

Causal Thinking

Michael R. Waldmann

Abstract The ability to acquire and reason with causal knowledge belongs to our most central cognitive competencies. Causal knowledge serves various functions: It enables us to predict future events, to diagnose the causes of observed events, and to choose the right actions to achieve our goals. The chapter gives an overview of the causal-model approach to causal reasoning and learning. It focuses on the contrast between traditional associationist theories and this more recent rational approach to causal thinking, and discusses this theory in light of recent experimental evidence.

1 Introduction

The ability of people to predict future events, to explain past events, and to choose appropriate actions to achieve goals belongs to the most central cognitive competencies that allow us to be successful agents in the world. How is knowledge about regularities in the world learned, stored, and accessed? A plausible theory that governs our intuitive thinking assumes that *causality* is the “cement of the universe” (Mackie 1974), which underlies the orderly relations between events. According to this view some event types, causes, have the capacity or power to generate their effects through hidden mechanisms.

The philosopher David Hume questioned this view in his seminal writings (e.g., Hume 1748/1977). He looked at situations in which he observed causal relations, and did not detect any empirical features that might correspond to evidence for causal powers. What he found instead was spatio-temporally ordered successions of event pairs, but nothing beyond that might correspond to power.

The psychology of learning has adopted Hume’s view by focusing on spatio-temporally ordered events. According to many learning theories, causal predictions are driven by associative relations that have been learned on the basis

M.R. Waldmann

Department of Psychology, University of Göttingen, Gosslerstr. 14, 37073, Göttingen, Germany
e-mail: michael.waldmann@bio.uni-goettingen.de

of observed covariations between events (e.g., Allan 1993; Shanks and Dickinson 1987). Similar to Pavlov's dog which has learned to predict food when it hears a tone (i.e., classical conditioning), or to a pigeon's learning that a lever press produces food (i.e., instrumental conditioning), we learn about predictive relations between observed events or interventional relations between our acts and their outcomes. According to the associationist view, there is no need for the concept of causality. Thus, consistent with the epistemology of empiricism the concept of causality as referring to causal powers was dropped and replaced by predictive covariational relations between observable events.

Hume's analysis leaves us with a puzzle. On the one hand, he seems to have correctly observed that covariations between observable events are the primary perceptual input for causal inductions. On the other hand, many researchers agree that when thinking about the world we go beyond the information given by assuming hidden capacities, forces, or processes beyond the surface of orderly event successions (Ahn et al. 1995; Cheng 1997). The following overview will summarize research showing that people (and some animals) indeed go beyond covariations in causal learning and reasoning.

2 Causal-Model Theory: Beyond Covariations

Hume seems to have correctly observed that the *input of causal learning* largely consists of covariation information. It can be shown, however, that mental representations that merely mirror this input cannot explain the competencies people have when dealing with causal situations (see also Buehner and Cheng 2005; Waldmann et al. 2006; Waldmann et al. 2008; Waldmann 1996, for overviews of causal-model theory). If we had no *causal* knowledge we could not represent the difference between causal and noncausal spurious statistical relations. For example, in the common-cause model in Fig. 1 the *Midosis* virus is a direct cause of two effects, symptom 1 and symptom 2. Inserting this virus into an organism would produce both effects. However, causing symptom 1 by other means would not affect symptom 2. Whereas the virus is causally related to either effect, the two effects only covary but are not causally related (i.e., spurious noncausal correlation).

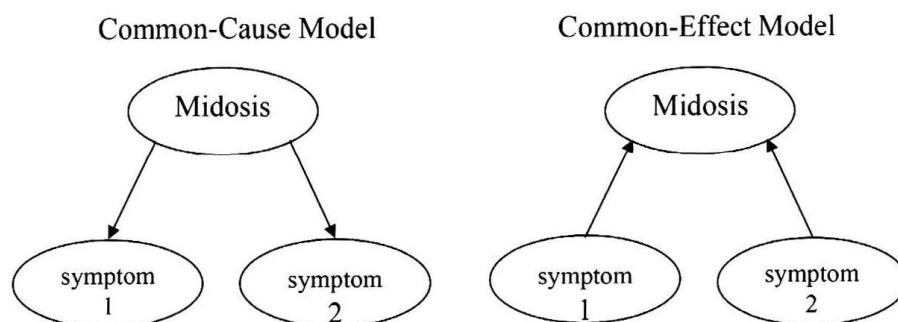


Fig. 1 Examples of a common-cause and common-effect model. The *arrows* represent causal relations directed from causes to effects

The difference between these two types of relations cannot be represented by theories that are only sensitive to statistical covariations (e.g., associative theories), whereas they are of utmost importance when we reason about the outcomes of interventions.

Covariational knowledge also fails to make the fundamental distinction between causes and effects, which is central for causality. Whereas causes are typically correlated with effects, and effects are typically correlated with causes, causal relations are asymmetric. Causes generate effects and not vice versa. Again, this distinction is important when we reason about causal systems. We can use information about causes to predict effects, and information about effects to diagnose their causes. However, we can only intervene in causes to achieve effects but not vice versa. Again we need to go beyond covariational information to correctly represent this knowledge.

Finally, causal models entail statistical relations between events that are helpful in learning. For example, multiple effects of a common cause but not multiple causes of a common effect are correlated in a predictable way. These correlations entailed by the structure of causal models allow us to limit the complexity of the representations we need to represent the covariations entailed by causal knowledge.

How can Hume's insight that the observational input only offers covariation information be reconciled with the observation that we represent our world in terms of causal models endowed with powers and mechanisms? A possible answer is that Hume's empiricist epistemology was incomplete. As many philosophers of science have revealed, apart from concepts referring to observable events (e.g., covariations) our theories also contain theoretical concepts which are only indirectly tied to the observable data (e.g., causal powers). Theoretical concepts are components of theories which specify how they can be estimated from data in a specific situation. Causal-model theory and related accounts claim that we have a tendency to assume the existence of deep causal relations behind the visible surface, which leads us to align covariational input with causal-model representations (see also Buehner and Cheng 2005). Similar to using sparse visual input to induce 3D object representations that go beyond the learning input, people have a tendency to represent some events as causes with the power to generate or prevent effects, and they build causal networks that can be used for inferences and planning (see Cheng 1997; Tenenbaum and Griffiths 2003; Waldmann et al. 2006, 2008).

In the following sections, I will first discuss empirical studies testing causal-model theory showing that humans (and some nonhuman animals) indeed go beyond covariations to infer causal structure (see also Gopnik et al. 2004; Lu et al. 2008; Sloman 2005; Tenenbaum and Griffiths 2003, for related views). In the final section I will add some speculations on the processes underlying these competencies.

2.1 Sensitivity to the Asymmetry of Causes and Effects

The distinction between causes and effects is a central feature of causal representations. Thus, one line of our research focused on whether there is evidence that people are sensitive to this distinction (see Waldmann 1996, for a summary of early work).

Fenker et al. (2005) have investigated this question in a *semantic memory task*. We were interested in whether causal relations are represented and accessed differently from associative relations in semantic memory. In the experiments, participants were shown pairs of words, one after another, either describing events that referred to a cause (e.g., spark) or an effect (e.g., fire) of a causal relation. Both the temporal order of word presentation and the question to which participants had to respond was manipulated. Interestingly, when we asked whether the two events are *causally* related, participants answered faster when the first word referred to a cause and the second word to its effect than vice versa. No such asymmetry was observed, however, when we asked about associative relations. The associative questions probed participants whether the words describing the events are related in some meaningful way. People appear to distinguish the roles of cause and effect when queried specifically about a causal relation, but not when the same information is evaluated for the presence of an associative relation.

In a follow-up study, a functional magnetic resonance imaging experiment was performed to investigate the hypothesized dissociation between the use of semantic knowledge to evaluate specifically causal relations in contrast to general associative relations (Satpute et al. 2005). Again, identical pairs of words were judged for causal or associative relations in different blocks of trials. Causal judgments, beyond associative judgments, generated distinct activation in left dorsolateral prefrontal cortex and right precuneus. These findings indicate that the evaluation of causal relations in semantic memory involves additional neural mechanisms relative to those required to evaluate associative relations.

Whereas semantic memory tasks target the results of learning, there is also evidence that people go beyond covariations in trial-by-trial learning tasks (see Waldmann and Holyoak 1992; Waldmann et al. 1995; Waldmann 2000). The general paradigm presents participants in different conditions with identical covarying events. If Hume was right, and learning simply consists of processing these observed spatio-temporally ordered covariations, the outcome of the learning process should be the same. However, if participants go beyond covariations and form representations of the underlying causal models, their reasoning should be sensitive to the structure of these models. In one study Waldmann (2001) presented learners first with cues that represented substances in hypothetical patients' blood and then gave feedback about fictitious hematological diseases (e.g., *Midosis*). Two conditions manipulated – through initial instructions – whether learners interpreted the substances (i.e., cues) as effects of the diseases (common-cause model) or as causes (common-effect model)(see Fig. 1). In the common-cause condition, participants were told that the diseases caused some of the substances in the blood which could be used to diagnose the diseases. In contrast, in the common-effect condition the very same substances were described as coming from food items, which were suspected to be causing the novel blood diseases.

According to associative learning theories, learners should learn to predict the diseases from information about the presence or absence of the substances, which was always provided first as cues in the learning trials. Since cues and outcomes were identical in both conditions, the learning process should be identical. In contrast, causal-model theory predicts that learners are sensitive to the distinction between

cues that represent causes versus effects, which should influence the learning and reasoning processes. The results showed that causal models indeed affected reasoning. Learners treated the substances as potentially competing explanations of the disease in the common-effect condition, whereas the substances were treated as collateral, collaborating effects of a common cause in the contrasting condition. Thus, despite the fact that all learners observed the same sequence of events, they assigned different causal roles to these events, and consequently made different inferences.

2.2 *Predicting Outcomes of Hypothetical Observations Versus Hypothetical Interventions*

Predicting or diagnosing on the basis of real or hypothetical observed events (e.g., observed substances) are both examples of observational inferences. We may also be confronted with the task to predict or diagnose on the basis of hypothetical interventions. Sometimes these two types of predictions coincide, but very often they do not. For example, hypothetically observing symptom 1 in the common-cause model depicted in Fig. 1 allows us to diagnose the *Midosis* virus and infer symptom 2 from there. However, a hypothetical intervention that causes symptom 1 by other means should not change our inferences about the virus and the other symptom. Again, to make correct inferences here, the learner needs to go beyond the given covariations, and assign causal roles to the observed events.

An associative learning theorist might react to this example arguing that human and nonhuman animals could distinguish between observing and intervening on the basis of observational (i.e., classical) and instrumental conditioning. We may, for example, have learned that symptom 1 predicts symptom 2 in an observational learning setting and in parallel may have tried to cause symptom 1 by other means with no effect on the virus and the other symptom. This solution only works, however, if learners are provided with both kinds of learning experiences, not if they only passively observe covarying events and then are requested to make both observational and interventional predictions.

Waldmann and Hagmayer (2005) tested people's competence to derive predictions for hypothetical observations and hypothetical interventions from causal models that had been learned purely through observation (see also Hagmayer et al. 2007; Sloman and Lagnado 2005). In a fictitious scenario, participants were either told that scientists hypothesized that the three hormones *pixin*, *sonin*, and *xanthan* form a common-cause or a causal chain model in animals (see Fig. 2). All participants in the two conditions received identical observational data indicating that the three hormones were connected by probabilistic causal relations. In the subsequent test phase learners were asked to make predictions about hypothetical observations of *sonin* in new animals, and hypothetical interventions, which increased *sonin* in the blood of new animals by means of inoculations. (In other conditions the *sonin* levels were hypothetically decreased.)

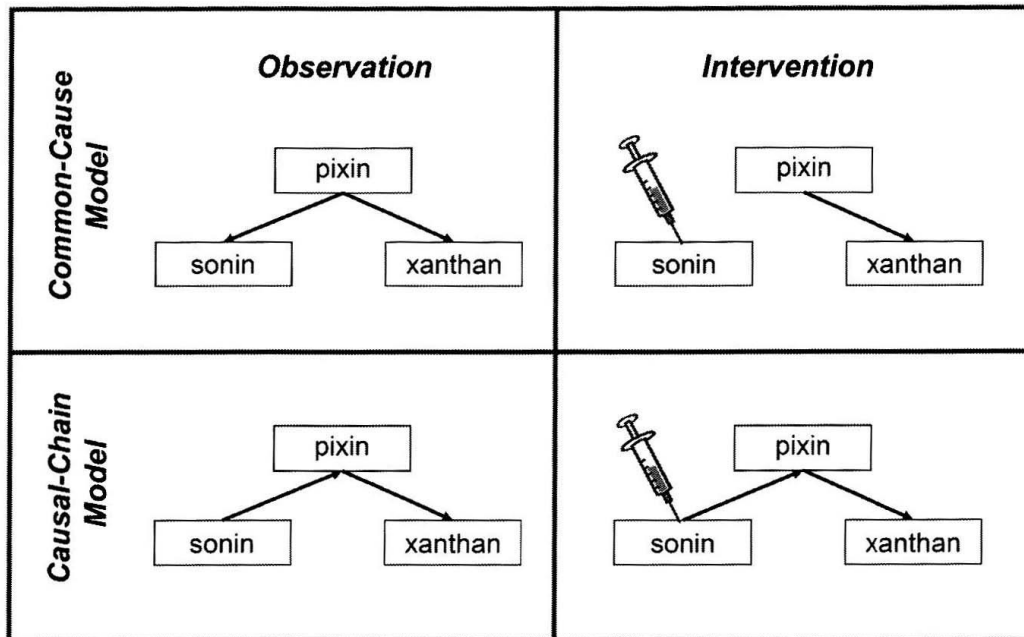


Fig. 2 Observational and interventional predictions in a common-cause and causal-chain model, in which three hormones are causally connected. The *left side* shows the models presented in the learning phase which can be used for observational predictions. The *right side* depicts the models underlying the predictions of the outcomes of hypothetical interventions. An intervention in *sonin* leads to full discounting of *pixin* in the common-cause but not in the chain model. Full discounting can be expressed by removing the *arrow* from *pixin* to *sonin*, which turns these two substances statistically independent in the test situation

The observational inferences can be modeled on the basis of the two presented causal models. Since the three events are statistically related in both causal models, the observation of the presence of *sonin* allows us to reason that *pixin* and consequently *xanthan* are also very likely to be present. Interventional predictions entail different predictions in one of the models. An intervention that adds *sonin* to the blood leads in the common-cause model to the consequence that the levels of *sonin* are now determined by this intervention and no longer by its usual cause (*pixin*), whose causal influence is preempted by the novel intervention (see Spirtes et al. 1993; Pearl 2000; Woodward 2003). One way to model this intervention is to remove the arrow from *sonin*'s normal cause, *pixin*, that is being explained away by the new intervention (see also Waldmann et al. 2008, for a more general theory). The removal expresses that *pixin* is no longer a cause of *sonin* (see Fig. 2, right) in the test situation. Due to the removal of the arrow in the common-cause model *sonin* becomes independent of *xanthan* so that regardless of whether *sonin* is increased or decreased by an intervention, the level of *xanthan* should remain at an identical level.

The chain condition served as a control that showed that observing and intervening do not always lead to different predictions (see Fig. 2). Since there are no alternative causes of *sonin* that are being discounted, there should be no difference whether *sonin* is hypothetically observed or generated by an intervention in this

model. As a consequence, participants should make identical predictions for the hypothetical observational and interventional questions in the chain condition. In our experiments, participants' responses corresponded to these predictions remarkably well. They were capable of predicting patterns they had never observed, which indicates that despite identical learning input they used causal model representations to transform identical covariational information into different types of predictions. In several further experiments we manipulated the statistical parameters of the models (base rates of the events; causal strength of causal links) and showed that participants' predictions were not only driven by the structure of the causal models but also by the learned parameters (see also Meder et al. 2008, 2009, for related evidence in trial-by-trial learning tasks).

2.2.1 Causal Reasoning in Nonhuman Animals

Humans apparently have the natural capacity to form causal representations. How about nonhuman animals? A number of researchers have asserted that causal reasoning and learning are faculties that form a dividing line between humans and nonhuman animals (Povinelli 2000; Tomasello and Call 1997). Recent research by Blaisdell et al. (2006) casts doubt on this conclusion (see also Beckers et al. 2006).

In one experiment, rats went through a purely observational learning phase in which the light was sometimes followed by a tone and at other times followed by food. Importantly, no instrumental learning took place. When in the subsequent observational test phase the rats again heard the tone, they showed that they expected food in the niche in which it was typically delivered. Apparently they reasoned through the causal model link-by-link from the tone through the light to the probable presence of food (see also Waldmann et al. 2008). In contrast, in a second test a lever which the rats had never seen before was introduced into the cage. Whenever the rats curiously pressed the lever, the same tone was presented. Now, although tone and food had been associated by the rats in the learning phase as indicated in the observational test phase, the rats were less inclined to search for food after the lever presses. Apparently they reasoned that they – and not the light – were the cause of the tone, which led to their reluctance to expect food.

In a second study of Blaisdell et al. (2006) a causal chain was presented in which the tone preceded light which in turn preceded food. Consistent with causal-model theory, the rats expected food regardless of whether they observed the tone or generated it with the lever. This shows that they were not generally reluctant to expect food after a novel intervention. The results revealed an understanding of causal relations and demonstrate that rats correctly differentiated between observing and intervening and different causal models.

Whereas associative theories predict associations between tone and food regardless of whether the tone is observed or generated by an action in the test phase, causal-model theory predicts that the intervention at test should be viewed as a potent alternative cause of the tone. Leising et al. (2008) report further tests of causal-model theory. One key prediction is that full discounting of the light should only be

observed when the new alternative cause is viewed as deterministic and independent of the previous cause, the light. Independence and determinism are two hallmark features of interventions but not necessarily of arbitrary events. Consequently, we (Leising et al. 2008) only observed discounting with interventions but not with other observable events. Moreover, rats were capable of flexibly switching between observational and interventional predictions. These results confirm that rats are capable of flexible causal reasoning.

Although this research documents remarkable causal competencies in rats, it nevertheless leaves some interesting questions open. It is true that rats in the two sets of experiments were capable of correctly inferring the outcomes of observations and interventions, but they did not display this knowledge in their actions. For example, although the rats strongly expected food when their intervention caused a tone that was directly causing food, they did not increase the number of lever presses to get more food in this situation. It may well be that rats only have partial incomplete knowledge of causal relations that serves their predictive competencies but falls short of underwriting the action system (see also Penn et al. 2008, for a skeptical view).

2.3 Estimating Causal Parameters

The primary difference between different types of causal models, such as common-cause or common-effect models, lies in the way directed causal arrows are combined. The previous sections have discussed studies showing that people are sensitive to the structural aspects of causal models and capable of coordinating identical learning input with different causal structures. Causal models do not only have a structure, the individual links also have attached strengths as parameters that need to be learned. According to associative theories strength corresponds to observed covariations. However, Cheng (1997) has shown that covariations do not directly mirror causal strength (or causal power in her terminology). According to Cheng, causal power is an unobservable property of causes which expresses the probability of causes generating or preventing effects in ideal circumstances in which no other causes are simultaneously present. This information is not directly provided by the learning input because typically multiple events co-occur. However, causal strength can be inferred. Again, learners need to go beyond covariations. Cheng has derived formulas that allow us to infer causal power under some background assumptions.

An example may illustrate why causal power and covariations do not necessarily correspond. Imagine a drug that generates itching as a side effect. If this drug is solely given to patients who already suffer from itching, no covariation would be observed. The probability of itching remains the same regardless of whether the drug is taken or not. However, it is intuitively clear that this situation does not allow us to assess causal power. The drug may still be a strong cause of itching although in this situation it does not display its power. Cheng and colleagues have shown that people take such properties of the learning situation into account when assessing causal power, and hence go beyond covariations (see Buehner et al. 2003).

Causal structures and their parameters are not independent entities but are deeply intertwined. The causal strength between a cause and an effect, for example, needs to be estimated differently depending on whether or not there is a confounding alternative cause. For example, if we learn the causal strength between the virus and a symptom, we need to control for possible confounds, but not for further effects of the symptom. Waldmann and Hagmayer (2001) have shown that learners are indeed sensitive to the causal roles of events when estimating causal strength.

Causal strength of individual links is not the only parameter of causal models that needs to be inferred, there are also different ways in which multiple causes can combine when they jointly cause a common effect. A typical assumption is that the combination of two generative causes increases the likelihood of the effect beyond what either cause would do. This so called “noisy-or” rule is a default assumption of many networks (Cheng 1997; Griffiths and Tenenbaum 2005), which is assumed to hold unless there are reasons to assume that the causes interact (see Novick and Cheng 2004). Waldmann (2007) has studied continuously varying effects, and has shown that people use background domain knowledge when choosing an integration rule. For example, in one experiment causes were differently colored liquids which could cause the increase of the heart rate of animals. When the liquids were described as affecting intensive quantities (e.g., taste) or preferences (e.g., liking) people were biased towards averaging the causal influences, whereas extensive quantities (e.g., liquids represented drugs with different strengths) led to a tendency to add.

2.4 Limitations of Causal Reasoning

Although people exhibit a sophisticated ability to reason with causal models, there is also evidence for limitations. For example, Waldmann and Walker (2005) have shown that people have difficulties with transforming covariation information into causal-model representations when the task is complex, presented abstractly, or when the learner operates at her information processing limit (see also De Houwer and Beckers 2003). Reips and Waldmann (2008) have similarly found that base rates may be neglected in complex learning tasks. Their results showed that learners are capable of incorporating base rate information in their judgments regardless of the direction in which the causal structure is learned. However, this only holds true for relatively simple scenarios. When complexity was increased, base rates were only used after diagnostic learning, but were largely neglected after predictive learning.

3 Inducing Causal Structures

The previous sections have focused on evidence showing that people and some nonhuman animals go beyond covariations to build causal model representations instead of mirroring statistical relations between cues and outcomes in the learning

input. This research demonstrates that we are not tied to the surface of covarying events. However, I did not address the question how people learn to separate causal model representation from statistical learning input. Why do we not just stick to the surface level? So far there is little research addressing this question. Different factors may be at play here. Infants may be born with a natural tendency to interpret causal events as caused by hidden forces, as suggested by Leslie and colleagues (e.g., Leslie and Keeble 1987). Others have suggested that the tendency to interpret events causally may be triggered by infants' experience of their own actions changing events in their environment, which might provide the basis for further causal knowledge (Dickinson and Balleine 2000; White 2006). Most likely both factors are at play, but we do know little about their relative contributions.

Independent of whether our bias to attribute a causal texture to the world is innate or learned, we need to learn to coordinate the learning input with hypothetical causal models. Where do these models come from? Lagnado et al. (2007) have suggested that we use several cues to form hypothetical models which in turn guide the processing of the learning input. The primary role of the learning input is to provide information about the existence and strength of the causal links (i.e., parameter estimation). The cues underlying structure inductions include temporal order (causes typically precede effects), interventions (interventions target causes, not effects), or coherence with prior knowledge or verbal instructions. Often these cues signal the same structure, but occasionally they may be in conflict. For example, a physician may see a symptom (i.e., an effect) prior to testing for its cause, which requires him to disentangle learning from causal order. Lagnado et al. (2007) summarize various experiments exploring how people coordinate different cues to induce causal models.

4 Conclusion

Hume has presented us with a puzzle: How do we acquire causal knowledge when we only observe covariation information? We have reported a number of studies showing that both human and nonhuman animals have a natural tendency to coordinate covariations with deep causal model representations.

One important question for future research is to explore the generality and differences of causal reasoning capacities across species. Another interesting question will be to analyze the relation between causal reasoning and rational models, such as causal Bayes nets (Gopnik et al. 2004; Lu et al. 2008; Griffiths and Tenenbaum 2005). Our findings on limitations of causal reasoning suggest that such models, if interpreted as psychological theories, may often exaggerate what human and nonhuman animals can do (see Waldmann et al. 2008). Answers to these questions promise to elucidate the structure, origin, and evolution of causal reasoning as an invaluable cognitive tool for surviving and succeeding in one's world.

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