THE CONDITIONING THEORY OF FEAR-ACQUISITION: A CRITICAL EXAMINATION

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Summary The conditioning theory of fear acquisition is outlined and the supporting evidence and arguments presented. It is argued that the theory lacks comprehensiveness and is also inadequate in other respects.

Six arguments against acceptance of the theory are advanced. People fail to acquire fears in what are theoretically fear-evoking situations (e.g. air raids). It is difficult to produce conditioned fear reactions in human subjects in the laboratory. The theory rests on the untenable equipotentiality premise. The distribution of human fears is not consistent with the theory. Many phobic patients recount histories inconsistent with the theory. Lastly, fears can be acquired indirectly, contrary to the demands of the conditioning theory. It is suggested that fears can be acquired by three pathways: conditioning, vicarious exposures and by the transmission of information and instruction. Vicarious and informational transmission of fears can take place in the absence of direct contact with the fear stimuli.

In our protracted attempts to understand the nature of fear we have failed to notice that, despite repeated exposures to dangerous or stressful situations, most people acquire comparatively few fears. Psychologists have been so pre-occupied with trying to understand how and why we acquire fears that the equally interesting questions of how and why we fail to acquire lasting fears in dangerous situations, have been neglected. As I hope to show, there are many circumstances in which, according to contemporary theories, people should acquire fears but fail to do so. Indeed, there are grounds for suspecting that the 'exceptions' outnumber the predicted outcomes. Although most people experience a wide range of theoretically fear-evoking situations, they remain comparatively fearless. For this reason and for several others, the conditioning theory of fear acquisition cannot be regarded as a comprehensive explanation. Like some other theories, the conditioning theory of fear acquisition explains more than is observed.

Before the arguments and evidence are introduced, it should be noted that the emphasis of the present examination is on the acquisition of fear; the persistence of fear and of fear-related behaviour is considered elsewhere (Rachman, 1976). Also, the emphasis of this paper is on human fear acquisition, but reference will be made to the laboratory literature on how animals acquire fear.

Although the conditioning theory of fear acquisition, in one or other version, appears to be influential there has been comparatively little debate about its merits. The theory is significant in its own right and also because it features in other theories, especially those dealing with avoidance behaviour. From a practical point of view, it underlies a good deal of the thinking associated with those forms of behaviour therapy that deal in fear reduction (such as desensitization, flooding etc; see Wolpe, 1958; Eysenck and Rachman, 1965; Rimm and Masters, 1974). One version of the theory originated in the important and influential work of Mowrer (1939) He stated that "the position here taken is that anxiety is a learned response, occurring to signals (conditioned stimuli) that are premonitory of (i.e. have in the past been followed by) situations of injury or pain (unconditioned stimuli)". He went on to argue that fear "may effectively motivate human beings" and that the reduction of fear "may serve powerfully to reinforce behaviour that brings about such a state of relief or security". Mowrer's theory owed most to the writings of Pavlov, Freud, James and Watson and the developments from Mowrer's position follow in the same tradition, amply encouraged by the fruitful research on the induction of experimental neuroses. For the remainder of the present
examination, I will take as my main example of a conditioning theory of fear acquisition a version with which I have been associated.

Basing their theory on a combination of research findings drawn from the field of experimental neuroses (including the original experiments of Wolpe, 1958), their clinical observations, and the influential writings of Mowrer (1939), Watson and Rayner (1920) and others, Wolpe and Rachman (1960) proposed the following: "Any neutral stimulus, simple or complex, that happens to make an impact on an individual at about the time that a fear reaction is evoked, acquires the ability to evoke fear subsequently... there will be generalization of fear reactions to stimuli resembling the conditioned stimulus". This conception was elaborated by Rachman and Costello (1961) who summarized the essentials of the theory in six statements. After restating the elements of the original proposition, they added three new features. It was argued that neutral stimuli which are of relevance in the particular situation, are more likely to become fear signals, that the repetition of the association between fear and the new phobic stimuli will strengthen the fear, and that associations in high intensity of fear situations and neutral stimuli are more likely to produce fear reactions. In 1965, three more elements were added and further consideration given to the determinants of the strength of the fear. Eysenck and Rachman (1965) proposed that fear reactions are more likely to occur under excessive conditions of confinement. They also incorporated the motivating qualities of the fear reaction in a manner originally proposed by Mowrer (1939). Shortly after this extension was published, some of the difficulties which are now acknowledged, began to appear. This led to a revision which for the first time incorporated the possibility, indeed the certainty, that emotional reactions, including fear, can be acquired vicariously (Rachman, 1968). This revision, which allowed that fears may be acquired directly or vicariously, led to a critical scrutiny of the whole theory.

Before embarking on an analysis of the limitations of the conditioning theory, it might be as well to give a concise account of some of the arguments and evidence in favour of the theory.

**ACQUISITION OF FEARS BY CONDITIONING**

The major features of the theory are as follows. It is assumed that fears are acquired and that the process of acquisition is a form of conditioning. Neutral stimuli which are associated with a fear or pain-producing state of affairs, develop fearful qualities, i.e. they become fear CS's. The strength of the fear is determined by the number of repetitions of the association between the pain/fear experience and the stimuli, and also by the intensity of the fear or pain experienced in the presence of the stimuli. Stimuli resembling the fear-evoking ones also acquire fearful properties i.e. they become secondary CS's. The likelihood of fear developing is increased by confinement, by exposure to high intensity pain and/or fear situations and by frequent repetition of the association between the new CS and the pain/fear. It was proposed further that once objects or situations acquire fear-provoking qualities, they develop motivating properties, i.e. a secondary fear drive emerges. Behaviour patterns such as avoidance actions, which successfully reduce fear, increase in strength.

Evidence to support the theory was drawn from five main sources (Eysenck and Rachman, 1965). Research on the induction of fear in animals, the development of anxiety states in combat soldiers, experiments on the induction of fear in a small number of children, clinical observations (e.g. dental phobias*) and a few experiments on the effects of traumatic stimulation.

The theory was bolstered by a multitude of experiments on laboratory animals and most strongly by the research on what became known as 'experimental neuroses' (see reviews by Wolpe, 1958; Broadhurst, 1960; 1972). Evidently it is easy to generate fear reactions in animals by exposing them to a conjunction of neutral and aversive stimu-

*Although it is sometimes useful to distinguish between fears and phobias, it is preferable to avoid this distinction when assessing the fear-acquisition theory. The term 'phobia' is however used whenever it seems to have significance in the original source.
The conditioning theory of fear-acquisition

lation, usually electric shock. These acquired fear reactions (usually inferred by the emergence of avoidance behaviour, physiological disturbance, disruptive behaviour or by some combination of these three indices) can be produced readily by employing conventional conditioning procedures. There is little room for doubt about the facility with which fear reactions can be conditioned—at least in animals tested under laboratory constraints. As we shall see, however, there are grounds for doubting whether the laboratory process of fear acquisition provides an adequate foundation for theorizing about fear acquisition in non-laboratory conditions, and in human subjects in or out of the laboratory. The demonstration that fears can be generated by conditioning does not mean that that is how they are ordinarily acquired.

Observations and analysis of people under combat conditions provide ample evidence (Gillespie, 1945; Lewis and Engle, 1954) of the production of intense fear as a result of traumatic stimulation. For example, Flanagan (1948) states that the overwhelming majority of combat air-crew surveyed had experienced fear during missions. Although these reactions were relatively transient, they often presaged the development of (reversible) combat fatigue. A minority of combat crew-members developed significant, lasting fears. Grinker and Spiegel (1945) provided detailed, vivid accounts of the fearful and disturbed behaviour that can arise as a result of air-combat. The affected fliers developed lasting feelings of intense apprehension, startle reactions produced by trivial sounds, uncontrollable tremors, marked tension, restlessness, nightmares and a range of bodily symptoms. According to the analyses carried out by Grinker and Spiegel, these intense fear reactions were precipitated either by a single catastrophic event or by repeated experiences of severe, long-lasting, fear-provoking missions. In addition, some of the airmen appeared to break down after repeated exposures to mildly or moderately fearful situations—what one might describe as repeated sub-traumatic experiences.

In clinical practice it is not uncommon for patients to give an account of the development of their fears (e.g. they date from a specific experience) that can easily be construed in conditioning terms. Similarly, from a study conducted on 34 cases of dental phobia (Lautch, 1971) we learn that every subject reported having had a traumatic dental experience (e.g. fearing suffocation from an anaesthetic mask) on at least one occasion in childhood. It is worth mentioning in passing however, that these 34 people were found to be generally neurotic while another 10 comparison subjects who had experienced comparable traumatic incidents with dentists during their childhood, showed little sign of dental fears.

Watson and Rayner (1920) provided a classical demonstration of the genesis of a fear in a young child, little Albert. This report is too well-known to bear repetition but it is worth drawing attention to the fact that their finding was tested by a number of research workers, with varying degrees of success and failure (see page 381 below).

A final source of support for a conditioning theory of fear acquisition comes from experiments in which subjects were given injections of scoline which produces a temporary suspension of breathing (Sanderson et al., 1963). Not surprisingly, most of the subjects who were subjected to this harrowing experience developed intense fears of the stimuli encountered in, or connected with the experimental situation. In fact there was a tendency for the intensity of their fears to increase, even in the absence of further unpleasant experiences.

Although the conditioning theory of fear acquisition does not require single trial or traumatic onset, fears which arise in an acute manner are more readily accommodated by the theory than are those of uncertain onset. Even though acute onset fears are easily accommodated (partly I suspect because of our conception of conditioned fear is based on laboratory experiments in which the UCS is typically traumatic), we also have to account for fears that are produced by experiences of a sub-traumatic or even of a non-traumatic nature (see Eysenck and Rachman, 1965).

Fears which emerge in the absence of any identifiable learning experience present difficulties for the theory. Hence, fears which develop gradually (e.g. social fears, see Marks, 1969) and cannot be traced to specific occurrences are a potential embarrassment.
Although the importance of the phenomenon of acquired taste aversions was not made evident until 1966, it can be used to provide buttressing for the conditioning theory. Garcia and Koelling (1966) and Garcia et al. (1966) were the first to demonstrate that strong and lasting aversive reactions can be acquired with ease when the appropriate taste stimulus is associated with illness, even if a long period elapses between tasting the food and the onset of illness. The phenomenon was given the catch-name of 'sauce Bernaise' by Seligman and Hager (1972) who further elucidated the theoretical significance of the research. If we allow that the genesis of these taste aversions is a form of conditioning, and if we also agree to an equation between the acquisition of a taste aversion and the acquisition of a fear, this recent literature (reviewed by Seligman and Hager, 1972) may yet provide the best evidence for a conditioning theory of fear acquisition. In Seligman's (1972) fascinating use of these findings, phobias are seen as instances of highly 'prepared' learning—that prepared learning is selective, highly resistant to extinction, probably noncognitive and can be acquired in one trial" (p. 451). The concept of prepared phobias is dealt with later in this paper; for the moment, we are concerned with the arguments adduced in support of the conditioning theory.

The idea that taste aversions and fear are related, receives some indirect support from findings such as the elevated incidence of food aversions among neurotic subjects. In an interesting study reported in 1945, Wallen compared 214 normal adults with 95 subjects of comparable age who had been rejected from the Navy on the grounds of neurosis. The neurotic subjects reported four times as many food aversions as the normal subjects. As neurotics are also known to have more fears than non-neurotic subjects (e.g. Adams and Rothstein, 1971), it seems possible that they have a sensitivity of some type that pre-disposes them to acquire fears and aversions more easily than other people. If the acquisition of taste aversions reported in the recent literature is used as a basis for supporting or expanding the conditioning theory of fear, it will have to take into account the unexpected temporal stretch of the taste aversion phenomenon (i.e. the delay between CS and UCS). Classical conditioning is of course expedited by temporal proximity between stimuli; although there are convincing examples of conditioned responses being established even with prolonged temporal delays, these tend to be exceptions. Experimentally-induced taste aversions on the other hand are powerful and rapidly established, even when long delays occur between the two stimulus events. It is plain therefore that if the taste aversion phenomenon is to provide the basis for a new or revised conditioning theory of fear we will have to de-emphasize the temporal qualities of classical conditioning processes.

It is hard to escape the conclusion that the evidence provides strong support for the notion that fears can be acquired by a conditioning process. This conclusion is justified even though some of the evidence is open to contrary interpretations, or is inherently weak. The strongest evidence, both in the sense of its replicability and completeness, comes from the genesis of fear in laboratory animals. This voluminous and hard evidence is supported by some limited findings on the induction of fear reactions in adult humans (but it should be noted that the stimuli involved were of a traumatic nature). The work on the induction of fear in children is not consistent, based on a very small number of instances, and all of the experiments can be criticised for errors of confounding, experimenter contamination and the rest. Psychiatric reports and other clinical observations have the great value of surmounting the artificiality of laboratory studies. Unfortunately the quality of the evidence is unsatisfactory, comprising as it almost always does a selected set of observations rarely supported by external confirmatory evidence. It also suffers from the serious weakness that the subject or the patient's account of the genesis of his fear relies on an accurate memory and powers of recall. Most people will agree that this is an unsatisfactory basis for theory-building. Because it has greater immediacy and the possibility of at least some external confirmation, the evidence on combat fears and neuroses, has something to recommend it. Regrettably, almost none of this valuable information was collected in a systematic manner; it therefore suffers from incompleteness, selection bias, and the interpretive gloss placed on
the accounts by the reporting psychiatrist or psychologist. Nevertheless it is material rich in interest, and authentic in quality. Fortunately military combat is an exceptional experience and from the point of view of psychological theorizing, it would be unwise to over-emphasize the significance of fears acquired in these unnatural circumstances. The intriguing research on taste aversions may well have opened the door for the development of an adequate theory of non-rational fears and, as already mentioned, Seligman (1972) has made an original and valuable contribution to this process.

THE NEED FOR REVISION

In giving this outline of the types and sources of evidence pertaining to the conditioning theory of fear acquisition, an attempt has been made to convey the impression that there is a good deal to be said in its favour. Why then is it necessary to revise it? Although the theory has merits, and some experimental and clinical support, its applicability is limited (Rachman, 1968). Whatever its value, the theory is not a satisfactory comprehensive account of the genesis and maintenance of fears.

There are six arguments against acceptance of the conditioning theory of fear acquisition. They are: (1) The failure of people to acquire fears in what are theoretically, undoubtedly fear-evoking situations (e.g. air raids). (2) It is difficult to produce conditioned fear reactions in human subjects, even under controlled laboratory conditions. (3) The conditioning theory rests on the equipotentiality premise (Seligman and Hager, 1972), which is now recognized to be untenable. (4) The distribution of fears in normal and neurotic populations is difficult to reconcile with the conditioning theory. (5) A significant number of phobic patients, psychiatric and military, recount histories that cannot be accommodated by the conditioning theory. (6) Fears can be reduced by vicarious processes and it seems highly likely that they can be acquired by similar processes.

1. Failures to acquire fear

On the face of it there can be few experiences more frightening than undergoing an air-raid. However, the well-documented information on the subject (see for example Janis, 1951) shows that during the Second World War the great majority of people endured air-raids extraordinarily well—contrary to the universal expectation of mass panic. Exposure to repeated bombing did not produce increases in psychiatric disorders. Although short-lived fear reactions were common, surprisingly few prolonged phobic reactions emerged. In the course of his official report to the Medical Research Council, Sir Aubrey Lewis (1942) said that “the doctors in Liverpool trained 18 volunteers as auxiliary mental-health workers for service in and after raids, but none of the 18 has been required: there was no such work for them to do,” (p. 178). His summary of his findings (“air-raids have not been responsible for any striking increase in neurotic illness”, p. 182) is in keeping with many similar reports from other workers. Wilson (1942, p. 284) noted that “the small number of psychiatric casualties that have followed aerial bombardment has been a matter for surprise”. Although he did obtain some evidence of fear induction and of an exacerbation of neurotic reactions, on the whole Lewis’s survey was remarklable in showing how uncommon these reactions were. In Coventry, Manchester, Liverpool and London, psychiatrists and other service-workers agreed that there had not been any significant increase in the number of patients attending psychiatric clinics. There was however evidence of more fear and related disturbances among the children. So for example, 4 per cent of 8000 school-children in Bristol (subjected to severe air-raids) were said to have developed anxiety symptoms attributable to raids. However, this leaves 96 per cent mildly affected or unaffected. The fears were particularly common and noticeable among children who had been subjected to traumatic experiences. It was also observed in Bristol and in Manchester, that “frightened mothers communicated their fears to the children” (Lewis, 1942, p. 181).

This British information is matched by the reports from Japan and Germany collated by Janis (1951). Immediately after an air-raid, many people experienced acute emotional reactions characterised by startle responses, tremor, fatigue and sleep disturbance. How-
ever, these acute reactions generally dissipated spontaneously, usually within the course of a day or two. People adapted to air-raids and became more courageous with increasing experience, even when as in London, the raids became progressively heavier.

The observations of comparative fearlessness enduring despite repeated exposures to intense trauma, uncontrollability and uncertainty, run contrary to the conditioning theory of fear acquisition. According to this theory, people subjected to repeated air-raids should acquire multiple conditioned fear reactions and these should be strengthened with repeated exposures.

2. Conditioning human fears

Bregman's (1934) thorough attempt to condition fear in 15 normal infants, was a failure. Evidence from a different source and of a different nature is consistent with Bregman's failure and with war-time observations, in showing that people fail to acquire fears in situations where the theory predicts that they should occur. Hallam and Rachman (1976, p. 183) point out that many writers on the subject of electrical aversion therapy, "appeared to assume that the successful administration of treatment would result in the development of a miniphobia—repeated associations of the conditioned stimulus with an unpleasant electrical shock would result in a situation in which the presentation of the stimulus produces fear reactions". To the contrary, Marks and Gelder (1967) found that most of their patients reported indifference to the conditioned stimuli employed in electrical aversion therapy; it was rare to find someone who complained of fear after undergoing the course of treatment. The same observation was reported by Bancroft (1971) and by Hallam et al. (1972).

Because the expected conditioned fear reactions did not emerge, and for some related reasons, Hallam and Rachman (1976) carried out two studies which were intended to provide an analogue for electrical aversion therapy. We were interested in finding out, among other things, whether repeated conditioning trials, carried out over numerous training sessions, and involving an electrical UCS, would result in the development of conditioned fear reactions. This research was carried out on 14 male research workers and postgraduate students, all of whom volunteered for at least one session of conditioning. Five of them also agreed to volunteer for a further nine training sessions. This latter group of subjects received 205 shocked trials over ten sessions. The other volunteer subjects received 20 trials during a single recording session. Cardiac, skin resistance and respiratory measures were recorded in the search for psychophysiological concomitants of what was expected to be a programme of fear conditioning. In the event, the results failed to confirm the hypothesis that conditioned fear reactions would develop. "The 'CR' did not resemble, in either magnitude or direction, the cardiac responses of phobic patients who are presented with their phobic stimulus, nor did subjects report anxiety or discomfort in the presence of the conditioned stimulus" (Hallam and Rachman, 1976, p. 192).

We were similarly unsuccessful in our search for evidence of conditioned fear reactions developing in alcoholic patients who underwent aversion therapy. "In effect, the results show that when alcoholics who have undergone aversion therapy are compared with alcoholics treated in other ways, there is no difference in their subjective anxiety responses to alcoholic stimuli or in the peripheral autonomic responses that usually accompany states of fear or anxiety. Subjective distaste for alcohol seems to be the only specific consequence of aversion therapy" (Hallam and Rachman, 1976, p. 194).

3. The equipotentiality premise

The conditioning theory of fear acquisition (see Eysenck and Rachman, 1965) assumes that any stimulus can be transformed into a fear signal. In addition the theory assumes that, given comparable exposures, all stimuli have roughly an equal chance of being transformed into fear signals. In some versions of the conditioning theory of fear, reservations were noted. For example, Eysenck and Rachman argued that "neutral stimuli which are of relevance in the fear-producing situation and/or make an impact on the
person in the situation, are more likely to develop phobic qualities than weak or irrelevant stimuli" (p. 81). Even when reservations of this sort were noted however, little attempt was made to specify their nature or implications.

This assumption of equal conditionability, or more properly in the term used by Seligman and Hager (1972), equipotentiality, was incisively criticised by Seligman (1970). His argument was developed by Seligman and Hager (1972) who observed that "general-process learning theorists believe that what an organism learns about is a matter of relative indifference. In classical conditioning the choice of the conditioned stimulus, unconditioned stimulus and response matters little; that is, all conditioned stimuli and unconditioned stimuli can be associated more or less equally well, and general laws exist which describe the acquisition, extinction, inhibition, delay of reinforcement, and spontaneous recovery of all conditioned and unconditioned stimuli" (p. 2). They proposed that the equipotentiality premise be replaced by the concept of preparedness and Seligman (1971) developed this alternative idea with particular reference to phobias. In brief, he argued that prepared phobias are of biological significance, are acquired rapidly, generalize broadly and are resistant to extinction.

The preparedness hypothesis has received partial support from the work of Ohman et al. (1975) who found that their normal human subjects acquired conditioned responses to pictures of (phobic) snakes and that these were more resistant to extinction than their conditioned reactions to supposedly neutral stimuli (human faces or houses). Further explorations of this interesting departure are awaited with interest, and it should be noted that they did not find differences in fear acquisition. Such differences are more likely to emerge with more trials and a better dependent variable than the GSR.

With hindsight we can now see that difficulties with the equipotentiality premise, particularly in its application to the acquisition of fear, began accumulating many years ago. For example, English (1929) was only partly successful in his attempt to replicate Watson's demonstration of the acquisition of a fear of white rats in his experimental subject, little Albert. English found that fear reactions could be conditioned only to selected stimuli. Along similar lines, Bregman (1934) failed in her thorough attempts to condition a group of 15 infants (median age 12 months) to fear a range of simple and biologically insignificant objects. The repeated presentation of geometrically shaped wooden objects and of cloth curtains, in association with a disagreeably loud and startling sound of an electric bell, did not produce conditioned fear reactions to the (biologically insignificant) stimuli. Other exceptions were quoted by Valentine (1946), who succeeded in producing in a 2-year-old child an unstable fear of a caterpillar, but failed to make her fear a pair of opera glasses. For the present, it is sufficient to notice that the equipotentiality premise cannot be sustained by the conditioning theory of fear acquisition.

4. The distribution of fears

Given the equipotentiality premise, the corollary for fear acquisition would be that all stimuli have an equal chance of being transformed into fear signals. However, this is not borne out by surveys of the distribution of fears, either in a general population or in psychiatric samples (see Rachman, 1974). For example, in an epidemiological study of common fears carried out in a small Vermont city, Agras et al. (1969) found that the prevalence of a fear of snakes was 390/1000 while fear of the dentist was only 198/1000—despite the fact that contact with the dentist was almost certainly far more frequent, and indeed, far more likely to be associated with painful episodes. To take another example from their survey, the prevalence of snake fears in the 30-year-old subjects was over 5 times as great as their fear of injections.

Anthropological observations of an isolated Indian community in Canada, are consistent with the view that the distribution of fears is non-random. Hallowell (1938) pointed out that the Indians were fearless of certain dangerous animals such as wolves and bears, but were considerably frightened by some harmless creatures notably frogs and toads.
Subject only to their prominence in the environment, many objects and situations should have an equal probability of becoming fear-provoking. What we find instead however, is that some fears are exceedingly common—far too common for the conditioning theory. Other fears are far too rare. Fear of the dark is commonly seen among children, but not pyjama phobias. In the case of animal phobias, one might expect that within a city population the prevalence of the fear of lambs should approximate that of the fear of snakes. In practice however, the fear of snakes is common and the fear of lambs is rare. Moreover, a genuine fear of snakes often is reported by people who have had no contact with the reptiles. Consequently one is forced to conclude that a fear of snakes can be acquired even in the absence of direct contact—and this significant concession opens three possibilities. Either the fear of snakes is innate or it can be transmitted indirectly, or the fear of snakes is 'lurking' and will appear with only slight provocation. The last of two of these three possibilities are of course compatible.

5. Patients' reports of fear onset

Whatever its value, the conditioning theory is not a satisfactorily comprehensive account of the genesis of fears. It had become apparent that "clinically it is often difficult to determine the origin of a patient's phobia" (Rachman, 1968, p. 32). In similar vein, Marks (1969, p. 92) described "many phobias where there was no apparent trauma to initiate the phobia". It can be extremely difficult to find a convincing precipitant of a phobia (de Silva et al., 1976). Goorney and O'Connor (1971) encountered this problem in their analysis of the excessive fears of peace-time air-crews. In a study of 97 cases of excessive anxiety encountered in R.A.F. crews, they were able to relate the fears of one quarter of all their cases to specific precipitants such as accidents or frightening incidents. In one third of the cases however, there was no discernible cause and in the remainder, the precipitants were not of a traumatic or conditioning type, e.g. a return to flying after a long absence.

6. The vicarious transmission of fear

The significant advances made in our understanding of the processes of observational learning and modelling (Bandura, 1969, 1971) made it plain that we acquire much of our behaviour, including emotional responses, by vicarious learning experiences. It was proposed (Rachman, 1968) that fears can be acquired either directly or vicariously and that stimuli are likely to develop fearful qualities if they are associated, directly or vicariously, with painful or frightening experiences. It has to be conceded however that at this stage, the evidence in support of vicarious acquisition of fear in humans is indirect and largely anecdotal—this is entirely understandable because of the ethical objections to experiments in which one sets out to induce lasting fears in subjects. As mentioned earlier, it was observed during the war that the fears or lack of fears displayed by mothers during the course of air-raids, was an important determinant of whether or not their children developed similar fears. Along the same lines, John (1941) commented on the social facilitation and inhibition of childrens' fears during air-raids. She obtained a correlation of 0.59 between the fears of mother and child. In normal conditions there is a good deal of correspondence between the fears of children within the same family, with correlations ranging between 0.65 and 0.74 (May, 1950). Similarly, Hagman (1932) found a correlation of 0.67 between the total number of fears exhibited by children and their mothers. Grinker and Spiegel (1945) provided clear examples of combat airmen who acquired fears after observing a crew mate expressing intense fear. In their survey of 1700 infantry troops in the Italian theatre during the Second World War, Stouffer et al. (1949) found that 70 per cent of the respondents had a negative reaction to seeing a comrade 'crack up'. Half of the total sample said that it made them feel anxious and/or like cracking up themselves.

It can of course be argued that in most of the examples cited here, a conditioning explanation can suffice. This defence is persuasive insofar as the examples involve the
occurrence, in temporal and spatial contiguity, of neutral and frightening stimuli. However, the social transmission of fear opens wide the possibility of the indirect acquisition of fears. That possibility, indeed likelihood, poses serious difficulties for a conditioning theory. If it is confirmed experimentally that fear reactions can be acquired to stimuli which the person has never encountered, then the conditioning theory cannot be adequate. Incidentally, there are good grounds for believing that fears can be acquired by indirect transmission—not necessarily by vicarious social learning only. This conclusion, that indirect transmission does occur, seems inevitable from a study of the distribution of fears—in that, people report fears of objects or situations which they have never actually encountered.

In sum, these 6 arguments lead to the conclusion that the conditioning theory of fear acquisition is neither comprehensive nor adequate.

INTEGRATION

Although they are serious, the weaknesses of the conditioning theory are not necessarily fatal. One can either search for an entirely new theory to replace it, or one can take the reformist view and search for modifications and extensions of theory. The conditioning theory draws most of its support from a substantial weight of laboratory findings on fear-induction in animals and is buttressed by lighter evidence extracted from tests on children, experiments on a small number of people, and from the experiences of clinical workers in dealing with phobic patients. At its best the conditioning theory can provide a partial explanation for the genesis of some fears. As we have seen, it cannot deal with the observed distribution of fears, the uncertain point of onset of phobias, the indirect transmission of fears, the ready acquisition of prepared phobias and the failure of fears to arise in situations demanded by the theory.

The impressive research findings on taste aversions indicate how intense and lasting reactions can be acquired very rapidly. The conditioning theory will receive an unexpected boost from these findings—if we are willing to assume that they are analogous to acquired fears. In my view, the evidence on acquired taste aversions is relevant to the conditioning theory of fear acquisition. The phenomena share two important features, i.e. the development of strong avoidance behaviour and of associated, intense psychophysiological reactions. However, taste aversions differ from fear in that the subjective reports are best described as repugnance rather than apprehension. Moreover, a reliance on the taste aversion findings will require a de-emphasis of the temporal qualities of classical conditioning.

In addition to providing an account of the conditions in which fear is acquired, an adequate theory of the genesis of fears needs to accommodate the following information as well. Fears can emerge suddenly (in the minority of instances) or gradually. There appear to be substantial individual differences in susceptibility to the acquisition of fears. Fears can be acquired indirectly—and that includes the acquisition of fears of objects or situations which the person has never encountered. All things considered, people acquire comparatively few fears. The distribution of fears in the population is non-random.

PATHWAYS TO FEAR

In my opinion, we have to acknowledge that there are at least three major pathways to the acquisition of fear. We can continue to regard conditioning (and include under this label the taste aversion phenomenon) as an important fear-induction process, but at least two other pathways can be identified. They are distinguished by being indirect processes of acquisition. The first, vicarious acquisition, has already been described and it was pointed out that the evidence to support this notion is still flimsy. The conviction that fears can be acquired vicariously is drawn more from the successful demonstrations of vicarious fear-reduction (Bandura, 1969; Rachman, 1972, 1976) than from the largely indirect and anecdotal evidence referred to earlier. A third pathway of significance, fear acquisition by transmission of information and/or instruction, has
been strangely overlooked—despite the fact that it is obvious, or perhaps because it is too obvious. Although I am unaware of any conventionally acceptable evidence that fear can be acquired through the transmission of information (and particularly, by instruction), it seems to be undeniable. Information-giving is an inherent part of child-rearing and is carried on by parents and peers in an almost unceasing fashion, particularly in the child’s earliest years. It is probable that informational and instructional processes provide the basis for most of our commonly encountered fears of everyday life. Fears acquired informationally are more likely to be mild than severe. Like the acquisition of fear by vicarious experience, informational and instructional processes have no difficulty in coping with the fact that people display fears of situations and objects which they have never encountered. Acceptance of the notion that fears can be acquired by informational processes, also enables us to explain some but by no means all of the failures to acquire fear in situations where it might, on the conditioning theory, have been expected to arise. Not only do we learn by information and instruction which situations to fear, we also learn to distinguish those situations and objects which are not dangerous and therefore not to be feared. We also learn and are taught to cope with dangers and to endure the accompanying discomfort or pain.

The best explanation offered so far for the non-random quality of fears is the concept of preparedness proposed by Seligman (1971). He hypothesized that “phobias are highly prepared to be learned by humans and, like other highly prepared relationships they are selective and resistant to extinction, and probably are non-cognitive” (Seligman, 1972, p. 43). He also suggested that most phobias are of biological significance and that the “great majority of phobias are about objects of natural importance to the survival of the species” (p. 465). Human phobias are ‘largely restricted’ to situations that threaten survival, potential predators, unfamiliar places, the dark, deformities etc. If this theory can be supported by experimental and other evidence, we will then be in the best-ever position to account for the non-random incidence of fears, and perhaps also to deal with the skewed distribution of fears both in the general population and in clinical samples. It may also provide the best basis for an explanation of why people fail to acquire fears despite exposure to considerable stress. It may not be stretching the point too far to say that civilian populations exposed to air-raids during the war failed to acquire many new fears because the stimuli associated with the trauma and stress of bombing (e.g. aircraft, bombs, etc.) are not ‘prepared’ stimuli. These modern artifacts do not feature in our biological inheritance.

The considerable individual differences in the range and intensity of fears need to be encompassed by any theory for which comprehensiveness is claimed. Just as the conditioning theory of fear acquisition rests on the assumption that most fears are acquired, so the 3 pathways hypothesis outlined here, is based on a similar assumption. This is not to deny that there are biological differences in the propensity to develop fears. From everything we know about the biological and genetic bases of personality (Eysenck, 1967) it seems certain that there is a genetic contribution to the general level of human fearfulness. Of greater interest is the question of whether or not the propensity to fearfulness is related to other important aspects of personality, e.g. neuroticism. If there is any value in the present tripartite scheme of fear acquisition, it may turn out that some people are particularly prone to develop fears by a process of conditioning (including the taste aversion phenomenon), while others are more susceptible to fears that are socially transmitted by vicarious learning or by information processes. Whether this speculation is borne out or not, it seems likely that we will uncover a connection between prepared fearful stimuli and acquisition of fears by a conditioning process. Those fears which are transmitted by informational and instructional processes are likely to comprise a large number of non-prepared stimuli.

It can be seen that we are moving towards a position in which it is postulated that there is an ease of connection between certain people and certain stimuli—and contrariwise, that some people are particularly invulnerable to certain fear stimuli. In
addition to postulating an appropriate fit between the person and the stimulus, we also have to take into account the occurrence of 'critical moments'. In attempting to explain those fears which have an acute onset, one needs to know why the fear emerges at the particular time that it does. It is plain from clinical experience that certain patients experience critical incidents in which the fear has its onset. What is particularly interesting is the fact that quite frequently these same people have been exposed to the same stimulus repeatedly in the past without acquiring the fear. It seems that for acute onset fears, there are certain psychological states in which the person is vulnerable to the acquisition of fear. To take a clinical example, in those agoraphobic patients who report an acute onset of fear, one needs to know why the fear arose on the day that it did, at the time that it did. And why do they acquire a fear of public transport, crowded or open spaces, or whatever the content of their phobia is, when on hundreds or thousands of previous exposures to the same set of stimuli, they remained unaffected?

So in framing an explanation for fears of acute onset, we have to try to match the person with the stimulus and the critical moment of onset. (In passing, it might be mentioned that at least as far as acute onset of agoraphobias are concerned, it seems likely that the critical incident occurs when the person is in an emotionally upset or apprehensive state before the critical incident occurs. Another predisposing factor seems to be physical illness and associated feelings of weakness, nausea, dizziness etc.)

To sum matters up, it is suggested that the classical conditioning theory of fear acquisition can account for only part of the available information. It is suggested in addition, that fears can be acquired by indirect processes. The two most important of these are the vicarious acquisition of fear and the informational and instructional transmission of fear. It is speculated that intense fears of biological significance (in Seligman's theory, prepared phobias) are more likely to be acquired by a conditioning process. The common everyday fears are probably acquired by the indirect and socially transmitted processes of the information-giving type and by vicarious exposure.

Any of these 3 pathways to fear, alone or in combination, can be implicated in the acquisition of fear. By appealing to one or more of these pathways, it should be possible to explain most of the common features of human fear including the observed distribution of fears in normal and clinical populations, the non-random incidence of fears, sudden or gradual acquisition of fears, the indirect transmission of fears, and failures of fears to arise under stress conditions. (The interesting questions of what happens when one or more of the pathways operate in opposing directions (e.g. information vs. preparedness), will be addressed in later analyses.)

Thus far we have considered fear solely in terms of the older conception sometimes known as the 'lump theory of fear' (see Rachman, 1974). If we adopt the more complex conception proposed by Lang (1970), according to which fear comprises at least three imperfectly related components, then the three pathways approach will in turn become more complex. Lang has argued that the three major components of fear are subjective report, avoidance behaviour, and psychophysiological disturbance. These three components can co-vary, vary inversely or vary independently (Rachman and Hodgson, 1975; Hodgson and Rachman, 1975). If this analysis is applied to the three pathways approach to fear acquisition we can hazard the speculation that fears acquired by a conditioning process (which for present purposes, includes the taste aversion phenomenon), the components which will be most markedly involved are the psychophysiological and behavioural, with the subjective component playing a comparatively minor role. In the case of fears transmitted indirectly (i.e. vicariously or informationally) one might expect the subjective aspect to be predominant and the psychophysiological changes and behavioural effects to be comparatively minor. This line of reasoning appears to be compatible with Seligman's concept of prepared phobias—which it will be remembered, he described as being non-cognitive and resistant to extinction. By contrast, one might expect non-prepared fears to have a larger cognitive element, i.e. they can be more readily acquired, and indeed reduced, by cognitive manipulations. It has to be admitted however, that so far, the attempts to relate prepared phobias to therapeutic
considerations have yielded unexpected (Rachman and Seligman, 1976) or unrewarding results (de Silva et al., 1976).

In conclusion, are we any closer to understanding the opening question of why people so often fail to acquire fears in dangerous situations? The Seligman hypothesis is of some utility in that biologically non-significant stimuli are said to be 'poorly prepared' for fear. Also, people are considerably more resilient than the conditioning theory allows. Not only is the equipotentiality (of stimuli) premise untenable, the comparable premise that all people are equally (and easily) vulnerable to fear, is similarly, hard to sustain. Despite the not inconsiderable casualty rate caused by air-raids on Britain, people developed relatively few fears—by contrast, the air-combat crews had a high incidence of (comparatively transient) fear. The casualty rate for airmen was far greater (it ran as high as 723 per 1000 in the early stages of the Second World War (Flanagan, 1948, p. 207)) and unlike the civilians they were not able to escape to the partial safety of a bomb-shelter at a given signal. When repeatedly subjected to extraordinarily dangerous, virtually inescapable trauma, most people acquire fears. But even under these most extreme circumstances, the large majority contained, and later recovered from their fears.

REFERENCES


The conditioning theory of fear-acquisition


