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Is everyday causation deterministic or probabilistic?

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ABSTRACT

One view of causation is deterministic: *A causes B* means that whenever *A* occurs, *B* occurs. An alternative view is that causation is probabilistic: the assertion means that given *A*, the probability of *B* is greater than some criterion, such as the probability of *B* given *not-A*. Evidence about the induction of causal relations cannot readily decide between these alternative accounts, and so we examined how people *refute* causal assertions. In four experiments most participants judged that a single counterexample of *A* and *not-B* refuted assertions of the form, *A causes B*. And, as a deterministic theory based on mental models predicted, participants were more likely to request multiple refutations for assertions of the form, *A enables B*. Similarly, refutations of the form *not-A* and *B* were more frequent for enabling than causal assertions. Causation in daily life seems to be a deterministic concept.

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1. Introduction

The everyday concept of causation is puzzling. No-one seems sure about what it means, and some theorists even seek to outlaw it from scientific discourse (e.g., Russell, 1912–13). A more recent skeptic wrote: "There is, in fact, no such thing as cause and effect. It is a popular chimera, a vague notion that will not withstand the batterings of pure reason. It contains an inconsistent set of contradictory ideas and is of little or no value in scientific discourse" (Salsburg, 2001, p. 185–6). One reason for such claims is that common assumptions about causation *are* inconsistent (Johnson-Laird, 2006, Ch. 22). On the one hand, you assume that you can initiate a causal chain. You serve in tennis; your opponent returns the ball; and you play to and fro until the rally ends. Each serve initiates a causal chain. Nature too can intervene to initiate a causal chain: an earthquake causes a building to collapse. The notion of an intervention initiating a causal chain is plausible (see Sloman, 2005; Woodward, 2003). On the other hand, you may assume that every event has a cause (see Lewis, 1986, for the role of this assumption in reasoning about causation). You are watching TV and suddenly the screen goes blank. You infer that something has caused this event—perhaps, the set has lost power, or the system transmitting the program has gone down. Yet, if every event has a cause, then an intervention that seems to initiate a causal chain does not really do so, because it too has a cause. But, now, you are on a slippery slope back to the uncaused cause that initiated all causal chains—perhaps the one great causal chain of being, of which all other chains are mere links. Hence, either you can initiate a causal chain or else every event has a cause, but not both.

An independent question concerns, not common assumptions about causality, but the meaning of the everyday concept itself, as underlying such verbs as *push* and *pull*, and such assertions as *the moon causes the tides*. Skeptics can hardly deny the existence of such a concept, and the question is whether it is deterministic or probabilistic. In the present paper, we outline various theoretical views about the answer, and distinguish between inductive *evidence* for causal relations and the intrinsic *meaning* of such relations. We then turn to some empirical studies of what facts individuals seek in order to refute causal assertions. We take these facts to reflect their conception of causation. Our aim is to make progress towards answering the question in the title of our paper.

The traditional view of causation is deterministic. As Hume (1748/1988, p. 115) wrote: "We may define a cause to be *an object followed by another, and where all the objects, similar to the first, are followed by objects similar to the second. Or in other words, where, if the first object had not been, the second never had existed*." Hume took causation to depend on nothing more than a constant conjunction of cause and effect, whereas Kant argued for a necessary connection between them, which he took to be part of the innate conception of causation. It demands "that something, *A*, should be of such a nature, that something else, *B*, should follow from it necessarily" (Kant, 1787/1934, p. 90). Mill (1874, p. 237) also held a deterministic view: "The invariable antecedent is termed the cause; the invariable consequent, the effect" (Mill, 1874, p. 237). And the reason that Russell (1912–13) argued that causation should be expurgated from philosophy was,

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ironically, because he *presupposed* that it was a deterministic concept, and that science demanded probabilities instead.

In the twentieth century, perhaps reflecting the irreducible probabilities of quantum mechanics, philosophers developed probabilistic accounts of causation. Reichenbach (1956) proposed such an analysis, and others followed in his steps (e.g., Salmon, 1980; Suppes, 1970, 1984). Reichenbach argued that for causation, as in *A causes B*, the following inequality should hold:

$$p(B|A) > p(B|\text{not} - A).$$

That is, the conditional probability of the effect *B* given the cause *A* should be greater than the conditional probability of the effect *B* given that the cause *A* did not occur. Reichenbach also noted that a cause can render irrelevant other events associated with an increase in the probability of its effect. Hence, if the probability of the effect given both the cause and the other event is the same as the probability of the effect given the cause alone, then the other event is irrelevant. Both deterministic and probabilistic views have current proponents in psychology, and we consider both sorts of theory in turn, and then why it is so difficult to decide between them from evidence about the induction of causality.

1.1. A deterministic theory of causality

The theory of mental models provides a deterministic account of the everyday meaning of causation (Goldvarg & Johnson-Laird, 2001; Johnson-Laird, 2006). *A causes B* refers to three temporally-constrained possibilities:

A	B
not-A	B
not-A	not-B

The temporal constraint is that *B* does not precede *A* in time, as corroborated in experimental studies (e.g., Bullock, Gelman, & Baillargeon, 1982; Tversky & Kahneman, 1980). When the events have occurred and are reported in the past tense, *A caused B*, the first of the possibilities above refers to a fact, and the other two cases refer to counterfactual possibilities, which support assertions such as, *if A hadn't happened then B might not have happened* (Goldvarg & Johnson-Laird, 2001; Byrne, 2005). Alternatively, when the cause failed to occur, an analogous change in reference occurs to support the counterfactual assertion, *if A had occurred then B would have occurred*.

Many theories deny that there is any distinction in meaning between causes and enabling conditions. Mill (1874) argued that the difference is capricious (see also Hart & Honoré, 1985). Cheng and Novick (1991) stipulate that the cause is inconstant and the enabling condition is constant within the relevant focal set. According to others, the cause violates a norm assumed by default whereas the enabling condition does not (see e.g., Einhorn & Hogarth, 1986; Kahneman & Miller, 1986). And, according to still another group of theorists, the cause is the factor that is relevant in any explanatory conversation: speakers describe the cause, not the enabler (Hilton & Erb, 1996; Mackie, 1980; Turnbull & Slugoski, 1988). Unlike these accounts, the model theory draws a sharp distinction between the *meaning* of causal and enabling assertions. *A enables B* refers to the following three temporally-constrained possibilities:

A	B
A	not-B
not-A	not-B

But, many assertions, such as, *a fortune enables you to live well*, have a weaker sense that is consistent with all four contingencies, i.e., even without a fortune you can live well. In daily life, however, there is often an implicature that only the antecedent, *A*, makes the consequent, *B*, possible (Goldvarg & Johnson-Laird, 2001).

To hold three distinct possibilities in mind is difficult (Bauer & Johnson-Laird, 1993; Bucciarelli & Johnson-Laird, 1999), and so a central assumption of the model theory is that individuals aim to minimize the number of models, and in particular, *mental* models normally represent explicitly only the case in which both the clauses in an assertion are true. Hence, both *A causes B* and *A enables B* have the same mental models:

A	B
. . .	

where the ellipsis denotes other implicit possibilities. It follows that individuals should not normally distinguish between the meanings of causal and enabling assertions, which may account for the common view that they do not differ in meaning.

The model theory's concept of causation is agnostic about assumptions concerning causation, such as whether every event has a cause or events can initiate causal chains. However, interventions are sometimes said to have their own special logic (e.g., Sloman & Lagnado, 2005). As an example, consider the causal assertion that eating too much causes obesity. Granted its truth, if you were to observe that Pat isn't obese, then you would infer that he doesn't overeat. But, suppose you learn that he takes a pill that prevents obesity. Now, you would no longer infer from his lack of obesity that he doesn't overeat. The pill disables the effects of overeating. No special reasoning is needed, but just an ability to understand the premises:

Overeating causes obesity.
Taking an anti-obesity pill prevents obesity.

and to realize that the second premise takes precedence over the first (see Johnson-Laird, 2006, p. 312 et seq.).

What observations in principle refute a causal assertion? According to the model theory, individuals grasp that a counterexample refutes an invalid inference (Johnson-Laird & Byrne, 1991), and evidence corroborates this hypothesis (e.g., Johnson-Laird & Hasson, 2003). A single observation of the occurrence of *A* without *B* should therefore suffice to refute the assertion, *A causes B*. Likewise, because the theory treats *A prevents B* as equivalent to *A causes B not to occur*, a single observation of the occurrence of *A* with *B* suffices to refute *A prevents B*. Recently, Mandel and Vartanian (2009) have made the same predictions about causation and prevention for similar reasons. They argue that individuals are prone to two biases: they focus on cases in which *A* and *B* co-occur in inferring a causal relation, which corresponds to the mental models of the concept, and they take a causal relation to mean that *A* is sufficient for *B*, and so the relation is refuted by the occurrence of *A* without *B*. It is unclear whether their account extends to enabling assertions.

Individuals often do not distinguish between causes and enabling conditions, which is why many have argued that the two concepts do not differ in meaning. The two relations have the same mental models, but cause is the stronger notion, because a claim that *A causes B*, always rules out the contingency, *A* and *not-B*, whereas the weaker interpretation of *A enables B* rules out no contingencies. The failure to distinguish between the two relations implies that if individuals are forced to think of a refutation of an enabling relation, they should tend to think of *A* and *not-B*. But, they may also realize that if *A* enables *B*, it makes *B* possible, and so what should not occur is *B* without *A*, i.e., that *not-A* and *B* refutes the enabling relation (see the three possibilities above). Likewise, a person who recognizes the distinction between causes and enabling assertions should consider all possibilities and seek an observation of *not-A* and *B* in order to refute the enabling

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