Part I INTRODUCTION

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Classical conditioning: data and theories

During classical (or Pavlovian) conditioning, human and animal subjects change the magnitude and timing of their conditioned response (CR), as a result of the contingency between the conditioned stimulus (CS) and the unconditioned stimulus (US).

In this chapter we briefly describe results of a number of classical conditioning paradigms that are discussed in detail in different chapters of the book (see Schmajuk, 2008a, 2008b). Then we introduce different types of learning theories. Finally, we present a number of computational models of classical conditioning.

Classical conditioning data

A. *Excitatory conditioning*

1. Acquisition. After a number of CS–US pairings, the CS elicits a conditioned response (CR) that increases in magnitude and frequency.
2. Partial reinforcement. The US follows the CS only on some trials, and might lead to a lower conditioning asymptote.
3. Generalization. A CS_2 elicits a CR when it shares some characteristics with a CS_1 that has been paired with the US.
4. US- and CS-specific CR. The nature of the CR is determined not only by the US, but also by the CS.

B. *Inhibitory conditioning*

1. Conditioned inhibition. Stimulus CS_2 acquires inhibitory conditioning with CS_1 reinforced trials interspersed with, or followed by, CS_1–CS_2 nonreinforced trials.

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Nestor Schmajuk
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2. Extinction of conditioned inhibition. Inhibitory conditioning is extinguished by CS_2–US presentations, but not by presentations of CS_2 alone.
3. Differential conditioning. Stimulus CS_2 acquires inhibitory conditioning with CS_1 reinforced trials interspersed with CS_2 nonreinforced trials.
4. Contingency. A CS becomes inhibitory when the probability that the US will occur in the presence of the CS, p(US/CS), is smaller than the probability that the US will occur in the absence of the CS (p[US/noCS]).

C. *Preexposure effects*

1. Latent inhibition (LI). Preexposure to a CS followed by CS–US pairings retard the generation of the CR.
2. Context preexposure. Preexposure to a context facilitates the acquisition of fear conditioning.
3. US–preexposure effect. Presentation of the US in a training context, prior to CS–US pairings, retards production of the CR.
4. Learned irrelevance. Random exposure to the CS and the US retards conditioning even more than combined latent inhibition and US preexposure.

D. *Compound conditioning*

1. Relative validity. Conditioning to *X* is weaker when training consists of reinforced *XA* trials alternated with *XB* nonreinforced trials, than when training consists of *XA* trials alternated with *XB* trials each type reinforced half of the time.
2. Blocking. Conditioning to CS_1–CS_2 following conditioning to CS_1 results in a weaker conditioning to CS_2 than that attained with CS_1–CS_2–US pairings.
3. Unblocking by increasing the US. Increasing the US during CS_1–CS_2 conditioning increases responding to the blocked CS_2.
4. Unblocking by decreasing the US. Responding to CS_2 can be increased by decreasing the US during CS_1–CS_2 conditioning.
5. Overshadowing. Conditioning to CS_1–CS_2 results in a weaker conditioning to CS_2 than that attained with CS_2–US pairings.
6. Potentiation. Conditioning to CS_1–CS_2 results in a stronger conditioning to CS_2 than that attained with CS_2–US pairings.
7. Backward blocking. Conditioning to CS_1 following conditioning to CS_1–CS_2 results in a weaker response to CS_2 than that attained with CS_1–CS_2–US pairings.

8. Overexpectation. Reinforced CS_1–CS_2 presentations following independent reinforced CS_1 and CS_2 presentations, result in a decrement in their initial associative strength.
9. Superconditioning. Reinforced CS_1–CS_2 presentations following inhibitory conditioning of CS_1 increase CS_2 excitatory strength compared with the case when it is trained in the absence of CS_1.

E. *Recovery from compound conditioning*

1. Recovery from latent inhibition. Presentation of the US in the context of preexposure and conditioning results in renewed responding to the preexposed CS.
2. Recovery from overshadowing. Extinction of the CS_1 results in increased responding to the overshadowed CS_2.
3. Recovery from forward blocking. Extinction of the blocker CS_1 results in increased responding to the blocked CS_2.
4. Recovery from backward blocking. Extinction of the blocker CS_1 results in increased responding to the blocked CS_2.

F. *Extinction*

1. Extinction. When CS–US pairings are followed by presentations of the CS alone, or by unpaired CS and US presentations, the CR decreases.
2. External disinhibition. Presenting a novel stimulus immediately before a previously extinguished CS might produce renewed responding.
3. Spontaneous recovery. Presentation of the CS after some time after the subject stopped responding might yield renewed responding.
4. Renewal. Presentation of the CS in a novel context might yield renewed responding.
5. Reinstatement. Presentation of the US in the context of extinction and testing might yield renewed responding.
6. Reacquisition. CS–US presentations following extinction might result in faster or slower reacquisition.
7. Partial reinforcement extinction effect (PREE). Extinction is slower following partial than following continuous reinforcement.

G. *Nonlinear combinations of multiple stimuli*

1. Positive patterning. Reinforced CS_1–CS_2 presentations intermixed with nonreinforced CS_1 and CS_2 presentations result in stronger responding to CS_1–CS_2 than to the sum of the individual responses to CS_1 and CS_2.

2. Negative patterning. Nonreinforced CS_1–CS_2 presentations intermixed with reinforced CS_1 and CS_2 presentations result in weaker responding to CS_1–CS_2 than to the sum of the individual responses to CS_1 and CS_2.

H. Occasion setting

1. Simultaneous feature-positive discrimination. Reinforced simultaneous CS_1–CS_2 presentations, alternated with nonreinforced presentations of CS_2, result in stronger responding to CS_1–CS_2 than to CS_2 alone. In this case, CS_1 gains a strong excitatory association with the US.
2. Serial feature-positive discrimination. Reinforced successive CS_1–CS_2 presentations, alternated with nonreinforced presentations of CS_2, result in stronger responding to CS_1–CS_2 than to CS_2 alone. In this case, CS_1 acts as an occasion setter.
3. Simultaneous feature-negative discrimination. Nonreinforced simultaneous CS_1–CS_2 presentations, alternated with reinforced presentations of CS_2, result in weaker responding to CS_1–CS_2 than to CS_2 alone. In this case, CS_1 gains a strong inhibitory association with the US.
4. Serial feature-negative discrimination. Nonreinforced successive CS_1–CS_2 presentations, alternated with reinforced presentations of CS_2, result in weaker responding to CS_1–CS_2 than to CS_2 alone. In this case, CS_1 acts as an occasion setter.

I. Temporal properties

1. Interstimulus interval (ISI) effects. Conditioning is negligible with short ISIs, increases dramatically at an optimal ISI, and gradually decreases with increasing ISIs.
2. Intertrial interval (ITI) effects. Conditioning to the CS increases with longer ITIs.
3. Timing of the CR. The CR peak tends to be located around the end of the ISI.
4. Temporal specificity of blocking. Blocking is observed when the blocked CS is paired in the same temporal relationship with the US as the blocking CS.
5. Temporal specificity of occasion setting. A serial feature-positive discrimination is best when the feature–target interval during testing matches the training interval.

J. Combination of multiple conditioning events

1. Sensory preconditioning. When CS_1–CS_2 pairings are followed by CS_1–US pairings, presentation of CS_2 generates a CR.

2. Second-order conditioning. When CS_1–US pairings are followed by CS_1–CS_2 pairings, presentation of CS_2 generates a CR.

Learning theories

Some classical conditioning theories stress the importance of mechanisms that act at the time of the presentation of the CS and the US. These theories assume that the association between events CS_i and CS_k, $V_{CSi,\ CSk}$, represents the *prediction* that the CS_i will be followed by CS_k (Dickinson, 1980). Neural network, or connectionist theories frequently assume that the association between CS_i and CS_k is represented by the efficacy of the synapses, $V_{\mathrm{CS}i,\ \mathrm{CS}k}$, that connect a presynaptic neural population excited by CS_i with a postsynaptic neural population that is excited by CS_k (event k might be another CS, or the US). When CS_k is the US, this second population controls the generation of the conditioned response (CR).

Following Hebb's (1949) ideas, changes in synaptic strength, $V_{\mathrm{CS}i,\ \mathrm{CS}k}$, might be described by $\Delta V_{\mathrm{CS}i,\ \mathrm{CS}k} = f(\mathrm{CS}_i) f(\mathrm{CS}_k)$, where $f(\mathrm{CS}_i)$ represents the presynaptic activity, and $f(\mathrm{CS}_k)$ the postsynaptic activity. Different $f(\mathrm{CS}_i)$ and $f(\mathrm{CS}_k)$ functions have been proposed. Learning rules for $V_{\mathrm{CS}i,\ \mathrm{CS}k}$ either assume variations in the effectiveness of CS_i, $f(\mathrm{CS}_i)$, the US, $f(\mathrm{CS}_k)$ (Dickinson & Mackintosh, 1978), or both.

Variations in the effectiveness of the CS during learning

Attentional theories assume that the effectiveness of CS_i to form CS_i–US associations (associability) depends on the magnitude of the "internal representation" of CS_i. In neural network terms, attention may be interpreted as the modulation of the CS representation that activates the presynaptic neuronal population involved in associative learning. Attentional theories include Makintosh's (1975), Grossberg's (1975) and Pearce and Hall's (1980) theories.

Variations in the effectiveness of the US during learning

A popular rule, proposed independently in psychological (Rescorla & Wagner, 1972) and neural network (Widrow & Hoff, 1960) domains, has been termed the "delta" rule. The delta rule describes changes in the synaptic connections between the two neural populations by way of minimizing the squared value of the difference between the output of the population controlling the CR generation, and the US. According to the "simple" delta rule, CS_i–US associations are changed until the difference between the US intensity and the "aggregate prediction" of the US computed upon all CSs present at a given moment, $(\mathrm{US} - \Sigma_j V_{\mathrm{CS}j,\mathrm{US}} \mathrm{CS}_j)$, is zero. The term $(\mathrm{US} - \Sigma_j V_{\mathrm{CS}j,\mathrm{US}} \mathrm{CS}_j)$ can be interpreted as the effectiveness of the US to become associated with the CS.

Schmajuk and DiCarlo (1992) introduced a model (the SD model) that, by employing a "generalized" delta rule (also known as backpropagation, see Rumelhart, Hinton & Williams, 1986) to train a layer of hidden units that *configure* simple CSs, is able to solve exclusive-or problems, and hence, negative patterning.

Variations in the effectiveness of both the CS and the US during learning

In order to account for a wider range of classical conditioning paradigms, some theories have combined variations in the effectiveness of both the CS and the US. For example, Frey and Sears (1978) proposed a model of classical conditioning that assumed variations in the effectiveness of both the CS and the US: $f(\text{CS}_i)$ is modulated by $V_{i,\text{US}}$. Wagner (1978) suggested that CS_i–US associations are determined by (a) $f(\text{US}) = (\text{US} - \Sigma_j V_{j,\text{US}}\text{CS}_j)$ as in the Rescorla–Wagner model; and (b) $f(\text{CS}_i) = (\text{CS}_i - V_{i,\text{CX}}\text{CX})$, where CX represents the context, and $V_{i,\text{CX}}$ the strength of the CX–CS_i association. Other theories that incorporate changes in the effectiveness of both the CS and the US include Wagner's (1981) sometimes opponent process (SOP) theory, Schmajuk, Lam and Gray's (1996) attentional–associative theory, Le Pelley's (2004) hybrid model, and Harris's (2006) elementary model.

Performance theories

Some classical conditioning theories stress the importance of mechanisms that act during performance to control the generation of the CR. Examples of this approach are Miller's comparator hypothesis (e.g. Miller & Schachtman, 1985), Wagner's (1981) SOP model, Schmajuk, Lam and Gray's (1996) attentional–associative model, and Harris's (2006) elementary model.

CS–US and CS–CS associations and decision processes during performance

The comparator hypothesis (Miller & Schachtman, 1985; Miller & Matzel, 1988; Denniston *et al.*, 2001; Stout & Miller, 2007) suggests that the magnitude of the CR is determined by a comparator that uses the CS–CS and CS–US associations of the CSs present at a given time as inputs.

CS–CS associations and inference generation during performance

During classical conditioning, animals learn to expect (predict) that a CS is followed by another CS, or by the US. Tolman (1932) proposed that multiple expectancies (predictions) can be integrated into larger units, through a reasoning process called inference. One simple example of inference formation is sensory preconditioning (see Bower & Hilgard, 1981, page 330). As summarized above, sensory preconditioning consists of a first phase in which two conditioned stimuli, CS_1 and CS_2, are paired together in the absence of the US. In a second phase, CS_1 is paired with the US. Finally, when CS_2 is presented alone, it generates a CR: the animal has inferred that CS_2 predicts the US. Tolman hypothesized that a large number of expectancies can be combined into a cognitive map (see Chapters 2, 7 and 16).

Dickinson (1980) suggested that knowledge can be represented in declarative or procedural form. Whereas in the declarative form knowledge is represented as a description of the relationships between events (knowing that), in the procedural form, knowledge is represented as the prescription of what should be done in a given situation (knowing how). Examples of declarative knowledge are classical CS–CS associations (CS_1 precedes CS_2) or CS–US (CS_2 precedes the US) associations. An example of procedural knowledge is the operant *S*–*R* association (if *S* is present, then do *R*). Dickinson indicated that declarative, but not procedural, knowledge can be integrated through inference rules.

By including CS–CS associations, some models of classical conditioning are able to generate inferences and, therefore, to describe sensory preconditioning. For instance, Gelperin, Hopfield and Tank (1985; Gelperin, 1986) proposed an autoassociative recurrent network capable of describing stimulus–stimulus associations during classical conditioning. The network can simulate first- and second-order conditioning, extinction, sensory preconditioning and blocking in the terrestrial slug, *Limax maximus*. Schmajuk (1987) proposed a dual memory architecture that incorporates an autoassociative nonrecurrent network capable of cognitive mapping in classical conditioning. The network separately computes CS–CS and CS–US predictions, and combines them to generate new expectancies. For instance, if CS(*A*) predicts (is associated to) CS(*B*), and CS(*B*) predicts the US, the network *infers* that CS(*A*) also predicts the US. Schmajuk defined first-order predictions as the prediction of the US by CS(*B*), and higher-order predictions as the predictions involving a chain of two or more predictions. The network describes complex classical conditioning paradigms such as sensory preconditioning, second-order conditioning, compound conditioning and serial-compound conditioning. Similarly, the models presented by Schmajuk and Moore (1988, 1989) and Schmajuk, Lam and Gray (1996) are able to describe sensory preconditioning and second-order conditioning.

Computational models of classical conditioning

As suggested by Hinzman (1991), the inherent unreliability of verbal intuitive reasoning for relating hypotheses and experimental results favors theories that provide precise quantitative descriptions. Furthermore, only formal models can be simultaneously examined at different levels. At the behavioral level, simulated behavioral results are compared with experimental data describing behavior. At the neuroanatomical level, interconnections among neural elements in the model are compared with neuroanatomical data, and the model performance is compared with animal performance after lesioning. At the computational level, simulated activity of the neural elements of the model is compared with the activity of single neuron or neural population activity. At the neurophysiological level, model performance is compared with animal performance after inducing long-term changes (e.g. lesions) or short-term changes (e.g. drug infusions) in different brain areas.

Below, we review in detail some computational models of classical conditioning that have been applied to a number of the conditioning paradigms described before. Some other models (e.g. Brandon, Vogel & Wagner, 2000; Kruschke, 2001; Pearce, 1994) are described later in the book, when they are relevant to the experimental results being discussed.

The Rescorla–Wagner (1972) model

In their classic article, Rescorla and Wagner (1972) indicated that the impetus for their new theoretical model was not new data which clearly disconfirmed existing theories, but rather the accumulation of a pattern of data which appeared to invite a more integrated account. The salient pattern of data the authors referred to was a set of observations involving Pavlovian conditioning with compound CSs. The central notion of the theory was that organisms only learn when the actual value of the US differs from its expected value. By proposing the novel principle that this expected value of the US is computed as a linear combination of the associative strength of all active CSs, the effect of reinforcement or nonreinforcement on the associative strength of a CS depends upon the existing associative strength, not only of that CS, but also of other CSs concurrently present.

Rescorla and Wagner (1972) proposed to formalize the basic idea of their theory by modifying Hull's (1943) account of the growth of habit-strength (stimulus–response associations), as described by Bush and Mosteller's (1955) linear operator. In the Rescorla–Wagner (RW) model, variations in the strength of the CS–US association, $V_{i,\mathrm{US}}$, are given by $\Delta V_{i,\mathrm{US}} = \alpha_i \beta_{\mathrm{US}}(\lambda_{\mathrm{US}} - B_{\mathrm{US}})$, where α_i represents the salience of CS$_i$, β_{US} represents the learning rate parameter corresponding to the US, and B_{US} is the linear combination of the prediction of the US by all active CSs. B_{US} is given by $B_{\mathrm{US}} = \Sigma_j V_{j,\mathrm{US}}$. By this equation, CSs compete to gain association with the US. The conditioned response (CR) was assumed to be proportional to B_{US}.

As observed by Sutton and Barto (1981), a rule similar to the RW equation had been described in the neural network field by Widrow and Hoff (1960). This rule, termed the "delta" rule (Rumelhart, Hinton & Williams, 1986), describes changes in the CS–US associations by way of minimizing the squared value of the difference between the predicted and observed values of the US. Most interestingly, as indicated by Duda and Hart (1973), the rule is able to solve simultaneous systems of equations; a power that provides a different perspective of the processes that take place during classical conditioning.

The RW model correctly described many Pavlovian conditioning phenomena such as acquisition and extinction of conditioned excitation, partial reinforcement, conditioned inhibition, overshadowing, blocking, unblocking by increasing the US strength, overprediction, generalization, US–preexposure effect and contingency effects. The success of the model in making specific correct predictions, inaugurated the modern era of experimental psychology.

In spite of its significant achievements, the RW model was unable to describe several aspects of classical conditioning including (a) the effects of temporal parameters, such as stimulus duration, interstimulus intervals (ISI) or intertrial intervals (ITI); (b) Pavlovian paradigms whose solution require a nonlinear combination of the prediction of the US by all active CSs, such as negative patterning; (c) conditioned inhibition not being extinguished by presentations of the inhibitory CS alone; (d) latent inhibition; (e) backward blocking; and (f) the recovery from blocking and overshadowing.

The Van Hamme and Wasserman (1994) version of the Rescorla and Wagner (1972) model

Van Hamme and Wasserman (1994) offered a modified version of the Rescorla and Wagner (1972) model that is able to explain some of the results mentioned above. Van Hamme and Wasserman (1994) proposed that the association of a CS with the US decreases when the CS is absent ($\Delta V_{i,\text{US}} < 0$), instead of staying constant, as in the original model ($\Delta V_{i,\text{US}} = 0$ because $\alpha_i = 0$). The model can explain the effects of extinction of the companion CS overshadowing and blocking.

Dickinson and Burke (1996) observed that the Van Hamme and Wasserman (1994) rule did not specify when an absent CS was allowed to decrease its association with the US. Following previous suggestions (Chapman, 1991; Markman, 1989; Tassoni, 1995), they indicated that the expectation of an absent CS, via its (within-compound) association with a present CS, could serve that purpose.

The Van Hamme and Wasserman (1994) version of the Rescorla–Wagner (1972) rule is able to describe that (a) extinction of the blocking CS results in the recovery of the response to the blocked CS (Blaisdell *et al.*, 1999); (b) extinction of the overshadowing CS results in the recovery of the response to the overshadowed CS (Matzel *et al.*, 1985); but cannot explain (c) extinction of the context following latent inhibition (LI) results in the recovery of the response