Associative analyses of reasoning-like behaviour in rats

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List of Publications

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Note: The text of Chapter 2 reflects closely the text of Burgess, Dwyer and Honey (2012), while that of Chapter 3 reflects closely the text of Burgess, Dwyer and Honey (under review), and that of Chapter 4 reflects closely the text of Dwyer, Burgess and Honey (2012).

List of Presentations

Katy V. Burgess, Dominic M. Dwyer, Robert C. Honey (2012). *No evidence for causal binding in rats from studies of timing relative to actions or external cues*. American Psychological Association 2012, Orlando, Florida.

Katy V. Burgess, Dominic M. Dwyer, Robert C. Honey (2012). *Timing from actions and observations: Implications for causal accounts of conditioned behaviour in rats.* Experimental Psychology Society Meeting, Hull.

Katy V. Burgess, Dominic M. Dwyer, Robert C. Honey (2012). *Sensory preconditioning with flavours: Avoidance not aversion.* Associative Learning Symposium, Gregynog, Newtown, Powys.

Katy V. Burgess, Dominic M. Dwyer, Robert C. Honey (2012). *Sensory preconditioning with flavours: Evidence against stimulus substitution.* Behavioural Neuroscience Lab Seminar Series, School of Psychology, Cardiff University.

Katy V. Burgess, Dominic M. Dwyer, Robert C. Honey (2012). *From causal reasoning to causal blindness*. Associative Learning Group Seminar. Sydney University, Sydney Australia.

Katy V. Burgess, Dominic M. Dwyer, Robert C. Honey (2012). *From causal reasoning to causal blindness*. Associative Learning Group Seminar. University of New South Wales, Sydney Australia.

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Katy V. Burgess, Dominic M. Dwyer, Robert C. Honey (2011). *Timing from an action or an external stimulus: Causal blindness in rats.* Associative Learning Symposium, Gregynog, Newtown, Powys.

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Thesis Summary

This thesis examines how rats represent relationships in their environment. There are currently two broad classes of account of how animals learn about such relationships: The associative account offers a relatively simple mechanistic account of behaviour; while the second account proposes that animal behaviour, like human behaviour, is underpinned by the processes of causal and deductive reasoning, that are beyond associative analyses. Chapter 1 identifies three domains in which these two classes of account provide quite different analyses of animal behaviour, which are experimentally investigated in Chapters 2, 3 and 4.

Chapter 2 reports three experiments that investigated the accuracy of predictions derived from the claim that rats are capable of forming and using causal models involving their own interactions with their environment (interventions) and external events (Blaisdell, Sawa, Leising, & Waldmann, 2006). The results failed to confirm these predictions and were instead more consistent with the operation of simpler processes.

The results from Chapter 2 left open two interpretations: either rats can represent causality but do not use such representations to reason, or they do not represent cause per se. Chapter 3 investigated these alternatives in three experiments using a timing task, which should be sensitive to whether rats are more likely to represent their actions as causal than external events (Buehner & Humphreys, 2009). The results provided no support for the view that causal binding occurs in rats.

Chapter 3 examined the possibility that sensory preconditioning might reflect a form of deductive reasoning (Hall, 1990). However, taken together, the results from four experiments provided no support for such an analysis; but instead helped to inform the nature of the associative processes that underlie sensory preconditioning.

In summary, while the results reported in this thesis provide no support for analyses of animal behaviour that rely on the processes of causal or deductive reasoning, they do help to inform the nature of the associative processes involved.

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Chapter 1: General Introduction

1.1. The study of animal behaviour: A selective review

The academic study of animal behaviour stems from the development of Charles Darwin's (1859) influential theory of evolution. In his book describing the mechanism and outcome of evolution, Darwin hinted at the idea that humans had evolved from simpler animals. This was a controversial proposition at the time of publication, and he did not expand upon this notion until the 1870s, when he also entertained the complimentary idea of mental and behavioural similarity between humans and animals (Darwin, 1871). It was this idea, that human intelligence has evolved, which sparked an interest in the study of mental processes of animals from an anthropocentric viewpoint.

The question of whether animals process information and think about the world in an analogous manner to humans was entertained by Romanes (1882), who began observing animal behaviour and explaining it in terms of human thought processes. He argued that evolutionary pressures resulted in the need for a development of intelligence, and that this intelligence is shared with animals, from ants to dogs. Nonhuman animals, he argued, have rich ways of representing the texture of their environment, and possess cognitive processes that are equivalent to those of humans. However, Romanes had little evidence for his claims, and Morgan (1894) was the first to suggest that experiments should be conducted in order to assess the cognitive abilities of animals. In his book *Introduction to Comparative Psychology*, Morgan directly considered the appropriate means to assess the mental capacities of other species. He took issue with people projecting human methods of thinking and reasoning onto animals, as evolution has resulted in an enormous variety of species, which in turn, he argued, must have also formed divergent types of "mind" (defined in his book as any type of mental activity). He noted, however, that people tend to see a final behaviour and explain it in terms of complex human cognitive ability without evidence of this kind of process. From his

own observations, he saw that method by which animals learned a particular response was slow, and achieved by simple trial-and-error learning. He thus argued that a more scientific method should be advocated, and that complex cognition should not be merely assumed. He thus stated his canon; *"in no case may we interpret an action as the outcome of the exercise of a higher psychical faculty, if it can be interpreted as the outcome of the exercise of one which stands lower in the psychological scale"* (p53). That is, if there are two mental processes available to explain an animal's behaviour, the simpler of the two alternatives should be favoured, until the more complex account can be proven. Comparative psychology, he argued, needs to discover the limits of animal psychology, and distinguish the cognitive level any given animal has obtained.

Although the canon has recently been criticised as the sole basis upon which to arbitrate between different accounts of animal behaviour (e.g., Heyes, 2012), it highlights the importance of at least considering simple accounts of behaviour. Logically, there is no reason that a simpler account is preferable, and Heyes' objection to the canon is centred on the idea that it appears to use associative learning as the default explanation of behaviour, rather than a contender. Perhaps the canon should not be taken at face value, but even so, it should be kept in mind that one should not assume complex behaviour without ruling out simpler accounts. In a similar vein, Heyes argued that in order to distinguish between associative and more cognitive explanations, further experiments need to be run to rule out one or the other as a potential explanation. Whether Morgan's canon or Heyes' critique is accepted, it seems clear that adjudication between alternative accounts is best served by experimental analysis.

At the turn of the 20th century, new approaches to the study of animal behaviour began to emerge. Thorndike (1911) and Pavlov (1927) provided clear descriptions of some of the earliest formal studies of animal cognition. Their experimental procedures arranged contingencies in an animal's environment (e.g., between an action and an outcome or

between one stimulus and an outcome), and measured changes in the animal's behaviour. For instance, Pavlov provided hungry dogs with food immediately following a tone, and measured salivation. He observed that initially only the delivery of food (the unconditioned stimulus, US) would elicit salivation (the unconditioned response, UR), but after a succession of tone-food pairings, the dogs salivated not only during the food, but also during the tone alone. This classical conditioning procedure differed from Thorndike's (1911) approach, wherein animals were only reinforced with an outcome (escape from a box and access to food) following certain behaviours (pressing a catch or lever). The measurement taken was the latency to escape the box, and it was observed that the time taken decreased as the animal learnt the relationship between their behaviour and escape. These two methods of studying changes in animal behaviour, based on contingencies arranged by the experimenter, have provided the foundation for a vast amount of research on animal learning that has followed.

Pavlov and Thorndike not only provided reliable methods of assessing animal learning, but also suggested a way of understanding the underlying basis of learnt behaviour. In fact, earlier than this, Morgan (1894) suggested that animals learn about the contingencies in their environments through mental representations, which connected events through associations. He argued that neural centres represent certain events, and when these events happen in close temporal proximity, the representations are activated and a connection is formed. So, for instance, Morgan noted that newborn chicks learnt to avoid pecking cinnabar caterpillars, which have an aversive taste. According to his theory, the chick would see the caterpillar, which would produce the motor response of pecking, shortly followed by an aversive taste. The mental representations of the caterpillar and taste would be activated in close succession, and upon later presentation of the caterpillar, the neural representation of the caterpillar would now activate the representation of the aversive taste, which would exert an inhibitory effect on the motor system and thereby prevent pecking.

This associative analysis, now supported by evidence (see Pavlov, 1927), had a large impact on how theorists think about how animals learn about the relationships between environmental stimuli. While early learning theorists focused on the nature of associative structure (i.e., whether the association was formed between a stimulus and a response, or between the stimuli themselves), researchers in the mid-20th Century began to investigate the mechanisms underlying association formation, rather than what constituted the associative content (Delamater, 2012). Early theorists believed that contiguous pairings of environmental stimuli was sufficient to activate mental representations of these stimuli in close succession, and that connections are formed automatically (Guthrie, 1935). However, modern theories of associative learning in animals, unlike their predecessors, now suppose that mere contiguity is an insufficient condition for learning to occur. Although the processes involved in association formation are still assumed to be automatic, they are more complex than previously imagined. That is, current theories acknowledge that it is not sufficient for the elements of association to be presented close together in time. To enter into an association, the stimuli must be attended to (e.g., Mackintosh, 1975), and be surprising (e.g., Rescorla & Wagner, 1972). The following paragraphs will explore these conditions of learning.

The suggestion that stimuli must be attended to was foreshadowed by Pavlov (1927), who observed that dogs presented with a novel light displayed an orienting response (OR), which allowed them to observe changes in its environment. Amongst many others, a study by Kaye and Pearce (1984) showed that the frequency of the orienting response in rats decreases the more a light is exposed. This effect is termed habituation. Habituation training also affects subsequent learning about a given stimulus. In the second phase of their study, Kaye and Pearce gave the experimental group of rats, who had pre-exposure to the light alone, and control rats with no experience with the light, pairings of light with food. That is, whenever the light illuminated, food would be delivered. The group who had been habituated to the

light showed a slower rate of learning about the relationship between the light and food than the control group (see also Hall & Channell, 1985). This effect, known as latent inhibition, supports the idea that attention must be paid to stimuli in order to learn about them (e.g., Mackintosh, 1975; Pearce & Hall, 1980)

The idea that the stimuli must be surprising in order to learn about them is perhaps best exemplified by experiments on blocking (Kamin, 1969). Kamin used a conditioned suppression procedure in which a CS (A) was first paired with the delivery of a shock. This training ordinarily results in the CS suppressing the instrumental baseline response of lever pressing. Kamin's critical finding was that should a second CS (X) be paired with shock in the presence of A, then X would not suppress responding when it was presented alone at test. This blocking effect, among others, was the motivation for the development of the model described by Rescorla and Wagner (1972). To evaluate whether a given behaviour is beyond an associative analysis (or associative analyses) one needs first to understand something about the details of these analyses. A brief review of such theories follows, by way of providing a theoretical backdrop against which to judge whether putative instances of animal behaviour, that might have a complex origin, are also explicable in more mundane, associative terms.

1.1.2. Formal theories of association formation

Rescorla and Wagner (1972) presented a mathematical algorithm to explain how associations form (see Formula 1.1). Here, ΔV represents the change in associative strength on a given trial, $\alpha\beta$ represents the associability of the stimulus (one for the associability of the CS (α) and another for the US (β)), λ is the asymptotic level of associative strength the US provides, and ΣV is the sum of associative strength for all stimuli present on a given trial. In very general terms, this formula can be used to make ordinal predictions about how much will be learnt on a given trial; which will be related to the salience of the stimuli (α and β),

what is known about the stimuli that are presented on the current trial from previous trials (ΣV), and the maximum amount of associative strength the US can support (i.e., λ). Learning is proportional to the difference between what is expected to happen and what actually happens.

$$\Delta V = \alpha\beta\ (\lambda - \Sigma V) \qquad \textit{Formula 1.1}$$

In a related model, Wagner (1981) showed how the central ideas that underpin the Rescorla-Wagner model could be implemented in a set of standard operating procedures (SOP) for animal memory. Here, the memory system consists of a series of directional associative links, and the memory of a stimulus can be in three states: inactive, whereby the stimulus is stored, and not capable of modifying behaviour; A2 when the stimulus is active but not present; and A1, in which the stimulus is active in the animal's attention. When a stimulus is presented, the nodal elements representing that stimulus are active in the A1 state. The representation is then thought to decay into the A2 state, and finally into the inactive (I) state (see Figure 1.1). Within this model, an excitatory association, in which the activation of a recipient node is increased, is formed when the CS and US nodes are in the A1 state. When the CS node is in the A1 state and the US node is in the A2 state an inhibitory association, in which the recipient node decreases in activation, is strengthened between them.[1] However, when the CS node is in the A2 state, no learning occurs. The final assumptions allow the

[1] Especially relevant to the empirical work presented in Chapter 3, is the fact that this already powerful model can be extended to deal with temporal order of stimuli, as Wagner (1981) argued that a stimulus activates a cascade of elements into A1. For instance, imagine that element A, then B, then C are activated during a CS. If a US is presented at the time that element C of the CS is activated, then an excitatory association will form between C and the US, and activate the US representation when element C is active, rather than during the activation of A or B. This differential conditioning between A, B, and C encodes the fact that the US is presented at the end, rather than at the beginning, of the CS.

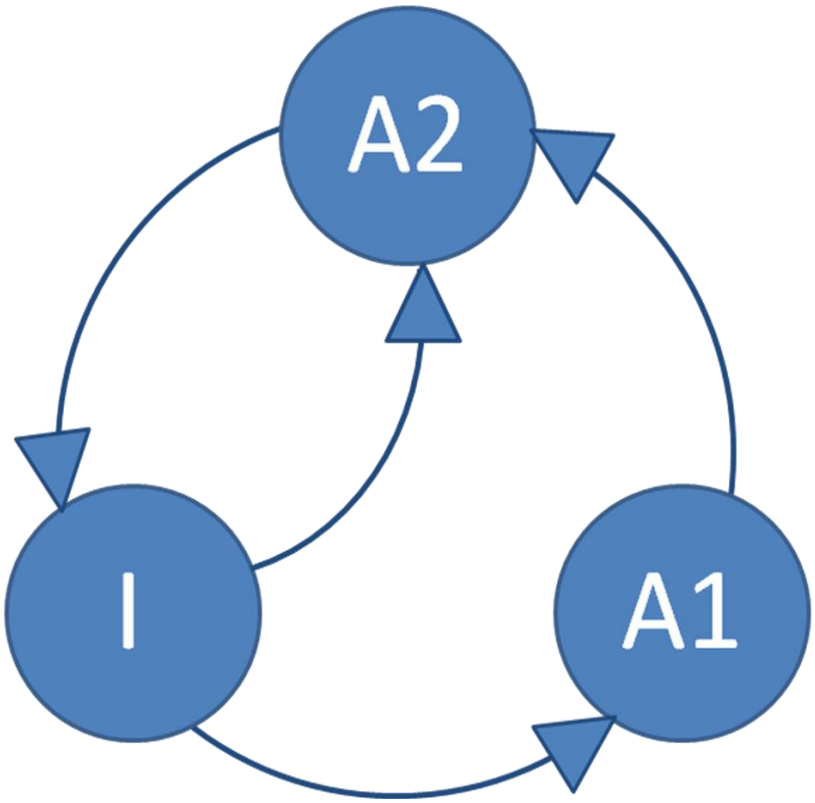


Figure 1.1. Wagner's SOP model, where A1 is the primary state of activation, A2 is the secondary state of activation, and I is the inactive state. The arrows represent the movement of nodal elements between the states of activation. The arrows from the inactive state represent activation of elements into the active states (experience of the stimulus activates elements from I into A1, activation by association activates elements from I into A2), whereas the other two arrows represent decay of elements.

model to explain latent inhibition and the fact that it is context specific, among a broad range of other phenomena (Honey, Iordanova, & Good, 2010). Latent inhibition involves repeated exposure to a stimulus before presenting trials in which the stimulus is paired with a motivationally significant event (e.g., food). The development of conditioned responding involving such a preexposed stimulus is usually retarded (as outlined in Section 1.1). Wagner's (1981) SOP explains this in terms of the initial exposure to the stimulus forming an association with the context in which it is presented. Thus, when the rat has learnt this association, whenever it is placed in the context, a memory of the stimulus will be activated into the A2 state. When the stimulus is presented, some elements from the inactive state will move into the A1 state, but elements in A2 cannot move directly into A1. The amount that can be learnt about the stimulus is limited to the elements active in the A1 state. As this amount is less in the context of pre-exposure, learning rate is reduced in comparison to control groups (Honey *et al.*, 2010). Due to its ready application to numerous phenomenon, the SOP model (along with its various modifications, such as Lin & Honey, 2011) will be used as a primary theoretical instrument to gauge whether a given behavioural observation is beyond an associative analysis. Of course, if there are yet simpler accounts, that require fewer theoretical assumptions (e.g., response competition, generalization decrement, or other performance-related constraints) then these will also be identified.

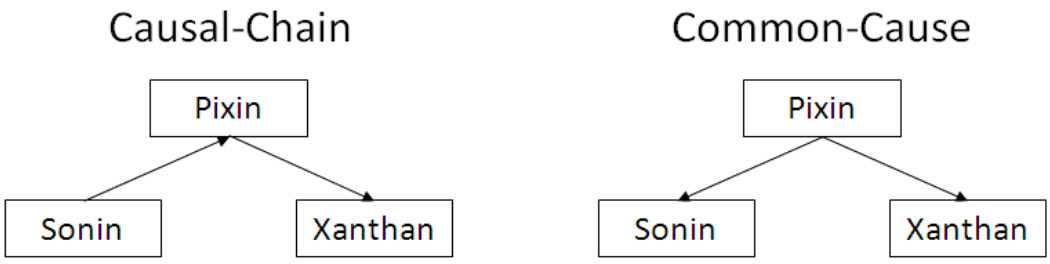
1.2. From representing casual relations to understanding causation

The development of formal models of associative learning has allowed an impressive range of phenomena to be explained, and the accuracy of novel predictions to be assessed. However, it should be noted that the models are relatively simple: a single construct, the association, still provided the bulk of the explanatory power. Yet, it has been argued that associative learning provides animals with a means of representing the causal structure of their environment (e.g., Dickinson, 1980). Indeed even evidence that, at face value, seemed to

undermine general process learning theory, can be reinterpreted as supporting the general idea that associative processes enable the causal structure of the environment to be represented. So, for instance, if you feel nauseous after eating a meal at a new restaurant, you would attribute the illness to the meal itself rather than anything associated with the restaurant, such as the decor. Similarly, Domjan and Wilson (1972) injected rats with lithium chloride (which induces nausea) or saline (a control substance, which does not affect the animal) after they had ingested a flavoured solution in the presence of an audiovisual stimulus. Rats learnt to avoid the solution but not the audiovisual stimulus. The reverse of this was found when rats were given a shock following the taste and audiovisual cues. That is, when shocked, rats avoided the audiovisual cues but not the solution. They suggested that rats learn about relationships depending on the nature of the stimuli involved (although there are other possible explanations reliant on the CS/US contiguity). Dickinson (1980) interpreted such findings as suggesting that illness and taste stimuli are more likely to be part of the same causal-chain than are illness and audiovisual stimuli (the idea of a causal-chain is discussed further below); but supposed that an associative analysis of such selectivity remained a viable account.

Even if one accepted the proposition that associative learning might provide animals, including humans, with a means of representing the causal structure of their environments this need not imply that associative learning provides the only means of representing causal relationships. For example, it has been argued that humans, and other animals, are capable of representing causal relationships with what are referred to as causal models (Blaisdell *et al.*, 2006; Waldmann & Holyoak, 1992). An experiment by Waldmann and Hagmayer (2005) illustrates the operation of such causal models in humans. They provided participants with a hypothetical scenario in which scientists hypothesised that the production of three substances

(a)



(b)

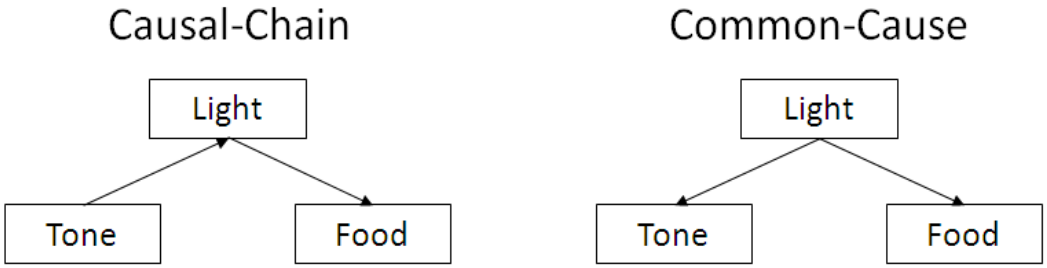


Figure 1.2. (a) Training conditions in Waldmann and Hagmayer (2005). The left diagram displays the causal relationship between the hormones Sonin, Pixin and Xanthan in a causal-chain arrangement. The right diagram displays the causal relationships in a common-cause model. (b) Training design of Blaisdell *et al.*'s (2006) study. The left diagram displays the causal-chain scenario, in which the tone predicts the light, which in turn predicts the delivery of food. The right diagram displays the common-cause scenario, in which the light is a cause of both the presentation of the tone and delivery of food.

(Pixin, Sonin and Xanthan) were related to one another (see Figure 1.2a). This scenario was presented in two conditions: a causal-chain model, whereby scientists hypothesised an increase in Sonin caused an increase in Pixin, which in turn increased Xanthan (that is, Sonin → Pixin → Xanthan); and a common-cause model, whereby an increase in Pixin caused an increase in Sonin and Xanthan (that is, Sonin ← Pixin → Xanthan). Participants were informed that other causal factors may influence the production of certain hormones. Alongside this information, the participants were provided with hypothetical data showing the levels of the three substances in chimpanzee studies; eight of which had elevated levels of all substances, eight with normal levels of the substances, and four with varying levels of the substances. Both conditions revealed a probability of .9 between all the causal links in the models. So, for instance, if Pixin changed in strength, there was a probability of .9 that Xanthan would behave in the same manner. The test presented participants with another hypothetical situation whereby the level of Sonin levels had increased or decreased either by means of an intervention or the change in Sonin levels was merely observed. That is, an intervention whereby an action directly manipulated the levels of substances, in comparison to an observed change with no known manipulation.

Participants in Waldmann and Hagmayer's (2005) study were asked how many animals would have an increased level of Xanthan given different variations in Sonin. The results revealed that participants can derive from this data that when an increase/decrease in Sonin is observed in the common-cause model, Pixin levels would have had to increase or decrease accordingly to cause the change in Sonin. In the causal-chain model, Pixin would have increased or decreased as an effect of the levels of Sonin. However, when an intervention is included, which directly changes the levels of Sonin, the predictions from the two types of model changes. In the causal-chain model, an increase in Sonin would cause an increase in Pixin, and in turn Xanthan. In the common-cause model, Sonin is an effect, not a

cause, and therefore, a change in the amount of Sonin will have no influence on the other substances.

The results revealed that humans distinguish between the effects that actions and external stimuli have on events in the environment. Causal model theory assumes that this is due to the fact that we are aware of the directionality of cause and effect, and understand the implications of actions through experience. One way to illustrate this formal derivation is through an everyday example described by Clayton and Dickinson (2006). When we know that rain (analogous to Pixin) can wet windows and clothing on a washing line (analogous to Sonin and Xanthan in the common-cause group), the observation of the wet windows can lead to the inference that the washing on the line is becoming damp (Clayton & Dickinson, 2006). This form of inference must reflect the assumption that the observed effect (wet windows) is the result of its frequently observed cause (rain), which should also produce the second effect (damp clothes); and should then set in train an appropriate course of action (retrieving the washing). However, if one knows that there is an alternative cause for the first effect (e.g., a sprinkler is operating near the windows, but not the washing) then it should no longer be inferred that the washing is also getting wet. That is, the sprinkler is a direct manipulation of the events in the causal model. Under these conditions, the response of retrieving the clothes is not required. Modulation of the behaviour that is provoked by a stimulus (wet windows), as a function of the origin of that stimulus, is predicted by causal model theory (for a review see, Waldmann, Hagmayer, & Blaisdell, 2006). However, in this paradigm, an observation only occurs within the framework of the three substances within the casual model. That is, there is no knowledge of an alternative cause, in this instance a sprinkler, manipulating the events. Thus, the predictions derived from observations and interventions will be different. However, this procedure did not require the participants to interact directly with the events within the model – they were provided with the relationships

and asked to make inferences from them. It would be interesting to see what results would be obtained if the participants had to derive the causal models themselves. Although this is not an immediate concern of this thesis, it should be evident that when rats are given an analogous situation they are required to learn or derive the putative causal models from experience. Section 1.2.1 will outline how the rat version of this task is conducted (see also Figure 1.2b).

1.2.1. Do animals form causal models?

One commonly used method for assessing the ability of animals to understand causality is by examining their ability to use tools in the trap-tube task (e.g., Fujita, Kuroshima, & Asai, 2003; Martin-Ordas, Call, & Colmenares, 2008; Santos, Pearson, Spaepen, Tsao, & Hauser, 2006; Tebbich & Bshary, 2004; Visalberghi & Limongelli, 1994). Here, animals are required to retrieve a piece of food from a transparent tube with a stick. On the face of things, this requires the animal to understand the cause (movement of the stick) and effect (movement of the food) of the situation, along with the effects of gravity. While this method could potentially be used to assess causal judgements in non-human primates and birds, extensive training with the apparatus was often required which raises the possibility that performance might be governed by trial and error rather than reasoning.

Hall (1990) stated that "given appropriate conditions of training, many animals may be better at reasoning than we have supposed" (p178). So, it appeared to be a fundamental advance when Blaisdell *et al.* (2006) developed a new paradigm which could be run in an operant chamber, using a simple behavioural paradigm to demonstrate a form of reasoning in rats. Their task was developed from an analysis of causal model theory (for a review see, Waldmann *et al.*, 2006), and was effectively a rat-based version of the type of reasoning task used in humans (see Section 1.2) by Waldmann and Hagmayer (2005). While humans were presented with causal diagrams along with data, rats were trained using individual links of a

causal model. More specifically, the training scenario that Blaisdell *et al.* (2006) gave their rats is analogous to the everyday situation described above (see Figure 1.2b). In their study, rats first received training in which one stimulus A (e.g., a light - analogous to raindrops) served as the "common-cause" for two separate effects: B (e.g., a clicker - wet windows), and the delivery of sucrose into a food well (damp clothes). This common-cause treatment was arranged by giving rats separate A→B and A→sucrose trials. During the subsequent test, B was presented and the tendency of rats to approach the food well was examined as a function of whether the presentation of B was contingent upon rats pressing a lever (intervene condition) or simply occurred without apparent cause (observe condition). Other rats were trained according to a "causal-chain" scenario, where the order of A and B were reversed (i.e., B→A and A→sucrose trials). In this condition, rats might be expected to respond to B irrespective of whether it occurred as a function of their own behaviour, because the chain of associations linking B to sucrose would be unaffected by the manner in which B appeared. Blaisdell *et al.* observed that when the rats had received common-cause training, they were less likely to approach the food well during B in the intervene condition than in the observe condition. In contrast, this was not the case when they had received causal-chain training. That is, there is an interaction, which mirrored the results on human participants conducted by Waldmann and Hagmayer (2005).

Blaisdell *et al.* (2006) claimed that rats had an understanding of the implication of the lever press within the causal model, and thus were capable of forming and using causal models. However, as mentioned previously in Section 1.1, Morgan's (1894) canon suggests that we should rule out simpler alternatives before appealing to more complex processes. Indeed, given certain simple assumptions, the results presented by Blaisdell *et al.* (2006) can be explained in terms of the operation of a very simple process (i.e., response competition). Referring back to the behavioural task presented to the rats in the study, the alternative cause

of the auditory stimulus was a lever press. For the rat to explore the lever, it would have to move away from the food magazine. Thus, it is clear that any tendency to interact with the lever would interfere with any tendency to interact with the food magazine. Thus, competition between responses could have contributed to the lower levels of magazine responding during the auditory stimulus when it occurred as a consequence of a rat's own actions. If correct, this very simple account suggests that, across experimental conditions, there should be a reciprocal relationship between the tendency to press the lever and the tendency to enter the food magazine during the auditory stimulus. That is, all experimental conditions ought to be assessed on lever pressing activity, as if, for instance, there are higher lever pressing rates during the intervene conditions, this can explain why there is less nosepoking activity in those conditions. The original data do not speak to the possibility of response competition because lever press responses were not recorded (for all conditions) as a function of whether they occurred during the auditory stimuli (Dwyer, Starns, & Honey, 2009).

Dwyer *et al.* (2009) noted the above criticism, and replicated the procedures used by Blaisdell *et al.* (2006) while recording lever press responses in a manner that allowed a direct test of any response competition. Briefly, during the critical auditory stimuli, a reciprocal relationship was found whereby food magazine responding was low when lever pressing was high. Unfortunately, however, they were not able to replicate the theoretically vital interaction from the original study whereby the actions only interfered with magazine responding during the auditory stimulus in the common-cause condition but not the causal-chain condition (but see, Leising, Wong, Waldmann, & Blaisdell, 2008). Thus, while there is direct evidence that lever press and food-magazine responses do compete with each other in this type of study (and so the idea that response competition might contribute to the observed behaviour is supported), I know of no experiment where this possibility can be directly tested

as an account of the interaction between the effects of the manipulation of training condition (common-cause versus direct cause or causal-chain) and the test condition (observe versus intervene).

It should be noted that the existence of competition between lever press and magazine responses does not, in itself, invalidate the claim that rats might be capable of causal reasoning. As aforementioned, logically, the fact that there is a simpler alternative does not mean that a cognitive process is not occurring. The response competition may be obscuring any causal behaviour, and thus the simpler alternative should not be assumed to be true (Heyes, 2012). That is, if response competition was removed as a variable, and the result is consistent with causal model theory, then the causal account may explain the behaviour. Thus, the first aim of this thesis was to extend the empirical investigation of the possibility that rats might be capable of causal reasoning using the procedures developed by Blaisdell and his colleagues. The results of this investigation will be presented in Chapter 2, where an account based on the idea that rats engage in causal reasoning using causal models, is contrasted with alternative theoretical analyses based upon response competition, in the first instance, and a modification of Wagner's SOP, in the second instance.

1.3. Causal binding

The studies conducted by Blaisdell *et al.* (2006) assume not only that rats can engage in causal reasoning, but that they do so in a particular manner. As such, any failure to observe results consistent with the predictions of causal model theory might be due to either a failure of the rats to represent causal structures at all, or perhaps be due to them not utilising causal knowledge in the way supposed by causal model theory. Thus a more general question is whether animals represent cause-effect relationships in a way that goes beyond associations between the events involved. One informative approach to this issue is to examine potential markers of causal representation that do not involve additional inferential processes.

It is generally accepted among cognitive psychologists that humans use more complex reasoning methods that work alongside an associative mechanism (see, Evans, 2008 for a review on the dual process account of human cognition). However, it is argued that the human sensory system does not perceive causal relationships directly; instead causality is inferred from evidence provided by the senses, such as contiguity and contingency of events (Shanks, Holyoak, & Medin, 1996). This idea was suggested by David Hume, who argued that causality is an illusion based on associations. This idea is supported by the fact that perceivable information in the environment has an impact on the perception of causality. A well-established phenomenon in the human literature is that of intentional or causal binding (Buehner & Humphreys, 2009; Haggard, Clark, & Kalogeras, 2002). This binding effect highlights the difference between observations and interventions as causes of events. A person's perception of time differs depending on whether an action is the cause of an event, or an observed external stimulus is.

People's sense of time was first measured formally by Libet, Gleason, Wright and Pearl (1983), who created a method whereby participants had to match an event with the time on a fast moving clock. This methodology was later employed to assess how the perception of causality was affected by whether the cause of an outcome was self-generated or not (Haggard *et al.*, 2002). Participants had to report when certain events occurred. In one condition, the outcome was produced by the participant's own action, in the other it was produced by an external stimulus (a tone). Haggard *et al.*'s (2002) results revealed that an action resulting in an outcome was perceived as closer together in time than the same outcome caused by an external event, and argued that people temporally bound actions to their effects. They termed this effect *intentional binding*, as they believed it provided evidence for the view that actions are different from external events in that actions are intentional. Haggard *et al.*'s (2002) analysis is consistent with the view expressed previously

that in human cognition, "the psychological antecedent of an instrumental act is the interaction of a belief (or representation) that there is a causal relation between the action and its outcome, with a desire for that outcome through a process of practical inference" (Dickinson & Shanks, 1995, p6). So, for example, when you turn on a light, you do so as you want the outcome of illumination to occur, and believe that the action of pressing the switch will turn on the light.

However, a potential issue with this methodology was noted in a later study (Buehner & Humphreys, 2009): Haggard *et al.* (2002) used subjective reports, rather than a more direct measure of behavioural data. Buehner and Humphreys (2009) used a stimulus anticipation task, whereby participants were given two conditions (as with Haggard *et al.*, 2002 study); one where an outcome was dependent on an action (causal-control condition), the other when it was the result of an external cue (baseline condition). Figure 1.3 shows the experimental procedure in diagrammatic form. In the exposure phase of training, participants were presented with the two trial types. In the baseline condition, two preparatory tones preceded Tone 1 (T1), which then had a delay followed by Tone 2 (T2). In the causal-control condition, however, participants would respond on a key (R1), and this would produce T2. In the training phase, participants were asked to synchronise their responses with the presentation of T1. The only difference at this point was that in the causal-control condition, the response at T1 is causally linked to the outcome, T2. Finally, in the experimental phase, participants were required to synchronise their responses to T1, but also to T2 (R2). Again, the only difference between the conditions was that in the causal-control condition, R1 and T2 were causally related. Participants were required to produce the same response in both conditions. The results of the experiment supported earlier findings of binding actions to their

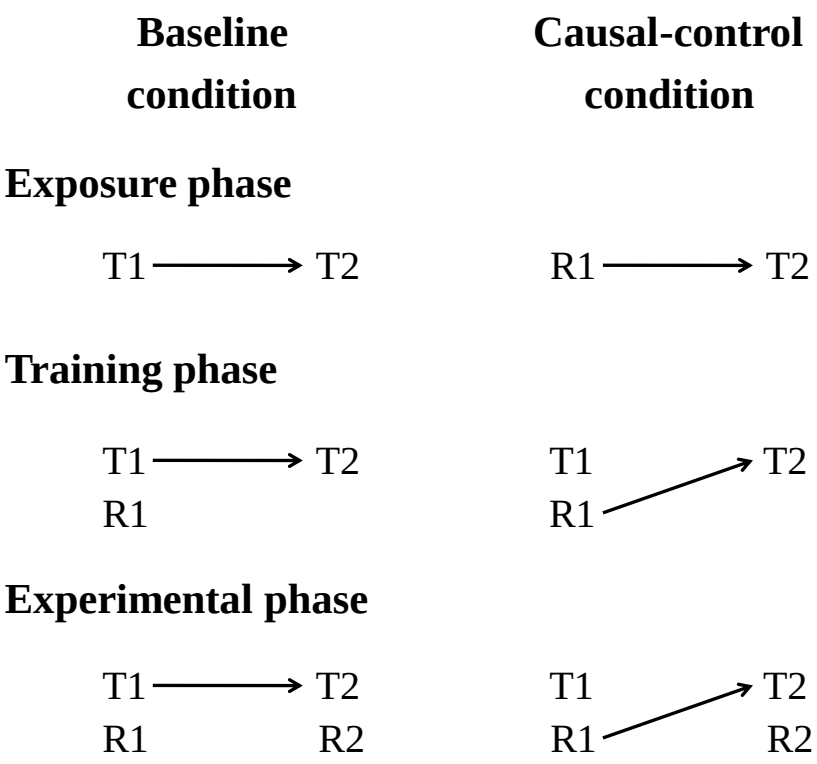


Figure 1.3. Methodology employed by Buehner and Humphreys (2009). T1 and T2 are a tones separated by a time interval, R1 and R2 are responses required to be synchronised with the two tones respectively. In the baseline condition, the two tones are related, whereas in the causal-control condition, R1 is causally related to T2.

outcomes, as participants judged T2 as earlier in the causal-control condition than in the baseline condition. However, Buehner and Humphreys termed the effect *causal binding*, as in both conditions there was an action involved, so intentionality and agency were controlled for. Their results instead suggested that it was the knowledge of causality that produced the effect.

The study of timing in animals has a long and illustrious history (Shettleworth, 1998). For example, Pavlov (1927) provided dogs with a three minute whistle predicting the delivery of weak acid into its mouth. Dogs salivated most in the final minute of the whistle, providing evidence for the notion that animals are sensitive to time intervals. More recently, a novel technique was developed for studying timing processes, where animals were reinforced with food a fixed length of time after a signal, such as a tone, had been presented (Roberts, 1981). These reinforced trials were intermixed with some non-reinforced trials, in which the accuracy of the animal's knowledge of elapsed time was assessed. So, for example, when a rat in a skinner box expects food a certain interval after a CS, the rate of responding would be measured as the length of time per second the rat has its snout in the food well. From this, a timing curve can be produced, where the response rates increase to a peak (where the animal most expects food), and then trail off to baseline rates of responding until the next trial. The most accurate measure of a rat's expectancy of food is through a curve fitting procedure, which reveals the peak time of responding using a line of best fit through the data points obtained. This procedure will be explained in more detail in Chapter 3.

Formal theories of timing (e.g., Scalar Expectancy Theory, SET; Gibbon, 1977) were developed to account for the behavioural characteristics of timing behaviour in animals. SET assumes that there is a pacemaker, which is used to send pulses to an accumulator, which compares the current number of pulses to the memory the rat has of the number of pulses that preceded reward delivery on previous trials. The key principles of this theory are that

responding will occur when the difference between the current number of pulses and the stored memory exceeds some threshold, and that this difference is scaled as a function of the size of the stored interval. These principles allow SET to explain the fact that the variability in the time of responding around an expected value scales with the size of that expected value. In light of the present concerns, it is important to note that there is no formal statement within the theory regarding the nature of the signal which begins the timing process. As such, SET makes no explicit predictions of differences between timing from external events or internally produced actions. This absence of a formal inclusion of the nature of the stimulus which initiates timing is shared by other formal theories of timing behaviour. For example, Machado's (1997), Learning to Time (LeT) theory assumes that a series of behavioural states is activated by an event, and that the activity of these states rises and falls in series. Because of this serial activation, the degree of activity in a particular state at the time of reward delivery will depend on the temporal interval between the event which began the timing sequence and the time of the reward delivery – the states that are most active at this time will become best with the response (e.g., lever pressing or nosepoke activity). LeT assumes that the rate at which activity transitions between states is inversely proportional to the temporal interval involved, and so it can also account for the scalar properties of timing behaviour. However, the rate of transition between states is not formally linked to the type of event which began the cascade, and so it also makes no explicit prediction regarding timing from external cues or actions.

In short, while formal timing theories make different predictions with respect to *what* animals learn in timing tasks, they make the same prediction about timing following an action versus timing following an external cue. That is, neither of the theories noted here, nor other related timing accounts, makes a distinction between whether the temporal referent is an action or an external cue. Although there have been many studies of animal timing, the

question of whether timing is influenced by whether the temporal referent is an observed stimulus or an intervention (e.g., a lever press response) has not been addressed, and seemingly the default assumption in theories of timing is that there is no difference. Waldmann and Hagmayer (2005) however, argued that observational and interventional cues are fundamentally different in causal learning, as predictions derived from events can differ depending on whether one has caused the outcome, or merely observed it. The findings of Buehner and Humphreys (2009) and Haggard *et al.* (2002) suggest that timing in humans is influenced by whether the referent is an action or an external stimulus. The question of interest here is whether timing in animals is influenced in a similar way. If animals represent cause-effect relationships, they would be expected to treat interventions and observations differently. That is, if timing behaviour is subject to causal binding then timing when food will occur should be less accurate on interventional trials than observational trials. More specifically, the peak in responding should occur earlier in the interval after a response than after an external stimulus. This issue is investigated in Chapter 3. It should be noted that this is an interesting prediction because current theories of animal timing (e.g., SET and LeT) make no fundamental distinction between the temporal referents used to time (e.g., Gibbon, 1977; Machado, 1997). Similarly, associative analyses of timing derived from Wagner's (1981) SOP make no distinction, in principle, between the two types of time marker: both a response and a stimulus might generate a cascade of elements that could be used as a basis for timing behaviour.

1.4. Sensory preconditioning as deductive reasoning

As discussed in Section 1.2.1, Blaisdell *et al.* (2006) provided their control rats with what they referred to as a causal-chain scenario. That is, rats were provided with an auditory cue (CS1) which predicted a light (CS2), which in turn predicted the delivery of food (see Figure 1.2b). This type of training procedure is better known in the associative learning

literature as sensory preconditioning (e.g., Rescorla, 1980; Rizley & Rescorla, 1972). While the associative analysis of sensory preconditioning will be discussed in detail later, what Blaisdell *et al.* (2006) claimed about this causal-chain condition is particularly important. As Section 1.2.1 outlined, this procedure resulted in nosepoke responding following the presentation of CS1. Presumably, this depends on rats learning both the relationship between CS1 and CS2, and between CS2 and food. A note in Blaisdell *et al.*'s (2006) supplementary materials suggests that rats given this training, in a pilot study, did not initially make magazine responses when presented with CS1 (the tone) alone. However, when they removed the light (CS2) from the conditioning chamber, rats responded as one would expect following this type of training procedure. Blaisdell and his colleagues (Blaisdell, Leising, Stahlman, & Waldmann, 2009) suggested that the rats in the pilot study did not respond to CS1, as they had learnt a chain of events, which required CS1 to cause CS2, and in turn CS2 to cause the delivery of food. According to this conception of events, CS1 does not directly predict food, but instead its relationship with food is mediated by CS2. Thus, if CS1 is not seen to produce CS2, then the causal chain has been broken and food might no longer be expected. However, if the source of CS2 (i.e., the light bulb) is removed from the chamber, then there can be no direct challenge to the causal chain by observing the unlit bulb and so CS1 should elicit responding for food.

This exact prediction, that the presence of the un-illuminated light was breaking the causal chain and thus resulting in a lack of responding, was tested using essentially the same sensory preconditioning/causal chain procedure as already described (Blaisdell *et al.*, 2009). They observed that rats did indeed respond more to CS1 when the bulb used to display CS2 was removed from the chamber. This was cited as evidence consistent with the idea that rats represented the relationship between events in a causal fashion, and so were sensitive to the difference between the explicit absence of a cue (un-illuminated light) and lack of

information about a cue (light removed from the chamber). However, Blaisdell *et al.* (2009) did note that the results might also be due to a context change due to the removal of the light from the chamber. Further work using patterning procedures (Fast & Blaisdell, 2011) has also been interpreted as consistent with the idea that rats are sensitive to the ambiguity resulting from the absence of information when the light is removed from the chamber.

Although this account of rat behaviour might be appealing, Morgan's canon suggests that simpler alternatives should be considered. This is precisely what Dwyer and Burgess (2011) did with relation to these results. Dwyer and Burgess simulated the formal associative theories described by Pearce (1994), Harris (2006) and Wagner (2003), to Fast and Blaisdell's (2011) data. All of these formal accounts were able to produce the same pattern of results observed by Fast and Blaisdell if it was assumed that an unilluminated bulb was a cue that could enter into the learning process and subsequently control responding. That said, as discussed previously (Section 1.2.1), the mere fact that associative accounts are not directly inconsistent with a particular result is not a direct demonstration that the associative account is to be preferred. More complex cognitive accounts remain, at least, tenable until directly invalidated by experimental evidence.

As noted at the beginning of this subsection, sensory preconditioning is a long established effect within the associative learning literature (e.g., Brogden (1939), who reported the effect in dogs). The fact that a response elicited to CS2 also becomes elicited by CS1, even though CS1 is never directly paired with the motivationally significant event, is of particular theoretical interest, as it speaks directly to general theoretical accounts of the associative structures produced by learning. Numerous alternative explanations have been advanced for the phenomenon, such as an elemental associative chain, or a configural representation of CS1 and CS2 becoming linked to the reinforcer (see, for example, Iordanova, Good, & Honey, 2011; Rescorla & Cunningham, 1978). Whether the effect is due

to elemental or configural representations is still a topic of debate, but for the moment I will focus on a principle underlying both of these accounts: that of stimulus substitution (Pavlov, 1927). This is the basic idea that one stimulus is substituted for another as a result of learning an association between them. That is, CS1 elicits the same CR as CS2 due to the association between CS1 and CS2. In an elemental account, CS1 will evoke a memory of CS2, which in turn activates a memory of the reinforcer. In a configural account, the presentation of CS1 will evoke a memory of the compound cue CS12, which in turn is associated with the reinforcer. The indirectly activated memory is thought to be the same as the directly activated memory. The predictions made by stimulus substitution are in fact the same predictions made by the causal account of behaviour, but for different reasons. The causal account predicts the response to the two events to be similar due to a causal relationship between them, that is, the reinforcer occurs because of the preceding stimuli. The stimulus substitution account, however, argues that it is due to the presentation of one stimulus activating a memory of the other.

Although Blaisdell and his colleagues' argument is perhaps somewhat contentious given the volume of sensory preconditioning studies that have found effect without manipulating the access to the source of CS2 (e.g., Rescorla, 1980; Rizley & Rescorla, 1972; Ward-Robinson & Hall, 1996), it remains possible that animals might represent events within their environment in a manner that is beyond associative analyses. Indeed, Hall (1990) has suggested that sensory preconditioning might involve a process of deductive reasoning. He argued that during a sensory preconditioning procedure a rat might learn a set of propositions (i.e., that noise causes food, and food causes illness, therefore a relationship is formed between noise and illness). However, sensory preconditioning still occurs when stimuli are presented simultaneously (Rescorla & Cunningham, 1978), and simultaneous presentation may even produce larger preconditioning effects than subsequent presentation (Rescorla,

1980). On one reading, the success of simultaneous presentation in producing sensory preconditioning might seem inconsistent with a propositional account, as causes must occur before their effects. In contrast, an advantage for simultaneous presentation can be explained by an associative analysis, such as Wagner's (1981) SOP, whereby both the CSs are activated into the A1 state at the same time, creating a strong association (Brandon, Vogel, & Wagner, 2003). Thus, when one CS is presented after simultaneous training, it evokes the memory of the other CS (thus, CS1 can excite the representation of CS2 and vice versa). Alternatively, with simultaneous presentation, it is possible that the animal does not form a representation of each of the stimuli, but that it forms a representation of the compound (i.e., stimulus CS12, rather than CS1 and CS2 (Rescorla, 1981). As each element shares properties with the compound, then either element might activate, thus supporting generalisation between them. All this said, a propositional account can easily be defended by arguing that, while causes must precede effects in the world, the perception of cause and effect might occur simultaneously if the interval between them is very brief. Consider, for example, the situation where blowing on a wind instrument causes a sound to be produced and the causal action of blowing is essentially simultaneous with the sound that it produced.

Propositional and stimulus substitution make the same prediction about the nature of the CR. However, other associative accounts, such as mediated conditioning, are possible. Wagner's (1981) SOP states that when a CS is in the A2 state it cannot enter into an excitatory association with other stimulus. However, Holland (1983) provided evidence that violated this assumption: after pairing a CS with food, pairing the same CS with illness results in an aversion to the food. Holland suggested that this finding reflected the fact that the CS associatively evoked a memory of food, and this evoked memory became associated with illness by a process of mediated conditioning (see also, Hall, 1996; Lin & Honey, 2011). This separation of direct conditioning, that results when memories are directly activated by

stimuli in the environment, from mediated conditioning, which is based upon associatively retrieved memories, allows that the two forms of conditioning might be dissociated. In particular, it allows that while direct conditioning might result in the development of one set of conditioning responses, mediated conditioning might result a different set.

Thus, while some associative accounts, the causal-chain analysis, and the idea that sensory preconditioning is based upon deductive reasoning, all predict that sensory preconditioning will adhere to a strict principle of stimulus substitution, the mediated conditioning account does not. The experimental work reported in Chapter 4 examines the relative merits of these distinct theoretic accounts of sensory preconditioning.

1.5. Summary

The study of animal intelligence began in the late 19th Century, when Darwin (1871) suggested that humans have evolved from simpler organisms through a process of natural selection. This idea of an evolutionary continuum provided one reason for the similarities and differences between human and non-human minds to be of scientific interest. Some comparative psychologists argue that non-human animals have complex methods of representing the environment, similar to humans (Blaisdell *et al.*, 2006). Meanwhile, other theorists have put forward the idea that animals represent the environment using much simpler mechanisms, by forming connections between the representations of stimuli, and between responses and stimuli (e.g., Rescorla & Wagner, 1972; Wagner, 1981). The overarching aim of this thesis is to investigate further the nature of cognition in rats in three experimental contexts (causal learning scenarios, causal binding procedures, and sensory preconditioning).

Chapter 2: Causal reasoning

2.1. Introduction

Chapter 1 introduced the idea that animals other than humans may possess the ability to reason about the relations between causes and effects. Blaisdell *et al.* (2006) presented evidence from rats, that was consistent with causal model theory. However, an alternative explanation for the results was suggested by Dwyer *et al.* (2009), whose experiments revealed that lever pressing interferes with magazine activity. So, the more a rat presses a lever, the less likely it is to investigate the food well. This idea of response competition does not, in itself, directly invalidate the claim that rats might be capable of causal reasoning. To return to the human scenario described in Section 1.2. (Clayton & Dickinson, 2006), the fact that a person cannot both turn on a sprinkler and remove their washing at the same time, does not mean that they are unaware that there is no need to bring in their washing (because the sprinkler is wetting the windows and not rain). Thus, response competition may be obscuring any behaviour consistent with causal model theory.

The analysis presented by Blaisdell *et al.* (2006), and by causal model theory more generally, suggests that the pattern of results that they observed should not be restricted to cases where the alternative cause is a response that is made by the rats. Returning to the example considered earlier, whether a person turns the sprinkler on themselves or merely observes its operation, the knowledge that there is a cause for wet windows other than rain should be sufficient to determine their behaviour. Leising *et al.* (2008) found that presenting a novel external alternative cue (C – a tone) prior to the target stimulus (B) during test failed to modulate performance to B. On the basis of these results, they argued that novel interventions (i.e., lever pressing) might have a special significance in understanding causal relations, and might quickly become established as alternative causes and modulate performance accordingly, while external cues would not automatically have such special significance.

They did, however, allow the possibility that external stimuli might well modulate responding if rats received training with them as an alternative cause of the target stimulus. Following on from previous analyses of causal reasoning (Waldmann, Cheng, Hagmayer & Blaisdell, 2008; Woodward, 2003), they stated that: "*Arbitrary nonaction events can be established as independent, deterministic causes if the strong contingency and the independence of the cause with the effect becomes apparent, such as through learning*" (Leising *et al.*, 2008, p. 516). This idea, that an external stimulus that is established as a cause of some target event through learning might play the role of an alternative cause in a reasoning model, is interesting for two reasons: first, it represents an untested prediction regarding the ability of rats to engage in causal reasoning; and second, in comparison to pressing a lever, an external stimulus does not require an animal to be in a particular place and it should produce comparatively little response competition.

The primary aim of the experiments in Chapter 2 is to reduce any effect that response competition might have in the experimental design used by Blaisdell *et al.* (2006) where the critical contrast is between rats given one of two training conditions: common-cause (B $\leftarrow$ A $\rightarrow$ F) or causal-chain (B $\rightarrow$ A $\rightarrow$ F). The attempt to reduce response competition will be approached in three ways: 1. In Experiments 1 and 3, a CS will be trained as an alternative cause, rather than a lever press, such that the rats do not have to interact with the alternative cause to produce the critical stimulus, B. 2. In Experiments 1 and 2, once the rats had pressed the lever, it was withdrawn, so that they could not continue pressing the lever during the critical auditory stimulus. In Experiment 2, the rats received training with an alternative cause (e.g., lever pressing followed by the auditory stimulus) which might reduce the tendency for lever pressing to interfere with magazine activity while enhancing its potential to serve as an alternative cause of the auditory stimulus.

2.2. Experiment 1

2.2.1. Introduction

In Experiment 1, I examined whether an environmental stimulus that had been trained as an alternative observed cause (cf. Leising *et al.*, 2008) would affect performance to the test stimulus (i.e., B) in the way that lever pressing (LP) does, as predicted by causal model theory. The design of Experiment 1 is depicted in Figure 2.1, and incorporates the critical conditions employed by Blaisdell *et al.* (2006). Thus, rats received either common-cause training (i.e., A→B, A→food; e.g., tone→clicker, tone→food) or causal-chain training (i.e., B→A, A→food; e.g., clicker→tone, tone→food); and both forms of training were supplemented by additional training trials intended to create an alternative observed cause (i.e., C→B; e.g., light→clicker). All rats then received three types of test trials: LP→B, C→B, and B alone. The LP→B trials test the prediction that when the actions of an animal provide an alternative cause for the presentation of B, then this will reduce the tendency of B to elicit magazine activity in the common-cause condition, but not in the causal-chain condition. Unlike in previous experiments of this type, each time the lever was pressed, it was withdrawn from the chamber as B was presented. This arrangement means that the lever would not be present at the same time as the tendency for the rats to approach the food magazine was being assessed (which will hopefully reduce the potential for responding on the lever to interfere with magazine-directed behaviour). The C→B trials test whether an environmental stimulus, previously trained as a signal for B, would act as an alternative cause in the manner predicted by causal model theory. That is, there should be less magazine approach during B when it followed C in rats given common-cause training than in those given causal-chain training. The B alone trials assess the baseline levels of responding elicited by B.

2.2.2. Method

Subjects. All experimental procedures in this thesis were conducted in accordance with the UK Animals Scientific Procedures Act 1986. Thirty-two experimentally-naïve male hooded Lister rats (*Rattus norvegicus*) were maintained at 85% to 90% of their free feeding weights (range: 304g – 339g, mean: 320g), by restricting the amount of food that they received in their home cages. The rats (obtained from Harlan, Bicester, UK) were housed in pairs in a room that was illuminated between 0800 and 2000, where they had unrestricted access to water.

Apparatus. Eight standard operant chambers (Med Associates Inc., St Albans, VT, USA) were used (L × W × H = 30 × 25 × 20cm). Each chamber consisted of two aluminium walls and two clear Perspex walls, with a clear Perspex ceiling and a floor constructed from 0.5cm diameter stainless steel rods, spaced at 1.5cm intervals from centre-to-centre. Each enclosure contained a ventilation fan, and this provided a constant background noise. The chamber was dimly lit by a 28-V, 100mA shielded house light mounted 2cm from the ceiling on one aluminium wall. Adjacent to the house light was a speaker (mounted outside of the chamber) that could deliver a 3000Hz tone and a train of clicks (both at and intensity of 80dB). These auditory stimuli served as the stimuli, A and B. Dwyer *et al.* (2009) used these stimuli in their Experiment 2, and stimuli from different dimensions in their Experiment 1 (cf. Blaisdell *et al.*, 2006). Although the patterns of results were quite different in the two papers, in neither case did the nature of the stimuli interact with the results that were observed. At the centre of the opposite wall (also aluminium) a food well was positioned close to the floor of the chamber. An infrared photo-detector, positioned across the entrance to the food well, was interrogated every 0.01s. Each time this interrogation revealed that the photo-detector had

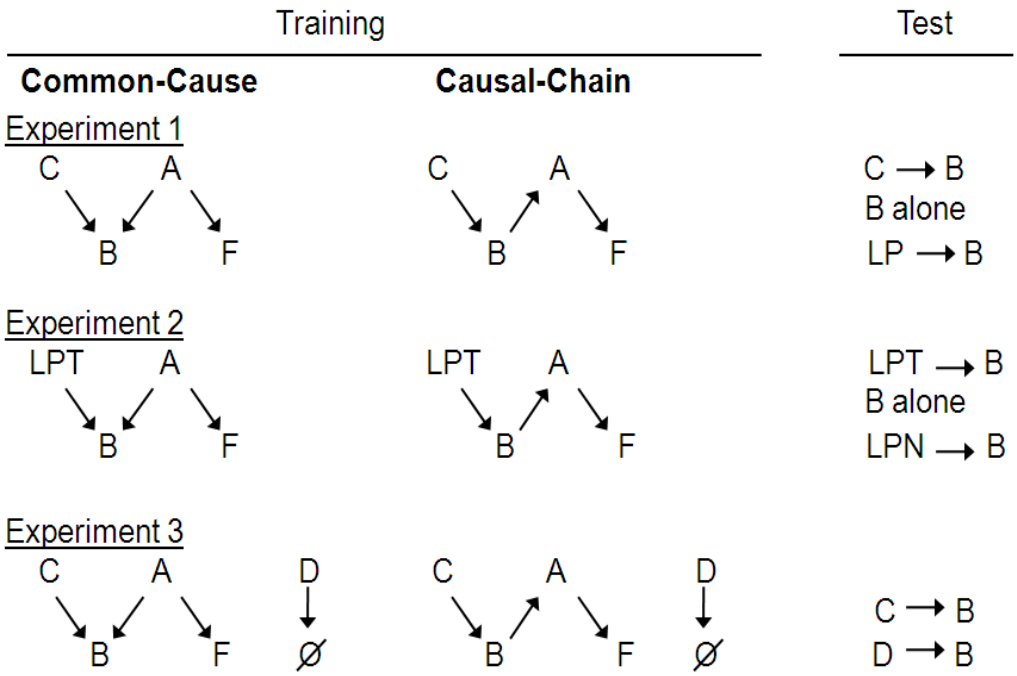


Figure 2.1. Schematics for the experimental designs. A and B were auditory stimuli (tone and buzzer), C and D were visual stimuli (left and right light), F is a food pellet, LP (in Experiment 1) is a lever press, LPN is the novel lever, LPT is a lever that has been paired with B, and ∅ refers to the absence of a programmed event.

been interrupted (upon entry of the rat to the food well) a nosepoke was recorded; and the next occasion on which a nosepoke could be recorded was once the detector had returned to its uninterrupted state and was interrupted again. A retractable lever, located 4cm to the right of the food well and 6cm above the floor of the chamber, was used; and a localized light mounted on the wall directly above the right lever served as the alternative cause (C). The chambers were controlled, and the data recorded, by a PC running MED-PC software (Med Associates Inc.).

Procedure. On the first day of training, food pellets were delivered to the food well on a VT-60s schedule (range: 30 – 90s). On the next four days, rats received training designed to establish parts of the causal models depicted in Figure 2.1. On each of days 2 and 3, rats received pairings of stimulus A (clicker or tone; counterbalanced within group) with stimulus B (tone or clicker; as determined by the identity of A). In the common-cause group, stimulus A immediately preceded B (i.e., A→B), whereas in the causal-chain group, stimulus B immediately preceded A (i.e., B→A). Trials on which stimulus C (i.e., the right light) was paired with stimulus B (i.e., C→B) were intermixed with the A→B trials (for the common-cause group) or B→A trials (for the causal-chain group). Each session consisted of four C→B trials and eight A→B trials or B→A trials. On each of days 4 and 5, rats received 8 trials on which A was immediately followed by the delivery of a food pellet (i.e., A→food). The stimuli (A, B and C) were each 10s; and the mean ITI (offset to onset) was 190s (range: 100 – 280s).

On day 6, rats received a single session in which there were three types of test trial: LP→B, C→B, and B alone. There were four presentations of each trial type, which occurred in a random order (with the constraint that there was one trial of each type in every block of three trials), and according to the same schedule as during training (i.e., mean =190s; range:

100 – 280s). In this case, the interval was between the offset of B, and the onset of the next trial. The results were pooled across test trials on this single test day for the purpose of statistical analysis to reduce trial-by-trial variability in responding (cf. Kutlu & Schmajuk, 2012). On LP→B trials, once the designated interval had elapsed, the lever was inserted into the chamber and remained extended until pressed. At this point, B was presented and the lever was withdrawn, to prevent further lever presses occurring during B and reduce the competition with magazine responding (cf. Dwyer *et al.*, 2009). C→B trials were the same as during training. B alone trials consisted of the presentation of B in the absence of any other programmed events. The duration of B on all trial types was 10s. The primary behavioural measure was the mean numbers of nosepokes per trial in the presence of A (during training) and B (during testing). The pattern of results observed using this measure did not differ appreciably from those seen when the duration of nosepokes was used as the measure of performance. The mean number of nosepokes per trial during the 10s periods without any programmed stimuli that preceded each trial during training and testing was also recorded.

2.2.3. Results

The mean numbers of nosepoke responses per trial during the two A→food sessions are shown in Table 2.1. Inspection of this table reveals that responding was similar in the common-cause and causal-chain groups, and that in both groups the mean levels of responding during the 10s periods that immediately preceded A were lower than during A. ANOVA confirmed that there was no effect of group, $F < 1$, there was an effect of period (i.e., before A versus during A; $F(1, 30) = 43.23$, MSE = 39.37, $p < .001$), and there was no interaction between these factors, $F < 1$. Table 2.1 also shows the overall levels of nosepoking during the 10s periods that preceded the test trials. Statistical analysis confirms, what inspection of this table suggests, that these rates were similar in the two groups, $t < 1$.

Table 2.1: Experiments 1, 2 and 3: Mean number of nosepokes per trial during training with A (both in the 10s before and 10s during A); and background levels of nosepoking during the tests that were conducted in extinction (these are reported as responses per 10s period).

Experiment 1			
Training			**Test**
Group	Pre-A	During-A	Pre-CS
Chain	10.9	20.1	2.0
Common	11.3	22.2	2.1
Experiment 2			
Training			**Test**
Group	Pre-A	During-A	Pre-CS
Chain	14.5	22.4	2.7
Common	14.6	20.6	2.7
Experiment 3			
Training			**Test**
Group	Pre-A	During-A	Pre-CS
Chain	9.4	14.2	1.5
Common	10.8	19.8	1.8

The critical results from Experiment 1, the levels of nokepoking during B, minus pre-trial responding, are shown in Figure 2.2. First consider the influence of the lever press on responding during stimulus B. Examination of Figure 2.2 suggests that, irrespective of whether the rats had received common-cause or causal-chain training, the level of responding during B was greater when it was presented alone than when it immediately followed a lever press. Now consider the consequences of providing an alternative trained cause (C) for stimulus B. Examination of Figure 2.2 reveals a tendency for the level of responding during B to be greater when it was presented alone than when it immediately followed C. However, this tendency was, if anything, more evident in the causal-chain group than the common-cause group. This pattern of results is inconsistent with the predictions of causal model theory.

The results were subjected to ANOVA with a between-subjects factor of training group (common-cause vs. causal-chain) and a within-subjects factor of test trial type (LP→B, C→B, and B alone). This confirmed that there was no main effect of training type, $F < 1$, but that there was an effect of trial type, $F(2, 60) = 5.95$, MSE = 16.46, $p = .004$, that did not interact with training group, $F < 1$. Analysis of simple main effects showed that responding on B alone trials was greater than on LP→B trials, $F(1, 30) = 10.64$, MSE = 36.83, $p = .003$; but there was no difference in responding either between the B alone trials and the C→B trials, $F(1, 30) = 2.80$, MSE = 33.72, $p = .104$, or between the C→B trials and the LP→B trials, $F(1, 30) = 3.59$, MSE = 28.22, $p = .068$.[2]

[2] There were never any significant effects in the post-CS period (cf. Blaisdell *et al.* 2006), indeed, responding in this period was typically no higher than in the absence of any stimuli. Thus these data will not be considered further here.

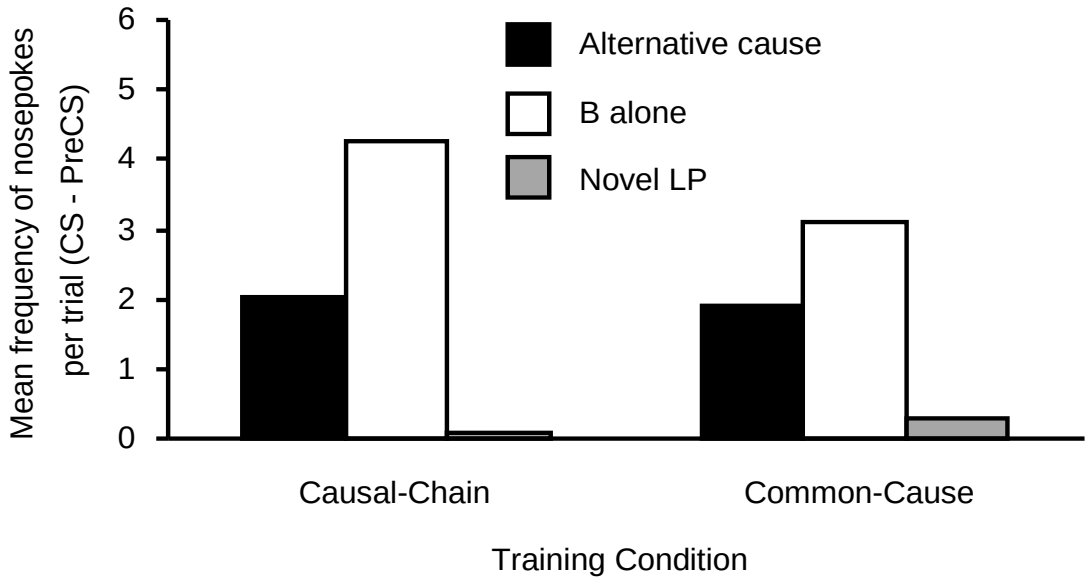


Figure 2.2. Experiment 1. Mean number of nosepokes per trial (minus pre-CS nosepokes) during the test with stimulus B as a function of training (common-cause or causal-chain) and test trial (C→B, alternative cause; B alone; and LP→B, novel LP)

2.2.4. Discussion

The effects of presenting an external stimulus as a trained alternative cause of B are difficult to assess conclusively because responding on the C→B trials lay between the levels of responding on LP→B and B alone trials without being significantly different from either of them. That said, there was no suggestion that any trend towards lower responding on C→B trials was stronger in the common-cause group as would be predicted by causal model theory. Indeed, any tendency for the light to interfere with magazine activity to stimulus B could also reflect response competition. For example, it seems entirely plausible to suppose that the light elicits an orienting response, and some vestige of this response interferes with magazine activity during the presentation of B (cf. Dwyer, *et al.*, 2009). This issue will be returned to, and a better control will be provided in Experiment 3.

The fact that the lever press intervention manipulation had an equivalent effect on magazine responding during B in the two training conditions is inconsistent with the predictions of causal model theory, and with the results reported by Blaisdell *et al.* (2006). However, these results are consistent with those reported by Dwyer *et al.* (2009). Although the lever was not physically present during the critical stimuli, it is certainly possible that both its initial presentation and subsequent removal would elicit a strong orienting response. In addition, as has been have noted before (see Dwyer *et al.*, 2009), a single lever press is merely the discrete endpoint of approach to, and contact with, the lever and so the ability of a lever press to interfere with other responses would extend beyond the time of the lever press itself. Thus, withdrawing the lever did not appear to eliminate response competition. Other manipulations of the lever will be considered in Experiment 2.

2.3. Experiment 2

2.3.1. Introduction

In Experiment 1, withdrawing the lever from the chamber as B was presented appeared to be insufficient, on its own, to eliminate response competition. One possible reason for this it that both the lever, and its removal upon being pressed, are novel and thus might elicit a large amount of orienting and other unconditioned behaviours. An obvious way to reduce such unconditioned behaviours is to simply familiarise the rats with the lever prior to test. However, presenting the lever without consequence could also reduce its tendency to be considered an alternative cause. Therefore, in Experiment 2, stimulus B was presented after each lever press. This should help to establish lever pressing as an independent alternative cause of B, while reducing its tendency to provoke competing behaviours based on its novelty.

Figure 2.1 shows the full design adopted in Experiment 2. During the first stage of training, rats either received common-cause training (i.e., A→B, A→food) or causal-chain training (i.e., B→A, A→food). Both forms of training were supplemented by additional trials in which a lever was inserted into the box. When the lever was pressed, it was retracted, and stimulus B was presented for 10s. At test, responding to B following either a press on the trained lever (LPT), or on a novel lever (LPN), was compared to responding on trials where B appeared alone. According to the causal analysis LPT should only influence responding in the common cause condition; and this influence should be at least as marked as for the untrained lever, LPN, which had not been trained as an independent cause. However, according to an analysis based upon competing responses alone, LPT should not interfere with responding to B; while LPN will do so to the extent that it is treated as entirely different to LPT.

2.3.2. Method

Subjects and Apparatus. Experiment 2 was conducted with two replications, each consisting of 32 experimentally-naïve male hooded Lister rats that were sourced and maintained in the same way as previously described. The mean free-feeding weight was 246g (range: 222-320g) for the first replication, while for the second replication the mean was 340g (range: 274-397g). Rats were trained in the same operant chambers as previously described.

Procedure. Magazine training was conducted in the same way as described above. On each of the next 2 days, rats were presented with four presentations of the lever and 8 pairings of A and B. The common-cause group were given presentations of A that were immediately followed by B, whereas the causal-chain group were given B immediately followed by A. On lever presentation trials, the lever was inserted into the box and remained extended until it was pressed, whereupon it was withdrawn and stimulus B was presented. The trained lever was counterbalanced within groups, with half of the rats in each group receiving the lever to the left of the magazine, and the remainder receiving the lever to the right of it. A and B were counterbalanced in the same manner as previously and were both 10s long. On each of training days 4 and 5, rats received 8 trials where stimulus A was followed by the delivery of a food pellet. The mean ITI during training (offset to onset of trials) was 190s, and ranged from 100 – 280s. On day 6, rats received one test session with the trial types shown in Figure 2.1 (LPT→B, LPN→B, and B alone). Trials were presented randomly (with the constraint that one trial of each type occurred in every 3-trial block), four times each, at an ITI matched to that of the training. On lever trials, the lever was inserted into the box and a lever press resulted in its retraction and the presentation of B. B alone trials consisted of the presentation of B in the absence of a lever or other programmed external stimulus.

2.3.3. Results and Discussion

The data from the two replications were combined for presentational purposes; but it should be noted that the critical statistical effects was the same in both replications when analysed separately. Training sessions two and three required animals to press a lever, without receiving a reinforcer for doing so. Under these conditions, it was unsurprising that some rats failed to complete these sessions, and thus were removed from the analysis. This left 28 rats in the causal-chain group and 25 in the common-cause. Table 2.1 shows responding during the critical stimuli in the final stage of training (A→F). Both groups nosepoked more frequently during A than in the pre-CS period. ANOVA confirmed that there was an effect of time period, $F(1, 51) = 48.17$, MSE = 38.18, $p < .001$, no effect of group ($F < 1$), but an interaction between these factors, $F(1, 51) = 4.04$, MSE = 38.18, $p = .114$, indicating a higher rate of responding to A in the causal chain group. Table 2.1 also presents mean pre-trial responding in the test phase, where again there was no difference between the groups, $t < 1$.

Figure 2.3 presents the frequency of nosepoke responding during the critical stimuli at test, minus the pre-trial responses. Inspection of this figure suggests that rats in the common-cause group responded at higher rates than those in the causal-chain group, but that in neither training group was there a tendency for responses to be lower on trials where B occurred after a lever press than on trials where B was presented alone. ANOVA revealed a significant effect of training group, $F(1, 51) = 5.60$, MSE = 6.44, $p = .022$, no effect of test trial type and no interaction between these factors, Fs < 1. The effect of group is inconsistent with that observed during training, and the basis for the difference is not immediately clear. An

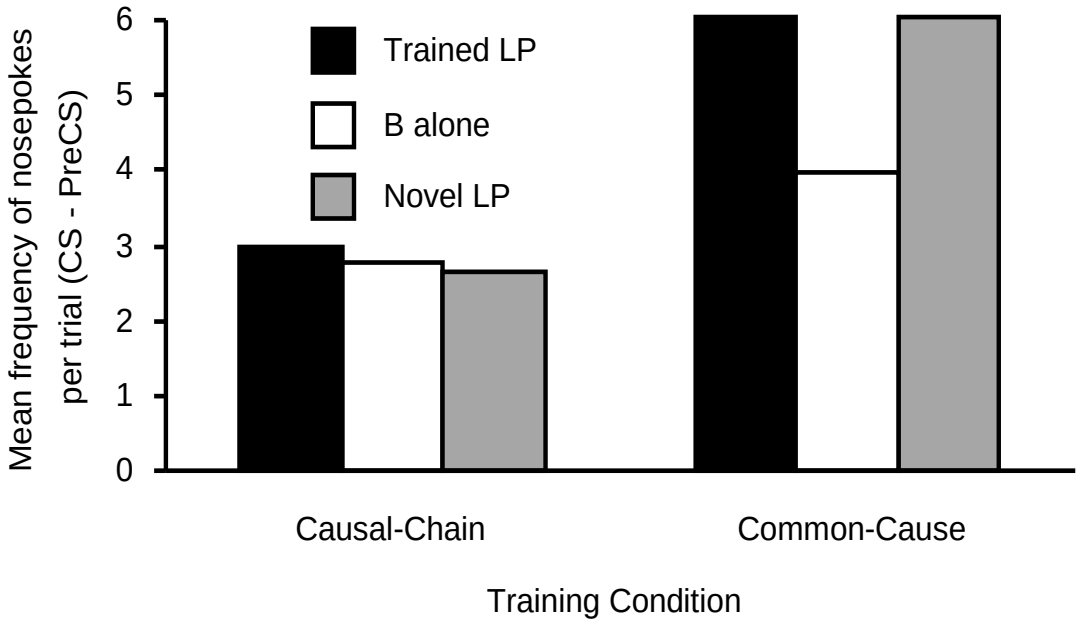


Figure 2.3. Experiment 2. Mean number of nosepokes per trial (minus pre-CS nosepokes) during the test with stimulus B as a function of training (common-cause or causal-chain) and test trial (LPT → B, C → B, LPN → B).

analysis including replication as a factor revealed no effect of replication, and no interactions involving it (*F*s < 1).

The 95% confidence intervals for the B minus pre-trial response scores did not include zero in any of the conditions in either group. That is, in all test conditions rats were approaching the magazine during B at a higher level than they were during the pre-trial period. Therefore, the absence of any effects of trial type does not reflect a general failure to respond. In contrast to both Experiment 1 and Dwyer *et al.* (2009), responding to B was not significantly reduced when it followed a lever press (either when the lever was novel or trained). Thus, exposure to a lever prior to test appears only to have attenuated response competition in a nonspecific way, which presumably reflects a process of generalization between the two levers or the responses that they prompt. Most importantly, even though rats had received training whereby a lever press resulted in the presentation of stimulus B, which should enhance its tendency to be treated as an alternative cause of B, lever pressing failed to modulate responding to B as predicted by the causal account.

2.4. Experiment 3

2.4.1. Introduction

As noted in the discussion of Experiment 1, the design of that experiment meant that the effect of an alternative trained cause was compared to a condition where no external stimulus was presented before responding to the critical test stimulus was assessed. A more adequate assessment of the effect of an alternative trained cause (C), and one that controls for the general consequences of stimulus presentation (e.g., orienting responses), should involve a comparison with an equivalent stimulus that has not been trained as an alternative cause (D). Under such circumstances, one might be more sensitive to any genuine effect that pairing C with B has on subsequent responding to B. The experimental design adopted in Experiment 3 is depicted in Figure 2.1. During the first stage of training, rats either received

common-cause training (i.e., A→B, A→food) or causal-chain training (i.e., B→A, A→food). Both forms of training were supplemented by additional training trials intended to create an alternative observed cause (i.e., C→B) and a control stimulus (D alone) that was not an alternative cause. All rats then received two types of test trials: C→B and D→B. The questions of interest were whether C and D would have a different effect on responding to B during the test, and whether any such effect interacts with the nature of training (common-cause or causal-chain).

2.4.2. Method

Subjects and Apparatus. Experiment 3 was conducted as two replications each using 32 experimentally-naïve male hooded Lister rats. The rats were obtained from the same source and maintained in the same way as those described in Experiment 1. The mean free feeding weight of the first replication was 321g, with a range of 305-344g, and in the second replication, a mean of 316g and a range of 290-336g. Rats were trained in the same operant chambers as Experiment 1, with the exception that the light above the left lever (that was in other respects equivalent to that above the right lever) was also employed.

Procedure. On the first day of training, rats were trained to collect food pellets from the food well in the same way as Experiment 1. On days 2 and 3, they received three types of trial in a pseudo-random sequence. Group common-cause received the following trials types: A→B (8 trials per session), C→B (4 trials per session) and D alone (4 trials per session); and group causal-chain received the same set and number of trials with the exception that the 8 A→B trials were replaced with 8 B→A trials. For both groups, the identity of the auditory stimulus (tone or clicker) that served as A or B was counterbalanced, and the identity of the visual stimulus (left or right light) that served as C or D was also counterbalanced in the subgroups created by the previous counterbalancing operation. On days 4 and 5, rats received presentations of A that were followed immediately by the delivery of food. On day 6, rats

received four presentations of two types of trial: C→B and D→B. These trials were presented in a pseudo-random sequence. The mean ITI during training and testing was 190s (range: 100-280s).

2.4.3. Results and Discussion.

The data from both replications were analysed together for presentational purposes. Table 2.1 shows the training data during the A→food days. It is apparent that responding during A is higher than during the 10s period before A for both groups. This was supported by an ANOVA, showing a main effect of time period, $F(1, 62) = 45.16$, MSE = 37.81, $p < .001$, but no effect of group, and no interaction between these factors, $Fs < 1$. Table 2.1 also shows the overall levels of nosepoking during the 10s periods that preceded CS presentation on test, which did not differ between groups, $t(62) = 1.11$, $p = .271$.

Figure 2.4 displays the mean nosepoking frequencies (less pre-CS responding) during stimulus B on trials on which it was preceded by the alternative cause (C) and the control stimulus (D) as a factor of training group (common-cause vs. causal-chain). In contrast to the predictions of causal model theory, responding to B was higher following the alternative cause (C) than the unpaired control stimulus (D) in the common-cause group, with this trend reversed in the causal-chain group. ANOVA confirmed that there were no main effects of the type of stimulus preceding B or training group, $Fs < 1$. There was an interaction between these two factors, $F(1, 62)=6.06$, MSE=7.45, $p=.017$. Responding to B, as a function of the preceding stimulus type (C or D), differed significantly in the common-cause group, $F(1, 31) = 4.65$, MSE = 8.06, $p = .039$, but not in the causal-chain group, $F(1, 31) = 1.67$, MSE = 6.84, $p = .207$. A combined analysis of the two experiments that included replication as a factor revealed no effect of this factor or interactions involving it ($Fs < 1$).

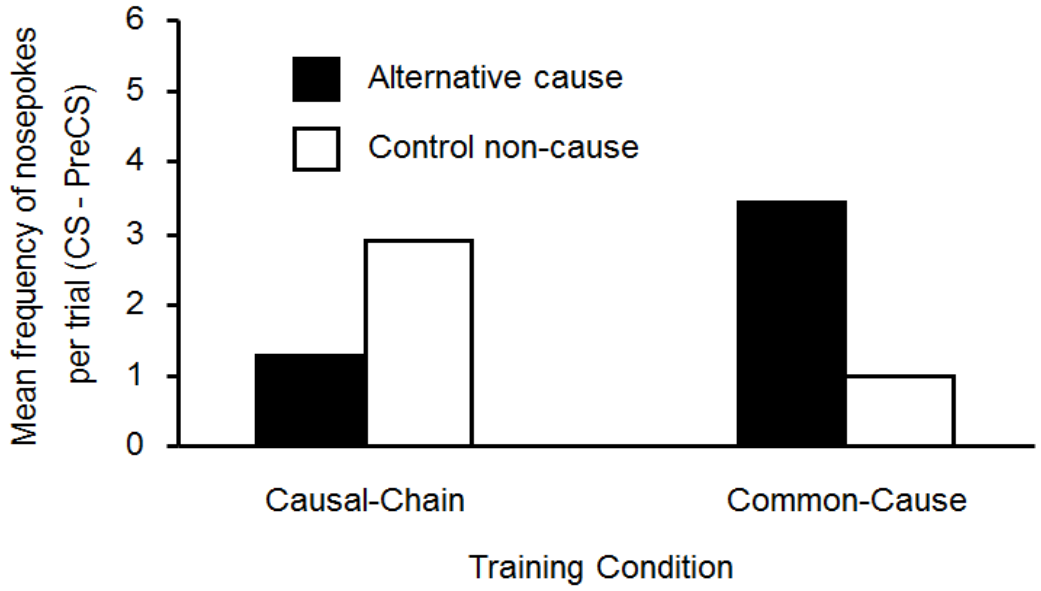


Figure 2.4. Experiment 3. Mean number of nosepokes per trial (minus pre-CS nosepokes) during the test with stimulus B as a function of training (common-cause or causal-chain) and test trial (C→B and D→B).

The results observed here were unexpected on the basis of causal model theory, indeed, they were the exact reverse of prediction derived from it. Possible explanations for these intriguing results are considered in the General Discussion (Section 2.5). For now it is sufficient to note that they provide no support for the idea that rats create causal models from the training that they received in Experiment 3; and this cannot be due to a failure of the rats to learn about C and D during training because these stimuli did modulate responding to B.

2.5. General Discussion

Blaisdell *et al.* (2006) reported that when rats are given training where one cue (A) separately predicts two outcomes (i.e., A→B and A→food; common-cause training) they approached the magazine when B was presented during a subsequent test. Moreover, responding to B was modulated by the manner in which it was presented: it was lower when B occurred after a lever press performed by the rat than when B occurred alone. This modulation of responding to B was not seen in various control conditions (e.g., when initial training established a chain of associations: B→A and A→food). These results were interpreted as providing support for the idea that the rats might have encoded a simple causal model of the training situation, wherein A was the common-cause of two separate events (B and food). Possession of this model would normally licence the inference that B had been generated by its usual cause (A), and that A would thus have also caused food to be delivered. This inference would not hold if there was an alternative cause for B (in this case the lever press). The modulation of responding to B, as a function of whether it was produced by a rat's own actions or not, was taken as evidence that magazine behaviour was under the control of causal inference mechanisms (and in particular, the mechanisms described by causal model theory).

Although this pattern of results has been replicated by the laboratory in which it was first reported (Leising *et al.*, 2008), direct replications of their procedures in the Cardiff

University laboratory have failed to reveal the original pattern of results (Dwyer *et al.*, 2009). Instead, Dwyer *et al.* (2009) consistently found that irrespective of the training that rats received (common-cause, causal chain or direct conditioning) responding to B was lower when it followed a lever press. Moreover, Dwyer *et al.* (2009) also observed (across experimental conditions) a reciprocal relationship between nosepoking to B and lever pressing – a relationship that suggests interference between these two forms of response. Thus, these previous results suggest that it is not necessary to invoke causal reasoning mechanisms to explain the rats' behaviour – a simpler account in terms of the competition between responses is also possible (see Morgan, 1894). Unfortunately, neither Blaisdell *et al.* (2006) nor Leising *et al.* (2008) reported the levels of lever pressing during the critical stimulus B (in all experimental conditions), thereby preventing an assessment of the potential role of response competition in their data. Thus, while considerations of parsimony support adopting the simpler explanation of the overall pattern of results, it remained possible that the response competition in our previous experiments had simply obscured any behaviour that was under the control of causal reasoning mechanisms. Therefore, the current series of experiments was performed to examine whether evidence could be found that was consistent with causal reasoning in rats in situations that might expected to reduce response competition.

Experiment 1 examined the effect of presenting an external stimulus that had been trained as an independent predictor or cause of the critical test stimulus, B, and by removing the lever from the chamber once it had been pressed. The response to B was lower when it followed the lever press than when it was presented alone, but again this effect was observed irrespective of the training that the rats had received (common-cause or causal-chain). Responding to B was not significantly affected by the presentation of a trained external cause, C, although there was at least some suggestion of a decrement in responding. Experiment 2 demonstrated that training the rats with the lever as an independent cause of B,

prior to test, abolished the influence of lever pressing on nosepoking during B. Taken together, Experiments 1 and 2 suggest that responding to B was only affected by lever pressing when the rats experienced levers for the first time in the test session. Experiment 3 examined nosepoking to B as a function of whether it followed an external cue, C, that had previously predicted B, or a control external stimulus, D, that had not. Responding to B was modulated by whether it followed C or D, and this interacted with training condition. However, the form of this interaction was the opposite to that predicted by causal model theory: in rats given common-cause training responding to B was *higher* when it followed a trained alternative cause, C, than the control stimulus, D, and this effect was not seen in the causal-chain control condition. The implications of these results will be discussed further in the General Discussion (Chapter 5).

Chapter 3: Causal binding in rats: Timing from an action or external event

3.1. Introduction

The results in Chapter 2 suggest that rats are unable to form and use causal models in a manner consistent with causal model theory. One possible explanation of this finding is that rats do not represent cause and effect relations in the same manner as humans. However, it is also possible that rats can represent cause and effect, but do not use that knowledge to reason in the same manner as humans. As discussed in Section 1.3, in humans, actions are more likely to be imbued with a causal status than are external stimuli, and it has been argued that this difference results in actions and outcomes appearing to share greater temporal contiguity than external stimuli and outcomes. In the context of animal cognition, it has also been argued that actions and external stimuli have a different causal status (Leising, *et al.*, 2008; but see, Burgess, Dwyer, & Honey, 2012). However, there have been no attempts to examine whether the perception of time in animals is influenced by whether the referent is a response or an external stimulus. This is a theoretically interesting issue, not least because according to theoretical analyses of timing behaviour, actions and external stimuli are merely treated as markers for the beginning of the interval. For instance, two prominent models of timing, Scalar Expectancy Theory (SET, Gibbon, 1977) and Learning to Time Theory (LeT, Machado, 1997) state that timing behaviour results from a series of processes that occur after a time marker which are generated irrespective of whether this marker is an instrumental response or an observed stimulus (for a review, see Gallistel & Gibbon, 2000).

In three experiments, the timing behaviour of rats was examined using a peak procedure in which either an action or an external stimulus served as the temporal referent. In this procedure, a referent was presented and then after *t* s food was delivered. The question of primary interest here was whether timing behaviour, as evident by a peak in responding at or

around *t* s after the referent, was influenced by whether the referent was an action or an external stimulus. An effect analogous to causal binding would be evident if the peak occurred closer in time to the action (e.g., *t* s – *n* s) than to the external stimulus (e.g., *t* s).

3.2. Experiments 4-6

3.2.1. Introduction

Experiments 4 and 5 used a within-subjects procedure to assess timing from an action (a lever press, LP) and an external stimulus (an auditory conditioned stimulus, CS). The interval between these two separately presented referents and the delivery of food was 5s. During the course of training and testing, nonreinforced trials were included in which the action and stimulus occurred, but no food was presented. These trials allow the accuracy of timing to be assessed in the absence of the effects of the presentation of food on behaviour. The design of Experiment 6 allowed a replication of the results observed in Experiments 4 and 5 under conditions in which one could assess whether instrumental performance was outcome or goal directed. On the one hand, if the rats are representing the outcome as the product of their actions (i.e., lever pressing is goal directed), then there are grounds for anticipating that timing should be influenced by a process of causal binding. On the other hand, if the rats are not representing the outcome in such a fashion (i.e., behaviour is habitual), then evidence of causal binding would not be expected.

To assess timing behaviour under conditions in which such goal directedness could be determined, rats were initially presented with two levers that were each paired with a different outcome (e.g., left lever→food and right lever→sucrose); and two CSs that were also paired with different outcomes (e.g., tone→food and buzz→sucrose). Once the timing behaviour in the two conditions (LP and CS) had been established, the rats were sated on one of the outcomes (e.g., food) and then given a test in which the two responses were made available in an extinction test. If instrumental responding was related to the outcomes with

which the different responses were paired, then rats should be less likely to perform the response that was paired with the now devalued outcome (left lever) than the response that was paired with the outcome that retains its value (right lever; e.g., Adams & Dickinson, 1981; Dickinson, 1985).

3.2.2. Method

Subjects. Each experiment used experimentally naïve male hooded Lister rats (*Rattus norvegicus*) obtained from Harlan, Bicester, UK. They were maintained between 85-90% of their free-feeding weights (Experiment 4, *N* = 12; mean = 346g, range: 330-360g; Experiment 5, *N* = 16; mean = 399g, range: 350g-428g; Experiment 6, N = *16*: mean = 356g, range: 328-385g) by feeding them a small quantity of food at the end of each day. Rats were housed in pairs in a room illuminated between the hours of 0800 and 2000.

Apparatus. The same operant chambers were used as those described in Chapter 2. The CSs were a 2s train of clicks (10 Hz: Experiment 4), a 0.5s tone (3000 Hz; Experiment 5), and a 0.8s buzz (100 Hz) or tone (3000 Hz, Experiment 6), and all CSs were presented at an intensity of 80dB. At the centre of the opposite wall (also aluminium), there was a food well positioned close to the floor of the chamber. This delivered food pellets and sucrose solution. An infrared photo-detector, positioned across the entrance to the food well, was interrogated every 0.01s. Each time this interrogation revealed that the photo-detector had been interrupted (upon entry of the rat to the food well) a nosepoke was recorded, along with its duration; and the next occasion on which a nosepoke could be recorded was once the detector had returned to it uninterrupted state and was interrupted again. The chambers were equipped with two 2cm long retractable levers, located 4cm to the right/left of the food well and 6cm above the floor of the chamber. The left lever was used in Experiments 4 and 5, and both levers were used in Experiment 6. The chambers were controlled and the data recorded by a PC running MED-PC software (Med Associates Inc.). The pre-feed boxes used for the

devaluation stage in Experiment 6 consisted of 32 × 15 × 12cm (L × W × H) white acrylic walls and floor, with a stainless steel wire lid. Food pellets were provided in small plastic trays; and 20% sucrose was made accessible through drinking spouts made of stainless steel, attached to 50ml cylinders. The amount consumed was assessed by weighing the bottles and trays before and after the pre-feed.

Procedure. On the first day of Experiments 4 and 5, rats received a pretraining session in which there were 20 trials on which the left lever was inserted into the chamber until it was pressed at which point it was retracted and a sucrose pellet was delivered; and 20 trials on which the offset of a 2s train of clicks (in Experiment 4), or the offset of a 0.5s tone (in Experiment 5), was paired with sucrose pellet. There was an interval of 60 – 80s (mean 70s) from the offset of one stimulus (CS or LP) and the onset of the next; and the order of trials was random with the constraint that there were no more than 2 trials of the same type in succession. In Experiment 6, rats were given the same form of pretraining with both levers (left and right), and two 0.8s auditory CSs (tone and buzz). The length of the CSs was increased from that in Experiment 5 to ensure that the rats could distinguish between them. Pressing one lever and the presentation of one CS was paired with the delivery food, and pressing the remaining lever and the presentation of the other CS were paired with 0.02ml of 20% sucrose solution. The design was fully counterbalanced. The session consisted of 40 trials, 10 of each trial type. The scheduling of trials types was otherwise the same as in Experiments 4 and 5.

On the next 24 days in Experiment 4 and 29 days in Experiment 5, rats received training trials where the LP and CS were followed by food after an interval of 5 s. There were 18 of each of the two trial types in each session; and an additional 2 nonreinforced LP and CS trials with the distribution of trials arranged in the same way as in the pretraining session. Food well activity was collected in 1s bins. In Experiment 6, rats received 34 days of training

in which the two CSs and LPs continued to be presented 9 times and paired with their designated outcomes after a 5s interval. One additional trial with each CS and LP was presented without reinforcement. The interval between stimuli was matched to that of the previous two experiments.

Test. The final stage in Experiments 4 and 5 involved test sessions that included an additional 4 non-reinforced LP and CS test trials. These test sessions consisted of 14 reinforced LP and CS trials, and 6 non-reinforced LP and CS trials. In Experiment 6 the test sessions consisted of 7 reinforced trials with the two LPs and CSs, and 3 nonreinforced trials with these LPs and CSs. The food-well entries on the non-reinforced trials were used to assess timing accuracy. Other details of these sessions were the same as for the immediately preceding stage of training.

Devaluation (Experiment 6). The within-subjects devaluation procedure involved presenting the rats with 30 minutes access to either food pellets or 20% sucrose solution in the pre-feeding boxes described above over two devaluation days. For half the cohort, food pellets were presented on the first day, and sucrose solution on the next; and for the rest, this order was reversed. After the rats were given access to either food pellets or sucrose solution for 30min, they were transferred to the operant chambers. During the test sessions, rats received separate access to the left lever and the right lever for 30s on 8 separate trials, with a variable ITI of 20 – 60s, and a mean of 40s. The frequency of lever presses during these 30s periods was recorded. Between the first and the second devaluation days, the rats were given another day of training (40 trials, 10 of each type).

Measures of timing. The rate of food well entries during successive 1s bins following the response and the offset of the stimulus were recorded. The location of the peak response rate was assessed using a Gaussian curve fitting procedure (Lejeune & Wearden, 2006). The resulting Gaussian curves were used to determine the accuracy of timing through the width of

the curve (i.e., the variance of the data). At the end of the Gaussian curve, responding should return to baseline levels. This can be fitted with a Gaussian curve and linear ramp function. The formula used is shown below (taken from Buhusi, Sasaki & Meck, 2002) where t is the current time, t_0 is the peak time, b is the estimate of precision of timing (variance), $a + d$ is the estimate of peak rate of response, and c is the slope of the tail.

$$A \times \exp\left(-.5 \times [(t - t_0) / b]^2\right) + c \times (t - t_0) + d$$

For the present purposes, the most critical parameter is t, the peak time of responding, as this represents the best estimate of interval between events. The accuracy of the curve fit (R^2) was taken to ensure the function accurately fits the data, and any rats not revealing a good curve fit were removed from subsequent analyses (where $R^2 < .80$).

3.2.3. Results

Experiment 4. Figure 3.1 displays the mean rates of responding during the 20s period following the LP and CS for the final 18 non-reinforced trials for each condition (i.e., the final 3 sessions of testing). All rats were included in the analysis, as each had a curve fit accuracy (R^2) of greater than .80. Inspection of this figure shows that in both conditions, responding peaks at around 5 – 6s, and then declines to a stable, low level by about 12s. It is also evident that the level of responding is initially higher after the CS than after the LP (see also Experiment 6). As will be seen in Experiment 5, in which a shorter CS was used, this effect appears to reflect the fact that the 2s presentation of the CS is less likely to compete with food well activity than is a lever press. The mean peak responding, along with the R^2 and variance in each condition, are presented in the top panel of Table 3.1. Inspection of this table shows that the peak response time for the CS trials are similar to the LP trials, $t(11) = 1.630$, $p = .131$, SEM = .274; and that the variability of the timing peak is smaller for LP

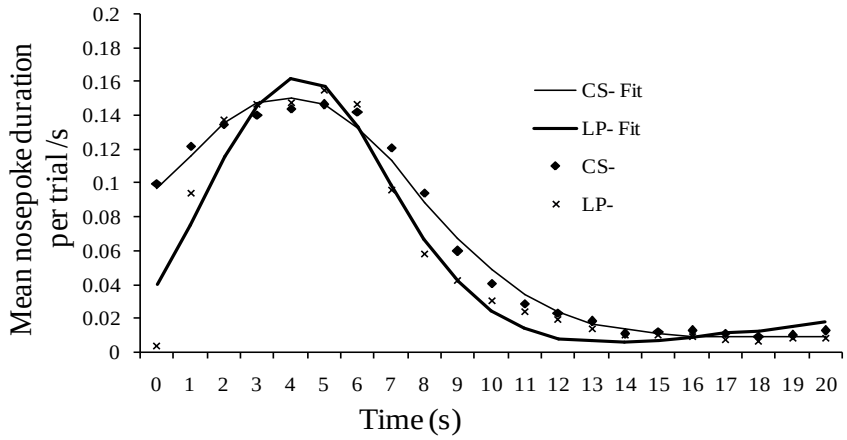


Figure 3.1. Experiment 4. Mean duration (in seconds) of nosepoke responding during the 20s periods that followed the nonreinforced CS and LP trials (presented as 0s on the *x* axis). The fitted curves show the mean curve fits in the CS and LP conditions.

trials than CS trials, $t(11) = 3.188$, $p = .009$, SEM = .312. The final variable is the mean R^2 value for each trial type. Inspection of these values in Table 3.1 indicates that the accuracy of the curve fits is generally better for the CS than the LP trials. This was confirmed using a t-test, $t(11) = 6.145$, $p < .001$, SEM = .008.

Standard null hypothesis tests of the sort performed above do not assess the extent to which the absence of a significant effect is consistent with a genuine absence of any difference in the population being sampled from. Rouder, Speckman, Sun, Morey and Iverson (2009) outline a method for using Bayesian analysis which does allow the evaluation of the degree to which the evidence actually supports the null hypothesis. In short, this analysis method provides a value known as the Bayes factor which is the odds ratio relating the probability of the null hypothesis being true to the probability that the alternative hypothesis is true, given the observed data. To evaluate the relative probability of the null and alternate hypotheses requires that both be specified. Although there are a range of different possibilities for each of these, Rouder et al. (2009) suggest that as a default, the null should be specified as it is in standard null hypothesis testing, and the alternate hypothesis (for a t-test situation) be set as 1 standard deviation between condition means. These default specifications were used for the Bayes analyses reported throughout this thesis. The Bayes factor can be interpreted as providing support for the null hypothesis if its value is greater than 3, the alternative hypothesis if its value is less than 1/3, or insufficient evidence for either hypothesis (if its value is between 1/3 and 3). An online calculator (http://pcl.missouri.edu/bayesfactor) was used for paired designs and the JZS (Jeffrey-Zeller-Siow) prior, because it makes the fewest assumptions regarding the prior distribution (Rouder *et al.*, 2009). The Bayes Factor for the peak time is 1.498, which provides inconclusive

Table 3.1. Test Data From Experiments 4 to 6: Mean Peak Responding (s), Curve Fit Accuracy (R^2), and Variance.

Trial Type	Peak Time (s)	R^2	Variance
Experiment 4			
CS-	4.6 (.201)	0.97 (.007)	4.1 (.203)
LP-	5.2 (.097)	0.92 (.012)	2.8 (.134)
Experiment 5			
CS-	5.3 (.178)	0.93 (.009)	3.49 (.263)
LP-	5.2 (.385)	0.92 (.010)	3.33 (.579)
Experiment 6			
CS-	4.6 (.404)	0.92 (.012)	4.6 (.639)
LP-	5.2 (.299)	0.85 (.019)	3.1 (.313)

evidence for either hypothesis. There is certainly no suggestion that the data is consistent with a causal binding effect where the peak time was shorter for LP trials.

Experiment 5. Figure 3.2 shows the combined data from the final 18 test trials (i.e., 3 sessions of training). Although these test sessions produced generally accurate curve fits, the performance of 5 rats were removed from the analysis because their curve fits revealed an accuracy of less than .80. The pattern of timing was very similar in the LP and CS conditions, with the only notable difference being that the spread of responding around the mean was somewhat smaller in the LP condition. The mean peak of responding, R^2 and variance of the LP and CS trials are presented in the middle panel of Table 3.1. Inspection of these scores reveals that there was little difference between the conditions. This description of the results is supported by the fact that there were no significant differences in peak responding, $t(10) = 0.064$, $p = 0.950$, SEM = .275, variance, $t(10) = 0.209$, $p = .839$, SEM = .538, or R^2, $t(10) = 1.571$, $p = .147$, SEM = .009.

As in Experiment 4, a Bayes analysis was run on the non-significant results, and a Bayes factor for the peak responding time revealed odds of 4.656, and the variance revealed odds of 4.943. This means that, given the observed data, the null hypothesis is over four times more likely than the alternative hypothesis – thus supporting the conclusion that timing from an action or an external cue does not differ with respect to the peak time or variability of responding. However, for R^2 the evidence for accepting either the null or alternate hypothesis is insufficient, with a Bayes factor of 1.282.

Experiment 6. Figure 3.3 depicts the mean rates of nosepoking following the nonreinforced LP and CS trials over the last 5 days of testing, pooled over the two LPs and CSs. This number of test days was necessary to produce more accurate curve fits, as there were fewer trials per condition in the test session compared to Experiments 4 and 5. The scores in Figure 3.3 therefore represent means of 15 non-reinforced trials. Only rats that

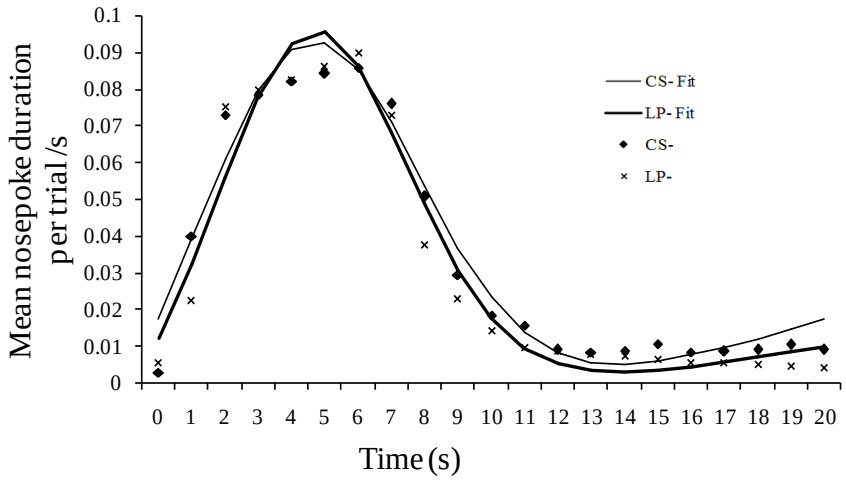


Figure 3.2. Experiment 5. Mean duration (in seconds) of nosepoke responding during the 20s periods that followed the nonreinforced CS and LP trials (presented as 0s on the *x* axis). The fitted curves show the mean curve fits in the CS and LP conditions.

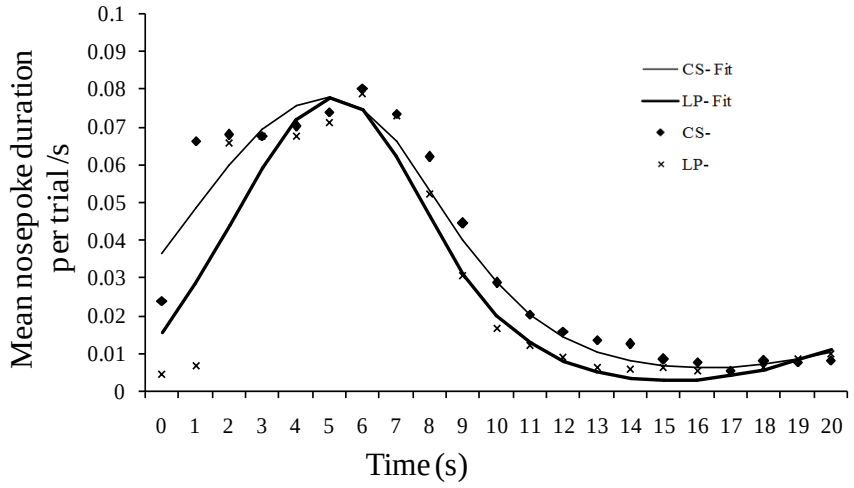


Figure 3.3. Experiment 6. Mean duration (in seconds) of nosepoke responding during the 20s periods that followed the nonreinforced CS and LP trials (presented as 0s on the *x* axis). The fitted curves show the mean curve fits in the CS and LP conditions.

provided an accurate ($R^2 > 0.80$) fit for responding to both CSs and both LPs were included. This led to the removal of eight rats from the analysis. The results shown in Figure 3.3 are more similar to those shown in Figure 3.1 than in Figure 3.2. Specifically, during the initial seconds after the LP and CS, nosepoking was lower on LP than CS trials. The length of the CSs were longer in Experiment 6 than for the CS in Experiment 5, and this might help to explain why the pattern of results was more like those seen in Experiment 4 than Experiment 5. Table 3.1 shows the mean curve fit data. Examination of this table suggests that the peak of responding was slightly longer for the LP than the CS condition, and that the variance and R^2 are smaller for the LP condition than the CS condition. This description of the results shown in the table is supported by *t*-tests, that revealed no difference between the peak time of responding in the LP and CS conditions, $t(7) = 2.206$, $p = .063$, SEM = .278, but a smaller variance for the LP than the CS condition, $t(7) = 3.672$, $p = .008$, SEM = .429, and a smaller R^2 for the LP than the CS condition, $t(7) = 4.376$, $p = .003$, SEM = .016. A Bayes analysis was run on peak response time data giving a Bayes factor of 0.700. As in Experiment 4, this is consistent with the idea that the peak time of responding is not meaningfully longer for LP than CS trials (despite the numerical difference) but again, there is no suggestion that the data is consistent with a causal binding effect where the peak time was shorter for LP trials.

During the devaluation procedure, rats consumed a mean of 6.06g (SEM = 0.324g) of food pellets and 5.83g (SEM = 0.648) of sucrose solution. The mean number of lever presses during the extinction test for the lever associated with the devalued outcome was 5.00 (SEM = 1.899) for the sucrose reinforcer and 9.75 (SEM = 2.938) for pellets. The number of lever presses for the non-devalued outcome was 11.00 (SEM = 1.783) for the sucrose reinforcer and 19.25 (SEM = 6.129) for pellets. Thus there were lower rates of responding on the lever associated with the devalued than the non-devalued outcome, and little difference in this devaluation effect depending on which of the outcomes was devalued. ANOVA confirmed

that there was an effect of devaluation, $F(1, 7) = 8.680$, MSE = 55.357, $p = .022$, no main effect of whether the reinforcer was food pellets or sucrose, $F(1, 7) = 1.742$, MSE = 194.00, $p = .228$, and no interaction between these factors, $F(1, 7) = 0.560$, MSE = 43.786, $p = .479$.

3.2.4. Discussion

Humans perceive the interval between their actions and a resulting outcome to be shorter than the interval between an external stimulus and an outcome (see Haggard *et al.*, 2002; Buehner & Humphreys, 2009). Chapter 3 investigated whether the timing behaviour of rats exhibits such a causal binding effect. In each experiment, an action (a lever press) or a CS (e.g., a tone) served as the referent for the start of a five-second interval that terminated in the delivery of an outcome. Rats were trained, using a peak procedure, until their peak rate of responding on non-reinforced probe trials was approximately five seconds. Using this procedure, evidence of causal binding would take the form of an earlier peak response time on lever press trials than on CS trials. In none of the experiments was this pattern of results observed. One potential explanation for the failure of instrumental responding to show evidence of causal binding is that lever press actions had become independent of the outcome during the extensive training that was required to establish reliable timing behaviour. However, in Experiment 6, it was established that causal binding was not observed under conditions in which instrumental responding was demonstrably outcome dependent: Satiation on one of two outcomes selectively depressed performance of the instrumental response associated with that outcome.

Although these experiments revealed no difference in timing behaviour between action and external cues, there is another potential explanation for the results obtained. As noted in the method section, the lever was withdrawn from the chamber once pressed. This may be acting as a cue for the rats to time from (albeit, this environmental event is under the control of the rat). The issue as to whether the rats are timing from their own action, or from

the external cue that this action produces, is worth exploring, as if it is the case that the rats are timing from the external cue, this takes away the critical comparison I am looking for in these experiments. It should be noted that external cues produced by the action are also present in the human studies (e.g., the button returning to its original position; for instance, see Haggard *et al*., 2002). However, in Buehner and Humphrey's (2009) study, both of the conditions involved an action, although the action was only causally relevant in one condition. Regardless, if both the lever press and its withdrawal are being used as cues for the timing, then the peak response time should reflect the combination of both. That is, the peak time should still be shorter in the LP condition (assuming causal binding effects) if the LP has any control over the timing of the nose poke response. However, numerically in two of the three experiments, the LP trials peak later than the CS trials.

From a causal model perspective, the fact that lever withdrawal was equivalent to the lever press as a cue for the timing of food should not be an issue, because Leising *et al.* (2008) argued that actions are more readily established as causes than external cues. Thus, the action of pressing the lever would be expected to control behaviour rather than the withdrawal of the lever. Nevertheless, suggestions for how my procedure could be improved to control for this possibility are discussed in Section 5.3.

Causal binding has been attributed to the internal "clock" or pacemaker running faster (e.g., see Humphreys & Buehner, 2010). If so, then the extended training used in these experiments might allow the rats to compensate for the change in clock speed by learning that the food arrived after a larger number of clock pulses in the LP than the CS case, thus supporting accurate timing for both. But the idea that the perceived LP to food interval will have more clock pulses than the CS to food interval also has implications of its own – in particular that the variability of the response should be higher in the LP case. That is, SET (Gibbon, 1977) suggests that the variability of a timing response should increase with its

mean value. However, the variability is always numerically less in the LP case, and this difference is significant in two of the three experiments. The data collected here are thus not consistent with the idea of more frequent pulses during the LP trials.

Although the results observed in Experiments 4-6 are inconsistent with the possibility that rats' judgments of temporal intervals is subject to a process of causal binding, they are consistent with formal models of timing. For example, SET (Gibbon, 1977) and LeT (Machado, 1997) both assume (implicitly) that timing will not be influenced by whether the temporal referent is an action and external stimulus. Until now, this assumption has not been directly assessed, and the novel results presented in this paper provide support for models of timing that have been developed and evaluated in the context of studies of animal behaviour. The implications of these results will be further discussed in Chapter 5.

Chapter 4: Sensory preconditioning as a form of reasoning

4.1. Introduction

The results of the experiments in Chapters 1 and 2 revealed no evidence supporting causal representation or causal reasoning in rats. However, the idea that rats are capable of propositional thought pre-dates Blaisdell *et al.*'s (2006) research. For instance, Hall (1990) suggested that sensory preconditioning might be considered to be an example of causal cognition (see Section 1.4). There are also associative explanations of sensory preconditioning, many of which rely on the principle of stimulus substitution, where whatever conditioned behaviours come to be elicited by A, as the result of it being directly paired with a US, might well be provoked by B, as the result of it being paired with A. As mentioned already (Section 1.4), the elemental chain and configural accounts provide good theoretical grounds for supposing that this will be so: either the memory of A or the configural memory of AB mediate conditioned the transfer of responding between A and B.

Here, four experiments are reported that assessed the prediction, derived from both propositional accounts and some associative accounts of sensory preconditioning, that there should be strict stimulus substitution. The procedure used produces a very robust sensory preconditioning effect (Rescorla & Cunningham, 1978). The procedure involves presenting rats with two flavour compounds (AB and CD), and then pairing one of the component flavours (A), but not the other (C), with injections of the nausea-inducing agent lithium chloride (LiCl). The standard outcome of this procedure is that rats will not only be less inclined to consume A than C, but they will also be less inclined to consume B than D. Although the most widely examined consequence of pairing a flavoured stimulus with LiCl is the subsequent reduction in its consumption, there are also reliably observed changes in the form of the consummatory response. For example, flavours previously paired with LiCl-induced illness elicit aversive orofacial behaviours in taste reactivity tests (e.g., Grill &

Norgren, 1978; Pelchat, Grill, Rozin, & Jacobs, 1983), suggesting that the test flavour is now unpalatable or aversive. In addition, flavours paired with LiCl elicit reduced lick cluster sizes (e.g., Baird, John, & Nguyen, 2005; Dwyer, 2009; Dwyer, Boakes, & Hayward, 2008). To consider the latter effect in a little more detail, first note that rats ingest fluids in sustained runs of licks (*clusters*) separated by pauses, and the mean number of licks per cluster (lick cluster size) is lawfully related to the nature of the solution being consumed: Lick cluster size shows a positive, monotonic relationship to the concentration of palatable fluids such as sucrose (e.g., Davis & Smith, 1992; Spector, Klumpp, & Kaplan, 1998) and decreases monotonically with increasing concentrations of unpalatable quinine solutions (e.g., Hsiao & Fan, 1993; Spector & St John, 1998). Moreover, benzodiazepine administration, which is thought to enhance hedonic reactions to foods in humans (Haney, Comer, Fischman, & Foltin, 1997), enhances lick cluster size (e.g., Higgs & Cooper, 1998). These, and other, results suggest that the size of licking clusters is a variable directly related to the palatability of the solution being consumed (for a review, see Dwyer, 2012). In this light, the reduction in lick cluster size observed in conditioned flavour aversion reinforces the idea from taste reactivity studies that pairing a flavour with LiCl-induced illness both reduces its consumption and results in the flavour becoming unpalatable.

A recent review has argued that theoretical analyses of learning must allow for separable representations of different aspects of the CS and US – and for manipulations of the learning situation to influence which aspects of these CSs and USs become associatively linked – if they are to provide a complete account of Pavlovian conditioning (Delamater, 2012). Notably, the same review also cited lick analysis methods as a key technique in addressing dissociable aspects of the US. Thus, this technique was used to understand the basis of sensory preconditioning. In the current context, direct flavour-aversion conditioning with A should result in both a reduction in consumption of A and a reduction in lick cluster

sizes indicating a concurrent reduction in palatability. The prediction of interest here, shared by propositional and some associative accounts, is that both of these robust conditioned responses of direct conditioning (to A) should also be evident to a flavour (B), that has been paired with A in a sensory preconditioning procedure. If it transpires that the prediction is accurate, then propositional and some associative accounts gain support; but if not, then other accounts for sensory preconditioning will need to be considered (e.g., those based on mediated conditioning) that are not constrained to make this prediction.

4.2. Experiment 7

4.2.1. Introduction

The design of Experiment 7 is shown in Table 4.1. All rats first received repeated exposure to two flavour compounds, AB and CD, prior to conditioning trials in which flavour A was paired with LiCl and flavour C was presented without consequences. The consumption of each of the flavours A-D was then examined in separate 1-bottle tests. Throughout the training and test phases, the timing of all licks was recorded to allow for the analysis of lick cluster sizes. Comparing responding to A and C during test provided the assessment of direct conditioning, while comparing B and D provided the assessment of sensory preconditioning. On the basis of previous analyses of conditioned flavour aversion, rats should consume less of A than C, and also that lick cluster sizes elicited by A will be smaller than those elicited by C. As described above, stimulus substitution accounts of sensory preconditioning suggest that equivalent effects will be seen with B and D. However, any dissociation between the effects of direct conditioning and sensory preconditioning on consumption and lick cluster size would be difficult to reconcile with such accounts.

Table 4.1. Design of Experiments 7, 8, and 10.

	Pre-training	Conditioning	Test
Experiment 7			
	3 × AB, 3 × CD	A→10ml/kg LiCl, C-	A, B, C, D
Experiment 8			
Low	3 × AB, 3 × CD	A→5ml/kg LiCl, C-	A, B, C, D
High	3 × AB, 3 × CD	A→15ml/kg LiCl, C-	A, B, C, D
Experiment 10			
Immediate	3 × AB, 3 × CD	A→10ml/kg LiCl, C-	A, B, C, D
Trace	3 × AB, 3 × CD	A→water→15ml/kg LiCl, C-	A, B, C, D

Note: AB and CD represent flavour compounds constructed from the elements (A-D; solutions of sucrose, maltodextrin, salt and lemon); "+" represents the intraperitoneal administration of 0.15M LiCl, while "-" represents no outcome. Contrasting the responses to A and C at test provides an assessment of a direct conditioning, while contrasting B and D assesses sensory preconditioning.

4.2.2. Method

Subjects. Twenty-four male Lister hooded rats (*Rattus Norvegicus*) were obtained from Harlan, Bicester, UK for the purposes of the study. Their weights before the beginning of the study ranged from 346g to 425g, with a mean weight of 378g. The rats were housed in pairs in a room illuminated between the hours of 0800-2000, where they had ad-lib access to food and received 60 min access to water per day, approximately 1hr after the experimental sessions.

Apparatus and Stimuli. Rats were trained and tested in eight custom-made drinking chambers (Med Associated Inc., St Albans, USA). These were 32 × 15 × 12 cm (L × W × H), with steel mesh flooring and with white acrylic walls. Fluids were made accessible through drinking spouts made of stainless steel, attached to 50ml cylinders. These could be inserted on the left or right hand side of the lid (made of wire mesh). The distance between the holes for the bottles was 8cm. Only the left hand side was used for the current studies. A contact sensitive lickometer registered the time of each lick to the nearest 0.01s. This was recorded by a computer using MED-PC software (Med Associates Inc.). The amount of fluid consumed by each rat was measured by weighing the drinking bottle before and after each session. The stimuli were solutions of 3% sucrose, 4% maltodextrin, 2% lemon juice and 1% salt (all w/w). When presented as simultaneous compounds, the two solutions were mixed so as to maintain these concentrations.

Procedure. All experimental sessions were 10min in duration and there was one session on each per. To acclimatise the rats to the experimental apparatus they were given one 10-min session with access to water. The following 6 sessions comprised three exposures to the pairs of flavour compounds in alternation (AB, CD,… or CD, AB…; see Table 4.1). The assignment of stimuli to condition was counterbalanced such that each of sucrose, maltodextrin, lemon and salt was used equally often as stimuli A, B, C, or D. In order to

minimize unconditioned differences in the responses to stimuli within a test pair (A vs. C for direct conditioning, B vs. D for sensory preconditioning) the assignment of solutions to conditions was constrained such that a test pair always comprised either sucrose and maltodextrin or salt and lemon.

On days 7 and 8 rats received access to A (on one day) and C (on the other) in a counterbalanced order. Access to A was followed by an intraperitoneal injection of 0.15M LiCl (at 10ml/kg bodyweight) and access to C was not. Sessions 9-12 comprised the test period where all rats received one exposure to each of the solutions. The solutions were given in the order sucrose, maltodextrin, salt and lemon for all rats: Thus, due to the counterbalancing of the assignment of solutions to conditions, across animals the order of testing was equally often ACBD, ACDB, CABD, CADB, BDAC, BDCA, DBAC, or DBCA.

Data Analysis. In addition to the consumption data, the mean cluster size for each rat was extracted from the record of licks for analysis. A cluster was defined as a set of licks each separated by an inter-lick-interval of no more than 0.5s. This criterion is used by Davis and his co-workers (e.g., Davis & Perez, 1993; Davis & Smith, 1992) and in the majority of our previous studies using lick analysis techniques (Dwyer, 2008, 2009; Dwyer, *et al.*, 2008; Dwyer, Lydall, & Hayward, 2011; Lydall, Gilmour, & Dwyer, 2010a, 2010b). Although other criteria have been used (e.g., Dwyer, Pincham, Thein, & Harris, 2009; Spector, *et al.*, 1998), parametric analyses suggest that there is little practical difference between them as most pauses greater than 0.5s are also greater than 1s (e.g., Davis & Smith, 1992; Spector, *et al.*, 1998). The data from pretraining and aversion conditioning was monitored to ensure that all solutions were being consumed, which they were by all rats. These data will not be considered further for this or subsequent experiments. Repeated-measures analyses of variance (ANOVA) were used to analyse the test data with factors of aversion condition (i.e., whether the stimuli should be subject to an aversion or not: A & B versus C & D) and

training type (direct conditioning or sensory preconditioning: A & C versus B & D). All tests reported here used a criterion for significance of $p = 0.05$.

4.2.3. Results

Figure 4.1 shows the data from the test sessions (consumption in Panel A and lick cluster size in Panel B). Inspection of Panel A suggests that consumption scores were lower for A than for C (direct conditioning) and lower for B than for D (sensory preconditioning), although the difference was larger for direct than for sensory preconditioning. ANOVA conducted on the amount consumed revealed significant effects of aversion condition (A & B vs. C & D; $F(1, 23) = 50.62$, MSE = 16.34, $p < .001$), training type (i.e., direct vs. sensory preconditioning; $F(1, 23) = 88.99$, MSE = 1.89, $p < .001$), and an interaction between them ($F(1, 23) = 34.66$, MSE = 8.41, $p < .001$); but, simple effects analyses revealed that the difference in consumption was significant for both the direct conditioning (A vs. C) and sensory preconditioning (B vs. D) ($F(1, 23) = 112.32$, MSE = 9.35, $p < .001$; and $F(1, 23) = 4.44$, MSE = 15.40, $p < .05$, respectively).

Inspection of Panel B suggests that while lick cluster sizes for were smaller for A than C (direct conditioning), there was little difference in this measure for B and D (sensory preconditioning). ANOVA on lick cluster sizes revealed significant effects of aversion condition ($F(1, 23) = 31.22$, MSE = 219.38, $p < .001$), training type ($F(1, 23) = 11.72$, MSE = 98.13, $p = .002$), and an interaction between them ($F(1, 23) = 9.07$, MSE = 286.12, $p < .01$). In contrast to the consumption data, simple effects analyses revealed a significant difference in lick cluster size between the A and C ($F(1, 23) = 69.72$, MSE = 128.19, $p < .001$), but not between B and D ($F(1, 23) = 1.34$, MSE = 377.30, $p = .259$).

As was noted in Chapter 3, Bayesian analysis can be used to assess the degree of support that a non-significant result gives to the idea that there is a genuine absence of an

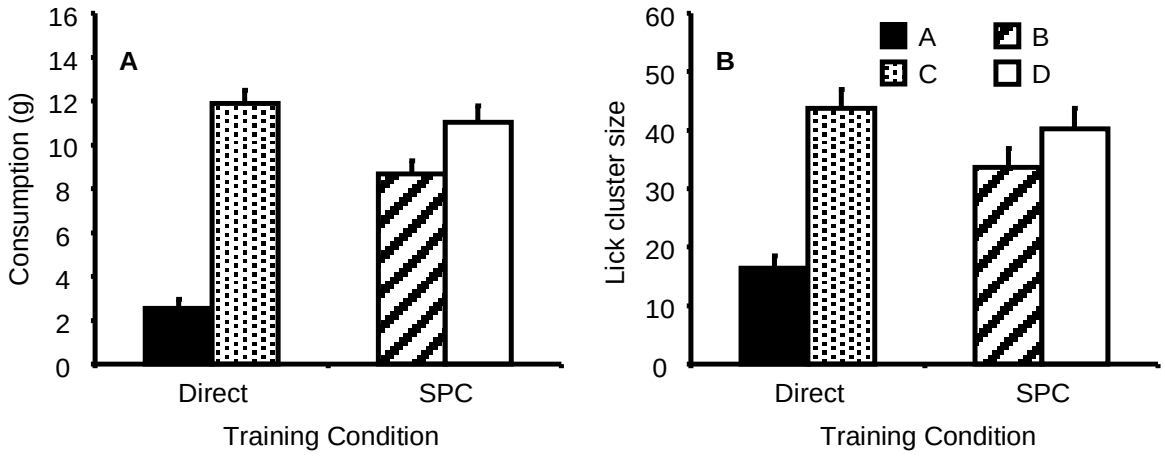


Figure 4.1. Experiment 7: Mean (+SEM) number of licks per trial (panel A) and lick cluster size (panel B) during the test with A and C (direct conditioning) and flavours B and D (sensory preconditioning).

effect. Therefore, the same technique was applied to the difference between B and D in the lick analysis data. The Bayes factor for this comparison was 1.970, which is inconclusive for the genuine absence of an effect.

The fact that differential direct conditioning to A and C was evident as differences in both the levels of consumption and cluster size, but sensory preconditioning was only evident in the levels of consumption of B and D, represents the type of dissociation that casts doubt on standard accounts of sensory preconditioning which rely on stimulus substitution. However, while the difference in the size of lick clusters for B and D did not approach statistical significance, they were numerically smaller for B than for D and the Bayes analysis did not provide support for accepting the null or alternative hypothesis. Thus, before considering the theoretical implications of the results of Experiment 7, I sought to replicate and extend them.

4.3. Experiment 8

4.3.1. Introduction

The design of Experiment 8, shown in Table 4.1, was equivalent to Experiment 7 with the exception that one group of rats received 5ml/kg (Low) and the other received 15ml/kg of LiCl (High). If the dissociation observed in Experiment 7 reflects a genuine difference between direct conditioning and sensory preconditioning then it should be observed across a range of conditioning parameters. However, if this dissociation reflects the fact that weak conditioning per se does not change lick cluster size then increasing the dose of LiCl might reveal a sensory preconditioning effect using the lick cluster size measure, while decreasing the dose of LiCl might serve to reduce the modulation of lick cluster size by direct conditioning.

4.3.2. Method

Forty-eight male Lister hooded rats, obtained from the same source as in Experiment 7 and maintained in the same fashion, were used. Their weights before the beginning of the study ranged from 333-389g, with a mean weight of 358g. All details of the stimuli, apparatus, and procedure were the same as in Experiment 7 with the following exceptions: A total of sixteen experimental chambers were used and half of the rats (Group Low US) received 0.15M LiCl at 5ml/kg while the remainder (Group High US) received 0.15M LiCl at 15ml/kg.

4.3.3. Results

Figure 4.2 shows the data from the test sessions (consumption in Panel A and lick cluster size in Panel B). As in Experiment 7, inspection of Panel A suggests that both direct conditioning (involving A and C) and sensory preconditioning (involving B and D) was evident in different levels of consumption; and that this was the case in both groups Low and High. The consumption data was assessed using a mixed ANOVA with a between-subject factor of LiCl strength and within-subject factors of aversion condition (i.e., whether subjected to an aversion or not: A & B versus C & D) and training type (direct conditioning or sensory preconditioning: A & C versus B & D). This analysis revealed no significant effect of LiCl strength ($F(1, 46) = 2.62$, MSE = 10.35, $p = .112$) or interactions involving this factor (Fs < 1). There were significant effects of aversion condition ($F(1, 46) = 53.35$, MSE = 13.83, $p < .001$), training type ($F(1, 46) = 32.11$, MSE = 6.44, $p < .001$), and an interaction between them ($F(1, 46) = 21.16$, MSE = 10.78, $p < .001$). In order to assess this interaction, the data from direct conditioning and sensory preconditioning were subject to separate 2-way ANOVAs examining the factors of aversion condition and LiCl strength. In the case of direct conditioning, consumption of A was lower than C ($F(1, 46) = 67.39$, MSE = 13.25, $p < .001$), but there was no effect of LiCl strength ($F(1, 46) = 2.77$, MSE = 7.23, $p = .114$), nor any

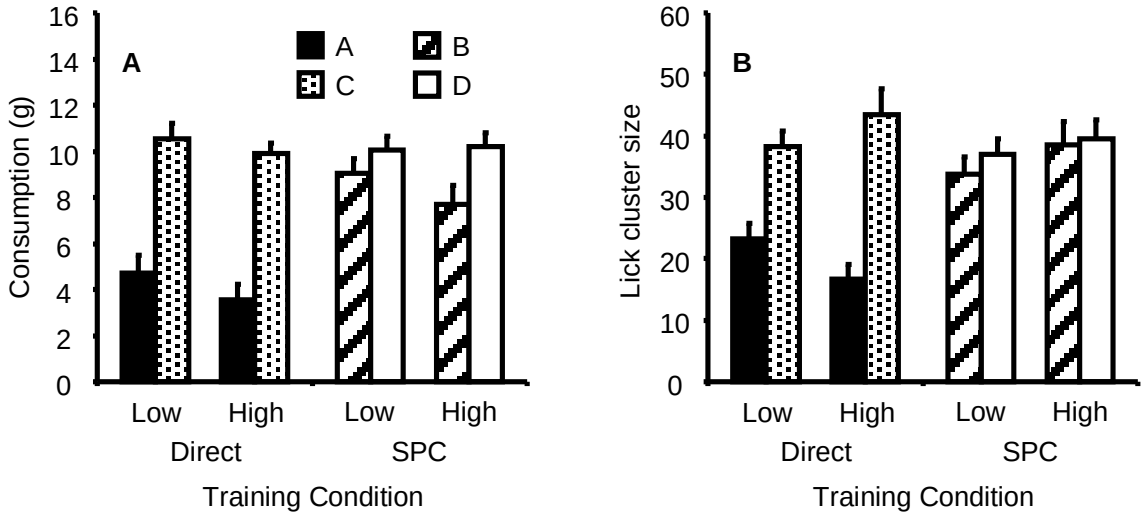


Figure 4.2. Experiment 8: Mean (+SEM) number of licks per trial (panel A) and lick cluster size (panel B) during the test with flavours A and C (direct conditioning) and B and D (sensory preconditioning). Half of the rats received 5ml/kg LiCl and the remainder received 15ml/kg LiCl.

interaction between these factors ($F < 1$). Similarly, the sensory preconditioning data revealed that consumption of B was less than that of D ($F(1, 46) = 6.41$, MSE = 11.36, $p = .015$), there was no effect of LiCl strength ($F < 1$), nor any interaction between these factors ($F(1, 46) = 1.23$, MSE = 11.36, $p = .274$). That is, as in Experiment 7, both direct conditioning and sensory preconditioning resulted in lower consumption of A than C and of B than D; albeit that the former, directly conditioned effect was larger than the latter sensory preconditioning effect.

Inspection of Panel B suggests that lick cluster sizes was lower for A than C (direct conditioning), but did not differ between B and D (sensory preconditioning); and this dissociation was evident in both groups High and Low. Again, there was no significant effect of LiCl strength ($F < 1$) or interactions involving this factor (largest $F(1, 46) = 3.02$, MSE = 203.33, $p = .089$, for the 3-way interaction between LiCl strength, aversion condition, and training type). There were significant effects of aversion condition ($F(1, 46) = 36.56$, MSE = 171.12, $p < .001$), training type ($F(1, 46) = 16.12$, MSE = 138.77, $p < .001$), and an interaction between them ($F(1, 46) = 20.86$, MSE = 203.33, $p < .001$). As with the consumption data, this interaction was assessed by examining separate 2-way ANOVAs for the direct conditioning and sensory preconditioning data. In the direct conditioning case lick cluster sizes were lower for A than C ($F(1, 46) = 48.36$, MSE = 217.12, $p < .001$), but there was no effect of LiCl strength ($F < 1$), nor a significant interaction between these factors ($F(1, 46) = 3.87$, MSE = 217.12, $p = .055$). In contrast, the sensory preconditioning data revealed no suggestion difference between B and D ($F < 1$), as well as no effect of LiCl strength ($F(1, 46) = 1.05$, MSE = 302.92, $p = .312$), nor an interaction between these factors ($F < 1$).

As in the previous experiment, a Bayes analysis was run on the B versus D lick analysis data. The Bayes factor for the 5ml LiCl group and 15ml LiCl group was 3.321 and

6.251 respectively, and thereby provide support for the idea that there genuinely is no effect of sensory preconditioning on lick cluster size.

4.4. Experiment 9

4.4.1. Introduction

Experiments 7 and 8 both produced the same dissociation: direct conditioning resulted in reduced consumption and lick cluster size for A compared to the C, but sensory preconditioning only resulted in reduced consumption of the B compared to D while leaving lick cluster sizes unaffected. Taken at face value, this pattern of results represents a direct challenge to standard accounts of sensory preconditioning that require strict stimulus substitution. However, it remains the case that in both of Experiments 7 and 8, sensory preconditioning produced smaller changes in consumption than did direct conditioning. Thus, it is possible that the same dissociation would be observed with direct conditioning parameters that produced weak conditioning. For example, it is possible that B is less able to activate A (or AB) or the US representation than is the direct application of A, and that it is these differences that produce the dissociation of the two measures of performance. Therefore, in Experiment 9, the intensity of the flavours and the US were manipulated in a direct conditioning procedure, to assess whether or not this would produce the same dissociation between the measures of performance that was evident in the sensory preconditioning procedure in Experiments 7 and 8.

All rats received four stimuli, A-D, that were arranged as two pairs (A, C and B, D). One of the pairs (A and C – "Normal" concentration) was presented at the same concentrations as in Experiments 7 and 8, and for the second pair of stimuli (B and D – Dilute concentration) these concentrations were halved. For half of the rats (those in group High) received 5ml/kg of LiCl (i.e., the weakest dose examined in Experiments 7 and 8) after A and B, and the remainder (those in group Low) received 2.5ml/kg of LiCl. C and D were

Table 4.2. Design of Experiment 9.

	Normal flavour concentration	Dilute flavour concentration	Test
High	A→5ml/kg LiCl, C-	B→5ml/kg LiCl, D-	A, B, C, D
Low	A→2.5ml/kg LiCl, C-	B→2.5ml/kg LiCl, D-	

Note: A-D represent flavours (solutions of sucrose, maltodextrin, salt and lemon). The concentrations at which these flavours were presented (Normal: A and C – equal to Experiments 8 and 9; or Dilute: B and D – half the concentrations used previously) was manipulated within subjects, and the amount of LiCl (High: 5ml/kg; or Low: 2.5ml/kg) was manipulated between subjects. For all rats, A and B were paired with LiCl and C and D were not. Contrasting the responses to A and C at test provides an assessment of conditioning with CSs of the same concentration as in Experiments 7 and 8, while contrasting B and D assesses condition with stimuli of lower concentrations than used previously.

not paired with LiCl (see Table 4.2 for the full design). If the finding that sensory preconditioning was evident as a difference in consumption, but not lick cluster size, simply reflected the fact that the presentation of B was only able to weakly activate A (or AB), then the same dissociation should be observed for B in Experiment 9. Similarly, if the dissociation reflected the fact that B was only able to weakly activate the representation of LiCl, then it should also be evident in group Low. Of course, if the dissociations reflected a combination of these two factors, then the dissociation should be most apparent for stimulus B in group Low.

4.4.2. Method

Thirty-two male Lister hooded rats, obtained from the same source as in Experiments 7 and 8 and maintained in the same fashion, were used. Their weights before the beginning of the study ranged from 314-348g, with a mean weight of 331g. The details of the stimuli, apparatus, and procedure are as in the previous experiments except as outlined below.

To acclimatize the rats to the experimental apparatus they were given two 10min sessions with access to water. The four following sessions comprised one exposure to each of stimuli A, B, C, and D. The assignment of stimuli to condition was counterbalanced such that each of sucrose, maltodextrin, lemon and salt was used equally often as stimuli A, B, C, or D. Stimuli A and C were presented at the same concentrations as in Experiments 7 and 8 while stimuli B and D were presented at half these concentrations. In order to minimize unconditioned differences in the responses to stimuli within a test pair (A vs. C and B vs. D) the assignment of solutions to conditions was constrained such that a test pair always comprised either sucrose and maltodextrin or salt and lemon. Moreover, for an equal number of rats in the counterbalanced subgroups, training sessions with the four stimuli were presented in the following orders: ABCD, BADC, CDAB, or DCBA. Half of the rats (group

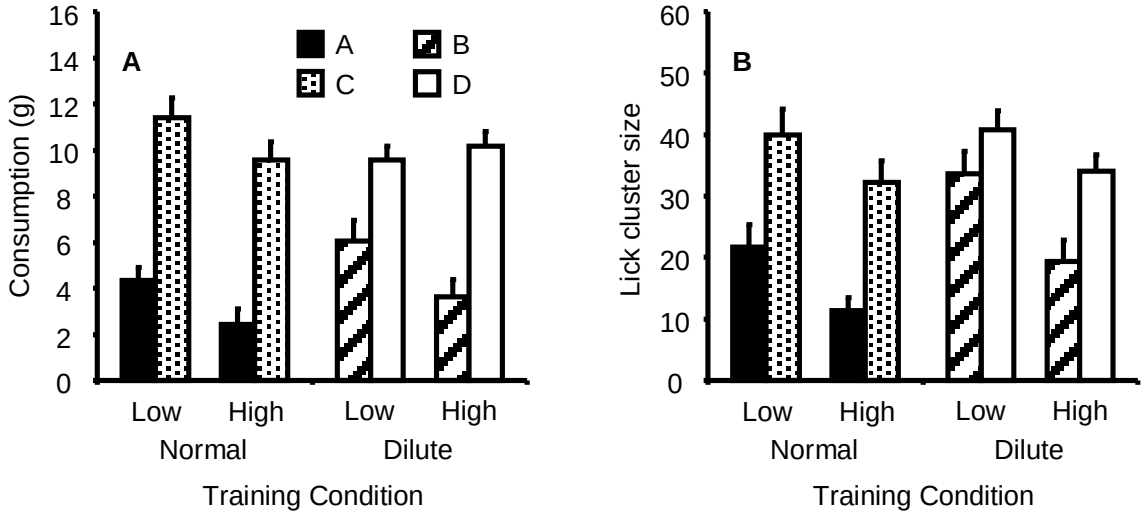


Figure 4.3. Experiment 9: Mean (+SEM) number of licks per trial (panel A) and lick cluster size (panel B) during the test with flavours A and C (high concentration) and B and D (low concentration). Half of the rats received 2.5ml/kg LiCl and the remainder received 5ml/kg LiCl.

High) received 0.15M LiCl at 5ml/kg while the remainder (group Low) received 0.15M LiCl at 2.5ml/kg after the designated stimuli, A and B. Sessions 7-10 comprised the test period where all rats received one exposure to each of the solutions. The solutions were given in the order sucrose, maltodextrin, salt and lemon for all rats. Thus, due to the counterbalancing of the assignment of solutions to conditions, across animals the order of testing was equally often ACBD, ACDB, CABD, CADB, BDAC, BDCA, DBAC, or DBCA.

4.4.3. Results

Figure 4.3 shows the data from the test sessions (consumption in Panel A and lick cluster size in Panel B). Inspection of Panel A suggests that consumption of the flavours paired LiCl (i.e., A and B) was lower than the flavours that were not (i.e., C and D) regardless of LiCl intensity. Inspection of Panel B suggests that lick cluster sizes were also lower for A and B than for C and D regardless of LiCl intensity. The test data were analysed as in Experiment 8: Namely, a mixed ANOVA with a between-subjects factor of the amount of LiCl and within-subjects factors of stimulus type, and flavour concentration, followed by separate 2-way ANOVAs examining the factors of stimulus type and amount of LiCl for groups High and Low.

The analysis of consumption revealed significant effects of amount of LiCl ($F(1, 30) = 10.85$, MSE = 5.76, $p = .003$) and stimulus type ($F(1, 30) = 105.68$, MSE = 11.19, $p < .001$). There were no other significant main effects or interactions (largest $F(1, 30) = 2.40$, MSE = 14.10, $p = .131$, for the interaction between flavour concentration and stimulus type). To provide a direct parallel with the analysis of Experiment 8, the data from A and C (the stimuli presented at the same concentrations as Experiments 7 and 8) and from B and D (the stimuli presented at half of these concentrations) were subject to separate 2-way ANOVAs examining the factors of stimulus type and amount of LiCl. For A and C, consumption of the A was lower than that of the C ($F(1, 30) = 60.19$, MSE = 13.43, $p < .001$) and consumption

was lower in group High than in group Low (F(1, 30) = 12.90, MSE = 4.35, p = .001). There was no interaction between these factors (F < 1). For B and D, consumption of B was lower than D (F(1, 30) = 34.41, MSE = 11.86, p < .001); but there was no effect of group (F(1, 30) = 2.82, MSE = 8.85, p = .103), nor a significant interaction (F(1, 30) = 3.11, MSE = 11.86, p = .088). That is, as in the previous experiments, direct conditioning resulted in lower consumption of flavours that were directly paired with LiCl than those that were not, even when the concentrations of the flavours and LiCl were reduced from the values used in Experiments 7 and 8.

The analysis of the lick cluster data revealed significant effects of the amount of LiCl (F(1, 30) = 12.28, MSE = 249.13, p = .001), stimulus type (F(1, 30) = 32.26, MSE = 230.26, p < .001), and flavour concentration (F(1, 30) = 11.32, MSE = 91.43, p = .002). There was also a significant interaction between flavour concentration and stimulus type (F(1, 30) = 4..56, MSE = 129.33, p = .041). There were no other significant interactions (Fs < 1). As before, the scores for A and C, and for B and D, were subject to separate 2-way ANOVAs examining the factors of stimulus type and amount of LiCl. For A and C, lick cluster sizes were lower for A than C (F(1, 30) = 33.15, MSE = 184.10, p < .001) and lower in group High than group Low (F(1, 30) = 8.42, MSE = 154.85, p = .007). There was no interaction between these factors (F < 1). For B and D, lick cluster sizes were lower for B than D (F(1, 30) = 10.92, MSE = 175.49, p = .002), and lower in group High than in group Low (F(1, 30) = 9.55, MSE = 185.71, p = .004). There was no interaction between these factors (F(1, 30) = 1.31, MSE = 175.49, p = .262). That is, as in the previous experiments, direct conditioning resulted in lower lick cluster sizes for flavours paired with LiCl than those that were not, even when the concentrations of the flavours and LiCl had been reduced from the values used in Experiments 7 and 8. Whereas Experiment 9 was intended to vary the strength of activation

of the CS, Experiment 10 attempted to manipulate the nature of the CS memory that was present immediately prior to the US.

4.5. Experiment 10

4.5.1. Introduction

One difference between direct conditioning and sensory preconditioning is that in direct conditioning the stimulus paired with the US and the test stimulus are both directly activated by a CS presented to the animals, whereas in sensory preconditioning the test stimulus was not presented during conditioning. In terms of Wagner's SOP model (1981), the critical stimuli during direct conditioning are in the A1 state during training and test, whereas sensory preconditioning involves evoked (i.e., A2) memories of stimuli. However, in some forms of conditioning, notably trace conditioning, the CS might be expected to decay, at least partially, into the A2 state. If the difference between direct and SPC, in terms of their effects on lick cluster size, is related to the representational state (A1 vs. A2) of the CS, then a simple manipulation should influence the dissociations observed. The design of Experiment 10, shown in Table 4.1, was equivalent to Experiment 7 with the exception that one group of rats received the LiCl US immediately following the presentation of solution A (group Immediate) while rats in the other group were allowed to consume water during an interval of 30 minutes that was left between presentation of A and the administration of LiCl (group Trace).

4.5.2. Method

Thirty-two male Lister hooded rats were used. These rats were obtained from the same source as in Experiment 7 and maintained in the same manner. Their weights before the study began ranged from 345g-461g, with a mean of 404g. All details of the stimuli, apparatus, and procedure were the same as described in Experiment 7 with the following

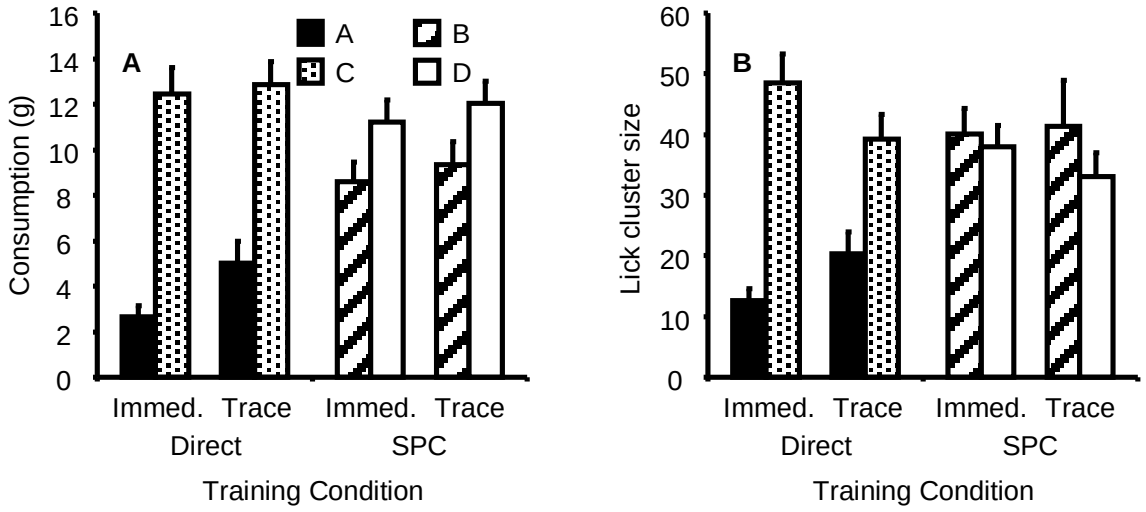


Figure 4.4. Experiment 10: Mean (+SEM) number of licks per trial (panel A) and lick cluster size (panel B) during the test with flavours A and C (direct conditioning) and B and D (sensory preconditioning). Half of the rats received LiCl immediately after presentation of A during the training phase and the remainder received LiCl following a 30 min delay.

exceptions: Sixteen experimental chambers were used. Half of the rats (group Immediate) received LiCl injections immediately after access to A, were replaced in the apparatus, and then allowed to consume water for 30 min. The remainder (group Trace) received flavour A, followed by access to water for 30 min in the apparatus, before LiCl administration. Once these treatments were complete, the rats were taken back to their home cages.

4.5.3. Results and Discussion

Figure 4.4 shows the results from the test sessions, with consumption in Panel A and lick cluster size in Panel B. As in Experiment 7, inspection of Panel A suggests that both direct conditioning (involving A and C) and sensory preconditioning (involving B and D) was evident in different levels of consumption; and that this was the case in both groups Immediate and Trace. The consumption data was assessed using a mixed ANOVA with a between-subject factor of LiCl delivery (immediate or trace) and within-subject factors of aversion condition (i.e., A & B versus C & D) and training type (direct conditioning or sensory preconditioning: A & C versus B & D). This analysis revealed no significant effect of the delay manipulation ($F(1, 30) = 2.71$, MSE = 13.88, $p = .110$) or interactions involving this factor (Fs < 1). There were significant effects of aversion condition ($F(1, 30) = 41.08$, MSE = 25.68, $p < .001$), training type ($F(1, 30) = 26.59$, MSE = 5.09, $p < .001$), and an interaction between them ($F(1, 30) = 24.04$, MSE = 12.70, $p < .001$). As in Experiments 8 and 9, to assess this interaction, the data from direct conditioning and sensory preconditioning were subject to separate 2-way ANOVAs examining the factors of aversion condition and LiCl delivery. In the case of direct conditioning, consumption of A was lower than C ($F(1, 30) = 71.11$, MSE = 17.54, $p < .001$), but there was no effect of LiCl delivery ($F(1, 30) = 2.90$, MSE = 10.52, $p = .099$), nor any interaction between these factors ($F < 1$). Similarly, the sensory preconditioning data revealed that consumption of B was less than of D ($F(1, 30) = 5.41$, MSE = 20.83, $p = .027$), there was no effect of LiCl delivery ($F(1, 30) =$

1.169, MSE = 8.45, p = .288), nor any interaction between these factors (F < 1). That is, as in Experiment 7, both direct conditioning and sensory preconditioning resulted in lower consumption of A than C and of B than D; albeit that the former, directly conditioned effect was larger than the latter sensory preconditioning effect.

Inspection of Panel B indicates that lick cluster sizes were lower for A than C (direct conditioning), but did not differ between B and D (sensory preconditioning); and this dissociation was evident in both groups Immediate and Trace. Moreover, the difference between A and C was smaller in group Trace than in group Immediate. Again, there was no significant effect of LiCl delivery (F < 1) or interactions involving this factor (largest F(1, 30) = 3.23, MSE = 334.99, p = .082, for the interaction between delay and aversion condition). There were significant effects of aversion condition (F(1, 30) = 11.85, MSE = 334.99, p = .002), training type (F(1, 30) = 8.97, MSE = 227.28, p = .005), and an interaction between them (F(1, 30) = 42.19, MSE = 200.22, p < .001). As with the consumption data, this interaction was assessed by examining separate 2-way ANOVAs for the direct conditioning and sensory preconditioning data. In the direct conditioning case, lick cluster sizes were smaller for A than C (F(1, 30) = 47.73, MSE = 251.39, p < .001), but there was no effect of LiCl delivery (F < 1). Critically, there was an interaction between these factors (F(1, 30) = 4.61, MSE = 251.39, p = .040). Despite this interaction there was a significant difference in lick cluster size between A and C in both cases (F(1, 15) = 37.03, MSE = 278.38, p < .001; and F(1, 15) = 12.70, MSE = 224.41, p = .003, for the Immediate and Trace conditions respectively). In contrast, the sensory preconditioning data revealed no suggestion of a difference between B and D (F(1, 30) = 1.48, MSE = 283.82, p = .234), as well as no effect of LiCl delivery, nor an interaction between these factors (Fs < 1).

As previously, Bayes analyses were run on the B versus D comparison in both the Trace and Immediate conditions. Although, the Trace condition revealed a Bayes factor of

2.264, formally providing inconclusive evidence for the absence of a difference between B and D, it should be remembered the observed values for B were greater than D, which is the opposite of what would be observed if sensory preconditioning reduced lick cluster sizes, whereas the Immediate condition revealed a Bayes Factor of 5.015, providing support for the absence of an effect.

Experiment 10 replicated the dissociation between direct conditioning and SPC in terms of their impact on consumption scores and lick cluster size; again, direct conditioning influenced both measures whereas SPC only influenced consumption. Experiment 10 also showed that introducing a trace interval between the CS and US produced a similar dissociation in response measures: this manipulation had no impact on consumption scores, but it did influence lick cluster size. The origin of the dissociations observed in conditioning and SPC will now be considered in greater detail.

4.6. General Discussion

Sensory preconditioning has been central to theoretical analyses of learning in nonhuman animals. Here, two type of account of sensory preconditioning were evaluated, associative and propositional, that both assume that after exposure to AB, any responses established to A will be mirrored in the responses elicited by B. I examined whether the two forms of conditioned responses established by direct conditioning to A were reflected in responding generated to B through prior exposure to AB. The conditioned responses examined were the decreases in consumption and lick cluster size produced by pairing flavours with LiCl. In Experiments 7-10, rats consumed less of a flavour that had been directly paired with LiCl than of a flavour which had not; and in all four experiments, the lick cluster sizes elicited by the flavour that had been paired with LiCl were smaller than those elicited by the flavour that had not. This pattern of results was evident across a range of flavour concentrations and doses of LiCl. These results are consistent with previous analyses

of lick cluster size (e.g., Baird, *et al.*, 2005; Dwyer, 2009; Dwyer, *et al.*, 2008) and confirm the fact that directly conditioned flavours produce both avoidance and aversion[3]. In Experiments 7, 8 and 10, after exposure to AB, the reduction in consumption in A resulting from its pairing with LiCl were also mirrored in the consumption of B. Critically, this SPC effect was not accompanied by a change in lick cluster sizes to B in any of the three experiments. That is, there was a clear dissociation between the effects of direct conditioning and those of SPC: Both produced changes in consumption of the test flavours, but only direct conditioning (and not sensory preconditioning) produced a change in lick cluster size.

As I have already noted, the dissociation described above is incongruent with standard, elemental and configural, accounts of SPC which assume that responses established by direct conditioning should transfer to other stimuli through an associative chain or a shared, configural representation. However, before completely rejecting these accounts, it is worth considering the types of arguments that have been proposed to explain why conditioned responses (CR) and unconditioned responses (UR) differ in direct conditioning, while maintaining a stimulus substitution framework. Perhaps the two most common arguments are that the precise form of a response (either CR or UR) depends upon the nature of the stimulus which elicits that response, and that many USs elicit multiple, and sometimes opposing, responses (for reviews, see Mackintosh, 1983; Rescorla & Holland, 1982). While these ideas can explain some of the discrepancies between CRs and URs, neither can explain the differences observed here between directly conditioned responses and those based on SPC. For example, Tolman (1932) noted that particular response components, such as chewing or licking, would not be seen to diffuse auditory or visual stimuli because such stimuli did not provide the stimulus support for those responses (i.e., there was nothing to

[3] All previous studies of licking microstructure in flavour conditioning have used between-subjects designs, and my results confirm these effects using within-subjects designs.

chew or lick). Indeed, when stimuli that were used did support chewing then this response to the CS can be observed (e.g., Boakes, Poli, Lockwood, & Goodall, 1978). However, in Experiments 7-10, both direct conditioning and SPC were based on the same flavoured solutions that were presented in the same way. So, while differences in the response enabling features of the CS and US can explain some discrepancies between CRs and URs, such differences cannot be used to explain the dissociations that were observed in the Experiments 7-10.

The second potential mechanism that has been used to explain the divergence of CRs and URs is the idea of opposing or compensatory responses. According to this analysis, a US elicits multiphasic responses and only some of these become linked to the CS. There is certainly evidence for such responses in many situations, especially those involving pharmacological and aversive stimulation (e.g., Solomon & Corbit, 1974). However, in the current situation, according to both the elemental chain and configural accounts, direct and SPC is mediated by the same pathway (involving either the memories of A or AB in these respective accounts). Consequently, even if LiCl elicited multiphasic responses, there is no mechanism – in these accounts – that would allow the directly conditioned stimuli and those that gain their behavioural tendencies through SPC to be linked to different aspects of these multiphasic responses. In short, while there are explanations of the divergence in the form of CRs and URs in direct conditioning that do not require one to abandon the principle of stimulus substitution[4], these explanations cannot account of the divergence observed here between SPC and direct conditioning.

[4] It should be noted that it has been concluded that a strict application of stimulus substitution is insufficient to explain all aspects of responding following Pavlovian conditioning (Mackintosh, 1983; Pearce, 2008; Rescorla & Holland, 1982).

A detailed consideration of the basis for the dissociation between responses acquired during conditioning and sensory preconditioning will follow in the General Discussion. It is sufficient to note here that the analysis that the results of Experiments 7-10 favour is based on the general idea that conditioning and sensory preconditioning are based on different associative pathways.

Chapter 5: General Discussion

5.1. Summary of Results

5.1.1. Causal Reasoning

Chapter 2 described three experiments in which rats were presented with a series of relationships that, it has been argued, should afford the development of different causal models: a common-cause model (B ← A → F) or a causal-chain model (B → A → F). Experiment 1 revealed that a novel lever press presented at test as an alternative cause of B, lowers responding rates compared to B presented alone. However, an external alternative cause that has been trained has no effect on the responding to B compared to baseline response rates. This pattern of results was evident in both training conditions. Experiment 2 examined the effect of training a lever press as an alternative cause of the test stimulus, B, and this was contrasted with the effect of pressing a novel lever at test. There were no effects of either the trained or the untrained lever at test. However, the rate of responding to the levers was higher in the common-cause group than the causal-chain group. The final experiment using this causal model methodology used solely external cues; that is, they did not require the animal to perform any action. A visual cue (C) was trained as the alternative cause of B, and this was compared to another visual cue (D) which had been pre-exposed during training, but not paired with B. Responding during B was higher following the alternative cause (C) than the unpaired control stimulus (D) in the common-cause group, and this trend was reversed in the causal-chain group. The theoretical implications of these results will be discussed in Section 5.2.1.

5.1.2. Causal binding

Chapter 3 presented a series of experiments using rats' timing behaviour. Rats were trained with an action (a lever press presented on a discrete trial basis) and an auditory cue (tone, clicker, or buzzer) which both predicted the outcome of food after a 5s delay.

Experiment 4 used a 2s clicker (CS) and a lever press (LP), and it was observed that the peak time of responding was the same in the CS condition as the LP condition. The next experiment shortened the length of the CS to a 0.5s tone, and again compared responding to that following a lever press. This comparison revealed no difference in responding between the conditions. The final experiment in Chapter 3 compared two CS conditions with two LP conditions, that is, a tone and a buzzer, each with a duration of 0.8s, and a left and right lever press. These led to one of two outcomes, food or sucrose solution (i.e., CS1 → food, CS2 → sucrose, LP1 → food, LP2 → sucrose). This revealed no differences in the peak rate of responding between the conditions. A devaluation procedure was conducted at the end of Experiment 6, where rats were presented with each lever separately following prefeeding of either food or sucrose solution. The results revealed that rats pressed the lever significantly more for the non-devalued substance than the devalued substance (e.g., if pre-fed food, rats would be more likely to press the lever corresponding to sucrose solution). These results will be further discussed in Section 5.2.2.

5.1.3. Sensory Preconditioning

The final experimental chapter (Chapter 4) investigated sensory preconditioning using compound cues. The stimuli were flavoured solutions (e.g., sucrose and lemon), and the consumption along with the size of licking clusters were taken as response measures. One of these elements (e.g., sucrose) was then given an aversion (lithium chloride, e.g., 10ml/kg), and responding to the directly paired stimulus (e.g., sucrose; A) and the indirectly paired stimulus (e.g., lemon; B) were compared to controls with no aversion (e.g., maltodextrin and salt; C and D). Experiment 7 showed that the directly and indirectly paired stimuli had lower consumption rates compared to the control substances. The directly paired stimulus revealed the same reduction in responding in the lick cluster size data, but the same effect was not observed in the indirectly paired stimulus. This effect was repeated in Experiment 8, which

varied the amount of lithium (5ml/kg, 15ml/kg). Experiment 9 used only direct conditioning, but varied the concentration of the solutions consumed (high concentration of A and C, low concentration of B and D), and the amount of lithium administered (2.5ml/kg or 5ml/kg). This experiment found that A was consumed less than C, and B consumed less than D, and the same pattern of results in the lick cluster data. The final experiment addressed whether there was a difference in responding following immediate or trace conditioning. This used the same procedure as Experiment 7, but one group were provided with lithium immediately following the stimulus (Group Immediate), and the other 30 minutes after the stimulus (Group Trace). The results revealed the standard sensory preconditioning effect for both the immediate and trace groups in the consumption data (A consumed less than C, B consumed less than D). In the lick cluster data, direct conditioning revealed a decrease in lick cluster size of A compared to C for both groups. This difference was larger in Group Immediate. Indirect conditioning revealed no difference between B and D in either group. The theoretical implications of these results will be discussed further in Section 5.2.3.

5.2. Theoretical implications

5.2.1. Causal Reasoning

The results of Experiments 1-3 in Chapter 1 provided little evidence that rats were engaging in a process of causal reasoning: The effects of intervention were as apparent for rats that had received causal-chain training as those given common-cause training; any effects were removed by familiarity with the lever, and were not evident with an explicitly trained external cause. It is thus appropriate to speculate about other explanations of the effects observed, which will be guided by associative analyses. The first step is to consider why, according to associative theorising, rats might respond during B at all. In the causal-chain condition, the most obvious basis for responding to B is that the presentation of B will activate the associative chain B→A→food. It should be clear that for rats given common-

cause training, the basis for responding during B is unlikely to be this associative chain: although A will be able to activate food, B should not be so able to activate A, because the nature of their pairing (A→B) means that any excitatory link from B to A is likely to be relatively ineffective (see Wagner, 1981). So, why do rats given this form of training visit the food well during B? One obvious possibility is based upon mediated conditioning (Dwyer, 2003; Holland, 1983, 1990). When A is paired with B, an association will form that will allow the associatively provoked memory of B to be active on trials where A is later paired with food. This could allow an association for form between the retrieved representation of B and food, thus providing a basis for rats to visit the food well during B.

Given that there are associatively-based accounts to explain why rats given either common-cause or causal-chain training visit the food well when B is presented, the question then becomes: Why is responding to B affected by what takes place immediately before it? I have already considered the possibility of response competition, whereby the tendency to interact with whatever preceded B (in particular a novel lever) would be inconsistent with producing a magazine response. Other things being equal, this mechanism should have equivalent effects regardless of the mechanism supporting the response to B: which is exactly what was observed when the lever was novel in Experiment 1, and previously (Dwyer *et al.*, 2009). In rats that had previously experienced lever presses paired with B, lever pressing did not affect responding to B at test (Experiment 2). But, what of the situation in Experiment 3, where the presentation of C (that had previously signalled B) and D (which had not) had different effects on responding to B depending on whether the rats had received common-cause or casual chain training?

Take first the common-cause condition. What is required is an account for why B elicits greater responding on trials on which it was preceded by C than when it was preceded by D. One possibility is based on the observation that as a result of C→B trials, during

training, the subsequent presentation of C, at test, will activate the memory of B into the same, associatively activated state, as it occupies when it is paired with food on A→food trials. This will not be the case when B is preceded by D. This idea receives direct support from the results of recent studies which used procedures (i.e., second-order conditioning and sensory preconditioning) that are similar to those used in the current experiments. Lin and Honey (2011) demonstrated that the associatively and directly activated representations of a stimulus are distinct and can be associated with different outcomes (see also Lin & Honey, 2010). This implies that there will not be perfect generalisation between the associatively and directly activated representations of a stimulus, and thus any response elicited by one of these will be elicited to only a lesser extent by the other.[5] Obviously, this associatively-based account was developed after the data were collected, and so requires further testing before it can be uncritically accepted. That said, it provides an explanation of why animals approach the food magazine during the presentation of B at all, and also of why this response to B will be modulated by the context in which B appears in some circumstances but not others.

Although there was no significant difference in responding to B as a function of whether it followed C or D in group causal-chain, it is worth briefly considering why the analysis described in the preceding paragraph would not also apply here. In the causal-chain condition, training should have resulted in the formation of a B→A association and an

[5] It might well be asked why similar effects are not seen in Experiment 2, where rats received training where a lever press predicted the presentation of the critical stimulus, and thus might be expected to evoke its associatively activated representation. However, in Experiment 3, the stimuli (A and C) that might evoke the associative representation of B were both externally presented visual cues, and so it would be reasonable to expect good generalisation between the representations of B that were elicited by both stimuli. This will not be the case in Experiment 2, where the associatively activated representation of B that was paired with food was elicited by an external visual cue (A) while on test B was preceded by a lever press.

A→food association; and responding to B at test will involve activation of the associative chain B→A→food as opposed to the form of mediated learning described in the previous paragraph. That is, activation of B by C during test will not mean that it is in the same representational state as during training. Moreover during the test, presenting C, but not D, should associatively activate the memory of B prior to its occurrence. There is a long history of research that has examined the effect of such associative primes (here C) on performance to a target stimulus (B). Most often, it is assumed that the provision of such a prime will have a detrimental effect on the processing of a target stimulus (see Wagner, 1981). Application of this analysis predicts that B should elicit less responding when it is presented after C than when it is presented after D; because B will be less capable of provoking activity in the putative associative chain (see Honey, Good & Manser, 1998; Honey, Hall & Bonardi, 1993). There was some indication that this was the case in Experiment 3.

It is certainly true that there are some differences between experiments conducted in Chapter 2 and those published by Blaisdell *et al.* (2006): for example, Blaisdell *et al.* (2006) used female rats tested in the dark portion of the cycle, while my studies used male rats tested in the light portion of the cycle, and the strain of animals differed (Long-Evans versus hooded Lister; for a further discussion see, Dwyer *et al.*, 2009; Kutlu & Schmajuk, 2012). Differences of this type might have influenced the results of the experiments. However, if rats possess the ability to control their behaviour according to the output of a causal reasoning process, then this should be generally apparent, rather than being restricted to a single strain and sex of rats housed under particular conditions. The fact that the majority of studies fail to find the critical interaction consistent with causal model theory, suggests that the observation of this interaction might be due to things unrelated to causal reasoning. The balance of evidence does not make a compelling *prima facie* case for the existence of causal reasoning mechanisms in the rat, especially as it is possible, at least in principle, for associative

accounts to explain both Blaisdell et al.'s (2006) experiments and other reported data (Burgess, *et al.*, 2012; Dwyer, *et al.*, 2009). For instance, Kutlu and Schmajuk (2012) demonstrated that it was possible to simulate both Blaisdell et al.'s (2006) results and Dwyer et al.'s results (2009) using the attentional-associative SLG model. In overview, this simulation relied on the fact that the lever press forms an inhibitory relationship with food, because the lever press would be followed by the absence of an otherwise expected reinforcer. They also note that their model can explain the lack of an effect of a novel external cue as the "alternative cause" because this will be less salient than the lever press and thus support less inhibitory learning. While these simulations can account for the previously reported data, they do not speak to the effects of pre-training the lever as an alternative cause (as this might reduce the inhibitory learning because there was no food expected during the training phase) or explain how training external cues as alternative causes reverses the observed pattern of results. Regardless, the simulations by Kutlu and Schmajuk (2012) reinforce the view that even the results reported by Blaisdell et al.'s (2006) cannot be taken as unambiguously supporting a causal reasoning account, even if both the previously reported failures of replication, and the results from Chapter 2 were discounted.

5.2.2. Causal binding

The experiments reported in Chapter 3 provided no evidence of causal binding in rats. This conclusion clearly resonates with that reached on the basis of the experimental work reported in Chapter 2. However, rats require a great deal of training before they display accurate timing behaviour. This training greatly exceeded that given to human participants, but the main concern involved in overtraining is that the action may become habitual rather than controlled by a goal-directed process. This was addressed in the devaluation assessment in Experiment 6, which found a significant reduction in lever press responses for the devalued reward. A second concern with extensive training during these experiments is that

rats may initially underestimate the time between a lever press and its outcome, but learn over extensive training that the reward was not until a 5s delay. This was not possible to observe during these experiments because the data gathered in the initial phases of training was not appropriate for a curve fitting procedure.

Although the results obtained here do not support a causal account of behaviour, they are in line with theories of timing, which implicitly assume that actions and observational stimuli are both time markers for the beginning of the interval. For instance, neither SET (Gibbon, 1977) nor LeT (Machado, 1997) make any distinction between timing from an action and external stimulus. Until now this assumption has not been directly assessed, and so the results of Chapter 3 provide support for current animal timing theories. The results are also in line with Wagner's (1981) SOP, which also makes no distinction between actions and external cues.

While the results of Experiments 4-6 might not be surprising in the context of models of timing, in other respects the similarity in timing functions across quite different temporal referents (instrumental responses and Pavlovian CSs) is a striking finding. For example, it has been argued that interventions (in the shape of instrumental responses) and observations (of CSs) differ in their access to the process of causal reasoning, with interventions having a privileged status (Leising *et al.*, 2008). The results of Experiments 4-6 provide no evidence for such privileged access to the mechanisms that underlie timing behaviour in rats, no evidence of causal binding, and thereby join others in suggesting that it is premature to suggest that interventions and observations have a fundamentally different status in the rat (see also Chapter 2).

5.2.3. Sensory Preconditioning

The results of the first two experiments in Chapter 4 demonstrated a standard sensory preconditioning effect in consumption, whereby the directly paired stimulus, A, was

consumed less than its control, C, and the indirectly paired stimulus, B, was consumed less than its control, D. The same pattern of results was found in the directly paired stimuli in the lick cluster data, but the indirectly paired stimulus showed no difference. That is, it seems that the rats avoid drinking much of the indirectly paired stimulus, due to its association with illness, but they drink it in the same way as another stimulus which has no association with illness. This pattern was seen across different LiCl doses. Thus, accounts based on some form of reasoning and some associative analyses are both undermined, as both these accounts would predict that the animals would respond in the same manner in both types of behavioural measures (i.e., B should show a marked aversion in both consumption and lick cluster data). The final experiment of Chapter 4 looked at whether introducing a trace affected the lick cluster data of the directly paired stimulus (i.e., whether introducing a trace affects how the rat responds to A). The critical finding in this experiment revealed that this trace to the directly paired stimulus made the rats respond more like their responding to B in the lick cluster data but not the consumption data.

There are a number of modifications, some more radical than others, that one can make to associative analyses that allow them to explain the results of Chapter 4. The first is based upon the distinction between preparatory and consummatory responses. It has been argued that preparatory responses (those involving approaching the drinking spout) influence the amount (e.g., the total number of licks) but not the form of consumption (e.g., the size of lick clusters); and consummatory responses (directed at the liquid spout) influence both the amount and form of consumption (Dwyer, 2009; Dwyer, *et al.*, 2009). Consummatory conditioning is typically thought to be supported by detailed sensory/perceptual representations of the stimuli involved, while preparatory conditioning relates to more diffuse/gnostic representations (e.g., Dickinson & Balleine, 2002; Konorski, 1967; Wagner & Brandon, 1989). Thus any analysis which suggests that direct conditioning and SPC differ in

the sensory or perceptual specificity of the critical representations would be entirely consistent with the observed data. Within an associative chain analysis of SPC, the memories of B and LiCl are more remote from one another than are those of A and LiCl. As increasing the remoteness of CS and US can reduce the sensory specificity of responding then this might lead to the lack of consummatory responding to B as compared to A (Colwill & Motzkin, 1994; see also, Honey & Hall, 1992). The results of Experiment 10 are consistent with this general analysis, insofar as introducing a trace interval during direct conditioning had no significant effect on consumption but did influence lick cluster size. The configural analysis might also imply differences in representational specificity, insofar as direct conditioning is mediated by activation of a memory of A, but SPC is mediated by the ability of B to activate a memory of AB.

Alternatively, it has been argued that SPC might reflect a form of mediated learning: after exposure to AB, the presentation of B associatively provokes either the elemental memory (A; or the configural memory, AB) into the A2 state and this associatively provoked memory becomes linked to the US (Iordanova *et al.*, 2011). Now, when B is presented at test it will be able to activate a memory of LiCl independently of any direct association that the memory of A has with LiCl (Rescorla & Freberg, 1978; Ward-Robinson, Coutureau, Honey, & Killcross, 2005; Ward-Robinson & Hall, 1996). This separation of representation-mediated conditioning from direct conditioning provides a basis upon which different types of associative knowledge, and response forms, might develop in SPC and standard forms of conditioning. For example, Lin and Honey (2010, 2011) have suggested that animals are able to form distinct associations involving the A1 and A2 states of a given stimulus. In the case under consideration, it must be supposed that what is learned when an associatively activated memory of B is paired with LiCl supports the development of preparatory responses, but not consummatory responses; whereas its directly activated counterpart supports the development

of both. This analysis received further support from the results of Experiment 10, wherein the A2 state was modulated by a trace interval rather than by association. The fact that the dissociation produced by trace conditioning was less profound than that observed in SPC might simply indicate that our trace interval was less effective in generating the A2 state than was SPC.

To conclude this section: The dissociation between the behaviours generated by direct conditioning and SPC undermine conventional associative accounts of SPC that assume stimulus substitution or a propositional account of behaviour. Each of the attempts to capture this dissociation assumes that there is a difference in the nature, and not just strength, of a representation that has been directly activated by its corresponding stimulus and one that has been associatively activated. Further research will be necessary to both establish the generality of the observations presented here and to enhance our understanding of them.

5.3. Future directions

The results of Chapter 2 clearly show that causal model theory does not account for rat behaviour (e.g., Waldmann, *et al*., 2008; Waldmann, *et al*., 2006; Waldmann & Holyoak, 1992). However, causal model theory is only one account of causal learning in humans. As mentioned in Chapter 1, before Blaisdell *et al.*'s (2009) study, most assessments of causal understanding in animals was by using the trap tube task. The adaptation of Waldmann and Hagmayer's (2005) study in rats really was a breakthrough in assessing causal understanding in animals. The methodology employed could easily be used with other species to assess the similarity (or dissimilarity) in cognition between humans and non-human animals. For instance, it may be the case that animals which have a more similar evolutionary background to humans (e.g., chimps) have evolved the capacity to reason in a manner consistent with causal model theory. Assessing different species would enable us to gain an idea of whether this capacity has evolved in animals other than humans. However, as noted in Chapter 1,

Waldmann and Hagmayer's design directly provided the participants with scenarios, meaning the participants did not have to form the causal models through experience. Thus it would also be interesting to observe how human participants would behave given similar training procedures to rats. If this procedure did not produce "reasoning" behaviour in human subjects, it would be necessary to re-evaluate its application to non-human animals.

Chapter 3 reveals no similarity in findings between the human studies of temporal binding and the rat adaptation of those studies. These results have no negative impact on current theories of timing, which already treat observations and interventions similarly, and include no mechanism for causal binding. However, there are clearly many differences in the methodology employed between rats and humans. For instance, the only difference in conditions in the Buehner and Humphreys (2009) study was that in one condition an action is trained as causally related to the outcome, and thus it may be worth assessing whether the methodology used in rats produces the causal binding effect in humans. That is, train humans that two stimuli (an external stimulus and an action) are both followed by an outcome 5s after their offset. As mentioned in the previous paragraph, it is important to assess whether the task produces the desired results in humans. If it does not, again its application to other species would need to be addressed. That said, MacPhail (1982) has eloquently explained that cross-species comparisons are fraught with difficulties due to differences in sensory or motor capacity (amongst many others). So the types of cross-species comparisons noted in the previous two paragraphs would only be the beginning of a genuinely comparative investigation of causal representation and reasoning.

In the discussion of Chapter 3, a caveat with the procedure employed was noted; that the rats may be timing from the lever withdrawal rather than the lever press itself. One potential alternative to the methodology used in Chapter 3 is to leave the lever extended into the chamber. The issue with using this method is that it is likely the rats will press the lever

more than once before the reinforcer is delivered (and thus it will be unclear which lever press caused the delivery of food). That said, it is possible to train rats to wait before pressing a lever again, by not reinforcing multiple presses. Food would be provided 5s after the last LP, but if the rat presses again in that interval, then the timer resets. The control (CS) condition would need to be yoked to the LP condition, such that the distribution of CS-food trials would be the same as the distribution of LP-food trials.

However, this still raises the possibility of providing an external cue to the animals once they have pressed the lever; the lever returning to its original position could act as a time marker. Another possible alternative procedure is to employ the methodology of the bisection task (Church & Deluty, 1977). To assess whether rats underestimate the interval following an action, two conditions would be assessed. In the action condition, rats would be required to pull a chain, and the external cue condition would present a CS. These would each be followed by a long or short delay to a tone, and the rats would be required to respond to a right lever if the delay was long, and a left lever if the delay was short. During the test, the interval between the chain pull, or the light, and the tone would be set to vary between 5 and 10s (intermixed with training trials). The proportion of long responses (i.e., right lever presses) can be plotted for each condition, and a curve fit conducted to assess whether the mean (that is, when the proportion of long responses is at 50%) is earlier for the action condition than for the external cue condition.

Chapter 4 produced perhaps the most thought provoking results. While an account for these results has been suggested (Section 5.2.3), it deserves further exploration. Firstly, it would be interesting to see how rats respond to compound cues at test (e.g., AB, AD, CB, CD; see also, Lin, Dumigan, Dwyer, Good, & Honey, in press). The account developed here suggests that the presence of the directly conditioned cues (presumably in A1) need not

prevent their corresponding A2 states from evoking the US. That is, the sensory preconditioning effects should survive the presence of the directly conditioned stimuli.

5.4. Conclusion

The results from all the chapters in this thesis point to the same conclusion: rats do not represent the causal texture of the environment in the same manner as humans. The notion that animals may not process information like humans has been around since Morgan (1894), who suggested that, as many different species had arisen from evolution, many different minds must have also arisen. Therefore, he argued that to attribute human thought processes to animals without excluding other alternatives is unscientific. This thesis has entertained the possibility that the behaviour of rats might be underpinned by various forms of reasoning; but the experimental results reported here have only served to reinforce the simpler alternatives, rather than ruling them out.

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