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Causal evidence in risk and policy perceptions: Applying the covariation/mechanism framework

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ABSTRACT

Today's information-rich society demands constant evaluation of cause-effect relationships; behaviors and attitudes ranging from medical choices to voting decisions to policy preferences typically entail some form of causal inference ("Will this policy reduce crime?", "Will this activity improve my health?"). Cause-effect relationships such as these can be thought of as depending on two qualitatively distinct forms of evidence: covariation-based evidence (e.g., "states with this policy have fewer homicides") or mechanism-based (e.g., "this policy will reduce crime by discouraging repeat offenses"). Some psychological work has examined how people process these two forms of causal evidence in instances of "everyday" causality (e.g., assessing why a car will not start), but it is not known how these two forms of evidence contribute to causal judgments in matters of public risk or policy. Three studies (n = 715) investigated whether judgments of risk and policy scenarios would be affected by covariation and mechanism evidence and whether the evidence types interacted with one another (as suggested by past studies). Results showed that causal judgments varied linearly with mechanism strength and logarithmically with covariation strength, and that the evidence types produced only additive effects (but no interaction). We discuss the results' implications for risk communication and policy information dissemination.

1. Introduction

Many of the judgments that people make about their policy preferences, political ideologies, and risk behaviors can be reduced to questions of cause and effect. Decisions ranging from health behaviors ("will consuming this food lower my cholesterol?") to voting patterns ("will this sales tax increase actually fix infrastructure problems?") depend on individuals' ability to make sound causal judgments. The psychological process of causal inference typically operates from direct observations and experience, but in contexts such as these it relies on the ability to reason about unfamiliar domains through the processing and synthesis of information. Many non-smokers, for instance, have no *direct* experience with the harmful impacts of tobacco on the lungs. Oftentimes, they have simply made causal judgments about its detrimental health effects based on evidence presented to them in some form or another, and have set their behavior accordingly. Thus, given the pervasiveness of cause-effect judgments in an information-rich society, it is important to understand how people typically weigh and synthesize different types of evidence in making these causal determinations, which is the primary focus of this paper. More specifically, we examine how individuals presented with risk-related and policy-related decisions infer causal relationships from two distinct yet complementary forms of information: evidence of *statistical covariation* and evidence of *causal mechanisms*.

The distinction between *covariation*-based and *mechanism*-based causal evidence is most relevant to causal inference from phenomena for which true experiments are impractical or impossible to perform. Consider the example of a train crash. If we assess the most likely cause for the mishap (Was it the operator's fault? Something about the train? Something about the tracks at that particular location?), we might assess past patterns of covariation: Are there frequent train crashes at this location? Is this specific train operator known for consistently crashing trains? Has this train been involved in other crashes before? Such inquiries attempt to glean causal information purely from real-world data on past occurrences—the covariation approach to causal reasoning. Yet we might also be interested in *how* exactly the train crashed—was the operator intoxicated, or perhaps not well rested? Did the train's brakes malfunction? Such questions are more qualitative in nature and attempt to uncover the process by which a causal relationship functions—this is the mechanism approach to causal reasoning.

It is important to stress here that a causal mechanism and covariation data are not, on their own, sufficient for a causal explanation (the fact that the operator has a history of train crashes and was intoxicated will still not implicate him as the cause if we later learn that someone

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had removed a piece of the train track several hours before). Yet the bifurcation of these two evidence types highlights two important components of an established causal relationship: the observable phenomena that it produces (the covariation between cause and effect) and the more qualitative understanding of *how* that relationship comes about (the causal mechanism). This is, perhaps, why the covariation-mechanism distinction has received a substantial amount of attention in existing psychological literature, to which we now briefly turn.

1.1. Philosophy of the covariation/mechanism distinction

The debate over the relative importance of covariation and mechanism information in causal inference dates to the 18th century, when philosopher David Hume (1987) argued that causal relationships could be reduced to contingency data between a cause and effect (e.g., the room gets warm whenever the fireplace is lit), while Immanuel Kant (1965) argued that causation required some a priori knowledge of a generative force behind the relationship (e.g., heat). Recently, psychologists have constructed competing theories of human causal induction based on these two philosophies. "Covariational" models of inference stipulate that humans learn causal relationships through observing which factors covary together across situations (see Cheng & Novick, 1990; Kelley, 1973; Rescorla & Wagner, 1972 for some of the most prominent covariation models). Cause and effect, under this paradigm, is simply a matter of matching events that happen to co-occur; as Kelley (1967) stated, "the effect is attributed to that condition which is present when the effect is present and which is absent when the effect is absent" (p. 194).

Others, more in line with the Kantian view, have argued for the importance of understanding the *process* behind a causal relationship. Ahn, Kalish, Medin, and Gelman (1995) define a causal mechanism as "some component of an event which is thought to have causal force or causal necessity" (p. 303). Mechanisms describe the process by which a cause is capable of bringing about its effect, and thus rely on more qualitative considerations than purely covariation-based approaches. Proponents of a mechanism-based view of causality claim that people do not simply infer causal relationships from "surface-level" data, but assume the existence of underlying theoretical entities (refer back to the example of "heat") that are responsible for causal relationships (Glennan, 1996; Gopnik & Wellman, 1994). Causal mechanisms are thus specified as the constructs that distinguish causal learning from mere associative learning, which have been shown to differ on the neurological level (Chen, Liang, Lei, & Li, 2015). Interested readers are referred to Newsome (2003) for a more comprehensive review of the covariation and mechanism philosophical traditions.

Furthermore, the conceptual distinction between covariation and mechanism information is not limited to the sphere of this one academic debate. Researchers in decision making have proposed that assessments of risk are based on "data-driven" processes (involving evaluating of past instances of covariation) and "theory-driven" processes (involving the mental construction of plausible scenarios; see Kahneman & Tversky, 1982; Jennings, Amabile, & Ross, 1982; Hendrickx, Vlek, & Oppewal, 1989); these researchers acknowledge that assessments of risk and probability likely rely on statistical/frequency data *and* on mental scenarios outlining how a certain event might come about (Kahneman & Tversky, 1982). This distinction very closely mirrors the covariation/mechanism distinction made in other corners of the psychological literature, and while these approaches differ in the behavior they predict (causal judgments versus risk judgments), the evidentiary distinction of "what happens" versus "how it happens" cuts across multiple lines of research.

1.2. Review of experimental findings

Perhaps unsurprisingly, many experiments have confirmed that causal inferences often depend on *both* covariation evidence (e.g., Cheng, 1997; Griffiths & Tenenbaum, 2005; Mendelson & Shultz, 1976; Rescorla & Wagner, 1972) and mechanism evidence (Ahn et al., 1995; Ahn & Bailenson, 1996; Chapman & Chapman, 1967; Koslowski, Spilton, & Snipper, 1981). Yet studies on causal inference often employ experimental paradigms that focus on only one of these evidence types in the absence of the other. Some studies have investigated how observers integrate different forms of contingency evidence in causal assessment (Arcuri & Forzi, 1988; Catena, Maldonado, Perales, & Cándido, 2008), while others investigate the psychological and neural underpinnings of causal learning in "mechanical causality" paradigms (e.g., two ball colliding), which rely purely on mechanism inference (Blakemore et al., 2001; Choi & Scholl, 2006).

Only a few experiments to date have specifically examined how respondents behave when presented with covariation-based and mechanism-based evidence for the same causal relationship. Koslowski et al. (1981) found that children viewing demonstrations of physical or electrical causation (such as controlling a wheel with a switch) typically ascribed moderate strength to causal relationships supported by mechanism evidence (but no covariation evidence) and ascribed low strength to relationships supported by covariation evidence (but no mechanism evidence); Ahn et al. (1995) interpreted these results as suggesting a slightly greater role for mechanism than covariation evidence in causal reasoning. In a separate study, Ahn et al. (1995) found that judgments of causality for simple event descriptions (e.g., "Dave got sick to his stomach after eating chicken at the restaurant") were well-predicted by a linear model that included covariation and mechanism information as separate, additive terms (with covariation and mechanism evidence *against* a causal relationship being subtracted for the model). They found that mechanism evidence was generally weighted greater than covariation evidence, but did not examine interactions between the evidence types.

In a series of experiments by Fugelsang and Thompson, respondents read about an event (e.g., a traffic accident) and candidate causes that implied either believable or dubious mechanisms (e.g., caused by the brakes vs. caused by the car radio), then were administered evidence of either high or low covariation between candidate causes and effects. They hypothesized that respondents would weigh covariation evidence greater in the presence of a plausible causal mechanism than in its absence, and found mixed results: Fugelsang and Thompson (2000) found evidence of the hypothesized positive (multiplicative) interaction, Fugelsang and Thompson (2001) found no evidence for an interaction, and Fugelsang and Thompson (2003) found conflicting results across multiple experiments. Contrasting these results are those of Hendrickx et al. (1989), who investigated respondents' subjective risk judgments for various activities and found that the effect of statistical (contingency) evidence was attenuated in the presence of "scenario" information (e.g., plausible scenarios describing *how* the risk might come about). Thus, with conflicting results regarding the interaction between covariation-based and mechanism-based evidence, the functional form of the relationship between the two is unclear.

Lastly, there has been no research examining how varying degrees of covariation or mechanism evidence impact causal judgments. Since past experiments on the issue have typically relied on dichotomous stimuli (presence versus absence of evidence, or strong versus weak evidence), it is unclear whether causal evidence that is *quantified* in some way will produce linear effects on causal judgments, or instead relies on some other functional form (such as a logarithmic relationship).

1.3. Application to policy and risk

The purposes of this paper are twofold: (1) to add to and clarify existing research findings on how covariation and mechanism evidence are synthesized in causal judgments, and (2) to extend this line of research into the more applied domains of risk perception and public policy. The debate between the covariation and mechanism accounts