PSYCHOSIS, DRIVE AND INHIBITION : A THEORETICAL AND EXPERIMENTAL ACCOUNT ¹

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INTRODUCTION

For the past 10 years or so the writer and his colleagues have been attempting to discover objective psychological tests in the cognitive, perceptual and motor fields which would differentiate psychotic patients from normal subjects and neurotic patients of similar age, sex and intelligence(10, 11, 15, 20). It was discovered that quite large numbers of tests did in fact differentiate the psychotic groups from the others, and it was further found that the one feature which all these tests seemed to have in common was a generalised pattern of slowness which seemed to cover the areas of perception, cognition and motor reaction(35). These results fit in rather well with those obtained by many other investigators(1, 2). and recent reviews have adequately covered this field so that it will be unnecessary to do so again here (17).

Another outcome of the series of researches mentioned above has been to clarify the *dimensional description* of mental abnormality. Analyses of objective test scores made by schizophrenic, manic depressive, hysterical, psychopathic, anxious, obsessional and depressed patients, as well as normals, have shown clearly that these diagnostic categories did not in any sense refer to categorical disease groups but are rather located at various points of a 3-dimensional framework, the three axes of which are psychoticism, neuroticism, and extraversion/introversion. These analyses have been carried out by making use both of factor analysis and of multiple discriminant function analysis, and the results of these quite divergent techniques have been astonishingly similar (10, 11, 13, 14, 18, 24, 36).

As regards the descriptive or nosological side of the investigation, therefore, the results seem to be clear-cut and relatively straightforward. Psychotic disorders are essentially unlike neurotic disorders; both

manic depressive and schizophrenic patients react very similarly on large batteries of objective tests; and it is possible to generalise a large number of different experimental findings by stating that psychotic subjects, in every aspect of their behaviour, tend to be abnormally *slow*. This statement of course does not exclude the possibility that psychotic subjects are also characterised in other ways which may not themselves depend on generalised slowness; it has however been our experience that many of the symptoms of thought and behaviour disorders of psychotic patients can ultimately be reduced to derivatives of general slowness. It is also not intended to convey the impression that there are no differences between schizophrenics and manic depressives; recent studies from this laboratory have indeed provided significant evidence of differentiation between these two groups (17, 36). Nevertheless, as far as mental slowness is concerned, both the two major groups of functional psychotics appear to be characterised by very similar reactions, and it will be the purpose of this paper to suggest a theory to account for these findings.

The sequence of events leading from psychiatric nosology through psychometric and empirical objectification to hypothetico-deductive theoretical and experimental analysis duplicates that which has characterised the set of studies carried out in the field of neurosis(9, 15, 18), and it is interesting to note that in the neurotic field this type of progress finally led to the elaboration of a rational system of classification, diagnosis and therapy (19). It is the writer's belief that advance in abnormal psychology and psychiatry is most likely to come by the pursuit of some such series of steps, and it may not be entirely fanciful to imagine that problems in the aetiology and treatment of psychosis may in the long run also find an answer along these lines.

THEORETICAL ANALYSIS

The concept of *explanation* in psychiatry

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and psychology has always presented some difficulties. It is customary to explain the less well-known by the better known, but in the field of the social sciences there has been so little agreement on what is known and what is merely hypothetical that it proved impossible to follow the usual course of scientific explanation. The writer(15), has argued that the recent advances in modern learning theory (26, 39) have furnished us with a set of concepts and laws which, while far from perfect, are nevertheless sufficiently precise and well validated to make it possible to account for behavioural observations in terms of these concepts and laws, and to make experimentally testable deductions from such theories. Hitherto the writer has concentrated on neurotic disorders in his attempts to apply modern learning theory to psychiatry(15); the present paper is a first attempt to make such an application to the field of psychosis.

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Without entering into all the complexities which a detailed analysis along Hullian lines would necessitate, we may perhaps state quite broadly that most modern learning theorists would agree with some such statement as this :

P = (f) D x H;

i.e., performance is a multiplicative function of drive and habit. Drive in this formulation means the motivational state of the organism which makes it perform the task in question, while *habit* relates to the particular connexions in the central nervous system which have been made through previous learning and which facilitate the performance in question. One immediate hypothesis which arises from this formula would be one which would account for the worse (slower) performance of the psychotic in terms of lower drive; indeed, this is of course the type of explanation which is normally given in text books of psychiatry and clinical psychology.

An alternative hypothesis suggests itself when we consider an additional concept, namely that of *reactive inhibition* (usually abbreviated and symbolised as IR). This is conceived to be a kind of neural fatigue, of central origin, which develops after any kind of perceptual, cognitive or motor performance and dissipates in time. During massed practice this inhibition accumulates, as there are no rest periods during which it can disappear. Now I_R, which subjectively is experienced as boredom, fatigue or even pain, is regarded as a negative drive, *i.e.*, a tendency to make the organism stop doing whatever it is doing. As such it is subtracted from D(30), so that the "drive" in our original equation is really equal to $(D-I_R)$; this combination of positive and negative drive may be symbolised as D (effective drive).

We now have an alternative hypothesis to account for the poor performance of psychotics. We may assume that reactive inhibition either accumulates faster or dissipates more slowly in psychotic patients or both; this would leave them in a state of lower D, even though they were equal or even superior to normal and neurotic subjects with respect to D. As inhibition grows fairly quickly even in normal subjects, the main interest of this hypothesis would centre on the hypothesised slower rate of dissipation, and in order to account for the very marked differences in behaviour between psychotics and other groups it would be necessary for this slowing down in the rate of dissipation to be quite severe. We thus end our consideration of theoretical possibilities with two alternative hypotheses, both or either of which would, if true, explain a good deal of psychotic behaviour :

- 1. Psychotics have lower D (motivation) than normal or neurotic subjects.
- 2. Psychotics have a slower rate of dissipation of I_R (reactive inhibition), and are consequently nearly always working under a lower D (effective drive).

It will be the task of the next section to deduce from general learning theory certain consequences, experimentally testable, which should follow if these hypotheses were true, and demonstrate that in fact these deductions are borne out.

DEDUCTIONS FROM THE THEORY

In order to test the two hypotheses outlined above it is clearly necessary to be able to measure drive and reactive inhibition. Measurement of IR fortunately presents little difficulty in principle. During massed practice IR accumulates, because there are no rest pauses during which it can dissipate. If now we introduce a rest pause into the proceedings sufficiently long for all or nearly all of IR to dissipate, then we would expect an improvement in performance after the rest pause. This prediction is based on the simple consideration that before the rest pause D<D, because $D = D - I_R$. After the rest pause, however, D = D, because IR has disappeared completely. Now H is not affected by the rest pause, and consequently the product of D x H, *i.e.*, the performance of the subject, will be greater after the rest pause than before. This well-known phenomenon is called reminiscence, a name given to it for historical reasons which would not recommend themselves to modern investigators, but which have embedded the the term so firmly in our theoretical framework that it would be impossible to dislodge it now(34). We may say then that reminiscence can be used experimentally as a measure of IR, provided certain simple conditions are fulfilled.

It has also been proved possible to use reminiscence as a measure of motivation or D. The early theoretical and experimental work of Kimble(31) and Wasserman(43)is relevant here. Kimble argued that reactive inhibition during massed practice would increase until it reached the same level as D. At that point $I_R = D$ and consequently $D = D - I_R = 0$. According to our formula performance should stop at this point as the equation would read : P =0 x H, which is itself zero. Accordingly at this point there would occur involuntary rest periods (I.R.P.s) during which the organism ceased functioning and during which inhibition would dissipate. When sufficient inhibition had dissipated for D to be a large enough positive quantity, performance would begin again, IR would accumulate once again, thus enforcing another rest pause. From the moment that the critical point had been reached where D = IR, the organism would therefore continue working in a series of fits and starts. There is good experimental evidence for the reality of such involuntary rest pauses in the analysis of behaviour(3, 4, 25, 33, 37, 42) and also in the electro-physiological analysis of EEG and other autonomic measures which demonstrate a momentary sleep-like state of the organism during these I.R.P.s. (44,5).

If the critical level at which these involuntary rest pauses begin occurs at the point where $D = I_R$, then an organism working under low drive would clearly reach the critical level earlier than an organism working under high drive, and equally the amount of I_R tolerated by the organism working under high drive would be greater than the amount of I_R tolerated by an organism working under low drive. This is illustrated in Figure 1, with the letters H and L referring respectively to groups of subjects having high and low motivation.





It will be seen from Figure 1 that after 6 minutes or so of practice both the high and the low drive group have reached their respective critical levels where $D = I_R$. Now we have already established that reminiscence is a good measure of I_R; in other words reminiscence = (f) I_R. We have also seen that at the point where involuntary rest pauses begin, and from thence onward, $I_R = D$. We can take these two equations together and make them read : reminiscence = (f) D; in other words the amount of reminiscence observed under the experimental conditions described is a function of the drive under which the organism is working.

That this hypothesis is correct can be seen from Figure 2 which is a summary of two studies carried out by Eysenck and Maxwell(21) and Eysenck and Willett(22) working with fairly large groups of industrial apprentices under conditions of high or low motivation respectively; these workers used as their experimental task the pursuit rotor. Rest pauses of 6 minutes were introduced at different points of practice for different groups of apprentices; as will be seen in Figure 2, the rest pause was introduced either after 2, 3, 6, or 8 minutes of practice. As predicted, the reminiscence scores of the low drive group remained at the same level throughout; those of the high drive group however grew as a straightline function of length of practice. We may conclude therefore that reminiscence is indeed a good measure of drive under certain specified conditions.





EXPERIMENTAL PROOF OF THE THEORY

We are now in a position to test our theory directly in relation to the performance of psychotic groups. First let us consider the kind of prediction which follows from both our two hypotheses. According to the first hypothesis, psychotics are characterised by low drive, and in view of the extreme nature of their performance defects we would have to postulate a very low drive state indeed to account for the defects. If this were true then it would follow directly from the considerations advanced in the last section that psychotics would have very little or no reminiscence regardless of the length of the rest pause, while normal and neurotic subjects under similar conditions would have reasonably high reminiscence scores. This result, it should be emphasised, would be expected to hold true regardless of the actual level of performance of our psychotic groups. It is one of the advantages of the method of measurement of motivation suggested here that differences in ability or previous training on the task do not affect the measurement to any considerable degree as reminiscence is measured in terms of relative change from pre-rest to post-rest performance, and not in absolute terms.

A similar prediction can be made from our second hypothesis. The phenomenon of reminiscence as produced by the dissipation of I_R; if, as the hypothesis states, I_R does not dissipate to any extent during the rest period in psychotics, then reminiscence would be low or nonexistent. We see therefore that both hypotheses predict unequivocally the absence of reminiscence in psychotic groups under conditions where reminiscence is clearly observed in normal and neurotic groups.

Two studies have been carried out in our laboratory giving strong support to this deduction from our theory. Broadhurst and Broadhurst(6), using both schizophrenic and manic depressive subjects of a chronic kind, demonstrated the complete failure of these subjects to show reminiscence effects on the pursuit rotor. At the same time, and quite independently, Claridge(7), also using the pursuit rotor, found a similar failure of reminiscence to appear in pursuit rotor performance on the part of young and undeteriorated schizophrenics (cf, Figure 3). Other investigations from the Institute of Psychiatry (40, 41) have also been interpreted in a similar manner although the results were not quite as clear-cut; this may have been due to the use of tasks less well suited than the pursuit rotor to the measurement of reminiscence. We may conclude therefore that the deductions from the general theory outlined in a previous section have good empirical support. It is interesting to note at this point that early investigations by Hoch(27) and Kraepelin(32), using the ergograph and the Rechenheft, suggested theoretical solutions not essentially different from those here attempted. These early studies were not adequately controlled, of course, and the type of activity studied did not produce results particularly well suited for this kind of analy-



A CRUCIAL EXPERIMENT TO DECIDE BETWEEN THE TWO HYPOTHESES

The results so far reported would follow regardless of which of our two theories happened to be correct. We must now consider the possibility of designing an experiment to discriminate between the two theories. It has been pointed out in the last section that if the first hypothesis were true, i.e., that accounting for psychotic performance in terms of low drive, then reminiscence scores should be low for psychotic subjects regardless of the length of the rest period. This, however, would not apply to our second hypothesis, which postulates that inhibition in schizophrenics would dissipate very much more slowly than in normals and neurotics. If the second hypothesis were true, therefore, we would expect that after a rest period of, say, 24 hours psychotics also would show strong reminiscence effects. Here then we would appear to have a crucial test to decide between our two hypotheses. It might of course be possible that both hypotheses were correct; in that case we would find a certain degree of reminiscence in psychotics after a 24 hour rest period, but this would still be below the reminiscence scores of comparable groups of normals and neurotics. The difference between the reminiscence scores of these groups would then

give us a measure of the difference in D. It would also be possible of course to record the rate of dissipation of IR in psychotics by varying the length of the rest interval (30 minutes, 1 hour, 6 hours, 12 hours, 24 hours, etc.); this should enable us to plot in detail the decline of IR and demonstrate in a quantitative fashion the differences between psychotics and other groups. It should also be possible to use reminiscence measures of this type as an index of recovery, assuming that recovery from psychotic illness was indeed accompanied by a lessening in slowness and an increase in D, or an increase in the speed of dissipation of IR.

Some preliminary data are available with respect to the crucial experiment here proposed. Ley, in an unpublished study, tested 10 psychotic males with a rest interval of 10 minutes on the pursuit rotor, and another 10 with a rest interval of 24 hours. He also tested two groups of 10 normal subjects with 10 minute and 24 hour rest pauses respectively. He found at a good level of statistical significance that while after the 10 minute rest pause the normals had high positive reminiscence scores, those of the psychotics were for practical purposes equal to zero. After 24 hours he found that the reminiscence scores of the normals were somewhat lower than after the 10 minute rest pause; those of the psychotics however were now higher than those of either of the two normal groups. It would appear, therefore, that the hypothesis relating psychotic behaviour to low drive is untenable and that we must conclude that psychotic reactions are characterised by an excessive slowness of dissipation of inhibition.

This conclusion is supported by a recent study by Rachman(38), in which he had 10 chronic schizophrenics perform on the pursuit rotor with a rest pause of 10 minutes, while another group of schizophrenics performed with a rest pause of 24 hours. As pointed out above, normal subjects under these conditions show less reminiscence after the longer rest interval; as will be seen from Figure 4, the schizophrenics produced the opposite effect, with the 24 hour period significantly superior to the 10 minute one with respect to the size of the mean reminiscence score. Rachman's subjects were all *male* patients of long standing; he benefited from Ley's finding that the poor performance of women generally, allied to the poor performance of schizophrenics generally, makes it almost impossible to test theories of this kind in schizophrenic women, as their performance does not easily leave the chance level; in order to work with women, the task has to be simplified, either by slowing down the rate of rotation of the target disc, or by enlarging the target disc. This has not to our knowledge been done hitherto.



It is possible to look upon the reports of Huston and Shakow(28, 29), supplemented by those of S. B. G. Eysenck(23), as providing some further support for the theoretical view under consideration. Huston and Shakow found with psychotic patients that reminiscence effects appeared after several months, using the pursuit rotor, while Eysenck failed to find such effects in normals after periods of 12 months. The analyses carried out by these workers are not sufficiently similar to place too much weight on the findings, but as far as they go they do seem to support the notion of slow rate of dissipation of IR in psychosis, rather than that of lack of drive.

The negative conclusion of these studies regarding the lack of drive in psychotics is in good agreement with the results of another experiment(13) in which the persistence of psychotics on mental tasks was compared with the persistence of neurotics and normals, the assumption being that persistence is to some extent a measure of drive. It was found that psychotics were significantly more persistent, as well as much slower. Elsewhere Eysenck(16) has shown that slowness in problem solving is a function of I_R. While this experiment on persistence is less conclusive than the series of experiments reported here, it nevertheless points in the same direction and may be used to strengthen the point made.

We may conclude therefore from this series of studies that an important aspect of psychotic behaviour is related to, and possibly caused by, the slow rate of dissipation of reactive inhibition in psychotics. It must remain the task of further experimental studies to verify these results, to indicate to what extent they apply to other types of test, to show to what extent the rate of dissipation of inhibition can be used to account for the observable defects of the psychotic, to what extent this rate is amenable to change by different types of treatment, and to what extent it mirrors the clinical state of the patient. This is a formidable list of research projects, to which should be added one additional project which takes us right out of the field of psychological investigation. There appears to be some evidence suggesting a link between inhibition and the activity of the ascending reticular formation(8), and a study of this link, with the slow rate of dissipation of inhibition of psychotics particularly in mind, would seem a very worth-while task.

SUMMARY AND CONCLUSIONS

An attempt has been made in this paper to account in theoretical terms for the generalised slowness which has been shown in previous work to characterise psychotic patients. Making use of the theoretical framework of modern learning theory, two hypotheses were put forward, relating psychotic slowness respectively to : 1. Lack of drive (motivation); 2. An exceedingly slow rate of dissipation of reactive inhibition. It was deduced from the theorems of learning theory that psychotics should be characterised by very low reminiscence scores on tasks involving massed practice if either of the two hypotheses were correct, and experimental evidence does indeed show that psychotics are differentiated from normals

and neurotics in precisely this way. It was then argued that a crucial test between the two hypotheses could be performed by varying the length of the rest period used for establishing reminiscence scores, and it was found that while with *short* rest periods psychotics showed *no* reminiscence, they did show very *strong* reminiscence effects after *long* rest periods. This is interpreted as irreconcilable with the hypothesis of low drive in psychotics, and may be used as support for the hypothesis that *psychotics are characterised by a slow rate of dissipation of inhibition*.

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