The inferential reasoning theory of causal learning: Towards a multi-process propositional account

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**1.Introduction**

In the present chapter, we describe the inferential reasoning theory of causal learning and discuss how our thinking about this theory has evolved in at least two important ways. First, we argue that it is useful to decouple the debate about different possible types of mental *representations* involved in causal learning (e.g., propositional or associative) from the debate about *processes* involved therein (e.g., inferential reasoning or attention; Moors, 2014). Second, at the process level we embed inferential reasoning within a broad array of mental processes that are all required to provide a full mechanistic account of causal learning. Based on those insights, we evaluate five arguments that are often raised against inferential reasoning theory. We conclude that causal learning is best understood as involving the formation and retrieval of propositional representations, both of which depend on multiple cognitive processes (i.e., the multi-process propositional account).

**2. The associative theory of causal learning**

The question of how we learn that one event causes the occurrence of another event has intrigued philosophers and psychologists since time immemorial. About three decennia ago, Dickinson, Shanks, and Evenden (1984) proposed a challenging answer: They suggested that human causal learning can be explained by association formation between the representation of cues and outcomes (i.e., causes and effects). In short, this theoretical proposal holds that repeated pairing of a cue and an outcome results in the formation of an association between cue and outcome. Such association is typically conceived of as an unqualified link that transmits activation from one representation to another, very much analogous to the way a strip of copper wire conducts electricity. Once it has been formed, presentation of the cue will result in the activation of its mental representation, and this will in turn produce an increase in the activation of the representation of the outcome. Interestingly, the hypothesis of Dickinson et al. (1984) implies that the established principles of associative learning theory, initially developed to account for animal conditioning, can be brought to bear on the issue of human causal learning (Le Pelley, Griffiths, & Beesley, this volume). This means that phenomena that are predicted by associative learning theory should also be observed in human causal learning. Dickinson and colleagues (1984) chose the blocking effect to put this suggestion to the test. This choice had some symbolic value: Arguably, blocking was (and still is) the most important phenomenon in the history of associative learning theory, because it inspired a whole generation of influential learning models (e.g., Rescorla and Wagner, 1972; Mackintosh, 1975; Pearce and Hall, 1980; Wagner, 1981).

Kamin (1967) pioneered the blocking procedure by presenting rats with pairings of a white noise stimulus with shock followed by pairings of a compound of the white noise stimulus and a new light stimulus with shock. This training procedure is commonly denoted as “A+ then AX+” training (where “A” means that a neutral stimulus, for example a burst of white noise, is presented; “AX” means that two stimuli, for example a burst of white noise and the flashing of a light, are presented together; and “+” means that those stimuli are followed by an outcome, for example a shock). Kamin (1967, p. 5) made the famous observation that “prior conditioning to an element might block conditioning to a new, superimposed element”: The light stimulus X elicited low fear responding when it was presented by itself during test, despite its being paired with the shock outcome. Importantly, Dickinson et al. (1984) observed blocking in a human causal learning study, mirroring the observations in animal conditioning. Procedurally speaking, a causal learning study is similar to an animal conditioning study in that it involves exposure to trials in which potential causes and effects may or may not co-occur (for a detailed description see Perales, Catena, Cándido, & Maldonado, this volume). An often-used procedure requires participants to imagine that they are an allergist who tries to discover the cause(s) of an allergic reaction. In a trial-by-trial fashion, participants are then presented with records of a fictitious patient that show one or more food items that the patients has supposedly eaten with an outcome message indicating whether an allergic reaction occurred following digestion of those food items. For example, in a blocking preparation, in a first series of trials, eating paprika would be followed by an allergic reaction and, in a next series of trials, eating paprika and coconut together would be followed by that same allergic reaction (i.e., A+, AX+ training). In this example, successful blocking would be reflected in the participant making the judgment that coconut does not produce an allergic reaction (i.e., cue X is not causally related to the outcome).

How do associative theories (e.g., Rescorla & Wagner, 1972) account for this phenomenon? These theories typically rely on an error-prediction mechanism that can be described in terms of expectancy and surprise: Learning is conceived as a function of how surprising the occurrence of the outcome is, which is determined by the extent to which the outcome is expected. Accordingly, the associative explanation of blocking holds that the preceding A+ training renders the outcome to be expected on AX+ trials and that therefore an associative link between cue X and the outcome cannot form (i.e., is "blocked"), which then (one way or another; see below) results in the judgment that X is not causally linked to the outcome. Going back to our example: People are supposed to display blocking, because they fail to acquire an association between the occurrence of eating coconut and the occurrence of the allergic reaction.

The observation of blocking in human causal learning prompted a wave of empirical studies, attesting to the popularity of this framework (for reviews, see De Houwer & Beckers, 2002, and Shanks, 2010). Successes of associative theories in explaining phenomena of causal learning did not remain limited to blocking but extended to a range of phenomena, further fuelling the view that causal learning might be understood in terms of association formation (for a detailed discussion see Le Pelley et al., this volume). However, for people who have taken part in such causal learning experiment, the associative explanation is rather counterintuitive: If you take part in such experiment, you might experience reasoning about whether eating coconut causally results in an allergic reaction. It is in this context that we must consider the significance of the inferential learning theory, which we will discuss below.

**3. The inferential theory of causal learning**

As an alternative to the associative view, inferential reasoning theory has stated that causal learning involves inferential reasoning, which can be defined as a slow and effortful process that starts from premises and returns a conclusion. In a blocking procedure, for example, participants supposedly infer that the blocked cue is unlikely to be causally related to the outcome, because the relation between the blocked cue and the outcome disappears if one controls for the relation between the blocking cue and the outcome (De Houwer, Beckers, & Vandorpe, 2005; Waldmann, 2000). More formally, the inferential reasoning process underlying blocking can be represented as a modus tollens argument (Beckers, De Houwer, Pineño, & Miller, 2005):

I. [if p, then q] If A and X are both causes of the outcome, then the outcome should be stronger when these causes are both present than when only one cause is present

II. [not q] The outcome is not stronger when A and X are both present than when A is presented alone

III. [therefore, not p] Thus, A and X cannot both be causes of the outcome

People can infer that cue X is not a cause of the outcome because they experienced that A results in the outcome when presented alone and therefore needs to be a cause. However, the validity of this modus tollens rule depends on a number of constraints. Hence, if blocking results from applying a modus tollens rule, then the blocking effect should vary as a function of those constraints. Manipulating the validity of the constraints therefore allows to empirically evaluate the inferential reasoning theory in at least two ways.

First, the conclusion of the modus tollens argument does not follow if premise I does not hold. In such case, blocking should not be observed. To test this prediction, Beckers, De Houwer, Pineño, & Miller (2005) presented to half of their participants pretraining that confirmed premise I. They did this by showing that two cues that resulted in a single outcome when presented individually resulted in a double outcome when presented together (e.g., eating cheese results in a moderate allergic reaction, eating mushrooms results in a moderate allergic reaction and eating cheese and mushrooms together results in a severe allergic reaction). The remaining participants received training that suggested that outcomes would be non-additive: Two cues that resulted in a single outcome when presented individually still resulted in a single outcome when presented together (e.g., eating cheese results in a moderate allergic reaction, eating mushrooms results in an allergic reaction and eating both cheese and mushrooms also results in a moderate allergic reaction). Only participants who received the addititive pretraining displayed blocking when subsequently exposed to a blocking contingency involving new food cues. This finding suggests that assumptions about cue additivity (sometimes) control blocking (for related evidence, see Lovibond, Been, Mitchell, Bouton, & Frohardt, 2003; Vandorpe, De Houwer, & Beckers, 2007). The additivity pretraining supposedly urges participants to go from using a noisy-or causal integration rule to using a linear-sum causal integration rule. The noisy-or integration rule assumes that causal influences are independent in producing a binary outcome (which should result in weak blocking), whereas the linear-sum causal integration rule assumes that causal influences are additive in producing a continuous outcome (which should result in strong blocking; Lu, Rojas, Beckers, & Yuille, 2015).

Second, the modus tollens argument is not applicable if outcomes occur to a maximal extent on all trials. If cue A causes the outcome to a maximal extent and if cues A and X together cause the outcome to the same maximal extent, one cannot be sure that X has no additive effect on top of A because of a ceiling effect. Stated differently, participants cannot verify the veracity of premise II. Therefore, if blocking depends on the modus tollens argument, one should observe weak blocking if outcomes that occur to a maximal extent are used during training. Beckers et al. (2005) tested this prediction by showing all participants outcomes of maximal and of submaximal intensity (i.e., allergic reactions of severe and moderate intensity) before presenting them with blocking training. In one condition, the intensity of the outcome used during blocking training corresponded with the maximal intensity shown during pretraining, whereas in the second condition this intensity corresponded with the submaximal intensity shown previously. As predicted, blocking was much stronger when the outcome occurred with submaximal strength during blocking training than when it occurred with maximal strength.

It is of interest that Beckers et al. (2006) later demonstrated that blocking in rats is also modulated by additivity and outcome maximality information, which suggests that inferential reasoning is not only involved in human causal learning but also in animal conditioning. This creates an interesting situation: Whereas Dickinson et al. argued that associative models developed on the basis of animal conditioning research can also account for human causal learning, Beckers et al. (2006) suggested that it might be the other way around: Inferential reasoning theory, originally developed to explain human causal learning, might also apply to animal conditioning.

 **3.1 Reasoning as an effortful cognitive process**

As stated, inferential reasoning theory assumes that causal learning (e.g., the sensitivity of causal learning to blocking) results from inferential reasoning, defined as the slow and effortful production of propositional conclusions on the basis of propositional premises. We now briefly consider the two core elements in our definition of inferential reasoning: In this section, we discuss reasoning as being an effortful cognitive process; in the next section, we discuss the propositional nature of the input (premises) and output (conclusions) of this cognitive process.

Inferential reasoning as a cognitive process is presumed to be effortful: Traditional reasoning theories invoke the idea of limited working memory capacity when explaining reasoning performance (e.g., Baddeley & Hitch, 1974; Johnson-Laird & Byrne, 1991; Rips, 1994). In line with this idea, research demonstrates that the number of errors in syllogistic reasoning tasks increases when working memory is overloaded (e.g., Toms et al., 1993; De Neys et al., 2005). Therefore, if the occurrence of blocking in causal learning results from inferential reasoning, one would expect blocking to be reduced under working memory load, which turns out to be the case (De Houwer & Beckers, 2003; Liu & Luhmann, 2013; Waldmann & Walker, 2005). Although this finding suggests that an effortful process is involved in blocking, it does not need to imply that inferential reasoning is involved. More direct evidence for the role of inferential reasoning was provided by Vandorpe et al. (2005) who asked participants to explain how they arrived at their causal ratings in a blocking task. Crucially, working memory load modulated the number of participants who were able to verbally report a valid blocking inference (i.e., “X did not add to the effect of A, hence it is not a cause of the outcome”). Also of interest is that the presence of blocking effects in children’s causal learning seems to go hand in hand with the development of working memory and general reasoning abilities (Simms, McCormack, & Beckers, 2012; McCormack, Simms, McGourty, & Beckers, 2013a). Moreover, encouraging children to engage in counterfactual reasoning, which is known to enhance inferential reasoning performance, enhances their propensity to show blocking in a causal learning task (McCormack, Simms, McGourty, & Beckers, 2013b).

Of note, although we argue that the evidence above points to a central role of inferential reasoning in causal learning, we by no means claim that inferential reasoning is the only process involved (see Section 4).

**3.2 Propositions**

We now focus on the second element in our definition of inferential reasoning, the propositional nature of the input it operates on (premises) and the output it generates (conclusions). Indeed, in logic, reasoning involves a [set](http://en.wikipedia.org/wiki/Set_%28mathematics%29) of propositions known as the premises along with another proposition known as the [conclusion](http://en.wikipedia.org/wiki/Logical_consequence). When describing propositions, we will, for reasons of clarification, also focus on how they differ from associations.

 A proposition is a bearer of truth-value (i.e., it can be true or false; e.g., "smoking causes cancer") and the object of propositional attitudes (e.g., "I believe that smoking causes cancer"). This is different from an association that cannot be true or false, but just does what it does: transmitting activation (e.g., the representation of smoking activates the representation of cancer). In addition to bearing truth-value, propositions are compositional, meaning that they are comprised of parts that can be recombined, just like words in sentences can be recombined to form other sentences (Moors, 2014). For example, the propositions "smoking causes cancer" and "cancer causes smoking" both comprise the elements "smoking", "causes" and "cancer"; yet, they have distinctly different meanings.

Most important for the present purposes is that propositions can contain qualified, relational information. For example, the propositions "smoking causes cancer" and "yellow fingers predict cancer" both specify the type of relation between cue and outcome. Relational representations are conceptualised as a set of elements (e.g., smoking and cancer, or elephant and mouse) that are bound together by a relational symbol (e.g., causes or is larger than; Halford, Wilson, & Phillips, 2010). In this regard, propositions again differ from associations: An association is an unqualified link that can at most have a direction, but even then this direction is determined only by the temporal order in which events are presented during training. Associations can differ on only one variable, i.e. the strength of the association. Variations in the events’ frequency, probability, and so forth can be mapped onto this single variable, but variations in the type of relation (e.g., predictive or causal) between events cannot be coded (Holland, 1993). It is worth noting that because of this mere theoretical reason it is difficult for associative theories to provide a full account of causal learning: Simply activating the outcome is not sufficient to form a statement about causality specifically (i.e., as different from non-causal relations). In response, theorists have suggested that the mind contains both associative and propositional representations. Low-level associations could then underlie higher-level propositions (Gawronski & Bodenhausen, in press). For example, the representation of smoking activating the representation of cancer would give rise to the conscious proposition "smoking causes cancer". Although this makes sense intuitively, the question remains how the organism can know which type of relation binds the elements: Does smoking predict cancer, cause cancer, enable cancer, prevent cancer, or is there still another relation at play (Moors, 2014)? We refer the interested reader to Johnson-Laird and Khemlani (this volume) for a more extensive discussion of the representation of causal relations.

With respect to the acquisition of propositions, the inferential theory of causal learning holds that the propositions on which causal inferential reasoning operates may themselves originate from inference, as well as from experience, observation and instruction (Gopnik, Sobel, Schulz, & Glymour, 2001). The underlying idea is that all knowledge is represented in a propositional form, in principle rendering it irrelevant how it is acquired (but see Perales et al., this volume). Accordingly, blocking can be modulated not only by experience that contradicts the proposition of causal additivity (Beckers et al., 2005; see above) but also by mere instructions that causes do not summate (e.g., Lovibond et al., 2003; Mitchell & Lovibond, 2002). Likewise, verbal information about the occurrence or absence of the outcome on A alone trials can retrospectively modulate blocking when outcome information was masked during the first phase of a blocking task (i.e., A? followed by AX+ training; De Houwer, 2002; for similar findings, see Boddez, Baeyens, Hermans, Van der Oord, & Beckers, 2013). It is difficult to see how associative models can account for learning by instruction or inference, as these models simply lack a plausible mechanism for this type of learning (for a detailed discussion see Lovibond, 2003).

Perhaps because inferential reasoning normally operates on and produces propositions, the terms “inferential reasoning theory” and “propositional theory” have often been treated as interchangeable. However, there is a fundamental asymmetry between inferences and propositions: In order to reason, one typically needs propositional premises. Therefore, an inferential reasoning account of causal learning presupposes propositional representations. Propositions, however, can be acquired and influence behaviour in the absence of inferences (and hence, in the absence of inferential reasoning; De Houwer, in press-a; De Houwer, in press-b; Moors, 2014). Accordingly, whereas evidence against the involvement of propositional representations casts doubt on inferential reasoning models, evidence against the involvement of inferential processes in causal learning does not necessarily invalidate the idea that propositional representations mediate causal learning and does not necessarily require invoking an associative system. In principle, one could even go so far as to develop and defend a propositional theory that does not make any claim about cognitive processes such as inferential reasoning.

**4. From an inferential reasoning to a multi-process account of causal learning**

In the previous section, we argued for the role of inferential reasoning in causal learning. However, we do not claim that inferential reasoning is the only process involved in causal learning. In fact, also learning principles traditionally associated with association formation models might play a role in the formation and retrieval of propositional representations involved in causal learning. For example, updating due to error-correction, a hallmark feature of many associative learning models, might motivate the formation of propositions. Let us argue by the example of scientific progress. An experimental observation that falsifies a prediction ideally results in an updated theory that can account for the observation that falsified the initial theory. So, falsification of theories results in updates of theories, which incrementally leads to increasingly better predictions. Yet, scientific theories consist of propositions, not associations.

Below, we will discuss some cognitive processes — other than inferential reasoning — that may play a role in the formation and retrieval of propositions about causal relations. Our aim is to illustrate that inferential reasoning is embedded in a variety of cognitive processes that affect the formation and retrieval of propositions involved in causal learning (Boddez, Haesen, Baeyens, & Beckers, 2014). This discussion will further clarify that claims about the involvement of propositional representations in causal learning are different from claims about cognitive processes.

**4.1 Perception**

There is an extensive empirical literature showing that the visual world is interpreted in terms of causality before slow, non-automatic causal reasoning processes begin to operate (Hubbard, 2013; White, Schulz & Muentener, this volume). If participants view a moving stimulus that strikes a stationary stimulus, and that latter stimulus then begins moving, there is a clear and immediate perception that this movement was caused by the initially moving object. This effect is termed the launching effect (e.g., Michotte 1946/1963) and it has been argued that it concerns an automatic impression of causality that does not involve inference (Hubbard, 2013). Although one might dispute whether the launching effect is a learning effect (because most observers would accurately predict that the one stimulus would launch the other before observing such launching trial, so nothing new would be learned), the phenomenon of causal perception at least illustrates that inferential reasoning is not the only process that plays a role in forming propositions about causal relations. For the sake of completeness, it nonetheless deserves mention that there are theories that assume that certain aspects of perception are produced by processes of automatic, probabilistic Bayesian inference (Knill & Pouget, 2004; Lee & Mumford, 2003).

**4.2 Attention**

Attention is another example of a process other than inferential reasoning that may play a role in the formation or retrieval of propositions about causal relations. Attention has long been the focus of theoretical consideration in the associative learning field. Evidence for the involvement of attention in, for example, blocking includes demonstrations that new learning about a blocked cue is slowed down: Blocking treatment interferes with subsequent learning, even when an outcome different from the one during blocking training is used. This interference effect is presumably due to a decrease in attention paid to the blocked cue, caused by the preceding blocking treatment (e.g., Le Pelley, Beesley, & Suret, 2007; Mackintosh & Turner, 1971). In line with this, studies that used the eye tracking method found that participants spent less time gazing at blocked cues (e.g., Beesley & Le Pelley, 2011; Eippert, Gamer, & Büchel, 2012; Kruschke, Kappenman, & Hetrick, 2005; also see Wills, Lavric, Croft, & Hodgson, 2007). Associative models explain these findings by assuming that the amount of attention determines the amount of associative learning. For example, Mackintosh (1975) proposed that a blocked cue is a poorer predictor of the outcome than a blocking cue, and that this leads to lower attention to and a failure to learn about blocked cues. However, attention may not only impact the formation of associations, but also of propositions involved in causal learning. Whereas blocking could be due to inferential reasoning (i.e., about how propositions are related after they have been formed) under some training conditions, similar blocking effects could be due to the effect of attentional processes on the initial formation of propositions. That is, attentional failure might cause the elements needed to form a proposition to not be represented, so one would simply not be able to form the proposition that X is causally related to the outcome. Note that this interpretation differs substantially from the way in which inferential reasoning theorists have previously explained effects of attention. Inferential reasoning theory holds that attentional shifts in a blocking procedure do not cause blocking, but rather, that they are a consequence of the organism’s non-automatic reasoning that the blocked cue is redundant. Obviously, those possibilities are not mutually exclusive: Selective attention may sometimes cause blocking, and blocking may sometimes give rise to selective attention.

**4.3 Memory**

Mitchell, Lovibond, Minard, and Lavis (2006) devised a blocking task with many different foods as the cues and with allergies as the outcomes, such that recall could be tested. In addition to revealing blocking on causal judgments, the results were clear in showing that recall of the outcome related to the blocked cue was poor in comparison with appropriate control cues. Shanks (2010) argues that these results provide positive evidence for associative explanations but challenge inferential accounts. The idea is that participants would need to remember that the blocked cue has been paired with the outcome in order to successfully make the modus tollens inference. Indeed, the proposition that A and X have been paired with the outcome is a crucial premise in the modus tollens argument described in the previous section of this chapter. However, in contrast to Shanks’s claim, not only associative learning theory, but also theories invoking propositions (e.g., inferential reasoning theory) can account for such findings: When many cues and outcomes are presented, as in Mitchell et al.'s study, these may not gain access to memory (e.g., Kastner and Ungerleider, 2000), so upon presentation of the blocked cue during testing no proposition about its causal efficacy would be retrieved from memory.

Until now we focussed on memory encoding, let us now turn to the role of memory retrieval processes. Boddez, Baeyens, Hermans, and Beckers (2011) investigated the effect of extinguishing a blocking cue on the causal judgment about the blocked cue (i.e., A+ and AX+ training followed by A– training) in a causal learning task. The results indicated that extinguishing A increased causal judgments about X. Crucially, this increase was context dependent: Increased judgment about X was limited to the context in which extinction of A took place. This finding can be used to illustrate that the blocking effect depends on memory retrieval of the blocking cue as an effective cause of the outcome, since memory accessibility is known to be context dependent (contexts can either facilitate or hamper retrieval of specific memories; Bouton, 2002). More precisely, our interpretation assumes that the proposition “A produces the outcome” is only retrieved in contexts that differ from the context in which A is extinguished. Because this propositional premise is required to come to the conclusion that X does not result in the outcome, recovery from blocking would be observed in the extinction context, whereas blocking would be observed in contexts that differ from the context in which A is extinguished.

In summary, we argue that causal learning is not a unitary phenomenon, but that different cognitive processes may be at play. We focused on additional roles of perception, attention and memory, but future research should obviously focus on the possible role of still other processes (e.g., the role of inhibition; for an extensive discussion see Boddez et al., 2014).

**5. Evaluating the inferential reasoning theory of causal learning**

We have described the nature of inferential reasoning theory and have clarified that (1) inferential theories should be distinguished from propositional theories and (2) inferential reasoning is never the only processes that is involved in causal learning. Based on those clarifications, we now turn to the evaluation of five of the most important arguments raised against inferential reasoning theory (e.g., McLaren et al., 2014; Penn and Povinelli, 2007; Shanks, 2010).

First, many people have intuitive sympathy for the argument that an associative system might better account for learning of non-verbal material or for trial-by-trial learning. However, several of the findings used as evidence for the inferential reasoning account come from experiments that make use of non-verbal stimulus material which is presented in a trial-by-trial fashion (e.g., the previously discussed studies in which assumptions about outcome additivity and outcome maximality were manipulated; Beckers et al. 2005). So, explaining findings obtained with this type of training procedure does no require invoking associative theories. In fact, even when trained using this type of procedure, people show behaviour that goes beyond the scope of associative theories (also see Meder, Hagmayer, & Waldmann, 2008).

Second, associative learning theorists (e.g., McLaren et al., 2014) have argued that irrational behaviour is out of scope for inferential learning theory and that, therefore, an additional associative system is needed to explain the often irrational behaviour of humans. However, adhering to an inferential account does not imply that we should assume people to always behave in a perfectly rational manner. Indeed, inferential processes and propositional representations can produce irrational learning effects (De Houwer, in press-a; Mitchell, De Houwer, & Lovibond, 2009). People can make errors when forming propositions (e.g., seeing relations where there are none) or make incorrect inferences on the basis of justified premises, and act irrationally on the basis of the resulting false beliefs. Still another possible reason for irrational inferences may be that subjects do not know the necessary logical inference rules (Johnson-Laird & Khemlani, this volume). The extent to which inferences are logically valid can vary with a number of factors such as time and effort. For instance, when given sufficient time, it may be evident that a particular reasoning is valid, whereas another reasoning is logically weak. But when time is limited, the weaker reasoning may nonetheless prevail (Chater, 2009). That is, different lines of argument or different sampling from all potentially relevant pieces of information can result in the formation or behavioural expression of different propositions, thus leading to dissociations in behaviour. People may also be inconsistent: For example, automatic retrieval of propositional beliefs that were endorsed in the past (e.g., spiders are dangerous) might guide behaviour at times when they are no longer endorsed. Verbal-autonomic response dissociations, that is, contradicting responses in different response channels (e.g., Perruchet, 1985), may also be explained in that way: A proposition that is verbally evaluated as false (e.g., spiders are not dangerous) may activate a (behavioural or physiological) response before being evaluated as false. In such case, behaviour can come to differ from beliefs that people report, therefore appearing irrational. Such effects are unlikely to be due to inferences and thus contradict purely inferential theories of causal learning. However, as pointed out above, these effects do not contradict propositional theories of causal learning.

Third, researchers have argued that an exclusively propositional account, let alone an inferential account, is incompatible with neuroscientific evidence. For example, McLaren et al. argue that there is neuroscientific evidence that associations exist in at least some animals (e.g., Aplysia californica), so that it must be the case that associative learning has evolved and that associations must exist in other animals including humans (McLaren et al., 2014). However, McLaren and colleagues do not distinguish between the neurological and the psychological level of analysis when they state that observed changes in the strength of synaptic connections in Aplysia provide neuroscientific evidence for the existence of associations. Inferential and propositional accounts are situated at the psychological level of analysis in that they specify assumptions about the mental processes and representations that mediate learning effects. Multiple candidate psychological representation and processes are compatible with any finite set of neuroscientific data: There is no way of knowing whether the changes in the strength of synaptic connections correspond with associations or not. Nonetheless, knowledge about the neural level does constrain theories at the mental level and, in contrast with McLaren’s claim, there is actually strong evidence that the properties of the changes in synaptic transmission align poorly with the properties of associative learning as revealed by behavioural experimentation (for a review see Gallistel and Matzel, 2013).

Fourth, Penn and Povinelli (2007) argued that it is problematic that inferential reasoning theory so far lacks a formal, computational specification, because it renders it impossible to refute these accounts. A couple of points can be made here. Most importantly, a model like Rescorla-Wagner is a rule that mathematically predicts behavioural output from environmental input. The same holds for Bayesian models (Kruschke, 2008). However, at a psychological level that rule can in principle be described as the operation of associations (i.e., Va is the strength of the association) as well as as the operation of propositions (e.g., Va is the extent to which the cue predicts or causes the US). So, in principle, existing formal computational models could be conceptualized equally well in terms of associations and propositions (Shanks, 2007). In fact, a Bayesian account for some of the inferential reasoning predictions concerning blocking has recently been proposed (Lu et al., 2015).

Moreover, formalization and the accompanying degree of precision would indeed allow one to easily refute computational models if predictions turn out to be incorrect. However, this is not what seems to happen in scientific practice: Many associative models, in particular the Rescorla-Wagner model, remain highly influential despite a history of falsification (Le Pelley at al., this volume; Miller, Barnet, & Grahame, 1995). Likewise, showing that causal judgments do not conform to the predictions of a specific formalized propositional theory would probably not make the research community refute the concept of inferential reasoning or propositional representations altogether. There are so many ways to formalize psychological concepts such as inferential reasoning and association formation that the refutation of one of those formalizations will have little or no impact on more abstract psychological theorizing.

It is also worth noting that the proliferation of computational models developed in the associative tradition has actually made it difficult to make precise predictions. Suppose one presents two cues with an outcome (e.g., coconut oil and paprika followed by an allergic reaction) and subsequently presents one of those cues without the outcome (i.e., extinction training; e.g., paprika without an allergic reaction) before testing the other cue for its relation with the outcome (i.e., the cue of interest; e.g., coconut oil). Computational models exist that predict that the extinction training will strengthen (Dickinson & Burke, 1996; Van Hamme & Wasserman, 1994), weaken (Holland, 1983, 1990; also see Dwyer, Mackintosh, & Boakes, 1998), or not affect (Rescorla & Wagner, 1972; Wagner, 1981) the association between the cue of interest and the outcome. More generally, the computational models developed in the associative tradition actually form a family of models in which divergent predictions are often made. Although having diverging predictions is not a problem if there is an empirical way of distinguishing between the candidate models, this realization still puts associationists’ claims of formal specification in perspective: If an effect does not follow a specific model, it is typically not seen as a falsification of the associative account in general (e.g., Le Pelley at al., this volume).

Importantly, however, formalization of inferential and propositional theories would still be useful because it is bound to further increase their predictive and heuristic function. An important future challenge for inferential and propositional accounts is to identify which environmental conditions favour the formation of which propositions and to identify how sets of simultaneously (and potentially contradictory) retrieved propositions affect the different response channels. Although post-hoc propositional accounts of dissociations can already be tested empirically, current accounts are too vague to allow for strong a priori predictions about when behavioural dissociations will occur. At this point, it is probably also worth noting that explaining response generation in general is challenging to propositional learning theory (the same holds for associative theories, though): Responses are assumed to be the behavioural expression of propositions entertained by the subject, but how this translation is done is poorly understood (Baeyens, Vansteenwegen, & Hermans, 2009; Mitchell et al., 2009).

Fifth, contrary to popular belief (e.g., McLaren et al., 2014), a convincing demonstration of automatic learning effects will not lead to a rejection of the inferential reasoning or propositional theory of causal learning: As argued extensively by Mitchell et al. (2009), automatic retrieval of propositions is entirely possible. For example, if subjects in for example a blocking procedure are able to form a proposition about the blocked cue before testing, they should be able to retrieve that acquired knowledge automatically. Interestingly, Morís, Cobos, Luque, and López (2014) recently demonstrated blocking using a priming test (a test which depends on automatic retrieval processes), providing some evidence for this possibility (it should be noted that the interpretation of the authors differs from ours). They used a standard learning task. However, instead of asking for verbal judgments at test, they used a priming task for testing. Results showed that a cue that underwent blocking training facilitated the recognition of the outcome to a lesser extent than a control cue and its outcome. This reveals that blocking can be detected through measures that are based on automatic retrieval processes.

With respect to the learning itself, association formation is often said to occur automatically and formation of proposition is often said to occur non-automatically. It is, however, questionable whether mapping these dichotomies onto each other is justified (Moors, 2014). One could, for example, readily build and program a (presumably) automatic and unaware robot that can work with propositional representations (also see Shanks, 2007). Interestingly, a recent study confirms that awareness is not a prerequisite for the formation of propositions: In a set of experiments it was demonstrated that predictive relations can be formed even when awareness of the relation is actively prevented (Custers & Aarts, 2011). As discussed above, the launching effect could also be regarded as a proof-of-principle that propositions about causal relations can be formed automatically (Hubbard, 2013). One important exception where the formation of a proposition must be non-automatic is the case in which a proposition results from a non-automatic inferential reasoning process. For example, if blocking is achieved through inferential reasoning with the modus tollens rule, then the proposition "cue X does not cause the outcome" will have been formed non-automatically. Under such circumstances, then, blocking should be reduced when effortful processing is prevented (e.g., under working memory load; see above).

In summary, we argue against the use of stimulus material, stimulus presentation mode, rationality, isomorphisms between psychological and neural mechanisms, formal specification, and automaticity as criteria for deciding whether causal learning depends on inferential processes and propositional representations. How to proceed then? Even in their present, non-formalized state, inferential and propositional theories do allow for interesting novel predictions. For instance, propositional theories allow for the impact of relational information. As said, propositions, but not associations, encode information about how events are related (see Lagnado, Waldmann, Hagmayer, & Sloman, 2007). Accordingly, future efforts in distinguishing between propositional and non-propositional (e.g., associative) accounts should focus on whether learned behaviour is always moderated by relational information (see Zanon, De Houwer, Gast, & Smith, in press, for a recent example). So, we believe that there is merit in proposing an inferential and propositional theory of causal learning, because of its ability to provide an interpretation for previous findings (i.e., heuristic value) and because of its potential to generate new predictions that can lead to new, empirical knowledge (i.e., predictive value).

**6. Summary, further thoughts and conclusion**

The present chapter is built on two pillars. First, we situated the inferential theory of causal learning within the context of a propositional account of behaviour. It is difficult for associative theories to provide a full account of causal learning, because simply activating the outcome is not sufficient to form a statement about causality. Such statement requires representation of the type of relation that exists between cue and outcome (e.g., causes, enables, etc.), which necessitates a propositional representation. Second, we argued that inferential reasoning does not suffice if one wants a cognitive account of causal learning: A variety of processes will need to be considered when writing the final story of causal learning.

Tension between higher-order and associative views of learning exists since over a century (Shanks, 2010). Regrettably, this debate sometimes seems to come down to a matter of personal preference – are we more impressed by the finding that causal learning often seems to follow associative principles of the sort formalized in the Rescorla-Wagner model or by the fact that this form of learning shows properties that lie outside the scope of models of this sort (see Hall, 2009, p. 210)? Arguably, clearer understandings and concepts are critical to break the stasis in this debate and to “prevent the field from wasting time chasing after ever-more-nuanced predictions in an attempt to differentiate ever-more-similar theories” (Liu and Luhmann, 2014). Contrasting associations with propositions might be more revealing than contrasting associative models with inferential reasoning models, because the latter debate mixes up aspects of representations and processes (Moors, 2014). Distinctions in terms of representational content (e.g., propositions or associations) do not necessarily map onto distinctions in terms of processes that operate on these representations. Following this line of reasoning, we argued that learning principles and processes typically invoked by associative theories (e.g., prediction error and attention) might very well facilitate the formation or retrieval of propositional representation. In principle, one could even go on to develop a propositional theory that remains completely silent with respect to the cognitive processes that operate on said propositions. The central tenet of such theory would be simply that behaviour is mediated by propositional representations, that is, by representational units that contain relational information. Interestingly, such theory would make fewer assumptions than the inferential learning theory, because it would not make any assumptions about the nature of the cognitive processes involved.

Most learning researchers strongly resist an account that explains all learning effects through inferential reasoning (McLaren et al., 2014; Penn and Povinelli, 2007; Shanks, 2010). For example, McLaren et al. (2014, p. 185) argued that associations are still needed to provide a full account of human associative learning. More precisely, they claimed that "no one disputes that we solve problems by testing hypotheses and inducing underlying rules, so the issue amounts to deciding whether there is evidence that we (and other animals) also rely on a simpler, associative system, that detects the frequency of occurrence of different events in our environment and the contingencies between them." Thus, the alternative to the propositional approach advocated in this chapter is the dual-system approach: behavior is determined by both propositional and associative representations. However, we made clear that the breach between propositional versus associative representations does not parallel the breach between rational versus irrational behavior, between non-automatic versus automatic behavior or between different kinds of stimulus material (e.g., verbal versus non-verbal stimuli, tabulated data versus trial-by-trial data). In summary, many arguments for a dual-process account disappear if one considers an inferential reasoning theory of causal learning tasks in tandem with a propositional theory of learning and if one considers inferential reasoning as one in a series of cognitive processes that contribute to causal learning.

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