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The conditioning model of neurosis

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Abstract: The long-term persistence of neurotic symptoms, such as anxiety, poses difficult problems for any psychological theory. An attempt is made to revive the Watson-Mowrer conditioning theory and to avoid the many criticisms directed against it in the past. It is suggested that recent research has produced changes in learning theory that can be used to render this possible. In the first place, the doctrine of equipotentiality has been shown to be wrong, and some such concept as Seligman's "preparedness" is required, that is the notion that certain CS are biologically prepared to be more readily connected with anxiety responses than others. In the second place, the law of extinction has to be amended, and the law of incubation or enhancement added, according to which the exposure of the CS-only may, under certain specified conditions, have the effect of increasing the strength of the CR, rather than reducing it. The major conditions favouring incubation are (1) Pavlovian B conditioning, that is a type of conditioning in which the CR is a drive; (2) a strong UCS, and (3) short exposure of the CS-only. Personality differences are also important in this connection. It is possible, using these two recent developments of conditioning theory, to show that a conditioning paradigm can be used to explain the major facts relating to the development of neurotic disorders, and that similarly the success of methods of treatment, such as behaviour therapy, can be explained. The evidence is reviewed for the extistence of the incubation phenomenon, both in the animal laboratory and in the clinic, and also for the relevance of the parameters suggested as being implicated in its occurrence. It is concluded that these modifications are sufficient to make the conditioning theory of neurosis and treatment the only viable scientific theory in the field, particularly when attention is paid to Pavlov's and Platonov's inclusion of the second signalling system.

Keywords: anxiety; behavior therapy; conditioning; incubation; learning; neurosis

1. The neurotic paradox

"Neurosis" is a word widely used in psychiatry and clinical psychology, but it is seldom defined in other than purely descriptive terms. In spite of the importance of the phenomena involved, both socially and scientifically, there is an absence of proper causal theories. Textbooks and monographs avoid any discussion of laboratory-based, experimentally testable theories that might account for the observationally established facts of neurotic behaviour; such theories either do not exist, or else they are too remote from reality to attract attention. The Freudian theory of human behaviour is at once too far reaching (it deals with all human behaviour, rather than with neurotic behaviour as such) and too ill defined (there is no way in which testable deductions can be made from it) to be of help in this connection; indeed, it is doubtful whether there is such a thing as a Freudian theory of neurosis in the sense that the term "theory" is used in science (Eysenck and Wilson 1973). There is one exception to these universal strictures, namely the conditioning theory of neurosis originally suggested by Watson and Rayner (1920); the purpose of this paper is to examine the viability of this theory, discuss the many justifiable criticisms to which it has been subjected, and finally suggest ways and means of bringing it up-to-date and rewriting it so that it can deal with the major known experimental and clinical facts that need to be accounted for.

We may provisionally define neurotic behaviour as maladaptive behaviour, accompanied by strong, irrelevant and persistent emotions, occurring in full awareness of the maladaptive and irrational nature of the behaviour in question. Typical instances of neurotic behaviour so defined are anxiety reactions, phobias, obsessive-compulsive behaviour, reactive depressions, and psychosomatic symptoms. Behaviour that is characteristic of the functional psychoses is quite different in character, and in causation, obeys different rules, and responds to different treatments (Eysenck 1973). The typical neurotic reactions here mentioned form a general syndrome which this writer has called "dysthymic"; it is differentiated from another group of symptoms, including hysteria, psychopathy, and antisocial behaviour generally (Eysenck and Rachman 1965). It is a moot point whether this second group should also be called "neurotic"; we have referred to dysthymic and antisocial reactions as "disorders of the first and second kind," respectively. In this paper we shall be concerned exclusively with disorders of the first kind; a theory of disorders of the second kind has been developed elsewhere (Eysenck 1977a, 1979; Eysenck and Eysenck 1978).

Neurotic behaviour of the kind defined and described presents a problem both for common sense as well as for psychological theory. This problem has been referred to by Mowrer (1950) as the "neurotic paradox." Given that most philosophical and psychological theories are essentially hedonistic, stressing what Thorndike has called the "law of effect" and Skinner the "law of reinforcement," how can we account for the existence of a class of behaviours which are at the same time "self-perpetuating and self-defeating?" (p. 486). As Mowrer points out,

> "common sense holds that a normal, sensible man, or even a beast to the limits of his intelligence, will weigh and balance the consequences of his acts: if the net effect is favorable, the action producing it will be perpetuated; and if the net effect is unfavorable, the action producing it will be inhibited, abandoned. In neurosis, however, one sees actions which have predominantly unfavourable consequences, yet they persist over a period of months, years, or a lifetime" (ibid).

Thus a proper theory of neurosis is of importance not only in psychiatry, and as an aid to treatment; it is a fundamental importance to psychology itself. Neurotic behaviour is not something isolated and relatively rare and unusual; one person in three is liable to neurotic breakdown at some time or other, a proportion that has not changed dramatically over the past three centuries (Eysenck 1977b), and minor "neurotic" reactions occcur in most if not all human beings. Thus an explanation of neurotic behaviour is central to a proper understanding of much of human activity. Conversely,

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so-called "laws" of behaviour that cannot account for such widespread and well-documented behaviour can hardly aspire to be accorded scientific respectability.

Clinical phenomena of neurosis play much the same part in relation to laboratory studies of emotions as do the celestial phenomena (black holes, quasars, and the like) in astronomy and physics, as compared with the phenomena produced and studied in the laboratory. The "real-life" phenomena are on a much larger scale and cannot in essence be reproduced in the laboratory; hence they pose a problem for the theoretical scientist who is required to derive his laws from the laboratory as well as encompass these extralaboratory phenomena. Success in so doing confirms in an unmistakable fashion the accuracy of his small-scale observations and hypotheses and allows him to extrapolate beyond the narrow confines of his laboratory. For the scientific psychologist, therefore, the phenomena associated with neurotic behaviour are of crucial importance in the attempt to gain a proper appreciation of the achievements and limitations of his scientific knowledge. It is from this point of view, rather than the more narrow applied one, that we shall be viewing the problem in this paper.

2. The Watson and the Miller-Mowrer models

The model proposed by Watson (Watson and Rayner 1920) was fundamental to future developments, but it does not constitute a properly developed theoretical system; we have to reconstitute it from a few words in his study of little Albert, the 11-month-old boy in whom he conditioned a fear of rats, and from the early behaviour therapy experiments of his student, Mary Cover Jones (1924a, 1924b). From these sources, it would appear that Watson thought of neurotic disorders essentially as conditioned emotional responses, the process of conditioning being entirely Pavlovian in nature. Watson never worked out his theory (if that is the right term for what is in effect merely a pointer in the direction that the prolegomena to a proper theory might take), and none of his successors has taken the task seriously, even though many have light-heartedly spoken of a "conditioning theory" of neurosis. Such a "theory" has never been accepted widely by experimental psychologists or by psychiatrists, for the simple reason that any attempt to construct a proper model along these lines immediately runs into insuperable difficulties. Before discussing these, it may be helpful to discuss the only serious attempt to develop Watson's hints into a more complete theory.

This attempt was made by Mowrer (1939, 1940) and Miller (1948, 1951a). In actual fact, in doing so they introduced an important sea-change into the theory; instead of a theory of neurosis, it became a theory of anxiety. "Conditioned fear" is the term by which they referred to anxiety, and together they worked out a conditioning paradigm to account for the appearance of anxiety, which had been made central to the neurotic disorder syndrome by Janet, Déjerine, and other French psychiatrists whose work became known to English-speaking psychologists through the distorting mirror of Freud and his colleagues (Ellenberger 1970). Conditioned fear referred to the emotional component of the unlearned reaction to a painful stimulus (e.g. an electric shock) which could be conditioned according to the Pavlovian paradigm to a previously neutral stimulus. Both authors tried to fit this Pavlovian fear-conditioning process into Hullian-type drive-reduction theory, and both finally gave up the attempt and shifted to some form of temporal contiguity theory (Kimmel 1975). The most important contribution of the Mowrer-Miller model was the demonstration that anxiety could act as a drive, and hence as a motivational state.

The general trend of the theory is given by the following quotation from Mowrer: it will be seen that it is much more detailed and experimentally verifiable than Watson's original suggestion. Furthermore, the connection between the Miller-Mowrer theory and the then popular Freudian "dynamic" views (Dollard and Miller, 1950) which these authors attempted to translate into learning-theory (particularly Hullian) language, gave this attempt at theory construction a powerful influence. Here is what Mowrer said:

"A so-called 'traumatic' ('painful') stimulus (arising either from external injury, of whatever kind, or from severe organic need) impinges upon the organism and produces a more or less violent defense (striving) reaction. Furthermore, such a stimulus-response sequence is usually preceded or accompanied by originally 'indifferent' stimuli which, however, after one or more temporally contiguous associations with the traumatic stimulus, begin to be perceived as 'danger signals,' i.e., acquire the capacity to elicit an 'anxiety' reaction. This latter reaction, which may or may not be grossly observable, has two outstanding characteristics: (i) it creates or, perhaps more accurately consists of a state of heightened tension (or 'attention') and a more or less specific readiness for (expectation of) the impending traumatic stimulus; and (ii) by virtue of the fact that such a state of tension is itself a form of discomfort, it adaptively motivates the organism to escape from the danger situation, thereby lessening the intensity of the tension (anxiety) and also probably decreasing the chances of encountering the traumatic stimulus. In short, *anxiety (fear) is the conditioned form of the pain reaction*, which has the highly useful function of motivating and reinforcing behaviour that tends to avoid or prevent the recurrence of the pain-producing (unconditioned) stimulus" (Mowrer 1939, pp. 554-555, italics in original).

The assumption made by Miller and Mowrer is that an explanation of the origins of anxiety explains the occurrence of neurosis; this is unfortunately not true. As Kimmel (1975) has pointed out, neurosis is characterized by long-continued persistence of maladaptive emotional reactions, while anxiety, viewed as a conditioned fear reaction, should quickly extinguish according to prevailing psychological theory. This, as we shall see, is one of the most serious objections to both the Watson and the Miller-Mowrer models of neurosis; many others will be noted. As Kimmel makes clear, the future of extinction to occur after anxiety has been acquired according to Mowrer's paradigm is precisely the point that calls for explanation; the theory fails to do so. Mowrer's (1947) two-factor theory of learning attempts to overcome this difficulty (Eysenck and Rachman 1965), and for some rather atypical neurotic reactions the postulation that the original Pavlovian conditioning of anxiety may be followed by a second stage of instrumental conditioning during which the neurotic learns to avoid the CS (conditioned stimulus), thus making the avoidance behaviour permanent, is acceptable. However, for the great majority this is plainly not so; case histories demonstrate time and again the frequent encounters of patients with unreinforced CS presentations. Hence the apparent reliance of the Watson and the Miller-Mowrer models on learning theory is indeed only apparent; predictions from traditional extinction theory would seem to make impossible the development of neurotic disorders of a long-continued kind along the lines of Pavlovian conditioning of anxiety. Perhaps it was the realization that this might be so that caused Miller and Mowrer to concentrate on anxiety, rather than on neurosis.

3. Criticisms of the conditioning model: I

In now taking up in detail the criticisms of the traditional conditioning model, and suggesting how these could be overcome by changing important properties of the model, we shall divide the criticisms into two main classes, and take these in turn, devoting a special section to each class. The first class of criticisms arises from the disregard by Watson (and most of his behaviourist followers until today) of the biological inheritance of the human race, its evolutionary development, and its instinctive reactions to different stimuli. By winning out over MacDougall on these issues in the famous "instinct debates" of the twenties, Watson set back the proper development of psychology by many years; in essence (though not in detail) MacDougall was right, and Watson wrong. The difficulties with Watson's theory that have arisen since its original presentation, and that derive from this particular error, are numerous, and will now be outlined briefly; we will then try to show how they can be overcome by the recognition of biological reality. The notion of the "empty organism," as Boring called it, has led psychology into a cul-de-sac; the difficulties here

noted are only some of those that inevitably arise when we fail to treat human beings as biosocial organisms, influenced both by deep-seated, genetically determined factors anchored securely in our nervous system, and by environmental influences mediated by the social setting in which we live. Watson's neglect of the former influences was in large part responsible for the failure of his theory.

1. One of the obvious objections to Watson's "theory," based as it is on one single case (little Albert), is that later investigators have been unable to replicate his results (English 1929; also Bregman 1934). This suggests that the phenomenon in question may be affected powerfully by individual differences, and Watson's model makes no room for these. He does indeed say that "one may possibly have to believe that such persistence of early conditioned responses will be found only in persons who are constitutionally inferior" (Watson and Rayner 1920, p. 14). However, this single sentence goes counter to his insistence in his major books on the absolute supremacy of environment and the absence of genetic causes in differentiating human behaviour. Furthermore, the notion of "constitutional inferiority" has no experimental backing or theoretical meaning, is untestable in its present form, and simply reinstates Victorian notions that psychiatry had already sloughed off. Watson is paying lip service to genetics, just as Freud and Skinner have done; none of these writers has taken seriously the task of specifying the precise nature of the genetic component, or of performing the necessary experiments to demonstrate the validity of the hypothesis.

While agreeing that individual differences are important in predisposing individuals to neurosis (vide infra), we will suggest that this is not the best explanation for this "failure to replicate."

2. Our second point relates to the postulation of *equipotentiality*, which is an important part of Pavlovian theory, as accepted by Watson. In Pavlovian conditioning, one CS is as good as another; "Any natural phenomenon chosen at will may be converted into a conditioned stimulus, any visual stimulus, any desired sound, any odor and the stimulation of any part of the skin" (Pavlov 1927, p. 86). This does not seem true of phobias, however;

"they comprise a relatively nonarbitrary and limited set of objects: agoraphobia, fear of specific animals, insect phobias, fear of heights, and fear of the dark, and so forth. All these are relatively common phobias. And only rarely, if ever, do we have pyjama phobias, grass phobias, electric-outlet phobias, hammer phobias, even though these things are likely to be associated with trauma in our world" (Seligman 1971, p. 312).

The set of potentially phobic stimuli thus seems to be nonarbitrary, and to be related to the survival of the human species through the long course of evolution, rather than to recent discoveries and inventions which are potentially far more rational sources of phobic fears, such as motor cars, aeroplanes, and guns (Geer 1965; Landy and Gaupp 1971; Lawlis 1971; Rubin, Katkin, Weiss, and Efran 1968; Wolpe and Lang 1964).

The nonarbitrary and limited choice of objects and situations which predominantly produce phobic fears in humans is difficult to explain along traditional lines of Pavlovian learning theory, or any of its behaviourist or neobehaviourist successors; Watson's theory (as well as Miller and Mowrer's) seems to break down in relation to this well-documented phenomenon.

3. Single-trial conditioning is sometimes (although far from universally) reported in connection with the genesis of phobic fears; yet single-trial conditioning is very rare in the laboratory (Kamin 1969; Seligman 1968), and it is by no means clear how events that usually do not appear very traumatic in the life of the patient can lead to such very clear-cut consequences. There appears to be something in the nature of the CS that makes it particularly easy to associate with a UCS (unconditioned stimulus), and that produces single-trial conditioning where another CS might not have done so. The problem is clearly associated with that mentioned in the previous paragraph; equipotentiality seems to be the exception rather than the rule. Fears of certain objects or situations seem to be so close to the surface that when these serve as CSs they acquire the fear-producing qualities of the UCS only too readily; this is difficult to explain on any simple environmentalistic hypothesis.

4. One very obvious characteristic of the typical laboratory CS-UCS connection is its dependence on very refined and precise experimental conditions, particularly the time relations involved. It is only when the CS precedes the UCS by between 500 msec. and 2500 msec. that eye-blink conditioning can be obtained, for instance; other types of human (and animal) conditioning are equally precisely circumscribed. But such precision is unattainable (except by chance, and occasionally) in real-life situations; attempts to use laboratory findings or Pavlovian conditioning (or operant conditioning, for that matter) as explanations of everyday life experiences and behaviour cannot overlook this very fundamental difficulty. It applies of course not only to Watson's theory, but also to other attempts to build bridges between laboratory conditioning and real-life events, such as this writer's theory of criminality and psychopathy (Eysenck 1977a, Eysenck & Eysenck 1978.)

An explanation of the four difficulties encountered by Watson's theory outlined above can be found in Seligman's hypothesis of "preparedness" (Seligman, 1970, 1971). According to this theory, which derives indirectly from MacDougall's theory of instincts, and more directly from modern work on ethology, "phobias are highly prepared to be learned by humans, and, like other highly prepared relationships, they are selective and resistant to extinction, learned even with degraded input, and probably non-cognitive" (Seligman 1971, p. 312). Seligman gives examples of the fact that some contingencies are learned much more readily than others, that is with highly degraded input such as single-trial learning, long delays of reinforcement, and so forth; the work of Garcia, McGovan, and Green (1971) has become a classical example of this.

This conception of preparedness helps to explain, among other things, why Bregman (1934) and English (1929) failed to get fear conditioning in their replication of Watson's experiment with little Albert; they used common household goods such as curtains and blocks or a wooden duck, none of which would have the "preparedness" value of furry animals. Another problem that may be explained by this concept is the choice of CS – why, in a traumatic situation (or in a series of subtraumatic situations) does the person concerned pick on one rather than another equally prominent stimulus to become the CS? On Seligman's evidence, the choice would be determined very much by innate preparedness, in addition to the usual chance factors.

The notion of preparedness integrates well with the hypothesis of innate fear (Seligman 1972; Hinde and Stevenson-Hinde 1973; Breland and Breland 1966); presumably *degree* of fear experienced separates the two concepts. When the fear upon first encountering the stimulus object is strong, it is considered innate; when it is weak, but easily conditioned, we think of preparedness. The underlying physiological connections and the hypothetical evolutionary development are identical. The concept is a valuable one and appears necessary for full understanding of phobic neuroses in particular. Presumably it, too, must be seen in the context of individual differences; it seems likely that extraversion and neuroticism are as relevant to preparedness as they are to the development of neurotic illnesses, or to incubation (vide infra).

The lack of equipotentiality is equally well explained in terms of "preparedness"; the most frequently experienced phobic fears are attached to especially dangerous objects or situations which made the acquisition of innate or "prepared" fears extremely useful to the individuals and the species concerned during the four million years of human evolution. Open spaces make it difficult to hide from enemies; closed spaces make it difficult to escape from enemies. Small animals used to be poisonous, as did snakes, with attendant dangers to humans. Heights had obvious dangers before protective fencing became customary. Altogether, the attractiveness of the hypothesis is obvious, although, like all evolutionary arguments, this one would be difficult to support experimentally. Fortunately, the recent work of Hugdahl, Fredrikson, and Öhman (1977) and Öhman, Eriksson, and Olofson (1975), Öhman, Erixon, and Löfborg (1975), and Öhman, Frederikson, Hugdahl and Rimmö (1976) has in a most ingenious fashion provided experimental support for the existence of "preparedness" with respect to the CS used for classical

conditioning in the laboratory; these investigators demonstrated very clearly that "prepared" stimuli (pictures of rats and spiders) acquired CS–CR (conditioned response) connections much more quickly than did nonprepared CS (pictures of flowers and mushrooms, or even rifles and revolvers, Öhman, Frederickson, and Hugdahl 1978). It was also found that conditioning mediated by such "prepared" stimuli was more resistant to extinction; this point is important for our subsequent discussion and should be kept in mind. There was also some interesting interaction with arousal; this too will be discussed again in a later section. (For a critique of the concept, see Rachman 1978.)

The last point that the postulation of "preparedness" explains is the fact that in typical laboratory conditions CS-UCS intervals are of such critical importance, while in real-life situations no such precise timing can be guaranteed. As Seligman points out, learning with a prepared CS can occur even with severely degraded input, that is to say, in circumstances that under typical laboratory conditions, that is with nonprepared CS, would lead to complete failure. Even with rats, Garcia, McGovan, and Green (1971) could delay the UCS by as much as one whole hour after the presentation of the "prepared" CS and nevertheless obtain significant evidence of conditioning. We thus see that all the difficulties with Watson's model listed above are resolved once we depart from his nonbiological, environmentalistic stance and accept the evidence for innate, instinctive, "prepared" fear-arousing stimuli. We must next turn to another set of difficulties which cannot be overcome by the same means.

4. Criticisms of the conditioning model: II

We have already mentioned the difficulties raised for Watson's theory by the postulate of *extinction*; this postulate has been retained almost unchanged since Pavlov's formulation in all modern textbooks.

5. Unreinforced conditioned reactions extinguish quickly (Kimble 1961), and neurotic reactions should be no exception to this rule. Eysenck and Rachman (1965) have suggested that the well-documented prevalence of spontaneous remission in neurosis (Rachman 1971) may be due to extinction of this type; however, in many cases extinction does not take place, and it is the task of a good theory to account for these nonfitting cases as well as for those that behave according to expectation. Mowrer (1947), as we have already noted, proposed his two-process theory of conditioning in part to account for this difficulty; according to this theory, the original conditioning is protected by a second stage of instrumental or operant conditioning, in which the relief from anxiety produced by avoidance of the CS leads to a conditioned avoidance reaction. This is analogous to the avoidance of "reality testing" that psychiatrists have postulated, and there is no doubt that for certain cases of neurosis Mowrer's theory fits the facts very well. However, Mowrer's theory does not seem to explain the majority of clinical cases, and in any case has been criticised on experimental grounds by Herrnstein (1969) and by Seligman and Johnston (1973). It is very doubtful if the Watson-Mowrer theory can really offer convincing arguments to explain the astonishing failure of extinction to occur after many years of exposure to the unreinforced CS. Unless this fact can be explained in a satisfactory manner, the theory becomes untenable. Indeed, as the essence of the "neurotic paradox" is precisely the failure of extinction to occur – anxiety fails to extinguish although no reinforcement is offered; behaviour that is punished continues to occur – we may say that the Watson theory fails to come to grips with the problem it was designed to solve.

Watson himself gave it as his opinion, based on his experiments with little Albert, that "conditioned emotional responses as well as those conditioned by transfer . . . persist and modify personality throughout life." (Watson and Rayner, 1920). (By "transferred responses" Watson means what would now be called "stimulus and response generalization," a term not then widely used.) Watson must have known of the experimental phenomenon of extinction; it is difficult to see why he did not mention the difficulties this would present to any such theory as that advocated by him. Of course, Pavlov's work had not been translated at that time, and he had to base his work on casual reports; this may account for his failure. Later writers do not have this excuse.

Several writers have of course noted the difficulty that the failure of extinction to occur in avoidance responding causes for a two-process fear-mediation theory, beginning with Ritchie (1951), Solomon and Wynne (1954), and Solomon and Brush (1956). Several systems would seem to generate predictions of almost total resistance to extinction (e.g. Miller 1951a, 1951b, 1963). Soltysik (1964, 1975) has suggested that the occurrence of an avoidance response may protect the warning stimulus from extinction. Solomon and Wynne (1954) have proposed the principle of the conservation of anxiety as a way out of this difficulty. Kimble and Perlmuter (1970) have suggested the principle of "automatisation," that is, a process by which well-practised responses come to be initiated without direct motivational antecedents. Schoenfeld (1950), Sidman (1953), and Dinsmoor (1954) have proposed a two-process aversion theory which aims to overcome some of the difficulties encountered by two-process fear-mediation theory. Herrnstein (1969) has formulated a discriminative stimulus theory that dispenses with both fear and aversion. Last, and perhaps most acceptable, Seligman and Johnston (1973) have proposed what they call a "cognitive theory," following the tradition of Ritchie (1951), Tolman (1949), and Irwin (1971). In this theory, learned avoidance responses come to be controlled by expectations and preferences, and responses can therefore be postulated to occur even in the absence of fear.

6. Even if we could agree that any of these attempts to explain, rather than postulate, the lack of extinction were successful, they would still leave unexplained a further difficulty in which the Watson-Mowrer theory is involved. In many neuroses we not only fail to observe the expected extinction of the unreinforced CS, but we find an incremental (enhancement) effect, such that the unreinforced CS actually produces more and more anxiety (CR) with each presentation of the CS. This fact is obvious when we consider the notion of "subtraumatic UCSs" which is sometimes introduced to salvage the Watson theory from the failure to discover traumatic UCSs in the history of the development of a neurotic disorder. In the theory of Pavlovian conditioning, there is no provision for CRs to achieve greater strength then UCRs (unconditioned responses); the dog never salivates more to the bell than to the food. As Mackintosh (1974) points out, "CRs, even if they resemble the UCR very closely, are usually weaker and of lesser amplitude" (p. 97). Yet the very notion of "subtraumatic UCS" implies something of this sort – the final CR (the neurotic breakdown) is stronger (involves more anxiety) than the UCR! This goes counter to all we know of the fate of UCRs; these are known to habituate, rather than to increase in strength.

7. The absence of a traumatic UCS, referred to in the above paragraph, deserves to be discussed separately from the major point raised there, namely the incrementation of CR effects when the CS is presented without reinforcement. Traumatic events do of course sometimes occur in connection with the development of a neurotic disorder, and indeed in wartime such traumatic events are relatively frequent (Grinker and Spiegel 1945). (Even there, experience has shown that many more neurotic breakdowns occur through separation from the family than through enemy action.) In peacetime neuroses, traumatic UCS are distinctly rare (Lautsch 1971; Gourney and O'Connor 1971); in the majority of cases there is some sort of insidious onset, without any single event that could be called "traumatic" even by lenient standards (Rachman 1968; Marks 1969). This fact is not accounted for in Watson's theory, and it is here classed with other criticisms related to extinction because, as we shall see, a revised theory that enables us to account for the nonoccurrence of extinction, and the enhancement of unreinforced CS, will also account for the absence of traumatic events.

8. The Watson and the Miller-Mowrer models tend to stress the importance of pain in connection with the UCR. "Pain" usually refers to simple physical pain, such as that experienced after the administration of shock. Shock, or any other obviously "painful" stimulus, clearly marks off the events in question as "traumatic"; the

absence of traumatic events of this kind in the development of most neuroses must cause us to doubt the omnipresence of "pain," at least in this obvious sense of the term. Watson, in his original formulation, postulated several natural causes of fear, such as loud noises, loss of support, and physical constraint; these are all "painful" in a physical sense.

Several alternative suggestions have been made for effectively substituting "mental pain" for physical pain in the conditioning paradigm. Thus Gray (1971) has shown that frustration ("frustrative non-reward") can have behavioural and physiological consequences identical with those of physical pain. We can thus substitute frustration for pain in the model, without losing touch with the experimental literature. Kimmel (1975) has suggested "uncertainty" as the basic UCS in the development of anxiety (Shenger-Krestovnikova 1921; Seligman, Maier and Solomon 1971; Masserman 1971); this is usually coupled with some unpleasant stimulus whose occurrence is uncertain and therefore evades proper control; but such a stimulus need not be accompanied by physical pain (Mineka and Kihlstrom 1978). Conflict is another UCS frequently adduced theoretically in lieu of physical pain (Yates 1962). Frustration, uncertainty, uncontrollability, and conflict are of course all related, although not synonymous; they all share the characteristic of representing "mental pain" (if this term be allowed) and thus introduce a cognitive element into the conditioning paradigm. This does not make the theory a cognitive one; this point will be discussed further below.

These four objections to classical conditioning theory present a powerful argument against the easy acceptance of Watson's model, or that of Miller and Mowrer. What is wrong, though, is not so much the conditioning theory of neurosis, as the classical theory of extinction. Eysenck (1968) has offered an alternative theory of extinction, which will be discussed in the next section.

5. The incubation of anxiety/fear responses

Classical extinction theory has always been beset by experimental anomalies. In a review of forty years of American and Russian experimentation, Razran (1956) stated that "extinction continues to be clearly a less than 100% phenomenon. Instances of difficult and even impossible extinction are constantly reported by classical CR experimenters" (p. 39). Eysenck (1968) has rewritten the law of extinction completely, suggesting that two consequences may follow upon the CS-only presentation. (We shall refer to the CS-only, or the unreinforced CS, by the symbol $\overline{CS}$.) Presentation of the $\overline{CS}$ may be followed by extinction of the CR, as predicted by the traditional formulation, or it may be followed by an actual enhancement of the CR. Eysenck has also suggested the parameters that control which of these contradictory consequences will in fact occur, and the reasons for the occurrence of *incubation*,[1] as he has called the underlying condition for the enhancement of the CR following $\overline{CS}$. In this section we will look at some of the theories underlying the postulation of the incubation-enhancement effect; in the next section we shall look at the parameters relating to this effect, and then at the facts supporting it. Let us merely note here that if this new version of the extinction law is correct, then the difficulties we have noted with the conditioning theory of neurosis disappear completely. Hence the importance we attribute to this new formulation of the law of extinction.

Traditionally, the term "incubation" refers to "a growth of fear over a time interval which follows some aversive stimulus. The increase in fear is assumed to be spontaneous, in the sense that the time interval is free of further exposure to the aversive stimulus" (McAllister and McAllister 1967, p. 180). Many studies of this phenomenon have been reported in the literature since Diven's (1937) original paper; the work of Bindra and Cameron (1953), Breznitz (1967), Brush (1964) and Brush and Levine (1966), Denny and Ditchman (1962), Desiderato and Wassarman (1967), Desiderato, Butler, and Meyer (1966), Golin (1961) and Golin and Golin (1966), Kamin (1957, 1963), McAllister and McAllister (1963, 1965), McMichael (1966), Mednick (1957), Saltz and Asdourian (1963), and Tarpy (1966) being perhaps best known. McAllister and McAllister (1967) conclude a review of this field by saying that "although the incubation-of-fear hypothesis has been tested in a wide variety of situations, the phenomenon has yet to be convincingly demonstrated" (p. 189). The view here taken is that this phenomenon, as defined and experimentally investigated, does not deserve a special title. It seems possible to explain the facts adduced simply in terms of a reminiscence-type theory, based in consolidation of the memory trace; such a theory has been advanced elsewhere in connection with pursuit-rotor learning (Eysenck and Frith 1977), and with suitable changes it would not seem impossible to apply this theory to the conditioning of the CS–UCS bond. The assumption here made is that consolidation is required to transfer the memory trace into long-term storage form (i.e. change it from some reverberatory circuit into a chemical form probably involving protein synthesis), that this consolidation takes time, and that conditioning phenomena do not in this respect differ from ordinary verbal and nonverbal learning (John 1967). If this brief outline of a theory is along the right lines, then it will be apparent why no special term is required for these rather weak phenomena; they merely exemplify certain consequences to be deduced from existing theories.

The possibility must also be considered that the traditional concept of incubation is merely a special case of incubation as here defined and discussed, that is, an instance of enhancement of the CR after $\overline{CS}$ exposure. The traditional definition of incubation only specifies exclusion of the aversive stimulus (UCS); it does not refer to the exclusion of the $\overline{CS}$. In most of the experiments cited, there is ample chance for the $\overline{CS}$, or parts of the $\overline{CS}$. to be presented explicitly or implicitly to the experimental animals. It is at present impossible to say which of the two hypotheses (reminiscence and $\overline{CS}$ exposure) is more correct in explaining traditional incubation phenomena, assuming these to be a reality; explicit testing of those two hypotheses should not present any difficulty. As we shall see, the evidence for incubation phenomena of the kind emphasised in the body of this article is much stronger than that for traditional incubation; this suggests that the explanation in terms of $\overline{CS}$ presentation may be the more correct one. Both of course may apply; the two explanations are not mutually exclusive.

In turning now to a theoretical account that might explain these curious phenomena which seem to run counter to the established facts of extinction, we do not wish to argue that extinction does not occur when the CS is presented repeatedly unaccompanied by the usual reinforcer. Our argument will be that the presentation of the CS unaccompanied by a UCS ($\overline{CS}$) always provokes a decrement in CR strength, but that, for reasons to be explained, it may also provoke an *increment* in CR strength, so that the observed CR is the resultant of two opposing tendencies; *extinction* will be observed if the decrementing tendencies are greater than the incrementing ones, while *incubation* will be observed if the incrementing tendencies are greater than the decrementing ones. We shall not be concerned here with the theoretical explanation of the nature of extinction (Kimble 1961), but take its occurrence as an established fact. Our concern will be exclusively with the reasons why a repetition of $\overline{CS}$s over a period of time should lead to an increment in CR.

Consider Grant's (1964) classification of conditioning paradigms. This is what he has to say about what he calls "Pavlovian B conditioning"; "Pavlovian A conditioning" is exemplified by the more familiar bell-salivation experiments:

"This subclass of classical conditioning could well be called Watsonian conditioning after the Watson and Rayner (1920) experiment conditioning fear responses in Albert, but Pavlov has priority. The reference experiment for Pavlovian B conditioning might be that in which an animal is given repeated injections of morphine. The UCR to morphine involves severe nausea, profuse secretion of saliva, vomiting, and then profound sleep. After repeated daily injections Pavlov's dogs would show severe nausea and profuse secretion of saliva at the first touch of the experimenter (Pavlov 1927, pp. 35–36). In Pavlovian B conditioning, stimulation by the UCS is not contingent on S's instrumental acts, and hence there is less dependence upon the motivational state of the organism,

and the CS appears to act as a partial substitute for the UCS. Furthermore, *the UCS elicits the complete UCR* in Pavlovian B conditioning whereas in Pavlovian A conditioning the organism emits the UCR of approaching and ingesting the food. A great deal of interoceptive conditioning (Bykov, 1957) and autonomic conditioning (Kimble 1961) apparently follows the Pavlovian B paradigm." (See also Kalat and Rozin 1973.)

The notion that "the CS appears to act as a partial substitute for the UCS" is basis to our new theory as is the fact that the UCS elicits the complete UCR. Consider the usual account of aversive conditioning. A CS is followed by a UCS, say shock, which produces a great variety of UCRs; some of these, or even one of these, may then be singled out for study. After a single pairing, or after repeated pairings of CS and UCS, $\overline{CS}$ produces some, or at least segments of some, of the responses originally produced by the UCS. Fear/anxiety responses are of particular interest in this connection; they are frequently produced by nocive UCSs and are readily conditioned. These CRs may be similar to the original UCRs, but they need not be; under certain conditions they may in fact be the exact opposite. Thus, in rats, shock (the UCS) produces parasympathetic responses, including heart rate decrement, but the $\overline{CS}$ produces sympathetic fear responses, including heart rate increment (Stern and Ward 1961, 1962; Fehr and Stern 1965). However that may be, CS and $\overline{CS}$ acquire the function of signalling danger and coming pain, discomfort, fear, and annoyance; let us denote these nocive consequences as NR (nocive responses). Through the intermediation of the UCS, the CS has become associated with the NR and signals their arrival to the organism. (For reasons that will become obvious later, we prefer the term NR in this connection to the terms UCR and CR. It will be argued that the classical account, which is implicitly accepted when we use the classical terms, is somewhat deficient, and that a novel nomenclature will be useful in formulating a theory that departs in some ways from the usual one.) Each reinforcement (which may be defined as an NR following a CS) increments the habit strength associating CS and NR; consequently, each CS/UCS pairing serves to increment the CR. When we administer a $\overline{CS}$, however, so classical theory assures us, this reinforcement is missing, and consequently extinction weakens the habit associating CS and NR.

We suggest that this account is partly erroneous. $\overline{CS}$, although unaccompanied by UCS or UCR, is in fact accompanied by CR, which is a partial, possibly weak, but real NR. Hence some reinforcement is provided, although perhaps this is so much weaker than that accompanying the UCS that its presence may not be very important under certain circumstances. Yet in principle it is always present, and its presence would theoretically lead to a strengthening of the CS/NR bond, and hence to some form of incubation. What is being suggested, in other words, is that conditioning sets in motion a positive feedback cycle in which the CR provides reinforcement for the $\overline{CS}$. Usually the extinction process will be stronger than this form of reinforcement, leading to overall extinction, and making the action of CS/NR reinforcement unobservable, but under certain circumstances (e.g. when the UCS is exceptionally strong) the extinction process may be weaker than the CS/NR reinforcement process and observable incubation will result.

Traditionally we would denote these NRs as response-produced stimuli, in the sense that measurable autonomic responses, such as changes in heart reate, breathing, and cessation of stomach contractions, are experienced by the organism as interoceptive stimuli. This division is unimportant for our argument. It might, however, serve to reassure critics who might feel that it is somewhat implausible to make a response (CR) act as its own reinforcer. It is not the CR itself that acts as reinforcer, but rather the response-produced stimuli; not the automatic, hormonal, and muscular reactions themselves but rather the experience of fear/anxiety based upon them. Insofar as these CR-produced stimuli are *identical* with the UCS-produced stimuli, it seems automatic that they will be reinforcing in exactly the same manner; insofar as they are *different* they will also act as reinforcers to the extent that they are painful and aversive.

What is proposed, then, is this. As Kimble (1961, p. 426) points out, "stimuli associated with painful events come, by a process of classical conditioning, to evoke fear. The status of fear as a motive is then inferred from the fact that it has the same properties as other motives, those of providing the basis for learning and of influencing the vigor of behaviour." What we propose to add is that fear, so generated, is itself a painful event, and therefore the stimuli associated with it (i.e. $\overline{CS}$) come, by classical conditioning, to evoke more fear, thus producing a positive feedback. This mechanism is well known descriptively in psychiatric disorders; it somewhat resembles Seneca's famous saying about having nothing to fear but fear itself.

The same mechanism must be assumed to be present when the CS is administered; we would postulate that to the NR produced by the UCS there is added an increment of NR produced by the CS. In this way, it becomes possible to account for the very strong NR achieved on the basis of a rather weak UCR (e.g. Anderson and Parmenter 1941; Liddell 1944). UCRs tend to decrement, due to habituation and adaptation, just as unreinforced CRs tend to extinguish; these trends are opposed sometimes successfully, sometimes not, by the CS/NR mechanism. (Wolpe 1958, has made a similar suggestion to account for the growth of neurotic disorder through many pairings of CS and weak USC; the growth of a final CR much stronger than the UCR is difficult to understand in orthodox terms.) To put the matter less technically, but perhaps more intelligibly: shock is followed by pain, $\overline{CS}$ is followed by fear. Shock + CS is followed by pain + fear; this combined NR is more potent (more disagreeable, more nocive, more aversive) than either alone and hence has greater reinforcing properties. $\overline{CS}$ is followed by fear as the CR, which is less reinforcing than pain + fear, but may be sufficiently reinforcing to more than counteract the decremental effects of extinction. When this occurs, incubation takes place. When shock is experienced a number of times, habituation/adaptation occurs. When shock is accompanied by CS, the addition of fear to pain may delay habituation/adaptation, or even become stronger in the balance and lead to the occurrence of NRs that are stronger than the original UCR. Thus there is a dynamic interplay between the components of the NR (UCR and CR) and the forces of habituation/adaptation and extinction which work against an incrementing and towards a decrementing of the CS/NR association.

It is interesting in this connection to note that Martin and Levey (1968) found, in their studies of eyelid conditioning, that the first unreinforced trial following a series of paired presentations of CS and UCS gave a response that was a combination of CR and UCR, showing clear traces of both, and *stronger than either alone.* This combined response quickly extinguished, probably because of the weak arousing properties of the UCS, but this experiment does seem to establish that the postulated evocation of the UCR by the $\overline{CS}$ can occur. Similar experiments with equally detailed attention to the nature of the response are required in the field of strong aversive conditioning to establish the hypothesis here presented.

The considerations discussed so far are likely to meet with some criticism on the grounds that CR and UCR are confounded. More than that, however, is implied in the theory, because the stress laid on the response must contrast inevitably with the usual stress laid on the *stimulus* in modern theorising. Where the classical account links the CS with the UCS, we would partly ignore the UCS and concentrate largely on the UCR. The differentiation between UCS and UCR is in any case somewhat artificial from the point of view of the organism that is being conditioned. Consider aversive conditioning, using shock. The shock is the UCS, and pain + fear the UCR; this makes sense from the point of view of the experimeter, who administers the UCS, while the S experiences the pain. However, the S does not feel shock (UCS) which produces pain (UCR); he experiences a painful shock, that is UCS and UCR are experienced simultaneously, and not as separate, consecutive entities. It is this Gestalt-like NR that is being linked with the CS through contiguity, and to which the CR eventually adds another increment of pain/fear which is introspectively very difficult or even impossible to differentiate from the original NR. In other words, the differentiation between UCS and UCR reflects preoccupation with control (the UCS is under control of the E and causes the UCR in the sense that what the E does produces

a response in the S;) from S's point of view (and, after all, conditioning process does take place in the subject) the differentiation is of doubtful relevance and value. UCS and UCR are temporally close together – so close that S often cannot differentiate between them – and in consequence it is difficult to disentangle the links that contiguity forges between CS and either; this difficulty can best be sorted out when UCS and UCR are temporally separated.

Such sorting out is possible in the case of apomorphine aversive conditioning, for example, for alcohol addiction; the drug (UCS) is given several minutes before the nausea it causes (UCR) supervenes. Orthodox opinion states that conditioning takes place when the CS precedes the UCS; when the UCS comes first, backward conditioning, which is weak at best and often nonexistent, is said to occur. In practice it is well known (Franks 1963, 1966) that strong, conditioned responses are also obtained when the CS immediately precedes the UCR, and follows the UCS by several minutes. This shows clearly the importance of the CS-UCR link, and the relative unimportance of the CS–UCS link, a distinction that has been hidden in most research because of the temporal contiguity (or even identity) of UCS and UCR. A particularly clear example of the irrelevance of the UCS is the experiment by Campbell, Sanderson, and Laverty (1964) in which temporary interruption of respiration (UCR) was produced by intravenous injection of succinylcholine chloride dihydrate (UCS). "The Ss were all unaware of the process of injection," which was part of a lengthy process of injection of saline solution and sometimes of atropine (to reduce salivation;) the CS was so timed as to precede the first sign of UCR – usually a sudden drop in skin resistance. Here the patient is completely unaware of the UCS; furthermore the UCS precedes the CS. This would mark this as a case of backward conditioning; yet as Kimble (1961) points out, "It is apparent that backward conditioning in which the UCS precedes the CS leads to little conditioning" (p. 158; but see Eysenck 1975b).

The traditional view seems to be based on the accidental temporal contiguity of UCS and UCR; it is the occurrence of the latter, not the former, that must be preceded by the CS. It must be clear that the view here taken requires substantial support from specially designed experiments before it can hope to take the place of orthodox views; such experiments are lacking because the orthodox view has seldom been challenged.

We are not proposing a special theory of extinction, but would like to draw attention to the fact that what has been said above agrees well with Razran's (1956) theory of extinction, and its more modern counterparts. The main point of this theory is that

"the automatic deconditioning in the early stage of extinction is a direct result of the loss of the interoceptive and the proprioceptive conditional stimuli (feedback CSs) which in the original conditioning were an integral part of the CR situation and which when the unconditional stimulus is withheld and the evoked reaction is reduced cease to be present."

In the case of anxiety conditioning these interoceptive and proprioceptive conditional stimuli remain, if in somewhat reduced form; they are sufficient to avoid extinction and instead produce enhancement. There is also some common ground between the concept of "incubation" in conditioning theory, and that of "sensitization" in habituation theory (Groves and Thompson 1970), although not too much should be made of the obvious similarities in view of the clear differences in pertinence and methodology. Nevertheless, the differentiation between extinction and habituation is easier to make in theory than to apply in practice. Consider a study (unpublished) performed by E. Nelson in our laboratories. He studied the decline in the extent of the penile reflex to the showing of 4 min. pornographic films, separated by 4 min. rest pauses; three films were shown per day, with 24 hours intervening between occasions. Extraverts were found to extinguish/habituate more quickly and strongly than introverts, both within films, between films, and between occasions, as predicted (vide infra). The question arises whether the process of diminution of penile reactivity is habituation to a UCS, or extinction of a $\overline{CS}$. Can films be regarded as in any real sense *unconditioned* stimuli? The distinction between UCS and CS is sometimes very difficult to make; often what we call the UCS is really a well-established CS, possibly removed by several steps from whatever may have been the true UCS. The rhetoric of conditioning paradigms does not always map easily into the realities of the experimental situation!

The theory here presented is probably deficient in not taking explicitly into account Pavlov's "second signalling system." Advocates of cognitive theories of emotion (Schachter and Singer 1962) have drawn attention to the important effects of cognitive recognition of autonomic feedback, whether genuine or experimentally falsified (Valins 1966; Valins and Ray 1967), and Lang, Sroufe, and Hastings (1967) have demonstrated the possibility of acquiring voluntary control over autonomic reactions, such as heart rate. A clear demarcation often appears in behaviour therapy between autonomic-behavioural and cognitive effects; Lang and Lazovik (1963) have reported immediate behavioural effects of their desensitisation therapy, but a long-delayed cognitive autonomic effect. While it would be desirable to go into these important but ill-understood matters, it would at the moment be purely speculative; there are too few facts available to make theorising fruitful. Nevertheless, the existence of a gap should be realised, and may lead to further work in this immensely important area. These comments are of course also relevant to our remarks about NRs being strictly speaking "response-produced stimuli"; cognitive theories are based on the stimulus properties of these responses.

So far our analysis has dealt entirely with aversive conditioning; would incubation phenomena also be produced in appetitive conditioning? Mowrer's (1956) theoretical concept of "hope" would suggest that such a possibility might not be too far-fetched, and there is ample experimental evidence for the motivational character of secondary (conditioned) reinforcers (Estes 1943, 1948; Walker 1942; Dinsmoor 1950). Direct evidence, however, is lacking, and as the point is not essential to the main purpose of this article, it might be best to leave this question open.

6. Parameters of the incubation paradigm

Demonstration that incubation occurs (see the next section), and the postulation of a theory (overlap of CR and UCR) explaining its occurrence, are not sufficient; we must also know under what conditions to expect incubation (enhancement), and under what conditions to expect extinction. Without knowledge of such parameter values, it is impossible to make and test predictions; any outcome of an experiment would be equally acceptable. In this section we suggest a number of parameters that are believed to be crucial to the occurrence of incubation. The first of these parameters concerns the nature of the CR and is connected with the requirement of "Pavlovian B conditioning" that the UCS elicit the complete UCR.

We must distinguish carefully between two types of CRs, namely those that have drive properties, and those that do not. Pavlov's bell produced a response (salivation), but this response had no drive properties. The experiment only worked when the hunger drive was already present in the dog; it did not produce hunger. (It may be possible to produce a conditioned hunger drive through periodic feeding schedules; the evidence is not too clear on this point. As Mackintosh (1974) points out, "the presentation of a stimulus established as a classical CS for appetitive reinforcement does not appear to increase the vigour or rate of appetitive instrumental responding" (p. 231). Also, "stimuli paired with a reinforcing event become signals for that event rather than generators of a motivational state" (p. 227). Giving rats shock after a CS (e.g. in a shuttle box) does produce a CS-induced drive (Miller 1951a); rats will learn new activities, and practice established ones, in order to avoid the CS. Sexual CSs may also be drive producing; tumescence can be conditioned to a CS (Rachman 1966b; Rachman and Hodgson, 1968), and being pleasurable, may constitute a drive. However, little research has been done on the drive-producing effects of CSs other than anxiety, and we shall here concentrate entirely on anxiety-producing CSs.

Our argument is that CSs that do not produce drives are subject to

the classical law of extinction, while CSs that do produce drives follow the law of enhancement (incubation).

The requirements of Pavlovian B conditioning, namely that the UCS should elicit the complete UCR, seem to be closely related to, and perhaps identical with, the requirement that the CR be a drive; in Pavlovian A conditioning the drive is already present when the experiment begins and is not elicited by the UCS. For that reason the CR has no drive properties. In Pavlovian B conditioning the UCS produces the drive, and hence the drive properties of the UCR can be transferred to the CR, thus making it possible for the CR to act as reinforcement for the CS–CR link. Neurosis cannot be understood in terms of Pavlovian A conditioning, but only in terms of Pavlovian B conditioning; yet the literature contains little work emphasising this difference.

The second parameter value we believe to be instrumental in mediating incubation is the strength of the UCS. Put in the form of a law, we would suggest that "incubation is more likely to follow strong UCS, extinction to follow weak UCS." (It is of course assumed that other things remain equal; this same assumption is made in our statement of our other laws.) We shall postpone discussion of this law until we have stated our second law, relating to the duration of the CS presentation. This law reads: "Incubation will follow upon short presentations of the $\overline{CS}$, extinction will follow upon long presentations of the $\overline{CS}$." The justification of these predictions runs along the following lines.

We have argued that when the CR acts as a drive (as in the case of anxiety), the CS can be regarded as a partial substitute for the UCS, and the CR as partly identical with the UCR. On this hypothesis, the CR acts as a reinforcement for the CS–CR connection, but will only do so provided it is strong enough to overcome the natural extinction tendency of all $\overline{CS}$. Hence incubation will only be observed if the UCS is strong enough in the first place for its partial substitute (the $\overline{CS}$) to overcome the extinction normally following the presentation of the $\overline{CS}$. The strength of the UCS is thus a crucial matter. Duration is important because the strength of the CR declines over time; the longer the exposure to $\overline{CS}$, the weaker will be the CR. (Empirical studies to justify this statement are available in Nunes and Marks 1975; Marks and Huson 1973; Stern and Marks 1973; Watson, Gaind, and Marks 1972; Borkovec 1972, 1974; Mathews and Shaw 1973; Mathews, Johnstone, Lancashire, Munley, Star, and Gelder 1974.)

Figure 1 illustrates the hypothetical events that take place upon the presentation of $\overline{CS}$. A strong CR is evoked which is felt as fear/anxiety by the patient (or the experimental animal). This CR habituates or extinguishes (Curve A) as $\overline{CS}$ presentation is prolonged, just as the UCR would habituate or extinguish. When strong, the CR can act as a reinforcement in much the same way the UCR does. Below a critical point, the CR is too weak to act in this manner, and hence beyond this point we get only extinction, rather than enhancement of the CR.

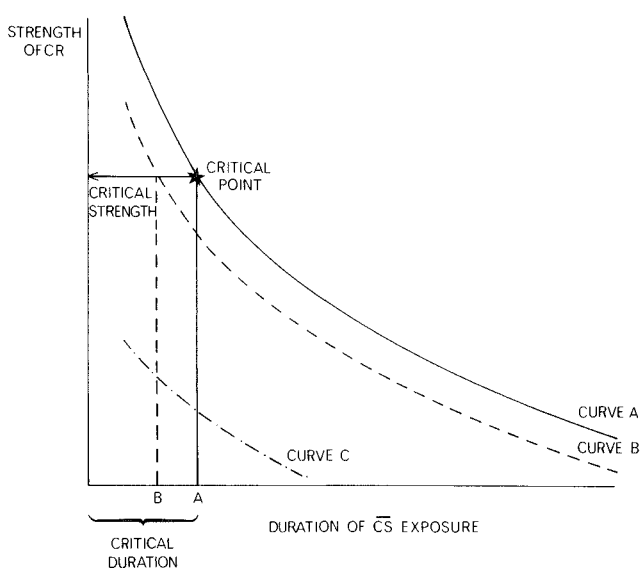

Figure 1. Strength of CR as a function of $\overline{CS}$ exposure.

This diagram also illustrates how this theory unravels the apparent inconsistency that exists in the clinical applications of behaviour therapy known as desensitization and flooding. In the former, the patient is protected against any strong anxiety arising during therapy by a procedure in which he is kept in a relaxed state, and is presented $\overline{CS}$, whether *in vivo* or in imagination, only at points on the hierarchy that will arouse him relatively little (i.e. points well below the critical point). If this point is ever exceeded, the $\overline{CS}$ is immediately withdrawn; it has often been demonstrated that when the critical point is reached or exceeded in desensitization, the success of treatment is imperilled, and the patient is actually made worse (Wolpe 1958.) In flooding, on the other hand, the patient is immediately confronted with the most threatening $\overline{CS}$, that is one at the top of the hierarchy; this procedure, which includes explicitly an element of response prevention, is continued for periods of an hour and more. Both desensitisation and flooding are successful in practice (Kazdin and Wilson 1978), although they appear to proceed in contradictory directions; this clearly is an anomaly which has posed considerable problems to a theoretical explanation of treatment success.

According to the theory embodied in Figure 1, the answer lies in the short duration of the exposure to high-anxiety $\overline{CS}$ which occurs in desensitization when the therapist makes an error; the critical point is exceeded, and consequently enhancement takes place, rather than extinction, which only occurs at levels of anxiety below the critical point. In flooding, exposure to the $\overline{CS}$ is continued long enough to get well below the critical point; hence extinction takes place, and no enhancement.

It can be deduced from this theory that flooding procedures that only exposed the $\overline{CS}$ for brief periods would make the patient worse, rather than better. We would also predict that when in desensitization the therapist in error evokes a too strong $\overline{CS}$, he should not follow the usual practice of immediately withdrawing the $\overline{CS}$, but should rather continue as if he were engaged in a flooding procedure. This has not hitherto been done (or at least, has not been reported); it would seem to constitute a crucial test of the theory. It may also serve to indicate that the theory is not without relevance to the practice of behaviour therapy.

What would be the consequence of successful presentation of the $\overline{CS}$, either through a desensitization procedure (i.e. using mild $\overline{CS}$ under conditions of relaxation) or through flooding, continued long enough to get the subject below the critical point? Either procedure would theoretically lower the curve indicating the strength of the CR (Curve A), as indicated by the broken line (Curve B); it would similarly shift the critical duration from point A to point B. This process of extinction would then be followed in desensitization by using stronger $\overline{CS}$ and thus raising the curve again to its original level, while in flooding the process of extinction would simply be continued along the same lines as before. (The diagram oversimplifies the situation by using the same curve to illustrate both desensitization and flooding; normally desensitization would use a much weaker $\overline{CS}$, as illustrated in Curve C. Curve C is never high enough to produce the critical strength of CR that would lead to enhancement.) To avoid excessive strength of the CR (which is advisable in clinical practice because patients may find the task too gruelling) tranquilizers may be used to lower the curve (Curve A), but not so much that it lies wholly below the critical point; this procedure has been used successfully in therapy (Marks, Viswanathan, and Lipsedge 1978).

Before leaving the discussion of parameter values, we must mention the importance of individual differences in this field, already adumbrated by Watson and Rayner (1920). There is strong evidence to indicate that personality dimensions of extraversion–introversion and neuroticism-stability are closely involved with the origins of neurosis (Eysenck and Rachman 1965; Eysenck 1967a, 1976c, 1977b, 1977c), in the sense that people who are high on N and low on E are much more likely to develop neurotic disorders than are people not in that quadrant of the personality space. There is equally

strong evidence for the genetic determination (to the extent of accounting for over half the variance) of both these personality factors (Eysenck 1976a, 1976b, 1977c, 1977d; Shields 1973, these references also document the partial genetic determination of neuroses as such). Last, there is good experimental evidence that N as a personality factor is produced largely by overreactivity of the limbic system, exerting a steering action on the autonomic system, while E is mediated by the ascending reticular formation, which in turn governs the arousal level in the cortex, producing a low resting level in extraverts and a high resting level in introverts (Eysenck 1967a; 1976c). Thus in theory the high N-low E person is predisposed to neurosis because he reacts strongly to emotionally arousing stimuli and strongly conditions these stimuli. (Conditioning is quicker and stronger when arousal is high; Pavlov 1927; Eysenck 1976c.) Introverts also extinguish conditioned responses more slowly than extraverts (Hemming 1979).

Such personality differences as these (or, in animal work, comparable differences) interact with the parameters already mentioned. (For animal subjects see Eysenck 1964; Chamove, Eysenck, and Harlow 1972; Sartory and Eysenck 1976; for human subjects Kantorowitz 1978; Sarason 1958; and Sipprelle Ascough, Detrio, and Horst 1977.) The prediction would be that weaker UCS are required to produce the same effect in high N scorers, or low E scorers, than would be required for low N or high E scorers. Similarly, duration of $\overline{CS}$ would need to be longer for high N or low E scorers to get below the critical point, as compared with low N or high E scorers. The possibility of "trading" personality variables against experimental variables has been discussed, in relation to experimental evidence, by Savage and Eysenck (1964); the hypothesis is supported at both the animal and the human level. Unfortunately, clinical psychologists have not shown much interest in the systematic investigation of personality parameters, in spite of the promise these hold out for more accurate prediction (Di Loreto 1971), so that on this point the evidence is rather meagre. We suggest, nevertheless, that personality interacts strongly with situational parameters and cannot be left out of any theory purporting to encompass the total situation.

A recent paper by Hugdahl et al. (1977) will serve to illustrate the relevance of personality differences, although instead of using personality questionnaires these authors used spontaneous electrodermal responses as their measure of arousal; this measure is known to correlate with introversion and anxiety (Eysenck 1967a). Using neutral and "prepared" CS in electrodermal conditioning, they found "that arousal and fear-relevance are additive factors in conditioning" (p. 353). They go on to say that

"if the present situation can be taken as a model of real-life phobic conditioning, the results suggest that persons with a habitual high arousal level, are more susceptible to acquire phobias than are people with low arousal levels, and that fear-relevance is an important factor in the content of the phobias. Furthermore, fear-relevance and activation seem to interact so that a high habitual arousal level is increased even more than when potentially phobic stimuli are encountered, since the number of spontaneous fluctuations was higher in the high-phobic than in the high-neutral group. At such high levels of arousal, there is a danger that the habituation mechanism becomes inoperative, which might lead to unselective, diffuse responding to all stimuli. For instance, in the present study, the high-phobic group did not differentiate between the significant CS+ and the unsignificant CS− during acquisition" (p. 352).

Spontaneous electrodermal fluctuation is a good measure of personality to use in this context, because it is correlated both with introversion and with neuroticism-anxiety (Lader 1967; Lader and Wing 1966). The results of the Hugdahl et al. work fit in well with the general theory here proposed and illustrate the importance of individual differences, the "preparedness' of CS, and the interaction between these two factors. Clearly, habituation and extinction are very much attenuated when suitable CS are used with suitable personality types, that is types showing high arousal whether this arousal is indexed directly by means of psychophysiological measures, such as spontaneous electrodermal fluctuations, or by means of personality questionnaires (Crider and Lunn 1971; Bohlin 1971; Lader 1967).

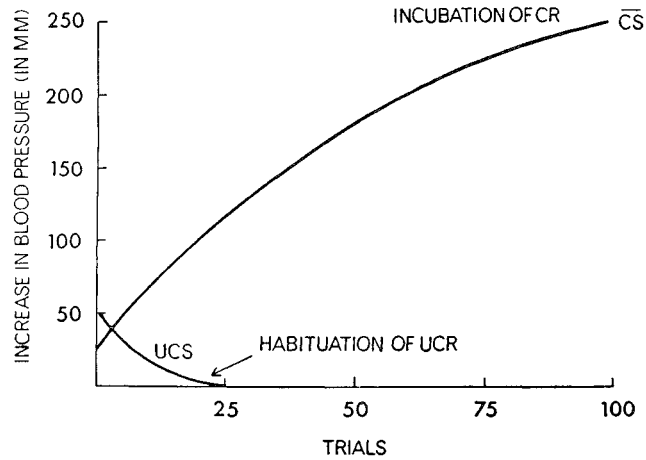

Figure 2. Diagrammatic representation of incubation of $\overline{CS}$ in Napalkov study (Eysenck 1976d).

7. Experimental and clinical evidence for incubation

In this section , we will very briefly mention some of the older studies that give support to the general notion of incubation (enhancement of the $\overline{CS}$), and then turn to some of the studies specially carried out in order to look at the suggested parameter values. Napalkov, (1963) working with dogs, found that various nocive stimuli produced increases in blood pressure of less than 50 mm., complete adaptation occurring after some 25 applications. A single conditioning trial, however, followed by repeated administrations of the CS (never the UCS) brought about increases in blood pressure of 30–40 mm. at first, rising to 190–230; the hypertensive state produced lasted over a year in some cases. The study has been discussed in some detail by Eysenck (1967b); Figure 2 shows in diagrammatic form the fate of the UCR and the $\overline{CR}$ in this experiment. The rapid habituation of this (subtraumatic) UCS, and the tremendous incrementation in the CR following presentation of the $\overline{CS}$, present a convincing contrast, illustrating the need for postulating an alternative to the traditional law of extinction, according to which the $\overline{CS}$ should have led to much more rapid extinction than is shown in the habituation of the UCR.

Lichtenstein (1950) reported on the inhibition of feeding responses in dogs following upon shock administered while the dog was eating. He noted that "a prominent feature of the anxiety symptoms is their tendency to develop and fixate after shock reinforcement has been discontinued" (p. 29). He writes:

"A further striking feature of anxiety symptoms is that they may be formed, increased in strength, and fixated some time after shock has been discontinued. We have mentioned, for example, the fact that resistance to entering the stock increased over a period of days. Tremors and tic-like movements, not observed directly after shock application, appeared later. The conditioned respiratory gasp likewise did not appear in some dogs until after the acquisition of the feeding inhibition."

He attempts to account for the phenomena in terms of a drive reduction theory. "Tics, tremors, struggle, etc. could . . . be fixated in anxious animals if they were followed by a drop in anxiety level. Since any response other than eating could be reinforced by anxiety-reduction there may be a trial and error factor accounting for the particular response which is stamped in." This does not appear a very likely explanation; Lichtenstein does not explain why "tics, tremors, struggle, etc." should lead to anxiety reduction or why they should appear in the first place; trial and error behaviour does not usually include such manifestations of anxiety. Furthermore, as components of an anxiety reaction, these types of behaviour would be more likely

to increase, rather than decrease anxiety, and hence be better understood as part of the incubation of fear/anxiety.

Dykman, Mack, and Ackerman (1965) arrived at somewhat similar conclusions in their work on conditioning and extinguishing specific and general responses in dogs; they stress the importance of nonspecific CRs and point out the importance of recording as many of these as possible. They summarise their findings by saying that "in general, extinction was more upsetting than conditioning, and this finding is contrary to expectation. *Apparently to some dogs the threat is more traumatic than the presence of shock.* The median of "symptoms" during all conditioning phases was 5.0, and the median number during extinction was 13.0 ($p<0.01$ binomial test)." In several other studies, Dykman, Murphree and Ackerman (1965) have suggested that

"the threat of trauma continues to operate in extinction . . . sometimes preserving the CR and sometimes interfering with it as real behavioral pathology appears. . . . We suggest that spontaneous recovery could stem from a failure of the experimenter to desensitize all relevant cues, the most important cue being, of course, the UCS. If this conjecture is correct, we could then obtain a more stable extinction by bringing back the UCS at reduced intensity, or better, by gradually decreasing the intensity of the UCS to a zero level" (p. 228; see also Dykman and Gantt 1958, 1960a, 1960b; and Galbrecht, Dykman, and Peters 1960).

Dykman et al. (1966) go on to demonstrate that longitudinal data, as well as genetic data from litter differences, "support the conception that the CR is dependent upon innate patterns of reactivity" (p. 430).

We have mentioned the relation of incubation to personality, with special reference to humans. Here let use merely note that the potency of "threats" (CSs) as compared with UCSs has also been demonstrated in the human field (Bridger and Mandel 1964); the principle appears to have wide applicability (see also Cook and Harris 1937). Maatsch (1959) has also reported a similar continued increase in an avoidance CR in rats subjected to a single shock trial, over a fixation criterion of 100 massed extinction trials.

Studies of "partial irreversibility" of conditioned fear responses, such as those of Solomon, Kamin, and Wynne (1953), and Solomon and Wynne (1953, 1954), show an increment in CR strength, indexed by decreased latency after withdrawal of the UCS; some of their data suggest close affinity with the concept of incubation put forward here. However, their data are complicated by the fact that their experiments used avoidance learning paradigms, so that simple incubation was complicated by newly acquired avoidance responses. Nevertheless, this work is valuable and partly relevant and will be borne in mind when coming to discuss our theory of incubation. Even a cursory look at their results will show that no consolidation-reminiscence theory, such as might be invoked for the majority of the orthodox "incubation" studies, can account for their data. One of the main reasons for not believing that consolidation can have been effective in the Solomon, Napalkov, Lichtenstein, and Campbell studies is the time element; consolidation is not expected to work over periods in excess of a few hours, while in these studies increments in CR strength were observed over weeks or even years. It is this temporal factor, coupled with the occurrence of CSs, that sets aside the few studies under consideration here from those reviewed by McAllister and McAllister (1967).

Next, we come to Reynierse (1966) who found that both time and duration of $\overline{CS}$ exposure were influential in deciding on the course of extinction/enhancement (see also Baum 1970, and Sartory and Eysenck 1976). In human subjects, Campbell, Sanderson, and Laverty (1964) found enhancement effects after a single, traumatic experience of respiratory paralysis; despite repeated extinction trials, 30 administered 5 min. after conditioning, 30 one week later, and 40 two weeks after that, the GSR (galvanic skin response) continued to gain strength over time. These and other slightly less relevant studies have been reviewed in Eysenck (1968).

More recently, Rohrbaugh and Riccio (1970), Silvestri, Rohrbaugh, and Riccio (1970), and Rohrbaugh, Riccio, and Arthur (1972) have attempted to test the writer's incubation theory directly. They exposed rats to $\overline{CS}$ in the form of apparatus cues, between conditioning and testing, and succeeded in demonstrating enhancement effect. They also tested the hypothesis that duration of $\overline{CS}$ exposure was an important variable and succeeded in demonstrating that short $\overline{CS}$ exposures produced enhancement, long $\overline{CS}$ exposures extinction. More recently still, Sartory and Eysenck (1978) studied five different strains of rats which were repeatedly subjected to extinction trials following Pavlovian fear conditioning, the duration of the extinction trials being varied for different groups of animals. Results showed that fearfulness of the animals (strain differences) and duration of extinction trials were jointly and severally causal in determining the degree of extinction of the conditioned fear response.

Last, Morley (1977) used the emotionally reactive and nonreactive Maudsley strains of rats (Eysenck 1964) and found that

"the two strains differ in the nature of their responses to the present experimental conditions. . . . The data confirm the hypothesis that individuals of the *emotional* type, are most likely to develop neurotic avoidance behaviour (Eysenck and Rachman 1965). Moreover the present experiment indicates that the avoidance behaviour may incubate and thus not be manifest until some time after the initial exposure to the CS–UCS pairing" (p. 367).

Morley's use of a punishment contingency procedure makes it somewhat more difficult to assess the relevance of his experiment to the theory here under discussion.

Of particular relevance to the hypotheses shown in diagrammatic form in Figure 1 is the work of Siegeltuch and Baum (1971) and Baum (1969b), in which they showed that the length of response prevention must be increased if the level of fear is to be enhanced, either by having a session of unavoidable shocks prior to training or by increasing shock during training. This is exactly the prediction that follows from our hypothesis about the "crucial point." Also relevant are findings by Baum (1969a, 1970) on the relationship between intensity of shock and duration of response prevention. Ward (1976) failed to replicate these findings, but Weinberger (1965), Spring, Prochaska, and Smith (1974), and Reynierse and Wiff (1973) all report that the longer the duration of response prevention, the quicker the extinction of the avoidance response.

The experimental evidence in favour of these deductions from our theory is impressive, both in the animal field and in the field of behaviour therapy with human patients. Rapid extinction of responses has been found with flooding in animals (e.g. Baum 1966; Page and Hall 1953; Polin 1959), and with neurotics (e.g. Rachman, Hodgson, and Marks 1971; Hodgson, Rachman, and Marks 1972; Rachman, Marks, and Hodgson 1973; see also review by Baum 1970). These studies all used lengthy $\overline{CS}$ presentations; with short presentations failures of extinction to occur have been observed, and in many cases incubation (enhancement) effects (review by Wood 1974). Thus in a study reported by Rachman (1966a); one of three spider-phobic subjects, exposed for ten sessions to $\overline{CS}$ presentation of spiders for 2-min. periods, reported that her fear of spiders *increased* during treatment. Periods of 1–1½ hours seem best for producing extinction effects.

A number of recent studies with human subjects have investigated experimentally the effects of short versus long exposure to $\overline{CS}$. Miller and Levis (1971) succeeded in verifying the importance of length of $\overline{CS}$ exposure on the fate of the CR. Proctor (1968) and Watts (1971) studied the influence of intraitem exposure time to aversive stimuli on systematic desensitization. Ross and Proctor (1973) found long single exposure to hierarchy items more effective in reducing avoidance behaviour than short exposure. Sue (1975) has reviewed a number of successful and unsuccessful extinction like studies in humans (exposure only) and found that success depended crucially on length of exposure; his own study gave similar results. There are also studies showing that exposure to symbolic representations of feared stimuli can elicit unexpected *increases* in autonomic responses, whether these stimuli were visual (Borkovec and Glasgow 1973), verbal descriptions (Boulougouris, Marks, and Marset 1971), or self-induced thoughts (Rankin, Nomikos, Opton, and Lazarus 1964; Breznitz 1967). Stone and Borkovec (1975) also found evidence of a paradoxical effect of brief $\overline{CS}$ exposure on analogue phobic subjects, in a study replicating that of Miller and Levis (1971), but with

certain additions that served to test (and disprove) hypotheses regarding the phenomenon of fear incubation advanced by Staub (1968) and others.

We may conclude this brief survey, as did Wood (1974) in his more detailed account, with the observation that incubation, paradoxical enhancement, or whatever we may wish to call the phenomenon of increased CR after short-term presentation the $\overline{CS}$, is a very real phenomenon, observable in both animals and humans, and having experimental parameters that can be deduced from more general theory, and can be tested in the laboratory. Most of these tests have borne out expectations that strength of the UCS, duration of exposure to the $\overline{CS}$, and personality variables would be of crucial importance in defining the properties of this phenomenon.

8. Cognitive factors and other problems

We have discussed some of the difficulties raised by the Watson and the Miller–Mowrer theories of neurosis and have attempted to show that these can be overcome by adapting our theories of conditioning to modern developments, such as the recognition of "preparedness" and incubation. Nevertheless, some problems remain that deserve at least passing mention (Beck 1976; Bandura 1974). The first of these is the problem of cognitive factors.

In 1969 Bandura wrote that

"all behavior is inevitably controlled, and the operation of psychological laws cannot be suspended by romantic conceptions of human behavior, any more than indignant rejection of the law of gravity as antihumanistic can stop people from falling. . . . The process of behavior change involves substituting new controlling conditions for those that have regulated a person's behavior" (p. 85).

Such a statement is in line with the thrust of this paper. However, more recently Bandura (1974) has changed his mind and has joined forces with critics of behaviourism who believe that it embodies an erroneously "mechanistic" view of human behaviour. Together with other advocates of a "cognitive theory" of human behaviour, he states that an adequate account of human learning must recognise that "contrary to the mechanistic metaphors, outcomes change human behavior through the intervening influence of thought" (p. 859). Such a view, and such a criticism of the conditioning model, has been brought forward by many other writers (e.g. Beck 1976; Ellis 1974; Goldfried and Goldfried 1975; Locke 1971; Mahoney 1977; Meichenbaum 1975). Replies have been equally many and varied (e.g. Rachman and Eysenck 1966; Eysenck 1971, 1972; Wolpe 1978). It is not the purpose of this brief comment to enter in any detail into this controversy, but it may be useful to state the position as the behaviourist sees it.

Let us begin by recognizing that Pavlov clearly introduced the language system into his theory of conditioning when he contrasted the second signaling system, found only in humans, with the more primitive conditioning systems found in animals; this certainly constitutes a recognition of "cognitive factors," even in the most strictly objective behavioural system. Platonov (1959), in his important book *The Word as a Physiological and Therapeutic Factor* continued this line of argument by showing experimentally that words and concepts can and do enter as elements into the conditioning process in the case of human subjects. And Martin and Levey (1978) have collected evidence to show that "evaluative conditioning" is a process that uses the principles of conditioning in a specifically human context. It would be difficult to argue in the face of this evidence (which could be multiplied many times, of course) that those who try to understand neurotic and certain other types of human behaviour in terms of conditioning have omitted an important "cognitive" element. This element has not been omitted; it has simply been shown to obey the same laws as do other types of human behaviour.

Wolpe has argued this point more extensively; he concludes: "The same lawfulness that applies to other behavior applies to cognitive behavior. We can easily see the error of Bandura's contention (1974, p. 860) that change in human behavior occurs 'through the intervening influence of thought.' Thoughts are responses whether they are perceptions or imaginings. Like other responses, they are evoked when the relevant neural excitations occur. They are a subset of learnable responses and, inasmuch as they have stimulus aspects, may be conditioned to other thoughts and to responses in other categories. They are not part of a separate *mechanism* of learning that only human beings possess" (1978, p. 441).

Cognitive psychology is really a dogma in search of a theory; there is no such thing as "cognitive theory," that is a body of experimental and theoretical knowledge from which deductions can be made, tested in the laboratory or the clinic, and then used to support or disprove the theory. In this it differs profoundly from conditioning theory, which clearly fulfills these requirements – even though of course, like all scientific theories, it encounters many anomalies and may in the end be found wanting. Cognitive theorists never criticise, as they ought to, the strong points of conditioning theory, nor do they attempt to show how these predictions and findings could be explained in cognitive terms. Consider for instance the prediction (and demonstration) that relapses in the bell-and-blanket conditioning treatment of enuretics can be prevented in large part by using an intermittent reinforcement schedule (Finley, Wansley, and Blenkarn 1977).

These predictions follow directly from a conditioning paradigm; there is nothing in cognitive theory that would make such predictions. This situation is typical; it could be repeated many times using the material referred to in this paper. Until cognitive theorists can account for the known facts in terms of an agreed theory, based on laboratory research, at least as well as do learning theory advocates, they cannot be said to offer a proper alternative. To point to the existence of cognitive elements in learning theory is not enough; these form part and parcel of any proper theory of human conditioning and are implicitly or explicitly incorporated in such a theory. The cognitive theorist claims far more than this, but has hitherto completely failed to demonstrate that these further claims are in fact justified.

9. Summary and conclusions

We have examined the evidence concerning the conditioning theory of neurosis, and we may conclude that, with suitable alterations, sometimes quite fundamental, the theory stands up to empirical test surprisingly well. In saying this we are of course well aware that we left the concept of "conditioning" itself without critical discussion; this is an obvious weakness of this paper, but to have done otherwise would have doubled its length. Our position is similar to that taken by Gray (1975), but we do not believe that one needs to accept in every detail Gray's model in order to make predictions in the field of neurosis, acquisition of fears, and treatment of symptoms. We recognise, of course, that many of the assumptions made by Pavlov, and many of the conceptualizations of conditioning paradigms by Watson, by Mowrer, and by later writers, are now merely of historical interest. Recent work on sign tracking (autoshaping), with its emphasis on informational variables, is but one example; this suggests a liberation from the narrow confines of conditioning theory as a theory of reflex action; it is much more realistic to speak of conditioned *responses* than of conditioned *reflexes*. We must also recognise the need to take into account operant processes, particularly negative reinforcement; these will complicate the picture, but they do not, in our view, force us to change the focus from Pavlovian conditioning as the most fundamental concept in the genesis and maintenance of neurotic behaviour.

Our theory, which may be regarded as the third model of the conditioning paradigm, with Watson's the first, and Mowrer's the second, posits the following stages. (1) First, it is postulated that organisms are born with certain fears of more-or-less specific stimuli; in animals these are likely to be quite specific, in humans less so. (2)

Some fears are "prepared," in the sense that while they may not be apparent on first encountering the object of the fear, these objects can easily become CS, either by being paired with a UCS, or by an increment in the arousal level of the autonomic system, or both. (3) Prepared fears can be conditioned even with degraded input, that is with nonoptimal CS–UCS interval, single trial conditioning, and relatively weak UCSs; with nonprepared fears this is not so. (4) Conditioned anxiety/fear responses will normally extinguish upon presentation of the $\overline{CS}$; this may be the basis of spontaneous remission in neurosis. (5) Under special conditions, incubation (enhancement) of the conditioned response will be observed, instead of extinction, upon presentation of the $\overline{CS}$. (6) Incubation takes place under specified conditions, that is having regard to the strength of the UCS, the duration of the $\overline{CS}$ presentation, and the personality of the subject. (7) Neurosis as a state is the product of this incubation process, which creates a positive feedback system. (8) The treatment of neurosis, through such methods as desensitization, flooding, and the like follows the laws of extinction, modified by the conditions giving rise to enhancement. (9) In the processes of acquisition and extinction of fear responses, cognitive factors (through the agency of the second signalling system) play a part, for example in defining the more complex stimuli, and in widening the gradient of stimulus generalisation; nevertheless, the major role in both acquisition and extinction is played by Pavlovian conditioning. (10) In defining Pavlovian conditioning, there has been a shift from a stress on reflex action to a stress on informational components; such shifts must be taken into account in making predictions in this field. (11) Conditioning theories like the one presented here apply to the genesis not only of neuroses, but also of psychosomatic disorders (Martin and Levey 1979). (12) The theory here presented accounts for such effectiveness in the treatment of neurotic disorders as is possessed by the different varieties of behaviour therapy and psychotherapy, and also for spontaneous remission (Eysenck, in press).

Theories in science are not right or wrong; they are fruitful or not depending on whether they lead to research with theoretically and practically important results. Judged by this standard, it seems that the conditioning theory of neurosis has been extremely fruitful, leading to extensive research activity both in the animal laboratory and in the clinic. The results of this research activity have inevitably led to a modification of the original theory; no doubt this most recent model, embodying many of these modifications, will in turn give way to a more advanced one. This is as it should be; in science no theory is sacrosanct, and no model lasts for very long. At the moment, the model here presented seems to incorporate most of the empirical findings relating to the development and treatment of neurotic disorders; inevitably the model is complex but then so are the facts it attempts to represent. All the different parts of the model are open to experimental testing; more cannot be asked of any scientific theory.

Summary

It is suggested in this paper that the only theory of neurosis to meet customary standards of scientific endeavour is the conditioning theory first put forward by Watson, and later amplified and brought up-to-date by Mowrer and Miller. It is also suggested that this theory, in its original form, is open to many criticisms, both from the side of the experimental psychologist and the learning theorist, and from that of the psychiatrist and clinical psychologist. A third version of the theory is proposed, taking into account modern developments and novel concepts, such as the notions of "preparedness" and "incubation." An attempt is made to show that with the adoption of these concepts the theory is found to be less susceptible to criticism, and that it can also explain and predict important phenomena in the field of treatment. It is argued that such a theory embodies cognitive features along lines already suggested by Pavlov and Platonov, and that so-called "cognitive theory" does not constitute a body of knowledge sufficient to serve as an alternative to the conditioning model.

NOTE

1. *Incubate:* cause development by creating suitable conditions. *Incubation:* phase through which germs of disease pass before development of first symptoms. *The Concise Oxford Dictionary.*

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Conditioning theory and neurosis. In developing a conditioning interpretation of neurosis, Eysenck relies less on the response-reinforcement or instrumental conditioning principles of learning and more on the Pavlovian or classical conditioning principles. He has been steadfast in holding to this view, and now his stand is amply supported by several theoretical and empirical developments (Bindra 1972, 1976; Bolles 1972; Estes 1972; Wasserman 1973). Eysenck's emphasis on differential associability of any specified conditional stimulus (CS) with various unconditional responses (UCRs) as a factor in determining the efficacy of conditioning is also well accepted, though not all would agree that the concept of "preparedness" or that of "phylogenesis" provides an explanation of the empirical principle of differential associability. Finally, Eysenck tellingly brings out the inadequacy of the instrumental learning principles to deal with the persistence and resistance to extinction of anxiety and avoidance responses and then points to the need for some such concept as "incubation."

Despite these important and useful features of his theoretical scheme, Eysenck's conditioning model of neurosis remains far from adequate. This is because his new formulation of the process of conditioning still deviates considerably from current understanding of the nature of classical conditioning and its relation to the production of behavior. Here I shall take up three points to illustrate that, by making use of certain current interpretations of the process of conditioning, Eysenck could have developed a more valid conditioning model of neurosis.

The first point to note is that Eysenck's account remains tied to the traditional view that conditioning is the establishment of an associative or signaling relation between a conditional stimulus (CS) and a conditional *response* (CR) and that response reinforcement has something to do with determining the strength of the conditioning. By retaining the dual assumption of stimulus-response links and response reinforcement, he makes his model inadequate for dealing with the innovative flexibility of adaptive action. Thus, while his model may well account for certain aspects of the stereotypy and persistence of neurotic symptoms, it is completely inadequate to deal with the changeability and adaptive flexibility of, say, the paralyses of a hysteric, the rituals of a compulsive, or the avoidance responses of a phobic.

An alternative to the CS-CR reinforcement view of conditioning (and neurosis) is provided by the perceptual-motivational account of conditioning and response production (Bindra 1974, 1978). According to this, a CS, by activating a neural representation of the UCS, generates the same *motivational state* (rather than the response) as would be generated by the unconditional stimulus (UCS) – a stimulus with reinforcing or incentive properties. The role of the unconditional (reinforcing or incentive) stimuli is then not one of strengthening a stimulus-response relation but one of generating, in combination with the prevailing organismic-condition ("drive") variables (food depletion, hormonal changes, etc.) a certain central motivational state (cms). It is this cms and the *relative* incentive value of various environmental stimuli (conditional and unconditional) that then determine the moment-to-moment response output. Contrary to the traditional views (e.g., Hull 1943; Tolman 1932), as well as the two-factor theory (Mowrer 1960; Rescorla and Solomon 1967), which make motivation ("drive") an instigator of *existing* responses or habits, the perceptual-motivational view emphasizes the fresh construction of each response on the basis of the *current* motivational states and the *current* perceptual input; motivation, instead of being a mere instigator of preformed responses, is regarded as a determiner of the variable and finely adjusted form of the response produced at a given moment. Such an account of conditioning appears to be required if the great variability and situational specificity of neurotic symptoms is to be explained. According to the perceptual-motivational view of conditioning, the stereotypy and persistence of responses (including symptoms) are also an outcome of fresh response construction, but this construction takes place in "confinement" situations where

the spatiotemporal layout of the behavioral environment remains stable; thus both (adaptive) flexibility and (maladaptive) stereotypy are accounted for in a single framework.

A second problem with Eysenck's view of conditioning is his acceptance of Mowrer's emphasis on the role of the stimulus consequences (sensory feedback) of viscerosomatic changes (CRs) produced by CS. While Mowrer regarded this feedback as motivational in character and called it "emotion," Eysenck regards the viscerosomatic changes as "drive" and calls their feedback a weak reinforcer of the relation between CS and the viscerosomatic (CR) changes. Both positions can be challenged. There is considerable evidence to show that the CS-induced viscerosomatic changes are the consequences, rather than the source, of central motivational processes (Bindra 1968; Rescorla and Solomon 1967). It appears now that the basis of such CS-induced motivational processes is the relation established between the central representations of CS and UCS and not any direct CS-UCR relation; Spence's (1960) and Miller's (1963) early idea that motivation arises from the sensory consequences of anticipatory conditioned *responses* elicited by the CS is no longer tenable (see Bindra 1968). Eysenck's argument in favor of a direct CS-UCR relation is based primarily on some old experiments of Franks (1963, 1966) on the conditioning of certain drug effects. More recent work with the conditioning of drug effects shows the conditioning effects to be highly variable, and there is as yet no consensus on their interpretation (Eikelboom and Stewart 1979; Siegel 1977, 1978).

The third difficulty with Eysenck's scheme arises from his argument that in Type A conditioning the drive-inducing (or reinforcing) properties of viscerosomatic CRs are different from those in Type B conditioning. In Type A conditioning, usually involving appetitive UCSs (e.g., food), the CS has no motivational properties, while in Type B conditioning, usually involving aversive UCSs (e.g., electric shock), the CS leads to drive stimuli that have reinforcing properties. The crux of Eysenck's argument is that Type B CSs are subject to the laws of enhancement (incubation) and Type A CSs are subject to the laws of extinction; he attributes the persistence of neurotic symptoms to the Type B enhancement effect. Eysenck makes the important point that the presentation of a nonreinforced CS, which typically leads to an extinction of the response may also, under certain conditions, lead to an enhancement of the response. His statement of the conditions that make enhancement more likely (than extinction) is a significant contribution to the literature of both conditioning and therapeutic procedures. However, Eysenck's explanation of enhancement effects in terms of differences in the properties of Type A and Type B conditioning can be called into question. (For a different view of resistance to extinction and possible enhancement effects, see Bindra 1976, ch. 12.)

Eysenck has missed the point, arising from the analysis of motivational mechanisms, that both appetitive and aversive motivational states are generated by a combination of incentive stimuli (e.g., food or a snake) and certain organismic conditions (e.g., deprivation state or an awake, nonsedated state). The organismic conditions are important because they serve as "gates" or limits within which particular incentive stimuli become effective generators of central motivational states. The difference between aversive and appetitive stimuli is not that the former generate "drive" and the latter do not, but that there is a wide range of organismic conditions under which the typical aversive stimuli (e.g., electric shock) can be effective and a relatively narrow range of organismic (deprivation) conditions under which the typical appetitive stimuli (e.g., food) can be effective. Further, viscerosomatic reactions (Eysenck's drive or reinforcing events) are produced not only by aversive CSs (e.g., adrenalin and changes in heart rate) but also by appetitive CSs (e.g., salivation and insulin secretion). Nor is there any fundamental difference between the effects of appetitive and aversive CSs on ongoing behavior (see Bindra 1974, 1976, ch. 9, 11, 12). Clearly, Eysenck's explanation of incubation and symptom persistence on the basis of special properties of Type B conditioning is inconsistent with the above considerations. Further, by making a false distinction between Type A and B (roughly appetitive and aversive motivational) CSs, and linking neurosis to the latter, Eysenck has essentially ruled that neuroses arise exclusively from aversive incentives and motivations, not from appetitive ones. In doing this, he ignores the variety of hysterical, compulsive, and character disorders that arise under conditions of little or no aversive incentive stimulation. Indeed, it has been suggested (Lipowski 1970) that the majority of neurotic disorders, at least of North American young men and women, arise from conflicts involving appetitive motivations rather than aversive motivations. (Of course, misery is as much a feature of "appetitive neuroses" as of aversive ones.)

Eysenck's paper is concerned not only with interpreting neurosis in terms of conditioning but also with revising conditioning principles in the light of what is known about the origin and treatment of neuroses. This is a welcome development, and it is to be hoped that his future revisions, taking into account the more recent developments in conditioning theory, will overcome some of the difficulties of his present models of neurosis and conditioning.

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The nonextinction of fear: operation bootstrap. A patient has some behavioral symptom. The therapist can see that this neurotic behavior is unrealistic and inappropriate in the sense that it fails to reflect the contingencies that apply in the patient's day to day world. For example, the patient may be defending against some nameless horror that poses no real threat and that warrants no defense. The question is: if the therapist can see this so clearly, why can't the patient see it too? Why does the patient's behavior resist control by the existing contingencies? Eysenck takes for granted the conventional answer to this question, namely, that back in the dark past of the individual there was a traumatic incident. It was not necessarily sexual, as Freud supposed, but it was painful, and that pain conditioned fear to CSs that happened to be present. And even to this day those CSs elicit fear. The patient has a clinical problem, the conventional answer continues, because those CSs keep recurring and the unrealistic fear does not extinguish.

Eysenck's primary concern here is to explain the nonextinction of fear. But I think that that is the least of his problems; I see the whole foundation of the model shaking. For one thing, one of the neurotic's problems, clinically, is not that the old CSs keep recurring, but that they keep changing. The symptom is triggered by ever-new events. The CS becomes generalized, verbalized, and symbolized. Where once the thing elicited fear, now the word or some remote association with the thing evokes fear. Symptoms grow (which is why patients go to therapists). Second, the US, the horrible thing that has to be defended against, keeps changing too. What started as a fear of rejection may end up as a fear of inadequacy or a fear of worthlessness. Anxiety can become depression, and vice versa. Again, the clinical problem is that symptoms grow, rather than that some fears persist. Often the anxious patient will have no idea of what he or she is afraid of; the US is inchoate. Furthermore, as Eysenck reminds us in this paper, fear-induced symptoms can grow without there ever having been a US. Thus, the phobic patient may have a very definite fear object, such as a snake, but the typical snake-phobe has never had a traumatic incident with a snake, and some snake-phobes have never seen a snake. The human organism seems "prepared," as they say, to fear certain animate objects as well as certain physical situations. Third, in the class of neuroses Eysenck is considering here, the strength of a symptom may bear little relation to the strength of the underlying fear. If the symptom is thought of as a kind of avoidance behavior, then there is little reason to expect its strength to reflect momentary levels of fear, because there is little evidence either from the laboratory or the clinic that the level of fear directly determines the strength of fear-related behavior. A person can be crazy as a fruitcake and yet be so well protected by various defenses that little of the initiating fear or the historically important fear is manifest. Once again, the clinical problem is that symptoms grow, not that fears persist.

Setting aside for the moment all these problems with a conditioning model of neurosis, Eysenck has posed an interesting academic question: why does fear sometimes incubate rather than extinguish? The laboratory evidence he cites is really impressive. Incubation does happen, and quite apart from whatever application it may have to the clinical situation, it is a phenomenon that requires an explanation. Eysenck's explanation of it is based on a bootstrap principle. The fear CR is maintained without a reinforcing US because the CR itself is unpleasant, just as the US would have been. The occurrence of the CR therefore reinforces the CS-CR association just as the occurrence of the US would have. In effect, the CR reinforces itself. Fear can raise its own level with its own bootstraps. The idea that fear can be a Pavlovian reinforcer provides a healthy and attractive alternative to the old adage that pain is the reinforcer for fear.

To limit the bootstrap effect, to prevent fear from going right through the roof, there are two decremental processes. One is what Eysenck calls normal extinction; some extinction of the CR always occurs, and it is only under certain circumstances that extinction is more than offset by the bootstrap mechanism. The second decremental process is something like habituation: the longer the CS remains on, the weaker the CR becomes. So, if the CS is short, then the CR may be strong enough to be self-reinforcing and generate an incubation effect. But if the CS is prolonged, then the CR will be too weak to reinforce itself, and all we get

is some extinction of fear. This second process is consistent with much that is known about avoidance extinction, but the case for fear extinction is not so clear. In many studies, when the avoidance response declines during extinction it is replaced by some other defensive behavior, such as freezing, which suggests that the fear is still strong. The first process, "normal" extinction, raises the tough question of what normal extinction is. Is it something other than the second process? Does it depend upon an inhibition mechanism? Or some kind of discrimination? At this point we cannot say.

The bootstrap principle may also have to be limited on motivational lines to prevent every CR from being affected by every other CR and US that comes along. If it is true that pain can reinforce fear, and that fear can reinforce fear, then we might wonder if fear can reinforce pain. Can fear reinforce guilt, or vice versa? And what about the happy emotions; does salivation inhibit fear, or vice versa? Can we think of such a mechanism providing the basis for counterconditioning and suppression, respectively? Eysenck tries to assure us that we need not worry about rampant bootstrapping occurring with all conditioning, but I find these assurances rather unconvincing. Thus, he says that fear is peculiar if not unique in supplying the drive for its own motivation. Salivation is not like that. But is salivation really so different? The occurrence of a salivary CR requires that the animal already be hungry, according to Pavlov, and the CS all by itself will not make the animal hungry. I grant that, but salivation is incentivelike if not drivelike (remember r_g?) and in that sense a source of motivation. In any case, it is apparent that we have yet to determine if bootstrapping only works with fear conditioning, or whether it is more widespread. It is also apparent that if we are to use conditioning theory as a useful model of neurosis, then conditioning theory is very much in need of conceptual clarification.

***by* T. D. Borkovec**

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Incubation and the relevance of functional CS exposure. Eysenck has presented the latest extension of the classical learning theory of neurosis, primarily via the addition of the concept of incubation. I have difficulty criticizing his overall approach, since I am in fundamental agreement with his purpose. Explanation of a phenomenon can occur at many different levels. At the level of scientific psychology, if a theory has been parsimonious and heuristic, it may be valuable not to abandon the theory but to gradually modify it in the simplest way to capture past empirical results and predict new events within its purview. However, the ultimate role of any theory is to generate useful bits of knowledge whose basic meaning will become apparent only after we discover some grander perspective on human nature and human experience. We are a long way from such a perspective. Eysenck's theory will serve the former purpose, however, because its elements relate to some rather fundamental aspects of the human condition.

Most important, the theory once again directs our attention to what remains the real challenge for anxiety theorists. In an evolutionary sense, organisms are required to learn to adjust to two exhaustive events if they are to survive: changing stimulation and repeated stimulation. Popular conceptions of the psychology of adjustment and its related research have emphasized adjustment to change, whereas basic research on habituation and extinction phenomena have focused on repetition. Obviously, rapid extinction of learned anxiety responses to unrealistically feared events is desirable for the species, and our frequent observation of extinction suggests the fundamental importance and pervasiveness of this process. What is significant is the fact that neurotic fears do not conform to our general expectation (the neurotic paradox). One may choose to seek elsewhere for the explanation, or one may attempt to identify the conditions under which repeated, unreinforced CS exposure results in decreases in fear (or in its failure to decrease), and to expand existing theory to include those conditions. Eysenck has very nicely reemphasized the challenge to those of us who choose the latter course. While evidence for actual enhancement of fear remains somewhat equivocal, circumstances that mitigate extinction appear to hold the key for solving the enigma of neurosis.

Assuming that extinction reflects a fundamental behavioral process, one confronts the critical question: what does the organism or environment do during $\overline{CS}$ that *prevents* the occurrence of this otherwise routine phenomenon? Mowrer's (1947) two-stage theory represented one classic attempt to answer this question. With the addition of incubation, Eysenck has identified two very reasonable environmental answers ($\overline{CS}$ duration and UCS intensity) and has pointed to personality variables for clues regarding organism behavior. Two comments are worth making here. First, any time parameter such as $\overline{CS}$ duration is important only in terms of the processes that are occurring during that period of time. Eysenck suggests CR decay, but other events (environmental or organismic) may also contribute, while all of these conditions probably share some as yet undefined, underlying principle. In a related matter, personality variables are important only in terms of their reflection of what the individual is doing (imaginally, conceptually, behaviorally, physiologically, etc.) during $\overline{CS}$ and how those activities relate to extinction and incubation outcomes. Thus, Eysenck's model has provided us with a heuristic conceptual framework within which we may elaborate the role of critical external and internal events that mitigate the extinction process.

Consider, for example, a notion labeled "functional CS exposure." As animal researchers or clinicians presenting feared CSs, we too casually assume that objective presentation of the CS results in a processing of that information (cf. Wilson and Davison 1971) or an engagement of relevant response systems on the part of the organism (cf. Lang 1977). However, we can be certain that the organism's response to the stimulus in this regard will occur somewhere along a continuum of functional stimulus impact. The assumption of an all-or-none dichotomy of response is inappropriate. Processes of perception, attention, and defensive response may all contribute to the impact of a given CS presentation at a given moment. Thus, what the organism is doing at those moments will very much influence the cumulative effects of repeated $\overline{CS}$. For several years, our own fear research has been guided by Eysenck's (1968) earlier theorizing and has examined the effects of various behaviors during $\overline{CS}$ on the retardation or facilitation of extinction. These effects appear to relate to the degree of functional $\overline{CS}$ exposure:

1. If subjects imagine an avoidance response during imaginal exposure to a feared situation, heart rate over exposure sessions remains elevated relative to equivalent amounts of exposure during desensitization or implosion. Thus, Mowrer's (1947) theory can be extended to simple cognitive (imaginal) events without loss in predictive capability.

2. Conditions which increase the likelihood of the subject's attending to the feared stimulus produce greater heart rate extinction with repetition than does the absence of such conditions. Thus far, both the presence of relaxation and of motivating, instructions, including positive expectational sets, have been demonstrated to produce such effects.

3. The extent to which subjects report an awareness of (attention to?) physiological cues, as measured by an Autonomic Perception Questionnaire (Mandler, Mandler, and Uviller 1958) has represented an individual difference variable which routinely interacts with numerous independent manipulations of CS exposure. Indeed, in one study (Stone and Borkovec 1975), a test of Eysenck's $\overline{CS}$ duration hypothesis found incubation according to his prediction only among subjects who reported high levels of awareness of physiological cues. Such an outcome makes sense in the context of Eysenck's current theory if one accepts his notion regarding the potential aversiveness of CR-produced proprioceptive cues and adds a dimension of individual difference with respect to responses of attending to such important components of the entire CS-CR complex.

The majority of nonhuman animals appear to be relatively free of neurotic anxiety except under laboratory conditions specially created by humans, whereas sizable numbers of people suffer from the problem. Such an observation would seem to support the role of cognitive factors in the maintenance of anxiety. However, these factors may require little elaboration beyond relatively simple processes of perception, attention, and avoidance behavior learned within those two systems, and I would agree with Eysenck that the usual laws of behavior can be applied without recourse to more complex cognitive theorizing. The objects, events, and concepts that we fear become avoided, may incubate upon periodic exposure, and produce distorted experience and expression of the behavior and affect of ourselves and others. Although both experimental and clinical experience suggest the likely importance of interpersonal factors in particular for understanding the majority of neuroses, Eysenck's latest theory provides the optimistic view that many of the basic processes underlying these events are simple, straightforward, and easily investigated in the scientific laboratory.

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The Gantt and Eysenck conditioning models for neurosis. In this paper, Eysenck has masterfully integrated much of the literature on neurosis and proposed a theory that will stimulate both controversy and research. But he has failed to mention some closely related ideas of Gantt, who, in turn, has generally ignored Eysenck. The first part of this commentary could thus be subtitled, "Two

remarkably similar theorists who hardly know each other." The second part of the commentary has to do with some of my reservations about both men's theories.

Eysenck should have acknowledged that Gantt developed, mainly out of his pioneering work on cardiovascular conditioning, three important principles of neurosis. Gantt, in fact, should be thought of as the originator of "Model 2 or 3" of neurosis and Eysenck the developer of "Model 4." Gantt's three principles are schizokinesis, autokinesis, and organ system responsibility. The first refers to the tendency for certain nonspecific (emotional) responses to condition far more rapidly (often in one trial) than more specific conditional responses such as salivation and motor flexion, and to outlast the latter in extinction. Schizokinesis, then, relates to Eysenck's Type B conditioning, the supposition that not all responses follow the same laws of conditioning. Autokinesis refers to the tendency for behavioral maladjustment or adjustment, once set in motion, to become self-perpetuating and relatively independent of feedback from the environment. As Gantt (1973c) puts it, autokinesis is "something the subject himself contributes, something novel, synthesized out of his experiences, of the traces that remain in the nervous system, and perhaps of functions peculiar to the nervous system of the individual." Gantt's autokinesis is Eysenck's incubation, and the term "incubation" may communicate the principle better. Eysenck has more clearly formulated incubation as a process antagonistic to extinction. But Gantt recognizes, and Eysenck does not, two possible kinds of "incubation," destructive and constructive. My own research (Dykman, Gantt, and Whitehorn 1956) was, I believe, influential in Gantt's recognition of the positive aspects of autokinesis.

The principle of organ system responsibility asserts that an organ cannot be conditioned to do what it is not innately equipped to do. For example, it is impossible, Gantt believes, to condition a diuretic response of the kidney in the absence of water load. There seems to be no comparable principle in Eysenck's theory, though some of the idiosyncracies of conditioning bespeak such a principle (see below). Gantt sees all of his principles as compatible with and not in opposition to Pavlovian theory.

Various conditioning theorists, including Eysenck, have attempted to deal with pain and anxiety in regard to neurosis and incubation, in particular. Pavlov simply did not discuss such matters subjectively; he referred to pain as a destructive stimulus. Cannon (1932) thought that the sensation of pain could be conditioned. "By experience agents which injure tissue and produce pain, become associated, so that our relations to them are conditioned by their effects." Gantt (1973a) argued that the sensation of pain is not conditioned, and more generally that no sensation of any unconditional reflex (or response) is ever conditioned. According to Gantt, the sensation of pain is not reported as a part of the conditional response to unavoidable shock. But once having been exposed to severe pain, the individual fears all stimuli that might trigger the sensation of pain. In describing his own trigeminal neuralgia, Gantt (1973a, p. 65) wrote: "The interesting point is that the signals for the stimulus, *viz*, walking, going into the cold, simulated touch to the sensitive areas, never result in the sensation of pain, only in the anxiety, and the movement of avoidance."

If the sensation (of pain or taste) were part of the conditional reflex, the response would, according to Gantt, never extinguish. And he asks, "What value is there in survival in having an unextinguishable sensation of pain with every learned action to avoid the pain?" Gantt cites the inability of sensation to become conditioned as an example of organ system responsibility. But Gantt overlooked the possibility that anxiety may be an exception to the rule that sensations are not a component of the conditional response. And even though anxiety is pathological in many instances, its conditionability has survival value.

The most innovative part of Eysenck's theory is, I believe, the notion that states, such as anxiety, are self-perpetuating and reinforcing. Both Eysenck and Gantt emphasize that conditional stimuli (once appropriately reinforced aversively) acquire signal value for danger and coming pain. Eysenck calls these internal sensations and their behavioral perturbations nocive responses. He goes a step further than Gantt, hypothesizing that nocive responses due to positive feedback explain incubation. And he makes explicit the point that the experience of fear and anxiety associated with bodily changes may not only prevent extinction but enhance pathology.

But why does anxiety extinguish in some persons and situations and not others? If we suppose anxiety to be an innate response elicited by threats to the organism's integrity, then perhaps the only way to extinguish anxiety is to remove or defuse the pain or threat that produces it; otherwise it will be, as Eysenck says, enhancing to behavioral pathology. This assures that anxiety will habituate but not extinguish because the stimuli eliciting anxiety produce both somatic-autonomic responses and the sensation of fear. In conditioning terms, anxiety could be conceived as an unconditional response tied to the threat of shock, and as such could be readily conditioned to various stimuli. To decrease anxiety, it would be necessary to reassure the animal that the shock will not reoccur or be painful if it does (a reinforcement of mildness).

I have criticized elsewhere Gantt's concepts of schizokinesis and autokinesis. My dissatisfaction with autokinesis is the emphasis the concept places upon spontaneous events occurring within the organism apart from everyday experiences (Dykman 1965). To Eysenck's credit he provides reasonable mechanisms for positive elaboration. I have suggested two bases for autokinesis (Dykman 1965, pp. 308–9): (a) disruption of nervous system timing mechanisms such that memory and reality are confused; (b) associations via evaluative (cognitive) mechanisms leading to indiscriminate generalization when there is high emotionality. A basic supposition is that in many life situations the threat of trauma may be more distressing than actual trauma.

Turning to matters other than incubation, Gestalt therapists, to mention one group, would disagree with the concept that neurotic behavior is paradoxical. Each of us consists of many parts in dynamic interrelation. Change is alien to our current sense of self; lifelong habit patterns, good or bad, are comfortable. According to Gestalt therapists, we hold on to phobias or obsessions because they help us to avoid unpleasant situations. Clients can give many good reasons for holding on to phobic behavior when forced to think about its value to them.

A final point is that Eysenck may be wrong in assuming that Pavlov adhered to a supposition that all conditional stimuli are equally effective. It is unlikely that Pavlov would have argued for differential effects of inhibitors (distractors), as he did (p. 45), and at the same time embrace a concept of equipotentiality. In discussing inhibitors, Pavlov pointed out that stimuli with survival value, such as the signal and sound of game for sporting dogs or rustling under the floor for others, are very powerful inhibitors of conditional responses (p. 45); that is, the concept of preparedness in an earlier guise. And Pavlov definitely recognized the more powerful effect of some conditional stimuli than others in delayed conditioning (p. 90).

To conclude, I reiterate that Eysenck has made an important theoretical contribution. He has put together a more explicit, integrated, testable theory of neurosis than has been heretofore accomplished. But theoretical issues remain, only some of which are amenable to a scientific solution.

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Is there any need for conditioning in Eysenck's conditioning model of neurosis? Eysenck suggests that existing explanations of neurosis in terms of conditioned responses are faulty, and he proposes a new explanation, but one that still uses the language of conditioning, to take their place. In this comment I shall ask, first, whether Eysenck has correctly identified the problem to which his paper was addressed; then whether his solution to the problem is acceptable; and, finally, whether there is an alternative solution.

I. *The problem.* Eysenck lists certain features of neurotic behaviour that, he claims, cannot be reconciled with existing conditioning theories of neurosis (the "standard" theory, as I shall call it). These features include the following:

1. The stimuli that elicit neurotic behaviour are not a random sample from the universe of possible stimuli; rather, a small group of stimuli (e.g., height, darkness, open or closed spaces, certain kinds of animals) are disproportionately represented.

2. Single-trial conditioning is sometimes reported in connection with the genesis of phobic fears, but single-trial conditioning is rare in the laboratory.

3. Neurotic responses are highly resistant to extinction, but unreinforced conditioned responses extinguish rapidly in laboratory studies.

4. "In many neuroses we not only fail to observe the expected extinction of the unreinforced CS, but we find an incremental (enhancement) effect, such that the unreinforced CS actually produces more and more anxiety (CR) with each presentation of the CS."

These arguments against the standard theory do not all have the same power. Weakest is (2), for the simple reason that single-trial conditioning, so far from being "rare in the laboratory," is widely used there, particularly in the study of memory. Argument (3) is also weak. As Eysenck himself notes, there have been several attempts to overcome the problem posed for the standard theory by the fact that laboratory avoidance behaviour and neurotic fears are both very resistant to extinction. Provided that one is prepared to accept that phobias include a component of instrumental avoidance behaviour, as well as a compo-

nent of Pavlovian conditioned fear, some of these attempts have been reasonably successful (Gray 1975, ch. 10), especially those making use of *Soltysik's* [q.v.] concept of conditioned inhibition of fear or the notion of safety signals. (Seligman and Johnstone's theory, regarded by Eysenck as the "most acceptable" of these attempts, contains nothing not already found in earlier, more "behaviourist" theories; it is merely couched in more fashionable, but less precise, "cognitive" terms.)

If we disregard arguments (2) and (3), we are left with (1) and (4) as substantial objections to the standard theory. To this we can add one argument that Eysenck did not use: (5) The onset of neurotic symptoms is not random with respect to time of life; rather, each syndrome (e.g., animal phobias, agoraphobia) has a characteristic peak age of onset (Marks 1969). The force of this argument is similar to that of (1), above. There is no reason to suppose that the conditioning of anxiety to a CS by associating it with an aversive UCS should be favoured by one age rather than another, any more than by one kind of CS rather than another. Thus neither (1) nor (5) is readily understood in terms of the standard theory. As to (4), this is in blatant contradiction, not only to theory, but also to hundreds of laboratory studies of classical conditioning in which the observed result of unreinforced presentation of a CS has invariably been extinction.

II. *Eysenck's solution to the problem.* Given that (1), (4), and (5) constitute serious obstacles to the standard theory, does Eysenck's new theory account for them adequately? As before, I shall pick out only the major features of his arguments.

Eysenck proposes that:

6. Certain CSs are biologically "prepared" (Seligman's term) to be more readily connected with anxiety than others.

7. Under certain conditions the presentation of a CS without reinforcement from the UCS may give rise to an increase in the strength of the CR ("incubation").

8. The conditions favouring incubation are, first, so-called Pavlovian B conditioning, that is, "a type of conditioning in which the CR is a drive"; second, a strong UCS; and third, a short duration of CS presentation.

It is then possible to account for (1) and (2) by reference to (6) and for (3) and (4) by reference to (7) and (8).

The trouble with these "explanations" is that they are circular. Only some stimuli are frequently found to elicit neurotic behaviour; so these stimuli are "prepared" to do so. Similarly, only some kinds of behaviour are peculiarly resistant to extinction; so these kinds of behaviour are subject to incubation. These moves would be truly explanatory only if independent criteria were offered by which one could detect prepared stimuli or those CRs that are subject to incubation. With regard to preparedness, no criteria are offered: all we can do is to list the stimuli that are frequently found to produce phobic reactions. With regard to incubation, criteria *are* offered, that is, those in (8) above; but when we examine these criteria closely, they too are found wanting.

The chief of these criteria is the requirement that the CR be a drive. Since the other two criteria in (8) can be applied to any CS-UCS combination, on their own they would allow *any* aversive UCS to be used to set up a neurotic reaction to any CS, thereby negating the very problem of selectivity that Eysenck emphasises. Thus we must ask what it means for a CR to be a "drive." Eysenck is very unclear on this point. This is doubly unfortunate, because "drive," especially when, as in the present case, it is entangled with "reinforcement," is probably the most confused and ambiguous concept in the whole of learning theory (Gray 1975), ch. 4).

Eysenck gives the following clues about the particular kinds of CR that constitute drives:

First, "the CS appears to act as a partial substitute for the UCS." But this is what Pavlov said about *all* conditioned reflexes, and, by and large, modern research has supported him on this point (Gray 1975, ch. 2; Gray, in press).

Second, "the UCS elicits the complete UCR in Pavlovian B conditioning whereas in Pavlovian A conditioning the organism emits the UCR of approaching and ingesting the food." It is very difficult to see what this distinction means. If an electric shock is used as UCS and the animal is free to move, it will undoubtedly attempt to run away from the place where it is shocked: does it not "emit" this UCR just as much as the UCR of approaching a place where it is fed? Conversely, it is possible to squirt water directly into the mouth (e.g., DeBold, Miller, and Jensen 1965): does this elimination of "emitted" locomotor behaviour convert Pavlovian A into Pavlovian B conditioning?

Third, "in Pavlovian B conditioning the UCS produces the drive, and hence the drive properties of the UCR can be transferred to the CR." But what does it mean to say that a UCS "produces a drive?" In terms of the distinctions between different senses of drive that I have drawn elsewhere (Gray 1975, ch. 4), Eysenck can only mean drive as "goal-directed behaviour." In the case he has in mind the goal would be the removal of an aversive UCS. And "transfer of the drive-properties of the UCR to the CR" then presumably means that the CS acquires (secondary negative) reinforcing properties similar to those possessed by the UCS. But this much is common ground to the standard theory, as used for example by Miller or Mowrer, and to Eysenck's new theory. Nor does it offer a useful distinction between the so-called A and B types of Pavlovian conditioning. For the food UCS used in salivary conditioning experiments (regarded by Eysenck as paradigmatic for Pavlovian A conditioning) also elicits goal-directed behaviour, as do CSs that have been paired with such a UCS.

Fourth, "in Pavlovian A conditioning the drive [e.g., hunger] is already present when the experiment begins;" whereas there is no drive in Pavlovian B conditioning until the UCS is presented (as indicated in the previous paragraph). This point is probably correct. But it is an argument that applies to *all* aversive UCSs. Thus incubation, not extinction, should be the norm in experiments using aversive UCSs. And this, of course, is not so; if it were, the discrepancy between laboratory experiments and clinical observations identified by Eysenck would not exist.

At the end of the day, therefore, both the preparedness and the incubation arguments are, it seems, irredeemably circular. Even if they were not, Eysenck's theory encounters major empirical difficulties. First, the data purporting to establish the phenomenon of incubation (which constitutes both part of the problem posed by Eysenck for the standard theory, and the cornerstone of his new theory) are by no means convincing (Bersh, in press). Second, the emphasis placed by Eysenck on the response (both UCR and CR) as the determining factor in conditioning is contradicted by much evidence that this plays at best a minor role in the formation of conditioned reflexes (Mackintosh 1974; Bersh, in press). Third (though not last: see Bersh, in press), Eysenck's emphasis on the response is also belied by the considerable amount of data accumulated in recent years (e.g., Kamin 1969, Rescorla and Wagner 1972) demonstrating the critical role played by stimulus-stimulus contingencies in classical conditioning.

We must conclude, therefore, that Eysenck's new theory fails both logically and empirically to solve the problem that he identifies.

III. *An alternative solution.* At this point it is worth asking what conditioning theory is *for*. Pavlov felt impelled to cross the great divide between the unconditioned and the conditioned reflex (Gray, in press) when he observed, in the phrase cited by Eysenck, that "any natural phenomenon chosen at will may be converted into a conditioned stimulus" (Pavlov 1928, p. 86). Had the salivary reflex been elicited only by tastes or smells closely related to nutritious substances, there would have been no need to take this step. There is a mass of data demonstrating that Pavlov was right: a host of reflexes have been conditioned to arbitrarily chosen lights, tones, metronomes, and whirligigs to which they have no conceivable biological relationship. To set against this, proponents of the fashionable hypothesis of preparedness can call on a handful of observations made under very special conditions. Nor is arbitrariness confined to the laboratory. Eysenck (1977b) himself describes a patient in whom sexual impotence was produced by exposure to a particular pattern of wallpaper which had once been the silent, but effective, witness of a thrashing he had received from an irate husband. It is to account for such findings as these that conditioning theory is needed. If *all* phobias were of heights, open or closed spaces, snakes, and so on (Marks 1969), there would be no need to call on conditioning theory at all: one would simply describe them as *innate* fears.

But why can we not do this in any case? Eysenck's problem only arises if we treat all phobias as identical in mechanism and cram wallpaper and snake phobias into the same suitcase. But rather than create a whole new variant of conditioning theory, it is much simpler to suppose (Gray 1971) that *some* fears and phobias are innate and *some* are conditioned. On this view, the ones that are conditioned are in no important way different from the conditioning phenomena studied in the laboratory; we need call on neither preparedness nor incubation to explain them. As to the ones that are innate (and these may well include the great bulk of clinically important phobias), to call them so offers by itself no fuller explanation than talk of preparedness, and the like. But it is a much simpler starting point from which to seek a more detailed account, it aligns phobias with other phenomena also known to be innate, and it calls on no new principles such as "incubation." In addition, it accounts neatly for point (5) above, that is, the fact that neurotic syndromes have characteristic ages of onset (Marks 1969). Neither

the old nor the new conditioning theory can easily do this, but there are many well attested examples of innate responses that require maturation to a particular stage of development before they become manifest.

There will be hostility to this account on the grounds of parsimony: it would be more satisfying to account for all neurotic reactions in essentially the same way rather than to divide them into two mutually exclusive classes. This is a hostility that I share. But there is a middle way, which preserves the distinction between innate and conditioned neurotic reactions and yet gives them a final common pathway. I have described this middle way in some detail in several publications (Gray 1971, 1975, 1976, 1978); here I shall simply indicate how it can be applied to Eysenck's problem.

The core of the approach is to suppose that anxiety consists of activity in a "behavioural inhibition system" (BIS). The BIS is activated by certain classes of stimuli to produce three major types of behavioural change: inhibition of ongoing behaviour, increased level of arousal, and increased attention to the environment. This is not the place to consider how these behavioural changes combine to produce neurotic reactions. The important issue for our present purpose is the nature of the stimuli adequate to activate the BIS. I have identified these principally as conditioned stimuli associated with punishment and conditioned stimuli associated with frustrative nonreward. This formulation stresses the fact that the BIS responds to *conditioned* stimuli. But I have also proposed that the BIS is an *innate* response system. These two features of the model may be encapsulated by saying that animals (including man) come equipped with an *innate* response to stimuli that have been followed by unconditioned punishing or frustrating events, and they only need to learn (probably by classical conditioning; but see Gray, Rawlins, and Feldon, in press) *which* originally neutral stimuli they should respond to (Gray 1975, p. 249)

It is now an easy move to add that the BIS can also be activated by certain classes of stimuli for which no conditioning is necessary at all. I have made this move before, by adding novel stimuli to the list (Gray 1975, pp. 352–54; 1977), as well as stimuli that occur during social interaction (Gray 1976); the latter are of very great importance for both normal (Gray 1971) and neurotic (Marks 1969) fears. The list can be extended further by including Seligman's (1971) "prepared stimuli," which I have more ponderously called "stimuli characteristic of special evolutionary dangers" (Gray 1971).

In this way, one can suppose that phobias of heights, snakes, closed spaces, and the like are mediated by the same mechanism that produces other fears, whether innate (social phobia, fear of strange environments) or conditioned (wallpaper).

But must we not pay the same price as Eysenck for parsimony regained? Is this ever-extending list more than a sheet to cover ignorance? Certainly, the danger of circularity remains real. It is not obvious that the description of phobic stimuli as those that activate the BIS is any more empirical than their description as those that are prepared, or subject to incubation. In defence of my alternative solution, beyond the points already made, I offer two last arguments.

First, the inclusion of novelty in the list of adequate stimuli for anxiety at once aligns the two processes described by Eysenck as being elicited by CS-only presentation – incubation and extinction – with similar processes known to be elicited by novel stimuli: sensitisation and habituation (Horn and Hinde 1970). This has the great advantage over Eysenck's position that the interplay between these two incremental and decremental processes has already been well studied in the laboratory, and that we no longer need to call on the wholly new principle of incubation. Furthermore, notions of habituation and sensitisation have already been applied to the experimental analysis of behaviour therapy with some success (Watts 1971). Finally, the most important advantage of Eysenck's approach – its capacity to deal with the variable of CS exposure duration as this affects the outcome of behaviour therapy – is preserved; indeed, it is embedded in a much better understood theoretical context.

Second, considerable progress has already been made in investigations of the physiology of the BIS. Work in my own laboratory has concentrated on reactions to stimuli associated with frustrative nonreward or with punishment (Gray 1977, 1978; Gray, Feldon, Rawlins, Owen, and McNaughton 1978). What is striking is that the brain systems apparently involved in these reactions have been independently implicated by Vinogradova and Brazhnik (1978) in the sensitisation and habituation of orienting responses to novel stimuli (Elliott and Whelen 1978, pp. 294 and 418). Thus the convergence for which I have argued above on psychological grounds appears to have a sound physiological basis.

In sum, while it is clear that Eysenck has identified a real problem, his new theory, calling as it does on the quite new principle of incubation, offers a less promising line of attack on this problem than that afforded by a combination of more familiar principles.

***by* H. D. Kimmel**

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Eysenck's model of neurotigenesis. For some twenty or more years, H. J. Eysenck (1960) has been an ardent advocate of a conditioning explanation for the etiology of neurotic behavior. His earlier attempts to place the origin of psychoneurosis in the individual's conditioning history suffered from being too vague and were criticized accordingly (Kimmel 1971). Unfortunately, his present effort to respond to some of the criticisms has fallen short of the mark.

In his earlier approach, Eysenck asserted that neurotic symptoms consist of learned patterns of behavior "which for some reason or other are inadaptive" (1960). But the learning mechanism on the basis of which these behaviors were acquired is, presumably, adaptive. That an adaptive mechanism can be the basis for the acquisition of maladaptive behavior patterns is, of course, what Mowrer (1950) called the "neurotic paradox," and little is gained by Eysenck's assertion that learning is involved in neurotigenesis unless the factors responsible for etiological differentiation between adaptive and maladaptive outcomes are made clear. In his earlier treatment of this question, Eysenck assigned significant roles to chance and environmental hazards, but nothing more specific was proposed.

In my criticism of Eysenck's earlier attempt to use conditioning principles to explain neurotic behavior (Kimmel 1971), I emphasized the fact that conditioned fear, the keystone of Eysenck's conditioning theory of neurosis, cannot be itself explain neurotic anxiety or phobias, since these are known to be highly persistent while conditioned fear is easily extinguished. In that paper I suggested that a solution to this problem might be found in the recently discovered fact that autonomic nervous system processes can be modified by instrumental conditioning (Kimmel 1974). Since that time I have elaborated on this proposal and have attempted to deal directly with the persistence problem via a theory of adventitious self-reinforcement of emotional reactions (Kimmel 1975).

In his revised conditioning model of neurosis, Eysenck joins me in criticizing traditional conditioning approaches on the grounds that conditioned fear is too easily extinguished to provide a firm basis for building a conditioning theory of neurosis. He also criticizes earlier conditioning approaches that fail to deal with the issue of "preparedness" to learn (Seligman 1970), although it would appear that the main implication of Seligman's arguments is that conditioning principles may be somewhat more narrowly applicable than was previously assumed.

Eysenck proposes to deal with these problems in his own conditioning model by adopting two concepts, first that the conditioned fear response can come to reinforce itself (i.e., substitute for an omitted unconditioned stimulus), since the experience of fear is an aversive condition in its own right (cf. Kimmel 1963), and second that fear incubation rather than extinction can take place when the shock or other primary aversive unconditioned stimulus is omitted and the conditioned stimulus is presented alone. Eysenck attempts to meet the requirement that a basis for differentiating between neurotic and nonneurotic outcomes must be provided by proposing that the strength of the conditioned response and the duration of exposure to the nonreinforced conditioned stimulus are the critical factors in determining whether the conditioned response will reinforce itself and result in persistent neurotic anxiety or will simply extinguish.

According to Eysenck, incubation is more likely to follow conditioning with a strong unconditioned stimulus than with a weaker one. Furthermore, incubation is more likely with short nonreinforced presentations of the conditioned stimulus than with longer presentations of the nonreinforced conditioned stimulus. The reason the strength of the unconditioned stimulus is an important factor, according to Eysenck, is that it determines how strong the conditioned response will be. The stronger the unconditioned stimulus, the stronger the conditioned response. At the same time, however, duration of the reinforced conditioned stimulus is important because the strength of the conditioned response declines over time. Thus, for each intensity of unconditioned stimulus there should be a duration of presentation of the nonreinforced conditioned stimulus that is optimal for fostering incubation. Any duration longer than this critical one will increase the likelihood of extinction.

Eysenck's theory suffers from several problems. First, there is very little hard evidence in support of his assertions about incubation following conditioning. He places great emphasis on a study by Napalkov (1963) which purported to show that a conditioned stimulus paired only once with the unconditioned stimulus and presented alone thereafter demonstrated incubation in the sense that it elicited blood pressure changes of increasing magnitude over trials. Unconditioned blood

pressure reactions to an unpaired unconditioned stimulus tended to habituate in about 25 trials. No subjects were given more than one paired trial prior to testing with the conditioned stimulus alone, so that it is impossible to know what would have happened with more training. Furthermore, unpaired presentations of the conditioned and unconditioned stimuli were not administered as a control procedure, so that one cannot even conclude from Napalkov's study that its result was due to an associative process. Another problem is that Eysenck tends to rely on quite arbitrarily selected features of clinical behavior therapy situations to find support for his theoretical arguments, shifting abruptly in his discussion of his model from Napalkov's laboratory findings, to desensitization and flooding, and back again to the laboratory study. It is difficult to avoid the feeling that less emphasis would be given to selected clinical observations had there been more laboratory evidence to cite.

The fact is that the evidence is quite slim even on the two basic assumptions made by Eysenck. First, extinction is known to be quite rapid with standard presentations of the conditioned stimulus alone following human electrodermal conditioning even with very intense shocks as unconditioned stimuli (Kimmel, Kimmel, and Silver 1969). In that study extinction was if anything more rapid with a stronger shock than with a weaker one. It is simply not true that a strong unconditioned stimulus will routinely result in incubation instead of extinction. Kimmel, Kimmel, and Silver (1969) did find greater spontaneous recovery two months following extinction in the group that was conditioned with the strong shock than in the weak shock group, but this was primarily a result of the fact that the strong shock group had extinguished to a lower level than the weak shock group. Probably more serious is that fact that there is almost no evidence of incubation under any circumstances. The Napalkov (1963) study cited by Eysenck has never been replicated and cannot be made to serve as the principle foundation for his theory.

Besides these rather serious criticisms of Eysenck's theory, it must be noted that it is put together in a somewhat haphazard manner and that it assembles arguments and evidence in a most arbitrary fashion from every conceivable source.

Even with all of this negative commentary, it must be said that Eysenck's efforts deserve great credit. No one ever expected the task of explaining the etiology of neurotic behavior to be simple. The entire field of conditioning is undergoing a transformation at present, and this is another reason why the working out of a genuinely effective conditioning model of the etiology of psychoneurotic behavior is bound to be difficult. But there is little reason to doubt that behavioristic approaches such as Eysenck's will in the long run provide the solution to the neurotic puzzle that has intrigued all of us.

***by* Leonard Krasner**

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Eysenck on Watson: paying lip service to lip service. Eysenck offers an exciting reconceptualization of conditioning theory which may well represent a major advance in theorizing about atypical human behaviors. The basis of the Eysenckian model is the "conditioning theory" first formulated by Watson and the "many criticisms" of Watson's work.

These criticisms stimulated this reviewer to investigate Watson's own verbalizations of his theories of conditioning and "mental illness." Thanks to Cedric Larson, who made these papers available, I perused several of Watson's writings on "mental disease" (Watson 1916, 1924, 1928; see also Larson 1969).

We were particularly intrigued by Eysenck's accusation that Watson was "paying lip service to genetics, just as Freud and Skinner have done." Eysenck quotes one sentence from Watson and Rayner (1920) which refers to individuals who are "constitutionally inferior." Eysenck then contends that this one sentence "goes counter to his [Watson's] insistence in his major books on the absolute supremacy of environment and the absence of genetic causes in differentiating human behaviour."

After reading Eysenck's evaluation of Watson it was with a high degree of expectancy that this environmentally biased reviewer approached Watson's own writings. Could it really be that Watson was indeed the true "environmentalist," as he was being labeled?

Alas, Watson's papers are replete with sensitivity to the importance of biological factors in their interaction with learned behavior. "The separation between hereditary reaction modes and acquired reaction modes can thus never be made absolute" (Watson 1924, p. 214). "An emotion is an hereditary 'pattern-reaction'" (Watson 1924, p. 215). In another passage Watson (1916, p. 596) refers to "hereditary mechanisms" which excite neural areas as a reaction to an emotionally exciting stimulus. In a fascinating chapter sardonically labeled "The unconscious of the behaviorist" Watson (1928, p. 103) refers to the kind of assessment material needed to enable "the physician to make a diagnosis of the genetic factors which have been operative in producing the deviations he observes" in a mentally disturbed patient.

The game of taking sentences out of context could go on and on. The point is that charges of "lip service" seem unfair to Watson, as well as to Freud and Skinner. The phrase "lip service" somehow seems inappropriate in a carefully written, closely reasoned, scholarly, and scientifically sound paper such as Eysenck's.

Speaking of awkward phrasing, particularly by a behaviourist, Eysenck refers to Bandura as having recently "changed his mind." But reaction to and interpretation of that observation could well be the basis of another paper at another time.

***by* Donald J. Levis**

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A reconsideration of Eysenck's conditioning model of neurosis. Eysenck offers a conditioning theory of neurosis that incorporates principles of learning, genetics, and individual differences into one viable model. This theoretical offering was found to be comprehensive, stimulating, and worthy of serious consideration. This commentator is in full agreement with the strategy of conceptualizing neurosis from a conditioning viewpoint. However, disagreement exists over whether Mowrer's two-factor avoidance theory has been exposed to damaging criticism and whether conditioning theory of neurosis needs, at present, to incorporate concepts like preparedness and incubation. Let us review Eysenck's criticisms of existing conditioning theory. They can be divided into issues requiring the concept of "preparedness" and those requiring the concept of "incubation."

Arguments for the concept of preparedness: 1. *Failure to replicate Watson and Rayner's (1920) reported conditioning effect – little Albert.* Eysenck's central point here is that English (1929) and Bregman (1934) reported failures to replicate the conditioning effect noted in the Watson and Rayner (1920) study, which are damaging to a conditioning theory of neurosis. Further, he argues that the failure to replicate may have been tied to the use of "nonprepared" CSs such as common household goods. Watson and Rayner's use of a furry animal as a CS was viewed by Eysenck as critical since it fits a "prepared" or genetically based stimulus classification.

The theoretical importance attributed to the above cases is surprising in that Watson and Rayner did not necessarily demonstrate that they had created a neurosis, only that they had successfully produced fear and avoidance of the CS. Numerous published, controlled studies already exist illustrating that aversive conditioning of infants, children, and adults is possible, and most of these used "nonprepared" CSs (for a review of the human studies using traumatic shock see Turner and Solomon 1962). Furthermore, a critical review of the English and Bregman studies questions the validity of Eysenck's conclusions.

In English's report three cases were discussed. The first replicated Watson and Rayner's finding using a black stuffed cat as a CS. The second failed to produce a conditioning effect when a nonprepared CS was used. However, the UCS, striking a large metal bar, did not elicit any fear, either to the CS or to the situational cues. Without an effective UCS, how can conditioning be expected, no matter what the CS is? The third case appears to be contrary to Eysenck's position in that a common household shoe, a nonprepared stimulus, was conditioned when paired with another shoe that evoked fear and avoidance.

Bregman's study also appears problematic. Fifteen children (8–16 months old) were tested with a set of six, nonprepared CSs to either a negative (loud bell) or positive (rattle or melody) UCS using a within-subject design. No indications of differential responding to the CSs were obtained. Although UCS effectiveness was established, Bregman failed to determine whether the CSs used were discriminable by the infants tested. Furthermore, acquisition and extinction trials were mixed, and so few CS-UCS pairings were given that it is doubtful whether sufficient differential-training could have occurred.

2. *Equipotentiality and the concept of preparedness.* Eysenck argues that the concept of equipotentiality, in which one CS is viewed as being as good as another, is central to a Pavlovian or Watsonian conditioning interpretation and is currently not supported by the literature. The notion of equipotentiality is believed to be weakened by the "taste-aversion" studies (Garcia, McGovan, and Green 1971) and by the clinical observation that phobias comprise a limited set of

objects such as fear of specific animals and insects, heights, and dark. In Seligman's (1971, p. 312) words: "And only rarely, if ever, do we have pyjama phobias, grass phobias, electric-outlet phobias, hammer phobias, even though these things are likely to be associated with trauma in our world."

As a major improvement to a conditioning theory of neurosis, Eysenck suggests the incorporation of Seligman's (1970, 1971) hypothesis of "preparedness." Basically, this concept suggests that the most frequently experienced phobic fears are attached to situations that threatened the survival of our ancestors, and that there is a genetic predisposition or preparedness for acquiring these fears. From this analysis, prepared stimuli (fear of dark, snakes, etc.) are more readily acquired and more resistant to extinction than unprepared stimuli (e.g., tones, shoes, clothes, etc.).

The addition to conditioning theory of a concept like preparedness may be required but with our current state of knowledge it seems premature. It should be noted that Seligman's hypothesis is not unlike Jung's concept of archetypes, which was scientifically disregarded because it was untestable. It should also be noted that the concept of equipotentiality only applies when everything else is held equal. For example, contemporary conditioning theory has readily established that nonreinforced preexposure to a CS significantly interferes with subsequent conditioning to that stimulus (Mackintosh 1974, p. 37). This finding is frequently referred to as latent inhibition and may well explain why certain phobic stimului are more easily conditioned than others.

Consider Eysenck's and Seligman's concern that phobias of electric outlets and hammers are rare despite the fact such objects may well have been a source of aversive conditioning. First of all, such stimuli are frequently exposed throughout life in nonaversive settings. Not only do any past conditioning effects to these stimuli have a chance to undergo extinction and latent inhibition effects, but early discrimination training is given in how to use these objects properly to avoid danger. Alternatively, the sensory consequence of common phobias that are characterized as prepared (stimuli involved in fear of snakes, insects, and heights) are more readily avoided, reducing CS exposure and exposure to other stored cues in memory that may be part of the total CS complex (Stampfl and Levis 1969; Levis and Hare 1977). Thus two factors must be considered when comparing the conditionability of prepared and nonprepared stimuli: (1) the preconditioning aversive level of each stimulus, and (2) the degree of latent inhibition from nonreinforced preexposure.

None of the studies cited by Eysenck in support of preparedness theory considered the above developmental issues by equating the stimuli involved. Furthermore, the Ohman studies referred to by Eysenck failed to show the acquisition differences between prepared and nonprepared stimuli predicted by preparedness theory. Eysenck also failed to quote damaging negative evidence conducted at his facility by Rachman and Seligman (1976) and de Silva, Rachman, and Seligman (1977). The latter study is particularly relevant. These authors conducted a retrospective study on a large number of phobic and obsessive cases. They failed to find a systematic relationship between evolutionary criteria of preparedness and either acquisition rate or therapeutic outcome.

On the other hand, the taste-aversion phenomenon clearly presents some interpretive difficulties for traditional conditioning theory. However, a critical review of this area, which space does not allow, confirms the contention that a genetic interpretation of the results is not directly supported. Not only has Bitterman (1975, 1976) raised important methodological criticisms of research in this area, but there has been a failure to consider the developmental history of the stimuli used, which may prove to be a critical variable.[1]

3 & 4. *Single-trial conditioning and the issue of CS-UCS time relations.* Eysenck noted that single-trial conditioning is sometimes reported in connection with the genesis of phobic fear. He maintains that single-trial conditioning is very rare in the laboratory, suggesting that phobic conditioning is enhanced because of prepared stimuli. This commentator takes issue with the conclusion that single-trial conditioning is rare. If the avoidance situation (active or passive) is made highly discriminable between safe and noxious situations, learning is very rapid using nonprepared CSs. For example, to reach a criterion of ten consecutive avoidances in a one-way situation the mean number of shock trials required is two or three, with many subjects learning after one (Levis 1970, 1971; Levis, Bouska, Eron, and McIlhon 1970). The above findings are not deemed unusual by researchers in the area.

Concerning CS-UCS time relationship problems, Eysenck concludes that the precision of having the CS precede the UCS by 500 msec. is unattainable in real life situations. This is a minor issue but it is hard for this reviewer to imagine a conditioning event in real life where some part of the CS does not precede the UCS by 500 msec. The issue of the critical time period for direct conditioning is confused with the issue of generalization of this effect across the CS and to other similar stimuli.

Arguments for the concept of incubation. The second set of criticisms offered by Eysenck is designed to illustrate the weakness of Mowrer's (1947, 1960a) two-factor avoidance theory. The purpose of such criticism by Eysenck is to justify the reformulation of Mowrer's theory by incorporating the concept of incubation elicited by short nonreinforced CS exposures. Four criticisms are offered, but only the first two are really central to the discussion.

5 & 6. *The issue of extreme resistance to extinction and CS enhancement.* Eysenck correctly notes that neurotic behavior is frequently highly resistant to extinction. Furthermore, laboratory evidence and clinical observation suggest that under certain circumstances unreinforced CS exposure results in an increase rather than a decrease in anxiety as might be expected by the law of extinction. Both of these findings require some modification of Mowrer's (1947) original version of two-factor theory.

These two issues have been addressed in the confines of two-factor theory by Stampfl and Levis (1967, 1969, 1976) who offer an alternative conditioning theory of neurosis which Eysenck failed to review. The model, which conceptualizes all human symptoms as avoidance behavior, extends Solomon and Wynne's (1954) conservation of anxiety principle to complex, serial ordered CS patterns hypothesized to be responsible for symptom maintenance. Space limitation precludes a review of this model here except to note that extreme resistance to extinction is a function of short-latency avoidance responses preventing the extinction of fear to longer CS exposures and eliminating the ability of longer CS exposures to recondition short latency responses. Thus the secondary intermittent conditioning effect produced by longer exposures to the CS is believed responsible for enhancing avoidance maintenance. The enhancement of fear to the CS in extinction (incubation) is explained by an increase in exposure to the nonextinguished part of the CS chain which can be dramatic especially if serial CS cues are used (see Levis and Hare 1977 and Levis and Boyd 1979 for a recent review).

7 & 8. *The issues of traumatic UCS and pain.* Of Eysenck's two remaining criticisms, the first is concerned with the establishment of extreme resistance to extinction without the use of a traumatic UCS. This has already been achieved in the laboratory. Brush's (1957) research demonstrated that the extreme resistance to extinction produced in Solomon's series of studies was not due to the traumatic shock used, and Levis (1966; Levis et al. 1970; Levis and Boyd 1979) has repeatedly obtained the effect with moderate shock intensities. Eysenck's last point concerning the issue of whether pain is attached to the UCR is really not germane to the discussion. However, this commentator was surprised that Eysenck considers frustration as "mental pain" (why not hunger pain?) which goes against the main theoretical account of this phenomenon (Amsel 1958).

In closing, it should be noted that Eysenck does acknowledge the similarity of his incubation notion with Solomon and Wynne's findings. He correctly suggests that a test of his position should be confined to a classical conditioning situation where CS duration can be controlled precisely. The critical issue is whether nonreinforced CS presentation results in an increase in anxiety over trials with CS duration held constant. Many of the studies Eysenck cites in support of his position have not met this control requirement. Unfortunately, Eysenck has a tendency in his citations not to discriminate poorly controlled from well-controlled research. An example involves the Napalkov (1963) study which Eysenck has labeled the "Napalkov effect" and discusses at length as supporting his viewpoint.

Napalkov reported that, in dogs, following a single conditioning trial, repeated administration of the CS brought about increases in blood pressures of 30-40 mm which, with repetition, increased to 190–230 mm. In some cases, this hypertensive state apparently lasted over a year. Given our current state of knowledge, such an effect is incredible to say the least and is very difficult to reconcile with the existing literature. It is difficult to see how these data can be given much weight since Napalkov only provided a one paragraph summary of his work and failed to cite a primary source of reference. Not knowing exactly what experimental procedures were used makes the study suspect since procedural artifacts can easily occur with this kind of measurement. Further, over fifteen years have passed, and apparently no replication of the effect has been reported. Until this phenomenon is replicated and presented in a detailed manner so a scientific evaluation can be made, one can only conclude that the effect is either nonexistent or procedurally artifactual.

Despite the above criticism, this commentator would like to reaffirm that

Eysenck has made an important theoretical contribution to the issues raised. Further research on these topics is clearly warranted.

NOTE

1. For a comprehensive review of the above and related issues, the reader is referred to an excellent unpublished paper, entitled "Hereditary determinants of fears and phobias; a critical review," by Dr. Dennis J. Delprato of Eastern Michigan University. It was found useful in preparing this commentary.

by **William Lyons**

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On some key concepts in Eysenck's conditioning theory of neurosis.

1. The notion of "preparedness." This is a most interesting concept, though I am not certain how informative it really is. Eysenck employs the notion of preparedness as part of his explanation of the neurotic paradox or the persistence of phobic or anxiety reaction to repeated presentations of some CS even though this CS has only once been paired with a negative UCS.

Preparedness is employed to explain three important sets of phenomena:

i. that humans are prone to phobias or conditioned fears;

ii. that such phobias are confined to a quite definite set of objects or stimuli, for example, spiders and rats (but not flowers or mushrooms);

iii. that such phobias are resistant to extinction.

But how informative or explanatory of these phenomena is this concept of preparedness? Suppose I am looking for an explanation of, say, the following three sets of phenomena:

a. that humans are prone to cancer;

b. that such cancers are connected with certain factors (e.g. smoking tobacco, ingesting certain chemicals) but not others (e.g. eating potatoes or chocolate);

c. that such cancers are resistant to extinction.

Am I really explaining anything if I say that the explanation is that humans are "prepared" for cancer? Cancer researchers would not be satisfied with such an explanation. They would say that to predict and cure cancers, they need to be able to isolate something tangible in humans that interacts with the carcinogenic agents. They look for physico-chemical factors in humans – *some* humans (those with cancer) – that interact with these agents.

In Eysenck's account, preparedness seems to be just shorthand for "prone to phenomena types (i), (ii), and (iii), above," and not any sort of explanation of the occurrence of these phenomena. But this can be seen more clearly from another angle. Preparedness does not explain why *some* humans, but not others, are prone to neuroses, yet this is what Eysenck sets out to explain. Indeed if we take preparedness as it stands as an explanation, then it would follow that we should *all* be neurotics. For the only further light that is shed on the concept of preparedness consists of the comments about how such phobic fears – as those of spiders, rats and the like – can be seen as "extemely useful to the individuals and *species* concerned"(italics mine). Thus, on Eysenck's account, we are all prepared in this way and, moreover, it is evolutionarily useful to be so.

The upshot is, it seems to me, that preparedness is just a shorthand or umbrella term for the phenomena whose appearance in some humans it is designed to explain. It turns out, then, not to be an explanation of the explananda but is (covertly) the explananda themselves in brief. To explain such explananda by means of the notion of preparedness is thus like explaining sleep in terms of "dormative power."

2. The notion of "incubation." This notion is also extremely interesting. It is designed to explain a fourth phenomenon associated with the neurotic paradox, namely

iv. enhancement, or the phenomenon of phobias increasing in strength over time even though the negative UCS is not presented (that is, increasing in strength when one would expect diminution and extinction).

Now Eysenck's use of this notion is certainly a genuine explanation this time and not shorthand for the phenomena it sets out to explain. "Incubation" does not just mean enhancement; it is an explanation of it in terms of a feedback mechanism. On Eysenck's account the neurotic person's reactions themselves become the stimulus for further such reactions. The neurotic person is fearful of fear itself, and this is why his neurotic fear increases in strength.

My worry here is that his feedback mechanism may not do the trick. Let us say that I have a traumatic experience in some open space, such that the open space (CS) becomes associated with the trauma (UCS-UCR). Now, when I am next in an open space, I again react fearfully because I associate the open space with my previous trauma. But since no trauma actually occurs, one would expect the fear reaction to be diminished. The reason why, with neurotics, this fear is not diminished but increased, suggests Eysenck, is that the neurotic is reacting to his own fear. But, and here is my worry, if the neurotic's fear as regards open spaces is diminished – and Eysenck gives us no reason to suggest that the first showing of this first order fear when there is no UCS will not diminish in relation to the original traumatic reaction – then it would seem to follow on Eysenck's model that the second-order fear (the fear of fear) should also diminish in parallel with the first-order one. In short, Eysenck's model does not seem to me to give him the result he desires, namely an account of how the neurotic's fear increases.

I also have a doubt about whether it makes much sense to postulate second-order fears in the face of no direct evidence that we have them. Eysenck does not bring forward any evidence that neurotics *say* that what they fear is their own fear, nor would it be easy – on his account – to divine from their behaviour that this is the object of their fear. For Eysenck's position seems to be that this second-order fear is fear of fear-of-open-spaces (that is, fear associated in a roundabout way with open spaces) and so should reveal itself in avoidance of open spaces just as a direct fear of open spaces would.

This means that the notion of "incubation" as a sort of feedback stands or falls by its power to make sense of enhancement and by its a priori plausibility, for there seems to be no possibility of gaining direct empirical evidence of this second-order fear. I have already aired my doubts about its ability to explain enhancement, so now I will air my doubts about its plausibility per se. As Eysenck himself has suggested, what is common to phobic objects is that, at least in our evolutionary past, they were *associated with danger*. The experience of fear, which is the object of this putative second-order fear – the trembling, avoidance behaviour, and so forth – may be *unpleasant*, but it is hardly dangerous. If this is so, why should we posit that it is the object of a fear?

If Eysenck explains this difficulty away by saying that the unpleasantness of fear is feared not because it is unpleasant but because it is associated with danger (the open spaces), then why say we fear fear at all? If *x* is a surrogate for danger, in fearing *x* I am fearing the danger. Its being a surrogate will make it no more nor less powerful a stimulus than the danger that it is a surrogate for. So if fear of fear is only a surrogate or stand-in for fear of open spaces, how does it explain that the fear of open spaces is not diminished but enhanced?

NOTE

With thanks for helpful discussion with my colleague Flint Schier.

by **Michael J. Mahoney**

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Reflections on the conditioning model of neurosi. Eysenck presents what he considers to be the third major model of the conditioning paradigm, a refinement and revision of two earlier models offered by Watson and Mowrer. In this very stimulating article he once again gives evidence of his impressive integrative skills and intimate familiarity with the conditioning literature. I have been cast in the role of a commentator, however, and in contemporary philosophy of science this is almost synonymous with critic (Lakatos and Musgrave 1970). Whether one leans toward either the Kuhnian or Popperian side in today's "great debate" on scientific progress, it is clear that criticism, falsification, and "essential tension" are the preferred paths toward progress (Kuhn 1977; Mahoney 1976; Popper 1972; Weimer 1979). The remarks that follow are therefore hopefully offered as constructive comments on what I consider to be a progressive and heuristic development in the conditioning approach to human behavior.

In my role as critic there are, of course, myriad points on which I might dwell. I could, for example, belabor the use of the term "neurosis" when it is clearly nebulous and currently being evicted from DSM-III. I could also decry the use of sexist language and the somewhat anachronistic realization that fear "is itself a painful event." Likewise, from the standpoints of epistemology and the philosophy of science, I could criticize Eysenck's apparent naiveté regarding the difference between description and explanation, the relation of a theory to a model, and the illusion of some discrete threshhold of scientific viability. But these criticisms are more peripheral to the paper than are others, and I prefer to focus more directly on his main points. In the interests of both clarity and conciseness, I shall enumerate my remarks.

1. I am in basic agreement on the point that genetically transmitted personality variables probably play a more important role in human behavior than has been acknowledged by behavioral researchers. The strength of my conviction is, however, lower than Eysenck's, both with respect to the percentage of variance they account for and the nature of their personality manifestation (e.g., introver-

sion/extraversion). I am also less convinced that we can confidently relate these personality patterns to hypersensitivities of the limbic system and reticular formation.

2. I am in basic agreement on the existence of the phenomenon of preparedness, although I believe we have just begun to unpack our ignorance in the realm of ethological relevancies.

3. The phenomenon of incubation is also difficult to deny, particularly from the perspective of the practicing therapist. Eysenck's *description* of th phenomenon is intriguing and, although it falls short of true *explanation,* his conditioning analysis (via Pavlovian B conditioning) is both innovative and heuristic. Particularly commendable are his efforts to translate the analysis into testable predictions.

4. I am not entirely convinced that "*anxiety (fear) is the conditioned form of the pain reaction.*" Although pain is clearly an associated element in many anxiety patterns, the universality of the above assertion is challenged by such phenomena as the apparent presence of anxiety in persons with congenital analgesia (Mandler 1975). The absence of peripheral pain sensitivity does not, of course, preclude some central (cognitive) form of dysphoria, which leads to my final point.

5. Since I am one of those behavioral "malcontents" (Wolpe 1976a) who has turned to cognitive processes as important psychological variables, it should not be surprising that I would be defensive about Eysenck's critical remarks on cognitive theory. Like Wolpe and Skinner, Eysenck does not deny that cognitive processes exist. Indeed, in the tradition of Kuhnian normal science, he claims to have always acknowledged them, under the less threatening labels of memory traces, enteroceptive stimuli, and a second signal system. Like other critics of cognitive approaches, however, Eysenck denigrates these approaches as less objective and, therefore, a less promising theoretical direction.

Despite my role as a critic, however, I have little interest in increasing the polarity between what I consider complementary positions. The reader does not face a dichotomous choice between conditioning and cognitive theories. With rare exceptions, the latter attempt to encompass rather than exclude the valuable findings harvested in eight decades of behavioral research. And, although the concept of conditioning is challenged in cognitive theories (Brewer 1974), the role of direct associative experience is not (Bandura 1977). Indeed, some of the recent research on the training of coping skills and on stress inoculation is directly relevant to Eysenck's conjectures about prolonged exposure to the $\overline{CS}$ (Mahoney and Arnkoff 1978). Just as relativity theory incorporated (rather than expelled) Newtonian physics, I believe that contemporary cognitive theories complement rather than compete with conditioning views.

To illustrate but one point of such complementarity, a cognitive approach to incubation might postulate that the optimal form and duration of exposure time to $\overline{CS}$ could be determined through a careful analysis of relevant perceptual and conceptual processes in the subject. This would move the level of analysis from a simple descriptive plane to one with more prescriptive information. Bandura's (1977) theory of self-efficacy is, in some ways, an attempt toward such movement. It does not negate the alleged "laws of learning" so much as cast them in a broader context.

Cognitive theories are, in my opinion, potentially more comprehensive in their scope and therefore more likely to lead to an adequate theory of human experience. They have their shortcomings, to be sure, and I would like to think that at least some cognitive theorists are focusing on their failures as energetically as they seem to be celebrating their triumphs. It is here, however, – in the realm of constructive dialectical exchange – that we most need the contributions of such writers as Eysenck.

***by* Isaac Marks**

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Conditioning models for clinical syndromes are out of date. "No theory is sacrosanct, and no model lasts for very long," as Eysenck has noted. The conditioning model of "neurosis" is already out of date and reminds one of Noel Annan's comment that Frazer's *Golden Bough* is "one of the most beautiful ruins in the history of thought." The model is plausible as long as it is not confused with the clinical facts. To be useful, models of psychopathology must bear strong resemblance to their natural counterparts. The conditioning model is at best a pale reflection of clinical phobias and has hardly been a model at all for obsessive-compulsive rituals, not to mention neurotic depression, social skills deficits, or sexual dysfunction.

The assertion that "typical neurotic reactions . . . form a general syndrome" remains mere assertion until it is substantiated by an operational definition of what is meant by a "general syndrome" and a demonstration that the similarities among various neurotic disorders are more important than their differences. Current knowledge does not allow this assumption to be made. Any model of neurosis worthy of the name would need to explain why the different varieties of problems such as phobias and obsessive-compulsive disorders tend to remain distinct when followed for four years (Marks 1971; Emmelkamp and Kuipers 1979); why there is a differential age and sex incidence for these phenomena (Marks 1969), why "free floating panic" is such a frequent feature of agoraphobia (Klein 1964), but rarely occurs in obsessive-compulsive disorders; why some patients become phobic, others get rituals, others develop occupational cramps, and yet others conversion hysteria, and why panics are a feature of both anxiety stages and agoraphobia yet agoraphobics show avoidance whereas anxiety states do not.

None of these issues is touched on by the conditioning model, whose tenuousness is readily apparent to those familiar with clinical patients. One wonders whether the confidence of model builders is directly proportional to their distance from the clinical scene. Eysenck himself has noted that "the rhetoric of conditioning paradigms does not always map easily into the realities of the experimental situation." He could have added "and maps hardly at all into the realities of the clinical situation."

Many of the problems are discussed by Marks (1977) and are repeated here. A crucial difficulty in the study of human psychopathology is the definition of "conditioned" and "unconditioned" stimuli and responses. Because there is usually no history of a clearly traumatic onset to human phobias or obsessions, we cannot assume that they have been "conditioned," – only that they have been acquired. In a typical animal experiment a single CS and a single US are arranged to produce fear. In contrast, a variety of situations usually evoke a patient's clinical distress, and these are seldom traceable to particular traumatic experiences. No one knows the original US, or indeed whether one ever existed. The phobia or obsession simply appears, and search for the equivalent of unconditioned shock is fruitless. Shock is an obvious (though unnatural) US in a rat experiment; what is its equivalent in a human phobic or obsessive? Are we to call anxiety the US, UR, CS, or CR? In clinical disorders it could be any or none of these. Clearly, an alternative terminology is desirable.

To illustrate this problem of definition, let us try to use Pavlovian labels for the typical phenomena found in a woman who has agoraphobia. She complains that each time she waits for a bus she has a wave of panic, breaks into a cold sweat, and wants little else than to rush back home. Just thinking about the bus stop evokes panic, and any panic triggers fear that her fear will get even worse, which aggravates it further in an increasing spiral of anticipatory anxiety, the fear of fear.

We are tempted to say that the bus stop is the CS that evokes the CR of panic. The panic could equally well be a UR. Had the woman once been physically attacked while waiting for the bus, we would surmise that this US produced a UR of pain. But such traumata are exceptional at the first onset of panic. Generally, panic just strikes suddenly. The phobics then blame their panic on those surroundings in which it chances to occur. Should we then call the panic a US? But we have already called it a CR. If we wished we could even label the panic a CS when the fear of fear comes into play, the first fear being a CS and the second a CR. The act of phobic avoidance could also be called a CR, but the "C" for "conditioned" implies an act of faith that conditioning in fact occurred. This confusion in trying to translate conditioning language into clinical practice is compounded when the labels are extended to obsessive-compulsive phenomena. In the case of a man who repeatedly checks the locks on his house for an hour, what is the US? CS? or CR? It is hard to say. When Eysenck refers to "unreinforced CS presentations" what does this mean operationaly for an agoraphobic, obsessive ruminator, or patient with social skills deficits, writer's cramp, or anorgasmia?

The operant (instrumental or Skinnerian) model is as unhelpful as the Pavlovian one. Operant language tells us little about the acquisition of phobias and obsessions. In this language the bus stop we considered before is a discriminative stimulus (S^D) for panic, but this simply means that the bus stop evokes panic. It is commonly assumed that avoidance is maintained (reinforced) by arrival in a neutral area that reduces panic (S^R-). Many patients describe this sequence, but they can be treated successfully despite continuation of their avoidance reactions. Therefore, other factors must play a part. Another operant assumption is that "histories of reinforcement" magically explain the psychopathology. In some cases such analyses do make sense, but often one is left to search in vain for other more plausible reinforcing factors.

Conditioning language and theory were developed as a means of describing and predicting experimental events in laboratory animals. In that context they are

very useful, but when they are applied to clinical data we see serious flaws developing. In scientific work it is accepted practice to evolve different languages for different universes of discourse, even though we need to build as many links as possible between these universes. Let us reserve conditioning terms for behaviour in the experimental laboratory that clearly has been conditioned. For clinical problems like neuroses it is more useful to evolve a simpler language that has clearer implications for treatment and for theory.

It is unnecessary to make an untestable assumption that phobias and obsessions are CSs. To do so would initiate a fruitless search for an unknowable US. Instead we can speak simply of the ES, the evoking stimulus that triggers phobias and obsessions. The phobias and obsessions are the ER, the evoked responses. Neither ES nor ER makes assumptions about antecedent conditions related to the psychopathology. The terms "ES" and "ER" have a major advantage for the clinician treating phobias and obsessions. They indicate at once the therapeutic strategy required – continued exposure to the ESs until the ERs subside. Why the ERs are extinguished when patients expose themselves to the ER during treatment is a vital and unsolved question, but this does not deter the clinician from mapping a successful treatment policy: to search for those situations (ES) that evoke the phobias and obsessions and then to maintain the patient in contact with them until he gets used to them.

The ES-ER paradigm has a second advantage, a theoretical one that focuses on crucial questions about mechanisms of improvement. We do not understand why the same experience before treatment evokes dread, yet continued exposure to that experience during treatment leads eventually to loss of discomfort. We need to work toward specifying the differences, so far unknown, between exposure that is traumatic or sensitizing (Ex_s) and exposure that is therapeutic (Ex_h). Our theoretical task is to discover these differences, which are likely to be multivariate. They may be biochemical; they may lie in duration of ES exposure, sessions or trials within sessions, or duration of intervals between trials or sessions, or all of these. They may lie in modification of the ES during exposure because of distraction or change in the meaning of the ES for a person when he accepts the need for treatment and the licence of the therapist to treat: that is, in the patient's definition of a situation as therapeutic instead of noxious. The type of ER emitted on contact with the ES may also be relevant.

Instead of making Procrustean attempts to fit clinical phenomena into an inadequate model, our aim should be to make precise statements of the necessary conditions that separate exposure (sensitizing) from exposure (therapeutic). Laboratory models of conditioning do not simulate both onset and extinction of phobias or obsessions. Most conditioning experiments have a different time span from that of patients' symptoms. Many produce acquisition of avoidance, treatment, and extinction testing on the same day (Baum 1970), although a few take several days. Most investigations study only one sequence of acquisition and extinction in a given animal species; only a few examine repeated acquisition and extinction. There is evidence that repeating the sequence over a long period can produce an outcome different from that following a brief experience (Akiyama 1968; 1971; Baum 1972b). By the time many humans seek treatment, their phobias and obsessions have been present for years, running a fluctuating course with repeated partial re-acquisition and re-extinction. To mimic these forms of human pathology more closely it is desirable to have animal experiments that extend over long periods, with recurrent sequences of acquisition and extinction. Furthermore, general rules would be easier to deduce from animal experiments if more species were included in many more stimulus response systems. It is dangerous to overgeneralize when the great bulk of experiments are based on a narrow range of stimuli and responses from one species – the rat.

Contiguity conditioning theories of fear and obsessions show serious clinical shortcomings on formal analysis. As Seligman noted (p.205 of Maser and Seligman 1977), Spence (1951) divided learning theory into two varieties. Sign-significant (S-S) theories held that reinforcement was unnecessary for learning, which was only one aspect of a larger problem, perceptual organization. The fact that in Pavlovian conditioning the CS and US are both stimuli that must be integrated perceptually makes this an S-S learning paradigm. Stimulus-response (S-R) theories emphasize the reinforced (instrumental) learning postulated by Hull and Thorndike. Spence noted that such distinctions were not always clear, and now this issue is often ignored.

With phobias and obsessions an S-S theory fits the clinical facts of extinction but not of acquisition, whereas S-R theory describes acquisition but not extinction. An S-S (CS-US) theory requires that a neutral stimulus – for example, a bus stop (CS) – become associated with an unpleasant stimulus (US), such as being struck by a bus. The resulting unconditioned response (UR) is pain and an attendant CR of fear. This S-S theory predicts that fear is extinguished when the CS occurs without the US. However, a phobia as seen in the clinic usually develops without a known history of trauma, and the CS becomes aversive without initial pairing with any discernible US. Furthermore, extinction of fear of the bus stop should constantly be occurring in the absence of a US, yet before treatment agoraphobics report steadily increasing fear during repeated encounters with the bus stop. S-S theory thus fails to account for the acquisition of most clinical phobias. It may accord better with the facts of extinction which are that repeated exposure to the CS (bus stop) without a known US, eventually leads to disappearance of the CR (fear).

To explain acquisition of a phobia of buses, an S-R theory associates the neutral bus stop (CS) with an unpleasant UR, which could be panic. The need for a US is disregarded. This accords with the clinical observation that those settings in which panic strikes (the bus stop) become the CS for phobias. So an S-R theory fits the clinical facts of acquisition, but not those of extinction, since extinction requires the CS (bus stop) to occur without the UR (panic). Unhappily for the theory, panic during exposure to the CS gradually disappears, as does avoidance of the CS. S-R theory predicts that exposure treatment will make the phobia worse, yet the opposite is generally true.

Animal models for the reduction of avoidance behaviour are more promising than the more global S-S and S-R theories. Continued exposure of both animals and humans to the stimulus that evokes discomfort (ES) usually leads to reduction of avoidance and fear: the patient eventually learns that the discomfort will gradually subside despite continuing contact with the ES. Avoidance or escape are therefore unnecessary for discomfort to be reduced and gradually drop out. This explanation could be tested by seeing whether reduction of discomfort by the end of a treatment session (ending on a "good note") produces improvement in avoidance. However, we would still not know why subjective fear or other discomfort was reduced by continuing exposure to the ES. Patients generally find the experience of fear noxious, and fear could be regarded as a US. Continued exposure to such a noxious stimulus should lead to increased, not decreased, fear. It is thus a mystery why most phobics and obsessives improve on exposure to situations in which their expectations of panic on contact with the ES are confirmed, at least in the first part of the treatment program.

Leaving aside these general shortcomings of a conditioning model of neurosis, there are further flaws in Eysenck's particular analysis. The hypothetical curve shown in his Figure 1 is not based on any evidence that the strength of the CR takes the shape shown as a function of $\overline{CS}$ exposure. It is quite possible that the "curve" might in fact be a straight line or a shape the inverse of that drawn, or it might assume different shapes with different criteria for strength of CS. We have no idea whether the shape is the same for subjective anxiety, for heart rate, for skin conductance, or for behavioural avoidance, or indeed whether phobias, rituals, and obsessive ruminations all follow similar patterns. Only when there is empirical evidence to support the shape of these imaginary curves can we talk sensibly about them.

Some inaccuracies also mar the article. The work of *Öhman* [q.v.] and his coworkers is said to demonstrate "very clearly" that " 'prepared' stimuli acquired CS-CR connections much more quickly than did nonprepared CS." In fact this is not so. For prepared versus unprepared stimuli Öhman et al. have in general found few differences in acquisition, and far more in *extinction*. The discussion on preparedness fails to take into account recent work which suggests that the preparedness hypothesis implying a readiness to form certain CS-CR connections is less convincing than the notion of "prepotency" (Marks 1969) or stimulus salience (Lang and also Eelen, personal communications).

Eysenck further claims to justify the statement that "the longer the exposure to $\overline{CS}$, the weaker will be the CR." In fact the studies cited by Nunes and Marks, Marks and Huson, Watson et al., and Mathews et al. did not control for long versus short periods of exposure. They only demonstrated that as exposure continues phobias and obsessions die down; but the same might have occurred with repeated brief exposures. Only the cited study by Stern and Marks, and also by Rabavilas, Boulougouris and Stefanis (1976, not cited) support the idea that longer exposure is better.

Finally Eysenck writes that he "has rewritten the law of extinction completely, suggesting that two consequences may follow upon the CS-only presentation," these being extinction or enhancement of the CR. A third obvious possibility not mentioned is that the CR simply continues unchanged at preexisting strength.

In brief, Eysenck's comment about the "distorting mirror of Freud and his colleagues" and his statement that "cognitive psychology is really a dogma in search of a theory" apply with equal force to his own conditioning model of neurosis. It ignores the fact that there are many neuroses rather than a neurosis, it

explains few known clinical facts about them, including those known about acquisition and extinction, and it makes large hypothetical assumptions without empirical data to support them. It is fortunate that we have effective treatments for certain neuroses well before we have adequate theories to explain their mechanisms of action. As often happens in science, theoretical advances may come from people in the field (in this case clinicians) as well as from those working in the laboratory.

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Are the concepts of enhancement and preparedness necessary? The suggestion that, at least under certain specified conditions, the occurrence of a conditioned fear response can serve to reinforce the association between that response and the CS and, thus, provide an increment in fear (enhancement/incubation) rather than a decrement (extinction) is provocative. It is interesting to note that such a possibility was previously considered by Miller (1951a, pp. 451ff) who provided a theoretical reason for its failure to be observed. However, Eysenck cites research that purports to provide support for the occurrence of such a process.

To be convincing, evidence for enhancement must demonstrate that it is the strength of the fear response itself that increases and not merely the strength of some performance measure used as an index of fear. Consider this example. Following inescapable CS-shock pairings, rats will learn to jump a hurdle to escape from the fear-eliciting stimuli in the absence of further shock, and response speed will increase monotonically for many trials (e.g., McAllister and McAllister 1963; 1971, pp. 107–10). The usual interpretation of such data is that although fear is presumably decreasing (extinguishing) with each trial, sufficient fear remains to provide the motivation and the reinforcement for the learning of the instrumental response. The same interpretation would apply to the improvement in performance reported in avoidance extinction trials (Solomon and Wynne 1953), which can be considered to be escape-from-fear trials. Inasmuch as CS-only presentations are administered during escape-from-fear training, the procedure is like that which holds when enhancement of fear ostensibly takes place. The question could, therefore, be raised as to whether the increase in performance could (partly) be attributed to an increase in fear. The research of Kalish (1954) indicates otherwise. Following classical fear conditioning, he presented the 5-sec CS alone either 0, 3, 9, or 27 times before hurdle-jumping training. Enhancement did not occur but extinction did: the fastest learning was found in the group that received 0 CS-only trials, and learning was increasingly poorer as the number of CS-only trials increased. Nevertheless, despite this extinction, sufficient fear was present to support the learning of the hurdle-jumping response in all but the group with 27 CS-only presentations.

Additionally, it should be noted that the simple maintenance over time of a response indexing fear (e.g., Maatsch 1959) or the observation that certain procedures slow down forgetting (e.g., Silvestri, Rohrbaugh, and Riccio 1970) do not implicate enhancement without additional evidence that fear has actually increased. Also, the fact that symptoms of fear shown by a subject may shift from one occasion to another or develop over time (e.g., Lichtenstein 1950) does not necessarily indicate that fear has strengthened. Such shifts may occur, for example, as a result of changes in relative strengths of different responses in the response hierarchy caused by extinction, habituation, adventitious reinforcement, or higher-order conditioning.

Perhaps the most persuasive experimental evidence for enhancement cited by Eysenck is that provided by Rohrbaugh and Riccio (1970) and Rohrbaugh, Riccio, and Arthur (1972). However, as pointed out by Weldin (1976), there is reason to believe that in each case the purported enhancement is attributable to artifacts in either the experimental design or procedure. In the Rohrbaugh et al. (1972) experiment, subjects from the two groups that are of interest here were first given tone-shock pairings and then were retained in the conditioning chamber for 12 min during which time they received either a 0-sec or a 15-sec exposure to the tone alone. The CS exposure was temporally centered in the 12-min period. The effect of this treatment was assessed by the degree to which the tone suppressed ongoing drinking behavior. It was found that the 15-sec group showed greater suppression than did the 0-sec group, a result interpreted as demonstrating enhancement of fear. Weldin pointed out that because the interval between the end of the 12-min exposure period and the test was constant and because the CS exposure was centered in the 12-min period, the groups of necessity differed with respect to the time between the end of their last exposure to the tone and the test. If some time-dependent process, akin to the Kamin effect (Brush 1971), begins with the termination of the conditioning trials, and if it can be restarted by a CS presentation, the results of Rohrbaugh et al. can be attributed to the decreased amount of fear that could be elicited in the 0-sec group, relative to the 15-sec group, rather than to an increase of fear (enhancement) in the 15-sec group. Weldin's study, in which the exposure time was held constant and the exposure-test interval was varied, confirms the presence of such a time-dependent process.

In the Rohrbaugh and Riccio (1970) study, animals were first shocked in one compartment of a two-compartment apparatus. Then, several groups were exposed to the fear-eliciting cues of the shock compartment, and a control group was not. Later, all subjects were placed in the shock compartment, and the amount of time spent in the safe compartment in a 20-min period was used as an index of fear of the shock compartment. Groups given brief exposure to the fear-eliciting cues spent more time in the safe compartment than the no-exposure control group, a result interpreted as showing enhancement of fear. The data from Weldin's replication of this study suggested that this interpretation may be erroneous because of the measure of fear employed. Weldin found that, as compared to the brief-exposure groups, the no-exposure group had a significantly longer initial latency to leave the shock compartment and, during the second 10 min of the test period, spent significantly more time in the safe box. Taken together, these results suggests that fear was greater in the no-exposure group than in the brief-exposure groups. Presumably, this greater fear initially elicited freezing in the shock compartment and later led to a greater preference for the safe compartment. Thus, his conclusion was that brief exposures resulted in extinction and not enhancement. It seems plausible to infer that these same events occurred in the Rohrbaugh and Riccio study. If so, their response measure, preference for the safe side over a 20-min period, would be confounded with the initial latency to leave the shock box and, thus, would not provide an accurate index of fear.

These remarks are not meant as a rejection of the enhancement hypothesis, but rather as a suggestion that no compelling experimental evidence has as yet, in our opinion, been published. The implications of the hypothesis are important, however, and as presented, are open to expiermental test. It may turn out to be the case that enhancement can be demonstrated with humans but not with animals. If this is so, it seems possible that the reported increases in neurotic symptoms which occur in the face of ostensible unreinforced CS occurrences are explicable in terms of symbolic processes. That is, the UCS and, hence, the UCR may be supplied through imaginal processes. If so, of course, "enhancement" would be simply the result of further, self-administered conditioning trials.

In developing his position, Eysenck contends that it is necessary to introduce a concept of preparedness to account for the ease with which some neuroses are established. Preparedness, a term introduced by Seligman (1970), refers to the proposition that certain organisms can, because of their genetic endowment, learn an association between certain stimuli and responses very quickly because they are prepared to do so, but cannot readily associate other stimuli and responses because they are unprepared or contraprepared. The opposite position, equivalence of associability or equipotentiality, holds that any response can be associated equally well to any stimulus. These contrasting positions are reminiscent of Leibnitz's conception of "veined marble" and Locke's conception of "tabula rasa" (Heidbreder 1933, pp. 60–61).

Eysenck, following Seligman, argues that the traditional learning theories adopted equipotentiality as a basic premise. This seems to be an overstatement of the case. At least, Hull (1943, pp. 206–9) listed several variables that would determine the amount of learning, if any, that would accrue to a given stimulus. These included, for example, the physical intensity of the stimulus, its ubiquity, whether the stimulus is dynamic rather than static, and so forth.

Although some form of preparedness probably holds, several instances are cited as supplying evidence for preparedness when alternative explanations are readily available. Thus, the failure of English (1929) and Bregman (1934) to condition fear to common household objects, in contrast to the success of Watson and Rayner (1920) in conditioning fear to a rat, may be understood as due to differences in familiarity of the conditioned stimuli. In the literature, difficulty in conditioning a response to a familiar stimulus is a well-known phenomenon called latent inhibition (e.g., Lubow 1973; McAllister, McAllister, Dieter, and James 1979). Further evidence is that taste aversions, considered to be prepared responses, are *not* readily learned with familiar CSs (e.g., Logue 1979, p. 286). Also, it should be noted that autoshaping, similarly cited by Seligman as an example of prepared learning, does not meet his definition. To be prepared, it is held that the response must come under control of the stimulus within a few trials. Yet, the autoshaped response cited in Brown and Jenkins (1968) required, on the average, about 40 trials to occur.

These comments suggest that given appropriate conditions (e.g., an unfamiliar, intense, or dynamic CS or a strong UCS producing a vigorous UCR), fear can be learned in one trial, and there is no need to modify traditional learning theory to account for one-trial learning, regardless of whether or not some principle like preparedness also operates. After all, literally dozens of experiments have reported the learning of fear in one trial as indexed by performance in passive avoidance tasks.

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New perspectives on conditioning models and incubation theory. The target article provides a useful review of the current status of the conditioning theory of neurosis. Although the paper does not differ substantially from the author's other recent statements on the same topic (e.g., Eysenck 1975a, 1976d), its appearance in this forum where others concerned with similar issues can provide commentary is a welcome step. My comments will be confined in large part to three general issues: (1) points where various learning models and their problems appear to be confused; (2) a discussion of problems with the author's intuitively appealing incubation theory; (3) two recent lines of research not cited by the author but relevant to the present discussion.

1. Conditioning versus avoidance models and their problems. Although Eysenck traces the evolution of the simple classical conditioning model of Watson to the more complex two-process model of Mowrer (1947), his discussion is at times confusing and even somewhat misleading. In the Mowrer avoidance model fear of the CS serves to motivate an avoidance response that serves to avoid the US. Yet Eysenck discusses the relevance of this model for "some rather atypical neurotic reactions" where the person "learns to avoid the CS" and thus avoids reality testing. In fact, there is no good model for such learning because animal investigators have generally been unsuccessful in their attempts to demonstrate learned avoidance of an avoidance CS (e.g., Sidman 1955; see Hineline 1977, for discussion). So an avoidance model must postulate the existence of an avoidance response (probably an unobservable one, cf. Seligman 1971) that allows the neurotic person to avoid the presumed occurrence of some traumatic *US*.

Even if such a response could be found to exist, the avoidance model then encounters other difficulties to which too little direct attention has been paid. Animals who become highly proficient at avoidance responding no longer appear very fearful of the CS (Kamin, Brimer, and Black 1963; Solomon, Kamin and Wynne 1953; Starr and Mineka 1977). Yet fear or anxiety is obviously the main persistent symptom of neurotic disorders. So while avoidance models evolved, at least in part, to explain the high degree of persistence of neurotic disorders, the model confuses the persistence of avoidance responding (a real phenomenon) with the persistence of high levels of fear (not true in avoidance responding). Of course, it is still possible that the preparedness notion may be of some help here – that is, avoidance learning with a "prepared" CS might show persistence both of the avoidance response and of fear of the CS. There are as yet, however, no relevant data on this possibility, and theorists of avoidance learning per se have clearly turned away from positing a major role for fear, at least in the maintenance of avoidance responding.

On a similar point, it should also be noted that in places the author alternates between discussing the fear conditioning model and the avoidance model and in so doing tends to obscure the distinctions between the two. As discussed above, with an avoidance model one would expect to see persistence of some response that serves to avoid some presumed US but not necessarily persistence of fear. With a classical conditioning model one should expect fear extinction. Since, in fact, fear or anxiety does persist, both modes fail, at least as originally stated.

A final point should be made regarding the author's discussion of the possibility of extending the domain of UCSs thought to be able to condition fear or anxiety states. Frustration as a UCS encounters the same difficulties as exist with traditional traumatic UCSs: CRs based on frustration should extinguish quite rapidly. Perhaps preparedness notions can come to the rescue here, but there are as yet no relevant data such as exist with the work of Öhman and his colleagues for fear conditioning. Finally, the discussion of "uncertainty" as a UCS is interesting and probably deserves more attention. However, it should be noted that uncertainty with a nonaversive (i.e. appetitive) UCS has never been conclusively shown by *controlled* studies to be able to condition fear or anxiety. The discussions by Kimmel (1975) and by Mineka and Kihlstrom (1978) of several studies from Pavlov's laboratory where experimental neurosis occurred in the absence of a noxious UCS contain post hoc hypotheses that "uncertainty" was the critical factor involved in the emergence of the neurotic behavior. However, until appropriate controlled studies have been done that directly manipulate "uncertainty" or "loss of predictability" in such appetitive conditioning situations, such ideas should not be stated as facts, but rather as hypotheses.

2. Problems with incubation theory. Although I personally have always found incubation theory to be intuitively very appealing, I am concerned about the paucity of evidence to support it. Incubation is defined as occurring when there is an *enhancement* of fear following some amount of nonreinforced CS exposure. Using these criteria the studies of Napalkov (1963), Rohrbaugh and Riccio (1970), and Rohrbaugh, Riccio, and Arthur (1972) are the only animal studies that have demonstrated incubation of *fear*, and the last two studies hardly found huge effects. Eysenck has a tendency to mislead us, however, into believing that there is a wealth of other data, both animal and human, that support the incubation notion. For example, he juxtaposes his discussion of the Sartory and Eysenck (1976) paper with that of the Rohrbaugh and Riccio papers. Yet Sartory and Eysenck found no evidence for enhancement of fear; rather, they simply found that short CS exposures were in some cases ineffective in producing fear extinction. Failure to demonstrate fear extinction should not be confused with fear enhancement, and only the latter can directly support incubation theory. Similarly, the discussion of the importance of the duration of response-prevention treatments in hastening avoidance response extinction is irrelevant to the viability of incubation theory per se. Incubation theory does predict the importance of this variable, but so do nearly all other theories of response prevention. Furthermore, it should be kept in mind that the cited studies on response prevention all assessed its effects on avoidance response extinction and not on fear. This is an important distinction because there is often a dissociation of the effects created by response prevention on avoidance extinction and on fear (cf. Mineka 1979; Mineka and Gino 1979).

Thus while incubation theory has considerable intuitive appeal, the apparent difficulty in finding empirical support for it must lead one to question how important a role it can play in the genesis of human neurotic disorders. Conditions such as encountered in the Campbell, Sanderson, and Laverty (1964) study are rarely encountered in real life and it would seem that more clear-cut demonstrations of incubation (i.e. fear *enhancement*) should be documented before this portion of Eysenck's theory is taken as more than speculative.

3. Recent work of relevance to the present discussion. In discussing attempts to explain why fear CRs often do not appear to obey the "law of extinction" attention should be drawn to the recent work of Rescorla and Heth (1975) on "reinstatement." These investigators have found that exposure to a traumatic UCS alone following extinction of a fear CR can serve to "reinstate" the fear CR even though no further CS-US pairings have occurred. Furthermore, the UCS used to reinstate the fear need not be identical to the UCS used in the conditioning. Thus, it is possible that random encounters with traumatic or aversive events could by themselves serve to reinstate partially extinguished fears and thereby contribute to their extreme persistence.

Finally, any theory that posits a major role for fear conditioning today should acknowledge the role that instrumental contingencies can play in affecting the dynamics of fear conditioning and extinction. For example, more fear is conditioned with inescapable shock than with the same amount of escapable shock (e.g., Desiderato and Newman 1971). In addition, the attenuation of fear seen over the course of avoidance learning (where instrumental contingencies operate) is not simply the result of a Pavlovian fear extinction process but rather is due to some aspect of the instrumental control the avoidance learner has (yoked animals do not show the same attenuation, Starr and Mineka 1977). Finally, the amount of control a subject has in an avoidance extinction situation may affect the degree of fear extinction that occurs there (Mineka and Gino 1979). (See Mineka 1979 for a more complete discussion of these issues.) Thus conditioning theorists, especially those interested in modeling the conditions in which human beings are likely to encounter fearful situations, must begin to study the dynamics of fear conditioning and extinction in more complex and varied situations than those used in the past.

by **J. M. Notterman**

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Toward an unpdated model of neurosis. I suspect that Eysenck's article will receive as wide a range of positive-to-negative comment as any heretofore published in this journal. For some, the facts and arguments presented by the

author will be as timely and intriguing as an international spy story in this morning's newspaper. For others, the article will be as stale as last week's five-day weather forecast.

Just sitting back and reflecting upon the overall contents of this contribution, I find that there is little to learn that has not already been reported. But to be fair about it, I have to acknowledge that Western psychologists, by and large, do need to be educated to the *nonmechanistic* character of Pavlovian conditioning. And if the author's focusing upon the phenomena of "preparedness" and "incubation" jars some behavioral and brain scientists into realizing that the stimulus substitution theory is nonsense, then the author's work has been productive.

Nonetheless, I feel obliged to offer a sample of instances where I believe that the author presses either too far, or not far enough.

1. A statement such as the following belongs in an elementary textbook; "It is much more realistic to speak of conditioned *responses* than of conditioned *reflexes*" (emphasis in original). Here, he presses too far. He may educate a few, at the expense of unnecessarily irritating at least as many, who might otherwise become sympathetic to the author's main argument, or already share it.

2. Insufficient distinction is drawn between anxiety, fear, and phobia, from the point of view of either therapist or theoretician. This is a case of not pressing far enough, for conditioning theory can help to refine the distinction.

3. Virtually no consideration is given to the CS-enhancing effects of partial reinforcement.

4. The interrelations among classical-instrumental aspects of anxiety, escape, and avoidance behavior are not articulated, even though Schoenfeld's (1950) seminal article is cited. I include in particular the consequences for automatic responses of conditioned and unconditioned sensory-motor feedback.

5. The comments on backward conditioning are reminiscent of Sullivan's "parataxic distortion," and comprise a strong feature of the article. (Parataxic distortion refers to the neurotic's tendency to disregard temporal and spatial contiguity of events.) Why confound the point with an example taken from (of all things) *delayed* reaction to injection of a drug. By this kind of analysis, temporal onset of CS can be assigned to the time of drug purchase, never mind drug injection.

6. What happened to Pavlov's theory of neurosis? Isn't it more substantive than Watson's treatment of little Albert?

7. Why are the neo-Pavlovians given such short shrift? Without an understanding of their philosophy and research, one cannot make solid contact with *current* views of the relations between conditioning and neurosis. Specifically, I include (a) the influence of levels of consciousness upon conditioning; (b) the autonomy of thought processes, as expressed in the philosophy of two-way dialectical materialism; (c) the importance of interpersonal transactions, as manifested in psychological *and* economic reinforcement contingencies (germane to all the foregoing are Razran's 1961 classic, and Cole and Maltzman's 1969 scholarly overview).

Enough. I hope I have not been merely bickering, for the subject matter is far too crucial for psychology and society. Conditioning theories of neurosis are implied in the formulation of *all* theories and therapies.

Professor Eysenck is one of the very few who could combine an underlying thriller with an updated forecast. He has not done so in this article. (Or have I stumbled on the truth — is the present effort but a test of a future such venture?)

ACKNOWLEDGMENTS

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by **Arne Öhman and Holger Ursin**

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On the sufficiency of a Pavlovian conditioning model for coping with the complexities of neurosis. The two crucial terms in Eysenck's article are "conditioning" and "neurosis." However, whereas the former term is extensively elucidated in his paper, the latter is almost totally neglected. Since his aim is to account for one of these phenomena in terms of the other, the success of his endeavour cannot be determined unless both components in the relationship are specified. By giving due attention to the specification of "neurosis," we think that it is possible to show that Eysenck's conditioning model at best delineates a set of necessary but not sufficient conditions for the development of neurotic behavior. In particular, we believe that a more complete account has to consider operant contingencies as well as the individual's evaluation of his coping efficiency.

Eysenck defines neurosis as "maladaptive behaviour, accompanied by strong, irrelevant, and persistent emotions, occurring in full awareness of the maladaptive and irrational nature of the behaviour in question." Although this definition is not without its problems, it can nevertheless provide a point of departure. Unfortunately, however, Eysenck neglects several of its central implications. The definition suggests that both functional and topographic aspects must be considered when neurosis is delineated. While giving some attention to the former in his treatment of "maladaptiveness," Eysenck totally neglects the latter aspect. However, even his treatment of "maladaptiveness" is inadequate. We will return to this problem in a discussion of operant contingencies and the difference between objective and subjective evaluation of the adequacy of a response. This last problem is essential for the "coping" construct as developed by Lazarus and Averill (1972) and Levine, Weinberg, and Ursin (1978).

The topographic perspective is necessary to deal with the fact that neurotic behavior is manifested in several distinct response modalities. According to Eysenck, the manifestations of neurosis are observed in overt behavior, in emotional reactions, and in cognitive activity (e.g. awareness of the irrationality of the behavior). Because Eysenck's theory rests on a unitary concept, that of Pavlovian conditioning, it follows that these response modalities must be viewed as alternative indicators of one and the same central process. Thus, it is predicted that they should covary closely. However, it is well documented in behavior therapy research that indices of neurotic fear such as overt avoidance behavior, physiological responses, and verbal reports of cognitive activity show minimal or at best moderate intercorrelations (e.g. Lang 1968; Grey, Sartory, and Rachman 1979). Thus, Eysenck's theory is in conflict with contemporary analyses of emotion, which have shifted the emphasis from inferred internal states of a unitary nature to a direct analysis of the interaction between different response systems and the environmental contingencies controlling them (Lang 1971; 1977; Rachman 1978a; Hodgson and Rachman 1974). From this perspective, any theory attempting to account for emotional behavior in terms of unitary mechanisms such as Pavlovian conditioning becomes untenable, because the different response systems are controlled by an intricate set of partly independent Pavlovian and operant contingencies. Thus, the parsimony and economy of Eysenck's formulation become the very reason for its failure to provide an exhaustive theoretical description of neurotic behavior.

However, this conclusion should not be taken to imply that Eysenck's efforts in developing the Pavlovian model are wasted. As argued elsewhere (Öhman 1979a) Pavlovian conditioning very likely provides a necessary link in the development of neurotic behavior such as phobias. The important point is that this process is insufficient, and that an adequate approach has to take a broader perspective by considering operant contingencies and the person's coping ability and strategies.

The insufficiency of the Pavlovian principles to account even for the physiological response component is illustrated by some recent data reported by Fredrikson and Öhman (1979a; 1979b). Whereas subjects conditioned to potentially phobic stimuli, in contrast to subjects conditioned to neutral stimuli, failed to extinguish skin conductance and digital vasomotor responses, they did not even show reliable conditioning of heart rate responses (Fredrikson and Öhman, 1979a). In a subsequent experiment, it was demonstrated that normal subjects decelerated their heart rate more to a previously reinforced stimulus than to a control stimulus, whereas phobic subjects showed acceleration to their phobic stimulus and deceleration to a control stimulus (Fredrikson and Öhman, 1979b). This difficulty in producing a conditioned heart rate acceleration in normal subjects has been noted by several authors (e.g. Obrist, Wood, and Perez-Reyez 1965; Hallam and Rachman 1976). To obtain heart rate acceleration, it appears necessary to consider the coping demands put on the subject. Thus, a sympathetically mediated heart rate acceleration is observed when the demands on coping competence are high, but not when they are low or absent (Obrist, Lawler, Howard, Smithson, Martin, and Manning 1974; Obrist, Gaebelein, Teller, Langer, Grignolo, Light, and McCubbin 1978).

The need to consider the coping dynamics is even more apparent when the functional maladaptiveness of neurotic behavior is focused. There is nothing in learning principles per se that makes for maladaptive outcomes. On the contrary, learning mechanisms have evolved because with overwhelming probability they provide for adaptive outcomes. Although popular psychological lore has it that Pavlovian conditioned responses are automatic, unconscious, and thus potentially maladaptive, the facts of the matter are that human conditioned autonomic responses especially are very versatile and responsive to cognitive factors (Dawson and Furedy 1976; Grings 1973; Maltzman 1977; Öhman 1979b). An important task for any learning theoretic approach to neurosis, therefore, is to

specify the conditions behind the development and maintenance of maladaptive behaviors.

Eysenck does not deal with this task explicitly, although one of the factors that he introduces in his version of the conditioning model speaks to this issue. The data on human conditioning to potentially phobic stimuli (reviewed by Öhman 1979a; Öhman, Fredrikson, and Hugdahl 1978) strongly indicate that such "prepared" conditioning conforms to the psychological lore referred to in the preceding paragraph – that is, it is little affected by cognitive factors once the responses are acquired (see Hugdahl 1978; Hugdahl and Öhman 1977).

As argued elsewhere (Öhman 1979a), however, even this factor is insufficient to deal with the maladaptivity of phobias. To address this question it is necessary to consider the coping dynamics prompted by the operant contingencies to which the individual is exposed. A Pavlovian conditioned fear response is likely to set the stage for avoidance behavior. Once such behavior is established, the fear is quite efficiently coped with, and there is little need for further worry (Costello 1970). It is only when this coping strategy is undermined by some approach contingencies that maladaptivity results (Hayes 1976). For example, a person with a strong fear of small closed rooms such as elevators can cope with his fear by choosing to climb stairs instead, thus even enjoying the positive side effect of exercising his cardiovascular system. However, when offered an attractive job with an office at the top of the company's 52-floor building, he is likely to term his fear a psychological problem requiring some form of professional help.

We believe that neurotic behavior can be elucidated further by considering physiological concomitants of fear and anxiety and the coping process itself. A high level of physiological activation is an important component of anxiety (Epstein 1972). Such activation is manifested in cortical, autonomic, and endocrinological measures (Lader 1975). Broadly viewed, coping may be understood as sets of mechanisms resulting in the reduction of states of activation (Levine, Weinberg, and Ursin 1978). For example, the behavioral transition from fearfulness to "nonchalance" as rats master an active avoidance task is paralleled by a dramatic decrease in the internal activation state as assessed by the plasma level of corticosterone (Coover, Ursin, and Levine 1973). Similar findings of reduced activation in several physiological systems once a fear-provoking situation has been mastered by the individual have also been demonstrated in humans (Ursin, Baade, and Levine 1978).

The coping process as developed by Levine and Ursin (Levine, Weinberg, and Ursin 1978; Ursin 1978; 1979) is closely related to concepts to control and predictability (cf. Levine, Goldman, and Coover 1972) and provides a means for controlling internal states. An important point is that the process does not relate directly to objective or external performance criteria (adaptiveness), but to the individual's subjective evaluation of the situation.

Coping processes may have maladaptive outcomes in two different ways. First, an individual may simply fail to cope with a threat to his psychological or physical well-being. This failure results in a continued state of overactivation, which in combination with a cognitive interpretation of the situation may provide the basis for aversive experiences of fear and anxiety (Mandler 1975). If this state continues over longer time spans, it may, depending on environmental and individual factors, result in different types of psychosomatic disease (Ursin 1979).

Second, coping may be successful in the sense that the individual manages to reduce his aversive internal state, but the psychological and social cost may be considerable. The "inadequacy" by external judgment of compulsive or agoraphobic behavior, for example, is irrelevant to the subject, because coping success is determined from his personal viewpoint. Thus, the person may complain about his particular neurotic habits but show little motivation to eliminate the peculiar ways of handling the environment, because such changes would threaten to undermine his coping strategy and throw him into an even more aversive situation.

By considering coping dynamics, one may not only be able to account for a broader spectrum of neurotic behavior, but also to simplify the required Pavlovian conditioning model. Thus, the clinical observations necessitating the central role of "incubation" in Eysenck's theory may be understood without rescourse to this additional process.

The most consistent finding with regard to the conditioning of autonomic responses to potentially phobic stimuli is that such responses fail to extinguish even under laboratory conditions involving minimal degrees of aversiveness (Öhman 1979a). The waxing and waning over time of neurotic symptoms is then due to the additional influence of subtle changes in environmental contingencies, in the internal state of the person, and in his coping ability. Thus, the conditioning process that we see as necessary in the development of neurotic fear may be even simpler than the one Eysenck suggests.

In summary, while sharing Eysenck's commitment to an experimental approach to neurosis based on learning theory, we have questioned the sufficiency of a unitary Pavlovian conditioning model to account for neurotic behavior. In addition, we suggest that the maintenance of neurotic behavior as well as the interaction between different response systems can be understood only by an intensive study of the operant contingencies and coping processes involved.

by **Howard Rachlin**

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Journey into the interior of the organism. It is gratifying to discover that the conditioning theory of neurosis is not dead. Eysenck's criticism of earlier conditioning theories and of cognitive theory is valid, and his modification of conditioning theory in the light of current developments is clever; it is in line with much experimental evidence from the animal laboratory and seems, to a nonclinician, to be in line with everyday experience. The remark at the end that theories cannot be "right or wrong" but must be judged by whether they are "fruitful or not" is well taken and, applied to Eysenck's own theory, seems likely to justify its existence.

Still, however, I read the article with a feeling of restlessness. There is an awful lot of *explanation* here but precious little in the form of guides to *prediction* and *control* of behavior. This is especially true with regard to variables (not explicitly part of conditioning) that modify conditioning, preparedness, and personality. With regard to preparedness, as Seligman (1970) has noted, the concept is useless unless a constellation of properties coheres to associations that are of a given degree of preparedness. For instance, taste aversion is easy to learn and also subject to long delays. Are other easy-to-learn associations also subject to long delays? Are they also difficult to extinguish? If this sort of grouping is not generally found, the concept of preparedness becomes nothing but a synonym for ease of learning and hence circular. Eysenck does not provide evidence here for noncircularity of preparedness (nor have I seen convincing evidence of this kind anywhere else). Thus, in Eysenck's theory, preparedness provides a way to disregard disconfirming evidence.

If, in a given instance, extinction is found where augmentation of the CR is expected, or vice versa, the fault could easily be attributed to preparedness and, still more easily of course, to previously unnoticed personality variables. Eysenck accuses Skinner of only "paying lip service" to genetics, but biological variables play at least as meaningful a role in Skinner's account of neurosis as they do here. "Lip service" does not become less fatuous when there is more of it – quite the contrary. There exist nontrivial accounts of the interaction between biological and conditioning variables (e.g., Staddon and Simmelhag 1971) that Eysenck might have incorporated into his theory of neurosis, but these accounts are not considered.

With regard to conditioning itself, Eysenck *could* have said:

a. Certain variables such as the duration of the $\overline{CS}$ and the intensity of the US have, in the laboratory, been of critical importance in determining whether extinction or augmentation occurs.

b. These variables correspond to such-and-such potential causes of neurotic symptoms in humans.

c. Manipulation of these variables in therapy, if done correctly, may reduce or eliminate the neurotic symptoms.

d. A guide to correct manipulation with humans may be found in corresponding manipulations with animals in the laboratory.

Finally, a theory of how these variables act, singly or in combination (along the lines of Eysenck's Figure 1, but I hope, with some quantitative precision), might be offered. If this were done, we would not have to ponder over: the difference between "mental pain" and physical pain; the meaning of the word "incubation" and what it might be analogous to in medicine; how one might "implicitly" present a stimulus; how the statement: "Shock is followed by pain, $\overline{CS}$ is followed by fear. Shock + CS is followed by pain + fear; this combined NR is more potent (more disagreeable, more nocive, more aversive) than either alone" can be reconciled with the fact (Badia, Culbertson, and Harsh 1973) that animals prefer a shock preceded by a signal to a shock not preceded by a signal, even when the former shock is four times as intense as the latter; what is a "Gestalt-like NR"?; what is a "complete UCR," as opposed to an incomplete UCR?; what is a drive, anyway, as opposed to the context in which reinforcement acts?; is fear a physiological

response or a cognitive one, or is it overt behavior?; and many other unnecessary questions that are introduced here for no reason apparent to me.

If pondering these issues has led Eysenck and others to do the interesting experiments they have done and to extend them to clinical issues in this potentially fruitful way, we should be grateful. But the empirical basis for Figure 1 (which deals with the only interesting theoretical question in this paper: how do the intensity of the US and duration of $\overline{CS}$ combine?) is weak as it stands. One wonders whether speculations about events in the interior of the organism (fears, hopes, drives of various kinds, neural mythology of various degrees) will help in providing this empirical basis, or whether they will prove to be distractions, as they have so often been before.

***by* Ted L. Rosenthal**

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Thesis and antithesis: S-R levers or meaning-perceivers? Intellectual friction between the heirs of Hull and of Tolman has, for the most part, benefited psychology. Hence, it is appropriate and stimulating to find Professor Eysenck seeking (a) to revitalize "conditioning" theory with his new views about CS duration, and (b) to rebut the burgeoning "cognitive renaissance." His two main emphases are independent and need separate comment.

In the effort to reformulate a conditioning view of neurosis, he contributes a most valuable analysis of the shortcomings in the traditional versions. The discussion of conditions that will promote extinction versus incubation can be especially expected to provoke debate and research. It appears to resolve some paradoxical findings, to point out weak welds in the structure of traditional conceptions and, most important, to suggest new research hypotheses open to experimental validation or refutation. For these, the key parts of Eysenck's contribution, a hearty "Well done!" is in order. Also, it is helpful to have biological variables called into play. Too often, extreme environmentalistic theories of the "black box" sort have ignored constitutional factors and psychophysiological processes [see Eibl-Eibesfeldt: "Human Ethology" *BBS* 2(1) 1979]. Yet one need not share an antibiological bias to have reservations about the "preparedness" theory: first, too few data are in, and some fail to support its assumptions (Rachman and Seligman 1976). Second, the capacity of some but not other cues to evolve into phobias does *not* require biological causation. For example, cultural differences may determine the likelihood that some cues will be perceived as dangers and then socially mediated, modeled, and attended to as threats. Differential cue *meanings* will in part govern how the person and the milieu react to alternative stimuli.

As to Eysenck's ripostes at "cognitive theory": one may ask which part(s) of the elephant are at issue? Occam's razor comes down to us blunted. It does *not* specify how to tell which hypothesis is "simplest." Lacking operational criteria to apply, parsimony ultimately becomes a value judgment. In like vein, many tribes now occupy the "cognitive" tent, some more partisan than critical. Also, if Pavlov's "Second Signal System" is intended to be taken as meaning whatever is scientifically known about human cognition, then argument reduces to nomenclature rather than substance.

However, since the paper specifically mentions the shift in Bandura's explanatory reasoning, some comments on the status of cognition in social learning theory seem called for. Mainly in the last decade, there has come a surge of careful, highly controlled, experimental work on covert information processing. Most of this research appears in such outlets as the *Journal of Experimental Psychology: Human Perception and Performance.* Despite extensive references, Eysenck's paper neglects these sources. Yet it is precisely this body of evidence – much of it hard, replicated fact – on which current social learning theory draws. At base, this work reveals that the routine complexity of human cognition far exceeds previcus, "classical," paradigms of learning. For example, between the presentation of a cue and the performance of an overt response, there typically intervene a host of events including (a) selective attention, (b) multiple recodings and transformations of the cue based on its perceived meaning and coherence with prior knowledge, (c) recursive and executive acts in which the practical significance of the cue is checked, reassessed, and weighted for relevance, and (d) interpretive planning for cue utilization. This evidence is elsewhere reviewed from the standpoint of social learning theory (Bandura 1977; Rosenthal and Bandura 1978; Rosenthal and Zimmerman 1978), but it is best evaluated from its many primary sources. Assuming these data as facts, *no* theory seems able to afford to neglect what they suggest about the nature of human thinking. But no "conditioning" view of neurosis has yet addressed this evidence in depth, or subsumed it by conditioning rubrics. Thus, one may ask how viable any psychological theory can be that ignores our present empirical grasp of human cognition, and the manner in which it seems to operate. For example, the difference in outcome between presenting high intensity fear scenes first (flooding) versus last (systematic desensitization) may have cognitive-perceptual origins. Perceived intensity is a relative product of the surrounding stimulus context – which governs the subjective intensity (assimilation or contrast) of cue events. Conditioning views do not readily suggest such an explanation. Thus, in Eysenck's own elegant phrase: "The rhetoric of conditioning paradigms does not always map easily into the realities of the experimental situation."

***by* Kurt Salzinger**

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Modeling neurosis: one type of learning is not enough. Eysenck's interesting model of neurosis suffers from three inadequacies: it includes at least one concept too many, namely, incubation; it fails to consider a part of the phenomenon to be explained, namely the apparently punishing consequences of neurotic behavior; finally, it does not exploit the operant conditioning paradigm.

In ignoring the operant conditioning approach, it does not distinguish between operant and classical conditioning, and perhaps most important, it does not make use of their critical interaction in explaining neurotic behavior. But no model is replaced by criticism; models are replaced by other (better?) models. We do not have the space here to fully explain such a model, but we can indicate the lines along which it will have to be built.

The appropriate model for neurotic behavior must begin by assuming the validity of the classical and operant laws of acquisition and maintenance of behavior. Such an assumption may be correct: the fault may well lie in the environment rather than in the person, but even if the assumption is unwarranted, one must start from that position, at least for neurosis (a relatively mild abnormality), and be ready to modify those basic laws only if it is found to be absolutely necessary.

By what mechanisms might we then be able to explain persistence of neurotic behavior? First, for both operant and classical conditioning, intermittent reinforcement results in greater resistance to extinction than does continuous reinforcement, particularly in avoidance behavior, the kind of behavior that Eysenck is largely discussing. "Neurotic" behavior may well "pay off" because it is often dramatic, but it probably does so only intermittently.

Second, when the UCS (unconditional stimulus) elicits a defense reflex to shock or other painful stimuli, it elicits a large number of responses controlled by the autonomic and the somatic nervous system. This plethora of responses, differing from one another in terms of speed of execution, latency, and degree of external versus internal control, such as, to take but one example, the difference between speaking and heart rate, practically assures a dyssynchrony among the responses. The lack of synchrony provides response-produced discriminative stimuli (and/or conditional stimuli) as well as response-produced reinforcing stimuli (and/or unconditional stimuli) for other responses over a much longer period than is provided when a simple discrete response class is conditioned. This continuing reverbation of responses may well result in the observed persistence of behavior and obviates such concepts as "incubation."

Third, the behavior that appears to be punished may, on the contrary, or in addition, be positively reinforced. An obvious example of such behavior is to be found in people who receive attention only by emitting behavior that eventually alienates them from others, an effect that results in their losing positive reinforcement only in the long run.

Fourth, the difference in delay interval between the "neurotic" response and the positive reinforcer, as opposed to that response and the punishing stimulus, follows directly from the last point and shows that a response that is punished in the long run can be maintained by positive reinforcement in the short run.

Fifth (and also following from the third point), stimuli that are topographically punishing (that is, appear to society to have that property) may well be functionally positively reinforcing. Such juxtaposition of positive and negative reinforcers occurs when people do strenuous exercises, as in jogging for miles or playing games that require much expenditure of energy. Under some circumstances, such energy expenditure is strictly avoided, as, for example, in climbing the stairs rather than taking the elevator. Ample data show that animals will endure high electric shocks when they receive positive reinforcement and the usually punishing stimulus is administered in small but successively increasing magnitudes.

Sixth, we cannot ignore the role of verbal behavior in maintaining behavior in human beings. We not only emit behavior that impinges on the environment; we also emit behavior that impinges only on ourselves – that is, we think and solve problems. Thus we may react to a situation by subvocally emitting a verbal chain that acts as a discriminative stimulus for our future behavior. Our verbal behavior may well turn out to be the proximal stimulus for many of our neurotic responses.

These are the kinds of complexities that behavior theory can readily cope with but Eysenck's model as described here cannot. I must therefore reluctantly conclude that the need for Eysenck's model is not demonstrated.

NOTE

1. On leave of absence from the New York State Psychiatric Institute and the Polytechnic Institute of New York.

***by* S. Soltysik,**

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Conditioned alpha fear responses and protection from extinction. Dr. Eysenck's paper is loaded with important issues on conditioning and reveals the weaknesses of the "standard" theory of learning. The central theme of his model is the extreme resistance to extinction or even enhancement of the "nocive responses" to conditioned stimuli despite the apparent lack of reinforcement in the form of CS-UCS pairing. The model attempts to incorporate this "neurotic" phenomenon in the general theory of conditioning as an extreme but still normal case of behavioral plasticity; neurosis is not so much a pathology or misfunction of the behavioral apparatus as an extreme case of learning under special conditions when the acquired drive reaches a critical intensity at which it starts reinforcing itself and successfully overcomes the law of extinction.

While sympathetic with the general direction of Eysenck's approach, I would disagree with his stress on the "intensity" of the drivelike conditioned response (CR) playing a critical role in the development of phobias. Instead I would argue that it is rather the "quality" or "mechansim" of the nocive responses to the synergistic (with the reinforcer) CS that plays the crucial role in the acquisition of robust phobic reactions. An additional role in the acquisition of such reactions may be played by the mechanism of protection from extinction by a conditioned inhibitor (or safety signal; Soltysik 1963).

Let me first enumerate the basic tenets of Eysenck's model that are addressed to the problem of intensity of the conditioned fear and its extinguishability.

1. Some stimuli are potentially phobic due to genetic predisposition, that is, neuronal prewiring so that when paired with painful or otherwise noxious stimuli they will readily acquire the capacity of eliciting fear. This readiness is exhibited in the promptness of learning, the intensity of acquired responses, and their persistence or even enhancement in a situation that should cause their extinction – for example when the CSs are presented without reinforcing noxious UCSs.

2. The intestity of the UCS is of prime importance, and the greater the strength of the UCR the more likely the "incubation of fear." However, the potentially phobic CS may readily acquire the full capacity to elicit phobic responses even with weak, sporadic, or otherwise "degraded" UCSs.

3. The nature of the UCR and consequently the CR is decisive in determining whether the presentation of nonreinforced CSs will result in extinction or potentiation of the CR. If the CR is a "complete" response, that is, includes the element of drive, potentiation instead of extinction may occur.

4. The hypothetical mechanism for potentiation of the nonreinforced CR consists in the strong drivelike CR generating stimuli which act as a reinforcer; thus a critically intense fear CR reinforces the CS and promotes further increments of CS-CR strength.

Strength of the CR seems to be a primary factor, and "prepared" stimuli or intense UCSs seem to be important only insofar as they contribute to the vigor of the CR. Even requiring the presence of the drive in the CR is only justified by the fact that nondrive conditioning (type A) cannot secure CRs stronger than the UCRs.

Let us reconsider some of these assumptions. First of all, it is not true that "dogs never salivate more to a bell than to the food." This belief is so firm that I have decided to include in this commentary a raw record of salivary responses to a bell CS and food UCS in one of my dogs; the average salivation to this CS and the effect of satiation on it were published in 1971. The rate of salivation to the CS is 1.6 times larger than the rate during eating. So it does happen. On the other hand, I have never seen incubation of fear in any of my dogs, goats, or cats, and they greatly outnumber the dogs in which I used food as a reinforcer. So the drive

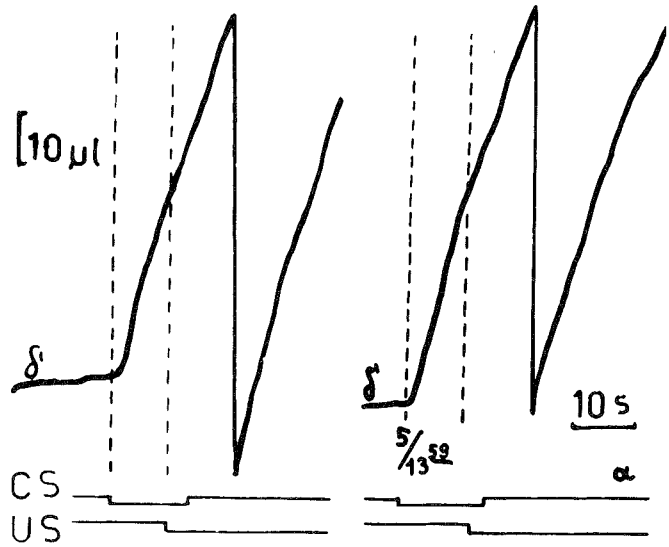

Figure 1 (Soltysik). Salivary responses to a bell and food in dog Rex: two original recordings. The rate of salivation to the CS exceeds by a factor of 1.6 the rate of salivation during eating. This voluminometric cumulative recording of salivation was used in a study by Solytsik (1971) where the averaged data from 3 dogs was presented.

conditioning (type B) is not necessary to promote very intense CRs. As a matter of fact, classical food conditioning could be considered more as antidrive conditioning (cf. Soltysik 1975a; Soltysik, Konorski, Holownia, and Rentoul, 1976). Of course, it could be the superimposed control by a satiety "center" that prevents the occurrence of incubationlike phenomena in food conditioning.

Second, although aversive conditioning is typically a case of drive conditioning, it seldom leads to neurotic phenomena. Even intense fear CRs are extinguishable. They extinguish more slowly than specific consummatory aversive CRs (such as leg flexion or eye blink), but normally they do not potentiate after removal of the reinforcing UCS. Gantt called such differential extinguishability of somatic and autonomic (presumably emotional) responses "schizokinesis" and aptly referred to the lingering emotional responses as representing the "museum of archaic emotions," which resides in the mind long after the overt behavior has adjusted (i.e., extinguished) to the changed CS-UCS contingencies (Gantt 1973b). It would be interesting to pursue the idea of overt versus covert extinction and of the potentially adaptive value of preserving hidden motivations even if they stopped controlling overt behavior. The mental pathology might develop around the mechanism of switching these "preserved" drive CRs from the behaviorally dormant state to actually influencing the conduct.

Third, by elimination, the most promising idea is that of innateness or preparedness (subthreshold innateness) of the fear "CR." Like Thorndike's "belongingness," the concept of prewired connections between CS and CR was used repeatedly to explain resistance to extinction in various learning contexts. Pavlov's "water CR" (in which injection of water into the mouth served as a CS signaling injection of acid solution) survived large brain ablations and was considered "under special conditions" as an unconditioned reflex. Konorski's "specific tactile stimulus" was shown to have additional neural connections of the CS-CR type which rendered the CR very resistant to extinction, but their surgical removal transformed the stimulus into an ordinary, easily extinguishable CS (Dobrzecka, Sychowa, and Konorski 1965). Even more familiar should be the so-called alpha response reported by the students of eye blink conditioning. The alpha response is an unconditioned (if often subthreshold before pairing with the US) eye blink reflex to the CS. It is characterized by a short latency S-R link or mechanism which is possibly different from the ordinary eye blink CR. A neurophysiological study by Black-Cleworth, Woody, and Niemann (1975) suggests that such a subthreshold reflex can be potentiated by pairing the CS (certainly *prepared* since it elicits the response at higher intensities than used in conditioning) with the activation of motoneurons controlling the UCR, without the necessity of participation of the sensory UCS.

That brings us closer to the solution of neurotic phenomena. Potentially phobic stimuli, when paired with noxious UCSs, also potentiate their own *alpha* fear reactions; if Black-Cleworth and Woody's result has any general significance, this process of incubation of the innate fear responses of these stimuli depends not

on the sensory UCS, not even on "response-produced" stimuli, but on the fact that the "motor" or "effector" neurons responsible for the fear reaction are activated in conjunction with the potentially fear-eliciting CS. Even if the initial pairings of such a CS with the nociceptive UCS established only an "ordinary" and extinguishable fear CR, the chances are that the regular succession of such CS by the fear CR will trigger development of an "alpha" fear response, which will mask the extinction of the true fear CR. Extremely relevant in this respect are the studies of Öhman, Erixon, and Löftberg (1975) and of Hugdahl, Fredrikson, and Öhman (1977) showing short latency electrodermal responses (alpha fear CRs?) to phobic CSs in human subjects.

An interesting consideration arises if the hypothesis of alpha fear CRs (Eysenck very correctly prefers to call them "nocive responses" instead of CRs) is accepted. Such nocive responses to prepared CSs should be relatively indifferent to manipulation of the UCS alone. This indifference could even account for the cases of second-order conditioning in which the fear CR elicited by the second order CS is not affected by the extinction of the first order CS (e.g., Rizzley and Rescorla 1972), but it cannot account for the "normal" (S-S type) second-order conditioning as exemplified by the work of Rashotte, Griffin, and Sisk (1977).

Briefly, what I find more congruent with the existing body of evidence is that the incubation of fear is not the result of a normal fear CR reaching a critical intensity and entering the vicious circle of self-reinforcement, but is rather a potentiated subthreshold innate "nocive" response. In intense forms it would cause maladaptive neurotic behavior; in less severe forms it would explain atypical cases of conditioning where mildly prepared stimuli were inadvertently used as CSs.

There remains a further mechanism for preservation of a CR, namely, when a nonreinforced CS is presented in conjunction with a so-called conditioned inhibitor (the term "safety signal" is also used). Although the original evidence was not too convincing (Soltysik 1960b) because of limited material and the use of a nonaversive type of conditioning, a current study in my lab at UCLA provides new data strongly supporting the original notion of inhibitory protection from extinction in aversive conditioning. Interestingly, a formal model of classical conditioning proposed by Rescorla and Wagner (1972) (which probably could not cope with the "incubation" phenomenon) does predict the protection from extinction by a safety signal (see Henderson and Harris 1979). Whether the conditioned inhibitory protection from extinction plays any role in preserving the alpha fear CRs remains unknown, and I rather doubt it does. But, the conditioned inhibitor (e.g., the feedback from the avoidance response) may contribute indirectly to the incubation of fear by maintaining the conditioned fear in the stage when the alpha fear response has not yet developed.

My final comment is addressed to the therapeutic measures described as desensitization and flooding. Both procedures have also been used effectively in extinguishing conditioned avoidance responses. First, Bregadze (1953) has pointed out that prolonging the duration of a CS eliciting an avoidance CR well beyond the latency of this (avoidance) response leads to easy extinction. A little later Dr. Pakovich described to me during the symposium in Osieczna in 1958 (personal communication) another method of extinguishing avoidance responses. He presented the CS intended for extinction for a very short duration so that the dogs did not respond with the motor avoidance reaction. After several such very short exposures to the CS he gradually extended its duration and could eliminate the avoidance response completely. Note that extinction of the fear CR is predicted by the notion of the protective conditioned inhibitor provided by the avoidance response whenever the CS is presented without a reinforcing UCS and the avoidance response does not occur, *regardless* of the duration of the CS (cf. Soltysik 1963). This convergence of the successful elimination of avoidance responses and neurotic symptoms, by two closely similar procedures, makes me suspect that in many cases of neurotic behavior the mechanism of preservation of fear may be analogous to that of avoidance behavior.

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Implications of recent research in conditioning for the conditioning model of neurosis. Conditioning theories of human neurosis usually draw their empirical support from two areas: from research on human neuroses (etiology, symptoms, and therapy) and from research in basic conditioning processes (frequently classical or avoidance conditioning of animals). The target article concentrates on the latter. Eysenck has updated the conditioning model of neurosis by adding the concepts of incubation and preparedness. My commentary will be concerned with other recently investigated conditioning phenomena, some of which may supplement or provide an alternative explanation for incubation and preparedness. Of course, some caution must be exercised in directly applying laboratory and animal-learning principles to human beings. Many of these ideas are new even to the area of animal behavior. However, the reviewed findings should offer new ideas for research and interpretation in the areas Eysenck has discussed. The three topics to be reviewed in the light of recent conditioning findings are incubation, preparedness, and backward conditioning.

Incubation. The hypothesis that $\overline{CS}$ can increase conditioning is an important notion in Eysenck's model. Therefore it is also important to understand the mechanism by which incubation occurs. Eysenck suggests that pairings of the CS and the CR (or NR) may be a means of increasing conditioning. However, the recent literature on conditioning suggests some alternative processes, each of which could be (though is not necessarily) a viable alternative interpretation in some settings. Three possibilities may be mentioned.

First, there is a hypothesis of postconditioning UCS inflation. Rescorla (1974) has shown, in a conditioned suppression task with rats, that postconditioning exposure to a more intense UCS produced increased fear to the CS when subsequently presented. Rescorla argues that manipulation of the UCS characteristics can affect the strength of CS conditioning, even though the CS itself is not present during this phase of the study. Possibly the fear in human neuroses can be intensified by exposure (either direct or imagined) to a UCS more intense than that originally used to condition the fear. UCS inflation would be experimentally distinguished from incubation in that the former does not require $\overline{CS}$ presentation while the latter does.

A second possibility is that $\overline{CS}$ trials do produce new conditioning, as suggested by Eysenck, but that it results from the pairing of the CS with a retrieved representation of the UCS from memory. According to one theory of conditioning (Wagner and Terry 1975), learning occurs through the joint rehearsal of CS and UCS representations in short-term memory. This hypothesis does not, in itself, distinguish between actually occurring UCSs and memory-retrieved UCSs (although some inhibitory effects of $\overline{CS}$ and the accompanying unexpected UCS absence are likely). This idea is similar to that commonly used to describe second-order conditioning, that is, that the second-order CS is paired with some mediated representation of the UCS. Of course, a decremental process like extinction must also be operating, so that extinction on $\overline{CS}$ trials eventually occurs because (a) the subject discriminates the actual from the retrieved UCS, (b) the retrieved UCS is not as potent a reinforcer as the actual UCS, or (c) the retrieved UCS does not contain all of the stimulus attributes of the actual UCS.

The above interpretation thus posits CS-UCS conditioning on $\overline{CS}$ trials while Eysenck stresses CS-NR conditioning. The two views should be separable through appropriate control procedures.

A third possibility results from the fact that the $\overline{CS}$ manipulation is similar to certain "reminder cue" manipulations used to reinstate memories lost because of, for example, amnesic treatments (electroconvulsive shock: ECS) in animals (see Miller and Springer 1973; Spear 1973). Posttrial ECS typically disrupts the appearance of learning that normally would have occurred on the preceding trial. Recent evidence has shown that certain reminder treatments (such as reexposure to CSs, apparatus cues, or UCSs) can reinstate the lost memories. These observations argue that ECS may sometimes cause failure or inability to retrieve certain memories rather than a failure to store such information in the first place. Just as the reminder cues act to enhance retrieval, so also could $\overline{CS}$ trials in incubation studies work to enhance the ability of the CS to retrieve the UCS (or even the NR) representation.

One strength of Eysenck's hypothesis is the proposed ability of $\overline{CS}$ to increase the strength of conditioning over the level achieved during training. By contrast, a reminder effect would simply alleviate forgetting or loss-of-conditioning after training. Thus, Eysenck's theory can be readily supported by data showing increments due to $\overline{CS}$ trials.

Unfortunately, some of the incubation studies do not provide such comparisons. For example, Silvestri, Rohrbaugh, and Riccio (1970) and Greenfield and Riccio (1972) only report the level of fear conditioning, as a function of various $\overline{CS}$ manipulations on test trials without giving measure of conditioning at the end of training. Thus, we do not know whether $\overline{CS}$ actually increased the strength of conditioning or simply reduced the amount of forgetting. Similarly, Reynierse (1966) monitored resistance to extinction without providing a comparison with performance attained at the end of training. Again, it is important to distinguish between actually increasing conditioning and only decreasing the loss of conditioning. Incubation studies should be designed and analyzed with this distinction in mind.

It should be noted that certain aspects of the incubation data available are

contrary to Eysenck's hypothesis. Thus, in one study Greenfield and Riccio (1972) found that three brief $\overline{CS}$s were no better than one long $\overline{CS}$, even though the former more ideally fits with Eysenck's conditions for producing incubation. A second experiment found that fear was comparable following $\overline{CS}$ (exposure to the previously shocked compartment of the shuttle box) and exposure to a safety signal (the unshocked compartment). Unless there was generalization present, it is not clear why a safety signal trial should produce incubation, since the NR should not have been elicited. The results are consistent with the reminder-cue hypothesis mentioned above.

Preparedness. The second innovation in recent conditioning theory that Eysenck emphasizes is the idea that organisms are prepared to associate certain stimuli. While the concept of preparedness is enjoying wide acceptance, caution is being expressed about it as an alternative to more traditional learning principles. For example, Mackintosh (1974, p.54) has argued that what may appear to be preparedness may actually relfect earlier learning experiences the organism has had with certain stimuli or classes of stimuli. Thus, the organism may have learned that exteroceptive CSs are generally uncorrelated with interoceptive UCSs. This "learned irrelevance" may inhibit future associations, just as if the animal was contraprepared to acquire these associations. In another example, Krane and Wagner (1975) demonstrated that a shock UCS can equally condition an exteroceptive CS (light and tone) and an interoceptive CS (novel taste), but that the optimal CS-UCS interval is different for the two classes of CSs. Thus, the alternative here to preparedness is an interaction of two familiar variables: interstimulus interval and CS modality.

Recent research in basic conditioning has revealed a number of new principles describing the conditioning process (see Dickinson and Mackintosh 1978), which may supplement the preparedness hypothesis and otherwise explain variations in the effectiveness of conditioning manipulations. Three examples may be mentioned.

Unsignaled, or "surprising" UCSs are more effective conditioners than are signaled, expected UCSs (Kamin 1969). The signals could be previously trained CSs, or even contectual or handling cues present during training. This principle has been used to describe why certain CSs are selected for conditioning over other CSs, to account for the variations in the effectiveness a given UCS has in producing conditioning, and to explain certain phenomena in short-term memory (see Wagner 1978; Terry & Wagner 1975). The implication for neuroses is that more conditioning may take place in situations or at times when the aversive UCS is most unexpected. The same notion might also apply to CSs: those stimuli that are expected or are familiar will be less conditionable when paired with a UCS.

Conditioning is more rapid the greater the similarity of CS and UCS. This has been demonstrated in both first- and second-order conditioned fear tasks (e.g., Testa 1975; Rescorla 1978). Possibly, in a situation with several CSs, all having comparable saliences and temporal contiguity with the UCS, the CS more similar to the UCS will acquire the most conditioning.

Conditioning depends on the correlation of CS and UCS, as well as their contiguity (Rescorla 1972). Learning is adversely affected by such conditions as presentation of either CS alone or UCS aone, occurring before, during, or after training. While there are differing theoretical interpretations of these effects, the empirical phenomena seem to be well documented. The important implication here is that conditioning occurring among stimuli is influenced by other experiences with these same stimuli.

Backward conditioning. It is interesting to note Eysenck's advocacy of backward conditioning in light of the recent reports that it does occur in some situations (see Moore and Gormezano 1977). Eysenck suggests that since conditioning depends upon CS-UCR (or NR) contiguity, learning will take place even if the CS and UCS are not contiguous. The experiments cited may not be the strongest support for this theory. In a situation in which an injection of morphine or poison is the UCS, no one would argue that the nominal onset of the UCS (the time of injection) is more important than the functional onset of the UCS (the time of the drug's effects). Not surprisingly, it has frequently been reported that "backward" pairings do produce stronger conditioning than do "forward" pairings in such tasks (e.g. Domjan & Gregg 1977). Of more interest are cases in which onset of the UCS is clearly perceived by the subject as such, and there is a delay before the UCR occurs. Jones (e.g., Champion and Jones 1961), advocating a theory much like Eysenck's, studied GSR conditioning to a shock UCS. Here the CS followed the shock, but was timed to coincide with the UCR. Backward conditioning was reported to be even stronger than forward conditioning. Some caution must be used in accepting these findings, since some of these older studies are lacking in sensitization and pseudoconditioning controls.

Eysenck's views may also be consistent with the observation that backward excitatory conditioning is more likely with a small number of trials (Heth 1976) or with surprising UCSs (Wagner and Terry 1975), both conditions that may be associated with larger, more persistent UCRs than expected in the opposite conditions.

In closing, it must be emphasized that the current state of knowledge about conditioning processes does not argue against a conditioning model of neurosis. The literature does suggest, however, a number of possible mechanisms for such phenomena as incubation and preparedness. These findings may only serve to introduce more complexity to the conditioning model, but it is hoped they will also stimulate more sophisticated research programs in human neuroses. Given that a complete theory of conditioning is not yet available, and that many new principles are currently being formulated, it may be too early to develop the best-fitting conditioning model of neuroses.

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The Eysenck and the Wolpe theories of neurosis. In his characteristically well written and well organized paper, Eysenck takes off from the perfectly accurate assertion that neither Watson nor Mowrer provided a satisfactory explanation for "the neurotic paradox" – the indisputable fact that neurotic responses, unlike almost all others, do not undergo extinction when they are repeatedly evoked without reinforcement and often show enhancement instead. However, Eysenck seems to believe that in the more than 30 years since Mowrer, no further attempts were made to come to grips with the neurotic paradox, until he produced his present theory.

The facts do not accord with this negative belief. A theory of neurosis was propounded a quarter of a century ago (Wolpe 1952, 1954, 1958) that comfortably handles the neurotic paradox and most other phenomena of neurosis as well. This theory grew out of experimentally established principles (Hull 1943), extending their application to the field of neurosis and became the framework of a therapeutic paradigm that generated the most widely used and most successful behavior therapy techniques (Wolpe 1954, 1958, 1974). It is really very surprising that Eysenck has been unaware of this. It may be relevant that his first, and perhaps only published reference (1959) to my work on experimental neurosis was not direct, but part of a quotation from Broadhurst (1960).

Experimental neuroses (Wolpe 1952, 1967) are remarkably similar to the neuroses of humans. They are habits of high-intensity anxiety response that are built up by repeatedly exposing an animal in a confined space to anxiety aroused either by ambivalent stimulation conflict or by noxious stimulation. (Fonberg 1956 showed the equivalence of the effects of these two agents.) Once the neurosis is definitely established, the animal is not further exposed to the causal agent.

The *autonomic* responses thus conditioned – the pupillary dilatation and the pilo-erection, for example – are not extinguished by simple exposure (even for hours or days) to the conditioned stimuli (Gantt 1944; Masserman 1943, Wolpe 1952), whereas related motor activity, such as clawing at the sides of the cage, is quite soon extinguished. Two factors seem to contribute to this difference (Wolpe 1952) – the relatively small amount of fatigue generated by autonomic activity (which is relevant insofar as reactive inhibition (Hull 1943) may be a factor in extinction) and the fact that reduction of anxiety drive leads to renewed reinforcement of anxiety responses whenever the animal is removed from the experimental cage (see Miller and Dollard 1941; Mowrer and Jones 1945). However, it now seems that extinction does not mainly depend upon reactive inhibition, but upon other ongoing responses that can successfully compete with and inhibit the conditioned response (e.g. Gleitman, Nachmias, and Neisser 1954; Amsel 1962, 1972).

The anxiety evoked in a neurotic animal in the experimental cage is of such intensity that it reciprocally inhibits virtually all other responses that the environment might tend to evoke. In point of fact, even responses to strong drives are inhibited; for example, a neurotic cat that has been placed in the experimental cage after being deprived of food for 24–48 hours will not eat fresh meat dropped in front of it (Wolpe 1958, p. 52). By contrast, *weak* anxiety responses do not inhibit eating and are weakened by its occurrence (Wolpe 1958, pp. 55 ff.). Very weak anxiety responses in experimental animals are readily extinguishable upon mere exposure to anxiety-evoking stimuli, presumably because of the competition of responses to various environmental stimuli (e.g. Berkun 1957). Similarly, weak clinical phobias are apparently often overcome by the competition of nonanxious responses to the therapeutic interview situation (Wolpe 1976b, p. 27). The more severe a neurosis, the more likely it is that specific deconditioning procedures will be needed to extinguish it (Wolpe 1975).

The one method by which experimentally induced neurotic anxiety responses can reliably be weakened is by systematically counterposing weak elicitations of them (by generalized stimuli) and an incompatible response (usually eating). The basis of this weakening is considered to be conditioned inhibition based on reciprocal inhibition (Wolpe 1954). Even the success of prolonged high-intensity exposure treatment (flooding) may be due to reciprocal inhibition, a view now favored by Levis (personal communication), one of flooding's foremost protagonists. (The anxiety-reinforcing effects of *brief* intense anxiety have been directly shown in neurotic animals; Wolpe 1958, pp. 59–60.)

The foregoing discussion presents a viable and fruitful theory of neurosis. The first question to be asked about Eysenck's theory is whether it, too, is viable. Its central point is the postulate that in the case of nociceptive conditioning, the conditioned response acquires part of the character of the *unconditioned* response – in contrast to all other conditionings. This is an entirely gratuitous suggestion for which Eysenck provides no substantive grounds. He contends that conditioned anxiety is "painful." While it is true that conditioned anxiety is painful in the sense that it is unpleasant, the *physical pain* of the noxious conditioned stimulus is *not* present with it. There is also *no other evidence* that in the particular case of conditioned anxiety some fraction of the *unconditioned* response is included in a way that does not apply to other conditioned responses. For "support" Eysenck can only point to the development of strong conditioned anxiety responses on the basis of weak unconditioned responses (as also described previously by Wolpe 1958, p. 63) and the conditioned phenomena that follow repeated daily narcotic injections. Neither set of observations is relevant, since unconditioned responses are involved in both of these conditionings.

Strikingly absent from Eysenck's presentation is any account of a mechanism of extinction, although he claims to have produced a "new version of the extinction law." His "new version" boils down to nothing more than the statement that unreinforced response evocation may lead to *either* extinction or enhancement. This statement subsumes the neurotic paradox but does not explain it, even if one adds that nonextinction or enhancement is more likely when the CR is strong and even if one states that fact "in the form of a law." Of course, if, with Eysenck, one *assumes* that when anxiety responses fail to extinguish, this "must" be because there is a hidden source of reinforcement, one obviously need not be concerned with the biological mechanism of extinction.

The broad conclusion is that, unlike Wolpe's theory, described above, Eysenck's theory contributes little to our understanding of neurotic phenomena.

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A critique of Eysenck's theory of neurosis. Attempting to stem the ever mounting tide of "cognitive" revolution, Eysenck contends that his conditioning theory of neurosis is "the only viable psychological theory at present." However, even as a staunch believer in Pavlovian conditioning as a major basis for emotional responses, I am not convinced by his argument or his supportive evidence. In the following, I will show that simpler alternative explanations are available and that serious faults exist in Eysenck's exposition.

Eysenck set out to explain why neurotic behaviour persists, since conditioning theory dictates that it should undergo extinction when the UCS (unconditioned stimulus) is no longer present. The failure of extinction may turn out to be a pseudoproblem, when we recognize that in many cases the neurotic response is a UCR (unconditioned response) rather than a CR (conditioned response) as assumed by Eysenck. In fact, Eysenck himself points out that it is not always easy to distinguish between CS (conditioned stimulus) and UCS (unconditioned stimulus). There are at least two reasons to believe what is considered as a phobic CS is actually a UCS capable of evoking an innate, unconditioned fear response.

The first reason is that, according to Eysenck, prepared fears and innate fears differ in degree rather than in kind. Since prepared fears are so close to the surface, they may readily become full-blown innate fears given the necessary precipitating factors. That one is more likely to be frightened by the dark after a horror movie is a case in point. Second, the common phobic stimuli, such as darkness, height, and animals, are all potential sources of danger over which individuals may not have a high degree of perceived control. In other words, it is the element of perceived uncontrollability that serves as a fear-evoking UCS. Whether perceived uncontrollability that serves as a fear-evoking UCS. Whether perceived uncontrollability will evoke an unconditioned phobic response may depend on both situational and organismic variables.

Even if we accept the presumption that all neurotic responses are conditioned, their persistence can be readily accounted for with Mowrer's two-factor theory by shifting the explanatory burden from Pavlovian conditioning to instrumental avoidance learning. According to this analysis, the self-defeating, unadaptive neurotic behaviour is primarily the avoidance behaviour that is reinforced by a reduction of the aversive drive state acquired through Pavlovian conditioning. Through the principle of contiguity, this avoidance behaviour may be evoked automatically by the phobic CS, even though the Pavlovian fear CR has extinguished. Two additional factors contribute to the persistence of avoidance. First, avoidance may have been partially reinforced and becomes highly resistant to extinction. Evidence has accumulated that the persistence effect of partial reinforcement cannot be eliminated by extinction (Amsel, Wong, and Traupmann 1971; Wong 1977). Second, avoidance may be maintained by a variety of subtle reinforcement such as attention, and such reinforcement could very well augment the avoidance tendency. From a systems point of view, it is not unlikely that an individual's neurotic avoidance behaviour is reinforced by other members of the family in order to maintain the equilibrium of the system.

Finally, the fact that extinction is less than 100% does not really pose any problem to conditioning theory; as a matter of fact, it provides empirical support to the widely held notion about the permanence of learned associations (see Kimble 1961). The term "extinction" is an unfortunate misnomer as it connotes that what is learned can be completely eradicated. The weight of evidence is clearly in favour of durability of learning rather than eradication, even with prolonged extinction treatment (e.g., Wong and Amsel 1976; Wong, Traupmann, and Brake 1974). Both retention and persistence are considered as indices of the permanence of learned associations (Wong and Amsel 1976). Desensitization is highly effective in eliminating phobia, precisely because it is based not on extinction but on relearning – the counterconditioning of a new, opposing response to the phobic stimulus.

In view of the above considerations, Eysenck's model III seems superfluous, to say the least. Furthermore, Eysenck has created more problems than he has solved by rewriting the law of extinction. I can briefly discuss only some of the problems here. First of all, Eysenck bases his entire analysis of neurosis on the incubation phenomenon observed primarily in animal experiments. Even if we accept incubation as a real phenomenon and Eysenck's analysis of incubation as basically correct, it still requires a great deal of faith to conclude that "neurosis as a state is the product of this incubation process, which creates a positive feedback system." There are compelling reasons against such an extrapolation. For example, Eysenck argues that a strong drive-producing UCS and short CS presentation are essential for the occurrence of incubation, yet there is no documentation that these two factors are consistently implicated in clinical neurosis. Individuals may develop phobia without having experienced a strong UCS; thus, one need not fall from high places to develop a phobic fear of height. In addition, there is no evidence that all neurotics have been exposed to short CS presentations only.

Extrapolation from laboratory to clinical populations is all the more risky to the extent that the data base is weak and the laboratory phonomenon is not well understood. The empirical basis of Eysenck's model is not particularly impressive. At present, there is no unequivocal evidence that conditioned fear increases to some asymptotic level and stays there in spite of prolonged extinction. In a number of unpublished studies in my laboratory (e.g., White and Wong 1975), incubation is invariably followed by a decline in conditioned fear if extinction is continued long enough, regardless of the duration of CS presentations. Studies cited by Eysenck as supporting his analysis of incubation typically have methodological limitations that render their findings inconclusive; unfortunately, a detailed critique of these studies is beyond the scope of this brief commentary. Suffice it to say that a much more rigorous and systematic data base is needed. We need to systematically vary the intensity, frequency, and temporal parameters of both different drive-producing UCSs and different modalities of CSs; we need such control groups as UCS alone, CS alone, and truly random control for both the acquisition and extinction phases; we also need a more detailed analysis of the CR and UCR through the use of a behavioural field approach (Wong 1979).

Apart from the problem of a weak and limited data base, Eysenck's model also suffers from the lack of good understanding of the incubation phenomenon. At present, we do not have a well-established theory of incubation. Eysenck's own theorizing concerning a positive feedback system as the basis of incubation is complex and unconvincing. His main thesis is that somehow during extinction CR is transformed to UCR and functions as its own reinforcement in a positive feedback system. The more he attempts to convince critics that a CR can indeed become its own UCR, the more untractable his theory becomes.

The following summary of Eysenck's reasoning will serve to reveal the complex nature of his model and many of its unsubstantiated implicit as well as explicit

assumptions. According to Eysenck, CS presentation in the absence of UCS initiates both incremental and decremental processes simultaneously. Whether extinction or incubation occurs depends on which process is stronger. When the UCS is a strong drive-producing stimulus and when the CS presentation is short, the incremental process will prevail. The mechanism of this incremental process involves several mediating steps: first of all, the CS elicits a nocive response, which results in a response-produced stimulus, and which in turn gives rise to the experience of fear/anxiety; it is this experience that functions as a reinforcement and a drive, "because the drive properties of UCR can be transferred to the CR," this transformed CR now increases the CS-CR bond in a positive feedback system.

Eysenck's analysis of incubation is further hamstrung by the fact that several deductions made from his theory are at variance with the literature. For example, according to his positive feedback system, a CS may become a stronger UCS in second-order conditioning if it has been subjected to a longer period of prior extinction. His reasoning also suggests that signaled shock (CS plus shock) is more aversive than unsignaled shock (shock alone). One may also argue that if the positive feedback system works for the CR-turned-UCR, there is no a priori reason why this system should not work for UCR and increase the UCS-UCR bond; in other words, the UCR should become stronger and stronger instead of showing habituation.

In Eysenck's model, the terminology of Pavlovian conditioning has lost its proper meaning, and acquisition and extinction processes can no longer be distinguished. It is debatable whether his positive feedback system is the same as Pavlovian conditioning. When the well-defined simple Pavlovian conditioning model is elaborated and modified beyond recognition, one wonders whether it should still be considered as a Pavlovian conditioning model.

In sum, Mowrer's two-factor theory seems to be much simpler and more convincing than Eysenck's model III as a conditioning theory of neurosis. There are serious problems to be overcome if Eysenck's theory is to gain acceptance in the scientific community.

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"Prepared fears" and the theory of conditioning. The paper by Eysenck, written chiefly to explain neurosis as a specific product of conditioning, directs attention to some phenomena that are important for the theory of conditioning itself. One of these phenomena is the "preparedness" of the organism, a term introduced by Seligman (1970) to describe an innate ability to react to each stimulus in a specific way; this predisposition either facilitates or inhibits the process of conditioning to a particular stimulus. Adapting the concept of preparedness to his own ideas, Eysenck postulates that fears related to certain stimuli are inborn; as a result, conditioning of fear responses to such "prepared" stimuli is much easier than to other stimuli. This point is worth discussion.

It may be questioned whether emotional responses such as fear of some specific stimuli are really inborn. To answer this question, it would be necessary to know the very first reactions of the subject to these stimuli, that is, to follow the subject's life from birth. It is true that a new stimulus evokes an innate orienting reaction which may include some fear. It is not sure, however, whether or not this kind of fear is specifically related to any stimulus. It may also be that fears related to specific stimuli are acquired by experience. Unfortunately, in the laboratory or in clinical research on conditioning, usually little is known about the history of the subject's relation to the stimulus. Consequently, it should be taken into account that the stimulus may not be completely new to the subject. In such cases previous experience related to this stimulus would be of great importance for the course of further conditioning.

A number of studies have shown that the effectiveness of conditioning to a stimulus strongly depends on the previous involvement of this stimulus in other cases of conditioning in the same subject. For example, Konorski and Szwejkowska (1956; cited by Konorski 1967, pp. 336–37) attempted to transform an alimentary conditioned stimulus (CS) into a defensive CS; such conversion turned out to be very difficult and was finally achieved only with the use of special intensive training. Similar difficulties were found during transformation of what was originally a defensive CS into an alimentary CS. The same authors also showed that the classical defensive response, established to an originally neutral stimulus and then chronically extinguished, was rapidly restored as soon as the noxious unconditioned stimulus (US) was used again (Konorski and Szwejkowska 1952, cited by Konorski 1967, p. 319). Other authors have reported that the acquisition of the conditioned response to a stimulus was significantly delayed when this stimulus was subjected to habituation prior to the conditioning training (Kostenetskaya 1949; Carlton and Vogel 1967; and others).

These examples suggest that facilitation or resistance in a stimulus's acquisition of conditioned properties can result from previous experience related to this stimulus. This could be the case with pictures of snakes and spiders, which appeared to be more effective than pictures of flowers and mushrooms in producing the conditioned response of fear related to an electric shock used as US (Hugdahl, Fredrikson, and Öhman 1977). It is likely that the subjects were already conditioned to these or similar CSs, directly or through the experience of others; in such cases, the sight of pictures of snakes and spiders could itself evoke a defensive attitude, while the sight of pictures of flowers and mushrooms could produce an appetitive response. Another example of such conditioning can be an aversive response to the sight of a domestic cockroach, an "obnoxious" and harmful insect which invades our privacy and destroys our food, as contrasted with a quite friendly response to the sight of another insect, similar in color and size to the cockroach, a harmless black beetle seen on a country trail during an outing. To an adult subject, a stimulus is rarely completely new or unlike any other stimulus previously encountered. Knowing whether the stimulus is specifically prepared for conditioning, either by an inborn tendency or by former experience, seems important for the understanding of each individual case of neurosis as well as for possible therapy.

In any case, treating the phenomenon of "prepared fears" as readily conditionable emotional responses, Eysenck undoubtedly adds a new aspect to the theory of conditioning. In fact, the theory of conditioning should not be considered a monolith, established once and for all and never changeable. On the contrary, the theory of conditioning should be treated as the general idea that associations between neural representations of stimuli can be formed and stored in the brain, and that through these associations a stimulus may acquire properties of other stimuli. The information as to where and how this happens is a matter of continuing progress in both experimental and theoretical research on problems of conditioning.

It should perhaps be recalled here that the problem of differences between stimuli to be conditioned was already being studied in the early period of research on conditioning. At that time Pavlov himself claimed that any noticeable change in the environment can be conditioned; he did not say, however, that all stimuli are equivalent in their ability to acquire conditioned properties. According to the observations made in Pavlovian laboratories, differences between the effects of various CSs are mostly of a physical nature: the higher the intensity of the stimulus, the stronger the conditioned response to it (within some limits); auditory CSs were found to produce stronger responses than visual and tactile CSs, at least in dogs (see a summary by Konorski 1967, pp. 290–91). It has also been pointed out that the final effect of the conditioned stimulus depends on the nature of the subject's nervous system (Pavlov 1928, ch. 17). More recent research on stimuli and responses, however, has obviously been taking a new course on these questions. The above mentioned studies of Konorski and Szwejkowska (Konorski 1967, Ch. 7) on the effect on the history of the stimulus on the conditioning process, and the theoretical work of Seligman (1970) on preparedness of the organism to react to various stimuli in a specific way, are eminent examples of progress in this matter.

Considered from this point of view, Eysenck's theory of neurosis as a result of prepared fears conditioned to specific stimuli deserves special attention. It should be welcome as a further step toward understanding behavioral mechanisms.

by **Kazimierz Zieliński***

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Short-latency avoidance responses. Dr. H. J. Eysenck has written an enlightening review in which "the neurotic paradox" is discussed in the framework of conditioning theory. His proposed version of the conditioning model attempts to overcome difficulties of earlier models, which to a great extent disregarded biological aspects of the stimuli used and the individual variability of subjects. Moreover, earlier models did not offer satisfactory explanations for numerous instances of the growth of fear in the absence of pain and the remarkable resistance of conditioned fear to extinction. Two main concepts incorporated into the model, "preparedness" and "incubation," are well documented by new experimental data obtained in many laboratories and from clinical observations.

The model proposed is consonant with modern understanding of the mechanisms that underlie the learning and performance of instrumental defensive

responses. In this short commentary I would like to present some additional relevant data recently obtained.

Comparisons of the distributions of latencies of bar-pressing responses performed by cats trained either to escape (Zieliński 1970, 1972b) or to avoid pain (Zieliński 1971, 1972a, 1972b, 1974) indicate that responses to acoustic stimuli signalling avoidable shock given 5 sec after CS onset are performed with shorter latencies than responses to nonsignalled shock which must be terminated by emission of similar motor responses. Thus, at the stage when performance reaches asymptote, the strength of the response terminating the acoustic fear-evoking stimulus and thus avoiding pain is greater than that of the response terminating the pain. The shorter latencies of avoidance responses, compared to escape responses may be considered another demonstration that the CR can be stronger than the UCR, and traditional conditioning models provide no explanation for such cases.

As a logical consequence of the Mowrer-Miller understanding of fear as a secondary drive having motivational properties, it may be assumed that a subject is able to learn to avoid not only pain but also the fear itself. Such a hypothesis has been proposed to explain changes in avoidance performance after prefrontal lesions in cats (Zieliński 1972b). It was shown that the decrease in avoidance performance after removal of the proreal and orbital gyri is due to the deterioration of short-latency responses, whereas long-latency responses are fully preserved and the postoperative recovery of avoidance performance is related to the further increase of long-latency responding. A postoperative drop in the cumulative response latency curves was in every case between 25 and 30 percent; however, the more intense the CS and the shorter the CS-UCS interval, the more the deterioration was restricted to the early portion of the CS-UCS interval.

Further, it was demonstrated that at later stages of training more short-latency than long-latency avoidance responses were acquired, and the rapid increase of avoidance performance was related to the increase in the proportion of short-latency responses (Werka and Zieliński 1978; Jakubowska and Zieliński 1979; Zieliński 1979). These results provide additional support for Eysenck's model, since a rapid increase in short-latency avoidances occurred at the stage of training when the proportion of shock trials and the overall duration of shocks were markedly reduced by the performance of well-trained instrumental responses (escape or long-latency avoidances).

It was hypothesized that two different mechanisms are involved in the performance of short- and long-latency avoidance responses. Whereas responses executed with long latencies may be considered as escape from fear, short-latency responses are avoidances of fear (Zieliński 1972b) driven by the nonspecific excitation of CS onset rather than by the fear reaction (Jakubowska and Zieliński 1979). This last point was supported by the positive correlation between the frequency of intertrial responses and the proportion of short-latency responses performed to a new CS never paired with shock, during go, no-go differentiation training or when the signalling properties of conditioned stimuli had been reversed (Zieliński and Czarkowska 1973, 1974; Kowalska, Dabrowska, and Zieliński 1975). Intertrial responses are thought to be performed on the basis of general arousal, including subthreshold fear conditioned to the entire experimental situation. The relation of the avoidance performance at early stages of training to the level of intertrial responding has also been demonstrated (Zieliński 1979), and again, these data support some important aspects of Eysenck's theorizing.

Summing up, the model proposed by Eysenck is consonant with experimental data and the conceptualization of defensive conditioned reflexes, both those reported in his paper and others presented in this commentary. Incorporation of our proposition about the nature of short-latency avoidance responses may further extend the implications of Eysenck's model.

NOTE

*Received too late for a response from the author. See Continuing Commentary.

by **Marvin Zuckerman**

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What and where is the unconditioned (or conditioned) stimulus in the conditioning model of neurosis? Eysenck's modification of the Watson-Mowrer conditioning model helps in dealing with some of the problems of that model, but it does not answer the broader questions. The model for the acquisition of a fear response through classical conditioning was based on the famous experiment by Watson and Rayner (1920). Not only has this study been unreplicable, as Eysenck notes, but the original study itself did not demonstrate much of what has been claimed in introductory psychology textbooks and behavior modification treatises. As Harris (1979) recently pointed out: "Critical reading of Watson and Rayner's (1920) report reveals little evidence that Albert developed a rat phobia or even that animals consistently evoked his fear (or anxiety)" (pp. 154–55). Seven trials failed to establish a clear phobic reaction in Albert. The response did begin to extinguish and required additional trials to maintain it. The failure of other investigators to replicate Watson and Rayner's findings is attributed by Eysenck to their use of nonanimate conditioned stimuli, thereby not capitalizing on the "biological preparedness" to fear small furry animals. Actually, the failures of replication appear to stem from the fact that loud noise is an unreliable unconditioned stimulus (Harris 1979).

Another fact makes the Watson and Rayner study inappropriate as a model for neurosis: Albert was an 11-month-old infant. The types of fears seen in infants, such as persistent crying after startle, are quite different from the neurotic phobias of adults, or even older children. Animal phobias are the rarest kind in clinics and hospitals; agoraphobia in adults and school phobias in children are the most common (Marks 1969). While small animal fears are common in the general population, providing an endless supply of subjects for behavior therapy analogue experiments, these fears are quite specific and are not correlated with general anxiety or neuroticism (Mellstrom, Cicala, and Zuckerman 1976). The typical phobic fear is of a broad class of situations rather than a discrete fear stimulus. These fears do not commonly originate in traumatic situations of the type demanded by the conditioning model, but emerge gradually in a general setting involving insecurity or change in the patient's life situation. The absence of clear and well-defined conditioned and unconditioned stimuli makes the application of the conditioning model to the typical neurotic fear difficult and clumsy.

Even if we do consider the fears of infrahuman species and infants as prototypes of neuroses in children and adults, it is still difficult to find the classical unconditioned stimulus of pain anywhere outside of the contrived experiments of behavioral psychologists. Hebb (1946) found that chimpanzees were terrified by such objects as models of detached chimpanzee heads, as well as by snake models and moving toys. Since the animals had no prior painful experience associated with these objects, their fears are difficult to account for in the classical conditioning model. Unconditioned stimuli for fear in the real world of animals and human infants involve stimulus factors of novelty, incongruity, unpredictability, size, and pattern, as well as intensity. The disappearance of such fears in older children suggests that the fears of infants depend on the lack of perceptual-cognitive schemata for assimilating novel stimuli. The universal development of fear of strangers in the human infant in the second half of the first year of life is a good example of this factor in fear development. This is not biological preparedness in the sociobiological sense of specific evolved fears which were once adaptive in the history of the species. An examination of typical phobias supports neither a sociobiological nor a conditioning explanation.

Why are fears of flying in airplanes more common than fears of driving in automobiles? Neither conveyance was part of our evolutionary history, and many more people have experienced accidents, or near accidents, in automobiles than in airplanes. Why would our humanoid ancestors, who evolved in the plains of Africa, be "prepared" to fear open spaces (or even closed spaces)? Such fears would certainly not be adaptive and any post hoc explanations of their adaptive value are bound to be strained and always unverifiable. A combination of *cultural preparedness* and lack of familiarity with stimuli may explain why certain creatures such as rats and snakes are more fearsome to most people than squirrels and small dogs.

While familiarity seems to be inimical to fear, not all novel stimuli are appraised as threatening (Zuckerman 1979). There must be an element of appraised threat in the context of a stimulus to elicit fear on first presentation. A rat running around in a cage is the same stimulus as a rat running free on the floor, but the latter is much more likely to elicit fear in people other than comparative psychologists. The difference in threat appraisal depends on prior learning, but rarely on directly reinforced experience (being bitten). There is ample evidence that fear can be learned through modeling or "vicarious conditioning" (Bandura and Rosenthal 1966).

Another cognitive factor that plays a large role in fear is helplessness. This factor undoubtedly plays a large role in the common fear of flying, since one is closely confined in a situation in which one is dependent on the skill of the faceless pilot and the conscientiousness of the builder of the airplane and those who service it. Helplessness is an essentially cognitive factor which cannot be easily accommodated in S-R learning theory. The role of helplessness is

documented in experiments such as those of Geer, Davison, and Gatchel (1976). They showed that subjects who *believed* that they had control of the aversive stimuli showed less fear arousal than subjects who *believed* that they were helpless in controlling the stimuli. The word "believed" is emphasized because actually neither group had control over the aversive stimuli.

It is not only the appraisal of the external stimulus and situation that determines fear; the attitude toward internal stimuli is also important. The internal sensations elicited by a roller coaster ride may be interpreted as fear or exhilaration depending on one's attitudes toward risk and arousal.

Eysenck has mentioned the individual difference factor which is usually ignored by both S-R and social learning theorists. Besides the heritable factor of emotional instability or neuroticism, there is another equally heritable trait called "sensation seeking," which plays a role in the fear response. High sensation seekers tend to appraise risk as less than low sensation seekers and to anticipate less fear arousal, and more positive affect arousal, in reacting to risky situations (Zuckerman 1979). Low sensation seekers are more likely to show fearful reactions to rats and exposure to heights and darkness than high sensation seekers, and this relationship between sensation seeking and fear response is independent of trait anxiety and neuroticism (Mellstrom et al. 1976).

Eysenck does not ignore the role of what others call "cognition" in the acquisition of fear, but he calls these factors other things such as "mental pain." This term seems to include the factors of frustration, uncertainty, uncontrollability, and conflict which influence conditioning through the "second signaling system" (Pavlov's term for the part of the brain system that controls symbolic operations). Eysenck's response-produced stimuli, which can act as unconditioned reinforcers in increasing the strength of a response, are the traditional S-R substitute for cognition.

How far should a theorist go in interpreting complex phenomena within a model that was not designed to encompass them? At some point parsimony must give way to impreciseness and simple inelegance. But as Eysenck aptly points out, the cognitive theorists have not yet developed a model from which deductions may be easily made. His modification of S-R theory is a challenge to the emerging cognitive models to go beyond mere demonstration experiments and to employ the more powerful hypothetico-deductive method to test clearly stated postulates. Researchers may want to design new studies that provide tests of one model as opposed to another.

The other major contribution of Eysenck, not fully addressed in this article, is his insistence that individual differences based on inherited biological traits must be included in any comprehensive theory of neurosis, in particular, or behavior in general. Any learning theory, whether S-R or cognitive, must be compatible with the facts of brain function and its individual variations.

Author's Response

by **H. J. Eysenck**

The conditioning theory of neurosis: criticisms considered

The criticisms evoked by my paper fall into several distinct categories; this makes answering them easier and tidier. The first great divide is between the more general and the more specific criticisms. I shall first of all take up some general comments – I hesitate to call them criticisms because they are obviously correct, and in principle I agree with them, although perhaps with some reservations. Thus it is suggested (**Wolpe, Kimmel, Wong, Mineka, McAllister & McAllister**) that there is insufficient evidence to support either of the central concepts in my theory, namely the concept of *preparedness* and the concept of *incubation*. (**Bolles** is more optimistic about the evidence for incubation). Again, it is suggested that even if the facts are as stated, alternative theories could perhaps explain them (**Levis, Terry, Soltysik, Salzinger**). Furthermore, the point is made with regard to both concepts that they are *circular*, and that such concepts are of little value in science (**Rachlin, Gray, Lyons**).

Now it is certainly true that the evidence is not sufficient to accept unquestioningly the theory put forward; were it sufficient then we would be dealing, not with a theory, but with a scientific law. It is of the essence of a theory, particularly a novel one, that it suggests something new, something that goes beyond the known facts. As T. H. Huxley puts it, "Those who refuse to go beyond fact seldom get as far as fact." Or, to quote J. J. Thomson, "A theory in science is a policy, not a creed." In other words, the value of a theory is not that it states a law, but that it suggests new experiments (which would not otherwise have been carried out) to either support or invalidate the theory. It outlines a new way of looking at old data, old problems, and old solutions; it is the beginning of what Lakatos calls a "research programme," not the end of one. In other words, it would be quite unreasonable to expect the evidence to be sufficient to "prove" my theory of incubation right; the research cited to give it some credence was not, for the most part, carried out in an attempt to test the concept, but was designed in relation to some quite different theory. What makes this research interesting is that it failed to support the old (extinction) theory, thus raising problems that eventually necessitated the elaboration of a new theory. This is in line with Kuhn's concept of "revolution" in scientific theory being the outcome of more and more anomalies accumulating in relation to orthodox theory, until finally the cracks cannot be papered over any longer. There were many obvious anomalies in the theory of extinction, as Razran already pointed out over twenty years ago. The particular theory I am putting forward is one rather revolutionary attempt to get over these difficulties within conditioning theory; even without the link with neurotic behaviour such an attempt had to be made inside laboratory experimental science.

What my theory does, therefore, is not to summarize a large body of relevant and well-designed research; in the nature of things such a body of research does not exist. It is rather a leap of the imagination, suggesting ways in which the obvious anomalies could be explained, and perhaps even more important, leading to experiments to be done in order to refute or support the theory. The studies cited, whether done before the first appearance of my theory, or done as tests of it, are relevant but not crucial; the former, having been done within the ambience of an older theory, can at best be suggestive, and the latter, as several critics point out (e.g. **McAllister & McAllister; Terry**), lack features that would rule out alternative interpretations. This is very much what one would expect of the first attempts to test a new theory; "normal science" can now be relied on to use its excellent equipment of "problem solving" potential to iron out the difficulties, check the alternative hypotheses, and come up with a reasonable decision between rival theories. I therefore agree with the criticism that the evidence does not as yet permit us to accept the theory; I can only express my hope that future experimental work will clarify the issue.

This possibility is denied by those who believe that the whole argument is circular, and that consequently the theory does not suggest any testable ("falsifiable") outcomes. This is both true and untrue. The whole concept of circularity in scientific theory is often misunderstood; it must be looked at in the context of Hume's criticism of causality. In his terms, we would have to conclude that all scientific theories are circular, but of course some are more circular than others! Take Newton's law of gravitation. He finds that there are a number of disparate facts (apples falling, planets circulating, tides running) and "explains" all of them in terms of a single law, that of universal gravitation. But what is the evidence for that law? Why, apples falling, planets circulating, tides running! Surely this is circular in the extreme. Even the fact that this circularity can be quantified very elegantly does not destroy its essential character, nor does the fact that the circle of facts can be extended to embrace many others not previously considered.

Or take Darwin's law of the survival of the fittest. Many critics have pointed out that this is circular – how do we know that the survivor is the fittest? Why, because he survives! Yet Darwin's contribution has been considered the greatest contribution to the science of biology in the nineteenth century. There is some degree of circularity in nearly every (possibly in every) scientific theory; the only question is whether the circle is narrow or wide. In other words, does it go beyond the facts originally considered, or is it confined within those facts? I would suggest that my theory clearly goes beyond the facts originally considered, and hence is falsifiable –

indeed, some critics consider that it has already been falsified! A theory cannot both be unfalsifiable (circular) and falsified at the same time. I would suggest that not only is it falsifiable, but even that it is quantifiable. The hypothesis embodied in Figure 1 of my target article, for instance, can lead to many quantitative deductions, including the shape of the curve (which can be monitored through psychophysiological recording, through the use of "fear thermometers," and through observation of behaviour). With the total duration of $\overline{CS}$ exposure held constant, and with all $\overline{CS}$s greater than the critical duration, it is possible to make predictions about the respective advantages for extinction of giving the subject a few lengthy $\overline{CS}$s or more numerous but brief $\overline{CS}$s (Bersh, in press). The work of Schiff, Smith, and Prochaska (1972) and of Stern and Marks (1973), with animals and humans respectively, is relevant here, but as neither was done with this theory in mind, results are again not conclusive.

Lyons puts the "circularity" argument particularly strongly, in relation to "preparedness," but his example (of cancer) suggests that the argument is not well taken. Cancer has a genetic basis (Eysenck 1979a), and to say that some humans are more "prepared" for cancer is not to argue in a circle. Of course we must go on from there and find out the biological details of this "preparedness," but a recognition of the importance of genetic factors is the first step in a long series of investigations. Similarly, the postulation of phobic preparedness links up with a large body of genetic studies in the personality field, referred to again later on; it is not suggested that all people are equally "prepared," but that some are more "prepared" than others. This is surely a testable hypothesis. Twin studies show that many different types of phobias have a strong genetic source, accounting for some 50% of the variance (Torgersen 1979). This surely is both support for the hypothesis, and proof that it is not circular in the narrow sense.

The point has been made by several critics that the very fact of incubation is still in doubt, and that the evidence in favour is not as strong as it might be. This is true; comparatively few researchers have found evidence for this phenomenon in animal work, and it may indeed be easier to find such evidence with humans. However, it is usual in science that evidence for a phenomenon is found mainly when one starts looking for it; incidental and accidental findings prior to a thorough search, informed by theoretical expectations, are relatively rare and not very impressive. Such search needs to clarify above all the question of the parameters that govern the appearance of the phenomenon; I have suggested several parameters (severity of UCS, preparedness of CS, duration of $\overline{CS}$ exposure, personality variables), but these are undoubtedly not the only relevant ones, and they may in fact not be the correct ones. Part of the task of "normal science" is precisely the search for the optimal parameter values, and the task of theory is to direct this search. In the course of such research, theory is, we hope, enriched and altered in line with empirical findings, and empirical search is improved and guided by theoretical advances. This is the traditional process by means of which science advances, and I can see no reason to expect such a process to be less advantageous in psychology than in physics.

A particularly important aspect of the theory is the connection it makes between the laboratory study of conditioning phenomena, on the one hand, and clinical work with neurotics (behaviour therapy) on the other; indeed, I have used my hypothesis to formulate a general theory of psychotherapy which tries to explain not only the success of the various behaviour therapies, but also of psychotherapy (in its various manifestations), psychoanalysis, and even spontaneous remission (Eysenck, in press). **Marks** has criticised my whole approach, advocating an atheoretical one, using concepts like "evoking stimulus" and "evoked responses," and avoiding altogether any theoretical treatment in favour of what seems a truly Baconian search for successful ES-ER combinations. He seems to believe that "theoretical advances come from people in the field (in this case clinicians) as well as from those working in the laboratory." He seems to forget that the whole process of behaviour therapy was set in motion by people working in the laboratory who applied (admittedly rather rough and ready) theories of extinction to clinical problems, who elaborated methods of desensitization, flooding, modeling, and the like on a theoretical basis, and who created the groundwork for the applied studies since carried out by Marks and many other clinicians. Here, as always, theory precedes successful and meaningful application, and it seems ungenerous of Marks not to acknowledge his debt to those whose theoretical work made possible the applications he mentions.

Marks makes another criticism that requires an answer; it is fairly general in nature, although for obvious reasons none of the other critics has made this particular point. He seems to suggest that the postulation of "neurosis" as a general category that requires a theory to account for its manifestations has little foundation in fact, and that it requires substantiation by operational definition, and demonstration that the similarities among various neurotic disorders are more important than their differences. Current knowledge, he claims, does not allow this assumption to be made. Such a statement betokens more a lack of knowledge of the literature than a lack of evidence in favour of the assumption; Figure 1, to give but one example, shows the results of a factor analysis of a large number of symptoms and traits, made at the Maudsley Hospital where both Marks and I work; this study of 819 male patients used item sheets filled in by psychiatrists in charge of the patients in question, there being over 500 items, intended to cover every psychiatrically significant aspect of the patients' history, symptoms, and course of illness (Trouton and Maxwell 1956). The figure only illustrates some of the items used, but it clearly demonstrates the existence of precisely that unity of neurotic symptoms which Marks questions as well as an unrelated psychotic factor which does the same job for psychotic disorders. Further evidence will be found in Eysenck (1950, 1961, 1970). The curious method used by psychiatrists to get rid of all neurotic disorders, namely by legislating the term out of existence (DSM-III; **Mahoney**) is surely a novel way of curing patients of their disorders; it does not alleviate the burden of finding an explanation for the occurrence of such regularities as exist in this field. There are undoubtedly anomalies in the postulated concept of "neurosis," but surely there are anomalies in all scientific concepts and theories; this one is no exception.

One difficulty in the general approach that I have advocated in the explanation of neurotic disorders is the fact of *desynchrony of symptoms* (Rachman 1974). The criticism has been made by **Öhman & Ursin** that in my treatment I omitted mention of this important fact; I can only plead that in my original draft there was a section on this topic, but that for reasons of space this had to be cut out. What

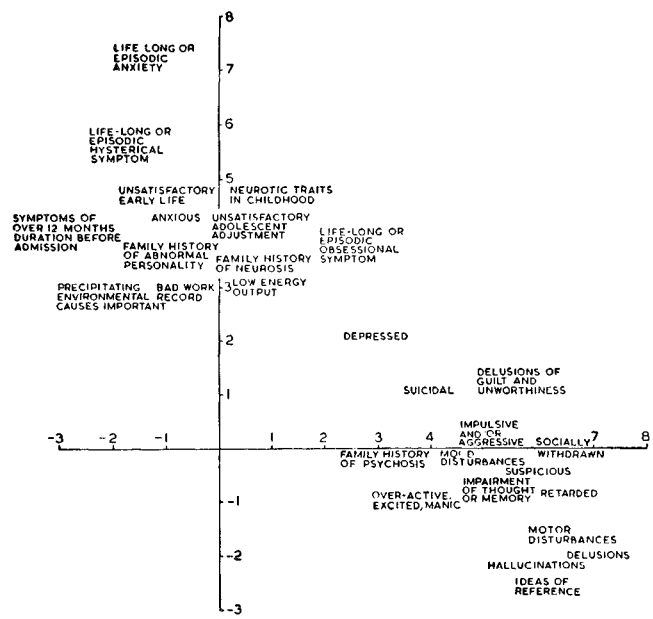

Figure 1. Items defining neuroticism and psychoticism factors. Symptoms or traits of 819 patients at the Maudsley Hospital. Results of a factorial analysis by Trouton and Maxwell. From Eysenck 1961.

can one say in answer to the suggestion that desynchrony argues against some such conception of neurosis as that here presented?

Desynchrony refers to the fact that anxiety and fear do not come as "lumps" (Rachman 1974). The three major aspects of anxiety, namely the physiological, the behavioural, and the introspective (Lang 1970), are far from perfectly synchronized, and may wax and wane almost independently of each other – at least as long as anxiety is not overwhelmingly strong (Hallam, Rachman, and Falkowski 1972; Hodgson and Rachman 1974; Rachman and Hodgson 1974). It may therefore seem that we are wrong in adopting a conditioning theory of fear, when in reality fear and anxiety are not unitary emotions, but are made up of behavioural, autonomic, and cognitive elements that do not cohere together at all closely. How does this fact fit in with our theory?

Quite briefly, we would say that the observed dissociation is more imaginary than real. Synchrony would only have been expected under *ceteris paribus* conditions; but such conditions never obtain. Consider a man who "goes over the top" in trench warfare; his behaviour seems indicative of lack of fear, although his autonomic system may be strongly involved, and his introspections may indicate strong fear. But his behaviour is not determined only by his autonomic responses and his cognitive anxieties; it is also determined by other factors, such as the fear that he will be court-martialled and shot for cowardice if he refuses to go over the top! Or consider a candidate in an examination, whose autonomic system is again strongly involved, but who denies any introspective fears; in his case the possibility exists that other cognitions, related to the examination situation, have preempted his thought processes.

Furthermore, it is well known that autonomic responses are highly specific, both to the person and to the situation; as Thayer (1970) has shown, different autonomic measures do not correlate at all highly together, but they do correlate quite well with introspection – it is as if introspectively we can integrate the sum of all autonomic responses, suitably weighing each in conformity with the situation and without particular patterns of individual reactivity. While therefore there will be considerable desynchrony between changes in introspection and changes in particular electrophysiological recordings, this does not mean that there is any pronounced desynchrony between the weighted sum of all electrophysiological recordings and verbal report (introspection).

Taking these arguments together with the fact that desynchrony mainly appears when relatively weak states of emotional arousal are concerned (i.e. well below the "critical point" in Figure 1 of the target article), we may conclude that while the discovery of desynchrony may present some problems for the theory, these are by no means insuperable. It seems desirable that further detailed work should be done on desynchrony, but using much more comprehensive recording of psychophysiological responses than has been customary hitherto. Even then it is of course quite likely that both the speed of conditioning and the speed of extinction of these components of the anxiety state may be correlated rather less strongly than might at first be supposed; again, there is no reason to expect that verbal responses should be conditioned at the same speed as behavioural or autonomic ones.

Rachman (1977) has made an interesting suggestion, namely that there are different combinations of the three components of the state of anxiety, and the three methods of acquiring fears that he recognizes (conditioning, vicarious, and informational). He says:

"We can hazard the speculation that (for) fears acquired by a conditioning process . . . 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."

He also expects prepared fears to have a large physiological and behavioural component, but nonprepared fears to have a larger cognitive element. These suggestions are worthy of being followed up by suitable experimentation.

As already noted, there are several authors who delineate in more or less detail alternative theories which, they believe, would explain neurotic behaviour and its origin better than mine (e.g. **Wolpe, Kimmel, Wong, Gray, Levis, Terry, Soltysik, Salzinger, Bindra**). This is of course possible, and it must be left to others to evaluate, both theoretically and experimentally, the possibilities opened up in this way. It would not be reasonable here to use up the restricted space available for consideration of criticisms of my theory in order to criticise in turn other theories. Instead of doing this, I would like rather to say a few words in answer to those who prefer some altogether different approach, whether cognitive, operant, or whatever. In particular, I would like to clarify my views about cognitive theories (**Rosenthal**). I have been critical of what to me seems an exaggerated stress on one aspect of the conditioning paradigm, namely the second signalling system, to the exclusion of the first signalling system, and of the curious belief (not, I think, justified by research) that we already possess some general cognitive theory of behaviour. The much more modest claims made by **Mahoney**, who in essence pleads for a combined onslaught by conditioning and cognitive theories on the frightening mystery of motivation and behaviour, are very much in line with my own views. I am as much opposed to the rigid dictates of orthodox behaviourism, with its elimination of mental processes and cognitive events from scientific consideration, as I am to those who would eliminate physiological and biological factors involved in conditioning. Man is a biosocial animal, and much of our trouble in making psychology into a science has been the mutual antagonism of those who would only look at the social, or only at the biological side, sometimes not even paying lip service to those aspects they disregard. Collaboration is always to be preferred to confrontation, and a constructive dialectical exchange, such as is asked for by Mahoney, is clearly the only way to resolve factual disputes and theoretical arguments. [see also Eibl-Eibesfeldt: "Human Ethology" *BBS* 2(1) 1979.]

With respect to the operant approach, several critics are clearly dissatisfied with my neglect of this fundamental set of concepts and theories (e.g. **Öhman & Ursin, Salzinger**). I can only plead lack of space; I believe that operant factors play an important part in nearly all manifestations of neurosis, and that they often help or hinder the process of therapy. I also believe, however, that the crucial element in behaviour therapy of the neuroses is Pavlovian conditioning and extinction. It may be useful to make a distinction between *behaviour therapy* – relevant to neurotic disorders, relying on Pavlovian conditioning and using extinction of conditioned responses as the mode of treatment – and *behaviour modification* – relevant to antisocial behaviour, relying on operant conditioning, and using positive reinforcement to condition approved behaviours. This distinction corresponds in some degree with that between disorders of the first and second kinds, introduced by Eysenck and Rachman (1965), to bring out certain important distinctions between two rather distinct kinds of mental and behavioural disorders; it does not carry with it any notion of an absolute and categorical separation of the two processes. It is very difficult to think of any real-life situation in which one or other of these two processes could be ruled out with any confidence, and even in the laboratory it has proved very difficult if not impossible to do so. This is often mistakenly believed to indicate that the two processes are not different in many ways; such a conclusion does not follow (Gray 1975). The fact that operant methods have not been widely used for the treatment of neurotics, or behaviour therapy for the improvement of antisocial conduct, suggests that in practice the distinction is well recognized. Hence I agree with those who argue for a recognition of operant processes in most if not all actual cases of neurosis and treatment, but I still maintain the primacy of classical conditioning in this field.

The critic who is perhaps closest to my own thinking is **Borkovec**, who first explicates the different levels of explanation in science and indicates the particular place that my own theory might occupy. Within the heuristic conceptual framework provided by the theory, Borkovec suggests important variables that may play a crucial role, in particular "functional CS exposure." His research in this area is of considerable value and provides an indication of the way in which I

had intended my theory to be used, that is, not as a finished law of nature, but rather as a guide to further research, in an attempt to discover the crucial parameters governing the various phenomena postulated by the theory.

Bindra is another critic with whose views I have much sympathy, and I do not feel that it would be impossible to incorporate his suggestions within my theory. In particular his view is that by activating a neural representation of the UCS the $\overline{CS}$ generates a *motivational state* similar to or identical with the one generated by the UCS; this is very close to my own postulation, particularly when we consider this stimulus to have reinforcing or incentive properties. The notion of a central motivational state (CMS) is a very appealing one and fits in very nicely with my theory; there are certainly experiments that can be better explained along Bindra's lines than along more classical lines. I think he would agree that his suggested improvements are not contrary to the intention of my theory, but extend and complement it. But my postulation of a direct CS-UCS relation is based on more than the "old experiments of Franks;" a recent review by Levey and Martin (1979) will make this clear. And Bindra is also wrong in thinking that I postulate that aversive stimuli generate drive, while appetitive stimuli do not; I have suggested several times that sexual stimuli are likely candidates for being "drive producing," as there is much evidence that (a) tumescence can be conditioned, and (b) tumescence constitutes a drive. Admittedly this is merely a suggestion, and crucial experiments are still to be done; nevertheless it illustrates that the view Bindra assigns to me is not really one I would maintain.

Now for some of the specific criticisms and suggestions. **Zuckerman** raises a number of difficult questions, to most of which the answer is not known, although hypotheses can be formulated. Why do we have many more phobias of flying than of car driving? Possibly because of the prepared stimulus of *height* being involved; fear of heights has always been one of the more popular and persistent phobias. Zuckerman's alternative of helplessness is of course another candidate; it is curious that so few clinical psychologists have tried to sort out such alternative hypotheses on an empirical basis. This should not be difficult to do, but until it is done we can only speculate. He is right in stating that pain as such is a very uncommon UCS in neurosis; that is why I suggested many more likely alternatives in human conditioning. Is fear of strangers really not prepared, as Zuckerman states? Introverted neurotics seem to develop such fear with a readiness that suggests that it is not confined to "human infants in the second half of the first year of life." Certainly fear of people is useful from the point of view of evolution; who else does us as much harm as other people? (This is an introvert speaking, although not a neurotic one!)

Wyrwicka points out some of the problems and difficulties attached to the concept of preparedness in humans, and to the postulation of innate fears. We have to re'y more than one would like on the ethological literature dealing with animals; there seems no direct way of experimenting with humans in such a manner as to rule out the possibilities of learning that Wyrwicka enumerates. It is simply the differential rate of learning that gives one a clue, but even that may be doubtful, as several critics have pointed out. The work of Konorski is certainly relevant, as she points out; it is perhaps not sufficiently well known in the west.

One further point may need answering, namely that raised by **Krasner** with respect to Watson's environmentalistic preconceptions. There is no doubt that he acknowledged some *phylogenetic* aspects of human behaviour, such as the innate fear-producing qualities of loud noises, or sudden loss of support. What he nearly always denied was the *ontogenetic* aspect of human diversity, that is, the determination of differences *between* human beings, in intelligence or personality, by heredity. From this point of view his solitary, unsupported statement about little Albert's "constitutional inferiority" is very puzzling. Yet it does suggest, in an environmentalist with a fundamentalist outlook, the homage that vice pays to virtue! I would suggest that there can no longer be any doubt about the importance of genetically based personality factors in linking conditioning theory to the genetic treatment of neurotic disorders. My colleagues and I have been engaged in a large programme of biometric genetic study of personality for a long time (e.g. Eysenck and Prell 1951; Eysenck 1956; Eaves and Eysenck 1975 and 1977), with very positive results, and two reviews of the field testify to the importance and fruitfulness of the genetic approach (Eysenck 1976b, 1977d). Altogether, I have rather played down the importance of individual differences in my target article, because it was mainly concerned with conditioning theory and incubation; in my view no theory of neurosis is viable that does not incorporate individual differences, largely genetic in origin, in its foundations (Hemming 1979).

Dykman draws attention to some interesting similarities between my theory and that of Gantt, involving principles of schizokinesis, autokinesis, and organ system responsibility. There is no doubt about the similarities, and had I had the space for historical treatment, I would gladly have acknowledged Gantt's important theoretical contribution. However, I think it is a little exaggerated to state that "Gantt's autokinesis is Eysenck's incubation." Gantt's explanation of the tendency for behavioural maladjustment to become self-perpetuating and relatively independent of feedback from the environment (which is not quite an accurate reflection of the phenomena I am trying to explain, either), is that this is "something the subject himself contributes, something novel, synthesized out of his experiences, of the traces that remain in the nervous system, and perhaps of functions peculiar to the nervous system of the individual." This seems to me a far cry from the conception that the CR to the $\overline{CS}$ acts as a reinforcement.

Bolles takes up the question of whether fear is peculiar if not unique in supplying the drive for its own motivation; this whole problem of the range of "bootstrapping" is of course an empirical one, and I count it as one of the plus points of my theory that it has brought the problem into focus, and thus enabled an experimental attack to be made on it. But ultimately I think Bolles's final words may be used to sum up the whole controversy. He says that "if we are to use conditioning theory as a useful model of neurosis, then conditioning theory is very much in need of conceptual clarification." This is quite apparent in contrasting the various criticisms made by the experts in this field; they disagree with each other more than they disagree with me! Obviously, unless we have an agreed theory of conditioning, motivation, and extinction, there can be no agreed verdict on my efforts to use these concepts to explain the facts of human neurosis and the effects of therapy on neurotic patients. Is the venture therefore condemned to complete failure from the beginning? I think not. What is usual in science is a kind of spiralling approach to theory development; there never arrives a moment when all are agreed on the substance of the theory, but gradually obvious fallacies are discarded, certain facts become universally acknowledged, some kinds of approach are recognized as useless, others as fruitful, new areas are drawn into the set of explicanda covered by the theory and in turn furnish new evidence for and against certain ways of thinking about the general problems. We certainly know much more about conditioning than we did thirty years ago, even though we are no nearer agreement. New problems have arisen, some of the older ones have turned out to be pseudo-problems; similarly, new solutions have arisen, and some of the older ones have turned out to be pseudosolutions. There is progress, even though there is still dissension. Under these circumstances, clearly my particular contribution cannot prove universally acceptable; this is impossible on simple a priori grounds. All that it can do is to encourage experimentalists to venture into this new field with new hypotheses, trying to verify or invalidate deductions from the combination of orthodox conditioning theory and novel mechanisms suggested here. This is bound to enrich our understanding of both conditioning mechanisms and neurosis, even though the particular theory in question may not survive for very long!

One last point. **Notterman** makes some shrewd criticisms and suggestions and ends up with an even shrewder guess when he says: "Have I stumbled on the truth – is the present effort but a test of a future such venture?" – referring to a more searching and detailed analysis of the problem. He is certainly right; I obviously had to cut short many promising theoretical discussions, and considerations of

empirical data, in cutting down my paper to the prescribed length. Had I discussed all the points raised by the critics, the article would have expanded into a book. Clearly, the next step is to get down to writing such a book, and in doing so I shall benefit considerably from the comments of the various critics who took the time and the trouble to make what in all cases have been thoughtful and valuable comments and suggestions. As critics *ex officio*, they have been kinder to my theory than it deserves; in following up Notterman's hint I hope to benefit from their erudition, their experience, and their wisdom. I am grateful to them all and shall acknowledge their help whenever and if ever I manage to complete the writing up of a more detailed and complete account of my theory than was possible in these pages.

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