# PROBABILITY-LOWERING CAUSES
# AND THE CONNOTATIONS OF CAUSATION*

## *Causas que disminuyen la probabilidad de sus efectos y las connotaciones de la causalidad*

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**ABSTRACT**

A common objection to probabilistic theories of causation is that there are prima facie causes that lower the probability of their effects. Among the many replies to this objection, little attention has been given to Mellor's (1995) indirect strategy to deny that probability-lowering factors are bona fide causes. According to Mellor, such factors do not satisfy the evidential, explanatory, and instrumental connotations of causation. The paper argues that the evidential connotation only entails an epistemically relativized form of causal attribution, not causation itself, and that there are clear cases of explanation and instrumental reasoning that must appeal to negatively relevant factors. In the end, it suggests a more liberal interpretation of causation that restores its connotations.

*Keywords:* D. H. Mellor, causation, probabilism.

**RESUMEN**

Una objeción común a las teorías probabilísticas de la causalidad es que aparentemente existen causas que disminuyen la probabilidad de sus efectos. Entre las muchas respuestas a esta objeción, se le ha dado poca atención a la estrategia indirecta de D. H. Mellor (1995) para negar que un factor que disminuya la probabilidad de un efecto sea una causa legítima. Según Mellor, tales factores no satisfacen las connotaciones evidenciales, explicativas e instrumentales de la causalidad. El artículo argumenta que la connotación evidencial sólo implica una forma epistémicamente relativizada de atribución causal y no la causalidad misma, y que hay casos claros de explicación y razonamiento instrumental que deben apelar a factores negativamente relevantes. Se sugiere una interpretación más liberal de la causalidad que reinstaura sus connotaciones.

*Palabras clave:* D. H. Mellor, causalidad, probabilismo.

## Introduction

The relation of positive statistical relevance is an essential characteristic of the probabilistic approach to causation; causes are required to increase the probability of their effects. The simplest and most natural way to formulate the relation of positive statistical relevance is in terms of conditional probability:

(1) Given any background B of causal circumstances, C causes E if and only if

*i.* $p(E|C) > p(E|\neg C)$

*ii.* There is no causal factor F in B such that $p(E|C \wedge F) = p(E|\neg C \wedge F)$.

The second condition guarantees that E and C will not be spuriously correlated. Notice that the background B of causal circumstances is not epistemically relativized. C must raise the probability of E given the conjunction of all the causally relevant factors in B, not only the ones we know of.[1] Notice also that this is not a reductive analysis of causation since the background conditions are framed in causal language. It is rather an attempt to establish the systematic connections between causation and probability. The question of whether causation can be reduced to probabilities is a thorny one, and I will stay clear of it because it is not relevant for the purpose of this paper.

Deborah Rosen's (1978) well-known example of the miraculous birdie illustrates a fundamental problem in this approach. A golf player tees off and the shot is badly pulled. By the sheerest accident, the ball strikes the branch of a tree near the green and falls directly into the cup for a spectacular hole-in-one. The golfer's pulled drive thus causes him to get a hole-in-one, even though he would have had a greater chance of doing so had he not pulled his drive.

In the light of (1), the only causal facts that we can accept in the example are that the golfer gets a hole-in-one (H) because he drives the ball (D), since $p(H|D) > p(H|\neg D)$; and also that he gets a hole-in-one because the ball hits the tree (T), since $p(H|T) > p(H|\neg T)$. If the ball had not hit the tree, the golfer's chance of getting a hole-in-one after pulling his drive would have been zero. On the other hand, according to (1), and despite being part of the causal chain, the golfer's hole-in-one is not caused by his pulled drive (P) since $p(H|P) < p(H|\neg P)$.

There have been many responses to Rosen's counterexample. In what follows I will present five strategies to deal with the objection. I will not discuss the success of most of them. My purpose is to provide a context for the discussion of the last strategy, suggested by Mellor (1995), which will be the focus of the paper.

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1    It is important to conditionalize the inequality on *any* causal background B in order to avoid Simpson's Paradox. See Cartwright (1979).

The first strategy to deal with the counterexample is to argue that causes will always raise the probabilities of their effects if we choose the "right" sort of effect. If we were to describe the effect in the example as "getting a tree-bouncing hole-in-one," the fact that the golfer pulled his drive would certainly increase the probability of *that* effect. In most cases it is easy to come up with effects that can be described in such a way that a prima facie negative causal factor ends up having a positive statistical relevance. But this *ad hoc* strategy just does not get rid of the original problem. Even if we find the "appropriate" way of describing some of the effects of a given cause to make it positively relevant, we must still explain why that same cause fails to raise the probability of other effects, or of the same effect under a different description.

A second, more promising strategy is to provide a sufficiently precise description of the causal chain in order to restore positive relevance. Under the new description, it is possible to interpolate additional causal links in order to reconditionalize the statistical relations. More precisely, if a cause C does not increase the probability of E, C can be the cause of E nonetheless if there is a sequence of events $<C, A_1, A_2, …, A_n, E>$ such that each element in the sequence increases the probability of its immediate successor. This strategy was first proposed by I. J. Good (1961) and refined in different ways by Lewis (1973) and Salmon (1980, 1984).[2] A closely related strategy is to improve on the theory, using formal tools to show that causes increase the probability of their effects in non-standard or less straightforward ways. Kvart's (2004) use of *ex post facto* probabilities and Eells' (1991) use of probability trajectories would be examples of this strategy.

A third strategy to deal with Rosen's counterexample is to deny the transitivity of causation. If causation is not transitive, the golfer's pulled drive is not a cause of his hole-in-one since there is no *direct* causal link between the two events. The transitivity of causation has been the matter of much debate. Most defenders of probabilistic causation have been unwilling to abandon the transitivity of causation, despite compelling examples to the contrary.[3] The main reason is that the strategy of successive reconditionalization discussed in the previous paragraph requires causation to be transitive.

A fourth and entirely different strategy is to use a more liberal notion of "cause" and include probability-lowering facts or events

2   The strategy is not without its critics. In my view, the main argument against it is that it assumes that all causal networks can be separated into separate causal chains (Humphreys 1986).

3   McDermott (1995) and Kvart (1997) are two notable exceptions of philosophers who defend the probabilistic approach but deny the transitivity of causation. For a more recent discussion of the problem of transitivity, see Hall (2000) and Paul (2000).

within a theory of causation. As part of this strategy one might avoid using the term "cause" altogether and opt instead for the more neutral "causal factor." This strategy is more in accord with the framework of structural equations, which is the basis for most work currently being done on causation (Hitchcock 2001, 2007), and with the literature on causal Bayes nets (Spirtes, Glymour & Sheines, 1993).

The fifth response to the counterexample, which is the one I will be focusing on, is to deny in an indirect and more principled way that any probability-lowering fact or event is a bona fide cause. The argument I will discuss was proposed by Mellor (1995) and rests on the claim that probability-lowering factors do not satisfy the evidential, explanatory, and instrumental connotations of causation: causes are always evidence for their effects, they explain them, and they are the means to obtain them. A probability-lowering factor can do none of these things. Therefore, a probability-lowering factor is not a bona fide cause. In the rest of the paper I examine and evaluate this argument. The analysis will require a fairly close look at the deep connections between causation and the notions of evidence, explanation, and manipulation. I must state at the outset that the discussion that follows does not turn on the details of Mellor's version of the probabilistic theory, in particular, on his conception of chance. I am only interested in the analysis of his overall strategy.

## The Connotations of Causation

Mellor argues that the formulation of the theory stated in (1) is entailed by the five "obvious and undeniable" (1995 79) connotations of causation:

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| Temporal: | Causes generally *precede* their effects. |
| Contiguity: | Causes are *contiguous* to their immediate effects. |
| Evidential: | Causes and effects are *evidence* for each other. |
| Explanatory: | Causes *explain* their effects. |
| Instrumental: | Causes are *means* of bringing about their effects. |

The temporal and spatial connotations of causation do not play a crucial role in the problem at hand, so I will only discuss the last three connotations. Notice that since Mellor believes in the transitivity of causation,[4] the contiguity connotation only applies to *immediate* effects.

Mellor's argument has the following structure. P represents any probability-lowering factor, for example, the golfer's pulled drive:

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4    In a previous article on the same topic, Mellor states that "the cause-effect relation is not intransitive" (1988 231).

*Premise 1*

P is a cause only if it satisfies the evidential, explanatory, and instrumental connotations of causation.

*Premise 2*

These three connotations imply the definition of probabilistic causation.

*Premise 3*

P violates the definition of probabilistic causation.

*Therefore*, by double *modus tollens*,

P is not a cause.

My aim will be to show that the second premise is false, that is, that neither an evidential nor an explanatory nor an instrumental relation implies a relation of positive probabilistic relevance.

## Causes as Evidence

Mellor establishes a very strong relation between causation and evidence, "No cause C that fails to [raise the probability of its effect] can be evidence for an effect E, and every cause C that does do it will be some evidence for E: weak evidence perhaps, if $p(E|C)$ is not much greater than $p(E|¬C)$, but some evidence nonetheless" (1995 70).[5] That is,

(2) C is evidence for E iff $p(E|C) > p(E|¬C)$.

The plausibility of (2) depends on how one understands the notion of evidence. (2) is supported by a principle common to both subjective and objective Bayesian accounts (e.g. Howson & Urbach 2006 and Maher 1996). According to the principle, for a fact *e* to be evidence for a hypothesis *h*, it is necessary and sufficient that *e* increases *h*'s probability. The sufficiency component of the principle can be criticized, however, along the following lines. Insurance companies have calculated the odds of an average golfer getting a hole-in-one at approximately 12,750 to 1, and the odds of a tour professional at 3,756 to 1 (Kindred 1999). Suppose the golfer in Rosen's example is an average player. In that case, the probability of getting a hole-in-one (H) given that he drives the ball (D) is about 0.000078. Given these facts, it would be a misuse of the concept of evidence to say that the fact that he drove the ball is *evidence* that he got a hole-in-one. There are many similar

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5   Throughout the paper I have changed Mellor's notation from $ch_C(E)$ to $p(E|C)$. Mellor is at pains to dissociate the two expressions because he claims that those who use the latter notion "tacitly treat chances as credences" (1995 33). As Edgington (1997) correctly points out, this is an odd statement because statisticians who work with empirical probabilities use the standard notion all the time. In her review of Mellor's book, Edgington clearly shows that Mellor's arguments for dissociating the two expressions are far from convincing.

standard examples in the literature: When Michael Phelps, a fourteen-time Olympic swimming champion, jumps into a pool, he thereby increases his probability of drowning; but knowing that he jumped into a pool is not evidence that he drowned. When I buy a ticket in the New York Lottery, I increase my chances of winning it; but buying a ticket is not evidence that I won the lottery. Likewise, D increases the probability of H, but no one who sees an average golfer drive would say that she has evidence that the drive resulted in a hole-in-one; she does not have a good reason to believe it. On the contrary, the fact that the person driving the ball is an average golfer, and not a pro, could be used as stronger evidence that the drive did not result in a hole-in-one.

It might be argued that D is better evidence for H than knowing, for example, what color shirt the golfer was wearing that day; not a lot of evidence, but some evidence nonetheless. I do not deny that positive relevance is sufficient to determine which facts are *potentially* relevant in the confirmation of a hypothesis. I can imagine a situation where D counts as evidence. Suppose I hear a lot of cheering and clapping coming from the green where the ball is supposed to fall. From my position I can see neither the golfer nor the trajectory of the ball. If someone tells me that the golfer just drove the ball, then under these circumstances D becomes evidence for H. Since the circumstances in the original example do not include the cheering and clapping, D is not evidence for H.

Positive relevance is not sufficient to determine what counts as evidence. As Achinstein (2001) argues convincingly, evidence is a threshold concept with respect to probability. In order for a cause to be evidence for its effect there must be a certain threshold of probability that C gives to E, not just any amount greater than zero. He believes that the threshold should be ½: I believe its value cannot be determined a priori because it might vary in different evidential contexts. A discussion of what this threshold should be would take us too far afield. What is important to note here is that Bayesian orthodoxy should not be taken for granted when discussing the relation between evidence and probability.[6]

In any case, Mellor's argument only requires positive relevance to be a necessary condition for evidence, not a sufficient one. We can therefore weaken (2) to:

(3) c is evidence for E only if $p(E|C) > p(E|\neg C)$.

This weakened version suffers from a different problem. Under almost any conception of evidence it is true that causal evidence is

defeasible, that is, it is always susceptible in principle of being *undercut or rebutted* as more evidence comes in (Pollock 1986). Its status as evidence will thus depend on the total evidence available in a given epistemic context κ.[7] Thus we must modify (3) and replace it by:

(4) c is evidence for E, given κ, only if $p(E|κ ∧ c) > p(E|κ ∧ ¬c)$.

The net result of this modification is that the evidential connotation of causation only supports an epistemically relativized version of probabilistic causation, while Mellor, and presumably most defenders of the probabilistic approach, argue for an absolute or ontic notion. In other words, the evidential component of Mellor's argument only allows us to say that probability-lowering factors are not causes *as far as we know*, but not that they are not causes *simpliciter*. Although this is not a decisive blow against Mellor's argument, we shall now see that the problems with the explanatory and instrumental connotations justify its definitive rejection.

### Causes as Explanations

Mellor's analysis of causation's explanatory connotation follows a very different line of argument. When we look for an explanation, Mellor argues, "we want to know why a state of affairs is a fact when, for all we know, it might not have been. In other words, a principal object of explanation is to close, or at least to reduce, the gap between what we know to be so and what we know to be necessarily so in some not-possibly-not sense" (1995 75). An explanans, therefore, must necessitate its explanandum, or at least raise its probability as much as possible, thereby reducing its probability of not existing.

The demand on the explanans to make its explanandum necessary is obviously satisfied by deterministic causal explanation. If c is sufficient for E, then $p(E|c) = 1$; c gives E no probability of not existing. But in many other cases there will be no sufficient cause for the explanandum. In such cases, Mellor argues, "since the relevant cause c does not make E necessary, knowing that c causes E does not close the gap that calls for explanation" (76). The gap may be narrowed,

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7   The only notable exception is Achinstein's (2001) objective epistemic theory of evidence. In his view, to say that the probability of a hypothesis *h* given evidence *e* is equal to *r* means that it is objectively reasonable to degree *r* to believe *h*. Objective reasonableness supervenes only on physical facts, that is, what it is reasonable to believe in this sense does not depend on anyone's knowledge or beliefs. But then it is difficult to see what makes *e* evidence. The evidential relation holds independently of any inferential processes, and consequently, it fails to show how inferences based on evidence are justified. As Roush (2003 204) justly point out, "it is easy to see what is objective about this view of probability, but it is hard to see what is epistemic about it."

nonetheless, if we know that C raises E's probability. In an indeterministic causal explanation we want to know how much C reduces the probability of ¬E by increasing the probability of E, *i.e.* we want to know how close C gets to making E, as opposed to ¬E, necessary. "The more C raises E's chance the better it explains it" (77).

This view of the purpose of explanation suggests a natural way of measuring how well C explains E: the higher $p(E|C)$ is, the better C explains E. But $p(E|C)$ alone does not tell us anything about C's causal influence; $p(E|C)$ is as contingent on E's other causes as it is on C. The only way to measure how much C contributes to the probability of E is by knowing the difference that C makes to the probability of E's occurrence, *i.e.* the difference between $p(E|C)$ and $p(E|¬C)$. Thus, if C and C′ are explanations of the same E,

(5) C explains E better than C′ iff $p(E|C) − p(E|¬C) > p(E|C′) − p(E|¬C′)$.

Whether an explanans must make its explanandum probable (Hempel 1965), or whether it can give its explanandum any probability (Jeffrey 1971) has long been disputed within the theory of explanation. Mellor rejects both possibilities, but acknowledges that C's explanatory virtue, like its evidential virtue, "derives from the virtue of making $p(E)$ high−or at least higher than $p(¬E)$. Hence our reluctance to call C a 'good' explanation of E unless it makes $p(E)$ greater than 1/2" (96). However, he believes that *any* cause that raises the probability of its effect provides an explanation for it, "a poor explanation no doubt if $p(E|C)$ is low and thus not much greater than $p(E|¬C)$, but some explanation nonetheless" (78). It seems, therefore, that Mellor is committed to the following claim:

(6) C explains E iff $p(E|C) > p(E|¬C)$.

I believe that (6), together with (5), are false, even if we weaken them to:

(5′) C explains E better than C′ only if $p(E|C) − p(E|¬C) > p(E|C′) − p(E|¬C′)$.

and

(6′) C explains E only if $p(E|C) > p(E|¬C)$.

I will concentrate my analysis on (6′).

The main question that an explanation must answer is, in Mellor's view, why did E, on this particular occasion, actually occur and why did it not fail to occur?[8] Within a deterministic context the

8   Mellor's view of explanation is just one among many. Under a different theory it is easy to dissolve the close connection between positive relevance and explanation. In David Lewis' (1986a, 1986b) theory, for example, to explain an event is just to cite one or more of

answer is straightforward: E was physically necessary relative to the explanatory facts. But when we make the transition to an indeterministic context, the close connection between explanation and the probabilistic theory is severed. Consider Hesslow's (1976) well-known example regarding birth control pills. One of the negative side effects of birth control pills is thrombosis. On the other hand, birth control pills prevent pregnancy, which is also a potential cause of thrombosis. Studies show that for sexually active women under 35 who are fertile, do not use other forms of contraception, and have a healthy lifestyle, the consumption of birth control pills lowers their probability of thrombosis. Therefore, according to Hesslow, within this subpopulation birth control pills cause thrombosis even though they lower its probability. Take the case of María, a healthy, fertile, sexually active women under 35 who takes birth control pills and who does not use any other contraception method. Suppose María develops thrombosis. Then her ingestion of birth control pills *explains* her thrombosis, but the explanans lowers the probability of the explanandum.

There have been many responses to Hesslow's example in the literature on probabilistic causation. Eells (1991 223-225) and Cartwright (1989 ch. 3), for example, stratify the background population into subpopulations and require a partial conditional probability increase; Hitchcock (2001) establishes a difference between net effects and component effects along a causal route to show that the consumption of oral contraceptives can both cause and prevent thrombosis in the same subpopulation. Mellor would not find these solutions satisfactory because they are more appropriate for type-level causation, that is, for causation between event or property types. In his view, all causation is singular and the probability of the effect given the cause is a genuine non-changing property of the specific chance situation.

In any case, my purpose here is not to discuss whether these are adequate responses to Hesslow's example, but rather to show that the explanatory connotation of causation does not imply a naïve version of the probabilistic theory, and therefore it cannot be used to deny probability-lowering factors the status of bona fide causes using Mellor's argument. One of the most surprising features of *The Facts of Causation* is Mellor's reluctance to engage with most of the literature on probabilistic causation. There is no discussion, for example, of preemption or of backup mechanisms. He seems to believe that a

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its causes. Since Lewis accepts that a cause can lower the probability of its effect, he can retain the explanatory connotation of causation without preserving positive relevance. Here, however, I want to show that Mellor's argument is misguided even if we accept his own theory of explanation.

bare-bones version of the probabilistic theory can be defended without further modifications, essentially because he thinks he can always deny the status of cause to any fact that does not fit the theory.

Although Hesslow's example weakens the connection between causation and the probabilistic theory, it does not weaken the connection between causation and explanation, provided causation is understood in a liberal way, that is, provided we use the phrase *causal factor* to speak of any circumstance that affects, positively or negatively, the statistical probability of the effect. If an instance of a negative causal factor is part of the causal history of a particular effect E, it *might* be part of the explanation of why E occurred. The decision to include a causal factor, positive or negative, in the explanation is purely pragmatic and dependent on the knowledge situation of the speakers. It would be odd to explain to someone why the golfer in Rosen's example got a hole-in-one without mentioning the fact that the golfer pulled his drive, because that fact is part of the causal history of the effect and it is highly relevant in this particular pragmatic situation.

We arrive here at the same problem that we found in the case of the evidential connotation of causation. Both connotations are highly dependent on epistemic and pragmatic situations, a fact that makes them unsuitable to be sufficient conditions for asserting the objective existence of a causal relation.

## Causes as Instruments

The instrumental connotation of causation states that causes are more or less effective means of bringing about their effects. Mellor bases this part of his argument on an objective version of Jeffrey's (1983) decision theory, with objective utilities, denoted $u(\text{M})$ in place of subjective valuations, and objective chances in place of subjective probabilities (credences). He then defines mean utilities, denoted $mu(\text{M})$, as follows:

$$(7)\ mu(\text{M}) = p(\text{R}|\text{M}) \times u(\text{M} \wedge \text{R}) + p(\neg\text{R}|\text{M}) \times u(\text{M} \wedge \neg\text{R})$$
$$mu(\neg\text{M}) = p(\text{R}|\neg\text{M}) \times u(\neg\text{M} \wedge \text{R}) + p(\neg\text{R}|\neg\text{M}) \times u(\neg\text{M} \wedge \neg\text{R})$$

Mean utilities are usually called objective expected utilities, but Mellor shuns the name because it "falsely suggests a dependence on what someone expects" (1995 81). When choosing between different actions, the theory recommends that one should choose the one with the highest mean utility. In other words, mean utilities obey the usual principle of maximizing objective expected utility. It prescribes doing M if $mu(\text{M}) > mu(\neg\text{M})$ and doing ¬M if $mu(\neg\text{M}) > mu(\text{M})$. If $mu(\text{M}) = mu(\neg\text{M})$, the principle is mute. The mean utility principle tells us, without invoking causation, "what it takes to make M a means to R

and, specifically, what chance any means must give any end to which it is a means" (86). Now something will be a means to an end only if the mean utility principle prescribes it, and it will do so if and only if it raises the probability of its end.

Often the choices one faces are not just between performing an action M and refraining from performing it. Often the choice is between actions that differ in their effectiveness in bringing about the intended end. If one chooses a suboptimal action and achieves the end, the action was a means that failed to raise the probability of its end. Consider the following example. Diana is suffering from a life-threatening cancer but for personal reasons she refuses to follow treatment A, which is effective 95% of the time, and prefers instead to follow treatment B, which is effective only 15% of the time. Diana wants to recover, but by refusing to follow treatment A and following treatment B, she is lowering the probability of her recovery. As it turns out, and despite its low success rate, Diana is cured from her cancer by following treatment B. She did not adopt the optimal means to her end, but a means it was. So a means need not raise the probability of its end. To be sure, either treatment, considered individually and compared to no treatment at all, raises the probability of Diana's recovery. However, in this particular case Diana's decision has to be based on a comparison between treatments; she cannot compare each treatment to no treatment at all because she wants to recover.

What can Mellor offer in response to this example? The key to his response is his definition of what it takes to make M a means to R. Since the suboptimal treatment B is not recommended by the mean utility principle when compared to treatment A, treatment B is simply not a means to its end. But why not? Mellor's definition of a means commits him to the same response he used in the golfer's example. In the latter, Mellor denied that the pulled drive is a cause: in the former, he has to deny that treatment B is a means to an end. But to assume that only optimal actions are means to an end is to beg the question against the possibility of probability-lowering causes. The definition is designed to exclude as means those actions that lower the probability of their ends. As a result, the instrumental component of Mellor's argument cannot be used on pains of circularity.

Just as the instrumental connotation of causation does not have to exclude suboptimal means, it does not have to exclude negatively relevant factors either. The instrumental use of causes is necessary not only to bring about an effect, but also to diminish the probability of factors that might prevent it, and to avoid effects that we view as harmful or undesirable. A more liberal view of causation in which both positive and negatively relevant factors are included thus allows a more comprehensive view of the instrumental connotation of causation.

## Conclusion

The foregoing analysis shows that Mellor has not offered compelling reasons to support the claim that the connotations of causation imply that all causes must raise the probability of their effects. I have argued that these connotations do not entail the relation of positive statistical relevance, either because they only entail an epistemically relativized form of causal attribution, not causation itself, or because there are obvious cases of explanation and instrumental reasoning that must appeal to negatively relevant factors. The connotations can be fully restored if we adopt statistical relevance in general as a criterion to characterize causation and a more inclusive sense of what evidence, explanation and instrumental reasoning are. An analysis of causation that appeals to both positive and negative factors will also be more in accord with current work on causation. As an added bonus, it will avoid any recourse to our common-sense intuitions about causation, a practice that has led to interminable and fruitless discussions during the past decades.

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