VI: A Framework for Causal Analysis

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Evaluation

2021

Topics of last week: Multiple regression and modeling probabilities

- Concepts
- Mechanics
- Estimation
- Categorical/ Qualitative x Interactions
- Case Study: Flood-Interaction Effects Causality
- Prediction
- Multiple regression and modeling probabilities LPM
- Logit and Probit
- Goodness of fit

Summary take-away

- Multiple regression are linear models with several x variables.
- May include binary variables and interactions
- Multiple regression can take us closer to a causal interpretation and help make better predictions.

Motivation: Modelling probabilities

- *What are the health benefits of not smoking? Considering the 50+ population, we can investigate if differences in smoking habits are correlated with differences in health status.*

Binary events

- Start with binary events: things that either happen or don't happen captured by binary variable
- How can we model these events?
  - We do not observe 'on average' larger values for y in this case.
- Solution - model instead the probabilities!

$$E[y] = P[y = 1]$$

- The average of a 0–1 binary variable is also the probability that it is one.
  - Frequency (25% of cases) — probability (25% chance)
- Expected value = average probability of event happening
  - Use the same tools, but interpretation is changing!

Section 10

LPM

Linear probability model - LPM

- Modelling probability – regression with *binary dependent variable*.
- *Linear Probability Model (LPM)* is a linear regression with a binary dependent variable

- Differences in average y are also differences in the probability that $y = 1$
- Linear regressions with binary dependent variables show
  - differences in expected y by x, is also differences in the probability of $y = 1$ by x.
- Introduce notation for probability:

$$y^P = P[y = 1|x_1, x_2, \dots]$$

- Linear probability model (LPM) regression is

$$y^P = \beta_0 + \beta_1 x_1 + \beta_2 x_2$$

Linear probability model - interpretation

$$y^P = \beta_0 + \beta_1 x_1 + \beta_2 x_2$$

- y^P denotes the probability that the dependent variable is one, conditional on the right-hand-side variables of the model.
- β_0 shows the probability of y if all x are zero.
- β_1 shows the difference in the probability that $y = 1$ for observations that are different in x_1 but are the same in terms of x_2.
- Still true: average difference in y corresponding to differences in x_1 with x_2 being the same.

Linear probability model - modelling

- Linear probability model (LPM) using OLS.
- We can use all transformations in x, that we used before:
  - Log, Polinomials, Splines, dummies, interactions, ect.
- All formulae and interpretations for standard errors, confidence intervals, hypotheses and p-values of tests are the same.
- Heteroskedasticity robust error are essential in this case!

Predicted values in LPM

- Predicted values - $\hat{y}^P$ - may be problematic, calculated the same way, but to be interpreted as probabilities.

$$\hat{y}^P = \hat{\beta}_0 + \hat{\beta}_1 x_1 + \hat{\beta}_2 x_2$$

- Predicted values need to be between 0 and 1 because they are probabilities
- But in LPM, they may be below 0 and above 1. No formal bounds in the model.
  - With continuous variables that can take any value (GDP, Population, sales, etc), this could be a serious issue
  - With binary variables, no problem ('saturated models')
- Problem if goal is prediction!
- Not a big issue for inference $\rightarrow$ uncover patterns of association.
  - But note in theory it may give biased estimates...

Does smoking pose a health risk?

The question of the case study is whether, and by how much less likely smokers are to stay healthy than non-smokers.

- focus on people of age 50 to 60 who consider themselves healthy
- ask them four years later as well

Research question: Does smoking lead to deteriorating health?

Data

- $y = 1$ if person stayed healthy
- $y = 0$ if person became unhealthy
- Data comes from SHARE (Survey for Health, Aging and Retirement in Europe)
  - 14 European countries
  - Demographic information on all individual
  - 2011 and 2015 participants are used
  - Being healthy means to report "feeling excellent" or "very good"
  - $N = 3,109$

LPM

Start with a simple univariate model with being a smoker.

$$stays\ healthy^P = \alpha + \beta smoker$$

Both dependent and independent models are using only dummy variables.

Estimated β is -0.072

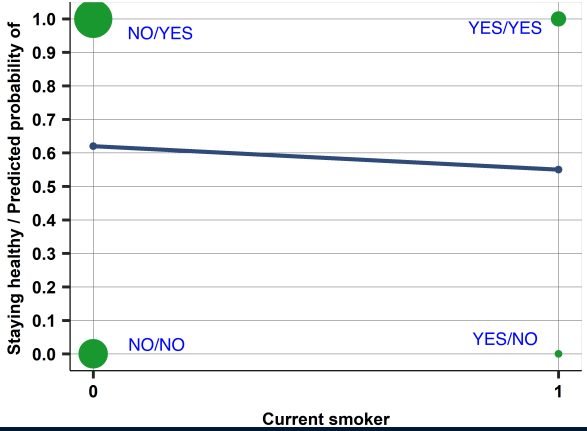
Can we draw a scatterplot?

Table: Probabilities

	Healthy: Yes	Healthy: No
Smoker: Yes	Case 1 : YY	Case 2 : YN
Smoker: No	Case 3 : NY	Case 4 : NN

Scatterplot

Figure: Staying healthy - scatterplot and regression line



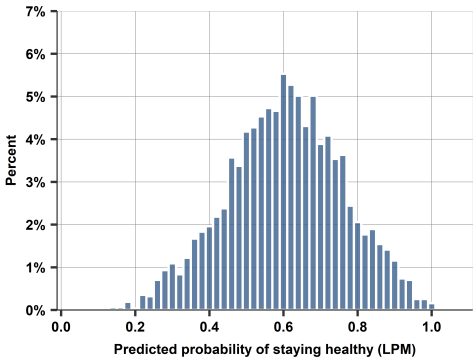
LPM Interpretation

- The coefficient on smokes shows the difference in the probability of staying healthy comparing current smokers and current nonsmokers.
- Current smokers are 7 *percentage points* less likely to stay healthy than those that did not smoke.
- Can add additional controls to capture if quitting matters.

LPM's predicted probabilities

- Predicted probabilities are calculated from the extended linear probability model.
- Predicted probability of staying healthy from this linear probability model ranges between 0.036 and 1.011
  - LPM means it can be below 0 or above 1...
  - Here, only marginally above 1

Histogram of the predicted probabilities



Source: `share-health` dataset.

Compare predicted probability distribution

- Drill down in distribution:
  - Looking at the composition of people: top vs bottom part of probability distribution
  - Look at average values of covariates for top and bottom 1% of predicted probabilities!

Top 1% predicted probability:

- no current smokers, women,
- avg 17.3ys of education, higher income
- BMI of 20.7, and 90% of them exercise.

Bottom 1% predicted probability:

- 37.5% current smokers, 63% men
- 7.6 years of education, lower income
- BMI of 30.5, 19% exercise

Section 11

Logit and Probit

Probability models: logit and probit

- Prediction: predicted probability need to be between 0 and 1

- For prediction, we use non-linear models
- Relate the probability of the $y = 1$ event to a nonlinear function of the linear combination of the explanatory variables -> 'Link function'
  - Link function is some $F(\cdot)$, s.t. $F(y)$ may be used in linear models.

- Two options: Logit and probit – different link function
  - Resulting probability is always strictly between zero and one.

Link functions I.

The logit model has the following form:

$$y^P = \Lambda(\beta_0 + \beta_1 x_1, \beta_2 x_2 + ...) = \frac{exp(\beta_0 + \beta_1 x_1, \beta_2 x_2 + ...)}{1 + exp(\beta_0 + \beta_1 x_1 + \beta_2 x_2 + ...)}$$

where the link function $\Lambda(z) = \frac{exp(z)}{1+exp(z)}$ is called the *logistic function*.

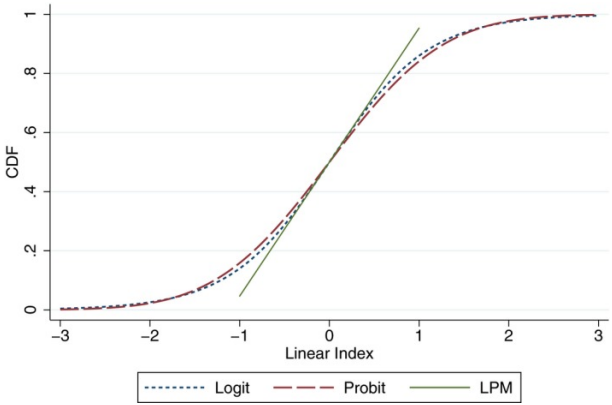
The probit model has the following form:

$$y^P = \Phi(\beta_0 + \beta_1 x_1 + \beta_2 x_2 + ...)$$

where the link function $\Phi(z) = \int_{-\infty}^{z} \frac{1}{\sqrt{2\pi}} exp\left(-\frac{z^2}{2}\right) dz$, is the cumulative distribution function (CDF) of the standard normal distribution.

Link functions II.

- Both Λ and Φ are increasing S-shape curves, bounded between 0 and 1. (Y here is $\Lambda(z)$ and $\Phi(z)$
- Plotted against their respective "z" values. (Here -3 to 3)
- Small difference (indistinguishable) - logit less steep close to zero and one = thicker tails than the probit.
- In our models, 'z' is a linear combination of β coefficients and x-s. The parameter estimates are typically different in probit vs logit.



Logit and probit interpretation

- Both the probit and the logit transform the $\beta_0 + \beta_1 x_1 + ...$ linear combination using a link function that shows an S-shaped curve.
- The slope of this curve keeps changing as we change whatever is inside.
  - The slope is steepest when $y^P = 0.5$;
  - it is flatter further away; and it becomes very flat if y^P is close to zero or one.
- The difference in y^P that corresponds to a unit difference in any explanatory variable is not the same.
  - You need to take the partial derivatives. It depends on the value of x
- Important consequence: no direct interpretation of the raw coefficient values!

Marginal differences

- Link functions makes variation in association between x and y^P – for logit and probit models, we do not interpret raw coefficients!
- Instead, transform them into 'marginal differences' for interpretation purposes
- The marginal difference for x is the average difference in the probability of $y = 1$, that corresponds to a one unit difference in x.
  - Software may call them 'marginal effects' or 'average marginal effects' or 'average partial effects'.
- Marginal differences have the exact same interpretation as the coefficients of linear probability models.

Maximum likelihood estimation

- When estimating a logit or probit model, we use 'maximum likelihood' estimation.
  - You specify a (conditional) distribution, that you will use during the estimation.
    - This is logistic for logit and normal for probit model.
  - You maximize this function w.r.t. your β parameters $\rightarrow$ gives the maximum likelihood for this model.
    - No closed form solution $\rightarrow$ need to use search algorithms.
- The maximum value for this function ℓ is then used for model comparisons (e.g. for Pseudo R^2)

Coefficient results for logit and probit

Dep.var.: stays healthy	(1) LPM	(2) logit coeffs	(3) logit marginals	(4) probit coeffs	(5) probit marginals
Current smoker	-0.061*	-0.284**	-0.061**	-0.171*	-0.060*
	(0.024)	(0.109)	(0.023)	(0.066)	(0.023)
Ever smoked	0.015	0.078	0.017	0.044	0.016
	(0.020)	(0.092)	(0.020)	(0.056)	(0.020)
Female	0.033	0.161*	0.034*	0.097	0.034
	(0.018)	(0.082)	(0.018)	(0.050)	(0.018)
Years of education (if < 8)	-0.001	-0.003	-0.001	-0.002	-0.001
	(0.007)	(0.033)	(0.007)	(0.020)	(0.007)
Years of education (if $>= 8$ and < 18)	0.017**	0.079**	0.017**	0.048**	0.017**
	(0.003)	(0.016)	(0.003)	(0.010)	(0.003)
Years of education (if $>= 18$)	-0.010	-0.046	-0.010	-0.029	-0.010
	(0.012)	(0.055)	(0.012)	(0.033)	(0.012)
Income group	0.008*	0.036*	0.008*	0.022*	0.008*
	(0.003)	(0.015)	(0.003)	(0.009)	(0.003)
Exercises regularly	0.053**	0.255**	0.055**	0.151**	0.053**
	(0.017)	(0.079)	(0.017)	(0.048)	(0.017)
Age, BMI, Country	YES	YES	YES	YES	YES
Observations	3,109	3,109	3,109	3,109	3,109

Does smoking pose a health risk?– logit and probit

- LPM – interpret the coefficients.
- Logit, probit - Interpret the *marginal differences*. Basically the same.
  - Marginal differences are essentially the same across the logit and the probit.
  - Essentially the same as the corresponding LPM coefficients.
- Happens often:
  - We could not know which is the "right model" for inference
  - Often LPM is good enough for interpretation.
  - Check if logit/probit very different.
    - Investigate functional forms if yes.

Section 12

Goodness of fit

Goodness of fit measures

- There is no comprehensively accepted goodness of fit measure...
  - This is because we do not observe probabilities only 1 and 0...
- R-squared is not the same meaning as before
  - Evaluating fit for probability models, we compare predictions that are between zero and one to values that are zero or one.
  - But predicted probabilities would not fit the zero-one variables, so we'd never get it right.
- R-squared less natural measure of fit, but we can calculate it as usual.
  - *But*: R-squared can not be interpreted the same way we did for linear models.

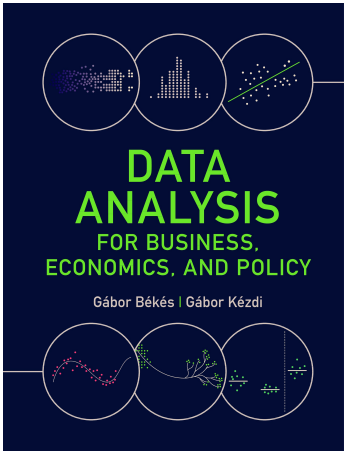
Probability models summary

- Find patterns with ease when y is binary - model probability with regressions
- Linear probability model is mostly good enough, easy inference.
  - Predicted values could be below 0, above 1
- Logit (and probit) - better when aim is prediction, predicted values strictly between 0-1
- Most often, LPM, logit, probit - similar inference
  - Use marginal (average) differences
- No trivial goodness of fit. Brier score or pseudo-R-Squared.
- Calibration is useful diagnostics tool: well-calibrated models will predict a 20% chance for events that tend to happen one out of five cases.

THIS WEEK's LECTURE

Overview

Slideshow for the Békés-Kézdi Data Analysis textbook



- Cambridge University Press, 2021
- gabors-data-analysis.com
  - Download all data and code: gabors-data-analysis.com/data-and-code/
- This slideshow is for Chapter 19

Causal questions

- How does having a major industrial investment affect house prices?
- Do vitamins have a beneficial health effect?
- Does better management yield greater revenues?
- Does a better diet makes you live longer?
- Does a merger between very large companies cause prices to rise?

Measuring causality require intervention and variation

- causality requires the presence of a possible intervention
  - Eating / not eating a food item
  - Replacing / educating managers
- Causality also requires variation
- How does taking vitamins effect health?
  - We need people who take and people who do not take vitamins.
- Does better management yield greater revenues?
  - We need firms to have a variation in the quality of management.

This lecture

- Heavy on vocabulary
  - Please read the running example on advertising in the book
    - Case study quickly sketched in lecture, more details in book

The setup: Intervention, treatment, subjects, outcomes

- Intervention describes a decision that aims changing the behavior or situation of people, firms. Also called Treatment.
- Subjects of an intervention are those that may be affected. Treated or untreated.
- Outcome variables, or outcomes, are variables that may be affected by the intervention.
- Causal variables, or treatment variables are the variables that indicate the intervention.
- Need idea why the intervention may affect an outcome variable. Mechanisms by which an intervention exerts an effect on a particular outcome variable or variables.
  - Other names for mechanisms: pathways or mediator variables

The causal question

Most important elements of a precise causal question are

- What's the outcome (Y) variable?
- What's the causal (X) variable?
  - The causal variable may be a binary variable (intervention takes place or not) or a quantitative variable (amount of intervention).
- What are the subjects (the outcome for whom?)
- What is the specific intervention (who, and how, would manipulate the cause to alter the outcome?)
- What is or could be the mechanism (why should one expect an effect of the intervention on the subject?).

Potential outcomes framework

- Potential outcomes framework is a structure to study causal questions.
- Thinking in this framework will make defining the effect of an intervention straightforward.
- The outcome variable Y, may be
  - Binary: whether an individual buys the product or not
  - Quantitative: the sales value of a house.

Potential outcomes framework

- Binary interventions: subjects may be either treated or untreated.
  - The outcome may be anything, including binary or multi-valued variables.
- Can always think about two potential outcomes for each subject:
  - what their outcomes would be if they were treated (their treated outcome),
  - what their outcomes would be if they were untreated (their untreated outcome).

Potential outcomes framework

- Of these two potential outcomes, each subject will experience only one: that's their observed outcome.
  - Treated subject: Observed outcome = their treated outcome.
  - Not treated subject: Observed outcome = their untreated outcome.
- The other potential outcome, unobserved, is their counterfactual outcome
  - what could have been observed had the subject experienced what did not happen.

Potential outcomes framework

- Each subject has two potential outcomes before the intervention, both unobserved.

- Then each subjects gets assigned to be treated or untreated.
- The intervention reveals one of their potential outcomes, the one that conforms their assignment.
- Their other potential outcome remains unobserved = counterfactual outcome.

The Individual Treatment Effect

- The individual treatment effect for subject i is the difference between their two potential outcomes: the value of the potential treated outcome for the subject minus the value of the potential untreated outcome:

$$te_i = y_i^1 - y_i^0 \tag{1}$$

  - $y_i =$ observable outcome
  - $y_i = y_i^1$ for subjects that end up being treated
  - $y_i = y_i^0$ for subjects that end up being not treated

Individual treatment effects

- te_i= the value of the treated outcome for the subject minus the value of the untreated outcome for the same subject i.
- te_i may be 0, positive or negative
- Consider binary outcomes (0 or 1), so the ITE=[0,-1,1].
  - $te_i = 1$ if the treated outcome is one and the untreated outcome is zero.
  - $te_i = -1$ if the treated outcome is zero and the untreated outcome is one.
  - $te_i = 0$ if both the treated outcome and the untreated is one, or both of them is zero.

Individual treatment effects

- Individual treatment - think cause and effect without observing them.
- The individual treatment effect is never observable.

- There is no way to know
  - what the outcome of untreated subjects would have been if they were treated,
  - what the treated outcome of untreated subjects would have been.
- Thus, data analysis cannot uncover individual treatment effects by simply observing them.

Heterogeneous treatment effects

- Individual treatment effects will vary, of course.
- For instance, vary across groups
  - Men vs women
  - Small vs large markets
- The possibility of effects being different across subjects = the possibility of heterogeneous treatment effects.
- Can't observe te_i – will not know if indeed heterogeneous among the subjects we care about.
- For some groups, we can actually look at it

Average treatment effect

- Instead of te_i, we can observe the average
- The average treatment effect, abbreviated as ATE, is the average of the individual treatment effects across all subjects.
- For binary outcomes, average outcomes are probabilities and average treatment effects are differences in probabilities.

ATE as average / expected ITE

- ATE is the expected (=average) difference between potential outcomes
  - Expectation operator ($E[]$)

$$ATE = E[te_i] = E[y_i^1 - y_i^0] \tag{2}$$

- The average of the differences is equal to the difference of the averages.
- Thus the average treatment effect is also the difference between the average of potential treated outcomes and the average of potential untreated outcomes:

$$ATE = E[y_i^1] - E[y_i^0] \tag{3}$$

Average treatment effect

- Think of the average treatment effect when they talk about the effect of an intervention.
- *ATE* can be viewed as the expected effect of the intervention for a subject randomly chosen from the population.
- *ATE* gives the total effect of the intervention if multiplied by the size of the population

Average Effects in Subgroups and ATET

- It is possible to get good estimates of average effects, at least under the right circumstances.
- Heterogeneity may be hidden behind the ATE.
- Consider $ATE = 0$:
  - all individual treatment effects are all zero.
  - the intervention has positive effects on some subjects and negative effect on other subjects but those cancel out.
- Any value may conceal a division of groups of subjects with very high and low effect.

Average Effects in Subgroups and ATET

- ATE = average of te_i across all subjects in the population that we defined.
- We can also calculate the ATE for subgroups

- One such subgroup is the treated group
- ATET = the average treatment effect on the treated - all subjects that end up being treated.
- ATET sometimes equals ATE, but other times it does not
- In some applications, we can calculate ATET only.

ATE when Quantitative Causal Variables

- Examples of interventions that lead to quantitative causal variables
  - setting prices of products or services;
  - deciding on the budget to be spent on advertising through a social media platform.
- PO framework - designed binary interventions.
- Concepts apply to quantitative causal variables
- But more complicated

Quantitative Causal Variables

- A quantitative causal variable - the intervention is not binary (happens to you or not), but the effect size varies by subject
- Many individual treatment effects beyond (0,1).
- Many potential outcomes for each subject (beyond -1,0,1)

ATE and Quantitative Causal Variables

- Quantitative causal variables lead to not one individual treatment effect but a series of them,
- One more step: average individual treatment effect beforetaking the average across subjects for ATE.
- Difficult to think about average effects of quantitative causal variables.
- But the idea is fundamentally the same.

- Often use quantitative variable and create a binary: low vs high

Ceteris Paribus: Other Things Being the Same

- What we really mean by potential outcomes.
- The difference between treated and untreated outcome is the intervention and only the intervention.
- All other things that may affect the outcome variable are the same.
  - Those other relevant things are things that may cause the outcome variable to change besides the intervention.
- "all other (relevant things) being the same" = "ceteris paribus".

Ceteris paribus vs multivariate regression

- Remember Chapter 10, with outcome y, causal variable x

$$y^E = \beta_0 + \beta_1 x + \beta_2 z \tag{4}$$

- In regression we condition on z
- Compare two observations that have the same z but are different in x by one unit. The observation with a one unit higher x is expected to have β_1 units higher y.

Ceteris paribus vs multivariate regression

- Can we condition on all potential confounders in regression?
- That would be ceteris paribus analysis
- Probably not
  - We can include only what we observed in data
  - We can be rarely sure that there are no confounders among what's not observed in data
  - How do we know that we controlled for everything relevant?
- So, in a regression, we compare observations that differ in x and are same in all other RHS variables that we observe and include in the regression

Average treatment effect

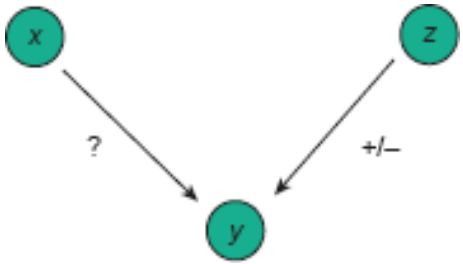
- How to calculate ATE - main issue for this course
- Because te_i cannot be calculated and averaged
- Because ceteris paribus exists as a theoretical concept and need to work hard to get close

Causal maps (DAGs) to uncover causal structure

- Causal maps: key tool to think about causality
- A causal map is a graph that connects variables (nodes) with arrows (directed edges).
- The arrows represent effects.

Causal maps: simplest case

- An example with x causing y, but also a variable z causing y.
- When an outcome variable is caused by the intervention of interest (x) but also other variables like z
- On this graph x and z are unrelated

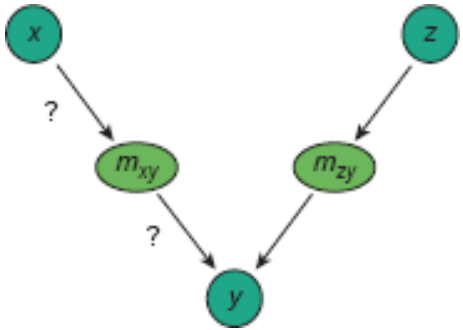


Causal maps to uncover causal structure

- Our aim: summarizing our assumptions about how variables affect each other.
- A causal map is a graph that connects variables (nodes) with arrows (directed edges).
- The arrows represent effects.
- Causal maps help understand whether and how we can uncover the effect we are after.
- Another name for causal map is directed acyclic graphs, DAG - graph of nodes and arrows.

DAG: mechanisms

- Add variables that measure the mechanisms (m) through which x and z affect y.
- m_{zx} = through which x affects y
- m_{zy} = through which z affects y.



Comparing Different Observations to Uncover Average Effects

- PO, DAG frameworks - think more precisely about the effect we want to measure.
- But: te_i cannot be measured
- = Counterfactual outcome ("what would have been") is never observed

- What is observable are:
- The potential treated outcome (y_i^1) for subjects treated.
- The potential untreated outcome (y_i^0) for subjects not treated.

Comparing Different Observations to Uncover Average Effects

- Uncover average potential outcomes from the average observable outcome IF two good approximations.
  - Average of the observed outcomes for treated subjects ($E[y_i|$ i is treated]) $\approx$ the average of the potential treated outcomes across all subjects.
  - Average of the observed outcomes for untreated subjects ($E[y_i|$ i is not treated].) $\approx$ the average of the potential untreated outcomes across all subjects.

$$E[y_i|\text{ i is treated }] \overset{?}{\approx} E[y_i^1] \tag{5}$$

$$E[y_i|\text{ i is not treated }] \overset{?}{\approx} E[y_i^0] \tag{6}$$

Comparing Different Observations to Uncover Average Effects

- Message: Data helps uncover ATE the closer observed groups represent theoretical concepts of PO.

Random assignment

- How can we get data where these assumptions would hold?
- The random assignment condition = assignment is independent of potential outcomes
  - whichever subject ends up being treated or untreated is independent of their potential outcomes
- Random assignment == independence of potential outcomes.
  - Not about how the data was collected (unfortunate name)

Random assignment and ATE

- Independence makes sure that treated and untreated groups are similar in terms of their potential outcomes, on average (on average = in expectation).
- And this means leads to a simple way to get a good estimate for the average treatment effect (ATE).

- So: if assignment is random, the difference between average observed outcomes of treated versus untreated subjects is a good estimate of ATE.
- Importantly, random assignment is a theoretical concept
- In practice, it is an aspiration to get close to, to get good estimate of ATE.

Random assignment, ATE and ATET

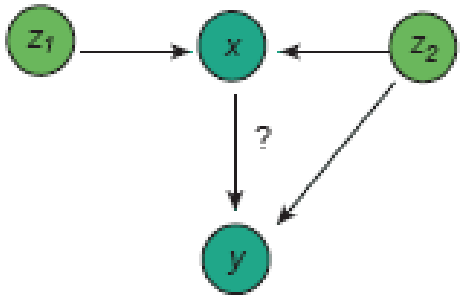
- Random assignment: observed difference is good estimate of ATE as well as ATET.
- Because, in this case, ATE and ATET are equal.
- Random assignment makes sure that those who end up being treated are no different in terms of their potential outcomes than the entire population.

Sources of Variation in the Causal Variable

- Sources of variation in the causal variable - thinking task
- An endogenous source of variation is when the source of variation in x is also related to y.
- An exogenous source of variation is when a source of variation that affects x is independent of y.

An exogenous and an endogenous source of variation in x

- Assumption 1: z_1 is an exogenous source of variation in x;
- Assumption 2: z_2 is an endogenous source of variation in x.



Sources of Variation in the Causal Variable

- Random assignment and exogeneity in the source of variation are close concepts.
- When assignment is random, there are only exogenous sources of variation in x.
- When assignment of x is not random, there are likely to be endogenous and exogenous sources of variation

Good and bad sources

- For the question of the effect of x on y, we need to assess all things that may make x vary across observations, and then divide them into
  - good ones (exogenous) and
  - bad ones (endogenous).
- To uncover the effect we'll need to keep the good ones and get rid of the bad ones.
- Next bits + most of the course is about how to do that.

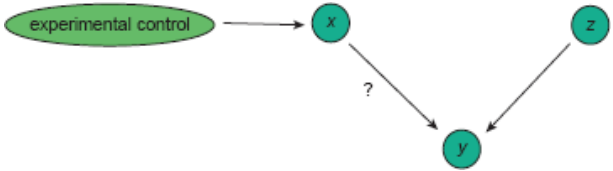
Experimenting versus Conditioning: 1 Controlled experiments

- Controlled experiments allows for controlling variation in the causal variable
- Variation in the causal variable x is controlled by assigning values of x to the observations.
- The intervention is hence done by the analyst

- This practice is called controlled assignment.
  - attempts to make sure that the value of x observations "receive" is not affected by the decisions of people who may be interested in the outcome.
  - It can also help avoid reverse causality by not letting the outcome y affect x in any way.
- If binary treatment x variable observations are assigned to a treated and an untreated ("control") group by the analyst.

Controlled experimental variation in x

- Experimental control is the only source of variation in x.
- Other variables,summarized by z, may affect y but are unrelated to x.



Experimenting versus Conditioning

- Sometimes controlled experiments are impossible, impractical, or would produce uninformative results,
- This is when data analysts will have to resort to using observational data.

Experimenting versus Conditioning: 2 Natural experiments

- In natural experiments - may assume that variation in x in observational data is exogenous,
  - ... as if it came from a controlled experiment.
- Natural experiments do not have experimenters who assign treatment in a controlled way.
- Assume that assignment in a natural experiment took place as if it were a well-designed controlled experiment.

- Key is indeed exogenous variation in x
- Example: Natural disasters, geography

Experimenting versus Conditioning: 3 Conditioning

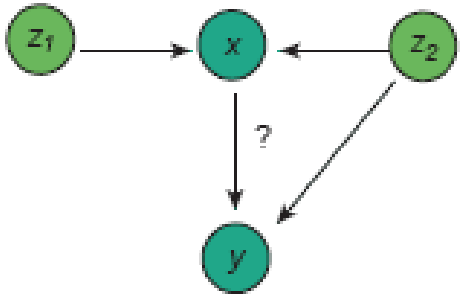
- Most often, no natural experiment situation
- Conditioning on endogenous sources of variation in the causal variable.
  - conditioning on the values of variable z when comparing the values of y by values of x.
- Let exogenous sources vary AND, not let endogenous sources vary.
- Comparing observations that are different in terms of exogenous sources of variation in x, while having similar values for the variables that are endogenous sources of variation.
- Why need difference in exogenous sources of variation in x?
- Conditioning = isolating exogenous sources of variation in x

Confounders in Observational Data

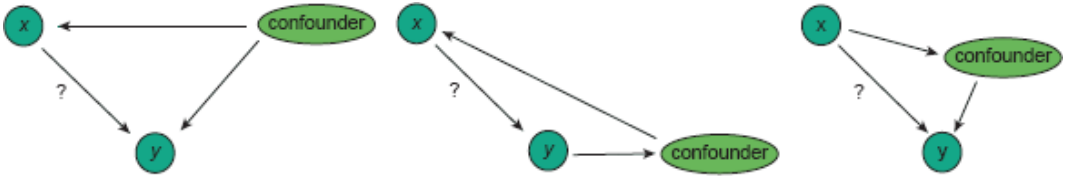
- Confounding variables (confounders) in observational data
  - endogenous sources of variation in a causal variable
- The key issue to think about when doing causal analysis with observational data

Confounders in Observational Data

- z_2 is an endogenous source of variation in x.
- Makes y and x correlated even though x not cause y and y not cause x.



Three types of confounders



(a) Common cause confounder

(b) Mechanism of reverse causality

(c) Unwanted mechanism confounder

Common cause confounder

- When we speak of confounders we often mean common cause confounders
- z affects y
- z also affects x
- Examples could be income, education affecting several choices and conditions of people

Mechanism of reverse causality

- The outcome variable y itself may affect the causal variable x: reverse causality.
- Here y affects x when, instead, we are interested in the effect of x on y.
- This reverse causality operates via the mechanism of z. Thus, here z is the mechanism of reverse causality.
- Example, if sales are going down the management of the firm may want to reverse that negative trend by advertising more.

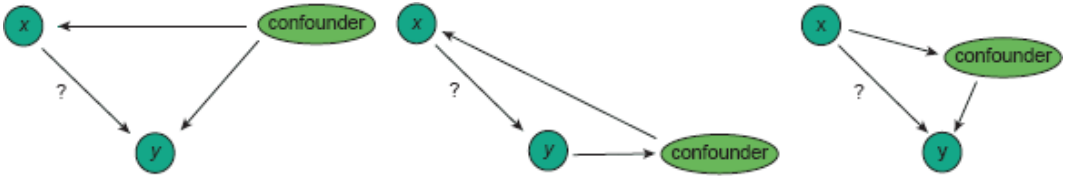
Reverse causality

- Even more complicated: feedback loop
- That may induce feedback loops: x affecting y, then y affecting x in turn, and so forth.
- Positive feedback loops reinforce the original effect of x; negative feedback loops diminish its effect.

Unwanted mechanism

- The third type of confounder is an unwanted mechanism confounder: a mechanism through which x affects y, but one that we want to exclude.
- Not actually a source of variation in x, but we want to condition on it nevertheless.
- It could be a mechanism of selection, that we want to exclude
  - Hard, more later...

Three types of confounders (repeated)



(a) Common cause confounder

(b) Mechanism of reverse causality

(c) Unwanted mechanism confounder

Confounders in practice: Selection

- In business, economics and policy applications most confounder variables represent some kind of selection.
- Self-selection - when subjects themselves decide on whether they are treated or not (with binary x), and that decision is related to confounder variable z that affects the outcome y as well.
  - or what level of the causal variable they get (with multi-valued x),
- Could be common cause or unwanted mechanism

From Latent Variables to Measured Variables

- From Causal map to data: latent and missing variables
- Causal map to data: two problems: (1) hard to measure, (2) not available.

- Confounders that we want to condition on are not directly measurable = latent variables.

- Variables in real data are often imperfect measures of the latent variables that we want to consider.
- Real data rarely includes variables that measure all of the confounders.

Omitted variable bias

- Failing to condition on some of the confounders, or conditioning on imperfect measures of them, leads to a biased estimate of the effect.
- This is the Omitted variable bias

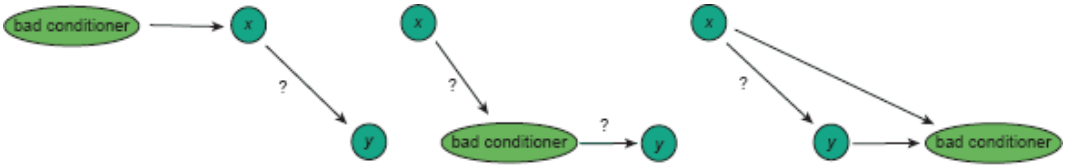
The three types of bad conditioning variables

- There are variables that we should not condition on when trying to estimate the effect of x on y. Bad conditioning variables.

- exogenous source of variation in the causal variable x.
- part of the mechanism by which x affects y – that is of course if we want to include that mechanism in the effect we want to uncover
- collider variable: a common effect, or common consequence, of both x and y;

- How to know if we should condition on a variable or not?
- Analyst must think and decide
- Causal map (DAG) helps

The three types of bad conditioning variables



(a) Exogenous source of variation

(b) Mechanism variable

(c) Common consequence (collider variable)

The three types of bad conditioning variables

- exogenous source of variation in the causal variable x.
- part of the mechanism by which x affects y – that is of course if we want to include that mechanism in the effect we want to uncover
- collider variable: a common effect, or common consequence, of both x and y;

- If you believe you have such variables, do NOT add them to a regression

Comparing pros and cons of approaches

- Causality can be established
  - Controlled experiment = great confidence
  - Natural experiment = good confidence, but work is needed to prove it
  - Conditioning on confounders = never be certain.
- This is about internal validity
  - The extent of which we can be certain that indeed, we uncovered a causal relationship

External validity

- However, there is another aspect
- External validity is measure of confidence about generalization
  - Will the causal relationship work in the future
  - Will the causal relationship work in other markets, countries
- Key issue throughout the course is discussing internal and external validity
  - Often a trade-off

Constructive skepticism

- No analysis is perfect
  - Weigh pros and cons of different approaches
- One can still learn from a well-designed analysis
  - Be that a controlled experiment or an observational study
- Solid knowledge from many studies
  - With different approaches
  - Pointing to similar conclusion if biases well understood
    - some studies mar be more biased than others
    - Need to take into account when summing up evidence from multiple studies

Case study: Food and health: data

- You are what you eat
- causal statement: some kinds of food make you healthier than other kinds of food.
- Does eating more fruit and vegetables help us avoid high blood pressure?

- Case study briefly in lecture, please read details

Case study: Food and health

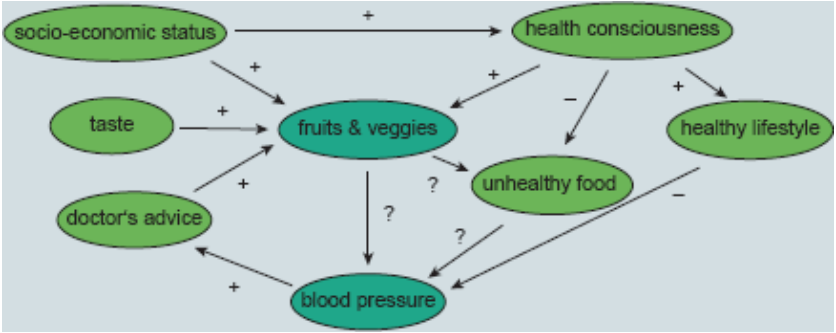
- The food-health dataset we use comes from the National Health and Nutrition Examination Survey (NHANES) in the United States.
- The amount of fruit and vegetables consumed per day and blood pressure
  - Measured by an interview that asks respondents to recall everything they ate in two days.
- Blood pressure is sum of systolic and diastolic measures.
- Fruit and vegetables is the amount consumed per day (g)
- Source: food-health dataset, USA,
- ages 30–59, 2009–2013. N=7358.

Case study: Food and health – descriptive statistics

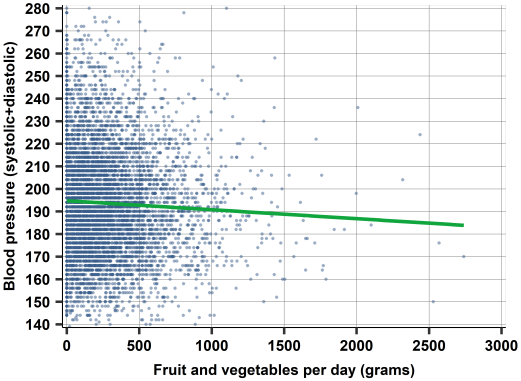
	Mean	Median	Std.Dev.	Min	Max	Obs
Blood pressure (systolic+diastolic)	194	192	24	129	300	7359
Fruit and vegetables per day, grams	361	255	383	0	3153	7359

Source: food-health dataset, USA, ages 30 to 59, 2009–2013.

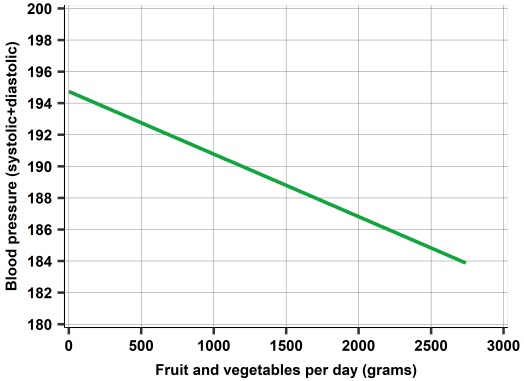
Case study: A causal map - effect of fruit and vegetables on blood pressure



Case study: Food and health- Correlation

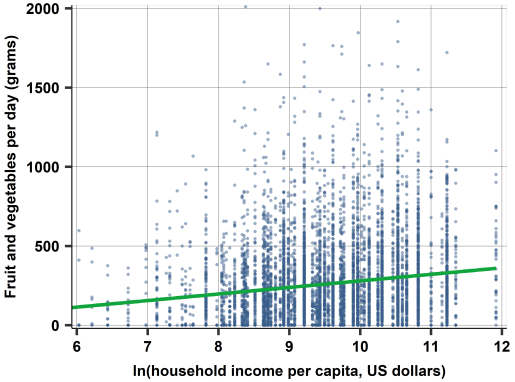


Scatterplot and regression line

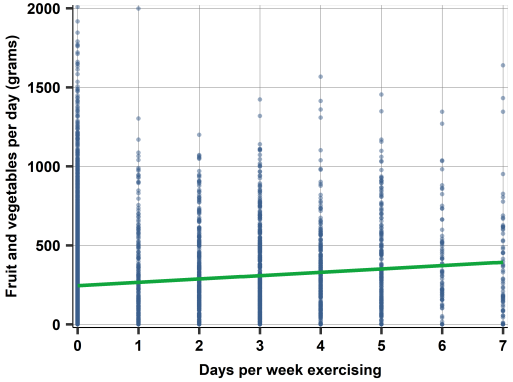


Regression line only

Case study: Food and health- two sources of variation in eating veggies



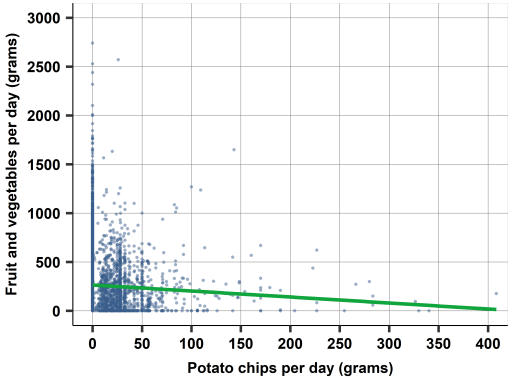
Log household income and amount fruit + vegetables



Days/week exercising and amount fruit + vegetables

Case study: Food and health- Consumption of an unhealthy food item

- Chips consumption. Should we condition on?
- Yes. chips eating is a common cause. Chip eating signal unhealthy diet could affect chance of veggies and health
- No. A potential bad conditioning variable: Veggie eating causes less chips that causes better health. Unwanted mechanism.



Summary

- Food and health correlated
- Many potential confounders
- Never be really causal
- But can offer insight and prompt experiments
- Can be informative - more likely causally true than not.