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CASE HISTORIES AND SHORTER COMMUNICATIONS

The theory of incubation: a reply to Bersh

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Summary—Bersh's critique of Eysenck's theory of incubation is summarized, and some arguments are presented to indicate that while some criticisms are pertinent, they do not invalidate the theory.

The writer has proposed a theory of neurosis which avoids some of the difficulties of Watson's (Watson and Rayner, 1920) original conditioning theory, according to which neurotic 'symptoms' are conditioned emotional responses, and the skeletal movements executed in response to these autonomic CRs (Eysenck, 1968, 1976, 1979, 1980). The main difficulties which this theory was designed to cope with were:

(1) According to classical theory, presentation of the unreinforced CS (symbolized as $\overline{CS}$) should be followed by extinction, yet neurotic CRs persist in spite of numerous presentations of $\overline{CS}$.
(2) According to classical theory, single-trial conditioning should start with a traumatic US, yet such USs are infrequent in the development of peacetime neuroses.
(3) CRs in classical conditioning theory are not expected to exceed in strength URs, yet typically the growth of neurotic CRs is insiduous, CRs growing in strength with repeated presentations of $\overline{CS}$.

It was suggested that these difficulties arose as a consequence of a form of a law of extinction which was not in accord with the facts: already in 1956 Razran in his review of the evidence pointed out that

> "extinction continues to be clearly a less than 100 per cent phenomenon. Instances of difficult and even impossible extinction are constantly reported by classical CR experiments." (p. 39)

And he goes on to state that CRs based on strong USs may be quite unextinguishable, and that in particular

> "CRs the magnitude of which approximates those of their URs are practically impossible to extinguish." (p. 41)

Razran, disillusioned with existing theories of extinction, went on to formulate his own, which was essentially a two-stage theory, of which only the first stage is here relevant. As he puts it,

> "the automatic deconditioning in the early stage of extinction is a direct result of the loss of the interoceptive and the proprioceptive conditional stimuli (feed-back 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." (p. 45)

In developing his theory, Razran states that

> "the dropping out of the interoproprioceptive components .... is simply a function of the fact that the CR is smaller in magnitude than the UR. If the magnitudes of the two are close to each other, the extinction of the CR should be slow or impossible, which is exactly what known findings indicate."

To this formulation the writer added two related additions.

(1) In Pavlovian B conditioning (Grant, 1964), the CR is identical with the UR, or at least very similar, whereas in Pavlovian A conditioning this is not so; hence in Pavlovian B conditioning the likelihood of extinction would be less.

(2) If the magnitude of the CR and the UR are close together, and if they are identical or reasonably similar, then surely the CR should act in a fashion similar to the UR, and hence provide substitute reinforcement for the whole conditioning sequence, leading to a positive feedback which was called "incubation" (Eysenck, 1968).

This phenomenon is the exact opposite to the one described by Razran when he pointed out that

> "up to a certain limit, the reduction of the CR should proceed as a sort of self-propelling mechanism: the reduced CR reducing the CS components and the reduced components reducing further the CR." (Razran, 1956, p. 46)

This negative feedback should occur when the US (and hence the CR) is weak; positive feedback when the US (and hence the CR) is strong. The general theory is presented in diagramatic form in Fig. 1, with a critical point dividing the ordinate (strength of CR) into two portions, giving, respectively, incubation and extinction (Eysenck, 1979).

The figure also illustrates the close relation between duration of CS exposure and occurrence of incubation or extinction, short exposure to supracritical CR leading to incubation, and lengthy exposure to infracritical CR leading to extinction. Data from both human (Rachman and Hodgson, 1980) and animal (Solomon and Wynne, 1953) experiments support the shape of the curve showing decline in the strength of the CR over time, in the presence of the CS. Bersh (1980) criticizes the evidence for the existence of incubation, and no doubt there is no absolute unblemished single experiment which would not be susceptible to such criticism. Nevertheless, the evidence is impressive, particularly when we add it to the large number of papers published since Razran's review, supporting his conclusion that the extinction phenomenon is certainly "less than

100 per cent". Given that a strong US is required to produce incubation (in order to achieve a position above the critical point), there is the danger that ceiling effects may make the demonstration of actual increments above the original CR difficult or impossible, so that failure to achieve extinction may be counted as support for the incubation hypothesis. The 'flooding' study by Rachman (1966) of spider phobics, in which the patients were exposed for 2 min to the CS, produced evidence of failure to extinguish, rather than incubation, but in view of the very strong CR it seems likely that ceiling effects were being approximated, so that little if any exacerbation of symptoms could be expected. In rat experiments, in particular, strong CRs are usually so strong that small increments are difficult to observe or measure. Possibly dogs are the best Ss, as in the Napalkov experiment (Eysenck, 1967), but there are few laboratories working with dogs, and hence the obviously highly desirable replication of the Napalkov experiment has not yet been done.

Clearly, Bersh is right in calling for more, and more convincing evidence; in particular, he asks for more direct evidence on the shape of our Curve A in Fig. 1, on the exact position of the 'critical point' on the relation between personality and incubation etc. He is not right, however, in suggesting that the theory does not make predictions clear enough to be testable. Such predictions appear, for instance, in the replication of Curve A by Rachman and Hodgson (1980), (their Fig. 14.1); the shape of the curve is identical with that of the hypothetical curve in our Fig. 1. Other predictions are given in Eysenck (1979). Thus a trade-off is predicted between (physical) strength of the US and neuroticism/emotionality in the *S*; the greater the emotionality of the *S*, whether man or rat, the lower the US required in order to push the CR above the critical point. Obviously much more work will be needed to quantify the precise details of the figure, and such quantification is more likely to be achieved when animals are used as experimental *S*s than when humans are used; ethical considerations make it impossible to administer USs to normal *S*s strong enough to produce incubation, or to treat patients with CS presentations so short as to be above the critical point, or at least sufficiently near to that point to make the outcome doubtful. The therapist must make sure that the CS exposure is terminated only when the critical point has clearly and unequivocally been passed; this makes experimentation to establish the precise position of that point impossible. However, a quite specific prediction has been made by the writer with respect to therapeutic activities and their effects when desensitization has gone wrong, and the therapist has allowed anxiety to reach a high level (Eysenck, 1978); many other such predictions would seem to follow from the theory. Quantification would certainly be a most desirable addition to the present support for the theory, but as usual quantification follows the elaboration of a theory; it does not precede it. The weakness Bersh discerns in the experimental support for the incubation hypothesis stems precisely from the fact that the experiments were for the most part not done to test the theory; they were designed with other theories in mind, so that although their outcome gives welcome support to the incubation hypothesis, quantitative evaluations in terms of that theory are not really possible.

Even as things stand, it is not accurate to say that in the absence of a precise, quantitative formulation "all outcomes are susceptible to *post-facto* and circular interpretation." There are many outcomes which could certainly not be so interpreted. Incubation following comparatively weak, extinction following comparatively strong USs would be one example. Short-term flooding with response prevention followed by remission of the symptom, or long-term flooding with response prevention followed by worsening of the symptom would be another example. A clinical application has already been cited which makes precise predictions as to what should and should not happen (Eysenck, 1978). Many other predictions, perhaps more qualitative than quantitative, but nevertheless precise enough to permit of falsification, follow from the theory. This criticism of Bersh's does not seem to be applicable to the theory, even in its present early form. If this be the most serious drawback of the theory, as Bersh avows (p. 15), then we may have hope for its future yet!

It may be useful to see this discussion in the historical context of what happens to theories in the hard sciences. Hubble's constant is perhaps the fundamental unit in cosmological theory, but it was determined only after the theory linking red-shift and stellar brightness was formulated, i.e. on the basis of a simple correlation, and its numerical value was wrong by a whole order of magnitude. This is not an unusual development; what usually happens in the exact sciences is that a qualitative theory is erected on the basis of some (often not very convincing) empirical findings; efforts are then made to quantify the theory, new experiments are instigated to test the theory and improve the provisional quantification, and gradually there emerges a properly predictive theory which may only have vague resemblance to the original hypothesis. This no doubt is what will happen to the theory under discussion, but it is unrealistic to expect quantitative predictive powers at this early stage of development.

One of the more serious criticisms of the theory made by Bersh is that

> "several implications ... of Eysenck's theory are completely at odds with the evidence." (p.15)

Bersh cites as his main example the following.

> "The strengthening or weakening effect of CS is assumed to vary with the degree to which the CR is above or below the critical point at the time of termination. With CS duration held constant, for example, each CS would progressively weaken the CR. Thus on successive CS trials, initial CR strength would be less, so that CR strength would fall further and further below the critical point by the time of CS termination. Accordingly, the size of the decrement in CR strength would *grow* with successive CS trials, yielding a *positively* accelerated extinction curve, or at least one that was positively decelerated until the occurrence of a floor effect as CR strength approached zero. It is well established, however, that the curve for Pavlovian extinction is *negatively* decelerated. Conversely, a constant CS duration, initially briefer than the critical duration, would progressively strengthen the CR, and, by the same logic, would generate a *positively* accelerated incubation curve, or at least one that was positively accelerated until the influence of a ceiling effect was sufficiently strong. The laboratory data presented by Eysenck (1979) to support the existence of incubation effect show a *negatively* accelerated incubation curve." (Bersh, 1980, p. 15)

The shapes of curves are always subject to rules of scaling, and assumptions regarding the use of scales have to be justified. Bersh is assuming that equal units on the ordinate in Fig. 1 are equal units of habit strength ($_SH_R$), but as Hull has shown (1943, p. 116) a transformation (scaling) is necessary to translate the one into the other. When this translation is undertaken, then *the prediction* is not contradicted by the resulting shape of the curves involved. The observed CR is scaled very roughly in terms of some such variables as ratings on a fear thermometer, or change in heart rate of G.S.P., or some other such

indirect measure of ${}_{S}H_{R}$. Now, as Hull (1943, p. 119) points out,

> "the determination of the correct functional relationship of an unobservable to an observable is to a considerable extent dependent upon trial and error."

We have provisionally accepted Hull's formulation, but clearly this is an empirical matter pure and simple, and an alternative method of scaling may prove superior in the long run. In any case, it does not seem that Bersh's criticism is well taken; the observed data are not at odds with theoretical expectation.

CONCLUSIONS

It is clear that Bersh (1980) was not speaking as a hostile critic, but as one who considered that

> "Eysenck's theory of incubation proposes a reasonable basis for an enhancement in the strength of a CR as the result of CS." (p. 16)

In response, the writer must agree with Bersh that there are weaknesses in the theory (particularly as far as quantified predictions are concerned), and in the data supporting the notion of incubation experimentally. Clearly, the theory is at an early stage of development; its strength is that it serves to unify animal and human experimental data, on the one hand, and clinical data, on the other, in a single formulation that has far-reaching consequences. If correct, this formulation would serve to explain the development and continuation of neurotic disorders, would suggest methods of treatment for these neurotic disorders, and would finally also explain the relative success of many different types of treatment, including psychoanalysis, psychotherapy, the various methods of behaviour therapy (desensitization, flooding, modelling etc.), and even spontaneous remission (Eysenck, 1978). It would seem that such a theory would probably repay detailed testing, and quantitative improvement along the lines suggested by Bersh.

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Note added in proof

Bersh has not criticized a weakness in Eysenck's original formulation of the incubation of anxiety theory, namely his failure to account for the *incrementation* of $\overline{CS}$ with repetition of exposure, while repetition of US leads to *habituation.* Eysenck (1982) has recently suggested a mechanism which would explain this phenomenon along neo-behaviouristic lines.