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## **Causal Pluralism in Philosophy:**

# **Empirical Challenges and Alternative Proposals**

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#### **Abstract**

An increasing number of arguments for causal pluralism invoke empirical psychological data. Different aspects of causal cognition—specifically, causal perception and causal inference—are thought to involve distinct cognitive processes and representations, and they thereby distinctively support transference and dependency theories of causation, respectively. We argue that this dualistic picture of causal concepts arises from methodological differences, rather than from an actual plurality of concepts. Hence, philosophical causal pluralism is not particularly supported by the empirical data. Serious engagement with cognitive science reveals that the connection between psychological concepts of causation and philosophical notions is substantially more complicated than is traditionally presumed.

#### 1. Introduction

Imagine a billiard ball rolling into a stationary ball, immediately followed by movement of the latter ball. Now imagine a healthy person who brushes against a plant and develops a rash an hour later. While both seemingly causal, these two sequences differ along many dimensions (e.g., timeframe, domain, reliability). In response, cognitive science research arguably points towards at least two distinct concepts of causation, one driven chiefly by perceptual features, the other statistical (e.g., Lombrozo 2010). At the same time, many philosophers of causation have argued—explicitly or otherwise—that the metaphysics of causation should depend partly on its psychological plausibility (e.g., Woodward 2011a, 2011b; Hitchcock 2012). These arguments are not simplistic inferences from psychological to metaphysical reality, but rather an observation that, for example, our causal concepts should be defeasibly anchored in actual relations in the world. The result of these two lines of work is a pluralistic metaphysical picture in which causation not only appears differently, but also comes in "basic and fundamentally different varieties" (Hall 2004, 1; see also Hitchcock 2003).

At a high level, the general argument-schema that unifies many different proposals of causal pluralism can be understood as:

- (1) Our lay concept(s) of causation are defeasibly correlated or connected with the metaphysical or scientific relation(s) of causation in the world
- (2) Cognitive science tells us that we have multiple distinct lay concepts of causation, realized through distinct cognitive processes and representations
- ⇒ (Conclusion) Metaphysical or scientific causal pluralism is defeasibly correct

One way to resist this argument is to challenge premise (1) by arguing that our lay concepts need not have any connection with metaphysical or scientific relations. In this paper, we instead

challenge premise (2): we argue that the appearance of multiple causal concepts in human cognition can be explained by methodological variations between communities of cognitive scientists. Moreover, we show that there are empirical data in support of complex interactions between the perceptually- and statistically-driven concepts of causation, thereby suggesting a single (perhaps complex) lay concept of causation. We conclude by showing how different empirically possible theories of causal cognition have different metaphysical implications, and so the need for philosophically-motivated cognitive science to resolve these issues.

### 2. Causal Pluralism in Cognitive Science

One cannot help but see a flying baseball break the window (not just be correlated with the breakage), or a person running at top speed because of (and not just in conjunction with) a barking dog. The standard cognitive science account of these phenomena (Michotte 1963) is that such impressions of causation result from a perceptually driven concept characterized by signature spatiotemporal features (e.g., spatiotemporal contiguity between purported cause and effect). The resulting causal perception exhibits a set of distinctive features: automatic, phenomenologically instantaneous, unamenable to top-down influences, and highly sensitive to spatiotemporal features. Causal perception has largely been studied through variations on the direct launching paradigm (Michotte 1963; Scholl and Tremoulet 2000): a stationary object *A* is on screen; a moving object *B* enters the screen and moves until it contacts object *A*; at that point, object *B* stops while object *A* moves until it disappears from screen. In ordinary circumstances, participants invariably claim that the moving object kicked, pushed, or launched the stationary object. Notably, a delay between contact and motion, or a gap between the two object at movement onset, destroys any impression of causality. Different spatiotemporal features can

signal different causal processes, though in all cases, causal perception emerges automatically without explicit reasoning.

In contrast, so-called causal inference<sup>1</sup> involves learning about causal relations from information about covariation, contingency, and other statistical information. This information might come from observed correlations (Rottman and Keil 2012) or interventions (Steyvers et al. 2003). For example, one often needs some data or trial-and-error to infer that an infant is crying because of a rash rather than hunger. Direct spatiotemporal connection is not a useful guide for this type of causal cognition (though see below). Strength judgments of a causal relation are sensitive to the degree of covariation between a purported cause and its effect (Shanks and Dickinson 1987; Buehner, Cheng, and Clifford 2003). Statistics also support causal structure learning or the ability to determine how different causal variables relate to one another (Griffiths and Tenenbaum 2005; Lu et al. 2008). In contrast with causal perception, causal inference is: cognitively effortful, has non-salient phenomenology, is largely independent of spatiotemporal features, and strongly amenable to top-down cognitive influences (Buehner and May 2002).

The different behavioral manifestations of the perceptual and statistical concepts of causation—causal perception and causal inference, respectively—are often taken to suggest that these concepts rely on distinct cognitive processes or systems. This suggestion is further supported by a behavioral study (Schlottmann and Shanks 1992), in which participants anecdotally reported that they "knew the collision was not necessary for Object B to move, but

<sup>&</sup>lt;sup>1</sup> The name is somewhat unfortunate, as causal perception arguably also involves some inferences. Nonetheless, 'causal inference' is the term used in cognitive science to refer to this kind of statistics-driving causal learning.

that it just looked as if it should be" (338). Further evidence for causal pluralism comes from neuroscientific research, which demonstrates a clear differentiation in the brain networks activated during causal perception and causal inference. The perception of causal launching events, compared to that of non-causal launching events, was accompanied by a higher activation level in bilateral V5/MT/MST, the superior temporal sulcus and the left intraparietal sulcus (Blakemore et al. 2001). These areas are involved in complex visual processing, which suggests that causal perception might involve the recovery of causal structures in an event from motion cues (Fugelsang and Dunbar 2009). In contrast, inferential or statistical tasks with causation involved the activation of prefrontal and occipital cortices, precentral gyrus, and parahippocampal gyrus when the data conformed to participants' expectations. A slightly different network—the anterior cingulate, left dorsolateral prefrontal cortex, and the precuneus—was activated when the data were incongruent with expectations (Fugelsang and Dunbar, 2005). Notably, all of these brain areas are typically associated with 'higher' cognition, such as decision-making, conflict resolution, and information integration. Additionally, patients who had a corpus callosotomy (severing the connection between brain hemispheres, usually to treat epilepsy) exhibited a double dissociation between causal perception (seemingly) localizing in the right hemisphere and causal inference (seemingly) in the left hemisphere (Roser et al. 2005). Insofar as one commits to the thesis that different brain network activations imply different brain mechanisms, these neuroscientific results all seem to imply that causal perception and causal inference recruit two different learning mechanisms.

On top of all of these results, causal perception and causal inference also seem to develop at different points during childhood. Humans develop sensitivity for rudimentary cues to causality such as spatial contiguity between 4 and 5½ months of age (Cohen and Amsel 1998), and

perceive direct launching as a causal event based on the appropriate causal roles between 6½ and 10 months of age (Leslie and Keeble 1987; Oakes and Cohen 1990). By 15 months, infants can perceive a three-object causal chain (in which the first object launches the second, which in turn activates the third) as involving a causal relationship between the first and third object (Cohen et al. 1999). In contrast, humans do not successfully solve the blicket detector task—a classic paradigm in causal inference research—until roughly two years of age (Gopnik et al. 2004; Sobel and Kirkham 2006). Children develop more complex causal reasoning abilities, such as the ability to infer unobserved causes (Schulz and Sommerville 2006) and integrate base rates (Griffiths et al. 2011), by four years of age, significantly later than all of the causal perception capacities. The different developmental timelines lend further empirical support to the initial claim that causal perception and causal inference result from two different cognitive processes and representations, which in turn depend on distinct psychological concepts of causation.

# 3. Causal Pluralism in Philosophy

Many metaphysical accounts of causation can be organized into two clusters of theories: transference and dependency. Transference theorists typically define causation by a transfer of energy or a conservation of quantities through transformation (e.g., Salmon 1984; Dowe 1992, 2000), all of which have signature spatiotemporal properties. For example, in a collision event between two billiard balls, spatiotemporal contiguity during contact enables a transfer of momentum from each ball to the other. In contrast, dependency accounts of causation characterize (though not necessarily define) a causal relation between two factors by their statistical relationship: generative causes make their effect more likely; preventative causes make their effect less likely. This statistical dependency is then grounded in different ways by different

authors, such as counterfactuals, hypothetical interventions, or statistical differences in appropriate reference classes (e.g., Lewis 1973; Woodward 2009).

Rather than arguing for one type of metaphysical account to the exclusion of the other, causal pluralists argue for the co-existence of transference and dependency as distinct kinds of causation that govern different phenomena (Godfrey-Smith 2009). Although arguments for these accounts can be purely metaphysical, a significant subset derive force from the empirical plausibility of these accounts (e.g., Hitchcock 2003; Woodward 2006, 2011a, 2011b; Hall 2004; Lombrozo 2010). The empirical observation that humans exhibit different behaviors and shift their criteria for causation in different scenarios, as presented above, is ostensibly a natural consequence of distinct philosophical causal concepts. For example, there is a straightforward mapping between the signature criteria for causation in many transference accounts and the specific spatiotemporal conditions encoded in the perceptually realized concept of causation. In particular, the immediacy and experiential richness of causal perception is inexplicable by many existing dependency theories of causation, but is more readily explained if causation involves transference (Wolff 2008; Beebee 2009). In the other direction, causal inference can often take place even in the absence of any obvious transfer of force or quantity between agent and recipient, such as cases of prevention in which there is no direct physical connection at all. This kind of causal cognition is often taken to support a dependency notion of causation (Woodward 2009). Causal pluralism seems to explain a wide variety of human causal intuitions—those of both laypeople and philosophers—at the cost of only a slightly more crowded ontology.

#### 4. Causal Pluralism in Cognitive Science: A Methodological Analysis

The previous sections presented the "standard view" in cognitive science of causal learning as consisting of two distinct cognitive processes and representations, and noted the important role that it plays in philosophical arguments about the nature of causation. In this section, we challenge the premise that the standard (cognitive science) view is well-supported. In particular, we contend that there are three key methodological differences between causal perception and causal inference experiments, and those differences can explain the differences in observed phenomena without appeal to multiple cognitive processes.

First, the presentation formats and response measures drastically differ between causal perception and causal inference experiments. In the former, participants usually watch a single event and answer questions about that particular event (Scholl and Nakayama 2002). Additionally, participants typically answer a forced-choice question of whether a causal relation exists, or give a quantitative rating of the extent to which a purported causal relation exists in this particular event. In contrast, causal inference experiments involve trial-by-trial presentations of cases (Fernbach and Sloman 2009) or a contingency table summarizing those cases (Hagmayer and Waldmann 2002). Moreover, the typical causal inference measures include (but are not limited to): ratings of proportions in sets of counterfactuals; numeric ratings of the strength of the cause; a categorical choices between causal models; construction or drawing of causal graphs; measures of intervention choices or post-intervention predictions; and more. Neuroscientific and developmental research on causal perception and causal inference typically use the same kinds of stimuli and measures as the corresponding cognitive/behavioral studies. Hence, the stimuli and measures lead directly to phenomenological and behavioral differences without any strong empirical justification, and—as we show next—vastly divergent theories.

Second, our theories of causal perception and causal inference aim to explain judgments about different things, but without directly considering whether they are distinct processes.

Causal inference theories aim to explain participant judgments that are based on multiple cases, and that generalize to future instances. In contrast, causal perception theories aim to explain judgments about single cases with no expectation of generalization. That is, causal perception judgments are about token events whereas causal inference judgments are about types. The very construction of the theories thus precludes direct comparison, since they do not attempt to explain the same phenomena. Moreover, the focus of each theory closely correlates with its experimental methods: experientially rich, automatic causal perception can only be captured by judgments of singular events; explicit statistical causal inference can only occur in judgments of multiple instances. The appearance of empirical difference between causal perception and causal inference can be explained by these methodological and focus divergences without any need to appeal to underlying differences in concepts or representations.

A third set of issues arises for the neuroscientific studies, which one might have thought to be immune from the other two worries. Those studies all relied on subtraction methods in which the brain activation map of a null condition is subtracted from that of the experimental conditions. This calculation reveals areas that are uniquely activated in the experimental conditions, yet omits areas that are commonly activated across conditions. Additionally, that an area activates more strongly during causal inference does not mean it is unactivated during causal perception, and vice versa. Cognitive scientists need a thorough investigation of the common areas of activation and the interactions between different brain regions during causal perception and causal inference. Liberal interpretations of early neuroimaging data without sensitivity to these

nuances could produce an overstated conclusion about distinct underlying brain networks for causal perception and causal inference.

### 5. Cognitive Science in Support of Causal Monism

A careful look at the cognitive science not only undermines the putative inference for pluralism in causal cognition, but actually provides some positive evidence for monism in causal cognition. In particular, consider causal inference and reasoning studies that use both mechanical and statistical information. For example, Kushnir and Gopnik (2007) tested 4-year-old children with a modified blicket detector task in which (i) statistical information matched standard blicket studies in which children infer causality; but (ii) objects were held over the machine rather than placed on it. Children in that task were more inclined to say that such objects were *not* related to the machine's activation; they smoothly integrated spatiotemporal information and constraints into a (seeming) causal inference task. Similarly, Schlottmann (1999) introduced 5-, 7-, 9-, and 10-year-old children to two systems whose inner mechanisms were hidden from view, but described as different. The experimenter dropped one ball (A) into one end of the system, followed by another ball (B) after 3 seconds; the bell rang roughly 1 second after that (i.e., the observed sequence was A-pause-B-bell). The experimenter then showed children the system mechanisms: the fast system had a two-arm seesaw system that rings the bell almost immediately; the slow system had a downward ramp along which the ball had to roll. Intuitively, the children should make different inferences about which ball caused the bell ring: ball A in the slow system and ball B in the fast system. Most children could diagnose the likely mechanism when only one ball was dropped, but 5- and 7-year-old children had difficulty predicting a delay in ringing even when they saw that the slow mechanism was at work. That is, children's causal

learning and inference up to 7 years of age depended on both statistical data accumulated over trials and the spatiotemporal contiguity cues of the events.

Spatiotemporal contiguity shapes causal judgments even among adults. In a series of experiments, Buehner and May (2002) tested the effect of prior knowledge about the time course of a causal relation on their causal judgments of observed contingencies. Participants were given two scenarios: either a light switch immediately causes a light bulb to turn on, or a grenade launch leads to detonation only after a delay. Experiment 1 used a within-subjects design, meaning that participants completed all experimental conditions. Prior experimentation suggested that these two scenarios produced different explicit assumptions about the causal time course, but results showed almost no difference between participants' causal ratings of the light switch and of the grenade launcher: as the delay period between purported cause and effect increased, participants' causal ratings of the cause decreased regardless of the domain or cover story. Buehner and May's preferred explanation is that the 'pull' of the temporal contiguity in the light switch scenario was so strong that it skewed participants' ratings and overshadowed their assumptions about delayed timeframes in the grenade condition. Even if their explanation is incorrect, spatiotemporal cues clearly play a significant role in adult causal inference.

If the previous section provides methodological reasons for doubting the empirical distinction between the perceptual and statistic concepts of causation, this section offers evidence that these concepts are more interconnected than previously thought. We suggest that a pluralism of psychological causal concepts is currently unwarranted by the data, and cautiously propose that monism should again be a feasible theoretical candidate. This monism clearly must allow different types of input, ranging from spatiotemporal cues to frequency and contingency information, but those could lead to distinct behaviors in light of variation in information and

task demands. Admittedly, many details remain to be provided about this kind of monism, but premise (2) in the original argument-schema clearly does not hold as straightforwardly as has been assumed in the philosophical literature. We now turn to the philosophical import of different possible ways of developing a monist account of causal cognition and learning.

## 6. Philosophical Implications of Alternative Cognitive Accounts

If we do not accept the standard view of pluralism in causal cognition, then we should consider some plausible alternatives. First, we could prioritize the developmental data, which suggest that the perceptual notion of causation develops first, and then the statistical or dependency notion emerges from it. As young learners gain perceptual exposure to simple causal events such as collision or pulling, their mental representations of these events include relevant perceptual features. Further exposure to new instances can result in automaticity of processing, which thereby manifests behaviorally as causal perception. For more complex causal events, some of the causal representations might not bear the same perceptual characteristics as those previously learned. Patterns of characteristics over multiple token perceptions can provide input to the later-developing statistical notion of causation, which are originally grounded in abstraction over spatiotemporal features. The resulting two concepts might develop to be distinct in adults; the developmental story underdetermines the final number of causal concepts in adults. On this theory, a transference notion of causation provides the historical basis for all causal judgments, and perhaps the actual conceptual basis if the concepts are *not* independent in adults. That is, a true causal relation must involve either a transference of energy or a preservation of some quantity from state to state during transformation, where those spatiotemporal features might be imputed on a system from statistical data.

Second, the order of development of causal cognition could be reversed: humans might develop the statistical notion of causation before the perceptual one. In particular, if causal inference occurs implicitly, then repeated exposures to a causal relation could enable learners to construct a representation of this causal relation that encodes statistical information, such as the frequency of occurrence of the purported effect (base rate), the strength of covariation between cause and effect, and so on. In practice, many of these relations are highly reliable or completely deterministic, and all exhibit reliable spatiotemporal characteristics (e.g., contiguity). Thus, the perceptual features of spatiotemporal contiguity might be encoded alongside—and perhaps even stand in for—statistical information: seeing that a rolling ball makes contact with a stationary ball is sufficient for the prediction that the stationary ball is likely to start moving. A philosophical monist account inspired by this psychological picture would take the dependency notion of causation as fundamental. Notably, causation in the physical, mechanical world typically has (statistically) reliable features such the appearance of determinism, or the ubiquity of spatiotemporal contiguity. The dependency framework can thus account for cases that are typically characterized by spatiotemporal features alone (such as causal perception): those features indicate an underlying, deterministic causal relation (see also Woodward 2011a).

Third, a unitary concept of causation might underpin all of human causal cognition.

Importantly, this underlying concept is irreducible to either the perceptual or the statistical concept alone, but is rather *inferred* from features of the seemingly distinct types of cognition. In this theory, people infer the existence of an unobserved causal relation using essentially anything that might be relevant, whether spatiotemporal constraints, information about mechanisms, or reliability of control interventions. One can even imagine other types of information being relevant in one's causal judgment, such as color: the color of a mushroom might suggest its

toxicity. These different information sources are integrated to infer the causal connection (if any), which could potentially require significant tradeoffs by the cognizer. Critically, in the case of events where people have had substantial 'perceptual practice' such as collision events, the spatiotemporal contiguity of the event might be the most important. In the case of events with only statistical information (e.g., determining if the peanuts or the shrimps caused an allergic reaction), people can use other types of information to both infer the relation and to suggest alternative control actions. On this account, causal perception and causal inference are only different behavioral manifestations—in response to different task demands and stimuli types—of the same process(es) and mechanisms whose goal is to yield usable representations of the causal web of the world. To the extent that a unitary concept of causation is psychologically plausible and distinct from the previous two alternatives, the third resulting monistic account of causation might plausibly depart from transference and dependency accounts in various ways. Instead of solely relying on transference or dependency between cause and effect, causation can be characterized by both, and which features are more salient depend on how epistemically accessible they are. In the case of billiard balls colliding into each other, the epistemically accessible features are the contact between the balls and the immediacy with which they move differently upon contact. In the case of prevention (e.g., plugging a hole in the sink prevents the leakage), the epistemically accessible features include the absence of leakage after plugging, and that leakage continues when one fails to plug the hole.

#### 7. Conclusion

Many variants of causal pluralism in philosophy, most of which lean on the distinction between transference-based and dependency-based causation, map onto the parallel development of two research clusters in cognitive science: the perceptually- and statistically-driven concepts of causation, respectively. In this paper, we argued that the foundation of the dualistic research program in cognitive science is shaky insofar as it traces an artifactual divergence between research paradigms and measures, rather than a natural fault line in the empirical landscape of causal cognition. We then discussed how cognitive science might point to a version of causal monism that includes interactions between the perceptual and statistical concepts of causation. Finally, we sketched three alternative accounts of how the statistical and perceptual concepts of causation might relate to each other, and briefly discussed the implications each of those account would have on the philosophical picture of causation. This sketch is not an endorsement of any particular theory, and the three alternatives are non-exhaustive: There are certainly other possibilities that we cannot explore here due to space limits. Rather, the sketch is an invitation for philosophers and psychologists alike to consider these un- and under-explored alternatives before settling for any particular theory.

#### References

- Beebee, Helen, Christopher Hitchcock, and Peter Menzies, eds. 2009. *The Oxford Handbook of Causation*. Oxford University Press.
- Beebee, Helen. 2009. "Causation and Observation." In Beebee et al. 2009, 471–97.
- Blakemore, Sarah-Jayne, Pierre Fonlupt, Mathilde Pachot-Clouard, Céline Darmon, Pascal Boyer, Andrew N. Meltzoff, Christoph Segebarth, and Jean Decety. 2001. "How the brain perceives causality: an event-related fMRI study." *Neuroreport* 12(17):3741–46.
- Buehner, Marc J., Patricia W. Cheng, and Deborah Clifford. 2003. "From covariation to causation: a test of the assumption of causal power." *Journal of Experimental Psychology: Learning, Memory, and Cognition* 29(6):1119–40.
- Buehner, Marc J., and Jon May. 2002. "Knowledge mediates the timeframe of covariation assessment in human causal induction." *Thinking and Reasoning* 8(4):269–95.
- Cohen, Leslie B., and Geoffrey Amsel. 1998. "Precursors to infants' perception of the causality of a simple event." *Infant Behavior and Development* 21(4):713–31.
- Cohen, Leslie B., Leslie J. Rundell, Barbara A. Spellman, and Cara H. Cashon. 1999. "Infants' perception of causal chains." *Psychological Science* 10(5):412–18.
- Dowe, Phil. 1992. "Process causality and asymmetry." Erkenntnis 37(2):179-96.
- ——. 2000. *Physical Causation*. Cambridge: Cambridge University Press.
- Fernbach, Philip M., and Steven A. Sloman. 2009. "Causal learning with local computations." Journal of Experimental Psychology: Learning, Memory, and Cognition 35(3):678–93.

- Fugelsang, Jonathan A., and Kevin N. Dunbar. 2005. "Brain-based mechanisms underlying complex causal thinking." *Neuropsychologia* 43(8):1204–13.
- 2009. "Brain-based mechanisms underlying causal reasoning." In *Neural correlates of thinking*. On thinking, vol. 1, ed. Eduard Kraft., Balázs Gulyás, and Ernst Pöppel, 269–79. Springer, Berlin, Heidelberg.
- Godfrey-Smith, Peter. 2009. "Causal pluralism." In Beebee et al. 2009, 326–37.
- Gopnik, Alison, Clark Glymour, David M. Sobel, Laura E. Schulz, Tamar Kushnir, and David Danks. 2004. "A theory of causal learning in children: causal maps and Bayes nets."

  \*Psychological Review 111(1):3–32.
- Griffiths, Thomas L., David M. Sobel, Joshua B. Tenenbaum, and Alison Gopnik. 2011. "Bayes and blickets: Effects of knowledge on causal induction in children and adults." *Cognitive Science* 35(8):1407–55.
- Griffiths, Thomas L., and Joshua B. Tenenbaum. 2005. "Structure and strength in causal induction." *Cognitive Psychology* 51(4):334–84.
- Hagmayer, York, and Michael R. Waldmann. 2002. "How temporal assumptions influence causal judgments." *Memory and Cognition* 30(7):1128–37.
- Hall, Ned. 2004. "Two concepts of causation." In *Causation and Counterfactuals*, ed. JohnDavid Collins, Edward J. Hall, and Laurie Ann Paul, 225–76. Cambridge, Massachusetts:MIT Press.
- Hitchcock, Christopher. 2003. "Of Humean Bondage." *British Journal for the Philosophy of Science* 54:1–25.

- ———. 2012. "Portable causal dependence: A tale of consilience." *Philosophy of Science* 79(5):942–51.
- Kushnir, Tamar, and Alison Gopnik. 2007. "Conditional probability versus spatial contiguity in causal learning: Preschoolers use new contingency evidence to overcome prior spatial assumptions." *Developmental Psychology* 43:186–96.
- Leslie, Alan M. and Stephanie Keeble. 1987. Do six-month-old infants perceive causality?. *Cognition* 25(3):265–88.
- Lewis, David. 1973. "Causation." Journal of Philosophy 70(17):556-67.
- Lombrozo, Tania. 2010. "Causal–explanatory pluralism: How intentions, functions, and mechanisms influence causal ascriptions." *Cognitive Psychology* 61(4):303–32.
- Lu, Hongjing, Alan L. Yuille, Mimi Liljeholm, Patricia W. Cheng, and Keith J. Holyoak. 2008.

  "Bayesian generic priors for causal learning." *Psychological Review* 115(4):955–84.
- Michotte, Albert. 1963. *The Perception of Causality*. Trans. T. R. Miles and E. Miles. Andover, Hants: Methuen. Original work published in 1946.
- Oakes, Lisa M., and Leslie B. Cohen. 1990. "Infant perception of a causal event." *Cognitive Development* 5(2):193–207.
- Roser, Matthew E., Jonathan A. Fugelsang, Kevin N. Dunbar, Paul M. Corballis, and Michael S. Gazzaniga. 2005. Dissociating processes supporting causal perception and causal inference in the brain. *Neuropsychology* 19(5):591–602.
- Rottman, Benjamin M., and Frank C. Keil. 2012. "Causal structure learning over time: Observations and interventions." *Cognitive Psychology* 64(1-2):93–125.

- Salmon, Wesley. 1984. Scientific Explanation and the Causal Structure of the World. Princeton, New Jersey: Princeton University Press.
- Schlottmann, Anne. 1999. "Seeing it happen and knowing how it works: How children understand the relation between perceptual causality and underlying mechanism." Developmental Psychology 35(1): 303–17.
- Schlottmann, Anne, and David R. Shanks. 1992. "Evidence for a distinction between judged and perceived causality." *The Quarterly Journal of Experimental Psychology* 44(2):321–42.
- Scholl, Brian J., and Patrice D. Tremoulet. 2000. "Perceptual causality and animacy." *Trends in Cognitive Sciences* 4(8):299–309.
- Scholl, Brian J., and Ken Nakayama. 2002. "Causal capture: Contextual effects on the perception of collision events." *Psychological Science* 13(6):493–98.
- Schulz, Laura E. and Jessica Sommerville. 2006. God does not play dice: Causal determinism and children's inferences about unobserved causes. *Child Development* 77(2):427–42.
- Shanks, David R. and Anthony Dickinson. 1987. "Associative accounts of causality judgment."

  The Psychology of Learning and Motivation 21:229–61.
- Sobel, David M., and Natasha Z. Kirkham. 2006. "Blickets and babies: the development of causal reasoning in toddlers and infants." *Developmental Psychology* 42(6):1103–15.
- Steyvers, Mark, Joshua B. Tenenbaum, Eric-Jan Wagenmakers, and Ben Blum. 2003. "Inferring causal networks from observations and interventions." *Cognitive science* 27(3):453–89.

Press.

Wolff, Phillip. 2008. "Dynamics and the perception of causal events." In *Understanding Events*: