



Review

Memory processes in classical conditioning

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Abstract

Classical conditioning provides a rich and powerful method for studying basic learning, memory, and emotion processes in animals. However, it is important to recognize that an animal's performance in a conditioning experiment provides only an indirect indication of what it has learned. Various remembering and forgetting processes, in addition to other psychological processes, may intervene and complicate what investigators can infer about learning from performance. This article reviews the role of context, interference, and retrieval in a number of classical conditioning phenomena (e.g. extinction), and provides an overview of how long-term and short-term memory processes influence behavior as it is studied in classical conditioning.

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Classical conditioning in animals provides a powerful tool for studying the biological processes underlying learning, memory, and emotion. In conditioning, once a conditional stimulus (CS) is associated with an unconditional stimulus (US), a constellation of conditioned responses (CRs) comes to be elicited by the CS.

Given proper control groups for alternative nonassociative processes such as sensitization and pseudoconditioning, the evocation of the CR is a reasonably good index of learning. But it is important to realize that what an animal does in a conditioning experiment is not the same as what it knows. Researchers in behavioral aspects of learning and memory have long separated *learning*, the hypothetical psychological and physical changes in the brain, from *performance*, the manifestation of that change in behavior. The present article selectively reviews the kinds of memory processes

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that separate learning and performance, and thus complicate simple inferences about psychological processes (learning, memory, emotion) from behavior.

The distinction between learning and performance was an important part of the thinking of early learning theorists. For instance, Edward Tolman, one of the most important theorists of the 20th century, made a convincing case for it. For example, in the well-known latent learning experiment, Tolman and Honzik [1] allowed rats to explore a 14-unit T-maze on a series of trials. One group received reward each time it reached the end of the maze, whereas another group did not. Not surprisingly, the rewarded group moved through the maze more efficiently, making fewer errors (entering fewer dead ends) over the first 11 trials. But when the non-rewarded group was then rewarded, they began to move efficiently through the maze beginning on the next trial. According to Tolman and Honzik, the nonrewarded group had been learning about the maze the whole time, even though that learning or knowledge had not been evident in their behavior. The function of reward was not to stamp behavior in, but instead to *motivate* the animal to perform. A motivational function of reward was widely accepted in subsequent theory [2–5]. Learning is not the same as performance. Motivation is required for the translation.

Modern thinking has followed another of Tolman's ideas. He argued, at a time when it was not fashionable to do so, that learning was not the simple attachment of a behavioral response to an environmental stimulus (so-called S-R learning). Instead, the animal represented its experience in some sort of cognitive way. For instance, Tolman [6] claimed that rats learned cognitive maps of the environment. Although the idea that animals learn a literal map is debatable, the cognitive view of what is learned in classical conditioning has become dominant [7,8]. This view assumes that there is a distinction between what is learned and what is manifest in behavior. For example, theorists now suppose that learning involves some sort of encoding of information, storage of that information in memory, and then the retrieval of it. Learning processes thus involve the encoding and storage of information. Performance, on the other hand, depends at least in part on successful retrieval. The current article is mainly interested in considering how memory and memory retrieval processes operate in classical conditioning. An appreciation of these processes is essential for a complete understanding of this deceptively simple learning process. They are methodologically important because they introduce a layer of complexity when inferring an animal's learning or knowledge from behavior or performance.

1. Extinction, context, and interference

Consider extinction, a learning phenomenon that has been investigated in our laboratory for many years. Just as a CS-US pairing comes to evoke responding, subsequently

presenting CS without the US on a number of trials eliminates ('extinguishes') that responding. The study of extinction is interesting in its own right, because something like it must be available to animals in order to allow them to update and modify their behavior in a changing environment. In addition, it has been explicitly linked to various therapies designed to eliminate unwanted cognitions, emotions, and behaviors that themselves depend on learning [9]. It is tempting to suppose that the loss of responding in extinction merely reflects the destruction of the original learning that led to it. But we know this is not the case; the original association is retained, perhaps fully intact. The extinction phenomenon is therefore another place where it is important to understand the distinction between learning (what the animal knows) and performance (what the animal does).

There are several ways to show that the original association is intact after extinction. First, there is 'reinstatement.' In reinstatement, if the US is now presented a few times after extinction is complete, responding will return to the CS when the CS is presented again [10]. One of the main reasons reinstatement occurs is that the animal associates the US with the context (background stimuli typically defined as emanating from the box in which the experiment is conducted) when the US is presented after extinction. When the animal is subsequently tested with the CS in the context, the contextual conditioning triggers responding to the CS. One of the most important types of evidence supporting this view is that the US must be presented in the context in which the CS will be tested. If the animal is presented with the US in an irrelevant context, it does not produce reinstatement [11–15]. Although the contextual conditioning that causes reinstatement is not always evident in behavior directly elicited by the context, the strength of reinstatement correlates with the strength of contextual conditioning when it is measured with sensitive context-preference tests [11,13]. Reinstatement indicates that extinction is not the same as unlearning. Responding to the extinguished CS can return depending on what the animal knows about the context.

A second phenomenon indicating that extinction is not unlearning, and that the CS has become dependent on the context, is the 'renewal effect' [13,16]. In renewal, an animal might receive conditioning trials with the CS in one context (Context A) and then extinction trials in another (Context B). When the animal is then returned to the original context and presented with the CS, responding to the CS recovers (is 'renewed'). Although most studies of renewal have used the ABA design (in which conditioning, extinction, and testing are conducted in Contexts A, B, and A, respectively), other work indicates that renewal can also occur if the contexts are ABC [17] or even AAB [18]. Such results suggest that extinction is especially dependent on the context. It would be a mistake to think, based on the animal's lack of responding in the extinction context, that the original learning is gone.

One of the interesting things about research on the renewal effect is that it suggests that extinction depends more on the context than conditioning does. Although it seems relatively easy to find that extinction performance is lost after a context switch, conditioning performance is rarely lost when the CS is switched to a familiar context, which would minimize possible external inhibition effects created by novel contexts used in some experiments [19–21]. A change of context after conditioning does not attenuate conditioned fear to a CS as measured by conditioned suppression [13,22,23] or by freezing [24], appetitive conditioning [23,25] or taste aversion conditioning [26,27]. The fact that extinction is more context-dependent than conditioning is consistent with the idea that the animal codes extinction as a kind of conditional exception to the rule—one that depends on the current context [28]. When the context is changed, extinction performance disappears and conditioning performance returns.

A related recovery-after-extinction phenomenon is ‘spontaneous recovery’. In this phenomenon, if the experimenter merely allows time to pass after extinction, the extinguished response can recover [29–31]. Just as extinction is more sensitive to context than conditioning, so it is more sensitive to the effects of the passage of time. In fact, we have argued that the passage of time affects extinction precisely because it is a kind of context [32]. Just as the renewal effect indicates that extinction is sensitive to the physical context, spontaneous recovery suggests that it is sensitive to the context provided by time.

Research thus clearly indicates that the current performance elicited by a CS can underestimate what the animal actually ‘knows’ about the CS. The major factor that influences performance after extinction—besides the latent CS–US association—appears to be the current context. How does the context operate? Conditioning theorists have had much to say about this. First, several influential conditioning models assume that the context enters into direct associations with the US, just as a CS might [33–37]. These context-US associations would be expected to summate with the CS–US association to generate performance. Unfortunately, an emphasis on this idea cannot explain details of the results just reviewed [38,39]. For example, it is not consistent with the fact that an extinguished CS is especially sensitive to context-US associations [11,40], and measurable context-US associations do not seem to be necessary for the renewal effect [13].

A second possibility is that the contexts have the properties of ‘occasion setters’ [41] (see [42,43] for reviews). On this view, the context is not merely associated with the CS (or the absence of the CS), but instead selects or activates the CS’s own current association with the US [44]. Thus, the extinction context activates something like the animal’s CS–no US association [44]. It is as if the context determines the current meaning of the ambiguous CS, much as verbal contexts disambiguate the meaning of ambiguous

words. (The word ‘fire’ can mean different things, and evoke different behaviors, depending on whether it is shouted in the movie theater or the shooting gallery.) Interestingly, occasion setters are not generally assumed to elicit performance by themselves, as ordinary CSs might; instead, they mainly operate by modulating performance to other CSs.

The occasion setting mechanism is similar to another possible role of context, namely, to enable retrieval of the CS’s association with the US. As mentioned above, when the animal is outside the extinction context, it might fail to retrieve extinction, which can be taken as a CS–no US association [28]. Consistent with this view, retrieval cues that remind the animal of extinction can abolish the renewal effect [45] and also spontaneous recovery [29,46,47]. On this view, the context and memory retrieval processes are necessary in the translation of knowledge into performance after extinction.

A growing literature on the brain processes involved in extinction is broadly consistent with the behavioral research just summarized (see [48] for one review). For instance, extinction appears to be linked to new brain plasticity mediated by NMDA receptors [49,50] and by L-type voltage-gated calcium channels in the shorter term [51]. One implication is that facilitation of these synaptic processes should help facilitate extinction, which has been shown with administration of an NMDA partial agonist, D-cycloserine [52,53]. It is not known whether facilitated extinction results from deeper extinction learning that is less context-dependent or merely learning that is easier to retrieve in the right context. At the systems level, fear extinction may be linked in part to activity in the infralimbic region of the medial prefrontal cortex [54, but see 55] or to possible GABAergic interneurons in the lateral amygdala [56,57]. A role for hippocampus, an area long thought to be involved in context learning [58] is also suggested by data indicating that the ABC renewal effect is suppressed by inactivation of the hippocampus [59], although the ABA renewal effect is not affected by lesions [60,61]. The pattern may be consistent with the idea that negative occasion setting by Context B, which is presumably a major source of the ABC effect, is especially dependent on the hippocampus [62]. Reinstatement, which depends on direct associations between the context and the US, also depends on an intact hippocampal system in fear conditioning [60,61], though not in appetitive conditioning [63]. The various brain processes are likely to contribute in different ways to the acquisition of new learning in extinction and its suppression of old learning in performance.

2. Long-term memory processes in animal conditioning and learning

It is interesting to note that the extensive literature on long-term memory in animals also reinforces the view that it

is important to distinguish between learning and performance. The word ‘forgetting’ describes a *behavioral* phenomenon in which performance assessed at Time 2 is shown to be inferior to that shown at Time 1, typically the time when the task is originally learned. Although it is tempting to assume that the behavioral phenomenon is explained by the decay or erosion of the original memory trace, we know that this often is not true. For example, it is often possible to reverse forgetting (and apparently recover a forgotten memory) by exposing the animal to a retrieval cue [64]. This sort of result has been taken to mean that forgetting often results from an inability to access or retrieve the target trace (see [65] for some of the relevant neurobiology).

Research on remembering and forgetting is often consistent with Tulving’s Encoding Specificity Principle, which emphasizes the importance of the similarity between background contextual cues present during learning and testing [66]. The general idea, which has been implicitly accepted in the extinction research presented above, is that retrieval depends on a match between the conditions present during learning and the conditions present during testing. In the original research with human participants, the specificity was defined by subtle semantic shadings of target, to-be-remembered words. Tulving and Thomson [66] showed that the memory for words on a list was affected by weakly-associated words that were present at input, which apparently influenced the semantic meaning encoded for the target. Their experiments dramatically showed that these weakly-associated input words were better able to retrieve the target words than were more strongly-associated words that were not present at input. Even though the presence of the strongly-associated words at the test almost certainly generated the target word through free association, the target words were not recognized. Although the initial work manipulated input cues that could profoundly affect the meaning of target words, subsequent work has shown that memory depends on a variety of contextual stimuli, including many external cues [67].

Consistent with this idea, memory for verbal material in humans is often inferior when the room is switched between learning and testing [68]. However, it is often disappointingly difficult to detect this effect [69]. In a recent meta-analysis, Smith and Vela [70] found overall evidence of such context-dependent memory, but suggested that it is sometimes difficult to find owing to a number of other processes that can help support recall at the time of the test. For example, ‘outshining’ refers to the idea that if other cues besides the context can support memory retrieval, and if they are present in both the training and testing context, then they will diminish any context switch effect. Contexts that are not particularly salient also would not support a context switch effect. And humans can also defeat a context switch effect by thinking about (mentally reinstating) the original context when they are tested in a different one [68].

It is worth observing that the weakness of the context switch effect in humans has a parallel in the animal research. As we noted above, a change of context after simple excitatory conditioning usually produces little effect on responding to the CS [13,25]. This sort of result can perhaps be seen as a case of outshining: The animal is responding to a highly salient cue for the US (the CS) that overcomes the absence of contextual support in the changed context. Conditioning theories essentially argue that the CS is a more informative cue than the context [35]. Other experiments in which context switches have been said to impair memory retrieval often have not included an overt CS; for example, investigators may test passive avoidance [71,72], in which the animal refrains from entering a compartment where it had previously been shocked, and thus responds directly to contextual (apparatus) cues. With few cues that can potentially outshine them, it may not be surprising to see an effect of switching the apparatus. Interestingly, in many cases an extant context switch effect can still be attenuated if the animal is given an extra retrieval cue [73–75]—a potential parallel to mental reinstatement in humans. The parallel between the animal and human literatures is even stronger than this. The fact that the context is especially important after extinction is consistent with other parts of the human memory literature, which indicate that context switch effects are similarly easier to detect in interference designs in which the participant learns a conflicting word list in a second phase [68]. Apparently analogous to extinction, the second word list produces context-specific interference with memory for the first.

One important implication of this work, of course, is that forgetting is not necessarily due to the erasure of the learned information. Instead, forgetting may be the result of a retrieval failure caused by a change of context [64]. Forgetting might also be caused by interference emanating from conflicting information learned at an earlier or later point. Interference has a long and distinguished history in human learning theory [76,77]. When first-learned information interferes with memory for second-learned information, we have ‘proactive interference.’ When second-learned information interferes with first-learned information, we have ‘retroactive interference.’ In the domain of animal learning, retroactive interference is represented by several possible paradigms, including extinction or counterconditioning (where Phase 2 interferes with Phase 1). Proactive interference is perhaps represented by phenomena like latent inhibition, in which initial exposure to the CS without the US can interfere with conditioning when the CS and US are subsequently paired.

Interestingly, it has been common in the animal learning tradition to assume that proactive and retroactive interference effects occur because of a failure at the level of learning. As we have already seen, extinction is sometimes thought to result from a destruction of the original learning. Similarly, latent inhibition is often attributed to a failure to learn during the second (conditioning) phase owing, for

example, to the habituation of attention to the CS [33,34]. Nonetheless, extinction and latent inhibition may both follow from performance processes [32,78]. Consistent with this idea, these and other proactive and retroactive interference phenomena are dependent on context (e.g. renewal effects have been obtained in both domains) and time (e.g. spontaneous recovery effects have been reported in both domains) (see [32] for a review). For example, in latent inhibition, if Phase 2 conditioning is assessed after a delay in the latent inhibition paradigm, the conditioned response becomes stronger, as if conditioning had been learned but performance was obstructed by a memory of latent inhibition, which was forgotten over the delay [78,79]. In addition, if preexposure and conditioning are conducted in different contexts, a return to the preexposure context after conditioning is complete can renew latent inhibition performance [80–82]. Once again, it would be a mistake to assume that the performance evident at a particular point in time is a simple product of the CS's associative strength.

Other research supports the idea that memory also depends on internal contexts provided by drugs [83]. In 'state-dependent retention', memory is inferior when there is a mismatch between interoceptive drug state at the time of learning and testing. For example, when fear extinction is conducted while the rat is under the influence of a benzodiazepine (chlordiazepoxide or diazepam), fear is renewed when the animal is subsequently tested in the sober state [84]. Fear extinction can thus be dependent on the drug context. State-dependent retention has important methodological implications for studies of the biological basis of learning and memory, where it is common to give a drug during or soon after training and then test memory in the absence of the drug. Although the drug may often be of interest because it potentially disrupts a biological consolidation process, if memory is being tested without a drug that was otherwise present during learning and storage, then retrieval failure is also a candidate explanation for poor performance during testing. For example, chemicals that are supposed to disable learning and consolidation processes, such as the protein synthesis inhibitor anisomycin and several NMDA antagonists (e.g. MK-801, ketamine, phencyclidine, and CGS 19155) can also generate state-dependent retention effects under some conditions (anisomycin [85], MK-801 [86], the others listed [87]). This means that performance tests in the absence of the chemical might overestimate its impact on learning as opposed to retrieval. Such a possibility highlights the importance of using control conditions in which learning is tested in the presence of the same drugged state; if a drug affects learning or consolidation when given at the time of conditioning, it should later affect performance in either the drugged or the nondrugged state.

Other research suggests that context effects can be created by a variety of contextual cues, including hormones (particularly in non-physiological doses) [88], time of day

[89], and deprivation state [90]. Mood effects have been reported in humans, but, perhaps as we have seen with exteroceptive context effects [69], they can be influenced by a number of other factors that make their effects small and difficult to demonstrate [91]. In animals, there is evidence that stress hormones that could be correlates of emotion can play the role of context in a renewal design. Specifically, Ahlers and Richardson [92] showed that administration of ACTH after passive avoidance extinction renewed avoidance behavior. The idea that ACTH provided a context is consistent with the fact that dexamethasone (which suppresses ACTH production) delivered during conditioning nullified this effect; ACTH needs to be part of the conditioning context to recover behavior after extinction. We have argued that the passage of time itself provides a gradually changing context [32]—as discussed above, spontaneous recovery can be understood this way [29,47]. Certain forgetting phenomena have been suggested as posing a challenge for this type of view [93], although the challenge has been addressed and arguably resolved [27,94]. Ultimately, it is almost universally held that 'context' can be provided by many kinds of cues [64,95,96], and all should therefore be kept in mind.

Our understanding of what kinds of events reactivate memories is not complete. As implied above, one of the main ideas is that presenting aspects of the original training situation is best for reactivating a target memory. Often, an effective cue can be thought of as part of the original context. Thus, the extinction cue in the experiments mentioned earlier [29, 45–47] worked to retrieve extinction because it was coded as part of the extinction context; presentation of even a part of a context can presumably trigger completion of the entire pattern of the context that it has been associated with [97]. As we will discuss in a later section, a CS is often thought to evoke behavior because it retrieves or activates a representation of the US. This implies that the laws of associative learning as developed in research on classical conditioning may be relevant to understanding how retrieval cues are learned and established [28]. However, the effectiveness of different cues at the time of testing might depend further on poorly-understood details about the timing of their presentation with respect to the memory test [98], the duration of their presentation [99], and what other cues are presented along with them [100,101]. In addition, the effectiveness of different cues may wax and wane as the retention interval increases [102]. The area would benefit from more systematic, theory-driven research on the specific factors that enable memory reactivation.

Another complication is that activation of a forgotten memory is not a neutral event, but itself enables further learning. For instance, when forgotten memories are reactivated in a new context, the new context becomes effective at retrieving the memory, as if it has been added to the original training memory [73,75]. (The word 'reactivation' is sometimes meant to imply susceptibility

to further modification.) In addition, events that disrupt memory when they are administered soon after learning can also disrupt memory when they are administered soon after reactivation. For example, Misanin et al. [103] found that exposure to electroconvulsive shock soon after a reactivation treatment made the memory difficult to retrieve. Similar evidence has been produced more recently by Nader et al. [104] who found that introduction of anisomycin likewise abolished a reactivated memory (see also [105] for a related effect of MK-801). The findings suggest that reactivation can make a memory susceptible to disruption again, and that similar neurobiological processes are engaged after initial learning and reactivation, although they say little about the mechanisms of retrieval itself [65]. One danger is to assume that the amnesic effect of any agent (e.g. ECS, anisomycin, or MK-801) is necessarily to abolish reconsolidation. For example, although the delivery of ECS soon after learning was originally thought to abolish consolidation [106], subsequent work suggested that it merely made the memory more difficult to retrieve [107]. Thus, the trace had been encoded after all. We have already seen that anisomycin and MK-801 might affect retrievability of a memory rather than merely disrupting consolidation [85,86]. The point is that if reactivation treatments initiate new learning, an amnesic agent may either interfere with reconsolidation or make the reconsolidated memory more difficult to retrieve. The distinction between learning and performance is all-important.

It is also interesting to note that reactivation treatments that involve exposure to a CS operationally resemble extinction trials. Typically, however, CS exposures that reactivate and engage reconsolidation do not create extinction (the CS improves performance, rather than weakens it, compared to control groups that receive no CS). It is possible that extinction learning, which involves the learning of new conflicting information, simply depends on more exposure to the CS. The relationship between these processes is interesting and important, but poorly understood at present.

3. Short-term memory processes in conditioning

Short-term memory processes have also been important in studies of animal learning. For example, there is a substantial amount of interest in variations of the delayed matching to sample procedure, in which animals are given a sample stimulus at one point in time and then given that stimulus and another a few seconds later, when they are required to respond to the stimulus that matches the sample in order to acquire reinforcement [108]. With typical methods, the pigeon's choice returns to chance when the interval between the presentation of the sample and then the choice stimuli is in the order of many seconds [109]. Short term memory for recent choices similarly influences behavior on the radial maze, where rats choose among a number of baited arms, almost without repeating

themselves, until all the baits have been removed [110]. These literatures provide insights into the nature of short-term memory processes in animals. For example, in both delayed match-to-sample and the radial maze, animals seem able to code short-term information either retrospectively (they remember the previous sample or previous arms that have been visited) or prospectively (they remember the correct upcoming comparison stimulus or arms that are yet to be visited) [111,112]. The implications for studies of classical conditioning have mainly been discussed in studies of occasion setting, where a feature CS informs the subject of whether or not a subsequent target CS will be reinforced [113].

There is also evidence that short-term memory processes are important even in simple cases of classical conditioning. Consistent with thinking about human memory processing, current theory assumes that conditioning requires some processing of the CS and US together in short-term memory after individual conditioning trials for the association to be stored in long-term memory. In a classic experiment, Wagner et al. [114] showed that learning of a particular CS–US relation was damaged in rabbits if a surprising episode was presented within 300 s after each learning trial.

The role of short-term memory in conditioning has figured importantly in the influential theories of Wagner [36,115]. The original formulation of his model, which paralleled models of human information processing [115–117], explicitly proposed that storage of the CS–US association in long-term memory depended on the CS and US being processed ('rehearsed') in short-term memory after each conditioning trial. Importantly, surprising CSs and USs—specifically, those that are not already 'primed,' or currently represented in short-term memory, when they are presented in a trial—were assumed to command more rehearsal than those that are not surprising. Priming can occur either through recent presentation of the event itself or through recent presentation of a retrieval cue associated with the event (which would retrieve the item from long-term memory and put it into short-term memory). Furthermore, consistent with an important characteristic of working memory in humans, the capacity of short-term memory was assumed to be limited. These ideas and assumptions allowed the model to explain an impressive amount of new data ([115] see for review).

In a more recent version of the model, Wagner [36,37, 118] put the ideas in a connectionist framework. In this model, known as the 'sometimes opponent process' model, or 'SOP,' CSs and USs are assumed to have corresponding 'nodes' in memory. The presentation of a CS or US is assumed to activate the node. Initially, the node is activated to a highly active state ('A1'), but this quickly decays to a less active state ('A2'), where it stays a bit longer before returning to the inactive (I) state. (An activated node is essentially one that is represented in short-term memory.) The animal learns about the CS only when it has been put into its maximally active state (A1). If the US node is

simultaneously activated to the same A1 state, then the animal will learn a connection or association between the two. As a consequence of conditioning, the CS is able to activate the US node into the A2 (not the A1) state; this activation ultimately produces a conditioned response.

Notice that if time were to elapse between presentation of the CS and US, the CS could decay from A1 to the less-active A2 state and therefore be unavailable for learning. The theory actually supposes that nodes are composed of many elements that move from A1 to A2 and then Inactivity in a probabilistic fashion. Given these dynamics, the theory explains the results of ‘trace conditioning’ experiments, in which one observes increasingly poor conditioning as the temporal gap between CS offset and US onset increases [119].¹ The US node similarly decays from A1 to A2 to Inactivity after each US presentation, although here the consequences are somewhat different. If the US is in A2 at the time the CS is in A1, an inhibitory CS–US association is learned. This is one account of inhibitory conditioning that can occur in ‘backward conditioning,’ when the CS is presented relatively soon after the offset of the US [120,121].

The dynamics of short-term memory in SOP can go some distance in explaining a number of other interesting effects in conditioning. For example, recent work in our own laboratory has investigated the well-known trial-spacing effect, in which trials that are spaced relatively widely in time produce better conditioning than trials that are massed in time [122–124]. Although a number of explanations of this effect have been proposed [122,125], recent research suggests that massing trials closely together in time may make conditioning inferior because the presentation of CS and US on one trial primes their representations into short-term memory (the A2 state) and makes them less surprising on the next trial [126,127]. The results were not consistent with other perspectives [125].

Interestingly, although it is once again tempting to assume that priming reduces learning, massed training may have its clearest effect on performance. For example, presentation of the CS a few seconds before it is presented again can reduce responding on the second CS presentation after conditioning has already occurred [128]. When conditioning occurs in a within-subject procedure that intermixes short and long intertrial intervals, there may be less responding in the short intertrial intervals [127]; a learning deficit caused by the short intervals should be manifest in behavior at all intervals. And when a CS that has received conditioning in a massed-trial procedure is

presented on trials that are more spaced in time, there may be a significant increase in conditioned responding on the spaced trials [126]. On the other hand, the increase in responding does not reach the level in animals that have received spaced trials throughout training [126,129]. This result might suggest that massed trials also have an enduring effect on learning, although it is also possible that the animals trained with massed trials and tested with spaced ones might not generalize perfectly between trials presented on the two schedules. The evidence suggests that recent presentations of the conditioning events can generate a short-term suppressive effect on performance and might also influence learning [130].

Of course, priming in short-term memory can only account for the effects of trial massing within the range of short intertrial intervals. In our appetitive conditioning experiments in which rats are given pairings of 10- or 30-s CSs with food pellet USs, priming effects are clearly evident when the trials are separated by 60 s, but not when they are separated by 240 s [126,127]. It is worth noting that additional spacing of trials beyond 240 s can have additional positive effects on conditioning [131]. This fact implies that additional mechanisms do contribute to trial-spacing effects (see [122] for review). Our own research suggests that long intertrial intervals may create better conditioning because they allow extinction of contextual cues that receive conditioning during CS–US pairings [131]. These otherwise compete with (or block [132]), conditioning of the CS. Quantitative conditioning models suppose exactly this process [33–35]. SOP gives the process psychological flesh by arguing that conditioned contextual cues would block CS conditioning because they activate the US node to A2, and thus reduce the surprisingness of the US when it is paired with the US on trial $n+1$; extinction in long intertrial intervals weakens the context’s ability to put the US in A2.

To summarize the trial-distribution findings, very short intertrial intervals can hurt conditioning because they prime the CS and US in short-term memory, making them less likely to command performance and/or learning on the next trial. At longer intertrial intervals, the context becomes less likely to retrieve the representation of the US (and perhaps the CS) and therefore allows better conditioning. Several psychological mechanisms thus play a role in trial-spacing effects. But a productive way to conceptualize them is to emphasize the influence priming effects in short-term memory during conditioning.

4. Conditioned stimuli as retrieval cues

Our discussion to this point has accepted the idea that once conditioning has occurred, the CS functions as a retrieval cue that activates a US node or representation. The idea has received direct empirical support. For example, Rescorla [133] conditioned fear in rats by pairing a light CS

¹ SOP also allows other mechanisms to contribute to the trace conditioning deficit. If the US occurs alone at the end of a very long gap (‘trace interval’), it could be associated with contextual cues that are present at the time [154]. Contextual conditioning would reduce any possible conditioning of the CS, because it would allow the context to activate (prime) the US node to the A2 state, reducing its surprisingness and therefore causing blocking [130].

with a loud noise (created by a klaxon). In a subsequent phase, he presented the klaxon repeatedly by itself to habituate fear to it. During a third phase, he presented the CS alone. At this point, the CS elicited less fear than it did in a control group that did not receive habituation to the noise. Rescorla [133] argued that habituation had modified the rat's memory representation of the noise; when the CS was then presented again, it activated a less frightening US representation. Other experiments revealed a corresponding inflation effect [134]: rats that received fear conditioning with a weak-intensity shock US showed augmented fear of the CS if they were then exposed to more intense shocks. The argument is thus that the response evoked by a CS depends in part on the status of the animal's US representation. A CS may generate behavior at least in part because it serves as a retrieval cue for that representation.

These findings have been extended in a number of ways. For instance, Holland [135] has presented striking evidence that the rat forms a rich representation of the US, and that responding to the CS depends on the status of that representation. In the basic experiment, rats receive pairings of different tone CSs with differently-flavored sucrose solutions. When one of these is then separately paired with illness (which creates a conditioned aversion to the flavor), the rat exhibits less appetitive responding to the paired tone during final tests. Remarkably, as if the tone evoked an almost palpable image of the flavor it was associated with, rats reacted to novel combinations of tones in a way that paralleled what they had separately learned about the corresponding combinations of flavors. In addition, the effect of taste-aversion revaluation of the US appears to be less noticeable when conditioning involves a more extended number of CS–US pairings, as if conditioned responding becomes more 'automatic,' rather than representation-mediated, with extended exposure to the conditioning procedure.

Related findings have been reported in operant conditioning, where animals learn to associate a behavior (such as lever pressing) with a reinforcer instead of a CS with a US. In this case, there is a similar literature indicating that the animal associates one event (the response) with a representation of the outcome. In a simple experiment, if a rat is trained to lever-press for a food pellet, separate pairings of the pellet and illness will condition an aversion to the pellet—and will also cause the rat to suppress its performance of the operant when it is tested in extinction [136,137]. As in classical conditioning, the current amount of responding depends on the strength of a hypothetical association and the extent to which the animal currently values the outcome. Interestingly, extended training in which the rat has many response–outcome pairings can decrease the sensitivity of the response to the devaluation procedure [138, but see 139]), once again suggesting that behavior has become 'automatized,' i.e. more habit-like and less dependent on the current status of the representation of the reinforcer [135].

Also importantly, how the animal currently values the representation of the outcome in instrumental learning depends upon a subtle process known as 'incentive learning,' [137,140,141]. For example, Balleine [142] trained rats to lever-press for food pellets while they were satiated and then tested lever pressing in extinction. At this time, the rats were either hungry or not. Hunger had no effect on the level of responding. But if the rats had been given a separate opportunity to eat the pellets while they were hungry, hunger strongly increased responding in the extinction test. The animal needed to learn about the effect of food on hunger. Hunger thus motivates behavior in a subtle way; the organism will perform an action while hungry if the behavior has been associated with a particular reinforcer, and if the reinforcer has been associated with something like amelioration of the hunger state.

The evidence thus suggests that animals form relatively rich representations as a result of conditioning. One effect of a CS is to retrieve a representation of the US. Consistent with this, conditioned responding (or performance) is a product of the animal's knowledge of a CS–US or response–reinforcer association and how the animal currently values the US. The assignment of value, especially in the operant situation, involves motivational as well as memory processes, which brings us full circle to the ideas of Tolman that we mentioned at the start of this article.

5. Summary and conclusion

This short review has only scratched the surface of the complexity of classical conditioning. In addition to describing some of the many roles for memory in conditioning, we have repeatedly noted that any study of conditioning must always distinguish learning from performance. We have seen that extinction does not depend on unlearning; erased performance at the end of extinction does not reflect erased knowledge. Instead, performance after extinction is a product of the animal's knowledge of two conflicting associations, and how the context selects between them. Similarly, forgetting in studies of long-term memory does not necessarily result from erasure or decay, but instead often results from reduced access, either due to retrieval failure or interference. Once again, the context plays a role. Short-term memory effects are also evident in classical conditioning; in this case, recent presentations of events can temporarily suppress performance and learning. Finally, the idea that a CS functions as a retrieval cue provides another link between conditioning and memory, as well as a further separation between what the animal does and what it knows.

Although we have focused primarily on memory processes, a number of other factors also matter in the translation of learning into performance. We have barely mentioned motivational factors (see [143] for one recent

review). In addition, the qualitative nature of the CS can influence the type of conditioned response one observes [144,145]. So can the duration of the CS: CSs that end in the US after a relatively brief interval can evoke conditioned responses that are qualitatively different from those evoked by CSs that end in the US after a longer interval [146–149]. In any of these cases, it would be a mistake to make inferences about learning from measurements of a single response, a point that has been made in other animal learning paradigms as well [150–152]. The behavioral product of classical conditioning is not a single, monolithic conditioned response. Conditioning instead engages whole behavior systems, or sets or constellations of behaviors that are evolutionarily adapted to help optimize the organism's interaction with the US [153]. Although the topic of behavior systems is beyond the scope of the present article, it further reminds us that classical conditioning involves a sophisticated and surprisingly varied set of behavioral processes that will always come into play whenever conditioning is used as a tool to study the brain.

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References

- [1] Tolman EC, Honzik CH. Introduction and removal of reward, and maze performance in rats. *U Cal Pub Psych* 1930;4:257–75.
- [2] Hull CL. *A behavior system; an introduction to behavior theory concerning the individual organism*. New Haven: Yale University Press; 1952.
- [3] Spence KW. *Behavior theory and conditioning*. New Haven: Yale University Press; 1956.
- [4] Mowrer OH. *Learning theory and behavior*. New York: Wiley; 1960.
- [5] Rescorla RA, Solomon RL. Two-process learning theory: relationships between Pavlovian conditioning and instrumental learning. *Psychol Rev* 1967;74:151–82.
- [6] Tolman EC. Cognitive maps in rats and men. *Psychol Rev* 1948;55: 189–208.
- [7] Hulse SH, Fowler H, Honig WK. *Cognitive processes in animal behavior*. Hillsdale, NJ: Erlbaum; 1978.
- [8] Rescorla RA. Pavlovian conditioning: it's not what you think it is. *Am Psychol* 1988;43:151–60.
- [9] Barlow DH. *Anxiety and its disorders: the nature and treatment of anxiety and panic*, 2nd ed. New York: Guilford Press; 2002.
- [10] Rescorla RA, Heth CD. Reinstatement of fear to an extinguished conditioned stimulus. *J Exp Psychol Anim Behav Process* 1975;1: 88–96.
- [11] Bouton ME. Differential control by context in the inflation and reinstatement paradigms. *J Exp Psychol Anim Behav Process* 1984; 10:56–74.
- [12] Bouton ME, Bolles RC. Role of conditioned contextual stimuli in reinstatement of extinguished fear. *J Exp Psychol Anim Behav Process* 1979;5:368–78.
- [13] Bouton ME, King DA. Contextual control of the extinction of conditioned fear: tests for the associative value of the context. *J Exp Psychol Anim Behav Process* 1983;9:248–65.
- [14] Frohardt RJ, Guarraci FA, Bouton ME. The effects of neurotoxic hippocampal lesions on two effects of context after fear extinction. *Behav Neurosci* 2000;114:227–40.
- [15] Wilson A, Brooks DC, Bouton ME. The role of the rat hippocampal system in several effects of context in extinction. *Behav Neurosci* 1995;109:828–36.
- [16] Bouton ME, Bolles RC. Contextual control of the extinction of conditioned fear. *Learn Motiv* 1979;10:445–66.
- [17] Bouton ME, Brooks DC. Time and context effects on performance in a Pavlovian discrimination reversal. *J Exp Psychol Anim Behav Process* 1993;19:165–79.
- [18] Bouton ME, Ricker ST. Renewal of extinguished responding in a second context. *Anim Learn Behav* 1994;22:317–24.
- [19] Penick K, Solomon PR. Hippocampus, context and conditioning. *Behav Neurosci* 1991;105:611–7.
- [20] Devenport L. Sampling behavior and contextual change. *Learn Motiv* 1989;20:97–114.
- [21] Gisquet-Verrier P, Alexinsky T. Does contextual change determine long-term forgetting? *Anim Learn Behav* 1986;14:349–58.
- [22] Hall G, Honey RC. Contextual effects in conditioning, latent inhibition, and habituation: associative and retrieval functions of contextual cues. *J Exp Psychol Anim Behav Process* 1989;15: 232–41.
- [23] Kaye H, Mackintosh NJ. A change of context can enhance performance of an aversive but not of an appetitive conditioned response. *Q J Exp Psychol B* 1990;42:113–34.
- [24] Harris JA, Jones ML, Bailey GK, Westbrook RF. Contextual control over conditioned responding in an extinction paradigm. *J Exp Psychol Anim Behav Process* 2000;26:174–85.
- [25] Bouton ME, Peck CA. Context effects on conditioning, extinction, and reinstatement in an appetitive conditioning preparation. *Anim Learn Behav* 1989;17:188–98.
- [26] Rosas JM, Bouton ME. Renewal of a conditioned taste aversion upon return to the conditioning context after extinction in another one. *Learn Motiv* 1997;28:216–29.
- [27] Rosas JM, Bouton ME. Context change and retention interval can have additive, rather than interactive, effects after taste aversion extinction. *Psychon Bull Rev* 1998;5:79–83.
- [28] Bouton ME. Conditioning, remembering, and forgetting. *J Exp Psychol Anim Behav Process* 1994;20:219–31.
- [29] Brooks DC, Bouton ME. A retrieval cue for extinction attenuates spontaneous recovery. *J Exp Psychol Anim Behav Process* 1993;19: 77–89.
- [30] Pavlov IP. *Conditioned reflexes*. Oxford, UK: Oxford University Press; 1927.
- [31] Robbins SJ. Mechanisms underlying spontaneous recovery in autoshaping. *J Exp Psychol Anim Behav Process* 1990;16:235–49.
- [32] Bouton ME. Context, time, and memory retrieval in the interference paradigms of Pavlovian learning. *Psychol Bull* 1993;114:80–99.
- [33] Mackintosh NJ. A theory of attention: variations in the associability of stimuli with reinforcement. *Psychol Rev* 1975;82:276–98.
- [34] Pearce JM, Hall G. A model for Pavlovian conditioning: variations in the effectiveness of conditioned but not unconditioned stimuli. *Psychol Rev* 1980;87:332–52.
- [35] Rescorla RA, Wagner AR. A theory of Pavlovian conditioning: variations in the effectiveness of reinforcement and nonreinforcement. In: Black AH, Prokasy WK, editors. *Classical conditioning II: current research and theory*. New York: Appleton-Century-Crofts; 1972. p. 64–99.
- [36] Wagner AR. A model of automatic memory processing in animal behavior. In: Spear NE, Miller RR, editors. *Information processing*

- in animals: memory mechanisms. Hillsdale, NJ: Erlbaum; 1981. p. 5–47.
- [37] Wagner AR, Brandon SE. Evolution of a structured connectionist model of Pavlovian conditioning (AESOP). In: Klein SB, Mowrer RR, editors. *Contemporary learning theories: Pavlovian conditioning and the status of traditional learning theory*. Hillsdale, NJ: Erlbaum; 1989. p. 149–89.
- [38] Bouton ME. Context and retrieval in extinction and in other examples of interference in simple associative learning. In: Dachowski L, Flaherty CF, editors. *Current topics in animal learning: brain, emotion, and cognition*. Hillsdale, NJ: Erlbaum; 1991. p. 25–53.
- [39] Pearce JM, Bouton ME. Theories of associative learning in animals. *Annu Rev Psychol* 2001;52:111–3.
- [40] Bouton ME, King DA. Effect of context on performance to conditioned stimuli with mixed histories of reinforcement and nonreinforcement. *J Exp Psychol Anim Behav Process* 1986;12: 4–15.
- [41] Bouton ME, Swartzentruber D. Analysis of the associative and occasion-setting properties of contexts participating in a Pavlovian discrimination. *J Exp Psychol Anim Behav Process* 1986;12:333–50.
- [42] Holland PC. Occasion setting in Pavlovian conditioning. In: Bower G, editor. *The psychology of learning and motivation*, vol. 28. Orlando, FL: Academic Press; 1992. p. 69–125.
- [43] Swartzentruber D. Modulatory mechanisms in Pavlovian conditioning. *Anim Learn Behav* 1995;23:123–43.
- [44] Bouton ME, Nelson JB. Mechanisms of feature-positive and feature-negative discrimination learning in an appetitive conditioning paradigm. In: Schmajuk NA, Holland PC, editors. *Occasion setting: associative learning and cognition in animals*. Washington, DC: American Psychological Association; 1998. p. 69–112.
- [45] Brooks DC, Bouton ME. A retrieval cue for extinction attenuates response recovery (renewal) caused by a return to the conditioning context. *J Exp Psychol Anim Behav Process* 1994;20:366–79.
- [46] Brooks DC. Recent and remote extinction cues reduce spontaneous recovery. *Q J Exp Psychol* 2000;53B:25–58.
- [47] Brooks DC, Palmatier MI, Garcia EO, Johnson JL. An extinction cue reduces spontaneous recovery of a conditioned taste aversion. *Anim Learn Behav* 1999;27:77–88.
- [48] Myers KM, Davis M. Behavioral and neural analysis of extinction. *Neuron* 2002;36:567–84.
- [49] Falls WA, Miserendo MJD, Davis M. Extinction of fear-potentiated startle: blockade by infusion of an NMDA antagonist into the amygdala. *J Neurosci* 1992;12:853–63.
- [50] Santini E, Muller RU, Quirk GJ. Consolidation of extinction learning involves transfer from NMDA-independent to NMDA-dependent memory. *J Neurosci* 2001;21:9009–17.
- [51] Cain C, Blouin A, Barad MG. L-type voltage-gated calcium channels are required for extinction, but not for acquisition or expression, of conditioned fear in mice. *J Neurosci* 2002;22:9113–21.
- [52] Ledgerwood L, Richardson R, Cranney J. Effects of D-cycloserine on extinction of conditioned freezing. *Behav Neurosci* 2003;117:341–9.
- [53] Walker DL, Ressler KJ, Lu KT, Davis M. Facilitation of conditioned fear extinction by systemic administration or intra-amygdala infusions of D-cycloserine as assessed by fear potentiated startle in rats. *J Neurosci* 2002;22:2343–51.
- [54] Millad MR, Quirk GJ. Neurons in medial prefrontal cortex signal memory for fear extinction. *Nature* 2002;420:70–4.
- [55] Gewirtz JC, Falls WA, Davis M. Normal conditioning inhibition and extinction of freezing and fear-potentiated startle following electrolytic lesions of medial prefrontal cortex in rats. *Behav Neurosci* 1997;111:712–26.
- [56] Goossens KA, Maren S. Pretraining NMDA receptor blockade in the basolateral complex, but not the central nucleus, of the amygdala prevents savings of the conditional fear. *Behav Neurosci* 2003;117: 738–50.
- [57] Harris JA, Westbrook RF. Evidence that GABA transmission mediates context-specific extinction of learned fear. *Psychopharmacology* 1998;140:105–15.
- [58] Holland PC, Bouton ME. Hippocampus and context in classical conditioning. *Curr Opin Neurobiol* 1999;9:195–202.
- [59] Corcoran KA, Maren S. Hippocampal inactivation disrupts contextual retrieval of fear memory after extinction. *J Neurosci* 2001;21: 1720–6.
- [60] Frohardt RJ, Guarraci FA, Bouton ME. The effects of neurotoxic hippocampal lesions on two effects of context after fear extinction. *Behav Neurosci* 2000;114:227–40.
- [61] Wilson A, Brooks DC, Bouton ME. The role of the rat hippocampal system in several effects of context in extinction. *Behav Neurosci* 1995;109:828–36.
- [62] Holland PC, Lamoureux JA, Han JS, Gallagher M. Hippocampal lesions interfere with Pavlovian negative occasion setting. *Hippocampus* 1999;9:143–57.
- [63] Fox GD, Holland PC. Neurotoxic hippocampal lesions fail to impair reinstatement of an appetitively conditioned response. *Behav Neurosci* 1998;112:255–60.
- [64] Spear NE. *The processing of memories: forgetting and retention*. Hillsdale, NJ: Erlbaum; 1978.
- [65] Sara SJ. Retrieval and reconsolidation: toward a neurobiology of remembering. *Learn Mem* 2000;7:73–84.
- [66] Tulving E, Thomson DM. Encoding specificity and retrieval processes in episodic memory. *Psychol Rev* 1973;80:352–73.
- [67] Godden DR, Baddeley AD. Context-dependent memory in two natural environments: on land and underwater. *Br J Psychol* 1975;66: 325–31.
- [68] Smith SM. Remembering in and out of context. *J Exp Psychol Hum Learn Mem* 1979;5:460–71.
- [69] Smith SM. Environmental context-dependent memory. In: Davies GM, Thomson DM, editors. *Memory in context: context in memory*. New York: Wiley; 1988. p. 13–34.
- [70] Smith SM, Vela E. Environmental context-dependent memory: a review and meta-analysis. *Psychon Bull Rev* 2001;8:203–20.
- [71] Land CL, Riccio DC. Nonmonotonic changes in the context shift effect over time. *Learn Motiv* 1998;29:280–7.
- [72] Zhou Y, Riccio DC. Manipulation of components of context: the context shift effect and forgetting of stimulus attributes. *Learn Motiv* 1996;27:400–7.
- [73] Gordon WC, McCracken KM, Dess-Beech N, Mowrer RR. Mechanisms for the cueing phenomenon: the addition of the cueing context to the training memory. *Learn Motiv* 1981;12:196–211.
- [74] Mowrer RR, Gordon WC. Effects of cueing in an ‘irrelevant’ context. *Anim Learn Behav* 1983;11:401–6.
- [75] Witttrup M, Gordon WC. Alteration of training memory through cueing. *Am J Psychol* 1982;95:497–507.
- [76] McGeoch JA. Forgetting and the law of disuse. *Psychol Rev* 1932; 39:352–70.
- [77] Postman L, Underwood BJ. Critical issues in interference theory. *Mem Cogn* 1973;1:19–40.
- [78] Kraemer PJ, Spear NE. The effect of nonreinforced stimulus exposure on the strength of a conditioned taste aversion as a function of retention interval: Do latent inhibition and extinction involve a shared process? *Anim Learn Behav* 1992;20:1–7.
- [79] Aguado L, Symonds M, Hall G. Interval between preexposure and test determines the magnitude of latent inhibition: implications for an interference account. *Anim Learn Behav* 1994;22:188–94.
- [80] Bouton ME, Swartzentruber D. Slow reacquisition following extinction: context, encoding, and retrieval mechanisms. *J Exp Psychol Anim Behav Process* 1989;15:43–53.
- [81] Maren S, Holt W. The hippocampus and contextual memory retrieval in Pavlovian conditioning. *Behav Brain Res* 2000;110: 97–108.

- [82] Westbrook RF, Jones ML, Bailey GK, Harris JA. Contextual control over conditioned responding in a latent inhibition paradigm. *J Exp Psychol Anim Behav Process* 2000;26:157–73.
- [83] Overton DA. Contextual stimulus effects of drugs and internal states. In: Balsam PD, Tomie A, editors. *Context and learning*. Hillsdale, NJ: Erlbaum; 1985. p. 357–84.
- [84] Bouton ME, Kenny FA, Rosengard C. State-dependent fear extinction with two benzodiazepine tranquilizers. *Behav Neurosci* 1990;104:44–55.
- [85] Radyushkin KA, Anokhin KV. Recovery of memory in chicks after disruption during learning: the reversibility of amnesia induced by protein synthesis inhibitors. *Neurosci Behav Physiol* 1999;29:31–6.
- [86] Harrod SB, Flint RW, Riccio DC. MK-801 induced retrieval, but not acquisition, deficits for passive avoidance conditioning. *Pharmacol Biochem Behav* 2001;69:585–93.
- [87] Jackson A, Koek W, Colpaert FC. NMDA antagonists make learning and recall state-dependent. *Behav Pharmacol* 1992;3:415–21.
- [88] Costanzo DJ, Riccio DC, Kissinger S. State-dependent retention produced with estrus in rats. *Physiol Behav* 1995;57:1009–11.
- [89] Holloway FA, Wansley RA. Multiple retention deficits at periodic intervals after active and passive avoidance learning. *Behav Biol* 1973;9:1–14.
- [90] Davidson TL. The nature and function of interoceptive signals to feed: toward integration of physiological and learning perspectives. *Psychol Rev* 1993;100:640–57.
- [91] Eich E. Searching for mood dependent memory. *Psychol Sci* 1995;6: 67–75.
- [92] Ahlers ST, Richardson R. Administration of dexamethasone prior to training blocks ACTH-induced recovery of an extinguished avoidance response. *Behav Neurosci* 1985;99:760–4.
- [93] Riccio DC, Richardson R, Ebner DL. Memory retrieval deficits based upon altered contextual cues: a paradox. *Psychol Bull* 1984; 96:152–65.
- [94] Bouton ME, Nelson JB, Rosas JM. Stimulus generalization, context change, and forgetting. *Psychol Bull* 1999;125:171–86.
- [95] Bouton ME. Context, ambiguity, and unlearning: sources of relapse after behavioral extinction. *Biol Psychiatry* 2002;52:976–86.
- [96] Spear NE, Riccio DC. *Memory: phenomena and principles*. Boston, MA: Allyn and Bacon; 1994.
- [97] Rudy JW, O'Reilly RC. Contextual fear conditioning, conjunctive representations, pattern completion, and the hippocampus. *Behav Neurosci* 1999;113:867–80.
- [98] Gordon WC, Smith GJ, Katz DS. Dual effects of response blocking following avoidance learning. *Behav Res Ther* 1979;17:479–87.
- [99] Miller JS, Jagielo JA, Spear NE. Differential effectiveness of various prior-cuing treatments in the reactivation and maintenance of memory. *J Exp Psychol Anim Behav Process* 1991;17:249–58.
- [100] Arnold HM, Spear NE. Order and duration of stimuli are important determinants of reactivation. *Anim Learn Behav* 1993;(2):391–8.
- [101] Gisquet-Verrier P. Coherence of retrieval cues, rather than additivity, determines prior cuing effectiveness in the rat. *Anim Learn Behav* 1992;20:382–92.
- [102] Gisquet-Verrier P, Dekeyne A, Alexinsky T. Differential effects of several retrieval cues over time: evidence for time dependent reorganization of memory. *Anim Learn Behav* 1989;17:394–408.
- [103] Misanin JR, Miller RR, Lewis DJ. Retrograde amnesia produced by electroconvulsive shock after reactivation of a consolidated memory trace. *Science* 1968;160:554–5.
- [104] Nader K, Schafe GE, Le Doux JE. Fear memories require protein synthesis in the amygdala for reconsolidation after retrieval. *Nature* 2000;406:722–6.
- [105] Przybylski J, Sara SJ. Reconsolidation of memory after its reactivation. *Behav Brain Res* 1997;84:241–6.
- [106] Duncan CP. The retroactive effect of electroshock on learning. *J Comp Physiol Psychol* 1949;42:32–44.
- [107] Miller RR, Springer AD. Retrieval failure induced by electroconvulsive shock: reversal with dissimilar training and recovery agents. *Science* 1972;177:628–30.
- [108] Blough DS. Delayed matching in the pigeon. *J Exp Anal Behav* 1959;2:151–60.
- [109] Roberts WA, Grant DS. Studies of short-term memory in the pigeon using delayed matching to sample procedure. In: Medin DL, Roberts WA, Davis RT, editors. *Processes of animal memory*. Hillsdale NJ: Erlbaum; 1976. p. 79–112.
- [110] Olton DS, Samuelson RJ. Remembrance of places passed: spatial memory in rats. *J Exp Psychol Anim Behav Process* 1976;2:97–116.
- [111] Cook RG, Brown MF, Riley DA. Flexible memory processing by rats: use of prospective and retrospective information in the radial maze. *J Exp Psychol Anim Behav Process* 1985;11:453–69.
- [112] Zentall TR, Urcioli PJ, Jackson-Smith P, Steirn JN. Memory strategies in pigeons. In: Dachowski L, Flaherty CF, editors. *Current topics in animal learning: brain, emotion, and cognition*. Hillsdale, NJ: Erlbaum; 1991. p. 119–39.
- [113] Swartzentruber D. Perspectives on modulation: modulator- and target-focused views. In: Schmajuk NA, Holland PC, editors. *Occasion setting: associative learning and cognition in animals*. Washington, DC: American Psychological Association; 1998. p. 167–97.
- [114] Wagner AR, Rudy JW, Whitlow JW. Rehearsal in animal conditioning. *J Exp Psychol* 1973;97:407–26.
- [115] Wagner AR. Expectancies and the priming of STM. In: Hulse SH, Fowler H, Hoing WK, editors. *Cognitive aspects of animal behavior*. Hillsdale, NJ: Erlbaum; 1978. p. 177–210.
- [116] Atkinson RC, Shiffrin RM. Human memory: a proposed system and its control processes. In: Spence KW, Spence JT, editors. *The psychology of learning and motivation*, vol. 2. New York: Academic Press; 1968.
- [117] Wagner AR. Priming in STM: an information processing mechanism for self-generated or retrieval-generated depression in performance. In: Tighe TJ, Leaton RN, editors. *Habituation: perspectives from child development, animal behavior and neurophysiology*. Hillsdale, NJ: Erlbaum; 1976. p. 95–128.
- [118] Wagner AR, Brandon SE. A componential theory of Pavlovian conditioning. In: Mowrer RR, Klein SB, editors. *Handbook of contemporary learning theories*. Hillsdale, NJ: Erlbaum; 2001. p. 23–64.
- [119] Smith MC, Coleman SR, Gormezano I. Classical conditioning of the rabbit's nictitating membrane response at backward, simultaneous and forward CS-US intervals. *J Comp Physiol Psychol* 1969;69: 226–31.
- [120] Maier SF, Rapaport P, Wheatly KL. Conditioned inhibition and the UCS-CS interval. *Anim Learn Behav* 1976;4:217–20.
- [121] McNish KA, Betts SL, Brandon SE, Wagner AR. Divergence of conditioned eyeblink and conditioned fear in backward Pavlovian training. *Anim Learn Behav* 1997;25:43–52.
- [122] Barela PB. Theoretical mechanisms underlying the trial-spacing effect in Pavlovian fear conditioning. *J Exp Psychol Anim Behav Process* 1999;25:177–93.
- [123] Holland PC. Trial and intertrial durations in appetitive conditioning in rats. *Anim Learn Behav* 2000;28:121–35.
- [124] Terrace HS, Gibbon J, Farrell L, Baldock MD. Temporal factors influencing the acquisition and maintenance of an autoshaped keypeck. *Anim Learn Behav* 1975;3:53–62.
- [125] Gallistel CR, Gibbon J. Time, rate, and conditioning. *Psychol Rev* 2000;107:289–344.
- [126] Bouton ME, Sunsay C. Importance of trials versus accumulating time across trials in partially reinforced appetitive conditioning. *J Exp Psychol Anim Behav Process* 2003;29:62–77.
- [127] Sunsay C, Stetson L, Bouton ME. Memory priming and trial spacing effects in appetitive Pavlovian learning. *Learn Behav* 2004; 32:220–9.

- [128] Pfautz PL, Wagner AR. Transient variations in responding to Pavlovian conditioned stimuli have implications for the mechanisms of 'priming'. *Anim Learn Behav* 1976;4:107–12.
- [129] Lattal KM. Trial and intertrial durations in Pavlovian conditioning: issues of learning and performance. *J Exp Psychol Anim Behav Process* 1999;25:433–50.
- [130] Barnet RC, Grahame NJ, Miller RR. Trial spacing effects in Pavlovian conditioning: a role for local context. *Anim Learn Behav* 1995;23:340–8.
- [131] Sunsay C, Bouton ME. A theoretical investigation of the trial spacing effect in appetitive Pavlovian conditioning; In Preparation.
- [132] Kamin LJ. Predictability, surprise, attention, and conditioning. In: Campbell BA, Church RM, editors. *Punishment and aversive behavior*. New York: Appleton–Century–Crofts; 1969. p. 279–96.
- [133] Rescorla RA. Effect of US habituation following conditioning. *J Comp Physiol Psychol* 1973;82:137–43.
- [134] Rescorla RA. Effect of inflation of the unconditioned stimulus value following conditioning. *J Comp Physiol Psychol* 1974;86:101–6.
- [135] Holland PC. Event representation in Pavlovian conditioning: image and action. *Cognition* 1990;37:105–31.
- [136] Colwill RM, Rescorla RA. Associative structures in instrumental learning. In: Bower GH, editor. *The psychology of learning and motivation*, vol. 20. New York: Academic Press; 1986. p. 55–104.
- [137] Dickinson A. Expectancy theory in animal conditioning. In: Klein SB, Mowrer RR, editors. *Contemporary learning theories: Pavlovian conditioning and the status of traditional learning theory*. Hillsdale, NJ: Erlbaum; 1989. p. 279–308.
- [138] Adams CD. Variations in the sensitivity of instrumental responding to reinforcer devaluation. *Q J Exp Psychol B* 1982;34:77–98.
- [139] Colwill RM, Rescorla RA. The role of response reinforcer associations increases throughout extended instrumental training. *Anim Learn Behav* 1988;16:105–11.
- [140] Balleine BW. Incentive processes in instrumental conditioning. In: Mowrer RR, Klein SB, editors. *Handbook of contemporary learning theories*. Hillsdale, NJ: Erlbaum; 2001. p. 307–66.
- [141] Dickinson A, Balleine B. Motivational control of goal-directed action. *Anim Learn Behav* 1994;22:1–18.
- [142] Balleine BW. Instrumental performance following a shift in primary motivation depends on incentive learning. *J Exp Psychol Anim Behav Process* 1992;18:236–50.
- [143] Dickinson A, Balleine B. The role of learning in the operation of motivational systems. In: Pashle H, Gallistel R, editors. *Steven's handbook of experimental psychology. Learning, motivation, and emotion*, vol. 3. New York: Wiley; 2002. p. 497–533.
- [144] Holland PC. Conditioned stimulus as a determinant of the form of the Pavlovian conditioned response. *J Exp Psychol Anim Behav Process* 1977;3:77–104.
- [145] Timberlake W, Grant DL. Auto-shaping in rats to the presentation of another rat predicting food. *Science* 1975;190:690–2.
- [146] Akins CK. Effects of species-specific cues and the CS–US interval on the topography of the sexually conditioned response. *Learn Motiv* 2000;31:211–35.
- [147] Akins CK, Domjan M, Gutiérrez G. Topography of sexually conditioned behavior in male Japanese quail (*Coturnix japonica*) depends on the CS–US interval. *J Exp Psychol Anim Behav Process* 1994;20:199–209.
- [148] Holland PC. CS–US interval as a determinant of the form of Pavlovian appetitive conditioned responses. *J Exp Psychol Anim Behav Process* 1980;6:155–74.
- [149] Vandercar DH, Schneiderman N. Interstimulus interval functions in different response systems during classical discrimination conditioning in rabbits. *Psychon Sci* 1967;9:9–10.
- [150] Wilkie DM, Spetch ML. Pigeon's delayed matching to sample errors are not always due to forgetting. *Behav Anal Lett* 1981;1:317–23.
- [151] Roberts WA, Grant DS. Interaction of sample and comparison stimuli in delayed matching to sample with the pigeon. *J Exp Psychol Anim Behav Process* 1978;4:68–82.
- [152] Roitblat HL, Harley HE. Spatial delayed matching-to-sample performance by rats: learning, memory, and proactive interference. *J Exp Psychol Anim Behav Process* 1988;14:71–82.
- [153] Timberlake W. Motivational modes in behavior systems. In: Mowrer RR, Klein SB, editors. *Handbook of contemporary learning theories*. Hillsdale, NJ: Erlbaum; 2001. p. 155–209.
- [154] Odling-Smee FJ. Background stimuli and the inter-stimulus interval during Pavlovian conditioning. *Q J Exp Psychol A* 1975;27:387–92.