Invariance, Mechanisms and Epidemiology

In her paper "Causality and Explanation: issues from epidemiology", Raffaella Campaner tries to put forth a challenge to current philosophical accounts of explanation. Of the latter, Campaner finds most relevant to epidemiological practices the mechanistic accounts of Glennan (2002) and Machamer, Darden, and Craver (2000) and Woodward's interventionist account of explanation (Woodward 2003). Campaner is not very specific about what her challenge to these views exactly amounts to, but I take it to consist of at least of the following core claim:

> (CC): Contrary to Woodward, invariances do not perform any explanatory work in epidemiology. Rather, it is mechanistic explanations that epidemiologists search for in their attempts to explain diseases as multi-level and multi-factor phenomena[1].

This challenge, I submit, fails on two counts. First it ignores the view expressed by Woodward (2002) that talk about mechanisms can perfectly be reduced to talk about counterfactual explanations. Second, some of the mechanistic concepts Campaner wishes to transfer from the biological context (in which it was developed) to the epidemiological context do not seem to be applicable there. Since these concepts nevertheless seem to be essential to the standard mechanistic account, this raises the question of whether a different conceptual framework might not be better suited for epidemiology.

Let us start with the first point. In the standard view about mechanistic explanation (see Machamer et al. 2001), mechanisms consist of entities and their activities that jointly *produce* phenomena. Providing an explanation of a phenomenon then means providing a description of the mechanism that produces or is capable of producing the phenomena to be explained. As Woodward (2002) rightly notes, the standard proponents of mechanistic explanations are somewhat vague about how the production relationship is to be understood, despite it obviously playing a crucial part in the explanation of the phenomena: if a mechanism is not capable of producing a phenomenon, describing the mechanism cannot constitute an explanation of the phenomenon in question. Woodward claims to be able to fill the lacuna left by the "mechanists". He suggests that the productive relationship between mechanisms and the phenomena be understood in terms of his interventionist account of explanation: if an intervention were to change the state of a mechanism from M to M* (by changing at least one of the components *m* of M), then the produced phenomenon would change its state from P to P*. Only productive relationships which conform to these "active" counterfactuals, as Woodward calls them in order to point to their interventionist component, are causal relationships. In contrast, to take one of Woodward's main examples, the "wiggling on" a barometer will not bring about the occurrence of a storm—the relationship is non-causal. Now one can of course ask how enlightening the reduction of the mechanistic production relation to an interventionist notion really is.

[1] There are various places in Campaner's paper, in which she makes claims to this effect. For example: "It is mechanistic understanding that is sought for *explanatory* purposes", "invariant generalizations, though, will not suffice for explanatory purposes", and "once the relevant properties are obtained through answers to what-if-things-had-been-different questions, the demand for mechanical explanations persists".

After all, Woodward's account of causal explanation is admittedly non-reductionist, i.e., it does even attempt to reduce causation to some other notion (other approaches try to reduce causation to e.g. some probabilistic notion; see Hitchcock 2010). In any case, Campaner does not really engage with any of this. In many places it looks as though she merely *asserts* that the interventionist notion of causation is not sufficient for explanatory purposes in epidemiology. In other places, Campaner appears to suggest that invariance relationships, contrary to mechanistic explanations, are too coarse-grained for them to serve as explanations:

> Recent epidemiologic literature stresses how what we want to know from a causal explanation – differently from a causal identification - is what goes on *between* the exposure to the risk factors and the appearance of the symptoms, i.e.: we want to uncover what constitutes the pathology's mechanistic working.

Campaner provides the following example to illustrate this idea: there seems to be some sort of invariant relationship between maternal overweight and infant schizophrenia. This alone, however, amounts to not more than a brute fact. Epidemiologists of course want more. They want to find out about the 'causes' or 'mechanisms' of this correlation. Various explanations have been suggested. Infant schizophrenia could be caused by their overweight mother's gestational diabetes or by amphetamines, which overweight mothers are often prescribed by physicians in order to limit weight gain. But I don't quite see why the interventionist account could not be rendered more fine-grained here. Why can one not probe the causal content of each of the above 'mechanistic explanations' by intervening on the relevant variables (by sampling only mothers without gestational diabetes and by refraining from prescribing amphetamines, respectively)? Although I do have sympathies with the idea that invariances are insufficient for explanations, I don't think Campaner does nearly enough to dismiss Woodward's account as being inappropriate for epidemiological explanations.

Let us now turn to the second major problem of Campaner's paper. Contrary to her assessment of Woodward's interventionist notion of causation, Campaner is largely sympathetic to the mechanistic account of explanation. For instance, Campaner finds useful for the epidemiologic context the mechanistic idea that multiple factors at different levels are involved in the causation of a phenomenon (here: a disease). But this is of course just one of the tenets of the mechanistic account. Unfortunately, Campaner does not exercise due care when discussing the other assumptions of the mechanistic account. Another central component of the mechanistic account, for example, is the idea that the levels of mechanisms are not reducible to each other (cf. Machamer et al. 2000). Although this is also true in the epidemiological context, it is trivially true. It simply would not make much sense to claim that environmental factors (as parts of higher level phenomena) are somehow reducible to genetic factors (as parts of lower level phenomena), for instance. Likewise, it would be odd to claim that environmental factors are somehow caused by genetic factors. Nevertheless, we can clearly talk about ion channels in neurons in the brain *causing* action potentials (*the* paradigmatic example of the mechanists; see Craver 2008). Also, as Campaner notes herself, "the very identification of the levels … is far from unproblematic and univocal". This is again clearly different from ion channels (lower level) and action potentials (higher level), where there is no doubt about a clear distinction between levels. Of course, this example

might be uncharacteristically simple even in the context of biology. Perhaps one should then go a step further than Campaner (who apparently does subscribe to the notion of "levels") and call for a broadening of the notion of mechanisms by for instance abandoning the idea of levels altogether. Doing so, however, might run the risk of trivializing the notion of mechanisms as merely multifactoral explanations of phenomena. Before giving up on the original idea of mechanisms in this way, it may be worth pondering whether the (philosophical) notion of mechanistic explanation is really appropriate to the context of epidemiology in the first place.

References:

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