Causal Inference

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IPL
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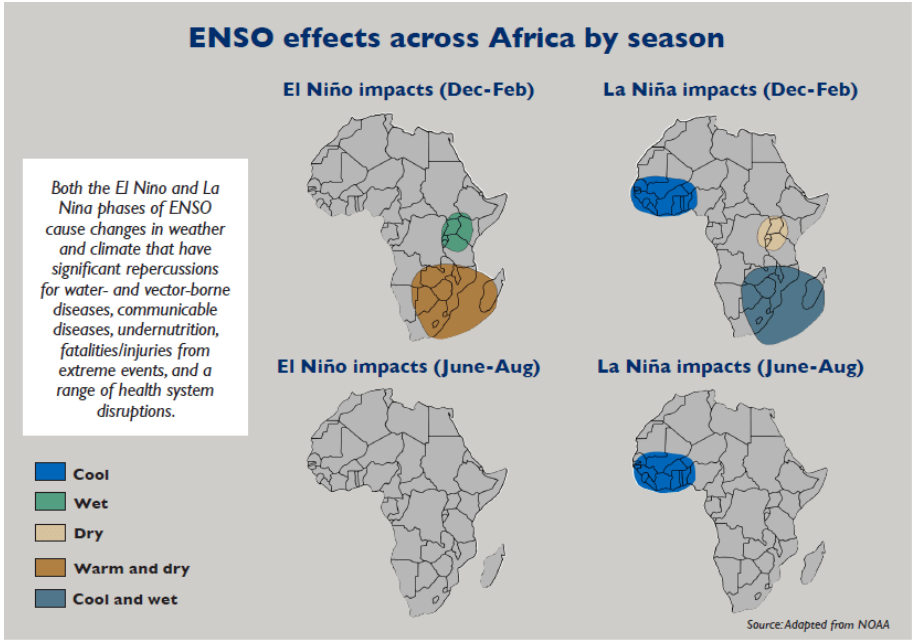
Causal Inference

- Estimate the strength of the effect of ENSO on vegetation greenness



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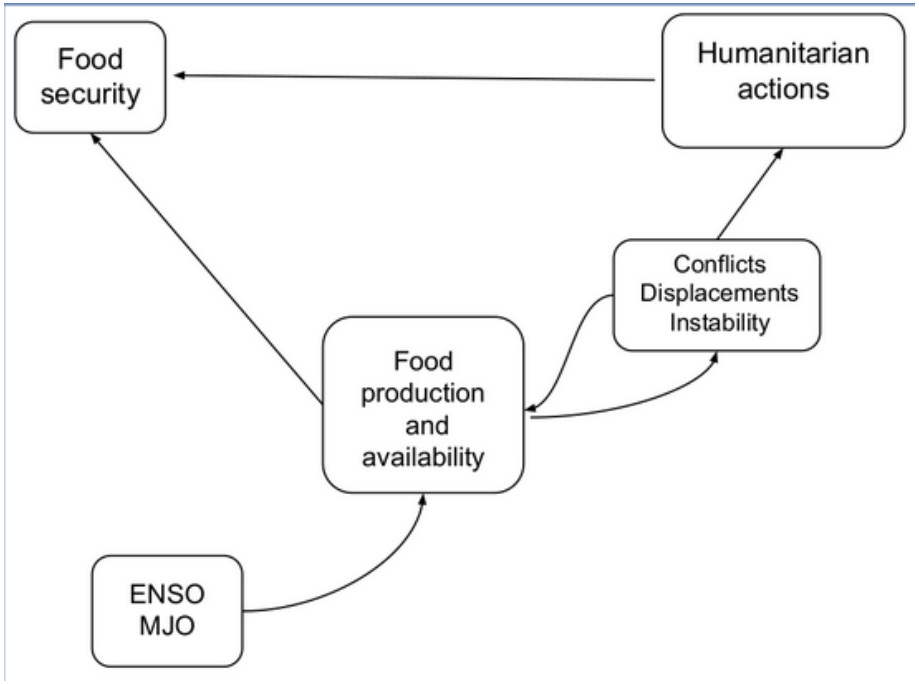
- Estimate the strength of the effect of ENSO on vegetation greenness
- How effective are humanitarian actions to fight food insecurity?



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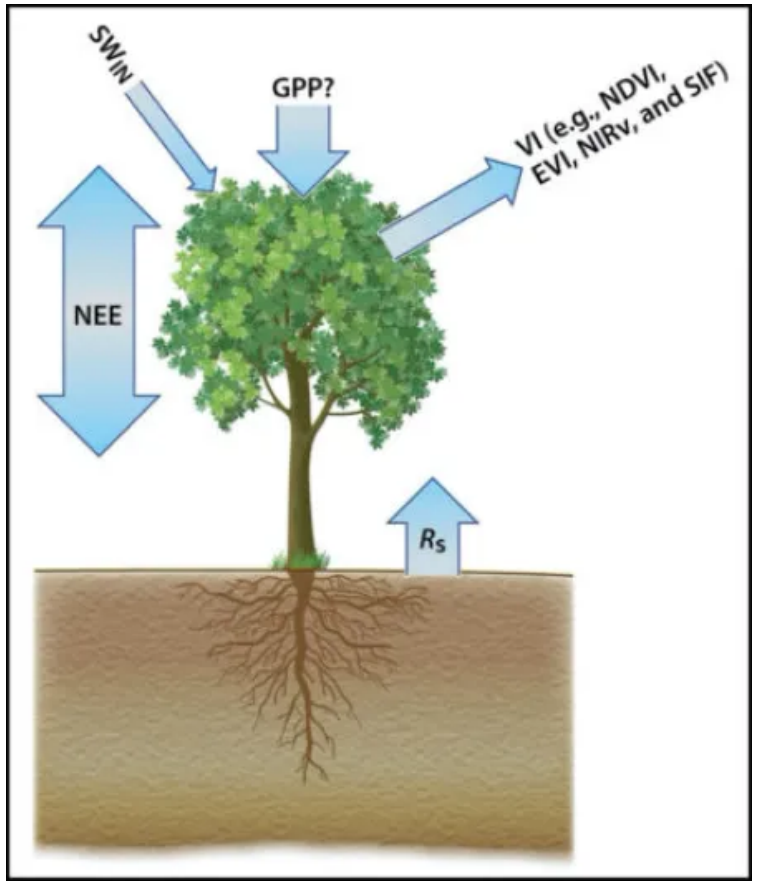
- How effective are humanitarian actions to fight food insecurity?
- there is an effect of precipitation in Denmark into precipitation in the Mediterranean region?
- What is the effect of radiation on NEE/GPP ? Or the effect of temperature on RECO?



- Causal link between forest management practices and burned area size, fire severity, post-fire regeneration
- Biomass yield drivers/what if scenarios (management alternatives to optimize productivity)
- Applying causal inference methods to study problems that feature social and climate variables
- Estimate the effect of droughts on crop production across Europe
- Estimate the conditional average treatment effect (CATE) of major crop rotations (in Europe) on yield and/or soil organic carbon using Double Machine Learning (DML)
- ...

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- Usually assume total or partial knowledge of the causal graph
- SCM or potential outcome frameworks

What we will discuss

1. Brief and fast summary of Part I from *Causal Inference: What If* book [Hernán and Robins, 2020]: randomized trials, observational studies and conditions for identification of causal effects, biases
2. Connections with SCM, graphical causal models
3. adjustment, backdoor criterion, do-calculus, front-door adjustment [Peters et al., 2017]
4. some examples

Potential outcomes - Counterfactual model

- consider a binary **treatment variable** A (1: forest management practice (thinning, controlled burns,...) , 0: wild/uncontrolled forest)
- and a binary **outcome** Y (1: burned area, 0: not burned)
- A, Y are random variables that take possible different values for each individual

Hernán and Robins [2020], Wasserman [2004]

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- denote with $Y^{a=1}$ (Y under treatment $a = 1$) the outcome variable that would have been observed under treatment $a = 1$, and similarly $Y^{a=0}$
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- for each individual, only one of the potential outcomes is actually observed/factual.

$$Y = Y^{a=A} \quad \text{(consistency equation)}$$

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Causal effects

Definition

Average causal effects An average causal effect of treatment A on outcome Y is present if

$$P(Y^{a=1} = 1) \neq P(Y^{a=0} = 1)$$

or equivalently (for binary outcomes)

$$\mathbb{E}[Y^{a=1}] \neq \mathbb{E}[Y^{a=0}]$$

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- causal risk ratio $\frac{P(Y^{a=1}=1)}{P(Y^{a=0}=1)}$
- causal odds ratio $\frac{P(Y^{a=1}=1)/P(Y^{a=1}=0)}{P(Y^{a=0}=1)/P(Y^{a=0}=0)}$

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- formally exchangeability is $Y^a \perp\!\!\!\perp A$ (**careful, not** $Y \perp\!\!\!\perp A$!!)

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What if we do not have enough money for treating (managing the forest patch) in half of the population? What if we have a limit on the funding available ? can we still perform an (ideal) randomized experiment ?

New problem: the firefighters do not like your randomized study they say that it is too dangerous not to manage some patches at all, and that some areas have a too high fire risk to be left completely untreated

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- this is called *stratification* and since the causal effect measured are different in each stratum we say that there is *effect modification* by L
- moreover we say that this procedure ensure **conditional exchangeability** $Y^a \perp\!\!\!\perp A|L$

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- **Inverse Probability Weighting** is an alternative, but equivalent, procedure to compute $P(Y^a = 1)$ by weighting each individual sample by $w_l = 1/P(A = a|L = l)$ and then we compute $P(Y^a = 1) = \sum w_l P(Y|A = a, L = l)$

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remember we obtained risk ratios $P(Y^{a=1} = 1|L)/P(Y^{a=0} = 1|L)$ $0.002/0.001 = 2$, $0.001/0.005 = 0.2$ and $0.01/0.2 = 0.05$ in strata with treatment probabilities 0.8, 0.5 and 0.2 for low, medium and high risk areas. What is the risk ratio in the entire population ?

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- still under certain conditions and assumptions we can identify the causal effect
- this conditions *assure that the observational study can be used somehow as a randomized trial*

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If we can assume this three conditions we can use the techniques such as IPW or standardization to compute ATE from observational data

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- they require exchangeability and positivity
- Standardization (or IPW), stratification and matching measure different causal effects: Average effects in the entire population, conditional causal effects (startification) and usually causal effects in the treated and untreated for matching

Counterfactual models associated with a causal DAG. In this book, a causal DAG G represents an underlying counterfactual model. To provide a formal definition of the counterfactual model represented by a DAG G, we use the following notation. For any random variable W, let $\mathcal{W}$ denote the support (i.e., the set of possible values w) of W. For any set of ordered variables $W_1, \ldots, W_m$, define $\overline{w}_m = (w_1, \ldots, w_m)$. Let R denote any subset of variables in V and let r be a value of R. Then V_m^r denotes the counterfactual value of V_m when R is set to r.

A nonparametric structural equation model (NPSEM) represented by a DAG G with vertex set V assumes the existence of unobserved random variables (errors) ϵ_m and deterministic unknown functions $f_m(pa_m, \epsilon_m)$ such that $V_1 = f_1(\epsilon_1)$ and the one-step ahead counterfactual $V_m^{\overline{v}_{m-1}} \equiv V_m^{pa_m}$ is given by $f_m(pa_m, \epsilon_m)$. That is, only the parents of V_m have a direct effect on V_m relative to the other variables on G. An NPSEM implies that any variable V_j on the graph can be intervened on, as counterfactuals in which V_j has been set to a specific value v_j are assumed to exist. Both the factual variable V_m and the counterfactuals V_m^r for any $R \subset V$ are obtained recursively from V_1 and $V_j^{\overline{v}_{j-1}}$, $m \geq j > 1$. For example, $V_3^{v_1} = V_3^{v_1, V_2^{v_1}}$, i.e., the counterfactual value $V_3^{v_1}$ of V_3 when V_1 is set to v_1 is the one-step ahead counterfactual $V_3^{v_1, v_2}$ with v_2 equal to the counterfactual value $V_2^{v_1}$ of V_2. Similarly, $V_3 = V_3^{V_1, V_2^{V_1}}$ and $V_3^{v_1, v_4} = V_3^{v_1}$ because V_4 is not a direct cause of V_3.

Robins (1986) called this NPSEM a finest causally interpreted structural tree graph (FCISTGs) "as fine as the data". Pearl (2000) showed how to represent this model with a DAG. Robins (1986) also proposed more realistic causally interpreted structural tree graphs in which only a subset of the variables are subject to intervention. For expositional purposes, we will assume that every variable can be intervened on, even though the statistical methods considered here do not actually require this assumption.

A FCISTG model does not imply that the causal Markov assumption of Technical Point 6.1 holds; additional statistical independence assumptions are needed. For example, Pearl (2000) assumed an NPSEM in which all error terms ϵ_m are mutually independent. We refer to Pearl's model with independent errors as an NPSEM-IE. In contrast, Robins (1986) only assumed that the one-step ahead counterfactuals $V_m^{\overline{v}_{m-1}} = f_m(pa_m, \epsilon_m)$ and $V_j^{\overline{v}_{j-1}} = f_j(pa_j, \epsilon_j)$, $j < m$, are jointly independent when $\overline{v}_{j-1}$ is a subvector of the $\overline{v}_{m-1}$, and referred to this as the finest fully randomized causally interpreted structured tree graph (FFRCISTG) model. Robins (1986) showed this assumption implies that the causal Markov assumption holds. An NPSEM-IE is an FFRCISTG but not vice-versa because an NPSEM-IE makes many more independence assumptions than an FFRCISTG (Robins and Richardson 2011).

A DAG represents an NPSEM but we need to specify which type. For example, the DAG in Figure 6.2 may correspond to either an NPSEM-IE that implies full exchangeability $\left(Y^{a=0}, Y^{a=1}\right) \perp\!\!\!\perp A$, or to an FFRCISTG that only implies marginal exchangeability $Y^a \perp\!\!\!\perp A$ for both $a = 0$ and $a = 1$. In this book we assume that DAGs represent FFRCISTGs whenever we do not mention the underlying counterfactual model.

Computing interventional distributions in SCM

truncated factorization [Pearl, 1993], G-computation formula [Robins, 1986], manipulation theorem [Spirtes et al., 2000]

Given an SCM $\mathcal{C}$ and an intervened SCM $\tilde{\mathcal{C}}$, obtained from $\mathcal{C}$ by intervening on some X_k with $k \neq j$, we have that

$$P^{\tilde{\mathcal{C}}}(X_j|X_{pa(j)}) = P^{\mathcal{C}}(X_j|X_{pa(j)})$$

- Combining the above property and the assumption of SCM we can sometimes compute interventional distribution from observational quantities

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- Combining the above property and the assumption of SCM we can sometimes compute interventional distribution from observational quantities
- Thus in practical terms we will be able sometimes to estimate interventional objects, such as treatment effects, from observational data alone
- This requires the *knowledge of the causal graph*

Peters et al. [2017]

Confounding and adjusting

- Consider an SCM $\mathcal{C}$, the causal effect from X to Y is called confounded if $P^{\mathcal{C},do(X=x)}(y) \neq P^{\mathcal{C}}(y)$

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- $\mathbf{Z}$ is called a valid adjustment set for X, Y if

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  2. **backdoor criterion** Any $\mathbf{Z}$ such that i) contains no descendant of X and ii) blocks all backdoor paths $\rightarrow X$
  3. **towards necessity** ...

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- Valid adjustment sets ensure conditional exchangeability, thus we can use standardization or stratification to compute average or conditional causal effect

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- Consider an SCM $\mathcal{C}$, the causal effect from X to Y is called confounded if $P^{\mathcal{C},do(X=x)}(y) \neq P^{\mathcal{C}}(y)$
- $\mathbf{Z}$ is called a valid adjustment set for X, Y if

$$P^{\mathcal{C},do(X=x)}(y) = \sum P^{\mathcal{C}}(Y|X, \mathbf{Z} = z)P^{\mathcal{C}}(\mathbf{Z} = \mathbf{z})$$

- Valid asjustment sets are:
  1. **parent adjustment** PA_X
  2. **backdoor criterion** Any $\mathbf{Z}$ such that i) contains no descendant of X and ii) blocks all backdoor paths $\rightarrow X$
  3. **towards necessity** ...
- Valid adjustment sets ensure conditional exchangeability, thus we can use standardization or stratification to compute average or conditional causal effect
- viceversa there are techniques that can handle confounding problems without relying on exchangeability: e.g. difference-in-differences, instrumental variables and the front door criterion

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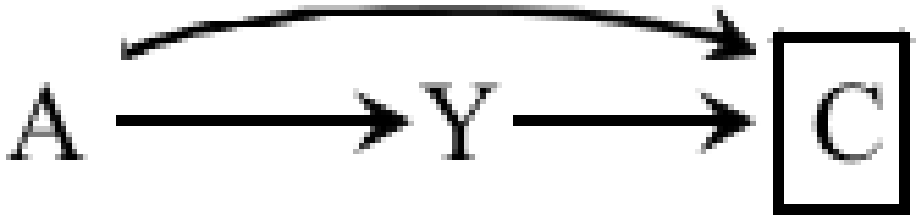
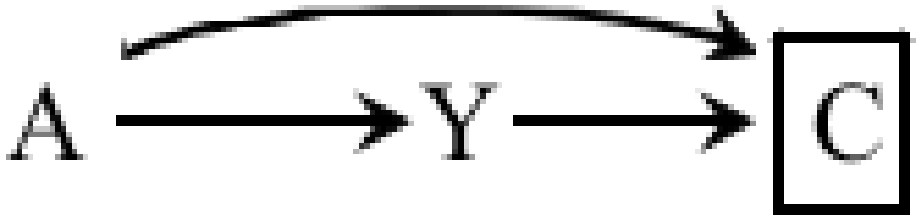
Peters et al. [2017]

Selection bias



- If we condition on C there are two open paths between A and Y

Selection bias



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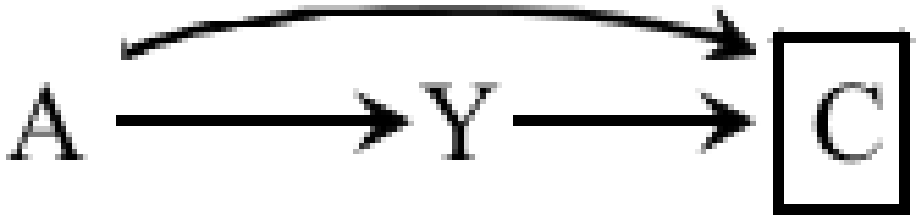

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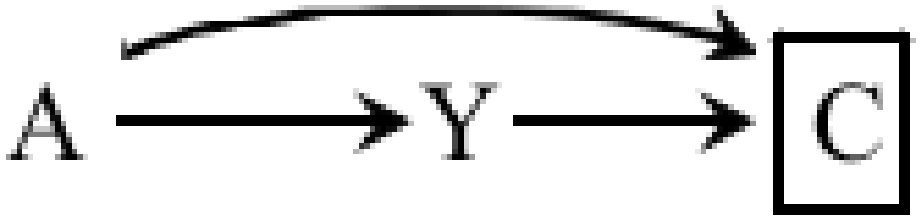

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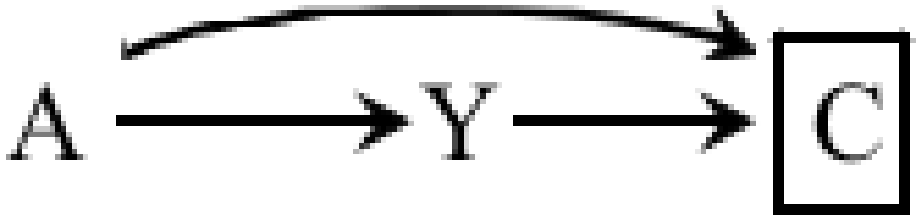

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- This can happen for example: differential loss to follow-up, missing data bias, nonresponse bias, healthy worker bias, self-selection bias, volunteer bias, and selection affected by treatment received before study entry
- Selection bias leads to a lack of exchangeability
- IPW or stratification can be used to control for selection bias
- randomization does not protect from selection bias

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- an interventional distribtuion in an SCM is called identifiable if it can be computed from observational quantities and properties of the graph structure

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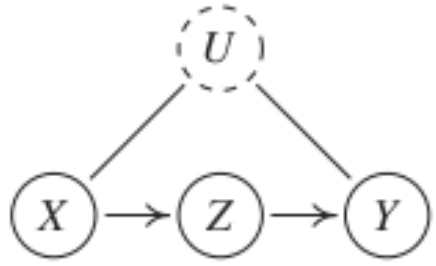
- do-caluclus is complete, every identifiable interventional distribution can be obtained
- one corollary of the do-calculus theorem is the **front-door adjustment**

Example 6.46 (Front-door adjustment) Let $\mathfrak{C}$ be an SCM with corresponding graph



If we do not observe U, we cannot apply the backdoor criterion. In fact, there is no valid adjustment set. But still, provided that $p^{\mathfrak{C}}(x,z) > 0$, the *do*-calculus provides us with

$$p^{\mathfrak{C};do(X:=x)}(y) = \sum_z p^{\mathfrak{C}}(z\,|\,x) \sum_{\tilde{x}} p^{\mathfrak{C}}(y\,|\,\tilde{x},z)\, p^{\mathfrak{C}}(\tilde{x}). \tag{6.23}$$

The fact that observing Z in addition to X and Y here reveals causal information nicely shows that causal relations can also be explored by observing the "channel" (here Z) that carries the "signal" from X to Y. □

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