# Hemsley DR. A simple (or simplistic?) cognitive model for schizophrenia. Behav Res Ther 31: 633-645

**Article** *in* Behaviour Research and Therapy · October 1993

DOI: 10.1016/0005-7967(93)90116-C · Source: PubMed



**1 author:**

David Roy Hemsley
King's College London
**185** PUBLICATIONS **7,710** CITATIONS

*Behav. Res. Ther.* Vol. 31, No. 7, pp. 633–645, 1993
Printed in Great Britain. 

0005-7967/93 $6.00 + 0.00


## INVITED ESSAY

# A SIMPLE (OR SIMPLISTIC?) COGNITIVE MODEL FOR SCHIZOPHRENIA

DAVID R. HEMSLEY
Psychology Department, Institute of Psychiatry, Denmark Hill, London SE5 8AF, England

*(Received 30 March 1993)*

**Summary**—An approach which views schizophrenia as a disturbance of information processing appears promising as a way of linking biological and clininal aspects of the disorder. A review of research in this area led to the suggestion that the basic disturbance in schizophrenia is "a weakening of the influences of stored memories of regularities of previous input on current perception". This formulation leads to the prediction that in certain circumstances, schizophrenics may perform better than normal subjects. Recent studies employing tasks derived from human experimental psychology provide evidence in support of the model. It is argued that the link between information processing disturbances and biological abnormalities may be facilitated by the use of paradigms derived from animal learning theory (latent inhibition and Kamin's blocking effect). On both tasks the pattern of performance of acute schizophrenics is consistent with the cognitive model. The ways in which such an information processing disturbance may lead to schizophrenic symptomatology are outlined, with particular reference to the formation and maintenance of delusional beliefs. The core cognitive abnormality may result from a disturbance in any of the brain structures involved in the prediction of subsequent sensory input. The proposed circuit, which draws heavily on Gray's model, implicates in particular the hippocampus and related areas and is consistent with studies of brain pathology in schizophrenia.

## INTRODUCTION

It will be argued in this paper that developments in animal learning theory may be relevant to our understanding of the abnormalities of behaviour and experience characteristic of schizophrenia. A viable animal model would permit the direct investigation of brain structures and functions thought to underlie such symptoms, and would facilitate the study of the neural bases of the cognitive abnormality which is "still the elusive core of the schizophrenia syndrome" (Shepherd, 1987 p. 37). Ellenbroek and Cools (1990) have recently reviewed several animal models with construct validity for schizophrenia. The present paper emphasizes the relationship between such models and the disturbances of information processing which have traditionally been studied in an attempt to integrate biological and social factors relevant to the disorder.

In 1914 Berze wrote that in schizophrenia "Perception and memory have become so hopelessly intermingled"; the observation still appears relevant to a number of phenomena which are prominent in the disorder. One would therefore expect models of normal human information processing to assist us in specifying the pattern of psychological dysfunction from which the various abnormalities resulting in a diagnosis of schizophrenia might be derived. The implementation of this research programme has proved far from straightforward. In part this is due to methodological problems associated with schizophrenia research generally (cf. Hemsley, 1988 pp. 104–105). However, it is also apparent that there is no agreed large scale model of normal cognitive functioning. Each model employs a somewhat different conceptual framework, and is frequently designed to explain performance on a small range of tasks. Their more general applicability (or ecological validity?) has yet to be demonstrated. As Shallice (1988) has noted "large scale information processing models are very loosely characterized; adding a connection, a constraint, or another subsystem is unfortunately only too easy" (p. 32).

A further problem is that many of the key symptoms of schizophrenia represent alterations in conscious experience, and there are obvious difficulties in mapping constructs which have been generated to explain task performance on to experiential phenomena. Anscombe (1987) points out that "there remains a gap between the computer terminology in which attentional theories are

couched, and the patient's experience of schizophrenia". This issue is made more complex by Shallice's (1988) argument that "in most operations that have a conscious correspondence, many subsystems are involved" (p. 321). A frequently employed distinction is between those subsystems which are responsible for perception/action and a higher lever executive system which controls the activity of the former. Morris (1992) proposes that consciousness is associated with information being made available to the executive system by lower level systems, and/or by feedback on its own output. A crucial feature of this model for the purposes of the subsequent discussion is that information is likely to achieve conscious representation when there does not exist any pre-formed subsystems "to control that particular perceptual-motor skill" (p. 5).

The current emphasis on diagnostic reliability, while in many ways commendable, has led to a relative neglect of the more subtle perceptual/cognitive abnormalities observed in, and reported by, schizophrenic patients. However, they feature prominently in the work of Huber and his colleagues (e.g. 1986) who have long argued that such 'basic symptoms' may be most closely related to the enduring neural bases of the disorder. There has recently been a renewed interest in early descriptions of the ways in which schizophrenics' perceptions and/or thinking are disturbed. As Cutting (1989) points out, Matussek (1952) and Conrad (1958) were among the first to argue that the early stage of schizophrenia could be explained in terms of a breakdown of Gestalt perception, and that such a disturbance could form the basis of delusional perception, Matussek (1952) describes a patient who was aware of "a lack of continuity of his perceptions both in space and over time. He saw the environment only in fragments. There was no appreciation of the whole. He saw only details against a meaningless background" (p. 92). Arieti (1966) later used the term 'perceptual and apperceptual fragmentation' for such phenomena. Matussek also noted another patient's report that "I may look at a garden, but I don't see it as I normally do. I can only concentrate on details. For instance I can lose myself in looking at a bud on a branch, but then I don't see anything else" (p. 92). In a similar vein, Shakow's experimental work had led him to the conclusion that a schizophrenic "can't see the wood for the trees . . . and examines each tree with meticulous care" (Shakow, 1950).

This introduction has argued for the crucial role of perceptual/cognitive abnormalities in our understanding of the phenomena characteristic of schizophrenia. The paper will go on to consider the development of the cognitive model, together with recent findings employing tasks derived from human experimental psychology. This will be followed by a discussion of experiments making use of paradigms derived from animal learning theory, and will suggest that the results are consistent with the proposed model. The ways in which the cognitive dysfunction may lead to schizophrenic symptoms will then be outlined. Finally, the possible neural bases for the disorder will be presented.

## THE DEVELOPMENT OF THE COGNITIVE MODEL

In a general sense, models of normal cognition accept that perception is dependent on an interaction between the presented stimuli and stored memories of regularities in previous input. The latter, in conjunction with the current context, result in 'expectancies' or 'response biases' (cf. Broadbent, 1971). It was originally suggested (Hemsley, 1975, 1987a) that schizophrenics fail to establish appropriate response biases and hence do not make use of temporal and spatial redundancy to reduce information processing demands. Collicutt and Hemsley (1985) proposed that such redundancy is involved in giving consciousness the distinctive 'stream-like' attributes emphasized by James (1890). Matussek's (1952) observation of a patient's "lack of continuity of his perceptions . . ." is therefore intriguing. If this formulation is correct it should be possible to construct tasks at which schizophrenics perform better than normals; this would be due to the latter forming expectancies which are inappropriate to the stimulus presented. It is this approach which characterizes some of the experiments to be described below. The aim is to overcome one of the methodological problems in this area, the existence of a generalized deficit, i.e. the poorer performance of schizophrenics on almost any task. This can make the interpretation of group differences in performance very problematic due to the confounding effects of such factors as poor motivation and task difficulty.

Clearly in considering schizophrenics' disturbances of perception and cognition, a number of theoretical models have been drawn upon. In addition to that of Broadbent (1971), those of

Schneider and Shiffrin (1977) and Posner and his colleagues (e.g. Posner, 1982) have been influential. Both suggest that awareness of redundant information is inhibited to reduce processing demands on a limited capacity system. Thus the change from controlled to automatic processing on a task as a result of prolonged practice may be seen as involving a gradual inhibition of awareness of redundant information (cf. Morris, 1992). A related position (Posner, 1982) distinguishes automatic processes and conscious attention, the former not giving rise to awareness, the latter involving awareness and closely associated with "a general inhibitory process" (p. 173). It is therefore tempting to link certain of the perceptual abnormalities prominent in schizophrenia to a weakening of the inhibitory processes crucial to conscious attention.

While it is obviously hazardous to attempt to interpret studies in a different framework from that in which each was designed, a review of influential views as to the nature of schizophrenics' cognitive impairment is suggestive of important common features (Table 1, from Hemsley, 1987a). There is an emphasis on a weakening of the influence of spatial and temporal regularities on perception, together with the suggestion of a disruption of performance by the intrusion of material normally below awareness. It was argued that although these views were based on a range of cognitive models, all acknowledged the important role of spatial and temporal regularities of past experience on the processing, and more speculatively awareness, of current sensory input. The 1987 model therefore suggested that "it is a weakening of the influence of stored memories of regularities of previous input on current perception which is basic to the schizophrenic condition" (Hemsley, 1987a p. 182).

It is illustrated in Fig. 1 the dotted line indicating the proposed dysfunction. A closely related position was put forward independently by Patterson (1987 p. 555) who suggested that there is "a failure in the automaticity with which prior experience may be recreated in parallel with current stimulus input in schizophrenia (with concomitant failures in future orientation or contextually generated expectancy)". Schizophrenia is therefore viewed as a disturbance in the moment by moment integration of stored material with current sensory input.

It is important to note that it is not claimed that the "memories of past regularities" are not stored, nor that they are inaccessible. They may indeed be accessed by consciously controlled processing. Rather the suggestion is that it is the rapid and automatic assessment of the significance or lack of significance of aspects of sensory input (and their implications for action) that is impaired. The proposed disorder would also influence the awareness of redundant information, and result in the intrusion into experience of aspects of the environment not normally perceived, as noted by Matussek (1952).

A study relevant to the proposed model has recently been reported by Jones, Hemsley and Gray (1991). It relates not only to the role of past regularities in determining schizophrenic performance, but also to the question of whether it is correct to describe the cognitive abnormalities of acute schizophrenia as a disturbance of 'selective attention'. Hemsley's (1987a) model deliberately avoided this terminology. The experiment used a choice reaction time paradigm developed by Miller (1987). *S*s were required to make one of two responses to two visually presented letters (e.g. A or B). These targets were regularly accompanied by two flanking letters (e.g. X, Y, making displays of the form XAX or YBY). Occasionally the flanking letters were interchanged, (YAY or XBX) but the correct response will still be cued by the target (A or B). Normal *S*s show a reliable slowing of reaction time on such context shift trials; clearly their performance is being influenced by the 'past regularities' within the task. If acute schizophrenics were simply to demonstrate a

Table 1. Current views on the nature of schizophrenics' cognitive impairment (from Hemsley, 1987)

1. "The basic cognitive defect . . . it is an awareness of automatic processes which are normally carried out below the level of consciousness" (Frith, 1979, p. 233).
2. "There is some suggestion that there is a failure of automatic processing in schizophrenia so that activity must proceed at the level of consciously controlled sequential processing" (Venables, 1984, p. 75).
3. Schizophrenics "concentrate on detail, at the expense of theme" (Cutting, 1985, p. 300).
4. Schizophrenics show "some deficiency in perceptual schema formation, in automaticity, or in the holistic stage of processing" (Knight, 1984, p. 120).
5. Schizophrenics show a "failure of attentional focusing to respond to stimulus redundancy" (Maher, 1983, p. 19).
6. "Schizophrenics are less able to make use of the redundancy and patterning of sensory input to reduce information processing demands" (Hemsley, 1985).
7. Schizophrenics do not maintain a strong conceptual organization or a serial processing strategy . . . nor do they organize stimuli extensively relative to others" (Magaro, 1984, p. 202).

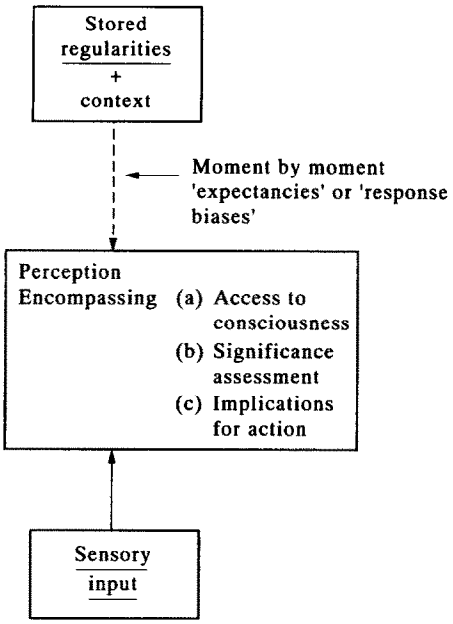


Fig. 1.

| | | Test |
|---|---|---|
| Pre-exposure (PE) group | A-A ---- A-A | A-X |
| No pre-exposure group (NPE) | ---------- | A-X |

Fig. 2. LI paradigm. In normals, pre-exposure to 'A' reduces rate of learning A–X association. This is usually interpreted as reflecting a reduction in the deployment of attention to a redundant stimulus.

broader span of attention, they should be more aware of the flankers, and hence show a greater than normal slowing of reaction time on context shift trials. However, if they are less influenced by past associations between focal stimuli and context, they should be less affected than normal *S*s by context shift. The latter was the result found by Jones *et al.* (1991) for acute but not chronic schizophrenics.

Findings from studies employing the negative priming paradigm (Tipper, 1985) can also be interpreted within the present framework. This paradigm was designed to measure the putative inhibitory processes involved in selective attention. Normal *S*s show an increase in reaction time (RT) when required to respond to a target which has previously been actively ignored as a distractor. Beech and his colleagues (e.g. Beech & Claridge, 1987) have examined the performance on such a task of normal *S*s classified according to a questionnaire measure of schizotypy. They demonstrated that high schizotypes actually showed facilitation in a negative priming paradigm, in contrast to low schizotypes who exhibited the expected delay in RT. An absence of negative priming was subsequently demonstrated in a group of schizophrenics (Beech, Powell, McWilliam & Claridge 1989). This finding has been replicated by Peters (1993), who has also demonstrated (Peters, Pickering & Hemsley, in press) that within a normal group the reduction in negative priming is particularly related to levels of 'positive symptomatology' as assessed by the Combined Schizotypal Traits Questionnaire (CSTQ, Bentall, Claridge & Slade, 1989). Once again therefore, we have evidence for a disturbance in the way current sensory input is integrated with previously presented material.

## PARADIGMS DERIVED FROM ANIMAL LEARNING THEORY

We have focused on two behavioural phenomena, Latent Inhibition (LI) (Lubow, Weiner & Feldon, 1982) and Kamin's (1969) blocking effect, both of which can be regarded as instances of the "influence of stored memories of regularities of previous input on current perception".

Lubow *et al.* (1982) have argued that the LI paradigm is an effective way of manipulating attention in animals and that it may provide a link with the attentional disturbance prominent in schizophrenia. It is illustrated in Fig. 2. In the first stage, a stimulus is repeatedly presented to the organism (PE); in the second stage, the pre-exposed stimulus is paired with reinforcement in any

of the standard learning procedures, classical or instrumental. When the amount of learning is measured, relative to a group that did not receive the first stage of stimulus pre-exposure (NPE), it is found that the stimulus-pre-exposure group learn the new association much more slowly. This is interpreted as indicating a reduction in the deployment of attention to a predictable redundant stimulus. The 'regularity' operating is that the stimulus has no consequence.

Animal studies employing the LI paradigm have demonstrated that the effect is disrupted by the administration of indirect dopamine (DA) agonists such as amphetamine (e.g. Solomon, Crider, Winkelman, Turi, Kamer & Kaplan, 1981). This affect is reversed following DA receptor blockade by neuroleptics. When administered alone, neuroleptics enhance the LI effect (Feldon & Weiner, 1991). The LI phenomenon can be demonstrated in human *S*s (e.g. Lipp, Siddle & Vaith, 1992) and Gray, Pickering, Hemsley, Dawling and Gray (1992b) have demonstrated that it is similarly affected by the administration of amphetamine. Lubow *et al.* (1982) wrote of the affect of amphetamine in animals "output is controlled, not like in the intact animal, by the integration of previous stored inputs and the prevailing situational conditions, but only by the latter" (p. 103). The normal integration of current sensory input and stored memories of regularities in previous input is absent. It is of note that LI is also disrupted by damage to the hippocampal formation (Kaye & Pearce, 1987), a point which will be returned to in the final section of this paper.

Clearly the effect of these manipulations is similar to the suggestion that schizophrenics fail to make use of the redundancy and patterning of sensory input to reduce information processing demands, and the prediction is therefore of disrupted LI in acute schizophrenia. This has been demonstrated previously (Baruch, Hemsley & Gray, 1988). The acute schizophrenics tended to perform better in the pre-exposure condition, and it was argued that these results are consistent with their being in a hyperdopaminergic state. Chronic medicated patients, less symptomatic than the acute group, performed more normally. In addition, LI for the acute group normalized following 6–7 weeks of anti-psychotic medication. It is therefore tempting to link disrupted LI to levels of positive symptomatology. All except the acute pre-exposure group showed a simple practice effect.

The performance of the acute group on the first occasion of testing was particularly interesting since the in pre-exposure condition they performed better than normal *S*s, due to their continuing to attend to the redundant stimulus. It cannot therefore be attributed to a non-specific loss of efficient cognitive functioning. These findings have recently been replicated (Gray, Hemsley &

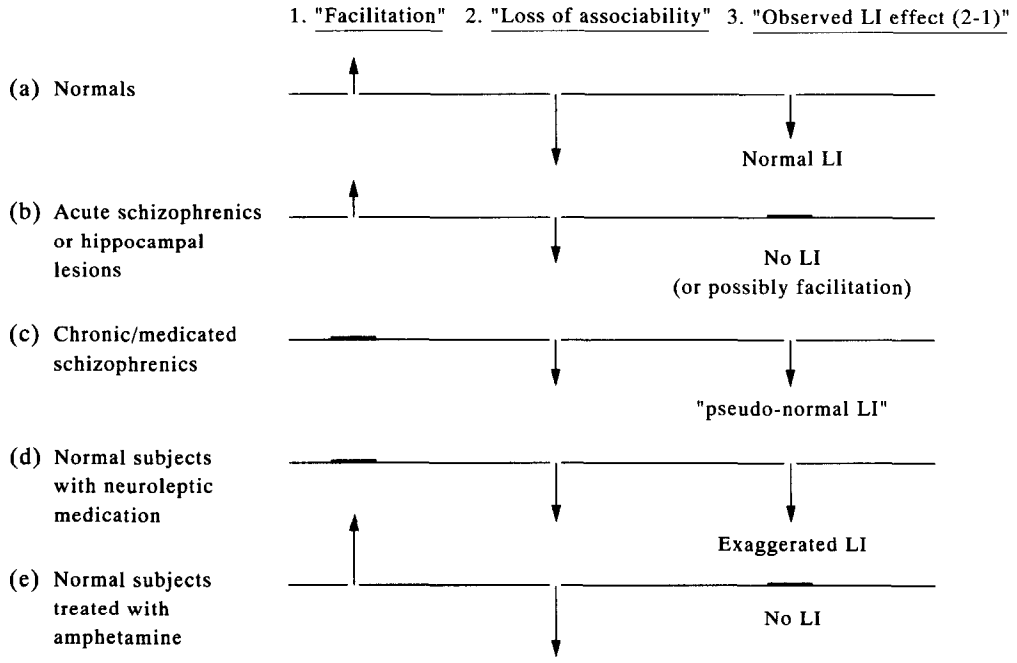


Fig. 3. The effects of a moderate number of pre-exposures (PE) to a stimulus. Horizontal line indicates no pre-exposure (NPE) condition.

| | Phase 1 | Phase 2 | Test |
|---|---|---|---|
| Blocking group | A-X | (A + B) - X | B - X |
| Control group | ------- | (A + B) - X | B - X |

Fig. 4. 'Blocking' paradigm. In normals, control group learns B–X faster than blocked group. Usually interpreted as reflecting a reduction in attention to B in phase 2 for blocked group, because it is found to predict nothing additional to that predicted by A, i.e. it is 'redundant'.

Gray, 1992a). However, the chronic patients in their study showed comparable levels of symptomatology to the acute group, but apparently normal LI.

A possible explanation for the complex relationship between LI and symptomatology may rest in the relatively crude measure of symptomatology employed [the Brief Psychiatric Rating Scale (BPRS), Overall & Gorham, 1962]. LI may relate specifically to perceptual disturbances upon which a system of delusional beliefs may then be based (cf. Maher, 1988). The latter may persist even after the successful treatment of perceptual abnormalities and may result in an elevated BPRS score. A more detailed assessment of current and past symptomatology would address this issue. However, a further possibility exists, and requires a more detailed consideration of the effects of pre-exposure to a stimulus.

Lubow (1989) has proposed a two factor theory for the effects of PE. He contrasts an initial property extraction phase with subsequent stimulus relationship processing, and his analysis therefore suggests a facilitation effect with small numbers of pre-exposures. The typically obtained decrement in performance is "traceable to negative transfer from $S_1$—no consequences association, and is superimposed on what otherwise would be a facilitatory outcome" (p. 216). However he notes that studies varying the number of stimulus pre-exposures at the low end of the continuum have not been carried out. Lubow's suggestion is illustrated in Fig. 3(a); the observed effect of a moderate number of pre-exposures is slower learning relative to the NPE condition (horizontal line). For acute schizophrenics (and animals with hippocampal lesions) a weaker 'loss of associability' is postulated—Hemsley's 'reduced influence of regularities of previous input'. Depending on the magnitude of this effect, reduced LI and even the possibility of an overall facilitation effect is suggested [Fig. 3(b)]. In the Baruch *et al.* (1988) study there was a suggestion of facilitated learning in acute schizophrenics due to pre-exposure.

One might then speculate that the apparent 'normalization' of LI following medication could reflect not genuinely normal functioning but the elimination of the facilitation effect, leaving the somewhat disrupted 'stimulus-relationship processing' predominant [Fig. 3(c)]. The effects of neuroleptic medication (enhanced LI) and amphetamine (reduction in LI) in normal *S*s are illustrated in Fig. 3(d and e), respectively. It appears probable that the discriminability of the pre-exposed stimulus will affect the relative weight of 'facilitation' and 'loss of associability' effects at a given number of pre-exposures (cf. Chamizo & Mackintosh, 1989). The model put forward in Fig. 3 may be tested by examining the effects of a small number of stimulus pre-exposures in both normals and schizophrenics, and their response to pharmacological manipulations.

It is important to note that LI does not simply represent a form of habituation. There is a major difference between the two: LI is disrupted by a change of context, whereas habituation is not. This may prove important for our understanding of schizophrenia, where a key disturbance may be the failure to relate specific associations to the context in which they occur. For LI, the association in question is one of stimulus—no consequence; a failure to link this association to its context should have the same consequences as a context shift, i.e. disruption of LI, and this may account for the performance of acute schizophrenics. Once again, Matussek's (1952) observations are relevant. He wrote, "When the perceptual context is disturbed, individual objects acquire different properties from those which they have when the normal context prevails," (p. 94) and suggests that the extent to which context is loosened crucially determines the severity of the disorder.

In a typical habituation experiment the dependent variable is measured during the 'stimulus pre-exposure' phase. In contrast, LI required at least a two phase procedure. Indeed as Lubow (1989) emphasized, "a major methodological concern is to keep the two measures—unconditioned response level and conditioned response level—independent of each other" (p. 147). However, the

issue of the relationship between LI and habituation is further complicated by Lubow's important distinction between habituation of unconditioned specific responses to the conditioned stimulus (CS), and habituation of the orienting response (OR). It is the latter which has been most frequently investigated in schizophrenics (e.g. Ohman, Nordby & D'Elia, 1986). There is some evidence of a relationship between OR habituation and LI (Kaye & Pearce, 1987), although a context change insufficient to dishabituate OR nevertheless interferes with LI (Hall & Channel, 1985).

A second paradigm, Kamin's (1969), blocking affect (KB) shares many of the same features as LI and is illustrated in Fig. 4.

It again involves a pre-exposure phase in which the experimental group learns an association between two stimuli (A–X); control *S*s learn either no association or a different one at this stage. Both groups are then presented with pairings between a compound stimulus (A–B) and X and are then tested for what they have learned about the B–X relationship. The pre-exposed group demonstrates less learning than controls; this is the blocking effect and it is generally agreed that it arises as a result of a process in which attention to B is reduced because it is found to predict nothing in addition to what is predicted by A (Pearce & Hall, 1980). It is viewed as 'redundant'. Like LI, the blocking effect in animals is abolished by amphetamine (Crider, Solomon & McMahon, 1982), and by damage to the hippocampus (Solomon, 1977). It was predicted that the blocking effect would be reduced in acute schizophrenics and this was confirmed (Jones, Gray & Hemsley, 1992). Acute schizophrenics actually performed worse in the control condition, whereas normals showed the usual blocking effect.

A further paradigm derived from animal learning theory may relate both to Hemsley's (1987a) model and also to the disturbance of Gestalt perception in schizophrenia discussed earlier. In negative patterning, reinforced component presentations ($CS_1$ or $CS_2$) are intermixed with non-reinforced compound ($CS_1$–$CS_2$) presentations. Negative patterning is attained if the response to the compound is smaller than the sum of the responses to the components. In positive patterning, reinforced compound ($CS_2$) presentation is intermixed with non-reinforced components ($CS_1$ or $CS_2$) presentation. Positive patterning is attained if the response to the compound is larger than the sum of the responses to the components. It has been suggested, (cf. Schmajuk & Di Carlo, 1992) that when both CSs are presented together they generate a 'patterned' or 'configural' stimulus.

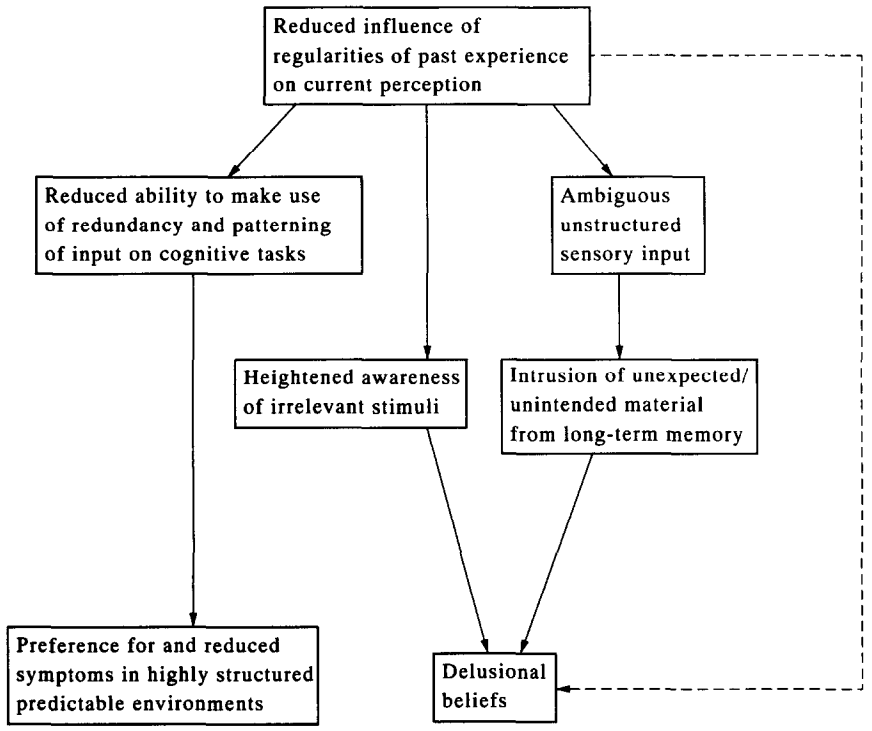


Fig. 5. Model of cognitive abnormalities and symptoms of schizophrenia (modified from Hemsley, 1987).

Rudy and Sutherland (1989) have demonstrated that hippocampal lesions preceding training prevent the acquisition of negative patterning and Schmajuk and Di Carlo (1992) present a detailed neural network model of hippocampal function in which its role in configural learning is emphasized. Given the evidence for abnormalities in the hippocampus and related brain areas in schizophrenia, (see below), it would clearly be worthwhile to investigate 'patterning' in this group.

## COGNITIVE DISTURBANCE AND SCHIZOPHRENIC SYMPTOMS

It is useful to distinguish two areas of theorizing and related experimentation. The first (e.g. Frith, 1979) seeks to account for the principal positive symptoms of schizophrenia in terms of the cognitive impairment. The second suggests that certain aspects of schizophrenics' functioning may reflect the action of control mechanisms which "involve conscious and unconscious psychological processes that focus on regulating the amount of demand faced to fit the adaptive capacity available" (Strauss, 1987, p. 85).

As indicated in the Introduction, the link between the proposed disturbance of information processing and psychotic symptoms diagnostic of schizophrenia may be more easily understood if we consider the well documented abnormalities of experience occurring early in the disorder. Huber and his colleagues (1986) have described how self-experienced disturbances of thought, perception, action and bodily sensations may be transformed into schizophrenic symptoms. A longitudinal study by Klostertotter (1992) has provided evidence for this progression. In a similar vein, Maher (1988) has argued for the crucial role of abnormal perceptual experiences, not only hallucinations, in the formation of delusions. This emphasis is also prominent in Matussek's (1952) observations. Following a loosening of the perceptual context, attention may be captured by incidental details of the environment. Normally such an aspect of the situation would not reach awareness, but its registration prompts a search for reasons for its occurrence. Frith's (1979) model adopts a similar position. As Fleminger (1992) has emphasized, abnormal beliefs, once formed, generate expectancies which may result in further misperceptions. Fleminger suggests that such misperception will be less likely when there are "strong links between sense data and memory" (p. 293), The present model (Fig. 1) proposes the disruption of such links.

Among the most prominent features of delusional thinking is an abnormal view of the relationships between events. As Schneider (1930) put it "meaningful connections are created between temporarily coincident external impressions, external impression with the patient's present condition, or perceptions with thoughts which happen to be present, or events and recollections happening to occur in consciousness about the same time". Similarly Arieti (1974) observed "patients see non-fortuitous coincidences everywhere" (p. 231). Matussek (1952) quotes a patient as saying "out of these perceptions came the absolute awareness that my ability to see connections had been multiplied many times over" (p. 96). For example, objects sharing certain qualities which had become prominent were seen as being linked in some significant way. Such feelings of relatedness, based on temporal or spatial contiguity between experiences, may proceed to an assumption of a causal relationship between them.

Such clinical observations may be linked to the present model. In reviewing the processes that underlie the judgement of causation, Einhorn and Hogarth (1986, p. 5) note that normal people engage in causal reasoning in order to make sense of the world, and that this is more likely to happen when perceptions violate expectations and become prominent. Spatial and temporal contiguity are clearly of great importance in concluding that there exists a causal relationship. However, in assessing the strength of such a relationship, account is also taken of instances of the occurrence of X in the absence of Y, and Y in the absence of X (i.e. past regularities). Consider now the case of the schizophrenic patient. Not only does the "weakened influence of past regularities of current perception" result in the intrusion of redundant material into awareness; it also influences the assessment of the covariation between X and Y. Hence abnormal causal relationships may be inferred on the basis of a single co-occurrence.

However, in emphasizing failures to make use of 'background knowledge' (cf. Anscombe, 1987), and alterations in the judgement of covariation (cf. Hemsley, 1990), there has clearly been a move away from Maher's (1988) view of delusions as essentially a product of normal reasoning, serving as explanations which the deluded individual uses to account for abnormal perceptual experiences.

Recent research suggests an abnormal reasoning style in some deluded *S*s in addition to well established perceptual disturbances.

A problem which is encountered in exploring the reasoning style of deluded *S*s is in setting a standard for 'correct' or 'incorrect' reasoning. Fishcoff and Beyth-Marom (1983) suggest that Bayesian inference provides a general framework for evaluating beliefs in the normal population and that it may be used to describe a person's consistency with, or departure from the model. Hemsley and Garety (1986) extended this approach to the inferences of deluded *S*s. Employing a probability inference task, two studies have demonstrated that deluded *S*s require less information before reaching a decision (Huq, Garety & Hemsley, 1988; Garety, Hemsley & Wessely, 1991). The second of these also indicated that after certainty is reached, deluded *S*s are more likely to make a dramatic change in probability estimate following an item of disconfirmatory information. This is consistent with those models of schizophrenia which emphasize greater influence of immediate environmental stimuli compared with the effects of prior learning (Salzinger, 1984; Hemsley, 1987a).

It is possible that the extensive literature on sensory/perceptual deprivation in normal *S*s is relevant to schizophrenic hallucinations. Under conditions of unstructured sensory input, no 'predictions' are possible, and it is clear that this may result in abnormal perceptual experiences which Leff (1968) suggested "overlap considerably with those of mentally ill patients" (p. 1507). It has also been possible to demonstrate the short term manipulation of auditory hallucinations in a group of schizophrenic patients by means of alterations in auditory input (Margo, Hemsley & Slade, 1981). The experiences reported were inversely related to the structure and attention commanding properties of the input.

The present model proposes that the schizophrenic condition is characterized by a reduction in the influence of the regularities of past experience on current perception. This, it is suggested, results in ambiguous, unstructured sensory input. One might therefore argue that hallucinations are related to a cognitive impairment which even under normal conditions results in ambiguous messages reaching awareness and hence fails to inhibit the emergence of material from long term memory (LTM) (Hemsley, 1987b). George and Neufeld (1985) have referred to an interaction between the "spontaneous retrieval of information stored in LTM and sensory processing, the latter having an inhibitory effect on the former" (p. 268). A similar argument is put forward by Rund (1986); "Schizophrenics, possibly because of a sensory overload . . . are more susceptible to such a direct flow between long term storage and the sensory storage level" (p. 532).

The preceding discussion has been concerned primarily with positive symptomatology. However, it is rare for schizophrenics to show only positive or negative symptoms. It remains unclear whether the distinction represents: (a) two underlying and distinct disorders; (b) differing severity of the same disorder; (c) individual differences in reaction to the same disorder; (d) different stages of the same disorder (acute–chronic), or a combination of (b), (c) and (d). Thus Pogue-Geile and Harrow (1988) conclude that "the evidence is supportive of the view that negative symptoms may represent a severity threshold on a continuum of liability to schizophrenia" (p. 437). Within the present formulation, a more drastic weakening of the influence of stored regularities on current perception might result in a level of disorganization such as to render difficult any goal directed activities.

Relevant to (c) is Strauss' (1987) argument that certain aspects of schizophrenics' functioning may reflect the action of conscious and unconscious regulatory processes. Such control processes were seen by Hemsley (1977) as crucial to negative symptomatology. Schizophrenics were viewed as being in a state of 'information overload'; symptoms such as poverty of speech, social withdrawal and retardation, represented adaptive strategies, learnt over time so as to minimize the effects of the cognitive impairment. One may also speculate that the search for meaning in the altered experiences may diminish over time, as actions based on these prove ineffective or counter-productive. As Anscombe (1987, p. 254) puts it, "less and less the subject forms his own impressions, and more and more he is impinged upon by his environment".

Frith (1992) has criticized this view of negative symptoms on the grounds that they may be apparent before the appearance of positive symptoms. However it is far from clear that they precede the more subtle disturbances of thinking and perception emphasized by such authors as Huber (1986). Nor is the fact that negative symptoms are associated with poor outcome and more severe cognitive impairment inconsistent with the suggestion that they represent a form of coping

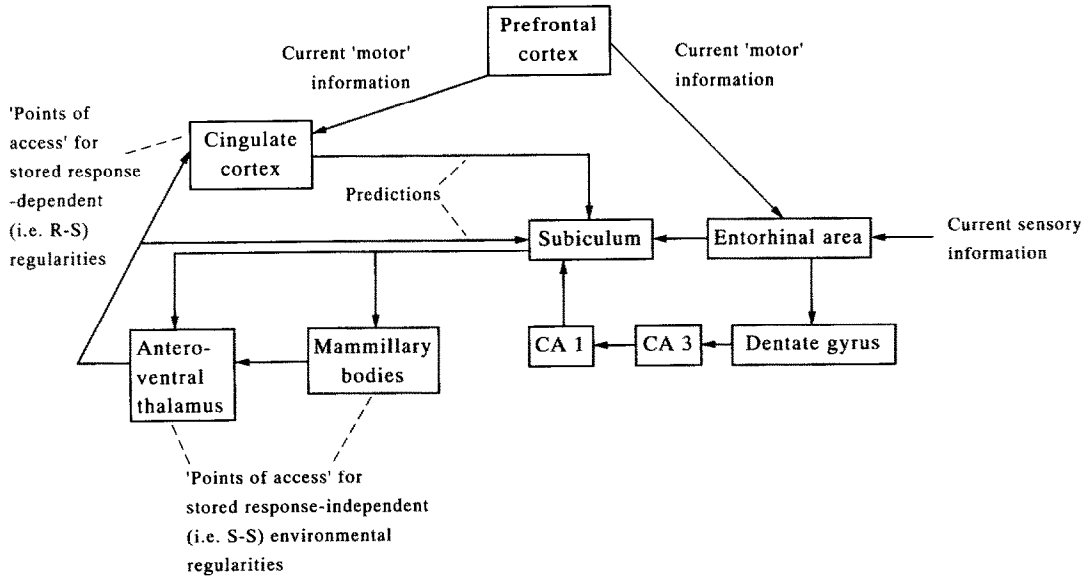


Fig. 6. Current sensory input integrated with stored material in this circuit. A "weakening in the influence of stored memories of regularities of previous input on current perception" (Hemsley, 1987) could result from structural abnormalities in any of the regions contributing to the generation of 'predictions' or 'moment by moment expectancies'.

strategy to minimize distress. As Hemsley (1977) wrote "The 'omission' and 'escape' adaptations (corresponding to under-responsiveness and social withdrawal) may be the only viable methods at high levels of overload" (p. 171).

The proposed model for psychotic symptoms is summarized in Fig. 5. The dotted line represents the modification of the 1987 model necessitated by the recent experimental studies of deluded patients.

## THE NEURAL BASES OF SCHIZOPHRENIA

In this section will be considered the possible bases of the proposed disturbance of information processing. As Weiner (1990) notes "there is a growing interest in the study of the neural mechanisms of learned behaviours" (p. 442). Several theoretical reviews (e.g. Huber, 1986; Schmajuk, 1987) link pathology of the limbic system with schizophrenic symptoms and neuropathological studies have provided some support for this position. Thus the recent review by Bogerts and Falkai (1991) suggests "neuroanatomical anomalies in the hippocampus, amygdala, entorhinal cortex, parahippocampal and cingulate gyri in a substantial proportion of schizophrenics" (p. 94). MRI studies are also indicative of "anomalies of limbic structures in the medial temporal lobe" (Degreef, Ashtari, Bogerts, Bilder, Jody, Alvir & Lieberman, 1992, p. 531).

The present model proposes that abnormalities of the hippocampus and related brain structures play a crucial role in the emergence of psychotic symptoms. The suggested functions of the hippocampus appear particularly relevant to the present cognitive model. Thus Olton, Wible and Shapiro (1986) have proposed that "The hippocampus may be the brain structure that allows each of the various components of a place and an event to be linked together and compared with other places and events". (p. 354) In a related formulation Squire (1992) argues that the hippocampus "contributes to the forming of new relationships, such as those established when associating stimuli with their spatial and temporal context" (p. 208). In more explicitly attentional terms Schmajuk and Moore (1985) suggest that the hippocampus controls modifications in stimulus associability, thus accounting for the disruption of latent inhibition resulting from hippocampal lesions. It is of interest that in recent neural network models of 'cognitive mapping', involving the hippocampus, there is postulated a mechanism which "prevents the formation of spurious associations." (Schmajuk & Thieme, 1992, p. 3).

The present model relies heavily on Gray's (1982) proposal that the hippocampus plays a crucial role in the comparison of actual and expected stimuli. Gray's model suggests that there exists a circuit responsible for the moment by moment generation of predictions of subsequent sensory input. It is just such a system which is claimed as defective within the cognitive model outlined above. This system (Gray, 1982; Gray, Feldon, Rawlins, Hemsley & Smith, 1991a, p. 11) brings together (i) information concerning the current state of the perceptual world; (ii) information concerning the current motor program; (iii) stored information concerning past regularities relating stimulus events to other stimulus events (S–S regularities); (iv) stored information describing regularities relating past responses to subsequent stimulus events (R–S regularities). From this information is predicted the next expected state of the perceptual world, and this is compared to the actual next state. If a 'mismatch' occurs, the ongoing motor program is interrupted, and attention is allocated to the new stimulus.

The brain systems considered to be involved in these processes are illustrated in Fig. 6 (modified from Gray, 1982, p. 268). The comparator function is attributed to the subicular area, and this is considered to receive elaborated descriptions of the perceptual world from the entorhinal cortex. Gray (1882, p. 271) suggests that "the direct input to the subicular area from the entorhinal cortex describes the current state of the world, the input via the hippocampus determines whether this description is treated as important". The subiculum receives predictions from, and initiates generation of the next prediction in the Papez circuit. Information concerning current and intended motor programmes is provided by the prefrontal cortex, through its projections to the entorhinal and cingulate cortices. The subicular loop requires access to stored regularities, and Gray (1982, p. 269) speculates that the anteroventral thalamus may be a point of access for S–S environmental regularities, and the cingulate cortex for R–S regularities. Storage of this material may be within the temporal lobes.

A failure to generate appropriate predictions could therefore result from a disturbance at one or more points in this crucial circuit. The final common pathway to psychosis, in psychological terms, is therefore the failure to relate current sensory input to stored regularities. The review of brain pathology in schizophrenia by Bogerts and Falkai (1991) is clearly indicative of abnormalities in the structures involved in the proposed circuit. Not indicated in Fig. 6 is the direct projection from temporal cortex to subiculum (Van Hoesen, Rosene & Mesulam, 1979). The cognitive model proposed that unstructured sensory input permits the unintended intrusion of material from LTM, and that this corresponds to hallucinations. Conceivably normal input to the subiculum is required to inhibit the direct flow of information from the temporal lobe.

The projection from the subiculum to nucleus accumbens makes it possible to link the proposed structural abnormalities to the evidence implicating dopaminergic hyperactivity in schizophrenia. Gray *et al.* (1991a) and Gray, Hemsley, Feldon, Gray and Rawlins (1991b) have argued that there is a disruption of the normal interaction between input from the hippocampus (via subiculum) to the nucleus accumbens and the mesolimbic dopaminergic system. This links with the known effects of amphetamine on such phenomena as latent inhibition. Schmajuk and Di Carlo (1991) argue that input to accumbens is directly proportional to the build up of expectancies. Faulty expectations generated within the above circuit could result in repeated 'mismatch' signals, and the inappropriate allocation of attention (cf. Hemsley *et al.*, 1993). It is apparent either that biological and psychological models of the disorder are converging in intriguing ways, or that the present author, in common with Arieti's patients, is seeing "nonfortuitous coincidences everywhere".

## REFERENCES

Anscombe, R. (1987). The disorder of consciousness in schizophrenia. *Schizophrenia Bulletin, 11*, 241–260.

Arieti, S. (1966). Schizophrenic cognition. In Hoch, P. H. & Zubin, J. (Eds), *Psychopathology of schizophrenia* (pp. 37–48). New York: Grune & Stratton.

Arieti, S. (1974). *Interpretation of schizophrenia* (2nd Edn). London: Crosby, Lockwood, Staples.

Baruch, J., Hemsley, D. R. & Gray, J. A. (1988). Differential performance of acute and chronic schizophrenics in a latent inhibition task. *Journal of Nervous and Mental Disease, 176*, 598–606.

Beech, A. R. & Claridge, G. S. (1987). Individual differences in negative priming: Relations with schizotypal personality traits. *British Journal of Psychology, 78*, 349–356.

Beech, A. R., Powell, J. H., McWilliam, J. & Claridge, G. S. (1989). Evidence of reduced 'cognitive inhibition' in schizophrenia. *British Journal of Clinical Psychology, 28*, 109–116.

Bentall, R. P., Claridge, G. S. & Slade, P. D. (1989). The multidimensional nature of schizotypal traits: a factor analytic study with normal subjects. *British Journal of Clinical Psychology, 28*, 363–375.
Berze, J. (1914). *Primary insufficiency of mental activity*. Leipzig: F. Deuticke.
Bogerts, B. & Falkai, P. (1991). Clinical and neurodevelopmental aspects of brain pathology in schizophrenia. In Mednick, S. A., Cannon, T. D., Barr, C. E. & LaFosse, J. M. (Eds), *Developmental neuropathology of schizophrenia* (pp. 92–120). New York: Plenum.
Broadbent, D. E. (1971). *Decision and stress*. London: Academic Press.
Chamizo, V. D. & Mackintosh, N. J. (1989). Latent learning and latent inhibition in maze discriminations. *Quarterly Journal of Experimental Psychology, 41B*, 21–31.
Collicutt, J. R. & Hemsley D. R. (1985). Schizophrenia: a disruption of the stream of thought. Unpublished manuscript.
Conrad, K. (1958). *Die Beginnende Schizophrenie*. Stuttgart: G. Thieme.
Crider, A., Solomon, P. R. & McMahon, M. A. (1982). Attention in the rat following chronic d-amphetamine administration: relationship to schizophrenic attention disorder. *Biological Psychiatry, 17*, 351–361.
Cutting J. (1989). Gestalt theory and psychiatry? Discussion paper. *Journal of the Royal Society of Medicine, 82*, 429–431.
Degreef, G., Ashtari, M., Bogerts, B., Bilder, R. M., Jody, D. N., Alvir, J. M. J. & Lieberman, J. A. (1992). Volumes of ventricular system subdivisions measured from Magnetic Resonance images in first episode schizophrenic patients. *Archives of General Psychiatry, 49*, 531–537.
Einhorn, H. J. & Hogarth, R. M. (1986). Judging probable cause. *Psychological Bulletin, 99*, 3–19.
Ellenbroek, B. A. & Cools, A. R. (1990). Animal models with construct validity for schizophrenia. *Behavioural Pharmacology, 1*, 469–490.
Feldon, J. & Weiner, I. (1991). The latent inhibition model of schizophrenic attention disorder. *Biological Psychiatry, 29*, 635–646.
Fischoff, B. & Beyth-Maron, R. (1983). Hypothesis evaluation from Bayesian perspective. *Psychological Review, 90*, 239–260.
Fleminger, S. (1992). Seeing is believing: the role of 'preconscious' perceptual processing in delusional misidentification. *British Journal of Psychiatry, 160*, 293–303.
Frith, C. D. (1979). Consciousness, information processing and schizophrenia. *British Journal of Psychiatry, 134*, 225–235.
Frith, C. D. (1992). *The cognitive neuropsychology of schizophrenia*. Hove: Lawrence Erlbaum.
Garety, P. A., Hemsley, D. R. & Wessely, S. (1991). Reasoning in deluded schizophrenic and paranoid patients. *Journal of Nervous and Mental Disease, 179*, 194–201.
George, L. & Neufeld, R. W. J. (1985). Cognition and symptomatology in schizophrenia. *Schizophrenia Bulletin, 11*, 264–285.
Gray, J. A. (1982). *The neuropsychology of anxiety: An enquiry into the function of the septo-hippocampal system*. Oxford: Oxford University Press.
Gray, J. A., Feldon, J., Rawlins, J. P. N., Hemsley, D. R. & Smith, A. D. (1991a). The neuropsychology of schizophrenia *Behavioural and Brain Sciences, 14*, 1–20.
Gray, J. A., Hemsley, D. R., Feldon, J., Gray, N. S. & Rawlins, J. N. P. (1991b). Schiz bits: Misses mysteries and hits. *Behavioural and Brain Sciences, 14*, 56–84.
Gray, N. S., Hemsley, D. R. & Gray, J. A. (1992a). Abolition of latent inhibition in acute but not chronic schizophrenics. *Neurology, Psychiatry and Brain Research, 1*, 83–89.
Gray, N. S., Pickering, A. D., Hemsley, D. R., Dawling, S. & Gray, J. A. (1992b). Abolition of latent inhibition by a single 5 mg dose of d-amphetamine in man. *Psychopharmacology, 107*, 425–430.
Hall, G. & Channel, S. (1985). Differential effects of contextual change in latent inhibition and in the habituation of an orienting response. *Journal of Experimental Psychology: Animal Behaviour Processes, 11*, 470–481.
Hemsley, D. R. (1975). A two stage model of attention in schizophrenia research. *British Journal of Social and Clinical Psychology, 14*, 81–88.
Hemsley, D. R. (1977). What have cognitive deficits to do with schizophrenic symptoms? *British Journal of Psychiatry, 130*, 167–173.
Hemsley, D. R. (1987a). An experimental psychological model for schizophrenia. In Hafner, H., Gattaz, W. F. & Janzarik, W. (Eds), *Search for the causes of schizophrenia*. Heidelberg: Springer.
Hemsley, D. R. (1987b). Hallucinations: unintended or unexpected? *Behavioural and Brain Sciences, 10*, 532–533.
Hemsley, D. R. (1988). Psychological models of schizophrenia. In Miller, E. & Cooper, P. (Eds), *Adult abnormal psychology*, (pp. 101–127). London: Churchill Livingstone.
Hemsley, D. R. (1990). What have cognitive deficits to do with schizophrenia? In Huber, G. (Ed.), *Idiopathische psychosen* (pp. 111–127). New York: Schattauer-Verlag.
Hemsley, D. R. & Garety, P. A. (1986). The formation and maintenance of delusions: A Bayesian analysis. *British Journal of Psychiatry, 149*, 51–56.
Hemsley, D. R., Rawlins, J. N. P., Feldon, J., Jones, S. H. & Gray, J. A. (1993). The neuropsychology of schizophrenia: Act 3. *Behavioural and Brain Sciences, 16*, 209–215.
Huber, G. (1986). Psychiatrische Aspekte des Basisstorungs Konzepts. In Sullwold, L. & Huber, G. (Eds), *Schizophrene Basisstorungen*. Berlin: Springer.
Huq, S. F., Garety, P. A. & Hemsley, D. R. (1988). Probabilistic judgements in deluded and non-deluded subjects. *Quarterly Journal of Experimental Psychology, 40A*, 801–812.
James, W. (1890). *The principles of psychology*. London: MacMillan.
Jones, S. H., Hemsley, D. R. & Gray, J. A. (1991). Contextual effects on choice reaction time and accuracy in acute and chronic schizophrenia: impairment in selective attention or in the influence of prior learning? *British Journal of Psychiatry, 159*, 415–421.
Jones, S. H., Gray, J. A. & Hemsley, D. R. (1992). Loss of the Kamin Blocking effect in acute but not chronic schizophrenics. *Biological Psychiatry, 32*, 739–755.
Kamin, L. J. (1969). Predictability, surprise, attention and conditioning. In Campbell, B. A. & Church, R. M. (Eds), *Punishment and aversive behaviour* (pp. 279–296). New York: Appleton Century Crofts.
Kaye, H. & Pearce, J. M. (1987). Hippocampal lesions attenuate latent inhibition and the decline of the orienting response in rats. *Quarterly Journal of Experimental Psychology, 39B*, 107–125.

Klostertotter, J. (1992). The meaning of basic symptoms for the development of schizophrenic psychoses. *Neurology, Psychiatry and Brain Research, 1*, 30–41.
Leff, J. P. (1986). Perceptual phenomena and personality in sensory deprivation. *British Journal of Psychiatry, 114*, 1499–1508.
Lipp, O. V., Siddle, D. A. T. & Vaith, D. (1992). Latent inhibition in humans: single cue conditioning revisited. *Journal of Experimental Psychology: Animal Behaviour Processes, 18*, 115–125.
Lubow, R. E. (1989). *Latent inhibition and conditioned attention theory*. New York: Cambridge University Press.
Lubow, R. E., Weiner, I. & Feldon, J. (1982). An animal model of attention. In Speigelstein, M. Y. & Levy, A. (Eds), *Behavioural models and the analysis of drug action* (pp. 89–107). Amsterdam: Elsevier.
Maher, B. A. (1988). Anomalous experience and delusional thinking: the logic of explanations. In Oltmann, T. F. & Maher, B. A. (Eds), *Delusional beliefs*. New York: Wiley.
Margo, A., Hemsley, D. R. & Slade, P. D. (1981). The effects of varying auditory input on schizophrenic hallucinations. *British Journal of Psychiatry, 139*, 122–127.
Matussek, P. (1952). Studies in delusional perception. *Psychiatric and Zeitschrift Neurologie, 189*, 279–318.
Miller, J. (1987). Priming is not necessary for selective attention failures: semantic effects of unattended, unprimed letters. *Perception and psychophysics, 41*, 419–434.
Morris. P. E. (1992). Cognition and consciousness. *The Psychologist, 5*, 3–8.
Ohman, A., Nordby, H. & D'Elia, G. (1986). Orienting and schizophrenia: stimulus significance, attention and distraction in a signalled reaction time task. *Journal of Abnormal Psychology, 95*, 316–334.
Olton, D. S., Wible, C. G. & Shapiro, M. L. (1986). Mnemonic theories of hippocampal function. *Behavioural Neuroscience, 100*, 852–855.
Overall, J. E. & Gorham, D. R. (1962). The brief psychiatric rating scale. *Psychological Reports, 10*, 799–812.
Patterson, T. (1987). Studies towards the subcortical pathogenesis of schizophrenia. *Schizophrenia Bulletin, 13*, 555–576.
Pearce, J. M. & Hall, G. (1980). A model for Pavlovian learning: variations in the effectiveness of conditioned but not of unconditioned stimuli. *Psychological Review, 87*, 532–552.
Peters, E. (1993). Cognitive processes involved in the formation of positive symptomatology in schizotypal, amphetamine treated, and psychotic populations. Unpublished Ph.D. thesis, Institute of Psychiatry, University of London.
Peters, E., Pickering, A. D. & Hemsley, D. R. (in press). Cognitive inhibition and positive symptomatology in schizotypy. *British Journal of Clinical Psychology*.
Pogue-Geile, M. F. & Harrow, M. (1988). Negative symptoms in schizophrenia: their longitudinal course and prognostic importance. *Schizophrenia Bulletin, 11*, 427–439.
Posner, M. (1982). Cumulative development of attentional theory. *American Psychologist, 37*, 168–179.
Rudy, J. W. & Sutherland, R. J. (1989). The hippocampal formation is necessary for rats to learn and remember configural discriminations. *Behavioural Brain Research, 34*, 97–109.
Rund, B. R. (1986). Verbal hallucinations and information processing. *Behavioural and Brain Sciences, 9*, 530–531.
Salzinger, K. (1984). The immediacy hypothesis in a theory of schizophrenia. In Spaulding, W. D. & Cole, J. K. (Eds), *Theories of schizophrenia and psychosis* (pp. 231–282). University of Lincoln and London: Nebraska Press.
Schmajuk, N. A. (1987). Animal models of schizophrenia: the hippocampally lesioned animal. *Schizophrenia Bulletin, 12*, 317–327.
Schmajuk, N. A. & Di Carlo J. J. (1991). A hippocampal theory of schizophrenia. *Behavioural and Brain Sciences, 14*, 47–49.
Schmajuk, N. A. & Di Carlo, J. J. (1992). Stimulus configuration, classical conditioning and hippocampal function. *Psychological Review, 99*, 268–305.
Schmajuk, N. A. & Moore, J. W. (1985). Real time attentional models for classical conditioning and the hippocampus. *Physiological Psychology, 13*, 278–290.
Schmajuk, N. A. & Thieme, A. D. (in press). Purposive behaviour and cognitive mapping: a neural network model. *Biological Cybernetics*.
Schneider, C. (1930). *Die Psychologie der Schizophrenen* Leipzig. Thieme.
Schneider, W. & Shiffrin, R. M. (1977). Controlled and automatic human information processing—I. Detection search and attention. *Psychological Review, 84*, 1–66.
Shakow, D. (1950). Some psychological features of schizophrenia. In Reyment, M. L. (Ed.), *Feelings and emotions* (pp. 383–390). McGraw Hill: New York.
Shallice, T. (1988). Information processing models of consciousness: possibilities and problems. In Marcel, A. J. & Bisiach, E. (Eds), *Consciousness in contemporary science*. Oxford: Clarendon Press.
Shepherd, M. (1987). Formulation of new research strategies on schizophrenia. In Hafner, H., Gattaz, W. F. & Janzarik, W. (Eds), *Search for the causes of schizophrenia* (pp. 29–38). Heidelberg: Springer.
Solomon, P. R. (1977). Role of the hippocampus in blocking and conditioned inhibition of rabbits nictating membrane response. *Journal of Comparative and Physiological Psychology, 91*, 407–417.
Solomon, P. R. Crider, A., Winkelman, J. W., Turi, A., Kamer, R. M. & Kaplan, L. J. (1981). Disrupted latent inhibition in the rat with chronic amphetamine or haloperidol induced supersensitivity: Relationship to schizophrenic attention disorder. *Biological Psychiatry, 16*, 519–537.
Squire, L. (1992). Memory and the hippocampus: a synthesis from findings with rats, monkeys and humans. *Psychological Review, 99*, 195–231.
Strauss, J. (1987). Processes of healing and chronicity in schizophrenia. In Hafner, H., Gattaz, W. F. & Janzarik, W. (Eds), *Search for the causes of schizophrenia* (pp. 75–87). Heidelberg: Springer Verlag.
Tipper, S. P. (1985). The negative priming effect: inhibitory priming by ignored objects. *Quarterly Journal of Experimental Psychology, 37A*, 571–590.
Van Hoesen, G. W., Rosene, D. L. & Mesulam, M. (1979). Subicular input from temporal cortex in the Rhesus Monkey. *Science, 205*, 608–610.
Weiner, I. (1990). Neural substrates of latent inhibition: the switching model. *Psychological Bulletin, 168*, 442–461.