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# What I Would Most Like to Know

*This is a series of short invited contributions from therapists and research workers who have volunteered to answer the following question: "If your fairy godmother were to wave her magic wand and give you an immediate answer to a question or questions, without having to do the relevant research, what would you most like to know?"*

# Why do Conditioned Responses Show Incrementation, while Unconditioned Responses Show Habituation?

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I have always maintained that most theories in the field of clinical psychology are lacking in scientific rigour (Eysenck, 1970a), that behaviour therapy is (or at least should be) based on theories of a more determinate character (Eysenck, 1970b), and that behaviour therapy, in fact, is not a dogma, but an applied science (Eysenck, 1976a). In pursuance of this belief, I have tried to bring up to date Watson's old conditioning theory of neurosis and treatment (Eysenck, 1976b, 1979, 1980), in the hope of aligning it more closely with modern developments, and avoiding many of the criticisms rightly levelled against the original form of the theory.

In doing so, my main difficulty has been the fact that the development of neurotic disorders seemed to show certain features which are not only incompatible with learning theory, but seem to go directly counter to it. Learning theory suggests that exposure to the unreinforced CS (CS only) produces extinction. In the development of neurotic disorders, however, it seems that exposure to CS only does not produce extinction, but if anything the opposite; I have named this process of enhancement of the CR when CS only is presented "incubation of anxiety," and have tried to account for it by postulating (a) that as far as neurosis is concerned, we are dealing with Pavlovian B rather than with Pavlovian A conditioning, and (b) that under these conditions the CR, being essentially identical with the UCR, can act as a positive reinforcement in the absence of the UCR. On the experimental side, I have used the Napalkov phenomenon (Eysenck, 1967, 1968) as an animal laboratory analogue of the

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process that takes place in the development of a neurotic disorder. Briefly, Napalkov showed that repetition of the UCS was followed by habituation of the UCR, but a single combination of a CS with the UCS, followed by repeated presentations of the CS only, was followed by an incrementation of the CR, i.e. by incubation of anxiety. This leaves us with the problem of accounting for the fact that while the UCR habituates, the CR augments. The problem is a crucial one for any theory of neurosis, and it is equally crucial for an understanding of the conditioning process in the laboratory. If I had one question to put to my presiding angel, to which I would like to receive an answer, this is it. In the absence of such divine revelation, it is perhaps possible to speculate on possible mechanisms which might mediate this effect.

Exposure of the UCS is usually short, followed immediately by the UCR, which is not usually prolonged. In the case of Napalkov's dogs, where the UCS was a shot, the consequent rise in blood pressure was relatively short-lived. Let us call the typical UCR PFA (i.e. a mixture of pain, fear, and anxiety), and consider what the fate is of the CR accompanying a CS only. Consider Figure 1, where the abscissa denotes the duration of CS-only exposure, the ordinate the strength of the CR, and *a* the moment when CS-only exposure is discontinued. The strength of the CR declines, from a maximum which is presumably equal to or less than the UCR. We now have to explain why the reinforcement produced by the CR (i.e. the PFA effects mediated by the CS) is apparently more powerful than the UCR, in leading to incubation rather than to habituation and extinction.

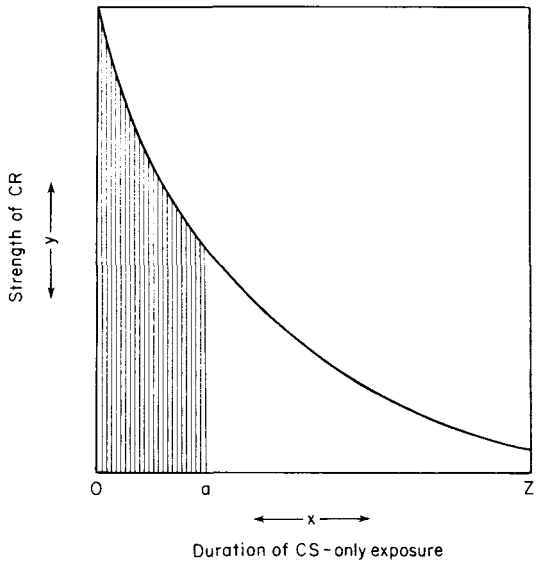


FIGURE 1. Change in CR strength with duration of CS-only exposure.

The most obvious reason would seem to be that because CS-only is continued over a period of time, the PFA effects build up, so that what we should take into account is not just the strength of the CR at point 0, but rather the whole area under the curve, as indicated by vertical lines. This suggestion therefore amounts simply to an hypothesis that after CS-only exposure the PFA effects, extending over a long period of time, will *summate* to produce a stronger effect than the UCR effects, which occupy a much shorter period of time.

This model, as it stands, is of course insufficient; it would suggest that if exposure of the CS-only were to be continued to point z, then the incubation effect should be even stronger than if it were terminated at point a. This is clearly implausible, and in fact untrue; if it were true, then "flooding" treatment could not work, as it is known to do (Rachman and Hodgson, 1980). We have to introduce one further step, namely the proviso that for the summation of the area under the curve, recency is an important variable, so that events at or close to point a count much more heavily than points remote from a. Thus as we approach point z, the very weak CRs recorded there outweigh the very strong CRs found at or near the point of the CS-only exposure, so that the total effect is very weak. Only termination of CS-only exposure relatively close to point 0 would, through integration of the area under the curve with this proviso, provide us with a CR stronger than the UCR generated by the UCS. We can express the hypothesis here suggested in terms of a formula:

$$I = \int_0^a \alpha(1 - e^{\beta x})e^{-[\gamma(a-x)]}\,dx$$

where $I$ is the total PFA experienced after exposure of the CS-only; alpha is the strength of the CR at point 0; beta is the decay factor or slope of the CR strength over time, and $a$ denotes the termination of the exposure of CS-only. It would also seem to be important to introduce a backward weighting factor, gamma, which may differ from person to person. Broadbent (1958) has suggested that introverts weight events distant in time more than do extraverts, who are more likely to weight heavily quite recent exposures.

It will be seen that this model makes adequate provision for individual differences, a feature that to my knowledge no other model of the development of neurosis and treatment has incorporated. Such individual differences would manifest themselves in terms of either alpha, beta or gamma, and from general personality theory it should not be difficult to make quite specific predictions for the interrelation of these three factors and extraversion–introversion, and neuroticism–stability. It would seem to me that the search for the

interaction between personality variables and treatment should not be random, but be governed by specific hypotheses related to a definite model, such as the one here presented.

This general hypothesis may serve to account for the facts of neurosis and experimental investigation; there is no direct evidence one way or the other, and indeed it would seem quite difficult to design an experiment to obtain such evidence. However, in the absence of divine intervention, this will have to do for the time being. If readers have a better answer to the problem, I would be delighted to hear of it.

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