



Review

Associations and propositions: The case for a dual-process account of learning in humans

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ARTICLE INFO

Article history:

Available online 2 October 2013

Keywords:

Implicit learning
 Explicit learning
 Cue competition
 Associative learning
 Cognition
 Rules
 Propositions

ABSTRACT

We review evidence that supports the conclusion that people can and do learn in two distinct ways – one associative, the other propositional. 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. There is neuroscientific evidence that associative learning occurs in at least some animals (e.g., *Aplysia californica*), so it must be the case that associative learning has evolved. Since both associative and propositional theories can in principle account for many instances of successful learning, the problem is then to show that there are at least some cases where the two classes of theory predict different outcomes. We offer a demonstration of cue competition effects in humans under incidental conditions as evidence against the argument that all such effects are based on cognitive inference. The latter supposition would imply that if the necessary information is unavailable to inference then no cue competition should occur. We then discuss the case of unblocking by reinforcer omission, where associative theory predicts an irrational solution to the problem, and consider the phenomenon of the Perruchet effect, in which conscious expectancy and conditioned response dissociate. Further discussion makes use of evidence that people will sometimes provide one solution to a problem when it is presented to them in summary form, and another when they are presented in rapid succession with trial-by-trial information. We also demonstrate that people trained on a discrimination may show a peak shift (predicted by associative theory), but given the time and opportunity to detect the relationships between S+ and S–, show rule-based behavior instead. Finally, we conclude by presenting evidence that research on individual differences suggests that variation in intelligence and explicit problem solving ability are quite unrelated to variation in implicit (associative) learning, and briefly consider the computational implications of our argument, by asking how both associative and propositional processes can be accommodated within a single framework for cognition.

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1. Introduction

In 1994 McLaren, Green and Mackintosh, Green and Mackintosh published a paper on “Animal Learning and the Implicit/Explicit Distinction” as a chapter in the book “Implicit and Explicit Learning of Languages” edited by N.C. Ellis. In it we argued for a dual process account of learning in humans, but against the use of awareness as a criterion for distinguishing between implicit and explicit learning and memory. Instead, we proposed a distinction between associative and cognitive processes, and took a comparative stance that equated associative processes across species. We characterized associative learning as that which involved the establishment of

links between representations (and here a representation can be as simple as the activation of a specific unit or neurone though often it will correspond to a pattern of activity over units), and cognitive processes were seen as allowing the induction and application of rules or propositions in a flexible manner not available to associative processes. Executive control was cited as possibly the purest example of the application of rule-based processing. We took the view that the cognitive system dealt in propositional knowledge and would therefore have beliefs, and reasons for those beliefs that could be verbalized, whereas the associative system would extract the statistical regularities from experience. Subsequently, Mackintosh (1997), starting from a rather different position, also argued for the importance of associative processes in human learning.

We are certainly not alone in suggesting that people may have two rather different sets of processes for learning and problem

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solving. Related ideas, albeit couched in different terminology, have been proposed by, among others, Evans (2003), Kahneman (2003, 2011), Stanovich and West (2000), and in the related area of memory by Jacoby (1991). But they have been strongly resisted by some. For example, Mitchell, De Houwer, and Lovibond (2009) and Lovibond and Shanks (2002), have argued that the case for a dual process account is not proven (at best), or non-existent (at worst). Both have suggested that all human learning is to be explained by a single process propositional account, there being no evidence for associative processes. We believe there is a need for proponents of a dual process account of human learning to respond to these analyses, and, in particular, to make the case for associative learning in humans. Our plan in this paper is to revisit the arguments we made in 1994 and 1997, and see how research to date has or has not substantiated the analyses we offered then. In the 1994 paper we discussed Pavlovian conditioning, peak shift, and the Overtraining Reversal Effect (ORE) to illustrate the application of associative processes, and in the 1997 paper additional evidence from studies of peak shift was used to support this argument. Here we will consider Pavlovian conditioning again (this time including the Perruchet effect), look at more recent evidence on peak shift and the ORE in humans to see if it parallels that in other animals. We will also bring to bear recent research on individual differences that offers a new perspective on the dual process debate. In what follows, we construct our argument in terms of making the case for associative learning, as the case for propositional learning naturally emerges as a counterpoint to this theme. Whilst doing this we are conscious of the need to exercise great care in drawing conclusions based on simple parallels between the animal and human literature on learning. We take the view that if we believe in dual process accounts of learning, then this places a responsibility on us to ensure that when we ascribe an effect to associative learning we take some care to show that it is not driven by means of rules or propositions. At the end of this paper, we conclude with a few thoughts on how associative and propositional processes might co-exist and jointly contribute to human learning and behavior.

2. The case for association–neural mechanisms

We start by asking why we might believe that there is such a thing as associative learning in animals and humans. The idea of an association as the basis for learning has its origins in the writings of philosophers such as Hartley and Hume (even Aristotle). Within experimental psychology, its popularity may be because it provides a simple and straightforward account of the basic phenomena of Pavlovian and instrumental conditioning. This in itself is not a sufficient reason to take it as the explanation for these phenomena; if we allow (as we do) that humans can learn by forming propositions, why could this not also be the case for other animals? A first answer is that it is demonstrably the case that for some animals learning is associative, because it has been shown to be so at a neural level. Neuroscientific studies of the substrate for learning have shown that in at least one invertebrate, *Aplysia californica*, learning is dependent on link formation. Kandel and his colleagues have shown in a series of papers (e.g. Hawkins, Abrams, Carew, & Kandel, 1983; Hawkins, Carew, & Kandel, 1986; and see Hawkins (1989) for a discussion) that learning in *Aplysia* takes place by means of either enhancing or degrading the effectiveness of synaptic connections between sensory and motor neurones. Here then, the activities of individual neurones are the representations and the synaptic connections between them are the links. *Aplysia* has also been shown to display many of the standard classical conditioning phenomena (acquisition, extinction, differential conditioning, ability to track contingency, blocking, conditional

discrimination; see Colwill, 1985; Colwill, Absher, & Roberts, 1988; Hawkins et al., 1986); thus the case for there being an associative basis for this type of learning in at least one animal is very strong indeed. Our first point, then, is that associative learning exists. The corollary must be that if it exists in *Aplysia*, it would be very surprising if it did not also exist in other animals, and not wholly surprising to find it in the laboratory rat or pigeon, (and perhaps even in humans for that matter). The demonstration of the existence of associative learning in *Aplysia* nullifies the single process argument that follows from conceding the existence of propositional learning in humans. It would seem that there are at least two types of process supporting learning in the world. The question becomes whether we are endowed with both of them or not, rather than whether either of them exists.

At this juncture it is worth stating that the evidence also points to an error-correcting form of associative learning in humans and other animals. We have already mentioned that *Aplysia* displays phenomena (e.g. blocking) that are often taken as evidence for error-correction. Whilst the basic neural mechanism for learning in *Aplysia* does not, at first sight, seem to be of an error-correcting nature, both Hawkins (1989) and McLaren (1989) have shown that an assembly of the type of neurones found in *Aplysia* will function as an error-correcting computational system. There can be little doubt that the eyeblink response in the rabbit (Thompson, 1965; Thompson, 1989) is controlled by an error-correcting circuit, and there is also evidence from Wolfram Schultz's laboratory that neural correlates of prediction error can be found in primates (see Schultz, Dayan, & Montague, 1997). Behavioral phenomena such as superconditioning and over-expectation (see Miller, Barnet, & Grahame, 1995 for a review) support the case for associative learning in animals being driven by some form of error correction. Rescorla and Wagner (1972) have provided the benchmark model for associative learning based on error correction, but it is worth mentioning that other models such as those of Mackintosh (1975), Pearce and Hall (1980) and Pearce and Mackintosh (2010) that are not often thought of as error-correcting do, in fact, depend on something akin to that process. Adjustment of associability in these models relies on a summed error-term, and this associability parameter controls learning (see McLaren & Dickinson, 1990, for a discussion of this issue, and how associability might be implemented neurally). Even Pearce's (1987) configural model is more error-driven than is commonly considered to be the case: It uses a simple, single error term to control learning between a configural CS and US, but the fact that it allows generalization from other trained configurations to contribute to that error term effectively allows a summed error term to influence learning. Each of these models is able to account for a wide range of the effects found in the associative learning literature. Thus there is considerable evidence, both empirical and theoretical, for learning being controlled (or at least influenced) by prediction error.

3. The case for association–Pavlovian conditioning

A second reason for believing that animal learning is associative is that it can be shown to be irrational in some circumstances, and produce behavior that is not at all what one would expect from a propositional system. It must of course be the case that if it is to be adaptive and enable the animal to survive, in most circumstances the outcome of associative learning should parallel that to be expected from a rational, propositional system. Thus, the ability to track contingencies (Rescorla, 1967; Wasserman, Chatlosh, & Neunaber, 1983) is exactly what you would expect from either system. The phenomenon of blocking (Kamin, 1968) is also susceptible to explanation in associative or propositional terms (see Beckers, Miller, De Houwer, & Urushihara, 2006, for

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