

Causal Inference

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Part 3a: Estimating a Causal Effect (of a Point Treatment)

Basic Setting



A = binary (point-)treatment

Y = some (numeric) outcome
(not survival / duration — that's special)

X = sufficient adjustment set of pre-treatment covariates

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Y = some (numeric) outcome
(*not survival / duration — that's special*)

X = sufficient adjustment set of pre-treatment covariates

Keeping it simple to focus on essentials!

General reference:

Goetghebeur, E, le Cessie, S, De Stavola, B, Moodie, EE,
Waernbaum, I. Formulating causal questions and principled statistical
answers. *Statistics in Medicine*. 2020; 39: 4922– 4948.

(Total) Causal Effect



Will focus on: Average causal effect

$$ACE = E(Y|\text{do}(A = 1)) - E(Y|\text{do}(A = 0))$$

or, with potential outcomes

$$ACE = E(Y(1) - Y(0)) = E(Y(1)) - E(Y(0))$$

aka: average treatment (ATE) or total causal effect (TCE), etc.

Key Assumptions

Consistency Assumption:

If we observe $A^i = a$ then $Y^i = Y^i(a)$ (for individual i)

i.e. the outcome we observe under the actual treatment is the potential outcome had the treatment been *set* to what it actually was.

Violated, e.g., if manipulation of A not well defined or so 'invasive' that observational setting not informative.

Example: A is 'BMI' — how to manipulate BMI itself?

Often: if violated, need more elaborate model; e.g. intervene in physical activity to change BMI.

Under consistency, and e.g. for binary A , can write

$$Y^i = Y(1)^i A^i + Y(0)^i (1 - A^i)$$

Note:

consistency implicit in graphical / $\text{do}(\cdot)$ approaches
→ invariance (at distributional level)

No-Interference



Common assumption: **no-interference**:

Vector \mathbf{a} = treatment assignments for n units, then

$$Y^i(\mathbf{a}) = Y^i(a^i),$$

i.e. PO does not depend on treatment **other** units received.

Violation: e.g. vaccines, social networks.

Stable unit–treatment value (SUTVA):

consistency + no-interference.

Conditional Exchangeability



Assumption of **cond. exchangeability** or **no unmeasured confounding** (or **random treatment assignment**, ignorability or ...):

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Interpretation: within values of X , can consider A like randomised wrt outcome.

Denote: X is **sufficient** to adjust for confounding;
or ‘valid adjustment set’.

Conditional Exchangeability

with $\text{do}(\cdot)$



Assumption of **cond. exchangeability** or **no unmeasured confounding** & '**consistency**' with **do**-notation:

$$p(y \mid x; \text{do}(A = a)) = p(y \mid x, a)$$

Interpretation: within values of X , whether $A = a$ obtained by intervention or observation makes no difference wrt. distribution of Y .

Useful software for querying DAGs: DAGitty ([Textor et al, 2016](#))

Pre-Treatment Covariates?



What makes X **pre-treatment covariates**?

⇒ must be **known** not to be affected by intervention in treatment A !

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Graphically: X non-descendants of A .

(Overview: methods for causal covariate selection

see Witte & Didelez, 2018:BiomJ)

Positivity Assumption

checking overlap



Often, methods for effect estimation require

Assumption of positivity:

$$p(a | x) > 0 \text{ for all } a, x \quad (p(x) > 0)$$

Interpretation: for all (suff.) covariate values, it must be possible that a subject receives any value of treatment.

In practice: often empirically (nearly) violated — modifications and adaptations of methods exist sometimes.

Positivity Assumption — Notes



- Target trial: can / should define eligibility criteria so that positivity is satisfied — requires domain knowledge

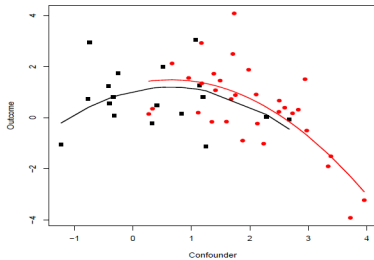
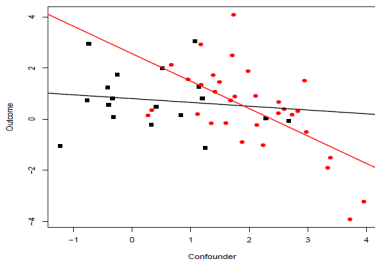
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- Do not include superfluous variables in X , especially: strong predictors of A that do not affect Y — can lead to apparent violations of positivity
- Regression-based approaches may mask lack of positivity as fitted models allow **extrapolation**.

Positivity Assumption

Extrapolation



We consider $p(y \mid \text{do}(A = a))$ or equivalently $p(Y(a))$.

With the above assumptions:

$$\begin{aligned} p(Y(a)) &\stackrel{(i)}{=} \sum_x p(Y(a)|x)p(x) \stackrel{(ii)}{=} \sum_x p(Y(a)|a, x)p(x) \\ &\quad \dots \stackrel{(iii)}{=} \sum_x p(y|a, x)p(x) \end{aligned}$$

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(i) probability calculus

(ii) valid adjustment set

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- (i) probability calculus
- (ii) valid adjustment set
- (iii) causal consistency & positivity

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- (i) probability calculus
- (ii) valid adjustment set
- (iii) causal consistency & positivity
- (iv) no-interference was needed for well-definedness of causal effect

Checking Assumptions?



- Consistency / no-interference:
domain knowledge, study design
- No-unmeasured-confounding: compare analysis of
observational data with actual randomised trial — often not
possible; triangulation, e.g. negative controls etc.
- Positivity
 - basic: boxplot of each variable in X by treatment group;
 - advanced: fit model for $\pi(x) = P(A = 1|X = x)$ — the
propensity score
obtain fitted values $\hat{\pi}^i = \hat{P}(A = 1|X = x^i)$ for each unit i
and check for near zero/one in treated and control group,
respectively.

Propensity Score: Checking Positivity

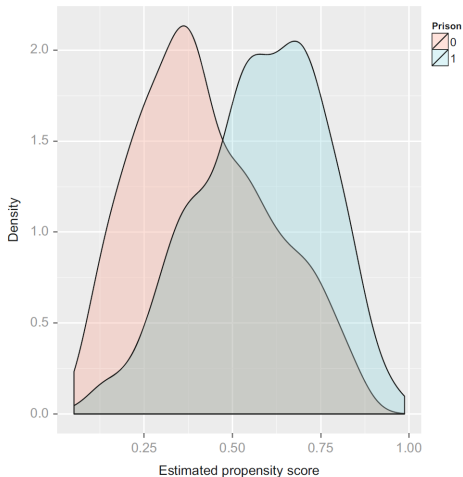


Example:

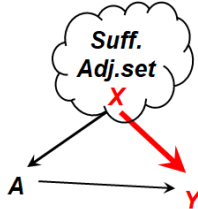
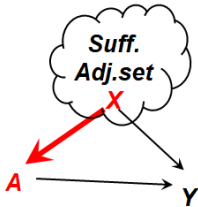
$n = 1022$ offenders
sentenced to either
probation $A = 0$
or prison $A = 1$;
 $X = 17$ covariates;
 $Y =$ recidivism (yes/no);
 \Rightarrow reasonable overlap.

Example taken
from [Guo et al. \(2016\)](#)

Covariate balance:
R package `cobalt`



Methods use either treatment model $p(a|x)$ (propensity score)
or outcome model $p(y|a, x)$ or both



Principles:

- regression (+standardisation),
- inverse-probability weighting (IPTW),
- stratification / matching,
- Hybrid: doubly-robust estimation (double-ML)

Standardisation

aka: G-Formula

(Robins, 1986)



Reminder: if X is sufficient set of covariates

$$E(Y \mid \text{do}(A = a)) = \sum_x E(Y \mid a, x) p(x)$$

An obvious way to use this is:

- fit flexible regression model for $E(Y \mid a, x; \beta)$ to data to avoid ‘g-null paradox’ (Evans & Didelez, 2023:JRSSB)
- average over empirical X -distribution: $\sum_i E(Y \mid a, x^i; \hat{\beta})/n$
- e.g. with R package `stdReg` (Sjølander and Dahlgvist, 2017)

Standardisation — Example



Y = 'low birth weight' (binary); X = 'mother smokes' (binary),
 $C = \{\text{'age', 'race'}\}$ (Sjolander, 2016)

```
> fit2 <- glm(formula=lbw~(smoker+race+age)^2,  
  family="binomial", data=clslowbwt)  
> fit.std <- stdGlm(fit=fit2, data=clslowbwt, X="smoker",  
  clusters="id")  
> summary(fit.std)
```

| | Estimate | Std. Error | lower 95 | upper 95 |
|---|----------|------------|----------|----------|
| 0 | 0.279 | 0.0406 | 0.199 | 0.358 |
| 1 | 0.407 | 0.0555 | 0.298 | 0.516 |

standardised means
control / treatment groups

```
> summary(fit.std, contrast="difference", reference=0)
```

| | Estimate | Std. Error | lower 95 | upper 95 |
|---|----------|------------|----------|----------|
| 0 | 0.000 | 0.0000 | 0.00000 | 0.000 |
| 1 | 0.128 | 0.0681 | -0.00544 | 0.262 |

difference, i.e.
estimated ACE

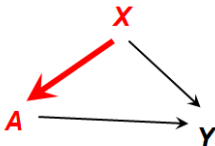
Why not just look at regression model $E(Y|A, X)$?

- Consider: marginal versus conditional causal effect and collapsible versus non-collapsible parameters;
- Logistic regression / odds ratios not collapsible.
- If set of sufficient covariates X not unique, **cond. effects may depend on choice of X (e.g. COR)**, but not marginal ones.
- Marginal $E(Y | \text{do}(A = a))$ corresponds to randomised trial where covariates X can be /are ignored.

Note: Consistency of effect estimation relies on correctly specified model for $p(y|a, x)$.

- R package `stdReg` (Sjølander and Dahlqwist, 2017)
- The method is special case of **G-formula** for sequential treatments (Robins, 1986).
- Population effect $E(Y \mid \text{do}(A = a))$ depends on *distribution* $p(x)$ of covariates in target population
⇒ not necessarily the same in different populations (e.g. age distribution). If $p(y|a, x)$ regarded as ‘stable’ across populations, then can just replace $\hat{p}(x)$ in the above by different covariate distribution for different populations (e.g. UK versus USA covariate distribution).

The following methods are all based on models for A given X instead of modelling Y given X .



The functionals

$$\sum_x p(y|a, x)p(x) \quad \text{or} \quad \sum_x E(Y|a, x)p(x)$$

might be ‘awkward’, especially if A continuous,
 $E(Y|a, x)$ non-linear with interactions,
or X high dimensional and/or partly continuous.

⇒ Parameterise $E(Y \mid \text{do}(A = a))$ itself?!

⇒ **Marginal structural models (MSM)**

Marginal Structural Models

(Hernán et al, 2001)



MSM: *semiparametric* model for

$$p(y \mid \text{do}(A = a)) \quad \text{or more typically} \quad E(Y \mid \text{do}(A = a))$$

e.g. linear, logistic, CoxPH, loglinear, probit etc.

Marginal: refers to time-varying covariates \rightarrow later

Structural: model under intervention in A (not observational)

Note: term ‘structural’ is used in many different ways — here it always refers to modelling the **underlying causal** relationships.

Inverse Probability Weighting (IPTW)



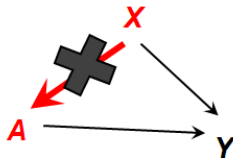
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Inverse Probability Weighting (IPTW)

Idea is based on:

$$\begin{aligned} p(y \mid \text{do}(A = a)) \\ &= \sum_x p(y \mid a, x) p(x) \\ &= \sum_x \frac{p(y, a, x)}{p(a \mid x)} \end{aligned}$$



Inverse Probability Weighting (IPTW)



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$$p(y \mid \text{do}(A = a)) = \sum_x p(y|a, x)p(x) = \sum_x \frac{p(y, a, x)}{p(a|x)}$$

\Rightarrow fit model for $E(Y \mid \text{do}(A = a))$ with weights $w^i = p(a^i|x^i)^{-1}$

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\Rightarrow creates 'pseudo sample' where X is balanced

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⇒ creates ‘pseudo sample’ where X is balanced

⇒ unbiased **estimating equations** for parameters of $E(Y \mid \text{do}(A = a))$.

Here, $w^i = p(a^i|x^i)^{-1}$ is the inverse of the probability that individual i receives ‘treatment’ a^i given they have covariates x^i .

IPTW Estimator

Basic Idea



Simple situation: binary exposure A ;
define $\pi(x) = P(A = 1 | X = x)$.

Can show (under our assumptions):

$$E\left(\frac{A}{\pi(X)}Y\right) = E(Y \mid \text{do}(A = 1))$$

and similarly

$$E\left(\frac{1 - A}{1 - \pi(X)}Y\right) = E(Y \mid \text{do}(A = 0))$$

Proof: iterated conditional expectation (exercise!)
→ see ‘Horvitz–Thompson’ principle

With model $\pi(X; \alpha) \Rightarrow$ plug-in $\pi(X; \hat{\alpha})$

IPTW yields consistent estimator for ACE:

- if $\pi(X; \alpha)$ correctly specified;
- can obtain sandwich standard errors or bootstrap, or theoretical asymptotical standard errors.

IPTW — Implementation



Easy to implement with standard software for regression models by specifying weights:
first, obtain weights, then fit chosen model.

```
p.i <- glm(treat~covar,family="binomial")$fitted  
w.i <- 1/(treat*p.i+(1-treat)*(1-p.i))  
msm <- glm(y.out~treat,family="binomial",weights=w.i)
```

Coefficients:

| | Estimate | Std. Error | z value | Pr(> z) |
|-------------|----------|------------|----------|----------|
| (Intercept) | -2.02055 | 0.01270 | -159.118 | <2e-16 |
| treat | 0.13202 | 0.01753 | 7.532 | 5e-14 |

```
cov.msm <- sandwich(msm)  
sqrt(cov.msm[2,2]) [1] 0.03218826
```

Note: default **standard errors** ignore variability in (estimated!) weights \Rightarrow **sandwich st.error (or: bootstrap)**

- Consistent when both, models for $E(Y \mid \text{do}(A = a))$ and $\pi(x)$ correctly specified.
- If $p(a|x) \approx 0 \Rightarrow$ large weights \Rightarrow use ‘stabilised’ weights, e.g. $w = \tilde{p}(a)/p(a|x)$, where $\tilde{p}(a)$ some distribution for $A \Rightarrow$ more efficient estimators.
- Check assumptions: in weighted population, observed covariates must be ‘balanced’ — e.g. package `cobalt` for balance plots.

Checking Assumptions: Balance

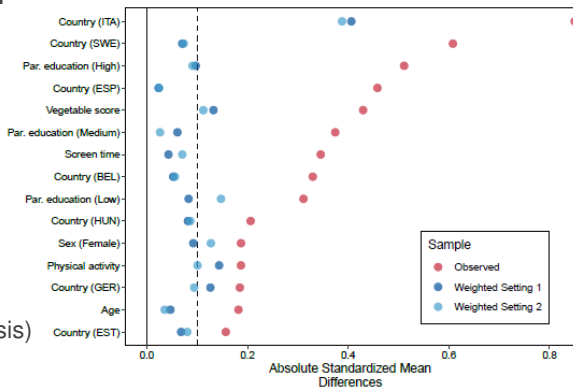
Example: causal effect of 'fibre intake' on children's BMI; large adjustment set (country, parental edu, vege-score, etc.)

Plot: std.mean-diff (abs)

treated / untreated^d

red: unweighted

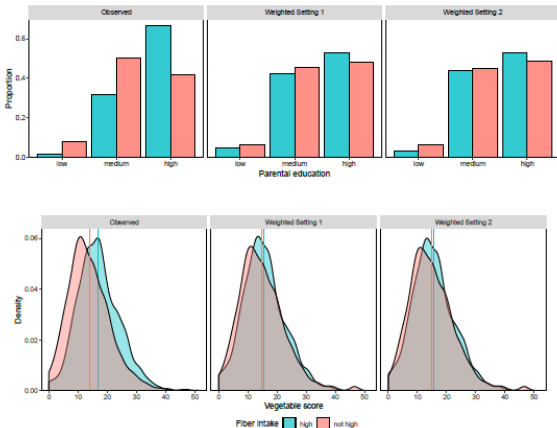
blue: weighted



(Nöhren, 2021:MAthesis)

Checking Assumptions: Balance

Example(ctd.), checking balance of whole distribution of covariates



- MSMs with IPTW mostly used in **longitudinal** situations / **sequential treatments** with time-varying confounding \Rightarrow 'marginal' over time-dependent confounders / covariates.
- IPTW especially useful when study design (or other) supplies background knowledge to model weights $p(a|x)$.
- Software: R package `ipw` for longitudinal data and correct standard errors
- Problem: estimation of weights $p(a|x)^{-1}$ not obvious, but possible, when A continuous.

Continuous Treatments?



Wanted: $E(Y(a))$ or $E(Y \mid \text{do}(a))$ as a function of a

- assume a (semi-)parametric model;
- but non-parametric methods exist

(e.g. Kennedy et al, 2017:JRSSB)

Issues:

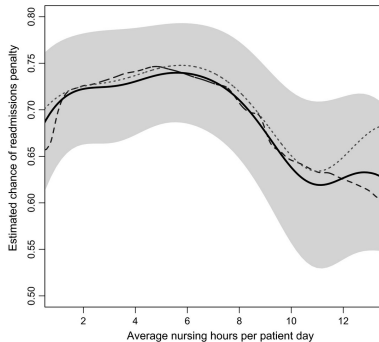
- positivity?
- meaningful interventions?

Example: in hospitals,
 A = nurse staffing hours per day
 Y = excess re-admission

dashed = regr.std

dotted = IPTW

full = “double-robust”



Propensity Score (PS)

(Rosenbaum & Rubin, 1983)



Have used $\pi(x) = P(A = 1|X = x)$

\Rightarrow **propensity score.**

Note: $\pi := \pi(X)$ is random variable.

MSM: used $\pi(X)$ for **weighting**.

But: can also use $\pi(X)$ for **adjustment-type** approaches, due to it being a **balancing score**...

Propensity / Balancing Score



(Still assuming: X sufficient set of covariates; A binary.)

Use of propensity scores (vs. IPTW) is based on

$$A \perp\!\!\!\perp X \mid \pi \quad \text{i.e. } \pi \text{ balances } X$$

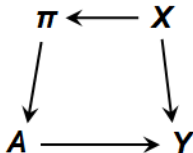
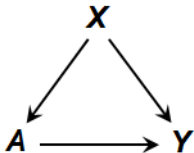
Hence (with properties of X):

$$Y(a) \perp\!\!\!\perp A \mid \pi$$

making π a **minimal sufficient reduction** of X (univariate $\in [0, 1]$).

Propensity Score — Graphically

Propensity score $\pi := \pi(X) = P(A = 1|X)$ satisfies these conditional independencies:



Left: assumption of X being sufficient set of covariates.

Right: π is deterministic function of X and $A \perp\!\!\!\perp X \mid \pi$.

- Estimate propensity score $\hat{\pi}$ with model for $\pi(X; \alpha)$.
- Required: correctly specified model $\pi(X; \alpha)$.
Non-parametric approaches: random forests etc.
- Note: **predictive** quality of $\pi(X; \alpha)$ for A **not important** because need X to be (A, Y) -confounders, not nec. strong predictors of A .
In fact: strong A -predictors \Rightarrow **bias amplification**
(Pearl, 2011).
- Check balancing property (various diagnostics).
- **Check positivity** / overlap \Rightarrow if necessary: prune!

Methods for using PS (other than weighting):

- **PS stratification**: divide into strata (often quintiles) and fit $p(y|a)$ to each stratum separately.
(Strata specific effects can reveal effect modification.)
Then weighted average to obtain overall population effect.

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- Sometimes: **PS adjustment** — specify model for $p(y|a, \pi)$ and fit with $\hat{\pi}$ plugged in.
- Extrapolation is automatically avoided.

Survival of Cancer Patients

Example



US National Cancer Institute's SEER data base; observational study.

Covariates: year of diagnosis, tumor size, geogr. registry, race, marital status

| Propensity score | Treatment | No. | 5-Year-Surv. | Difference |
|------------------|-----------|------|--------------|------------|
| 1st quintile | A | 56 | 85.6% | |
| | B | 1008 | 86.7% | -1.1% |
| 2nd quintile | A | 106 | 82.8% | |
| | B | 964 | 83.4% | -0.6% |
| 3rd quintile | A | 193 | 85.2% | |
| | B | 866 | 88.8% | -3.6% |
| 4th quintile | A | 289 | 88.7% | |
| | B | 978 | 87.3% | 1.4% |
| top quintile | A | 462 | 89.0% | |
| | B | 604 | 88.5% | 0.5% |

Overall estimated (weighted average) $ACE = -0.68$.

From strata specific results: slight suggestion that treatment B is better for those who are more likely to receive it.

- Best with **binary** treatment / exposure.
- PS stratification consistent if $\pi(X; \alpha)$ correctly specified, but can be markedly biased due to residual confounding within strata possible.
- Consistency can be achieved by increasing number of strata when sample size is 'large' or by additional modelling of $E(Y|X = x; \text{do}(A = a))$ within strata.
- PS popular especially for matching: π is 'one-dimensional reduction' of covariates — but at cost of first modelling / estimating $\pi = p(a|x; \alpha)$.
- PS matching / stratification not really suitable for **sequential** treatments.

- Danger: modelling $\pi(X; \alpha)$ may focus on strong predictors of $A \Rightarrow$ can **amplify** bias! \Rightarrow selection of X as adjustment set should be separate process from fitting $\pi(X; \alpha)$.
- **Interpretation** of PS sometimes regarded as difficult compared to actual covariate values.
- Simulations suggest that IPTW use of π superior to stratification. (Lunceford & Davidian, 2004)
- Critique of PS matching: King & Nielsen (2019)

Estimating Causal Effects

Summary (no unobs. conf.)



Given sufficient obs. confounders X (& positivity):

- Traditional: regression adjustment or...
- ... standardise to obtain population effect (g-formula in time-varying context) – underused in practice
- or MSMs fitted by IPTW – easy to use, also with time-varying data – but can be inefficient
- propensity score methods (stratification / matching) – overused?
- Combination of above approaches leads to *doubly robust* estimation procedures
- Should always check positivity / overlap!

Thank You!

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