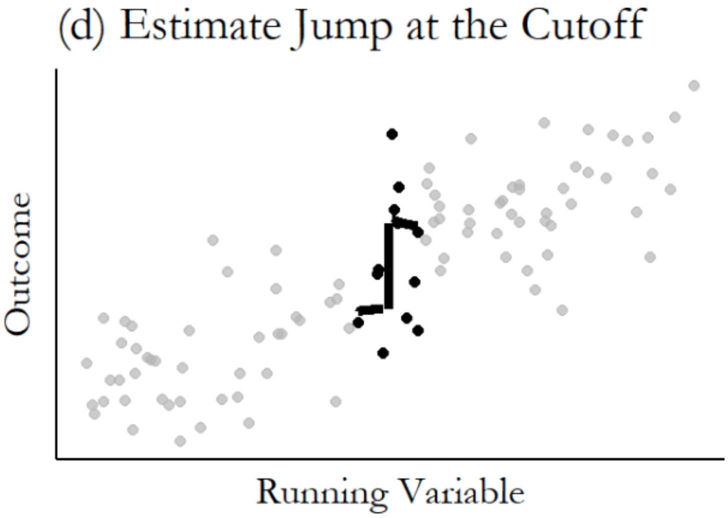
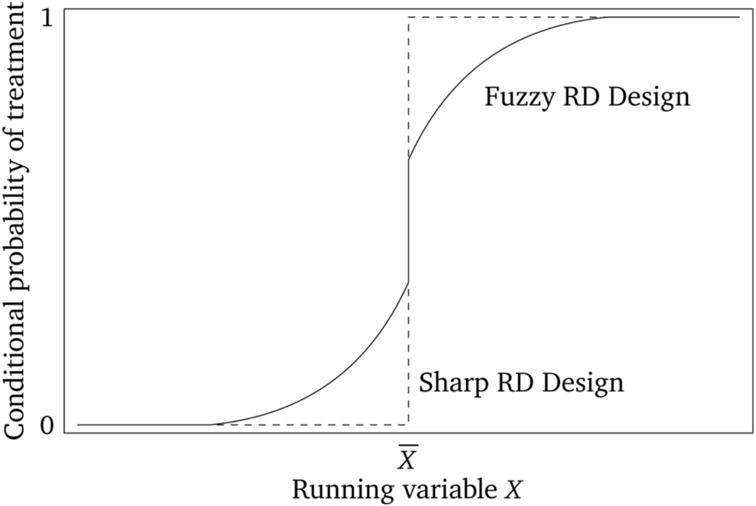


Sharp vs. fuzzy RDD

- In fuzzy RDD the threshold is not discrete and only changes the probability of being assigned an intervention.



Key RDD assumptions

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- However, this can be violated when:
  - Units can sort their treatment status.
  - Cutoff is endogenous to unobservables that influence the outcome.
- Analyst must know the assignment rule!

RDD with geographic boundaries

- Geographic borders can act as a discontinuity.
- When one a policy is arbitrarily implemented on one side of a boundary and not the other.
- Places or observational either side are more likely to be similar - comparable on unobservables and potential outcomes.
- Challenges:
  - Sorting - individual can sort across geographic boundaries
  - Interference - aka spatial diffusion/spillover
  - Context - borders not randomly assigned and thus endogenous to outcome and potential outcomes - think gerrymandered districts and political outcomes.

Important ideas not discussed

- Estimands/treatment effects
- Heterogeneous treatment effects
- Instrumental variables (see back of slides)
- Synthetic controls
- Spatial causal inference
- Causal machine learning

Summary (cont'd)

Table 1 Summary of the key analytical methods used to assess health interventions and their relative trade-offs

Analytical method	Description	Advantages	Disadvantages	Trade-offs relative to other methods
Interrupted Time Series (ITS)	A before-after comparison in the level and trend of outcomes pre and post intervention [17, 21, 22]	Straightforward methodological approach without reliance on simplifying assumptions [17, 21, 22]	Influenced by simultaneous events occurring at the time of intervention [17, 21, 22]	No control group to compare intervention effects against a group exposed to the intervention which can bias estimated intervention effects [23]
Difference-in-differences (DiD)	A contrast of outcome changes pre and post intervention using a naturally occurring control group and treatment group subject to the intervention change [18, 24]	Using the intervention itself as a naturally occurring experiment, allows to difference out any exogenous effects from events occurring simultaneously [18, 24]	The parallel trends assumption is based on counter-factual intervention trends which cannot be tested [18, 24]	Use of a naturally occurring control group to compare intervention effects naturally isolates group differences from intervention effects. No statistical test to verify the parallel trends assumption can bias estimated effects [18, 24]
Synthetic Control (SC)	Comparison of treatment effects between a treatment group and a constructed control i.e. a synthetic control using weights similar to treatment outcomes pre-intervention [25, 26]	Can complement other analytical methods particularly when a naturally occurring control group cannot be established and/or when simplification assumptions do not hold e.g. the parallel trends assumption in DiD [25, 26]	Requirement of sufficient data pre and post intervention containing sufficient detail of control weights similar to the treatment group [19]	Can overcome parallel trends assumption required for DiD. Cannot test for similarity of controls used to construct the synthetic control which may bias estimated intervention effects. Heavy data requirement pre and post intervention [19, 25]
Matching	A comparison of outcomes between treatment and control groups pre and post intervention post matching groups with similar observable factors [18, 27]	Reduction of biases within groups is eliminated due to matching [18, 27]	Requirement of sufficient data pre and post intervention for matching similar observable characteristics between treatment and control groups. No statistical means to testing 'similarity' [27]	Heavy data requirement to match similar characteristics. Matching is limited to observable factors and does not account for non-observable factors. 'Similarity' determined using subjective judgment and cannot be statistically measured and can bias estimates [27].
Instrumental Variables (IV)	An observable variable i.e. the instrument is selected to randomise the estimation of treatment effects [18, 20, 28]	Introduction of randomness when estimating treatment effects to reflect similarity to a RCT [18]	Dependence on choosing the most appropriate instrument to satisfy the assumption of no relationship between the outcome and assuming outcome is affected only via intervention exposure [18, 29]	Imposed randomisation using an instrument useful for estimating intervention effects. Randomisation is imposed and not naturally occurring like with DiD and can bias estimated effects [18, 20, 28, 29]

Instrumental variables

A way of identifying causal effect of an intervention by identifying a source of random variation in treatment assignment that is not affected by unobservables.

The instrument, Z, mimics the explicit random assignment of T in RCTs with something that has already randomised T in the real-world.

Use Z to statistically isolate variation in T driven by Z and identify causal effect of T on Y:

- 1) Use Z to explain T
- 2) Remove any part of the T that is not explained by Z
- 3) Use Z to explain tY removing any Y not explained by Z
- 4) Assess relationship between Z-explained part of T and Z-explained part of Y

How IV works

$$\begin{tikzcd}
& U \arrow[dl, dashed] \arrow[dr, dashed] & \\
T \arrow[rr] & & Y
\end{tikzcd}$$

Figure: Endogeneity

$$\begin{tikzcd}
& & U \arrow[dl, dashed] \arrow[dr, dashed] & \\
& T \arrow[rr] & & Y \\
Z \arrow[ur] & & &
\end{tikzcd}$$

Figure: Instrumental variable

$$\begin{tikzcd}
& & U \arrow[dl, dashed] \arrow[dr, dashed] & \\
& T \arrow[rr] & & Y \\
Z \arrow[ur] \arrow[urrr, bend right] & & &
\end{tikzcd}$$

Figure: Exclusion restriction violation

How IV works (cont'd)

Instrument variables estimation - For each Z-explained movement in T, how much Z-explained movement in Y was there?

Actual estimation is comparatively simple.

Most commonly performed via Two-stage least squares (2SLS):

$$T = \gamma_0 + \gamma_1 Z + \gamma_2 W + \upsilon \tag{4}$$

$$Y = \beta_0 + \beta_1 \hat{T} + \beta_2 W + \epsilon \tag{5}$$

Where W are controls, γ are first stage regression coefficients, and $\hat{T}$ are predicted values of T.

Choosing instruments

Credible inference in IV depends upon the choice of IV

A valid instrumental variable must satisfy three key criteria:

- Relevancy: $Cov(Z, Y) \neq 0$. Statistical vs substantive relevancy. Does Z theoretically *cause* Y?
- Exogeneiety: Z is assigned randomly or conditionally on controlled covariance, $\gamma_2 W$ in first-stage equation.
- Exclusion restriction: Z affects Y only through its influence on T. No "backdoor" between Z Y.

Choosing instruments (cont'd)

Selecting an instrument:

- Theoretically identify all possible source of variation in T
- Select ones that are least likely to be correlated with U. Exclusion resctrivtion.
  - DAGs are especially helpful here
- Estimate first stage equation to see if Z is a sufficiently strong (relevant) predictor of T.

Bad instruments

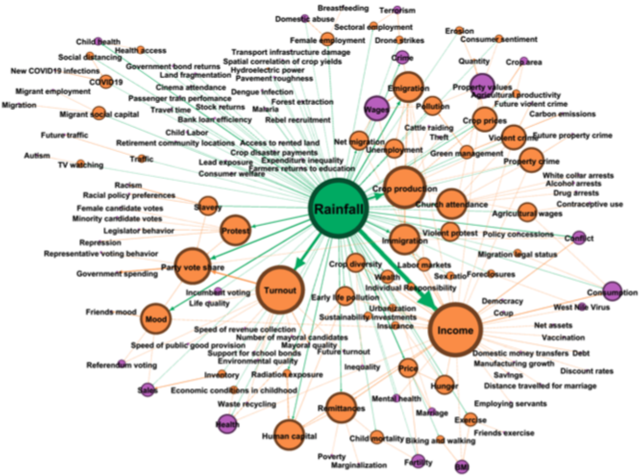

Figure: Rainfall IV

Good instruments?

Do highways cause suburbanisation?

Baum-Snow (2007) - did contruction of radial highways cause population decentralisation in US cities?

Baum-Snow et al., (2014) - did contruction of radial highways cause population decentralisation in Chinese cities?

IV summary

- Well identified ID can recover causal effects of urban policy interventions.
- However, credible inference from IV is not mechanistic.
- Requires strong theoretical consideration of the instrument and variation in T.
- Strong theory must be used to justify the two main identifying assumptions:
  - Relevance: Z is relevant predictor of T.
  - Exogenous: Z is assigned randomly or "as if" by random
  - Exclusion restriction: Z is uncorrelated with Y.
    $Z \rightarrow T \rightarrow Y$