# Causal questions and principled answers: exposures, populations and effect estimation

#### **STRATOS Topic Group 7**

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This is where STRATOS TG7 aims to contribute

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- 2 The PO Framework
- **3** From questions to estimands

#### 4 Estimation

5 Simulated example

## 6 Summary



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- > There is no agreed complete characterization of causation
- The currently dominant approach in biostatistics and epidemiology to quantifying causal effects relies on potential outcomes (PO; or counterfactuals) \* [Neyman, 1923; Rubin, 1974; Robins, 1986; Pearl, 1995]

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- In general, contributions from within this framework are concerned with causal questions formulated as contrasts of outcomes that would occur under hypothetical interventions on the exposure of interest:

Would the outcome of an individual differ if that individual had been with versus without that exposure?

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Would the outcome of an individual differ if that individual had been with versus without that exposure?

Formally, for a binary exposure: Is  $Y(1) \neq Y(0)$ ?

Here Y(a) denotes the outcome that would have occurred had exposure A been set to take value a <sup>†</sup>

<sup>†</sup>Assuming no interference; see slide 18

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  - Cluster RCT of  $\sim$  20,000 expecting mothers in Belarus carried out in 1996-97
  - Intervention: breastfeeding (BF) encouragement program
  - Primary outcomes: BF uptake and infection rates in infancy
- Imagine we wish to ask a new question regarding, not the effect of the intervention, but of BF (downstream from the randomization):

To what extent BF influences an infant's weight at 3m?

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- What is distinctive of the PO perspective when considering these choices is that they should be selected in such a way that the PO under each level of the exposure is well-defined for each individual in the population
- If this were not the case, then contrasts of POs, used to define causal effects, would be ambiguous

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- Let's select the following for our example:
- 1. population: all singleton births in Belarus in 1996-7 for whom BF is not counter-indicated
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- We could choose a finer and finer definition ... But even the finest may not be sufficiently unique, or may be present in an insufficiently large portion of the data





These considerations lead to invoking the 'technical assumption'<sup>‡</sup> of consistency, defined as (in its simplest form):

Y(a) = Y, for everyone with A = a

This says that, when *A* is set to a certain level for all individuals, it would not change the outcome of those who actually have that exposure level, from the outcome that was actually observed (*"setting the exposure is non-invasive"*)





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- ▷ It also links the selected exposure levels to the data via the equality in Y(a) = Y





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- ▷ A pragmatic approach:
  - aims for a sufficiently well-defined POs which are relevant for the data
  - interprets the POs as averages of the various POs that correspond to the multiple versions of the exposure





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- ▷▷▷ A different definition of exposure would lead to a different average



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- ▷▷ This estimand captures the best case scenario of what an encouragement intervention would achieve



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- ▷ How to proceed?



## Alternative approaches to estimating these estimands are available

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    - Direct confounder adjustment: Outcome regression/stratification/matching based (may or may not involve propensity score as an aid)
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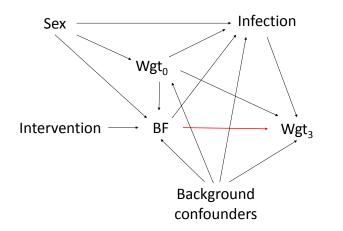
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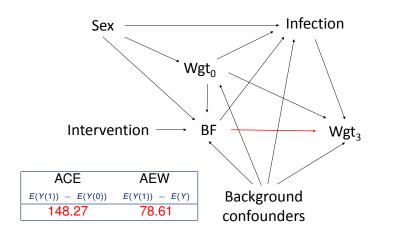
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  - (B) Those that assume there is valid instrument
- All invoke parametric assumptions for some/all of their models, while positivity is invoked when the propensity score is used











(a)



	True value	148.27	
Class	Method	Estimate	(SE)
	Crude regression	253.42	(5.45)
Α	Regression adjustment	151.03	(1.85)
	Regression with PS*	156.14	(2.04)
	PS stratification* (6 strata)	157.49	(6.48)
	PS matching	154.46	(3.96)
	PS IPW	147.16	(2.44)
В	IV (simple)	136.00	(29.38)
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- restricting causal investigations to exposures that are humanly feasible
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Mostly it is accused of *paralysing* research by:

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- not being able to deal with complex exposures

Our view is that the PO framework:

- provides sufficient conditions for the quantitative assessment of certain causal effects
- has achieved important methodological advances where standard methods fail



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- However application of this framework is demanding, conceptually and technically
- Improving our understanding of this approach should be beneficial across many applied fields of research: STRATOS has a role to play!

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Bang and Robins, Biometrics 2005; 61(4):962-73 Cole and Frangakis, Epidemiology 2009;20(1):3–5 Kramer et al., JAMA 2001; 285(4):413-20 Neyman, Statistical Science 1923; 5(4), 465–480 Pearl, Biometrika 1995; 82(4), 669-710 Pearl, Causal Inference in Statistics: A Primer 2016 Rubin, Journal of Educational Psychology 1974; 66, 688–701 Robins, Mathematical Modeling 1986; 7, 1393–1512 Schwartz et al., Ann Epidemiol 2016; Apr 30 Vanderbroucke et al., Int J Epidemiol 2016; Jan 22 VanderWeele, Epidemiology 2009; 20(6):880-3 ◆□▶ ◆□▶ ▲□▶ ▲□▶ □ のので





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one individual's outcome does not depend on the exposure status of others

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- This would occur *e.g.* if babies set not to be BF-ed suffer from infections that themselves affect the infection status, and hence the POs, of other babies
- We should use an extended notation using external information/assumptions regarding who is interfering with whom



- ▷ Pr(A = a | L = l > 0 for all *l* and with  $Pr(L = l) \neq 0$  in the population of interest
- Positivity holds when there are people at all levels of treatment in every level of the confounder.