Commentary on **Graham C. L. Davey (1995). Preparedness and phobias: Specific evolved associations or a generalized expectancy bias? BBS 18:289–325.**

Abstract of the original article: Most phobias are focussed on a small number of fear-inducing stimuli (e.g., snakes, spiders). A review of the evidence supporting biological and cognitive explanations of this uneven distribution of phobias suggests that the readiness with which such stimuli become associated with aversive outcomes arises from biases in the processing of information about threatening stimuli rather than from phylogenetically based associative predispositions or "biological preparedness." This cognitive bias, consisting of a heightened expectation of aversive outcomes following fear-relevant stimuli, generates and maintains robust learned associations between them. Some of the features of such stimuli which determine this expectancy bias are estimates of how dangerous they are, the semiotic similarity between them and their aversive outcomes, and the degree of prior fear they elicit. Ontogenetic and cultural factors influence these features of fear-relevant stimuli and are hence important in determining expectancy bias. The available evidence does not exclude the possibility that both expectancy biases and specific evolved predispositions coexist, but the former can explain a number of important findings that the latter cannot.

Selective associations and associative learning: Multiple mechanisms, multiple measures

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Abstract: Davey presents expectancy bias as an alternative explanation for preparedness effects in conditioning; this commentary mentions some of the problems in interpreting UCS expectancy designs vis-à-vis conditioning. A further complication is that other CS characteristics also influence conditioning: A recent experiment provided support for Rescorla and Wagner's (1972) hypothesis that the amount of conditioning is smaller when the CS and UCS elicit similarly valenced initial affective responses (as with a fear-relevant CS and shock UCS). Finally, different physiological systems measure different aspects of the conditioning process, a further consideration in interpreting the results of selective association studies.

Davey's (1995) target article comprises two parts. The first is a critique of conditioning studies performed to test various aspects of the preparedness hypothesis. The second is a review of work by Davey and others regarding differential bias in the expectation of further aversive consequences following fear-relevant stimuli. The expectancy and covariation bias studies form an interesting literature in their own right, and may have potential in illuminating the conditioning data. Comments on cross-cultural effects are also a useful reminder of social factors that can affect a predisposition to acquire fear. To date, however, these studies seem more independent than one would like with respect to the conditioning data. Davey claims that the preparedness effect can be accounted for by a greater expectation of shock following "prepared" stimuli such as snakes or spiders, but I have three concerns about the UCS expectancy paradigm. First, asking subjects to estimate, on a trial-by-trial basis, the probability of shock changes the procedure in multiple and potentially unknown ways. It becomes a dual-task situation, in which subjects must estimate the probability that the experimenter will administer a shock at the same time that they are anticipating potential shock. Due to the experimenter's request for probability estimates throughout, the subject may also direct attention to aspects of the contingencies and develop various covert strategies that might not otherwise occur. Also, it is not clear what the referent for the subject's probability estimate might be, since this is obviously an arbitrary matter controlled entirely by the experimenter. These factors make it difficult to know whether emotional state and/or action dispositions of the subject are comparable in conditioning versus expectancy bias paradigms. Second, where physiological data provide some parallels to the expectancy measures (e.g., Davey 1992, Experiment 3), it is not compelling to argue that the expectancy data therefore explain the physiological data; this seems simply to illustrate a particular example of synchrony in terms of a three-systems model of emotion. Third, recent data from Öhman's laboratory (1993) regarding preparedness effects with backwardly masked stimuli (which subjects cannot recognize) indicate that these effects can occur even when an expectancy cannot be generated. Davey, in his response, suggests that expectancy of threat that has become associated with CS+ can drive pre-attentive effects; however, it is difficult to suppose how expectancy could be established quickly and precisely enough, over just 10 conditioning trials, to account for differential responses to fear-relevant CS+ and CS− pictures presented for 30 msec during extinction, whether extinction is instructed or not (Öhman 1993).

Another consideration is that there are also other effects, in addition to selective associations, which may affect the nature and degree of conditioning. For instance, Davey discusses evidence for the semiotic similarity between CS and UCS as affecting the degree of selective association effects. In contrast to this "belongingness" effect, however, other theorists predict that the degree of conditioning will vary inversely with the initial CS–UCS relationship (Rescorla & Wagner 1972). This position received support in a recent study by Hamm et al. (1993). Different categories of stimuli, occupying different locations in an affective space defined by pleasure (valence) and arousal dimensions, were used as CSs in a differential conditioning design with shock UCS. An overall differential conditioning effect was observed for the eyeblink startle reflex, evoked by presentation of an intense, rapid-onset white noise burst during the CS; this measure has been shown to be a sensitive probe of an ongoing affective state, with larger eyeblink responses linearly related to increasing unpleasantness of foreground stimuli (Lang et al. 1990). Startle magnitude was greater for probes presented during CS+ than CS− throughout extinction. However, the strength of the effect varied significantly with CS content, with stronger differential conditioning for pictures initially rated as more pleasant (e.g., nature scenes, erotica). It is interesting to note that the data for skin conductance also followed the Rescorla-Wagner rule, but in a different manner: pictures initially rated high in arousal, independent of pleasantness (erotica or mutilated bodies), showed the smallest amounts of discrimination between CS+ and CS−, and a similar pattern was observed in post-experimental verbal reports of arousal change.

These results inform the current discussion in multiple ways. First, it appears that any effects of selective association and those reflecting the Rescorla-Wagner rule may interact in determining the extent of conditioning. Snake/spider slides, for instance, are rated as unpleasant and arousing, but to a relatively modest degree compared to such images as mutilated bodies and violence (e.g., Lang 1995). Thus, the Rescorla-Wagner rule would work against a selective association effect, in predicting less conditioning for snake/spider content compared to the flower and nature slides often used as controls; on the other hand, the same rule would predict better conditioning for snake/spider contents compared to stimuli with putative ontogenetic significance, for example, weapons. Second, experimental results could also be affected by individual differences in initial affective reactions – not only to "preparedness" stimuli, but to other categories as well. Finally, the data discourage a simplistic equating of physiological measures in

assessing conditioning, suggesting rather that the function of each physiological system must be considered. The startle probe has been interpreted as indexing the current state of the organism along a biphasic dimension of pleasure–displeasure; skin conductance appears to reflect changes along an arousal dimension; cardiac acceleration has been interpreted in terms of mobilization for escape or avoidance (e.g., Lang 1995). All of these factors act in concert to affect the conclusions regarding any given experiment, and may well contribute to the lack of consistency observed in the preparedness literature.

It is not clear how the expectancy bias hypothesis that Davey presents in the target article would address these multiple issues. At this point, expectancy bias seems to be a promising area of research that may account for some of the variance in the results of selective association studies, but does not represent an overarching theory which can subsume all of the conditioning data.

No convincing evidence for a biological preparedness explanation of phobias

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Abstract: The nonrandom distribution of fears is not as clearly related to phylogenetically survival relevance as preparedness theory seems to imply. Although delayed extinction reflects some of the best human evidence for preparedness, even this phenomenon is not as robust as it once seemed to be. Apart from the evidence reviewed by Davey, recent studies from our laboratory provide further evidence for an expectancy bias model of selective associations.

We strongly agree with Davey's (1995t) position that an expectancy bias can account more parsimoniously for the pertinent characteristics of fears and phobias than biological preparedness. In this commentary we first argue that Davey is too mild in his evaluation of the preparedness theory of phobias. We then present some recent data that add to the evidence in favour of Davey's expectancy bias explanation.

The greatest virtue of Seligman's (1971) preparedness theory was that it could explain the nonrandom distribution of fears. Although several fear surveys clearly showed that some stimuli are more frightening than others, however, it should be stressed that an interpretation of this finding in terms of biological preparedness is not without problems. The most important drawback of these studies is that the results obtained are highly dependent on the items covered by the questionnaires that are used. For instance, Agras et al. (1969) used items derived from traditional fear scales and fund that fear of snakes is the most prevalent fear. Clearly this finding invites a preparedness interpretation of phobias. However, using a different questionnaire, Kirkpatrick (1984) found that, for women, fears of roller-coasters and untimely death were most prevalent, whereas for men the fear of being punished by God ranked highest. Note that the fear rank order in the latter study cannot easily be explained in terms of preparedness (i.e., it seems rather hopeless to trace back, say, the fear of God to some "phylogenetically relevant" avoidance). Apart from this, a more fundamental point can be raised. As long as there are no independent criteria for defining phylogenetically survival relevance, one runs the risk of becoming a victim of a circular argument: certain stimuli are considered more survival relevant just because they appear more frequently.

Davey (sects. 1, 3.4.1, and 3.4.4) states that delayed extinction reflects some of the best human laboratory evidence for preparedness. Note, however, that this phenomenon is not as robust as it once seemed to be. Several authors reported failed replications of the Uppsala studies (Hugdahl & Johnsen 1989; Kirsch & Boucsein 1994; but see Schell et al. 1991; McNally & Foa 1986; Merck-

elbach et al. 1987). Furthermore, in the recent backward masking studies from Öhman's lab, it appears that no resistance to extinction emerged while nonmasked FR stimuli were used (Öhman & Soares 1993; Exp. 2). From a preparedness point of view, this variability in the delayed extinction phenomenon is hard to explain. Meanwhile, this pattern of results can be explained easily by assuming some variability in subjects' UCS expectancies (cf. Davey 1992).

Whereas some evidence of delayed extinction emerged from laboratory experiments, the results of clinical studies are embarrassing for a preparedness theory of phobias. For instance, Öst (1989) showed that most simple phobias can be satisfactorily treated within 2.5 hours. Also, in our spider phobia research project, some 300 severe spider-phobic women were successfully treated as in Öst (1989).

Another feature of phobia that is misrepresented in the older preparedness literature is the suggestion that "prepared" phobias are typically noncognitive. This position is exemplified by several studies of Öhman and colleagues. These studies (e.g., Öhman et al. 1975) showed that even when "no shock" instructions were given, fear-relevance effects still appeared. Öhman and Hugdahl (1979) argued that these effects provide a laboratory model for the irrationality of phobias. That is, the finding that subjects responded with autonomic fear-reactions despite knowing that no further shocks would be presented was taken as an *a fortiori* argument for aversive autonomic conditioning as a proper model of phobias. However, in contrast to Öhman and Hugdahl's (1979) suggestion, small-animal phobias are not at all noncognitive anxiety disorders. For instance, spider phobics seem to have highly developed sets of negative ideas about spiders and about their own reactions during confrontation with spiders (e.g., Arntz et al. 1993). The finding that animal phobics have an abundance of attack- and predator-related ideas about their phobic object fits better with a cognitive (S-S) than with a biologically prepared S-R account of phobic fear.

Apart from the delayed extinction phenomenon, several other predictions from the preparedness theory were tested to destruction in laboratory studies. For instance, Siddle and colleagues made several idle attempts to show that aversive conditioning to FR (fear-relevant) stimuli is not susceptible to latent inhibition (Booth et al. 1989) or UCS devaluation (Siddle et al. 1988). Furthermore, several authors failed to show that FR stimuli are contra-prepared for safety-signal conditioning in humans (McNally & Reiss 1982; Wilkinson et al. 1989).

A final point that can be raised about predictions that flow from the preparedness theory concerns the results of twin studies. Eysenck (1987) remarked that "it is curious that Seligman and the Uppsala School, in advocating their model of preparedness, fail to mention the most significant evidence in its favor, namely the genetic evidence" (p. 396). But is there such genetic evidence? Andrews et al. (1990) found in their twin study on anxiety disorders evidence for the inheritance of a specific diagnosis (see also Kendler et al. 1992). This is not what one would expect on the basis of the preparedness theory. Obviously, the genetic process implied in the preparedness theory would lead to the prediction that there is a strong concordance in monozygotic twins, for example, for simple phobias. This prediction is not supported by these genetic studies.

Whereas the evidence for a preparedness explanation of selective associations is rather weak, there are a number of studies that sustain cognitive accounts of selective associations. Apart from the evidence reviewed by Davey, recent studies in our laboratory using an illusory correlation paradigm, provide further evidence for an expectancy bias model of selective associations. De Jong (1995) and De Jong et al. (1995) showed that high as well as low spider-fearful subjects have an UCS (unconditioned stimulus) expectancy bias for both weapons and spiders. Similar results were obtained for facial expressions (De Jong & Mulkens 1994). Both high and low socially-anxious subjects displayed an UCS expectancy bias for angry faces.

Perceived dangerousness was found to be one of the determi-

nants of UCS expectancy bias (De Jong 1995; De Jong & Mulkens 1994). The finding that shocks after fear-relevant slides have a relatively large physiological impact (De Jong & Merckelbach 1991; De Jong et al. 1995) supports the idea that the similarity in emotional responses might be another factor promoting fear-relevant expectancy biases and illusory correlations (IC). A study of Tomarken et al. (1995) further supports the view that the affective profile of cue and consequence is an important determinant of the generation of ICs (illusory correlation). Note that neither of these findings necessarily presumes preparedness; both can be readily explained by cultural transmission or experiential factors. This position is supported further by De Jong et al. (1990). They showed that even apart from *a priori* characteristics of cue and consequence, it is possible to induce an IC between FI (fear irrelevant) stimuli and shock by means of a conditioning-like procedure. Most important, the results of that study demonstrated that an IC, once induced, can become "self-supporting." Thus, an acquired (illusory) UCS expectancy can act in such a way as to promote the assessments of differential associations.

To summarize, as the preparedness theory relies heavily on evolutionary arguments, it is a hypothesis that cannot be subjected to direct experimental tests (DeSilva et al. 1977). Hence such a hypothesis can only be accepted if it generates a set of predictions that meet two criteria. First, these predictions must be corroborated by research. Second, these should not flow from alternative hypotheses. As the available data violate both criteria, we must conclude that there is no convincing evidence for a preparedness explanation of phobias. The available evidence seems to converge upon Davey's conclusion (sect. 6.3) that there are theoretical (and empirical) reasons for exploring more fully the possible cultural influence on fear distribution.

Author's Response

The merits of an experimentally testable model of phobias

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Abstract: A series of arguments are presented by **De Jong & Merckelbach** which suggest that biological preparedness has been received significantly less critically than it should have been. I agree fully with their assessment. **Cuthbert** raises four questions about the applicability of the expectancy bias hypothesis to selective associations in human conditioning. This response argues that none of these four examples is necessarily problematic for the hypothesis.

What is perhaps most striking about the commentary by **de Jong & Merckelbach** is the phrase towards the end which claims that "we must conclude that there is no convincing evidence for a preparedness explanation of phobias." Many would express surprise that a theory which is already enshrined as psychological fact in most introductory psychology text books should be criticised in this way. However, I have to agree that their commentary points to many features of the relevant literature which are ambiguous in their support of biological preparedness or are clearly inconsistent with it. In the early 1970s, biological preparedness was being debated as a possible explanation of the uneven distribution of phobias at a time when evolutionary

approaches to the understanding of learning were becoming fashionable and persuasive. Although ecological analyses are clearly necessary for an understanding of learning, the preparedness theory of phobias appears to have been uncritically carried along on the evolutionary tide. De Jong & Merckelbach point out that over the last 25 years, many findings potentially contrary to the preparedness theory have been conveniently ignored. These include the many failures to obtain experimental findings predicted by preparedness theory, the failure of preparedness theory to find any support in the realm of clinical phobias, and the ease with which so-called prepared phobias can now be treated using contemporary treatment packages. Added to this is the difficulty of finding the evidence that might refute an evolutionarily-based explanation such as preparedness.

I can only agree whole-heartedly with **de Jong & Merckelbach** that most of the data relevant to biological preparedness can be explained by an expectancy bias account. Not only that, but expectancy bias provides a testable model of phobias in a way which the preparedness hypothesis does not.

Cuthbert suggests that asking the subject to respond to a conditioned stimulus (CS) while assessing unconditioned stimulus (UCS) probability may change the procedure from a simple conditioning one in unknown ways. There are two points to make here. First, performance on conditioning procedures does not seem to differ in any substantial way when subjects are asked to make UCS probability assessments either on-line or post-experimentally (cf. Davey 1992). Second, if there was any effect of on-line UCS probability rating on, say, contingency awareness, then this should not substantially change any conclusions drawn from conditioning studies that have compared fear-relevant (FR) and fear-irrelevant (FI) CSs, since on-line UCS probability rating is a condition that is common to both – unless, of course, there are some good grounds for supposing that on-line judgments differentially influence conditioning to FR and FI stimuli.

Cuthbert argues that although there are parallels between the expectancy data and the physiological data the claim that the former explain the latter is not compelling. This is true. However, I have pointed out in the past that there is some evidence that changes in UCS expectancy precede physiological changes in the conditioned response (CR). Thus, at the very least, the UCS expectancy ratings are not simply *post hoc* rationalisations based on the subject's perception of CR strength (Davey 1992).

Cuthbert suggests that the apparent preparedness effects observed in the subliminal backward masking studies of Öhman and colleagues (1993) cannot be due to expectancy bias – even preattentive expectancy biases – because it is difficult to suppose how the expectancy could be established so quickly over just 10 conditioning trials. However, the bias hypothesis does not assume that the expectancy develops over acquisition trials; it exists pre-experimentally in the form of judgments that the subject will have already made about the nature of the CS and its fear- and danger-relevance (Davey 1992; Davey & Dixon 1996).

Cuthbert claims that the Rescorla-Wagner (1972) model of associative strength in Pavlovian conditioning studies would predict that semiotic similarity between CS and UCS results in weaker conditioning between CS and UCS than when there is no semiotic similarity between the

two stimuli. This, claims Cuthbert, is because the degree of conditioning will vary inversely with initial CS-UCS similarity. The Rescorla-Wagner model and its contemporaries (e.g., Pearce-Hall model, 1980) state that the amount of associative strength that will accrue to a CS on any conditioning trial will be inversely proportional to the amount of associative strength that has already accrued to that CS. The expectancy bias model assumes that associative strength between CS and threatening outcomes (UCS) has already accrued prior to explicit conditioning trials, and that any semiotic similarity between CS and UCS will have contributed to this pre-experimental associative strength. Thus, at the end of the first conditioning trial it is true that the increment in associative strength between a fear-relevant CS and an aversive UCS will be smaller if the CS and UCS share semiotic similarity than if they do not. However, at the end of the first conditioning trial using a fear-relevant CS, the associative strength between CS and UCS will not just be equal to the associative strength generated on that trial, but to the associative strength generated on that trial plus the associative strength generated by the pre-experimental expectancy bias. The study cited by Cuthbert (Hamm et al. 1993) uses a startle probe to assess conditioned response strength. However, there is no reason to suppose that the magnitude of the startle response elicited during a CS is an indicator of the degree of associative strength that has accrued to the CS (which is what the Rescorla-Wagner model is predicting); it may simply be a performance effect resulting from the interaction between the similar or dissimilar affective states elicited by the startle probe and the CS.

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