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DISCUSSION NOTE

CAUSAL, *A PRIORI* TRUE, AND EXPLANATORY: A REPLY TO LANGE AND ROSENBERG

Mehmet Elgin and Elliott Sober

Sober [2011] argues that some causal statements are *a priori* true and that *a priori* causal truths are central to explanations in the theory of natural selection. Lange and Rosenberg [2011] criticize Sober's argument. They concede that there are *a priori* causal truths, but maintain that those truths are only 'minimally causal'. They also argue that explanations that are built around *a priori* causal truths are not causal explanations, properly speaking. Here we criticize both of Lange and Rosenberg's claims.

Elliott Sober [2011] argues for the anti-Humean thesis that some causal statements are *a priori* true. The ones he discusses have the form '*X* would causally promote *Y*.' Sober also suggests that *a priori* causal statements are central to the theory of natural selection, and to population biology more generally, in that they form the backbones of many evolutionary explanations. We will call this two-part thesis *CAPEC* (= causal *a priori* + explanatory centrality).

In their reply to Sober's paper, Marc Lange and Alexander Rosenberg [2011] argue that *a priori* causal truths are 'minimally causal' [598] and that explanations that are built around those truths aren't *causal* explanations, properly speaking. They say their main goal is to defend the 'Humean dictum' that 'there are no causal explanations containing *a priori* knowable statements specifying the causal relevance of certain features of events' [591]. Although L&R say they aim to 'controvert Sober's argument', it isn't clear that they deny either conjunct in *CAPEC*. Even so, we have doubts about L&R's defence of their Humean dictum. In particular, we challenge their argument for the thesis that *a priori* causal truths are always 'minimally causal'. We also question their thesis that an explanation that is built around an *a priori* causal truth isn't causal.

L&R focus their discussion on the following example that Sober [2011] discusses:

the principle of natural selection (PNS): Trait *A*'s being fitter than trait *B* in a population would causally promote¹ *A*'s increasing in frequency and *B*'s declining if the two traits were heritable.

¹L&R [592] argue that there are true statements of the form '*x* would promote *y*' where the relation isn't causal. We find this dubious, but it doesn't matter, since Sober meant 'causal promotion' when he said 'promotion'.

In what sense is the statement that *A*'s being fitter than *B* caused *A* to increase in frequency and *B* to decline 'minimally causal'?

If 'minimally causal' just means that the statement has omitted some causal fact that is explanatorily important, we agree. For example, the statement has failed to describe *why* *A* is fitter than *B*. The problem is that *all* causal statements omit some causal fact or other that is explanatorily important. It is worth noting that PNS and its variants differ markedly from the example that L&R cite of an obvious triviality, that 'the causes of sleep, if there are any, cause sleep' [598]. The parallel triviality would be 'the causes of evolution, if there are any, cause evolution', but that is not what PNS says.

There is a second reading of L&R's claim that PNS is 'minimally causal'. We think this is the reading that L&R intend, given the way they formulate their 'Humean dictum'. L&R argue that when '*A*'s being fitter than *B* caused trait *A* to increase in frequency' is true, the statement fails to specify the causally efficacious property of the cause that brought about the effect. We agree that *some* true causal statements fail to describe causally efficacious properties. For example, suppose that the first event described in the first chapter of Gibbon's *Decline and Fall of the Roman Empire* caused the second event described in the second chapter. This statement, though true, does not say what it is about the first event that produced the second. Statements of the form 'event E_1 caused event E_2 ' can have this deficiency. Matters change, we believe, when one considers statements of the form '*a*'s having *F* would causally promote *b*'s having *G* in circumstance *C*.' If a statement with this form is true, then it *does* single out a causally efficacious property (namely, *F*). It is statements with this logical form that Sober argues are sometimes *a priori*; if they are true, they cannot leave you in the dark in the way that the statement about Gibbon's book does. L&R grant that there are *a priori* true statements that have this form, but deny that these statements tell you which properties are causally efficacious. We think this is a misreading of what statements of that form assert.

L&R explain their denial when they discuss a proposition they call DORM ('ingesting under certain conditions something with a dormitive virtue would promote falling asleep'). They say that it 'does not specify any first-order properties instantiated in an event *C* of opium ingestion that enables *C* to cause a falling-asleep event *E*' [595]. They then offer the following justification for their claim that a causally efficacious property must be first-order [594–5]:

To argue that the second-order property was causally active threatens to require us to accord causal relevance to *C*'s instantiating a third-order property, and a fourth, and so on. Accordingly, we suggest that what it is about *C* that gives it the power to bring about *E* is its involving the ingestion of a substance with a certain intrinsic, non-dispositional, natural property involving opium's chemical structure. The properties of *C* that are causally relevant to *E*, then, do not include *C*'s involving the ingestion of a substance possessing the second-order property of being soporific.

We think that fitness is a dispositional property and involves the relationship of an organism (or a trait) to an environment. L&R conclude that fitnesses

(and presumably fitness differences), understood in this way, don't cause anything. We are sceptical about their thesis that only intrinsic and nondispositional properties have causal efficacy, especially if 'intrinsic' means *nonrelational*. We don't see much of an argument in L&R's paper for their thesis. They mention a looming regress, but don't develop the idea. As it happens, Lange [2013] has abandoned the claim that dispositional properties are always causally inert.

Even if L&R are right that dispositional and nonintrinsic properties are always causally inert, this point applies to the example of PNS, but not to the other two biological examples that Sober [2011] describes at some length—R.A. Fisher's model of sex ratio evolution and a population genetics model of heterozygote superiority. In the first of these, the mix of sons and daughters that a parent produces is said to causally influence how successful the parent will be in producing grand-offspring. Even if fitness is a dispositional property, it is hard to see how having five sons and five daughters can be a dispositional property that the parent has. We say this even though we worry that the distinction between dispositional and occurrent properties may be misconceived. Perhaps L&R will reply that having five sons and five daughters is not an 'intrinsic' property of a parent. Our response is that this makes their claim that only intrinsic properties can be causally efficacious very implausible indeed. Similar points apply to the model of heterozygote superiority: being a heterozygote doesn't sound like a dispositional property (unless all properties are dispositional), and to us it sounds pretty 'intrinsic'. These two examples were important in Sober's paper. He mentions that readers of his paper may be tempted to dismiss the idea that PNS is explanatory because they think it is trivial and unilluminating, but that it is harder to defend this attitude with respect to these two other examples from mathematical biology. L&R never discuss these examples.

We turn, finally, to L&R's discussion of what a causal explanation is. Here is a key passage [593–4]:

suppose Mother tries to distribute her strawberries evenly among her children without cutting any (strawberries or children). That she has three children and 23 strawberries would promote her attempt's failing. *That Mother's having three children and 23 strawberries would be causes of her failure* (if she tried to distribute the strawberries evenly, under controlled conditions) *is the common verdict of many accounts of causal relations . . .*

Thus, we recognize that there are *a priori* 'would promote' causal statements . . . the 'would promote' statement about Mother is sufficiently informative to figure in scientific explanations.

[W]hen we explain Mother's failure by appealing to the fact that her failure would be promoted by her having three children and 23 strawberries, we do not give a causal explanation. Rather, our explanation works by pointing out that, given the numbers of strawberries and children, Mother cannot succeed—where this necessity is stronger than causal necessity. Mother failed

because failure was inevitable. The numbers of children and strawberries, though they make failure inevitable, do not function in this explanation as causes.

Notice that L&R grant that the strawberry statement they italicize, which has the form ‘*a*’s having *F* would causally promote *b*’s having *G*’, is *a priori* true and explanatory. However, the explanation that embeds this *a priori* causal truth is not, they say, a *causal* explanation. In a footnote [593–4], they explain why:

This example shows that to be a ‘causal explanation’, it is not enough that the *explanans* essentially include a ‘would promote’ statement specifying the causally relevant features of causes and their effect. What makes an explanation ‘causal’ is not what it includes, but how it works: that it explains by delineating contextually relevant parts or features of the world’s causal nexus.

This clarification does not permit L&R to draw the conclusion they want. The strawberry statement *does* delineate ‘contextually relevant features of the world’s causal nexus’.

According to L&R, the facts they mention about Mother, her children, and her strawberries fail to provide a *causal* explanation, because ‘Mother cannot succeed—where this necessity is stronger than causal necessity’, and this is so even though they concede that the generalization in the *explanans* is causal. This concession, we think, means that a second premise in their *explanans* describes the cause and the *explanandum* describes the effect. We find it strange that L&R want to withhold the label ‘causal explanation’, given all this. We also note that probabilistic statements about X’s causally promoting Y do not entail that if X happens, then Y cannot fail to occur. This means that even if L&R are right about the strawberries, their thesis about what a causal explanation is fails to show that the biological examples that Sober discusses aren’t causal.

In summary, L&R grant that there are *a priori* causal truths, but claim that they are only minimally causal. They also claim that explanations that are built around *a priori* causal truths aren’t causal explanations. We fail to see an interesting sense in which these truths are only minimally causal. And we don’t think that L&R have offered a convincing reason to abandon the idea that an explanation that is built around a causal statement (whether it is minimal or not, and whether it is *a priori* or not) is a causal explanation. At the same time, we have no problem with the idea that there are noncausal explanations: they obviously exist in pure mathematics and in the reduction of one empirical law to another by a derivation. All in all, we think that CAPEC is alive and well.

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