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Is conditioning a useful framework for understanding the development and treatment of phobias?☆

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Abstract

Despite the prevalence of therapeutic interventions based on conditioning models of fear acquisition, conditioning has been seen by many as a poor explanation of how fears develop: partly because research on conditioning has become less mainstream and models of learning have become increasingly more complex. This article reviews some of what is now known about conditioning/associative learning and describes how these findings account for some early criticisms of conditioning models of fear acquisition. It also describes how pathways to fear such as vicarious learning and fear information can be conceptualised as forms of associative learning that obey the same learning rules. Some popular models of conditioning are then described with a view to highlighting the important components in learning. Finally, suggestions are made about how what we know about conditioning can be applied to improve therapeutic interventions and prevention programs for child anxiety.

Conditioning as an explanation of phobic responding arose from Watson and Rayner's (1920) famous demonstration that aversive and avoidant responses towards a previously neutral stimulus could be learned. In their study, a 9-month-old child, Albert B, was pre-tested to see whether he was initially fearful of various stimuli (including a white rat and the noise made by banging a claw hammer on an iron bar). Having established that Albert was not fearful of the rat but was scared by the noise, Albert was placed in a room with the rat and every time he touched the rat, or the rat approached him, Watson hit the iron bar, thus scaring the child. After several pairings of the rat with the loud noise, Albert began to show signs of anxiety when the rat was presented without the loud noise. Although Watson himself did not formulate a coherent theory of phobia acquisition, the implication from the study was that excessive and persistent fear (i.e. a phobia) could be acquired through experiencing a stimulus in temporal proximity to some fear-inducing or traumatic event.

In conditioning terminology, this stereotyped and oversimplified account would suggest that the rat was a conditioned stimulus (CS) and the loud noise an unconditioned stimulus (US), which is a stimulus that evokes a natural response called the unconditioned response (UR): in this case anxiety. Through pairing of the CS and US, and the formation of a CS–US association in memory, the CS comes to evoke a response called the conditioned

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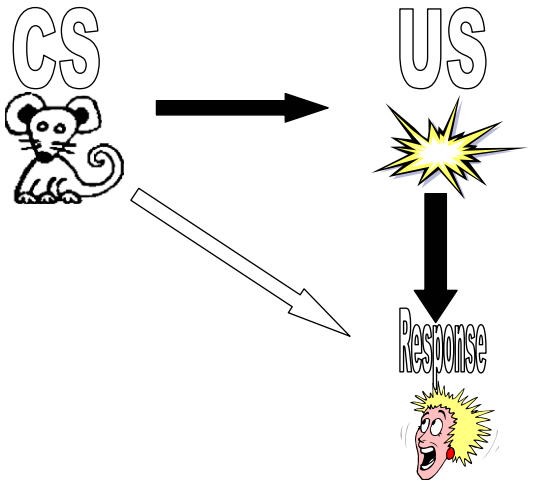

Fig. 1. Shows the stereotypical conditioning model of phobia acquisition using the example of Watson and Rayner (1920). A previously neutral stimulus, CS (a white rat) is paired with an aversive outcome, US (in this case a loud noise) which naturally evokes an anxiety response (UR). Through association with the aversive outcome, the CS comes to evoke an anxiety response (CR).

response, CR (see Fig. 1). Clinically speaking, conditioning at the time had two important characteristics: *Equipotentiality* and *Extinction*. Equipotentiality refers to the observation that any predictor should be able to enter into an association with any outcome; the implication is that a phobia of anything can develop provided that it is at some point experienced alongside trauma. Extinction refers to the well-demonstrated effect that if a predictor (CS) is presented alone (i.e. without the outcome) after a response has been acquired, then the strength of that response will decline over successive trials until the predictor stimulus no longer elicits a CR. The implication of extinction for clinical psychology was that exposure to the CS (the target of the phobia) without the trauma would allow the anxious response to extinguish. This simple idea formed the basis of behaviour therapy (see Wolpe, 1961) which even to this day is successful in treating specific phobias (Öst, Svensson, Hellstrom, & Lindwall, 2001).

1. Evidence for conditioning as a pathway to fear

Although ethical considerations prevent detailed study of the role of conditioning in the development of anxiety in childhood, naturalistic studies support the idea that conditioning is a mechanism through which fears develop. For example, compared to control children, 29 child survivors of a severe lighting-strike showed more numerous and intense fear of thunderstorms, lightening and tornadoes (Dollinger, O'Donnell, & Staley, 1984) and 25 teenage female survivors of a sinking cruise ship also had an excess of fears relating to ships, water travel, swimming and water, and their fear even generalised to other modes of transport (Yule, Udwin, & Murdoch, 1990). Both of these studies support the idea that a single traumatic event can lead to intense fears of objects related to the trauma. There is also retrospective evidence that anxious children (or their parents) will attribute fears to direct traumatic experiences. King, Gullone, and Ollendick (1998) reviewed seven studies that had looked at self-reported attributions of childhood fear and found that conditioning was endorsed as an explanation in 0% (for fear of water) to 91% (fear of dogs) of the children. The weighted mean endorsement of conditioning across these studies was 38%. With the notable exception of fear of water (in which 78% endorsed a 'fear at first contact' explanation), nearly all of the endorsements were direct conditioning, learning through observing others (vicarious learning) or through receiving fear-relevant information. In fact the weighted mean percentage of children or parents endorsing these combined categories was 94%. What I hope to demonstrate in due course is that all three of these categories are, in fact, forms of associative learning with the same underlying mechanism. As such, conditioning, as a theoretical framework, has enormous power to explain how fears develop (at least in terms of phobics' attributions of the origin of their fear). However, before then we shall look at some of the criticisms traditionally directed at conditioning as an explanation of fear, and then explore some lesser-known aspects of conditioning theory.

2. Criticisms of conditioning as an explanation of phobias

Despite the success of therapies based upon it, conditioning as an explanation of how phobias develop has come under fire. Rachman (1977) identified several problems with the conditioning model as it stood at the time:

1. *Some phobics cannot remember an aversive conditioning experience at the onset of their phobia*: Many phobics, such as snake and spider phobics (Davey, 1992) and height and water phobics (Menzies & Clarke, 1993a,b), do not remember an aversive conditioning episode. However, others do (e.g. Davey, 1989a; Dinardo et al., 1988, have shown that dental and dog phobics respectively can remember traumatic events). Also, for a particular feared stimulus some individuals do remember an associated traumatic event while others have no such memory (Withers & Deane, 1995). Although much of the evidence suggesting that phobics do not report traumatic memories is retrospective (often many years after the onset of the phobia) and, therefore, prone to memory biases (see King et al., 1998), these findings are nevertheless problematic for simple conditioning explanations, which rely on a traumatic learning episode.
2. *Not all people experiencing fear or trauma in a given situation go on to develop a phobia*: I have already suggested that the memory profiles of individuals with the same fears differ. However, there is specific evidence that not all people who have traumatic incidents while at the dentist (Lautch, 1971) or flying (Aitken, Lister, & Main, 1981), or who experience severe thunderstorms (Liddell & Lyons, 1978) go on to develop phobias. A simple conditioning account of fear acquisition simply cannot explain why similar experiences produce phobias in some, but not others.
3. *Incubation*: Eysenck (1979) noted that although conditioning theory would predict that fear should decrease over successive non-reinforced presentations of the CS, the opposite is often true. For example, when a spider phobic subsequently comes into contact with spiders, each spider is unlikely to be paired with a traumatic event, so fear should decrease (extinction), but instead the phobic becomes more fearful of spiders. Eysenck called this phenomenon the incubation of fear.
4. *The uneven distribution of fears*: the law of equipotentiality already described suggests that all stimuli are equally likely to enter into an association with an aversive consequence. Consequently, fears and phobias should be evenly distributed across stimuli and experiences. However, they are not: phobias of spiders, snakes, dogs, heights water, death, thunder, and fire are much more prevalent than phobias of hammers, guns, knives, and electrical outlets yet the latter group of stimuli seem to have a high likelihood of being associated with pain and trauma (see Seligman, 1971). There are also common themes that appear in the fears of 'normal' children (Ollendick & King, 1991) across different countries (Burnham & Gullone, 1997; Gullone & King, 1992). In addition, attempts to replicate the 'little Albert' study have been successful when the CS had some biological relevance (Valentine, 1946) but not when the CS was not ecologically significant (Bregman, 1932). Relatively recent laboratory studies have also shown that people acquire conditioned fear responses more rapidly to some stimuli (so-called 'fear-relevant' stimuli) such as snakes and spiders than to 'fear-irrelevant' stimuli such as rabbits and flowers (see Öhman & Mineka, 2001, for a review).
5. *Indirect pathways to fear*: Rachman (1968, 1977) noted that fears can be acquired not only directly but through verbal information and vicarious learning (learning through watching other's reactions). This observation implies that conditioning alone is not an adequate explanation of how phobias develop.

3. What is conditioning?

The term 'conditioning' can mean many things to many people. Already I have used the term to describe a paradigm (Watson and Rayner's pairing of a rat with an aversive outcome), a response (the anxiety acquired by little Albert is said to be 'conditioned') and a theoretical framework or underlying mechanism (I've talked about conditioning 'models'). Eelen (1980—cited in De Houwer, Baeyens & Field, 2005) believes that the term can and should be applied to all of these situations and should not be tied to a particular dogma (for example, the idea that conditioning effects result from automatic rather than conscious processes). As such, in considering whether conditioning is a useful framework for understanding the development of phobias I will consider conditioning as an effect (anxiety is an effect of pairing a stimulus with trauma) and explore the utility of applying the functional characteristics of conditioning (as a procedure) to the development of anxiety. Theories of conditioning have

changed over the years (and continue to develop), but they all agree that learning is driven by an association between stimuli (*associative learning*—see below). As such, I will also consider whether viewing anxiety development in terms of associative learning is useful.

4. Some things you might not know about classical conditioning

The conditioning model described until now and the criticisms of it ignore much of what is now known about conditioning processes in non-human animals and humans. In addition, many widespread myths exist about conditioning which have hindered conditioning being taken seriously as a framework for studying the development of phobias. However, conditioning research has established a great deal about what happens during a conditioning procedure and what factors moderate conditioned responses. This section explores some aspects of conditioning (and ignores a great many more) that are, perhaps, not widely known within the clinical domain. First, we will consider what is learnt during a conditioning episode and the impact of experiences prior to a conditioning episode.

4.1. Conditioning is driven by CS–US associations

Broadly speaking there are two possibilities about what happens during conditioning: (1) an association is formed between the CS and the UR (i.e. an association is formed between the potential phobic stimulus and the organism's *reaction* to the trauma); or (2) an association is formed between the CS and the US (i.e. an association is formed between the potential phobic stimulus and the trauma itself). It has been long-established that the later of these is true: both animals and humans tend to learn associations between the CS and US during classical conditioning, and it is this association that mediates the CR. There are two reasons to think that CRs are mediated by CS–US associations. First, in studies in which the UR is prevented, conditioned responses still occur: when the drug curare, which blocks all skeletal responses, is used during conditioning, conditioned skeletal responses to the CS are still present when the drug has worn off (Soloman & Turner, 1962). Second, when the US is revaluated in some way it has an effect on the CR (Rescorla, 1974). Rescorla (1974) conditioned a CR using a weak shock as a US in rats. Following conditioning, these rats were exposed to a stronger shock than that used during conditioning (but bear in mind the CS was not present during these exposures). Subsequent presentations of the CS gave rise to greater intensity conditioned responses than after conditioning. Rescorla concluded that exposure to the greater intensity shocks had led to *US-revaluation*, that is, the US was re-valued by the organism as more aversive than it had been during conditioning. As a result of this revaluation, conditioned responses changed accordingly. This experiment demonstrates that conditioned responses are mediated by CS–US associations (because revaluing the US changes the CR), and that, therefore, conditioned responses can be altered by experiences outside of the original learning trials.

The fact that conditioning is mediated by CS–US associations is now well-known in learning (e.g. Davey, 1989b, Mackintosh, 1983) and anxiety literatures (e.g. Davey, 1997; Field & Davey, 2001), which is why it is often called *associative learning*. However, this picture of what is learned during conditioning is still overly simplistic. In an elegant paper, Rescorla (1988) highlighted several pervasive myths about Pavlovian conditioning. One of these myths was that conditioning represents an extremely restricted form of learning in which a single stimulus becomes associated with a single outcome. As we shall see in due course, conditioning is a much more complex process in which past learning and contextual variables are accounted for. During conditioning, associations are formed between representations (which themselves can include relations generated by other associations) of multiple events. As we shall see, conditioning, far from being a simple learnt association between two events, is a complex process providing an organism with a detailed representation of its environment.

4.2. Conditioning depends on past experience

Organisms do not enter conditioning episodes as *tabula rasa*, instead they bring with them information about prior experience, and prior relationships between CSs and USs. Several conditioning phenomena illustrate this fact. One such phenomenon is *blocking* (Kamin, 1968). In a typical blocking paradigm there are two groups of animals. The first group experiences a set of conditioning trials in which a CS (CS1, a light perhaps) predicts a US. The second group does not receive these trials. In the second stage both groups experience a compound stimulus (CS1

and CS2, the light and a tone) predicting the US from stage 1. These experiments show that the pre-exposed group show reduced conditioned responding to CS2: the fact that CS1 already reliably predicted the US 'blocks' learning about CS2 in the second stage. The group that was not pre-exposed to CS1–US contingencies does, however, show strong CRs to CS2.

Two related phenomena also demonstrate learning when, behaviourally speaking, no learning appears to have taken place. The first phenomenon is *latent inhibition*, which refers to the discovery that it takes significantly longer to generate conditioned responses to a CS if an animal is pre-exposed to that CS before the conditioning trials, compared to when no pre-exposure takes place (Lubow, 1973; Lubow & Moore, 1959). A subtly different phenomenon is *learned irrelevance*, which occurs when an animal is pre-exposed to uncorrelated presentations of CSs and USs (so, unlike latent inhibition, there is pre-exposure to both CSs and USs but the CS does not reliably predict the US). Again, this kind of pre-exposure retards subsequent learnt responses from correlated CS–US presentations (Mackintosh, 1973). Both of these phenomena reiterate that the overall power of a CS to predict a US is crucial (in both cases pre-exposure reduces the overall correlation between the CS and US), but also that animals can learn about the absence of relationships (Mackintosh, 1973, for example, believed that pre-exposure to uncorrelated CS–US presentations harmed learning because the animal learns that the CS is irrelevant).

Another moderator of learnt responses occurs when the CS is presented in compound with a stimulus that already has an inhibitory relationship with the US. Imagine in a training phase an animal experiences a stimulus (A) in the presence of an outcome (say, shock); they also experience this same stimulus at the same time as another stimulus (B) and the absence of shock. Given that A reliably predicts shock, B will form an inhibitory relationship with shock (i.e. the organism will learn that B predicts absence of shock). The organism now experiences this inhibitory stimulus, B, alongside a new stimulus, C, in the presence of the shock US. Given that B already has a negative relationship with the CS, the new stimulus should acquire a magnified conditioned response to the US compared to if that CS had been presented in isolation with the US. This phenomenon is known as *super-learning* (see Aitken, Larkin & Dickinson, 2000; Rescorla, 1971). Super-learning again suggests that learning history has implications for the strength of magnitude of an acquired fear response: if at the time at which a CS becomes associated with trauma an inhibitory stimulus (one which has a negative prior relationship with trauma) is present also then it would be reasonable to expect a much greater acquired fear response.

These characteristics of conditioning clearly demonstrate that organisms enter conditioning episodes with information about prior experience and prior relationships between CSs and USs. In clinical terms this means that the power of a particular conditioning episode will depend, to some extent, on prior experience. Let's take an example of a girl who sees a wasp land on her hand and is then stung. Prior exposure to a potential phobic stimulus in the absence of any biologically significant outcome (or the presence of a positive outcome) will protect the girl from learning aversive responses in a subsequent traumatic conditioning episode (latent inhibition). In this example, prior (non-traumatic) exposure to wasps should protect the girl from acquiring a fear of wasps after being stung. Likewise, uncorrelated pre-exposure to trauma and the phobic stimulus will weaken future associations between the two (learned irrelevance). Prior experience of trauma in the absence of the potential phobic stimulus should lead to an inhibitory relationship, which again will protect that stimulus from becoming a phobic stimulus. If the girl has been stung many times before but has not known what stung her or has associated the sting with different unrelated stimuli, then this should weaken the association between wasps and trauma after she sees the wasp sting her. Blocking also implies that existing relationships between a stimulus and a trauma should retard future stimuli from acquiring aversive CRs from *the same* trauma. For example, if she already associates similar painful bites with another animal then this should block the association between the wasp and trauma. Finally, super-learning can explain individual differences in acquired fear: the extent to which a fear CR is acquired depends on contextual variables at the time of learning. For example, if the girl is playing with her pet cat (a stimulus not predictive of trauma) at the time when she sees the wasp sting her then she should acquire a greater fear CR to the wasp than if she had not been playing with her cat.

All of this evidence explains why people who experience the same trauma differ in their acquisition of fears: because they have different conditioning histories, both with the trauma itself, and the stimulus with which it was paired. The other implication is that fear acquisition through conditioning processes is more likely to happen at younger ages because learning histories become more varied and complex as an organism gets older. To use the example above, *ceteris paribus*, a girl of 8 years old has had more time to have experienced wasps, experienced them more often and acquired a positive learning history that will be longer and more protective than a girl of 1 year old.

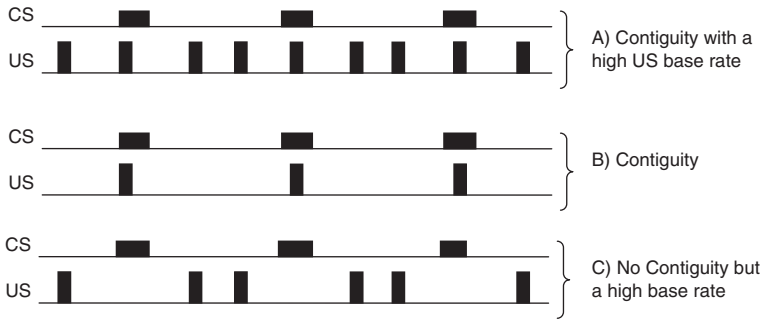

Fig. 2. Time lines showing different levels of contiguity between a CS and a US. In A and B, the contiguity is the same (the CS and US always coincide), but A has a high base rate of the US (it also occurs in the absence of the CS). In C, there is no contiguity (the US occurs only when the CS is absent).

Having looked at the impact of events before conditioning, I will now focus on the impact of events during conditioning.

4.3. Conditioning does not depend upon contiguity

Rescorla (1988) also noted that there are widely held misconceptions about the conditions under which conditioning occurs. It is widely acknowledged that temporal contiguity between the CS and US is a central concept in conditioning (in clinical terms the potential phobic stimulus should occur at the same time as a traumatic event); however, it is less widely acknowledged that contiguity is neither necessary nor sufficient for conditioning to occur. Rescorla (1968) conducted a study using two conditions that had identical pairings of a tone CS with a shock US (see A and B of Fig. 2). However, in one of the conditions the shock US was also presented in between conditioning trials (A in Fig. 2). As such, the contiguity between the CS and US was identical in the two conditions, but they differed in terms of the information that the CS provided about the US: in one condition the CS perfectly predicted the US but in the other the likelihood of the US occurring when the CS was present was equal to the likelihood of the US when the CS was absent. In this later condition, no conditioned responding was found demonstrating that conditioned responding is sensitive to the base rate of the US. Put another way, the predictive power of the CS is key, not the contiguity. This evidence suggests that base rate experience of the trauma will have an impact on its power to 'create' phobic stimuli.

Contiguity is not only insufficient, it is actually unnecessary! The earlier discussion of super-learning referred to inhibitory relationships in which a stimulus predicts the absence of a US. These inhibitory relationships demonstrate that contiguity is unnecessary. Imagine a situation in which a group experiences the US only when the CS is not present (see C in Fig. 2). The expectation, based on simple contiguity, is that nothing is learned in this situation. As we have already seen, the animal does learn something: it learns that there is a negative relation between the CS and the US. This phenomenon is known as *conditioned inhibition*. So, even in the absence of CS–US contiguity, animals learn something.

4.4. Conditioning can occur after only 1 trial

Another misconception about conditioning is that it occurs over many trials. This view is problematic for conditioning models of fear acquisition because it is unlikely that an organism would have repeated exposure to a particular stimulus and the same traumatic outcome (it would be a very unlucky person who experienced a traumatic outcome every time they saw a spider!). However, conditioned responses can be acquired very rapidly: for example, Rescorla (1980) has demonstrated conditioned responding in only 8 trials. Even 8 trials though is a lot in terms of phobia acquisition: the assumption is that fear is acquired in a single trial. Nevertheless, there is evidence of one-trial learning especially when the outcome is aversive: Garcia, McGowan, and Green (1972) have shown one-trial learning using illness and shock as an aversive US (see also Izquierdo et al., 2000; Izquierdo, Barros, Medina, & Izquierdo, 2000); Campbell, Sanderson, and Laverty (1964) demonstrated conditioned responses in humans when respiratory paralysis was used as a US; and most relevant, Öhman, Eriksson, and Olofsson (1975) showed one-trial learning to

both fear-relevant (snakes) and fear-irrelevant (houses) stimuli when shock was used as a US. These studies demonstrate that conditioned responses to stimuli co-occurring with an aversive outcome can be established in one trial.

4.5. Associations formed after 1 trial are persistent

Perhaps more compelling evidence of one-trial learning comes from Rescorla's (2000) demonstration that even when CSs and USs are randomly presented learning takes place: CS–US associations are formed very early and rapidly as are CRs, however, as training continues the CS loses its power to evoke a CR, but the CS–US associations *remain intact*. This finding is particularly interesting because it implies that CS–US associations form in very few trials and even subsequent random presentation of the CS and US does not eliminate the association between the two. The first implication is that a stimulus–trauma association can be created very quickly (in a single trial). The second, more subtle, implication is that the absence of a behavioural manifestation of the stimulus–trauma association (i.e. anxiety) cannot be taken as evidence that the association has not been formed, or that this association could not have a behavioural effect at some later stage. In fact, given that this association is resistant to subsequent random experience with the CS and US, then even in cases in which a person re-experiences the phobic stimulus or the trauma in the absence of the other, the initial association between the two will not be diminished[1] and may acquire the power to evoke a CR *at some later time*. We shall return to this point.

4.6. The CR and UR are not necessarily the same

Another common misconception about conditioning is that the conditioned response is the same as the unconditioned response. The reason why this belief is held is probably because in classic studies such as the little Albert study, and Pavlov's original experiments (Pavlov, 1927), the CR and UR *were* the same. In fact, early theories of classical conditioning suggested that the CS becomes a substitute for the US and so the response elicited by the US is transferred to the CS (*stimulus substitution*). However, subsequent work has shown that the CS can elicit several responses that are relevant to the US, but are not necessarily the same as the response elicited by the US: for example, rats respond to aversive outcomes such as shock with behaviours such as increased heart rate, squeaking, and jumping, whereas a CS paired with this shock comes to elicit the opposite anticipatory responses such as a decrease in heart rate and 'freezing' (Black, 1971).

Also, different CSs conditioned to the same US can produce different CRs: Pinel and Treit (1979) report that if a tone CS predicts a shock US then freezing occurs as already described, however, if a localised prod is used as a CS to the same shock US, then the CS comes to evokes behaviours aimed at burying the prod. The implication for conditioning accounts of phobias is that for any given phobic stimulus, the experiences at the time of conditioning may not be as simple as experiencing that stimulus during a traumatic event and subsequently feeling anxiety. The CR a person experienced may only be *related* to anxiety rather than being anxiety itself, and will depend on the CS and the US. As such, retrospective studies (in adults and children) of whether conditioning led to fear are likely to underestimate conditioning experiences simply because they present phobics with an inaccurate and over-simplified explanation of the type of experience they should be reporting.

4.7. The CS does not always produce a CR following conditioning

Rescorla (1991) has also demonstrated that if a CS is reinforced if, and only if, it is accompanied by another stimulus (called a 'feature'), the conditioned responses likewise occur only if that feature is present. This phenomenon is called *feature modulation* or *occasion setting*. Feature modulation again emphasises the point made earlier that conditioning does not occur in a vacuum but is dependent on context. The first important implication for clinical accounts of phobia acquisition is that feature modulation can, perhaps, explain differences between subjective experiences of stimuli following conditioning. For some people conditioning may occur in the presence of a modulating stimulus: if this modulating stimulus is absent in subsequent encounters with the CS then conditioning theory predicts that no CR should occur. As such, although a CS–US association has been formed, it has no behavioural impact because the

[1] However, the response to the CS will be diminished.

modulating stimulus is never encountered. Conversely, if at the time of conditioning there is no modulating stimulus, or the modulating stimulus is one highly likely to co-occur with the CS, then subsequent encounters with the CS should produce anxiety. For example, a boy who embarrasses himself during a school play may not develop general social anxiety because the context in which learning took place was so unusual. However, he may develop anxiety specifically about performing in school plays. The second implication is that treatment programs must aim to discover if such moderators exist, and if they do, exposure to the CS should be done within the context of the moderator (see Bouton, Mineka, & Barlow, 2001).

A second, better-known, phenomenon is that of *overshadowing* in which a compound CS (i.e. two stimuli presented at the same time) is conditioned to a US. In subsequent presentations of the stimuli that made up the compound CS it is only the more salient of the two that will evoke a CR: responses to the less salient stimuli are overshadowed. The important point here is that there is a tendency to assume that conditioning takes place between one stimulus (viewed as a whole) and another (also viewed as a whole) and yet a given stimulus is a combination of many features. In fact, conditioning is not simply the association of one stimulus with another, but the association of stimulus features with other stimulus features (see the section on conditioning being driven by CS–US associations). As such, fear may be evoked by quite specific features of the CS and conditioned responding during subsequent exposure to phobic stimuli will be modulated by the degree to which these features are present in that particular exemplar of the object.

4.8. Conditioning can occur without an actual CS and US

As ridiculous as it might sound, recent evidence suggests that conditioned responses can be acquired (using flavour CSs) without the CS or US being present at the time of learning: all that is required is a mental representation of these stimuli. Learning from representations rather than actual stimuli has been shown in two ways. First, when a mental representation of a CS is evoked in the absence of the actual stimulus and is paired with a US, the CS acquires a conditioned response. Second, if a mental representation of a stimulus (CS_1) is paired with a second stimulus (CS_2), which has previously been paired with a US and hence should evoke a mental representation of it, then CS_1 acquires a conditioned response. The implication is that learning has occurred in the absence of both the CS and US: a representation of the CS has been paired with a representation of the US (see Dwyer, 1999, 2001, 2003; Dwyer, Mackintosh, & Boakes, 1998). Although more work needs to be done to see whether these effects extend beyond flavours, these findings have important implications for conditioning accounts of fear acquisition. First, it implies that it may be possible to acquire a conditioned anxiety response to a stimulus without experiencing that stimulus at the time of trauma: all that is necessary is that the person is *thinking* about the stimulus at the time of the trauma. Second, it may be possible to acquire a conditioned fear response merely by thinking about a stimulus and a traumatic event simultaneously.

A related phenomenon is *second-order conditioning* (Rescorla, 1980): once a predictor can elicit a reliable CR through its association with another stimulus, that predictor can act as outcome for other potential predictors. This phenomenon can be demonstrated using the following procedure: (1) CS_1 (e.g. a bell) is paired with an outcome (e.g. shock) until it reliably evokes a response (anxiety); (2) a second predictor, CS_2 (e.g. a light), is paired with CS_1 (the bell); (3) CS_2 (light) is presented alone and is found to elicit a CR (anxiety) despite having never been directly paired with a US (shock). The second predictor comes to elicit a response because it is associated with CS_1, which in turn is associated with an aversive outcome. Although there are many possibilities (and some conflicting evidence—see Davey, 1989b) regarding what associations govern responding to CS_2, there is some evidence in humans that devaluing the US eliminates CRs to CS_2 (Davey & McKenna, 1983). This finding suggests that CS_2 forms a direct association to the original US that was paired with CS_1; implying that when CS_1 and CS_2 are paired, CS_1 evokes a representation of the US that becomes associated with CS_2. The aforementioned evidence that US representations are sufficient to uphold conditioning is consistent with this possibility.

The evidence in this section supports the idea that fear acquisition can occur through more complex scenarios than simply experiencing a stimulus in the presence of trauma. Specifically, stimuli can become associated with aversive USs without ever occurring in the presence of that US, all that is required is that they are experienced in the presence of a *representation* of an aversive US (a representation that may or not be evoked by a different CS). In these circumstances a person would have no memory of the object of their fear co-occurring with a traumatic event because it never did (but the acquired fear still stems from conditioning processes). As such, these possibilities go some way to explaining why phobia sufferers often do not remember explicit conditioning episodes.

4.9. The US need not be biologically significant

Typically, USs are defined as a biologically significant event (because they evoke an unconditioned response). However, humans can readily associate stimuli, without one of them needing to evoke an unconditioned response. For example, studies of causal learning show that humans can learn predictive relations between neutral stimuli and neutral outcomes (see De Houwer & Beckers, 2002; Dickinson, 2001; Shanks, Holyoak, & Medin, 1996 for reviews). Examples of the paradigms used include predicting whether pictures of butterflies (CS) will mutate (US) when exposed to radiation (Collins & Shanks, 2002; Lober & Shanks, 2000) and whether certain foods (CS) predict an allergic reaction US (Aitken et al., 2000; Le Pelley & McLaren, 2003). Note in both cases that the apparent US does not evoke an unconditioned response; the outcome of these learning trials is not a US in the traditional sense. Nevertheless, humans readily learn the contingencies in these tasks (e.g. they can accurately predict whether a butterfly will mutate or not after several trials on which they receive feedback about whether their predictions are correct). This form of learning can be successfully characterised as associative learning in which the cause acts as a CS and the outcome is viewed as a US (see Dickinson, Shanks, & Evenden, 1984). When characterised in this way and models of associative learning (notably the Rescorla–Wagner and Mackintosh models described later) are applied, many aspects of the data accurately fit predictions from these models (De Houwer & Beckers, 2002; Dickinson, 2001; Le Pelley & McLaren, 2003; Lober & Shanks, 2000). For example, phenomena such as the importance of statistical contingency between the CS and US (Perales & Shanks, 2003), blocking (Dickinson, 2001; Dickinson et al., 1984), learnt inhibition and learning under negative contingency (Chapman & Robbins, 1990; Dickinson, 2001), and super-learning (Aitken et al., 2000; Dickinson, 2001) are found in causal learning tasks.

Although this review is an over-simplified look at the vast literature on causal learning, the main point is that associative learning (or 'conditioning') applies not just to associations between neutral and biological significant events. Models of associative learning can be applied with some success to situations in which the US is innocuous, and there is no obvious CR (or UR for that matter). This observation is important because it shows the generality of this form of learning: throughout childhood many associations will be formed between potentially phobic stimuli and any number of seemingly innocuous USs (and resulting in no anxiety at the time the association is formed). Once formed, however, these associations may still have power to generate anxiety through processes such as US revaluation. These associations also seem, to some extent, to be governed by the same laws as traditional conditioning.

So far I have described several phenomena relating to events prior to conditioning, and factors that influence learning during the conditioning episode itself. I now turn to the impact of events subsequent to a conditioning episode.

4.10. Extinction does not break the CS–US association

The finding described earlier that CS–US associations form very quickly and remain in tact even after random CS–US presentations has interesting implications for extinction, in which the CS is presented alone resulting in a gradual decline in the CR until it is no longer evoked at all. If CS–US associations survive random presentations then they should also survive extinction (even if the CR is extinguished).

There are two clinical issues related to extinction. The first is that conditioning models of fear have been criticised because they predict extinction of fear responses, yet this is not seen in phobics. The idea behind this criticism is that having been unlucky enough to be on a ship that sunk (as in Yule et al., 1990) in all probability subsequent encounters with ships will not be accompanied by traumatic outcomes so the anxiety CR should decline. The second issue is that having been treated through exposure therapy (which is in its simplest form is a series of extinction trials) anxiety sometimes returns.

The lack of extinction seen subsequent to traumatic conditioning episodes can be explained by avoidance: the children in Yule et al. (1990) who acquired anxiety about boats would be likely to avoid travelling on them subsequently and therefore never experience ships in the absence of trauma. We have also seen that a US need not be a real object: a mental representation is sufficient. This too explains a lack of extinction and possibly even incubation: when the fear-evoking stimulus is encountered it evokes a mental representation of the traumatic event that maintains the fear response.

The issue that anxiety can return after extinction arises out of another misconception about conditioning: that extinction is forever. In fact, recent research into extinction is completely consistent with the view that CS–US associations not only survive subsequent random CS–US presentations (as described earlier) but also survive

extinction procedures (see Bouton, 1994). So, even though conditioned responding to a CS declines over presentations of the CS in the absence of the US, several phenomena suggest that the underlying CS–US association survives.

The *renewal effect* occurs when conditioning (e.g. a tone CS with a shock US) is conducted in one context (for example a specific location), but extinction trials (the tone alone) are conducted in a different context. CRs to the tone extinguish, however, if the extinguished tone is presented in the original context (the context in which it was conditioned), the extinguished response is renewed. This effect can be very strong: Going back to the children in Yule et al. (1990), the obvious implication is that the association between boats and the traumatic event will stay intact even if subsequent encounters with boats are not accompanied by trauma, provided these encounters are in a different context.

The second phenomenon is *reinstatement* which describes the finding that subsequent to extinction trials, if the US is presented again on its own, CRs are reinstated upon subsequent presentation of the CS. Again, reinstatement suggests that the CS–US association survives extinction. However, as with renewal, this effect is context dependent: the CS must be experienced in the same context as the reinstating stimulus (the US) for reinstatement to work. In terms of fear acquisition, reinstatement implies that even if anxiety CRs are extinguished, a subsequent experience of the original trauma could reinstate the conditioned anxiety response if the phobic stimulus is later experienced in the same context.

The oldest phenomenon that demonstrates how CS–US associations survive extinction is *spontaneous recovery*, in which extinguished CRs return (at least partially) merely after the passage of time. Bouton and Swartzentruber (1991) view this effect as an example of renewal but in temporal rather than physical contexts. In essence, the idea is that the extinction trials occur in a different temporal context to conditioning (although, of course, the CS cannot be re-experienced in the original temporal context but in an entirely new context to both conditioning and extinction).

In terms of the clinical profile of developing anxiety, it is clear that the prediction that anxiety CRs should diminish over subsequent exposure is not as simple as it first seems. In fact, the extent to which anxiety should subside is dependent upon context, and the success of exposure therapy may also depend on matching the context of exposure to the context in which the initial conditioning occurred.

4.11. Traumatic incidents might not be traumatic at the time

If, as we have seen, stimulus–trauma associations can be formed very rapidly, and exist even when the CS does not have sufficient power to evoke a CR, or when the CR has been extinguished, then what does it take to re-evoke the CR? We saw at the beginning of this section that conditioned responses are driven by CS–US associations and that re-valuation of the US, therefore, can influence the CR. If a person has formed a stimulus–trauma association (that survives extinction and subsequent random exposure to the stimulus and/or trauma) then all it would take to re-evoke a conditioned response would be for the US to be revalued (see Davey, 1997; Field & Davey, 2001).

Davey and McKenna (1983) conditioned fear responses in humans and then exposed them to the US to allow participants' fear to it to habituate. This habituation resulted in both a more favourable assessment of the US and a diminished CR when the CS was next presented. Conversely, Davey, De Jong, and Tallis (1993) report the case of a person (L.L.) who experienced mild anxiety and intestinal unease (US) in social situations (CS). On one occasion while L.L. was at home and not anxious, similar symptoms of intestinal unease led to an uncontrollable attack of diarrhoea. This event re-valued the US (the negative consequences of intestinal unease were magnified) and in subsequent social contexts L.L. became extremely anxious and developed severe agoraphobic symptoms.

There are two important points here (see Davey, 1997; Field & Davey 2001): (1) US revaluation rules out the need to find specific traumatic conditioning events in a patient's history because the US may become traumatic subsequent to the learning episode in which it was associated with the CS; and (2) US revaluation explains why some individuals can experience a stimulus in the presence of a traumatic event yet not develop phobic symptoms—because the traumatic event is subsequently devalued.

4.12. Summary

So far we have looked at some recent (and not so recent but poorly understood outside of the conditioning literature) phenomena of classical conditioning. The stereotyped view of conditioning presented at the start of this paper is hugely

inaccurate: conditioning is not simply a response acquired through experiencing one stimulus in the presence of another. Instead it is a rich environment in which context, past experience, and cognition all play a part in learning. We now turn our attention to other pathways to fear and look at whether conditioning can inform us about these routes to anxiety.

5. Vicarious learning and information

One of the limitations of early conditioning models was that learning occurs through other processes such as vicarious learning and information. The assumption here is that these pathways operate in different ways to direct conditioning (i.e. the underlying mechanism is different). However, this assumption is not necessarily true. In vicarious learning, a person experiences a stimulus (a CS) and someone else's reaction to it. Mineka and Cook (1993) suggest that in such a learning episode the observed reaction to the CS acts as a US (that is, someone else's distress is itself anxiety-evoking). As such, the observer experiences the CS in the presence of an anxiety-evoking event (someone else's distress). In which case, observational learning is procedurally the same as so-called direct conditioning. Mineka and Cook also present evidence suggesting that observer Monkeys do exhibit fear responses (unconditioned responses) to other Monkey's distress, which supports the idea that other's fear responses can act as a fear-evoking US.

A second possibility is that vicarious learning represents a form of second-order conditioning (see above). In this interpretation, a person's fear response is actually a CS which has acquired a fear-evoking quality through some prior co-occurrence with a traumatic event (US). The observational learning episode itself is, therefore, the co-occurrence of a new CS_2 with the CS_1 of the observed fear response. As such, a stimulus (CS_2) acquires a second-order fear response through co-occurrence with a fear response which has previously been associated with a traumatic outcome. Unfortunately, there is no available evidence to distinguish these two interpretations of what occurs in observational learning (see Mineka & Cook, 1993). However, for the purposes of this review all that is important is that vicarious learning is a form of conditioning at the procedural level and the associative structure of a vicarious learning episode can be conceptualised in the same way as a direct conditioning episode.

In terms of how children acquire fear, very few studies have addressed the issue. Gerull and Rapee (2002) showed that toddlers would show greater fear expressions and avoidance of novel stimuli if their mothers had previously displayed negative facial expressions towards those stimuli. However, this study was not intended to look at the underlying mechanism of this learning. Recent work from our own laboratory (Askew, Zioga, & Field, 2004) is looking at these mechanisms by using a paradigm in which 7–9-year-old children are shown pictures of 3 novel Australian marsupials (CSs) presented on a computer screen alongside either pictures of smiling or fearful facial expressions (the third marsupial is not paired with any CSs and acts as a control for exposure). Initial results suggest that such pairings can produce changes in children's self-reported fear beliefs about the animals and produce reliable differences in attitudes to the animals when measured using reaction-time based tasks such as affective priming. The interesting thing about this paradigm is that comparisons to the animal that does not enter into an association with a US (and also comparisons with non-paired control groups) indicate that the changes are caused by a CS–US association: The results reflect associative learning. As such, like 'direct' conditioning, observational learning appears to be driven by CS–US associations. However, these studies have looked only at changes in subjective fear *beliefs* and so are still a step removed from showing acquired *physiological* fear.

All of this evidence suggests that vicarious learning can be conceptualised as a conditioning procedure and is driven by CS–US associations; however, more research is needed to determine whether it conforms to the same rules as so-called direct conditioning.

The second indirect pathway to fear (Rachman, 1977) is fear acquired through verbal information. Up until recently the evidence for the role of fear information in fear acquisition was based on retrospective reports in which adult phobic patients are asked to assign their learning experiences to one of the three pathways some 10–20 years after the onset of their phobia. Although such studies support the role of fear information in the development of fears (see King et al., 1998; Merckelbach, De Jong, Muris, & Van den Hout, 1996, for reviews), these reports will be prone to memory bias and forgetting of potentially important learning episodes and typically rely on questionnaires such as the Phobic Origin Questionnaire (Öst & Hugdahl, 1981), which fails to identify components of the conditioning process resulting in misattributions of the cause of phobia (see

Menzies & Clarke, 1994). These studies also force respondents to classify learning episodes as belonging to prescribed categories of learning experience (such as direct conditioning, vicarious learning and verbal information). Such retrospective studies also say little about the mechanism underlying the effect of fear information.

Recently, our laboratory has investigated the effects of negative information about novel animals in children. Field, Argyris, and Knowles (2001) developed a prospective paradigm for looking at the effects of fear information in the development of animal fear beliefs in children: in two experiments, 7–9 year olds received either positive or negative verbal information about previously un-encountered toy monsters. The results showed that children's self-reported fear beliefs about the monster about which they had received negative information significantly increased. Similar results were found when the paradigm was adapted to look at social rather than animal fear beliefs (Field, 2002; Field, Hamilton, Knowles, & Plews, 2003). Field and Lawson (2003) refined the paradigm by using real animals (3 Australian marsupials: the quoll, quokka and cuscus) that were unfamiliar to children in the UK, as stimulus materials. For a particular child, one of the animals was associated with positive information, one was associated with negative information and they were given no information about the third (which acted as a control). In these studies, negative information significantly increased children's fear beliefs as indexed by self-report, implicit measures of the beliefs (the implicit association task) and behavioural avoidance as measured by reluctance to approach a box inside which the animal was believed to be. Muris, Bodden, Merckelbach, Ollendick, and King (2003) have also shown in a similar paradigm that the effect of negative information persisted a week after it was given and Field, Lawson and Banerjee (submitted for publication) have shown that both implicit and explicit fear beliefs can last up to 6 months. In addition, Field (2004) reported data suggesting that actual fear increased too in a behavioural task following negative information (relative to the control animal). This body of work, although still in its infancy, shows that fear information can change fear beliefs, produces behavioural avoidance, and increases actual fear.

The next issue is how fear information has its effect. Associative learning is a prime mechanism to explain how fear information operates. In the studies above, the novel animal is a CS, and the information a US. Typically, USs are defined as biologically significant, so in a strict conditioning sense, the information would need to be fear-evoking. As yet, there is no evidence that the information given in these studies is fear evoking (in fact, the content of the information is highly unlikely to be fear-evoking) which suggests that conditioning models cannot be applied in this situation. However, as we saw in the previous section, conditioning models can also explain causal learning in humans—in which USs are not biologically significant. It is plausible, therefore, to consider that throughout childhood, a child experiences many causal learning/conditioning trials in which a stimulus (such as an animal) consistently predicts negative information ('don't touch that', 'be careful of the animal' 'that animal bites'). These experiences are directly analogous to the type of experiences in human causal learning experiments: a potentially phobic stimulus acts as a CS and negative information as a US. Procedurally, this situation is conditioning—it results from the pairing of a stimulus with an outcome. At the effect level too, it is conditioning: the beliefs or behaviours are acquired through the pairing of a stimulus with some meaningful information about it.

If we consider fear information as having its effect through associative learning then the first implication is that if the negative information gets revalued in some way (perhaps by some salient experience such a friend being bitten by the animal) then through its association with the animal CS, conditioned anxiety could emerge (remember that in direct conditioning studies, if an innocuous US is revalued in this way a CS that previously did not have the power to evoke a measurable CR can come to evoke one). The second implication is that fear information should exhibit the other functional characteristics of direct conditioning. For example, the base rate of information should be important, positive information should protect against subsequent negative information (latent inhibition) and so on. Research is ongoing to see if this is the case, however, initial results do suggest that, consistent with models of associative learning, acquired fear beliefs are sensitive to the statistical contingency between a novel animal and negative information (Field, in preparation).

In this section we have seen that the effects of both vicarious learning and negative information of the development of fear are procedural examples of conditioning. We have also explored the possibility that both are forms of associative learning. In both cases there is initial evidence that they show some of the characteristics that would be expected if they are governed by the same underlying mechanism as so-called direct conditioning. Although only time will reveal whether these 'indirect pathways' can be fully described by models of associative

learning, I hope to have convinced you that there are good reasons to suppose that they might. If you accept this possibility then, remembering that 94% of anxious children and their parents endorsed direct conditioning or one of these indirect pathways as an explanation for their fear, then associative learning is an extremely good prospect for a mechanism that underlies the acquisition of fear.

6. Models of conditioning/associative learning

Assuming you are suitably convinced of the merits of conditioning theory as an explanation of developing fears, it is worth looking as some of the popular models and what they imply for fear acquisition. This section reviews some of the most popular models of the past 30 years but is by no means exhaustive.

6.1. The Rescorla–Wagner (1972) model

Rescorla and Wagner (1972) proposed a model of conditioning that, in essence, is based on processing of the US. The model formalises the idea that associations are formed between cues and surprising outcomes and incorporates a term representing a cue's individual associability, which represents an individual learning rate that stems from differential attention. The model is formalised in the following equation:

$$\Delta V_A = \alpha_A \beta(\lambda - \sum V)$$

This equation simply means that the change in associative strength of a given cue or CS (ΔV_A) is a function of the intensity or salience of the cue itself (α_A), known as its *associability*, and the intensity of the US (β). The crucial part of the equation is the part in parenthesis, which reflects the difference between the maximum associative strength that the US can support (i.e. the maximum amount of conditioning possible with a given US, λ) and the sum of the associative strengths of *all cues* presented on the trial ($\sum V$). This summed error term allows the model to explain cue competition effects such as blocking: in initial training stages the first cue, A, will acquire associative strength up to the maximum allowed by the US (λ), then in the second stage, the amount of associative strength gained by the second cue B depends upon the discrepancy between the amount of conditioning supported by the US (λ) and the summed associative strength of A and B ($\sum V$). The pre-training of A to its maximum associative strength means that the summed associative strength of A and B will equal the amount of conditioning supported by the US ($\lambda = \sum V$), and the discrepancy between the two is, therefore, zero. The end result is the associative strength acquired by B will also be zero: a blocking effect.

In clinical terms, this model summarises some important characteristics. The change in associative strength of a potentially phobic stimulus will depend on several things. First, it will depend upon the intensity of the US (β): more traumatic experiences should support greater fear acquisition. Second, it will depend upon the salience of the potentially phobic stimulus (α_A), which itself could be influenced by any number of factors such as novelty, and prior information. Finally, it will depend on the extent to which it is presented with other cues that already have some associative connection to the US (cue competition effects).

6.2. Mackintosh's (1975) model

Mackintosh (1975), in a seminal paper, extended the Rescorla–Wagner model to suggest that the attention devoted to a given cue is a function of its importance in predicting an outcome: that is, animals will attend to relevant stimuli at the expense of not attending to irrelevant ones. In this sense, Mackintosh's model rather than looking at US-processing focuses on CS-processing. The model can be formalised as follows:

$$\Delta V_A = \alpha_A \beta(\lambda - V_A)$$

in which V_A is the existing associative strength of the cue. The key to this model, as I've already suggested, is that the associability of the cue (α_A) is not constant as in the Rescorla–Wagner model, but updates as a function of the degree to which that cue predicts the outcome relative to any other cues presented on the same trial. If the cue is better than all

other predictors then α_A will remain high, but if there are better predictors within the environment then α_A will decrease. These ideas are formalised as follows:

$$\Delta\alpha_A^n > 0 \quad \text{If} \quad |\lambda^n - V_A^{n-1}| < |\lambda^n - V_X^{n-1}|$$

$$\Delta\alpha_A^n < 0 \quad \text{If} \quad |\lambda^n - V_A^{n-1}| \geq |\lambda^n - V_X^{n-1}|$$

The first equation simply means that for a given trial, n, the salience of the CS on that trial, $\Delta\alpha_A^n$, will be positive if the discrepancy between the maximum amount of conditioning possible (λ^n) and the associative strength of the CS on the previous trial (V_A^{n-1}) is smaller than the discrepancy between the maximum amount of conditioning possible and the associative strength of all other cues on the previous trial (V_X^{n-1}). So, as the associative strength of the CS approaches the maximum (or is closer to the maximum for that CS than any other cues) then the salience of that CS increases. The second of the two equations means that for a given trial, n, the salience of the CS on that trial, $\Delta\alpha_A^n$, will be negative if the discrepancy between the maximum amount of conditioning possible (λ^n) and the associative strength of the CS on the previous trial (V_A^{n-1}) is greater than or equal to the discrepancy between the maximum amount of conditioning possible and the associative strength of all other cues on the previous trial (V_X^{n-1}). Therefore, when the associative strength of the other cues is as close or closer to the maximum possible than the CS, then the salience of the CS will decrease.

Kruschke (2001) has extended these ideas. He suggests that learning involves an attentional system that aims to implement the assumption that any CS should receive some attention, and to decide how attention should be distributed over multiple CSs. Finite attentional resources are assumed so increased attention to one CS necessarily implies less attention to another. The system receives feedback and shifts attention in such a way as to reduce error, these shifts in attention lead to changes in the association weights (of the CSs), which themselves act to reduce the error in learning.

These attentional models explain failures to learn such as blocking: after pre-training, the first CS is known to predict the US reliably and, therefore, it attracts most or all of the attentional resources when it is presented alongside a second CS. As such, the second CS receives little or no attention and no learning about it takes place (Kruschke & Blair, 2000; Mackintosh, 1975).

6.3. The Pearce–Hall (1980) model

Mackintosh's model has some problems in explaining phenomena such as latent inhibition, which led to Pearce and Hall (1980) to propose an alternative model, which like Mackintosh's model is based on CS-processing. However, they viewed learning in completely the opposite way to Mackintosh, arguing that attention would not be placed on CSs that reliably predict a US, but instead would be given to stimuli about which the predictive significance was unclear. The rationale was that it is costly to expend resources on stimuli already known to predict an outcome, when those resources could be allocated to trying to establish the significance of the 'unknowns' in the environment. In this model, different equations determine the change in associative strength depending on whether the trial is excitatory (CS–US) or inhibitory (CS–No US). For excitatory trials, the change in associative strength is determined by a function of a learning rate parameter related to US Intensity (β_E), the maximum possible conditioning (λ^n) and the associative strength of the CS on the previous trial (α_A^{n-1}):

$$\Delta V_A^n = \beta_E \alpha_A^{n-1} \lambda^n.$$

In inhibitory trials, the change in associative strength is determined by a function of a learning rate parameter related to US Intensity (β_I), the associative strength of the CS on the previous trial (α_A^{n-1}), and the discrepancy between the maximum possible conditioning (λ^n) and the extent to which a US is predicted by all stimuli presented on that trial ($\sum V_{NET}^{n-1}$).

$$\Delta V_A^n = \beta_I \alpha_A^{n-1} (\lambda^n - \sum V_{NET}^{n-1}).$$

Having determined the change in associative strengths on a given trial, the associability (salience) of a given CS is determined by:

$$\alpha_A^n = \gamma|\lambda^n - \sum V_{NET}^{n-1}| + (1-\gamma)\alpha^{n-1}.$$

In which γ is a parameter lying between 0 and 1 that defines the degree to which associability is governed by the preceding trial ($\gamma=1$ when the previous trial is solely responsible for the salience of the CS) or by earlier trials ($\gamma=0$ when the salience of the CS is solely determined by earlier trials and not the directly preceding one).

6.4. Hybrid models

Both the Rescorla–Wagner and Mackintosh models have their problems (Le Pelley, 2004; Pearce & Bouton, 2001) and subtle modifications have been made (see De Houwer & Beckers, 2002; Pearce & Bouton, 2001). The Rescorla–Wagner model breaks down by assuming that the individual associative strength of cues is unimportant and that all that matters is the *combined* associative strength. Mackintosh's model does not have this problem because it does not rely on a summed error term but instead views cue competition in terms of associability (rather than error). However, by not using a summed error term Mackintosh's model is unable to explain conditioned inhibition (when there is a negative relation between the CS and US). These problems have recently led to the development of hybrid models such as that by Le Pelley (2004). The technicalities of this model are beyond the scope of this article, but in essence it combines features of the Rescorla–Wagner, Mackintosh and Pearce–Hall models. The end result is a model that has at its core both CS (attentional as in Mackintosh's model and salience as in Pearce–Hall's model) and US processing (as in Rescorla–Wagner). This hybrid model allows explanations of learned irrelevance, blocking, latent inhibition, conditioned inhibition, superconditioning and associative history (see Le Pelley, 2004, for further details).

7. Conditioning and the development, treatment and prevention of anxiety

This paper has reviewed some features of conditioning and has attempted to dispel the myth that conditioning explanations of fear simply involve isolated learning episodes that occur in an experiential vacuum. On the contrary, learning theory has developed, and continues to develop, rapidly and we now have elegant demonstrations in animals and humans that conditioning is a rich and complex system that allows organisms to learn predictive relationships within their environment. Conditioning can not only account for how fears might be acquired, but also offers a framework for understanding how associations between mental representations are formed that themselves may lead to fear. For example, an association between a novel animal and fear information may lead to an activation of a fear response if that fear information is subsequently revalued. Conditioning is also not incompatible with the idea that some stimuli may more readily acquire fear responses in that models of learning see stimulus salience as key in the associative processes. Social and cultural processes will contribute to the extent to which a stimulus grabs attention and is, therefore, associated with an outcome. Conditioning also can explain differences in fear acquisition in children experiencing similar events: learning history and contextual cues are both important in whether CS–US associations are formed and whether conditioned responses are activated by a CS. There are also now a range of models that summarise the wealth of knowledge about conditioning processes. These models offer predictive mechanisms through which fears are acquired (or at least a mathematical approximation of the biological mechanism in the brain). From these models come predictions about when fears will be acquired and how associations between stimuli and trauma/negative information/vicarious experiences might be weakened and anxiety responses reduced.

The importance of understanding underlying mechanisms of fear acquisition cannot be underestimated. First, understanding mechanisms that cause fears to develop has important implications for prevention and treatment because it opens up several avenues through which preventive/corrective measures can be taken. In terms of prevention and rapid intervention, our knowledge of conditioning and the models that describe it allow several predictions:

1. *The contingency between a CS and US is important*: as such programs can be developed that actively attempt to reduce the contingency between a given stimulus and bad outcomes be they direct, vicarious or negative

information. Exposure to or positive learning trials involving common fear-evoking objects should reduce the impact of future learning episodes because the stimulus–bad outcome contingency will be weak.
2. *Learning history*: related to the above, the earlier that such preventions are initiated the more successful they will be (because there is less chance of a negative learning history existing).
3. *Quick intervention*: given that bad experiences do occur, parents and teachers might be encouraged to follow up bad experiences with positive ones. Again, this is important even if no behavioural anxiety is obvious because it will again weaken the stimulus–bad outcome association. For example, if a child is frightened by a dog jumping up then it should be beneficial to calm the dog and encourage the child to stroke it.
4. *Real CSs and USs are not necessary*: The evidence suggesting that conditioning can occur using only mental representations of CSs and USs opens up a range of prevention possibilities by using imagery, rather than real conditioning episodes. In the example of the dog frightening a child above, it may be sufficient to remind the child of past positive encounters with dogs, or to get them to think about or imagine those positive encounters.
5. *CS salience*: if CS salience is important in learning then preventions can be aimed at devaluing the salience of particular stimuli. For example, following a negative learning experience, if a child strongly associated a single stimulus, A, with the trauma, then drawing attention to other features that predicted the trauma should reduce the associative change that will occur to A in future negative episodes. This intervention should work because in effect you are reducing the discrepancy between A as a predictor of the outcome and other events as a predictor of the outcome in Mackintosh's equations which will reduce the change in associative strength of A in subsequent trials).
6. *US devaluation*: devaluing traumatic events quickly after they have happened may prevent anxiety from developing. For example, children who experienced a bad thunderstorm (e.g. Dollinger et al., 1984) might be reassured that storms of that magnitude are very rare and unlikely to happen again.

We can also use this wealth of knowledge to inform treatments.

1. *Contextual cues*: Although exposure and other interventions based on conditioning have already proved successful, it is clear that therapeutic interventions are relatively simplistic in that they are still based on the notion of a simple stimulus–trauma association. Given the complexities of learning, more detailed accounts of learning histories may help to inform interventions. Of course, therapists already search for contextual cues and triggers, but realising that such cues fit into formalised models of learning can help therapists to make firm predictions about the effect of manipulating these contextual variables (or conditioned inhibitors). For example, exposure therapy using imagery for a child who is socially anxious because of a mistake in a school play will be most effective if the child imagines the hall, the stage and any other salient features from the time of the initial conditioning episode. Honing in on these contextual cues from the time when the fear was acquired could reduce the probability of renewal of the initial fear.
2. *Formulation*: Conditioning may also be useful as a framework for explaining to patients or parents the kinds of processes involved in fear acquisition and how the various interventions have their effects. For example, it may be useful to explain the concept of spontaneous recovery to help explain setbacks in therapy.

Of course, the bleaker picture is that we have evidence that CS–US associations are formed quickly and are difficult to break. This evidence leads to the pessimistic conclusion that whatever preventions and interventions we try, negative learning experiences will win through. Perhaps the way forward is to target common fears at very early ages to help build positive learning histories that will protect children from developing anxiety after at least some of the negative learning experiences that life brings.

8. Summary

This paper has reviewed some of what is now known about phenomena and theories of conditioning (or associative learning) and has argued that it is a fruitful way to conceptualise how fears might develop. Not only do a broad range of learning phenomena conform to similar principles (human causal learning seems to obey similar rules to 'basic' conditioning in animals), but the various models of associative learning allow firm predictions about when and how fears might develop in children. By using conditioning as a framework for understanding direct fear acquisition,

vicarious learning and fear information we stand some chance of gaining better insight into how to prevent and treat phobias.

References

Aitken, M. R. F., Larkin, M. J. W., & Dickinson, A. (2000). Super-learning of causal judgements. *Quarterly Journal of Experimental Psychology. Section B, Comparative and Physiological Psychology*, *53*(1), 59−81.

Aitken, R. C. B., Lister, J. A., & Main, C. J. (1981, Jul). Identification of features associated with flying phobia in aircrew. *British Journal of Psychiatry*, *139*, 38−42.

Askew, C., Zioga, M., & Field, A. (2004). *Vicarious learning and the development of fear in children.* British Psychological Society Annual Conference, London (15th–17th April).

Black, A. H. (1971). Autonomic aversive conditioning in infrahuman subjects. In F. R. Brush (Ed.), *Aversive conditioning and learning* (pp. 3−104). New York: Academic Press.

Bouton, M. E. (1994). Context, ambiguity, and classical-conditioning. *Current Directions in Psychological Science*, *3*(2), 49−53.

Bouton, M. E., Mineka, S., & Barlow, D. H. (2001). A modern learning theory perspective on the etiology of panic disorder. *Psychological Review*, *108*(1), 4−32.

Bouton, M. E., & Swartzentruber, D. (1991). Sources of relapse after extinction in Pavlovian and instrumental learning. *Clinical Psychology Review*, *11*(2), 123−140.

Bregman, E. (1932). An attempt to modify the emotional attitudes of infants by the conditioned response technique. *Journal of Genetic Psychology*, *45*, 169−196.

Burnham, J. J., & Gullone, E. (1997). The fear survey schedule for children: 2. A psychometric investigation with American data. *Behaviour Research and Therapy*, *35*(2), 165−173.

Campbell, D., Sanderson, R. E., & Laverty, S. G. (1964). Characteristics of a conditioned-response in human-subjects during extinction trials following a single traumatic conditioning trial. *Journal of Abnormal and Social Psychology*, *68*(6), 627−639.

Chapman, G. B., & Robbins, S. J. (1990). Cue interaction in human contingency judgement. *Memory and Cognition*, *18*, 537−545.

Collins, D. J., & Shanks, D. R. (2002). Momentary and integrative response strategies in causal judgment. *Memory and Cognition*, *30*, 1138−1147.

Davey, G. C. L. (1989a). Dental phobias and anxieties—evidence for conditioning processes in the acquisition and modulation of a learned fear. *Behaviour Research and Therapy*, *27*(1), 51−58.

Davey, G. C. L. (1989b). *Ecological learning theory.* London: Routledge.

Davey, G. C. L. (1992). Characteristics of individuals with fear of spiders. *Anxiety Research*, *4*, 299−314.

Davey, G. C. L. (1997). A conditioning model of phobias. In G. C. L. Davey (Ed.), *Phobias: A handbook of theory, research and treatment* (pp. 301−322). Wiley: Chichester.

Davey, G. C. L., De Jong, P. J., & Tallis, F. (1993). UCS inflation in the aetiology of a variety of anxiety disorders: Some case histories. *Behaviour Research and Therapy*, *31*, 495−498.

Davey, G. C. L., & McKenna, I. (1983). The effects of postconditioning revaluation of CS1 and UCS following Pavlovian second-order electrodermal conditioning in humans. *Quarterly Journal of Experimental Psychology*, *35B*, 125−133.

Dickinson, A. (2001). Causal learning: An associative analysis. *Quarterly Journal of Experimental Psychology*, *54B*, 3−25.

Dickinson, A., Shanks, D. R., & Evenden, J. L. (1984). Judgment of act-outcome contingency: The role of selective attribution. *Quarterly Journal of Experimental Psychology*, *36A*, 29−50.

De Houwer, J., Baeyens, F., & Field, A. P. (2005). Associative learning of likes and dislikes: Some current controversies and possible ways forward. *Cognition and Emotion*, *19*, 161−174.

De Houwer, J., & Beckers, T. (2002). A review of recent developments in research and theories on human contingency learning. *Quarterly Journal of Experimental Psychology*, *55B*, 289−310.

Dinardo, P. A., Guzy, L. T., Jenkins, J. A., Bak, R. M., Tomasi, S. F., & Copland, M. (1988). Etiology and maintenance of dog fears. *Behaviour Research and Therapy*, *26*(3), 241−244.

Dollinger, S. J., O'Donnell, J. P., & Staley, A. A. (1984). Lightening-strike disaster: Effects on children's fears and worries. *Journal of Consulting and Clinical Psychology*, *52*(6), 1028−1038.

Dwyer, D. M. (1999). Retrospective revaluation or mediated conditioning? The effect of different reinforcers. *Quarterly Journal of Experimental Psychology. Section B, Comparative and Physiological Psychology*, *52*(4), 289−306.

Dwyer, D. M. (2001). Mediated conditioning and retrospective revaluation with LiCl then flavour pairings. *Quarterly Journal of Experimental Psychology. Section B, Comparative and Physiological Psychology*, *54*(2), 145−165.

Dwyer, D. M. (2003). Learning about cues in their absence: Evidence from flavour preferences and aversions. *Quarterly Journal of Experimental Psychology. Section B, Comparative and Physiological Psychology*, *56*(1), 56−67.

Dwyer, D. M., Mackintosh, N. J., & Boakes, R. A. (1998). Simultaneous activation of the representations of absent cues results in the formation of an excitatory association between them. *Journal of Experimental Psychology. Animal Behavior Processes*, *24*(2), 163−171.

Eysenck, H. J. (1979). The conditioning model of neurosis. *Behavioral and Brain Sciences*, *2*(2), 155−166.

Field, A. P. (2002). *Does fear information create fear beliefs about animals and social situations in children?* British Psychological Society Developmental Psychology Section Annual Conference. University of Sussex (5th–9th September).

Field, A. P. (2004). *Indirect pathways to fear in children: Are fear information and vicarious learning viable pathways to fear?* Annual Congress of the Association for the Advancement of Behaviour Therapy, New Orleans, USA (November 19th–21st).

Field, A. P. (in preparation). *The consistency of fear information and its effect on children's fear beliefs*.

Field, A. P., Argyris, N. G., & Knowles, K. A. (2001). Who's afraid of the big bad wolf: A prospective paradigm to test Rachman's indirect pathways in children. *Behaviour Research and Therapy*, *39*, 1259−1276.

Field, A. P., & Davey, G. C. L. (2001). Conditioning models of childhood anxiety. In W. K. Silverman, & P. A. Treffers (Eds.), *Anxiety disorders in children and adolescents: Research, assessment and intervention* (pp. 187−211). Cambridge: Cambridge University Press.

Field, A. P., Hamilton, S. J., Knowles, K. A., & Plews, E. L. (2003). Fear information and social phobic beliefs in children: A prospective paradigm and preliminary results. *Behaviour Research and Therapy*, *41*, 113−123.

Field, A. P., & Lawson, J. (2003). Fear information and the development of fears during childhood: Effects on implicit fear responses and behavioural avoidance. *Behaviour Research and Therapy*, *41*, 1277−1293.

Field, A. P., Lawson, J., and Banerjee (submitted for publication). *The longitudinal effects of fear information on implicit and explicit fear beliefs in children*.

Garcia, J., McGowan, B. K., & Green, K. F. (1972). Biological constraints on conditioning. In A. H. Black, & W. F. Prokasy (Eds.), *Classical conditioning II: Current research and theory* (pp. 3−27). New York: Appleton-Century-Crofts.

Gerull, F. C., & Rapee, R. M. (2002). Mother knows best: Effects of maternal modelling on the acquisition of fear and avoidance behaviour in children. *Behaviour Research and Therapy*, *40*, 279−287.

Gullone, E., & King, N. J. (1992). Psychometric evaluation of a revised fear survey schedule for children and adolescents. *Journal of Child Psychology and Psychiatry and Allied Disciplines*, *33*(6), 987−998.

Izquierdo, L. A., Barros, D. M., Ardenghi, P. G., Pereira, P., Rodrigues, C., Choi, H., et al. (2000). Different hippocampal molecular requirements for short- and long-term retrieval of one-trial avoidance learning. *Behavioural Brain Research*, *111*(1–2), 93−98.

Izquierdo, L. A., Barros, D. H., Medina, J. H., & Izquierdo, I. (2000). Novelty enhances retrieval of one-trial avoidance learning in rats 1 or 31 days after training unless the hippocampus is inactivated by different receptor antagonists and enzyme inhibitors. *Behavioural Brain Research*, *117*(1–2), 215−220.

Kamin, L. J. (1968). 'Attention-like' processes in classical conditioning. In M. R. Jones (Ed.), *Miami symposium on the prediction of behavior: Aversive stimulation* (pp. 9−32). Coral Gables, FL: University of Miami Press.

King, N. J., Gullone, E., & Ollendick, T. (1998). Etiology of childhood phobias: Current status of Rachman's three pathways theory. *Behaviour Research and Therapy*, *36*(3), 297−309.

Kruschke, J. K. (2001). Toward a unified model of attention in associative learning. *Journal of Mathematical Psychology*, *45*, 812−863.

Kruschke, J. K., & Blair, N. J. (2000). Blocking and backward blocking involve learned inattention. *Psychonomic Bulletin and Review*, *7*, 636−645.

Lautch, H. (1971). Dental phobia. *British Journal of Psychiatry*, *119*(549), 151−157.

Le Pelley, M. E. (2004). The role of associative history in models of associative learning: A selective review and a hybrid model. *Quarterly Journal of Experimental Psychology. Section B, Comparative and Physiological Psychology*, *57*, 193−243.

Le Pelley, M. E., & McLaren, I. P. L. (2003). Learned associability and associative change in human causal learning. *Quarterly Journal of Experimental Psychology. Section B, Comparative and Physiological Psychology*, *56B*, 68−79.

Liddell, A., & Lyons, M. (1978). Thunderstorm phobias. *Behaviour Research and Therapy*, *16*(4), 306−308.

Lober, K., & Shanks, D. R. (2000). Is causal induction based on causal power? Critique of Cheng (1997). *Psychological Review*, *107*, 195−212.

Lubow, R. E. (1973). Latent inhibition. *Psychological Bulletin*, *79*(6), 398−407.

Lubow, R. E., & Moore, A. U. (1959). Latent inhibition—the effect of nonreinforced pre-exposure to the conditional stimulus. *Journal of Comparative and Physiological Psychology*, *52*(4), 415−419.

Mackintosh, N. J. (1973). Stimulus selection: learning to ignore stimuli that predict no change in reinforcement. In R. A. Hinde, & L. S. Hinde (Eds.), *Constraints of learning* (pp. 75−96). London: Academic Press.

Mackintosh, N. J. (1975). A theory of attention: Variations in the associability of stimuli with reinforcement. *Psychological Review*, *82*, 276−298.

Mackintosh, N. J. (1983). *Conditioning and associative learning.* Oxford: Oxford University Press.

Menzies, R. G., & Clarke, J. C. (1993a). The etiology of childhood water phobia. *Behaviour Research and Therapy*, *31*(5), 499−501.

Menzies, R. G., & Clarke, J. C. (1993b). The etiology of fear of heights and its relationship to severity and individual-response patterns. *Behaviour Research and Therapy*, *31*(4), 355−365.

Menzies, R. G., & Clarke, J. C. (1994). Retrospective studies of the origins of phobias: A review. *Anxiety, Stress, and Coping*, *7*, 305−318.

Merckelbach, H., De Jong, P. J., Muris, P., & Van den Hout (1996). The etiology of specific phobias: A review. *Clinical Psychology Review*, *16*, 337−361.

Mineka, S., & Cook, M. (1993). Mechanisms involved in the observational conditioning of fear. *Journal of Experimental Psychology. General*, *122*, 23−38.

Muris, P., Bodden, D., Merckelbach, H., Ollendick, T. H., & King, N. (2003). Fear of the beast: A prospective study on the effects of negative information on childhood fear. *Behaviour Research and Therapy*, *41*, 195−208.

Öhman, A., Eriksson, A., & Olofsson, C. (1975). One-trial learning and superior resistance to extinction of autonomic responses conditioned to potentially phobic stimuli. *Journal of Comparative and Physiological Psychology*, *88*(2), 619−627.

Öhman, A., & Mineka, S. (2001). Fears, phobias, and preparedness: Toward an evolved module of fear and fear learning. *Psychological Review*, *108* (3), 483−522.

Ollendick, T. H., & King, N. J. (1991). Origins of childhood fears—an evaluation of Rachman theory of fear acquisition. *Behaviour Research and Therapy*, *29*(2), 117−123.

Öst, L. -G., & Hugdahl, K. (1981). Acquisition of phobias and anxiety response patterns in clinical patients. *Behaviour Research and Therapy*, *19*, 439−447.

Öst, L. G., Svensson, L., Hellstrom, K., & Lindwall, R. (2001). One-session treatment of specific phobias in youths: A randomized clinical trial. *Journal of Consulting and Clinical Psychology*, *69*(5), 814−824.

Pavlov, I. P. (1927). *Conditioned reflexes.* Oxford: Oxford University Press.

Pearce, J. M., & Bouton, M. E. (2001). Theories of associative learning in animals. *Annual Review of Psychology*, *52*, 111−139.
Pearce, J. M., & Hall, G. (1980). A model for Pavlovian learning: variations in the effectiveness of conditioned but not unconditioned stimuli. *Psychological Review*, *87*, 532−552.
Perales, J. C., & Shanks, D. R. (2003). Normative and descriptive accounts of the influence of power and contingency on causal judgement. *Quarterly Journal of Experimental Psychology. A, Human Experimental Psychology*, *56*, 977−1007.
Pinel, J. P. J., & Treit, D. (1979). Conditioned defensive burying in rats—availability of burying materials. *Animal Learning & Behavior*, *7*(3), 392−396.
Rachman, S. (1968). *Phobias: Their nature and control.* Springfield: Thomas.
Rachman, S. (1977). Conditioning theory of fear-acquisition—critical-examination. *Behaviour Research and Therapy*, *15*(5), 375−387.
Rescorla, R. A. (1968). Probability of shock in presence and absence of Cs in fear conditioning. *Journal of Comparative and Physiological Psychology*, *66*(1), 1−7.
Rescorla, R. A. (1971). Variations in the effectiveness of reinforcement following prior inhibitory conditioning. *Learning and Motivation*, *2*, 113−123.
Rescorla, R. A. (1974). Effect of inflation of the unconditioned stimulus value following conditioning. *Journal of Comparative and Physiological Psychology*, *86*, 101−106.
Rescorla, R. A. (1980). *Pavlovian second-order conditioning: Studies in associative learning.* Hillsdale, NJ: Erlbaum.
Rescorla, R. A. (1988). Pavlovian conditioning: It's not what you think it is. *American Psychologist*, *43*(3), 151−160.
Rescorla, R. A. (1991). Combinations of modulators trained with the same and different target stimuli. *Animal Learning & Behavior*, *19*(4), 355−360.
Rescorla, R. A. (2000). Associative changes with a random CS–US relationship. *Quarterly Journal of Experimental Psychology. Section B, Comparative and Physiological Psychology*, *53*(4), 325−340.
Rescorla, R. A., & Wagner, A. R. (1972). A theory of Pavlovian conditioning: Variations in the effectiveness of reinforcement and non-reinforcement. In A. H. Blake, & W. F. Prokasy (Eds.), *Classical conditioning II: Current research and theory* (pp. 64−99). New York: Appleton-Century-Crofts.
Seligman, M. E. P. (1971). Phobias and preparedness. *Behavior Therapy*, *2*(3), 307−320.
Shanks, D. R., Holyoak, K. J., & Medin, D. L. (Eds.). (1996). *The psychology of learning and motivation. Causal learning*, *vol. 34*. San Diego: Academic Press.
Soloman, R. L., & Turner, L. H. (1962). Discriminative classical conditioning in dogs paralyzed by curare can later control discriminative avoidance response in the normal state. *Psychological Review*, *69*, 202−219.
Valentine, C. W. (1946). *The psychology of early childhood* (3rd ed.). London: Meuthen.
Watson, J. B., & Rayner, R. (1920). Conditioned emotional reactions. *Journal of Experimental Psychology*, *3*, 1−14.
Withers, R. D., & Deane, F. P. (1995). Origins of common fears—effects on severity, anxiety responses and memories of onset. *Behaviour Research and Therapy*, *33*(8), 903−915.
Wolpe, J. (1961). The systematic desensitization treatment of neurosis. *Journal of Nervous Mental disorders*, *132*, 189−203.
Yule, W., Udwin, O., & Murdoch, K. (1990). The 'Jupiter' sinking: Effects in children's fears, depression and anxiety. *Journal of Child Psychology and Psychiatry*, *31*(7), 1051−1061.