Learning and conditioning
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Chapter preview

When you have read this chapter, you should be able to

- understand what is a theory of learning, and why you should be interested in developing a good theory
- understand how humans and animals learn about the relationships between the events in their environment
- detect instances of Pavlovian and instrumental conditioning in everyday life
- evaluate the strengths and weaknesses of some of the major learning theories
- understand that many different types of learning, such as Pavlovian and instrumental conditioning, acquisition of causal and predictive relations, categorization, and spatial learning, appear to be subject to the same learning principles

Developing a theory of learning

You probably have some ideas about what learning is. You may think of the many years spent at school and the many hours you have spent reading books and learning things. But think not only about how you acquired your school knowledge, but also about how you first learned to walk, to find your way back home when on an unknown street or in an unknown city, to predict a potentially dangerous situation, to relate with other people, to show emotions when it is appropriate to show them and to hide them otherwise. You might also think about how we learn our phobias and our preferences, how we learn the value of money, or how we learn appropriate behaviour for different situations (and inappropriate behaviour as well).

How does learning take place? Answering this question has motivated psychologists to study the learning process, to conduct experiments and to develop theories that should help us understand how we learn. That is, no matter how distinct the many things that we learn are, there must be something fundamental to all of these situations that allows learning to take place. Running experiments on learning and developing a theory of learning is simply trying to answer that question. If you are curious about it, as many psychologists have been, you should try to run your own experiments and to develop a theory of learning that could improve the current ones. You can start by imagining a simple theory that could explain the basic learning experiments which will be described below. Once you have your own theory, compare it with that developed by your classmates, find the pros and cons of each of those theories, and try to reach an agreement on one or two theories that appear to explain the examples of learning that are provided here as well as any other example of learning that you could think of. Then, compare your theory with those that other psychologists have developed. You will probably find that you arrived at many of the ideas that psychologists have proposed; you will also find that some of those ideas have been found problematic, and that many of those ‘faulty’ theories are still a matter for discussion at the present time. If possible, when you finish the chapter, try to find out ways in which you could improve the theories presented here, find further readings that will help you improve your theory, and run experiments that would test whether or not you are correct. Trying to solve the riddle of learning can be fun.

While trying to develop a simple theory of learning, you may want to think not only about how people learn but also about how other animals learn. You have probably heard about Pavlov’s dog, which salivated upon hearing a tone that had previously been presented as a signal for food, or about Thorndike’s cats, which learned how to escape from the puzzle boxes that Thorndike constructed to study how they did this. These are good instances of animal learning, but non-human animals also learn a great deal of other things. Finding their way home, relating with people and with other animals, differentiating friends from strangers, asking for a walk, differentiating edible from non-edible foods, how (and where) to obtain food, how to predict danger and how to escape from it, and so on. These things that animals learn are not too different from the ones that we learn. Additionally, we learn more. But the basic idea that most learning theorists have entertained is that it is just a matter of quantity, not quality. You may or may not agree on this assumption, but that again is part of the excitement of studying learning. If you do not agree with the idea that the basic learning principles are general principles that apply across species, you should try to run experiments and find good arguments to challenge this general view, and develop a more satisfactory one. You must know that even though the psychology of learning has made great improvements over the years, and even though we know a lot more now than such pioneers as Pavlov and Thorndike knew when they started the experimental study of learning about 100 years ago, there is still much room for improvement, new knowledge, ideas, experiments and new and better theories.

Associative theories

Even if you do not agree with the idea of a general learning process that can suffice to explain all forms of animal and human learning, let us agree, at least for the time being, that there must exist a very simple mechanism underlying most types of learning, something that allows learning to occur in different situations and species — something that simply allows learning to take place.

Many of the psychologists who study learning have agreed that the most basic learning mechanism which underlies most types of learning must be an associative mechanism. That is, something that allows the animal (or human) to associate the mental representation of one event with the mental representation of another event (see Dickinson 1980). These two events could be two environmental stimuli (S) which occur close in time and space, such as when Stimulus 1 (S1) is immediately followed by Stimulus 2 (S2). For example, chil-
children usually learn to associate black sky (S1) with a storm (S2) after they experience some pairings of black skies followed by storms. In this case, the children learn an association between their mental representation of these two stimuli, and the adaptive value of such learning is that once they have formed such S1→S2 association, they will be able to predict when a storm is about to occur. But of course, such learning of S→S associations would be of much help if it were not complemented by an additional type of learning that would allow us to learn what we should do after we have made the prediction (e.g. how to protect ourselves from the storm). Thus, not only do we need to form associations between environmental stimuli, but also we need to form associations between our behaviour and its consequences. For example, if we see a black sky and we remain outdoors, we will associate the black sky (S1) with the storm (S2) but we will also learn to associate our behaviour (staying outdoors) with a rather unpleasant outcome (getting wet and cold in the storm). In addition, we can also learn that a different behaviour (e.g. going indoors) produces a nicer outcome (staying dry and warm). When we learn an association between the representation of our own response (R) and that of the outcome (O) that it produces, we speak of having acquired an R→O association.

Thus, after acquiring many of those simple associations, we are able to better adapt and adjust our behaviour to our changing environment. Learning S→S associations allows us to predict our environment, and learning of R→O associations allows us to better adapt our behaviour to our environment. The main purpose of research in learning has been to analyse how those associations are acquired and used. The rest of this chapter presents a summary of some of the research which has been conducted on S→S and R→O associations.

Stimulus–stimulus associations

Pavlovian or classical conditioning

Let us start by a very well known experiment. A hungry dog receives food from time to time and every single presentation of the food is preceded by a tone that signals that the food is about to be presented. After several trials in which the dog is exposed to the tone–food relation, the dog will salivate upon hearing the tone even if the food is not presented. This was demonstrated by Ivan Pavlov (1927) and has become the best known example of classical (or Pavlovian) conditioning, which is a form of stimulus–stimulus (S→S) learning.

Note that what the dog learned here is very similar to our storm example above, in which children learned to predict storms after being exposed to several trials in which a black sky was followed by a storm. In Pavlov’s experiment, the dog can easily learn to predict when food will be available, if it pays attention to the tone–food relationship. Interestingly, once the dog has learned that the tone predicts food, the dog will salivate upon hearing the tone. This is what Pavlov called the conditioned response (CR). Why? Because it was a response that was conditional on learning that the tone would be followed by food. No dog would salivate upon hearing the tone unless it had previously learned that the tone would be followed by food. Thus, such salivary responding to the tone was conditioned (i.e. learned).

Although in the storm example above we did not mention the occurrence of a conditioned response (CR), acquisition of a black sky–storm association also produces CRs when we see a black sky. In that case, the CRs do not take the form of salivary responses, of course, but may take the form of fear responses. The child who has already learned to predict storms may feel fear upon observing a black sky (S1). Thus, CRs can take very different forms (e.g. salivation, fear, sexual arousal, and many others); they occur when the organism perceives S1, and S1 predicts the occurrence of an S2 which is biologically significant (e.g. food, storm, sex, water, sources of pain, drugs, illness). By contrast, if S1 predicts an S2 which is biologically irrelevant, conditioned responses will not be observed even though this does not mean that an S1→S2 association has not been learned. For example, if we hear a sequence of two numbers, S1 and S2, a few times (e.g. 2-5, 2-5, 2-5, 2-5, 2-5) we can perfectly predict S2 (5) upon hearing S1 (2) but we will not give a conditioned response. Similarly, we can learn an association between the name of a person (S1) and a phone number (S2), but this association will not normally produce a conditioned response. Thus, it is important to remember that although the occurrence of a CR indicates that an association has been acquired (as in the dog’s example above), the absence of a CR does not mean that learning has not taken place: there are many S→S associations that are not ordinarily expressed in conditioned responses.

The term classical or Pavlovian conditioning should be reserved, therefore, for a certain type of S→S learning. In particular, the type of S→S learning that involves stimuli which are biologically significant. According to
Pavlov (1927), in order to obtain a conditioned response, S2 must be a biologically significant stimulus. For example, food, sex, water, electric shock or any other stimuli that would elicit responses unconditionally, that is, without any prior learning, are biologically significant stimuli. For this reason, in Pavlovian conditioning, S2 is generally called the unconditioned stimulus (US), because it does not require any special conditions in order to produce a response. For example, food produces salivation without any prior learning, or, if the US were an electric shock, it would produce, among other responses, a leg flexion reflex. These responses that are produced unconditionally in all members of the same species by a US are called the unconditioned response (UR).

S1 is called in Pavlovian conditioning the conditioned stimulus (CS), because it produces the response (e.g. salivation) conditional on learning having taken place. The CS is usually a tone, a light, a colour, an odour or a taste, initially of low biological significance. The CS does not elicit the CR in all members of the same species, but only in those individuals that have undergone training with the same or similar USs (e.g. only dogs exposed to tone-food pairings will end up salivating at tones).

Figure 3.1 illustrates the different steps that take place during classical conditioning and may help you better remember the different terms that we are using. Before the CS becomes conditioned (see panel A of Figure 3.1), the CS is still a neutral stimulus which does not produce the CR, but which does produce an orienting response (OR), when it is first presented. The orienting response consists of paying attention to the stimulus and noticing that something unexpected has occurred. Pavlov called it the 'what is that?' reflex. If we observe that a particular CS does not produce an OR when it is first presented, that means that we had better use a different stimulus for our conditioning experiment because a stimulus which does not command the attention of the organism will not become conditioned.

Panel B of Figure 3.1 shows the conditioning stage. During conditioning, the CS is presented immediately before the US. That is, the tone sounds and the food follows. It is important that the food follows the sound immediately or at most after a very short delay because contiguity between the CS and the US helps the animal to associate the two stimuli. The salivation that is produced during conditioning is produced solely by the food during the early tone-food pairings (thus, salivation is a UR during the early stages of conditioning) and it gradually begins being produced also by the CS as learning proceeds (thus, salivation gradually becomes a CR).

Panel C of Figure 3.1 shows that after conditioning has taken place, if we present only the tone (that is, the CS but not the US), the dog salivates to the tone. Only when salivation occurs on presentation of the CS alone (either on CS-alone trials, or in the presence of the CS before the US is presented on CS→US trials) can we say that the salivation observed is a CR. The CR serves the purpose of preparing the organism for the occurrence of the US (e.g.
salivation prepares the gastric system to receive food). Moreover, the CR can be used as an index of learning because its occurrence means that the dog has now learned to anticipate food in the presence of the tone.

Non-salivary Pavlovian conditioning

It should be clear by now that not all forms of Pavlovian or classical conditioning involve salivary responses. By simply looking at TV for a short while, we can discover many more examples of Pavlovian conditioning because it is widely used in advertising. For example, food commercials generally try to produce salivary conditioning, decaffeinated coffee advertisements generally involve associations that are aimed at producing relaxation CRs; the way in which perfumes are advertised almost always includes the establishment of an association between a given trade mark, which is an initially neutral stimulus (i.e. a CS, as the tone was in Pavlov's experiment) and an attractive person of the opposite sex, who is a biologically significant stimulus (i.e. the US, as was the food in Pavlov's work). The establishment of such associations is of course aimed at producing an appetitive reaction when we see the advertised brand at the store. Moreover, some governmental prevention campaigns make use of fear conditioning by associating, for example, alcohol with a fatal accident. The purpose is to produce relevant fear responses that might help prevent traffic accidents. We shall now look at some forms of Pavlovian conditioning that have been extensively studied in the laboratory: fear conditioning and taste aversions. The case study on p. 73 also shows how drug addictions can be explained as an example of Pavlovian conditioning.

Fear conditioning

A dog hears a tone, but this tone does not signal food. Instead, this tone (CS) is always followed by a footshock (US). The UR produced by the footshock US is, among others, a suppression of any ongoing behaviour. For example, if the animal is pressing a lever to obtain food, its lever-pressing behaviour will be suppressed if we shock the animal. Instead of lever pressing, the animal behaves defensively by freezing. As in the preceding examples, these pairings of a relatively neutral event (the tone) with a biologically significant event (in this case a painful stimulus) generally occur over several trials, but this time the CR elicited by the tone will be a fear reaction! The fear CR can be easily assessed through the behavioural suppression that it produces (Annaud and Kamin 1961; Estes and Skinner 1941). That is, if we present a CS (such as a light or other) that tells the animal that a footshock will be forthcoming, the animal experiences fear of footshock and will suppress any ongoing behavior motivated by a positive outcome such as food (e.g. lever pressing). It is not that the animal can escape shock by suppressing lever pressing or other behaviours when the tone is presented. It is simply that when animals (and humans) feel fear, they frequently suppress other behaviours (see Figure 3.2), probably because, in the past evolutionary history of many species, freezing was an effective way of avoiding being detected by sources of harm such as a hunting carnivore. In this way, we can assess whether the animal is expecting the footshock to occur after the CS. If the animal feels fear, its ongoing behaviour will be suppressed. The index of

Figure 3.2. Conditioned suppression is now a common way of assessing Pavlovian conditioning. In this case, instead of assessing the number of drops of saliva, as in Figure 3.1, we can assess the degree to which the rat freezes upon observation of the light (CS). Before conditioning, the rat in the figure was not afraid of lights and did not freeze when we turned the light on. However, if the rat has learned that the light (CS) will be followed by an electric shock (US), the rat will freeze when we present the light. This shows that fear conditioning has been acquired (i.e. the rat has learned the CS-US association). If we have a computer recording the rat's activity, such as, for example, the number of times that the rat presses a lever to obtain food, and the rat freezes when we present the light, the computer will record a suppression of lever-pressing behaviour when the light is turned on. Thus, suppression of ongoing behaviour can be used to assess whether animals have learned a CS-US association. This technique is more convenient than assessing salivary conditioning, and hence is now more frequently used.
learning (the CR), in this case, is the degree to which an ongoing behavior such as lever pressing is suppressed.

Thus, we have here a procedure which is very similar to the tone-food procedure, but the effect of the tone-footshock pairings is now the conditioning of a fear response. This raises new and interesting questions that go beyond salivary responding. For example, if fear is the result of conditioning because a neutral event is paired with a painful event, does this mean that we can reduce fear if we do the opposite type of conditioning (e.g. would pairing the tone with an appetitive event after it has been paired with shock reduce the fear reaction)? The answer to this question is yes. This technique is called counterconditioning. In brief, once a CS has been paired with a US which is aversive (such as footshock — or a scolding by a parent), we can perform counterconditioning which consists of pairing that CS with a US which is of the opposite valence (i.e. appetitive), such as food. A clear application of counterconditioning is the treatment of fears in clinical psychology. Alternatively, we can also countercondition an appetitive stimulus by pairing it with an aversive stimulus. This is sometimes used in the treatment of alcoholics by clinical psychologists. Thus, counterconditioning can either be used to give appetitive valence to a previously aversive stimulus or vice versa.

Taste aversion learning
Imagine now a person who eats a certain food and relatively soon feels sick. You have probably experienced something similar yourself. Conditioning here is said to occur because a person (or another animal) that experi-

Drug addiction learning: tolerance and overdose effects
The food or footshock, drugs (e.g. heroin) are also USs that produce URs. Thus, when they are taken, they become associated to the US that are present (e.g. group of friends or a certain location) and those CSs will end up producing CRs. However, whereas the UR produced by food (salivation) is very similar to the CR produced by a CS which has become associated with food (i.e. the tone also produces salivation after conditioning has taken place), the CRs produced by CSs which are associated with drugs are generally just a reaction (opponent response) to that by the drug itself (e.g. alcohol produces a) but the CSs associated with alcohol produce more like in the case of food, in which drug served the purpose of preparing the organism to receive the drug, the opponent CR that produces the CSs associated with drugs also serves the purpose of preparing the organism to receive the drug compensating for the effect that the drug will produce in that way, the organism compensates for the effect of the drug, so that, when conditioning has taken place (when one has become a drug addict), the CSs will prepare the organism to receive the drug so that it will be less harmful. However, this opponent reaction produced by the CSs also produces the need to take the drug in order to compensate for that opponent reaction. Therefore, the initial dose no longer produces the effect the drug addict was seeking because the UR effect is now being compensated for by the opposite CR. This compensation normally results in drug tolerance. As a consequence, drug addicts tend to increase the dose of the drug that they take (see Siegel 1983 for a review on drug tolerance and drug dependence).

One of the most dramatic effects of this process can be seen when, once a day, the drug addict takes the drug in a new context (e.g. in a different location or with very different and unfamiliar people). In that new context, the CSs that produce the opponent CR are not present. Hence, the drug addict's body is not prepared to receive the dose. The addict is taking interviews with people who have survived after an "overdose" (Siegel 1984) as well as studies with animals (Siegel et al. 1982) suggest that what is commonly known as death by overdose is not always due to an overdose (i.e. the doses are generally not larger than those to which the same is not used to). Instead, it is sometimes due to taking the drug in an unfamiliar context. That is, in a context which does not produce the opponent CRs that should have prepared the organism to receive the drug. Thus, death by "overdose" is sometimes due to taking a regular dose in a new context which does not produce the CRs that should compensate for the effect of the drug.
ences such food–illness relation will probably develop an aversion to that particular food and the mere odour or taste of that food may become a CS which will produce a CR of nausea in the future. If you have been sick after eating a certain food, it is likely that you developed an aversion to that food and avoided eating it again on subsequent occasions. Just thinking of eating it may produce a rather unpleasant CR. Of special interest in this type of conditioning is why you attribute the illness to that particular food among the many things that you ate that day. Moreover, why did you attribute it to the food instead of to, say, the people with whom you were eating (or the conversation that you had with them)?

The phenomenon of conditioned taste aversion was first studied by Garcia and Koelling (1966). They gave rats water to which they had added two CSs: a flavour CS and an audio-visual CS. That is, the water was flavoured and the apparatus from which the rats had to lick in order to obtain water produced a flashing light and a sound whenever the rats licked. The primary question was whether the flavour or the audio-visual stimulus would better serve as a CS. The US was either a drug that made the rats feel sick in their stomach or an electric shock to their feet. Thus, the question was, would the rats equally associate illness with a flavour and an audio-visual cue? And would they equally associate the footshock with the flavour and the audio-visual stimulus? In order to test this question, Garcia and Koelling later gave some test trials to the rats with either the flavoured water (and no audio-visual CS present) or the audio-visual water (with no flavour CS present). Garcia and Koelling found that the rats that had been given the drug which made them feel sick refused to drink the flavoured water but not the audio-visual water. Conversely, the rats that had been given the shock US refused to drink the audio-visual water but not the flavoured water. This means that rats tend to associate gastric illnesses more readily with flavours than with audio-visual stimuli; and that, by contrast, flavours do not work very well as cues for pain on the skin such as that produced by footshock (the flavour–shock associations were weaker than the associations formed between the audio-visual cues and the shock). Thus, contrary to what Pavlov thought, not all types of CSs can be equally associated with all types of USs. According to Garcia and Koelling, there is a predisposition which makes us (and other animals) better associate some CSs with some USs. This associative predisposition probably reflects both genetically coded information and prior experience of the human or animal.

Note that this does not mean that we (or rats) cannot associate illness with audio-visual cues. It only means that flavours are more readily associated with illness than are other cues. Indeed, other research has shown that although Garcia and Koelling were correct in concluding that illness is more readily associated with flavours than with other types of CSs, animals do also associate illness with other CSs (P.J. Best et al. 1977; Boakes et al. 1997; Loy et al. 1993). As an example, if you should feel sick after having eaten in a new restaurant, you will probably acquire a conditioned taste aversion to what you ate, but you will also probably acquire an aversive reaction toward the restaurant.

An important clinical application of taste aversion learning can be found in cancer patients who are exposed to chemotherapy (Bernstein 1978, 1991). Because chemotherapy often produces nausea and vomiting, the patient may come to associate that sickness with the CSs that occur close in time (e.g. the flavour of the food that they ate that day), and after some sessions of chemotherapy, many stimuli (flavours of lots of different foods) can become conditioned and will produce CRs of nausea and sickness, which creates a very serious eating problem for the patient. Although important progress has been made concerning how those taste aversions are produced, there is still a need for more research on how taste aversions can be treated. One possibility that researchers are exploring is presenting patients with unusual tastes immediately before chemotherapy so that the taste aversions get conditioned to those unusual flavours rather than interfere with the normal diet. (See also p. 51)

S–S associations between stimuli that are biologically neutral

So far we have seen that in Pavlovian conditioning, organisms associate the mental representations of two stimuli (S1 and S2) and S2 is generally a biologically significant stimulus (a US) which produces URs in the absence of any prior learning. However, as previously mentioned, stimulus–stimulus (S–S) associations can also occur between stimuli that are biologically irrelevant (such as, for example, the learning of an S–S association when these stimuli are both numbers). This type of S–S learning is not called classical conditioning and does not produce CRs (but it is also a form of learning).

Because the learning of associations between representations of neutral stimuli does not produce CRs, its study with animals has been more difficult than with
humans. Thus, whereas much of the research on classical conditioning has been performed with non-human animals, much of the research on associations between neutral events has been conducted with humans (but see the case study to learn about studies with neutral events with animals).

Another difference between experiments on Pavlovian conditioning and experiments on associations between biologically neutral events has to do with whether the scientist is interested in the development of the CRs per se (e.g., how taste aversions are developed or extinguished in chemotherapy patients) or whether the researcher is interested in how learning occurs. Whereas in the past researchers were primarily concerned with the development of CRs (such as in Pavlov’s experiments), many of the current researchers are more concerned with the underlying learning process. In this later case, the CRs, or other types of responses, are used as a mere index to assess whether learning has occurred. Thus, if the researcher is interested, not in the development of a CR, but in whether or not the organism is acquiring an association between the mental representation of two events, such association can be equally assessed through a CR, through a verbal response, or through other means. The generalized use of computers allows present-day researchers to design computer programs in which S1 and S2 are biologically neutral stimuli that are presented through the
computer's screen or speakers, and the keyboard responses (not CRs) of the human volunteers are automatically recorded in a data file in the computer. Thus researchers can easily assess how the learning process takes place under different neutral conditions. This has produced an enormous increase in the amount of research that is being conducted with neutral stimuli and human subjects. Today, if a researcher wants to know about how learning occurs rather than about the formation of a CR, the experiment can often be simpler with humans than with animals.

One very common type of S-S experiment with humans consists of learning causal relations between two events (e.g. Allan and Jenkins 1983; Matute et al. 1996; Shanks and Dickinson 1987; Wasserman 1990a, 1990b). This may sound strange because we have not yet mentioned any relationship between conditioning and causal learning, but conditioning experiments with animals have also been frequently viewed as the animal's learning causal relations between events (e.g. Mackintosh 1977; Tolman and Brunswik 1935). Moreover, we know that the conditions that govern the acquisition of causal relations in humans are similar to those that govern the acquisition of conditioned responses (Shanks 1987). Although the details vary from one experiment to another, the following example illustrates how one of these experiments on human causal learning can be constructed.

Participants are seated in a room with a computer and given instructions on the computer's screen. These instructions tell the participants that they should imagine that they are an allergist who is going to see the medical records of several patients that have developed allergic reactions after taking certain medicines. The task of the participant will be to learn which medicines caused which reactions. Once the participants have read the instructions, the experiment begins. The screen then shows the records of fictitious patients, one at a time. It first shows the medicine that Patient 1 has taken (S1), and then it shows the allergic reaction that this patient has developed (S2). This process will be repeated for Patients 2, 3 and so on. Thus, in this example, each S1-S2 trial is represented by one fictitious patient which has taken one medicine (S1) and has developed a reaction (S2). After the participants have seen a series of trials (i.e. patients), we can ask them to which degree they think that each of the medicines was the cause of the allergic reaction. They normally respond by giving their judgement on a numerical scale (e.g. from 0 to 100) provided by the experimenter. The number that they give is called their causal judgement and it is the index that is used in these experiments to assess whether learning has taken place. Thus, as you can see, using this index is different from assessing CRs, but serves the same purpose of assessing whether learning has taken place.

But not all experiments involve medicines and allergic reactions, or even test questions and causal judgements. Other experiments may use video games in which S2 is a tank which explodes if it enters a mine field (S1) in the computer screen (Shanks 1985), yet other experiments use a Martians' invasion (S2) which is predicted by some cues (S1) in the screen (Arcediano et al. 1996). In this later case, the acquisition of the association is not necessarily assessed through a causal judgement. Instead, the computer ordinarily records the keys that the participant presses throughout the experiment, and a behavioural change is usually observed when the participant is expecting a Martians' invasion. This behavioural change is not observed in the early trials in which S1 predicts the Martians' invasion but it is gradually observed as learning proceeds. This produces a learning curve similar to that observed in Pavlovian conditioning (see Figure 3.3).

The interesting point when comparing the research on associations between neutral events and the research on associations between biologically significant events is that the same type of results are generally being observed in both cases. That is, even though some studies are more frequently conducted with animals and the other ones with humans, even though some studies produce CRs and the others do not, and even though there are many other important differences in the way that these two types of experiments are being conducted, in both cases, we can see that learning proceeds in a similar way and is similarly sensitive to various experimental manipulations. Thus, in the remainder of this chapter, we will indistinctly refer to either conditioning data or causal judgment data, to animal data or to human data. It must be noted, however, that as this trend to compare these two situations is increasing, some differences between the two situations are starting to show up. The research update on p. 85 shows one of such differences observed between experiments that use biologically significant USs versus those that use biologically neutral stimuli.

Learning to predict that S2 will not occur

So far, we have only considered conditions in which Stimulus 1 (S1) predicted Stimulus 2 (S2). But can we also learn the opposite relation? That is, can we learn that S1 predicts the absence of S2?
Consider, for example, the following experiment with humans using the causal judgement preparation described above. Student subjects are shown the medical records of fictitious patients who have developed allergic reactions after taking some medicines, and are told to figure out which medicines caused the allergic reactions in those patients. The students see the following types of patients: on the one hand, they see many patients that have taken medicine A and have developed the allergic reaction (we can represent this as $A+$). On the other hand, they also see many patients that have taken medicines A and X simultaneously and these patients have not developed the allergic reaction (represented as $AX$). Now, if you were one of the students taking part in such an experiment and were then asked to give your causal judgement about the likelihood of medicines A and X causing the allergic reaction, what would you say? The rating scale in these cases goes from +100 to -100, with +100 meaning that you are absolutely certain that the medicine produced the allergic reactions, 0 meaning that there is no relation between the medicine and the allergic reaction, and -100 meaning that you are certain that the medicine absolutely prevents (or inhibits) the occurrence of the allergic reaction. Most students give a rating close to +100 for medicine A (i.e. they attribute the allergic reaction to A) and a rating close to -100 for medicine X (i.e. they attribute to medicine X inhibitory or preventive properties with respect to the allergic reaction). Thus, this shows not only that we can learn when one stimulus (e.g. A) predicts $S_2$ (the allergic reaction), but also that we can learn when another stimulus (e.g. X) predicts that $S_2$ will not occur.

This has been demonstrated with human subjects (e.g. Chapman 1991; Chapman and Robins 1990; though their procedure was slightly different from the one described here) as well as with animals. With animal subjects, this was demonstrated long ago by Pavlov (1927) and has been generally called Pavlovian Conditioned Inhibition. When this type of experiment is run with animals, A and X are usually lights and tones, $S_2$ (the allergic reaction in our example) is usually food or footshock, and the index of learning is the degree to which the CR is inhibited when X is presented (e.g. inhibition of a salivary or a fear CR) rather than a causal judgement. Otherwise, the experiment works much the same way, and animals also learn to predict when the US will not be presented, and show it by inhibiting the CR in the presence of X that should otherwise have been observed (see also Rescorla 1969).

One important factor in the development of these inhibitory reactions is that they can be acquired only if the context in which they are learned is a context which has been associated to $S_2$. That is, imagine a dog that is given $X$→no shock training. Unless the dog had any reason for expecting the shock to occur in such a context (i.e. unless the context was associated with shock), the dog would not interpret such $X$→no shock pairings as $X$→no shock, but simply as $X$. The same is true for our medicine example above. If participants judge medicine X as preventing the development of an allergic reaction it is because they had a reason to expect the allergic reaction (i.e. medicine X was taken in compound with medicine A, which was a medicine strongly associated with the development of the allergic reaction, and hence, A played the role of the context which should produce the allergic reaction unless X prevented its occurrence).

Extinction and spontaneous recovery

One of the things that Pavlov noted in his classical conditioning experiments was that, after conditioning had occurred, if the CS was repeatedly presented alone (i.e. without the US), the CR would gradually disappear. This is called extinction of the CR. You have probably experienced a similar process many times in the past. For example, you may have associated a given perfume or music (CS) with a given person (US) and this may have produced some emotional CRs after several pairings. Moreover, even if you had not listened to that music or smelled that perfume again in many years, the CRs would still occur if you were now exposed to the CS again. That is, CRs are generally not 'forgotten' by the mere passage of time (although there may be some decrease in the magnitude of the CR). However, if you continue exposing yourself to the CS when the US is not present (you listen to that music over and over in the absence of the US), the CRs will eventually extinguish (i.e. become successively smaller and finally vanish).

This is shown in Figure 3.3, which first shows (Panel A) how the responses are gradually acquired in most learning situations, and then shows (Panel B) how the responses are gradually extinguished. Moreover, we also know that extinction does not imply unlearning or forgetting (Bouton 1993; Pavlov 1927; Rescorla 1996). For example, once extinction has taken place, if the subject is not exposed to either the CS or the US for some time (Panel C), and then we present again the CS in order to test whether it would produce a response (Panel D), it does produce a response. This restoration of responding
is called spontaneous recovery because the response is spontaneously recovered (by the mere passage of time) after it had been extinguished. Once a response has been recovered, it can either be re-conditioned or re-extinguished, and this time, either process will usually be easier than the first time. This has important implications in clinical psychology. After therapists have been able to extinguish a problematic response in a patient (e.g. a fear response), they must be aware that the response is potentially subject to spontaneous recovery over time. Thus, the patient will need to be monitored for future possible re-treatments, after the response is extinguished.

Generalization and discrimination

Another process of which we have to be aware is generalization. If subjects learn an association between two stimuli, S1 and S2, they will probably transfer what they learned to other stimuli that are similar to S1. Thus, if a dog has learned that a tone is followed by food, it will probably salivate not only at the sound of that particular tone, but also at other tones which are relatively similar to the original S1. The greater the similarity between the original S1 and the probe stimulus, the greater the transfer will be. This process allows us and other animals to efficiently use previous knowledge in new situations.

Otherwise, we would have to undergo the whole process of new learning every time that we encountered a situation which was not exactly the same one in which our learning had taken place. And in practice exactly the same situation never arises twice.

But there might be situations in which we do not want the CR (or the causal judgement or the predictive behaviour) to generalize to other similar situations. In this case, we must use a discrimination procedure to teach the subject to discriminate between those stimuli that are followed by S2, and those that are not followed by S2. For example, learning that a given medicine produces an allergic reaction could produce the generalized belief that all medicines produce the allergic reaction. Thus, in order to discriminate, the subject must also be exposed to medicines that do not produce the allergic reaction (e.g. Matute et al. 1996). The way in which Pavlov used to do this with his dogs was by exposing them to some CSs that were followed by food, and also to CSs that were followed by the absence of food. In that way, the animals were able to discriminate between the CSs that produced food and those that did not. In consequence, the CR tended to concentrate only around the CSs that produced the food. Similarly, in a human experiment using the Martians video game mentioned above, college students were trained to predict that colour blue would be followed by a Martians’ invasion, whereas colour yellow would not (Arcudi et al. 1996). But in order to learn this, subjects needed to be exposed to both conditions. If they were exposed only to the blue-invasion relation, then they would also expect the invasion in the presence of other colours that we might test after learning had occurred (i.e. generalization).

This discrimination training procedure is also known as differential inhibition, because, presumably, inhibition is acquired with respect to the stimulus that signals the absence of S2, but not with respect to the stimulus that signals the presence of S2. The stimulus that signals that S2 will occur is called the excitatory stimulus (or S+) and the stimulus that signals that S2 will not occur is called the inhibitory stimulus (or S–).

Theories of S–S learning

S–R versus S–S theories

Many psychologists believed that the important association going on in classical conditioning was the one that become established between the CS and the response (e.g. Hull 1943), that is, the S–R association. For
example, after several trials in which the tone was followed by food (and thus by salivation), the dog came to associate, according to S–R theories, the tone with the salivary response. And this was the reason why the tone ended up eliciting salivation. If you think carefully about the implications of this view, you will notice that S–R theory does not assume that the dog has learned to predict when food will be available. It simply assumes that the tone will produce salivation because tone and salivation have occurred together many times. This theory was more mechanical than current theories, and did not assume the existence of any cognitive processes in the mind of the dog or the human which had been conditioned (see Rescorla 1988 for further elaboration on differences between this view and today’s view).

Instead, according to current stimulus–stimulus (S–S) theories, the important association is the one formed between the mental representations of the two stimuli, that is, the one that allows the animal to predict Stimulus 2 (S2) on the presence of Stimulus 1 (S1). Modern psychology has shown that there are many cases in which CRs do not occur (and therefore, S–R associations cannot be formed) and even so, organisms are able to learn that one stimulus predicts another. As an example, consider sensory preconditioning (see case study on p. 75). In sensory preconditioning experiments, animals are able to learn an association between two stimuli that are biologically irrelevant, and which do not produce URs or CRs. Thus, those experiments cannot be explained by assuming that an S–R association is what the animal acquires. Moreover, S–R theories would not be able to account for any of the examples of learning of causal or predictive relations between neutral stimuli in humans. Although modern psychology assumes that S–R associations are formed in many situations (Dickinson 1989), it also assumes that S–S associations, rather than S–R associations, are necessary to explain the vast majority of findings in learning research (Dickinson 1980).

Contiguity theory

Temporal and spatial contiguity is one of the most important conditions that modulate the establishment of associations (Pavlov 1927). As an example, consider an experiment in which there are several groups of subjects. All of them are exposed to the same CSs and USs (same intensity, etc.) and for the same number of trials. However, in one group the US always occurs immediately after the CS, whereas in other groups we vary the interval between the CS and the US. The longer the CS–US interval (onset to onset), the weaker the CR that will be observed. Even in situations such as taste aversion learning, in which the delay between the CS and the US can be relatively long, the effect of varying the CS–US interval can also be detrimental in the formation of associations (e.g. if we feel sick one day at 6 p.m. we will probably associate our illness with something that we ate for lunch a few hours earlier, but not with something that we ate the day before). Thus, contiguity between S1 and S2 modulates the acquisition of associations.

But Pavlov’s theory stated that contiguity was the only important factor which determined the formation of associations. According to Pavlov, when a CS and a US occurred contiguous in time, a connection was formed between the CS centre and the US centre in the brain and, in that way, the CS gradually came to substitute for the US in eliciting the response. Pavlov’s contiguity theory has been challenged by the results of several experiments that showed that contiguity was not the only condition that enabled the formation of associations. Some of those experiments are described next.

Some problems with contiguity theory

Contingency

Rescorla (1968) conducted an interesting experiment that showed that contiguity between two events was not the only important factor that affects associative learning. He used several groups of animals that he exposed to several pairings of CS–US (tone–shock). However, he also inserted trials in which the US was presented in the absence of the CS. For some animals, there were few of those US-alone trials, for other animals there were as many USs in the absence as in the presence of the CS. Now, what would you think if you were exposed to those conditions? For example, consider the case in which the probability of the US when the CS is presented, p(US | CS), equals the probability that the US will occur when the CS is not present, p(US | noCS). That is, a footshock (US) occurs with the same probability when a tone (CS) is on or when the tone is off (noCS). Do you think that the animal should acquire any special fear reaction to the tone? Surely not. The tone has no informative value. It does not help the animal predict when a footshock is about to occur.

Thus, what Rescorla observed was that, despite all subjects being exposed to the same number of CS–US pairings and to the identical CS–US contiguity, reducing the informative value (i.e. contingency) of the CS by
inserting several US-alone trials, produced a dramatic decrease in the magnitude of the CR. The strongest CRs were observed when the positive contingency was strongest. That is, when the probability of the US in the presence of the CS was much larger than the probability of the US in the absence of the CS. In those cases, the occurrence of the US was contingent (depended) on the occurrence of the CS. Thus, the CS had a great predictive value, and subjects learned that the CS was an accurate signal of when the US would be presented. According to Pavlov, all groups should have acquired a similar fear response because all of them had been exposed to the identical contingency during the identical number of CS-US pairings. Thus, this effect shows that, in addition to contiguity, the informative value of the CS (or, in other words, the contingency between the CS and the US) is also an important factor. Note, however, that contingency alone is also unable to explain learning because it cannot explain how the learning curve occurs in a trial-by-trial basis. That is, if contingency were the only important factor, we would expect a flat learning curve, with subjects detecting the degree of contingency in the same way in any learning trial. However, the learning curve typically observed in learning situations (see Figure 3.3) suggests a gradual process in which the acquired associations are strengthened or weakened over the learning trials.

Overshadowing
Imagine now a situation in which two CSs are presented in compound as signals of a US. For example, two different tones are presented simultaneously and followed by food. Would the two tones acquire the capacity to elicit a CR? According to contingency theory, if the two tones are equally contiguous to the food, the two of them should acquire a CR of similar magnitude. However, many experiments have shown that one of the two CSs frequently acquires greater response potential and overshadows the other CS. The weaker CS elicits a response if trained individually, but becomes overshadowed if trained in compound with the stronger CS. Apparently, the stronger CS competes with the weaker one. This was first observed by Pavlov (1927) and has been later demonstrated in many different situations. Contiguity theory cannot explain why this occurs.

Forward blocking
Another experiment that was critical in showing that Pavlov's contiguity theory did not suffice to explain learning was Kamin's (1968) blocking experiment. (The word 'blocking', unless otherwise stated, normally refers to forward blocking; later in this chapter you will see some examples of a different type of blocking which has been demonstrated more recently and which is ordinarily called backward blocking.) Kamin exposed an experimental group of animals to several pairings of CS1 followed by a US during the first phase of the study (i.e., CS1 → US). Then in Phase 2, he continued using the CS1 → US pairings but he added a new CS (CS2) which was presented in compound with CS1. Thus, during Phase 2, both CS1 and CS2 were potential predictors of the US (i.e., CS1 → CS2 → US). According to Pavlov, both CSs should become associated with the US because both of them were contiguous with the US. To test this expectation, Kamin then presented CS2 alone during a subsequent test phase. If it had become associated to the US, it should produce the CR. But it did not! Moreover, this failure to respond to CS2 did not occur in a control group that had not been exposed to the initial CS1 → US pairings during Phase 1 but that had received the identical CS1 → CS2 → US treatment during Phase 2. Thus, the control group demonstrated that the number of CS2 → US pairings had been sufficient because good responding was observed in the control group. Then why was responding to CS2 blocked in the experimental group? According to Kamin, the experimental subjects had previously learned that CS1 produced the US, and thus the subsequent introduction of CS2 as a predictor was absolutely redundant. The animals did not need an additional predictor to know when the US was going to occur. Animals were already responding to CS1 and the occurrences of the US were not surprising at all. As a consequence, according to Kamin, the association between CS2 and the US could not be acquired. This suggests that only USs that are surprising can become associated to CSs.

For an example of how blocking works in humans, consider the medicines and allergies experiments previously described. Imagine that you are taking part in one of those experiments and you see many patients who have developed the allergic reaction after taking medicine A, and that later, you see patients who have taken medicines A and X simultaneously and they also develop the allergic reaction. Would you attribute the allergic reaction to medicine X? Why not? If contiguity theory were correct, seeing a sufficient number of patients that have developed the allergic reaction after they had taken A and X (thus allowing for contiguity between X and the reaction) should produce good learning. However, we tend to attribute the allergic reaction to medicine A rather than X if we have previously learned that A alone
is sufficient to produce the effect and it is present on the subsequent training trials with X.

Note that the effect of forward blocking can also be regarded as the effect of previous knowledge. For example, you may develop an allergic reaction after having eaten shrimps (A) and potatoes (X). But despite the allergic reaction being equally contingent and con
gnous with both the shrimps and the potatoes you will probably attribute the allergic reaction to the shrimps, and will probably discount the potential causal role of the potatoes, correct? Why? You probably have some previous knowledge that shrimps may produce allergic reactions. Previous knowledge comes from a previously established association between shrimps and allergic reactions. And this association competes with the association between potatoes and allergic reactions.

Thus we now know that, in addition to contiguity (Pavlov 1927) and contingency (Rescorla 1968), it is also important that there are no competing associations in order to observe a CR or a causal or a predictive judgment. Note, however, that the concept of contingency can be regarded as showing the importance of an absence of competing associations. That is, contingency refers to whether S2 occurs in the absence of S1 (as well as continguously with it), but this can be regarded as the formation of competing associations because when the S2-alone trials occur, S2 necessarily becomes associated to other cues (e.g. background cues that are present in the experimental context). Thus, in addition to contiguity, most current associative theories have emphasized the importance of an absence of competing associations for the occurrence of a CR or a causal judgement (e.g. Rescorla and Wagner 1972).

Inhibitory learning
Consider also the examples on inhibitory learning described above in which a subject (human or animal) learned to predict that S2 would not occur (e.g. when subjects attributed to medicine X preventive or inhibitory properties with respect to the allergic reaction). If S2 (e.g. an allergic reaction) does not occur when S1 is presented (medicine X), S2 is not contiguous to S1. Thus, contiguity theory cannot explain such learning. Then, how does inhibitory learning occur?

The concept of contingency could perhaps provide an explanation for inhibitory learning because, in these cases, S2 (the allergic reaction) is occurring with greater frequency in the absence than in the presence of S1 (medicine X). Indeed, if you recall the examples of inhibitory learning described above, the allergic reaction occurred when medicine A was taken but not when medicine X was taken. Thus, the probability of the reaction (S2) was greater in the absence than in the presence of S1 (medicine X). This is a negative contingency situation in which S2 is contingent (depends) on the non-occurrence of S1. These negative contingency situations result in the inhibition rather than elicitation of the CR or the causal judgement. Also, if you think about this, you will note that this probably occurs because the allergic reaction (S2) was associated with medicine A rather than with our critical S1 (medicine X). Thus, we could also say that inhibitory learning occurs because S2 is associated with a competing stimulus (such as medicine A or background contextual cues), during the trials in which S2 occurred in the absence of S1. This could explain inhibitory learning as a type of competition by the CSs (or contextual cues) that are present in the experimental situation. Several other possibilities have also been considered and it is not yet clear how we should best explain inhibitory learning. What appears clear is that learning to predict the absence of an event implies that such event was in some way expected (e.g. because it was associated to something in that context) and its absence when the CS was presented was noticed (see Mackintosh 1983 for further elaboration of these issues).

CS pre-exposure and US pre-exposure effects
Now imagine that one day you feel sick after having eaten bread. You will probably attribute your illness to some other thing that you ate rather than to the bread. Moreover, if you ate something new that day you will almost certainly attribute your illness to that new food. The reason is that you have probably eaten bread many times in the past and those experiences have never been followed by illness. Thus, even though you now felt sick after eating bread, you probably would not associate your illness with bread. In this case, we say that you were pre-exposed to the CS (bread) in the absence of the US and this made the formation of a CS-US association unlikely. That is, novelty is one more factor that influences conditioning.

Once again, we can see that contiguity between the CS and the US is not sufficient for the establishment of an association. If subjects receive exposure to either the CS alone or the US alone before the CS is paired to the US in a conventional conditioning paradigm, conditioning becomes retarded (e.g. Lubow and Moure 1959; Randich and LoLordo 1979). For example, a dog that has heard a tone several times before conditioning will
later have difficulties in associating that particular tone with food during the conditioning stage. The same occurs when the subjects are exposed to the US alone before conditioning. If contiguity between a CS and a US during the conditioning stage were the only necessary condition for the formation of associations, pre-exposure to either the CS or the US alone should be irrelevant. But it is not. Why? There are several potential explanations. On the one hand, we could assume that if a subject is exposed to either the CS alone or the US alone, the subject will learn that the CS or the US is unrelated to anything significant. Thus, subjects should later have difficulties in associating that stimulus to another one (e.g. Baker and Mackintosh 1977). Another potential explanation is that the pre-exposed stimulus becomes associated to the context in which it is being presented, and thus, its potential to become associated to another stimulus during the conditioning stage becomes reduced (e.g. Wagner 1981).

Current theories

Psychologists have developed several associative theories that improve on Pavlov’s view concerning how the associations are acquired and strengthened in a trial by trial basis (e.g. Mackintosh 1975; Pearce and Hall 1980; Rescorla and Wagner 1972). Most current associative theories are formalized by a mathematical equation which captures the basic assumptions that these theories make about how the learning process occurs during each learning trial. At first, it may sound strange to see that a learning theory can be summarized as an equation. Note, however, that qualitative theories are generally imprecise. That is, they do not allow us to make specific predictions for specific situations. For example, so far we have seen that for learning to occur, S2 must be surprising (i.e. the degree to which a CS will be blocked is directly related to the extent to which the US is predicted by another CS). But what we do exactly mean, for example, by being predicted; or when exactly can we say that the US is surprising? Formalizing these ideas into a mathematical model allows researchers to better know what the specific predictions of each theory are. Moreover, this allows researchers to write the theories into computer programs that should be able to ‘learn’ in the way predicted by the theory (e.g. see Mercier 1996 for a computer simulation of Rescorla and Wagner’s and Pearce and Hall’s theories).

By writing computer simulations of learning theories, the data from experiments can be more easily contrasted with the predictions of each of the different theories and a better assessment of these theories is possible as a function of how well each computer simulation fits the experimental data. Perhaps the most widely known of these theories is the one developed by Rescorla and Wagner (1972). Although the Rescorla-Wagner model is not a perfect theory of learning, it is probably the theory that has generated more experiments than any other model (see R.R. Miller et al. 1995, for a detailed assessment of the successes and failures of this theory). Interestingly, this theory is very similar to the delta rule, commonly used in cognitive science as well as in the construction of machine learning programs by computer scientists (see Gluck and Bower 1988; Lieberman 1990). This similarity between current learning theories and the delta rule is another factor which has contributed to the current interest in associative theories, which are now no longer restricted to the psychology of learning, but are being explored in many different areas of scientific research.

The Rescorla and Wagner Model

Without going into the details of the equation (see Rescorla and Wagner 1972 and Mercier 1996 if you would like to know more about it), according to the Rescorla-Wagner model, when a CS and a US occur contiguous in time during a learning trial, the association between them will be strengthened only if the US was unexpected. Consider for example the learning curve in Panel A of Figure 3.3. During the early trials, the US is unexpected (there are no previous associations to the US); thus, it becomes more and more strongly associated to the CS. Note also that the increment of the associative strength during each of the early learning trials is large, and certainly much larger than the increase that occurs during each of the later trials (see Panel A of Figure 3.3). This is because the discrepancy between what the subject is expecting to occur after the CS and what actually occurs is large during the early trials (at first the subject does not expect the US to occur after the CS). However, this discrepancy between the subject’s expectations and what actually occurs becomes gradually reduced as learning proceeds and the subject learns to predict the US after the CS. Thus, for each trial, the equation captures the difference between what the organism is expecting and what actually occurs, and this produces the typical learning curve.
Learning and conditioning

If you now look at the extinction curve in Panel B of Figure 3.3, this process works symmetrically. During the first extinction trials, the subject is fully expecting the US to occur, but it does not occur. Thus, its absence is surprising and much learning (in this case, unlearning) can be accomplished. Gradually, the subject comes to expect that the US will no longer occur, and thus, its absence is not surprising anymore. At this point, no more extinction can take place. Thus, this model can explain the shape of the acquisition curve (Panel A), and superficially it also explains the shape of the extinction curve (Panel B). But does it really explain extinction? The Rescorla-Wagner model fails because it explains extinction as if it were unlearning. Recall that the phenomenon of spontaneous recovery—and several other findings—show that extinction is not unlearning (see Bouton 1993; Pavlov 1927; Rescorla 1996). Although the phenomena of extinction and spontaneous recovery have been known since Pavlov’s days, their explanation has remained elusive. Indeed, they are still a matter of research (e.g. Bouton 1993; Rescorla 1996).

Consider now a blocking experiment. During Phase 1, the subjects learn to fully predict the US any time that CS1 is presented. Thus, when in Phase 2 we introduce CS2 in compound with CS1 (i.e. CS1→CS2→US), the US is by then fully predicted by CS1 and thus, no more learning can occur. The CS2→US association cannot be acquired according to the Rescorla-Wagner model. Thus, according to this model, blocking is a failure to learn the CS2→US association that occurs because the companion stimulus of CS2 (i.e. CS1) fully predicts the US when CS2 is first presented.

If you think of other phenomena described so far, you will notice that this same process can also explain the US-pre-exposure effect (but not the CS-pre-exposure effect). That is, when the US is pre-exposed before conditioning, it becomes associated to the context in which it is being pre-exposed. Thus, the pre-exposure phase can be regarded as a CS1-US training, with CS1 being the experimental context. Then, when we present our target CS (let us call it CS2) followed by the US during the conditioning stage, the context is also present and can play the role of a blocking CS that blocks responding to our target CS (i.e. CS2). Similarly, contingency experiments can also be explained by assuming that when we present the US in the absence of the CS, what we are actually doing is pairing the experimental context with the US. Thus, by the same reasoning as above, the context-US association will again compete with the association between the target CS and the US.

In summary, the association between a CS and a US that are present in a given learning trial will be strengthened only if the CS is not yet fully predicting the US, and also, if there are no other CSs present that are fully predicting the US because they have strong associations to the same US. If other CSs that are associated to the same US were present (including background contextual CSs), and these CSs were already fully predicting the US, then the US would not surprise the subject and thus, would not become associated with the target CS.

Problems with the Rescorla-Wagner model

Apart from the extinction and spontaneous recovery problems mentioned above, there are several other phenomena that are problematic for the Rescorla-Wagner model (see R. R. Miller et al. 1995 for a detailed review). One of these problems is the CS-pre-exposure effect. The Rescorla-Wagner model assumes that learning depends on variations on the effectiveness of the US; as conditioning proceeds and the US becomes being predicted by the CSs present in the situation, the ability of the US to enter into associations becomes reduced. Thus, this model cannot explain why presentations of the CS alone before conditioning should affect subsequent learning of a CS-US association (Baker and MacKintosh 1977). Indeed, explaining the CS-pre-exposure effect is problematic. One possibility is that during the pre-exposure stage subjects learn that the CS does not predict anything important. Then at the conditioning stage they should learn that the CS predicts a US but their initial expectation that the CS predicts nothing makes the new learning difficult (MacKintosh 1975). Several other possibilities have also been suggested (e.g. Pearce and Hall 1980; Wagner 1981) but it is not yet clear how we should best explain this effect.

One of the major problems for associative theories such as that of Rescorla and Wagner (1972; see also MacKintosh 1975; Pearce and Hall 1980) has been the observation of backward blocking and other retrospective revaluation effects. If you recall the forward blocking effect previously described, it referred to a situation in which the subjects had previous knowledge that, for example, medicine A produced an allergic reaction, and this made them discount the potential causal role of a second medicine, say medicine X, that the patients had taken in compound with medicine A. The interesting point for our present purposes is that blocking has also been observed in situations in which the medicine A— ALLERGIC reaction association is acquired subsequently. That is,
imagine that you have taken two medicines, A and X, and have developed an allergic reaction. You might well attribute your allergic reaction to both A and X. However, if you later learn that medicine A, taken alone, frequently produces allergic reactions, you will probably retrospectively discount the potential causal role that you might have initially attributed to medicine X. This is called a backward blocking effect because it is the result of subsequent rather than previous knowledge. This has been demonstrated using several different procedures with either human (Chapman 1991; Shanks 1985) or non-human animals (Denniston et al. 1996; Miller and Matute 1996). In addition to backward blocking, several other types of retrospective revaluation effects, showing that organisms re-evaluate their initial attributions, have also been reported (Baker and Mercier 1989; Miller and Matzel 1988). Backward blocking and retrospective revaluation effects are something that traditional associative theories (e.g. Rescorla and Wagner 1972) cannot explain because they assume that only stimuli that actually occur during a given learning trial can be learned about (only the stimuli that are present in a given learning trial enter into the equation for that trial). That is, those models cannot explain learning about a stimulus (medicine X) which is presented neither during or after acquisition of the competing A-US association. Thus, the subsequent learning about A cannot affect what subjects had already learned about X, because X is no longer present at the time in which subjects are learning about A. According to Rescorla and Wagner, what we learn about X cannot be retrospectively revaluated once we have learned it.

One possibility to explain backward blocking and retrospective revaluation effects is the so-called Comparator Hypothesis, suggested by Miller and Matzel (1988). According to these authors, blocking is not a failure to acquire associations as it is regarded to be by the Rescorla and Wagner model. Instead, they suggest, like Pavlov did, that contiguity between a CS and a US is sufficient for the formation of associations. Thus, even the 'blocked' CSs are learned about if they have occurred contiguously with a US, regardless of whether or not other CSs are present. Thus, in a typical forward blocking experiment, subjects would learn about both medicines, A and X, despite the allergic reaction being fully predicted by A by the time that X is initially presented. However, they also point out that some associations are stronger than others. Thus, after learning has taken place, if the associative strength of A (i.e. the strength of its association with the allergic reaction) is stronger than the associative strength of X (and X and A are themselves strongly associated because they have occurred in compound), this will result in our discounting the potential role of X in producing the allergic reaction. The same would also be true for an animal discounting the potential role of a light in predicting the US, if the light had been trained in compound with a tone and the strength of the tone-US association were stronger than the light-US association. Thus, according to these authors, a comparison between the strength of the different associations after learning has taken place (not during learning) is what determines our favouring some CSs and blocking other CSs as potential causes of S2. This allows this theory to predict both traditional (forward) blocking and backward blocking, because, if the comparison that leads to blocking occurs once learning has proceeded, then the order in which we encounter the information (either previously or subsequently) should be irrelevant (see also Baker and Mercier 1989 for a related account).

But there is also a different possibility that several researchers have considered. Imagine, for example, a backward blocking experiment in which subjects first are exposed to the CS1-CS2-US pairings and then are exposed to the CS1-US pairings. We could assume that what the subjects learn during Phase 2 is not only that CS1 produces the US, but also that CS2 does not produce the US. Thus, each of the CS1-US pairings that occur during Phase 2 will strengthen the CS1-US association but will weaken the CS2-US association because CS2 is not presented when the US occurs during Phase 2. Several researchers have suggested that this assumption can be incorporated into the Rescorla-Wagner model by making a very slight modification in the original Rescorla-Wagner equation. This consists of assuming that when a stimulus is expected to occur in a given trial but it does not occur (e.g. when subjects have been experiencing compound presentations of CS1 and CS2 during the first phase of a study, and then only CS1 occurs during Phase 2), the representation of the absent stimulus (CS2) becomes negatively activated in the trials in which it is not presented. Representing the stimuli that are absent (such as CS2 in this case) with a negative activation value in the equation produces a decrease in the strength of its association to the US (Dickinson and Burke 1996; Markman 1989; Tassoni
Biologically significant stimuli appear to be resistant to backward blocking

In the past, experiments on learning used to be conducted almost exclusively with animals. Moreover, they used to be almost exclusively conditioning experiments with biologically significant outcomes (i.e., USs). Since the mid-1980s, however, there has been an enormous increase in the amount of research that is being conducted with human subjects as well as in the amount of research that is not primarily concerned with the development of CRs (e.g., pure S1-S2 learning research with S2, not being biologically significant). The clearest finding of this recent trend is that much of the major learning phenomena can be observed with either animal or human subjects; as well as with CS-US, cause-effect tasks, or absolutely neutral S1-S2 situations. For example, Kamín’s (1986) forward blocking effect was initially demonstrated in animal conditioning but has now also been shown in human Pavlovian conditioning (Martin and Lavey 1991), as well as in human causal judgement situations (Shanks 1985).

However, the phenomenon called backward blocking was initially demonstrated in human causal judgement (Shanks 1986) but could not be demonstrated in animals during many years (see R.R. Miller et al. 1990). Backward blocking implies retrospective re-evaluation of initial attributions (see text), and thus, it appeared possible that perhaps humans were able to retrospectively re-evaluate their initial attributions, but animals were not able to do so. It could also mean that perhaps causal judgement is subject to retrospective re-evaluation, but Pavlovian conditioning is not. However, recent experiments have shown that backward blocking can occur in rats, as long as rats are exposed to S1-S2 pairings in which S1 and S2 are neutral stimuli rather than S2 being a biologically significant US (Denniston et al. 1996; Miller and Matute 1996). Thus, we now know that rats, as well as humans, are able to retrospectively re-evaluate their initial attributions. In consequence, the theories that explained these effects in humans (e.g., Dickinson and Ruerke 1988; Miller and Matzel 1988) can also be applied to animal learning. However, these experiments also suggest that backward blocking can occur only in neutral S1-S2 learning situations. This is at variance with the general view stated through this chapter that Pavlovian conditioning is equivalent to S1-S2 learning, where S1 and S2 are neutral stimuli. Thus, even though conditioning and the acquisition of associations between neutral events generally work in the same way, and even though most learning phenomena can be observed in both situations, there appear to be some exceptions. Some phenomena, like backward blocking, can be observed in neutral S1-S2 learning with either humans or animals, but has not yet been observed in conditioning situations with either humans or animals. For some yet unknown reason, stimuli that are inherently biologically significant (such as food, water and sex, i.e., USs), or stimuli which have previously acquired biological significance by means of their pairings with a US, appear to be resistant to backward and forward blocking (Denniston et al. 1996; Hall et al. 1977; LoLordo et al. 1982; Miller and Matute 1996).

1995; Van Hamme and Wasserman 1994). In this way, each of the CS1-US pairings of Phase 2 results in some unlearning of the association between the absent CS (CS2) and the US. Thus, this particular revision of the Rescorla-Wagner model can also explain backward blocking and other retrospective reevaluation effects. At the present time, either this negative activation view or the comparator view mentioned in the above paragraph (Baker and Mercier 1989; Miller and Matzel 1988) appear to be equally able to explain most of the data available on retrospective reevaluation effects. The main difference between these two views is that the negative activation view assumes that backward blocking is due to unlearning whereas the comparator view assumes that it is the result of a post-learning comparison of the strength with which each of the CSs is associated with the US. Further research is needed to better discriminate between these two views.
We generally speak of stimulus–stimulus (S–S) learning when an association is formed between the mental representation of two stimuli. The acquisition of these associations allows us to predict that the second stimulus will occur upon observation of the occurrence of the first one. Pavlovian (or classical) conditioning is an example of this type of learning. In this case, an initially neutral stimulus (the CS) becomes associated to a biologically significant US such as food or a painful event. Fear conditioning, taste aversions and drug addictions are some examples of classical conditioning. The S–S learning that occurs in classical conditioning frequently results in the elicitation of a conditioned response (CR). But S–S learning can also occur in situations in which no USs are involved and no CRs are elicited (as in the learning of associations between neutral events by humans or animals). The learning of causal relations is an example of S–S learning which does not necessarily involve USs and CRs. In S–S learning, contiguity between the two stimuli is an important factor in the formation of associations, but not the only one. Current theories on associative learning, such as that of Rescorla and Wagner, take into account not only contiguity, but also the degree to which the second stimulus is surprising. This theory can explain many of the learning phenomena described in this section but it also presents several problems which have not yet been fully solved (see text).

1. How many examples of Pavlovian conditioning can you think of?
2. What does the learning of causal relations have to do with Pavlovian conditioning?
3. How does the Rescorla–Wagner model explain blocking? Which are the strengths and weaknesses of this theory?

Response–outcome associations

So far, we have seen many examples of stimulus–stimulus (S–S) learning, but you have probably noted that what we call S–S learning does not cover all types of learning. One important feature of S–S learning is that it enables us and other animals to predict our environment (e.g., to predict a dangerous situation). However, it does not allow us to control our environment (for example, it does not allow us to avoid danger once we have predicted that a harmful event is about to occur). To use a different example, imagine a dog which is exposed to tone–food pairings. The animal can learn to predict when the food is about to be presented and will learn to react accordingly (e.g., the dog will salivate when the tone is presented). However, S–S learning alone does not allow the animal to control its environment. That is, the dog could not obtain more food if its only way of learning and responding were Pavlovian (i.e., S–S learning). This is because, in classical conditioning, the food and the tone are contingent (i.e., the presence of the food depends upon the tone), whereas the food and the dog’s behaviour are not contingent.

Thus, there are other forms of learning in which, instead of learning about the relationship between environmental events (e.g., tone and food), organisms can also learn about relationships between their own behaviour and environmental events. For example, a rat may learn to predict when a shock is about to occur, but it may also learn that pressing a lever prevents the shock from occurring. In such cases, we say that the rat has learned a relationship between its response (in this case, lever pressing) and an environmental outcome (no shock).

The learning of response–outcome (R–O) associations can be said to occur in much the same way as does the learning of S–S associations, although it raises some additional and interesting issues because the first event in the association is not an environmental event such as a CS, but is our own behaviour. Thus, how does our future behaviour change when our own behaviour, rather than a CS, is followed by a reinforcer such as food? Consider the rat in Figure 3.4. If the rat presses the lever, it will obtain food. Moreover, the occurrence of the food is now contingent on the lever-pressing response rather than on the occurrence of a CS. Of course the rat will also salivate in this situation, much the same as Pavlov’s dog did. However, we can also observe how the rat’s lever-pressing behaviour changes as it learns how to obtain the reinforcer (e.g., food). Once the rat has learned the relationship between the food and lever pressing, it will press the lever anytime that it is hungry. This process is called instrumental conditioning (or learning). Tolman (1932) defined instrumental behavior as purpserive behavior, because, unlike Pavlovian responding, instrumental responding occurs with the purpose of obtaining something or avoiding something (but not all psychologists agreed on this defi-
tion; see e.g. Skinner 1953). Thus, the responses that are acquired or maintained in this way are generally referred to as instrumental responses because they serve as instruments to obtain certain outcomes. Instrumental conditioning refers to how such instrumental responses are acquired and maintained; the learning of R-O associations is a more theoretical term which refers to the underlying learning which presumably takes place.

The learning of an R-O association may or may not be always reflected in an instrumental response. For example, a rat may have learned that pressing a lever produces a certain outcome, but the rat will ordinarily not press the lever unless it wants to obtain that outcome (e.g. Dickinson and Balleine 1994). Thus, the observation of instrumental responses implies that the R-O association has been acquired, just as much as the occurrence of a CR during Pavlovian conditioning indicated that the CS-US association had been acquired. However, like in Pavlovian conditioning, an absence of an instrumental (or Pavlovian) response does not necessarily mean that the organism has not learned the R-O (or the CS-US) association. As an additional example, if you learn an instrumental response that will produce money, such as accepting a baby-sitting job, you will probably be inclined to exhibit this response when you need money but not otherwise.

Edward Thorndike, who was the first scientist to study this type of learning in the laboratory, used cats and some ingenious puzzle boxes that he constructed to study instrumental learning (Thorndike 1898). His experiments consisted of putting a cat inside the box, and the food reinforcer nearby outside the box (see Panel A of Figure 3.5). If the cat wanted to get out of the box, it had to open the door by pressing a pedal that was located inside the box. In the mean time, Thorndike would assess how much time the cat took to get out of the box during successive learning trials. As you can imagine, a cat takes a long time to get out of such a box when it is first placed inside it. Then seemingly by accident, the cat presses the pedal by chance, the door opens and the cat gets out and then eats the food (see Panel B of Figure 3.5). The next trial would look much the same as the first one, though maybe a bit shorter. Gradually, the cat learns how to get out of the box. Once the cat has learned the relationship between the pedal and the escaping from the box, it will get out in just a few seconds. In this way, Thorndike was able to draw many

Figure 3.4 Schematic representation of the Skinner box. In the front wall, there is a lever that the rat can press. Lever presses are the instrumental response through which we assess learning in this box. Next to the lever, there is a cup where food pellets are delivered. This provides reinforcement for lever-pressing behaviour. The floor is constructed of metal rods, through which an electric current can be applied if the experiment deals with the effects of aversive stimuli (e.g. punishing the rat for lever pressing).

Figure 3.5 Thorndike’s puzzle box. Panel A shows the cat inside the box and the food outside. Pressing the pedal becomes reinforced by the opening of the door and access to food (Panel B). A learning curve is evident if we assess the time that the cat needs to get out of the door in the successive learning trials.
learning curves and, most importantly, developed the law of effect, which is the law that basically defines the way in which instrumental responding works. When a given response is followed by a pleasant consequence, this response will become more firmly connected to the environmental situation in which it occurred so that, in the future, the organism will tend to repeat the same response when in the same environmental situation. Conversely, when a given response is followed by an unpleasant consequence, the connection between the environmental stimuli and the response becomes weaker, and this reduces the probability that the response be repeated the next time that the subject is in the same environmental situation (Thorndike 1911).

Thorndike’s work has been continued by B.F. Skinner (1953) and many other psychologists over the years. Skinner preferred to work with rats and pigeons, rather than cats, and he constructed a new experimental apparatus which became very popular and is probably the most widely used apparatus in animal research even today. He called it the Operant Chamber, but most people call it the ‘Skinner box’. A schematic representation of it is shown in Figure 3.4. The front wall generally has a lever protruding from it that the rat has to press down to obtain food (or a plastic disc that the pigeon has to peck), as well as a cup where the food pellets (reinforcers) are delivered when lever presses occur. If the experiment is to study the consequences of aversive stimulation on the rat’s behaviour (e.g. the effect of punishment on the rat’s lever-pressing behaviour), an electric current can be applied through the floor of the box, which is made of metal rods. In present-day laboratories, the box is connected to a computer which presents the stimuli and reinforcers, as well as records the animal’s responses.

What type of experiments are generally conducted in instrumental learning? In instrumental learning we can study several phenomena that are highly analogous to those that were described with respect to S–S learning. That is, we can study acquisition, extinction, generalization, discrimination, contingency, blocking, overshadowing, and many other phenomena. In general, they work in much the same way, although there are also some specific aspects of R–O learning about which you should know.

**Aspects of R–O learning that are similar to those observed in S–S learning**

Extinction, generalization, discrimination, blocking, contiguity and contingency were described with regard to stimulus-stimulus (S–S) learning but also work in a similar way in response–outcome (R–O) learning.

After an instrumental response has been acquired, it will become extinguished if it no longer is followed by the outcome. In S–S learning, extinction occurred when Stimulus 1 (S1) no longer predicted Stimulus 2 (S2); in instrumental learning the process is similar but now it is the instrumental response (e.g. lever pressing) that is extinguished because it no longer is followed by the outcome. For example, the rat in Figure 3.4 would stop lever pressing if lever pressing no longer produced food. Similarly, when children try to obtain the attention of their parents by making a noise, that instrumental response could be extinguished if the parents did not produce the outcome for which the child is looking (i.e. giving the child their attention).

Generalization can also be observed in instrumental learning. If we teach the rat that food will be available only when a light is on, the rat will learn to press the lever with greater frequency when the light is on. However, if we test generalization by presenting a slightly different light, the rat will probably generalize the response and will press the lever in the presence of this second light, although probably somewhat less vigorously than in the presence of the original light. If we want the rat to press the lever only in the presence of one specific light, we have to use a discrimination training procedure similar to that shown with respect to S–S learning. That is, we must teach the rat that pressing the lever in the presence of one light (S+) will produce food, whereas pressing the lever in the presence of the other light (S−) will not produce food. Once this discrimination is acquired, we speak of stimulus control because the response is only produced in the presence of S+ (Skinner 1953).

Instrumental responses can also result blocked in both animal (Poulte and Hall 1978) and human subjects (Hammerl 1993; Shanks 1985) if there exists a more valid predictor of the reinforcer. Recall that blocking in S–S learning occurred when there were two potential causes of S2 (e.g. CS1 → US). If subjects had previous knowledge that CS1 produced the US, they tended to discount the potential causal role of the other stimulus (CS2) in producing the US. Thus, if you now imagine a situation in which both CS1 (an environmental event) and your own response (R) are potential causes or predictors of an outcome, and you have previous knowledge that CS1 can produce the outcome, you will probably discount (i.e. block) the potential causal role of your own response in producing the outcome.
For example, Shanks (1985) demonstrated this effect in an experiment with college students who played a video game. The students were supposed to发射 shells (R) at tanks that passed through a minefield (CS). 1) in the computer’s screen. The reinforcement was the explosion of the tank that occurred during some of the trials. But of course, it was not easy to know whether the explosion had occurred because the shot had been accurate or because the tank had hit a mine. Some of the students were allowed an observational phase before they tried to destroy the tanks. During the observational phase they did not perform any responses, and thus, they were able to observe the probability of the tanks exploding if they hit a mine. Thus, when the game started, these students already had formed the mine—explosion association (i.e. they had acquired some previous knowledge of the mine—explosion relation). What Shanks observed was that the attribute of causality that these students made with regard to the effectiveness of their own responses when they played the game was weak (i.e. was blocked) in comparison to the attribute that had been made by the students who had not been given the observational period (i.e. they had no previous knowledge of the mine—explosion association). Moreover, blocking occurred regardless of whether the observational period was given before (i.e. forward blocking) or after (i.e. backward blocking) the phase in which both the response and the minefield were paired with the outcome. This demonstrates that blocking of R—O associations can occur, just as blocking of S—S associations.

Contiguity and contingency also affect R—O associations in a way similar to S—S associations. For example, Wasserman (1990b) and Shanks and Dickinson (1987, 1991) have reported numerous experiments in which human participants were sensitive to the response—outcome contingency and contiguity under very different conditions. The response could be pressing a telegraph key, in some of the experiments (Wasserman), or shooting at tanks in a video game in the experiments (Shanks and Dickinson). The outcome could be, for example, a flashing light, or a tank exploding. In all conditions, the greater the R—O contiguity and contingency, the greater the attribution of causality that the subjects made concerning their response. If the outcome occurred with greater probability in the presence than in the absence of a response, the subjects would conclude that there was positive contingency between the response and the outcome (i.e. they concluded that the response produced the outcome). If the outcome occurred with greater probability in the absence than in the presence of a response, the subjects concluded that the contingency was negative (i.e. they concluded that the response prevented the outcome from occurring). If the two probabilities were alike, the students were also able to learn that the outcome was independent of their behaviour. Thus, the effect of contingency is similar in R—O and in S—S learning.

Finally, there is an issue which we have not yet mentioned, but that you may have missed. What about reinforcers such as money or social praise? Why do these stimuli reinforce our behaviour? They are what we call conditioned reinforcers or second-order reinforcers. That is, they have become reinforcers because they have been associated with primary reinforcers such as food or sources of pain. In human instrumental behaviour, conditioned reinforcers are often more important than primary reinforcers. And the same is true for Pavlovian conditioning. Second-order CSs can be conditioned if we first pair a CS (e.g. a light) with a US (e.g. food) and then we use that CS to reinforce another CS (e.g. tone—light). The second-order CS (in this case the tone) will also come to produce a conditioned response by means of its having been associated with a first-order CS.

Effects of reinforcement and punishment on instrumental responses

The process by which we increase the probability of an instrumental response by pairing it with a pleasant outcome is called reinforcement, and the process by which we reduce the probability of an instrumental response by pairing it with an unpleasant outcome is called punishment. Thus, although both types of learning involve the learning of response—outcome (R—O) associations, the effect on the response will be diametrically opposite, depending on whether the response is reinforced or punished.

There are two different types of reinforcement. For example, if we want to reinforce the rat in Figure 3.4 for pressing the lever, we can give the rat a positive reinforcer (e.g. food) for pressing the lever. However, we could also reinforce the rat with a negative reinforcer. Consider a rat in the Skinner box receiving shocks from time to time. If pressing the lever stops or prevents the shock from occurring, the lever-pressing behaviour will be reinforced. In this case, we speak of negative rather than positive reinforcement because it is the omission of the second event in the association (the shock) that reinforces the subject. Note that both positive and negative reinforcement can be seen as cases in which the
response produces a pleasant outcome. The effect of both types of reinforcement is an increase in the probability of the instrumental response. In the case of negative reinforcement we can subdivide it into escape behaviour (when the behaviour terminates the aversive stimulation, but does not prevent it from occurring) and avoidance behaviour (when the behaviour avoids or prevents the aversive stimulation from occurring). Thus, in summary, an instrumental response can be positively reinforced when its outcome is the occurrence of an appetitive event (e.g. money) and it can be negatively reinforced when the outcome of the response is either the termination (escape) or the avoidance (non-occurrence) of an aversive situation.

Similarly, we do not always need to give a shock to the rat in order to reduce its lever-pressing behaviour (punishment). We can also use omission training, which consists of omitting the presentation of food if the rat presses the lever and this should also reduce the probability of the response. You can probably think of many other examples of omission training, as when, for example, a child is being kept from watching TV after having done something that the parents want to prevent from happening in the future.

Interval schedules of reinforcement
In most real-life situations, reinforcement is not provided for every single response that the subject performs. Consider, for example, a parent who is not always present to provide reinforcement every time that the child produces a certain behaviour. Or consider a supervisor who provides feedback on a fixed weekly interval schedule (e.g. every Friday). Or consider also rain reinforcement for farmers, which occurs on a variable interval schedule. What type of behaviour can we expect when reinforcement occurs according to interval schedules?

Imagine two rats pressing a lever in the Skinner box to obtain food. For one of these rats, food is available on a fixed interval schedule. For example, one food pellet will be available every minute. This means that, regardless of how many responses the rat gives, these responses will not be reinforced until after that minute has elapsed and one food pellet is ready to be delivered. At this point, the first response that occurs once reinforcement is available will be reinforced. Thus, once the rat has learned how this schedule works, it will probably concentrate its responses around the last part of the interval, which is when food can be expected. The other rat is exposed to a variable interval schedule. For this rat, one pellet per minute will be available on average. However, the rat will never be able to know when exactly food is available because the schedule is a variable one. That is, sometimes food will be available after 30 seconds, sometimes after 2 minutes. Thus, even though the average is also 1 minute, the rat needs to respond more often and more regularly than under the fixed interval schedule because in the variable schedule, the rat cannot know when food will be available. Thus, many laboratory experiments have shown that rats learn to press the lever more often if they are given food on variable rather than fixed interval schedules, even though getting the food does not depend on how many times they press the lever (Ferster and Skinner 1957). As an additional example, consider the effects that may be produced in students' behaviour with a variable as opposed to a fixed interval schedule for examinations in a particular course. Studying habits would of course be more regular if the examinations were delivered on a variable rather than fixed interval schedule.

Ratio schedules of reinforcement
Now consider a company which does not pay their employees for work based on some time interval, but on the basis of the number of responses that they give, for example, the number of cars that they sell, or the number of letters that they send out during a mailing campaign. Or consider a rat that has to emit a certain number of lever-pressing responses in order to obtain a food pellet. If reinforcement is given on a fixed ratio schedule (e.g. payment is given after the mailing is complete for each of 1,000 customers), the person will probably work very hard in order to obtain payment and will probably then take a break before starting on the mailing for the next 1,000 customers. However, if reinforcers were given on a variable ratio interval (e.g. also 1,000 customers on average, but randomly distributed, so that sometimes takes more and sometimes takes less than 1,000 letters), these people cannot be certain of when reinforcement is going to occur, and thus, they tend to be more regular in their behaviour. Many animal experiments have shown that a much more regular rate of responding can be obtained if subjects are reinforced according to a variable, rather than fixed, ratio interval. This is a well-known principle in companies that make their profits out of gambling machines. Variable ratio schedules induce very high and regular response rates
Superstitious behaviour

Reinforcers are not always contingent (dependent) on the subject's behaviour. Sometimes, they occur contiguently (i.e. regardless of whether or not the subject responds). For example, in ancient civilizations, people invented rain dances that they thought would bring rain, but of course rain was response-independent. Even so, many people acquired response

outcomes (R-O) associations between the dances and

rain if their dancing behaviour was accidentally followed by rain from time to time (e.g., variable ratio schedule).

The first laboratory experiment on superstitious behaviour was conducted by Skinner in 1948. He gave

‘false’ food to pigeons every 15 seconds. That is, the

pigeons did not have to peck any key or do any other

type of job to obtain food. However, he observed that

pigeons developed a specific pattern of responding,
as if it ‘thought’ that that was the way to obtain food.

According to Skinner, this could allow us to explain human superstition. That is, perhaps humans develop superstitious behaviour in the same way that pigeons do. In brief, the first time that the food reinforcer-occurred, the pigeon must have been doing something, for example, RT. Thus, RT becomes reinforced and in consequence, its probability of occurring again in the near future was increased. This also augmented the probability of the next reinforcer occurring again in the presence of RT and in turn, of RT being reinforced again. According to Skinner, human superstitious behaviours are established in a similar way when they become accidentally reinforced (see Herrnstein 1968; Skinner 1948, for a more detailed elaboration of this view; see the main text to learn about some potential problems of this view as well as to know about related research conducted with humans).

(Ferster and Skinner 1957). Moreover, the behaviour reinforced under variable ratio schedules becomes highly resistant to extinction because the subject knows that reinforcement depends on the number of responses, but does not know how many responses are needed to obtain it.

Superstitious behaviour and illusion of control

You can probably think of many examples of superstitious behaviour. Some of them are acquired through social learning. For example, someone tells us that 13 is a bad-luck number, and most people try to avoid it, 'just in case'. As a result, many hotels avoid labelling floor 13 as such in their lifts and simply label floor 14 after floor 12. This is a social superstition which is transmitted by social interaction and we cannot be certain about how it came to be established many years ago. But there are also many personal superstitions and the establishment of these can be studied in the laboratory. Perhaps you and your friends have developed some of these personal superstitions that you may use when you are going to take an exam or when playing football or tennis. Some students like to wear black (or blue) when they are going to take an exam; others, in contrast, are more preoccupied by the route they take when they go to school; yet others think that the necklace that they wear is a good-luck amulet. Among football, tennis or golf players, these types of personal superstitions are also well known.

Skinner (1948) showed that pigeons, as well as humans, developed superstitious behaviours if non-contingent reinforcers occurred from time to time which accidentally reinforced what they were doing (see case study). Skinner’s experiment, however, presented some problems of interpretation (Staddon and Simmelhaug 1971). One of the problems was that Skinner’s data could be interpreted as the result of classical conditioning rather than superstitious conditioning. That is, Skinner had been giving food to the pigeons every 15 seconds, and thus the 15 seconds interval could have played the role of a classically conditioned CS which predicted food. If this were true, we should expect Pavlovian CRs to occur every 15 seconds or so. According to Staddon and Simmelhaug, what Skinner had observed in his pigeons were not superstitious instrumental behaviours but Pavlovian CRs. The responses were not arbitrary responses, as would be expected from Skinner’s view, but were responses appropriate to the nature of the anticipated US and timing of that US.
Nevertheless, many researchers have reported superstitious behaviour in human experiments which cannot be interpreted as classical conditioning (see case study). For example, Wright (1962) reported an experiment in which college students tended to develop different sequences of key presses despite none of them being related to the production of reinforcement. Reinforcement was simply given at different random intervals, but students tended to believe that the sequence of key presses that they were developing was what produced the reinforcers. Moreover, several researchers have shown that human subjects tend to perceive reinforcers that occur independently of their behaviour ('free reinforcers') as if they had produced them (e.g. Alloy and Abramson 1979; Langer 1975; Wortman 1975). For instance, Alloy and Abramson (1979) performed a study with college students who were pressing keys and obtaining different types of reinforcers under different R–O contingencies. Under some contingencies, the reinforcer was absolutely independent of the subject's behaviour (it was pre-programmed to occur with the same probability regardless of whether or not the student responded). However, students tended to believe that they were producing the reinforcers. This is called the illusion of control, because the subjects not only behave superstitiously, but also believe that they are controlling an outcome which is uncontrollable. More recently, Matute (1994, 1995) also observed superstitious behaviour and illusion of control in college students who were exposed to an uncontrollable negative reinforcer. The students were told that their task was to find the way to stop the noises that would be produced by a computer from time to time. Thus, the negative reinforcer was the termination of noises. But it was actually uncontrollable. That is, noise duration was pre-programmed for each trial and the student's responses did not have any effect on it. However, most students developed illusions of control. Very few students noticed that their responses were not controlling noise termination.

Thus, apparently, we can sometimes form R–O associations even when reinforcers are independent of the response. The probability of the occurrence of the reinforcer is one of the factors that affect this result. That is, the students who received a larger number of free reinforcers developed higher illusions of control than the students who received a smaller number of free reinforcers. But this is surely not the only factor affecting the illusion of control. For example, Alloy and Abramson also showed that non-depressed subjects developed stronger illusions of control than did depressed subjects, and that in normal subjects, receiving uncontrollable reinforcers produced stronger illusions of control than receiving uncontrollable punishment (see also research update on p. 93 to learn more about this).

Nevertheless, it is important to note that despite the many experiments demonstrating superstitions and illusions of control, there are also many experiments that have shown that when reinforcers are uncontrollable, animals (e.g. Killeen 1981) and people (e.g. Shanks and Dickinson 1987; Wasserman 1990b) are able to realize that they are not controlling the reinforcers. For example, Killeen (1981) performed an experiment to test whether pigeons would discriminate between an outcome produced by their own behaviour and an outcome produced by a computer. The pigeons could peck at three different keys in a row. Pecking at the central key sometimes produced the effect of turning off the light. However, from times to time, the light was also turned off by the computer. The pigeon's task was to learn whether the light had turned off because of its pecking or because the computer had turned it off. If the computer had turned off the light, the pigeon had to 'say' this by pecking at the left-side key, and if it was true, the pigeon was reinforced with food. By contrast, pecking at the right-side key meant 'I turned the light off', and this response was followed by food if it was true that the pigeon, rather than the computer, had turned the light off. The pigeons were incredibly accurate in their responses. Thus, if an accurate detection of non-contingency is possible, why, then, do people and animals sometimes behave superstitiously? The research update addresses this problem.

Learned helplessness

The term learned helplessness refers both to an experimental effect and to one of the theories that attempts to explain that effect. Moreover, it also refers to a model of human depression which was later developed as an extension of the theories and data that were developed in the animal and human laboratories.

The learned helplessness effect was first demonstrated by Overmier and Seligman (1967) and has been extensively investigated in animal and human research (e.g. Hiroto
Illusion of control and superstitious behaviour versus accurate judgements of R-O independence and learned helplessness

When we are exposed to uncontrollable reinforcers, do we learn that reinforcers are independent of our behaviour, or do we, by contrast, behave superstitiously and develop illusions of control? As we have seen, the experimental results are quite mixed. Some experiments show that we notice that we do not have control; others show that we do not notice. Why?

One potential explanation is that in order to learn that an outcome occurs independently of our behaviour, we have to test both what happens when we perform the response and what happens when we do not, and this is something that we do not always do. For example, Matute (1986) observed that student subjects who were trying to turn off uncontrollable noises produced by a computer tended to respond at every opportunity. In consequence, although the noise termination was always controlled by the computer, the subjects did not notice that the noises would have equally stopped even if they had done nothing. Because uncontrollable reinforcers occur with the same probability regardless of whether we respond or not, if we respond at every opportunity they will always become associated to our own behaviour rather than to other causes. Indeed, when a different group of subjects was explicitly instructed that they should refrain from responding from time to time in order to test how much control they had over noise termination, the uncontrollable reinforcers (noise termination) occurred both in the presence and in the absence of the response, and these subjects were able to learn that they had no control. Possibly, in this latter group, the reinforcers become associated to causes different from the subject behaviour (e.g., the computer program) and hence, S-O associations were formed that competed with the R-O association, thus reducing the illusion of control.

Similar processes can also be found outside the laboratory. For example, a person who has been diagnosed with an incurable illness has several courses of action. One is to do nothing and acknowledge that reinforcers do not depend on her or his behaviour; this may produce depression and helplessness. Another possible course of action may be to try all types of therapies, including the most superstitious ones (e.g., witchcraft). If transient recoveries occur, they will be associated with those ‘therapies’. This will produce an illusion of control and a superstition, but note that it might also be preventing a depression. Indeed, in cases in which the outcome is uncontrollable, letting patients keep their illusions and superstitions, or even favouring the development of new ones, can sometimes have prophylactic effects against helplessness, depression and other problems (see Alloy and Abramson 1988, Alloy and Clements 1992).

Note also that there are many factors which reduce the tendency to respond and which, therefore, should tend to reduce the illusion of control (e.g., fatigue, extinction, punishment, depression and many others). For example, the finding by Alloy and Abramson (1979) that depressed people are less prone to develop illusions of control than non-depressed people could be due to depressed people being generally more passive than non-depressed people. As noted by E.A. Skinner (1985), the low rate of responding probably prevents the development of R-O associations in depressed patients because uncontrollable reinforcers are more likely to occur in the absence than in the presence of their responding (see Matute 1986; Skinner 1985, for further elaboration of this view). Of course, this is not to say that the rate of responding is the only factor affecting the development of the illusion of control versus accurate judgements of response-outcome independence. The important point for our present purposes is that even though helplessness and superstition, and accurate versus illusory acquisition of R-O relations, may seem to be contradictory experimental results in the scientific literature, they all appear to occur also in real life, and are, probably, opposite ends of the same continuum.
and Seligman 1975; Maier and Seligman 1976; Seligman and Maier 1967). In brief, many experiments have shown that dogs can be trained to escape shocks by jumping over a barrier that leads to a safety compartment or by performing other types of instrumental responses. However, if the dogs are previously given shocks that are unpredictable and uncontrollable (i.e. shock termination does not depend on the dog's behaviour), dogs will later be less able to escape shocks when given the opportunity to do so. Moreover, the consequences of being exposed to uncontrollable and unpredictable shock were not limited to the subsequent failure to escape from shocks that were escapable. The dogs also developed several other deficits which have been described as emotional deficits (which are presumably similar to the sad and depressed state that a person feels after learning that reinforcers are uncontrollable), motivational deficits (the dogs did not even try to escape the shocks when they were later given the opportunity to do so), and cognitive deficits (the dogs were unable to learn that the shocks were controllable during the last phase of the studies).

These effects could in principle be explained in several different ways. For example, perhaps all of the dog's responses were extinguished because none of them was followed by the reinforcer (shock termination). Or perhaps a passive behaviour became superstitionally associated with shock termination (e.g. Balleine and Job 1991). Another potential explanation, and the one that has become most popular, is the so-called learned helplessness theory (Abramson et al. 1978; Seligman 1975).

According to this theory, the reason that the dogs showed these deficits was because they had learned to be helpless during Phase 1 of the study. That is, they learned that nothing that they could do allowed them to control the reinforcer (shock termination). In each trial, shock termination was controlled by the experimenters, and the dogs were able to learn that they had no control on shock termination. It is important to note that, according to learned helplessness theory, it is the lack of control over reinforcers, rather than the absence of reinforcers, that produces the deficits (see Abramson et al. 1978; Seligman 1975). The dogs in the control condition had been exposed to the identical reinforcers (shock termination occurred with the same probability and identical parameters), but these dogs were not helpless because they were not actually the experimenters, were the ones that were controlling shock termination. If the effect were due to an absence of reinforcers, the effect could be explained by the extinction of all voluntary responses. However, because the effect appeared to be due to a lack of control over reinforcement, Seligman and his colleagues concluded that subjects had actually learned that reinforcers were uncontrollable, and that this was what produced the helplessness effect.

The learned helplessness theory has become very popular as a model of human depression and of several other human disorders (e.g. school failure). According to the learned helplessness model of depression (Abramson et al. 1978), a person who is exposed to uncontrollable outcomes will learn that no one response can control those outcomes and thus this person may develop the expectancy that the desired outcomes will remain uncontrollable in the future. If this occurs, this person will become depressed. As in the animal learned helplessness experiments, human depression frequently includes an emotional deficit (being sad and anxious), a motivational deficit (a depressed person frequently says: 'nothing will work, so why try?') and a cognitive deficit (according to this theory, depressed persons frequently have problems in learning that an event is controllable).

But can subjects really perceive the lack of relation between their responses and the outcome? Learned helplessness research has not tested this question (see Maturi 1994). However, if we look at the superstition and the illusion of control literature, they suggest that subjects receiving uncontrollable reinforcers frequently (not always) perceive them as controllable (see above). According to learned helplessness theory, learned helplessness effects could not take place if subjects do not detect that reinforcers are uncontrollable. Of course, if reinforcers never occur, subjects would perceive such absence of reinforcers and would stop responding, but that would be extinction rather than helplessness. Thus, learned helplessness theory needs to specify under which conditions animals and humans can detect that reinforcers are uncontrollable, because only under those conditions can they become helpless (Maier and Seligman, 1976). The study of the illusions of control (see pp. 91–2 and also the research update) can probably shed some light on the question of when can uncontrollable reinforcers be perceived as uncontrollable.

S–R versus R–O associations

As previously mentioned, according to Thorndike's (1911) law of effect, whenever a response was followed by a pleasant consequence, this strengthened the stimulus–response (S–R) connection between the environmental stimuli (S) present in that situation and the response (R) that the subject had performed. In this way, the probability that under the same environmental situation (same S), the same response would be repeated in
the future was increased. Thus, Thorndike, as well as many other psychologists, thought that instrumental behaviour was due to S-R associations that were acquired by contiguity. However, modern psychology has shown that organisms also learn response-outcome (R-O) associations and that these associations are important in determining their instrumental performance (Colwill and Rescorla 1986; Dickinson and Shanks 1995). Moreover, we also know now that, as in stimulus-stimulus (S-S) learning, contiguity is not the only important factor determining the learning of R-O associations and instrumental performance. As in Pavlovian conditioning, contingency and blocking effects also show that instrumental learning is more complex than the initial contiguity theories had assumed. But how do we really know that organisms can acquire R-O associations rather than simply strengthening the S-R connections? Some of the experiments that demonstrated the existence of R-O associations are described next.

Figure 3.6 shows a rat in a T-shaped maze, which is a very commonly used apparatus in instrumental learning. Generally, there is food in a box which is located at the end of one arm of the maze and there is no food at the end of the other arm. S-R theorists believed that although the rat at first chose at random (represented in Figure 3.6 by the rat tossing a coin), the finding of food in one of the end-boxes served to reinforce the response that the rat had just performed (e.g. turn left), so that, in the future, the probability that that response would occur when the rat is in the T-maze was increased. The rat would no longer chose which way to turn in a random manner.

One experiment by Tolman and Gleitman (1949) demonstrated that the rat's behaviour was not that simple. They used a maze with a distinctive box at the end of each arm (one was white, the other one was black). During Phase 1 of the study, food could be obtained in either box. Thus, according to the S-R view, both responses should have become equally reinforced and the rat should have kept choosing at random which way to go. And this was true. The rats kept 'tossing a coin' in deciding to go right or left because food could be obtained in either direction. However, during Phase 2 of the study, the rats were put into each box by hand during several trials. In one box, say the white box, they were shocked. In the other one they found food. Then in Phase 3, the rats were allowed to run the maze again. Which way would they go? According to S-R theory, both responses, right and left, had been equally reinforced and thus rats should keep on choosing at random. However, if they had learned an R-O association (left leads to white) and an S-S association (white leads to shock), then rats should certainly choose right. And of course, that is what they did.

Another study that showed the importance of R-O associations in instrumental learning was reported by Adams and Dickinson (1981). They trained hungry rats to press a lever to obtain food pellets in an operant chamber. Later, they conditioned a taste aversion to the pellets. This was accomplished by injecting the rats with a drug that made them feel sick after they ate some pellets, but the lever was not present at this stage and the rats were simply given the pellets without the requirement of any instrumental response. After this aversion to pellets was acquired, the rats were again given the opportunity to lever press in the operant chamber. No pellets were provided during this test. But would the rats press the lever? According to S-R theory, the acquired taste aversion should not affect the lever-pressing behaviour. That is, if the reason why they lever press is simply that such a response has been strengthened by reinforcement during the first stage of the study, rats should again lever press when given the opportunity to do so. However, if rats had really learned an R-O association (i.e. lever pressing produces pellets), they should not show much interest in lever pressing once they feel nausea for pellets. And this is just what the results showed. Rats no longer pressed the lever after they had acquired a taste aversion to the pellets. Thus, this study also demonstrated that an R-O association was what had been acquired and that it was governing the rat's behaviour (see also Colwill and Rescorla 1985 for further evidence of this). Now we may ask, how are these R-O associations acquired? We do not yet have a complete theory of R-O learning, but many researchers assume
that, at least in principle, the same theories that explain the acquisition of S–S associations can explain the acquisition of R–O associations (see e.g. Mackintosh 1983).

We generally speak of response–outcome (R–O) learning when the events in the association are an instrumental response and the outcome that it produces. This type of learning is presumably responsible for the behavioral changes that are often observed in instrumental learning. In general, organisms will tend to perform those responses that they expect to be reinforced (either positively or negatively reinforced) and will not perform those responses that they expect to be punished. Although older theories did not speak of R–O associations, we now know that rats (and humans) will not perform an instrumental response (no matter how strongly that response has been reinforced), if they expect that response to produce an outcome that they do not want. Thus apparently organisms acquire a mental representation of the outcome produced by their response and this allows them to decide whether or not they want to perform the response. Although R–O associations generally reflect an accurate causal relation between the response and the outcome, superstitious behaviors and illusions of control can also be sometimes acquired in situations in which the outcome does not depend on the response. Extinction, generalization, discrimination, blocking and many other phenomena that were described with regard to S–S learning have also been reported in R–O learning situations.

1. What is the difference between Pavlovian and instrumental conditioning?
2. How do we know that animals acquire R–O associations?
3. What does superstitious behavior have to do with the learning of R–O associations?

‘Special’ types of learning

There are many other types of learning that we have not yet discussed. Imitative learning, category learning and spatial learning are some examples of situations that have traditionally been thought of as ‘special’ forms of learning that could not be explained by associative theories. But this was in part due to the old theories of learning not being able to explain those effects. For example, imitative learning occurs when we observe someone doing something and being reinforced for it, and then we tend to imitate that behavior (Bandura 1977). Of course, the old stimulus response (S–R) theories could not explain such learning because the subject was not performing any response while learning was taking place through observation of the model’s behavior. That is, not one of the subject’s responses could be reinforced because the subject was not performing any response. However, current learning theories could explain imitative learning as a form of associative learning. Throughout this chapter, we have seen evidence suggesting that associations are formed between mental representations of events and that responses are not necessary for learning to occur. For example, in sensory preconditioning the animals were able to associate the representation of two neutral events. And in response–outcome (R–O) learning the animals were able to associate a response with an outcome and to refrain from producing the response if the outcome had been devalued (as in the cases in which the outcome ‘food pellets’ was paired with nausea after the R–O association had been acquired). Thus, we know that rats (or people) would perform a response if they have learned that that response produces a desirable outcome and would not perform the response if they know that it leads to an undesirable outcome. Thus, if we observe a person performing a response and obtaining a given outcome, we can form an association between that response and that outcome, just as we can associate any other type of events that we observe. Once we have acquired such R–O association we can later imitate or not the response as a function of whether we wish to obtain that particular outcome. Similarly, spatial learning and category learning are also being found to be explicable in terms of associative learning. Some examples of this are described below.

Spatial learning

Many researchers have been curious about the way we learn spatial relations. How do we find our way through a new city, for example? Or to use a laboratory example, how does a rat find its way through a new maze? Tolman (1948) suggested that rats, and humans, build up cognitive maps of spatial locations
such as mazes and cities while they learn, and they later use these maps when they need them (see Figure 3.7). This seems a reasonable explanation with which many scientists agree. But it has the problem of being rather vague. That is, what exactly is a mental map? Moreover, this explanation presents the problem of explaining spatial learning as a type of learning which is different from other types of learning; that is, spatial learning is treated as an exception to the general rules of learning. In consequence, many researchers have tried to determine if spatial learning is really an exception. The principle of parsimony in science tells us that, if we can have one simple explanation that can explain all forms of learning, such an explanation should be preferred over the use of different explanations for each individual situation. Thus, researchers have tried to determine if spatial learning could be explained by our already well tested associative mechanisms and have apparently found one such way. Seemingly, rats (and people) use cues in their mazes (and cities) that help them find their way to their goal (see Figure 3.8). These cues can play the role of S1 in an S1-S2 learning and become associated with the goal (S2). Moreover, they can also play the role of discriminative stimuli which indicate to the subjects when an instrumental response will be reinforced (as when a certain light in the Skinner box indicated that food was available and a different light indicated that food was not available). In this way, the subject can learn in a maze which cues indicate that a particular response (e.g., turn left) will be reinforced or punished.

In an experiment which illustrates this point, Rodrigo et al. (1997) used a circular swimming pool to study spatial learning in rats. The pool was full of water, but there was an underwater platform in a certain location. The rats had to swim through the pool and their goal was to reach the platform which would allow them to step on it and take a rest. These researchers also added a few cues through the pool that would help the rats find where the platform was located. Indeed, the only way by which this problem could be solved was by learning about those cues because the platform was consistently located in relation to them. The rats had no trouble in getting to the platform after they had learned where it was located in relation to the cues (which can be regarded as several S1s which became associated to the platform, i.e. S2). However, when a new (and redundant) cue was added during Phase 2 of the study, the rats did not learn to use it. Apparently, they had already learned to predict where the platform was by using the other cues, and thus the learning about the cue which was added during Phase 2 was blocked. This shows that the learning of spatial relations can also be blocked by using a procedure which was known to produce blocking in S-S and in R-O associative learning. This suggests that spatial learning is probably just one more instance of associative learning rather than a special case of learning.

**Category learning**

Consider now when in your childhood you were learning to categorize items as furniture as opposed to non-furniture or as games versus non-games. Consider how medical students learn to categorize symptoms under disease categories. Moreover, consider how pigeons may learn to distinguish the concept of fish from that of non-fish by pecking only at pictures that show fish on them (Herrnstein and deVilliers 1980). Category learning has
been frequently classified as a 'non-associative' form of learning. However, researchers are now finding that the same associative principles that we have described throughout this chapter can be applied to category learning. Moreover, the observation of category learning in animals, such as the example above of pigeons learning to classify pictures as fish versus non-fish adds support to the idea that category learning is not a special type of learning limited to the human species (see also Wasserman et al. 1992, to learn more about concept learning in animals).

Gluck and Bower (1988) noted that category learning could be explained by associative principles similar to those used to explain conditioning experiments. They described a simple computer simulation that was able to learn to categorize items using the Rescorla-Wagner model as its learning equation. After this, many other computer programs have been written that have improved upon the initial simple program described by Gluck and Bower, and that have contributed to a current renewed interest in associative theories, not only with regard to the psychology of learning, but also within other areas of psychology. The new programs can learn to categorize new items with great accuracy (e.g. Kruschke 1992). Moreover, the results provided by these programs are similar (both in successes and in errors) to those produced by animal and human subjects who are trained to perform the equivalent categorization tasks. Thus, we can now also explain category learning through associative principles.

As a final exercise, consider writing a learning equation (e.g. Rescorla and Wagner 1972; see Mercier 1996) in your computer. By doing so, you would be telling your computer how the associations between S1 and S2 should be strengthened every time that S1 and S2 occur together. Once your program is finished, you can start the learning stage by introducing several S1–S2 pairings. For example, describing a particular chair (S1) followed by the label 'furniture' (S2) will be an example of an S1–S2 pairing. When you present each new S1, your computer should give a response by telling which S2 is it expecting (e.g. furniture or non-furniture). Then, you can provide feedback on whether the computer's response was correct, just as you would do with a child or a pigeon, in order to allow learning to take place. In this way, the system will start acquiring associations between S1s and S2s and those associations will be strengthened or weakened according to the learning rule that you have implemented in your equation. Of course, as in any other learning situation, there will be many failures during the earlier phases of learning. But as learning proceeds, the responses should be more and more accurate, assuming that the theory that your equation represented was a good theory. Once you have introduced a sufficient number of different S1–S2 pairings, the system should have learned to categorize those items. That is, the system should be able to recognize each S1 as a member of its corresponding S2 category. Moreover, responding to new items that the system has never 'seen' before can be accomplished by generalization (the more similar a new item is to those stored under the category representation, the greater the probability that the new item will be classified as a member of that category).

Thus, like spatial learning, category learning does not appear to be a 'special' type of learning. Whether other forms of learning which currently may still appear to be outside of the domain of associative learning will someday be integrated within the same body of associative knowledge that applies to most forms of learning is something that will be decided by the research performed by future generations of psychological researchers, or, in other words, by you and your colleagues.

Several types of learning have been traditionally regarded as exceptions to the laws of learning because the older stimulus–response (S–R) theories were not able to provide a satisfactory explanation of how they occurred. Examples of these are imitative learning, spatial learning and category learning. Current theories, however, assume that responses are not necessary for learning to occur, and that learning consists of the acquisition of associations between mental representations of events (be they responses or environmental stimuli). As a consequence, current developments are showing that many of those 'special cases' no longer need to be regarded as exceptions to the laws of learning. Current associative theories, such as that of Rescorla and Wagner, can provide a reasonable explanation of these types of learning.

1 Are cognitive maps always necessary to explain spatial learning? Why?
2 How can we explain category learning as an instance of associative learning?
Developing a theory of learning

If you are curious about how we learn, developing a theory of learning is no more than trying to solve that question. Most current theories explain learning as the process through which we acquire associations between mental representations of events. In this chapter, the strengths and weaknesses of major theories are described, and critical human and animal experiments that help assess the contributions of each theory are discussed.

Stimulus-stimulus associations

We generally speak of stimulus-stimulus (S-S) learning as an association is formed between the mental representations of two stimuli. Pavlovian conditioning is a type of S-S learning in which an initially neutral stimulus (the conditioned stimulus or CS) becomes associated to a biologically significant stimulus (the conditioned stimulus or US). As a result of this learning, a conditioned response (CR) is often observed. But S-S learning can also occur in situations in which no USs are involved and CRs are elicited, such as, for example, in many human social learning situations.

Response–outcome associations

We generally speak of response–outcome (R-O) associations when the events in the association are an instrumental response and the outcome that it produces. This type of learning is presumably responsible for the behavioral changes that are often observed in instrumental learning. Current research suggests that organisms acquire a mental representation of the outcome produced by their response and this allows them to decide whether or not they want to perform the instrumental response.

Special types of learning

Several types of learning have been traditionally regarded as exceptions to the law of learning because older theories were not able to provide a satisfactory explanation of how they occurred. Examples of these are imitative learning, spatial learning and categorization learning. Current developments, however, are showing that many of those special cases no longer need to be regarded as exceptions to the laws of learning.

Further reading

- Dickinson, A. (1980) *Contemporary Animal Learning Theory*. Cambridge: Cambridge University Press. This book provides excellent discussions on current learning theories and on the type of learning research that psychologists are conducting today. It is a worth reading it after you have read a more introductory textbook (e.g. Domjan or Tarpy).
- Pavlov, I. (1927) *Conditioned Reflexes*. London: Clarendon Press. This is a classical book on learning which has inspired generations of psychologists. Even though many of Pavlov's views have been challenged by later discoveries, many are still of great value and you should read this book to form your own opinion on learning and conditioning.
- Skinner, B.F. (1953) *Science and Human Behavior*. New York: Macmillan. Like Pavlov's, Skinner's book is another classic. It has often been challenged by later discoveries but has had a profound influence in the psychology of learning and in the development of applications of learning theory. A must-read book.

Acknowledgements

This chapter was written under support of grants PB95-0440 and PB96-006 from Dirección General de Enseñanza Superior (Spain) and from Departamento de Educación, Universidades e Investigación (Basque Government), respectively. I would like to thank Lourdes Albóniga, Victoria D. Chamizo, Ralph R. Miller, Oskar Pineño and Pedro Villegas for their highly valuable comments on an earlier version of this chapter. Special thanks are due to Martha Escobar for the excellent artwork that she has contributed to this chapter.