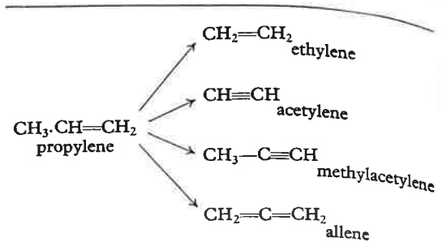
We are using this method to study the pyrolysis of the simple hydrocarbon, propylene, C_3H_6. The products which have been detected are indicated in Table 9. Although it is not possible to obtain such useful kinetic data as with the previous methods, since any reactive intermediates present disappear during quenching, it would not have been possible to detect the products shown above by present spectroscopic methods.

This example, in particular, shows that the uses of the shock tube for scientific investigations are many and varied and suggests that many possible applications of the technique have yet to be realised.

TABLE 9

PRODUCTS OF SHOCK-TUBE PYROLYSIS OF PROPYLENE



DISAPPOINTMENT AND DRUGS IN THE RAT*

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IN RECENT YEARS our knowledge of the basic neurophysiology of motivation has been greatly increased by experiments, pioneered by James Olds in the United States in which animals (usually rats) with electrodes implanted deep in the brain are allowed to press a bar which either causes a small electric current to flow in their own brains or, alternatively, turns off a current which the experimenter caused to flow in their brains. It has been found that with certain placements of electrodes in

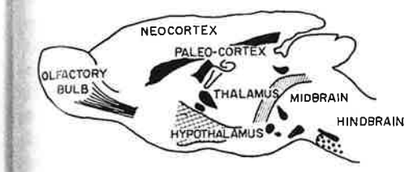

FIG. 1. The reward (medial forebrain bundle) and punishment (periventricular) systems in the rat brain. Olds and Olds (1965).

the hypothalamus and mid-brain (see Fig. 1) the rat will press a bar to stimulate its own brain electrically for hours on end. With certain other placements, the rat will be equally eager to press a bar to terminate or prevent the occurrence of electrical stimulation. Now it requires no great imagination to make the guess that these results indicate the existence in the brain of two fundamental motivational systems, a reward system and a punishment system; that is to say, the common denominator of the events which an animal finds rewarding (e.g. food, water, copulation, etc.) is that they cause an increase in the activity of the brain reward system, while the common denominator of such diverse punishments as electric shock, loud noises, sudden loss of support and so on is that they cause neurons to fire in the brain punishment system. Moreover, there is some evidence that these two systems interact with one another in important ways. It seems likely that activity in the one leads automatically to inhibition of activity ('reciprocal inhibition') in the other. It has also been suggested (Stein, 1964) that, after such a period of dampened activity, the inhibited system, when finally released from reciprocal inhibition, undergoes a short period of *increased* activity which has been termed a 'rebound effect'.

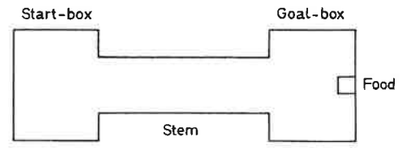

FIG. 2. Simple runway for learning experiments.

Now it seems possible that such a rebound effect may underlie the emotion of 'disappointment' or 'frustration'. Consider a rat which has been trained to run down a simple straight runway (Fig. 2) for a food reward which it finds in the goal-box at the end of the runway. We suppose that, while it is running down the runway, there is an anticipatory activation of the reward system of the brain. In more technical terms, the increase in activity in the reward system which occurs innately when a hungry rat ingests food comes to be set into operation, through the process of classical conditioning, by

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stimuli (such as those along the stem of the runway) which regularly precede the ingestion of food. This is supposed to occur in exactly the same way that a dog which is exposed to the regular sequence, bell–food, in Pavlov's famous conditioning experiments, comes to salivate at the sound of the bell alone. If, now, the rat fails to find food at its expected place in the goal-box (i.e. if it is exposed to 'frustrative non-reward'), there will be a sudden decrease in the activity of the reward system of the brain. The punishment system, which has been reciprocally inhibited while the reward system was activated, is now suddenly released from inhibition and there is a rebound increase in its activity of the punishment system which, subjectively, we feel as 'disappointment' or 'frustration'. If this is so, it follows that there is an important similarity, and perhaps even identity, between the effects of punishment and the effects of frustrative non-reward, for both involve activity in the same neural system, in the one case as a result of direct stimulation by a punishing event, in the other, as a rebound release from inhibition. We are led, therefore, to the hypothesis that, physiologically and functionally, the effects of punishment and those of frustrative non-reward are the same; or, in the language of the emotions, fear = frustration. It is to the testing of this hypothesis that the experiments I am going to describe were directed.

If we are to take this hypothesis at all seriously, it must first of all be shown that frustrative non-reward shares the most obvious properties of punishment. Now the defining characteristic of a punishment is that it is aversive, i.e. the organism will work to terminate or avoid it. The very least we must show, therefore, is that an animal will work to terminate or avoid frustration. The best demonstration of this is an experiment by Adelman and Maatsch (1956). They trained a group of rats to run down the familiar straight alley for a food reward. They then removed the reward from the goal-box, but carried on placing the animals in the start-box as before. (Technically, this is described as 'extinction' and the number of trials for which the animal continues to run to zero reward is taken to be a measure of 'resistance to extinction': I shall ask you to note these terms, as we shall meet them again.) They introduced one modification into the usual extinction schedule, which we can see in Fig. 3. Usually, what is done in extinction is that the animal is repeatedly placed in the start-box and allowed to run to the goal-box until eventually it gives up, stopping either in the stem of the runway or in the start-box itself. What Adelman and Maatsch did was to allow an alternative route of exit from the goal-box, by putting a ledge around this box on to which the rat could jump. A control group of hungry rats were rewarded with food on the ledge for jumping up to it. A second control group was never given food, either in the goal-box or on the ledge, but merely exposed to the goal-box and allowed to jump out. The remarkable finding made by Adelman and Maatsch was that the

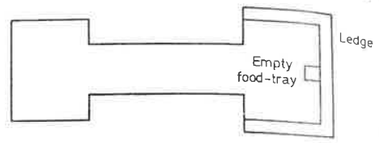

FIG. 3. Modified runway as used by Adelman and Maatsch (1956).

experimental group, which was jumping to get away from an environment in which it was being exposed to frustrative non-reward, learnt to jump out more quickly even than the food-rewarded controls, while both did much better than the control group which was neither rewarded nor frustrated. I think you will agree that this is a very powerful demonstration of the aversive nature of frustrative non-reward.

Another demonstration of the aversive properties of frustrative non-reward depends on showing that an animal will work to avoid stimuli *associated* with non-reward. Just as we assumed earlier that activity in the reward system is subject to classical conditioning (i.e. stimuli which are not in themselves rewarding may, if they regularly precede reward, acquire the power to activate the reward system), so we also assume that activity in the punishment system may be conditioned in the same way. This means that, if the fear = frustration hypothesis is correct, stimuli which regularly precede frustrative non-reward should become aversive. This has been shown to be the case by Wagner (1963). He trained rats to run down an alley but rewarded them on only a proportion of the trials; this is called a 'partial reinforcement schedule', another term which we shall be using a great deal. On the trials when they were not rewarded a distinctive stimulus (noise plus

light) was presented to them. They were then tested in an apparatus in which they were able to turn off this stimulus by jumping across a small barrier. It was found that they jumped the barrier significantly more often than control rats for which the stimulus had never been associated with frustrative non-reward. Thus it appears that associating the stimulus with non-reward had given it aversive properties similar to those acquired by a stimulus which is associated with punishment.

One other obvious feature of punishment is that it increases the vigour with which an immediately subsequent response is performed. We are all familiar with the excited behaviour which is likely to follow the receipt of a painful stimulus. The same is true of stimuli which are not themselves punishing but which have been associated with punishment. For example, Brown, Kalish and Farber (1951) showed that a stimulus which had regularly been followed by shock increased the magnitude of the startle reflex if it was presented to rats just prior to presentation of the stimulus (a gunshot) for the startle itself. Another preliminary demonstration which must be made, therefore, is that frustrative non-reward and stimuli associated with frustration have the same invigorating, or, as we say, 'drive-inducing' properties. Fortunately, there is evidence for both these claims. Thus, in the experiment by Wagner (1963) already described, in which a stimulus was associated with the non-rewarded trials of a partial reinforcement schedule, this stimulus was found to increase the magnitude of the startle reflex in exactly the same way that a stimulus associated with electric shock did in the experiment by Brown, Kalish and Farber (1951). The demonstration that frustrative non-reward itself (as distinct from stimuli associated with frustration) is drive-inducing was made by Amsel and Roussel (1952) in an experimental situation which has since been widely used. In this situation, the rat is trained to run down two alleys with two goal-boxes placed successively as shown in Fig. 4. In goal-box 1 it is on a partial reinforcement schedule, i.e. on average it is rewarded on 50 per cent of the trials, the sequence of rewards and non-rewards being determined at random. In goal-box 2 it is rewarded on every trial (described as a 'continuous reinforcement schedule'). If frustrative non-reward is drive-inducing we should expect that the rat will run faster in the second alley on trials on which it has not been rewarded in the first goal-box than on trials on which it has been rewarded there. And this is exactly what Amsel and Roussel found. Furthermore, in a later experiment, Wagner (1959) showed that this increase in speed of running the second alley after non-reward in the first goal-box only occurs if animals are *sometimes* rewarded in the first goal-box; for controls who are *never* rewarded there run no faster than the partially reinforced group does after its *rewarded* trials. It is clear, then, that the increase in speed is indeed due to the disappointment of an expectation, or, as we say, to frustrative non-reward. This interpretation of these results is now sufficiently well established for the increase in speed of running the second alley found by Amsel and Roussel to be called simply the 'frustration effect'.

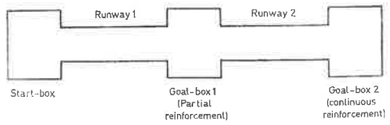

FIG. 4. Double runway as used by Amsel and Roussel (1952).

With these preliminary points out of the way, we turn to the experiments which have set out to test the hypothesis that frustration is the same state as fear. There are three main kinds of experiments which have been carried out in this attempt. One kind uses the general theory of learning in which the terms 'fear' and 'frustration' both figure to derive predictions concerning interactions between punishment and frustrative non-reward. A second (which includes my own work) takes drugs which are known to affect fear and uses them to try to affect responses to frustrative non-reward. The third kind takes animals known to differ in their susceptibility to fear and tries to find parallel differences in their susceptibility to frustration. Predictions of all three kinds have been verified, giving considerable support to the fear = frustration hypothesis.

We shall consider the evidence from a learning-theory experiment first, as this will be of some help in understanding the other kinds of experiment. This experiment, conducted by Brown and Wagner (1964), depends on the application of the concept of frustration to the phenomena associated with

partial reinforcement schedules. One of the best established findings in the whole study of animal (and human) learning is that a partial reinforcement schedule, compared to a continuous reinforcement schedule involving the same number of trials (and therefore a greater number of rewards), *greatly increases resistance to extinction*: that is to say, when reward is removed from the goal-box altogether the partially reinforced animal continues running down the runway for many more trials than does the continuously reinforced animal. Now, it is obvious that on the non-rewarded trials of a partial reinforcement schedule the rat is exposed to frustrative non-reward, so we would expect that a satisfactory theory of frustration would be able to offer an explanation for the partial reinforcement extinction effect. Such an explanation has been

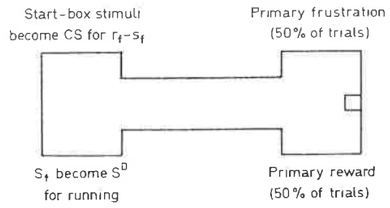

FIG. 5. The frustration theory as applied to the effects of partial reinforcement (see text for further explanation).

elaborated by Amsel (1958, 1962). We have already seen that stimuli which regularly precede a frustrating event are believed to acquire, by the process of classical conditioning, the capacity themselves to elicit a state of frustration. It is clear that, in a simple runway situation, the stimuli in the start-box and in the stem of the runway will become conditioned frustrating stimuli (CS) of this kind (see Fig. 5). Let us symbolize the conditioned response of frustration which these stimuli produce by r_f. We need now to introduce two new principles. First, we suppose that the state of frustration (or conditioned frustration, in the present case) can itself set up stimulation which is perceptible to the organism. This is a reasonable assumption, for, if it were not so, there would be no basis on which we could learn to say of ourselves that we are 'very disappointed' or 'feeling rather frustrated'. Let us symbolize the stimuli which result from the conditioned frustration response, r_f, as s_f. All that we need do now is to bring in the concept of a 'discriminative stimulus' (S^D). Consider a rat in a box in which it can obtain food by pressing a lever (a so-called 'Skinner box'). Let us arrange things so that food will be available for lever-pressing only when the box is lit; when it is in darkness pressing the lever has no consequences. We shall soon find that the rat will press the bar very frequently while the light is on, but hardly at all during darkness. 'Light on' has come to play the rôle of a signal to the rat that, if he presses the bar, he can obtain food. We call such a signal a 'discriminative stimulus'. If we now return to the special kind of stimulus, s_f, we can see that, on a partial reinforcement schedule, this stands to the act of running down the runway in the relation of discriminative stimulus, since there will very often occur the following sequence of events: r_f–s_f–run–food in goal-box. A continuous reinforcement schedule, by contrast, cannot set up an association of this kind. Thus when animals trained on the two kinds of reinforcement schedule are put on to full extinction, with its accumulating load of frustration, running will be relatively more probable in the partially reinforced animal for which the stimulus feedback from frustration has become a signal to 'keep on trying'. In other words we shall get the observed partial reinforcement effect.

With this preamble we can return to Brown and Wagner's experiment. If, as our hypothesis holds, the effects of frustrative non-reward are closely similar to the effects of punishment, it should be possible to substitute a punishment for frustrative non-reward, and vice versa, and make very little difference to the results obtained. Now, just as a partial reinforcement schedule is a way of training an animal to continue running down a runway in spite of frustration, so there is a method to keep an animal responding in spite of punishment. What you do is to introduce the punishment (in this case, an electric shock) initially at a very low level of intensity and then gradually increase the intensity from trial to trial. In this way, you can eventually get an animal to tolerate a much more intense shock for the sake of the reward (which, of course, must also be present) than if you suddenly introduce the shock at full strength. What Brown and Wagner did, then, was to train three groups of rats: one on an ordinary continuous reinforcement schedule (as controls), a second on an ordinary partial reinforcement schedule, and the third on the special schedule of continuous reinforcement plus gradually increasing punishment which has just been described. Each group was then divided into two, one half being put on a normal extinction schedule, the other being given continuous reinforcement plus punishment. As would be expected from the fear = frustration hypothesis, the partial reinforcement group showed greater resistance to *punishment* in this second phase of the experiment than did the continuous reinforcement group; and the punishment group showed greater resistance to *extinction* than did the continuous reinforcement groups (see Fig. 6). Thus, tolerance for frustration carries with it tolerance for punishment, and vice versa.

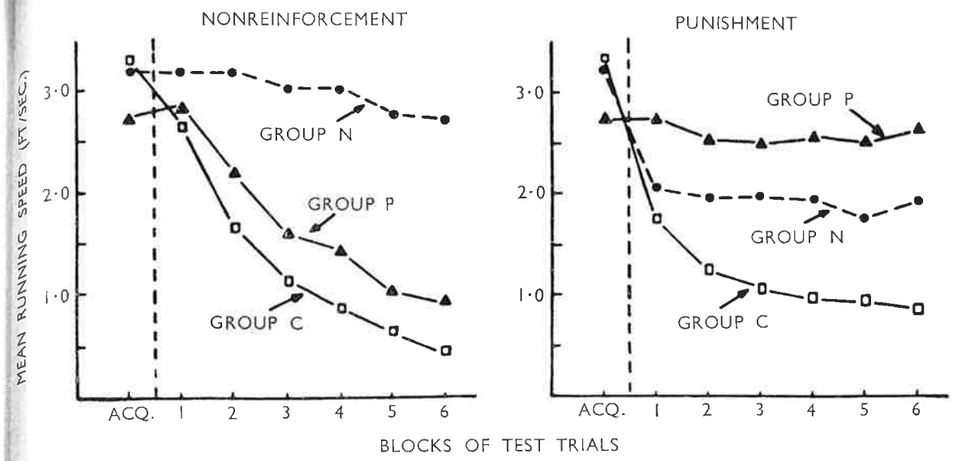

FIG. 6. Mean running speeds on the last day of acquisition and subsequent daily blocks of six test trials. Left-hand graph: performance of groups in extinction. Right-hand graph: performance of groups exposed to continuous reward and punishment. Group C was trained on a continuous reinforcement schedule. Group N was trained on a partial reinforcement schedule. Group P was trained with continuous reinforcement and gradually increasing punishment. Brown and Wagner (1964).

I want now to turn to the experiments which have tested the fear = frustration hypothesis by the use of drugs. Some time ago Neal Miller (1964) at Yale showed that alcohol and the barbiturate drug, sodium amylobarbitone, have an antagonistic effect on fear. In the case of the former, this will not surprise anyone familiar with the concept of 'Dutch courage'. Clearly, our hypothesis must predict that these drugs will also be antagonistic to frustration. The most obvious derivation of this argument is that the fear-reducing drugs should increase an animal's resistance to extinction, for we have argued that, in extinction, it is frustration which brings an animal's responding to a halt. This hypothesis was tested by Barry, Wagner and Miller in 1962 and upheld, and I have recently confirmed this finding, as you can see from Fig. 7.

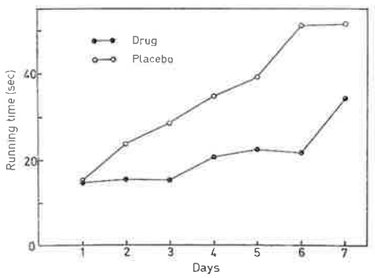

FIG. 7. Increased resistance to extinction produced by injection of amylobarbitone (the 'drug' curve) during extinction. The 'placebo' animals received a control injection of saline. In this and subsequent figures the dose of amylobarbitone used was 20 mg/kg i.p.

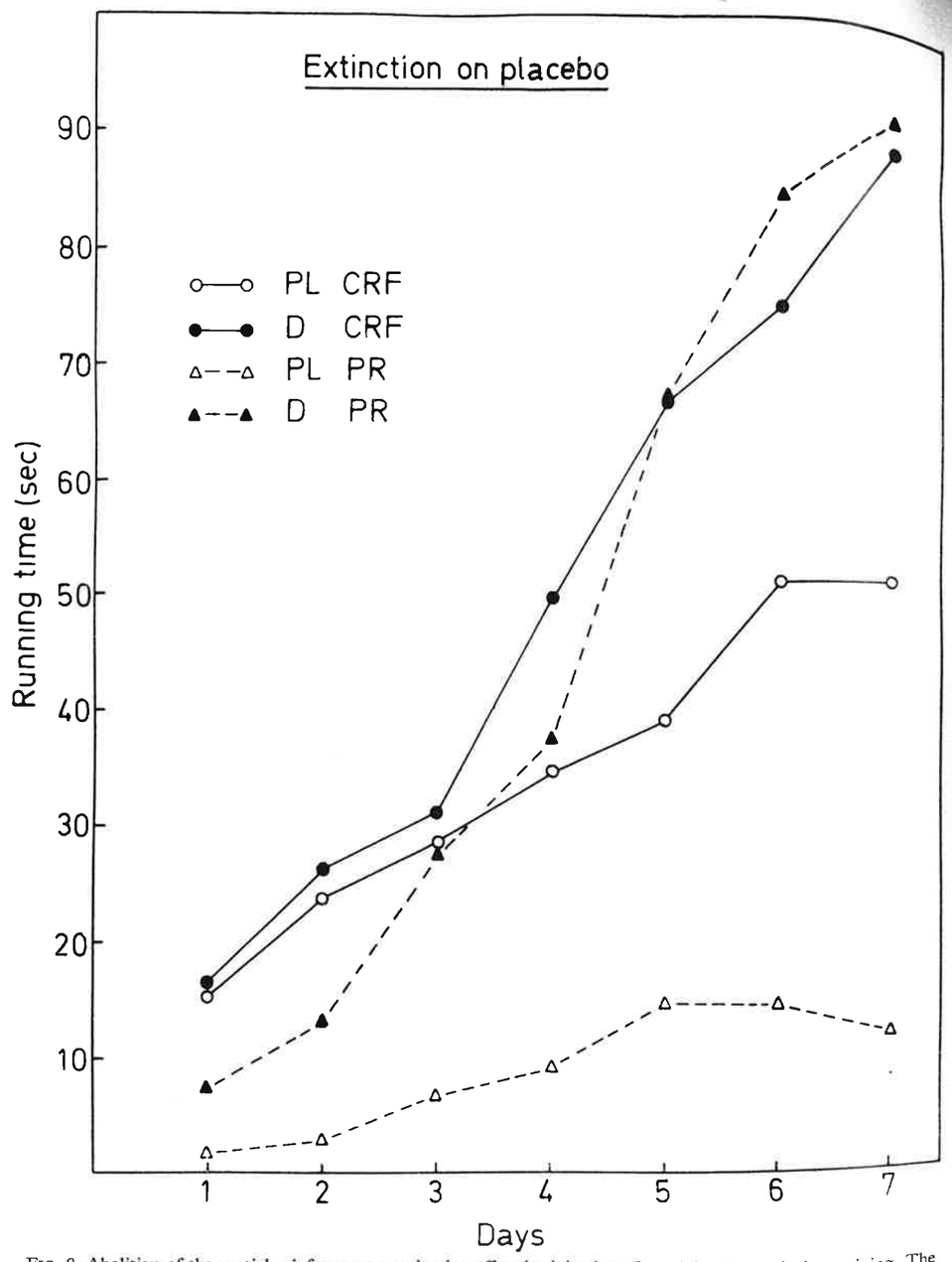

FIG. 8. Abolition of the partial reinforcement extinction effect by injection of amylobarbitone during training. The curves show running time as a function of days of testing during extinction. All animals received saline injections during extinction. CRF=continuous reinforcement during training; PR=partial reinforcement during training. D=amylobarbitone during training; PL=saline during training.

Next, consider the increased resistance to inction produced by partial reinforcement, and e explanation offered for this phenomenon by ustration theory. We have seen that this theory pposes that, on a partial reinforcement schedule, e animal learns to tolerate frustration, to use it, in ct, as a signal that reward is available for further ving. Clearly, if the fear-reducing drugs also reduce frustration, administering them to an animal on a partial reinforcement schedule should remove his opportunity to learn frustration-toleration, and thus block the partial reinforcement effect. Figure 8 shows that this is indeed the case: there was no difference in resistance to extinction between partially reinforced and continuously reinforced animals when both groups were given

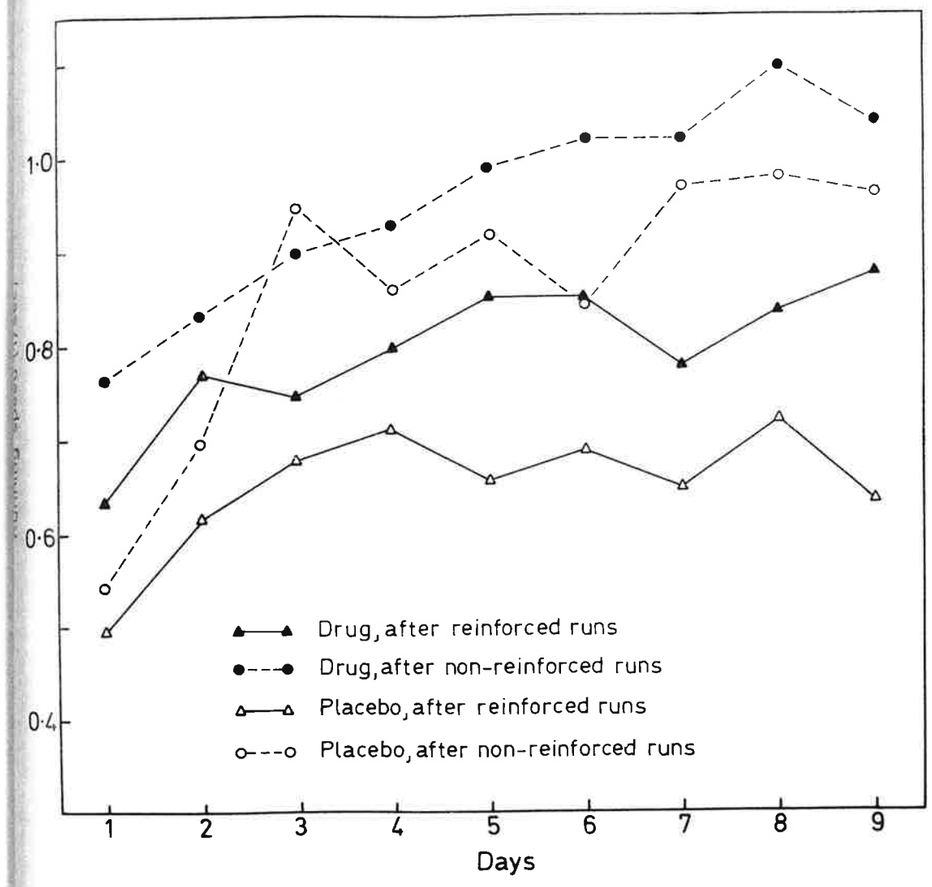

FIG. 9. Failure to reduce the double runway frustration effect by injections of amylobarbitone. The figure shows unning speed in the second alley on successive days of training, separately for trials after reward in the first goal box riangles) and trials after non-reward in the first goal box (circles). The 'drug' group received daily injections of mylobarbitone; the 'placebo' group received saline injections.

amylobarbitone during training and tested with a placebo (saline) injection during extinction.

Having successfully tested these rather complex derivations from the hypothesis, I naturally expected no trouble when I tested a simpler prediction, namely that the fear-reducing drugs would reduce the Amsel double-runway frustration effect (the increased speed of running in a second runway after non-rewarded trials in a first goal-box; see Fig. 4). However, upon doing the experiment, I found no effect of amylobarbitone on this phenomenon whatsoever, as you can see from Fig. 9. This was all the more surprising as in the earlier experiment the drug *had* abolished a phenomenon which is theoretically very similar to the Amsel frustration effect. This is the phenomenon known as the partial reinforcement *acquisition* effect, as distinct from the partial reinforcement *extinction* effect. It consists in the fact that, when training has proceeded to the point at which the animals are running as fast as they ever will, partially reinforced animals run faster than continuously reinforced animals. We attribute this to the effect of the conditioned frustration which we believe the animal to be subjected to in the start-box: this is thought to have drive-inducing effects (leading to the partially reinforced animal's greater speed) exactly similar to the drive-inducing effect observed in the double-runway situation. Figure 10 shows that, as we would expect, amylobarbitone abolished this

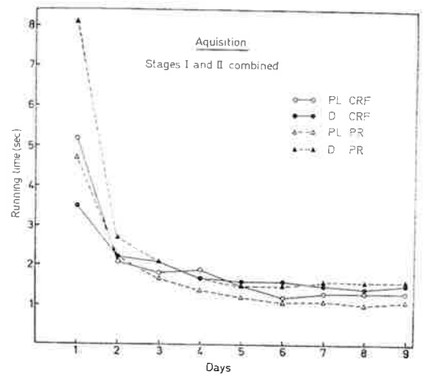

FIG. 10. Abolition of the partial reinforcement acquisition effect by amylobarbitone. The figure shows running time on successive days of acquisition training. Conventions as in Fig. 8.

superiority in speed for the partially reinforced animal just as it abolished the increased resistance to extinction. Why, then, did it not reduce the Amsel frustration effect?

At this point, the experiments seemed to be at an impasse. However, there appeared to be one possible explanation. The effects of frustrative non-reward which amylobarbitone *had* succeeded in blocking were all theoretically due to the action of *conditioned* frustrating stimuli. Both the termination of responding which occurs in extinction and the effects of partial reinforcement are thought to be due to the action of stimuli *prior* to the point of non-reward which have acquired their aversive properties as the result of classical conditioning. The Amsel frustration effect, on the other hand, is an *unconditioned* effect of frustrative non-reward: it is observed as a change in behaviour *after* the point of non-reinforcement. Moreover, a similar phenomenon exists in the effects of the fear-reducing drugs on behaviour motivated by punishment: Barry and Miller (1965) had shown that amylobarbitone and alcohol reduce the intensity of *avoidance* behaviour, where the animal reacts to stimuli associated with punishment, without experiencing the punishment itself, but have less effect on *escape* behaviour, where the animal is first exposed to the punishment and is then able to make some response which terminates it. Perhaps then, the puzzling results I had obtained could be accounted for by the hypothesis that amylobarbitone affects conditioned fear and frustration but not unconditioned fear and frustration. I resolved, therefore, to test this hypothesis by using the Adelman and Maatsch situation which I have talked about earlier.

You will remember that the Adelman and Maatsch technique of extinction differs from the usual kind in that the animal, instead of stopping in the stem of the runway, i.e., prior to the point of non-reward, enters the now empty goal-box and then escapes from it by jumping up on to a ledge (Fig. 3). This offers us an ideal way of testing the hypothesis that amylobarbitone affects conditioned frustration but not unconditioned frustration. We know already that this drug increases resistance to extinction on a normal extinction schedule. If the hypothesis is correct, it should *not* alter behaviour in Adelman and Maatsch's extinction situation. Figures 11 and 12 show the results I obtained when I

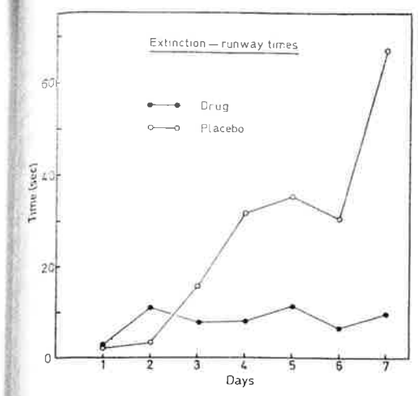

FIG. 11. Replication of the increase in resistance to extinction produced by amylobarbitone injections during extinction. Same conventions as Fig. 7, which shows the same result.

did this experiment. As you can see from Fig. 11, amylobarbitone once more slowed down extinction carried out in the normal manner. But (Fig. 12) it also slowed down 'jump-out' extinction—the drugged animals learnt to jump out of the frustrating situation more slowly than the placebo controls. Note that this experiment leads to several conclusions. On the one hand it disproves the special hypothesis I introduced to account for the failure of amylobarbitone to reduce the double-runway frustration effect (namely, that *unconditioned* frustration is resistant to the drug). On the other hand, it increases our confidence in the general theory. It would be impossible to account for the effects exerted by amylobarbitone in this experiment by supposing that this drug alters *motor* behaviour; for the drugged animals ran *faster* into the empty goal-box but jumped *slower* out of it. But these results are easy to understand on the hypothesis that amylobarbitone reduces frustration, so that the drugged animals are both less reluctant to enter a frustrating situation and less eager to get away from it.

We are left then with the problem of why the drug had no influence in the double-runway experiment. This brings us to the important question of individual differences. It so happens that the animals we used for the double-runway experiment were from a hooded strain of rats, whereas all the other experiments involved albino rats of the Wistar strain. The importance of individual differences (or 'personality') in susceptibility to frustration had already been shown by Savage and Eysenck (1964). They tested the fear = frustration hypothesis by taking animals known to differ in fearfulness and testing them on the Amsel double runway. The animals used were the Maudsley Reactive and Non-reactive strains, which have been selectively bred for over twenty generations to be, respectively, very susceptible to fear and very insusceptible to fear. The hypothesis clearly predicts that the former will show a greater frustration effect (i.e. a greater increase in speed after non-reward in the first goal-box) than will the latter. And this was indeed the case, as you can see from Fig. 13. We decided, therefore, to repeat our experiment on the effects of amylobarbitone on the double-runway frustration effect using the same albino rats as in the other drug experiments. So far

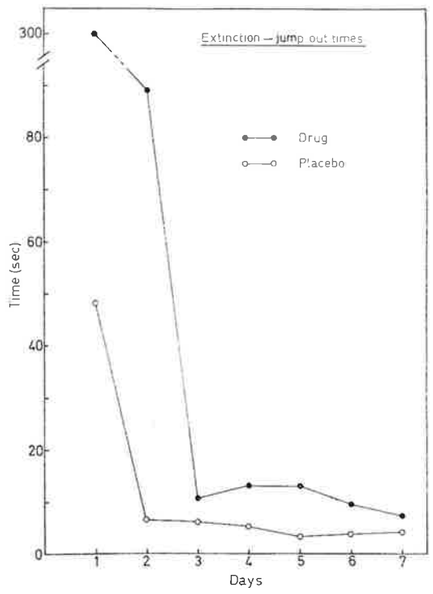

FIG. 12. Reduced speed of jumping out of now empty goal-box as a result of injections of amylobarbitone during extinction with the Adelman and Maatsch (1956) technique (see Fig. 3).

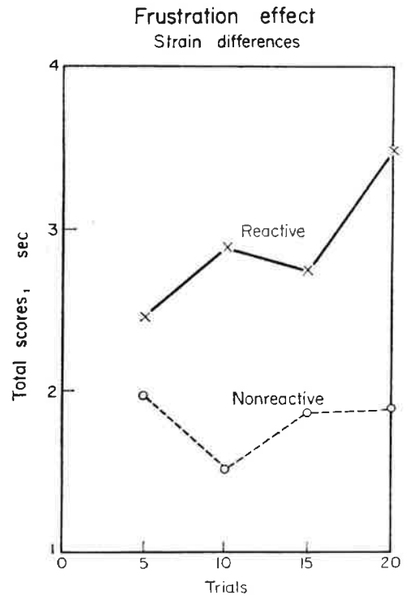

FIG. 13. The greater double runway frustration effect shown by Maudsley Reactive ('fearful') animals as compared to Non-reactive ('fearless') animals. The figure shows the difference between second alley running times on trials with and without reward, respectively, in the first goal box for each of the two kinds of animals. Savage and Eysenck (1964).

we have only obtained preliminary results, but these are encouraging: there are definite indications that amylobarbitone *does* reduce the frustration effect. Our earlier failure to obtain this result, then, was probably due, in part, to the fact that unconditioned frustration is *more* resistant to the effects of drugs (though not entirely resistant); and, in part, to the use of animals which are either highly susceptible to frustration or relatively insusceptible to the effects of amylobarbitone.

In sum, I think we might conclude that a fair amount of evidence supports the fear = frustration hypothesis. I think it is plausible that both emotions consist, neurophysiologically, in the activation of the punishment system of the brain described by Olds on the basis of self-stimulation studied with implanted electrodes; but that is far more speculative. I hope the experiments I have reported have thrown some light on the mechanism of action of the barbiturate drugs (and perhaps of alcohol as well). We are, I believe, better placed to understand the phenomenon of addiction to these drugs if we realize that they can be used to counteract the effects of disappointment and frustration, which we must all contend with often enough. Finally, the finding that rats selectively bred to be very fearful are also highly susceptible to frustration means that we must greatly expand our concept of the nature of neuroticism; it might well be that human neurotics, who have usually been thought of as especially prone to anxiety, are also highly susceptible to frustration.

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