

Estimating Heterogeneous Treatment Effects in Randomized Control Trials

by Christopher Adams

Discussed by me (Salvador Navarro)
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What I'll do

- Having never been graced with an invitation from NBER, I have never participated in this kind of conference. So I'll wing it.
- The paper is way to contrived for me to follow it as written.
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- This, for reasons unknown, is not what the author does which leads to confusion (on my part) later.
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Missing Data and other stuff we all know

- As we all know by now that is an impossible mission, EVEN if we have a perfect RCT since we only get marginals

$$F(Y_1|X = 1, R = 1) = F(Y|R = 1)$$

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- Author makes the confusing claim that an RCT gives us

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- Chris proposed to think of the problem as a mixture problem with multiple (but less than “usual”) measures.
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Key result

- Forget potential outcomes for a second and just think about the following problem: two variables that are correlated Y, S with the key assumption that, conditional on some other UNOBSERVABLE variable U

$$Y \perp\!\!\!\perp S | U$$

- If this is true then

$$P(Y < y, S < s) = \sum_u \pi(u) F_{Y|U}(y) G_{S|U}(s)$$

- Important assumption not mentioned (as an assumption and only mentioned later in the paper): discreteness. Should be made explicit.
- This may sound trivial but most results on mixtures are discontinuous at the limit (and I suspect this one is too)

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- In matrix notation (more discreetenes)

$$P_{I \times J} = \begin{bmatrix} F & D_{\pi} \\ I \times K & K \times K \end{bmatrix} G'_{K \times J}$$

- Assumption (needs to be made explicit) $I \geq K$, $J \geq K$. What this means is that we have at least as many states that Y (I) and S (J) can take as there are on the unobservable U (K).
- We know that it is also true that

$$P = \begin{matrix} W & H \\ I \times K & K \times J \end{matrix}$$

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Theorem 2

- With this we can state theorem 2 (the main theorem): if blah, blah, blah then

$$P = FD_{\pi}G'$$

is unique up to relabeling.

- What is the if blah, blah? A bunch of uninteresting technical stuff and an interesting one: for each type (K) we want there to exist states (outcomes) that are not possible but highly likely for the other types.
- It seems to me that this is “like” identification at infinity. If I had regressors I would like to vary them so that I know that a type will never visit a state.
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- It is not quite that as this are joint states (i.e., Y, S states) and there are no regressors.
- I would have liked to see an example of when this is likely to happen.
- It seems to me that this is more likely if $I, J \gg K$.
- If $K = 2$ say, then I want there two be a pair of Y, S that $k = 1$ will not visit (very young people will not die immediately if they are type 1 but may if they are type 2) and also for $k = 2$ (very old people will not survive long if they are type 2 but type 1's will).
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- We can do it for each Y_j and test that π and G are the same, or we can do it jointly imposing that $\pi(u)$ and $G(S)$ is the same for all j .
- Estimation is then “simply” a matter of doing (constrained) minimum distance (or GMM)
- He does a Montecarlo exercise.
- More interestingly he estimates this on Chemotherapy patients (Stage III colon cancer, incidentally what my father died of a year ago)

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- Two treatments: “observation” after surgery, just Lev or chemotherapy (5-Fu) .
- The mean effect is that Chemotherapy reduces the risk of recurrence by 41% and the death rate by 33%.
- The author instead models it as a mixture of two types of patients with histology and number of lymph nodes affected being the signals S .
- He actually studies three treatments (although he only talks about two at the beginning). Observation, Lev and Lev + 5Fu.

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- 77% are type 1. For these types all treatments are virtually identical.
- For type 2 patients though observation is a death sentence (no one survives 4 years), 15% do with Lev and 51% do with Lev + 5-Fu.
- Little discussion about how to identify the types. Clearly we can try to via the signals we observe.
- In his case, type 2 patients are those that are likely to have more than 4 lymph nodes affected (and poorly or well differentiated tumors).
- Some mention should be made that the more signals we have the better we can predict patient type. So we can do it with just 1, but having more is better from a practical (forget identification) view.
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- Very interesting idea
- But it is not going after the joint in the sense that he seems to describe in the introduction.
- In the papers we have worked on we are talking about

$$F(Y_1, Y_0) = \int F(Y_1|U) F(Y_0|U) dF(U)$$

but that is what you have as you are interpreting this as types so $\pi(u)$ is the probability of type u regardless of $X = 1$ or $X = 0$.

- For that, I would write the model as
 $X_i = a_i(U_i), Y_{i1} = b_{i1}(U_i), Y_{i0} = b_{i0}(U_i), S_i = c_i(U_i).$

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- That is, we can identify something about how Y_1 and Y_0 are linked because there is a common equation between the two: S . Otherwise, we would be simply imposing that π is the same without anything linking them.
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