

A THEORY OF THE INCUBATION OF ANXIETY/FEAR RESPONSES

H. J. EYSENCK*

Institute of Psychiatry, University of London

Summary—This paper presents a theory of *incubation*, defined as an increment in CR strength occurring during a period of time when only unreinforced presentations of the CS are made, i.e. when traditionally extinction would be expected to occur. Experimental evidence for the occurrence of this phenomenon is cited, both with humans and with animals, and its relevance to the phenomena of neurotic disorder and of behaviour therapy is considered. The theory developed to account for the phenomenon stresses the nocive nature of the CR in aversive conditioning, and suggests that if sufficiently strong this may serve as a reinforcement for the CS-CR link. This theory stresses the importance of the UCR, rather than that of the UCS, in mediating the original conditioning, and suggests the formation of a generalized “nocive response” which is made up of both UCR and CR. Applications of the theory are suggested to desensitization therapy, aversion therapy, “implosion” therapy, and the genesis of neurotic disorders generally.

1. INTRODUCTION

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 are 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 *et al.* (1966), Golin (1961) and Golin and Golin (1966), Kamin (1957, 1963), McAllister and McAllister (1963) and McAllister *et al.* (1965), McMicheal (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 on consolidation of the memory trace; such a theory has been advanced elsewhere in connection with pursuit-rotor learning (Eysenck, 1965), 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

* I am indebted to the Maudsley and Bethlem Royal Research Fund for the support of work leading to this theoretical statement. I am also indebted to Dr. R. Ramsey for making available to me a pre-publication draft of his paper on “incubation” which stimulated me to differentiate more clearly between his use of this term and mine.

consolidation takes time, and that conditioning phenomena do not in this respect differ from ordinary verbal and non-verbal 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 not very strongly appearing phenomena; they merely exemplify certain consequences to be deduced from existing theories.

In this article the term "incubation" will be used in quite a different sense; it will be used to refer to an increment in CR over a period of time when the CS is applied once or a number of times, but without reinforcement. Normally this procedure would give rise to extinction; the fact that it may instead give rise to an increment in the size of the CR (an increment which may be substantially larger than that which in control experiments follows paired CS/UCS exposures) makes the alternative explanations given of the "empty interval incubation" experiments inapplicable. We must take care to keep these two types of incubation clearly separate, and the use of a common name, such as "incubation", is unfortunate in that it disguises this difference. It is for this reason that the writer has previously referred to the second type of incubation as the "Napalkov phenomenon" (Eysenck, 1967).

2. THE INCUBATION PHENOMENON

Napalkov (1963) working with dogs, found that various noxious 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. Campbell *et al.* (1964), working with humans, gave single-trial temporary interruptions of respiration lasting for about 100 sec, an extremely harrowing experience. One hundred extinction trials were given over a period of 3 weeks, and autonomic responses recorded. Instead of extinction, there appeared an increase in size of the CER, together with stimulus generalisation. In both cases, therefore, the administration of the CS unaccompanied by UCS resulted in an increment of the CR, rather than in extinction. This effect seems far more notable, and far more difficult to explain, than the consolidation effect studied by most previous writers under the title of "incubation."

Lichtenstein (1950) reported on the inhibition of feeding responses in dogs following upon shock administered while the eating response was occurring. 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 days 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 behavior does not usually include such

manifestations of anxiety. Furthermore, as components of an anxiety reaction, these types of behavior 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 specifically stress the importance of non-specific CRs and point out the importance of recording as many of these as possible. They summarize 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 number 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 and Gantt, (1958, 1960a, 1960b) and Galbrecht *et al.* (1960) 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 (see also Gantt, 1953.) . . . 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." (Dykman *et al.*, 1965, p. 228.) 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 will return later to a discussion of incubation as related to personality, with special reference to humans. Here let us 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 Cooks and Harri, 1937, and Warner, 1938.)

Studies of "partial irreversibility" of conditioned fear responses, such as those of Solomon *et al.* (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 here put forward. However, their data are complicated by the fact that these experiments employed avoidance learning paradigms, so that simple incubation is 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, which sets aside the few studies under consideration here from those reviewed by McAllister and McAllister (1967).

It should be noted, however, that even the experiments giving rise to the traditional notion of "incubation", which we have explained in terms of reminiscence and consolidation, may include an element of genuine incubation as here defined. Admittedly no \overline{CS} (unreinforced CS) is in fact presented during the period intervening between conditioning and testing after rest, but this is true only of stimuli chosen by the E and intentionally presented to the animal or human S. In addition, as we have pointed out, there are

many other stimuli associated with apparatus, room, experimenter, temporal sequence etc which go to make up a $\Sigma \overline{CS}_n^a$; some of these are likely to have occurred during the rest period supposedly free of \overline{CS} . As this point was not considered by the writers of the papers quoted, it is impossible to be certain on this point, or to evaluate the frequency or the strength of stimulation thus received; the point is mentioned rather for the sake of completeness, and to indicate that in future work it may repay attention.

A final series of experiments which may be relevant to our concept of incubation relates to higher-order conditioning, i.e. attempts to use the CS of one experiment as the UCS in another. Work on "covert sensitization" (Cautela, 1967) or "verbal aversion" techniques (Anant, 1967) suggests that verbal and imagined stimuli (i.e. CSs acquired through a long-continued previous process of conditioning) may be used as UCSs and produce very strong responses (Ashem and Donner, 1968, Gold and Neufeld, 1965). This work is too recent, and its theory too uncertain as yet, to do more than mention it; the absence of extinction during a long series of evocation of what must be considered an aversive \overline{CS} suggests that this work may in fact be relevant to our theory. More than that cannot be said at the moment.

3. A THEORY OF INCUBATION

In now turning to a theoretical account which 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 a CS unaccompanied by a UCS (to be symbolized in this paper as \overline{CS} , while CS stands for a reinforced stimulus) always provokes a decrement in CR strength, but that for reasons to be explained it also provokes 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 here be concerned with the theoretical explanation or 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 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 NRs (nocive responses.) Through the intermediacy of the UCS, CS has become associated with the NRs and signals their arrival to the organism. (For reasons which will become obvious later, we prefer the term NR in this connection to the use of 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 putting over a theory which departs in some ways from the usual one.) Each reinforcement (which may be defined as a NR following 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 strength associating CR and NR.

It is here suggested 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 feed-back 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.*

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 behavior." What we propose to add is that fear, so generated, is itself a painful event, and the stimuli associated with it (i.e. \overline{CS}) therefore by classical conditioning, come to evoke more fear, thus producing a positive feedback. This mechanism is well known descriptively in psychiatric disorders; it resembles somewhat Seneca's famous saying about "having nothing to fear but fear itself." Take two clinical examples. Mr X suffers from impotence on a particular occasion, due to drink, fatigue, or illness; the CSs associated with the occasion produce fear/anxiety as a CR. On the next occasion these CRs follow upon \overline{CS} and cause reciprocal inhibition of sexual reflexes; this failure causes additional anxiety/fear CRs, which produce an even stronger reaction the third time, thus setting in motion a positive feedback circle which continues without the necessity of a new UCS-CS combination. Or consider a person unable to go sleep to because of overfeeding, or noise; his failure (UCR) is associated with anxiety responses, which thus condition to the CSs involved (bed, night, etc.) The next night the UCS is missing, but the \overline{CS} -CR association produces anxiety; the patient is worried that he may not be able to go to sleep, and now the worry keeps him awake. Thus his anxiety builds up by each repetition of the \overline{CS} . Biologically, the principal usefulness of the extinction mechanism may lie in the fact that it interferes

* Traditionally we would denote these NRs as response-produced stimuli, in the sense that autonomic responses, which are measurable, such as changes in heart rate, breathing, cessation of stomach contractions etc. 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 CR itself which acts as reinforcer, but rather the response-produced stimuli; not the autonomic, hormonal and muscular reactions themselves but rather the experience of fear/anxiety based upon them. Insofar as these CR-produced are *identical* with the UCR-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 nocive and aversive.

(successfully in most cases) with the development of the positive feedback implicit in the conditioning principle.

The same mechanism must be assumed to be present when 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 NRs achieved on the basis of rather weak UCRs (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 UCS; 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 fear + pain; 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 which 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.*

The considerations discussed so far are likely to meet with some criticisms 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 which 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 experimenter, who administers the UCS, while the S experiences the pain. However, from the S's point of view he does not feel a shock (UCS) which produces pain (UCR); he experiences a painful shock, i.e. UCS and UCR are experienced simultaneously, and not as separate, consecutive entities. It is this Gestalt-like NR which is being linked with the CS through contiguity, and to which CR eventually adds another increment of pain/fear which is introspectively very difficult or even impossible to differentiate from

* 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 which was a combination of CR and UCR, showing clear traces of both, and stronger than either alone. This combined response quickly extinguished, probably due to 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. Brady's (1965) experiment with the "executive" monkey may perhaps be cited as additional evidence; here both monkeys experience UCS and UCR, but only the "executive" monkey receives the CS, and only he acquires ulcers (here regarded as a "response") and dies. Clearly CS and the CR produced by it have additional properties to merely signalling the arrival of the UCR, and the CR adds importantly to the emotional response mediated by the UCR. (This experiment used instrumental conditioning, and is therefore only indirectly relevant.)

the original NR. In other words, the differentiation between UCS and UCR reflects pre-occupation with control (the UCS is under the control of the E, and causes the UCR in the sense that what the E does produces a response in the S;) from the S's point of view (and after all it is in the subject that the process of conditioning takes place) the differentiation is of doubtful relevance and value. UCS and UCR are temporally close together—so close that the S often cannot differentiate between them—and in consequence it is difficult to disentangle the links which 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, e.g. 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 is said to occur which is weak at best and often non-existent. In practice it is well known (Franks 1964, 1966) that strong, conditioned responses are obtained only 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 which has been hidden in most researches 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 *et al.* (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 at Kimble (1961) points out, "it is apparent that backward conditioning in which the UCS precedes the CS leads to little conditioning" (p. 158.)

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, which 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.

The theory here presented is probably deficient in not taking explicitly into account Pavlov's "second signalling system." Advocates of cognitive-type 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 *et al.* (1967) have demonstrated the possibility of acquiring voluntary control over autonomic reactions, such as heart rate. A clear demarcation often appears in behavior therapy between autonomic-behavioral and cognitive effects; Lang and Lazovik (1963) have reported immediate behavioral effects of their desensitization 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 realized, 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-type 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.

4. INCUBATION AND BEHAVIOR THERAPY

The theory of incubation (as contrasted with simple consolidation and reminiscence effects) here stated has important corollaries for behavior therapy. We will consider several of these, the first related to the genesis of neurotic disorders. Consider the three-stage theory of neurosis suggested by Eysenck and Rachman (1965), according to which (1) a traumatic event (UCS) leads to emotional turmoil (UCR); (2) a previously neutral stimulus or set of stimuli (CS) becomes conditioned to the UCS and now has the power to produce emotional turmoil as a CR. (3) Extinction is prevented by instrumental conditioning, the organism being rewarded for avoiding $\overline{\text{CS}}$ by reduction in strength of CR, thus making "reality testing" impossible (avoidance conditioning.) This account may serve to describe certain types of neurotic disorders, but frequently psychiatric reports show a rather different course which would be difficult to explain in terms of this three-stage theory. A traumatic event may fail to show immediate symptoms, or where there are immediate symptoms they may be rather weak and quickly die out; it is later, often after a considerable period of time, that symptoms appear and grow, sometimes again over a considerable period of time, until they reach the proportions of a full-blown neurosis. Incubation would seem to suggest itself as a rational mechanism which could account for facts such as these; $\overline{\text{CS}}$ presentation at first gives only slight effects, but as long as these are larger than the decremting effects of extinction they are likely on our showing to build up into a proper NR which may even be more debilitating than the original UCR. Clearly the actual course of a neurotic case history is much more complex than this, with partial reinforcement interspersed among casual and non-systematic CS evocations, and with the intrusion of adventitious emotion-arousing incidents which may be irrelevant to the CR but act so as to sensitise the organism. However, in principle the notion of incubation makes the general theory more life-like and less inapplicable to reported histories of neurotic illness.

Let us now look at aversion therapy. Here we may consider a point emphasised by Rachman and Teasdale (1969), when they say: "The surprising thing about aversion therapy is *not* that its effects are uncertain, but rather that it works at all." The reasons why this is surprising are the same as the reasons why it is surprising that all neurotic disorders do not extinguish; indeed, it is useful to regard aversion therapy as the experimental inculcation of an experimental neurosis in a S—with the sole proviso that this "neurosis" is not maladaptive, but on the contrary counteracts maladaptive practices on the part of the S. This "neurosis" is subject to extinction, and in virtue of its tenuous nature (laboratory provenance, weak UCS, few CS-UCS pairings) extinction under reality conditions should be rapid. Yet, in fact, although extinction (relapse) does occur in a fair portion of cases (Eysenck and Rachman 1965), yet it takes a long time, and often does not seem to occur at all. Incubation would seem to present us with a mechanism which would counteract extinction, and lead to a positive feedback preserving and even strengthening the encapsulated "neurosis".

Next, let us consider incubation in relation to desensitization therapy and Stampfl's "implosion" therapy (London, 1964; Stampfl, 1967; Stampfl and Levis, 1968, 1969.) The latter is a method similar to what Polin (1959) has called "flooding", i.e. the presentation of \overline{CS} evoking strong CRs; Stampfl appears to suggest that the stronger the CR the better the treatment. Both Polin and Stampfl expect quick extinction to take place, and the former adduces some animal experiments in this connection, while the latter claims something like 100 per cent success in treatment of human neuroses (see also Hogan, 1966, and Hogan and Kirchner, 1967.) Malleon (1959) has also reported successful treatment along these lines in human subjects, as have Wolpin and Raines (1966.) Also relevant are response prevention technique studies like those of Baum (1966), Black (1958) Page (1955) and Weinberger (1965.) Kimble and Kendall (1953) in the animal field, and Rachman (1966) in the human field, failed to find supporting evidence, and concluded that some form of desensitization was more effective. Theoretically, "implosion" therapy should produce extinction quickly and efficiently as long as we consider only orthodox views; the introduction of incubation into the process suggests that "implosion" may either lead to extinction or to aggravation of the neurotic disorder, depending on which process is the stronger at the time of initiation; this in turn would presumably depend on such factors as strength of the UCR, of the CR, and the personality of the subject. We will return to these points again later. Here let us note merely that successes reported with human Ss in experimental situations (like the Wolpin and Raines study) have dealt with non-neurotic Ss suffering from monosymptomatic phobias; one would predict that with genuine neurotics aggravation rather than cure would result from "implosion" therapy. However, this whole region is clearly of considerable interest and relevance to our theory, and much more empirical study of high quality will be needed before any definite conclusions can be drawn.

The same can perhaps be said of a related topic which arises in connection with desensitization therapy, namely the frequently noted tendency for patients to relapse when in the course of therapy a part of the habit-family hierarchy is touched upon which is still too sensitive to be sufficiently counteracted by relaxation (Wolpe, 1958.) This reaction is of course akin to "implosion" therapy or "flooding", but instead of being welcomed as leading to extinction it is shunned by practitioners as retarding the curative process. Yet curiously enough there is nothing in orthodox theory to justify this practice; as we have already noticed in the last paragraph, the presentation of the \overline{CS} should not lead to an increment in CR, however anxiety-producing the \overline{CS} . The fact that such incremental reactions are widely documented in the literature on behavior therapy (Eysenck and Rachman, 1965) suggests the need for a concept like incubation, without of course proving that the particular theory of incubation here adopted is in fact the correct one.

It will in any case be clear that the incubation process envisaged in relation to the formation of neurosis and to aversion, desensitization and "implosion" therapy is completely theoretical; while it fits in with clinically suggested facts there is no direct evidence, such as might be provided by direct CR measurement under conditions of \overline{CS} presentation. Such experiments should not be too difficult to devise, and until they have in fact been carried out not too much stress should be laid on the potential usefulness of this theory. Even in the laboratory the support for it rests on just a few experiments, none of which can be said to be unequivocal; alternative hypotheses have not been entirely ruled out. Yet the potential usefulness of the incubation hypothesis, whether in the precise form given it here or not, is so large that it may repay experimental study.

In devising such experiments, it may be useful to specify certain parameter values which may make more likely the evocation of incubation responses. Two such parameters in particular suggest themselves, one related to the strength of the UCR, the other to the personality of the subject. As regards the former, it would seem likely that strong, traumatic UCRs are more likely than weak ones to produce incubation phenomena; weak UCRs are likely to lead to extinction prevailing over incubation. The literature certainly suggests that this variable is an important one; Napalkov, Campbell *et al.* and Solomon and his associates all used what were unusually strong stimuli. (In the case of Napalkov this cannot be said with any degree of assurance as his account is not explicit on this point.) It will be noted that we have specified that the UCR should be strong, rather than the UCS; the two will of course usually be correlated, but this correlation is far from perfect. Identical electric shocks of medium intensity will have different effects on two people of whom one is already terrified of electricity, while the other is well accustomed to dealing with it; identical UCSs will lead to quite different UCRs. This obvious point is often neglected in theoretical discussions.

The other parameter in question also has a bearing on the possible lack of relation between UCS and UCR. People high on neuroticism-anxiety-emotionality are more likely to show a strong UCR to a given UCS than are people low on this personality dimension; hence they are more likely to demonstrate incubation phenomena. People who condition well and strongly are more likely to show incubation, which after all is a conditioning phenomenon; hence we may postulate that the personality dimension of extraversion-introversion, which is associated with cortical arousal and inhibition, and through this with conditioning, will also be involved in the genesis of incubation phenomena (Eysenck and Levey, 1967.) These relationships, if confirmed, may explain why apparently similar environmental conditions lead to incubation in some subjects, to extinction in others. The complex dynamic interplay involved will obviously require a great deal of experimental clarification.*

In summary, then, it is suggested that (1) there are two varieties of incubation so-called, of which the first (increment of CR after period of non-evocation) is probably nothing more than a slight excess of consolidation-remembrance effect over retroactive inhibition. (2) A second variety of incubation, of much greater interest and the only kind here considered, refers to increments in CR after several evocations of the unreinforced CS. (3) Incubation so conceived has some experimental backing, both in animal work and in work with humans. (4) Incubation seems a necessary concept to account for certain phenomena in the formation of neurotic disorders, and also in the methodology of aversion therapy. (5) A theory of incubation can be envisaged which proposes that CRs are themselves reinforcing, and add their force to the UCR, or may take its place when CS is not reinforced. (6) Incubation is postulated to occur whenever aversive conditioning takes place, but is usually not observed because extinction, which is also postulated to occur, is stronger than incubation, thus leading to a performance decrement when CS is not reinforced. (7) When CS is reinforced,

* The relationship between strength of UCR and personality is of course reciprocal as pointed out by Savage and Eysenck (1964). They quote as an example the studies of Rosenbaum (1953, 1956) who demonstrated that threat of a strong shock led to greater generalisation than did threat of a weak shock; similarly anxious subjects showed greater generalisation to identical stimuli than did non-anxious subjects. It is likely that among the CRs which one would want to measure in an experimental test of the hypothesis here presented would be the generalisation of stimuli and responses; several authors have noted greater generalisation in the consolidation-remembrance type of incubation phenomenon mentioned at the beginning of this paper (e.g. Desiderato and Wassarman, 1967; Desiderato *et al.*, 1966; Lacy and Smith, 1954; McAllister and McAllister, 1963; Perkins and Weyant, 1958; Thomas and Lopez, 1962.)

incubation increments are added to the UCR and counteract adaptation/habituation phenomena. (8) Incubation is more likely to occur in certain types of persons, such as subjects with high scores on neuroticism-anxiety-emotionality inventories. (9) Incubation is more likely to be observable where the UCR is exceptionally strong. (10) In general, it is suggested that orthodox theories of conditioning throw too much weight on the UCS, and tend to neglect the importance of the UCR.

REFERENCES

- ANANT S. (1967) A note on the treatment of alcoholics by a verbal aversion technique. *Can. J. Psychol.* **8**, 19-22.
- ANDERSON O. D. and PARMENTER R. (1941) A long-term study of the experimental neurosis in the sheep and dog. *Psychosom. Med. Monogr. Suppl.*
- ASHEM B. and DONNER L. (1968) Current sensitization with alcoholics; a controlled replication. *Behav. Res. & Therapy* **6**, 7-12.
- BAUM M. (1966) Rapid extinction of an avoidance response following a period of response prevention in the avoidance apparatus. *Psychol. Rep.* **18**, 59-64.
- BINDRA D. and CAMERON L. (1953) Changes in experimentally produced anxiety with the passage of time: Incubation effect. *J. exp. Psychol.* **45**, 197-203.
- BLACK A. H. (1958) The extinction of avoidance responses under curare. *J. comp. physiol. Psychol.* **51**, 519-525.
- BRADY J. V. (1965) Ulcers in "executive" monkeys. In *Frontiers of psychological research* (Ed. COUPERSMITH S.). W. H. Freeman, London.
- BREZNITZ S. (1967) Incubation of threat: duration of anticipation and false alarm as determinants of the fear reaction to an unavoidable frightening event. *J. exp. Res. Person.* **2**, 173-179.
- BRIDGER W. N. and MANDEL I. J. (1964) A comparison of GSR fear responses produced by threat and electric shock. *J. Psychiat. Res.* **2**, 31-40.
- BRUSH F. R. (1964) Avoidance learning as a function of time after fear conditioning and unsignalled shock. *Psychonomic Sci.* **1**, 405-406.
- BRUSH F. R. and LEVINE S. (1966) Adrenocortical activity and avoidance learning as a function of time after fear conditioning. *Physiol. Behav.* **1**, 309-311.
- CAMPBELL D., SANDERSON R. E. and LAVERTY S. G. (1964) Characteristic of a conditioned response in human subjects during extinction trials following a simple traumatic conditioning trial. *J. abnorm. soc. Psychol.* **68**, 627-639.
- CAUTEA J. R. (1967) Covert sensitization. *Psychol. Rep.* **20**, 459-468.
- COOK S. W. and HARRIS R. E. (1937) The verbal conditioning of the galvanic skin reflex. *J. exp. Psychol.* **21**, 202-205.
- DENNY M. I. and RITCHMAN R. E. (1962) The basis of maximal "Kamin effect" in rats. *J. Comp. physiol. Psychol.* **55**, 1069-1070.
- DESIDERATO O., BUTLER B. and MEYER C. (1966) Changes in fear generalisation gradients as a function of delayed testing. *J. exp. Psychol.* **72**, 618-682.
- DESIDERATO O. WASSARMAN M. E. (1967) Incubation of anxiety in effect on generalisation gradients. *J. exp. Psychol.* **74**, 506-510.
- DINSMOOR J. A. (1950) A quantitative comparison of the discrimination and reinforcing functions of a stimulus. *J. exp. Psychol.* **40**, 458-472.
- DIVEN K. (1937) Certain determinants in the conditioning of anxiety reactions. *J. Psychol.* **3**, 291-308.
- DYKMAN R. A. and GANTT W. H. (1958) Cardiovascular conditioning in dogs and humans. In *Physiological Basis of Psychiatry* (Ed. GANTT W. H.), pp. 171-195. C. C. Thomas, Springfield.
- DYKMAN R. A. and GANTT W. H. (1960a) A case of experimental neurosis and recovery in relation to the orienting response. *J. Psychol.* **50**, 105-110.
- DYKMAN R. A. and GANTT W. H. (1960b) Experimental psychogenic hypertensions: blood pressure changes conditioned to painful stimuli (Schizokinesis). *Bull. Johns Hopkins Hosp.* **107**, 72-89.
- DYKMAN R. A., GANTT W. H. and WHITEHORN J. C. (1956) Conditioning on emotional sensitization and differentiation. *Psychol. Monogr.* **70**, 15.
- DYKMAN R. A., MACK R. L. and ACKERMAN P. T. (1965) The evaluation of autonomic and motor components of the unavoidance conditioned response in the dog. *Psychophysiology* **1**, 209-230.
- DYKMAN R. A., MURPHREE O. D. and ACKERMAN P. T. (1966) Litter patterns in the offspring of nervous and stable dogs: II. Autonomic and motor conditioning. *J. nerv. ment. Dis.* **141**, 419-431.
- ESTES W. K. (1943) Discriminative conditioning: I. A discriminative property of conditioned anticipation. *J. exp. Psychol.* **32**, 150-155.

- ESTES W. K. (1948) Discriminative condition. II. Effects of a Pavlovian conditioned stimulus upon a subsequently established operant response. *J. exp. Psychol.* **38**, 173-177.
- EYSENCK H. J. (1965) A three-factor theory of reminiscence. *Br. J. Psychol.* **56**, 163-181.
- EYSENCK H. J. and LEVEY A. (1967) Konditionierung, Introversion-Extraversion und die Stärke des Nervensystems. *Z. Psychol.* **174**, 96-106.
- EYSENCK H. J. and RACHMAN S. (1965) *The Causes and Cures of Neurosis*. Routledge and Kegan Paul, London.
- EYSENCK H. J. (1967) Single-trial conditioning, neurosis and the Napalkov phenomenon. *Behav. Res. & Therapy* **5**, 63-65.
- FEHR F. S. and STERN J. A. (1965) Heart rate conditioning in the rat. *J. Psychosom. Res.* **8**, 441-453.
- FRANKS C. M. (1963) Behaviour therapy, the principles of conditioning and the treatment of the alcoholic. *Quart. J. stud. Alcohol.* **24**, 511-529.
- FRANKS C. M. (1966) Conditioning and conditioned aversion therapies in the treatment of alcoholics. *Int. J. Addict.* **1**, 61-98.
- GALBRECHT C. O., DYKMAN R. A. and PETERS J. E. (1960) The effect of traumatic experiences on the growth and behavior of the rat. *J. gen. Psychol.* **50**, 227-251.
- GOLIN S. (1961) Incubation effect: Role of awareness in an immediate versus delayed test of conditioned emotionality. *J. abnorm. soc. Psychol.* **63**, 534-539.
- GOLIN S. and GOLIN A. R. (1966) Incubation and inhibition. *J. exp. Psychol.* **71**, 208-211.
- GOLD S. and NEUFELD I. L. (1965) A learning approach to the treatment of homosexuality. *Behav. Res. & Therapy* **2**, 201-204.
- HOGAN R. (1966) Implosive therapy in the short term treatment of psychotics. *Psychotherapy* **3**, 25-31.
- HOGAN R. A. and KIRCHNER J. H. (1967) A preliminary report of the extinction of learned fears via short-term implosive therapy. *J. abnorm. Psychol.* **72**, 106-111.
- HULL C. L. (1949) Stimulus intensity dimension (V) and stimulus generalisation. *Psychol. Rev.* **56**, 67-76.
- JOHN G. R. (1967) *Mechanisms of memory*. Academic Press, New York.
- KAMIN L. J. (1957) The retention of incompletely learned avoidance response. *J. comp. physiol. Psychol.* **50**, 457-460.
- KAMIN L. J. (1963) Retention of an incompletely learned avoidance response: some further analyses. *J. comp. physiol. Psychol.* **56**, 713-718.
- KIMBLE G. I. (1961) *Hilgard and Marquis' Conditioning and Learning*. Appleton-Century-Crofts, New York.
- KIMBLE G. and KENDALL J. (1953) A comparison of two methods of producing experimental extinction. *J. exp. Psychol.* **45**, 87-89.
- LACEY J. I. and SMITH R. L. (1954) Conditioning and generalisation of unconscious anxiety. *Science* **120**, 1045-1052.
- LADER M. H. and WING L. (1966) Physiological measures, sedative drugs and morbid anxiety. *Maudsley Monogr.* No. 14.
- LANG P. J. and LAZOVIC P. O. (1963) Experimental desensitization of a phobia. *J. abnorm. soc. Psychol.* **66**, 519-525.
- LANG P. J., SROUFE I. and HASTINGS J. E. (1967) Effects of feedback and instructional set on the control of cardiac-rate variability. *J. exp. Psychol.* **75**, 425-431.
- LICHTENSTEIN C. E. (1950) Studies of anxiety: I. The production of a feeding inhibition in dogs. *J. comp. Physiol. Psychol.* **43**, 16-29.
- LIDDELL H. G. (1944) Conditioned reflex method and experimental neurosis. In *Personality and the behaviour disorders* (Ed. MCV HUNT J.). Ronald Press, New York.
- LONDON P. (1964) *The modes and morals of psychotherapy*. Holt, Rinehart & Winston, New York.
- MALLESON, N. (1959) Panic and phobia. *Lancet* **i**, 225-227.
- MARTIN I. and LEVEY A. (1968) *The genesis of the classical conditioned response*. Pergamon Press, Oxford.
- MCALLISTER V. R. and MCALLISTER E. E. (1963) Increase over time in the stimulus generalization of acquired fear. *J. exp. Psychol.* **65**, 576-582.
- MCALLISTER W. R. and MCALLISTER D. E. (1965) Variables influencing the conditioning and the measurement of acquired fear. In *Classical conditioning: 9th Symposium* (Ed. GRABRARY W. F.), pp. 172-191. Appleton-Century-Crofts, New York.
- MCALLISTER D. E. and MCALLISTER W. R. Incubation of fear: an examination of the concept. *J. exp. Res. Person.* **2**, 180-190.
- MCALLISTER W. R., MCALLISTER D. E. and FRANCHISA J. J. (1965) Dependence of equality judgements upon the temporal interval between stimulus presentations. *J. exp. Psychol.* **30**, 602-605.
- McMICHAEL J. S. (1966) Incubation of anxiety and instrumental behavior. *J. comp. physiol. Psychol.* **61**, 208-211.
- MEDNICK M. T. (1957) Mediated generalization and the incubation effect as a function of manifest anxiety. *J. abnorm. soc. Psychol.* **55**, 315-321.

- MOWRER O. H. (1938) Preparatory set (expectancy)—a determinant in motivation and learning. *Psychol. Rev.* **45**, 62–70.
- MOWRER O. H. (1956) Two-factor learning theory reconsidered, with special reference to secondary reinforcement and the concept of habit. *Psychol. Rev.* **63**, 114–128.
- NAPALOV A. V. (1963) Information process of the brain. In *Progress of Brain Research* (Eds. WIENER N. and SEFADE J. C.), Vol. 2, pp. 59–69. Nerve, Brain and Memory Models.
- PAGE H. A. (1955) The facilitation of experimental extinction by response prevention as a function of the acquisition of a new response. *J. comp. physiol. Psychol.* **48**, 14–16.
- PERKINS C. C. and WEYANT R. G. (1958) The interval between training and test trials as a determiner of the slope of generalisation gradients. *J. comp. physiol. Psychol.* **51**, 596–600.
- POLIN A. T. (1959) The effect of flooding and physical suppression as extinction techniques on an anxiety-motivated avoidance locomotor response. *J. Psychol.* **47**, 253–255.
- RACHMAN S. (1966) Studies in desensitization—II: Flooding. *Behav. Res. & Therapy* **4**, 1–6.
- RACHMAN S. and TEASDALE J. D. Aversion therapy. In *Behavior Modification* (Ed. FRANKS C. M.). (To be published).
- SALTZ G. and ASDOURIAN D. (1963) Incubation of anxiety as a function of cognitive differentiation. *J. exp. Psychol.* **66**, 17–22.
- SAVAGE R. D. and EYSENCK H. J. (1964) Definition and measurement of emotionality. In *Experiments in Motivation* (Ed. EYSENCK H. J.). Pergamon Press, Oxford.
- SCHACHTER S. and SINGER J. E. (1962) Cognitive, social, and physiological determinants of emotional state. *Psychol. Rev.* **69**, 379–399.
- SEWARD J. P. (1950) Secondary reinforcement as tertiary motivation: a revision of Hull's revision. *Psychol. Rev.* **57**, 362–374.
- SEWARD J. P. (1951) Experimental evidence for the motivating function of reward. *Psychol. Bull.* **48**, 130–149.
- SEWARD J. P. (1952) Introduction to a theory of motivation in learning. *Psychol. Rev.* **59**, 405–413.
- SEWARD J. P. (1956) Drive, incentive, and reinforcement. *Psychol. Rev.* **63**, 195–203.
- SOLOMON R. L., KAMIN L. J. and WYNNE L. C. (1953) Traumatic avoidance learning: the outcome of several extinction procedures with dogs. *J. abnorm. soc. Psychol.* **48**, 291–302.
- SOLOMON R. L. and WYNNE L. (1953) Traumatic avoidance learning. *Psychol. Monogr.* **67**, 4.
- SOLOMON R. L. and WYNNE L. (1954) Traumatic avoidance learning: the principles of anxiety conservation and partial irreversibility. *Psychol. Rev.* **61**, 353–385.
- SPENCE K. W. (1956) *Behavior Theory and conditioning*. Yale University Press, New Haven.
- STAMPFL T. G. (1967) Implosive therapy, Part I: The theory. In *Behavioral Modification Techniques and the Treatment of Emotional Disorders* (Ed. ARMITAGE S. G.). V. A. Publications, Michigan.
- STAMPFL T. G. and LEVIS D. J. Implosive therapy—a behavioral therapy? *Behav. Res. & Therapy* **6**, 31–36.
- STAMPFL T. G. and LEVIS D. J. Implosive therapy. In *Handbook of Direct and Behavior Psychotherapies* (Ed. Jurjevich R. M.). North Carolina Press, North Carolina.
- STERN J. G. and WARD T. J. (1961) Changes in cardiac response of the albino rat as a function of electroconvulsive seizures. *J. comp. Physiol. Psychol.* **54**, 389–391.
- STERN J. G. and WARD T. J. (1962) Heart rate danger during avoidance conditioning in the male albino rat. *J. Psychosom. Res.* **6**, 157–162.
- TARPY R. M. (1966) Incubation of anxiety as measured by response suppression. *Psychonomic Sci.* **4**, 189–190.
- THOMAS D. R. and LOPEZ L. J. (1962) The effects of delayed testing on generalization slope. *J. comp. physiol. Psychol.* **55**, 541–544.
- VALINS S. (1966) Cognitive effects of pulse heart rate feedback. *J. person. Soc. Psychol.* **4**, 400–408.
- VALINS S. and RAY A. A. (1967) Effects of cognitive desensitization on avoidance behavior. *J. person. soc. Psychol.* **7**, 345–350.
- WALKER R. C. (1942) Effect of a discriminative stimulus transferred to a previously unassociated response. *J. exp. Psychol.* **31**, 312–321.
- WEINBERGER N. M. (1965) Effects of detainment on extinction of avoidance responses. *J. comp. physiol. Psychol.* **60**, 135–138.
- WOLPE J. (1958) *Psychotherapy by reciprocal inhibition*. Stanford University Press, Palo Alto.
- WOLPIN M. and RAINES J. (1966) Visual imagery, expected roles and extinction as possible factors in reducing fear and avoidance behavior. *Behav. Res. & Therapy* **4**, 25–37.