COMMENTS AND CRITICISM

IS ACTUAL DIFFERENCE MAKING ACTUALLY DIFFERENT?*

All agree that genes are part of the causal history of any phenotypic trait. Equally, all agree that many other molecules and cellular structures play a necessary causal role. The usual conclusion has been that this implies causal parity, but C. Kenneth Waters seeks to establish that the causal contribution of genes can (sometimes) be ontologically privileged over those of the other factors. The issue is, of course, the classic one of causal selection that dates to Mill—ontologically, can we distinguish causes from mere background conditions? Against the prevailing philosophical consensus, Waters thinks that we can, and to that end has recently outlined in this JOURNAL his own ingenious argument why.[1]

I certainly agree with Waters that the role of genes in the development and functioning of cells and organisms can be illuminated considerably by recent work on causation—as he himself has notably shown.[2] I also agree with him that theories of causation should be sensitive to scientific practice. However, although sympathetic to much in his paper, in this note I shall argue that, contrary to advertising, it fails to defeat the traditional causal parity thesis.

I. WATERS'S THEORY

Waters's theory supplements James Woodward's influential one.[3] According to Woodward, causation is defined relative to, in Waters's phrase, an *invariance space*. (If X is a cause variable and Y an effect variable, this space is the range of values over which they may vary while preserving an invariant functional relation.[4] For instance, Hooke's Law holds for a spring stretched over a certain range of values; but too much stretching, and the relation breaks down.) That is, Woodward's theory of causation, like other contrastive theories, accords a semantic role to invariance space or its equivalent. This will prove significant.

* I would like to thank Kenneth Waters for helpful discussion on earlier drafts of this comment.

[1] Waters, "Causes That Make a Difference," this JOURNAL, CIV, 11 (November 2007): 551–79.

[2] Waters, "Genes Made Molecular," *Philosophy of Science*, LXI, 2 (March 1994): 163–85; "Molecules Made Biological," *Revue Internationale de Philosophie*, CCXIV (2000): 539–64.

[3] Woodward, *Making Things Happen* (New York: Oxford, 2003).

[4] As Waters points out, the range of values of X and Y for which invariance holds may vary depending on the values of other variables.

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As Waters points out, the causal parity issue then becomes—what determines the portion of the invariance space that is deemed admissible? Different theorists have put forward different formal requirements, but up to now, reflecting the attitude of Mill, one way or another all have allowed a significant role for pragmatic factors. For instance, for any given *Y* there will typically be many weird and wonderful *X* variables that have an invariant relation with it over some range of values, at least some of which in turn make a difference, that is, imply a change in *Y*'s value. All of these are therefore endorsed by Woodward as causes. (Waters labels them potential difference makers.) Their embarrassing weirdness and wonderfulness is explained away as being the result of our intuitive causal judgments being focused elsewhere in invariance space, in particular, fastening only onto those portions of it that are contextually—that is, pragmatically—salient, and that therefore typically do not seem so weird or wonderful. But ontologically, according to Woodward, there remains strict causal parity.

Waters's innovation aims to challenge all that. He adopts Woodward's framework, but augments it with a scheme whereby one portion of the invariance space is privileged—and now privileged ontologically, not just pragmatically. As a result, at least in the simplest cases, just one cause is picked out for extra ontological endorsement.[5] Waters labels this cause the actual difference maker (hereafter *ADM*).

How does Waters's scheme achieve this nonparity result? The key is that the definition of an ADM is explicitly relativized to the choice of an actual difference in a particular population of interest (567). In a given population, only some of the potential difference makers will actually vary. To cite Waters's own example, consider the fruit fly experiments of classical genetics that investigated variation in eye color. Although there were many potential difference makers with respect to eye color, none varied in the actual experimental population of flies except for one particular gene (or, more precisely, the alleles in that gene). The latter did vary across the population, and moreover, as a result, the actual difference to be explained—namely the variation in eye color—is fully explained by this genetic variation alone. Thus the gene is endorsed as the unique ADM, and this is plainly an ontological fact about eye color in this population, independent of any pragmatic concerns.

Waters argues that his ADM apparatus enables us to escape Mill's parity thesis. This is the claim that I shall dispute. As just noted, an ADM is relativized to choice of an actual difference in a population.

[5] Generally, several causes may be so endorsed. I shall focus only on the cases most favorable to Waters's analysis, namely those in which the ADM is unique.

So the obvious retort is that this latter choice is clearly interest-relative, that is, pragmatic; that the specification of an ADM is therefore similarly pragmatic; and so that nothing has been gained over the orthodox view. But Waters is well aware of this objection, and it is worth quoting his answer to it in full:

> [The objection] conflates the identification of a cause with the specification of an effect. Of course, being interested in one effect rather than another depends on more than the ontology of causal processes. Our interests lead us to inquire about the causes of some things and not others. The question here is whether the cause(s) of an effect are fixed by ontology (569).

This is the heart of the matter. I reconstruct Waters's argument as follows:

(P1) Determination of an ADM is fixed by ontology once given an actual difference in a given population.
(P2) Specifying an actual difference in a given population is merely equivalent to specifying an effect.
(P3) While specifying an effect is of course in part pragmatic and thus not fixed by ontology, that does not render the subsequent determination of an ADM pragmatic too.
(C1) Therefore the determination of whether a cause is an ADM is fixed by ontology.
(P4) The distinction between a cause that is an ADM and a cause that is not, (often) tracks the distinction between a cause that is "the" cause and one that is a mere background condition.
(C2) Therefore (often) the traditional causal parity thesis fails.

II. A CRITIQUE

Like Waters, I am sympathetic to contrastive theories such as Woodward's, especially in the context of special sciences.[6] In the situations that concern us here, typically a change in a variable *X* will lead to a change in another variable *Y* but not to one in a third variable *Z*. Roughly speaking, it follows on Woodward's account that *X* is a cause of *Y* but not of *Z*. In other words, whether *X* is a cause indeed depends on the specification of the effect, just as Waters claims. And, as Waters notes, although choice of effect variable *Y* is interest-relative, nobody would for that reason claim that the causal relation between *X* and *Y* is not ontological. I also agree that once given an actual difference in a population, the specification of an ADM is thereafter fixed by ontology. Moreover, I further agree that, at least sometimes, the distinction

[6] Northcott, "Causation and Contrast Classes," *Philosophical Studies*, XXXIX, 1 (May 2008): 111–23.

between actual and potential difference-makers tracks the traditional one between causes and background conditions.

Putting these points together, I accept all of P3, P1, and P4. However, I believe that a problem still remains, and it lies in premise P2. P2, recall, states that specifying an actual difference in a population merely specifies the effect. But I think it does rather more than that—it also partly identifies the cause, too. In particular, it constrains the admissible range of invariance space and thereby the range of admissible variation for the cause variables. (Alternatively put, choice of an actual difference in a population does not just specify *Y* and then leave to ontology whether *X* is a cause of that *Y*; rather, it also constrains what range of *X* is available for consideration.) And for that reason, Waters's nonparity result is not purely ontological but rather also partly pragmatic.

In the fruit fly example, for instance, two causes of variation in eye color are: having one particular allele rather than another in the crucial gene; and perhaps later having some protein available, in contrast to not having it available. Both of these causes are equally endorsed by a contrastive definition in general, but only one is an ADM, namely the gene. That is because, of the two contrast cases—the alternative allele and lack of the protein—only the first actually occurred in the experimental population. Thus the constraint on admissible invariance space resulting from this choice of population impacts the cause variables. Intuitively, on a contrastive theory such as Woodward's, part of the *definition* of a cause is the range of values the cause variable may take. So any constraint on that range, such as restricting attention to just a particular population, is also a constraint on the determination of the cause.

The sequence of argument here is subtle. First, all agree that specification of effect is allowed to be pragmatic. Second, all agree further that, once a population and effect are specified, nonparity is thereafter indeed purely ontological, just as Waters emphasizes. But, to requote Waters himself, "the question here is whether the *cause(s)* of an effect are fixed by ontology" (569, my italics). And the answer to that critical question is 'no', because the causes—not just the effect—are also partly fixed by choice of population. Therefore the nonparity result is not purely ontological, and so there is no dramatic discontinuity with previous theory after all.

Alternatively put, given restriction to those portions of invariance space instantiated in the chosen population, then indeed the specification of ADMs is thereafter purely ontological. But on any current view, once the admissible invariance space has been fixed, then whether something is still a difference-maker is thereafter purely

ontological. There is nothing new about that. The decisive issue is whether *the prior fixing* of the admissible invariance space is purely ontological. That would be new. But it is not what we have here. Rather, the prior fixing is (partly) by choice of population.

Here is a final way to express the point. We can think of a causal relation's invariance space as forming part of an overall state space of all relevant variables. Specification of an actual difference in a population then does two things. First, specification of the actual difference serves merely to specify the effect of interest, just as Waters says. This in turn specifies the invariance spaces of the various candidate causes of that effect. So far, no problem. But second, specification of the population specifies a part of the overall state space too, namely that part corresponding to the variable values that actually occur in that population. In general, there will be overlaps between this population space and each of the invariance spaces, and these regions of overlap are what fix the ADMs. Now there is a problem. Formally, an ADM is a cause whose invariance space has a nonempty overlap with the population space. Mere potential difference makers, on the other hand, are those causes whose invariance spaces do not overlap with the population space. The asymmetry between ADMs and other causes rests on this difference between the admissible ranges of their invariance spaces, that is, between the portions of those spaces that fall in the overlap. That is, the initial specification of an actual difference in a population does not just determine the invariance spaces; it also—crucially—determines what portions of those invariance spaces are admissible.

On a contrastive view, to repeat, part of the definition of a cause is its range of variation or contrasts. Therefore constraints on the admissible range of that variation amount to partially fixing the cause. And restricting attention only to the portion of an invariance space that overlaps with the population space is precisely to constrain that admissible range of variation. Therefore, finally, choice of population does not just specify the effect; it also partly identifies the cause.

III. CONCLUSION

It is perfectly true that once given an experimental fruit fly population, say, it may thereafter be fixed by ontology that a gene and not a supporting protein is the unique ADM with respect to eye color. Perhaps as a result, at least sometimes, scientists are indeed justified in focusing their attentions only on the gene. But none of that implies any breach of the traditional causal parity thesis.

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