# Do causes raise the chances of effects?

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#### 1. Introduction

The issue of whether or not a cause must raise the chance of its effect is one which philosophers seem unable to agree on. Take a hackneyed example: the squirrel's kick. A golfer takes a swing at a golf ball. Fortunately it's a good shot, and the ball heads for the cup. Unfortunately, a squirrel, rather dangerously positioned near the cup, kicks the ball away, thus decreasing the ball's chance of landing in the cup. Fortunately, the ball then hits the branch of a nearby tree and is deflected into the cup.<sup>1</sup>

Question: was the squirrel's kick a cause of the hole-in-one? According to some philosophers' intuitions, the answer is yes.<sup>2</sup> According to others, the answer is no: the ball went in *despite* the kick.<sup>3</sup> According to a third view, the kick was a cause of the hole-in-one *and* the hole-in-one occurred despite the kick.<sup>4</sup>

Clearly what's needed is a principled way of resolving this dispute. Fortunately, help is to be found in Hugh Mellor's book, *The Facts of Causation*. Mellor lists five 'connotations of causation' and argues that three of them (the evidential, explanatory and means-end connotations) entail that causes raise the chances of effects. If Mellor's argument succeeds, we have at our disposal a way of resolving the squirrel problem which relies on solid facts about the concept of causation, rather than on the usual rather unstable intuitions about the causal status of the kick. Unfortunately, as I shall argue, Mellor's connotations fail to establish that causes raise the chances of effects; hence we shall have to look elsewhere to resolve the squirrel problem.

In what follows, I mean the notion of chance-increase to be understood in the counterfactual sense of Mellor 1995 and Lewis 1986a: to say that C raises the chance of E is to say that E has a greater chance of occurring (or of being true) than it would have done in the absence of C. ' $ch_C(E)$ ' (Mellor's notation) is to be read as 'the actual chance of E' (where it's taken for granted that C occurred); and ' $ch_{C}(E)$ ' as 'the chance E would have had in the absence of C'. Also, I shall be concerned solely with causation

<sup>&</sup>lt;sup>1</sup> The original version of this example is in Rosen 1978.

<sup>&</sup>lt;sup>2</sup> See for instance Eells and Sober 1983, and Sober 1986.

<sup>&</sup>lt;sup>3</sup> See for instance Papineau 1986 and Eells 1991.

<sup>&</sup>lt;sup>4</sup> See Menzies 1989.

between particular events or facts, and not with generic or population-level causation.

Mellor's central claim, then, is this: that the evidential, explanatory and means-end connotations of causation entail that if C is a cause of E, then

(\*) 
$$ch_{C}(E) > ch_{\sim C}(E)$$
.

I shall argue that the connotations entail no such thing.

### 2. The evidential connotation

According to Mellor, the evidential connotation of causation is that causes and effects are evidence for each other. He starts out by noting that it is only by comparing  $ch_C(E)$  with  $ch_{\sim C}(E)$  that we can measure how much, or whether, C 'contributes' to the chance of E. He considers the facts that cause an explosion (E): the presence of gas (G), oxygen (O), and a spark (C). In the actual world the explosion just has the chance it has: it does not have one chance 'given' to it, as it were, by C, another by G, and so on. Thus:

What does measure how much there being a spark contributes to the chance of an explosion is the difference this makes to that chance, i.e. the difference between  $ch_C(E)$  and  $ch_{\sim C}(E)$ . For if these chances were equal, the chance of an explosion would be the same whether there was a spark or not. In that case there being a spark would be evidence neither for nor against there being an explosion. While if  $ch_C(E)$  were less than  $ch_{\sim C}(E)$ , there being a spark would lessen the chance of an explosion: C would be evidence against E. To be evidence for E, C must in the circumstances raise the chance of an explosion:  $ch_C(E)$  must be greater than  $ch_{\sim C}(E)$ .

Causation's evidential connotation does therefore require a cause to raise the chances of its effects. A cause need not determine its effects by raising their chances from 0 to 1, but it must raise their chances somewhat: it must satisfy [(\*)]. No cause C that fails to do this 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  $ch_C(E)$  is not much greater than  $ch_{C}(E)$ , but some evidence nonetheless. (Mellor 1995: 72)

So how plausible is Mellor's claim that in order for a cause C to be evidence for an effect E, C must raise the chance of E? Well, notice that the evidential connotation says that causes and effects are evidence *for each other*. Note also that it is crucial to any counterfactual-based analysis of causation that effects do not raise the chances of causes. Put the two together and you get the result that in general – contrary to what Mellor says in the quoted passage – A can be evidence for B without raising B's

chance, for instance when A is an effect of B, or when A and B are effects of a common cause. So the fact that C fails to raise the chance of E does not exclude C from being evidence for E; hence the evidential connotation does not entail that such a C cannot be a cause of E.

## 3. The explanatory connotation

'I think,' says Mellor, 'we require explanations to raise the probability of what they explain because 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'. (Mellor 1995: 75)

Now, as he points out, in indeterministic situations no explanans which cites causes can show why the explanandum is necessary. But Mellor thinks that chances 'measure possibilities' (75): 'the less possible ~E is, i.e. the less ch(~E) is and hence the greater ch(E) is, the closer the fact E is to being necessary. This is the sense in which a cause C may explain E better or worse, depending on how close it comes to making E necessary, i.e. on how much it raises ch(E)' (77). In order to count as an explanation, then, C must contribute to 'closing the gap' between ch(E) and 1; and, he says, C will do this only if it raises the chance of E. 'Thus however high Bill's chance of getting cancer may be, no one would take his smoking to explain his getting cancer if they thought that chance would have been higher still had he not smoked. What makes us take Bill's smoking to explain his getting cancer, ... is that we think his chance of getting it if he did not smoke would be even less' (77).

Does all this show that the explanatory connotation of causation – that causes explain their effects – requires that causes raise the chances of effects? Well, I suppose it does if we accept Mellor's account of explanation. But Mellor's account of explanation is by no means the only account on the market; nor is it obviously the most plausible one. And there are other models of explanation which do allow a C which *fails* to satisfy (\*) to count as an explanation of E. The most obvious candidate here is a causal theory of explanation, for instance David Lewis' (1986b). For Lewis, to explain an event just is to cite one, or some, of its causes. So of course if you think (as Lewis does<sup>5</sup>) that a cause can lower the chance of an effect, you can retain the explanatory connotation of causation by adopting a causal theory of explanation.

Another plausible model of explanation is the 'statistical-relevance' (S-R) model proposed by Wesley Salmon (1971, 1984). The S-R model of explanation is the 'statistical-relevance' (S-R) model proposed by Wesley Salmon (1971, 1984).

<sup>&</sup>lt;sup>5</sup> See Lewis 1986a, Postscript B.

nation says roughly that for C to explain E, for instance for the squirrel's kick to explain the hole-in-one, P(E/C) must differ from  $P(E/\sim C)$  in the 'objectively homogeneous reference class' to which the kick belongs. (Very roughly, think of the class of all golfing situations that are exactly similar to the present one, and think of what proportion of those end up with a hole-in-one. This gives you P(E/C). Now think of the class of all golfing situations that are exactly similar to the present one except that they lack the squirrel, and think of what proportion of *those* end up with a hole-in-one. This gives you  $P(E/\sim C)$ .)

The important point here is the requirement that P(E/C) must *differ from* – not necessarily be greater than – P(E/~C): 'a complete explanation of an event must make mention of the causal factors that tend to prevent its occurrence [i.e. lower its probability in an objectively homogeneous reference class] as well as those that tend to bring it about' (Salmon 1984: 46). Now, for simplicity's sake, and without begging any relevant questions, suppose that we can equate the conditional probability P(hole-in-one/kick) with the actual chance of the hole-in-one just after the kick, and P(hole-in-one/no kick) with what the chance of the hole-in-one would have been without the kick. Then P(hole-in-one/kick) < P(hole-in-one/no kick). According to Salmon's S-R model, then, we *can* explain the hole-in-one by reference to the squirrel's kick (although not, perhaps, without also citing some positive relevant factor too). But of course the kick decreased the chance of the hole-in-one. So again, if a chance decreaser can be a cause, we have an example of a chance-decreasing but nonetheless explanatory cause.

Admittedly this aspect of Salmon's theory has come in for some criticism (see for instance Cohen 1975). But all this shows is that the claim that the kick can feature in an explanation of the hole-in-one is just as controversial as the claim that it can be a *cause* of the hole-in-one. So it isn't as if there are accepted facts about the application of the concept of explanation to which we can appeal in order to settle the causal status of the kick.

There are plausible models of explanation, then, which – together with the explanatory connotation of causation – do not have as a consequence that causes must raise the chances of effects. Of course, this is not a problem for Mellor himself, since his analysis of explanation does entail (\*). But it does show that the explanatory connotation by itself does not entail that causes raise the chances of effects: one can perfectly well agree with Mellor that causation has an explanatory connotation without being required to accept (\*).

#### 4. The means-end connotation

Causes, Mellor says, are means of bringing about their effects. Moreover, he says, 'causation's means-end connotation is even more basic than its

evidential and explanatory connotations, being to my mind the very core of the concept: causation is essentially the feature of the world that gives ends means. But essential or not, the fact is undeniable: causation is in fact what gives ends means.' (Mellor 1995: 79–80)

He goes on to point out that if the means-end relation were spelled out in causal terms (e.g. so that C is a means to E iff C could or does cause E), appeal to the means-end relation would not tell us anything about causation. Fortunately, however, he claims that we can give a non-causal account of what it is to be a means to an end; and the account he gives entails that means (and hence causes) must raise the chances of ends (effects).

I shall argue that Mellor faces a dilemma: If we accept his analysis of the means-end relation, it follows that causation is not after all what gives ends means, and hence we have no reason to suppose that facts about the means-end relation (in particular the fact that means raise the chances of ends) constrain facts about causation. But if we do *not* accept his analysis, we have no reason to suppose that means must raise the chances of ends. Either way, we have no reason to suppose that causes must raise the chances of effects.

I am happy to grant Mellor's argument that on his definition of the means-end relation means raise the chances of ends, so I shall not present the argument in detail.<sup>6</sup> The basic claim is that the means-end relation can be defined using non-causal decision theory, as follows:

Mellor employs an 'objectivized' version of standard utility-maximizing decision theory, so that subjective 'valuations' are replaced by objective utilities u, and subjective probabilities (credences) are replaced by objective chances. The mean utilities (mu) of performing and not performing an act M with respect to an outcome E are thus:

$$\begin{split} & mu(\mathbf{M}) = \mathrm{ch}_{\mathbf{M}}(\mathbf{E}) \cdot u(\mathbf{M} \otimes \mathbf{E}) + \mathrm{ch}_{\mathbf{M}}(\sim \mathbf{E}) \cdot u(\mathbf{M} \otimes \sim \mathbf{E}) \\ & mu(\sim \mathbf{M}) = \mathrm{ch}_{\sim \mathbf{M}}(\mathbf{E}) \cdot u(\sim \mathbf{M} \otimes \mathbf{E}) + \mathrm{ch}_{\sim \mathbf{M}}(\sim \mathbf{E}) \cdot u(\sim \mathbf{M} \otimes \sim \mathbf{E}). \end{split}$$

According to the 'mean utility principle', the objective analogue of the principle of maximising subjective expected utility, it follows that the agent should do M iff  $mu(M) > mu(\sim M)$ .

Given the further assumption that M is a 'pure' means to E iff it is prescribed by the mean utility principle *and* has no intrinsic value or disvalue, i.e. iff

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mu(M) > mu(\sim M) and u(M \& E) = u(\sim M \& E) \& u(M \& \sim E) = u(\sim M \& \sim E),
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it follows that M is a (pure) means to E iff  $ch_M(E) > ch_{\sim M}(E)$ . Hence means

<sup>&</sup>lt;sup>6</sup> See Mellor 1995: 80-88.

raise the chances of ends; so if all causes are means, it follows that all causes raise the chances of effects.

So what's wrong with Mellor's argument? Well, first of all I just want to focus on the crucial assumption – that what it is for M to be a means to end E is for the mean utility of M to exceed the mean utility of ~M. My point will be simply this: the defender of chance-decreasing causes is under no obligation to accept this assumption, and can easily replace it with another analysis of the means-end relation which allows for chance-decreasing means.

Consider the following case. A sensible climber – one who knows, and is keen to avoid, the dangers of hanging around on an exposed summit – is deciding which of the possible routes to take down a mountain. In fact she chooses the worst alternative (M) – if she'd taken any other route her chance of reaching the bottom (E) would have been much greater. Of course if she hadn't taken *any* route, she would have been very much less likely to get the bottom. But that's no reason to equate  $ch_{\sim M}(E)$  with this much lower number: given her desire to get off the summit and start her descent as soon as possible, we may suppose that if she hadn't chosen route M, she would have chosen some other route instead. Luckily, she makes it to the bottom alive and well.

Was what the climber did a means of getting to the bottom? Mellor says not, since her chance of getting there was lowered by what she did. But does one really want to say that what she did was not a means to her end? After all, she achieved what she wanted to achieve, and she did so by way of a perfectly respectable causal process. Of course, she could have chosen a better means. But that doesn't entail that the route she chose was no means at all.

We have a straightforward intuitive disagreement about what counts as a means to an end here. Mellor holds that means maximize mean utilities of ends; the defender of chance-decreasing causes might want to hold instead that to be a means to an end is to be part of a causal process leading to – and hence a cause of – that end. Now, I don't claim that the latter view has a better claim to being the correct analysis of the means-end relation than does Mellor's view. My point is just that there is no obvious way of settling this dispute, and *a fortiori* no obvious reason to suppose the balance to be tipped in Mellor's favour.

More seriously, Mellor's claim that causation is what gives ends means is incompatible with his analysis of the means-end relation. Note first of all that Mellor does *not* claim that C is a cause of E *if and only if* C is a means to E; hence he doesn't claim that C is a cause of E iff  $ch_C(E) > ch_{\sim C}(E)$ . Rather, he construes increase in chance as a *necessary* condition of causation but he doesn't claim that it is a *sufficient* condition. And it's a good

thing that he doesn't, for chance-increase is *not* a sufficient condition of causation.<sup>7</sup> Example: I want X dead, so I poison his soup. You want X dead, so you poison his coffee. Unsuspecting X decides which to drink by activating some indeterministic device: flipping a coin, say. The coin lands heads, he drinks the coffee, and promptly dies. My action raised the chance of his death – from 0.5 to 1, say – so by Mellor's analysis it was a means to my desired, and realised, end. But I did not cause it; he didn't so much as dip his spoon in the soup.

So Mellor apparently holds that although all causes are means, not all means are causes. But he also holds that causation is what gives ends means; and it is just this claim that he uses to warrant his assertion that causation has an 'obvious and undeniable' means-end connotation (Mellor 1995: 79). But, it seems to me, to deny that all means are causes, as Mellor must, is just to *deny* that causation is what gives ends means. If an act can perfectly well be a means to an end without causing that end, as is my poisoning X's soup according to Mellor's definition, then what gives the end (X's death) that particular means cannot be causation.

So one can't hold *both* that causation is what gives ends means, *and* that the means-end relation can be defined in the way Mellor defines it. So we have a choice about what to give up. We can go along with Mellor's analysis of the means-end relation and hence accept that some means are not causes. But that involves severing just the conceptual link between the means-end relation and causation that motivates taking the means-end connotation to be a genuine connotation of our concept of causation. If we can live with the fact that causation is not after all what gives ends means, it's not at all obvious on what basis we could claim intuitive support for the view that facts about the means-end relation nevertheless constrain the concept of causation.

Alternatively we can retain the view that causes are what give ends their means and therewith the means-end connotation of causation. But if we do *that*, given that some chance-increasers are not causes, we cannot identify the means-end relation with chance increase; hence we cannot straightforwardly appeal to the means-end relation to justify the claim that causes must raise the chances of effects.

Might there be some analysis of the means-end relation which does the job Mellor needs to be done? Such an analysis would have to keep the thought that causation is what gives ends means – it would have to yield the result that all means are causes. And, given that the purpose of such an analysis would be to show how the means-end relation constrains causa-

<sup>&</sup>lt;sup>7</sup> I don't just mean this in the trivial sense that C can raise the chance of E and yet E not occur, so that C fails to cause it. Rather I am concerned with cases where C raises the chance of E *and* E occurs, yet not caused by C.

tion, it would also have to yield the result that all causes are means (since if it didn't yield this result – if it allowed that there are causes which are not means – then telling us what conditions means must satisfy would not thereby tells us what conditions causes must satisfy).

Such an analysis of the means-end relation, then, would need to have it that something is a means to E iff it is a cause of E. But, as we've seen, intuitions about means-end relations are no more settled than intuitions about *causal* relations. So it's just not clear that *any* analysis of the means-end relation will tell us anything interesting about causation, since there are no settled intuitions about the means-end relation which holders of rival theories of causation will agree on. Mellor's own analysis is a case in point: his claim that means must raise the chances of ends can just be denied by someone who wants to hold that causes can lower the chances of effects. Such a person can appeal to a causal account of the means-end relation which allows means to lower the chances of effects.

What all this shows, then, is that the means-end connotation of causation cannot settle any interesting facts about causation; in particular it cannot show that causes must raise the chances of ends.

## 5. Concluding remarks

Mellor's connotations of causation don't help settle the squirrel problem because it's perfectly possible to retain those connotations *and* hold that causes can lower the chances of their effects. And we cannot settle the squirrel problem by appeal to common-sense intuition, since, as the wide variety of intuitions garnered in the introduction shows, common sense simply can't make up it mind. What cases like the squirrel's kick reveal is that 'the' common-sense concept of causation is not sufficiently determinate to decide one way or the other on the squirrel case and others of its ilk – not even when we try, as Mellor does, to draw connections between the concept of causation and other, related concepts.

The moral of all this, I suggest, is that the project of analysing our common-sense concept of causation is a hopeless one – or at least, it is if the desired outcome is a list of 'platitudes' or 'central tenets of our folk theory of causation' – a project which Peter Menzies has recently attempted (see Menzies 1996). If common sense can't even decide on whether causes raise the chances of effects, there can be little hope that it will be able to decide on, say, whether causation typically hold between facts or events, or whether causation is transitive – both of which are questions to which Menzies takes the 'folk theory' to yield a definite answer.

This is not to say that we should give up on causation all together. Rather, we should accept that common-sense intuitions about the nature of causation are ill-defined, heterogeneous and even, in some cases, incompatible with one another; and therefore not be too surprised if the project of analysing causation requires that some of our pre-theoretical intuitions be abandoned.

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