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Causation and prediction in epidemiology: A guide to the "Methodological Revolution"

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ABSTRACT

There is an ongoing "methodological revolution" in epidemiology, according to some commentators. The revolution is prompted by the development of a conceptual framework for thinking about causation here referred to as the *Potential Outcomes Approach* (POA), and the mathematical apparatus of directed acyclic graphs that accompanies it. But over and above the mathematics, a number of striking theses about causation are evident, for example: that a cause is something that makes a difference; that a cause is something that humans can intervene on; and that causal knowledge enables one to predict under hypothetical suppositions. This is especially remarkable in a discipline that has variously identified factors such as race and sex as determinants of health, since it has the consequence that factors of this kind cannot be treated as causes either as usefully or as meaningfully as was previously supposed. In this paper I seek to explain the significance of this movement in epidemiology, to understand its commitments, and to evaluate them.

1. Introduction

At the World Congress of Epidemiology (WCE) in August 2014, Miguel Hernán of the Harvard School of Public Health told a plenary session that "causal questions are well-defined as long as interventions are well-specified." What did he mean, and why did he think this was worth saying?

The answer to the second question, according to some commentators, is that there is currently a "methodological revolution" going on in epidemiology. Hernán is a leading "revolutionary", if I may be permitted to use that term to denote the (thoroughly respectable) leading figures in this methodological movement. Maybe "revolution" is too strong, but there is little doubt that something is going on. During the course of the 2014 WCE, the conference Twitter feed featured numerous comments on the apparent methodological revolution. One of the plenary sessions was devoted to "causal inference", which in some circles has come to stand not for causal inference in general, but rather for the particular set of methods and principles that are endorsed by the revolutionaries. Discussion of the revolution also dominated the session devoted to the development of the sixth edition of the Dictionary of Epidemiology.[1] To an observer from outside the discipline, as I was, the overriding feeling was one of uncertainty. On the one hand, there are new exciting methods accompanied by persuasive rhetoric. But on the other hand, the rhetoric includes some messages that directly challenge longstanding epidemiological ways of thinking. Epidemiologists are not sure what to think, whether to be for or against, and—most importantly for the majority of epidemiologists—what to do.

My main concern in this paper is not to explain why this movement is currently attracting discussion in epidemiological circles, nor whether it is truly methodological in character (rather than social, political, etc.). Instead, in this paper I seek to

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[1] Edited by Miquel Porta, also editor-in-chief of the *Journal of Epidemiology and Community Health*, an important journal of the BMJ group and an active presence on Twitter.

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understand the *first* question posed above, namely, what Hernán and other revolutionaries *mean* by assertions of the kind cited in the opening paragraph. I seek to understand the content of the revolution, and to evaluate its conceptual and methodological components.

In Section 2 I identify two papers that are both representative and influential statements of revolutionary thinking, and explain what makes them revolutionary. In Section 3 I seek to extract the underlying conceptual elements of the "revolution". I come up with four theses which I think the revolution is committed to: semantic, metaphysical, pragmatic, and epistemic. In Section 4 I evaluate these theses. I endorse the semantic and metaphysical theses, but reject the pragmatic thesis on the grounds of circularity, and the epistemic thesis on the grounds of falsity. In Section 5 I ask whether I have misrepresented the "revolution", and whether there are methodological lessons to be learned either from the revolution itself or from my critique.

Before going further, let me issue a number of important caveats.

First, I do not pretend to contribute to the technical aspects of the methodological revolution. Undeniably, developments of a technical nature, both within epidemiology and in statistics more widely, have been important. In the last couple of decades there have been widely-discussed mathematical developments in the representation of causal claims and inferences, most famously associated with the work of Judea Pearl (see esp. Pearl, 2009). These developments have had an effect on epidemiology, along with many other social sciences; but in the case of epidemiology, the effect has only followed the successful application of these techniques to analyse data sets in revealing ways. In a seminal pair of papers (a different pair from those discussed in the next section), Miguel Hernán, Jamie Robbins and Babette Brumback developed a mathematical tool ("marginal structural models") and then applied it retrospectively to show that an early trial of an anti-HIV drug in 1980s San Fransisco had been wrongly understood (Hernán, Brumback, & Robins, 2000; Robins, Hernán, & Brumback, 2000). The trial was stopped early on the basis that higher mortality appeared to occur among the intervention group; the re-analysis showed that, in fact, the test group showed a slightly reduced mortality.[2] Although this had been appreciated before the 2000 paper, the new framework offered a much clearer way of expressing what had gone wrong; the new methods provided something that was much more elegant and seemed to offer a much clearer formulation of the causal nature of the situation. As one senior epidemiologist put it to me in conversation, before 2000, nobody took the new "causal inference" stuff seriously; but after this pair of papers, they had to. There have since been other brilliant analyses that have also effectively formalized problems that were previously only informally manageable, and have substantially contributed to or altered our understanding of the data in question.[3]

Second, and relatedly, in this paper I am not questioning the validity or even the usefulness of these technical developments. I am engaging with the conceptual framework within which they have been interpreted and presented. The mathematics is by no means the whole of the methodological revolution. Hernan's claim that causal questions are well-defined as long as interventions are well-specified is not a mathematical claim. It is the conceptual framework giving rise to statements such as this that I want to unearth and then evaluate.

Third, I need to restrict the scope of "Potential Outcomes Approach" and "POA". Clearly not everyone who has advocated a stance using these or related words is saying the same thing, and some are not saying anything that falls at all within the scope of what I am here calling a methodological revolution. It might help to distinguish "broad" from "narrow" senses of POA. In a very narrow sense (and perhaps there are degrees of narrowness and broadness), the POA is just a certain collection of mathematical tools. Someone might promote these tools, and even encourage epidemiologists to formulate their questions in a way amenable to applying those tools, yet still count as an advocate of the narrow POA. They are not advocating the replacement of any existing ways of thinking about causation and causal inference; they are merely advertising the existence of some new, useful tools.

In the broad sense of "POA", on the other hand, the POA is a conceptual framework for understanding causal concepts. This framework happens to be a necessary precursor for the application of certain powerful mathematical tools, but the framework is advocated not on these grounds but on independent grounds which are most naturally described as philosophical—whether or not the advocates admit that they are being philosophical. It is the broad POA that constitutes a methodological revolution, and the broad POA that is my focus in this paper. When I use "POA" in this paper, unless otherwise specified, I mean it in the broad sense.

A fourth thing that I do not set out to do is survey the entire history or scope of the debates around the Potential Outcomes Approach. The sentiments expressed by Hernán and other proponents of the POA have both a history and a number of counterparts in other disciplines. The work of Donald Rubin (Rubin, 1974), vigorously taken up by Paul Holland (Holland, 1986), is a prominent source of the contemporary POA. Holland in particular makes a number of remarks that are similar in tone as well as content to Hernán's—for example, "the effect of a cause is always relative to another cause" (Holland, 1986, 946). The question as to whether race can be a cause was raised in Holland's, 1986 paper and disputed in a response by Clark Glymour (Glymour, 1986). Economists such as James Heckman have also argued that the Rubins/Holland framework has limited econometric application (Heckman, 2008). And, of course, within philosophy, the debate about counterfactual analyses of causation (especially in response to David Lewis's (1973) work) is enormous. The scope of the present paper is thus quite restricted; I am not looking at the whole intellectual development of the POA, but at its significance and recent development in the context of contemporary epidemiology. A study of this whole line of thinking about causation in philosophy, statistics, and the sciences over the last few decades would also be interesting, but very ambitious. The present paper is not such a study, although I hope it might offer something to that more ambitious kind of project.[4]

The main source of misunderstanding that this paper has encountered in draft form concerns a charge of straw man. This charge may be justified, but it arises in part from a misunderstanding of the logical structure of the paper. The goal is to reconstruct a conceptual framework which would provide the necessary materials to turn a certain non-sequitur into a valid

[2] The drug was zidovudine, and the difficulty, briefly, was that the biological indicator used to measure effectiveness of the drug was also a confounder of the causal effect of the drug on survival. CD4 lymphocyte count is both affected by past zidovudine treatment, and is a confounder of the causal effect of zidovudine on survival (Hernán, Brumback, & Robins, 2000, 561).

[3] Interestingly, the first "live", non-retrospective application of marginal structural models (Haight, Tager, & Sternfeld, 2005; Tager, Haight, & Sternfeld, 2004) is criticized by Hernán. His criticism is based on exactly the point discussed in the present paper, namely, the lack of a well-defined causal questions owing to underspecified interventions (Hernán, 2005), confirming that there is more to the POA than the use of certain statistical tools.

[4] I am grateful to an anonymous referee for helping me to distinguish the project of this paper from the more ambitious one.