CASE HISTORIES AND SHORTER COMMUNICATIONS

UCS Inflation in the aetiology of a variety of anxiety disorders: some case histories

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Summary—This paper reports a number of case histories which illustrate the involvement of UCS inflation processes in the aetiology of a variety of anxiety disorders including agoraphobia, simple phobia, PTSD, obsessive-compulsive disorder and panic disorder. These cases are discussed in the context of: (i) renewed interest in cognitive features of conditioning models of anxiety disorders; and (ii) the implications that such models might have for an understanding of the aetiology and treatment of anxiety-based disorders.

INTRODUCTION

In recent years there has been a revival of interest in the application of conditioning models to the understanding of anxiety disorders, and this revival has largely been stimulated by both recent advances in our understanding of conditioning in humans and the implication of cognitive factors in conditioning processes (cf. Davey, 1987, 1989, 1992a). Of particular importance in this revival has been the discovery that the strength of a conditioned response (CR) can be affected not just by associative processes that govern the strength of an association between the conditioned stimulus (CS) and the unconditioned stimulus (UCS), but also by a variety of processes which influence the S's evaluation of the UCS (cf. Davey, 1989, 1992a; Poulos, Furedy & Heslegrave, 1979; Baeyens, Eelen, Van den Berg & Crombez, 1992). Davey (1992a) has reviewed these recent developments and described in detail the implications that this kind of two-process model of human classical conditioning has for our understanding of the aetiology and treatment of anxiety-based disorders. In particular, when processes which influence UCS evaluation are added to an associative model of classical conditioning, most of the traditional criticisms of conditioning models of fears and phobias no longer apply.

This two-process model of human classical conditioning basically suggests that once an association has been formed between a CS and UCS (either through experience with the contingencies, socially transmitted information or observational learning), subsequent presentations of the CS will activate a representation of the UCS. However, the strength of the CR that is mediated by this representation will depend on the S's evaluation of the UCS; if the S evaluates the UCS as being particularly aversive it will evoke a strong fear CR, if it is evaluated as relatively benign it will evoke, at best, only a weak fear CR. Hence, those factors which influence the evaluation of the UCS will have an important role to play in determining the strength of the CR.

Davey (1989, 1992a) identifies a number of processes which appear to be involved in the evaluation of an aversive UCS, and hence will play an important role in modulating the strength of any CR. These include: (i) experience with a similar UCS of either greater or lesser intensity. For instance, post-conditioning experience of individual presentations of a UCS of greater intensity than that used during conditioning immediately produces a greater magnitude CR on the next CS presentation [normally called a UCS inflation effect (White & Davey, 1989; Merckelbach & de Jong, 1991)]. Similarly, post-conditioning experience of UCSs of weaker intensity immediately lead to a reduction in the magnitude of the CR [a UCS devaluation effect (Davey & McKenna, 1983)]; (ii) socially or verbally transmitted information about the UCS. For example, Ss may be told that on future occasions the UCS will be of a different intensity or will change in its probability of occurrence. Following such procedures, Ss usually emit a CR which is changed in magnitude in accordance with the content of this information (Davey, 1983; Davey & McKenna, 1983); and (iii) discrimination and interpretation of the intensity of the S's own reactions to either the CS or UCS. For instance, if Ss believe they are emitting a strong fear reaction to an aversive UCS, this will increase the magnitude of the fear CR to the CS (Cracknell & Davey, 1988). Similarly, if Ss are given false CR feedback suggesting they are emitting either a strong or a weak CR to the CS, the kind of feedback given will directly influence the actual physiological CR that is evoked by the CS (Davey, 1987; Russell & Davey, 1991). In both cases, change in the strength of the CR as a result of the S's perceptions of their own responses appears to result from changes in their evaluation of the UCS (Davey, 1988).

Clearly, when a conditioning analysis is applied to the acquisition and remission of anxiety disorders, these processes of UCS valuation will take a central role—providing, as they do, a means of explaining fluctuations in responding without concomitant changes in associative strength between CS and UCS. However, although Davey (1989, 1992a) and White and Davey (1989) have provided a number of hypothetical examples of the way in which UCS revaluation processes might influence the acquisition and remission of anxiety-based disorders, there has been no systematic attempt to collect actual clinical examples of UCS revaluation in the aetiology of anxiety disorders. The present paper represents a preliminary attempt to rectify this by reporting a number of case histories which appear to display the prima facie involvement of UCS revaluation processes in the acquisition of the clinical disorder.

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UCS inflation through experience with a similar UCS of greater intensity

Case 1. L.L. (male, aged 37 yr) had always been mildly anxious in social situations, and this mild anxiety was mostly accompanied by physical symptoms. The most salient symptom was intestinal unease. On one occasion, when L.L. was alone at home and not anxious at all, similar symptoms of intestinal unease led to an uncontrollable attack of diarrhoea. From that moment on, L.L. catastrophically interpreted the symptoms of intestinal unease that he regularly experienced in company as a signal for losing control again. He became extremely anxious and developed severe agoraphobic symptoms.

Comment. This represents a fairly simple example of UCS inflation as a result of experience with a UCS of greater intensity or severity. The consequence (UCS) that was originally associated with social situations (CS) was only mild intestinal discomfort. As the result of an 'off-the-baseline' experience which inflated the aversiveness of this consequence, subsequent experiences with the CS evoked a significantly more aversive UCS representation which mediated severe phobic reactions.

UCS inflation through socially or verbally transmitted information

Case 2. H.B. (female, aged 35 yr) applied for therapy complaining of a severe spider phobia (DSM-III-R criteria of simple phobia). Because of her father's occupation, she had lived in Rio de Janeiro in Brazil during her childhood. Once, at the age of 10 yr, she woke at night when a large tropical spider walked over her face. At the time she reported not being particularly frightened. However, when she told her parents about the incident the following morning, they were extremely concerned and looked very alarmed. From that moment on, H.B. was extremely frightened of spiders and exhibited severe phobic behaviour.

Comment. All three examples illustrate how relatively innocuous events or situations (respectively spiders, working in a bank and knives) become, first of all, CSs predicting relatively minor consequences (nominal UCSs). However, because of subsequent exposure to socially and verbally transmitted information, the aversive properties of these consequences become dramatically inflated such that the CSs would ultimately activate an aversively-evahiated UCS representation which mediate anxiety related responses. These examples also demonstrate how UCS revaluation can be a factor in the aetiology of a variety of anxiety disorders, including simple phobias, post-traumatic stress disorder and obsessive-compulsive disorder.

DISCUSSION

The case histories discussed in the present paper provide a number of prima facie examples of UCS inflation in the aetiologies of a variety of anxiety disorders including agoraphobia, simple phobia, PTSD, obsessive-compulsive disorder and panic disorder. The apparent modes of UCS inflation also range across those already identified from laboratory studies of fear conditioning, including experience with a similar UCS of greater intensity, social and verbally transmitted information about the UCS, and observation and reinterpretation of the individual's own physiological responses. Such clinical evidence is important in helping to validate contemporary conditioning models of anxiety disorders, and, in particular, in demonstrating the central importance of UCS revaluation processes in such accounts (cf. Davey, 1989, 1992a).

The process of UCS inflation in the aetiology of anxiety disorders also helps to understand instances where: (i) onset is significantly delayed after trauma; and (ii) patients appear unable to identify the event that precipitated the first symptoms.

The best example of the former is PTSD, where it is necessary according to DSM-III-R to specify delayed onset. It is interesting to note that when Vietnam veterans returned from the war, it was originally thought that the Vietnam war had produced fewer psychiatric casualties than World War II, but subsequent findings have shown otherwise (Davey & Neale, 1992a).
1982, p. 148). It may be possible that the dimension of the tragedy did not become apparent to veterans until some time after returning from action, when the aversiveness of their combat experiences may have been inflated by exposure to TV film of events, fatality figures, etc. (see resemblances with Case 3).

In relation to an understanding of patients' inability to identify precipitating events, a consistent observation among clinicians is that, particularly with panic disorder, the first panic attack is often preceded by one or more negative life events, yet few patients can identify the precipitating event when specifically asked questions like "what caused your first panic attack?" Nevertheless, systematic questioning reveals that about 80% of these patients describe one or more negative life events preceding their first panic. Often patients are unaware that the two may be connected (Barlow, 1988, p. 216), and this may be because the critical mediating variables are revaluation incidents of the kind that lead to trauma inflation [see White and Davey (1989) for a specific hypothetical example of how this may occur].

The list of factors discussed in this paper which might lead to UCS revaluation is not meant to be exhaustive, but represents those factors that have been positively identified in the laboratory. Other factors may be operating on a more general level to influence the evaluation of a potentially traumatic UCS, and thus determine whether an anxiety disorder becomes manifest. One such general factor is the possession of generalised coping strategies which permit the individual to cognitively devalue or neutralise potentially traumatic experiences. Such cognitive strategies have been identified as a distinctive set of coping strategies which individuals utilise in order to neutralise or devalue potentially stressful or traumatic events (Davey, 1992b). Not surprisingly, recent studies have shown that the use of these generalised devaluing strategies is inversely related to scores on the Fear Survey Schedule (FSS), and both simple phobics and panic disorder patients report significantly less use of such strategies than normal control Ss (Davey, Burgess & Rashes, 1992). Further research comparing normal control Ss with anxiety-disordered populations may reveal other generalised coping strategies which may help to insulate the individual from traumatic experiences which may precipitate anxiety disorders.

Finally, if UCS inflation can be positively identified as a primary precipitating factor in a variety of anxiety disorders, what implications might this have for ameliorative therapy? First, the most obvious implication is that the disorder is centred around an event whose traumatic impact has been inflated. Arguably, therefore, successful treatment would seem to require the effective devaluation of this traumatic event. However, while this principle itself might be an important one, having knowledge of the specific process by which the UCS was inflated in individual cases (e.g. whether by direct experiences with the UCS or socially transmitted information about the UCS, etc.) does not necessarily provide unambiguous information about the best way to effectively devalue the inflated UCS. For instance, while information provided about the UCS in instructions can generate immediate fear CRs in laboratory fear conditioning studies (e.g. Davey & McKenna, 1983; Dawson & Grings, 1968; McComb, 1969; Brewer, 1974), providing similar information about the non-occurrence of the UCS in instructed extinction procedures regularly fails to eliminate an already-conditioned fear CR (Bridger & Mandel, 1965; Mandel & Bridger, 1967; Fuhrer & Baer, 1969; Wilson, Fuhrer & Baer, 1973; Davey, 1992c). Clearly, because a process contributes to UCS inflation does not necessarily imply that it will also contribute to effective UCS devaluation. An illustrative example might help to clarify how those processes which contribute to UCS inflation in the aetiology of an anxiety disorder might not be effective contributors to UCS devaluation during therapy. For instance, an individual may become airplane phobic as a result of contact with socially or verbally transmitted information about airplane travel: they may read news stories of terrifying airplane crashes or have a close friend or relative involved in an airplane crash. This information would inflate the aversive consequences of airplane travel for the individual. However, as the phobia develops, the effective consequences of airplane travel for that individual may shift from fear of crashing (as experience shows that the risk of airplanes crashing is very slim) to fear of panicking whilst in the airplane. The effective UCS is no longer the fear of crashing but the fear response itself. This being the case, only those treatment procedures which address the devaluation of this response are likely to be effective, and this may explain the success of those therapies, such as exposure therapies, which enable the individual to perceive reductions in their fear responses to their phobic cue. The relevance of this kind of approach depends very much on what anxiety-disorder patients perceive as the effective consequence of interaction with their anxiety-provoking situations, and research still needs to be carried out to identify the distribution of effective cue-consequence relationships that accompany different types of anxiety disorder. Such research may help to answer the time-honoured question "what is the UCS in conditioning models of anxiety-disorders?" The answer may be that the effective UCS actually changes during the development of a disorder.

What is additionally required before UCS devaluation can make a significant contribution to therapy is an understanding of the way in which various sources of information are given priority during both UCS inflation and UCS devaluation. While social and verbally transmitted information about the UCS may be sufficient to produce effective and reliable UCS inflation, such a source of information might be relatively less important in effecting UCS devaluation. While more laboratory work needs to be done on this subject, some indication of the hierarchical importance of various sources of information about the UCS might be obtained by analysing the effectiveness of existing therapies. It may be, for instance, that while a variety of processes can contribute to UCS inflation, relatively fewer contribute effectively to UCS devaluation, and some sources of information (e.g. self-observation of the reduction in physiological responses to the CS or UCS) may be given considerably more importance than others.

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REFERENCES


