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# Anxiety: one label for many processes*

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There is no agreement among psychologists as to the exact nature or definition of anxiety. This paper considers some aspects of biology which can account for this lack of agreement.

Emotional reactions involve skeletal, autonomic and hormonal effector systems. In any particular situation each of these systems will be subject to unique selection pressures not shared by the other systems. Evolutionary adaptations develop in an incremental, piecemeal fashion. The underlying control mechanisms of each effector system are, therefore, likely to be different. The apparent coordination of separate effector systems in emotion may be fortuitous. The nameability of an emotion would then depend not on some unitary control process within the animal but rather on a high correlation in the phylogenetic environment between a variety of eliciting stimuli.

The piecemeal progress of evolution has interesting consequences for our view of reactions of individual effector systems. A generally adaptive response will confer some selective advantage even if it can be elicited only in a limited set of situations. Superficially similar reactions of any one effector system may, as a result, depend on different 'rules of thumb' under slightly different environmental circumstances. For example, experiments with anxiolytic drugs demonstrate that there are several independent processes which give rise to the behavioural inhibition consequent on anxiety.

Both within and between effector systems, therefore, we can expect considerable independence of the processes which constitute 'an emotion'. However, given a usual co-occurrence of effector reactions, or the occurrence of one effector reaction where a second is also advantageous, there will be selection pressure for dialectical links between such reactions. For example, each reaction could act as a stimulus for the other. This would result in some apparent integration of observed reactions — which would not depend on any unitary control process.

So, in analysing anxiety, we must be prepared for independent neural control of apparently integrated components of reactions; for multiple processes underlying the same observable reaction in apparently similar situations; and, for unexpected dialectical interactions between such components. Anxiety as a term is likely to be more easy to define in terms of common evolutionary origins, or pharmacological sensitivities than in terms of any unitary psychological construct.

The physiologists who, during the past few years, have been so industriously exploring the functions of the brain, have limited their attempts at explanation to its cognitive and volitional performances. Dividing the brain into sensorial and motor centres, they have found their division to be exactly paralleled by the analysis made by empirical psychology, of the perceptive and volitional parts of the mind into their simplist elements. But the *aesthetic* sphere of the mind, its longings, its pleasures and pains, and its emotions, have been so ignored in all these researches that one is tempted to suppose that if (physiological psychologists) were asked for a theory in brain-terms of the latter mental facts they might . . . reply either that they had as yet bestowed no thought upon the subject, or that they had found it so difficult to make distinct hypotheses that the matter lay for them among the problems of the future, only to be taken up after the simpler ones of the present should have been definitely resolved.

And yet it is even now certain that of two things concerning the emotions one must be true. Either separate and special centres affected to them alone are their brain-seat, or else they correspond to processes occurring in

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the motor and sensory centres, already assigned, or in others like them, not yet mapped out.

William James, (1884)

It might seem embarrassing, to one of a physiological persuasion, that William James's complaint seems as apposite today as it was more than a century ago. However, a lack of a 'theory in brain-terms' of emotion in general and anxiety in particular is not surprising given that there is no apparent agreement among psychologists of a non-physiological persuasion as to the nature or definition of anxiety.

I believe that James is in part to blame for this. His question 'What is an emotion?' has led psychologists to search for unitary 'brain-seats' or at least unitary control processes specific to individual emotions. I do not think that there are any such things to find. On the other hand, I do not accept Duffy's (1941,1962) view that there is no such thing as an emotion.

The idea that a word such as anxiety can have scientific meaning without implying some unitary control process derives from biological analysis. This paper makes two main points. First, our successful everyday use of emotional referents such as anxiety depends on the fact that behavioural, autonomic, and hormonal reactions have evolved in response to regularly co-occurring sets of adaptive requirements. Second, that there need be no unitary central state which controls all reactions of any particular emotional class such as anxiety. Our use of emotion words, I would contend, is like our use of colour words. We have good agreement as to the use of a large number of discrete colour words. But the physical basis of colour depends on a continuum and the physiological basis for our colour discriminations is trichromatic. Indeed, many nameable colours can be perceived in response to the presence of only two frequencies of light (Land, 1959). The ostensive validity of the words is, thus, no guarantee as to referents at the process level of analysis.

This paper is divided into four main parts. First, a discussion of the theoretical implications of evolution for emotion. Second, an argument for the independent evolution of the separate effector systems which contribute to emotional responses. Third, presentation of experimental evidence for the use of many 'rules of thumb' to support a superficially unitary type of anxious reaction. Finally, consideration of the dialectical links which could serve to provide some integration of emotional responses in the absence of a central controlling state.

## Evolution and anxiety

In this section I will consider some of the general implications of evolution for anxiety. The general principles involved, as opposed to the specific examples considered, are likely to be true of most emotions. A more general and detailed treatment of the subject is presented in McNaughton (1989).

A critical feature of evolution for all of the arguments in this paper is that it is opportunistic and incremental rather than in any sense goal-directed. Any advantageous reaction will come under genetic control only if a suitable mutation occurs and only if there is a local increase in adaptive fitness. This has a variety of implications for the evolution of the kind of responses which we would normally consider to contribute to anxiety.

Let us first consider the 'situation' which is to generate anxiety. For the purposes of the argument, we will assume that this can be characterised as one of threat. As is represented diagrammatically in Figure 1, where there is the threat (as opposed to actuality) of predation there will be a variety of effector reactions which may be advantageous. The advantage to be gained by each reaction will depend less on the precise nature of the threat (e.g., lion versus tiger) than on the fact that the situation is threatening.

It may be that there is a set of stimuli (S1–S7) which are each highly correlated with most threatening situations. In each case, therefore, there will be selection pressure favouring the elicitation of adaptive responses by those stimuli. Whether such elicitation comes under genetic control will also depend on mutation. As a result, it is possible, and indeed relatively likely, that each separate adaptive response will come under the control of a different stimulus. This result is demonstrated diagrammatically by the separate connections made by S1, S3, S4, S5 and S6 in the lower part of the figure.

A second reason for separate control of different reactions is that some reactions may be advantageous in non-threatening situations. As a result, they may be best controlled by

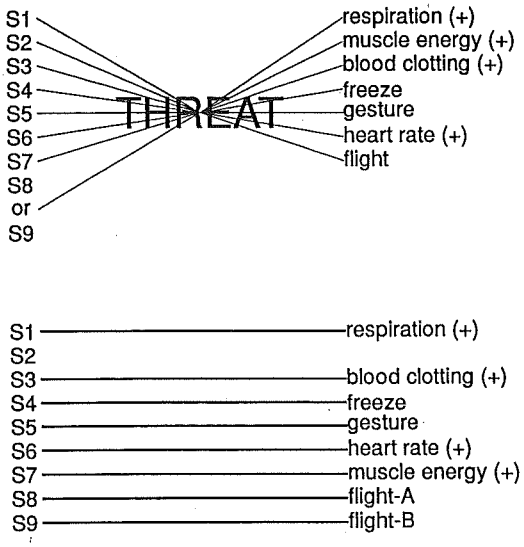


*Figure 1:* Diagrammatical representation of the relation between stimuli in the environment, threat, and various effector outputs. All stimuli represented are positively correlated with a variety of types of threat, S8 and S9 are mutually exclusive. All the effector outputs are of advantage in all, or most, threatening situations. The bottom half of the figure shows the control relationships which can result from random mutations. An 'obvious' connection between e.g. S2 and muscle energy may not be made because of a lack of a suitable mutation. Likewise a single type of response may come under the control of two separate stimuli (S8, S9) each of which results in its occurrence in only some threatening situations.

a stimulus which is correlated with both types of situation. However, even if this is true of S2 and muscle energy, for example, there is no guarantee that an appropriate mutation will occur. So, as is shown in the figure, while we might *a priori* expect S2 to control muscle energy, S7 may do so simply because of the occurrence of the appropriate mutation.

We cannot assume, either, that any particular adaptive requirement will be fulfilled by any particular pairing. In the figure, S8 and S9 are presented as being highly correlated with threat but negatively correlated with each other. In this case, elicitation of flight by S8 will only result in adaptive advantage in, say, half the appropriate situations. Additional selective advantage will be gained, therefore, if S9 can also come to elicit flight. In the diagram flight is subdivided here into flight-A and flight-B as there is no requirement that identical responses be elicited. Provided the animal is removed from the threatening situation the topography of the response sequences could be distinct, or, as we will see below the underlying control processes may differ even when the superficial topographies do not. In the jargon of evolutionary theorists, therefore, we may have a variety of 'rule of thumb' strategies each of which results in similar adaptive advantages to the others and each of which operates within only a limited stimulus domain.

A final point to notice is that S1–S9 need not be stimuli within the environment. They may be the result of cognitive processes, or, more importantly, they may be detectable aspects of changes in other effector systems. Clearly, if a mutation has resulted in the attachment of one response to a stimulus which is highly correlated with threat then that response will, itself, be as good a predictor of threat as the stimulus to which it is attached. If a particular response is attached to a range of stimuli, each operative in only a subset of threatening situations, then the response will be a better predictor of threat than any of the individual stimuli. The critical determinant of what occurs will be the occurrence of appropriate mutations. With sufficient time, phylogenetically, the control processes could become quite complicated. For example, when two separate responses occur, each within a limited stimulus domain, each may come to elicit the other. Likewise, where two responses co-occur each may alter the adaptive equation for the other. (If you are very good at running away you may not need particularly good blood clotting.) There can therefore be selection of reactions which modulate effector outputs.

## Independent effector systems

The skeletal nervous system is the most obvious source of 'responses' of interest to the psychologist. Its complex neural organisation clearly provides for a wide range of separate classes of integrated response. As can be seen from Figure 2, the autonomic nervous system also has the capacity to produce a wide variety of responses. Even a brief perusal of the variety of target organs involved should be enough to dispel the myth that autonomic responses are undifferentiated. The sympathetic and parasympathetic branches of the autonomic system constitute, therefore, an internal effector system similar to the external effector system provided by the skeletal nervous system.

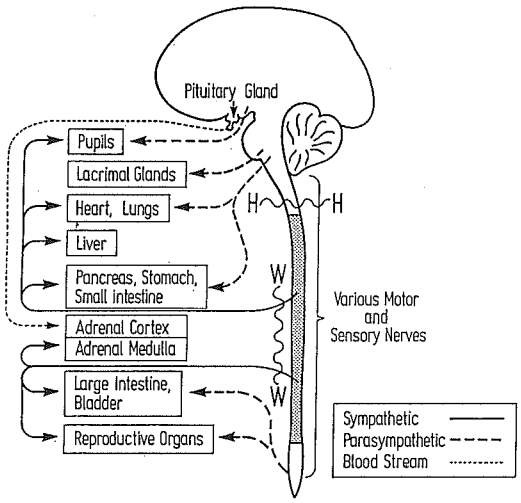


*Figure 2:* Cross section of the brain and spinal cord showing the outflow of the autonomic nervous system. Note the wide variety of organs which receive separate innervation. This implies a large capacity for differential reponse of the different organs.

Hormonal systems, although they are chemically rather than neurally organised, are also capable of complex integrative action with effects which impinge on both skeletal and autonomic target structures. As Mason (1975) states 'the endocrine apparatus . . . represents a *third effector or motor system* of the brain'.

Each of the different effector systems will be subject to somewhat different selection pressures. Equally importantly, the control of outputs from different components of any one of these effector systems will be subject to idiosyncratic selection pressures. We should expect, therefore, some independence in the control of separate components of apparently integrated emotional responses. The types of pressure involved are detailed for a threatening situation in table 1.

As can be seen from the table some outputs may, independently, achieve the same type of adaptive advantage (e.g. increased oxygen availability). Other outputs will achieve entirely separate adaptive advantages. In each case there is no reason why the different outputs should not be obtained, via mutation, through completely separate control mechanisms.

Hofer (1972) carried out an experiment which directly addressed the question of the independence of response components in an emotion. He was interested in the complex behavioural and autonomic reactions which are observed in young animals when their mother is removed. These reactions are frequently labelled with the term 'separation anxiety'. He asked 'Is the young rat responding to all aspects of the experience taken together as a form of gestalt or complex patterned stimulus? If this were the case, omission of one or two components would alter the pattern . . . Alternatively, the individual components of the experience could be additive . . . [or] the response to separation might depend primarily on one aspect . . . the other components being incidental'.

He removed rats from their mother at 2 weeks of age and measured heart rate and a variety of behavioural responses. Both heart rate and behaviour were changed by loss of the mother. Frequent feeding with milk returned heart rate to normal but did not eliminate the behavioural aspects of 'separation anxiety'. The presence of a non-lactating foster mother blocked the separation response for most of his behavioural measures but did not normalise heart rate. So, rather than there being a single central state or control system for 'separation anxiety' (Figure 3), separate stimulus aspects of the missing mother control separate effector outputs. Of course, in the

Table 1: *Some outputs of the effector systems of the body, subdivided by system, with details of their possible selective advantages in a threatening situation.*

| SYSTEM | OUTPUT | ADVANTAGE |
|---|---|---|
| autonomic | heart rate (+) | oxygen (+); energy (+); waste (–) |
| | bronchodilation | oxygen (+) |
| | blood clotting (+) | bleeding (–) |
| hormonal | blood glucose (+) | energy (+) |
| skeletal | fight | escape predation |
| | flight | escape predation |
| | expression | warn/recruit conspecifics |

normal phylogenetic environment, all stimulus aspects of the mother will be present or absent together and so 'separation anxiety' can be used to describe the various reactions which the animal shows under these circumstances — provided we do not assume that this implies any common central control of the reactions.

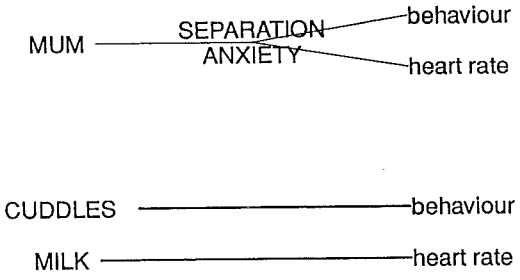


*Figure 3:* At the top of the figure is the relationship which might be assumed to obtain between separation from the mother, separation anxiety and changes in behaviour and heart rate. Below is shown the relationship which obtains in practice between loss of discrete stimulus components of the mother and separately controlled behavioural and autnomic reactions.

## Multiple rules of thumb

The idea that separate effector systems should be independently controlled in their responses to some common situation seems reasonable (if less parsimonious than we would like) given the different selection pressures which are faced by each system, or separate system component. In this section we must deal with a more counterintuitive result of mutation and selection — the use of multiple rules of thumb to achieve some single adaptive advantage.

The theoretical basis for multiple rules of thumb was discussed above. Essentially, where a mutation allows an adaptive response to occur in only some of the appropriate circumstances, selection will none the less occur because there has been some (albeit limited) increase in fitness. Successive mutations may, then, extend the range over which that class of response occurs. Ultimately, a superficially standardised response can occur throughout the animal's phylogenetically usual environment — but as a result of a set of independent locally-effective 'rules of thumb' rather than some single solution to the general adaptive problem. It is particularly important to note that the rule of thumb used by an animal to solve a problem under its normal environmental circumstance may bear no resemblance to the calculations required to solve the problem in a more general case (Krebs, Stephens & Sutherland, 1983). As a result, behaviour in the laboratory or in entirely novel circumstances may be quite maladaptive.

To make this point in the context of anxiety, let us restrict ourselves to those aspects of anxiety which are affected by anti-anxiety drugs and to those effects of the anxiolytic drugs which can be assumed to result from changes in functioning of the hippocampus (Gray, 1982). Let us further restrict ourselves to situations involving conflict between a rewarded response and conditions under which it is not rewarded. It might be thought that this degree of restriction would result in a unitary control process. However, as we will see this is far from the case.

An example of such a conflict is 'successive discrimination'. In a conventional successive discrimination the animal (typically a rat) will be pressing a lever to obtain food when a stimulus will be presented which signals the fact that lever pressing will no longer be rewarded. After some experience with this schedule, responding during the signal will be suppressed. This can be contrasted with a simultaneous discrimination where the positive and negative stimuli are presented together and where, after training, animals regularly choose the correct one of two available responses and no prepotent responses needs to be inhibited. Anxiolytic drugs do not affect simultaneous discriminations. But, their capacity to impair successive discrimination by releasing behavioural inhibition is one of the more reliable assays of their clinical efficacy.

Drug companies have to screen large numbers of compounds and so they administer drugs briefly to animals whose performance on successive discrimination has stabilised. It should be noted that this is unlike the clinical situation where drugs are administed chronically. It is interesting, therefore, that we found that chronic administration of the anxiolytic benzodiazepine chlordiazepoxide (Librium) has no effect on well-learned successive discrimination (Figure 4). The effects of the drug on performance are attributable to state-dependency rather than a truly anxiolytic action (McNaughton 1985).

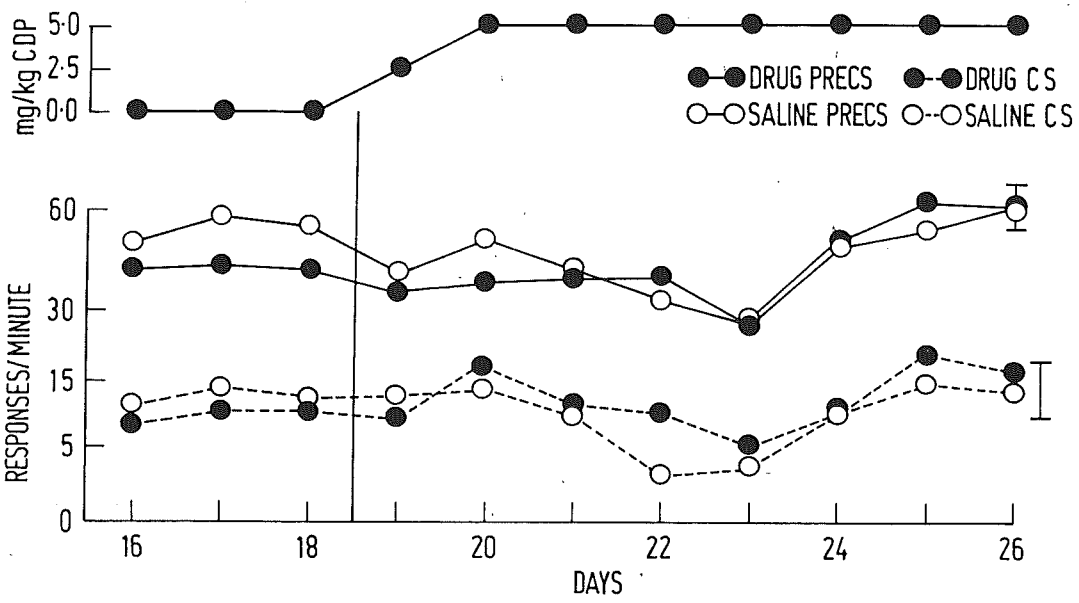


*Figure 4:* Effect of chronic administration of the anxiolytic benzodiazepine chlordiazepoxide on well-learned successive discrimination. The difference between CS and PRECS response rates reflects behavioural inhibition which is unaffected by the drug.

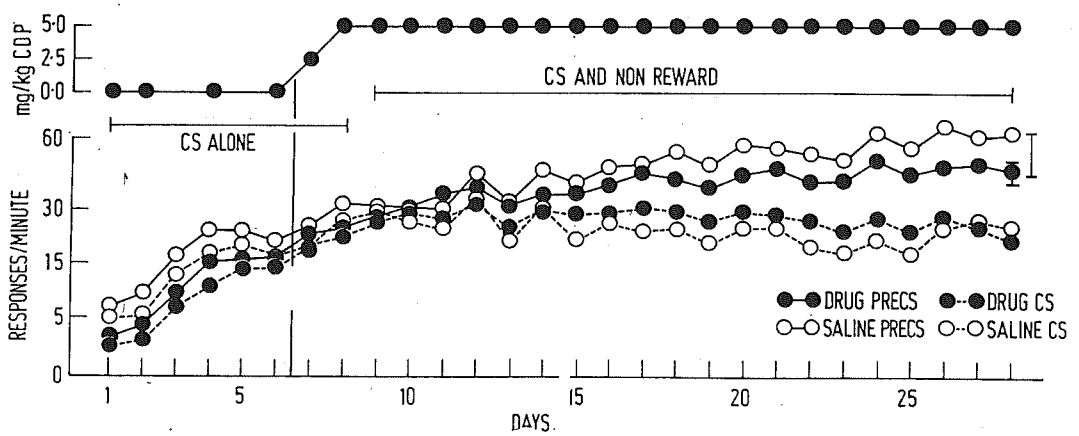


*Figure 5:* As figure 4, but showing that chlordiazepoxide does impair acquisition of successive discrimination.

By contrast with the lack of effect of chronic chlordiazepoxide on well-learned successive discrimination, we found that chronic administration does impair learning of the task (Figure 5). This shows that well-learned behavioural inhibition is controlled by different processes than those during learning of the response. We appear, then, to have what could be called behavioural-inhibition-1 and behavioural-inhibition-2.

In the classic successive discrimination the presentation of stimuli and availability of rewards is totally under the control of the experimenter. In a signalled differential reinforcement of low rates of response task, by contrast, if the animal makes a response which follows a previous response by less than the criterion interval (e.g. 15 seconds), not only does it not receive a reward but the nonrewarded period is extended for a further 15 seconds. The length of the signalled nonrewarded period, therefore, depends on the

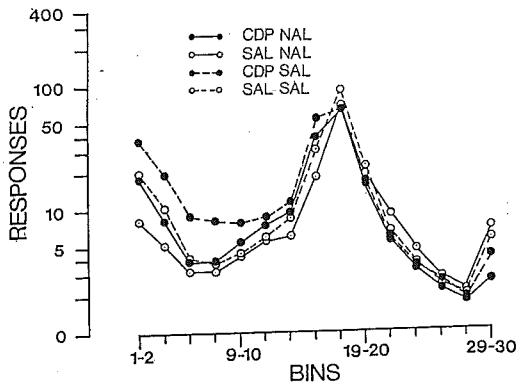


*Figure 6:* Interresponse time (IRT) distribution for animals responding on a signalled DRL 15 seconds schedule. All responses with an IRT less than 15 seconds were nonrewarded, all responses with an IRT greater than 15 seconds were rewarded. A light signalled when reward was unobtainable. Chlordiazepoxide releases inhibition of responding at IRTs between 0 and 15 seconds. Naloxone, an opiate receptor antagonist blocks the effects of chlordiazepoxide at IRTs of above 5 seconds but has no effect at very short IRTs. (Data from Tripp, & McNaughton, in preparation).

animal's responding. In other respects the schedule is formally similar to successive discrimination. Periods of reward and nonreward follow each other and nonreward is signalled. As you might expect, experience of this schedule results in response inhibition and administration of chlordiazepoxide during learning increases nonrewarded responding. One might be forgiven, therefore, for thinking that this task involves the same underlying processes as successive discrimination.

We found (Figure 6) that the effects of chlordiazepoxide on signalled DRL are blocked (at intermediate inter-response times) by administration of the opiate receptor antagonist naloxone. With the same drugs at the same doses, there is no sign of an interation of chlordiazepoxide and naloxone on conventional successive discrimination (Tripp & McNaughton, 1987). If the naloxone-insensitive effect of chlordiazepoxide, in successive discrimination, is on behavioural-inhibition-2 then the naloxone-sensitive effect of chlordiazepoxide, in DRL, must be behavioural-inhibition-3.

We were curious to determine what might be the difference between behavioural-inhibition-2 and behavioural-inhibition-3. There are a variety of parametric differences between the successive discrimination and DRL schedules we had used. These are itemised in Table 2. We therefore designed an experiment to reduce the number of possible differences between the two schedules. We created a successive discrimination in which random number generators were used to produce a distribution of rewarded and signalled nonrewarded periods similar to that obtained with DRL. Unlike DRL the animals's responses had no effect on the distribution. We argued that if response control determines opiate sensitivity this new schedule should show similar pharmacology to successive discrimination, but if schedule timing is critical then it should be similar to signalled DRL.

In one sense our results suggested that schedule timing is the important factor — naloxone blocked the effects of chlordiazepoxide. However, as can be seen in Figure 7, chlordiazepoxide *increased* behavioural inhibition rather than blocking it! There are a variety of different *ad hoc* ways of accounting for this result, but all seem to require some form of behavioural-inhibition-4.

The rather tortuous series of experiments I have described shows that the observation of simple behavioural inhibition in response to a signal which predicts the omission of an expected reward depends on at least four separate underlying processes. Equally impor-

Table 2: *Parametric differences between the successive discrimination schedule used by Tripp and McNaughton (1987) and the signalled DRL schedule which generated the results of Figure 6. Operant boxes, stimulus lights, food deprivation schedule, food reward and similar factors were all the same.*

| | DRL | SUCCESSIVE DISCRIMINATION |
|---|---|---|
| SCHEDULE CONTROL | Response/Rat | Time/Experimenter |
| CS+ LENGTH | 1+ seconds | 7 Minutes |
| CS- LENGTH | 15+ seconds | 3 Minutes |
| CS CHANGES | Frequent | Infrequent |
| CS VARIABILITY | Large | None |

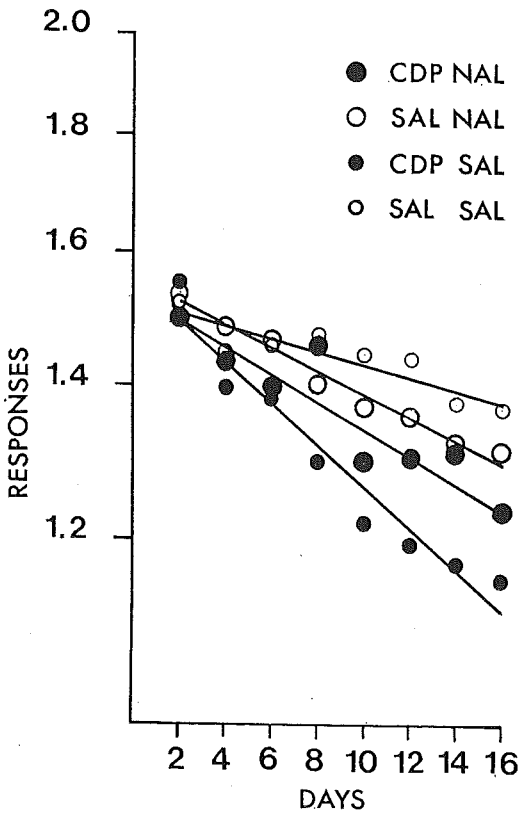


*Figure 7:* Responding in the nonrewarded component of a successive discrimination which modelled DRL (see text). Rates are adjusted by covariance for any variations in overall response rate. Chlordiazepoxide *increasse* behavioural inhibition — an effect which is blocked by naloxone. (Data from Tripp, & McNaughton, in preparation).

tantly, the behavioural distinction between these different processes is negligible. Behavioural-inhibition-2 (acquisition of successive discrimination) and behavioural-inhibition-1 (well learned successive discrimination) must transfer smoothly from one to the other. Similarly, within Figure 6 it can be seen that behavioural-inhibition-2 (naloxone-insensitive, bins 1-4) gives way smoothly to behavioural-inhibition-3 (naloxone-blocked, bins 5-8). So, within an apparently unitary type of reaction (inhibition) of a single response system (lever pressing) we have different 'rules of thumb' operating depending on the situational parameters. Superficial inspection of the rats' behaviour gives no sign of this — pharmacological probes have been necessary to discover the lack of unitary control processes.

## Dialectical interactions

I have so far argued that responses of separate effector systems are likely to have evolved separate control mechanisms because they are subject to separate selection pressures and, in any case, will have resulted from separate mutations. I have also argued that even apparently unitary responses may depend on a variety of control mechanisms each of which results in the response in only a restricted range of circumstances. Both within and between effector systems I have provided examples of such unparsimonious responses.

I believe such parallel control will prove sufficiently general that it will be impossible to refer to anxiety, or any other emotion, as a unitary controlling state, central construct or any other coherent entity within the orgaznism. As I indicated at the beginning of the paper this does not mean that words such as anxiety cannot be used but rather that their referent is the evolutionary situation (in the case of anxiety, possibly 'threat') which provides the basis for the co-occurrence of sets of responses.

However, I would not want to be taken as suggesting that there is no functional relation between different components of anxiety. I argued earlier that once a reaction of any effector system has evolved it can affect the evolution of other effector systems both in the capacity of a potential signal and also because of its effects on the general adaptive equation.

Note that interactions between effector systems would not constitute centralised control. Rather they would involve dialectical links which could give the superficial appearance of central control. That is to say a change in one effector system could produce a change in a second which would then feedback on the first and so on until the system as whole had settled into a stable state.

There is good evidence for such a messy organisation of emotional systems. For example, autonomic reactions in most species depend on cognitive assessements of situations. It is also clear that, at least in some cases (Lader and Tyrer, 1975), autonomic changes can affect cognition. A particularly counterintuitive example of this type of interaction is that of

the effect of facial expression on autonomic response. Ekman, Levenson & Friesen (1983) carried out an experiment in which 'subjects were not asked to produce an emotional expression but instead were told precisely which muscles to contract'. To produce an expression of fear, therefore, subjects were told to 'raise your brows and pull them together'; then 'raise your upper eyelids'; and finally 'also stretch your lips horizontally, back towards your ears'. This 'directed facial action' produced 'autonomic changes of large magnitude that were more clear cut than those produced by reliving emotions'.

Thus, each effector system may affect other systems, directly or indirectly, and in turn be affected by them. In part these dialectical interactions will constitute positive feedback. If the interactions are sufficiently strong the result, at least superficially, will be very similar to that which would be produced by some single central control process.

## Conclusions

So, in analysing anxiety, we must be prepared for independent neural control of apparently integrated components of reactions; for multiple processes underlying the same observable reaction in apparently similar situations; and for unexpected dialectical interactions between such components. The apparent coherence of anxious reactions in particular and emotions in general is, on this view, due to the regular coincidence, in the phylogenetic environment, of sets of adaptive requirements. Anxiety as a term is likely, therefore, to be more easy to define in terms of common evolutionary origins, or pharmacological sensitivities, than in terms of any unitary psychological or neural construct.

As Monod (1974) emphasised, even the most apparently elegant productions of evolution are a product of the interaction between 'Chance and Necessity'. Selection has moulded systems so that their output tends to conform to the adaptive requirements of a situation. But the raw material from which selection sculpts its works is the result of (usually) chance mutations. We cannot assume therefore evolution has achieved any particular end through means which we would think sensible. Nor can we assume that evolution will produce any particular end at all, however desirable. As Horace Shipp put it:

> There lived a happy coelocanth
> In dim primordial seas,
> He ate and mated, hunted slept
> Completely at his ease.
> Dame Nature urged: 'Evolve!'
> He said: 'Excuse me Ma'am,
> You get on making Darwin,
> I'm staying as I am'.

### References


Duffy, E. (1941). An explanation of 'emotional' phenomena without the use of the concept 'emotion'. *Journal of General Psychology, 25,* 283-293.

Duffy, E. (1962). *Activation and behaviour.* New York: Wiley

Ekman, P., Levenson, R.W., & Friesen, W.V. (1983). Autonomic nervous system activity distinguishes among the emotions. *Science, 221,* 1208-1210

Gray, J.A. (1982). *The neuropsychology of anxiety: an enquiry into the functions of the septo-hippocampal system,* Oxford: Oxford University Press

Hofer, M.A. (1972). Physiological and behavioural processes in early maternal deprivation. In R. Porter, & J. Knight, (Eds) *Physiology, emotion and psychosomatic illness,* CIBA Symposium No:8 (new series). Elsevier.

James, W. (1968). What is an emotion? In M.B. Arnold, (Ed) *The nature of emotion,* Harmondsworth: Penguin Books (Penguin Modern Psychology Series — reprinted from *Mind, 9,* 188-205)

Krebs, J.R., Stephens, D.W., & Sutherland, W.J. (1983). Perspective in optimal foraging. In G.A. Clark, & A.H. Brush, (Eds) *Perspectives in ornithology.* Cambridge: University Press.

Lader, M., & Tyrer, P. (1975). Vegetative system and emotion. In L. Levi, (Ed) *Emotions: Their parameters and measurement.* New York: Raven Press

Land, E.H. (1959). Experiments in colour vision. *Scientific American, 200,* 84-89

Mason, J.W. (1975). Emotion as reflected in patterns of endocrine integration. In L. Levi, (Ed) *Emotions: Their parameters and measurement.* New York: Raven Press

McNaughton, N. (1985). Chlordiazepoxide and successive discrimination: different effects on acquisition and performance. *Pharmacology, Biochemistry and Behaviour, 23,* 487-494

McNaughton, N. (1989). *Biology and emotion.* Cambridge: Cambridge University Press.

Monod, J. (1974). *Chance and necessity.* Glasgow: Fontana Books/ William Collins

Tripp, G., & McNaughton, N. (1987). Naloxone fails to block the effects of chlordiazepoxide on acquisition and performance of successive discrimination. *Psychopharmacology, 91,* 119-121