Part X

CAUSATION

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General Causation[1]

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1. Introduction

Philosophers of science, e.g. Cartwright (1979) and Skyrms (1980), have given accounts of general causation in probabilistic terms; not surprising since we often accept general causal sentences as true despite being aware of apparent counterinstances. Consider:

(E1) Smoking is a cause of heart attacks.

We accept (E1) as true though aware of smokers who never have a heart attack. Here I quickly review familiar problems in two existing probabilistic accounts, offer a new objection, and suggest the beginnings of an alternative account.

2. Probabilistic Accounts of General Causation

The *traditional model* of general causation maintains that causes, and only causes, raise the probability of the effect. That is:

(TM) F is a cause of G if and only if Prob(Gx/Fx) > Prob(Gx/~Fx).[2]

Despite some intuitive appeal, the traditional model faces at least three basic objections. First, consider cases of epiphenomena in which there are properties F, G, and H such that F is a cause of G, F is a cause of H, but G is not a cause of H and H is not a cause of G. Since F is a common cause of G and H, G will raise the probability of H and H will raise the probability of G. Thus, the traditional model will have the untoward consequence that G is a cause of H and H is a cause of G. Second, the traditional model fails to establish the temporal directedness of general causation. A temporally indexed property, F* at t, can raise the probability of a temporally indexed property, G* at s, even if t is later than s. Third, the traditional model is subject to counterexamples arising from the fact that effects raise the probability of the cause and causes raise their own probability.

With the hope of avoiding the latter two objections, one might be tempted to complicate the traditional model by adding (i) a necessary condition requiring that the

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effect not "begin" before the cause and (ii) a necessary condition requiring that the cause not be identical to the effect. But these moves are *ad hoc*; one would prefer that the characteristics of general causation not be so derivative. Worse, the temporal condition does not rule out all counterexamples arising from the fact that effects raise the probability of the cause if simultaneous causation is possible, and the irreflexivity condition, as I will argue–somewhat surprisingly–in section three, is too strong if a certain sort of simultaneous causation is possible.

The standard alternative to the traditional model has come to be called the *contextual unanimity model*. It's *locus classicus* is Cartwright's (1979) article, but there are many variations (cf. Skyrms 1980, Eells and Sober 1983). Cartwright's version holds that F is a cause of G if and only if F raises the probability of G in every causally homogeneous situation, i.e. situations; roughly, in which all other causes and preventatives of G are held fixed. While the contextual unanimity model has an advantage over the traditional model in that it avoids the problem of epiphenomena, it too fails to establish the temporal directedness of general causation and is subject to the same simple counterexamples deriving from the fact that effects raise the probability of the cause and causes raise their own probability. In addition, it unlike the traditional model is subject to problems regarding the specification of the factors to be held fixed in the homogeneous situations. Cartwright's original specification, for example, makes the account circular by appealing to general causation. Though Eells and Sobers (1983, 41) have a suggestion which removes the circularity, more complications are required to address problems due to the possibility of alternative sufficient causes (Cartwright 1979, 428) and cases of interaction (Cartwright 1979, 427-428, and Eells 1986).

There is another, not so familiar, problem for the traditional model and the contextual unanimity model. It begins with the observation that general causation is sometimes between properties typically instantiated by *distinct* individuals. For example:

(E2) Dumping sewage is a cause of pollution.

(E3) Careless smoking is a cause of forest fires.

Prima facie, (E2) relates the property of dumping sewage and the property of being polluted and (E3) relates the property of careless smoking and the property of being a forest fire. These properties are typically instantiated by distinct individuals in the sense that, in ordinary circumstances, it is not the dumper of sewage who is polluted nor the careless smoker who is a forest fire. (E2) and (E3) contrast with sentences like (E1) reporting causation between properties typically instantiated by a *single* individual. In ordinary circumstances, it is the smoker who has the heart attack.

Neither the traditional model nor the contextual unanimity model gives the correct results when applied to sentences like (E2) or (E3). Consider (E2). The traditional model has the unintuitive consequence that dumping sewage is not a cause of pollution because dumping sewage does not raise the probability of being polluted. Similarly, careless smoking does not raise the probability of being a forest fire. So, the traditional model has the unintuitive consequence that careless smoking is not a cause of forest fires. (Even if those probabilities happened to be raised, that would not be the correct explanation of the facts reported by (E2) and (E3); that is not *why* dumping sewage is a cause of pollution nor *why* careless smoking is a cause of forest fires.) Though I will not go through it, each of these examples creates a similar problem for the contextual unanimity model.

This problem suggests that philosophers have focused too closely on sentences like (E1) relating properties typically instantiated by a single individual. In fact, it looks

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as if they have been focusing on a secondary reading of those sentences. Their accounts suggest that (E1) can be paraphrased as:

(E4) Smoking is a cause of *the smoker's* heart attacks.

And, that surely is *one* reading of (E1). However, sentences like (E2) and (E3) do not admit of an analogous reading. Nor does the following:

(E5) Smoking is a cause of coughing;

because it is the coughing of the smoker and the coughing of others that can contribute to the truth of (E5).

There may be a way to accommodate general causation between properties of distinct individuals which preserves at least the spirit of the traditional model or the contextual unanimity model, but I am skeptical. The two most plausible attempts I have been able to formulate are not without difficulties. The first suggests the following revision of the traditional model:

F is a cause of G if and only if:

Prob[(∃y)(Gy & x=x)/Fx] > Prob[(∃y)(Gy & x=x)/~Fx].

A similar revision might be made in the contextual unanimity model. Unfortunately, the property expressed by the open sentences '(∃y)(Gy & x=x)' is much too easily satisfied for this account to work. For example, the corresponding relative frequencies:

Freq[(∃y)(Gy & x=x)/Fx] and Freq[(∃y)(Gy & x=x)/~Fx]

would both be one in any population in which there is a single satisfier of G no matter how distant and unrelated it was to satisfiers of F. Thus, it would be very fortuitous if the probabilities picked out the correct pairs of properties as causally connected. The second way to revise the traditional model is more drastic and more plausible:

F is a cause of G if and only if:

Prob[(Gy & x=x)/y is exposed to x's having F] > Prob[(Gy & x=x)/~(y is exposed to x's having F)].

Again, similar changes could be made to the contextual unanimity model. The problem in this suggestion stems from the fact that it imports the mysterious relation of *being-exposed-to*. The relation cannot, as one might initially think, be a purely spatiotemporal notion for one cannot set any spatiotemporal limits without thereby ruling out causation at a greater distance.[3] Being-exposed-to has to be a causal relation of some sort, something like "being under the causal influence of." That it is a causal relation is not a problem in and of itself. It is a singular causal relation and so does not make the account circular. As a matter of fact, I am about to argue that an account of general causation should appeal to singular causation. I resist the move because it is a non-ordinary, rather mysterious causal relation that is being invoked. If an account of general causation is going to appeal to a singular causal relation, it is better off appealing to the ordinary singular causal relation. In the next section, I propose the beginnings of an account that does just that.

3. An Alternative Approach

It is useful to think of general causal sentences as akin to *generic sentences*.[4] Examples of generic sentences include:

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(E6) Cheetahs are faster than men.

(E7) Men are taller than women.

As is the case with general causal sentences, we often accept generic sentences as true though aware of apparent counterinstances. We accept (E6) as true though aware that there are crippled cheetahs. We accept (E7) as true though aware of some very tall women and very short men. We should expect there to be ties between generics and probability; ties which may be instructive in our attempt to understand general causation.

A natural probabilistic understanding of (E6) and (E7) goes as follows. Cheetahs are faster than men because the probability of a cheetah being faster than a man, i.e. Prob(Fxy/Cx & My),[5] is sufficiently high. Men are taller than women because the probability of a man being taller than a woman, i.e., Prob(Txy/Mx & Wy), is sufficiently high.[6] What I find instructive about this understanding of (E6) and (E7) is that it makes no attempt to account for the fact that cheetahs are faster than men or the fact that men are taller than women in probabilistic terms without an appeal to the singular faster-than relation or the singular taller-than relation. Indeed, it is *prima facie* implausible to think it could be done. I am inclined to think that the same is true of general causal sentences. Plausible probabilistic understandings of general causal sentences will appeal to the singular causal relation.

Given the similarities between general causal sentences and generics, one might even hope that general causal sentences admit of the same probabilistic understanding as (E6) and (E7). Unfortunately, that is not the case. Consider (E1). If (E1) were tied to probability in the same way as (E6) and (E7), smoking would cause heart attacks just in case Prob(SxCHy/Sx & Hy) were sufficiently high. ('FxCGy' abbreviates 'x's having F is a cause of x's having G'.) However, given the number of particular heart attacks and smokings which are causally unrelated, I suspect that Prob(SxCHy/Sx & Hy) is quite low. So on this understanding (E1) incorrectly turns out false. Thus, (E1) does not admit of the same probabilistic understanding as (E6) and (E7).

We need to recognize that the causal locution shifts the probabilistic understanding slightly. I suggest:

(SM) F is a cause of G if and only if Prob[(∃y)(FxCGy)/Fx] is sufficiently high.[7]

What my account suggests is that smoking is a cause of heart attacks because the probability of a smoker being such that there is a y such that the smoker's smoking is a cause of y's having a heart attack is sufficiently high. The account applies in just the same way to general causal sentences relating properties typically instantiated by distinct individuals. For example, dumping sewage is a cause of pollution because the probability of a dumper of sewage being such that there is a y such that the dumper's dumping is a cause of y's being polluted is sufficiently high.

(SM) is only meant as a first approximation. Further issues, need to be addressed. For example, I have said nothing about how (SM) handles cases of interaction discussed by Cartwright (1979, 427-428) and Eells (1986). Also, the account as stated suggests that general causal sentences are vague because the account does not require the relevant probability to exceed some precise value but only requires that it be sufficiently *high*. But I have said nothing by way of argument to indicate that general causal sentences are vague. Similarly, I am inclined to think that general causal sentences are context sensitive in significant ways. One thought I have is that the context of utterance will fix to varying degrees of precisification how high is *sufficiently* high. In the most demanding contexts only a probability of one will count as sufficiently high. In the

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most undemanding contexts, any probability greater than zero will be sufficient. But again, I have not argued for the context sensitivity of general causal sentences.

Even at this early stage of development, there are certain advantages to (SM). For instance, it is not subject to the problem of epiphenomena. Suppose that F is a cause of G and F is a cause of H, but that G is not a cause of H and H is not a cause of G. According to the account, that F is a cause of G and that F is a cause of H entails that Prob[(∃y)(FxCGy)/Fx] is sufficiently high and that Prob[(∃y)(FxCHy)/Fx] is sufficiently high. However, these facts give us no reason to expect that Prob[(∃y)(GxCHy)/Gx] is sufficiently high nor that Prob[(∃y)(HxCGy)/Hx] is sufficiently high. Indeed, we expect these probabilities to be quite low. So, (SM) is not stuck with the untoward consequence that G is a cause of H, or vice versa.

Another advantage of (SM) is that general causation, so characterized, is temporally directed. Suppose that t is later than s and consider two arbitrary, temporally indexed properties: F = having F* at t, and G = having G* at s. Since the singular causal relation is temporally directed, it necessarily follows that:

Prob[(∃y)(FxCGy)/Fx] = 0.

So, it follows according to (SM) that if t is later than s then it is not the case that F* at t is a cause of G* at s. Hence, (SM) establishes the directedness of general causation. The directedness of general causation is inherited from directedness of singular causation.

My account also avoids the counterexamples due to the fact that effects raise the probability of the cause and causes raise their own probability. In typical cases in which F is a cause of G, the probability required for it to be the case that G is a cause of F will not be sufficiently high. Similarly, the probability required for a property to be a cause of itself typically will not be sufficiently high.

I use to think that the general causation was irreflexive and asymmetric as well as temporally directed; that a property is never a cause of itself and that an effect is never a cause of its cause. The issue is a little complicated, but that interestingly is not the case; at least not if a certain sort of simultaneous singular causation is possible. To see this, suppose simultaneous singular causation between states of affairs involving distinct individuals and a single property is possible. That is, suppose there could be objects b and c, a property F*, and a time t such that b's having F* at t is a cause of c's having F* at t. If this were the case and the relevant probabilities were sufficiently high, then (SM) would have what I now take to be the intuitive consequence that F* at t is a cause of F* at t. Thus, if simultaneous causation between states of affairs involving distinct objects and a single property is possible, then general causation is not irreflexive and, hence, not asymmetric. Believers in the possibility of simultaneous causation of this sort thus should find another advantage for (SM) over the traditional model and the contextual unanimity model. It allows the possibility of a property being a general cause of itself without having the absurd consequence that every property is a general cause of itself.[8]

4. Conclusion

Most of the advantages of my approach over earlier models arise from the fact that (SM) appeals to singular causation. Some of these same advantages might be retained by a slightly different account which also included an appeal to singular causation.[9] Given the early stage of development of (SM), I would not be all that surprised if some other such option had much to recommend it. I do think, however, that a strong case has been made for an appeal to singular causation in the account of general causation.

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Future attempts to explicate general causation should overlap with (SM) at least to that extent.

Notes

[1]Thanks to John Pollock, Roy Sorensen, Mark Johnston, and David LeWine for helpful discussions on causation and probability.

[2]My treatment of probability in most respects parallels Pollock's (1984) development. Those not familiar with Pollock's treatment can think of the probability function as some kind of hypothetical frequency function.

[3]To avoid this consequence while keeping being-exposed-to as a purely spatio-temporal notion, one could make the move sometimes made in analyses of singular causation (e.g. Lewis 1973, 563). First define a relation of proximate general causation and then use it to characterize general causation (simpliciter) in terms of causal chains. But, this move is inelegant and rules out the possibility of true causation at a distance, i.e. causation at a distance with no mediating causal chain.

[4]Roy Sorensen's "Process Vagueness" (manuscript) suggested to me the connection between generics and general causal sentences.

[5]Here and throughout the remainder of the paper my abbreviations will be the obvious ones, unless otherwise noted.

[6]It is worth noting that many generic sentences are not, at least not in a direct fashion, amenable to this analysis (cf. Carlson 1980, 1-32). My discussion of generic sentences is intended only as suggestive of the correct approach to general causation. I am not concerned to give a complete treatment of generic sentences.

[7]Pollock suggested invoking singular causation in the account of general causation in a discussion about the work of Cartwright. He deserves credit for the original insight leading to (SM). Cartwright, in conversation and in a paper read at the 1986 meeting of the *Society for Exact Philosophy,* has indicated that she now favors introducing singular causation in the account of general causation.

[8]Of course, it is controversial whether simultaneous causation of any sort is possible and I cannot address that issue here. What's interesting, however, is that I see no *additional* reason to be suspicious of this particular kind of simultaneous causation. Specifically, notice that the possibility does not call into question the irreflexivity or asymmetry of singular causation.

[9]For example, in the spirit of the traditional model one might suggest: F is a cause of G if and only if Prob[(∃y)(FxCGy)/Fx] > Prob[(∃y)(FxCGy)/~Fx]. I also find the following idea interesting: F is a cause of G if and only if Prob(FxCGy/y is exposed to x's having F) is sufficiently high.

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