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DISCUSSION:

HOW EVERYONE CAN HAVE A RARE PROPERTY: RESPONSE TO SOBER ON FREQUENCY-DEPENDENT CAUSATION*

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In a recent discussion note Sober (1985) elaborates on the argument given in Sober (1982) to show the inadequacy of Ronald Giere's (1979, 1980) causal model for cases of *frequency-dependent causation*, and denies that Giere's (1984) response avoids the problem he raises. I argue that frequency-dependent effects do not pose a problem for Giere's original causal model, and that all parties in this dispute have been guity of misinterpreting the counterfactual populations involved in applying Giere's model.

1. Introduction. Ronald Giere (1979, 1980) offers an interesting model of simple causal hypotheses for populations of deterministic and stochastic systems. Both cases are covered in the following expression of Giere's model:

> (*G*): *C* is a positive causal factor for effect *E* in a population *P* if and only if the *mean value* of *E*[1] were every member of population *P* to have *C* would exceed the mean value of *E* were no member of *P* to have *C*.

On this view, "*C* is a positive causal factor for *E* in population *P*" entails something about two *counterfactual populations*: the first, in which all members of the actual population *P* were to have causal input *C*; the second, in which none were to have *C*. In particular, it entails that the mean value (for example, average incidence) of effect *E* in the first would exceed the mean value of *E* in the second.

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[1]I am allowing *E* to be a *qualitative* as well as a *quantitative* variable property. When the effect is qualitative, such as "has an excellent view of a ballgame" (to consider a property to which I will refer in this discussion), the mean of *E* refers to the *incidence rate* of *E* over members of *P*, attendees at a game. With a qualitative effect, such as "the proportion of the ballfield that one is able to view" (at a given time or on the average), the mean of *E* is the *arithmetic* average of this quantity, the mean proportion of the ballfield viewed over *P*.

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Elliott Sober (1982) has criticized Giere's model as inadequate for cases of *frequency-dependent causation*. A causal factor C is frequency-dependent if its effects in population P vary with the relative frequency of C in P. In a recent discussion note ("What Would Happen If Everyone Did It? . . .") Sober (1985) adds some further comments to the recent responses by Giere (1984) and Collier (1983) to the criticism he raises. The question raised by Sober's criticism is essentially this: *How can a property causally act on all members of a population if the property is effective only when it is rare?* Both the responses of Giere and Collier reflect the belief that the question posed by Sober's problem calls for a revision in Giere's model (*G*). My aim in this paper is not to rehearse the revisions they propose,[2] but to argue that Giere's original model (*G*) adequately answers the above question—provided (*G*) is correctly and consistently interpreted.

2. Illustration of the Problem. While Sober's initial criticisms of Giere concerned cases of frequency-dependent causality from biology, Sober (1985) illustrates his criticism of Giere's model with the following non-biological example:

> At a baseball game *at which everyone is seated,* each individual would have his or her view improved by standing up. Yet, if everyone stood up, the average quality of view would be no better than it would be if no one stood up. Biology aside, this example seems sufficient to show that Giere's theory does not get to first base. In the actual circumstance, standing up is a positive causal factor for seeing better. *Giere's theory, however, apparently denies that standing up is a positive causal factor* (p. 142, emphasis mine).

In this example, the frequency-dependent causal hypothesis is this:

> (***H***): "In the actual circumstance [at a baseball game *at which everyone is seated*], standing up is a positive causal factor for seeing better."

The substance of Sober's argument may be sketched as follows:

> (1) [Given] "At a baseball game *at which everyone is seated,*[3] each

[2]I shall, in note 10, make a single point concerning Giere's (1984) proposed revision to his model for frequency dependent claims as it relates to Sober's recent (1985) discussion note. In "Understanding Frequency-Dependent Causation" (Mayo 1986) I argue that neither Giere's (1984) nor Collier's (1983) proposed revisions adequately answer the question posed by Sober's problem.

[3]The same argument can be and is made where it is stipulated only that most attendees are sitting (Sober 1985, p. 149). My remarks follow essentially the same pattern for this latter case. See note 5.

individual would have his or her view improved by standing up." So, *hypothesis* ***H*** is true.

(2) "Giere's theory, however, apparently denies that standing up is a positive causal factor"; that is, *Giere's theory denies* ***H*** since "if everyone stood up, the average quality of view would be no better than it would be if no one stood up."

(Conclusion) Giere's model (*G*) reaches the wrong conclusion about causal hypothesis ***H***.

The problem, then, is that comparing the mean value of the effect in the two counterfactual populations (100% with factor *C* versus 0% with *C*) may fail to reflect the causal influence *C* has in the actual population, where the frequency of *C*'s is neither of these extreme values. The effect of standing on the ability of attendees to view the game varies depending upon how many other attendees are standing; it is frequency-dependent. While standing up affords a better average quality of view in the actual situation (a baseball game where everyone (else) is seated), the two extreme cases (100% standing and 0% standing) yield the same average quality of view. So here we have an example where causal hypothesis ***H*** is true, and yet no difference shows up when these two extreme cases are compared. Sober concludes that applying Giere's model (*G*) to this case leads to erroneously denying hypothesis ***H***.

This conclusion, however, requires an additional premise concerning Sober's construal of the two counterfactual populations at which Giere's model (*G*) instructs one to look. In particular,

(3) (Sober's Construal): According to Giere's analysis (*G*), if ***H*** is true then the average quality of view were everyone to stand up would exceed the average quality of view were no one to stand up.

According to Sober (in premise (3)), the first counterfactual population in (*G*) is the one in which all of the attendees at the ball game stand *simultaneously*. I will refer to this as the *simultaneous construal* of the counterfactual population. But is this the first counterfactual population at which Giere's model (*G*) instructs one to look? I shall argue that it is not.

3. The Error in the Simultaneous Construal. Let us be clear on what the causal factor *C* is and in what population *P* hypothesis ***H*** is asserted to hold. From the statement affirming ***H*** in premise (1) it is clear that *C* is standing up at a baseball game *at which everyone* [*else*] *is seated*. More simply put, *C* is the property of viewing the game as the *sole stander*. By definition, then, possessing property *C* (that is, being the sole stander)

is a unique property; only one attendee can possess this "unique viewing position" at a given time.

Substituting *C* in Giere's model (*G*) gives as the first counterfactual population the one that would result were all members of *P* to be in this unique viewing position. According to Sober (in premise (3)), the first counterfactual population is the one in which 100% of the attendees [in *P*] stand simultaneously. That, we saw, is the basis of his claim (in premise (2)) that Giere's theory denies ***H***. But the population of *P*'s with all attendees standing at once is *not* the same as the one in which all of the *P*'s have this "unique viewing position". For if, as in the former counterfactual population, all *P*'s stand simultaneously, then standing is not a unique property in population *P*; *none* of the *P*'s enjoys the unique viewing position of "the sole stander". This clearly differs from the latter population where *all* of the *P*'s are sole standers. But we saw that it is this latter population to which the first counterfactual population in Giere's model (*G*) should be taken to refer. (That is, it results from substituting "sole stander" for *C* in "all *P*'s have *C*".) It follows that Sober's premise (3) misconstrues this first counterfactual population.

To put this in another way, if the phrase "when everyone (else) is sitting" appears in premise (1) in affirming the truth of ***H***, it should also appear in applying Giere's model (*G*) in premise (3). However it does not, and because of this premise (3) is false. That a unique property causes some effect (that is, the truth of ***H***) does *not* entail that some shared property causes that same effect. If the causal factor in ***H*** is consistently interpreted throughout, it is seen that the correct instantiation of Giere's model (*G*) is this:

> (3)*: According to Giere's model (*G*), if ***H*** is true, then the quality of view on the average were 100% of the *P*'s to have the unique view of the sole stander would exceed the average view were 0% to be sole standers.

The idea of all attendees possessing the unique property of standing may appear self-contradictory unless it just means all attendees stand (at once). But then (3)* is identical to the simultaneous construal in (3), and Sober's claim that (*G*) leads to the erroneous denial of ***H*** follows soundly.[4] However, while it is contradictory for all *P*'s to have a unique (or a rare) property *at the same time,* it is not contradictory for all *P*'s to have a

[4]Because Giere himself accepted this identity in Giere's (1984) response to Sober, he proposed a special extension of (*G*) to accommodate frequency-dependent cases. As such, it may be claimed that in offering my interpretation of Giere's model in (3)*, I am actually offering a model that is entirely different from the one Giere intended. That may be so, but it is my understanding that Giere would no longer accept the simultaneous construal (identifying (3) with (3)*), and that he generally endorses the solution put forward here.

unique property. My solution rests on providing an interpretation of the latter. For given this interpretation, it will follow that the mean value of effect E when all P's have C is identical to the (mean) effect of C in population P, which is what (G) asserts.

4. How Everyone Can Have a Unique Property. For simplicity, let us continue to take the causal factor C to be the unique property "viewing the game as the sole stander" as in Sober's statement of the example, although an analogous treatment obtains were C required merely to be rare (see note 5). Suppose all k members of the population P of attendees are seated. When a single individual i stands in this situation, that individual has the causal factor C of interest. We can then talk about the effect (on quality of view) that individual i experiences when having factor C in much the same way statisticians talk of the outcome of an experiment on a trial i. There are a variety of measures of the quality of view of the game that can be chosen to represent the possible outcomes of effect E. For instance, the possible outcomes could be represented by the numbers 1, 2, 3, according to whether one's view is poor, fair, or excellent; or the effect can be expressed in terms of the proportion of the ballfield visible. With either sort of measure individual i has some quantitative outcome effect, which can be abbreviated as $E(i)$, when in the experimental situation C of interest (that is, when i is the sole stander). It is easy to see that each of the k members of P can have his or her turn being in experimental situation C; that is, each can have his or her turn being the only one standing at the game. What makes it easy in this case is that here the causal factor can be assigned, and later *undone*. An attendee who is the only one standing in one trial (that is, who possesses C) can be among those sitting in the next, and conversely.

Each of the k attendees would have a single value of the outcome variable E when in the experimental situation C (the only attendee standing).[5] Then the arithmetic mean of these k values yields the mean view when each member of the population of attendees P has the property of "being the only attendee standing". *This is the mean effect in the first counterfactual population in model* (G).[6] Similarly, each of the k attendees would

[5]Since we are letting the causal factor be the unique property (standing while everyone else is sitting), each attendee would only need to have his or her effect considered for a single standing experience. Had the causal factor been defined as standing while most others are sitting, then each would have to be tested as many times as there were "rare standing slots". Also see note 6.

[6]Following Sober's presentation of this example, I am assuming the effect of standing is not stochastic, in the sense that each individual i has only one value for the viewing effect E when everyone else is sitting. However, it is easy to apply this treatment were the effect of C considered stochastic. Each individual would have a mean value of effect

have a single value of the outcome variable E when in the experimental situation represented by not-C, regardless of how this is defined.[7] Then the arithmetic mean of these k values yields the mean view when 0% of P's have C. *This is the mean effect in the second counterfactual population in model* (G). If hypothesis $\boldsymbol{H}$ is true, the mean value of effect E (that is, mean quality of view) in the first counterfactual population (thus defined) would exceed the mean of E in the second. That is to say, if hypothesis $\boldsymbol{H}$ is true, Giere's model (G) (on this interpretation) correctly affirms $\boldsymbol{H}$. Thus (3)* (instantiated for $\boldsymbol{H}$) is true.

Although the baseball example is exceptional in that the counterfactual populations can actually be generated by a series of experiments, the same interpretation of the counterfactual populations, I maintain, is available when they are only hypothetical.

For each member of the actual (finite) population, the causal factor C of interest would produce some value, or (for a stochastic case) some mean value of the effect of interest. The mean of these values over all members of the population would give the mean value of the effect in the first counterfactual population. Similarly, the absence of C (defined relative to the causal process of interest) would produce some (mean) value of effect E for each member, and averaging over the entire population yields the mean value of E in the second counterfactual population in model (G). *Whether or not C is frequency-dependent,* if C is a cause of E in population L, it follows that the mean value of E in the first counterfactual population would exceed that of the second, and conversely—which is precisely what (on the present reading) Giere's model

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E, and these k means would be averaged to yield the mean value of E in the first counterfactual population. We can imagine, for example, that one's viewing capacity would vary with varying weather conditions, or with different permutations of the seating of the other attendees. Those stochastic cases require considering for each individual i, his or her E-values at each of the various weather conditions, or at each of the different permutations of the seating of the other attendees. The resulting set of E-values would be averaged for each individual i. Since the result attached to each individual is a *single* quantity, stochastic considerations, even if made more elaborate, do not require changes in the form of the solution I propose.

[7]Not-C can be defined in various ways depending upon the effect of interest. If one is interested in the average improvement in view afforded by being a sole stander in comparison to the average view at the actual game, then an attendee has property "not-C" when in his or her actual viewing position in the game of interest, with all other members in their actual positions. Or, an attendee may be seen to possess "not-C" whenever he or she is in any of the (finite) positions other than the "sole stander". As can be seen from our discussion of the first counterfactual population (see for example note 6), the interpretation of (G) I propose can accommodate either type of definition of not-C. It should also be noted that for either type of definition of not-C, the second counterfactual population (0% with C, or equivalently, 100% with not-C) that results differs from the one emerging from Sober's simultaneous construal, which is 100% sitting.

(*G*) asserts.[8] Thus, Giere's model (*G*), interpreted in the manner I have suggested, avoids Sober's criticism.[9]

5. A Further Application of (*G*). It is not at all unusual to be interested in the effect of a property that involves the possession of some rare or unique trait. Let us consider such an example so as to distinguish clearly the interpretation of (*G*) I am recommending from the simultaneous construal underlying Sober's criticism. In so doing, it is also to be hoped that the intuitive plausibility of my interpretation will be evident. Suppose one were interested in the effect of being the sole rider on a subway car of an *A*-train on the chance of being mugged in a certain population of Manhattan dwellers. It obviously would not do to consider the accident rate that would occur were the entire population to ride on an *A*-train at the same time (even if there were enough trains to go around)—which is what the simultaneous construal of the counterfactual population would yield. Doubtless, having the entire population on *A*-trains at once would have an effect on the rate of muggings; but it would not be the effect of the causal factor of interest. On the other hand, it seems clear that one could begin to learn about the effect of interest by studying statistics of how often muggings occurred to lone riders as opposed to more crowded conditions.

This intuition is made plausible if one understands the first counterfactual population as the (hypothetical) one that would occur were Giere's model (*G*) to be applied to this case. To see precisely what this amounts to, imagine that attached to each member of the population is a badge indicating the average incidence of muggings (over the time period of interest) when the sole rider on a car of an *A*-train. Imagine the arithmetic average of all the averages on these badges; we may refer to it as the *first counterfactual population average*. My interpretation amounts to identifying this first counterfactual population average with the first counterfactual population in Giere's model (*G*) (that is, with the mean incidence of mugging were all members of the population to be "lone riders" on a car of an *A*-train). This interpretation explains why, although obtaining this population average is impossible, one can expect to learn about the effect of interest by observing certain statistical samples.

[8]We can go further, for not only is *some* increase in the effect detected by the trials described by my construal of (*G*) when *C* is a positive causal factor for *E* in *P*. The counterfactual trials described also correctly measure the *extent* of the difference in the mean of effect *E* due to *C*, which Giere terms the *effectiveness* of *C* for *P*. See for example Giere 1980, p. 266.

[9]While I shall not discuss the biological examples Sober considers here, both the problem they allegedly pose for Giere's model (*G*) and the solution to that problem recommended here, are analogous on all key points. (This is shown in my "Understanding Frequency Dependent Causation" cited in note 2.)

6. Advantage Over Sober's Probabilistic Causal Criterion. An additional point of misunderstanding that seems to underlie Sober's final comments (p. 149) on the value of Giere's causal model merits clarification.

There he says:

> I was surprised to learn in Giere's reply ([1984] pp. 385–386) that he thinks his theory is based on the idea that a positive causal factor must raise the probability of its effect in all (or most) causally relevant background contexts. I also subscribe to this *probabilistic criterion*. . . . Although it is *false* that the average quality of view at a ballgame would be better if everyone stood up than it would be if no one did, it is *true* that each person at a given time would see better by standing, given that few people are doing so [initial emphasis mine]. (Sober 1985, p. 149)

In other words, hypothesis ***H*** is correctly affirmed by what Sober terms the *probabilistic criterion*, while erroneously denied by Giere's model (*G*). The supposition that Giere accepts this probabilistic criterion leads Sober to ask:

> Even if Giere's proposal can be modified so that it coincides in all cases with the dictates of the probabilistic criterion, what is the gain, if the probabilistic criterion is used to evaluate whether Giere's counterfactual models generate the right judgments about examples? (Sober 1985, p. 149)

But Giere does not subscribe to the probabilistic criterion favored by Sober and others. According to this criterion, "a positive causal factor must raise the probability of the effect in at least one causal background context and must not lower it in any" (Sober 1984, p. 315). So for ***H*** to be true on this probabilistic criterion, it cannot be that any of the members of *P* have their quality of view lowered by standing. The effect, for it to be positive in a population *P*, must be in the same direction for all members—at least according to the probabilistic criterion Sober favors.

But for Giere, as is made clear in Giere's initial discussion of his causal model (Giere 1980), the fact that *C* is a causal factor for *E* in a population *P* "is compatible with their being some *other* population for which the reverse is true. And indeed, that other population might be a subgroup of the original" (p. 288). So Giere does not accept the probabilistic criterion as Sober alleges. The substantial difference between requiring (as Sober does) that the probabilistic criterion be satisfied (that the mean value of *E* be 0 or positive for each member of *P*) and requiring (as Giere does) that *C* increases the mean value of *E* in population *P* (in the aggregate) is not as striking in the baseball example as in many other causal hy-

potheses.[10] For in the baseball hypothesis ***H*** we are dealing with a case in which it is likely that each attendee's view is increased. However, even here it may be that certain subpopulations would have their view hampered and not helped by standing even were everyone else sitting; for example, they may be seated under overhangs which would block their view were they to stand.[11]

Yet, if (as in actual baseball stadiums) the vast majority are not situated under overhangs (behind pillars or blind), it would still be deemed correct to affirm ***H***, that standing contributes to an improved view in the population *P* (when most or all are sitting). Giere's model (*G*) would affirm ***H*** even if a small handful of attendees would have their views obstructed by standing. In contrast, the probabilistic criterion favored by Sober would deny ***H*** if any attendees would have their views hampered by standing.

More generally, the probabilistic criterion would endorse a methodological strategy that rejected a causal claim of the form: *C* is a positive cause for effect *E*, were the chance of *E* to be decreased for *any* member of *P*. Although in some cases such a procedure might be warranted, it is at variance with numerous causal hypotheses that are accepted and with

[10]Interestingly, if one *were* to require that the probabilistic criterion held, (that is, that all members of *P* be effected in a 0 or positive direction) before asserting that *C* is a positive cause for effect *E*—as Sober does—then the alternative model Giere offers to accommodate frequency-dependent causal claims would not be open to the very criticism Sober raises. Giere's alternative version of (*G*), which I denote by (*G*: Frequency-Dependence), recommends replacing the counterfactual versions of population *P* with counterfactual versions of a given *subpopulation* of *P*. It can be stated as follows:

> (*G*: *Frequency-Dependence*): If a) *C* is a positive (frequency-dependent) causal factor for effect *E* in population *P*, whose effectiveness depends on the proportion of *C*'s in *P* (that is, on the value of *r*), *and* b) *P-sub* is a subpopulation of *P* where changes in the proportion of *C*'s in *P-sub* leave the proportion of *C*'s in the full population (*r*) approximately constant, *then* c) the mean value of effect *E* in *P-sub* would be greater were all members of *P-sub* to have *C*, than if none were to have *C*.

On the probabilistic criterion, if *C* is a positive cause for effect *E*, then *C* produces a 0 or a positive difference in the mean of *E* for *each* member of *P*. Since it would then follow that any subpopulation *P-sub* would be affected in a 0 or a positive direction by *C*, (*G*: Frequency-Dependence) would correctly affirm that *C* causes *E* in *P*. It is only in cases where *C* may effect a subpopulation in a *different direction* from the average effect in the whole population that (*G*: Frequency-Dependence) may fail to detect the population increase. But if one accepts the probabilistic criterion (as Sober does), then failing to detect effects in such cases is not erroneous; it is precisely what a causal model embodying the probabilistic criterion should do.

In contrast, Giere has acknowledged (unpublished communication) that such cases (where subpopulations are effected in a reverse direction from the whole population) will yield counterexamples for (*G*: Frequency-Dependence), further illustrating his rejection of the probabilistic criterion Sober favors. See also note 12.

[11]The example of being seated behind overhangs is attributable to Jim Fetzer in his comments on my "Understanding Frequency-Dependent Causation" at the December 1984 meeting of the APA.

methodological strategies that have been shown to be valuable.[12] In contrast, on Giere's model (*G*), the value attached to fundamental experimental principles (and the disvalue to others) receives an explanation. Since on his model causal claims are understood as assertions about a certain set of counterfactual populations, an experimental strategy that informs about these counterfactual populations is obviously valuable for arriving at and testing such claims. For example, on my construal, (*G*) implies that the only thing that alters the mean value of the effect in the two counterfactual populations is the presence or absence of causal factor *C*. This explains the importance attached to experimentally creating "treated" and "control" groups that differ (with regards to the effect *E*) solely as a result of the presence or absence of the treatment (that is, factor *C*), respectively. The reason is that in this way it is possible to observe, *in effect,* a sample from the two counterfactual populations of interest. And, the rationale of certain methods of taking samples[13] is that they enable inferences about these counterfactual populations to be sustained. Perhaps the greatest support for Giere's causal model is this ability to shed light on appropriate methodological strategies for investigating causal claims. It would be desirable for critiques of this or other empirical models of causal inquiry to include a realistic comparison of their respective values for the methodology of causal inquiry in science.

REFERENCES

Collier, J. (1983), "Frequency-Dependent Causation: A Defense of Giere", *Philosophy of Science 50*: 618–625.

Giere, R. (1979), *Understanding Scientific Reasoning*. New York: Holt, Rinehart, and Winston.

———. (1980), "Causal Systems and Statistical Hypotheses", in L. J. Cohen (ed.), *Applications of Inductive Logic*. New York: Oxford University Press, pp. 251–270.

[12]Since there is typically at least one member of a population *P* for whom a causal factor *C* has an effect in the opposite direction than it does in the overwhelming majority of cases in *P*, common causal claims are prevented from holding on the probabilistic criterion that Sober favors. For example Sober (1984, p. 294) claims that on the causal model he favors

> What is still precluded, however, is that smoking increase some people's chances and reduce the risks that others run. One can't conclude, in this case, that smoking is either a positive or a negative causal factor; there is no such thing as *the* causal role it plays vis-à-vis heart attacks.

[13]I have in mind here Orthodox methods of randomization and stratification. While these play an important role in distinguishing spurious from genuine causal effects in the received theory of experimental design, philosophical discussions of statistics have typically denied that this role is justified. If the goal is to compare a sample treated with *C* to hypothetical results that *would have* occurred were none to have been so treated, then the importance of such methods becomes evident. Thus, standard frequentist (or Orthodox) methods of data generation and analysis, which are directed toward this goal, receive an important justification from Giere's counterfactual causal model.

———. (1984), "Causal Models with Frequency Dependence", *Journal of Philosophy 79*: 384–391.
Mayo, D. (1986), "Understanding Frequency-Dependent Causation", *Philosophical Studies 49*: 109–124.
Sober, E. (1982), "Frequency Dependent Causation", *Journal of Philosophy 79*: 247–253.
———. (1984), *The Nature of Selection*. Cambridge: Bradford/MIT Press.
———. (1985), "What Would Happen if Everyone Did it? A Reply to Collier and Giere on Frequency-Dependent Causation", *Philosophy of Science 52*: 141–150.