STELIOS GEORGIADIES

INVESTIGATION OF COGNITIVE DEFICITS IN SCHIZOPHRENIA THROUGH PERFORMANCE ON A REVERSAL LEARNING TASK

Submitted in fulfilment of the degree
Doctorate of Psychology in Clinical Psychology

University of Surrey
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Dedicated to my brother Petros Georgiades whose memory will accompany me everywhere in this lonely passage of life.
AKNOWLEDGEMENTS

I would like to thank Dr Marie Clark, the supervisor of the present portfolio for all her useful comments and corrections as well as for all her patience to respond to my requests for last minute supervision.

Thanks are also due to Professor David Winter for supporting my application for financial assistance from Barnet NHS Trust and to all psychiatrists in St Anns and Napsbury Hospitals for assisting me with the selection and the ratings of patients. To all the patients who agreed to participate in the studies and to the nursing staff of the wards in the above hospitals who informed patients about the study and ensured that they attended their appointments with me.

Last but by no means least I would like to thank my wife Vasso and my two sons Markos and Christos for their incredible support and tolerance while I was writing this thesis.
SUMMARY

The present portfolio includes three dossiers: academic, clinical/professional and research.

The academic dossier consists of two literature reviews. The first, titled “the efficacy of cognitive behavioural therapy in the reduction of positive symptomatology in schizophrenia” reviews the results of recent studies on the application of Cognitive Behavioural Therapy in schizophrenia and evaluates the extent of which the approach contributes to the treatment of the disorder. The second review, titled “the value of schizotypy in enhancing our understanding of schizophrenia” looks at evidence from different areas of current schizotypy research in order to evaluate the advantages of the concept of schizotypy as an alternative method for investigating schizophrenia.

The study included in the clinical/professional dossier investigates the efficacy of cognitive behavioural therapy in the treatment of Post-Traumatic Stress Disorder (PTSD) as derived from the clinic in the Adult Section of the Psychology Department at Barnet NHS Trust. The results of the study support the hypotheses that Cognitive Behavioural Therapy contributes significantly to the reduction of PTSD symptomatology especially in people who sought help soon after their traumatic experience.

The research dossier includes the findings of a research study in which it investigates cognitive deficits in schizophrenia, as described by Hemsley and colleagues (1987), using the behavioural phenomenon of reversal learning. The performance of normal controls (n=40), chronic schizophrenics (n=34) and acute schizophrenics (n=31) on this task appeared to support the hypothesis that acute schizophrenics less influenced by previous learning tend to shift faster than chronic schizophrenics and normal controls at the outset of reversal. However, the same findings also point out to the difficulty experienced by this group of subjects to maintain their attention, and consequently their response, to the selected stimulus for prolonged periods of time.
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Dissertation Submitted for the Degree
Master of Science in Clinical Psychology,
Institute of Psychiatry, University of London, 1993

Schizotypy in Normal Controls and their Performance on the
behavioural Phenomenon of Reversal Learning Task

Abstract

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PERSONAL STUDY PLAN

FOR

DOCTORATE OF PSYCHOLOGY IN CLINICAL PSYCHOLOGY

NAME: Stelios Georgiades
DATE OF REGISTRATION: October 1997
REGISTRATION NUMBER: 3716333

SECTION I: ACADEMIC DOSSIER

Review I: The Efficacy of Cognitive Behavioural Therapy in the reduction of positive symptomatology in Schizophrenia

Since the descriptions of Kraeplin (1909-1913/1971) and Bleuler (1911/1950) nearly a century ago, schizophrenia was regarded as a biological disorder and consequently all attempts for treatment were predominantly focused on the development of more effective psychopharmacological agents. However, recent findings indicating that medication is not equally effective to all patients (e.g., Johnson et al, 1987; Davis and Casper, 1997) led to the development of Cognitive Behavioural Therapy techniques where the main aim is to alleviate symptoms in people with psychosis (e.g., Kingdon and Turkington, 1991; Fowler et al., 1995). In the last decade several studies were reported suggesting that this treatment modality can be quite effective in the reduction of drug resistant psychotic symptoms (e.g., Garety et al., 1994). The main aim of this review is to examine these studies and to evaluate the extent of which such an approach contributes to the treatment of the disorder.
Review II: The Value of Schizotypy in Enhancing our Understanding of Schizophrenia

The results reported in most studies of schizophrenia research are often difficult to draw any conclusions from or to interpret them due to the presence of a number of confounding variables such as medication, hospitalization and low motivation. During the past thirty years, under the wider umbrella of Mehl's (1962; 1990) model which suggests that schizophrenia and schizotypy are genetically related, research in the area has progressively shifted so that greater emphasis is placed in understanding schizophrenia through the investigation of schizotypy. The main aim of this review is to draw together and review evidence from different areas of current schizotypy research in order to evaluate the advantages of the approach in enhancing our understanding of the disorder.

SECTION 2: CLINICAL DOSSIER

Service Development of Evidence Based Practice: Investigating the efficacy of Cognitive Behavioural Therapy in the treatment of Post-Traumatic Stress Disorder

The Adult Acute Section of Barnet's Healthcare NHS Trust Clinical Psychology Department, at the time of writing this study, employs 10.1 Whole Time Equivalent Clinical Psychologists and covers the areas of Barnet, Edgware Borrhamwood and Potters Bar with an average population of 240,000 people. Due to the small number of clinical psychologists and the large population that the department needs to offer its services to, it is important to ensure that all referrals receive the most effective service possible in terms of the time seen for initial assessment and the type of therapy that is subsequently offered to them. Decisions as to the type of therapy offered to clients are predominantly based on findings and recommendations produced by clinical research studies. Roth and Fonagy (1996) suggest that these findings do not necessarily reflect the clinical needs of Psychology Departments but rather the intentions, the resources and the interests of the researcher(s) carrying out the study. It is for this reason that the findings reported by clinical research studies often cannot be replicated in clinics. The problems
associated with current clinical research practices were highlighted in the National Health Service Psychotherapy Services in England (1996) review. It was suggested that one way of improving clinical effectiveness is by developing evidence-based practices where the efficacy of psychotherapeutic interventions is constantly monitored. Such an approach will ensure that specific groups of clients will receive psychotherapeutic treatments that are more relevant and more effective to their particular psychological needs.

Predominantly based on the recommendation of National Health Service Psychotherapy Services in England (1996), the main aim of the present study is to investigate the efficacy of Cognitive Behavioural Therapy in the treatment of PTSD in the course of routine clinical practice using outcome measures on initial assessment and on discharge. In the study, this will be presented in four parts: (a) presentation of clinical literature review arguments in support for the need to develop such a service, (b) description of the type of intervention offered to clients using the service (c) evaluation of the intervention with the aid of inventories completed by clients on assessment and on discharge and (d) suggestions for improving the treatment protocols.

SECTION 3: RESEARCH DOSSIER

Title of Research Project
Investigation of Cognitive Deficits in Schizophrenia through performance on a reversal learning task

Research Supervisor
Dr Marie Clark, Research Fellow, Department of Psychology, University of Surrey

Background and Relevance
Hemsley (1987) postulates that basic to the schizophrenic condition “is a weakening of the influences of stored memories of regularities of previous input on current perception”
Gray and his colleagues (1991) in their proposed neuropsychological model of schizophrenia suggested that the cognitive deficit described by Hemsley (1987) could correspond to an underlying neurological deficit specified in the projection from the hippocampus to the nucleus accumbens. The research reported in the present part of the portfolio seeks to examine Hemsley’s (1987) and to a certain extent Gray’s and colleagues’ (1991) models of schizophrenia. By following a similar procedure to the one used by other researchers (e.g., Baruch, 1988, Jones et al., 1992) examining the same models (but with different experimental paradigms) the hypothesis that people with acute schizophrenia fail to learn from previous experiences will be investigated using the behavioural phenomenon of reversal learning (Lawrence, 1949). The reason for selecting this particular task derives from the fact that it has been shown in animal (e.g. Weiner et al., 1986) as well as in human (e.g. Nolan, 1974) studies that reversal learning depends on the control of attention by prior experience and that it can facilitate direct comparisons of the extent by which prior experiences may influence new learning.

Methodology

(i) Subjects
Three groups of subjects will be employed. Two groups of people with schizophrenia (acute and chronic) and a group of normal controls.

(ii) Design
A between subject design will be adopted in this study

(ii) Apparatus and Stimuli
The experimental procedure will be carried out using an ATTARI 1040 computer and a colour monitor. The task to be employed is the modified and computerised version of the original card task employed by Nolan (1968). Two stimuli differing along the dimensions of both shape and colour will be presented in a colour monitor. The two colours appearing on screen will always be red and green and the two shapes always a
circle and a triangle. The stimuli will appear in randomised combinations (e.g., green
circle-red triangle, and red circle-green triangle) and each stimulus of the combinations
will appear either on the right or on the left of the screen. The main task of the subjects
will be to decide by trial and error which one of the two colours on the computer will
regard shapes as the correct one. The subjects will respond by pressing the appropriate
response keys. In addition to the computerised tasks, all subjects will be tested for their
overall intellectual functioning using the Standard Progressive Matrices (Raven, Court and
Raven 1988). People with schizophrenia will be rated by the experimenter and the
psychiatrist responsible for their care on two symptom scales, the Brief Psychiatric
Rating Scale (BPRS; Overall and Gorham 1962) and the MAINE scale (Magaro et al.
1981)

(iii) Procedure
All subjects will be tested individually with the experimenter present. Following initial
instructions, subjects will commence the computerised task. The first task of the subjects
will be to discover by trial and error which one of the two stimuli appearing on the screen
is the correct one. In this first part of the experiment, the stimulus that will be regarded by
the computer as the correct one is the colour red. Upon reaching the learning criterion,
(L) which is to score correctly on nine consecutive trials, the reversal learning condition
(RL) will commence. In this part of the experiment, subjects will be rewarded for
choosing the alternative stimulus within the same dimension (colour green). The task
will end after the subject reaches the learning criterion; i.e., scoring correctly on nine
consecutive trials

On completion of the task, two scores will be obtained for each subject. The first score
will be referred to the number of trials taken by the subject to acquire the initial
discrimination and establish which one of the stimuli is the correct. The second score
will refer to the number of trials taken by the subject to establish the correct stimulus
during the reversal learning task.
Results

For all three groups, analysis of the results will be carried out using standardised statistical procedures. Demographic data between groups will be tested for significance using the Mann-Whitney non-parametric test. The ratings of people with acute and chronic schizophrenia on the BPRS and MAINE that will be carried out by the psychiatrist and the experimenter will be examined for inter-rater reliability by using the Pearson’s correlation test. Statistical differences between the scores of L and RL on each group of subjects will be investigated using t-test (related). Differences across all three groups on each of their scores will be examined using one way ANOVA.
References


SECTION II: Academic Dossier
Review I: The Efficacy of Cognitive Behavioural Therapy in the reduction of positive symptomatology in Schizophrenia

1. Aim of Review

Following the traditions of Kraeplin (1909-1913/1971) and Bleuler (1911/1950) schizophrenia up until recently was regarded as a biological disorder with treatment developments being solely on the psychopharmacological domain. The discovery of neuroleptic drugs and their impact in the alleviation of psychotic symptoms provided further support for the biological basis of the disorder strengthening the belief that the future of the disorder relied on the development of more effective medication.

Beliefs that psychotic experiences were categorically different from normal experiences and that symptoms such as delusions were unresponsive to rational arguments (e.g., Jaspers, 1963) were so prevalent that there was virtually no research interest in psychological therapies. The recent introduction of atypical antipsychotic medication although quite effective in the reduction of psychotic symptoms also indicated that these drugs were not equally beneficial to all patients (e.g., Johnson et al, 1987; Davis and Casper, 1997). The conflicting evidence about the efficacy of pharmacological treatments and the development of cognitive theories that attempted to account for the emergence of symptoms led to the development of Cognitive Behavioural Therapy for people with psychosis (e.g., Kingdon and Turkington, 1991; Fowler et al., 1995).

In the last decade, several studies were published pointing out that Cognitive Behavioural Therapy as an approach is very promising for the future of the treatment of schizophrenia. The main aim of the present paper is to briefly review the current psychological theories of positive symptoms in schizophrenia and by examining a sample of these studies to evaluate the extent of which such an approach contributes to the treatment of the disorder.
2. Background to the treatment of Schizophrenia

The contribution of psychological factors in schizophrenic symptomatology was recognised almost concurrently with the formulation of the disorder (see Bleuler (1911/1950). For an illness however that seemed to reflect dissociation between the functions of emotions and cognition (see McKenna, 1994), research into the possible role of psychological therapies in the treatment of schizophrenia up until the last decade were few and sporadic (Milton et al, 1978; Hartman and Cashman, 1983). According to Bellack (1986) the lack of motivation and scientific interest in this area of schizophrenia research was due to the influence of four misconceptions: (i) that schizophrenia as an illness does not exist, (ii) that it is a biological disorder (iii) that it is adequately handled by medication, and (iv) that schizophrenia is too severe for behavioural therapy. As a result and despite the evidence indicating the potential of cognitive therapy in reducing delusions (Beck, 1952; Hole et al, 1979; Watts et al, 1973), the study and the application of this treatment modality remained neglected.

The discovery of neuroleptics (Davies, 1975) and the subsequent introduction of atypical antipsychotic medication advanced the management of schizophrenia (Tarrier, 1992; Zipursky and Schulz, 2002) due to their profound impact in reducing positive symptomatology (see Zipursky, 2002) such as delusions (Davis et al., 1980) and hallucinations (e.g., Kopala et al., 1996; Keshavan et al., 1998). Subsequent studies however, indicated that between 30 to 40 per cent of patients with schizophrenia relapse while on pharmacotherapy (Johnson et al, 1987) whereas a significant proportion of them continue to experience symptoms in spite of medication (Davis, 1997). Moreover, it was pointed out that some drugs may produce unpleasant side effects, which in turn causes patients to default (Buchanan, 1992) or to be poorly compliant to medication (Kissling, 1992). As a consequence of the number of problems associated with neuroleptic medication, other treatment methods for schizophrenia had to be investigated.
3. Cognitive Theories of Delusions and Hallucinations

The symptoms that characterise schizophrenia fall into two broad categories (Crow, 1980; 1985), the positive symptoms that reflect an excess or distortion of normal functions (e.g., hallucination, delusions) and the negative that reflect a diminution or loss of normal functions (e.g., grossly disorganised or catatonic behaviour). Although several attempts were made by cognitive theorists to account for the development of negative symptomatology (e.g., Hemsley 1987) greater emphasis was placed in the conceptualisation and understanding of delusions (e.g., Hemsley, 1993; Garety and Hemsly 1994) and hallucinations (e.g., Slade and Bentall, 1988; Morrison and Haddock, 1997).

3.1 Delusions

From these, delusions were recognised quite early in the history of schizophrenia to constitute a key feature in the diagnosis of the disorder (see Maher and Spitzer, 1992). Psychological investigation of this category of symptoms however, was neglected due to early accounts (e.g., Jaspers, 1963) which pointed out that normal beliefs and delusions are separated by an unbridgeable gap and that delusions are nothing but empty speech acts that do not refer neither to world nor self (see Bentall et al., 1994).

More recently various attempts have been made by cognitive scientists to define what is a delusional belief without necessarily taking into account the underlying neurological deficits described by neuropsychological theories (Frith, 1992; Hemsley, 1993). Strauss (1969) in an attempt to emphasise the modifiability of delusional beliefs, he proposed that they should be viewed as points on a continuum along with normal functioning. Moreover, it was proposed that their position on this is to be determined by factors such as the client’s degree of belief conviction and the amount of time spent preoccupied by the belief. According to the same theorist delusions could be modifiable to varying extents, depending on the degree of the belief conviction. Continuing with the conceptualisation of delusions, Maher (1988) proposed that they are rational attempts to explain abnormal or unusual perceptual experiences. Within this framework it was suggested that the process of reasoning that produces delusions is similar to the process
involved in producing ‘normal’ beliefs, but that ‘bizarre perceptions demand bizarre explanations’.

Deriving from current theoretical models several cognitive studies investigating cognitive biases in people with delusions and the way by which they distort their perception of life experiences (for a review see Rector and Beck, 2002) indicated that delusional thinking shares a number of common characteristics such as egocentric, externalising and a self-serving biases (see Haddock et al., 1998). These biases in their reasoning processes are thought to help individual patients to make sense of the world and protect their vulnerable self esteem (e.g., Bentall et al., 1991; Kaney and Bentall, 1989).

### 3.2 Hallucinations

Auditory hallucinations are regarded as the most frequently reported symptom in schizophrenia (Rector and Beck, 2002). Although there is not an agreed model by which to explain the formation of this group of symptoms, most cognitive scientists at the core of their theories agree that auditory hallucination are the result of misinterpretation of externally and/or internally generated events. More specifically it is proposed that ambiguous external noises are interpreted by people with schizophrenia as voices (e.g., Bentall and Slade, 1985; Young et al., 1987).

To some researchers (Frith and Done, 1987; Frith, 1992) hallucinations are regarded to be the result of an underlying neurological deficit that is located in the temporal lobe (e.g., Gray et al., 1991; Hemsley, 1987; 1987; 1993; Frith, 1992). As a result of this impairment quite often willed intentions are not adequately monitored the actions are not recognised as self-initiated and they are attributed to external agents (Frith, 1987). Alternative theoretical explanations consider the development and the content of hallucinatory experiences to be highly influenced by cognitive biases (Slade and Bentall, 1988; Bentall and Slade, 1985) such as the individual patient’s idiosyncratic belief with regard to the identity of the voice (Bentall, 1990) and the expectations they might have (Birchwood and Chadwick, 1997; Chadwick and Birchwood, 1994).
4. Cognitive Behavioural Therapy for Schizophrenia

The indications that delusions and hallucinations are strongly associated with biases in reasoning and explanatory processes paved the way for the development of psychological therapies. Moving away from the biological view that schizophrenia is the sole area of psychopharmacology, the main aim of psychotherapeutic interventions is to help patients identify and correct cognitive distortions that according to theoretical models play a contributory role in the development of psychotic symptoms (see Garety and Hemsly 1994).

Although several small scale studies (e.g., Hole et al., 1979; Hartman and Cashman, 1983) and single study cases (e.g., Beck, 1952; Garcelan and Yust, 2000) pointed out to the potential impact that cognitive behavioural therapy (CBT) could have in reducing delusions and hallucinations it is only during the last decade that cognitive behavioural interventions developed into a more structured and systematic approach of treatment. Although the common target of all treatment packages is to help patients identify and by extent correct their cognitive distortions, alleviate or reduce the symptom in question and improve their quality of life, each of the packages differ on the emphasis they place on specific aspects of the disorder (see Dickerson, 2000). For example, the main aim of the approach described by Kingdon and Turkingdon (1991) is to guide patients to recognise their symptomatology and through inductive questioning and challenging techniques to assist patients to normalise their responses and hence their symptoms. In other approaches the emphasis is shifted to enhance the patients' repertoire of coping skills (Tarrier et al., 1992) or to assist them re-evaluate and restructure their beliefs with regard to their self and the world (Chadwick et al., 1996). In another approach recognising the importance of all targets set by previous treatment manuals encompasses within its framework most elements described earlier in a comprehensive treatment manual starting with the role of establishing rapport and concluding with the relapse prevention component (Fowler et al., 1995). The introduction of these manuals and therapeutic guidelines stimulated an increased number of clinical studies which despite their differences within their specific targets they all aim to investigate the impact of CBT in its wider form in the alleviation of symptomatology.
4.1 Empirical studies investigating cognitive behavioural therapy in schizophrenia

One of the early nonrandom controlled studies following the principles of a treatment manual was carried out by Garety and colleagues (1994). In that pilot study, a group of patients with a diagnosis of either schizophrenia or schizo-affective psychosis, who presented unremitting (at least six months), drug-resistant, positive psychotic symptoms were offered a treatment package that included a variety of cognitive behavioural techniques (see Fowler et al., 1995). The results were promising in that it showed the advantage of CBT and Routine Care (RC) compared to RC only in the reduction of symptoms in people with chronic schizophrenia.

The first large randomised controlled trial of intensive treatment for medication-resistant symptoms of psychosis was conducted by Kuipers and colleagues (1997). The main aim of the study was to offer long-term CBT and to evaluate any changes in outcome. Participants were randomly allocated between a CBT and RC condition and a RC only control condition. Therapy lasted for nine months, was individualised, and involved the use of similar strategies as the ones used by Garety and colleagues (1994). The results of the Kuipers and colleagues (1997) study showed by means of scores on the Brief Psychiatric Rating Scale (BPRS; Overall and Gorham, 1962) that CBT and RC combined can reduce delusions and hallucinations up to 25% in people with medication-resistant psychosis. This improvement was shown to be maintained on a nine-month follow up (Kuipers et al, 1998).

The above studies, although indicative of the potential benefits of CBT and RC combination in the reduction of delusions and hallucinations (see Rector and Beck, 2001), it is extremely difficult to ascertain with any degree of accuracy as to whether the results obtained can be attributed to CBT or to any other parameters such as the patient-therapist frequent contact or to potential differences in idiosyncratic beliefs and characteristics of patients in the two groups (Garety et al, 1997).

Randomised controlled studies aiming to clarify the role of CBT in the reduction of symptomatology in addition to the CBT-RC and RC only conditions they included in
their design a third group of patients that received an alternative form of therapy. In a controlled study reported by Tarrier and colleagues (1993) CBT (termed as 'coping strategy enhancement') and RC was compared with the 'problem solving' (PS) technique (Hawton and Kirk, 1989) and RC and RC (waiting list) alone. The expectation of the study was that the PS technique although generally beneficial to patients it would not contribute to the reduction of psychotic symptoms. The results of patients receiving CBT-RC indicated that they experienced greater reduction in their positive symptoms than the PS-RC and the RC groups who showed no improvement. Moreover, patients who received CBT-RC showed greater improvement on symptom-related assessments such as anxiety and delusions as indicated from their scores on the subscales of the administered Psychiatric Assessment Scale (Krawiecka et al., 1977). Considering as significant change an at least 50% reduction in symptomatology it was shown that 60% of the CBT-RC group experienced significant reduction of symptoms compared to the 25% of the PS-RC. Despite the significant symptom reduction observed in the CBT-RC condition the possibility that might have been differences in terms of the severity of symptoms experienced by patient between conditions is not clarified.

Similar results to the Tarrier and colleagues (1993) study where reported by the randomised controlled study of Tarrier and colleagues (1998). In that study subjects were assessed for severity and were randomly allocated in one of three conditions ensuring that in each condition patients experienced symptomatology of similar severity and took similar doses of medication. Clinical raters where blind to treatment condition and subjects were allocated to CBT-RC, Supportive Counselling (SC)-RC and RC only. Again, considering the 50% symptom reduction criterion it was found that patients receiving CBT-RC experienced greater reduction in their symptomatology than subjects in the other two conditions. In a 12-month follow-up it was found that patients on the CBT-RC condition continued to experience less positive symptoms than patients on the other two conditions with 4 patients in the RC only condition to relapse and to remain in hospital for an aggregate total of 204 days.
The effectiveness of CBT in the reduction of symptoms compared to other forms of psychological therapy was also reported by Pinto and colleagues (1999). In that study subjects with drug resistant psychosis were randomly allocated either to CBT-RC or to Supportive Therapy (ST)-RC. The CBT approach was based on the modules described in the Fowler and colleagues (1995) manual whereas the ST included elements such as psychoeducation and crisis management. On completion of therapy the group of patients receiving CBT-RC showed greater reduction in all symptom measures than the ST-RC group. On a six-month follow-up, the CBT-RC group continued to have significantly less symptoms than their ST-RC counterparts.

The studies described compared CBT, RC and another therapeutic approach. The introduction of the second psychotherapeutic modality aimed at providing general therapeutic benefits to people with schizophrenia but not to target specific symptoms. The results reported although indicative of a CBT advantage compared to the alternative modality, the findings produced also showed that when compared to the RC only patients in the alternative treatment group also experienced significant reduction of symptoms (e.g., Tarrier et al., 1993; Tarrier et al., 1998). On the light of these findings a relevant question that can be asked is with regard to the actual role of CBT in the reduction of symptoms in schizophrenia. Is it valid to assume that the only value of CBT in the treatment of schizophrenia compared to other psychotherapeutic modalities is the extent by which it alleviates symptomatology?

In a study carried out by Sensky and colleagues (2000) whereby employing the treatment approach described in the manual by Kingdon and Turkington (1994) they attempted to investigate and compare the impact of CBT-RC to a placebo therapy (termed as “Befriending Therapy” - BT-RC) in the reduction of symptoms in schizophrenia. The results indicated a significant and equivalent reduction of symptoms in both groups as derived by a number of measures. In a nine-month follow-up however it was found that patients on the CBT-RC condition were significantly superior to BT-RC in all measures of psychopathology pointing to the potential value of CBT in relapse prevention and in maintaining the beneficial effects of therapy.
All of the above studies are primarily concerned with patients with long term schizophrenia. There is available evidence however that indicates the efficacy of CBT in the acute phase of the disorder. Following the treatment protocol described by Chadwick and colleagues (1996) Drury and colleagues (1996) carried out a randomized control study employing patients while on an acute episode of schizophrenia whereby CBT-RC was compared to Informal Support Therapy (IST)-RC. The later group was engaged regularly on social and leisure activities as well as they received informal support. Again, like in earlier studies, although both groups showed significant reduction in their symptomatology the CBT-RC group showed that the reduction of their positive symptoms was significantly more than that of the IST-RC group. These changes continued to be present six months later with a significant higher number of CBT-RC participants reporting full remission or very low symptoms. In a five year follow up it was found that from those participants that had relapsed, the group of patients that had received CBT-RC had significantly lower symptomatology that those who received IST-RC (Drury et al., 1999).

All studies reviewed thus far referred primarily to patients receiving individual therapy. The potential impact of CBT in groups was investigated in a preliminary study by Gledhill and colleagues (1998). The group comprised of two men and two women with schizophrenia. The group ran for eight consecutive weeks, with each session lasting one hour comprising elements such as psychoeducation, formulation and problem solving techniques. The results of the study demonstrated that this therapy format can have a beneficial effect not only in reducing positive symptoms and associated symptoms such as depression but also in increasing the participants self esteem and repertoire of skills in coping with their symptomatology.

4.2 Evaluation of empirical studies

The importance of the findings reported in the preceding section seems to be strengthened by a number of methodological considerations. All studies employ structured treatment protocols as derived from therapy manuals (Kingdon and Turkingdon, 1991; Fowler et al., 1995; Chadwick et al., 1996). Also in the majority of
them there are control conditions in the form of RC only (e.g., Garety et al., 1994; Kuipers et al., 1997) and in others there is the addition of a third condition of RC plus another psychotherapeutic intervention (e.g., Tarrier et al., 1993; Tarrier et al., 1998). Most importantly however is that all studies employ reliable diagnostic criteria (see Cormac et al., 2002) and use valid outcome measures for positive symptoms such as the BPRS (Overall and Gorham, 1962), the Psychiatric Assessment Scale (Krawiecka, 1977) and the Schedule for the Assessment of Insight (David, 1990). Some studies acknowledge and investigate associated symptomatology such as depression and anxiety (e.g., Sensky et al., 2000) whereas in others the severity of symptoms amongst patients between conditions was controlled (e.g., Tarrier et al., 1998). Recognising the impact of medication in therapy outcome in some studies medication dosage during trials was monitored (e.g., Tarrier et al., 1998). Finally it is worth noting that despite the inherent difficulties of employing double blind evaluation of the outcomes of psychosocial intervention, attempts were made to minimise the effect biases by employing blind raters (e.g., Tarrier et al., 1998).

Although the results of these research studies in their more general form seem to be promising in terms of the potential of CBT in the reduction and treatment of positive symptoms in schizophrenia, the same studies also suffer from a series of methodological limitations. As indicated above, studies were conducted using different treatment protocols. The employment of published protocols although positive in that the steps followed during therapy are precise, each protocol describes different techniques and places greater emphasis to some aspects of the disorder than to others. As a result, when talking about the impact of CBT in reducing positive symptomatology (e.g., Garety et al., 1994; Kuipers et al., 1997) or improving medication compliance (Kemp et al., 1998) we cannot talk of the effects of a specific treatment approach but of a different variation of a more general approach. As a result, the targets set and the techniques used by each study vary making the results obtained between studies difficult to compare. This points to the need for greater investigation of each particular protocol and the specific impact that each one of them has on specific aspects and/or symptoms of the disorder.
Another problem associated with studies investigating the efficacy of CBT in the treatment of people with schizophrenia, is with regard to the way by which improvement is considered. In some studies the emphasis was on the reduction of delusion and/or hallucinations whereas in others the stabilisation of the same symptoms and insight and dysphoria (Drury, 2000). In studies where the targets were similar, the measures employed were not the same (see Tarrier et al., 1993; 98; Garety et al., 1994; Kuipers et al., 1997). Also the percentage of symptom reduction -as derived from questionnaire measures- considered by researchers to constitute significant improvement is not stable across studies. For example, in the Tarrier and colleagues (1993; 1999) significant improvement was defined by a 50% or more reduction in symptoms whereas in Kuipers and colleagues (1997) significant improvement was defined by a 25% reduction of symptoms. The variability in the symptoms targeted for treatment and the differences across studies as to what can be considered significant reduction of symptomatology indicate the need for developing therapy specific standardised outcome measures or even employing in a more consistent rate other outcome measures such as self harm, relapse, re-admission to hospital or increases in medication.

Also as shown above the emphasis on therapy results is either on the reduction of symptoms or on specific behaviours known to be closely associated with the disorder such as medication compliance and insight. However none of the studies reviewed reported the impact of CBT on the subsequent socioeconomic functioning of these individuals compared to patients on the other conditions.

In most studies apart from CBT-RC and RC only there is also the inclusion of RC plus an alternative treatment approach spanning from supportive psychotherapy (Drury et al., 1996) to psychoeducation (Pinto et al, 1999). Looking at the results of such studies in the majority of them there is evidence to suggest that a variety of psychological interventions can reduce significantly the positive symptoms by the end of the treatment period. None of the studies however reports for which aspects of the disorder in the initial improvement CBT is more effective than other psychological interventions.
Finally although some studies do investigate associated symptomatology and attempt to control for medication intake amongst patients between groups during trials the majority of them do not. Similarly, only in few studies there is evidence of attempting to reduce the effect biases by employing blind raters (Tarrier et al., 1998) whereas none of them reports the employment of independent raters in assessment and their inter-rater reliability.

5. Conclusions and suggestions for future research

The development and application of Cognitive Behavioural Therapy for people with schizophrenia undoubtedly marked the beginning of a new trend in the treatment of the disorder. Considering the limitations of pharmacotherapy and the results reported by a number studies investigating the efficacy of cognitive behavioural therapy it seems that this later treatment modality can be developed as an integral part of the overall treatment of schizophrenia.

The random controlled studies reported indicate that Cognitive Behavioural therapy is quite effective in reducing drug resistant or otherwise symptoms of schizophrenia both in the acute and in the chronic phases of the illness. The value of these findings seems to be strengthened by the fact that all studies used patients with reliable diagnoses and that their responses to therapy were measured by reliable outcome measures. Furthermore most studies followed manual based treatments whereas in addition to the routine care treatment most of the reported studies included and compared Cognitive Behavioural therapy to another treatment modality spanning from supportive psychotherapy to problem solving techniques. Most of these studies seem to report that in addition to the immediate improvement in terms of reducing symptomatology this beneficial effect seems to be maintained over time. Finally more recent studies began to take into consideration the role of medication and the severity of symptomatology so that attempts were made to monitor the first and control for the later.
Of course, as indicated in the evaluation section of the present paper, these studies are not without their methodological limitations. The small sample of subjects employed in these studies, the variation in the treatment protocols used, the different outcome measures used by different studies and the fact that none of the study investigates the socioeconomic situation and the quality of life of patients following their discharge from therapy are all important issues that need further clarification. Another important observation that is worth mentioning (see Rector and Beck, 2002) is that all of the studies reported were carried out in the United Kingdom. This reflects the fact that Cognitive Behavioural Therapy was developed and applied in this country. However, until this treatment modality is tried using non-British population in other countries it will not be possible to ascertain with any degree of certainty whether these findings are specific to this part of the world or across cultures.

Finally, considering the short history of Cognitive Behavioral Therapy into the treatment of the disorder and the relatively small number of studies reported it seems that all available results are quite encouraging in suggesting that Cognitive Behavioural therapy could play a significant role in the future of the treatment of schizophrenia. Also the fact non-drug treatments for schizophrenia are likely to be of low risk, have no physical side effects, are not physically intrusive and may be more immediately acceptable to patients (Kuipers 1996) strengthens even more the potential advantages of the approach. However, taking into consideration the methodological limitations outlined above and the absence of any long term follow up data one has to be cautious in drawing any final conclusions with regard to the efficacy of the approach as more research accounting for the present limitations is necessary.
6. References


Review II: The Value of Schizotypy in Enhancing Our Understanding of Schizophrenia

1. Aim of Review

Schizophrenia is a severe and highly disabling disorder that affects almost 1% of the population (Gottesman, 1995). According to McKenna (1994) no war or any other disaster in the history of mankind has ever cost or claimed so many lives as schizophrenia does in a single year (see Wyatt et al., 1995). Since the description of the disorder by Kraeplin (1909-1913/1971) and Bleuler (1911/1950) scientific attempts to understand the pathogenesis and the phenomenology of the disorder led to the investigation of a vast array of factors. Despite the intensity of research in the area, progress in our understanding of the disorder has been hampered by the fact that any findings in terms of the behaviour, perception, response or even neurophysiology of people with schizophrenia are complicated by third degree confounding variables.

During the past thirty years, highly influenced by genetic research (Kety et al., 1968) and phenomenological indications (see Vollema, 1999) it was proposed that schizophrenia and schizotypal personality disorders might be genetically related and hence share the same underlying biological construct (Mehl, 1962; 1990). Under the wider umbrella of this hypothesis there is a progressive shift in the methodology of schizophrenia research today where emphasis is placed in understanding schizophrenia through the investigation of schizotypy.

The main aim of the present study is to draw together and review evidence from different areas of current schizotypy research in order to evaluate its advantages as a potential alternative method for investigating schizophrenia. Emphasis will be placed in examining the available methods of identifying and selecting schizotypes for research purposes as well as on examining the extent of which schizotypal individuals and people with schizophrenia resemble on behavioural, cognitive as well as on other characteristics relevant to schizophrenia. Finally, taking into consideration the difficulties associated
with the treatment of schizophrenia despite recent advances in pharmacotherapy (Zipursky and Schulz, 2002) and psychotherapy (for a review see Rector Beck, 2001), an attempt will be made to examine the potential role of schizotypy in the development of preventative treatments for people that are on high risk to experience a psychotic episodes.

2. Background to the Concept of Schizotypy

The possibility that schizophrenia can manifest in an alternative manner was first introduced by Kraeplin (1909-1913/1971) almost concurrently with his descriptions of dementia praecox. Similar to Kraeplin’s views, Bleuler (1911/1950) proposed that all core symptoms of the disorder can exist in a covert form in what he named as “latent schizophrenia”. Rado (1953), predominantly based on clinical observations introduced the term schizotypy as an abbreviation for “schizophrenic genotype” and referred as such to the group of people that are hereditary predisposed towards developing schizophrenia. Solely explaining schizotypy through the psychoanalytic prism it was proposed that schizotypes are experiencing an “integrative pleasure deficiency”. Continuing, he suggested that these individuals develop adaptive mechanisms that enable them to cope more or less effectively without necessarily developing schizophrenia.

Although these early suggestions appeared to indicate the existence of a continuum between psychosis and normality, research into the potential link between psychotic proneness and schizophrenia remained neglected up to the 1960’s. The introduction of the third edition of the Diagnostic and Statistical Manual for Mental Disorders (DSM-III; APA, 1980) and the recognition that individuals who under the DSM II (APA, 1968) were diagnosed as suffering with schizophrenia in this newer edition of the DSM were regarded as schizotypals, it was indicative that schizotypy and schizophrenia might be related. Also influential in the possible relationship between schizophrenia and schizotypy were the findings of the Danish Adoption Study that reported elevated levels of “borderline” and “latent schizophrenia” in the biological relatives of people with schizophrenia (Kety et al., 1968; Kety et al., 1975).
One of the earliest attempts to describe the relationship between schizotypy and schizophrenia in the form of a coherent theory was proposed by Mehl (1962; 1989; 1990). Highly influenced by Rado's (1953) explanation, at a very general level Mehl's model suggests that schizophrenia is genetically determined by a specific genetic factor which activates the emergence of the disorder through its interaction with other genetic factors (e.g., social introversion, anxiety) and environmental stressors. According to the same model this genetically specific factor of schizophrenia is transcribed into a functional synaptic control aberration in the central nervous system and results a synaptic slippage in the brain. This synaptic slippage termed schizotaxia almost always develops into schizotypy via social learning experiences. Continuing, the same theorist suggests that although schizotypy is a personality organisation that predisposes individuals to schizophrenia it does not manifest in overt clinical symptoms. On the contrary, schizotypes are characterised by subtle behaviours and cognitive responses such as thought disorder that are only evident in laboratory and/or experimental measures. Finally, the same model emphasises that most schizotypal individuals remain at this state throughout their lifespan and only a small percentage of them develop schizophrenia. This suggestion was recently supported by the study of Lenzenweger and Korfine (1992) which indicates that only 10% of schizotypes develop the disorder.

Mehl's model (1962; 1990) seems to bear certain similarities with other models that point out to the existence of a continuum between normality and psychiatric illness (Eysenck and Eysenck, 1976). Also compatible to Mehl's theory is the more recent stress vulnerability model (Zubin and Spring 1977) in which schizophrenia is considered to be the result of an interaction between genetic predisposition and environmental stressors.

3. The Role of Schizotypy in Schizophrenia Research
Investigating schizotypy in itself can be regarded as important as attempting to increase our understanding of any other psychiatric illness. However, its real value in current research lies on its contribution in providing the tool by which to investigate schizophrenia (e.g., Coleman et al., 1996). As it is indicated by studies (e.g., Squires-
Wheeler et al, 1989; Titelman and Nilsson, 1992) schizotypy, schizotypal personality disorder and schizophrenia are all genetically related. These findings seem to accord well Mehls’s (1962; 1990) model which suggests that schizotypes and people with schizophrenia share the same biological vulnerability for developing the disorder. As a result, when investigating schizotypic psychopathology we do not employ a clinical analogue of schizophrenia but rather a variation of the disorder on individuals that potentially can go on to develop schizophrenia.

It is known schizophrenia is a highly disabling psychiatric illness that affects 1% of the general population. The experience and response of patients with this disorder vary not only in terms of symptomatology and severity of symptoms but also in terms of treatment and prognosis (see Frangou and Murray, 2000). Due to the complexity of the illness, research aiming to investigate the possible causes, the characteristics and the cognitive deficits associated with schizophrenia is often plagued by a series of direct or indirect confounding variables (see Claridge 1997).

A major problem that was recognised relatively early in schizophrenia research (e.g., Cromwell and Dokecki 1968) was the “worse than normal” performance of people with schizophrenia in experimental tasks. Such results can be attributed to a variety of factors (Lenz et al., 1995) such as the low motivation and the symptomatology of this group of patients that could mask their true cognitive abilities. Of course the impact of the schizophrenia experience itself, hospitalisation, chronicity and the associated low self esteem also seem to contribute negatively to their overall performance on experimental tasks. In addition to the above, the fact that people with schizophrenia are usually on neuroleptic medication complicates matters even more as one can never be certain as to which of the findings are specific to schizophrenia and which are due to the effects of medication.

In contrast to the methodological limitations and the large number of confounding variables associated with schizophrenia research, studies employing schizotypes not only potentially can be free of these disadvantages but also as a group of subjects...
they are easier to recruit due to their higher prevalence in the general population. In fact, in a study carried out by Korfine and Lenzenweger (1995) it was estimated that the prevalence for schizotypy is about 7% compared to the 1% prevalence for schizophrenia (Guttesman, 1995). Another advantage of schizotypy research is that as Raine and Manders (1989) suggested by being able to distinguish schizotypes in the general population it would facilitate the employment of schizotype-free controls in schizophrenia research and therefore it would increase the likelihood of obtaining results that are specific to the disorder and not to the masking effects of confounding variables.

4. **The Recruitment of Schizotypes**

Despite the fact that at a theoretical level the prevalence for schizotypy is significantly higher than schizophrenia the recruitment process for this group of subjects is not as straight forward as it is for people with schizophrenia. Traditionally schizophrenic subjects were recruited from hospitals and there was no doubt or concern about their diagnosis and/or the severity of their symptomatology. In contrast people with schizotypal personality disorder or high schizotypy rarely come to hospitals. From the small minority that do attend clinics they do not usually seek help for their personality disorder itself but rather for secondary symptoms such as depression or increased tendencies for suicide (Millon et al, 2001). As a result most of these individuals cannot be employed for schizotypy research as any findings yielded will be complicated by their secondary symptomatology and circumstances.

According to Lenzenweger (1997) there are three methods to recruit schizotypes for research purposes. The first method is termed clinical and refers to the usage of diagnostic criteria for identifying people with schizotypal or paranoid personality disorder (e.g., DSM IV-TR; APA, 2000). As mentioned above, the problem with this approach is that this group of people rarely present themselves to the clinic and therefore no researcher can rely on this method for subject recruitment. The second method is the so called familial-biological. Utilising evidence that biological
relatives of people with schizophrenia are at higher risk than the general population for developing schizophrenia (e.g., Gottesman, 1991) it is considered that this group of people are more likely to display schizotypic characteristics. The problem of solely relying on biological relationship is that not all first degree relatives possess these characteristics. Therefore, any result obtained by using this method runs the risk of considering as a homogenous group a combination of people with high and low schizotypy. The third approach is the psychometric-laboratory method where the identification of schizotypes is achieved by the usage of reliable and valid psychometric measures. The advantage of this later method is that the only criterion for selection is the scores of individual subjects on specific scales. However, as pointed out by several researchers (e.g., Lenzenweger, 1997) by employing the psychometric-laboratory or the clinical approach it is possible that the identified schizotypal individuals might not be perfectly related to an underlying schizotypy construct as one would have expect to find in biological relatives of people with schizophrenia. Therefore, on the basis of the above, one could suggest that by combining at least two methods (e.g., familial-biological and psychometric-laboratory) may be regarded as the most appropriate method in selecting schizotypes for research purposes. This is because on the basis of their scores the selected schizotypes are more likely to be perfectly related to an underlying schizotypy construct.

4.1 Measurement of Schizotypy by Self Report Questionnaires

Current schizotypy research indicates that measuring schizotypy can be achieved in a variety of ways spanning from employing self-reported questionnaires (e.g., Chapman et al., 1978; Eckblad and Chapman, 1983; Nielsen and Petersen, 1976; Launay and Slade, 1981) to structured interviews (Kendler et al., 1989) to psychological tests (e.g., Jones, 1977). The present review will be focused on the development and usage of questionnaires due to their overall advantages over the other methods (see Grove et al., 1982).
Taking into consideration the complexities associated with schizophrenia as an illness and the variety of symptoms experienced by sufferers of the disorder, research into the area of schizotypy saw the development of a number of schizotypal scales during the past thirty years. According to Bentall and colleagues (1989) due to the multidimensionality of schizophrenia different researchers developed scales that would tap the underlying construct of schizotypy and by extent the liability for schizophrenia from different perspectives.

Scales such as the Perceptual Aberration (PAS; Chapman et al., 1978), Magical Ideation Scale (MI; Eckblad and Chapman, 1983), Physical Anhedonia Scale (PhA; Chapman et al., 1976), Social Anhedonia Scale (SA; Chapman et al., 1976), Impulsive Nonconformity Scale (IN; Chapman et al., 1984), Schizophrenism (NP; Nielsen and Petersen, 1976), Hallucination Scale (LSHS; Launay and Slade, 1981), Rust Inventory of Schizotypal Cognitions (RISC; Rust, 1988), Experiential Word Inventory (EWI; El-Melgi and Osmond, 1970), Social Fear Scale (SF; Raulin and Wee, 1984), Intense Ambivalence Scale (IA; Raulin, 1984) and the Cognitive Slippage Scale (CS; Miers and Raulin, 1985) are all symptom oriented scales aiming to derive indices that would detect the schizoid taxon (see Mason et al., 1995; Mason, 1995). The Psychoticism sub-scale (P) of the Eysenck Personality Inventory (EPI; Eysenck and Eysenck, 1975), Schizoidia Scale (GM; Golden and Meehl, 1979) and the Unfriendly World Scale (UW; Tellegen, 1978) as a group of measures can be regarded as belonging to the personality oriented approach emphasizing the view of a continuity between normality and psychosis.

The development of the clinical and the personality oriented scales although important in schizotypy research they are also separated by a gap. More recently, in order to bridge this gap several scales were developed by combining items from both clinical and personality questionnaires. Such scales are the Schizotypal Personality Scale (STA; Claridge and Broks, 1984), the Schizotypal Personality Questionnaire (SPQ; Raine, 1991) and the subscales of the Minnesota Multiphasic Personality Inventory (MMPI; Lachar, 1974).
An important aspect of the above scales that increases their validity as tools for investigating schizotypy is that despite their different psychometric properties most of them are internally consistent with adequate test re-test reliability value (e.g., Muntaner et al., 1988, Bentall and Slade, 1985; Rust, 1989). Furthermore, all researchers regardless of the approach they have adapted in developing these measures they all seem to agree that schizophrenia and schizotypy share the same underlying construct (Bentall et al., 1989).

A major problem associated with these self report questionnaires however is the low correlation between different scales (see Chapman et al., 1980). Although this low correlation might be explained through the multidimensionality of the disorder and therefore the predisposition might be restricted to a specific cluster of symptoms, the problem as to which scales to select when attempting to carry out high risk research remains. Considering the wide spectrum of symptomatology associated with schizophrenia and the number of traits linked with the disorder, studies on schizotypy should be able to account for all potential symptoms/traits. On the basis of this, it could be argued that the development of such a wide range of heterogenous schizotypal scales, each set measuring different aspects of the same disorder, ensures the investigations of all aspects of schizotypy.

However, can it be considered possible that a complex concept such as schizotypy can be described fully by a predisposition towards a heterogeneous set of symptoms which collectively compose a unified underlying construct that potentially leads to schizophrenia? Taking into account the heterogeneity of scales and by extent the different symptoms and/or traits associated with schizophrenia, Chapman and colleagues (1980) suggested that the different symptoms of schizophrenia might be linked to different factors and proposed that schizotypy may be also composed of more than one factor. This suggestion paved the way towards a series of factor analytic studies (e.g., Muntaner et al., 1988; Raine and Allbutt, 1989; Bentall et al., 1989; Venables, 1990) all aiming to unveil the factors associated with the schizotypy construct and by extent making it easier for research purposes to select the group of scales with high loading on
the specific factor that is more relevant to the hypothesis investigated (see Vollema and Van den Bosch, 1999).

By combining a range of schizotypal scales, Bentall and colleagues (1989) have formed the Combined Schizotypal Trait Questionnaire. When factor analysis was carried out in the scores of subjects participated in their study three factors were extracted. These were (i) the positive schizotypy, (ii) the negative schizotypy and (iii) the social anxiety and disorganization factor. As derives from the Crow (1980) distinction of schizophrenia symptomatology, positive schizotypy refers to a predisposition towards positive symptomatology (e.g., delusions and hallucinations) and negative schizotypy to a predisposition towards negative symptoms (e.g., catatonia, anhedonia). The results reported by a number of other studies also indicated quite consistently that schizotypy is a multidimensional construct composed of at least three dimensions. These dimensions were termed as positive schizotypy (e.g., Muntaner et al., 1988; Raine and Allbutt, 1989; Bentall et al., 1989; Venables, 1990; Kelley and Coursey, 1992), negative schizotypy (e.g., Kendler and Hewitt, 1992; Bentall et al, 1989) and Nonconformity (e.g., Muntaner et al., 1988; Raine and Allbutt, 1989; Bentall et al, 1989). The factor namely Social Anxiety/Cognitive Disorganisation (Bentall et al., 1989; Claridge et al., 1996) was not found in these later group of studies.

According to Raine and Allbutt (1989) one of the main reasons for identifying the factors that constitute the schizotypy construct is for making possible its direct comparison with the schizophrenia construct. Recent factor analytic studies on schizophrenia symptomatology (e.g., Liddle, 1987; Liddle and Barnes, 1990; Liddle and Morris, 1991) suggested that schizophrenia might also be a multidimensional construct and therefore direct comparison between people with schizophrenia and schizotypes might be possible.

In the factor analytic study reported by Liddle (1987) it was shown that schizophrenia is associated with three factors which were termed as “Reality Distortion”, “Disorganisation” and “Psychomotor Poverty” each one of them representing an independent schizophrenic syndrome. Comparing these three factors to the results of
schizotypy studies it seems that the factors extracted by these two groups of subjects are very similar (see Bentall et al., 1989; Claridge, 1996). More specifically the factors of Positive Schizotypy, Negative Schizotypy and Social Anxiety/Cognitive Disorganisation extracted by the Bentall and colleagues (1989) study seemed to correspond to Liddle’s (1987) factors of Reality Disortion, Disorganisation and Psychomotor Poverty.

The findings that both schizotypy and schizophrenia are multidimensional and that they are composed by similar factors appears to support the earlier suggestion that both constructs are represented by different points in the same continuum with schizotypal scales measuring an underlying predisposition towards any particular cluster(s) of symptoms.

Identification of the schizotypy construct and its potential to facilitate research in the area without the complications of confounding variables marked the beginning of a new trend in schizophrenia research (see Claridge, 1997). Recent studies aiming to investigate, through experimental paradigms, deficits that are considered to play a contributory role in the aetiology of schizophrenia they often included in their experimental designs groups of high and low schizotypes. The former aims to facilitate the comparison between the performance of people with schizophrenia and the possible impact of confounding variables whereas the later to determine the inherent differences in the performance between psychotics and schizotype free subjects. However, before the value of schizotypy is fully appreciated as a tool of enhancing our understanding of the disorder it is quite important to identify the similarities of schizotypes and people of schizophrenia in their wider functioning rather than simply on their predisposition towards a particular cluster(s) of symptoms.

Although schizotypy as a tool for investigating schizophrenia is a fairly recent one, research in the area has produced a proliferation of studies (e.g., Hall and Habbits, 1996;
Cadenhead et al., 1996). One of the main targets of psychological research into schizophrenia today is to identify the cognitive abnormalities that underlie the clinical picture of the disorder (see McKenna 1994). Predominantly based on highly developed models of normal cognitive functioning (e.g., Broadbent, 1971) within this approach symptoms of schizophrenia are conceptualised in terms of deficits in the individual components of the particular model employed. In order to investigate the deficit that is thought to play a contributory role in the emergence of symptoms research in this area takes the form of developing specific experimental tasks that are considered to be tapping the normal cognitive functioning suggested by the model. Potential differences in the performance of people with schizophrenia and controls in such tasks are interpreted as indicating a deficiency in the stage or process described by the model and considered to be critical for the emergence of schizophrenic symptomatology.

Regardless of the theoretical model they are based on and the deficit they aim to investigate, the majority of studies that incorporate within their design schizotypes, in their more general form seem to support the notion that high schizotypes, as derived by their scores in schizotypy scales, react and perform in experimental tasks similar to their schizophrenic counterparts. As examples to the above we may be referred to a series of studies in which people with schizophrenia were tested on behavioural tasks such as latent inhibition (e.g., Baruch et al., 1988a), reversal learning (Nolan 1968), Kamin blocking effect (e.g., Jones et al, 1992), negative priming (Beech and Claridge, 1987; Beech et al., 1989; Peters et al., 1994) and time estimation and crossover effect (e.g., Rosenbaum, 1988). The results of these studies have shown quite consistently that the performance of high schizotypes resembles to a great extent to that of people with schizophrenia (for examples see Baruch et al., 1988b, Jones et al., 1992, Georgiades, 1993; Steel et al., 2002; Sarkin et al., 2002).

Apart from including and comparing the performance of people with schizophrenia with that of high schizotypes on experimental tasks that potentially tap the underlying deficit common to these two groups of subjects, several neuropsychological studies (e.g., Shallice et al., 1991) have investigated the possible similarities of people with
schizophrenia and schizotypes in their overall cognitive functioning (e.g. Giraldez et al., 2000). Previous findings appear to relate schizophrenia with impaired abstraction (Saykin et al, 1991), language and verbal learning (e.g., Allen et al., 1993), attention as well as with memory deficits (e.g., Gruzelier et al, 1988). These deficits were regarded to reflect involvement of left hemisphere frontal and temporolimbic structures (Gur et al., 1991; Nestor et al., 1993). In a study reported by Voglmaier and colleagues (1997) where the overall cognitive functions of schizotypal personality individuals was investigated it was found that this group of subjects show cognitive deficits in verbal learning, and on abstraction that requires concept formatting and mental flexibility. The results of the Voglmaier and colleagues (1997) study seem to accord well with other studies that indicate consistently that people classified as prone to psychosis show deficits in abstraction (e.g., Kremen et al., 1996). Similarly, in a more recent study investigating cognitive slippage, as derived by scores on the Wisconsin Card Sorting Test in high schizotypes it was found that subjects scoring high on Magical Ideation and Perceptual Aberration Scales are more likely to report cognitive slippage and therefore do not achieve as many categories on the test as the non-schizotypy controls (Gooding et al., 2001). Further support to the similarities between people with schizophrenia and high schizotypes comes from a number of studies which point out that parents of people with schizophrenia (and presumably high schizotypes) demonstrate subtle language (e.g., Docherty et al., 1997), cognitive functioning (e.g., Kremen et al., 1996) and personality (Kendler et al., 1996) characteristics. These characteristics were also found in people with schizophrenia (Docherty, et al., 1998) but not in people who did not have patients with schizophrenia in their families.

The similarities between schizotypes and people with schizophrenia as indicated by recent studies (e.g., Alleman et al., 2000; Shean and Wais, 2000; Cameron and Kwapis, 2000) can be extended to a number of other areas of their overall functioning providing further support to the hypothesised similarities and genetic relationship between schizophrenia and schizotypy.
5.1 Possible Applications of Schizotypy into the Treatment of Schizophrenia

The above findings apart from emphasising the underlying similarities between schizophrenia and schizotypy and provide a way to reduce the methodological complexities associated with schizophrenia research they also point to a route by which to identify with some degree of accuracy as to what really schizophrenia is and what it entails. The potential value of utilising the concept of schizotypy though cannot be restricted to the better understanding of the phenomenology of the disorder or in the accumulation of data that potentially would contribute to the identification of the factors or deficits relevant to the development of the illness.

Without underestimating the importance of such knowledge one cannot ignore that a major problem associated with schizophrenia today is with regard to its treatment. Recent findings that pharmacotherapy is not equally effective to all patients (e.g., Davis and Casper, 1997) and that the addition of psychotherapeutic modalities can only reduce drug resistant psychotic symptoms only to a percentage of patients (for a review see Cormac et al., 2002) it might be possible that the future on the treatment of schizophrenia might lie on the development of treatment modalities that would prevent high risk individuals from experiencing psychotic episodes.

The results of recent studies investigating the role of therapy in schizotypes (see Senst et al., 2002) and in people at the prodromal stage of schizophrenia (e.g., Rosen et al., 2002) were quite encouraging. Also encouraging were the findings reported by Tsuang and colleagues (2002) in which it was indicated that cognitive deficits in people with predisposition towards negative symptomatology (schizotaxia) of schizophrenia can be identified and potentially reversed with low doses of neuroleptic medication. Finally, the findings of Morrison and colleagues (2002) were very optimistic in their suggestion that schizotypal scales as risk indicators can predict psychotic episodes and that psychological intervention in the form of cognitive therapy may facilitate its prevention.
6. Conclusions and suggestions for future research

The results of schizophrenia research often are open to criticism and contradiction due to the inherent difficulties of distinguishing which of the findings reported are specific to the disorder or the outcome of confounding variables. Recently, the renewed scientific interest on the concept of schizotypy (Rado 1953; Mehl, 1962; 1990) and the recognition that schizotypy and schizophrenia might be genetically related provided researchers an alternative route in the investigation of the disorder by employing high schizotype individuals instead of people with schizophrenia. Available evidence indicates that schizotypes are an easier group to recruit due to their higher prevalence in the general population. Moreover development of self-report questionnaires and subsequent factor analytic studies have shown that it is possible to identify high schizotypes who are predisposed not only globally to schizophrenia but also to specific symptoms (e.g., positive and negative symptoms) and to be compared to groups of schizophrenic individuals who experience similar symptomatology (e.g., Bentall et al., 1989; Liddle 1987).

The hypothesis that schizotypes share the same personality traits and genetic liability for experiencing psychotic breakdowns led many researchers to investigate whether such a possibility implies similarities in the overall functioning between the two groups. A large number of studies reported during the past two decades, seem to indicate quite consistently that high schizotypes resemble people with schizophrenia on a number of areas of their overall functioning spanning from cognitive and behavioural to more subtle characteristics such as mental imagery (e.g., Alleman et al., 2000). Finally, recent attempts that examined the possibility of utilising the concept of schizotypy in order to provide preventative treatments to high schizotypes indicated that research in this area might have an important role to play in the treatment of schizophrenia both in the pharmacological (e.g., Tsuang et al., 2002) and in the psychotherapeutic (e.g., Morrison et al., 2002) domains.

On the basis of the information reviewed one cannot avoid feeling tempted to suggest that schizophrenia research as it is known has to be abandoned and re-direct its focus towards
investigating and understanding the disorder via schizotypy. However, looking at the core of schizotypy models and research, the assumption made is that schizotypes and people with schizophrenia share the same underlying biological construct. Does this imply that all aspects of the disorder derive from a biological deficit and schizotypy can facilitate the investigation of this deficit without the presence of confounding variables? Modern psychological studies (e.g., Bentall, 1990; Birchwood and Chadwick, 1997; Chadwick and Birchwood, 1994) indicate that the psychological element in the development of schizophrenic symptomatology is quite strong, whereas a number of researchers emphasise that often these symptoms emerge as a mechanism to protecting the individual’s vulnerable self esteem (e.g., Bentall, et al., 1991). Current schizotypy research does not show how this characteristic is linked biologically to schizophrenia nor does it explain whether this group of people developed low self-esteem prior or after the onset of the disorder. Another issue that is worth raising here is that research suggests that only 10% of schizotypes develop schizophrenia. What are the contributory factors that differentiates that 10% from the others who never experience psychotic episodes? It seems that current schizotypy research does not take into account the role of environmental factors and stressors in the development of the disorder nor does it consider that there might be significant individual differences between those who remained high schizotypes all their lives and those who go on to develop schizophrenia.

Finally the issue of interest here is schizophrenia as a highly disabling illness, the phenomenology and the impact that the experience of the disorder has on people. Schizotypy research however, does not seem to concentrate on these aspects of the disorder. Instead, it focuses on the investigation of “healthy individuals” who although show a tendency towards developing the disorder due to a presumed biological predisposition, they have never surpass the threshold that separates sanity from insanity.

As a concluding remark it is quite important to emphasise that considering schizotypy as an alternative approach of investigating schizophrenia is not a straight forward process. Undoubtedly this approach holds several advantages and current research seems to indicate consistently that there are similarities between schizophrenia and schizotypy.
However, taking into consideration the many aspects of schizophrenia that cannot be encompassed or have not been investigated as yet it seems that schizotypy as a tool is very valuable in facilitating our better understanding of the disorder but not adequate enough to replace research using people with schizophrenia.
7. References


Kety, S.S., Rosenthal, D., Wender, P.H., Schulsinger, F., and Jacobson, B. (1975): Mental illness in the biological and adoptive families of adopted individuals who have become


SECTION III: Clinical Dossier
ABSTRACT

Background: Cognitive Behavioural Therapy can be regarded today as probably the most effective treatment for Post Traumatic Stress Disorder. Research however, into the efficacy of any type of psychotherapy to specific psychological disorders seems to be plagued by a number of methodological issues. These make the utilisation and application of research findings into clinical practice problematic. A possible way to improve clinical effectiveness is by developing evidence-based practice where the efficacy of psychotherapeutic interventions is constantly monitored.

Method: The Revised Impact of Events Scale (IES), the Beck Anxiety Inventory (BAI) and the Beck Depression Inventory (BDI) were completed by 40 participants (11 constituting the waiting list control group and 29 the clinical group). Both groups were reassessed on the same symptom measures 12 weeks later. In the intervening period the clinical group received 12 sessions of Cognitive Behaviour Therapy whereas the waiting list control group was offered therapy after their re-assessment.

Results: There was clear indication that Cognitive Behavioural Therapy contributes significantly to the reduction of PTSD symptomatology of people who received therapy quite early after their experience of trauma.

Conclusions: Cognitive restructuring appears to be an effective form of treatment for people who were seen for therapy early after their traumatic experience. In instances of an increased time gap between trauma and onset of therapy this seems to reduce tolerance for anxiety and by extent the effectiveness of exposure treatment to Post Traumatic Stress Disorder.
1. INTRODUCTION

People using mental health services usually experience a range of psychological problems. In order to meet the needs of these clients it is necessary for the clinician to employ forms of psychological therapy that are easily accessible and appropriate to the type of problems they experience. According to Kazdin (1996), when psychotherapy began to emerge as a formal form of intervention within the medical profession in the early 1900’s, research about the efficacy of these forms of treatment was not a priority and the only way to demonstrate their effectiveness was through the publication of study cases. Today, the several hundreds of psychotherapeutic approaches available (for a review see Kazdin, 1986), the great variability of problems experienced by patients and the financial implications associated with the delivery of psychotherapy point out that decisions with regard to therapy need to be guided by research based findings about the efficacy of specific psychotherapeutic approaches to particular problems rather than on the individual clinician’s view, training or preference. Elaborating on the excessive financial cost of this type of service and the vast amount of research generated during the past fifty years (Shapiro, 1996) about the effectiveness of psychotherapy Kazdin (1996) argues that “...it is surprising and regrettable that the demand for greater accountability in services has peaked only now” (p.6).

Although taking clinical decisions about the employment of particular types of therapy to specific problems on the basis of research findings poses several advantages, this approach is not without its limitations. According to Roth and Fonagy (1996), a major problem associated with research into the efficacy of psychotherapeutic treatments is that the methodology employed in these studies quite often constitutes a compromise that reflects the interests, the intentions and the resources available to the researcher. Moreover, the usual practice in studies to implement therapies where the influence of confounding variables is kept to the minimum often has direct impact on the outcome of the therapy making it extremely difficult to draw any conclusions about the effectiveness of these therapies in everyday clinical practice. These problems, which are only a small sample of the many difficulties encountered by clinicians in transferring skills and
suggestions deriving from research into their day to day interaction with patients, point out the great gap that separates clinical practice from research-based knowledge.

The complexities associated with the interpretation of these findings are acknowledged in the review of the National Health Service Psychotherapy Services in England (1996) that emphasises the need to improve clinical effectiveness of NHS services by developing evidence-based practice where the efficacy of psychotherapeutic interventions on individuals or groups of patients is constantly monitored by ongoing clinical audit and reviews using where possible outcome measures. It is anticipated that the employment of this strategy not only would enhance the clinicians’ understanding of the efficacy of psychotherapeutic approaches to the treatment of specific disorders but also it will facilitate direct comparisons with controlled clinical trials. However, the number of difficulties associated with the ongoing evaluation and investigation of therapy makes it extremely difficult to adopt this strategy as part of the everyday clinical practice. The shortage of clinical psychologists, the great variability of problems experienced by patients, the high rate of attrition and the spontaneous remission of some patients prior to the end of therapy are only few of the obstacles associated with this approach. To complicate matters even more, the different training experiences of clinical psychologists and the need to keep waiting lists down by usually seeing patients on the basis of the date of referral rather than by problem makes it almost impossible to incorporate within the ongoing activity of a department the investigation of therapies without placing additional burden to the already limited resources.

The Adult Acute Section of Barnet’s Healthcare NHS Trust Clinical Psychology Department where the research reported in sections 2 and 3 was carried out, is not immune to the above problems. The Adult Acute Clinical Psychology Service which at present employs 10.1 Whole Time Equivalent (WTE) Clinical Psychologists, covers the areas of Barnet, Edgware, Borrhamwood and Potters Bar with an average population of 240,000 people. The advantage of this section of the department however, is that in addition to its full-time members of staff (7.5 WTE) it also attracts a number of clinical psychologists who
although for the greater part of their time they work in other sections, they devote some sessions per week to seeing patients in the Adult Acute Clinical Psychology Section. Due to the limited number of sessions spent within this section, these "part-time" clinical psychologists are encouraged to form specialist clinics where they see for therapy clients with specific diagnoses as defined by the diagnostic tools. The advantage of the approach where the same clinical psychologist sees all clients from a specific diagnostic category is that it facilitates research into the efficacy of the psychological therapies employed within the context of clinical practice as proposed by the review of the National Health Service Psychotherapy Services in England (1996).

The main aim of the research study reported in the next section was to examine the efficacy of Cognitive Behavioural Therapy (CBT) in the treatment of Post-Traumatic Stress Disorder (PTSD) in the course of routine clinical practice. Since the treatment package employed in this study does not derive from any particular theoretical framework but it eclectically draws on a number of theoretical suggestions within the Cognitive Behavioural model the first part of the study presents a general review of the background and treatment of the disorder. The methodology and the presentation of the results follow the general introduction whereas the last section discusses these findings in relation to implications they might have in clinical practice and in future research.

1.1 The Concept of Post-Traumatic Stress Disorder

1.1.1 Background to Post-Traumatic Stress Disorder

Descriptions of human responses to traumatic and life-threatening experiences can be found in abundance in sociohistorical and literary writings from the beginning of recorded history (e.g., Dally, 1983; Trimble, 1985; Joseph et al., 1997). In recent times, the psychological difficulties experienced by many soldiers that fought in the Vietnam War after they had returned to America brought to the attention of health professionals the detrimental effects that exposure to traumatic and life threatening events can have on
people's lives (Shalev, 2000). The realisation that this type of experiences could lead to
the emergence of a specific cluster of symptoms that potentially could have horrendous
impact on the individuals' personality was possibly the most important factor that led to
the conceptualization of the new diagnostic category of PTSD. The wealth of data
derived from work with Vietnam Veterans and the lack of a diagnostic framework by
which to describe those findings, pointed out the clinical need to develop a system that
would provide a common language amongst professionals to describe these phenomena
and to facilitate the scientific generalisation that is so important for research and
diagnostic purposes.

1.1.2 The Diagnosis and Prevalence of Post-Traumatic Stress Disorder
In response to the need for a tool that would facilitate both diagnosis and research, PTSD
was included for the first time as a separate diagnostic entry on the third edition of the
Diagnostic and Statistical Manual for Mental Disorders (DSM III; APA 1980) whereas
some ten years later the disorder also constituted a distinct entry in the tenth edition of the
International Classification of Diseases (ICD-10; WHO, 1992). In order to emphasise the
importance and the contributory role of the work with Vietnam veterans in our current
understanding of the disorder, Yule (1999) pointed out that PTSD syndrome can be
regarded as "... one of the legacies [of a] futile conflict" (p. 2).

Within the DSM III (APA, 1980) criteria, the primary requirement for an individual to be
considered for PTSD diagnosis was the "existence of a recognisable stressor that would
evoke significant distress to almost everyone". This criterion however, has not been a
stable pre-requisite for the diagnosis of PTSD as in subsequent editions of DSM this has
been modified to reflect the increased knowledge of the disorder deriving from
continuous research in the area (Shalev, 2000). In the DSM III-R (APA, 1987) the
primary criterion was the experience of "a psychologically traumatic event that is
generally outside the range of human experiences and that would be markedly distressing
to almost anyone" (APA, 1987) whereas in the DSM IV (APA, 1995) and DSM IV-TR
APA, 2000), the necessary criteria for the diagnosis of the disorder were elaborated even more to incorporate the concept of vicarious experiences.

In addition to the experiencing of a traumatic event, the symptoms considered to be stemming from these experiences are grouped into three distinct classes. Although there are slight differences in the description of PTSD symptoms between the different editions of the DSM, at a more general level they all point out to the presence of three clusters of symptoms, (a) the persistent re-experience of the traumatic event, (b) the persistent avoidance of stimuli associated with the trauma and numbing of general responsiveness, and (c) the persistent symptoms of increased arousal. For a diagnosis of PTSD to be made the cluster of symptoms described above need to be present for a period of at least one month. Moreover, the revised criteria for PTSD described in the DSM IV (APA, 1995) distinguish between different types of PTSD not only in terms of the duration of symptomatology but also with regard to the onset of these symptoms.

The ICD-10 (WHO, 1990) and the DSM IV (1995) as diagnostic systems share a number of common features such as the identification of a life threatening event, the re-experiencing of the trauma and avoidance of stimuli that remind the trauma. In addition to their similarities the two systems also differ in a number of ways. Most important of which is that in the ICD-10 "emotional numbing" although not an essential requirement for the diagnosis of PTSD is regarded to constitute an important component as it accompanies the disorder frequently (Yule 1999).

Regardless of the system used to diagnose the disorder, epidemiological studies investigating the "incidence" and "prevalence" of PTSD in the western world have produced a great variability of results (Keane et al, 2000). These findings indicate that the frequency of new cases (incidence) and the extent by which traumatic experiences lead to a pathological reaction at any given time (prevalence) is determined by the type of trauma investigated and the number of people exposed to this trauma (Foia and Rothbaum, 1998). Commenting on the heterogeneity of traumatic events that potentially
could lead to symptoms of PTSD, O'Donahue and Elliot (1992) has suggested that traumatic experiences should be sub-grouped into several categories (e.g., war, sexual abuse, natural disaster, accident etc.) and that the different rates of prevalence should be estimated within each category. On the basis of the above, Tomb (1994) has suggested that with serious trauma, the lifetime prevalence rate for PTSD is approximately 30% whereas the current prevalence after several years is below 10%. Tomb's (1994) statistical proposition appears to accord well with Green's (1994) report, which also suggests similar figures.

1.2 The Factors Influencing the Development and Course of Post-Traumatic Stress Disorder
The general consensus of researchers working in the area of PTSD is that the main symptoms of PTSD (re-experiencing and numbing) develop as a result of the person's system being overloaded. It is proposed that following exposure to a traumatic event, individuals try to integrate the experience into their life. However, due to the distressing nature of what they are required to come to terms with, individuals begin to experience symptoms of avoidance and numbness which disrupts the process of integration and maintains the symptomatology.

According to some researchers symptoms of avoidance are adaptive in the short term and serve as a defense of denial (Titchener, 1985) whereas perseverance of this type of response could develop into a chronic pattern that would prevent the process of integrating the traumatic experience into the individuals' lives. The suggestion that symptoms of avoidance are adaptive in the short term and that their presence do not necessarily predict the course of development of the disorder seem to be supported by McFarlene's (1992) findings which indicate that the levels of intrusive symptoms are more likely to predict the development of PTSD than symptoms of avoidance. In contrast to McFarlene's (1992) suggestion Shalev (1992) suggests that the development of symptoms of intrusion immediately after exposure to trauma could be also considered as
normal reaction to trauma and therefore not predictive of subsequent development of PTSD. According to the same researcher, the predictive value of these sets of symptoms increases only when they persist after a period of time as such a response may be considered as indicative of a continued failure of the traumatic event to be integrated into the person's experiences.

On the basis of the above it seems that for a large number of people exposure to a traumatic event is followed by symptoms of avoidance and intrusion which characterise the development of acute PTSD. For most of these individuals, the symptoms will serve as short term mechanisms of self-defense and their intensity will decrease with the progression of the process of integrating the trauma within their experiences (e.g., Foa et al., 1992; Foa and Kozak, 1986). The failure to process and integrate the trauma into the person’s experiences lead to a more chronic pattern of PTSD symptomatology and consequently the difficulties experienced by these individuals are more severe (e.g., Dangleish, 1999). This suggestion although useful in assisting the distinction between acute and chronic PTSD and how these phases of the disorder are linked to the processing and integration of the trauma into the individuals' experiences, this explanation by itself cannot be regarded as being complete. For example, the proposal does not account for the factors influencing the development of these two different patterns of response nor does it explain why for some people exposure to traumatic events is not followed by any of the typical symptoms of PTSD.

In the search for the possible causes influencing the development of PTSD symptoms several factors were investigated including age, gender and pre-morbid personality. Research studies investigating the above factors did not reveal that age or gender are consistently related to the development of PTSD symptomatology (for a review see Gibbs, 1989). Similar conflicting findings were also reported in studies investigating the predictive value of previous psychopathology in the development of PTSD (e.g., Kilpatrick et al., 1985, Blanchard et al., 1996).
Studies where the predictive value of the attributional style of people suffering from PTSD was investigated indicated that perceived control during the traumatic situation is related to reduce psychological difficulties (Gibbs, 1989) whereas perceived helplessness during such events is more likely to be associated with increased symptoms of intrusion (Joseph et al., 1994; Joseph et al., 1997). Moreover, there is evidence to suggest that great internal attribution for traumatic events such as self blame (Janoff-Bulman, 1985; Joseph et al., 1991) and negative attitudes towards emotional responses (Joseph et al., 1996) are associated with poor psychological outcome and predict the severity of PTSD symptomatology.

Finally, another factor that has also been considered to play a significant role in the development and maintenance of PTSD symptomatology is the duration and the frequency of exposure to trauma. According to some researchers, people who have experienced frequent and/or prolonged traumatic events in their life (often referred as Type II trauma) such as incest and ongoing sexual, physical or emotional abuse are more likely to experience more severe symptoms of PTSD (see Padesky 1995).

1.3 Assessing Post-Traumatic Stress Disorder

As indicated in section 1.1.2 the rate of prevalence for traumatic experiences and the impact that these events can have on individuals' overall life functioning vary from moderate to severe Carlson (1997) depending on the population sampled and the methods employed in investigating these experiences (e.g., Green, 1994; Keane et al. 2000). Taking into consideration the variability of the symptomatology presented by PTSD sufferers following exposure to a traumatic event, it is quite important both for research and therapeutic purposes to be able to assess with some degree of accuracy the clusters as well as the severity of symptoms experienced by patients. Since the inclusion of PTSD in the DSM III (APA, 1980) as a separate diagnostic entity (in addition to clinical assessment interviews based on the guidelines offered by the diagnostic system) several attempts were made to identify patterns of responses relevant to PTSD stemming either
from the utilisation of existing psychometric tests or by the development and standardisation of an array of new ones (Joseph et al., 1997). However, the wide range of psychological reactions experienced by people exposed to trauma and the impact that potentially these events could have on the individuals’ social and cognitive functioning probably constituted the most important problem in the development of these tools. But despite the limitations derived from the lack of an “all inclusive” test that can account for all aspects of the disorder, the contribution made by the majority of these tools in enhancing our understanding of the disorder both in research and in clinical settings has been quite significant.

The most widely used tools in the assessment of PTSD are grouped into two main categories, the structured interviews and the self report scales (for a detailed discussion of these tools see Wilson and Keane, 1997). In the structured interviews the items derive predominantly from the clusters of symptoms that are included in the diagnostic systems and they measure the extent and the severity of the symptomatology. Two of the most well known tools that have been specifically developed for the assessment of PTSD are the PTSD Interview (Watson et al., 1991) and the Clinician’s Post-Traumatic Stress Disorder Scale (Blake et al., 1990).

With regard to self-report scales probably the most well known is the Horowitz’s and colleagues (1979) Impact of Events Scale (IES). This scale assesses within its 15 items the two main clusters of symptoms that are regarded to constitute the cornerstone of the disorder: (a) the intrusively experienced ideas, thoughts and images and (b) the avoidance of situations feelings or ideas associated with the traumatic event. The items included in the inventory derived from the most common statements made by people who have experienced recent life threatening and/or traumatic events. Horowitz’s and his colleagues (1979) reported that the scale poses satisfactory internal and test re-test reliability and this was subsequently supported by the findings of other studies (e.g., Zilberg et al., 1982; Joseph et al., 1993). Additional advantages of this scale is that it correlates with other PTSD measures (e.g., Schlenger et al., 1992), it has been translated
to a number of languages and used in several countries (e.g., Schwarzwald et al., 1987; Brom et al., 1986) but most importantly the IES can be used for any traumatic life-event making data across studies comparable (for a review see Weiss and Marmar, 1996).

1.4 Treatment of Post-Traumatic Stress Disorder

Although the general consensus amongst theoreticians working in the area is that PTSD arises as a result of information overload and mediated by a number of different factors (see section 1.2), there has never been an agreed model by which to account for the way by which this overload of information occurs. Therefore, the proposed process has been formulated within a number of different theoretical frameworks spanning from biological (e.g., Kolb, 1987; Jones and Barlow, 1990; Van der Kolk, 1987) and psychoanalytic (see Horowitz, 1979; 1986) to Cognitive Behavioural (e.g., Lyons and Keane, 1989) and the more recent model of Eye Movement Desensitisation and Reprocessing (EMDR; Shapiro, 1989) each of these proposing their corresponding therapeutic approach.

These formulations and their associated therapeutic approaches although important in the provision of interesting insights about the nature of the disorder (Dangleish, 1999) the evidence available with regard to the efficacy of these treatment models indicate a variable degree of success (for a review see Meichenbaum, 1994; Wilson et al., 2001). This variability of success seems to be emphasised by the results of Green’s (1994) review study which suggested that fifty per cent of individuals who received treatment for their PTSD symptomatology continued to meet the full criteria for PTSD diagnosis at the end of therapy. More recent research findings suggest consistently that from all available psychotherapeutic models Cognitive Behavioural Therapy can be considered as the most developed approach (e.g., Horowitz et al., 1980; Foa et al., 1992; Foa and Jaycox, 1999; Brewin, 1989; 1996) that provides the most “... coherent and successful attempts at accounting for the range of factors implicated in PTSD” (Dangleish, 1999 p.194). This view seems to accord well with Roth and Fonagy’s (1996) review findings where they concluded that Cognitive Behavioural Therapy is probably the most effective
psychological therapy for PTSD and that on the basis of available evidence (e.g., Thompson et al., 1995) it may be regarded as the treatment of choice for PTSD (for detailed review of theoretical models see Horowitz, 1973; 1976; 1979; 1986; Epstein, 1991; Janoff-Bulman, 1992; Brewin et al., 1996; Blanchard et al., 1996).

1.4.1 Cognitive Behavioural Therapy and Post-Traumatic Stress Disorder

Cognitive behavioural interventions which include a variety of procedures such as exposure, anxiety management, cognitive therapy and/or their combinations have been shown repeatedly to be superior to controls as well as to other forms of therapy (e.g., Meichenbaum, 1994). Exposure is regarded as highly effective in the treatment of a variety of anxiety disorders including phobia, panic and obsessive compulsive disorder (Hawton et al, 1989). Within the context of PTSD several exposure techniques differing along the dimensions of type (imaginal vs in vivo), length (short vs long) and level of arousal (high vs low) were used for the treatment of the disorder (Richards and Rose, 1991; Zoellner, et al., 2001).

The development and successful application of Cognitive therapy (e.g., Beck, 1976) in the treatment of a number of psychological disorders led researchers and clinicians working in the field to propose the potential role played by cognitive processes in the development and maintenance of PTSD symptomatology. Within the framework of the cognitive approach patients are directed towards identifying the negative automatic thoughts and beliefs associated with the traumatic experience and are helped to develop a more appropriate view of the event in relation to themselves and the world. Studies investigating the efficacy of this treatment modality to PTSD although fairly recent compared to the investigation of the approach to other psychological disorders (e.g., Hollon et al., 1993; Chambless and Gillis, 1993; Clark, 1991) seem to indicate that cognitive techniques such as modifying negative automatic thoughts and dysfunctional assumptions are quite effective in the treatment of PTSD (e.g., Resick and Schnick, 1993, Thrasher et al., 1996).
1.5 Aims and Hypotheses of Clinical Study

According to Roth and Fonagy (1996), the majority of studies investigating the efficacy of psychotherapeutic interventions in the treatment of psychological disorders are characterised by a number of serious methodological problems (see section 1). These problems make the transfer of research findings into clinical practice and their comparison with other treatment modalities extremely difficult. A possible way to resolve these problems is by developing evidence-based practice where research about the efficacy of psychological interventions to specific disorders is incorporated into the clinician's ongoing clinical activity. Taking into consideration that Cognitive Behavioural Therapy is regarded as one of the most effective psychological therapies for PTSD (Dangleish, 1999; Roth and Fonagy, 1996), the main aim of the present study is to investigate the efficacy of a Cognitive Behavioural Therapy package (for description see section 2.5) in a routine clinical practice through the following hypotheses:

(i) On the waiting list control group, there will be no significant differences on any of the symptom measures between initial assessment and on assessment at a twelve week interval;

(ii) On the clinical group there will be significant differences in all symptom measures between initial assessment and on assessment after twelve sessions of Cognitive Behavioural Therapy. It is hypothesised that on discharge the scores of this group of subjects and hence their symptomatology will be significantly lower than their scores on initial assessment.
2. METHOD

2.1 Subjects

Two groups of subjects suffering from PTSD as described by the DSM IV (APA, 1995) were employed in this study. The first group constituted the waiting list control group and the second the clinical group. All subjects were seen for assessment and therapy by the present author. The demographic details of each group are described below:

2.1.1 Waiting List Control Group

A total of 11 subjects (4 males and 7 females) were assessed in this group. All subjects were referred to the Adult Clinical Psychology Services of Barnet Psychiatric Unit for assessment by the Barnet NHS Trust’s Psychiatrists (8 subjects: 3 males and 5 females) and the Local General Practitioners (3 subjects: 1 male and 2 females). All participants had experienced type I trauma (single traumatic event) and met the criteria for PTSD as described by the DSM IV (APA, 1995). None of the subjects were on medication at the time of assessment and no one of them had received previously psychological therapy for their current difficulties. All subjects were native English speakers and three of the subjects assessed were unemployed, one was a student, two were housewives and the rest employed in various occupations.

The mean age of the eleven subjects was 37.73 years, ranging from 20 to 53. The time gap between exposure to the trauma and assessment was calculated in months and the mean time gap for all 11 subjects was 24.18 months, ranging from 2 to 44 months. Finally, the source of the trauma of these subjects was as follows: 4 subjects were involved in a car accident, three were victims of rape, two had experienced abuse at their place of work, one was physically assaulted and one subject was robbed (see table 2.1 for summary of demographic data).
Table 2.1  Means and standard deviations (in parenthesis) of demographic data of waiting list control group.

<p>| | | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>AGE</strong></td>
<td>37.73 (09.78)</td>
<td>n=11</td>
</tr>
<tr>
<td><strong>TYPE OF TRAUMA</strong></td>
<td>Accident 4</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Rape 3</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Physical Assaults 1</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Robbery 1</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Abuse 2</td>
<td></td>
</tr>
<tr>
<td><strong>TIME GAP BETWEEN TRAUMA AND ASSESSMENT (IN MONTHS)</strong></td>
<td>24.18 (16.12)</td>
<td>n=11</td>
</tr>
<tr>
<td><strong>SEX</strong></td>
<td>MALES 04</td>
<td></td>
</tr>
<tr>
<td></td>
<td>FEMALES 07</td>
<td></td>
</tr>
</tbody>
</table>

2.1.2 Clinical Group

In the clinical group, a total of 41 subjects (20 males and 21 females) were employed. Again, all subjects were referred to the Adult Clinical Psychology Services of Barnet Psychiatric Unit for assessment by the Barnet NHS Trust’s Psychiatrists (31 subjects: 17 males and 14 females) and the Local General Practitioners (10 subjects: 3 males and 7 females). All the subjects had experienced type I trauma (single traumatic event) and met the criteria for PTSD as described by the DSM IV (APA, 1995). None of the subjects were on medication at the time of assessment and no one of them had received psychological therapy for their PTSD symptomatology.

Thirty-three of the subjects were native English speakers and eight had adequate command of the English language for the purposes of assessment and therapy. Nine of the subjects assessed were unemployed, three were housewives and the rest employed in various occupations. Their mean age was 36.46 years ranging from 18 to 55 and the time gap between trauma and assessment was 21.78 months ranging from 2 to 65 months. The source of traumatic experiences was as follows: 22 subjects were involved in a car
accident, eight had been raped, two had experienced physical and/or emotional abuse, seven were assaulted and two subjects were robbed.

Finally, from the 41 subjects who initially agreed to receive therapy, 10 had dropped out of therapy within the first six weeks of treatment and two subjects between the sixth and the twelfth session. Therefore only the data obtained by 29 participants were used for statistical comparisons of pre and post-treatment. Table 2.2 shows the demographic data of subjects employed in this group of the present study.

Table 2.2  Means and standard deviations (in parenthesis) of demographic data of clinical group.

| AGE | 36.46 (09.80) n=41 |
| TYPE OF TRAUMA | |
| Accident | 22 |
| Rape | 08 |
| Assaults | 07 |
| Robbery | 02 |
| Abuse | 02 |
| TIME GAP BETWEEN TRAUMA AND ASSESSMENT (IN MONTHS) | 21.78 (15.49) n=41 |
| SEX | MALES | 20 |
| FEMALES | 21 |

2.2  Symptom Measures
Subjects on the waiting list and on the clinical groups were asked on the initial assessment session and twelve weeks later to complete the following three symptom measures:

**Impact of Events Scale (IES):** This is a 15-item scale (see appendix 1) that has been specifically designed to measure the effects of traumatic events. It is comprised of two sub-scales measuring the levels of intrusion (I) and avoidance (A), the two major sets of
symptoms relevant to PTSD. Together these two subscales give a total IES score that is a useful indicator of the overall difficulties experienced by subjects following exposure to a traumatic event. The scale was originally developed by Horowitz and colleagues (1979) and was normalised on a 66 subjects sample. The mean score for the Intrusive sub-scale is 21.4 (s.d. 9.6) ranging from 0 to 35, and for the Avoidance sub-scale the mean score is 18.2 (s.d. 10.8) ranging from 0-38. The total average score for men is 34.5 and for women 42.1.

**Beck Anxiety Inventory (BAI):** This is a 21-item scale (see appendix 2) that was developed by Beck and Steer (1990) to measure the severity of anxiety in adults and adolescents. The items included in this scale were drawn from three earlier self-report instruments, the Anxiety Check List, the PDR Check List and the Situational Anxiety Check List that measured the various aspects of anxiety. The inventory, which has been normalised in a sample of 1,086 (456 men and 630 women) subjects consists of 21 descriptive statements relating to a 4-point scale. The obtained total score gives an indication of the severity of anxiety in the following way: scores from 0 to 9 represent normal levels of anxiety, from 10 to 18 mid-moderate levels of anxiety, from 19-29 moderate to severe levels of anxiety and scores from 30 to 63 severe levels of anxiety.

**Beck Depression Inventory (BDI):** This is a 21-item instrument (see appendix 3) that was developed by Beck and his colleagues (1979) to assess the severity of depression in adults and adolescents. Within this 21-item inventory, subjects are assessed on a number of areas relevant to depression spanning from irritability to suicidal ideas to fatigability and loss of appetite and libido. The psychometric properties of the scale have been investigated by a number of studies (e.g., Beck and Steer, 1987) and the responses selected are related to a 3-point scale. The total score obtained by the subject gives an indication of the severity of symptoms of depression experienced. Scores from 0 to 9 represent normal range of depression, from 10 to 18 mid-moderate, from 19-29 moderate to severe levels of depression and scores from 30 to 63 severe levels of depression.
2.3 Design
A within subject design was adopted in this study. All subjects regardless of the group they were allocated to were asked to complete the above three symptom measures on initial assessment and twelve weeks later. Ethical approval was granted for this project as part of a wider project to be completed by three members of the department.

2.4 Procedure
All subjects were seen for assessment and therapy individually. They were all informed of the research protocol on their initial assessment appointment and subsequently, if they agreed to participate in the study, they were asked to sign a consent form (see appendix 4). Subjects who refused to participate in the study were seen for therapy as planned but their questionnaire data were not used for statistical analysis. The procedure followed for subjects on the clinical group was different than the one followed for the waiting list group. These procedures are described separately below:

**Waiting List Control Group:** The first twelve subjects referred to the department for assessment and treatment were assigned to the waiting list control group and within a month from the date referred were invited to the department for an initial assessment. This process was in line with the practice of the Clinical Psychology Department where all referrals are seen within a month for an assessment and subsequently placed on the waiting list. During initial assessment subjects who fulfilled the criteria for the diagnosis of PTSD as described in DSM IV (APA, 1995) and agreed to participate in the study were asked to complete the set of questionnaires mentioned above. At the end of the session subjects were informed that their names were going to be placed on the waiting list and that a therapy appointment was to be offered to them in twelve weeks. On their first therapy appointment subjects were asked to complete again the same set of questionnaires.

**Clinical Group:** Subsequent subjects who were referred to the department for PTSD assessment and therapy were also invited within a month of their referral for an initial
assessments. Patients who fulfilled the criteria for PTSD diagnosis and agreed to participate in the study were asked to complete the same set of questionnaires completed by the waiting list control group. At the end of the assessment session they were offered their first of twelve therapy appointments starting a week later. Subjects were seen on a weekly basis for twelve weeks and on the twelfth week, which was also the discharge date they were asked to complete again the same set of questionnaires.

2.5 Description of Therapy

The first therapy session started with the psychoeducation component of the treatment package. In this part of the session clients were offered the opportunity to learn more about PTSD, the associated symptomatology, how it develops and to discuss in some length the reasons as to why these symptoms should be considered as normal reactions to their traumatic experiences and not as indicative of mental illness. Moreover the therapist provided the clients with an outline of the treatment programme they were going to follow and the rationale for each of the components included in the programme. Subsequent to that, clients were assisted to learn to rate their levels of anxiety based on aspects or instances of their traumatic experiences and record the different levels of anxiety on the provided record (see appendix 5).

The second session began with discussing briefly the previous session and then, in collaboration with the therapist, clients drew a hierarchical list of situations that they avoided as a result of their traumatic experience. Finally, clients were asked to rate their level of anxiety record on the provided record (see appendix 6) and were introduced to “imaginal exposure”. In this part of the session clients were instructed to sit as comfortably as they possibly could and to start “recounting the event in the first person and in the present tense as though the incident was happening during the session”. The recounting of the event was taped and the involvement of the therapist during imaginal exposure was minimal apart from instances where the clients obviously distressed from exposure reported difficulties in holding certain images/memories of the event. In such instances the therapist intervened to
encourage them to hold on to those images. At the end of the recounting, clients were asked to rate and record their levels of anxiety again. The remaining few minutes before the end of the session the emotions and the difficulties experienced by the clients during exposure were discussed with the therapist. At the end of the second session clients were asked, as homework assignment, to listen to the exposure part of the session (from the tape) at least every other day until their next session. Also they were provided with a record sheet (see appendix 7) and were asked to rate their levels of anxiety prior to and after listening to the tape.

After reviewing the homework assignment and discussing the possible difficulties encountered by the clients in carrying out the assignment the third session began, just like in the second session, with clients rating and recording their levels of anxiety and recounting of the event. The expectation at this stage was that as the clients continued to recount and stimulate the memory of the trauma, they would have been able to recall more information about the event. At the end of imaginal exposure and after they had recorded their levels of anxiety, in the course of discussing their emotions associated with the memory of the event, clients were introduced to identifying any negative automatic thoughts that they might have experienced. By employing cognitive therapy techniques (e.g., challenging and modifying the automatic thoughts) they were helped to look for alternative explanations and learn how to modify those thoughts (cognitive restructuring). In order to be assisted with this process, clients were furnished with a set of diaries where they were encouraged to use between the session in recording and modifying their negative automatic thoughts (see appendix 8). Also they were asked, as a homework assignment, to continue to listen to the tape every other day in between the sessions. For the 4th, 5th and 6th session the procedure employed was identical to the one followed on the 3rd session.

In the seventh session, and while imaginal exposure and cognitive restructuring continued, clients were introduced to in vivo exposure. By using the hierarchical list constructed at the beginning of the treatment clients were encouraged to begin approaching the situations listed there by starting from somewhere in the middle of the list. In this stage of the treatment, the
clients were encouraged while approaching these situations to utilise the cognitive skills (identifying and challenging negative automatic thoughts) developed earlier in the programme. Any difficulties encountered by clients in approaching such situations were discussed in subsequent sessions and on occasions it was possible for the therapist to accompany the clients in approaching some of those situations. The process of imaginal exposure was usually stopped at the ninth session when by that time no more new information was added to their recounting. Between the ninth and the twelfth session the client in addition to continuing with the cognitive work was also prepared for discharge. Finally, on the twelfth session all clients were asked to complete the same set of questionnaires they had completed on initial assessment.
3. RESULTS
Data were analysed using SPSS for Windows (9.0 Release) software. All significance levels are two tailed.

3.1 Waiting List Control Group
The principle outcome measures in this part of the study were the symptoms of patients as derived by their scores on the Impact of Events Scale (Avoidance, Intrusion and Total Score), the Beck Anxiety Inventory and Beck Depression Inventory on initial assessment and twelve weeks later. As shown on figure 1 and on table 3.1 statistical analysis using a related t-test did not reveal any significant differences on any of the three measures between initial assessment and twelve week re-assessment (IES (T): t=1.54, d.f.=10, p>0.05; IES(A): t=1.07, d.f.=10, p>0.05; IES(I): t=1.60, d.f.=10, p>0.05; BAI: =2.07, d.f.=10, p>0.05; BDI: t=1.76, d.f.=10, p>0.05).

![Figure 1](image_url)

Scores on all symptom measures of waiting list control group on initial assessment and on assessment after twelve weeks.
Table 3.1  Means and standard deviations (in parenthesis) of waiting list control group scores on IES (T), IES (A), IES (I), BAI and BDI.

<table>
<thead>
<tr>
<th>Measures¹</th>
<th>Initial Assessment N=11</th>
<th>Assessment on 12 Weeks N=11</th>
<th>Significance²</th>
</tr>
</thead>
<tbody>
<tr>
<td>IES (T)</td>
<td>35.36 (4.34)</td>
<td>36.73 (5.59)</td>
<td>ns</td>
</tr>
<tr>
<td>IES (A)</td>
<td>19.54 (3.93)</td>
<td>20.45 (4.27)</td>
<td>ns</td>
</tr>
<tr>
<td>IES (I)</td>
<td>15.82 (3.40)</td>
<td>16.36 (3.93)</td>
<td>ns</td>
</tr>
<tr>
<td>BAI</td>
<td>22.80 (5.49)</td>
<td>27.00 (6.89)</td>
<td>ns</td>
</tr>
<tr>
<td>BDI</td>
<td>25.60 (10.99)</td>
<td>27.20 (9.12)</td>
<td>ns</td>
</tr>
</tbody>
</table>

¹ increases in scores correspond to increases in symptomatology

² ns p>0.05  * p<0.05  ** p<0.02  ***p<0.01  ****p<0.001

3.2 Clinical Group
As indicated on figure 2, statistical analysis of the scores for this group of subjects indicated that on discharge they had experienced a significant reduction on all symptom measures.

Figure 2  Scores on all symptom measures of clinical group on initial assessment and on discharge
(assessment (IES (T)): $t=3.85$, d.f.=28, $p<0.001$; IES(A): $t=4.60$, d.f.=28, $p<0.001$; IES(I): $t=2.72$, d.f.=28, $p<0.02$; BAI: $t=3.75$, d.f.=28, $p<0.001$; BDI: $t=3.78$, d.f.=28, $p<0.001$).

Tables 3.2 shows the mean scores of subjects on all measures on both assessments. Due to the high rate of clients dropping out of therapy an additional statistical analysis was carried out in order to look for possible differences on the initial scores on all symptom measures between subjects who had completed therapy and those who had dropped out of therapy. The results of this analysis using t-test (unrelated) did not reveal any significant differences in the IES(T) between the two groups ($t=1.38$, d.f=39, $p>0.05$).

Table 3.2  Means and standard deviations (in parenthesis) of clinical group scores on IES (T), IES (A), IES (I), BAI and BDI.

<table>
<thead>
<tr>
<th>Measures</th>
<th>Initial Assessment n=29</th>
<th>Assessment on Discharge N=29</th>
<th>Significance$^1$</th>
</tr>
</thead>
<tbody>
<tr>
<td>IES (T)</td>
<td>39.14 (5.28)</td>
<td>28.55 (15.02)</td>
<td>****</td>
</tr>
<tr>
<td>IES (A)</td>
<td>20.34 (6.94)</td>
<td>15.14 (10.10)</td>
<td>****</td>
</tr>
<tr>
<td>IES (I)</td>
<td>18.79 (6.80)</td>
<td>13.41 (6.92)</td>
<td>**</td>
</tr>
<tr>
<td>BAI</td>
<td>31.13 (6.42)</td>
<td>22.27 (9.58)</td>
<td>****</td>
</tr>
<tr>
<td>BDI</td>
<td>26.96 (9.21)</td>
<td>21.48 (14.27)</td>
<td>****</td>
</tr>
</tbody>
</table>

$^1$ increases in scores correspond to increases in symptomatology

$^2$ ns $p>0.05$  * $p<0.05$  ** $p<0.02$  ***$p<0.01$  **** $p<0.001$

However, as shown on figure 3 and table 3.3, comparison of the scores on the other measures indicated that on initial assessment clients who completed therapy had scored significantly lower on IES (A) ($t=3.77$, d.f=39, $p<0.001$) and BDI ($t=4.36$, d.f=39, $p<0.001$) than those who dropped out of therapy.
With regard to IES (I) ($t=2.61$, d.f=39, $p<0.02$) and BAI ($t=5.48$, d.f=39, $p<0.001$) the scores of people who completed therapy were significantly higher.

### Table 3.3
Assessment scores on IES (T), IES (A), IES (I), BAI and BDI of clients who completed therapy and those who dropped out of therapy.

<table>
<thead>
<tr>
<th>Measures</th>
<th>Complete Therapy n=29</th>
<th>Incomplete Therapy n=12</th>
<th>Significance$^2$</th>
</tr>
</thead>
<tbody>
<tr>
<td>IES (T)</td>
<td>39.14 (5.28)</td>
<td>42.08 (8.17)</td>
<td>ns</td>
</tr>
<tr>
<td>IES (A)</td>
<td>20.34 (6.94)</td>
<td>28.91 (5.74)</td>
<td>****</td>
</tr>
<tr>
<td>IES (I)</td>
<td>18.79 (6.80)</td>
<td>13.16 (4.66)</td>
<td>**</td>
</tr>
<tr>
<td>BAI</td>
<td>31.13 (6.42)</td>
<td>20.25 (3.70)</td>
<td>****</td>
</tr>
<tr>
<td>BDI</td>
<td>26.96 (9.21)</td>
<td>41.00 (9.79)</td>
<td>****</td>
</tr>
</tbody>
</table>

1 increases in scores correspond to increases in symptomatology

2 ns $p>0.05$ * $p<0.05$ ** $p<0.02$ ***$p<0.01$ **** $p<0.001$
The significant differences obtained on four of the symptom measures between clients who had completed therapy and those who had dropped out necessitate the investigation of the possible role of demographic data. Statistical analysis using t-test (related) although did not reveal any significant differences in the age of subjects in the two subgroups (t= .75, d.f=39, p>0.05), did suggest that for subjects who failed to complete therapy the time gap between trauma and assessment was significantly greater (t=5.06, d.f=39, p<0.001) than those subjects who completed therapy (see table 3.4).

Table 3.4 Comparison of age and time gap between trauma and assessment for clients who completed therapy and those who dropped out of therapy.

<table>
<thead>
<tr>
<th>Measures</th>
<th>Complete Therapy n=29</th>
<th>Incomplete Therapy N=12</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (in Years)</td>
<td>35.72 (10.53)</td>
<td>38.25 (7.89)</td>
<td>ns</td>
</tr>
<tr>
<td>Time Gap (in Months)</td>
<td>15.58 (10.27)</td>
<td>36.75 (16.05)</td>
<td>****</td>
</tr>
</tbody>
</table>

1 increases in scores correspond to increases in symptomatology

2 ns p>0.05 * p<0.05 ** p<0.02 *** p<0.01 **** p<0.001

The possible influence of the time taken between trauma and assessment on the scores of inventories of those subjects who had completed therapy was also investigated. Median split performed in the variable of time gap between trauma and assessment established two subgroups, those who had completed therapy and the time between trauma and assessment was below the Median (M=15) and those who had completed therapy and the time gap was above the median. Statistical comparisons of the scores in each of the inventories between these two sub-groups (see table 3.5) using t-test (unrelated) revealed
no significant differences on the IES (T) \((t=.35, \, d.f=27, \, p>0.05)\). However, people who were seen for assessment within the first fifteen months of their trauma had scored significantly higher on the IES (I) \((t=2.89, \, d.f=27, \, p<0.01)\) and significantly lower on the IES (A) \((t=3.23, \, d.f=27, \, p<0.05)\) than those people who were seen after fifteen months. A similar pattern was observed with regard to the scores on BAI and BDI (see figure 4).

**Figure 4** The scores of Clinical Group based on time gap between trauma and initial assessment

Subjects who completed therapy and were seen for initial assessment within fifteen months from the trauma had scored significantly lower on the BDI \((t=3.88, \, d.f=27, \, p<0.001)\) than the group whose time gap between trauma and initial assessment was higher than the median.
Table 3.5 Means and standard deviations (in parenthesis) of Scores on Inventories based on time gap between trauma and initial assessment.

<table>
<thead>
<tr>
<th>Measures 1</th>
<th>Time Gap Lower than 15 Months</th>
<th>Time Gap Higher than 15 Months</th>
<th>Significance 2</th>
</tr>
</thead>
<tbody>
<tr>
<td>IES (T)</td>
<td>38.80 (5.38)</td>
<td>39.50 (5.36)</td>
<td>ns</td>
</tr>
<tr>
<td>IES (I)</td>
<td>21.93 (7.78)</td>
<td>15.43 (3.25)</td>
<td>***</td>
</tr>
<tr>
<td>IES (A)</td>
<td>16.87 (6.19)</td>
<td>24.07 (5.81)</td>
<td>***</td>
</tr>
<tr>
<td>BAI</td>
<td>33.20 (7.31)</td>
<td>28.93 (4.60)</td>
<td></td>
</tr>
<tr>
<td>BDI</td>
<td>21.73 (7.25)</td>
<td>32.57 (7.75)</td>
<td>****</td>
</tr>
</tbody>
</table>

1 increases in scores correspond to increases in symptomatology

2 ns p>0.05  * p<0.05  ** p<0.02  *** p<0.01  **** p<0.001

With regard to the BAI, subjects who were seen early had scored higher than those who were seen late but the differences between the groups failed to reach statistical significance (t=1.87, d.f=27, p>0.05).

Table 3.6 Scores on symptom measures between initial assessment and discharge for subjects seen within fifteen months from the trauma

<table>
<thead>
<tr>
<th>Measures 1</th>
<th>Initial Assessment</th>
<th>Assessment on Discharge</th>
<th>Significance 2</th>
</tr>
</thead>
<tbody>
<tr>
<td>IES (T)</td>
<td>38.80 (5.38)</td>
<td>21.53 (13.15)</td>
<td>****</td>
</tr>
<tr>
<td>IES (A)</td>
<td>16.87 (6.17)</td>
<td>10.27 (9.49)</td>
<td>****</td>
</tr>
<tr>
<td>IES (I)</td>
<td>21.93 (7.79)</td>
<td>11.33 (4.52)</td>
<td>***</td>
</tr>
<tr>
<td>BAI</td>
<td>33.20 (7.31)</td>
<td>18.93 (8.43)</td>
<td>****</td>
</tr>
<tr>
<td>BDI</td>
<td>21.73 (7.28)</td>
<td>14.27 (11.89)</td>
<td>****</td>
</tr>
</tbody>
</table>

1 increases in scores correspond to increases in symptomatology

2 ns p>0.05  * p<0.05  ** p<0.02  *** p<0.01  **** p<0.001
Investigating the response to therapy, it was revealed using t-test (related) that the scores on IES (T) \( (t=4.38, \ d.f.=14, \ p<0.001) \), IES (I) \( (t=3.67, \ d.f.=14, \ p<0.01) \), IES (A) \( (t=5.09, \ d.f.=14, \ p<0.001) \), BAI \( (t=4.11, \ d.f.=14, \ p<0.001) \) and BDI \( (t=4.30, \ d.f.=14, \ p<0.001) \) on discharge of those who were seen for therapy within fifteen months from their traumatic experience were significantly smaller than their scores on initial assessment (see table 3.6).

As shown on table 3.7, although there were some reductions on the scores between initial assessment and discharge of those subjects who were seen for therapy after 15 months from the trauma, the differences failed to reach significance \( (p>0.05) \) on all symptom measures.

Table 3.7  Scores on symptom measures between initial assessment and discharge for subjects seen after fifteen months from the trauma

<table>
<thead>
<tr>
<th>Measures(^1)</th>
<th>Initial Assessment</th>
<th>Assessment on Discharge</th>
<th>Significance(^2)</th>
</tr>
</thead>
<tbody>
<tr>
<td>IES (T)</td>
<td>39.50 (5.36)</td>
<td>36.07 (13.51)</td>
<td>ns</td>
</tr>
<tr>
<td>IES (A)</td>
<td>24.07 (5.81)</td>
<td>20.36 (8.11)</td>
<td>ns</td>
</tr>
<tr>
<td>IES (I)</td>
<td>15.42 (3.25)</td>
<td>15.64 (6.90)</td>
<td>ns</td>
</tr>
<tr>
<td>BAI</td>
<td>28.92 (4.60)</td>
<td>25.86 (9.71)</td>
<td>ns</td>
</tr>
<tr>
<td>BDI</td>
<td>32.57 (7.75)</td>
<td>29.21 (12.72)</td>
<td>ns</td>
</tr>
</tbody>
</table>

\(^1\) increases in scores correspond to increases in symptomatology

\(^2\) ns \( p>0.05 \)  * \( p<0.05 \)  ** \( p<0.02 \)  *** \( p<0.01 \)  **** \( p<0.001 \)

Finally, considering that the study examines the efficacy of this mode of therapy in the course of routine clinical practice, further analysis was carried out in order to investigate the extent of which subjects continued to meet the criteria for diagnosis for PTSD at the end of therapy. As indicated on table 3.8, on the basis of their scores on each particular inventory
both pre and post intervention assessment all subjects were allocated into the above or below the threshold category for clinical symptomatology as suggested by the standardised norms. Non-completers were also included in this analysis assuming that their scores did not change over time.

Table 3.8 Number and percentages (in parenthesis) of subjects scoring above and below the cut off point for clinical symptomatology during pre and post treatment assessments on IES (T), IES (A), IES (I), BAI and BDI.

<table>
<thead>
<tr>
<th>Scores on IES-T</th>
<th>Pre-Treatment n=41</th>
<th>Post-Treatment n=41</th>
<th>Significance¹</th>
</tr>
</thead>
<tbody>
<tr>
<td>Above 34.5 (for Males)</td>
<td>32 (78.05)</td>
<td>18 (43.90)</td>
<td>*</td>
</tr>
<tr>
<td>42.1 (for Females)</td>
<td>9 (21.95)</td>
<td>23 (56.10)</td>
<td>**</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Scores on IES-A</th>
<th>Pre-Treatment n=41</th>
<th>Post-Treatment n=41</th>
<th>Significance¹</th>
</tr>
</thead>
<tbody>
<tr>
<td>Above 18.2</td>
<td>32 (78.05)</td>
<td>16 (39.02)</td>
<td>*</td>
</tr>
<tr>
<td>Below 18.2</td>
<td>9 (21.95)</td>
<td>25 (60.98)</td>
<td>***</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Scores on IES-I</th>
<th>Pre-Treatment n=41</th>
<th>Post-Treatment n=41</th>
<th>Significance¹</th>
</tr>
</thead>
<tbody>
<tr>
<td>Above 21.4</td>
<td>33 (80.49)</td>
<td>15 (36.59)</td>
<td>***</td>
</tr>
<tr>
<td>Below 21.4</td>
<td>8 (19.51)</td>
<td>26 (63.41)</td>
<td>***</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Scores on BDI</th>
<th>Pre-Treatment n=41</th>
<th>Post-Treatment n=41</th>
<th>Significance¹</th>
</tr>
</thead>
<tbody>
<tr>
<td>Above 30</td>
<td>29 (70.73)</td>
<td>15 (36.59)</td>
<td>*</td>
</tr>
<tr>
<td>Below 30</td>
<td>12 (29.27)</td>
<td>26 (63.41)</td>
<td>*</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Scores on BAI</th>
<th>Pre-Treatment n=41</th>
<th>Post-Treatment n=41</th>
<th>Significance¹</th>
</tr>
</thead>
<tbody>
<tr>
<td>Above 30</td>
<td>31 (75.61)</td>
<td>15 (36.59)</td>
<td>**</td>
</tr>
<tr>
<td>Below 30</td>
<td>10 (24.39)</td>
<td>26 (63.41)</td>
<td>***</td>
</tr>
</tbody>
</table>

¹ ns p>0.05   * p<0.05    ** p<0.02    ***p<0.01    **** p<0.001
Subsequently a chi-square analysis was calculated for each inventory for above the diagnostic threshold scores both pre and post intervention (for IES a further chi-square analysis was calculated for each of the subscales). The results indicated a significant reduction in the number of people who scored above the threshold during initial assessment on IES (T) \((\chi^2=6.13, \text{df}=1, p<0.02)\), IES(A) \((\chi^2=7.53, \text{df}=1, p<0.01)\), IES (I) \((\chi^2=9.53, \text{df}=1, p<0.01)\), BDI \((\chi^2=4.45, \text{df}=1, p<0.05)\) and BAI \((\chi^2=5.57, \text{df}=1, p<0.02)\) compared to the number of people at post treatment. Also, a significant increase was observed in the number of subjects who scored below the threshold during post treatment assessment on IES (T) \((\chi^2=3.92, \text{df}=1, p<0.05)\), IES(A) \((\chi^2=5.33, \text{df}=1, p<0.05)\), IES (I) \((\chi^2=6.75, \text{df}=1, p<0.01)\), BDI \((\chi^2=5.16, \text{df}=1, p<0.05)\) and BAI \((\chi^2=7.11, \text{df}=1, p<0.02)\) compared to the number of subjects on initial assessment.
4. DISCUSSION

The inclusion of PTSD in the diagnostic tools DSM III (APA, 1980) and ICD-10 (WHO, 1992) as a separate diagnostic entity constituted the recognition of the suffering experienced by individuals exposed to traumatic and life threatening events. Also it marked the beginning of health professionals' systematic investigation of the factors contributing to the development of the symptoms that characterise the disorder (e.g., Joseph et al., 1997). Most theories of Post-Traumatic Stress Disorder at their basic level suggest that the disorder arises as a result of information overload mediated by a number of different factors such as premorbid personality, frequency and duration of exposure to trauma. According to Roth and Fonagy's (1996) review, from the available psychotherapeutic approaches deriving from these theoretical models Cognitive Behavioural Therapy is considered as probably the most effective form of psychological intervention in the treatment of PTSD. In addition to the potential value of the approach in alleviating the symptoms of the disorder, the same authors also acknowledge the difficulties associated with current practices of clinical research. Taking into consideration that research into the efficacy of specific psychotherapeutic approaches often reflect the interests, the intentions and the resources available to the researcher rather than the conditions of everyday clinical practice, they emphasise that this type of results need to be interpreted with some caution before being transferred to the clinic. The problems associated with the generalisation and application of research findings into clinical settings were also recognised by the review of the National Health Service Psychotherapy Services in England (1996). In that review it was pointed out the need to improve clinical effectiveness of NHS services by developing evidence-based practices. In such practices the efficacy of psychotherapeutic interventions on individuals or groups of patients should be constantly monitored by using, where possible, outcome measures.

Despite the many obstacles that usually accompanies the implementation of the approach described by the National Health Service Psychotherapy Services in England (1996) review, the main aim of the present study was to investigate the efficacy of a Cognitive Behavioural Therapy package in the treatment of a group of PTSD sufferers referred to
the Adult Section of the Clinical Psychology Department of Barnet NHS Trust. Based on Roth's and Fonagy's (1996) suggestion that CBT programs can be regarded as the treatment of choice for PTSD it was proposed that subjects receiving this type of therapy would experience significant reduction in their overall symptomatology than their waiting list control counterparts. A summary of the results within the context of the two hypotheses (see section 1.5) will be presented in the next section. This will be followed by a discussion of the limitations of the study and the possible implications of these findings to future research.

4.1 Summary of Results

According to the first hypothesis the expectation was that there would be no significant differences in the symptomatology displayed by subjects on the waiting list control group on initial assessment and twelve weeks later. Statistical analysis on each of the three outcome measures supported the hypothesis as no significant differences were found in the scores of subjects in all symptom measures (p>0.05). With regard to the second hypothesis, it was revealed that subjects who had received 12 sessions of Cognitive Behavioural Therapy had scored significantly lower on IES (T) (p<0.001) IES (A) (p<0.001) IES (I) (p<0.02), BAI (p<0.001) and BDI (p<0.001) on discharge compared to their initial assessment scores. A problem associated with these findings however is that the significant reduction of scores reported above does not take into account the high number of subjects that dropped out of therapy within that 12 week period.

Although attrition of approximately 30% is very much within the limits reported by a number of other PTSD studies (for a review see Meichenbaum, 1994) further statistical analysis was performed on the initial assessment scores between those that completed therapy and those that dropped out prior to discharge. Our findings revealed significant differences between the two sub-groups in all measures apart from the IES (T). Statistical analysis indicated that people who had completed therapy scored significantly higher on the IES (I) and the BAI whereas those who failed to attend all 12 sessions of
therapy had scored significantly higher on the IES (A) and BDI. Post hoc investigation of factors that potentially could have played a contributory role to the differences in the scores between these two sub-groups suggested that the time gap between traumatic experience and onset of therapy was significantly higher in those subjects who had dropped out of therapy. Median split on the time gap between trauma and onset of therapy (M=15) performed in all symptom measures for all subjects on initial assessment revealed that those who had come for therapy within the first fifteen months after the trauma had scored significantly higher on the IES (I). On the BAI there was a trend in the same direction but the obtained difference failed to reach significance. Subjects however who came for therapy after 15 months had scored significantly higher on the IES (A) and BDI.

Since these post hoc findings appeared to suggest that with the passage of time the symptomatology experienced by people who have been exposed to trauma changes significantly, the question raised at that stage was with regard to the effectiveness of therapy to subjects that came for therapy within fifteen months from trauma exposure compared to those that came later. Statistical analysis of the scores on initial assessment and on discharge on the first subgroup revealed significant reduction in all symptom measures. For subjects who came for therapy after the initial fifteen months although their scores in most symptom measures indicated a consistent trend of a reduction in their overall symptomatology none of the obtained differences had reached significance.

4.2 Implications of Study to Clinical Practice
As suggested by the above discussion, the results of the research study supported our two hypotheses. However, the high rate of attrition, the different scores obtained by those who had completed therapy and those who had dropped out and the differences in the impact that therapy had for subjects who sought help within the first fifteen months from trauma compared to those who sought help after a longer period of time all seemed to point out that the Cognitive Behavioural Therapy package employed in this study is not
equally effective to all PTSD sufferers. In fact the findings suggest that the pattern of symptoms experienced by individuals suffering from PTSD change as the time gap from trauma exposure increases and that these changes in symptomatology predict the degree by which the Cognitive Behavioural therapy techniques employed can assist in the alleviation of symptoms.

In summary, the results obtained in the present clinical study show that patients who are seen for therapy within fifteen months from trauma usually experience high levels of anxiety as indicated by their scores on the IES (I) and BAI. Moreover, it seems that Cognitive Behavioural Therapy is quite effective in reducing their symptomatology based on the scores of these subjects on initial assessment and on assessment after they had received twelve sessions of therapy. In subjects where the time gap between therapy and trauma is greater than fifteen months not only they are more prone to drop out of therapy but also on the basis of their questionnaire scores they appear to be experiencing higher symptoms of avoidance and depression. Finally, in this group of subjects Cognitive Behavioural Therapy does not appear to be as effective in reducing symptomatology.

A possible explanation for this pattern of response to the Cognitive Behavioural Therapy employed in this study is that as the time from trauma exposure increased subjects, in the later group have learnt to reduce their anxiety and discomfort stemming from their trauma by avoiding thoughts, memories and stimuli that were associated with the traumatic event. Although this mechanism of avoidance in the short term appears to be quite adaptive, in the longer term the same mechanism can lead to social and occupational impairment which in turn contributes to the rising of symptoms of depression. One of the basic principles of the Cognitive Behaviour Therapy employed in the study was to expose patients to the memory of the incident. The technique of exposure however, appeared to counteract with the mechanism of avoidance and increased their levels of anxiety. Due to heightened levels of anxiety subjects found imaginal exposure difficult to tolerate and as a result they either dropped out of therapy or, if they continued to come to sessions, they persistently avoided aspects of the trauma that they considered difficult to cope with. In
contrast to the above response, subjects who were seen for therapy within the first fifteen months from their traumatic experience their levels of intrusions and anxiety were already quite high and therefore exposure to the incident was more tolerable and therefore the therapy package used effective.

As reviewed in section 1.4. many researchers (see Joseph et al., 1997) have proposed that for individuals with low tolerance for anxiety it might be quite beneficial to incorporate within the treatment package anxiety management techniques. Linking this proposal to the findings of the present study, it seems that the group of subjects that would benefit from such an approach are those who due to the increased time gap between trauma exposure and onset of therapy have developed elaborate techniques of avoidance in order to reduce the discomfort associated with their anxiety. It is anticipated that the inclusion of anxiety management and general coping skills in the treatment of this group of PTSD sufferers prior to the onset of therapy might be quite effective in enhancing therapy outcome. This is because it is anticipated it will provide patients with alternative skills to deal with the high levels of anxiety and discomfort that they will encounter in the course of treatment.

4.3 Limitations of Study
The findings of the clinical study reported seem to indicate quite consistently that the Cognitive Behavioural Therapy techniques employed are quite effective for subjects that sought therapy quite early whereas the time gap between trauma and onset of treatment appears to reflect differences in the pattern of symptoms experienced. A relevant conclusion that was drawn from these findings was that treatment programmes that incorporate anxiety management techniques could be quite beneficial for subjects who were not seen for therapy early enough and therefore have developed excessive symptoms of avoidance and presumably low tolerance for anxiety.
But despite the consistency of the results, any elaboration and/or application of these findings to clinical practice should be done with caution, as the study is not free of any limitations. One of the major problems that characterise research about the efficacy of treatment modalities to specific psychological problems is that quite commonly researchers try to implement therapies where the influence of confounding variables is kept to the minimum. As pointed out in section 1 any findings derive from these studies often has direct impact on the outcome of the therapy making it extremely difficult to draw any conclusions about the efficacy of the approach. Although in the present study the main aim was to investigate the effectiveness of a specific treatment modality in subjects suffering from PTSD as referred to the department without attempting to minimise the confounding variables, this was not fully achieved. For example, in the study there were no people who had experienced type II (prolonged and repeated) trauma or who had been traumatised as a result of vicarious experience. Similarly, there were not any subjects who had been seen for therapy regarding the same problem in the past or had reported history of a psychotic illness and/or history of drug and alcohol dependency and none of the people taking part in the study were on any medication.

Another limitation of the study is with regard to the type and number of symptom measures employed. In order to minimise the time spent in completing questionnaires only three sets of symptom measures were used. As a result, the findings regarding the overall picture presented by patients can be described as quite restricted and incomplete. An additional limitation of the study is the lack of a long term follow up data. Although a 12 week follow up appointments were offered to all people who completed therapy unfortunately only a very small percentage of them kept their appointments making it impossible to analyse their responses statistically and to draw any meaningful conclusion. A final limitation is with regard to the size of the sample used. Although the original sample of 41 subjects appeared to be adequate for the purpose of this clinical research, considering the annual number of referrals received in Clinical Psychology Department, the subsequent high rate of attrition reduced the sample. As a result, post hoc investigation of the characteristics of the different sub-groups of patients was based on an
even smaller number of subjects and this by extent weakens the explanatory power of our findings. Also a true randomised design involves an intention-to-treat analysis, which counts all outcomes in the treatment group as a consequence of treatment. In those cases where patients did not complete their treatment and dropped out of therapy should be considered as not making any further change and their last recorded scores should also be considered as their final scores and be included in any statistical analysis. In the present study, although the scores of all subjects were taken into consideration when examining caseness, in all other statistical analyses presented in section 3 only the scores of those completing treatment were used. A limitation that potentially could lead to a false impression with regard to the benefits of this type of therapy to all PTSD patients. As a consequence of these limitations, the current results although quite encouraging can be regarded only as relevant to a certain subgroup of patients and any generalisation of these findings across all PTSD sufferers might be problematic.
5. CONCLUSION AND SUGGESTIONS FOR FUTURE RESEARCH

The inclusion of PTSD as a separate category into the diagnostic tools marked the beginning of a systematic investigation of the factors contributing to the emergence of PTSD and the efficacy of the psychotherapeutic approaches employed in the treatment of the disorder. Although research studies indicate that most treatment modalities are significantly more effective than waiting list control groups (e.g., Hudson and Pope, 1990; Scarvalone et al., 1995; Rothbaum, 1997) the findings of Roth and Fonagy's (1996) review indicate that Cognitive Behavioural Therapy is probably the most effective approach in reducing the symptomatology of PTSD sufferers. But despite their recommendation that this treatment modality can be considered as the treatment of choice for PTSD they also emphasise that most studies investigating the efficacy of the approach are characterised by several methodological problems that limit the potential value of findings to actual clinical settings. The most important of these limitations is that most research studies investigating the efficacy of psychotherapeutic approaches is usually carried out away from everyday clinical practice. The need and the potential value for developing evidence-based practice where the efficacy of psychotherapeutic interventions on individuals or groups of patients is constantly monitored was also identified in the review of the National Health Service Psychotherapy Services in England (1996).

The research reported in this study aimed to adopt the approach proposed by the National Health Service Psychotherapy Services in England (1996) with regard to the efficacy of Cognitive Behavioural Therapy in the treatment of PTSD. The results obtained showed that subjects who received Cognitive Behavioural Therapy experienced a reduction in their overall symptomatology. However, post hoc investigation of the data revealed that exposure and cognitive restructuring are quite effective in alleviating the symptomatology of subjects experiencing high symptoms of intrusion and anxiety as indicated by IES (I) and the BAI which seem to characterise subjects who sought therapy within the first fifteen months from their traumatic experience. Subjects who came for therapy after a longer period of time, based on their scores on the IES (A) and BDI, experienced increased symptoms of avoidance and depression which were less amenable
to the Cognitive Behavioural Therapy techniques mentioned above. Based on these results it was proposed that due to the incompatibility of avoidance, which has been developed by individuals to reduce their anxiety stemming from trauma and the increased levels of anxiety experienced as a result of exposure techniques, subjects in this later group found it difficult to cope with this type of treatment and they either dropped out of therapy or they came to sessions but continued to avoid aspects of the trauma that they found difficult to cope with. Moreover, it was suggested that the incorporation of anxiety management techniques into the Cognitive Behavioural Therapy package offered to this group of PTSD sufferers would provide them with the skills to cope better with their increased anxiety and therefore benefit more from the other components of the treatment.

Although these findings and suggestions appeared to accord well with proposals that incorporation of anxiety management techniques into Cognitive Behavioural Therapy packages can be highly beneficial to subjects with low tolerance for anxiety (Joseph et al. 1997), the usefulness of this approach has never been tested within the context of everyday clinical practice. Since any evidence supporting this proposal can potentially contribute to the development and improvement of the treatment offered to different subgroups of people with PTSD, it might be worth investigating the role of this proposal further.

As mentioned in section 4.3 the present study is associated with three major problems. These were the size of the sample employed, the restricted number of symptom measures used and the lack of follow up data. A possible way to improve the study and strengthen its explanatory power with regard to the efficacy of this type of therapy is by replicating it using a larger sample with more symptom measures. The problem of follow up data is a problem that is typical of most studies. A route to overcome the problem of obtaining follow-up data is by considering the possibility of asking the local General Practitioners to ask their patients referred to the department for psychological therapy, on any of their subsequent visits to the surgery to complete again the set of questionnaires used at the beginning and at the end of their treatment.
Finally, the aims of the recommendation made by the review of the National Health Service Psychotherapy Services in England (1996) about the development of evidence-based practices within the NHS system were twofold. It was anticipated that such an approach apart from increasing our understanding of the effectiveness of treatment modalities to specific problems it would also promote the accumulation of data that would facilitate direct comparisons with the results of controlled clinical trials and of studies used in a different therapeutic approaches for the treatment of the same disorder(s). On the basis of the above recommendation, a possible way to investigate the efficacy of Cognitive Behavioural Therapy in reducing PTSD symptomatology compared to other psychotherapeutic approaches is by attempting to replicate the study using another treatment approach instead of Cognitive Behavioural Therapy.

In concluding, the evidence obtained in the present study indicates that exposure and cognitive restructuring techniques seem to be quite effective for a subgroup of PTSD sufferers whereas for others it might be more appropriate to incorporate within the treatment package anxiety management training. Taking into consideration however the number of limitations associated with the study, the results obtained cannot be regarded as conclusive and the application of these findings to clinical practice needs to be preceded by further investigation.
6. References


APPENDIXES
## Impact of Event Scale (IES)

**Appendix 7.1**

**On** *(date)*

**You experienced** *(life event)*

<table>
<thead>
<tr>
<th>Frequency</th>
<th>Not at all</th>
<th>Rarely</th>
<th>Sometimes</th>
<th>Often</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. I thought about it when I didn't mean to</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2. I avoided letting myself get upset when I thought about it or was reminded of it</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3. I tried to remove it from memory</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4. I had trouble falling asleep or staying asleep, because of the pictures or thoughts about it that came into my mind</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5. I had waves of strong feelings about it</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6. I had dreams about it</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>7. I stayed away from reminders of it</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>8. I felt as if it hadn't happened or it wasn't real</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>9. I tried not to talk about it</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>10. Pictures about it popped into my mind</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>11. Other things kept making me think about it</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>12. I was aware that I still had a lot of feelings about it, but I didn't deal with them</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>13. I tried not to think about it</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>14. Any reminder brought back feelings about it</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>15. My feelings about it were kind of numb</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

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Appendix 7.2

BECK ANXIETY INVENTORY (BAI)

Name  
Date 

Below is a list of common symptoms of anxiety. Please carefully read each item in the list. Indicate how much you have been bothered by each symptom in the PAST WEEK, INCLUDING TODAY, by placing an X in the corresponding space in the column next to each symptom.

<table>
<thead>
<tr>
<th></th>
<th>Not At All</th>
<th>Mildly</th>
<th>Moderately</th>
<th>Severely</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Numbness or tingling</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2.</td>
<td>Feeling hot</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3.</td>
<td>Wobbliness in legs</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4.</td>
<td>Unable to relax</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5.</td>
<td>Fear of the worst happening</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6.</td>
<td>Dizzy or light-headed</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>7.</td>
<td>Heart pounding or racing</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>8.</td>
<td>Unsteady</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>9.</td>
<td>Terrified</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>---</td>
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<td>10.</td>
<td>Nervous</td>
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<td>11.</td>
<td>Feelings of choking</td>
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<td>12.</td>
<td>Hands trembling</td>
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<tr>
<td>13.</td>
<td>Shaky</td>
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<td>14.</td>
<td>Fear of loosing control</td>
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<tr>
<td>15.</td>
<td>Difficulty breathing</td>
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<tr>
<td>16.</td>
<td>Fear of dying</td>
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<tr>
<td>17.</td>
<td>Scared</td>
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<tr>
<td>18.</td>
<td>Indigestion or discomfort in abdomen</td>
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<tr>
<td>19.</td>
<td>Faint</td>
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<tr>
<td>20.</td>
<td>Face flushed</td>
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<tr>
<td>21.</td>
<td>Sweating (not due to heat)</td>
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</tbody>
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Appendix 7.3

BECK DEPRESSION INVENTORY (BDI)

On this questionnaire are groups of statements. Please read each group of statements carefully. Then pick out the one statement in each group which best describes the way you have been feeling the PAST WEEK, INCLUDING TODAY: Circle the number beside the statement you picked.

1 0 I do not feel sad.
1  I feel sad.
2  I am sad all the time and I can’t snap out of it.
3  I am so sad or unhappy that I can’t stand it.

2. 0 I am not particularly discouraged about the future.
1  I feel discouraged about the future.
2  I feel I have nothing to look forward to.
3  I feel that the future is hopeless and that things cannot improve.

3 0 I do not feel like a failure.
1  I feel I have failed more than the average person.
2  As I look back on my life, all I can see is a lot of failures.
3  I feel I am a complete failure as a person.

4 0 I get as much satisfaction out of things as I used to.
1  I don’t enjoy things the way I used to.
2  I don’t get real satisfaction out of anything any more.
3  I am dissatisfied or bored with everything.

5 0 I don’t feel particularly guilty.
1  I feel guilty a good part of the time.
2  I feel quite guilty most of the time.
3  I feel guilty all of the time.
| 6 | 0 | I don't feel I am being punished. |
|   | 1 | I feel I may be punished.        |
|   | 2 | I expect to be punished.         |
|   | 3 | I feel I am being punished.      |
| 7 | 0 | I don't feel disappointed in myself. |
|   | 1 | I am disappointed in myself.     |
|   | 2 | I am disgusted with myself.      |
|   | 3 | I hate myself.                   |
| 8 | 0 | I don't feel I am any more than anybody else. |
|   | 1 | I am critical of myself for my weaknesses or mistakes. |
|   | 2 | I blame myself all the time for my faults. |
|   | 3 | I blame myself for everything bad that happens. |
| 9 | 0 | I don't have any thoughts of killing myself. |
|   | 1 | I have thoughts of killing myself, but I would not carry them out. |
|   | 2 | I would like to kill myself.     |
|   | 3 | I would kill myself if I had the change. |
|10 | 0 | I don't cry any more than usual. |
|   | 1 | I cry more now than I used to.   |
|   | 2 | I cry all the time now.         |
|   | 3 | I used to be able to cry, but now I can't cry even though I want to. |
|11 | 0 | I am no more irritated now than I ever am. |
|   | 1 | I get annoyed or irritated more easily than I used to. |
|   | 2 | I feel irritated all the time now. |
|   | 3 | I don't get irritated at all by things that used to irritate me. |
|12 | 0 | I have not lost interest in other people. |
|   | 1 | I am less interested in other people than I used to be. |
|   | 2 | I have lost most of my interest in other people. |
|   | 3 | I have lost all of my interest in other people. |
|13 | 0 | I make decisions as well as I ever could. |
|   | 1 | I put off making decisions more than I used to. |
|   | 2 | I have greater difficulty in making decisions than before. |
|   | 3 | I can't make decisions at all any more. |
14 0 I don't feel I look any worse than I used to.
1 I am worried that I am looking old or unattractive.
2 I feel there are permanent changes in my appearance.
3 I believe that I look ugly.

15 0 I can work about as well as before.
1 It makes an extra effort to get started at doing something.
2 I have to push myself very hard to do anything.
3 I can’t do any work at all.

16 0 I can sleep as well as usual.
1 I don’t sleep as well as I used to.
2 I wake up 1-2 hours earlier than usual and find it hard to get back to sleep.
3 I wake up several hours earlier than I used to and cannot get back to sleep.

17 0 I don’t get more tired than usual.
1 I get tired more easily than I used to.
2 I get tired from doing almost anything.
3 I am too tired to do anything.

18 0 My appetite is no worse than usual.
1 My appetite is not as good as it used to be.
2 My appetite is much worse now.
3 I have no appetite at all any more.

19 0 I haven’t lost much weight, if any, lately.
1 I have lost more than five pounds.
2 I have lost more than ten pounds.
3 I have lost than fifteen pounds.

I am purposely trying to lose weight by eating less.
Yes ________  No ________

20 0 I am no more worried about my health than usual.
1 I am worried about physical problems such as aches and pains.
2 I am very worried about physical problems and it's hard to think of much else.
3 I am so worried about my physical problems that I cannot think about anything else.
I have not noticed any recent change in my interest in sex.

I am less interested in sex now.

I am much less interested in sex now.

I have lost interest in sex completely.
FORM OF CONSENT SIGNED BY ALL PARTICIPANTS OF THE STUDY

CODE : ___________________________
DATE : ___________________________
NAME : ___________________________
ADDRESS : _________________________
                            _________________________
                            _________________________

CONSENT FORM
I consent to participate in the research study carried out by S. Georgiades, Clinical Psychologist at Barnet NHS Trust, in which the effectiveness of treatment of Post-traumatic stress disorder will be investigated.

I understand that all information obtained will be strictly confidential and will only be used for research purposes.

______________________________
Signature

______________________________
in the presence of
Stelios Georgiades,
Clinical Psychologist
**Appendix 7.5**

**MEASURING THE RATE OF ANXIETY**

Insert on each line, a situation that corresponds to the anxiety situated on the right hand side of the scale.

<table>
<thead>
<tr>
<th>0</th>
<th>10</th>
<th>20</th>
<th>30</th>
<th>40</th>
<th>50</th>
<th>60</th>
<th>70</th>
<th>80</th>
<th>90</th>
<th>100</th>
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</table>
## Appendix 7.6

### Name:

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**RECORD OF LEVELS OF ANXIETY DURING SESSION**

<table>
<thead>
<tr>
<th>Date AND SESSION</th>
<th>RATE OF ANXIETY PRIOR TO RECOUNTING THE EXPERIENCE (0-100)</th>
<th>RATE OF ANXIETY AFTER RECOUNTING THE EXPERIENCE (0-100)</th>
<th>RATE OF ANXIETY END OF SESSION (0-100)</th>
</tr>
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<tbody>
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</table>
Appendix 7.7

Name: ____________________________________________

**RECORD LEVELS OF ANXIETY**

<table>
<thead>
<tr>
<th>Date</th>
<th>RATE OF ANXIETY BEFORE LISTENING TAPE (0-100)</th>
<th>RATE OF ANXIETY AFTER LISTENING TAPE (0-100)</th>
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</tbody>
</table>
# DAILY RECORD FOR NEGATIVE AUTOMATIC THOUGHTS

<table>
<thead>
<tr>
<th>DATE</th>
<th>EMOTIONS</th>
<th>SITUATION</th>
<th>AUTOMATIC THOUGHTS</th>
<th>EVIDENCE FOR</th>
<th>EVIDENCE AGAINST</th>
<th>RATIONAL RESPONSE</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>How bad is it (0-100)?</td>
<td>What were you doing Or thinking about</td>
<td>What exactly were your thoughts? How far do you believe each of them (0-100)?</td>
<td>What evidence do you have supporting these thoughts?</td>
<td>Any evidence that does not support these thoughts?</td>
<td>What are your rational answers to the automatic thoughts? How far do you believe each of them (0-100)?</td>
</tr>
</tbody>
</table>

 NAME: ____________________________

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Appendix 7.8
SECTION 4: RESEARCH DOSSIER

INVESTIGATION OF COGNITIVE DEFICITS IN SCHIZOPHRENIA THROUGH PERFORMANCE ON A REVERSAL LEARNING TASK

ABSTRACT

Background: It is postulated that the positive symptoms of schizophrenia are associated with disturbances in selective attention due to the weakening of influences of stored memories of regularities of previous input to current perception. Neuropsychological theories suggest that this cognitive disturbance could correspond to an underlying neurological deficit specified in the projection from the hippocampus to the nucleus accumbens. The hypothesis that people with acute schizophrenia due to the cognitive deficit described above, will fail to learn from previous experiences is investigated through the behavioural phenomenon of reversal learning.

Method: The performance of normal controls (n=40), people with chronic schizophrenia (n=34) and acute schizophrenia (n=31), was investigated on a computerised version of a learning and a reversal learning task.

Results: The results seem to support the hypothesis that acute schizophrenics less influenced by previous learning tend to shift faster than chronic schizophrenics and normal controls at the outset of reversal.

Conclusions: In addition to the finding that people with acute schizophrenia tend to be less influenced by previous learning and therefore shift faster at the outset of reversal, the same results also point out to the difficulty experienced by this group of subjects to maintain their attention, and consequently their response, to the selected stimulus for prolonged periods of time. These results are discussed within an integrated approach based on the above two models and on other contemporary cognitive and neuropsychological theories of schizophrenia.
disorder might be corresponding to a specific underlying neural dysfunction (e.g., Grace et al., 1998; Gray et al., 1991). Since these recent formulations suggest that the core problem of schizophrenia is cognitive, it is necessary to investigate the specificity of the cognitive dysfunction relevant to the disorder by developing experimental tasks that would tap the attentional processes proposed to play a crucial role in the development of schizophrenic symptomatology.

According to Hemsley’s (1987; 1991) cognitive theory, the positive symptoms of schizophrenia are associated with disturbances in selective attention attributed to the weakening of influences of stored memories of regularities to current perception. Linking the proposed disturbance to their neuropsychological model of schizophrenia, Gray and his colleagues (1991) argued that the cognitive deficits proposed by Hemsley (1987; 1991) could correspond to an underlying neural dysfunction in the subiculo-accumbens projection. Predominantly based on Hemsley’s (1987; 1991) theory, and to a lesser extent on Grays and colleagues’ (1991) model of the disorder, the main aim of the present research study is to investigate the cognitive deficit suggested by Hemsley (1987; 1991) by employing the behavioural phenomenon of reversal learning.

Since the main aim of the present study is focused on the investigation of a theoretical model that draws together evidence from different fields of current schizophrenia research, it is necessary in the introductory section to review a number of areas in order to provide the context within which the research reported in the next section of the thesis was conducted. The findings of the research in relation to the theoretical models reviewed in the introduction will be discussed in the final section.

1.1. General Background to the Concept of Schizophrenia

1.1.1 Historical Background

Behavioural patterns that potentially could qualify for the diagnosis of schizophrenia cannot be considered as a phenomenon of our century. In contrast, descriptions of
such behaviours can be found in abundance in the history spanning from as early as
the biblical times and ancient Greece to the more recent Shakespearean era (Smith,
1986) and the early 19th century asylum patient (Foucault, 1967). The classification
and definition however of the current concept of schizophrenia came much later
through the influential works of Kraeplin (1899) and Bleuler (1911/1950).

The major contribution of Kraeplin was that he was the first to suggest that what was
then regarded as insanity comprised of two distinct disorders (Birchwood et al. 1988)
which he classified as the endogenous psychoses. In the first category, for which he
used the term “dementia praecox” (Morel 1860)*, Kraeplin grouped together the
previously described conditions of heberphrenia (Hecker, 1871)*, monomania (Snell,
1865)*, catatonia (Kahlbaum, 1874)* and primary madness (Griesenger, 1868)*
proposing these as different manifestations of a disease of organic origin characterised
by an early onset and a progressive deterioration. The major symptoms thought to be
associated with dementia praecox were hallucinations, delusions, thought disorder,
behavioural stereotypy and emotional dysfunction. The second disorder was that of a
manic depressive illness which was considered to be a disorder of affect rather than
insanity per se and was distinguished from dementia praecox on the basis of its
symptoms (e.g., euphoria, pressure of speech and grandiose ideas) and its prognosis
(Johnstone, 1994).

Bleuler (1911) who expanded on the concept of dementia praecox to include paranoia
and paraphrenia took a more optimistic attitude on the course of the disorder
suggesting that an early onset and a progressive intellectual deterioration were not
inevitable. Highly influenced by the psychoanalytic schools he went beyond
Kraeplin’s primarily descriptive conceptualisation and proposed that the primary
disorder (which again assumed to be of organic origin) is that of an associative

* cited in McGhie, 1969
disturbance. His term schizophrenia, deriving from the Greek words “σχίσμα” and “φρένα”, meaning split mind, was introduced in order to describe what he called “a loosening of associations between the different functions of the mind” manifesting in the inability of people with schizophrenia to organise single thoughts coherently (Johnstone 1994). In Bleuler’s conceptualisation, symptoms such as delusions and hallucinations were viewed as secondary to the primary associative disturbance and thought to be resulting from the patients’ interaction with the environment; an interaction that is mediated by the underlying associative dysfunction.

1.1.2 Problems Associated with the Concept of Schizophrenia

Nearly a century after the publication of the influential works of Kraeplin and Bleuler our knowledge with regard to what schizophrenia is and the causes underlying the symptomatology associated with the disorder continues to remain limited. In the search for identifying the causes of schizophrenia over the past hundred years, several lines of investigation have been pursued. The results produced from genetic (e.g., Kendler and Diehl, 1993; Kringlen, 1987; Moldin, 1994), biochemical (e.g., Carlsson 1987, 1988, 1991; Angrist et al. 1974; Seeman et al. 1984; Farde et al. 1990), cognitive (e.g., Payne, 1964; Maher, 1983), familial (e.g., Gottesman 1991; DeLisi and Lovett, 1991; Gershon et al. 1988), environmental (e.g., Cutting 1985; Dohrenwend et al. 1987) and neuropathological (Weinberger 1991; Brown et al., 1986; Crow et al. 1989; Falkai et al. 1988; Bogerts 1991) studies failed to provide consistent and conclusive evidence that schizophrenia has an underlying physical pathology or that it is definitely connected with emotional trauma and/or childhood deprivation (McKenna, 1997).

The difficulties encountered in successfully identifying the factors causally linked with schizophrenia led scientists in the 1960’s to propose that schizophrenia as an illness does not really exist (e.g., Laing 1960; Szasz 1961). More recently however, taking into consideration the large number of factors implicated in the disorder and the diverse symptomatology that is associated with it, researchers seem to
increasingly take the more moderate view of referring to the schizophrenias not as a
unitary concept but as a group of related illnesses where the emphasis is on individual
symptoms or clusters of symptoms. Although this view is progressively reflected by
current research strategies (see, Garety and Hemsley 1994; Costello, 1993), the
approach is not without its disadvantages. In contrast to the broad definition where
schizophrenia is regarded as a unitary entity and the main aim is to identify a single
factor that would account for the entire causal network, the present approach may be
regarded as too narrow and may risk reducing the disorder to an incomplete feature
where not all its characteristics are taken into consideration.

Due to the complexities associated with attempts to identify the causes of
schizophrenia, scientists working in the area are left with a basic alternative. That is
to propose (or utilise existing) models that would take into account available data,
knowledge and hypotheses with regard to the disorder from different fields. It is
anticipated that such models potentially would enhance our understanding of the
disorder by providing a more “coherent picture” of schizophrenia as well as they
would highlight areas or aspects of the disorder requiring further experimental
investigation.

1.1.3 The Diagnosis and Classification of Schizophrenia

A major problem that still remains regardless of whether schizophrenia is considered
as a unitary entity or a dimensional concept is with regard to the way by which the
disorder is defined and diagnosed. Since Bleuler (1911) coined the term
schizophrenia there has been considerable diversity amongst scientists working on the
area with regard to what constitutes schizophrenia.

In recent years psychiatry has seen the development of a number of diagnostic tools
for schizophrenia in particular and mental disorders in general. These include the
Diagnostic and Statistical Manual for Mental Disorders IV [DSM IV] (APA 1994),
the Research and Diagnostic Criteria [RDC] (Spitzer et al. 1978), the Present State
Examination [PSE] (Wing et al. 1974) and the Brief Psychiatric Rating Scale [BPRS] (Overall and Gorham, 1962). Detailed reviews of research in the psychiatric phenomenology however indicate that schizophrenia as a diagnostic entity is related to a large number of symptoms ranging from hallucinations and delusions to catatonia and that its diagnosis can be made only on the basis of very few of these symptoms. This lack of homogeneity in the symptomatology observed in people with schizophrenia has led researchers to question the validity and reliability of the concept of schizophrenia as well as to doubt whether there is indeed a diagnostic entity that corresponds to the disorder (Bentall, 1986; Bentall et al., 1988; Bentall, 1990).

According to Bentall (1990), for a diagnostic system to be clinically useful it needs to be reliable so that “...one psychiatrist’s schizophrenic must be another psychiatrist’s schizophrenic” (p.25). Research studies aiming to investigate the reliability of diagnostic systems have indicated that although diagnostic reliability can be achieved within a specific diagnostic tool the rate of concordance between different diagnostic systems is low. The problem becomes even greater when one considers the need for a diagnostic system that would enable the diagnosis of patients not within the same hospital or between diagnostic centres within the same country but across countries (Cooper et al., 1972; Leff et al. 1991).

The development of classificatory typologies that within their framework scientists attempt to understand the clinical heterogeneity of the symptoms associated with schizophrenia is a tradition that started almost concurrently with the works of Kraeplin (1896) and Bleuler (1911). The main aim of classifying the symptoms into categories is to describe and to a certain extent explain the groups of symptoms that seem to be appearing together and on the basis of these descriptions to predict the course of the illness.

Despite the number of dichotomies proposed since the beginning of research in the area of schizophrenia, probably the best known classification is that of the acute versus chronic (e.g., Spitzer et al. 1978). This is associated with the distinction made by Crow (1980) between positive and negative symptoms. According to this distinction acute schizophrenia is characterised by a short period of illness during
which patients display primarily positive symptoms. The description of positive symptoms derives from the distinction made by Crow (1980) between Type I, or positive schizophrenia and Type II, negative schizophrenia. According to Crow (1980) positive symptoms are those symptoms which are abnormal because of their presence and they are in addition to normal mental states. Such symptoms refer to hallucinations, delusions and thought disorder and are less prominent in chronic schizophrenia, which is mostly defined by the presence of negative symptoms. Negative symptoms refer to the absence of some normally presented behaviour and it includes stereotyped behaviour, loss of affect, social withdrawal etc.. According to Research and Diagnostic Criteria (e.g., Spitzer et al. 1978), chronic schizophrenia is characterised by a continuous period of illness of at least two years with no obvious improvement in the mental state of the patient.

Although it seems that the optimal set of criteria that would enable the reliable and valid diagnosis of schizophrenia are yet to be achieved, research in schizophrenia has benefited considerably over the years by employing the specific diagnostic tools mentioned above. This view, seems to be supported by Andreasen and Carpenter (1993) who although acknowledging that the current knowledge of the disorder in terms of its symptomatology and its aetiology is quite limited, they suggest that the findings of studies using current diagnostic tools have been generally valid and useful for clinical, research and demographic purposes.

1.2 Aetiological Factors

During the last hundred years, in the search for the causes that underlie schizophrenia a large number of factors have been hypothesised to have a causal role in the genesis of the disorder (Johnstone, 1994). As a result of these hypotheses and the extensive research studies that followed their formulation many individual areas of schizophrenia research have enjoyed considerable advances. However, up until recently, the contribution made by such advances in describing schizophrenia as a
complex sociopsychobiological phenomenon continued to remain limited because of the wide gap that separated the factors investigated.

More recent attempts to incorporate environmental, psychological and biological factors into single models that would account for the emergence of schizophrenia led to the development vulnerability–stressor models (e.g., Nuechterlein and Dawson, 1984; Nuechterlein 1987). The basic suggestion made by these models is that schizophrenia emerges as a result of the failure of individuals to cope with stressful events in their lives due to a cognitive vulnerability that it is presumed to be the product of a genetic neurobiological deficit (e.g., Nuechterlein and Dawson, 1984; Zubin and Spring, 1977). Within this context it is maintained that people who are predisposed to psychosis as indicated by genetic research (e.g., Gottesman and Shields, 1982; Kety et al., 1975; 1976; Zerbin-Rudin, 1972) are more sensitive to environmental stressors such as stressful life events (e.g., Dohrenwent et al., 1987; Cutting 1985), family relations/behaviour (e.g., Parker et al., 1988; Kuipers et al., 1988) and social class (e.g., Eaton and Boyd, 1985). Linking these with findings from studies on autonomic reactivity and arousal of schizophrenics (e.g. Gruzelier and Venables 1972; 1975; Claridge 1985), which suggest that as environmental stressors increase individuals with predisposition towards schizophrenia begin to engage in protective inhibition, it was proposed that the positive symptoms of schizophrenia might be the result of an imbalance between inhibition and the genetic sensitivity of these individuals (Jones 1991).

The stress vulnerability models have been influential in their attempts to draw together data from different areas of schizophrenia research and to emphasise the presence of both vulnerability and stress in the aetiology of the disorder. A major criticism associated with these models however, is that the causal links assumed between the factors that are considered to play a significant role in the emergence of schizophrenia are generic purely hypothetical and lack the support of experimental evidence (Bentall and Kinderman 1998). These criticisms when considered within the wider context of the disorder, seem to highlight the important need of current schizophrenia research to have models that would attempt to bridge the gap between
aetiological factors with the utilisation of hypotheses that are admissible to experimentation.

On the basis of the evidence reviewed thus far it seems that schizophrenia as a disorder is not the product of a single aetiological factor but rather the result of a sequence of interactions between heredity and environment modulated by biological and cognitive factors. Therefore, although the basic argument of the present thesis is that the symptoms, and more specifically the positive symptoms observed in schizophrenia, are due to cognitive abnormalities of the type suggested by Hemsley (1987) and to a certain extent by Frith (1979; 1989; 1992), one cannot ignore the possible role played by biological factors in providing the essential mechanism through which the cognitive deficits considered here may be expressed (see Shenton et al., 2001; Harvey and Sharma, 2002).

1.2.1 The Biology of Schizophrenia

Consideration of the role of biological factors into the aetiology of schizophrenia began at the turn of the century with Kraeplin's (1899) and Bleuler (1911) hypotheses. Early attempts to unveil the neuropathology of the disorder by microscopic examinations and subsequently by air encephalography however, did not consistently reveal any brain abnormalities due to the crude methods available for studying the brain in life (see David, 1957 for a review). Another problem associated with research in the area is that even if one does accept that schizophrenia is related to structural brain abnormalities, as more recent findings seem to suggest (see section 1.2.1.2), this explanation does not really account for the variability of symptoms of schizophrenia which to a great extent are different than those found in neurological diseases (McKenna 1997). As a result of the repeated failures of early neuropathological studies to convincingly demonstrate that schizophrenia is causally linked to structural brain abnormalities, scientists returned to the investigation of an earlier hypothesis about the causes of schizophrenia which ascribed a pathogenic role to abnormal biochemistry (Birchwood et al. 1988).
1.2.1.1 The Dopamine Hypothesis

The idea that alterations in the brain chemistry could give rise to abnormal mental states has existed for many centuries. However, according to McKenna (1997) it is the synthesis of LSD and its subsequent widespread illicit use that most likely has stimulated the formulation and the subsequent investigation of several biochemical hypotheses of schizophrenia. However, for reasons that are beyond the scope of this thesis none of these theories stood the test of time (see Iversen, 1978; Smythies 1983) and research into the neurochemistry of schizophrenia has been dominated by the dopamine hypothesis (O'Donnell and Grace 1998).

The dopamine hypothesis, in its most general form proposes that schizophrenia, and more specifically the positive symptoms of the disorder, are the result of a dysfunction in at least one dopaminergic system causing the brain to be in a hyperdopaminergic state. This theory has emerged out of several indirect but converging lines of evidence from different areas. Probably the most important line of evidence to support this hypothesis arose out of the discovery of neuroleptic drugs (Davis 1965) which were shown to be quite effective in ameliorating and controlling the positive symptoms of schizophrenia. The therapeutic effect of neuroleptics was attributed to their ability to block dopamine receptors and act as dopamine antagonists (Carlsson and Lindquist 1963). Indirect support for this view comes from the observation that effective neuroleptics lead to extrapyramidal-side effects identical to the ones observed in Parkinson disease; a disease that is known to be caused by depletion of the caudate nucleus (Hornykiewicz 1973). Further evidence supporting the role of dopamine in schizophrenia comes from studies that indicate high correlation between dopamine (DA) receptor blocking action and the clinical potency of neuroleptics (e.g., Seeman et al. 1976; Janssen 1976). Also, in studies where the effects of the DA receptor agonists in non-schizophrenic subjects was examined it was shown that prolonged administration of amphetamine (e.g., McKinney and Moran 1981; Angrist et al. 1970) or of drugs of similar structure such as phenylpropanoline (Lake et al. 1988) can produce psychotic like symptoms (e.g., paranoid ideation,
delusions, hallucinations and stereotyped behaviours) which can be alleviated by neuroleptics (Angrist et al. 1974).

The above findings although indicative of the involvement of dopamine in schizophrenia also seem to be associated with a number of unresolved problems that require further clarification. An important issue arising from this formulation is with regard to the relationship between neuroleptics' dopamine receptor blocking action and their clinical potency. The results produced by research studies showed that neuroleptic drugs block dopamine receptors almost immediately after administration (e.g., Bradeley 1986). In contrast to those findings, clinical observations indicate that neuroleptics do not exert their effectiveness for up to six weeks (e.g. Crow 1980; Pickar 1988) and even then a percentage of patients do not experience any real change in their symptomatology (e.g., Crow et al. 1986). Another source of contradictory evidence is with regard to the action of dopamine agonists. As mentioned earlier there is evidence to suggest that psychostimulants and more specifically amphetamine induces psychotic like symptoms to non-schizophrenic subjects. On the basis of these findings it would be predicted that dopamine agonists would lead people with schizophrenia to experience an exacerbation of their symptomatology. The results of studies investigating the impact of psychostimulants in people with schizophrenia showed that only 40% of patients have experienced exacerbation of their symptoms (for a review see Lieberman et al. 1987) and even then the findings were more relevant to positive symptoms (Chiarello and Cole, 1987).

Although the evidence discussed thus far is characterised by several inconsistencies, the proposition based on circumstantial evidence that dopamine plays a key role in schizophrenia and more specifically in the positive symptoms of the disorder is hardly debatable (Carlsson, 1988; Floresco et al, 2001). Of course, the problem with this formulation is that regardless of how strong and convergent are the indications of a functional excess of dopamine in the brains of schizophrenics these indications require confirmation by direct investigation (McKenna 1997). As McKenna (1997) suggests “… functional excess of brain dopamine can only be brought about in a limited number of ways” (p.150). However, research studies aiming to investigate
these and to provide direct evidence of the role of dopamine in schizophrenia has produced more equivocal data than the circumstantial findings discussed earlier. To complicate matters even further, Gray (1991) has pointed out two further anomalies of the dopamine hypothesis. It was reported that even if one does accept that medication does not affect DA levels and that there is indeed an increase in DA receptor density (Owenes et al. 1978), the relation between hyperdopaminergia and schizophrenic symptomatology is still not clear. As it is known, the key symptoms of schizophrenia are cognitive (e.g., delusions, hallucinations, thought disorder), and the most salient feature of the disorder is a deficit in attention (e.g., Garety and Hemsley, 1994). Animal research however has indicated that the two major functions of dopaminergic systems are in motor programming (Robbins and Everitt 1982) and approach to reward (Swerdlow and Koob, 1987), none of which is specifically implicated in acute schizophrenia.

1.2.1.2 Neuroanatomy and Temporal Lobe Pathology

Recent advances in brain imaging techniques in the 1970’s and the recognition that a heterogeneous disorder like schizophrenia is very unlikely to be associated with uniform or specific anatomical changes led to a renewed interest in this area of schizophrenia research (Green 1998) yielding a substantial amount of data.

The evidence reported by a number of studies did not reveal in any consistent manner that schizophrenia is associated with structural abnormalities in areas that are rich in dopaminergic cells e.g., Farde et al, 1990; Hietala et al, 1994). In contrast to the above inconsistencies, there has been during the past few years a growing body of evidence indicating that schizophrenia is associated with ventricular enlargement and anatomical changes in the temporal lobe both in the temporal cortex and the medial aspects of the temporal lobe (Brown et al. 1986; for a review see, Shenton et al, 2001). These findings seem to be quite compatible with earlier knowledge that organic lesions in limbic and paralimbic structures of the temporal lobe are often associated with schizophrenialike symptoms (e.g., Torrey and Peterson 1974).
More precisely, there are several reports that seem to show quite consistently that schizophrenia is associated with structural changes in the major temporolimbic structures such as the hippocampal formation (hippocampus, prosubiculum, subiculum, presubiculum, parasubiculum and ethorhinal cortex), parahippocampal gyrus, the amygdala and the internal segment of the palladium (e.g., Bogerts et al. 1985; Falkai et al., 1988; Dwork 1997).

Functionally, the anatomical structures implicated in schizophrenia (e.g., the hippocampus and the amygdala) are thought to have an important role in the convergence of sensory information. It is now known that sensory information from the external world, after passing unimodal and polymodal cortical associations converge in the hippocampus and the amygdala. These temporolimbic structures are regarded as having a very important role in sensory information processing, in context analysis, in filtering out irrelevant information and in making the comparisons between present and past experiences (Gray 1982; Schmajuk 1987). The functions of these structures seem to point out to an association with selective attention, the dysfunction of which has been implicated in the symptomatology of the disorder almost since the development of the present concept of schizophrenia. Further support of the possible role of temporolimbic structures in schizophrenia comes from Berman and colleagues (1986) who propose that the hippocampus and the associated temporal lobe memory systems have an essential role in the learning of relationships of exteroceptive stimuli and of contextual cues whereas the amygdala plays an important role in coupling exteroceptive sensory information with interoceptive information in relation to affect and emotion. A proposition which according to Bogerts (1997) could lead to the plausible assumption that “… structural and functional deficits in these brain regions are associated with the failure of many schizophrenia patients in the higher integrative and associative brain functions, leading to distorted interpretations of the external reality” (p.424).

Finally, although the evidence reviewed in this section seems to indicate a strong relationship between temporal lobe pathology and schizophrenia, it is important to emphasise that structural brain abnormalities are not confined only to this region of
the brain. In fact there are numerous studies suggesting that other brain areas might be also implicated in the disorder. However, taking into consideration the lack of specificity in the brain areas implicated in the disorder and the way by which these areas are interconnected, it is difficult to determine whether the primary pathology of schizophrenia involves structural changes in many areas of the brain or in only some of them with the remaining being the secondary effects of a primary pathology (Weinberger 1987).

1.2.2 The Role of Cognition in Schizophrenia

In the psychological domain over the years there has been a steady stream of attempts aiming to identify the role of psychological factors in the onset and subsequent development of schizophrenia. Although the importance attributed to the contributory role of a variety of factors in psychotic breakdowns is quite significant, the most dominant trend in contemporary psychological research on schizophrenic impairments is guided by the cognitive theory. Within the general framework of cognition, it is proposed that regardless of what causes schizophrenia, the pathological process underlying the disorder must affect one or more of the higher cognitive functions (see Sharma and Harvey, 2000; Harvey and Sharma, 2002).

Today, the research strategies employed in investigating the possible cognitive deficits associated with schizophrenia can be divided into two main categories: those concerned with the abnormalities that underlie the clinical picture of schizophrenia and those aiming to identify the abnormalities that lie behind it (see McKenna 1997). Although both types of research have been highly useful in the investigation of cognitive deficits that might be associated with schizophrenia, during recent years it is the later strategy that has been applied with increasing success in yielding an important body of data. Predominantly based on highly developed models of normal cognitive functioning, within this approach schizophrenic symptoms are conceptualised in terms of deficits in the individual components of these models.
Research in this area takes the form of developing specific experimental tasks that are considered to be tapping the normal cognitive functioning suggested by the model. On the basis of the differences in the performance of non-schizophrenic and schizophrenic individuals on these tasks, the main aim of these studies is to determine which stage or process described by the model is critical for the emergence of schizophrenic symptoms and, as demonstrated by more recent researchers (Gray et al., 1991; Grace et al., 1998), to attempt to identify the neurobiological substrates of these cognitive deficits. An important problem associated with this type of research though is that the differences obtained in the performance of schizophrenics and non-schizophrenic subjects on specific tasks does not specify whether the findings are inherent to schizophrenia or the result of other variables such as the stage of illness, the pattern of symptoms, the influence of medication and the length of hospitalisation. In order to account for the variability of the disorder and the influences that these variables might exert on findings it has been quite useful to classify patients according to clinical subtypes such as chronic vs acute, positive vs negative symptomatology etc..

1.2.2.1 Early Cognitive Theories of Schizophrenia

The relevance of cognitive deficits in schizophrenia was recognised at the turn of the century almost concurrently with the formulation of the disorder and it was suggested that a deficit in attention is one of the most salient features of the disorder (e.g. Stransky, 1907). However, early attempts to specify the cognitive dysfunction(s) that would account for the symptoms of schizophrenia were hampered by the lack of normal information processing frameworks within which to conceptualise the results obtained (Banister, 1968).

One of the first conceptualisations to derive from the early experimental work with people with schizophrenia was that of over-inclusiveness proposed by Cameron (1938) in which the poor sorting performance observed in schizophrenics was attributed to their “inability to preserve conceptual boundaries”. Cameron’s proposal
gave rise to a series of experimental studies that supported the concept of over-inclusiveness while at the same time it gave rise to the hypothesis that over-inclusiveness was the result of a defective “filtering” mechanism (Payne, 1964; Payne et al., 1970). According to Payne (1964), in a non-defective information processing filter there is a mechanism that inhibits both internal and external stimuli that are irrelevant to the task in hand in order to allow efficient processing of stimuli relevant to the task. On the basis of this it was proposed that the over-inclusiveness observed in people with schizophrenia reflects one aspect of a more general breakdown in this filtering mechanism.

The concept of over-inclusiveness proposed by Cameron (1938) and the subsequent proposition of a “defective filter” made by Payne (1964) gave rise to the development of an array of tasks where the performance of people with schizophrenia was compared to the performance of non-schizophrenic individuals. As a result of those studies several hypotheses were advanced all of which pointed to the relation between schizophrenia and attentional deficits (Silverman 1964a; 1964b; 1967; McGuie and Chapman 1961).

Despite the list of findings indicating the “worse than normal” experiences and performance of people with schizophrenia on attentional tasks, the interpretation of these results within this rather generic framework was not a straightforward process. Most of the early research studies were based on descriptive accounts of a cognitive deficit in terms of a disturbance in the function of selective attention. The unitary concept of attention however, which constituted the main focus of research was so broadly and ill defined that made it impossible to identify with any degree of accuracy which type of attentional disturbances was relevant to schizophrenia and how such deficits could be coherently linked to the observed symptomatology. In fact attempts to verify some of the ideas by examining the performance of schizophrenic patients on a battery of tasks, not only failed to show consistently a clear attentional deficit but they confirmed the overall poor performance of this group of people as compared to that of normal controls (e.g., Kopfstein and Neale 1972).
1.2.2.2 Broadbent's Theories of Information Processing

In addition to the difficulties outlined above, early schizophrenia research also suffered from the lack of normal information processing frameworks within which to conceptualise the findings. One of the early theories of normal information processing which played a significant role in schizophrenia research was Broadbent's (1958) "limited capacity filter model". In that model, the filter was hypothesised to be functioning as a single limited capacity information channel. Since within that hypothetical limited capacity system the input exceeded by far its output capacity, irrelevant or non-salient stimuli were selected on the basis of their physical characteristics and excluded from further processing preventing this way the system from becoming overloaded.

The application of this theory to schizophrenia research became apparent following the publication of the phenomenological studies of McGhie and Chapman (1961). These studies, which indicated the inability of people with schizophrenia to concentrate on any particular cognitive activity due to the presence of increased associations with irrelevant thoughts and stimuli, gave rise to a number of theoretical interpretations most of which emphasised the relevance of Broadbent's model to schizophrenia. According to these theorists, the filter described by Broadbent (1958) was needed to function efficiently at all times in order to prevent irrelevant stimuli and information from further processing. Within this formulation, people with schizophrenia were assumed to have a defective filter, which disrupted their ability to attend selectively and hence the inappropriate associations.

The filter proposed by Broadbent (1958) was investigated in people with schizophrenia by employing dichotic listening tasks. The findings of these studies however indicated that the performance deficits observed in people with schizophrenia was more due to a slow and to a certain extent insufficient processing of relevant stimuli rather than due to the interference of non-salient stimuli as suggested by the defective filter (Shakow 1963; Hawks and Robinson 1971).
The limitations of the filter theory in relation to schizophrenia research and the experimental evidence indicating that people with schizophrenia are not distracted by any stimulus but only by those bearing a relationship to the correct one (e.g., Chapman 1961) led scientists to consider Broadbent’s (1958) model as insufficient to account for the disorder and to seek alternative explanatory frameworks. Although for a number of years several response based theories were proposed (e.g., Shakow 1963; Broen, 1968) it was Broadbent’s (1971) revised model that could accommodate the finding that representation of information appears to be processed before any selection processes act upon it. In this version of Broadbent’s theory, a distinction is made between stimulus set (filtering) and response set (pigeonholing). According to the model, at the filtering stage only the characteristics necessary to classify the stimulus as belonging to a particular category are processed. In this context the filter was proposed to act on a probabilistic rather than on a binary basis as previously postulated. In the pigeon-holing stage response biases deriving from semantic cues are attached to certain categories of responses so that output can be triggered on the basis of minimum perceptual input reducing this way information processing demands. On the basis of this model, when irrelevant information is presented to the system, this information is not excluded but it is attended and will reach awareness depending on its nature or the circumstances by which it is presented as determined in the stage of pigeon holing. In this context the response biases attached to categories refer to the integration between current stimulus input and stored memories of previous regularities and its influence on perception.

The concept of pigeon holing which has been described by Broadbent (1971) as a stage to his proposed model for normal information processing has been useful in schizophrenia research for setting the criteria for responses and for its applicability to a variety of processing models that have been utilised to investigate attentional deficits at the response stage of selection in relation to schizophrenia (e.g., Pogue-Geile and Oltmanns 1980; Hemsley and Zawada 1976; Hemsley and Richardson 1980; Harris et al. 1990).
1.2.2.3 Modern Theories of Attentional Processing in Schizophrenia

Predominantly derived from Broadbent’s (1971) model, several theories of attentional processing were advanced and research in the area of schizophrenia has been focused in the investigation of the disorder within the context of these theories.

A theoretical formulation that has been considered relevant to the schizophrenic condition was the concept of automatic/controlled or automatic/conscious processing developed by Schiffrin and Schneider (1977) and Schneider and Shiffrin (1977) where a distinction between automatic and controlled processing is proposed. According to the model, normal automatic processing refers to the activation of fixed sequence of mental operations in response to a particular input configuration. This type of processing is established by training and as it occurs outside conscious awareness, it does not require attention or processing capacity. Controlled processing however refers to voluntary temporary sequences of mental operations and because it occurs within the conscious awareness it requires attention and involves demands on the processing capacity.

Although the state of automatic processing in schizophrenia has not been investigated to any great length (e.g., Larsen and Fromholt, 1976; Oltamnns, 1978; Koh and Patterson, 1978), a closer examination of the above model indicates that the inhibition of awareness during automatic processing plays an important role in reducing processing demands on a system with limited processing capacity. Elaborating on the above, Knight (1984) argued that any dysfunction in the automatic processing would lead to greater demands on the controlled processes. Since controlled processes are generally limited to the processing of novel tasks, any increased dependency on this type of processing which occurs sequentially and within conscious awareness would have an impact on the overall performance on tasks with increased demands.

The assumption that schizophrenia is associated with a dysfunction in the automatic processing has received the support of a number of theorists who working on similar lines have also proposed that a dysfunction at this level would lead to the processing of all stimuli at a consciously controlled level (Venables 1984) and therefore,
irrelevant information will enter into consciousness (Dixon 1981). In accord with the formulation that deficits in the automatic processing would lead to greater reliance on controlled processing and therefore to an impairment in the overall performance of people with schizophrenia (see Spring et al. 1991 for a review) are the findings reported by several researchers (e.g., Granholm et al. 1991) and the reports of newly admitted schizophrenics who stated that "everything seems to grip my attention, although I am not particularly interested in anything" or "I have to do everything step-by-step, nothing is automatic now" (McGuie and Chapman 1961).

1.2.2.4 Frith's Theories of Schizophrenia

Frith (1979) working along similar theoretical lines pointed out that these theories although important in providing the framework by which to investigate cognitive deficits in schizophrenia, they fail to provide important information that would enhance our understanding of the disorder. Elaborating on that he stated that the proposed models although partially successful in explaining some of the cognitive disorders that are present in schizophrenia, they failed to account for the principle positive symptoms of the disorder. In response to these criticisms and drawing on the Freudian terms of conscious and unconscious, Frith (1979) pointed out the relevance of these terms to cognitive processes explaining that unconscious cognitive processes are the processes that are carried out outside awareness and are not open to introspection and conscious those that operate within the level of awareness. Within the unconscious greater emphasis is placed to the set of processes which although operating outside awareness they were or they can become conscious with appropriate stimulation. These processes are referred to as the cognitive equivalent of the Freudian preconscious and drawing on available experimental evidence (e.g., Allpert et al., 1976) he postulates that these processes which include both perceptual inputs and motor outputs occur within a proposed "brief high capacity iconic store [that exists] below the level of consciousness" (Frith, 1979, p.226).
In addition to the difference mentioned above, Frith (1979) based on earlier research emphasises that conscious and preconscious processes differ in other important ways too. Conscious is described as a limited capacity process, information is dealt in a serial manner and that only one meaning of a stimulus can reach awareness at any one time. With regard to preconscious, there is evidence to suggest that it is a larger capacity process and information processing is carried out in parallel. Finally, preconscious processing is described as automatic and lacking flexibility because in this type of processing “the repertoire of appropriate stimuli and response is already known and there is a fixed relationship between stimulus and response”. In contrast to preconscious processes, conscious processes are flexible, the relationship between input and output is not fixed and the performance of any novel task is carried within the level of awareness. According to this explanation a skill or task can be transferred gradually from conscious to the automatic preconscious process when a repertoire of responses to the particular stimulus is acquired.

Taking into account the cognitive formulation discussed, Frith (1979) proposed that the basis of the cognitive deficit evident in schizophrenia is an “awareness of automatic processes which are normally carried out below the level of consciousness” (Frith, 1979, p.233). However, on this model Frith (1979) emphasises that schizophrenia is not associated with a generalised fault in automatic processing (see Venables 1984) but rather with the inability to inhibit the product of automatic processing due to a breakdown in the mechanism that controls and limits the contents of consciousness. This formulation seems to share a number of features with the descriptions of newly admitted schizophrenics who were reported to have lost their automaticity and to experience an increased awareness of every movement composing their actions (McGhie and Chapman 1961). Also in accord with the model is the observation made by several researchers that schizophrenics are slow processors (Yates, 1966; Granholm et al., 1991; Hirt and Pithers, 1991). Elaborating on Frith’s (1979) formulation, this deficit in performance can be attributed to the fact that fast parallel processing that under normal circumstances is carried out below the level of awareness opens to introspection and is influenced by slow serial processing. As a
result, individuals become aware of the different interpretations that words or events can have and therefore in cases where people with schizophrenia have to respond to specific task requirements they experience greater difficulty than their non-schizophrenic counterparts in selecting and carrying through the right course of action (McKenna 1997). This explanation seems to indicate that the inhibitory mechanism assumed by the model acts as a selective filter which is placed at the later stages of attentional processing determining which items of information in preconsciousness should enter consciousness facilitating this way the focussing ability of conscious attention.

In addition to the above, Frith (1979) also proposed that the increased awareness of automatic processes due to a breakdown in cognitive inhibition could be associated with the emergence of positive symptoms central to schizophrenia. According to him in the normal course of events both internal and external sounds are represented first in the preconscious and then, after the incorrect early representations are rejected the relevant sounds reach consciousness as meaningful message(s). In people with schizophrenia however, due to a deficit in the inhibitory mechanism, early erroneous interpretations, that under normal circumstances would not have entered consciousness, reach awareness leading individuals to experience them as voices speaking to them and hence the auditory hallucinations. Similarly, due to a breakdown in the inhibitory mechanism that controls and limits the contents of consciousness a large amount of preconscious information reach consciousness. Since any arrival of information to consciousness is normally considered significant and needs to be accounted for individuals will start employing normal reasoning to account why he/she notices these events, this way giving rise to delusions. Finally, positive thought disorder which is usually inferred from problems in speech and language is considered to be the result of the inability of people with schizophrenia to inhibit the product of attentional processing that normally occurs at preconsciousness. As a result they become aware of the alternative meanings of words that are not normally available to consciousness leading to an inappropriate speech production.
In recognition of the fact that his model could not account for the specific nature of delusions and hallucinations and its weakness to account for the negative symptomatology, Frith rejected his 1979 formulation that the positive symptoms of the disorder are due to a deficit underlying perceptual input and proposed that schizophrenic symptomatology is the result of a defect in the area of output (Frith, 1987; Frith and Done, 1988).

Elaborating on William James' (1890, see Cahill and Frith, 1996) definition of "willed actions" which are described as the actions we deliberately choose from a number of other possibilities and distinguishing them from the automatic actions which are performed by ourselves unintentionally, Frith (1987) proposed a model that incorporated these classes of actions and described how a dysfunction in this system could lead to the emergence of schizophrenic symptoms. According to Frith (1987) central components to the model are the two routes to action namely the stimulus driven or automatic action and the internally driven or willed action. These actions are monitored by a hypothesised self monitoring system whose main function is (a) to distinguish events caused by our own actions from those resulting from the actions of others and (b) to distinguish actions resulting from our own goals and plans (willed actions) from those that are stimulus driven (automatic responses). This monitoring process is depicted in diagram 1.

In order to emphasise the value of the idea of self monitoring described in the model, Frith (1987) suggested that this is the neurocognitive equivalent of the physiological mechanism applied to eye movements which is termed "corollary discharge". According to descriptions of the function of this mechanism the main aim of corollary discharge is to inform a central monitoring system of the intention to make an eye movement so that it can compensate for the anticipated movement. This enables us to distinguish between a moving retinal image caused by us (self-generated action) versus the one caused by moving objects in the outside world (stimulus driven action).
Diagram 1  The key components of Frith’s revised model of schizophrenia

Source:
On the basis of the above analogy and by referring to Feinberg's (1978) suggestion that apart from the eye and limb movements corollary discharge may be applied to covert actions such as thinking, Frith (1987) proposed that our perception of the world can be modulated through a cognitive process of self monitoring that functions in a similar manner to that of corollary discharge. Moreover, he stated that the positive symptoms of schizophrenia, or as what he collectively terms “passivity experiences”, are caused by a dysfunction in this monitoring system. He suggested that when willed intentions are not adequately monitored, the individual fails to recognise these actions as self-initiated and attributes them to an external source. Within this framework hallucinations are viewed as the result of the patient’s inner speech (see Johnstone 1978) which instead of being experienced as a self initiated internal action is perceived to have been caused by an external agent.

With regard to delusional experiences Frith (1987) proposes that all thought processes normally involve some sense of effort or intention which he considers to be stemming from the monitoring system. When this central monitoring system breaks down the patient loses the sense of intention and consequently, thoughts can be experienced as alien or implanted into his/her head by an external source leading into delusions of control. Finally, a deficit in the ability to monitor the intentional actions of others could lead to other types of schizophrenic symptoms such as paranoid delusions and delusions of reference (Cahill and Frith, 1996). In addition to positive symptoms, Frith’s (1987) model also attempts to account for the negative symptoms of the disorder. This set of symptoms however, are not attributed to any deficiency in the monitoring system but rather the result of a dysfunction in the willed intention component. Due to a dysfunction at this level the patient experiences problems in producing spontaneous behaviour in the absence of external stimuli and shows poverty of action.

The information reviewed thus far indicates that Frith’s revised model attempts to account for the entire spectrum of schizophrenic symptoms through three types of deficits: (a) failure to monitor the intentions of self leads to hallucinations and delusions of control, (b) failure to monitor the intentions of others to symptoms such
as delusions of reference, third person auditory hallucinations and paranoia and (c) a
dysfunction of willed intentions leads to negative symptomatology. However the
most important innovation of this model over his earlier one is that Frith (1987)
having produced cognitive theories concerning the symptoms of the disorder he
attempted to account for how these theories might be expressed in terms of brain
function (see Frith, 1992)

Finally, in an updated version of the model, Frith (1992) proposed that most of the
negative symptoms of schizophrenia can be described as behavioural abnormalities
and therefore it would have been more appropriate to be referred as “behavioural
signs” instead of negative symptoms. In support of this view he referred to several
studies pointing to the difficulties in assessing this cluster of symptoms (Lewine,
1985) as opposed to positive symptoms emphasising at the same time that as a
primary feature of the disorder negative symptoms are associated with distinct
structural brain abnormalities (e.g., Andreasen et al., 1982). On the basis of this view
he proposed the subdivision of “behavioural signs” into two groups which he termed
“positive signs” (incoherence of speech, incongruity of affect and stereotypies) and
“negative signs” (poverty of speech, flattening of affect, retardation and social
withdrawal). Frith’s (1992) inclusion of positive (incoherence of speech and
incongruity of affect) and negative symptoms (stereotypies) into the same group
appears to be quite consistent with Liddle’s (1987) three-factor theory which indicate
that that these traditionally positive symptoms in factor analytic studies they form a
separate cluster than delusions and hallucinations.

1.2.2.5 Hemsley’s “Weakening of the Influences of Regularities of Previous
Input on Current Perception”

Hemsley (1987), in his attempt to integrate the perceptual organisation deficit seen in
schizophrenia with the attentional impairment described by several theorists, has
proposed a comprehensive cognitive model where a significant role is attributed to the
perceptual/cognitive abnormalities in the emergence of the disorder. In prefacing the
theory, Hemsley (1987) stated that despite the lack of an agreed model of normal
information processing, the majority of information processing theories (e.g., Broadbent, 1971; 1977; Schneider and Shiffrin, 1977; Shiffrin and Schneider, 1977; Posner et al, 1982) at their basic level illustrate how “spatial and temporal regularities of past experiences influence the processing and awareness of current sensory input” (Hemsley, 1987 p.182). Linking this view with the observation that most theoretical approaches to schizophrenic cognitive impairment suggest either a disruption to cognitive performance due to an intrusion of material that normally exists below awareness (Frith 1979; Cutting 1985) or relate to the weakening of the influence of spatial and temporal regularities on perception (Venables 1984; Knight, 1984; Maher, 1983; Hemsley, 1987; Magaro 1984) he proposed that:

It is a weakening of the influences of stored memories of regularities of previous input on current perception which is postulated as basic to the schizophrenic condition  (Hemsley, 1987 p.182)

Hemsley’s (1987) model is predominantly based on the limited capacity system model of normal cognitive functioning in which awareness of irrelevant or redundant information is inhibited in order to reduce information processing demands on the system (Underwood, 1976). The gradual inhibition of awareness of irrelevant information is seen as the result from the change from controlled to automatic processing with the later not giving rise to awareness (Schneider and Shiffrin, 1977; Shiffrin and Schneider, 1977; Posner et al, 1982).

As discussed in 1.2.2.2, Broadbent’s (1971; 1977) model of selective attention makes a distinction between stimulus set (filtering) and response set (pigeon holing), with the later mechanism acting as a bias towards certain categories on the expense of others. According to Hemsley (1987) the “response biases” stem from the interaction between stored memories of past spatial and temporal regularities and the current context by means of top-down processing. On the basis of the above, Hemsley’s (1987) model suggests that in people with schizophrenia the employment of top-down processing, in which new stimuli are compared with stored memories of past spatial and temporal regularities for their interpretation, is reduced. This failure to make use of the redundancy and the patterning of sensory input in the process of integrating
current stimuli with stored regularities for reducing processing demands in a limited capacity system is attributed to a deficit of a general inhibitory process which is important to conscious attention. As a result of the failure to inhibit redundant perceptual details, information that usually exists below consciousness intrudes into awareness causing the individual to experience percepts by their individual components rather than in a Gestalt fashion, a view that appears to be compatible with the reports of patients' experiences (McGuie and Chapman 1961; Matussek 1952) which indicate that they pay excessive attention to detail. According to Gray and his colleagues (1991), excessive attention to detail can be linked to the phenomenon of “overattention” which is described as being the result of “weakening of the capacity to select for cognitive processing only those stimuli that, given past experience of similar contexts, are relevant” (Gray et al., 1991, p.3). Continuing, the same authors define the term relevant as “… the subject's current plans or motor programs” (p.3).

On the basis of the above, Hemsley (1993) points out that his proposal that “[basic to schizophrenia] is the weakening of the influences of stored memories of regularities of previous input on current perception” (Hemsley, 1987 p.182) does not suggest “that the memories of past regularities are stored, nor that they are inaccessible” because according to him they can be “accessed by consciously controlled processes” (Hemsley, 1993 p.635). What he suggests however, is that it is “… the rapid and automatic assessment of the significance or lack of significance of aspects of sensory input (and their implication for action) that it is impaired” (Hemsley, 1993, p.635) and that schizophrenia is “… a disturbance in the moment by moment integration of stored material with current sensory input” (Hemsley 1994, p.164). As a result of this disturbance in the way by which past regularities influence current perception, individuals would experience the intrusion of irrelevant or redundant information into consciousness as well as making more inferences about the causal relation of events. Hemsley's (1987; 1993) view that people with schizophrenia are less influenced by past regularities than normal controls seems to be supported by several studies (e.g., Jones et al., 1991; Peters 1993) which indicate a deficit in the integration of current sensory input with previous experiences.
As suggested by the model, people with schizophrenia are less able than normals to inhibit attention to irrelevant aspects of their environment and hence fail to make use of the redundancy and patterning of sensory input in order to reduce information processing demands. Although the general experience with schizophrenics is that they perform poorly in most experimental tasks, Hemsley (1987) suggested that on the basis of the above it might be possible to construct tasks where the performance of people with schizophrenia could be superior to that of their normal counterparts. More specifically, he proposed that if people with schizophrenia are unable to make use of the redundancy and patterning of sensory input then it is possible that in tasks where the automatic selection process of normals are not adaptive, schizophrenics should be able to perform better. In support of this view, Hemsley (1987; 1993; 1996) refers to two experimental paradigms deriving from the animal learning theory and which can be "...regarded as instances of the influence of stored memories of regularities of previous input on current perception" (Hemsley, 1996, p. 144). These experimental paradigms are the Latent Inhibition (LI; Lubow et al., 1982) and Kamin's Blocking Effect (KBE; Kamin 1969).

Both LI and KBE appear to fulfil the cognitive requirements of Hemsley's (1987) proposition that the basic deficit in schizophrenia lies in their inability, based on past experiences, to select only relevant information. In a more general framework the research findings seem to indicate quite consistently that both tasks are sensitive to the same cognitive and neurochemical processes. More specifically available evidence suggests that in the animal laboratory both LI (e.g., Solomon et al, 1981; Warbuurton, et al., 1994) and KBE (e.g., Solomon et al, 1981) are disrupted by the administration of the dopamine releasing drug and that the disruption is reversed by the administration of neuroleptics (Crider et al, 1982; 1986; Feldon and Weiner, 1991). Moreover there are findings to suggest that the same disruption could be found on the hippocampally lesioned animal (e.g., Solomon et al, 1977). Consistent with animal studies are the reports of a similar disruption in the performance of acute but not in chronic schizophrenics as well as in normal volunteers following administration of low doses of the dopamine releasing drug amphetamine. These findings, although compatible to the hypothesis that acute schizophrenics are in a hyperdopaminergic
state and that their performance in these tasks can be interpreted within this context, detailed examination of these results indicate that there are several important issues that need to be addressed. For example, in contrast to early findings which showed that LI is disrupted in acute but not in chronic schizophrenics (Baruch et al., 1988) subsequent studies indicated that this disruption is more relevant to the stage of the illness rather than to the severity or the type of symptoms (N.S. Gray et al., 1992). Also, in a more recent study where the KBE was investigated in normal controls following administration of amphetamine the results did not support the prediction of a disrupted KBE in this group of subjects (N.S. Gray et al., 1997). These rather contradictory findings raise some serious questions regarding the methodology employed and the way by which findings were interpreted pointing out to the need for further research not only in terms of replicating previous studies but also in investigating the performance of similar groups of subjects on other cognitive tasks with similar characteristics.

According to Chapman and Chapman (1973), any theory of schizophrenic cognitive functioning should be able to give an account of the unusual experiences of people with schizophrenia. Hemsley’s (1987) model is consistent with this view as he proposes an explanation of the way by which schizophrenic symptoms can be related to his theory (see diagram 2).

At the beginning of his account of the link between his model and schizophrenic symptoms, Hemsley (1993) draws attention to the role of abnormal experiences in the overall development of the disorder. Specifically he refers to the study reported by Klostertottler (1992) which appears to support the suggestion of Hauber that abnormal experiences of thought, action, perception and bodily sensations early in the disorder could be transformed eventually into schizophrenic symptoms. This proposal seems quite compatible to Maher’s (1988) view which emphasises that abnormal perceptual experiences play a significant role in the formation of delusions. Moreover, Hemsley (1993) refers to the work of Einhorn and Hogarth (1986) who suggest that normal people engage in causal reasoning in order to make sense of the world with
Diagram 2  Hemsley’s (1987) model of cognitive abnormalities and symptoms of schizophrenia

Reduced influences of regularities of past experience on current perception

Reduced ability to make use of redundancy and patterning of input on cognitive tasks

Ambiguous unstructured sensory input

Heightened awareness of redundant stimuli

Intrusion of unexpected/unintended material from long-term memory

Preference for and reduced symptoms in highly structured predictable environment

Delusional beliefs

spatial and temporal contiguity play a significant role in their conclusion of a causal relationship between events. Linking the above to the model, Hemsley (1993) proposes that a weakened influence of past regularities of current perception not only would result the intrusion of redundant material into awareness which in turn would lead the individual to search for their meaning, but also it will influence the assessment of the co-occurrence of two events. Since spatial and temporal contiguities play a significant role in considering events as being causally related, then abnormal causal relationships may be inferred on the basis of a single co-occurrence. Continuing, he proposes that positive thought disorder stems from abnormal associations between current contextual (both spatial and temporal) and stored information. As a result of these abnormal associations attention is directed towards incidental details of the percept resulting in the deviation from the current motor programme (Gray 1991).

In relation to hallucinations Hemsley (1987; 1993) refers to the findings of sensory deprivation studies which indicate that the lack of unstructured sensory input could play significant role in giving rise to hallucinatory experiences in normal individuals. In accounting for these findings, George and Neufeld (1985) have suggested that this phenomenon occurs because long term memory and sensory processing work in a reciprocal inhibitory manner. More specifically it was suggested that in the absence of normally patterned sensory input thoughts and ideas from long term memory begin to intrude unexpectedly into consciousness giving rise to hallucinations (Jakes and Hemsley, 1986). Linking these findings to his model Hemsley (1987; 1993) proposes that the weakening of the influences of past experiences and regularities on current perception would result to an ambiguous, unstructured sensory input. Therefore, hallucinations in people with schizophrenia could be seen as analogous to the experiences of normal subjects in sensory deprivation experiments.

As indicated above, heightened awareness of perceptual input that would not normally reach consciousness leads to the emergence of abnormal perceptual experiences. The attempts made by the individual to rationalise this divergent information and to account for the abnormal experiences constitute the basis for delusions. Hemsley
(1993) proposes that delusions can be maintained when deluded individuals refuse to consider information that is incompatible to their delusional system. The indication however, that deluded individuals fail to use background information (Ancombe 1987) or to consider information incongruent to the delusional system (Hemsley 1990) seems to be in contrast with Maher's (1988) view which suggests that delusions are the product of the application of normal reasoning into accounting for abnormal experiences. In support of the suggestion that deluded individuals tend to employ abnormal as opposed to normal patterns of reasoning, Hemsley (1993) refers to a number of studies which seem to confirm this view (e.g., Garety et al. 1991; for a review see Garety and Hemsley 1994).

According to McKenna (1997) "... the application of [cognitive] theory to negative symptoms is due primarily to Hemsley" (p.168). Hemsley (1993) although acknowledges the difficulties associated with research into negative symptomatology, he proposes that negative symptoms such as poverty of speech, social withdrawal and retardation are developed by people with schizophrenia as adoptive strategies in response to their state of information overload. In response to the criticism that positive symptoms do not always precede negative symptoms (Frith 1992) Hemsley (1993) argued that they may develop as a coping mechanism to early subtle disturbances of thinking and perception which according to Hauber and his colleagues (1986) could progress to schizophrenic symptoms (Klostertotter, 1992). This explanation however, does not account for those instances where the two types of symptoms occur concurrently nor does it explain the role of symptoms such as stereotypy of content that do not seem in any obvious way to reduce stimulation. In defence of the model however, it is important to emphasise at this stage that the main body of Hemsley's theory is predominantly concerned with the positive symptoms of the disorder and with only secondary reference to the negative symptomatology.

An important aspect of Hemsley's (1987) theory is that it constitutes an integral part of Gray's and colleagues (1991) neuropsychological model of schizophrenia which at a basic level it suggests that the cognitive deficit proposed by Hemsley to play a significant role in the emergence of positive symptoms correspond to an underlying

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neural dysfunction. Based on evidence that indicates a link between schizophrenic symptoms and pathology of the limbic system (e.g., Schmajuk, 1987; Bogerts et al., 1991) he proposed that damage to the hippocampus and related brain structures may be responsible for the cognitive deficit similar to the one described in his model (Hemsley, 1993; 1996).

More specifically, the Grays and colleagues' (1991) neuropsychological model of schizophrenia propose the presence of a neural circuit responsible for the moment-by-moment generations of predictions of subsequent sensory input with the hippocampus playing a significant role in this monitoring of whether actual and expected stimuli match. When an external stimulus matches with information already present in the comparator then the nucleus accumbens interrupts the ongoing motor programme. According to Gray and his colleagues (1991) positive symptoms of schizophrenia arise due to a defect in this circuit which as a result of the failure to identify the match between external stimuli and those already present in the comparator, the motor programmes are not interrupted and individuals continue to perceive familiar stimuli as though they are new to them. An elaboration of this theory seems to suggest that people with schizophrenia fail to relate current sensory input to stored regularities, and as Hemsley (1987; 1993) proposed a disturbance at this level could lead to the emergence of positive symptomatology.

1.2.3 Conclusion on the Aetiology of Schizophrenia

As discussed in section 1.2.1.1 the most dominant theory in the neurochemical domain is the dopamine hypothesis. However, the evidence supporting this hypothesis has been described as circumstantial whereas any attempts to provide direct evidence in its favour has yielded rather inconclusive results. In contrast to the failure to provide conclusive evidence that would indicate that schizophrenia is associated with an elevation in the amount of dopamine cell receptors and terminals in areas that are rich in dopaminergic cells, more recent findings seem to indicate quite consistently that schizophrenia is associated with abnormalities in a number of areas
in the temporal lobe. In the psychological domain, schizophrenia research has been predominantly guided by the cognitive theory which at its most basic level suggests that regardless of what causes schizophrenia the underlying pathological processes must affect one or more cognitive processes.

As a result of the proposed relationship between cognition and schizophrenia, several models have been suggested culminating in the cognitive models of schizophrenia proposed by Hemsley (1987; 1991) and Frith (1987; 1992). Hemsley's (1987; 1991) model proposes that schizophrenia, and more specifically the positive symptoms of the disorder are associated with disturbances in the attentional processes. According to him, this disturbance occurs in the connection between Long Term Memory and the generation of action regardless of whether the action is stimulus elicited or self-generated. In Frith's (1987; 1992) model the emphasis is on internal monitoring and planning of action. On the basis of this model, positive symptoms emerge as a result of a disturbance in the route that links willed intentions and the monitor, whereas negative symptoms are considered to be the result of a disruption in the route that links willed intentions and stimulus intentions to the generation of action.

Although both models are considered by their respective authors to reflect the primary cognitive deficit that leads to schizophrenia, each of the models differ from the other on several points. Frith's (1987; 1992), for example, distinguishes between stimulus-driven and self-generated actions and within the self-generated actions he makes the further distinction between monitoring and execution of willed intentions. Within the context of the descriptions of these different routes of action Frith (1987; 1992) provided a distinction between positive and negative symptoms in terms of their cognitive and neural bases while at the same time he gave negative symptoms an equal status, if not more important, to that of positive symptoms. On the other hand, Hemsley (1987; 1991) proposes within his formulation that negative symptoms are secondary to positive symptomatology and are thought to emerge as adaptive strategies to a state of information overload. But despite their differences, the models also share several common characteristics. For example, both models consider
hyperdopaminergic activity to play a crucial role in the emergence of positive symptoms. Also, they propose ways by which to integrate psychological and biological findings in order to account for the positive and, to a certain extent, the negative symptoms of the disorder. Of course when considering the basis from which these two models have developed it seems that they had more in common in the past than in the present. Hemsley’s (1987) model and Frith’s (1979) theory of an “awareness of automatic processes which are normally carried out below the level of consciousness” (Frith, 1979, p.233) were based on the proposal that the cognitive deficits relevant to schizophrenia are due to a defective filter. However, as discussed in section 1.2.2.4, in a revised version of the theory, Frith (1987; 1992) rejected his initial formulation and proposed that schizophrenia is associated with a dysfunction in the monitoring of willed intentions and in the generation and control of action.

But above all considerations, it would have been more appropriate, when referring to Hemsley’s (1987; 1991) and Frith’s (1987; 1992) models, to consider them not as conflicting but as complimentary to each other describing different aspects of the same dysfunction. In fact, Gray and his colleagues (1991) in their attempt to provide a comprehensive hypothesis about the emergence of positive symptomatology, by integrating neurochemical, neuropathological and cognitive data, have incorporated in their neuropsychological model the cognitive theories developed by Hemsley and Frith due to their compatibility in their proposed neural machinery.

1.3 Reversal Learning

The experimental part of the present thesis is predominantly concerned with the investigation of Hemsley’s (1987; 1991) model which proposes that basic to schizophrenia “is a weakening of the influences of stored memories of regularities of previous input on current perception” (Hemsley, 1987 p.182). As discussed earlier, one of the most important components of Hemsley’s (1987; 1991) theory is that he refers to two behavioural phenomena namely, the LI and KBE that have been originally developed in the animal laboratory. According to Hemsley (1987; 1991),
an elaboration of these paradigms can be considered as "instances of the influence of stored memories of regularities of previous input on current perception" (Hemsley, 1996, p. 144) and may be viewed as indicative of the way by which subjects inhibit attention to a predictable redundant stimulus. On transferring the tasks to the human laboratory employing clinical populations as well as normal volunteers although produced rather encouraging results they also pointed out that any conclusions drawn about the cognitive deficit proposed by Hemsley (1987; 1991) needs to be preceded by further research. The main purpose of the present study will be to investigate the cognitive deficit proposed by Hemsley (1987; 1998) by employing a different experimental task to the ones already described by the above authors. The tool of investigation chosen for this study is the Reversal Learning (RL; Lawrence, 1949) task because, like LI and KBE, it has been shown in the animal laboratory (e.g., Weiner and Feldon 1986) as well as in human studies (Nolan 1974; Kamis and Muzekari 1973) to depend on the control of attention by prior experience and therefore can facilitate direct comparisons of the extend by which prior experiences influences new learning in people with schizophrenia.

1.3.1 Background to Reversal Learning

The behavioural phenomenon of "Reversal Learning", which will be reviewed in this section in some detail due to its direct relevance to the experimental part of this study, was originally developed in the animal laboratory by Lawrence (1949) and was subsequently used and further developed by other researchers (e.g. Sutherland and Holgate 1966; Mackintosh 1965).

In a typical reversal learning task, the animal is initially rewarded for one response (e.g. going to the lighted arm of a T-maze) in a two choice paradigm. After a learning criterion has been reached, the reward is given in a subsequent phase for the alternative response (e.g., going to the dark arm). In accounting for the processes involved in this behavioural phenomenon, Jones and Mishkin (1972) have proposed that discrimination reversal learning in animal studies can be explained through the three-stage theory.
Based on the theory it has been suggested that when reward contingencies change it is necessary for subjects in the first stage to undo the old contingencies. In the second stage subjects' performance is characterised by chance responses while the new associations are being formed and in the third stage, the actual shift at the outset of reversal, the new associations are established.

In an alternative explanation provided by Mackintosh (1983; Mackintosh, 1965; Sutherland and Mackintosh 1971), it was proposed that reversal learning can be explained within the framework of the two-stage theory. Elaborating on the above, it was suggested that discrimination learning involves several processes most important of which are the excitatory conditioning of the positive stimulus and inhibitory conditioning of the negative. The positive stimulus refers to those instances where its presentation signals the availability of reinforcement and negative stimulus signals the absence of reinforcement. Reversal learning, involves two specific processes, the extinction of the original discriminative response and the acquisition of a new discriminative response. According to these researchers, the process of extinction of the original discriminative response reflects the difference in the associative values between positive and negative stimuli. When, for example, subjects are rewarded consistently whenever they select a particular stimulus, the excitatory strength of that stimulus is increased whilst the inhibitory strength of the alternative stimulus is also increased. Due to this increase of the excitatory strength of the positive stimulus, at the outset of reversal (when the positive stimulus will become negative and the negative positive) subjects will persist to select the original positive. The shift to the new positive stimulus is considered to reflect the attention to the relevant stimulus and occurs when the associability of the relevant stimulus and the reinforcement is increased. The second process, which is described as the acquisition of a new discriminative response, is considered to reflect the attention to the relevant stimuli. Within this process, as the associability of the relevant stimuli is increased, a more rapid shift of choice to the new positive stimulus is observed (Weiner and Feldon 1986).
The findings that adults reverse faster than children and animals led Kendler and Kendler (1969) to formulate a "mediating response theory" of discrimination learning. Based on their theory they proposed that the differences observed in the performance of different groups in reversal learning tasks are due to differences in mediating responses, the later described as "... a developmental progression from a single unit process to a more complex mediational process that involves symbolic representation of sets of discrete stimuli" (Kendler and Kendler, 1970, p.70). Since, according to the above authors, mediatonal responses develop as a correlate of human growth, when mediating responses occur subjects shift relatively fast at the outset of reversal learning. This is because the same mediational response learnt in the initial discrimination is still effective during reversal.

In responding to this formulation, Mackintosh (1965) argued that the concept is vaguely described and if the reversal is indeed influenced by mediating responses then one would expect significant differences between adults and children in the number of trials required to learn the initial discrimination. However, he emphasised in his response that "there is no evidence that older children and adults are notably superior at solving simple discrimination problems to these subjects" (Mackintosh, 1965, p. 144)

1.3.2 Reversal Learning and Schizophrenia

Despite the several theoretical formulations proposed in order to account for the processes involved in this behavioural phenomenon, reversal learning can be also considered to reflect the way by which inhibited attention to a predictably redundant stimulus can influence subsequent learning when the redundant stimulus becomes relevant to the task. Following from the above, in a two choice reversal learning task (A-B) the subject is rewarded for response A but not for response B. When the initial discrimination is acquired and the reward contingencies alter so that reward at the outset of reversal is associated with response B but not with A, subjects influenced by their previous learning, will continue for a period of time to select response A. Linking this to Gray's and colleagues (1991) and Hemsley's (1987; 1991) theories which propose
that basic to schizophrenia is their failure to learn from previous experiences, one would expect that people with acute schizophrenia will be less influenced by previous regularities and consequently will reverse faster than non-acute schizophrenics.

The reversal learning paradigm in relation to the acute schizophrenia has been investigated in animal studies as well as in studies employing clinical populations and normal volunteers. Evidence produced in the animal laboratory has shown that amphetamine administration dramatically facilitates reversal learning. (e.g., Calhoun and Jones 1974; Weiner and Feldon 1986). These findings were interpreted by researchers as indicating that amphetamine decreases the effects of phase 1 learning on subsequent cognitive processing. Since the amphetamine treated rat has been proposed to constitute an animal model for schizophrenia (e.g., McKinney and Bunney, 1969; Randrup and Munkvard, 1967), these findings are considered as indirect evidence that supports the association between acute schizophrenia and faster reversal.

However, in studies where the performance of normal controls on reversal learning tasks was compared to that of schizophrenics have yielded rather conflicting results. In some of these studies there was evidence to suggest that schizophrenics shift faster than normal controls at the outset of reversal (e.g., Nolan 1968) whereas in others, no difference was reported (e.g., Nolan 1974; Kamis and Muzekari 1973). A closer examination of the characteristics of subjects employed in these studies though indicates that none of these studies have adopted the positive versus negative symptoms (Crow, 1980) nor the acute versus chronic (e.g., Spitzer et al. 1978) distinction that is so important in Gray’s and colleagues (1991) and Hemsley’s (1987; 1991) formulations. Therefore, drawing conclusions on the basis of studies (e.g., Nolan 1968) where the symptomatology of the groups of subjects employed is not so well defined can be problematic.

As discussed in section 1.2.2.5, Hemsley (1993) and Gray and colleagues (1991) models consider damage to the hippocampus to play a significant role in the emergence of positive symptoms. Studies investigating the performance of animals as
well as humans in the LI and KBE tasks following hippocampal damage have shown similar disruption as the one reported in the animal laboratory after amphetamine administration and in studies employing acute schizophrenics. In contrast to the consistency of findings reported for the LI and the KBE, the evidence produced in animal as well as in human studies have indicated that hippocampal damage is associated with impaired reversal learning (e.g., Mahut 1971; Jones and Mishkin 1972; Daum et al. 1991). However, a more detailed examination of the findings produced by these studies also point out to several methodological problems. In most of the studies investigating the performance of hippocampally damaged humans and animals the damage sustained by the subjects is global whereas the hippocampal dysfunction proposed by the models (Gray et al., 1991; Hemsley, 1992) is very specific and refers to the neural projection from the subiculum to the nucleus accumbens. Although any global damage to the hippocampal region would also imply damage to the specific projection considered by the above authors as relevant to the emergence of positive schizophrenic symptoms, it is possible that the overall performance on a reversal learning task may require components of the hippocampal region to be intact.

1.4 Objectives of Thesis

The main aim of this study is to attempt to investigate the cognitive abnormality underlying the positive symptoms of schizophrenia and more specifically the deficit proposed by Hemsley’s (1987; 1991) cognitive model of the disorder. This theory seems compatible with Gray’s and colleagues (1991) neuropsychological model which proposes that the cognitive deficit suggested by Hemsley corresponds to a neural impairment in the subiculo-accumbens projection. They argued that people with schizophrenia tend to treat familiar stimuli as though they are novel, and attribute that to the absence of a matched message from the subicular comparator.

Research studies investigating these hypotheses although reported some encouraging results, in some of these studies the findings produced were rather inconclusive pointing
out to the need for further research. As mentioned at the opening of this section, the main aim of the experimental part of the present thesis is to investigate the cognitive deficit proposed by Hemsley. This will be done by employing a reversal learning paradigm which although different than the ones already employed in previous studies, it shares the same characteristics as LI and KBE this way enabling us to make direct comparisons of the extent by which prior experiences influence new learning in people with acute schizophrenia. Since this is a comparative study, in addition to people with acute schizophrenia the performance on a reversal learning task of two other groups, chronic schizophrenics (e.g., Spitzer et al. 1978) and normal controls will be also investigated.

1.4.1 Hypotheses

Based on Hemsley’s model, one would expect that if people with acute schizophrenia are indeed less influenced by previous learning, then they would respond to new stimuli faster than chronic schizophrenics and normal controls. In order to test this prediction the following hypotheses will be tested:

(i) People with acute schizophrenia will require fewer trials to shift at the outset of reversal than chronic schizophrenics and normal controls, and

(ii) The number of trials taken by acute schizophrenics to reverse in relation to their initial learning (RL/L) will be significantly smaller than the number of trials taken by chronic schizophrenics and normal controls.
2. METHOD

2.1 Subjects

Three groups of subjects were employed in this study, normal controls, chronic schizophrenics and acute schizophrenics. The demographic details of each group are described below:

2.1.1 Normal Controls

A total of 43 subjects (22 males and 21 females) were tested in this group. Three subjects (one male and two females) were discounted due to their failure to acquire the initial learning discrimination. Therefore, only the results obtained by 40 subjects (21 males and 19 females) were used for statistical analysis. Twenty-one of the subjects were people who responded to advertisements placed in the Camberwell and Wood Green Job Centres and Haringey Library, seven were clerical staff at St. Ann's Hospital and twelve subjects were recruited from the subject pool of the Institute of Psychiatry (a group of adults drawn from the general population who consented to be contacted for research purposes). All subjects were native English speakers and at the time of testing, six subjects were students, eight were unemployed and the rest were employed in various occupations. Subjects with any sight difficulties, a history of mental illness, drug or alcohol dependency were excluded from the study. Table 2.1 shows the demographic data of all subjects employed in this study.

| Table 2.1. Means and Standard deviations (in parenthesis) of age, education and sex of Normal Controls group |
|-----------------|-----------------|-----------------|
| Age (in yrs)    | 37.07 (9.84)    | n=40            |
| Yrs of Education| 12.80 (2.74)    | n=40            |
| IQ (Progressive Raven Matrices) | 101.07 (5.46) | n=40 |
| SEX             | Males           | 19              |
|                | Females         | 21              |
The mean age of the 40 subjects was 37.07 years, ranging from 19 to 58. Education was calculated by accepting that education up to the age of 16 is 11 years. Any further years of education were added to the basic 11 years. Similarly in older subjects, who left school before the age of 16, the number of years prior to the age of 16 were subtracted from the basic 11 years. The mean education of subjects in years was 12.8 (range 9-18). IQ scores were derived by converting the score obtained by each subject on the Standard Progressive Matrices (Raven, Court and Raven 1988) to IQ. The mean IQ for the whole group was 101.07, ranging from 86 to 111.

2.1.2 Chronic and Acute Schizophrenics

In the group of chronic schizophrenics, 34 subjects (18 males and 16 females) were tested. Twenty-four subjects were patients at St Anns Hospital and eleven were patients at Napsbuiy and Barnet General Hospitals. All subjects were selected on the basis of Research Diagnostic Criteria (Spitzer, Endicott and Robins 1978) by the psychiatrist responsible for their care.

In the group of acute schizophrenics, a total of 35 subjects were tested (20 males and 15 females). Four subjects (three males and one female) were discounted due to their failure to acquire the initial learning discrimination, and therefore only the results obtained by 31 subjects were used for statistical analysis. Twenty-five of the subjects were patients at St Anns Hospital and six were patients at Napsbury and Barnet General Hospitals. All subjects were selected on the basis of Research Diagnostic Criteria (Spitzer, Endicott and Robins 1978) by the psychiatrist responsible for their care. All acute schizophrenic subjects were tested within two weeks of their admission to hospital.

Table 2.2 shows the demographic data of both groups of schizophrenic subjects employed in this study. Statistical analysis of demographic data revealed that chronic schizophrenic subjects were significantly older (t=2.92, d.f.=63, p<0.01), had been in hospital for longer (t=2.67, d.f.=63, p<0.01) and had a greater number of previous admissions (t=3.48, d.f.=63, p<0.001) than their acute counterparts. Acute
Schizophrenics on the other hand were receiving significantly higher daily dosages of medication (t=5.15, d.f.=63, p<0.001) than chronic schizophrenics. Comparison of medication was carried out by converting subjects’ medication at the time of the study, into chlorpromazine equivalents (Davis 1976).

Table 2.2 Means and Standard deviations (in parenthesis) of demographic data of people with chronic and acute Schizophrenia

<table>
<thead>
<tr>
<th>DEMOGRAPHIC DATA</th>
<th>CHRONIC SCHIZOPHRENICS N=34</th>
<th>ACUTE SCHIZOPHRENICS n=31</th>
<th>Significance¹</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (in yrs)</td>
<td>39.35 (8.42)</td>
<td>33.03 (8.96)</td>
<td>***</td>
</tr>
<tr>
<td>Yrs of Education</td>
<td>12.06 (2.27)</td>
<td>11.77 (1.50)</td>
<td>ns</td>
</tr>
<tr>
<td>IQ (Prog. Matrices)</td>
<td>99.29 (5.00)</td>
<td>97.97 (7.19)</td>
<td>ns</td>
</tr>
<tr>
<td>Age of 1st Admission</td>
<td>25.50 (5.74)</td>
<td>23.77 (4.29)</td>
<td>ns</td>
</tr>
<tr>
<td>Previous Admissions</td>
<td>4.68 (2.28)</td>
<td>3.00 (1.57)</td>
<td>****</td>
</tr>
<tr>
<td>Prior Time in Hospital</td>
<td>20.97 (13.27)</td>
<td>12.87 (11.17)</td>
<td>***</td>
</tr>
<tr>
<td>(in months)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Medication (converted into</td>
<td>287.68 (136.99)</td>
<td>328.61 (100.28)</td>
<td>****</td>
</tr>
<tr>
<td>(Chlorpromazine equivalents)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SEX</td>
<td>Males</td>
<td>Females</td>
<td></td>
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<tr>
<td></td>
<td>18</td>
<td>16</td>
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<tr>
<td></td>
<td>17</td>
<td>14</td>
<td></td>
</tr>
</tbody>
</table>

¹ ns p > 0.05     * p < 0.05     ** p < 0.02     *** p < 0.01     **** p < 0.001
2.2 Symptom Measures

Chronic and acute schizophrenic subjects were also rated on two symptom scales, the Brief Psychiatric Rating Scale (BPRS; Overall and Gorham 1962) and the MAINE Scale (Magaro, Abrams and Cantrell, 1981). These scales are described briefly below:

*Brief Psychiatric Rating Scale (BPRS)*: A scale that has been developed by Overall and Gorham (1962) in order to provide a rapid assessment tool that would enable clinicians to evaluate changes in the symptomatology of psychiatric, predominantly schizophrenic, patients. The scale consists of 16 items that can be rated from 1-7 during an interview with the patient. Each of the items corresponds to a discreet symptom area and for increased reliability of the scale its authors recommend that the interview is carried out by two independent raters. A sum of the ratings of all 16 items denotes a total pathology score (see appendix 1).

*The MAINE Scale*: This scale has been developed by Magaro, Abrams and Cantrell (1981). The scale consists of ten items five of which correspond to a nonparanoid (N) subscale and five to a paranoid (P) subscale. The ratings of the items on each subscale can be summed to give two total scores each corresponding to paranoid and nonparanoid schizophrenia (see appendix 2).

| Table 2.3 | Means and Standard deviations (in parenthesis) of Symptom Rating Scales for Chronic and Acute Schizophrenics |

<table>
<thead>
<tr>
<th>GROUPS</th>
<th>BPRS</th>
<th>MAINE</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>a</td>
<td>b</td>
</tr>
<tr>
<td>CHRONIC SCHIZOPHRENICS</td>
<td>14.29</td>
<td>15.56</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ACUTE SCHIZOPHRENICS</td>
<td>31.94</td>
<td>30.65</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Table 2.3 shows the total scores of both chronic and acute schizophrenics in the BPRS and Maine-N and Maine-P symptom scales. The BPRS measures were taken by the psychiatrist responsible for their care (a) and the experimenter (b) whereas the Maine scale(s) only by the experimenter.

The total scores of the acute schizophrenics in all symptom measures were significantly higher (BPRS (a): t = 12.03, d.f. = 63, p < 0.001; BPRS(b): t = 10.45, d.f. = 63, p < 0.001; Maine-N: t = 4.78, d.f. = 63, p < 0.001; Maine-P: t = 7.90, d.f. = 63, p < 0.001) than the scores of their chronic counterparts. The reliability of the various symptom ratings was also investigated. As it can be seen from table 2.4 the inter-rater reliability for BPRS is high.

Table 2.4 Intercorrelations\(^1\) between Symptom Rating Scales of Chronic and Acute Schizophrenics

<table>
<thead>
<tr>
<th>SCALES</th>
<th>BPRS (a)</th>
<th>BPRS (b)</th>
<th>MAINE-N</th>
<th>MAINE-P</th>
</tr>
</thead>
<tbody>
<tr>
<td>BPRS(a)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BPRS(b)</td>
<td>0.96 ****</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MAINE-N</td>
<td>0.62 ****</td>
<td>0.60 ****</td>
<td></td>
<td></td>
</tr>
<tr>
<td>MAINE-P</td>
<td>0.72 ****</td>
<td>0.70 ****</td>
<td>0.57 ****</td>
<td></td>
</tr>
</tbody>
</table>

\(^1\) ns p > 0.05 * p < 0.05 ** p < 0.02 *** p < 0.01 **** p < 0.001

2.3 Design

A between subject design was adopted in this study. All subjects regardless of their diagnostic category were asked to complete a learning and a reversal learning computerised tasks.
2.3.1 Apparatus and Stimuli

All experimental procedures were carried out using an ATTARI 1040 computer and a colour monitor. The tasks employed in this study were modified and computerised versions of the original "card task" employed by Nolan (1968). Two stimuli differing along the dimensions of both shape and colour, were presented on a colour monitor. The two colours appearing on screen were always red and green and the two shapes always a circle and a triangle. The stimuli appeared in randomised combinations (e.g., green circle-red triangle, red circle-green triangle etc.) and each stimuli of the combinations appeared either on the right or on the left of the screen. The main task of the subjects was to decide by trial and error which one of the two colours or shapes was regarded by the computer as the correct one. The subjects responded by pressing the appropriate response keys. The response keys were situated directly below the monitor and each of the keys corresponded directly to the stimulus as presented on the screen (e.g., the stimulus appearing on the right side of the screen corresponded to response key that was situated on the right of the box whereas the stimulus appearing on the left side of the screen corresponded to the response key that was situated on the left side of the response key box). Correct responses were followed by a smiling face on the screen whereas incorrect responses were followed by a crying face.

2.3.2 Procedure

In the testing of all subjects the experimenter was present. All subjects were also asked to complete the Raven's Coloured Progressive Matrices (Raven, Court and Raven 1988). The procedure however, followed for normal controls was slightly different than that of schizophrenic patients.

Normal Controls: All normal controls approached the experimenter either in person or by telephone to enquire about the tests and to arrange for a testing appointment. All individuals who wished to participate in the experiment and fulfilled the criteria outlined in section 2.1 were invited to come either to the Institute of Psychiatry or to St Ann's Hospital. All subjects were tested individually and testing was carried out either at a
testing room at the Institute of Psychiatry or in one of the interview rooms at St Ann’s Hospital after they had sign a consent form (see appendix 3).

Schizophrenics: All schizophrenic subjects were approached by their psychiatrist to obtain informed consent about their participation on the research study. Once this was done the patient was introduced to the experimenter and subsequently they participated in semi-structured interview carried out by the experimenter and the psychiatrist guided mainly by items of the BPRS and the Maine scale. The BPRS’s ratings were completed by both the experimenter and the psychiatrist whereas the Maine scale only by the experimenter. Following that semi-structured interview, all patients were usually allowed fifteen minutes break before the experimenter took them to a quite room on a secluded part of the wards to complete the learning and the reversal learning computerised tasks.

2.3.2.1 Instructions to Subjects

At the beginning of the testing session, all subjects were given the following instructions: “In a little while, you will see on the screen two different shapes; one of which will be a triangle and the other a circle. Each of the two shapes will sometimes appear in the colour red and sometimes in the colour green. One of the two items you will see on the screen is correct either on the basis of its shape or on the basis of its colour. Your task will be to discover by pressing the corresponding response key, which one of the two items is regarded by the computer as being the correct one; each response key corresponds directly to the place of any given item on the screen. Whenever you press a response key and your choice is correct, you will see on the screen a smiling face. Whenever your choice is wrong, you will see a crying face. Once you have established which one of the two items on the screen is correct, the task will automatically shift in such a way so that another item will become the correct. Again your task will be to establish which one of the two items appearing on the screen is regarded by the computer as the correct one. Please continue to respond by pressing the appropriate response keys for as long as the stimuli keep appearing on the screen. When the
computer is satisfied that you have discovered the overall pattern will write on the screen \textit{end of session}.

2.4 Description of Tasks

Following the above instructions, subjects commenced the computerised task. The first task of the subjects was to discover by trial and error which one of the two stimuli appearing on the screen was the correct one. In this first part of the experiment, the stimulus that was regarded by the computer as being the correct one was the colour red. Upon reaching the learning criterion, which was to score correctly on nine consecutive trials, the reversal learning condition commenced. In this part of the experiment, subjects were rewarded for choosing the alternative stimulus within the same dimension (colour green). The task ended once the subject reached the learning criterion; i.e., scoring correctly on nine consecutive trials.

On completion of the task, two scores were obtained for each subject: (a) the learning score (the number of trials taken by the subject to acquire the initial discrimination) and (b) the reversal learning score (the number of trials taken by the subject to establish the correct stimulus during the reversal learning task).
3. RESULTS

Data were analysed using SPSS for Windows (9.0 Release) software. All significance levels are two tailed.

3.1 Trials to criterion on Learning, Reversal Learning and RL/L Ratio

3.1.1 Normal Controls

As described in section 2.4 on completion of the task, two scores were obtained for each subject, the learning and the reversal learning scores. The last nine responses on each of the two conditions that represented the learning criterion were not taken into account during statistical analysis. Statistical analysis using a t-test (related), indicated that reversal learning (M=13.60, s.d. 10.34) was learnt significantly faster than initial learning (M=16.68, s.d. 10.34; t=2.49; d.f.=39; p<0.02).

The possible association of the pattern of performance on learning and reversal learning with the factors of sex, age, education and IQ scores, was also investigated. Median splits were performed on each of the factors in order to establish for each factor two groups of subjects and subsequently to test whether possible differences in their performance on the tasks could be associated to age (median=37.50), education (median=11.00) and IQ score (median=101). Due to the non-normal distribution of the groups created the non-parametric test of Man-Whitney was used in all comparisons. The pattern of performance on the tasks did not appear to be associated with any of the above variables. Comparing the performance between males and females also we did not find any significant differences between the groups (see table 3.1).

Post hoc analysis of Learning and Reversal Learning created a further variable; the ratio of the number of trials taken by subjects to reverse in relation to the number of trials taken to establish the original discrimination (RL/L; M=0.93).
Table 3.1  Means and standard deviations (in parenthesis) of learning and reversal learning scores based on median splits for age, education and I.Q.

<table>
<thead>
<tr>
<th>TASKS</th>
<th>AGE LOWER THAN 37.5</th>
<th>AGE HIGHER THAN 37.5</th>
<th>SIGNIFICANCE(^1)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Learning</td>
<td>18.05 (08.84)</td>
<td>15.30 (11.73)</td>
<td>ns</td>
</tr>
<tr>
<td>Reversal Learning</td>
<td>15.30 (08.61)</td>
<td>11.90 (07.82)</td>
<td>ns</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>TASKS</th>
<th>EDUCATION LOWER THAN 11.0</th>
<th>EDUCATION HIGHER THAN 11.0</th>
</tr>
</thead>
<tbody>
<tr>
<td>Learning</td>
<td>15.50 (09.68)</td>
<td>18.11 (11.21)</td>
</tr>
<tr>
<td>Reversal Learning</td>
<td>13.64 (08.79)</td>
<td>13.56 (07.91)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>TASKS</th>
<th>IQ LOWER THAN 101</th>
<th>IQ HIGHER THAN 101</th>
</tr>
</thead>
<tbody>
<tr>
<td>Learning</td>
<td>17.38 (09.69)</td>
<td>15.90 (11.24)</td>
</tr>
<tr>
<td>Reversal Learning</td>
<td>14.52 (09.22)</td>
<td>12.58 (07.25)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>TASKS</th>
<th>MALES N=21</th>
<th>FEMALES N=19</th>
</tr>
</thead>
<tbody>
<tr>
<td>Learning</td>
<td>16.81 (10.76)</td>
<td>16.53 (10.16)</td>
</tr>
<tr>
<td>Reversal Learning</td>
<td>13.48 (07.36)</td>
<td>13.74 (09.43)</td>
</tr>
</tbody>
</table>

\(^1\) increases in scores correspond to increases in the number of trials

\(^2\) ns p>0.05  * p<0.05  ** p<0.02  *** p<0.01  **** p<0.001

The possible relationship of this variable to age, sex, education and I.Q was also investigated. Again median splits were performed on each of the above establishing two groups of subjects for each factor and subsequently to test whether any differences in the ratio RL/L could be associated to age (median=37.50), education (median=11.00) and IQ score (median=101). Statistical comparisons using the Mann-
Whitney test did not reveal any significant differences (p>0.05) on any of the demographic variables (see table 3.2).

Table 3.2  Means and standard deviations (in parenthesis) of RL/L based on sex and on median splits for age, education and I.Q.

<table>
<thead>
<tr>
<th>TASKS</th>
<th>AGE LOWER THAN 37.5</th>
<th>AGE HIGHER THAN 37.5</th>
<th>SIGNIFICANCE</th>
</tr>
</thead>
<tbody>
<tr>
<td>RL/L</td>
<td>0.93 (0.37)</td>
<td>0.92 (0.39)</td>
<td>ns</td>
</tr>
<tr>
<td>EDUCATION</td>
<td>LOWER THAN 11.0</td>
<td>EDUCATION HIGHER THAN 11.0</td>
<td></td>
</tr>
<tr>
<td>RL/L</td>
<td>0.97 (0.39)</td>
<td>0.87 (0.35)</td>
<td>ns</td>
</tr>
<tr>
<td>IQ</td>
<td>LOWER THAN 101</td>
<td>IQ HIGHER THAN 101</td>
<td></td>
</tr>
<tr>
<td>RL/L</td>
<td>0.92 (0.40)</td>
<td>0.94 (0.35)</td>
<td>ns</td>
</tr>
<tr>
<td>SEX</td>
<td>MALES N=21</td>
<td>MALES N=21</td>
<td></td>
</tr>
<tr>
<td>RL/L</td>
<td>0.93 (0.36)</td>
<td>0.92 (0.40)</td>
<td>ns</td>
</tr>
</tbody>
</table>

1 increases in scores correspond more trials to reverse compared to the original learning score

2 ns p>0.05  * p<0.05  ** p<0.02  ***p<0.01  ****p<0.001

3.1.2 Chronic Schizophrenics

Similar to their normal counterparts, two scores were obtained for each subject on completion of the task, the first score represented the number of trials required to reach the learning criterion for learning and the second for reversal learning. Statistical analysis of the number of trials taken to acquire initial discrimination and
the number of trial taken to shift at the outset of reversal using a t-test (related) revealed that reversal learning \((M=11.50 \text{ s.d.}=05.32)\) is learnt significantly faster than initial learning \((M=20.32 \text{ s.d.}13.31; t=4.26; \text{d.f.}=33; p<0.001)\). The possible association of the pattern of performance on learning and reversal learning with the variables of sex, age, education, IQ scores, medication, age on first admission, number of previous admissions and total number of months spent in hospital were also investigated. Median splits were performed on all of these variables and subsequently the number of trials to criterion for learning and reversal learning between the two new groups for each variable was investigated. Due to the non-normal distribution of the groups created the non-parametric test of Mann-Whitney was used in all comparisons. The results produced did not reveal any significant differences between the performance of subjects on any of the two tasks and the above variables \((p>0.05)\).

Post hoc analysis of the ratio of learning and reversal learning \((RL/L; M=0.80 \text{ s.d.}=0.48)\) and investigation of a possible association with demographic data using the non-parametric test of Mann Whitney again did not reveal any significant differences \((p>0.05)\) in the RL/L between the newly created groups.

### 3.1.3 Acute Schizophrenics

Statistical analysis of the scores obtained by this group of subjects on learning and on reversal learning using a t-test (related) indicate that reversal learning \((M=18.5 \text{ s.d.}10.31)\) is learned significantly faster than initial discrimination \((M=34.29 \text{ s.d.} 18.45; t=6.99; \text{d.f.}=30; p<0.001)\). The performance of subjects in each of the two stages of the task did not seem to be affected by variables such as sex, age education, IQ scores, medication, age on first admission, number of previous admissions and total number of months spent in hospital.
Post hoc analysis of the ratio of learning and reversal learning (RL/L; M=0.59 s.d.=0.22) and investigation of a possible association with the demographic data mentioned above using the non-parametric test of Mann Whitney also did not reveal any significant differences (p>0.05) in the RL/L between the newly created groups.

3.2 Normal Controls, Chronic Schizophrenics and Acute Schizophrenics: Trials to criterion on Learning, Reversal Learning and RL/L

As shown on table 3.3 one way ANOVA performed for each of the variables across groups revealed significant differences in Learning (F=14.6, d.f.=2, p<0.001) and Reversal Learning (F=6.39, d.f.=2, p<0.01). In order to control for type 1 error, the Bonferroni approach was used at this stage across the pairwise comparisons with the corrected significance level of p<0.017.

Table 3.3 Means and Standard deviations (in parenthesis) of number of trials to criterion on Learning, Reversal Learning and RL/L

<table>
<thead>
<tr>
<th>Tasks</th>
<th>Normal Controls</th>
<th>Chronic Schizophrenics</th>
<th>Acute Schizophrenics</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Learning</td>
<td>16.68 (10.34)</td>
<td>20.32 (13.31)</td>
<td>34.29 (18.45)</td>
<td>****</td>
</tr>
<tr>
<td>Rev. Learn.</td>
<td>13.60 (08.30)</td>
<td>11.50 (05.32)</td>
<td>18.58 (10.31)</td>
<td>***</td>
</tr>
<tr>
<td>RL/L</td>
<td>00.93 (00.37)</td>
<td>00.79 (00.48)</td>
<td>00.59 (00.22)</td>
<td>***</td>
</tr>
</tbody>
</table>

1 increases in scores correspond an increase in the number of trials

2 ns p>0.05 * p<0.05 ** p<0.02 ***p<0.01 **** p<0.001

Paired comparisons using t-test (unrelated) revealed that in Learning, normal controls (t=5.10, d.f.=69, p<0.001) and chronic schizophrenics (t=3.52, d.f.=63, p<0.001) reached the learning criterion with significantly fewer trials than acute schizophrenics. In Reversal Learning, chronic schizophrenics reached the learning criterion with
significantly fewer trials (Reversal Learning: $t=3.52$, d.f.=63, $p<0.001$) than acute schizophrenics. The difference in the number of trials taken by normal controls to reverse compared to acute schizophrenics, although failed to reach significance (Reversal Learning: $t=2.26$, d.f.=69, $p<0.05$) there was an obvious trend in the right direction (i.e., normal controls required fewer trials than acute schizophrenics - see figure 1).

**Figure 1**  
Number of trials on Learning, Reversal Learning and RL/L

![Number of trials on Learning, Reversal Learning and RL/L](image)

One Way ANOVA also revealed significant differences in the RL/L ($F=6.88$, d.f.=2 $p<0.01$) ratio across the groups. Paired comparisons showed that the ratio RL/L for acute schizophrenics is significantly smaller than chronic schizophrenics ($t=2.20$ d.f.=63, $p<0.05$) and normal controls ($t=4.41$ d.f.=69, $p<0.001$).

### 3.3 Normal Controls, Chronic Schizophrenics and Acute Schizophrenics: New Learning Criterion for Learning, Reversal Learning and RL/L

Retrospective observation on subjects' number of trials in acquiring learning in each of the two tasks indicated that in a number of cases, subjects responded correctly to a
smaller number of stimuli but could not maintain this level of response for the nine consecutive trials required for the shift onto the next task. In order to investigate the possibility of a significantly different performance on each of the tasks taking into account this observation, an additional analysis of the data was carried out. Subjects' scores on each of the tasks were considered as reaching the learning criterion after they had responded correctly to five consecutive trials instead of the original nine. The number of trials between the initial five correct trials and up to the shift into the next task were disregarded and not used for statistical analysis.

This later criterion was selected on the basis of the observation that people with acute schizophrenia although they could often respond correctly up to five trials they could not maintain their response to the remaining number of trials in order to reach the learning criterion. In contrast, normal controls and acute schizophrenics in most cases when they responded correctly for five consecutive trials they continued with reaching the learning criterion for nine consecutive trials.

3.3.1 Normal Controls, Chronic Schizophrenics and Acute Schizophrenics: Comparison of number of trials between new and original criterion on Learning, Reversal Learning and RL/L

Statistical comparisons on the number of trials taken by normal controls to complete Learning and Reversal Learning in each of the learning criteria using t-test (related) were carried out. The results did not reveal any significant differences between the two sub-groups in the number of trials taken to acquire the initial discrimination and to shift at the outset of reversals. As indicated in table 3.4, statistical analysis of the RL/L ratio between the two learning criteria did not yield any significant differences.

Similar to the performance of normal controls, statistical comparisons for people with chronic schizophrenia using t-test (related) indicated that the number of trials taken by subjects to acquire initial learning and to shift at the outset of reversal were not
significantly different (p>0.05) between the 9-trials and the 5-trials criteria. Insignificant
differences were also found when the RL/L ratio was investigated.

Table 3.4  Means and Standard deviations of number of trials to new and original
criteria on Learning, Reversal Learning and RL/L in all groups

<table>
<thead>
<tr>
<th>TASKS ¹</th>
<th>LEARNING CRITERION 9 TRIALS</th>
<th>LEARNING CRITERION 5 TRIALS</th>
<th>SIGN.²</th>
</tr>
</thead>
<tbody>
<tr>
<td>LEARNING</td>
<td>16.68 (10.34)</td>
<td>16.38 (10.07)</td>
<td>ns</td>
</tr>
<tr>
<td>REV. LEARN.</td>
<td>13.60 (8.30)</td>
<td>13.15 (8.07)</td>
<td>ns</td>
</tr>
<tr>
<td>RL/L</td>
<td>00.93 (0.37)</td>
<td>0.91 (0.37)</td>
<td>ns</td>
</tr>
</tbody>
</table>

CHRONIC SCHIZOPHRENICS:

<table>
<thead>
<tr>
<th>TASKS ¹</th>
<th>LEARNING CRITERION 9 TRIALS</th>
<th>LEARNING CRITERION 5 TRIALS</th>
<th>SIGN.²</th>
</tr>
</thead>
<tbody>
<tr>
<td>LEARNING</td>
<td>20.32 (13.31)</td>
<td>19.79 (12.79)</td>
<td>ns</td>
</tr>
<tr>
<td>REV. LEARN.</td>
<td>11.50 (5.32)</td>
<td>11.12 (5.31)</td>
<td>ns</td>
</tr>
<tr>
<td>RL/L</td>
<td>0.79 (0.48)</td>
<td>0.81 (0.51)</td>
<td>ns</td>
</tr>
</tbody>
</table>

ACUTE SCHIZOPHRENICS:

<table>
<thead>
<tr>
<th>TASKS ¹</th>
<th>LEARNING CRITERION 9 TRIALS</th>
<th>LEARNING CRITERION 5 TRIALS</th>
<th>SIGN.²</th>
</tr>
</thead>
<tbody>
<tr>
<td>LEARNING</td>
<td>34.29 (18.45)</td>
<td>19.61 (10.82)</td>
<td>****</td>
</tr>
<tr>
<td>REV. LEARN.</td>
<td>18.58 (10.31)</td>
<td>8.32 (3.64)</td>
<td>****</td>
</tr>
<tr>
<td>RL/L</td>
<td>0.59 (0.22)</td>
<td>0.55 (0.40)</td>
<td>ns</td>
</tr>
</tbody>
</table>

¹ increases in scores correspond an increase in the number of trials

² ns p>0.05  * p<0.05  ** p<0.02  ***p<0.01  **** p<0.001
Statistical analysis of the performance of acute schizophrenics in the 5-correct trials and the 9-correct trials criteria revealed that this group of subjects required significantly fewer trials for learning ($t=5.21 \, \text{d.f.}=30, \, p<0.001$) and reversal learning ($t=5.81 \, \text{d.f.}=30, \, p<0.001$) during the new learning criterion than in the previous one (see Figures 2 and 3).
Comparison of the RL/L ratios between the two learning criteria did not reveal any significant differences (see table 3.4).

3.3.2 Normal Controls, Chronic Schizophrenics and Acute Schizophrenics: Comparison of trials to new criterion on Learning, Reversal Learning and RL/L

One-way ANOVA revealed significant differences across the three groups on Reversal Learning (F=5.36, d.f.=2, p<0.01) but not on initial discriminations (F=1.09, d.f.=2, p>0.05). In order to control for type 1 error, the Bonferroni approach was used at this stage across the pairwise comparisons with the corrected significance level of p<0.017. Statistical comparisons using a t-test (unrelated) indicated that acute schizophrenics required significantly fewer trials to shift at the outset of reversal than normal controls (t=2.99, d.f.=69, p<0.01). Similarly acute schizophrenics required fewer trials to reverse than chronic schizophrenics but the difference in the number of trials failed to reach significance (t=2.45, d.f.=63, p<0.02).

Figure 4 Number of trials on the 5-correct trials criterion across the three groups on Learning, Reversal Learning and RL/L
One Way ANOVA revealed significant differences between the RL/L (F=6.36, d.f.=2, p<0.01). Paired comparisons showed that the ratio RL/L for acute schizophrenics is significantly smaller than chronic schizophrenics (t=2.77, d.f.=63, p<0.01) and normal controls (t=3.94, d.f.=69, p<0.001). Finally, no significant differences were found on initial discrimination (t=1.29, d.f.=72, p>0.05) on reversal learning (t=1.25, d.f.=72, p>0.05) and on RL/L (t=1.05, d.f.=72, p>0.05) between normal controls and chronic schizophrenics (see table 3.5).

Table 3.5 Means and Standard deviations (in parenthesis) of number of trials to new criterion on Learning, Reversal Learning and RL/L

<table>
<thead>
<tr>
<th>TASKS 1</th>
<th>Normal Controls</th>
<th>Chronic Schizophrenics</th>
<th>Acute Schizophrenics</th>
<th>Significance2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Learning</td>
<td>16.38 (10.07)</td>
<td>19.79 (12.79)</td>
<td>19.61 (10.82)</td>
<td>ns</td>
</tr>
<tr>
<td>Reversal Learning</td>
<td>13.15 (8.07)</td>
<td>11.12 (5.31)</td>
<td>8.32 (3.64)</td>
<td>***</td>
</tr>
<tr>
<td>RL/L</td>
<td>0.91 (0.37)</td>
<td>0.81 (0.51)</td>
<td>0.55 (0.40)</td>
<td>***</td>
</tr>
</tbody>
</table>

1 increases in scores correspond to increases in the number of trials

2 ns p>0.05 * p<0.05 ** p<0.02 ***p<0.01 **** p<0.001
4. DISCUSSION

In a review carried out by Hemsley (1987) it was pointed out that most theories of information processing (e.g., Broadbent, 1971; 1977; Schneider and Shiffrin, 1977; Shiffrin and Schneider, 1977; Posner et al, 1982) at their basic level illustrate how “spatial and temporal regularities of past experiences influence the processing and awareness of current input” (Hemsley, 1987, p.182). Moreover it was noted that the majority of cognitive theories of schizophrenia either suggest a disruption in the cognitive performance of people with schizophrenia due to an intrusion of material that normally exists below awareness (Frith 1979; Cutting 1985) or relate to the weakening of the influence of spatial and temporal regularities on perception (Venables 1984; Knight, 1984; Maher, 1983; Magaro 1984). In an attempt to unite these influential models of information processing that have been proposed as possible explanations for the emergence of the symptoms observed in schizophrenia, he argued that the basic deficit to the schizophrenic condition “is a weakening of the influences of stored memories of regularities of previous input on current perception” (Hemsley, 1987 p.182). In addition to being consistent with Chapman’s and Chapman’s (1973) recommendation that theories of schizophrenia should be capable of describing the way by which the proposed deficit leads to the observed symptomatology, the model also appears to be compatible to the more elaborate neuropsychological model of schizophrenia proposed by Gray’s and his colleagues (1991). In their formulation, the cognitive deficit proposed by Hemsley (1987) to play a key role in the emergence of positive symptomatology is considered to correspond to an underlying neural dysfunction which, according to them, is placed in the interaction between the hippocampal formation and nucleus accumbens.

In support of the view that the emergence of positive symptomatology is related to the inability of people with schizophrenia, based on previous experiences, to select for processing only relevant information Hemsley (1987; 1991; 1993) refers to two experimental paradigms, namely the LI and the KBE. According to the above author,
these paradigms, which were originally developed in the animal laboratory, can be "regarded as instances of the influence of memories of stored regularities of previous input on current perception" (Hemsley, 1987 p.144). Evidence produced in the animal laboratory investigating the performance of animals in the above tasks consistently indicated that administration of the dopamine agonist amphetamine disrupts LI (e.g., Solomon et al., 1981; Warburton et al., 1994) and KBE (e.g., Solomon et al., 1981; Weiner et al., 1981) and that the disruption is reversed with the administration of neuroleptics (e.g., Solomon et al., 1981; Warburton et al., 1994). Similar disruption was also found in the performance of the hippocampally lesioned animal. Linking the above findings to the proposal that the basic cognitive deficit of people with schizophrenia lies in their inability to learn from previous experiences and that this deficit is considered to correspond to an underlying neural dysfunction in the subiculo-accumben projection, it was hypothesised that the performance of acute schizophrenics and of normal volunteers who have taken low doses of amphetamine on the above tasks would also be disrupted. Research studies investigating these hypotheses although reported some rather encouraging results (N.S. Gray et al., 1992) other studies in the same area pointed to several inconsistencies (N.S. Gray et al., 1997) emphasising the need for further research.

According to Knight (1984), research into cognitive deficits of schizophrenia should aim at finding tasks in which the results obtained across tasks will follow a similar pattern to the one predicted by the specific deficit and therefore disconfirm a general deficit explanation. Predominantly influenced by this suggestion, the experimental study described in the previous section sought to further investigate the cognitive deficit proposed by Hemsley by employing a different experimental procedure than that of LI and the KBE. Taking into consideration research evidence indicating that reversal learning also seems to depend on the control of attention by prior experience and therefore it can facilitate direct comparisons of the extend by which prior experiences influences new learning in people with schizophrenia, in the course of the study the following hypotheses were addressed.
(i) people with acute schizophrenia would require fewer trials to shift at the outset of reversal than chronic schizophrenics and normal controls; and

(ii) the number of trials taken by acute schizophrenics to reverse in relation to their initial learning (RL/L) would be significantly smaller than the number of trials taken by chronic schizophrenics and normal controls.

In the next section a summary of the data within the context of the above hypotheses will be presented followed by a discussion of the implications of this study in relation to Hemsley’s (1987) and Grays and colleagues’ (1991) models.

4.1 Summary of Results

The first set of results indicated that people with acute schizophrenia required significantly more trials to acquire the initial discrimination than chronic schizophrenics and normal controls. As it can be derived from the above results, the main hypothesis that people with schizophrenia would be less influenced by previous learning and therefore would reverse faster than chronic schizophrenics and normal controls was not supported by our data. Further analysis of our data however indicated that when considering the ratio of the number of trials taken by subjects to reverse in relation to their original discrimination, people with acute schizophrenics required significantly fewer trials to reverse than chronic schizophrenics and normal controls.

These later findings appear to be quite compatible with evidence suggesting that people with schizophrenia experience difficulties in acquiring new knowledge (Hemsley, 1991). Moreover, a similar pattern of results was also reported by a study where the performance of high schizotypy individuals was compared to that of low schizotypy individuals in an identical reversal learning task. In that study it was shown that the RL/L ratio of high scorers on schizotypal scales was significantly smaller than the RL/L ratio of low scorers indicating that the former group required
significantly fewer trials to reverse than the number taken to acquire the initial discrimination (Georgiades 1993). Although these findings seem to support Hemsley’s (1991) formulation that people with acute schizophrenia are less influenced by previous learning and therefore require fewer trials to reverse compared to their initial discrimination, the fact that people with acute schizophrenia are in a disadvantageous position, when the absolute number of trials is taken into consideration, remains. As indicated throughout this section in contrast to our hypothesis, acute schizophrenics required significantly more trials to acquire the initial discrimination and to shift at the outset of reversal compared to the number of trials taken by chronic schizophrenics and normal controls.

The observed difficulties of people with acute schizophrenia in both the initial learning and in the subsequent reversal learning could be attributed to several possible factors spanning from the possibility that the experimental task employed does not really measure the cognitive deficit described by Hemsley to schizophrenia being associated with a more generalised cognitive deficit (as proposed by earlier studies) rather than the specific deficit considered here. The findings of several studies however pointing that schizophrenics do seem to be less influenced by previous learning (e.g., Baruch et al., 1988) and the compatibility of several components of Hemsley’s (1987) model with other theories of schizophrenia (see section 1.2.2.5) makes it rather difficult to regard any of the above factors as possible explanations for the observed performance of acute schizophrenics in this task. An alternative explanation that might be offered here is that due to the increased number of trials required for reaching the learning criterion for both stages of the task, acute schizophrenics were more easily “cognitively overtired” than their chronic schizophrenics and normal controls counterparts. The concept of cognitive fatigue in this group of subjects was discussed by Everett and his colleagues (1989) who suggested that the performance of people with acute schizophrenia in attentional tasks deteriorates significantly when selective attention had to be maintained. According to these authors the decrement in performance can be attributed to cognitive fatigue rather than to a selective attention deficit.
With regard to this experimental task however, even if we do accept that cognitive overtiredness was indeed the factor that affected the performance of acute schizophrenics then one would have expected subjects to experience difficulty to completing the task or at least the difficulty experienced due to overtiredness in initial discrimination to increase as they progressed on to the next stage of the task. These difficulties would have been reflected in the greater number of trials taken by subjects to reverse compared to their initial discrimination. As indicated from our data though acute schizophrenics not only did not take longer to shift at the outset of reversal but in fact they required significantly fewer trials to reverse than the number of trials taken to acquire the initial discrimination. Moreover, the RL/L ratio of acute schizophrenics was significantly smaller than that of chronic schizophrenics and normal controls suggesting that the “overtiredness assumption” might not be adequate to explain the results obtained in this study.

A closer observation of the raw data appeared to indicate that people with acute schizophrenia began their learning and reversal learning acquisition earlier than shown by their actual performance. However, a problem that appeared to be associated with the deterrence of their overall performance was that this group of subjects could not maintain their attention and consequently their response to the appropriate stimulus for long enough to fulfil the learning criteria for the task.

In order to investigate the effect that the nine trials learning criterion had in the performance of subjects in all three groups, an additional analysis was carried out where subjects were considered to have reached the learning criterion after selecting the correct stimulus for five instead of the previous nine consecutive trials in both initial discrimination and reversal learning. All additional trials after the initial five were disregarded and were not used for analysis. Statistical analysis of the number of trials taken by chronic schizophrenics and normal controls for learning and reversal learning did not indicate any significant differences in the number of trials taken for subjects to acquire learning and reversal learning when their performance employing the five and the nine trials learning criterion were compared. These insignificant
differences between the two criteria in the performance of these groups suggested that for these subjects once they had selected correctly the appropriate stimulus for five consecutive trials they were likely to continue selecting the appropriate stimulus up to ninth trial meeting this way the nine trials learning criterion. In contrast to the findings for chronic schizophrenics and normal controls, the picture emerged for people with acute schizophrenia was different when their performance to the five trials was compared with the nine trials criterion. In this later analysis it was revealed that in the five consecutive trials learning criterion acute schizophrenics acquired initial discrimination and shifted at the outset of reversal with significantly fewer trials than the number of trials taken when the learning criterion was nine consecutive trials. This later set of findings were interpreted to indicate that this group of subjects even after selecting the appropriate stimulus for five consecutive trials often shifted their attention and selected the alternative stimulus. On the basis of the above, these results may be considered to provide further evidence of the inability of this group of subjects to let past experiences influence current behaviour.

Finally, when the performance across all three groups was statistically analysed for both stages of the task within the context of our initial hypotheses it was revealed that there were no significant differences in the number of trials taken by each group to acquire the initial discrimination. With regard to the number of trials taken to shift at the outset of reversal it was found that people with acute schizophrenia reversed significantly faster than chronic schizophrenics and normal controls. The differences in the number of trials taken by chronic schizophrenics and normal controls to reverse were insignificant.

4.2 Implications of Study to Schizophrenia Research

As shown in the previous section, subsequent analysis of the data when the learning criterion was reduced from nine consecutive trials to five revealed that people with acute schizophrenia reversed faster than chronic schizophrenics and normal controls. These findings seem to support the hypotheses deriving from Hemsley’s (1987) model
which suggest that the basic cognitive deficit of people with acute schizophrenia lies in their inability to learn from previous experiences. Within the context of this experimental paradigm the results that people with acute schizophrenia require fewer trials to reverse than the number of trials taken by subjects from the other two groups and the significantly smaller RL/L ratio are interpreted to suggest that people with acute schizophrenia are less influenced by their previous learning, in this case initial discrimination, and approach the alternative stimulus earlier.

At this point it is quite important to note that although these later findings are the product of a post hoc analysis with a modified learning criterion, the basis of this approach appears to be quite compatible with the way by which data are recorded and analysed in the animal laboratory. In most animal studies (e.g., Weiner and Feldon, 1986), animals were considered to have reached the learning criterion for both stages of the task not when they have approached the appropriate stimulus for any number of consecutive trials but rather when they have selected the correct stimulus nine times in a twelve-trial-block. As suggested in the present experimental study, the major difficulty associated with the performance of people with acute schizophrenia derives from their inability to let past experiences influence their current behaviour. As a consequence of this they find it difficult to maintain their response for the number of trials required by the task to reach the learning criterion. Due to the design of the task, a single selection of the alternative stimulus, prior to reaching the ninth trial, would automatically disregard all the previously correct trials and would require the subject to start from the beginning selecting the appropriate stimulus for nine consecutive trials as only then they would be considered to have reached the learning criterion. The approach of nine correct out of a twelve-trial-block required by animal researchers appears to counteract the specific difficulties identified by the present study with regard to the overall performance of acute schizophrenics.

Taking into consideration the differences found on the above two analyses of the data, a relevant question that needs to be addressed at this stage is with reference to the factors affecting the ability of people with acute schizophrenia to maintain their
attention to the appropriate stimulus for the necessary period to fulfil the learning criteria for the task. The possible explanations that the specific experimental task employed in this study does not really measure the cognitive deficit described by Hemsley (1987) or that acute schizophrenics experience cognitive fatigue (Everett et al., 1989) more easily than the two other groups do not seem to be substantiated by these later findings. In fact any explanation offered within the context of this paradigm needs to account the two facets of the findings; that people with acute schizophrenia do tend to reverse faster but their overall performance is affected by their inability to maintain their attention to the selected stimulus.

The first aspect of their performance appears to be in accord with Hemsley’s (1987) proposal that people with acute schizophrenia are less influenced by previous learning. In the context of this task previous learning is considered to be the knowledge acquired during initial discrimination that the colour red is associated with positive feedback and therefore is the appropriate stimulus and green the inappropriate. The tendency observed in this group of subjects to reverse quite early in the task is facilitated by their difficulty to learn from previous experiences.

With regard to the finding that this group of subjects cannot maintain their attention and response to the appropriate stimulus for long, it may be attributed to an overattention to all the stimuli present within the prevailing situation including those that, based on previous experience, might be redundant; i.e. stimuli not associated with any consequence. In accounting for the concept of overattention, Gray and his colleagues (1991) described it as being the result “of weakening of the capacity to select for cognitive processing only those stimuli that, given past experience of similar contexts, are relevant” (Gray et al., 1991). Linking this description to Hemsley’s (1987) theory and to the way by which the performance of acute schizophrenics on the LI and KBE tasks were interpreted, the expectation within the present task was that acute schizophrenics would have been less influenced by the learning acquired during initial discrimination and would reverse quicker than chronic schizophrenics and normal controls. However, Frith (1991) argued that the description provided by
Gray and his colleagues is inadequate and that the concept of overattention implies attention to all stimuli within any given situation regardless of their relevance to the task implying attention to both the target and the redundant stimuli. In accord with Frith’s (1991) argument is our finding that people with acute schizophrenia continue to attend to redundant stimuli even after they have selected the appropriate stimuli and received positive feedback for a number of consecutive trials. A possible explanation for this pattern of response may be attributed to the inability of this group of subjects to inhibit the product of automatic processing due to a breakdown in the mechanism that controls and limits the contents of consciousness (Frith, 1979; see section 1.2.2.4). As a result of this dysfunction redundant information that normally exists below the level of consciousness would enter awareness influencing the subjects’ response which by extent may be considered to affect “... [their] current plans or motor programmes” (Gray et al., 1991, p.3). Linking this with Frith’s (1991) model where it is proposed that failure to monitor the intentions of self leads to positive symptoms of schizophrenia (hallucinations and delusions of control) it is possible that a dysfunction at this level may be caused by the intrusion into consciousness of information that exists below awareness (see Frith 1979).

A relevant issue that also needs to be addressed at this stage is with regard to the extent by which awareness of redundant information influences current responses. On the basis of the explanation provided above one could imply that the deficit described by Frith (1979) is permanent and therefore the intrusion of redundant information into consciousness continuous. A problem associated with this explanation however, is that if we accept that redundant information intrudes continuously into awareness, then this not only would deterred the performance of acute schizophrenics but in effect it would have disrupted it altogether with subjects finding it extremely difficult to complete the task. In contrast to these later expectations the findings of the present study indicated that people with acute schizophrenia completed the task and the trials taken to reverse were significantly less than those taken to acquire initial discrimination. Also the RL/L ratio for this group of subjects is significantly smaller than the RL/L ratio of their chronic schizophrenic and normal control counterparts.
Elaboration of these findings seem to indicate that intrusion of redundant stimuli into consciousness and the consequent attention to that stimuli is not continuous but rather it progressively decreases as the formation of the association between the appropriate stimulus and positive feedback increases.

On the basis of the above it seems that people with acute schizophrenia due to a weakening of the influences of previous regularities of stored memories to current perception fail to learn from previous experiences and they approach each situation as being novel. Within the context of our present study novel does not necessarily imply sole attendance to the new relevant stimulus but rather uninfluenced by their previous experiences they attend both the stimuli present in the prevailing situation. Although at this stage acute schizophrenics tend to recognise the relevance of the appropriate stimulus quite early in the task, presumed intrusions and subsequent attention to redundant information (which in this case redundant information refers to the alternative stimulus) deters subjects from maintaining their attention and consequently their response to the selected stimulus. However, as the association between positive feedback and appropriate stimulus increases the more likely it is for the appropriate stimulus to be established as a response leading into the inhibition of redundant stimulus.

The above explanation, in addition to accounting for the results of the present study also seem to be quite compatible to the large body of evidence pointing to the difficulties of schizophrenics in acquiring new knowledge (see Hemsley, 1992). Within the context of the suggestion proposed in this thesis, the difficulties experienced by this group of people may be associated with the process of establishing new knowledge and responses while experiencing the detrimental effects of being consciously aware of the presence of redundant information. Finally, since strengthening of the association between appropriate stimulus and positive feedback facilitates the inhibition of redundant information, it is quite probable that as the strengthening of this association increases (by means of increasing the learning criterion for the initial discrimination from nine consecutive trials to a much larger
number of trials) then the inhibition of attention to the alternative stimulus will also be enhanced. On the basis of the above one would expect that in an overtraining reversal learning task, when the reward contingencies change so that the irrelevant stimulus becomes relevant, subjects will continue to respond to the old stimulus due to the enhanced inhibition of the redundant stimulus. In fact by employing an identical task to the one used here but with subjects continuing to receive positive feedback when selecting the same stimulus for twenty more trials after they had reached the nine trials learning criterion, Georgiades (unpublished data) has found that the overall performance of people with acute schizophrenia in this overtraining condition of a reversal learning task resembled that of normal controls in a non-overtraining condition. These findings appear to be quite compatible with the principles governing most rehabilitation programmes for people with schizophrenia. In such programmes, the expectation is that regular and excessive training on specific skills would eventually inhibit previous pattern of behaviour and the new skills would be added to their repertoire of appropriate skills.

As mentioned in previous sections studies investigating Hemsley’s (1987) and Grays and colleagues’ model theory using the LI and the KBE paradigms have produced rather conflicting findings. As discussed earlier in some studies there was evidence to suggest that people with acute schizophrenia and normal volunteers who have taken low dosages of amphetamine show a disrupted LI and KBE whereas more recent findings either have found the opposite results or their findings indicate that the disruption might not be associated with symptomatology but with the time spent in hospital. A review of the methodology employed in those studies indicated that the task requirements in terms of establishing associations between stimuli were similar to the one used by the present study; i.e., subjects were required to select for a number of consecutive trials specific stimuli. Although it is quite difficult on the basis of the results reported on the published papers to ascertain with any degree of accuracy that the response of those subjects in terms of their ability to maintain attention to specific stimulus might have had any impact on their overall performance, it would have been interesting to replicate those findings focusing on this aspect of their performance.
4.3 Limitations of Study

The results discussed in the preceding sections of this part of the thesis seem to provide some support of Hemsley's (1987) and Gray's and colleagues (1991) models which propose that the basic cognitive deficit associated with the emergence of positive schizophrenic symptoms lies in the inability of this group of subjects to learn from previous experiences. In addition to supporting our hypotheses the findings of this study also indicated that it is possible for the performance of acute schizophrenics in a reversal learning task to be further complicated by the attention of subjects to redundant information.

Despite the rather encouraging results reported here, the present study is not without its limitations. Probably the most important problem associated with the results is the way by which the data were analysed. As described in the methodology section and in sections 4.1 and 4.2, the original task requirement was for subjects to select the appropriate stimulus for nine consecutive trials in order to consider them as having reached the learning criterion. As indicated above, the findings produced on the basis of this criterion not only failed to confirm the hypotheses but they also masked an important component of the overall performance of this group of subjects.

However, the approach employed during this later analysis does not take into consideration the number of trials taken by subjects between the fifth consecutive trial (considered in this later analysis to constitute the learning criterion) and the actual shift to reversal which was based on nine consecutive trials. As it was discussed earlier, the inhibition of redundant stimulus appears to occur in relation to the association between the selected stimulus and the positive feedback. On the basis of the above, it is possible that subjects who have managed to select the appropriate stimulus quite early in the initial discrimination but failed to maintain their attention for the nine consecutive trials and had to start from the beginning on more than once, the task would resemble an overtraining condition which in effect might have some impact on the number of trials taken to reverse (see section 4.2). A possible way to counteract this problem is by re-designing the experimental task so that the learning
criterion would either be reduced from nine consecutive trials to five or to employ a similar criterion as the one employed in the animal laboratory where the expectation for acquiring learning was to select the appropriate stimulus for nine trials in a block of twelve.

The findings of recent studies investigating the LI and KBE between acute and chronic schizophrenics with similar BPR scores suggested that it might be possible that the disruption found might not be associated with the symptomatology per se but rather with the time spent in hospital. In the present study the BPRS scores were significantly different between acute and chronic schizophrenics and therefore it is quite difficult to identify whether the findings of the study are related to the symptomatology or to any other variable. A possible way to clarify this issue is by replicating the study employing chronic and acute schizophrenics with similar BPRS scores. Also in future studies it would have been quite appropriate to include a psychiatric control group in order to investigate whether the obtained pattern of results is only relevant to schizophrenia and not typical of psychiatric patients.

A final limitation of the study that is worth mentioning here is with regard to the suggestion that people with acute schizophrenia in addition to the selected stimulus also attend the redundant information. Within the context of a reversal learning task it was assumed that the redundant stimulus was the alternative colour. As shown in the description of the task the colours were presented within two specific shapes, a circle and a triangle, which may also be considered as redundant stimuli. The explanation offered however, does not take into account the possibility that subjects might have been attending to more than one redundant stimulus nor does it take into account the possible effect that such an eventuality would have in their overall performance. Again, the role played by stimuli that can be classified to belong to a different dimension that are present within a reversal learning task might also need to be investigated. This can be achieved by including an extradimensional shift condition (see Nolan and Anderson, 1973) where following reversal, subjects will be required to select a stimulus from a different dimension (e.g., red-triangle). Any possible
differences in the performance of subjects in these two conditions might be indicative of the way by which people with acute schizophrenia attend to different types of redundant information.

4.4 Suggestions for Future Research

In the previous section in addition to discussing the limitations associated with the present study several recommendations were proposed with regard for potentially improving the study and clarify some of the current findings. However, even with the above recommendations the information that can be derived from a study predominantly employing normal controls, chronic and acute schizophrenics is quite limited.

According to Hemsley's (1987) model, the basic cognitive deficit of people with schizophrenia stems from a weakening of the influences of stored memories of previous regularities to current perception whereas Gray’s and colleagues (1991) propose that a cognitive disturbance at this level may correspond to an underlying neural deficit in the subiculo-accumben projection, which by extent causes DA overactivity to one or more dopaminergic systems. Studies that investigated these models by employing the experimental paradigms of LI and KBE in addition to the three groups mentioned above they also investigated the performance of healthy volunteers that have taken low doses of amphetamine. Since animal studies have shown that amphetamine enhances reversal learning and acute schizophrenics are presumably in a hyperdopaminergic state, it will be quite important to investigate within the context of the models (Hemsley, 1987; Gray et al., 1991) possible differences in the performance of acute schizophrenics and healthy volunteers following amphetamine administration. Moreover, the strategy adopted within the study derives from Knight’s (1984) suggestion that research into cognitive deficits of schizophrenia should aim to identify and investigate tasks in which the pattern of results across tasks would follow a similar predicted pattern. On the basis of the above, it will be quite useful to investigate the reversal learning paradigm using
similar procedures and groups of subjects to the ones used for the investigation of LI and the KBE as this would facilitate direct comparisons of the performance of subjects across tasks as suggested by Knight (1984).

Finally, taking into consideration the number of confounding variables such as medication, institutionalisation and symptomatology often associated with schizophrenia research, studies investigating the performance of people with schizophrenia on the LI and KBE in order to account for these confounded variables have also examined the performance of normal volunteers with high scores on several schizotypal questionnaires. Although findings from such studies indicated some similarities on the overall performance of acute schizophrenics on the LI (e.g., Baruch, 1988), the KBE (Martins, 1995) and on Reversal Learning (Georgiades, 1993), the small number of subjects and the different scales employed in these studies not only weakens their explanatory power but also it makes it extremely difficult to make any direct comparisons of the performance of subjects between tasks. Due to the potential value of schizotypy research in the future of schizophrenia, studies that would facilitate direct comparisons of the performance of high schizotypy individuals and acute schizophrenics on these tasks not only would increase our knowledge of the possible relationship between schizotypal traits and schizophrenia itself but also they would enhance our understanding of the cognitive deficit relevant to acute schizophrenia without the complications of the confounding variables mentioned above.
5. CONCLUSIONS
The search for the factors causing the emergence of schizophrenic symptoms led to the development of several prominent theories and hypotheses and consequently to a vast amount of research data. Unfortunately, up until recently these theories although important in enhancing our understanding of the disorder their value in unveiling the mystery that surrounds the aetiology of the disorder has been quite limited. The limitations of these theories emphasised the need to identify a model that would take into consideration the different facets and characteristics of the illness described in several experimental and observational studies.

The two most prominent theories of schizophrenia that managed to withstand the test of time and considered to play an important role in the genesis of the disorder are the cognitive (see section 1.2.2) and the neurochemical theories (see section 1.2.1.1). But despite the attractiveness of these theories they also seem to be separated by a wide gap and this is especially evident when schizophrenia is regarded as a heuristic concept based on a unitary concept where all aspects of the disorder, including aetiology, symptomatology and treatment are mutually and coherently related.

Recent advances in our understanding of temporal lobe pathology however and the proposals that damage in the hippocampal region could be related to dopamine overactivity led Gray and his colleagues (1991) to propose an elaborate neuropsychological model of schizophrenia. In that model, it was suggested that the cognitive deficit considered to play a significant role in the emergence of positive symptoms in people with schizophrenia may correspond to an underlying neurological dysfunction placed in the projection from the hippocampal formation to the nucleus accumbens. The cognitive deficit relevant to their model was Hemsley's suggestion that "[basic to schizophrenia] is the weakening of the influences of stored memories of regularities of previous input on current perception" (Hemsley, 1987 p.182).

The main aim of the present study was to investigate the cognitive deficit described by Hemsley (1987). Following a similar approach to the one employed by researchers
investigating the above models using the behavioural paradigms of LI (Baruch, 1988) and KBE (Jones, 1989) the present study sought to examine the extent by which prior experiences influences new learning in people with acute schizophrenia by employing the experimental paradigm of reversal learning (Lawrence, 1949; Mackintosh, 1965; Sutherland and Holgatee, 1966)). Since reversal learning could be also considered as an "...instance of the influence of stored memories of regularities of previous input on current perception" (Hemsley, 1996, p.144) it was hypothesised that acute schizophrenics less influenced by their previous knowledge they would shift faster than their normal controls and chronic schizophrenics at the outset of reversal.

The initial findings however, not only failed to support the hypotheses but in contrast to our expectation people with chronic schizophrenia and normal controls reversed significantly faster than acute schizophrenics. However, based on the findings that the RL/L ratio for acute schizophrenics was significantly smaller than the other groups and the observation that acute schizophrenics would have reversed faster if the learning criterion was smaller than the nine consecutive trials, further analysis was carried out using as a learning criterion five consecutive trials instead of nine. The results of this second analysis confirmed the observation that people with acute schizophrenia experience difficulties in maintaining their attention and consequently their response to the appropriate stimulus for the necessary period to fulfil the initial learning criterion. Moreover, it was also revealed that within the revised criterion their performance supported the hypothesis that people with schizophrenia less influenced by their previous experience would reverse faster than chronic schizophrenics and normal controls.

However, the data indicating that the RL/L ratio of acute schizophrenics was significantly smaller than that of the other groups and the initial findings of a disrupted performance on both the initial discrimination and the reversal stages of the task when subjects had to maintain their attention for longer periods of time could not be fully explained on the basis of Hemsley’s (1987) theory. Therefore, in order to account for our findings it was proposed that due to the weakening of the influences
of regularities of stored memories to current perception, people with acute schizophrenia less influenced by their previous learning tend to attend equally all stimuli present in any given situation until such time that attention to the redundant stimuli is inhibited by the strengthening of the association between the appropriate stimulus and positive feedback.

This suggestion appears to accord with Hemsley’s (1987) as well as it takes into consideration Frith’s (1979) suggestion that the disruption in the cognitive performance of people with schizophrenia may be attributed to an intrusion into consciousness of material that normally exists below awareness (Frith, 1979). According to Frith’s (1991) revised model, the positive symptoms of schizophrenia and most specifically hallucinations and delusions of control are proposed to be due to a failure to monitor the intentions of self. Linking the experimental findings reported in the present study to Frith’s (1991) model, it might be possible, within the suggestion made above, that the cognitive equivalent of the disruption at the level of monitoring the intentions of self described by Frith (1991) to be due the intrusion into awareness of redundant information. Continuing, it is possible that due to this type of deficit people with acute schizophrenia experience difficulties to monitor their intention to select a particular stimulus manifesting in an inability (to this group of patients) to learn from previous experiences. As a result of this difficulty they randomly select any one of the presented stimuli until such time that the redundant stimuli is inhibited by the strengthening of the association between the appropriate stimulus and positive feedback.

The above explanation although predominantly attempts to account for the results of the present study, it also appears quite compatible with findings in the animal laboratory (e.g., Weiner and Feldon, 1986) using the same task. Also, it provides an alternative framework by which to examine the conflicting data reported by studies investigating the phenomena of LI and KBE. However, taking into consideration the many limitations associated with this study it is quite important to consider the
explanations and suggestions offered in this thesis as tentative requiring further investigation.
6. References


positron emission tomography study with raclopride. Archives of General Psychiatry, 47, 213-219.


Hemsley D.R. (1991): What have cognitive deficits to do with schizophrenia? In G. Huber (Eds): Idiopathische psuchosen. Shattarer Stutte


APPENDIXES
APPENDIX 7.1

PATIENT

NUMBER ----------------   GROUP ----------------

RATER -------------------------------

DATE -------------------------------

DIRECTIONS

DRAW A CIRCLE AROUND THE TERM UNDER EACH SYMPTOM WHICH BEST DESCRIBES THE PATIENT'S PRESENT CONDITION.

SOMATIC CONCERN
Degree of concern over present bodily health. Rate the degree to which physical health is perceived as a problem by the patient, whether complaints have realistic basis or not.

Not very mild moderate moderate-severe severe extremely
Present mild severe

ANXIETY
Worry, fear, or over-concern for present or future. Rate solely on the basis of verbal report of the patient's own subjective experiences. Do not infer anxiety from physical signs or from neurotic defence mechanism.

Not very mild moderate moderate-severe severe extremely
Present mild severe
EMOTIONAL WITHDRAWL
Rate degree to which the patient gives the impression of failing to be in emotional contact with other people or in the interview situation.

Not very mild moderate moderate-severe severe extremely
Present mild

CONCEPTUAL DISORGANIZATION
Degree to which the thought process are confused, disconnected or disorganized. Rate on the basis of integration of the verbal product of the patient; do not infer from the patient's subjective impression of his own level of functioning.

Not very mild moderate moderate-severe severe extremely
Present mild

GUILT FEELINGS
Over-concern or remorse for past behaviour. Rate on the basis of the patient's subjective experience of guilt as evidenced by verbal report with appropriate affecdt; do not infer guilt feelings' from depression, anxiety or neurotic defences.

Not very mild moderate moderate-severe severe extremely
Present mild

TENTION
Physical and motor manifestation of tension, "nervousness", and heightened activation level. Tension should be rated solely on the basis of physical signs and motor behaviour and not on the basis of subjective experiences of tension reported by the subject.

Not very mild moderate moderate-severe severe extremely
Present mild

MANNERISMS AND POSTURING
Unusual and unnatural motor behaviour which causes certain mental patients to stand out in the crowd of normal people. Rate only abnormality of movements; do not rate simple heightened motor activity here.

Not very mild moderate moderate-severe severe extremely
Present mild
GRANDIOSITY
Exaggerated self-opinion, conviction of usual abilities or powers. Rate only on the basis of patients statements about himself or self in relations to others.

Not

very

mild

moderate

moderate-severe

severe

extremely

Present

mild

moderate

moderate-severe

severe

DEPRESSIVE MOOD
Despondency in mood, sadness. Rate only degree of despondency; do not rate on the basis of inferences concerning depression based upon general retardation and somatic complaints.

Not

very

mild

moderate

moderate-severe

severe

extremely

Present

mild

moderate

moderate-severe

severe

HOSTILITY
Animosity, contempt, belligerence, disdain for other people. Rate solely on the basis of the verbal report of feelings and actions of the patient towards others. Do not infer hostility from neurotic, defences, anxiety nor somatic complaints.

Not

very

mild

moderate

moderate-severe

severe

extremely

Present

mild

moderate

moderate-severe

severe

SUSPICIOUSNESS
Belief (delusional or otherwise) that others have now, or have had in the past, malicious or discriminatory intent toward the patient. On the basis of verbal report, rate only those suspicions which are currently held whether they concern present or past situations.

Not

very

mild

moderate

moderate-severe

severe

extremely

Present

mild

moderate

moderate-severe

severe

HALLUCINATORY BEHAVIOUR
Perception without normal external stimulus correspondence. Rate only those experiences which are reported to have occurred within the last week and are described as distinctly different from the thought and imagery processes of normal people.

Not

very

mild

moderate

moderate-severe

severe

extremely

Present

mild

moderate

moderate-severe

severe
MOTOR RETARDATION
Reduction in energy level evidenced in slowed movements and speech, reduced body tone, decreased number of movements. Rate on the basis of the observed behaviour of the patient only; do not rate on basis of patient's subjective impression of own energy level.

Not very mild moderate moderate-severe severe extremely severe
Present mild

UNCOOPERATIVENESS
Evidence of resistance, unfriendliness, resentment, and lack of readiness to cooperate. Rate only on the basis of your impression.

Not very mild moderate moderate-severe severe extremely severe
Present mild

UNUSUAL THOUGHT CONTENT
Unusual, odd, strange or bizarre content. Rate here the degree of unusualness, not the degree of disorganization of thought processes.

Not very mild moderate moderate-severe severe extremely severe
Present mild

BLUNTED AFFECT
Reduced emotional tone, apparent lack of normal feeling or involvement.

Not very mild moderate moderate-severe severe extremely severe
Present mild

THANK YOU FOR YOUR COOPERATION
APPENDIX 7.2

MAINE SCALE

PATIENT ...................................................... GROUP ..............................
NUMBER ......................................................
RATER ......................................................
DATE ......................................................

INSTRUCTIONS

DRAW A CIRCLE AROUND THE NUMBER UNDER EACH SYMPTOM WHICH BEST DESCRIBES THE PATIENT'S CONDITION.
MAINE SCALE

P1. Does he tend to suspect or believe on slight evidence or without good reason that people or external forces are trying to or now do influence his behaviour, control his thinking?
   1. No unjustified suspicions.
   2. Will admit suspicion when pressed.
   3. Easily admits suspicion.
   4. Openly states others are trying to control him.
   5. Has firm conviction that he is influenced or controlled.

P2. Does he tend to suspect or to believe on slight evidence or without good reason that some people are against him (persecuting, conspiring, cheating, depriving, punishing) in various ways?
   1. No unjustified suspicions expressed.
   2. When pressed expresses belief that he is conspired against.
   3. Frequently inclined to suspect.
   4. Frank inclination to believe in persecution.
   5. Strongly expresses conviction of persecution.

P3. Does he have an exaggeratedly high opinion of himself of an unjustified belief or conviction of having unusual ability, knowledge, power, wealth or status?
   1. No expressed high opinion of himself.
   2. When pressed expresses high opinion of himself.
   3. Frequently expresses high opinion of himself.
   4. Open conviction of unusual power, wealth et.
   5. Strongly expresses conviction of grandiose or fantastic power, wealth etc.

P4. Does he tend to suspect or believe on slight evidence or without good reason that some people talk about, refer to or watch him?
   1. No unjustified suspicions.
   2. Will admit suspicion.
   3. Easily admits suspicion.
   4. Openly states that he is watched.
   5. Has firm conviction of being watched.

P5. Compared to others how openly hostile is he? Does he show hostility or a high degree of ill will, resentment, bitterness or hate?
   1. No open hostility.
   2. Relatively little hostility.
   3. Some hostility.
   4. Rather hostile.
   5. Very hostile.
N1. Does he have perceptions (auditory, visual) without normal external stimulus correspondence?
   1. None.
   2. When pressed admits hallucinations.
   3. Easily admits hallucinations.
   4. Openly admits frequent hallucinations.
   5. Openly hallucinations.

N2. On the basis of the integration of the verbal production of the patient, does he exhibit thought processes which are confused, disconnected or disorganised?
   1. As normal.
   2. Slight disorganisation.
   4. Marked disorganisation
   5. Complete disorganisation.

N3. How incongruous are his emotional responses? Eg., giggling or crying for no apparent reason or not showing any emotion when emotion would be appropriately shown.
   1. As normal.
   2. Slightly different from normal.
   3. Responses somewhat incongruous.
   4. Distinctly incongruous
   5. Very markedly incongruous.

N4. How well oriented is he as to time? For instance, does he know (a) the season (b) the month (c) the calendar year (d) the day of the week (e) how long has he been in hospital?
   1. As normal.
   2. Occasional confusion.
   4. Frequent confusion
   5. Marked continuous confusion.

N5. Does he assume or maintain peculiar, unnatural or bizarre positions?
   1. None.
   2. On rare occasions.
   3. For short periods.
   4. Frequently.
   5. All the time.
FORM OF CONSENT SIGNED BY ALL PARTICIPANTS PRIOR TO TESTING

DATE

NAME :

ADDRESS :

CODE :

CONSENT FORM

I consent to participate in the research study carried out by S. Georgiades, Clinical Psychologist at the Barnet NHS Trust, in which the different ways of responding to a specific task in the general population is studied.

I understand that all information obtained will be strictly confidential and will only be used for research purposes.

_____________________________
Signature

_____________________________
in the presence of
Stelios Georgiades,
Clinical Psychologist
SECTION V: Dissertation
Submitted for the Degree
Master of Science
in Clinical Psychology,
Institute of Psychiatry,
University of London, 1993
SCHIZOTYPY IN NORMAL CONTROLS AND THEIR PERFORMANCE ON THE BEHAVIOURAL PHENOMENON OF REVERSAL LEARNING

ABSTRACT
The research reported in this thesis examines the Gray's et al. (1991) and Hemsley's (1987) model of schizophrenia by employing the behavioural phenomenon of reversal learning. In order to eliminate the possibility of producing data confounded by uncontrolled variables such as medication, hospitalisation etc., often associated with the conflicting results produced in schizophrenia research, the subjects employed in the present study were drawn from the general population. They were grouped as high positive or low positive schizotypy on the basis of their scores on the subscales and factors of the Combined Schizotypal Trait Questionnaire (CSTQ; Bentall et al. 1989), and the RISC. Prior to the presentation of our data, in the introductory section a number of areas related to the models are reviewed. Reference is also made on the concept of schizotypy and its role to current schizophrenia research. The results, provide some support for the hypothesis that subjects scoring high in positive schizotypy scales do tend to reverse faster than low schizotypy individuals, when their performance on the behavioural phenomenon of reversal learning is not taken as an actual number but rather as the ratio between original learning and reversal learning. A final section discusses these findings within the framework of the theoretical models put forward in the introduction.
1. INTRODUCTION

The two most prominent theories of schizophrenia today are the cognitive and neurochemical theories, each of which has been the subject of a large body of literature in the area. But despite the importance attributed to these two areas of current schizophrenia research, a wide gulf seems to separate the cognitive and neurochemical bodies of knowledge (Baruch et al. 1988a). Attempts to provide support for the model introduced by Gray et al. (1991), which aims to relate findings in the neurochemical field, relating schizophrenia to hippocampal dysfunction (see Schmajuk 1987 for a review), and subsequently to link them to cognitive theories of schizophrenia (Hemsley 1987), has produced rather conflicting results. These conflicting results were mainly attributed to the large number of confounding variables such as medication, symptomatology and institutionalisation, often associated with schizophrenia research (e.g., Hemsley 1988).

The research reported in the present study, sought to examine the Gray's et al. (1991) and Hemsley's (1987) models by investigating information processing in schizophrenia, employing the behavioural phenomenon of reversal learning, without the influences of the confounding variables mentioned above. Drawing on evidence produced by current schizotypy research, which seems to suggest that the performance of high schizotypy individuals in cognitive tasks is similar to that of schizophrenics (e.g., Balogh and Merit 1985; Merit et al. 1986), the present study was carried out using psychiatrically healthy individuals. The performance on the reversal learning task of those subjects scoring high and of those scoring low on schizotypy scales was investigated.

Since main aim of the present study is to attempt to draw together evidence from different fields of current schizophrenia research, it is necessary, in the introductory section, to review a number of areas in order to provide the context within which the research reported in the next section of the thesis was conducted. A final section, discusses the results with respect to the theoretical models reviewed earlier.
1.1 Cognitive Theories

Historically, attentional disturbances have been viewed as being central to schizophrenia. In 1919, Kraeplin, became convinced of the relationship between attentional disorder and schizophrenia. He noted that "it is common for those patients to lose both the inclination and the ability on their own initiative to keep their attention fixed for any length of time ... there is occasionally noticed a kind of irresistible attraction of the attention to casual external impressions" (p.p. 6-7). Disturbances at this level were also reported by Bleuler (1950) following his detailed clinical observations. In addition, the extensive structured interviews reported by McGhie and Chapman (1961) concentrating on the experiences of newly admitted schizophrenics were influential in emphasising a relationship between schizophrenia and attentional disturbances.

Following in the tradition of Bleuler (1950), psychological research in this area is aiming at specifying a single cognitive dysfunction from which the abnormalities observed in schizophrenia might be derived (Hemsley 1988). Working on similar lines and highly influenced by the reported attentional abnormalities of schizophrenics, early research on the area has generated a large amount of data, most of which had indicated the "worse than normal" performance of schizophrenics on a variety of cognitive tasks (eg., Harris 1957; Weckonicz, 1957).

One of the early concepts proposed as being crucial in accounting for the poor performance of schizophrenics in such tasks, was that of "overinclusiveness"; i.e., the inability of schizophrenics to preserve conceptual boundaries (Cameron 1938). In subsequent research studies (Payne 1964; Payne et al., 1970), the problem of attentional deficits in schizophrenia was also highlighted and it was suggested that these deficits could be attributed to a defective filtering mechanism.

Similar attempts to produce models that would account for the attentional problems encountered in schizophrenia were made by a number of researchers (eg., Mednick 1958; Broen et al., 1966; Salzinger et al., 1964). The broad theoretical concepts though, on which
most of those research studies were based, made the interpretation of the data, in relation to the attentional disturbances observed in schizophrenia, almost impossible.

In a review carried out by Banister (1968), it was pointed out that in order for any progress to be made in schizophrenia, it was not enough to formulate models that would attempt to explain the observed abnormalities in schizophrenia. It was proposed that studies on the area, should be based on models of normal functioning within which the performance of schizophrenics could be explained and understood. Despite the problematic start of research in this area, more recently the acceptance of the "normal information processing approach" concerned with models of normal adult cognitive processes, has played a significant role in increasing our understanding of attentional functioning in the schizophrenic.

Influential in early studies of schizophrenics' cognitive disturbances was the model proposed by Broadbent (1958). This model, in its basic level, suggests a hypothesised filter that is functioning in a limited capacity channel. Due to the limited capacity of the information channel, this filter has the role of selecting stimuli on the basis of their physical characteristics filtering out and preventing the further processing of all irrelevant ones. Studies of schizophrenia carried out on the basis of this model employed mostly dichotic listening tasks (eg., Payne et al., 1970). But although the results reported indicated the poor performance of schizophrenics, the similar results obtained when psychiatric controls were employed, suggested that the poor performance of schizophrenics might not be due to a defective filter but rather due to ineffective processing.

The equivocal results led researchers to adopt the more recent probabilistic model of Broadbent (1971) in which the distinction between stimulus set (filtering) and response set (pigeon holing) is made. This distinction between stimulus set and response set has been more useful in subsequent research in the area due to its applicability to a number of other models.

Schneider and Shiffrin (1977) and Shiffrin and Schneider (1977) have proposed another theory of information processing in which they distinguished between automatic and
controlled processing. In that theory, it is maintained that automatic processing does not require attention or processing demands as it occurs outside the consciousness. For controlled processing though, the opposite is true; i.e., it requires attention, awareness and involves demands on the processing capacity.

Following these lines of theorisation, Frith (1979) proposed that one basis of the cognitive deficits observed in schizophrenia lies in the awareness of automatic processing which normally should be carried out below the level of consciousness. Frith suggests that the positive symptoms experienced by schizophrenics are their attempt to make sense of the abnormal input they are experiencing. Similar suggestions were also made by Maher (1983), who asserted that the observed symptomatology of schizophrenia is the outcome of the inability of schizophrenics to exclude from consciousness irrelevant information. Experimental studies based on Frith's hypothesis employing the "negative priming" paradigm have supported his model (e.g., Peters 1992).

Recently, in an attempt to unite the number of different models of "information processing" that have been proposed as possible explanations of the abnormalities observed in schizophrenia, Hemsley (1987) argued that most models at their basic level may be seen as illustrating the way in which "spatial and temporal regularities of past experiences influence the processing and awareness of current sensory input" (cited in Hemsley 1991 p.113). Linking this with the abnormal experiences reported by schizophrenics, he proposed that basic to schizophrenia, "is the weakening of the influences of stored memories of regularities of previous input on current perception" (Hemsley 1987).

Experimental studies attempting to examine whether schizophrenics are less able than non-schizophrenics to ignore irrelevant stimuli in a coherent manner, indicated that this might be the case. Strong support for this comes from studies which examine performance on tasks whose the demands are characterised by a strong association bias and thus the automatic processes in normals are non-adaptive; in such tasks, schizophrenics perform better (eg., Baruch et al., 1988a). Further support for the plausibility of this model, comes from the animal laboratory. There are studies available (eg., Lubow et al., 1982) in which
it is indicated that the performance of amphetamine-treated rats, unlike intact animals, is controlled by the prevailing situational condition, i.e., failing to make use of previous regularities and respond to any stimulus as if it was presented to them for the first time.

1.2 The Dopamine Hypothesis

One of the most prominent theories of schizophrenia today, is the dopamine hypothesis. In this hypothesis, it is maintained that schizophrenia, and more specifically the positive symptoms observed in the disorder, are due to the dysfunction of at least one dopaminergic system (DA) causing the brain to be in a hyperdopaminergic state. Evidence indirectly supporting the above hypothesis, comes from a number of different areas.

The first line of evidence, emerged after the discovery of therapeutically effective neuroleptic drugs (Davis 1965). These drugs, in addition to alleviating at least the positive symptoms of schizophrenia, produce side effects resembling Parkinson's disease; a disease which is known to be caused by DA depletion of the caudate nucleus (Homykiewicz 1973). On the basis of this observation, it was proposed that schizophrenia is caused by dopaminergic hyperactivity and that effective neuroleptic drugs must share some DA antagonistic action.

This proposition, seems to receive support from studies in which it was shown that the DA receptor blocking action and the clinical potency of neuroleptics is correlated (eg. Seeman et al., 1976; Jansseen 1976). More supportive evidence, arises from studies where the effects of the DA agonist amphetamine in normal subjects was examined. In a number of such studies, it was indicated that prolonged administration of amphetamine (eg. Connel 1958; Angrist and Gershon 1970; Griffith, et al., 1972; McKinney and Moran 1981) or of drugs of similar structure to that of amphetamine (eg., phenylpropanoline; Lake, Mason and Quirk, 1988) generate symptoms similar to those experienced by acute schizophrenics (amphetamine psychosis). In addition to the above, there have also been reports indicating that neuroleptic drugs can effectively alleviate the symptoms caused by amphetamine (Angrist, et al., 1974). Similar findings to the ones reported by human studies have also been reported in a number of animal studies. Utena and his colleagues (1975) for example, has reported that monkeys
become hyper-vigilant following amphetamine administration; a finding which was viewed by the researchers as a manifestation of paranoia.

The suggestion, that amphetamine should also exacerbate the symptoms of patients who have been diagnosed as suffering from schizophrenia, has not been fully supported by research studies. Reviewing the literature in the area published since 1937, Van Kammen and his colleagues (1982; cited in Birchwood, Hallet and Preston, 1988) noted that of the 285 drug free patients listed, only 25% of them have shown a symptomatic exacerbation following amphetamine administration. In a more recent comprehensive review carried out by Liberman and his colleagues (1987), about the effects of psychostimulants in schizophrenia, it was reported that only 40% of the schizophrenics receiving amphetamine have experienced an exacerbation in their symptomatology. This finding, when coupled with the fact that the majority of schizophrenics participating in the study were chronic institutionalised patients, seems to suggest that the usefulness of the dopamine hypothesis might be limited in accounting for the acute positive schizophrenic symptoms.

At this point, one needs to assert that although the evidence reviewed so far appears to support the dopamine hypothesis, the results have often been criticised as being circumstantial due to the indirect methods of investigation employed. Attempts to produce evidence supporting the dopamine hypothesis in its original form by employing more direct methods of investigation have been more elusive. In studies where it was attempted to provide direct evidence of elevated amounts of DA and its metabolite homovanillic acid (HVA), the results obtained were rather contradictory (e.g., Bowers 1973; Sedvall, et al., 1974; Post, et al., 1975; Beckman, et al., 1987). Similar contradictory results have also been noted in post-mortem studies. Studies which have shown consistently elevations of DA receptor number in the post-mortem brains of schizophrenics (eg. Bird et al., 1979; Mackay et al., 1980) are difficult to interpret due to the fact that most of the schizophrenic subjects used in those studies had been on neuroleptic medication. As a result, it is unclear as to whether observed elevation of DA receptors is directly linked with the disorder itself or is merely the side effects of medication (Creese and Sibley 1981).
But despite the many shortcomings of this hypothesis, even if we do accept that medication does not affect DA levels (Owen et al. 1978) and that there is indeed an increase in DA receptor density, as more recent studies have reported (e.g., Crawley et al., 1986; Wong et al., 1986), the relation between the dopamine hypothesis and the clinical symptoms of schizophrenia is still far from clear. As it is known, the key symptoms of schizophrenia are cognitive (e.g. hallucinations and delusions), and the most salient cognitive features of the disorder is a deficit in attention and more specifically in the ability to inhibit irrelevant information (e.g., Hemsley 1987). Animal studies though have suggested that the major functions of DA systems are in the selection of motor programmes (Swerdlow and Koob 1987) and the motivational effects of rewards (Robins and Everitt 1982); neither of which seems directly relevant to the symptomatology or the most salient cognitive features of the disorder.

1.3 Temporal Lobe Pathology
Evidence provided by morphological studies on the brains of schizophrenics did not reveal in any consistent manner that schizophrenia is associated with damage or abnormality in those areas of the brain that are rich in dopaminergic cells. Evidence produced by research on other areas of the brain, seems to indicate that schizophrenia may be associated with abnormalities in a number of areas of the temporal lobe such as the hippocampal formation, the cingulate cortex and the amygdala (e.g., Beckman et al. 1987; Falkai and Bogerts 1986).

In an attempt to resolve and integrate these rather ambivalent findings of the dopamine hypothesis with the existing advances in temporal lobe pathology, Gray and his colleagues (1991) proposed that the neural dysfunction specific to schizophrenia is the result of an abnormal input from the hippocampal formation (subiculum) to nucleus accumbens; the latter being the major target of the mesolimbic DA system.

On the basis of the evidence (Gray 1982) that the septohippocampal system plays an important role in monitoring as to whether expected and actual outcomes match, it was
suggested that when features of any particular stimulus match with those that are already present in the comparator, the brain labels them as not being novel. Once input from the hippocampus indicates that an incoming stimulus is not novel, basal ganglia, and more specifically the nucleus accumbens, terminate the initiated motor programme (Swerdlow and Koob 1987). Furthermore, integrating the above with evidence that hippocampal damage reduces DA utilisation in the nucleus accumbens (Springer et al. 1982) and that neuroleptic medication can alleviate some of the symptoms produced by hippocampal damage (Isaacson 1980), they suggested that a dysfunction in the interaction between the hippocampus and the nucleus accumbens, causes reduced DA utilisation in the nucleus accumbens, compensatory up regulation in other areas of the mesolimbic DA systems and therefore DA overactivity in these areas.

As discussed above, the major role assumed by the septohippocampal system is that of monitoring incoming stimuli. On recognition that stimuli are no longer novel, the nucleus accumbens has the major function of terminating the initiated motor programme. From this, one would assume that the effects of a dysfunction in the interaction between the subiculum and the nucleus accumbens would be most evident on those occasions when the subiculum would be expected to label a stimuli as no longer being novel due to the occurrence of a match. If due to the dysfunction, abnormal input from the subiculum does not lead nucleus accumbens to terminate the motor programme, the subject will continue to treat familiar stimuli as being unfamiliar. Elaborating from this, one could suggest that due to this dysfunction, schizophrenics would fail to learn from earlier experiences and they will continue to approach any stimuli as though it is presented to them for the first time. It is argued by Gray et al. (1991) that this disruption of the information processing in acute schizophrenics (see section 1.2) results in the formation of positive symptoms, drawing on Hemsley's (1987) proposition that basic to schizophrenia "is the weakening of the influences of stored memories of regularities of previous input on current perception".
1.3.1 Experimental Evidence

Evidence in attempting to provide support for Gray's et al. (1991) model comes from three different directions in schizophrenia research, from animal studies, studies using normal volunteers who have received different doses of d-amphetamine and from studies employing groups of acute and chronic schizophrenics. Three of the cognitive paradigms employed with regard to this model, are the latent inhibition (Lubow 1973; Lubow et al. 1982), the Kamin blocking effect (Kamin 1968) and the partial reinforcement extinction effect (Gray 1975). The reason for employing these behavioural phenomena derives from the fact that all three of them appear to share the following characteristics: (i) they depend on the control of attention by prior experience, (ii) they are affected by DA releasing drug amphetamine and by neuroleptics and (iii) they are affected by damage to hippocampus.

Evidence produced in the animal laboratory, seems to indicate rather consistently that low doses of amphetamine and damage to the hippocampal formation leads the nucleus accumbens in a hyperdopaminergic state. Moreover, both hippocampal damage and low dosage of amphetamine, seem to abolish latent inhibition, (Weiner et al. 1984 Solomon et al. 1981, Weiner et al. 1990; Tarrasch et al., 1992) the Kamin blocking effect, (Crider et al. 1982; Solomon 1977; Ricket et al. 1979) and the partial reinforcement effect (Weiner et al. 1985; 1987; Jarrard et al. 1986; Rawlins et al. 1980; Feldon and Gray 1979a, 1979b). An effect which is reversed by the administration of neuroleptics.

Attempts to investigate these behavioural phenomena in humans, following the administration of amphetamine, has failed to produced any straightforward, easy to interpret results (e.g., Gray 1991).

As discussed in section 1.4, Gray and his colleagues (1991) have proposed that the positive symptomatology observed in schizophrenics may be the result of a disruption in the information processing level due to their hyperdopaminergic state. Moreover, as it is known, positive symptomatology is regarded as the typical cluster of symptoms experienced by schizophrenics when in the acute phase.
On the basis of this distinction, Baruch et al. (1988a) using a latent inhibition task tested a group of acute schizophrenics within 2 weeks following admission to the hospital, and some weeks later when medication was stabilised. A second group consisting of chronic schizophrenics were also tested. The results obtained were that the acute group on first testing did not show any latent inhibition whereas normal latent inhibition was shown in chronic schizophrenics and in acute patients during the second testing when medication was stabilised. Findings such as these seem to provide us with evidence in support of the Gray's et al (1991) model, i.e., that the differences obtained in the performance of acute and chronic schizophrenics, in a latent inhibition task, may be due to the hyperdopaminergic state of the acute schizophrenics before fully medicated and the presence of positive symptoms.

A closer examination of these results though, seems to indicate that the explanation offered accounting for them is rather simplistic. This becomes especially evident when considering that chronic schizophrenics (not in a hyperdopaminergic state), may also experience positive symptoms. How does one account for these symptoms in the absence of dopamine overactivity? Moreover, high scores in the brief psychiatric rating scale (as obtained by the above authors) cannot be viewed as a valid way of indicating the presence of positive symptomatology in acute schizophrenics, as certain patterns of behaviour included in the questionnaire cannot be classified as being either positive or negative.

These rather conflicting findings, become even more difficult to interpret when confounded with other variables present in studies employing schizophrenics. Variables such as the bizarre perceptual experiences that patients might experience during the early stages of the disorder, the negative effect it might have on them when moved into an unfamiliar environment, the heterogeneity of symptoms, medication or institutionalisation (in the case of chronic patients) seem to highlight the problems encountered in schizophrenia research pointing out to the need of a more homogeneous group of subjects who are unaffected by extraneous factors.
1.3.2 Reversal Learning

Another behavioural phenomenon that also seems to share the same predispositions as the three behavioural phenomena discussed in the previous section is the "reversal learning". This behavioural phenomenon, which will be reviewed in some detail here due to its direct relevance in the present study, was originally developed in the animal laboratory by Lawrence (1949). Subsequently reversal learning was used and further developed by other researchers (eg. Sutherland and Holgate 1966; Mackintosh 1965). In a typical reversal learning task, the animal is initially rewarded for one response (e.g. going to the lighted arm of a T-maze) in a two choice paradigm. After a learning criterion has been reached, the reward is given in a subsequent phase for the alternative response (e.g., going to the dark arm).

According to Mackintosh (1983), discrimination learning involves several processes. Such processes may include excitatory and inhibitory conditioning to S+ (presentation of which signals the availability of reinforcement) and S- (signalling the absence of reinforcement); conditioning to irrelevant stimuli; attentional learning etc.. Reversal learning, on the other hand, is assumed to involve two specific processes: (a) extinction of the original discriminative response and (b) the acquisition of a new discriminative response. The first process, may be seen to reflect the difference in the associative values of the original S+ and S-. By consistently offering reward whenever a particular stimulus is selected (e.g., a particular colour, shape or direction) it increases the excitatory strength of that stimulus (S+) and the inhibitory strength of the alternative stimulus (S-). As a result of this increase in the excitatory strength of S+ and the inhibitory strength of S-, greater persistence will be observed in selecting the former S+ at the outset of reversal. The second process, is considered to reflect the attention to the relevant stimuli. As the associability of the relevant stimuli is increased, a more rapid shift of choice to the new S+ is observed (in Weiner and Feldon 1986).

According to Gray's et al (1991) model and to Hemsley's (1987) proposition, basic to schizophrenia is the failure to learn from previous experiences and in most tasks, schizophrenics continue to approach any stimulus as though it is presented to them for the first time. Linking this to the reversal learning task, one would expect schizophrenics to be...
less influenced by previous learning at the outset of reversal, and consequently to reverse faster than normal controls.

Experimental studies attempting to provide support for this hypothesis come from three different directions in schizophrenia research; the animal laboratory, from studies in which groups of schizophrenics and normal controls were employed and from studies where the performance on reversal learning tasks, following hippocampal lesions, was studied.

Evidence produced in the animal laboratory, has shown that amphetamine administration dramatically facilitates reversal learning (e.g., Calhoun and Jones 1974; Kulig and Kulhoun 1972; Weiner and Feldon 1986). This finding seems to indicate that amphetamine decreases the effects of phase 1 learning on subsequent cognitive processing. In studies where the performance of normal controls on reversal learning tasks was compared to that of schizophrenics have yielded rather conflicting results. In some of these, there was evidence to suggest that schizophrenics shift faster than normal controls at the outset of reversal (e.g., Nolan 1968) whereas in others, no difference was reported (e.g., Nolan 1974; Kamis and Muzekari 1973). A closer examination of the design of those studies though, seems to suggest that drawing conclusions from such studies is rather problematic due to the not so well defined symptomatology of the schizophrenic groups employed (e.g., Nolan 1968).

Evidence produced in animal as well as in human studies where the performance on reversal learning tasks following hippocampal damage was investigated, indicates that, unlike the behavioural phenomena mentioned in section 1.4.1 (latent inhibition, Kamin blocking effect and partial reinforcement extinction effect), the effects of hippocampal damage differ from those of the amphetamine treated rat. A large body of evidence, seems to point out that hippocampal damage is associated with impaired reversal learning (e.g., Mahut 1971; Jones and Mishkin 1972; Daum et al. 1991).

The well documented findings that amphetamine improve reversal learning, appears to be in line with the hypothesis that schizophrenics should reverse faster than normal controls. The evidence though that hippocampal damage disrupts reversal learning and the conflicting
findings of studies employing schizophrenics, seem to suggest that drawing conclusions on the basis of these findings is rather problematic emphasising this way the need of further research employing more homogenous groups of patients.

1.4 The Problem with the Concept of Schizophrenia

The evidence reviewed so far, was mainly focused on the two most prominent theories in current schizophrenia research (cognitive and neurochemical theories). Moreover, as indicated, attempts to provide support for a model (Gray et al. 1991) intending to integrate these two different approaches have failed to provide solid evidence in favour of any of the above theories. This was mainly attributed to the influences of extraneous factors, often associated with schizophrenia research, such as the effects hospitalisation or medication.

In addition to the problem of confounding variables present in schizophrenia research, another problem that also appears to play an important role in the future of research in this area, is the way in which the diagnosis of the disorder is made. Despite the large body of evidence produced by research investigating the psychological processes underlying schizophrenia over the last century, the problem of diagnosis continues to remain the target of many criticisms.

Schizophrenia, as a diagnostic category, encompasses a large number of heterogeneous symptoms ranging from delusions and hallucinations to catatonia, and individuals can be classified as being schizophrenics on the basis of only few of those symptoms. As a result, the symptoms, on the basis of which one individual is labelled as suffering from the disorder can be completely different from those present in someone else who is also labelled schizophrenic (Bentall 1990). In order to overcome these problems of diagnostic classification, a number of operational criteria for schizophrenia have been developed in recent years (e.g., DSM III-R). The end result though was that despite acceptable reliability within any given system, concordance between diagnosis based on different systems has been very low (Brockington et al. 1978), failing to provide us with any clear dividing lines between different types of schizophrenia, between schizophrenia and other disorders or indeed between the disorder itself and normality.
On the light of all these, researchers have often expressed the view that although empirical attempts to understand madness or psychosis should not be rejected, nevertheless, they also propose that the concept of a single diagnostic entity, schizophrenia, as we know it, does not exist (e.g., Bentall et al. 1988).

1.5 The Concept of Schizotypy

1.5.1 Measures of Schizotypy

The view that a continuum may exist between psychosis and normality, as expressed in the "stress diathesis model" (Zubin and Spring 1977) cannot be described as a recent trend in schizophrenia. On the contrary, according to Manfred Bleuer (1978), this has been represented in the literature from as early as the time of Bleuler (1911) when the concept of schizoid personality was first reported. Currently, evidence supporting this continuity model, comes from a number of different directions in schizophrenia research.

Findings from the psychiatric phenomenology research, indicate that the diagnosis of schizophrenia depends on arbitrary cut-off points which define the boundaries between schizophrenia and other diagnostic categories as well as between illness and health. This, has led a number of researchers not only to question the validity of the concept of schizophrenia as a unitary illness (e.g., Bentall et al. 1988), but also to argue that a continuum may indeed exist between psychosis and various borderline states. This argument, seems to accord well with the identification of a number of personality disorders (as described according to their clinical features in the DSM III-R) which appear to share a number of characteristics with major psychosis. As an example to the above, we may be referred to the schizotypal personality disorder, disorder which although its difference with the normal personality is quantitative rather than qualitative, clinically, it falls in the schizophrenic spectrum.

Over the past two decades or so, there have been increasing attempts to provide more concrete evidence that would support the continuity model of psychosis. These attempts have been
mostly focused in devising questionnaire scales that would be capable of identifying and measuring schizoid or schizophrenic like traits in individuals who are regarded as being psychiatrically healthy. Some of the most important of these scales are described below.

**The Eysenck Psychoticism Scale:** The Eysenck's Psychoticism scale, can be described as one of the earliest serious attempts to measure predisposition towards psychoticism in psychiatrically healthy people. Approaching the issue of psychosis proneness from the personality theory perspective, Eysenck had identified a personality dimension which he named psychoticism.

**Claridge's Schizotypy Scales (STQ):** After reviewing early psychometric approaches to the measurement of schizotypy, Grove (1982) has proposed that assessment strategies based on clinical signs and symptoms were more successful than alternatives employing highly inferential constructs. Following this proposition, Claridge has devised two scales which attempted to model the diagnostic criteria delineated in DSM III for schizotypal personality disorder (STA) and borderline personality disorder (STB).

**The Nielsen Petersen Schizophrenism Scale (NPS):** Is another scale which uses the symptoms of already defined syndromes as the basis for constructing questionnaires. In this scale Nielsen and Petersen (1976) by adopting the term schizophrenia instead of psychoticism (as they did not want to include the affective psychoses which they considered as being unrelated to the concept) attempted to construct an ad hoc schizophrenia scale that would tap sub-schizophrenic traits such as withdrawal and attentional failures.

**The Chapman Scales:** Taking the actual phenomenology of psychosis Chapman and his colleagues have attempted to construct a number of different scales each of these concentrating on a specific symptom. According to Chapman's view, devising such scales serves two purposes: firstly, it might provide an insight about the possibility that different clusters of prepsychotic symptoms might presage different psychoses hidden within "schizophrenia" and other categories of psychosis. Secondly, they observed that some non-psychotic individuals who, in the more general questionnaires, can be categorised as low
schizotypy, report isolated psychotic experiences. Therefore, by tapping separately individual symptoms does not only provide us with a tool of identifying schizotypy but also it opens up the possibility of examining individual differences among schizotypal individuals. These scales, have been designed to measure physical (61 items) and social (40 items) anhedonia (PhA and SoA respectively) (Chapman, Chapman and Raulin 1976), perceptual aberration (PAb) (35 items; Chapman, Edell and Chapman 1980), magical ideation (Mgl) (30 items - Eckblad and Chapman 1983), and hypomanic traits (HoP) (48 items - Eckblad and Chapman 1986).

**Delusions Symptoms States Inventory:** Four symptom scales derived from the Delusions Symptoms States Inventory (Foulds and Bedford 1975), namely delusions of contrition (dC), delusions of persecution (dP), delusions of Grandeur (dG) and delusions of disintegration (dD). These scales, according to Bentall et al (1989) are mostly intended to assess the presence of active psychotic symptomatology rather than measuring schizotypal traits.

**The Lunav Slade Hallucinatory Scale (LSHS):** This scale, which can be described as being conceptually similar to the Chapman's scales, attempts to identify and measure the vulnerability of non-psychotic individuals to hallucinations.

**Schizoidia Scale (GMS):** A brief schizoidia questionnaire, derived from the MMPI by employing a complex taxonomic theory, that discriminates schizophrenics from non-schizophrenics (Golden and Meehl 1979).

**The Rust Inventory of Schizoid Cognitions (RISC):** Rust's (1987,1988) questionnaire (26 items), was devised by taking into consideration aspects of schizotypy tapped by other questionnaire scales. Although this questionnaire is mainly concerned with the measurement of schizotypal cognitions associated with the positive symptoms of acute schizophrenia, its main difference from the other questionnaires is that it was developed and standardised with special attention to a normal distribution in the general population.
1.5.2 Schizotypy and Cognition

Currently, a growing body of evidence exists indicating that individuals scoring highly on these scales show similarities with psychotic patients not only at the level of phenomenology but also in neuropsychological functioning (e.g., Gur 1978; Rawlings and Claridge 1984) and psychophysiological responding (e.g. Claridge and Chappa 1973; Claridge and Birchall 1978; Geyer et al., 1990). The performance of high schizotypy individuals on cognitive tasks, is another area that has been investigated. This area will be reviewed somewhat more extensively here, due to its immediate relevance to the present study.

The possibility that psychiatrically healthy individuals scoring highly on the schizotypy scales might perform in cognitive tasks in a similar way to schizophrenics is a line of research pursued extensively during the past decade with promising results. The generally poor performance of schizophrenics in comparison to normal or psychiatric controls on most cognitive tasks has always been a major obstacle in identifying the underlying deficit of the disorder. Although the problem of identifying the underlying deficit of schizophrenia continues to persist, an increasing body of evidence seems to suggest that high schizotypy individuals show similar impairments in certain cognitive tasks to those of schizophrenics. These findings are promising in that they might eventually allow us to make inferences about the underlying deficit of the disorder without being confounded with variables such as medication, institutionalisation or symptomatology.

As an example of the above, we may be referred to the evidence produced by experimental studies in which the cognitive task of backward masking was employed. In these studies, it was shown that schizophrenics tend to need longer inter-stimulus intervals than psychiatric controls for identifying correctly the test stimulus (Saccuzzo et al 1974; Braff 1989). This finding was interpreted as indicating that there might be a deficit either in the early visual processing or in the transfer from iconic to short term memory. Subsequent studies employing similar tasks in non-psychiatric subjects, showed that the performance of high scorers in the Perceptual Aberration, Magical Ideation and Physical Anhedonia scales (Balogh and Merit 1985; Merit et al, 1986) in these tasks, was similar, but less severe, to that of schizophrenics. Similar findings were also noted in another study where the performance
of students with high schizotypic MMPI profile on a backward masking task was compared with that of schizophrenics (Nakano and Saccuzzo 1985).

In a study where the performance of normal individuals in the cognitive task of latent inhibition was investigated, it was shown that high P scores in the EPQ showed a reduced latent inhibition (Baruch et al. 1988b). This finding, to a certain extent, appears to resemble the performance of acute schizophrenics and the amphetamine treated rat.

The availability of a number of different schizotypal scales identifying different groups of schizophrenic like traits seem to be of some value as it recognises the wide heterogeneity of symptoms present in psychosis. Experimental studies where an array of schizotypal scales are employed in conjunction with a cognitive task, could potentially indicate that a certain cognitive deficit, as mapped out by the cognitive task, might be specific to a particular type of schizotypy. In a study carried out by Kendler and his colleagues (1991), it was shown that attentional dysfunction and eye tracking error as measured by a battery of attentional tests was related to negative symptomatology. In another study where the incidental learning task and the Kamin blocking task were employed, there was evidence that performance was affected in acute but not in chronic schizophrenics (Jones et al., 1992a). Attempts to investigate the possibility of these tasks being affected in high schizotypal individuals indicated that only the "unusual perceptual experiences" factor as taken from the STA (Hewitt and Claridge 1989) was related to their previous findings.

In the light of the large number of studies supporting the view that there might be a continuum of personality and cognitive traits that predispose psychiatrically healthy individuals to schizophrenia, Claridge (1985), argued that just as blood pressure acts both as a disposition to disease and as a trait of normal functioning, schizotypal characteristics have similar status with respect to schizophrenia; an argument which appears to have received support from the prospective study carried out by Chapman and Chapman (1987). In that study, control subjects were compared with groups of students who scored highly on one or more of their various psychosis proneness scales. The results obtained, indicated that a significant
The proportion of these high schizotypal subjects had sought professional help for emotional problems during the follow-up period.

1.5.3 The Value of Schizotypy in Understanding Schizophrenia

Up to this point the emphasis has been placed in discussing the development of questionnaire scales that measure schizophrenic like traits and how these are used to provide support for a continuum of personality and cognitive traits that predispose psychiatrically healthy individuals to schizophrenia. At this stage though, one could ask what is the value of understanding the structure of schizotypal traits in increasing our knowledge about schizophrenia.

Researchers working in the area seem to suggest that any increase in our understanding in the structure of schizotypal traits may be of considerable importance in schizophrenia research. One of the most optimistic arguments about the value of understanding schizotypal traits postulates that by employing an integrated set of schizotypy scales, it may be possible to identify individuals who show many features that characterise psychosis and yet have not been affected by variables such as confusion, withdrawal, medication, institutionalization that complicate most of the research carried out in psychiatric patients. Moreover, as suggested by Raine and Manders (1988) more significant differences might be obtained in studies comparing schizophrenics with normal controls when these schizotypal characteristics are taken into consideration. Finally, investigating the high schizotypal individual might enhance our understanding about the factors that play a significant role in increasing the vulnerability to a psychotic breakdown.

1.5.4 Problems with Schizotypal Scales

One problem associated with the use of these questionnaire scales though, stems from the important recognition of the heterogeneity of symptoms found in psychotic syndromes leading to the development of a large number of schizotypal scales. Although one cannot deny that all of them have the common purpose of identifying and measuring schizophrenic
like features in normal individuals, the problem of deciding which of these scales are most appropriate to employ in a research study remains. And this is mainly due to the lack of knowledge regarding the extent of which schizotypal characteristics are measured by each of these questionnaire scales and the relationship between the different scales. The findings that each of these scales is related to psychosis cannot guarantee that all the scales measure the same or even overlapping traits.

In a study carried out by Chapman and his colleagues (1980) in which a group of students were tested, it was found that scores on their perceptual aberration and physical anhedonia scales were uncorrelated. In another study, subjects were given the EPQ P scale, the Golden & Meehl's schizodia scale, two composite schizotypy scales derived from the MMPI, the physical anhedonia, perceptual aberration, magical ideation scales, and a non conformity scale. An analysis of the results showed high correlations only between some of the scales (Chapman, Chapman and Miller 1982).

An explanation for this low correlation between the different schizotypy scales, might be offered by accepting that predisposition might occur towards only some aspects of the disorder and not in others. Like schizophrenia itself, the presence of one cluster of symptoms does not necessarily imply the presence of another. Similarly, individuals scoring high on a set of schizotypy scales measuring predisposition towards a particular group of schizophrenic symptomatology, might, at the same time, score low on some others measuring predisposition to different symptoms.

Although the current diagnosis of schizophrenia encompasses a wide range of heterogenous symptoms ranging from paranoid to catatonic, these symptoms are often broadly categorised as representing either a positive or a negative set of symptoms. According to Garety (1992; in Birchwood and Tarrier 1992), most authors regard as positive symptoms, those symptoms which are regarded pathological by their presence (e.g., hallucinations, delusions, thought disorder and bizarre behaviour) rather than by the absence of some aspects of functioning. The absence of some aspects of functioning (such as flatness of affect, poverty of speech, apathy, inattention and social withdrawal) characterises negative symptoms. The division
however of schizophrenia into positive and negative, has often been criticised by researchers as being inadequate in describing all aspects of the disorder, and it has been suggested that the additional category of cognitive disorganisation need to be added (e.g. Liddle 1987).

Returning to the problem of low correlation between the different schizotypy scales, Muntaner and his colleagues (1988), in an attempt to overcome this problem, gave student subjects a number of different schizotypy scales. This included a Catalan versions of the EPQ, the two Claridge scales and the Chapman anhedonia, magical ideation and perceptual aberration scales. By performing factor analysis on the data obtained, they have managed to extract three factors. These factors appeared to correspond to cognitive aberrations, resembling those found in the positive aspects of psychotic symptomatology, anhedonia and social deviancy. Working on similar lines, Raine and Albutt (1989) carried out a series of factor analyses on a short battery of schizotypy scales administered to student subjects. From one of these analyses two factors were provided, one with a high loading for Eysenck's P and the anhedonia scale and the second with a high loading on the two Claridge scales, the Lunay-Slade Hallucination scale, the Chapman perceptual aberration scale and the Nielsen and Petersen schizophrenism scale.

By combining a wider range of scales measuring psychotic traits or symptoms with the Eysenck Personality Questionnaire, Bentall, Claridge and Slade (1989) have formed a Combined Schizotypal Traits Questionnaire (CSTQ) and administered it in 180 subjects. When factor analysis was carried out on the scores obtained, three factors were extracted. These factors appeared to measure traits relating to (i) positive psychotic symptomatology (ii) negative symptomatology, and (iii) aspects of schizotypy involving social anxiety and cognitive disorganisation. By including the scores derived from the Delusions Symptoms States Inventory of Foulds and Bedford (1975) a fourth factor was obtained which seemed to measure an asocial component of schizotypy. The results produced by this study seem to support earlier findings that psychotic traits are not unidimensional but rather they are distributed in at least three dimensions. But the most important aspect of this study seems to be the promising advantages for future research when a single questionnaire (consisting of a
combination of schizotypy questionnaires) would be capable of measuring an array of psychotic traits in normal individuals.

With respect to the three dimensions of psychotic traits (positive, negative and social disorganisation), the subscales of the CSTQ, can be grouped (by taking as an arbitrary criterion loadings of subscales of over 0.5 on any factor in the Bentall et al. 1989 study) as follows: those measuring predisposition towards positive symptomatology (Factor 1 - STA; HoP; PAb; Mgl; dC; dP; dD; dG; LSHS), those referring to social anxiety aspects of schizotypy (Factor 2 - EPQ (N); STA; STB; NP; GMS), those measuring predisposition towards negative symptomatology (Factor 3 - SoA; PhA) and those referring to a form of asocial schizotypy (Factor 4 - EPQ(P)). As it can be seen from above, the STA although designed to measure predisposition towards positive symptomatology, it also loads with Factor 2 (measuring social anxiety aspects of schizotypy), a category which is often regarded by many researchers as part of the positive symptoms category (see Garety 1992). The EPQ (E), the only scale not included in the above categorisation, shows a high negative loading to Factor 3 (predisposition to negative symptomatology).

1.6 Conclusion

On the basis of the evidence reviewed so far, it appears that the cognitive and the neurochemical theories are the most prominent theories of schizophrenia today. But despite the great volume of research produced in each of these areas, they are not without their critics and a great gap seems to separate them in terms of the way in which the concept of schizophrenia is viewed. This gap becomes even more evident when schizophrenia is regarded as a heuristic concept based on a unitary system where all aspects of the disorder such as aetiology, symptomatology and treatment are mutually and coherently related.

Recently, Gray and his colleagues (1991), in order to resolve the ambivalent findings of DA research, have proposed a model in which DA findings are related to the available evidence relating schizophrenia hippocampal function. In this model, it is maintained that basic to acute schizophrenic symptomatology is a disruption at the information processing level.
Elaboration of this model, seems to accord well with the proposition of Hemsley (1987) which suggests that schizophrenic symptoms are due to a "the weakening of the influences of stored memories of regularities of previous input on current perception".

Attempts to provide support for this model, although encouraging when considering the evidence produced in the animal laboratory, on transferring them to human studies, where schizophrenic subjects were employed, the evidence produced was far from readily interpretable. This was mainly due to a number of confounding variables often associated with schizophrenia research such as symptomatology, medication, institutionalisation etc..

Although main aim of the present study is to attempt to examine the Gray et al. (1991) model, by employing the behavioural phenomenon of reversal learning, emphasis is also given to the role played by schizotypy research as it might provide us with an alternative method of studying the underlying deficit of schizophrenia without the influences of confounding variables. Evidence produced by current research on schizotypy indicates that individuals scoring high on schizotypy scales, resemble (but to a lesser extent) psychotics not only in the phenomenology but also in their neuropsychological and psychological functioning.

Attempts to provide evidence of performance on certain cognitive tasks on the basis of the scores produced in schizotypy scales measuring predisposition towards a particular category of symptoms, have produced a rather mixed picture. Although the evidence available making a distinction between predisposition towards positive and negative symptoms is sparse, there are however, some indications pointing out towards a stronger association between predisposition towards a positive symptomatology and learning tasks such as latent inhibition (Baruch et al. 1988b) and Kamin Blocking Effect (Jones et al. 1992b).

Elaborating from this, in the present study the performance on the reversal learning task of those individuals scoring high on schizotypy scales measuring predisposition towards positive symptomatology (STA; HoP; PA;b; MgI; dC; dP; dD; dG; LSHS) will be investigated. On the basis of the results obtained, inferences will be made as to whether the evidence produced supports or otherwise the Gray's et al. (1991) and Hemsley (1987) model on schizophrenia.
1.7 Hypotheses

As it was discussed in section 1.4, Gray's model (1991), postulates that a dysfunction in the interaction between the hippocampus and the nucleus accumbens, causes disruption of the information processing in acute schizophrenics resulting in the formation of positive symptoms. Linking this to Hemsley's (1987) proposition, it was suggested that basic to schizophrenia is the failure of schizophrenics to learn from previous experiences and that they respond to any stimulus as if it was presented to them for the first time. Based on the above model, in the case of the reversal learning task, one would expect acute schizophrenics to be less influenced by previous learning and therefore to shift faster at the outset of reversal than normal controls. In order to test the prediction of faster reversal learning associated with the symptoms of acute schizophrenia without the influences of confounding variables such as medication, institutionalisation etc., the following hypotheses will be tested using normal controls.

1.7.1 Hypothesis 1

Individuals with a high scores in the positive symptomatology factor of the CSTQ (factor 1) and in the scales included in the CSTQ measuring predisposition to positive symptoms (STA; HoP; PAb; Mgl; dC; dP; dD; dG; LSHS) as well as on the RISC, are expected to reverse faster on a reversal learning task than subjects who generally score low on the CSTQ positive factor and on the subscales measuring predisposition to positive symptomatology and on the RISC (as opposed to no difference between groups expressed by the null hypothesis).

1.7.2 Hypothesis 2

With respect to factors 2, 3, and 4 derived from the CSTQ and the scales included in the CSTQ which do not measure positive symptomatology (SoA; PhA STB; EPQ(E); EPQ(N); EPQ (L); EPQ (P), NPS; GMS), it is predicted that there will be no difference in the performance on the reversal learning task between high and low scorers in these scales (as opposed to significant differences between high an low scorers expressed by the null hypothesis).
2. METHODOLOGY

2.1. Subjects
Subjects were recruited from the subject pool of the Institute of Psychiatry (a group of adults drawn from the general population who consented to be contacted for research purposes) and from those responded to advertisements placed in the Camberwell Job Centre, Wood Green Job Centre and Haringey Library. Subjects with any sight difficulties, a history of mental illness and drug or alcohol dependency were excluded from the study.

All subjects constituted part of the normal sample whose scores were grouped down in relation to their score on the Combined Schizotypal Traits Questionnaire (Bentall et al. 1989).

2.2. Design
A within subject design was adopted in this study. All subjects were asked to complete a reversal learning computerised task as well as to complete the following set of questionnaires: the Combined Schizotypal Traits Questionnaire (Bentall et al. 1989) and the Rust Inventory of Schizotypal Cognitions (RISC; Rust 1987). All subjects were also asked to complete the Raven's Coloured Progressive Matrices (Raven, Court and Raven 1988).

On the basis of the scores obtained in the factors as well as on the eighteen subscales included in the Combined Schizotypal Traits Questionnaire (Bentall et al. 1989) and the Rust Inventory of Schizotypal Cognitions (RISC; Rust 1987), subjects were divided into high and low schizotypy groups and differences in the performance of the reversal learning task were investigated.

2.3. Apparatus and Stimuli
Two stimuli differing along the dimensions of both shape and colour, were presented on an ATTARI 1040 ST computer and a colour monitor. The task, is a computerised version of the original "card task" employed by Nolan (1968). The two colours appearing on screen were always red and green and the two shapes always a circle and a triangle. The stimuli appeared
in randomised combinations (e.g., green circle - red triangle; red circle - green triangle; each of the combinations appearing either on the right or on the left of the screen). The main task of the subjects was to decide by error and trial which one of the two colours or shapes was regarded by the computer as being the correct one. The subjects responded by pressing the appropriate response key; response keys were situated directly below the monitor, each key corresponding to each stimulus. Correct responses were followed by a smiling face on the screen whereas false responses were followed by a crying face.

2.4 Questionnaire
In addition to the computerised task, all subjects were asked to complete the Combined Schizotypal Traits Questionnaire (Bentall et al. 1989), the Rust Inventory of Schizotypal Cognitions (RISC; Rust 1987) and the Standard Progressive Matrices (Raven, Court and Raven 1988) - see Appendix 7.2.

The Combined Schizotypal Traits Questionnaire consists of 420 items; these items derive from the following eighteen scales designed to measure different aspects of schizotypy:

1. Schizotypal Personality (STA; Claridge and Broks, 1984)
2. Borderline Personality (STB; Claridge and Broks, 1984)
3. Extraversion (E), (E.P.Q.-Eysenck and Eysenck, 1975)
5. Lie (L), (E.P.Q.-Eysenck and Eysenck, 1975)
6. Psychoticism (P), (E.P.Q.-Eysenck and Eysenck, 1975)
7. Social anhedonia (SoA; Chapman et al., 1976)
8. Physical anhedonia (PhA; Chapman et al., 1976)
9. Hypomanic personality (HoP; Eckblad and Chapman, 1986)
10. Perceptual aberration (PAb; Chapman et al., 1980)
11. Magical ideation (Mgl; Eckblad and Chapman, 1983)
12. Delusions of contrition (dC; Foulds and Bedford, 1975)
13. Delusions of persecution (dP; Foulds and Bedford, 1975)
14. Delusions of disintegration (dD; Foulds and Bedford, 1975)
Delusions of grandeur (dG; Foulds and Bedford, 1975)

Predisposition to hallucinations (LSHS; Launay and Slade, 1981)

Schizophrenism (NPS; Nielsen and Petersen, 1976)

Schizoidia (GMS; Golden and Meehl, 1979)

From the Combined Schizotypal Traits Questionnaire, four factors can be extracted (Bentall et al. 1989) which measure traits relating to positive symptomatology (factor 1), social anxiety and cognitive disorganisation (factor 2), negative symptomatology (factor 3) and asocial component of schizotypy (factor 4).

2.5. Procedure

All subjects were tested individually. Testing was carried out either at the Institute of Psychiatry or at the subjects' houses depending on the subjects' preference. At the beginning of the testing session, all subjects were told that the tests constitute part of a research study in which we investigate aspects of thinking in the general population and that testing, which would not exceed 90 minutes in duration, is divided into two parts. In the first part, they would have to do a task in the computer and in the second part, after they take a ten minute break, they would have to complete some questionnaires. Following the above instructions, subjects were asked to sign a consent form (see Appendix 7.3) and then proceeded with the testing.

Part A: Once the subject was settled in front of the computer screen, the following instructions were given:

"In a little while, you will see on the screen two different shapes; one of which will be a triangle and the other one a circle. Each of the two shapes will sometimes appear in the colour red and sometimes in the colour green. One of the two items you will see on the screen, is correct either on the basis of its shape or on the basis of its colour. Your task, will be to discover, by pressing the corresponding response key, which one of the two items is regarded by the computer as being the correct one; each response key corresponds directly to the place of any given item in the screen. Whenever you press a response key and your
choice is correct, you will see on the screen a smiling face. Whenever your choice is wrong, you will see a crying face. Once you have established which one of the two items on the screen is the correct one, the task will automatically shift in such a way so that another item will become the correct one. Again, your task will be to establish which item is the correct one.

Following the above instructions, the first set of stimuli appeared in the screen. During the first part of the computerised task, the subjects were to discover by trial and error the first correct stimulus, which was the colour red. Upon reaching the learning criterion, which was to score correctly on nine consecutive trials, the reversal learning condition was immediately commenced. In this part of the task, subjects were rewarded for choosing the alternative stimulus (colour green) as being the correct stimulus. The task ended once the subject reached the learning criterion; scoring correctly in nine consecutive trials. On completion of the task, two scores are obtained for each subject. The first score (learning) refers to the number of trials taken by the subject to acquire the initial discrimination and establish which one of the stimuli is correct. The second score (reversal learning), refers to the number of trials taken by subject to shift at the outset of reversal.

**Part B:** During the second part of the testing, all subjects were asked to complete a set of three questionnaires: the Raven's Coloured Progressive Matrices (Raven, Court and Raven 1988), the Combined Schizotypal Traits Questionnaire (Bentall et al. 1989) and the Rust Inventory of Schizotypal Cognitions (RISC; Rust 1987).
3. RESULTS
Data were analysed using SPSS-PC (V. 31) software. All significance levels are two-tailed.

3.1 Subjects
In the present study, 30 subjects were tested, 15 males and 15 females. Two subjects (one male and one female) were discounted due to their failure to acquire the initial learning in less than 100 trials. Therefore, only the results obtained by 28 subjects were used for statistical analysis.

Nine subjects were unemployed, two were students and the rest were employed in various occupations. All subjects were native English speakers. The mean age was 34.25 years, ranging from 20 to 55. I.Q. scores were derived by converting the score obtained by each subject on the Standard Progressive Matrices (Raven, Court and Raven 1988) to I.Q.. The mean IQ of the whole group was 104.93, ranging from 97 to 121. Education was calculated by accepting that education up to the age of 16 is 11 years. Any further years of education were added to the basic 11 years. Similarly, in older subjects, who left school before the age of 16, the number of years prior to the age of 16 were subtracted from the basic 11 years of education. The mean education of subjects in years was 13.25. Table 3.1.1 shows the demographic data for all subjects employed in the study.

Table 3.1.1 Means and standard deviations (in parenthesis) of age, IQ, education and sex.

<table>
<thead>
<tr>
<th></th>
<th>Mean (Standard Deviation)</th>
<th>n=28</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>34.25 (10.71)</td>
<td></td>
</tr>
<tr>
<td>IQ</td>
<td>104.93 (6.94)</td>
<td></td>
</tr>
<tr>
<td>Education</td>
<td>13.25 (2.59)</td>
<td></td>
</tr>
<tr>
<td>Sex</td>
<td>14 males and 14 females</td>
<td></td>
</tr>
</tbody>
</table>


3.2 Reversal Learning Scores

On completion of the reversal learning task, two scores were obtained for each subject. The first score (learning), represents the number of trials required by each subject to acquire the initial discrimination; i.e., until he/she establishes that the correct stimulus is any item appearing on the screen in colour red. The second score (reversal learning), represented the number of trials taken by the subject in shifting to the new stimulus at the outset of reversal; i.e., until subjects establish that the correct stimuli this time is any item appearing on the screen in colour green. In addition to the above two scores, a third score was obtained during our statistical analysis, representing the ratio between reversal learning and learning (RL/L); i.e., the number of trials taken by subjects to reverse in relation to the number of trials required to establish the original discrimination.

Table 3.2.1 shows the mean number of trials taken by the whole group in the original learning task, reversal learning and the ratio of trials between learning and reversal learning (RL/L).

### Table 3.2.1

<table>
<thead>
<tr>
<th>TYPE OF SCORE</th>
<th>MEAN NUMBER OF TRIALS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Learning</td>
<td>15.07 (14.43) n=28</td>
</tr>
<tr>
<td>Reversal Learning</td>
<td>09.32 (03.80) n=28</td>
</tr>
<tr>
<td>Reversal Learning/Learning (RL/L)</td>
<td>01.07 (0.83) n=28</td>
</tr>
</tbody>
</table>

Median splits were performed to establish two groups and test whether differences in scores were associated with the variables of age (median=33), education (median=13) and IQ (median=104.5). Because the scores on the reversal learning task were not normally distributed, a non-parametric test (Mann-Whitney) was used in order to test for any differences between the groups in their performance on the reversal learning task. As it can be seen from table 3.2.2, no significant differences were found between the groups. No
significant difference was also found in the performance of males and females on the reversal learning task.

Table 3.2.2 Means and standard deviations (in parenthesis) on learning and reversal learning scores based on median splits on age, IQ., education and sex.

<table>
<thead>
<tr>
<th></th>
<th>AGE OLDER THAN 33 YRS n=14</th>
<th>AGE YOUNGER THAN 33 YRS n=14</th>
<th>SIGN.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Learning</td>
<td>13.21 (12.41)</td>
<td>16.93 (16.47)</td>
<td>ns</td>
</tr>
<tr>
<td>Rev. Learn.</td>
<td>08.64 (3.10)</td>
<td>10.00 (4.40)</td>
<td>ns</td>
</tr>
<tr>
<td></td>
<td>EDUCATION MORE THAN 13 YRS n=14</td>
<td>EDUCATION LESS THAN 13 YRS n=14</td>
<td>/</td>
</tr>
<tr>
<td>Learning</td>
<td>12.36 (13.49)</td>
<td>17.79 (15.32)</td>
<td>ns</td>
</tr>
<tr>
<td>Rev. Learn.</td>
<td>09.64 (04.01)</td>
<td>09.00 (03.70)</td>
<td>ns</td>
</tr>
<tr>
<td></td>
<td>I.Q. HIGHER THAN 104.5 n=14</td>
<td>I.Q. LOWER THAN 104.5 n=14</td>
<td>/</td>
</tr>
<tr>
<td>Learning</td>
<td>11.27 (06.12)</td>
<td>12.10 (5.73)</td>
<td>ns</td>
</tr>
<tr>
<td>Rev. Learn.</td>
<td>09.40 (03.51)</td>
<td>09.25 (4.12)</td>
<td>ns</td>
</tr>
<tr>
<td></td>
<td>MALES (n=14)</td>
<td>FEMALES (n=14)</td>
<td>/</td>
</tr>
<tr>
<td>Learning</td>
<td>13.43 (13.49)</td>
<td>16.71 (15.65)</td>
<td>ns</td>
</tr>
<tr>
<td>Rev. Learn.</td>
<td>09.14 (02.66)</td>
<td>09.50 (04.78)</td>
<td>ns</td>
</tr>
</tbody>
</table>

3.3 Scores on the Combined Schizotypal Traits Questionnaire (CSTQ)

In the present study, the mean scores obtained by all subjects in the 18 subscales included in the CSTQ were similar (within one standard deviation) to those obtained in the study reported by Bentall et al. (1989) - see table 3.3.1.

ns p>0.1; * p<0.1; ** p<0.05; *** p<0.02; **** p<0.01
Table 3.3.1  Means and standard deviations (in parenthesis) of CSTQ and RISC scores as obtained in the present study and as they compared to Bentall et al. (1989)

<table>
<thead>
<tr>
<th>SCALE</th>
<th>Scores Obtained in present study n=28</th>
<th>Scores Obtained in Bentall et al. (1989) n=180</th>
</tr>
</thead>
<tbody>
<tr>
<td>Schiz. Person.</td>
<td>11.71 (06.21)</td>
<td>13.97 (06.56)</td>
</tr>
<tr>
<td>Bord. Person.</td>
<td>04.79 (03.19)</td>
<td>06.61 (03.68)</td>
</tr>
<tr>
<td>E.P.Q. (E)</td>
<td>16.75 (03.03)</td>
<td>12.97 (05.17)</td>
</tr>
<tr>
<td>E.P.Q. (N)</td>
<td>09.86 (04.44)</td>
<td>11.01 (05.51)</td>
</tr>
<tr>
<td>E.P.Q. (L)</td>
<td>07.36 (03.76)</td>
<td>05.21 (03.27)</td>
</tr>
<tr>
<td>E.P.Q. (P)</td>
<td>03.32 (02.18)</td>
<td>04.11 (03.19)</td>
</tr>
<tr>
<td>Soc.Anhedon. (SoA)</td>
<td>10.07 (05.13)</td>
<td>10.28 (05.23)</td>
</tr>
<tr>
<td>Phys. Anhedon. (PhA)</td>
<td>13.14 (06.68)</td>
<td>12.33 (06.91)</td>
</tr>
<tr>
<td>Hypoman. Pers. (HoP)</td>
<td>13.46 (07.53)</td>
<td>15.41 (08.49)</td>
</tr>
<tr>
<td>Percep. Aber. (PAb)</td>
<td>03.36 (03.90)</td>
<td>04.63 (05.07)</td>
</tr>
<tr>
<td>Mag. Ideation (MgI)</td>
<td>05.29 (03.54)</td>
<td>05.88 (04.81)</td>
</tr>
<tr>
<td>Delus. Contr. (dC)</td>
<td>00.32 (00.82)</td>
<td>00.56 (01.09)</td>
</tr>
<tr>
<td>Delus. Persec. (dP)</td>
<td>00.18 (00.39)</td>
<td>00.27 (00.61)</td>
</tr>
<tr>
<td>Delus. Disint. (dD)</td>
<td>00.25 (00.97)</td>
<td>00.43 (00.90)</td>
</tr>
<tr>
<td>Delus. Grand. (dG)</td>
<td>01.14 (01.69)</td>
<td>01.27 (01.60)</td>
</tr>
<tr>
<td>Pred. To Hal. (LSHS)</td>
<td>02.07 (02.29)</td>
<td>03.17 (02.56)</td>
</tr>
<tr>
<td>Schizophrenism (NPS)</td>
<td>03.71 (02.37)</td>
<td>05.95 (03.48)</td>
</tr>
<tr>
<td>Schizoidia (GMS)</td>
<td>01.43 (01.23)</td>
<td>02.07 (01.36)</td>
</tr>
<tr>
<td>(F1)</td>
<td>01.16 (00.80)</td>
<td>Not Available</td>
</tr>
<tr>
<td>(F2)</td>
<td>00.91 (01.81)</td>
<td>&quot; &quot;</td>
</tr>
<tr>
<td>(F3)</td>
<td>-0.79 (01.71)</td>
<td>&quot; &quot;</td>
</tr>
<tr>
<td>(F4)</td>
<td>01.24 (03.12)</td>
<td>&quot; &quot;</td>
</tr>
<tr>
<td>(RISC)</td>
<td>29.86 (08.56)</td>
<td>&quot; &quot;</td>
</tr>
</tbody>
</table>
3.4 Comparison Between High and Low Schizotypy Scores and Performance on Learning and Reversal Learning

Subjects were grouped for each scale to those scoring above the mean obtained by the Bentall et al. (1989) and to those scoring below that mean (criterion split). As means on factors 1-4 were not available, median splits were performed on the scores of all subjects on factor 1 (median=0.97), factor 2 (median=0.75), factor 3 (median=-0.25) and factor 4 (median=0.66). Median split was also performed on their score on the RISC (median=31). Again, subjects were divided into those scoring above the median of each of the factors and to those scoring below the median.

Generally speaking, in most studies reported (e.g., Chapman et al. 1980; Chapman et al. 1982) individuals are regarded as being high schizotypy on the basis of their scores on any of eighteen subscales included in the CSTQ and/or the RISC. For the purposes of the present study, we regarded high positive schizotypy those individuals with high scores on the subscales (STA; HoP; PAb; MgI; dC; dP; dD; dG; LSHS) or factor of the CSTQ (factor 1) or the RISC which measure predisposition towards positive symptomatology. Subjects whose high scores were confined on factors (factor 3) or subscales (SoA; PhA) measuring predisposition to negative symptomatology or predisposition towards any other aspect of the disorder (factor 2; factor 4; STB; EPQ(E); EPQ(N); EPQ (L); EPQ (P), NPS; GMS) were not regarded as being of high positive schizotypy.

The scores obtained in all the schizotypy scales were not normally distributed, so a non parametric statistic (Mann-Whitney) was used to test whether there were possible differences between the performance of subjects with high and low positive schizotypy scores on the number of trials needed both in the acquisition of the initial learning task and during reversal learning. As it can be seen from table 3.4.1, the differences on the number of trials between high and low positive schizotypy, as obtained by employing Factors 1 and the RISC, both in the acquisition of the initial discrimination task and at the reversal learning task, were not significant. No significant difference was also observed in high and low scorers in the CSTQ factors (factors 2, 3, 4) not measuring positive schizotypy.
Table 3.4.1 Means and standard deviations (in parenthesis) on learning and reversal learning based on median splits on Factors 1-4 and RISC.

<table>
<thead>
<tr>
<th></th>
<th>FACTOR 1 score higher than 0.97</th>
<th>FACTOR 1 score lower than 0.97</th>
<th>SIGN.</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n=14</td>
<td>n=14</td>
<td></td>
</tr>
<tr>
<td>Learning</td>
<td>18.50 (17.73)</td>
<td>11.64 (09.64)</td>
<td>ns</td>
</tr>
<tr>
<td>Rev. Learn.</td>
<td>08.64 (03.18)</td>
<td>10.00 (04.35)</td>
<td>ns</td>
</tr>
<tr>
<td></td>
<td>FACTOR 2 score higher than 0.75</td>
<td>FACTOR 2 score lower than 0.75</td>
<td></td>
</tr>
<tr>
<td></td>
<td>n=14</td>
<td>n=14</td>
<td></td>
</tr>
<tr>
<td>Learning</td>
<td>18.57 (17.71)</td>
<td>11.57 (09.62)</td>
<td>ns</td>
</tr>
<tr>
<td>Rev. Learn.</td>
<td>08.71 (03.17)</td>
<td>09.93 (04.38)</td>
<td>ns</td>
</tr>
<tr>
<td></td>
<td>FACTOR 3 score higher than -0.25</td>
<td>FACTOR 3 score lower than -0.25</td>
<td></td>
</tr>
<tr>
<td></td>
<td>n=14</td>
<td>n=14</td>
<td></td>
</tr>
<tr>
<td>Learning</td>
<td>13.07 (12.42)</td>
<td>17.07 (16.42)</td>
<td>ns</td>
</tr>
<tr>
<td>Rev. Learn.</td>
<td>09.93 (04.70)</td>
<td>08.71 (02.67)</td>
<td>ns</td>
</tr>
<tr>
<td></td>
<td>FACTOR 4 score higher than 0.66</td>
<td>FACTOR 4 score lower than 0.66</td>
<td></td>
</tr>
<tr>
<td></td>
<td>n=14</td>
<td>n=14</td>
<td></td>
</tr>
<tr>
<td>Learning</td>
<td>14.93 (17.51)</td>
<td>15.21 (11.22)</td>
<td>ns</td>
</tr>
<tr>
<td>Rev. Learn.</td>
<td>09.43 (04.00)</td>
<td>09.21 (03.75)</td>
<td>ns</td>
</tr>
<tr>
<td></td>
<td>RISC ^1 score higher than 31.00</td>
<td>RISC ^1 score lower than 31.00</td>
<td></td>
</tr>
<tr>
<td></td>
<td>n=14</td>
<td>n=14</td>
<td></td>
</tr>
<tr>
<td>Learning</td>
<td>15.49 (17.32)</td>
<td>14.86 (03.08)</td>
<td>ns</td>
</tr>
<tr>
<td>Rev. Learn</td>
<td>09.64 (03.84)</td>
<td>09.00 (03.88)</td>
<td>ns</td>
</tr>
</tbody>
</table>

^1^ load with F1 (positive symptoms); ^2^ load with F3 (negative symptoms); ^3^ load with F2 and F4 (refer. to cognitive disorganisation and asocial component of schizotypy respectively)

ns p>0.1; * p<0.1; ** p<0.05; *** p<0.02; **** p<0.01.
By dividing subjects as being high and low positive schizotypy on the basis of their scores on all subscales included in the CSTQ measuring predisposition towards positive symptomatology, it appears that there were significant differences between the groups to acquire the initial discrimination (learning) in STA (p<0.01), dP (p<0.05) and dD (p<0.01).

Table 3.4.2  Means and standard deviations (in parenthesis) on learning and reversal learning based on a criterion split (Bentall et al. 1989).

<table>
<thead>
<tr>
<th></th>
<th>STA(^1) (higher 13.97) n=8</th>
<th>STA(^1) (lower 13.97) n=20</th>
<th>SIGN.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Learning</td>
<td>29.00 (20.94)</td>
<td>09.50 (04.49)</td>
<td>****</td>
</tr>
<tr>
<td>Rev. Learn.</td>
<td>09.13 (03.87)</td>
<td>09.40 (03.87)</td>
<td>ns</td>
</tr>
<tr>
<td></td>
<td>STB(^3) (higher 6.61) n=10</td>
<td>STB(^3) (lower 6.61) n=18</td>
<td></td>
</tr>
<tr>
<td>Learning</td>
<td>23.60 (21.49)</td>
<td>10.33 (04.46)</td>
<td>ns</td>
</tr>
<tr>
<td>Rev. Learn.</td>
<td>09.00 (02.63)</td>
<td>09.50 (04.38)</td>
<td>ns</td>
</tr>
<tr>
<td></td>
<td>EPQ(^3)(E)- higher 12.97 n=24</td>
<td>EPQ(^3)(E)-lower 12.97 n=4</td>
<td></td>
</tr>
<tr>
<td>Learning</td>
<td>15.50 (15.47)</td>
<td>12.50 (05.45)</td>
<td>ns</td>
</tr>
<tr>
<td>Rev. Learn.</td>
<td>09.33 (03.77)</td>
<td>09.25 (04.57)</td>
<td>ns</td>
</tr>
<tr>
<td></td>
<td>EPQ(^3)(N)-higher 11.01 n=9</td>
<td>EPQ(^3)(N)-lower 11.01 n=19</td>
<td></td>
</tr>
<tr>
<td>Learning</td>
<td>27.89 (19.83)</td>
<td>09.00 (04.07)</td>
<td>****</td>
</tr>
<tr>
<td>Rev. Learn.</td>
<td>11.22 (05.02)</td>
<td>08.42 (02.80)</td>
<td>ns</td>
</tr>
<tr>
<td></td>
<td>EPQ(^3)(L)-higher 5.21 n=18</td>
<td>EPQ(^3)(L)-lower 5.21 n=10</td>
<td></td>
</tr>
<tr>
<td>Learning</td>
<td>18.78 (16.87)</td>
<td>08.40 (03.24)</td>
<td>*</td>
</tr>
<tr>
<td>Rev. Learn.</td>
<td>10.06 (04.11)</td>
<td>08.00 (02.91)</td>
<td>ns</td>
</tr>
<tr>
<td></td>
<td>EPQ(^3)(P)-higher 4.11 n=4</td>
<td>EPQ(^3)(P)-lower 4.11 n=24</td>
<td></td>
</tr>
<tr>
<td>Learning</td>
<td>20.25 (24.76)</td>
<td>14.21 (12.62)</td>
<td>ns</td>
</tr>
<tr>
<td>Rev. Learn.</td>
<td>10.00 (02.83)</td>
<td>09.21 (03.98)</td>
<td>ns</td>
</tr>
</tbody>
</table>

\(^1\) load with F1 (positive symptoms); \(^2\) load with F3 (negative symptoms); \(^3\) load with F2 and F4 (refer. to cognitive disorganisation and asocial component of schizotypy respectively)

ns p>0.1; * p<0.1; ** p<0.05; *** p<0.02; **** p<0.01.
Table 3.4.3  Means and standard deviations (in parenthesis) on learning and reversal learning based on a criterion split (Bentall et al. 1989) on SoA, PhA

<table>
<thead>
<tr>
<th></th>
<th>SoA² (higher 10.28) n=12</th>
<th>SoA² (lower 10.28) n=16</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Learning</td>
<td>13.42 (14.63)</td>
<td>16.31 (14.64)</td>
<td>ns</td>
</tr>
<tr>
<td>Rev. Learn.</td>
<td>08.58 (02.50)</td>
<td>09.88 (04.54)</td>
<td>ns</td>
</tr>
<tr>
<td></td>
<td>PhA² (higher 12.33) n=14</td>
<td>PhA² (lower 12.33) n=14</td>
<td></td>
</tr>
<tr>
<td>Learning</td>
<td>09.86 (05.22)</td>
<td>20.29 (18.62)</td>
<td>ns</td>
</tr>
<tr>
<td>Rev. Learn.</td>
<td>09.36 (02.98)</td>
<td>09.29 (04.60)</td>
<td>ns</td>
</tr>
</tbody>
</table>

On the reversal learning task, no significant differences were found between the number of trials taken to shift at the outset of reversal in any of the scales apart from the scale assessing dP (p<0.05) - see tables 3.4.2, 3.4.3, 3.4.4 and 3.4.5.

With respect to the scores on the scales which do not measure predisposition towards positive symptomatology, significant differences were found in the number of trials required to acquire the initial discrimination task (learning) between high and low scorers in the subscales EPQ (N) (p<0.01) EPQ (L) (p<0.1) and NPS (p<0.01). On the reversal learning task, no significant differences were found between high and low scorers on any of these subscales on the number of trials required to reverse (see tables 3.4.2, 3.4.3, 3.4.4 and 3.4.5).

1 load with F1 (positive symptoms); 2 load with F3 (negative symptoms); 3 load with F2 and F4 (refer. to cognitive disorganisation and asocial component of schizotypy respectively)

ns p>0.1; * p<0.1; ** p<0.05; *** p<0.02; **** p<0.01.
### Table 3.4.4

Means and standard deviations (in parenthesis) on learning and reversal learning based on a criterion split Bentall et al. (1989) on HoP, PAb, Mgl, GMS, dC, dP, dD, dG.

<table>
<thead>
<tr>
<th></th>
<th>HoP¹ (higher 15.41) n=16</th>
<th>HoP¹ (lower 15.41) n=12</th>
<th>SIGN</th>
</tr>
</thead>
<tbody>
<tr>
<td>Learning</td>
<td>16.06 (15.91)</td>
<td>13.75 (12.76)</td>
<td>ns</td>
</tr>
<tr>
<td>Rev. Learn.</td>
<td>08.69 (02.68)</td>
<td>10.17 (04.93)</td>
<td>ns</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PAb¹ (higher 4.63) n=4</td>
<td>PAb¹ (lower 4.63) n=24</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Learning</td>
<td>24.00 (22.55)</td>
<td>13.58 (12.73)</td>
<td>ns</td>
</tr>
<tr>
<td>Rev. Learn.</td>
<td>11.75 (03.30)</td>
<td>06.92 (03.79)</td>
<td>ns</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mgl¹ (higher 5.88) n=9</td>
<td>Mgl¹ (lower 5.88) n=19</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Learning</td>
<td>21.33 (19.59)</td>
<td>12.11 (10.63)</td>
<td>ns</td>
</tr>
<tr>
<td>Rev. Learn.</td>
<td>09.44 (03.75)</td>
<td>09.26 (03.93)</td>
<td>ns</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>dC¹ (higher than 0.56) n=4</td>
<td>dC¹ (lower 0.56) n=24</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Learning</td>
<td>21.50 (24.23)</td>
<td>14.00 (02.58)</td>
<td>ns</td>
</tr>
<tr>
<td>Rev. Learn.</td>
<td>10.00 (03.56)</td>
<td>09.21 (03.90)</td>
<td>ns</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>dP¹ (higher 0.27) n=5</td>
<td>dP¹ (lower 0.27) n=23</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Learning</td>
<td>32.00 (24.77)</td>
<td>11.39 (08.05)</td>
<td>**</td>
</tr>
<tr>
<td>Rev. Learn.</td>
<td>08.20 (03.27)</td>
<td>13.57 (03.93)</td>
<td>**</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>dD¹ (higher 0.43) n=3</td>
<td>dD¹ (lower 0.43) n=25</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Learning</td>
<td>42.33 (22.03)</td>
<td>11.80 (09.48)</td>
<td>****</td>
</tr>
<tr>
<td>Rev. Learn.</td>
<td>11.00 (03.61)</td>
<td>09.12 (03.84)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>dG¹ (higher 1.27) n=12</td>
<td>dG¹ (lower 1.27) n=16</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Learning</td>
<td>21.00 (21.63)</td>
<td>12.70 (10.14)</td>
<td>ns</td>
</tr>
<tr>
<td>Rev. Learn.</td>
<td>08.75 (02.71)</td>
<td>09.55 (04.20)</td>
<td>ns</td>
</tr>
</tbody>
</table>

¹ load with F1 (positive symptoms); ² load with F3 (negative symptoms); ³ load with F2 and F4 (refer to cognitive disorganisation and asocial component of schizotypy respectively)

ns p>0.1; * p<0.1; ** p<0.05; *** p<0.02; **** p<0.01.

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Table 3.4.5  Means and standard deviations (in parenthesis) on learning and reversal learning based on a criterion split Bentall et al. (1989) on LSHS, NPS, GMS.

<table>
<thead>
<tr>
<th></th>
<th>LSHS (^1) (higher 3.17) n=14</th>
<th>LSHS (^1) (lower 3.17) n=14</th>
<th>SIGN (^1)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Learning</td>
<td>23.33 (24.63)</td>
<td>12.82 (09.93)</td>
<td>ns</td>
</tr>
<tr>
<td>Rev. Learn.</td>
<td>08.00 (02.97)</td>
<td>09.68 (03.98)</td>
<td>ns</td>
</tr>
<tr>
<td></td>
<td>NPS (^3) (higher 5.95) n=14</td>
<td>NPS (^3) (lower 5.95) n=14</td>
<td></td>
</tr>
<tr>
<td>Learning</td>
<td>32.20 (20.87)</td>
<td>11.35 (09.76)</td>
<td>****</td>
</tr>
<tr>
<td>Rev. Learn</td>
<td>09.20 (03.56)</td>
<td>09.35 (03.93)</td>
<td>ns</td>
</tr>
<tr>
<td></td>
<td>GMS (^3) (higher 2.07) n=14</td>
<td>GMS (^3) (lower 2.07) n=14</td>
<td></td>
</tr>
<tr>
<td>Learning</td>
<td>16.43 (18.63)</td>
<td>14.62 (13.27)</td>
<td>ns</td>
</tr>
<tr>
<td>Rev. Learn</td>
<td>10.43 (05.47)</td>
<td>08.95 (03.15)</td>
<td>ns</td>
</tr>
</tbody>
</table>

3.5. Comparison Between High and Low Schizotypy Scores and Performance on Ratio RL/L

Statistical analysis of the ratio between the number of trials taken to acquire initial discrimination and the number of trials taken to shift at the outset of reversal (RL/L) was carried out in the Bentall Slade factor 1 and the RISC (see tables 3.5.1, 3.5.2, 3.5.3). Since some of the schizotypy scales were not normally distributed a non-parametric test (Mann-Whitney) was also used in this case in order to test possible differences in the ratio RL/L between high and low schizotypy groups.

\(^1\) load with F1 (positive symptoms); \(^2\) load with F3 (negative symptoms); \(^3\) load with F2 and F4 (refer. to cognitive disorganisation and asocial component of schizotypy respectively)

ns p>0.1; * p<0.1; ** p<0.05; *** p<0.02; **** p<0.01.
Table 3.5.1  Means and standard deviations (in parenthesis) on the ratio of reversal learning/learning scores based on median splits on Factors 1- 4 and RISC.

<table>
<thead>
<tr>
<th></th>
<th>FACTOR 1</th>
<th>FACTOR 1</th>
<th>SIGN.¹</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>score higher than 0.97</td>
<td>score lower than 0.97</td>
<td></td>
</tr>
<tr>
<td></td>
<td>n=14</td>
<td>n=14</td>
<td></td>
</tr>
<tr>
<td>RL/L</td>
<td>0.087 (0.69)</td>
<td>0.028 (0.92)</td>
<td>ns</td>
</tr>
<tr>
<td></td>
<td>FACTOR 2</td>
<td>FACTOR 2</td>
<td></td>
</tr>
<tr>
<td></td>
<td>score higher than 0.75</td>
<td>score lower than 0.75</td>
<td></td>
</tr>
<tr>
<td></td>
<td>n=14</td>
<td>n=14</td>
<td></td>
</tr>
<tr>
<td>RL/L</td>
<td>0.090 (0.74)</td>
<td>0.025 (0.89)</td>
<td>ns</td>
</tr>
<tr>
<td></td>
<td>FACTOR 3</td>
<td>FACTOR 3</td>
<td></td>
</tr>
<tr>
<td></td>
<td>score higher than -0.25</td>
<td>score lower than -0.25</td>
<td></td>
</tr>
<tr>
<td></td>
<td>n=14</td>
<td>n=14</td>
<td></td>
</tr>
<tr>
<td>R/L</td>
<td>0.021 (0.94)</td>
<td>0.094 (0.69)</td>
<td>ns</td>
</tr>
<tr>
<td></td>
<td>FACTOR 4</td>
<td>FACTOR 4</td>
<td></td>
</tr>
<tr>
<td></td>
<td>score higher than 0.66</td>
<td>score lower than 0.66</td>
<td></td>
</tr>
<tr>
<td></td>
<td>n=14</td>
<td>n=14</td>
<td></td>
</tr>
<tr>
<td>RL/L</td>
<td>0.020 (0.86)</td>
<td>0.095 (0.80)</td>
<td>ns</td>
</tr>
<tr>
<td></td>
<td>RISC¹</td>
<td>RISC¹</td>
<td></td>
</tr>
<tr>
<td></td>
<td>score higher than 31.00</td>
<td>score lower than 31.00</td>
<td></td>
</tr>
<tr>
<td></td>
<td>n=14</td>
<td>n=14</td>
<td></td>
</tr>
<tr>
<td>RL/L</td>
<td>0.012 (0.73)</td>
<td>0.010 (0.94)</td>
<td>ns</td>
</tr>
</tbody>
</table>

Differences in RL/L between high and low positive schizotypy, as obtained by factor 1 and the RISC failed to reach significant levels. Insignificant differences were also found in RL/L between high and low scorers on factors 2, 3 and 4. Despite these insignificant differences, a trend is observed indicating that subjects scoring high on factors 1 (positive symptomatology) and 2 (social anxiety and cognitive disorganisation) of the CSTQ tend to take fewer trials to shift at the outset of reversal in relation to the number of trials taken during their initial

¹ load with F1 (positive symptoms); ² load with F3 (negative symptoms); ³ load with F2 and F4 (refer. to cognitive disorganisation and asocial component of schizotypy respectively)

ns p>0.1; * p<0.1; ** p<0.05; *** p<0.02; **** p<0.01.
Learning, (i.e., scores below 1 represent faster reversal learning than initial learning, and scores over 1 represent faster initial learning than reversal learning).

Table 3.5.2. Means and standard deviations (in parenthesis) on the ratio of reversal learning/learning based on criterion split (Bentall et al. 1989) on STA, STB, EPQ (E), EPQ(N), EPQ (L), EPQ (P), SoA, and PhA.

<table>
<thead>
<tr>
<th></th>
<th>STA(^1) (higher 13.97) n=8</th>
<th>STA(^1) (lower 13.97) n=20</th>
<th>SIGN.(^1)</th>
</tr>
</thead>
<tbody>
<tr>
<td>RL/L</td>
<td>00.67 (00.85)</td>
<td>01.24 (00.78)</td>
<td>****</td>
</tr>
<tr>
<td>STA(^2) (higher 6.61) n=10</td>
<td>STB(^2) (lower 6.61) n=18</td>
<td>00.86 (00.63)</td>
<td>ns</td>
</tr>
<tr>
<td>RL/L</td>
<td>01.20 (00.91)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>EPQ(^3)(E)-higher 12.97 n=24</td>
<td>EPQ(^3)(E)-lower 12.97 n=4</td>
<td>1.11 (00.87)</td>
<td>ns</td>
</tr>
<tr>
<td>RL/L</td>
<td>00.85 (00.43)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>EPQ(^3)(N)-higher 11.01 n=9</td>
<td>EPQ(^3)(N)-lower 11.01 n=19</td>
<td>00.96 (01.24)</td>
<td>ns</td>
</tr>
<tr>
<td>RL/L</td>
<td>01.35 (00.57)</td>
<td></td>
<td>**</td>
</tr>
<tr>
<td>EPQ(^3)(L)-higher 5.21 n=18</td>
<td>EPQ(^3)(L)-lower 5.21 n=10</td>
<td>01.05 (00.92)</td>
<td>ns</td>
</tr>
<tr>
<td>RL/L</td>
<td>01.11 (00.66)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>EPQ(^3)(P)-higher 4.11 n=4</td>
<td>EPQ(^3)(P)-lower 4.11 n=24</td>
<td>00.99 (00.60)</td>
<td>ns</td>
</tr>
<tr>
<td>RL/L</td>
<td>01.09 (00.87)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>SoA(^2) (higher 10.28) n=12</td>
<td>SoA(^2) (lower 10.28) n=16</td>
<td>01.01 (00.53)</td>
<td>ns</td>
</tr>
<tr>
<td>RL/L</td>
<td>01.12 (01.01)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>PhA(^2) (higher 12.33) n=14</td>
<td>PhA(^2) (lower 12.33) n=14</td>
<td>01.22 (00.63)</td>
<td>ns</td>
</tr>
<tr>
<td>RL/L</td>
<td>00.93 (00.99)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Statistical analysis carried out on RL/L between high and low positive schizotypy based on the scales measuring predisposition towards positive symptomatology included in the CSTQ, showed significant differences between high and low positive schizotypy in the following subscales STA (p<0.01), dP (p<0.02) and dD (p<0.05). Significant differences were also

\(^1\) load with F1 (positive symptoms); \(^2\) load with F3 (negative symptoms); \(^3\) load with F2 and F4 (refer. to cognitive disorganisation and asocial component of schizotypy respectively)

ns p>0.1; * p<0.1; ** p<0.05; *** p<0.02; **** p<0.01.
found when statistical analysis was carried out between high and low scorers on the subscales included in the CSTQ which do not measure predisposition towards positive symptomatology. Significant differences were found on the basis of their scores on the subscales EPQ (N) (p<0.05) and NP (p<0.01).

Table 3.5.3 Means and standard deviations (in parenthesis) on the ratio of reversal learning/learning based on criterion split (Bentall et al. 1989).

<table>
<thead>
<tr>
<th></th>
<th>HoP¹ (higher 15.41) n=16</th>
<th>HoP¹ (lower 15.41) n=12</th>
<th>SIGN.¹</th>
</tr>
</thead>
<tbody>
<tr>
<td>RL/L</td>
<td>00.91 (00.62)</td>
<td>01.29 (01.03)</td>
<td>ns</td>
</tr>
<tr>
<td>PAb¹</td>
<td>(higher 4.63) n=4</td>
<td>(lower 4.63) n=24</td>
<td></td>
</tr>
<tr>
<td>RL/L</td>
<td>00.93 (00.95)</td>
<td>01.10 (00.82)</td>
<td>ns</td>
</tr>
<tr>
<td>Mgi¹</td>
<td>(higher 5.88) n=9</td>
<td>(lower 5.88) n=19</td>
<td></td>
</tr>
<tr>
<td>RL/L</td>
<td>00.81 (00.77)</td>
<td>01.20 (00.84)</td>
<td>ns</td>
</tr>
<tr>
<td>DC¹</td>
<td>(higher 0.56) n=4</td>
<td>(lower 0.56) n=24</td>
<td></td>
</tr>
<tr>
<td>RL/L</td>
<td>00.82 (00.44)</td>
<td>01.12 (00.87)</td>
<td>ns</td>
</tr>
<tr>
<td>DP¹</td>
<td>(higher 0.27) n=5</td>
<td>(lower 0.27) n=23</td>
<td>***</td>
</tr>
<tr>
<td>RL/L</td>
<td>00.57 (00.54)</td>
<td>01.18 (00.84)</td>
<td></td>
</tr>
<tr>
<td>DD¹</td>
<td>(higher 0.43) n=3</td>
<td>(lower 0.43) n=25</td>
<td>**</td>
</tr>
<tr>
<td>RL/L</td>
<td>00.36 (00.30)</td>
<td>01.16 (00.83)</td>
<td></td>
</tr>
<tr>
<td>DG¹</td>
<td>(higher 1.27) n=12</td>
<td>(lower 1.27) n=16</td>
<td></td>
</tr>
<tr>
<td>RL/L</td>
<td>00.84 (00.55)</td>
<td>01.17 (00.91)</td>
<td>ns</td>
</tr>
<tr>
<td>LSHS¹</td>
<td>(higher 3.17) n=14</td>
<td>(lower 3.17) n=14</td>
<td></td>
</tr>
<tr>
<td>RL/L</td>
<td>00.70 (00.45)</td>
<td>01.18 (00.88)</td>
<td>ns</td>
</tr>
<tr>
<td>NPS³</td>
<td>(higher 5.95) n=14</td>
<td>(lower 5.95) n=14</td>
<td></td>
</tr>
<tr>
<td>RL/L</td>
<td>00.37 (00.22)</td>
<td>01.23 (00.83)</td>
<td>****</td>
</tr>
<tr>
<td>GMS³</td>
<td>(higher 2.07) n=14</td>
<td>(lower 2.07) n=14</td>
<td></td>
</tr>
<tr>
<td>RL/L</td>
<td>01.24 (01.15)</td>
<td>01.02 (00.71)</td>
<td>Ns</td>
</tr>
</tbody>
</table>

¹¹ load with F1 (positive symptoms); ²² load with F3 (negative symptoms); ³³ load with F2 and F4 (refer, to cognitive disorganisation and asocial component of schizotypy respectively)

ns p>0.1; * p<0.1; ** p<0.05; *** p<0.02; **** p<0.01

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4. DISCUSSION

According to Gray's 1991 model, a dysfunction in the interaction between the hippocampus and the nucleus accumbens, causes disruption of the information processing in acute schizophrenia resulting in the formation of positive symptoms. Elaborating from this model, it could be suggested that due to this disruption in the information processing, acute schizophrenics would fail to learn from previous experiences and that they would continue to approach any stimulus as though it is novel (see section 1.4). This model appears to accord well with Hemsley's (1987) proposition, that basic to schizophrenia "is the weakening of the influences of stored memories of regularities of previous input on current perception".

Attempts to provide support for the above by employing behavioural phenomena that depend on the control of attention by prior experience (latent inhibition, Kamin blocking effect, partial reinforcement extinction effect and reversal learning) has produced rather conflicting results. In most studies reported, the results obtained are rather difficult to interpret or to draw any conclusions from them as they are often confounded with other variables that are present in studies employing schizophrenics. Variables such as the heterogeneity of symptoms, medication or institutionalisation (e.g., Hemsley 1988), seem to highlight the difficulties of current schizophrenia research pointing to a need of a more homogenous group of subjects that are unaffected by extraneous factors.

During the past two decades there have been increasing attempts to provide evidence supporting an earlier view that a continuum may exist between psychosis and normality. These attempts have been mostly focussed in devising questionnaire scales that would be capable of identifying and measuring schizophrenia like traits in individuals who are regarded as being psychiatrically healthy. Currently, there is a large body of evidence indicating that individuals scoring highly on these scales, show similarities with psychotic patients both in the phenomenology as well as in their psychological functioning and responding (e.g., Balugh and Merit 1985; Merit et al. 1986). Such findings appear rather promising for the future of schizophrenia research as it might provide us with the opportunity of studying the underlying
deficit of the disorder in non-schizophrenic individuals and thus any results produced will be unaffected from the confounding variables mentioned above.

Drawing on the findings produced by current schizotypy research, the present study sought to examine, by solely employing normal controls, the Gray's et al. (1991) and Hemsley's (1987) models which suggest that schizophrenics manifesting positive symptomatology, fail to make use of earlier experiences and as a consequence any stimulus is approached as though it is presented to them for the first time. By employing the behavioural phenomenon of reversal learning, the following hypotheses were addressed:

1. that individuals with a high scores in the positive symptomatology factor of the CSTQ (factor 1) or in the scales included in the CSTQ measuring predisposition towards positive symptoms (STA; HoP; PA; Mg; dC; dP; dD; dG; LSHS) or on the RISC, are expected to reverse faster on a reversal learning task than subjects who generally score low on the CSTQ positive factor and/or on the subscales measuring predisposition to positive symptomatology and on the RISC.

2. that there will be no difference in the performance of the reversal learning task between those subjects whose scores are either high or low in the factors (factor 2, factor 3 and factor 4) and in the scales included in the CSTQ that do not measure positive symptomatology (SoA; PhA STB; EPQ[E]; EPQ[N]; EPQ [L]; EPQ[P], NPS; GMS).

In the following discussion, a brief summary of the general results will be given and prior to a fuller discussion of the implications of the data, each hypothesis will be addressed.

The first set of results obtained in the present study indicated that variables such as age, I.Q., education and sex, did not yield any significant differences in the performance of neither the original discrimination task nor in the reversal learning task (p>0.1).

With respect to hypothesis one, subjects scoring high on the positive symptomatology factor (factor 1), the RISC and the subscales included in the CSTQ measuring predisposition
towards positive symptomatology did not show significant differences in the number of trials required to shift at the outset of reversal (p>0.1). In the only scale where differences in the number of trials taken in the reversal learning task reached significance level (p<0.05) was in the dP; partly supporting our prediction that positive symptomatology is associated with faster reversal.

With respect to our second hypothesis a more clear picture has emerged. No significant differences were found in any of the factors (2, 3, 4) or on the sub-scales of the CSTQ not measuring predisposition towards positive symptomatology and thus our hypothesis that no differences in the performance of the reversal learning task between high and low scorers in these scales and factors was supported. But although these latter findings seem to support our hypothesis, on the light of the earlier finding that of the ten measures of positive schizotypy analysed, only one showed significant differences between high and low positive schizotypy in the performance of the reversal learning task, makes it rather difficult to draw any conclusions.

In order to investigate whether these findings could be better clarified, the number of trials taken to learn the initial discrimination by high and low positive schizotypy subjects was examined. The results obtained showed no significant differences in learning between subjects scoring high on the CSTQ factor 1 and the RISC and those scoring low. Similarly, no significant differences were found in learning when high and low scorers on the other factors (2, 3, 4) were compared. By looking at the subscales constituting the CSTQ a different picture appeared to emerge. In terms of high and low positive schizotypy scales, it was shown that in three of those subscales there were significant differences in the number of trials taken between high and low positive schizotypy scorers (STA p<0.01; dP p<0.05; dD p<0.01), indicating that high positive schizotypy subjects on these scales required a greater number of trials to acquire the initial discrimination. Although failed to reach significance, the same trend (i.e., high positive schizotypy subjects taking longer than low positive schizotypy subjects to acquire initial learning) was also observed in all of the scales measuring predisposition towards positive symptoms. A similar trend was also observed in Factors 1 (positive symptomatology) and 2 (measuring social anxiety and cognitive
disorganisation; an aspect of schizophrenia often regarded by researchers as belonging to the positive symptoms category) of the CSTQ and the RISC.

Looking at the subscales not measuring positive schizotypy (STB; EPQ (E); EPQ (N); EPQ (L); EPQ (P); SoA; PhA; NPS and GMS), a rather mixed picture was observed. In some subscales significant differences (EPQ (N) (p<0.01); EPQ (L) (p<0.1) and NPS (p<0.01)) or a trend (STB; EPQ (P); EPQ (E); GMS) was observed indicating that high scorers on these scales require greater number of trials to acquire learning than low scorers whereas in other scales the opposite trend was observed (SoA; PhA). With respect to factors 3 (negative symptomatology) and 4 (measuring an asocial component schizotypy) of the CSTQ a trend was found indicating that subjects scoring high on those factors required less trials to acquire the initial discrimination task than those scoring low.

A closer examination of our results, and prior to statistical analysis, seemed to suggest that although in general high positive schizotypy individuals tended to reverse slower than low positive schizotypy subjects, high positive schizotypy subjects also needed more trials than subjects in the low positive schizotypy group to acquire the initial discrimination. This observation appears to accord well with a large body of evidence pointing to the difficulties of schizophrenics in acquiring new knowledge (e.g. Hemsley 1992). In the light of this observation, one could suggest that high positive schizotypy individuals, as predicted by our hypothesis and thus supporting the Gray's et al (1991) and Hemsley's (1987) models, could reverse faster than low positive schizotypy, when the trials needed to shift at the outset of reversal, is not taken as an absolute number of trials but rather the comparison is made on the basis of the ratio between learning and reversal learning (RL/L) of each group.

Statistical analysis using the non-parametric test Mann Whitney, revealed significant differences in the RL/L ratio between high and low positive schizotypy groups when the division was made on the basis of their scores in the following subscales: STA (p<0.01), dP (p<0.02) and dD (p<0.05). In those positive schizotypy scales where the number of trials required between high and low positive schizotypy individuals did not reach levels of
significance (HoP, PAb, Mgl, dC, dG, LSHS), there was a trend indicating that high positive schizotypy individuals need fewer trials than the original learning to reverse.

Although significant differences were also observed in subscales not measuring positive schizotypy (EPQ (N) (p<0.05) and NPS (p<0.01) again, not a clear trend was observed in these latter group of scales. This, rather inconsistent trend obtained on this group of subscales, might be due to the fact that a number of these scales measure aspects of schizotypy that, although not clearly positive (SoA and PhA being the only scales that solely measure predisposition towards negative symptomatology), they do contain items which correlate with positive schizotypy scales (e.g., EPQ-N) or tapping some positive schizotypal traits (e.g., EPQ (P); NPS) too.

The behavioural phenomenon employed in the present study, appears to bear some intriguing similarities to the probabilistic inference learning task employed by Garety et al (1991). In that study, it was reported that schizophrenics manifesting positive symptomatology (delusions) were more responsive to new information (disconfirming an existing hypothesis) than a normal control group. Elaborating from this, one could suggest that in the present study there is some evidence for a similar pattern of response. Subjects scoring high in positive schizotypy on three scales (and with trends on certain other scales), are less influenced by their previous learning and associations and are making inferences, as to which stimulus is the correct at the outset of reversal, faster than individuals scoring low on these positive schizotypy scales.

In the light of these latter findings, one could propose that there is some evidence (from the three subscales STA, dP, dD) and a trend (HoP, PAb, Mgl, dC, dG, LSHS) that high positive schizotypy individuals do tend to reverse faster than low positive schizotypy individuals when performance on the reversal learning task is not taken as an absolute value but rather as the ratio of the number of trials taken to acquire initial discrimination. Moreover, significant differences on three subscales and a trend in all other subscales suggest that high positive schizotypy individuals take longer than low positive schizotypy individuals to acquire the
initial discrimination, but once learning has occurred reversal can be achieved by taking less trials than those taking for the initial learning.

Drawing on experimental evidence produced by current schizotypy research, indicating that the performance of high schizotypy individuals on learning tasks resemble to those of schizophrenics, and more specifically to those manifesting positive symptomatology (e.g. Baruch et al. 1988b and Jones et al. 1992b), the present findings seem to accord well with Gray's (1991) model and Hemsley's (1987) proposition that basic to schizophrenia "is the weakening of the influences of stored memories of regularities of previous input on current perception". Unlike the subjects in the high positive schizotypy group, low positive schizotypy individuals generally tended to need a similar or more trials to reverse than the number of trials taken at the initial discrimination. According to Mackintosh's (1983) model, as the excitatory strength of S+ (presentation of which signals reward) and in the inhibitory strength S- (signalling the absence of reward) increases, greater persistence is observed in selecting the former stimulus at the outset of reversal. This is what appears to happens in low positive schizotypy individuals (continuing to select former stimulus at the outset of reversal) whereas, possibly due to a disruption in the information processing of schizophrenics (Gray et al. 1991) this expected pattern is less obvious in individuals with high scores in positive schizotypy scales (less influenced by previous associations, they shift faster at the outset of reversal). On the basis of the above, one could assert that what is interesting in the present study, is that by employing a sample drawn from the general population with no psychiatric history and solely on the basis of questionnaire measures of a predisposition to positive symptomatology, the results obtained appear consistent with models (Gray et al. 1991 and Hemsley 1987) intending to account for the aetiology of the observed positive symptomatology in schizophrenia. These findings become even more encouraging when they are considered in the context of current schizophrenia research. A major obstacle in producing evidence relevant to any theoretical framework in current schizophrenia research, is the influence of confounding variables such as medication, institutionalisation and symptomatology. As a result of this, most studies reported, instead of increasing our understanding of the disorder, emphasise the shortcomings of the methodology employed pointing to the need for a more homogenous group of subjects. Research studies employing
normal, high schizotypy individuals, might indeed provide us with one route to the solution of the methodological problem that has plagued research in the area of schizophrenia for more than a century.

Despite the fact that our results seem to provide some support for our hypotheses the present study is not without its limitations. Although trends indicating faster RL/L were observed in most positive schizotypy scales (the RISC being the only scale where such a trend was not observed), one also needs to point out that differences in the RL/L reached significance in only three scales (STA; dP; dD). Additionally, the small size of our sample, the even smaller number of our high positive schizotypy group (ranging from n=3 to n=16 as opposed to the size of low positive schizotypy group which ranged from n=9 to n=25) and the large standard deviations obtained, weakened the statistical power of the study.

On the basis of the above, one could suggest that before any conclusions are drawn on the basis of the general trend obtained, a replication of the study by employing a larger sample is necessary, as it might provide us with more encouraging results.
5. SUGGESTIONS FOR FURTHER RESEARCH

A possible way of improving on this research, is by attempting to replicate the current findings in subsequent research by employing a subjects who are more likely than the average population to have high positive schizotypal traits.

Research into the possible role played by genetic factors in the aetiology of schizophrenia, rests on the assumption that if the disorder follows the laws of heredity, then, genetic overlap between individuals will increase the degree of their similarities. Familial studies, seem to confirm that schizophrenia run in families and that the morbidity risk of relatives of schizophrenics is higher than it is in relatives of normal controls (eg. Tsuang, et al. 1980). Moreover, there now exist a convincing body of literature indicating that as individuals are becoming more genetically related to the index case, the greater the probability of them becoming schizophrenics. In a review, compiled by the most important investigations in the area, carried out by Zerbin Rudin (1972), it was estimated that the morbidity risk of children of two affected parents is around 40-48%, for parents of a schizophrenic around 9-16% and for siblings 8-14%. In the same study, it was also indicated that as the genetic relation becomes more distant the morbidity rate decreases. Evidence produced by this type of research also seems to support earlier findings in that genetic influences play an important role in the aetiology of the disorder (Ketty, et al. 1968, 1975; Heston 1966; Kendler, et al. 1981).

Based on the evidence presented above, it appears that first degree relatives are more likely than the general population to be predisposed towards psychosis. Elaborating from this, one could infer that it is also more likely that the first degree relatives of schizophrenics to be higher in schizotypy than the average population. On the basis of this, we suggest that one possible way of investigating the validity of our present findings might be by attempting to replicate the present study by employing a larger group of normal controls as well as by including a group consisting of first degree relatives of schizophrenics.
6. REFERENCES:


Hemsley D.R. (1991): What have cognitive deficits to do with schizophrenia?. In G. Huber (Eds): Idiopathische psuchosen. Shattarer Stutte


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APPENDICES
APPENDIX 7.1
THE COMBINED SCHIZOTYPAL TRAIT QUESTIONNAIRE

The following questionnaire is being used as part of a study being carried out by the Universities of Liverpool, Oxford and London. The questionnaire is designed to measure personality variables related to peculiarly vivid mental experiences. We believe that these kind of vivid mental experiences are much more common than has sometimes been supposed and that most people have had some such experiences during their lives. We believe that these experiences are important because they may be related to such characteristics as imagination, creativity and sensitivity to stressful events.

The following pages consist of 420 questions in two parts. The first part consists of questions which can be answered 'yes' or 'no'. The second part consists of statements which you can answer 'True' or 'False'. In each case, record your answer by ticking the appropriate part of the two page score sheet provided. The questionnaire is quite long so we would advise you not to spend too much time deliberating over each question or statement.

When completing the questionnaire please bear the following points in mind:

1) Please answer honestly. There are no right or wrong answers, and we expect there will be great variation in the way that people respond to the individual items.

2) Because of this it is important that you do not discuss your answers with anyone else you know who is completing the questionnaire, as this may affect their answers.

3) Your answers will be treated in the strictest confidence and will be revealed to no one. (We are interested in the overall results for particular groups of people, rather than individual scores).

Thank you for your help. With your co-operation our study should be a success.
THE COMBINED SCHIZOTYPAL TRAIT QUESTIONNAIRE (CSTQ)

**Part 1**

<table>
<thead>
<tr>
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<th>Name</th>
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PART ONE

All questions are answered by YES or NO.

1. (E) Do you enjoy meeting new people?
2. (L) Would you dodge paying taxes if you were sure you could never be found out?
3. (E) Are you a talkative person?
4. (P) Do you lock up your house carefully at night?
5. (P) Do you enjoy practical jokes that can sometimes really hurt people?
6. (L) Have you ever blamed someone for doing something you knew was really your fault?
7. (STA) Are you very hurt by criticism?
8. (E) Do you like mixing with people?
9. (E) Do you have many friends?
10. (STB) Do you often feel the impulse to spend money which you know you can't afford?
11. (N) Do you worry a lot about your looks?
12. (STA) Do you dread going into a room by yourself where other people have already gathered and are talking?
13. (E) Can you usually let yourself go and enjoy yourself at a lively party?
14. (N) Are your feelings easily hurt?
15. (STB) Do you often overindulge in alcohol or food?
16. (STA) Do your thoughts ever stop suddenly causing you to interrupt what you are saying?
17. (STA) Do you feel it is safer to trust nobody?
18. (L) As a child did you do as you were told immediately and without grumbling?
19. (STA) When in the dart do you often see shapes and forms even though there's nothing there?
20. (STB) Do you at times have fits of laughing or crying that you can't control?
21. (L) Have you ever been late for an appointment or work?
22. (P) Do you stop to think things over before doing anything?
23. (STA) Have you ever felt when you looked in a mirror that your face seemed different?
24. (STB) Do you often experience an overwhelming sense of emptiness?
25. (E) Can you easily get some life into a rather dull party?
26. (STB) Do you ever have the urge to break or smash things?
27. (N) Are you an irritable person?
28. (P) Do you believe insurance schemes are a good idea?
29. (STA) Do you ever have a sense of vague danger or sudden dread for reasons that you do not understand?
30. (N) Are you easily hurt when people find fault with you or the work you do?
31. (E) Are you mostly quiet when you are with other people?
32. (E) Do you usually take the initiative in making new friends?
33. (P) Do good manners and cleanliness matter much to you?
34. (STB) Do you at times have an urge to do something harmful or shocking?
35. (E) Do you like telling jokes and funny stories to your friends?
36. (E) Would you call yourself happy-go-lucky?
37. (L) Do you sometimes talk about things you know nothing about?
38. (STA) Do you ever suddenly feel distracted by distant sounds that you are normally not aware of?
39. (P) When you catch a train do you often arrive at the last minute?
40. (STA) Does your sense of smell sometimes become unusually strong?
41. (STA) Do you feel at times that people are talking about you?
42. (N) Have you often felt listless and tired for no reason?
43. (P) Does it worry you if you know there are mistakes in your work?
44. (E) Do other people think of you as being very lively?
45. (STA) Have you ever thought you heard people talking only to discover that it was in fact some nondescript noise?
46. (P) Do most things taste the same to you?
47. (P) Do people tell you a lot of lies?
48. (L) Have you ever taken advantage of someone?
49. (STA) Are you easily distracted from work by daydreams?
50. (STA) When coming into a new situation, have you ever felt strongly that it was a repeat of something that has happened before?
51. (STB) Do you frequently gamble money?
52. (L) As a child were you ever cheeky to you parents?
53. (N) Do you worry too long after an embarrassing experience?
54. (N) Have you ever wished that you were dead?
55. (STB) Do you hate being alone?
56. (STA) Do you often feel that people have it in for you?
57. (E) Do you nearly always have a 'ready answer' when people talk to you?
58. (STB) Do you often feel like doing the opposite of what other people suggest even though you know they are right?
59. (L) Do you always practice what you preach?
60. (E) Are you rather lively?
61. (STA) Are you sometimes sure that other people can tell what you are thinking?
62. (STA) Does your voice even seem distant, faraway?
63. (P) Do you try not to be rude to people?
64. (N) Do you often feel life is very dull?
65. (N) Are you a worrier?
66. (STB) Do you have difficulty in starting to do things?
67. (STA) Do you feel that you cannot get 'close' to other people?
68. (N) Do you prefer reading to meeting people?
69. (N) Do you often feel 'fed up'?
70. (L) Were you ever greedy by helping yourself to more than your share of anything?
71. (E) Do you like doing things in which you have to act quickly?
72. (P) Is (or was) your mother a good woman?
73. (P) Do you like to arrive at your appointments in plenty of time?
74. (STA) Do you feel lonely most of the time even when you are with people?
75. (STB) Do you often feel that there is no purpose to life?

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76. (P) Would you feel very sorry for an animal caught in a trap?
77. (L) Have you ever said anything bad or nasty about anyone?
78. (E) Do you like plenty of bustle and excitement around you?
79. (P) Do you sometimes like teasing animals?
80. (STA) Are your thoughts sometimes so strong that you can almost hear them?
81. (E) Do you tend to keep in the background on social occasions?
82. (STA) Do you often have vivid dreams that disturb your sleep?
83. (STB) Have you ever felt the urge to injure yourself?
84. (N) Do you worry about awful things that might happen?
85. (N) Do you often feel lonely?
86. (STA) Do you ever feel that your speech is difficult to understand because the words are all mixed up and don't make sense?
87. (N) Do you worry about your health?
88. (P) Do you think marriage is old-fashioned and should be done away with?
89. (L) Are your thoughts about sex often odd or bizarre?
90. (L) Have you ever taken anything (even a pin or a button) that belonged to someone else?
91. (P) Would you take drugs which may have strange or dangerous effects?
92. (STA) Do everyday things sometimes seem unusually large or small?
93. (N) Do you ever feel 'just miserable' for no reasons?
94. (N) Do you ever suffer from 'nerves'?
95. (STA) Have you ever felt that you were communicating with someone telepathically?
96. (L) Are you always willing to admit it when you have made a mistake?
97. (STA) Do you ever become oversensitive to light or noise?
98. (STB) Does life seem entirely hopeless?
99. (STB) Have you ever taken the praise for something you knew someone else had really done?
100. (STA) Do you ever have the sensation that your body or a part of it is changing shape?
101.(N) Would you call yourself tense or 'highly strung'.
102.(L) Have you ever insisted on having your own way?
103.(N) Are you often troubled by feelings of guilt?
104.(E) Do you have many different hobbies?
105.(STA) Do you believe that dreams can come true?
106.(N) Does your mood often go up and down?
107.(STB) Do you often have the urge to hit someone?
108.(N) Would you call yourself a nervous person?
109.(P) Would you like other people to be afraid of you?
110.(STA) Do you ever get nervous when someone is walking behind you?
111.(L) Are all your habits good and desirable ones?
112.(L) Have you ever cheated at a game?
113.(L) Have you ever broken or lost something belonging to someone else?
114.(L) Do you sometimes boast a little?
115.(STB) Do you often have periods of such great restlessness that you are not able to sit still for more than a very short time?
116.(P) Do people who drive carefully annoy you?
117.(N) Are you touchy about some things?
118.(STB) Do you ever have suicidal thoughts?
119.(P) Do your friendships break up without it being your fault?
120.(STA) Do you feel that you have to be on your guard even with your friends?
121.(P) Would it upset you a lot to see a child or an animal suffer?
122.(E) Can you get a party going?
123.(L) Do you always wash before a meal?
124.(STA) When in a crowded room do you often have difficulty in following conversation?
125.(STA) When you are worried or anxious do you have trouble with your bowels?
126.(N) Do you suffer from sleeplessness?
127.(STA) Do you believe in telepathy?
128.(P) Are there several people who keep trying to avoid you?
129.(STB) Do you often change between intense liking and disliking of the same person?
130. (L) If you say you will do something, do you always keep your promise no matter how inconvenient it may be?
131.(STA) Do things sometimes feel as though they were not real?
132.(L) Do you sometimes put off until tomorrow what you ought to do today?
133.(STA) Do you sometimes feel that your accidents are caused by mysterious forces?
134.(P) Would being in debt worry you?
135.(STA) Are you ever bothered by the feeling that people are watching you?
136.(P) Do you enjoy hurting people you love?
137.(STA) Do you ever feel that your thoughts don't belong to you?
138.(P) Do you think people spend too much time safeguarding their future with savings and insurance?
139.(STA) Do you ever feel sure that something is about to happen even though there does not seem to be any reason for you thinking that?
140.(E) Do you often take on more activities that you have time for?
141.(STA) Does it often happen that nearly every thought immediately and automatically suggests an enormous number of ideas?
142.(N) Are you sometimes bubbling over with energy and sometimes very sluggish?
143.(N) Do you often worry about things you should not have done or said?
144.(P) Do you have enemies who want to harm you?
145.(E) Do you like going out a lot?
PART TWO

All questions are answered by TRUE or FALSE.

1. (SoAn) I would be happy living alone in a cabin in the woods or mountains.
2. (PhAn) When I see a statue I have the urge to feel it.
3. (PhAn) Sunbathing is not really more fun than lying down indoors.
4. (HoP) There are so many fields I could succeed in that it seems a shame to have to pick one.
5. (NPS) I do not like to mix with people.
6. (PAb) I have sometimes had the feeling that my body is decaying inside.
7. (Mgl) When introduced to strangers, I rarely wonder whether I have known them before.
8. (dC) I have felt that I am condemned for ever.
9. (HoP) I have sometimes felt that nothing can happen to me until I do what I am meant to do in life.
10. (SoAn) When someone close to me is depressed, it brings me down also.
11. (HoP) I seem to have an uncommon ability to inspire others.
12. (PhAn) I have always had a number of favourite foods.
13. (HoP) People often come to me when they need a clever idea.
14. (HoP) When I go to a gathering where I don't know anyone, it usually takes me a while to feel comfortable.
15. (PhAn) The sound of rustling leaves have never much pleased me.
16. (Mgl) The hand motions that strangers make seem to influence me at times.
17. (PAb) Sometimes when I look at things like tables and chairs, they seem strange.
18. (dP) People have been secretly plotting to ruin me.
19. (Mgl) I almost never dream about things before they happen.
20. (HoP) I expect that someday I will succeed in several different professions.
21. (SoAn) If given the choice, I would much rather be with others than be alone.
22. (HoP) I often get into moods where I feel like many of the rules of life do not apply to me.
23. (HoP) Sometimes ideas and insights come to me so fast that I cannot express them all.
24. (SoAn) I find that people too often assume that their daily activities and opinions will be interesting to me.
25. (PhAn) I don't know why some people are so interested in music.
26. (HoP) I seem to be a person whose mood goes up and down easily.
27. (PAb) I have had the momentary feeling that the things I though remain attached to my body.
28. (LSHS) Sometimes my thoughts are as real as actual events in my life.
29. (HoP) There are often times when I am so restless that it is impossible for me to set still.
30. (PAb) I have felt as though my head or limbs were somehow not my own.
31. (dP) Someone has deliberately tried to make me ill.
32. (PhAn) The first winter snowfall has often looked pretty to me.
33. (SoAn) Although I know I should have affection for certain people, I don't really feel it.
34. (Mgl) At times I perform certain little rituals to ward off negative influences.
35. (PhAn) A brisk walk has sometimes made me feel good all over.
36. (NPS) I often have difficulties in controlling my thoughts when I am thinking.
37. (PAb) I have never felt that my arms or legs have momentarily grown in size.
38. (SoAn) People who try to get to know me better usually give up after a while.
39. (dG) I have considered myself superior to everyone.
40. (PhAn) I think that flying a kite is silly.
41. (SoAn) When others try to tell me about their problems and hang-ups, I usually listen with interest and attention.
42. (Mgl) I have never really had the feeling that certain thoughts of mine really belonged to someone else.
43. (PhAn) I have been fascinated with the dancing of flames in a fireplace.
44. (NPS) I am much worried over humiliating experiences.
My moods do not seem to fluctuate any more than most people's do.

No matter how hard I try to concentrate, unrelated thoughts always creep into my mind.

I have sometimes had the feeling of gaining or losing energy when certain people look at me or touch me.

I can remember when it seemed as though one of my limbs took on an unusual shape.

I am easily distracted when I read or talk to someone.

I have had the momentary feeling that my body has become misshapen.

Sometimes I am so nervous that I am 'blocked'.

I often feel excited and happy for no particular reason.

When things are going really good for my close friends, it makes me feel good too.

Although there are things which I enjoy doing myself, I usually seem to have more fun when I do things with other people.

I have sometimes felt that strangers were reading my mind.

I have seldom cared to sing in the shower.

I have always found organ music dull and unexciting.

The warmth of an open fireplace has not especially soothed and calmed me.

My feelings have been taken over by someone.

I often get so happy and energetic that I am almost giddy.

When I pass by flowers, I have often stopped to smell them.

I have felt that I have special, almost magical powers.

Just being with friends can make me feel really good.

I would rather be an ordinary success in life than a spectacular failure.

Sometimes I have had feelings that I am united with an object near me.

I have always hated the feeling of exhaustion that comes from vigourous activity.

I often get into excited moods where it's almost impossible to stop talking.
68. (PhAn) The taste of food has always been of importance to me.
69. (PAb) Sometimes I feel like everything around me is tilting.
70. (HoP) I often have moods when I feel so energetic and optimistic that I feel I could outperform almost everyone at anything.
71. (SaAn) My emotional responses seem very different from those of most people.
72. (HoP) There have often been times when I have had such an excess of energy that I felt little need to sleep at night.
73. (GMS) One food tastes as good as another to me.
74. (SoAn) I have been disappointed in love.
75. (SoAn) When I am home alone, I often resentment people telephoning me or knocking on my door.
76. (PhAn) I have always loved having my back massaged.
77. (PhAn) Poets always exaggerate the beauty and joys of nature.
78. (PhAn) When eating a favourite food, I have often tried to eat slowly to make it last longer.
79. (LSHS) Sometimes a passing though will seem so real that it frightens me.
80. (PhAn) The smell of dimmer cooking has hardly ever aroused my appetite.
81. (MgI) I often daydream.
82. (PhAn) Things sometimes seem to be in different places when I get home, even though no one has been there.
83. (PhAn) I have usually found soft music boring rather than relaxing.
84. (HoP) I am frequently so 'hyper' that my friends kiddingly ask what drug I'm taking.
85. (LSHS) I have heard the voice of the devil.
86. (LSHS) In the past I have had the experience of hearing a person's voice and then found that no one was there.
87. (HoP) In unfamiliar surroundings, I am often so assertive and sociable that I surprise myself.
88. (LSHS) I often hear a voice speaking my thoughts aloud.
89. (PAb) Sometimes I have had a passing thought that some part of my body was rotting away.
90. (HoP) I am no more self-aware than the majority of people.
91. (PAb) I have sometimes had the feeling that my body is abnormal.
92. (dC) I have thought that the world is such an evil place that I, and those nearest to me, would be better out of it.
93. (PAb) I have sometimes felt that some part of my body no longer belonged to me.
94. (PhAn) Sex is okay, but not as much fun as people claim it is.
95. (SoAn) Making new friends is not worth the energy it takes.
96. (dD) Someone else has been doing the thinking that goes on in my head.
97. (PAb) The boundaries of my body always seem clear.
98. (SoAn) I have often found it hard to resist talking to a good friend, even when I have other things to do.
99. (PhAn) Beautiful scenery has been a great delight to me.
100. (PhAn) I have sometimes enjoyed feeling the strength in my muscles.
101. (PhAn) It has often felt good to massage my muscles when they are tired or sore.
102. (PAb) Occasionally I have felt as though my body did not exist.
103. (NPS) I am easily confused if too much happens at the same time.
104. (PhAn) On hearing a good song I have seldom wanted to sing along with it.
105. (HoP) I find it easy to get others to become sexually interested in me.
106. (PhAn) When I have walked by a bakery, the smell of fresh bread has often made me hungry.
107. (PhAn) Dancing, or the idea of it, has always seemed dull to me.
108. (HoP) I have often persuaded groups of friends to do something really adventurous or crazy.
109. (SoAn) People often expect me to spend more time talking with them than I would like.
110. (HoP) I am frequently in such high spirits than I can't concentrate on any one thing for too long.
111. (dP) Voices have spoken to me when no one was there at all.
112. (Mgl) At times I have felt that a teacher's lecture was meant especially for me.
113. (Dp) I have felt that an organisation or group has been planing my downfall.
I don't understand why people enjoy looking at the stars at night.

I never had really close friends in school.

Sometimes people whom I know well begin to look like strangers.

Often I have a day when indoor lights seem so bright that they bother my eyes.

The sound of organ music has often thrilled me.

I cannot keep interested in the same thing for a long time.

I don't really feel very close to my friends.

I very frequently get into moods where I wish I could be everywhere and do everything at once.

A hundred years after I'm dead my achievements will probably have been forgotten.

My sex life is satisfactory.

I have wondered whether I am male or female.

The colour that things are painted has seldom mattered to me.

I think I would make a good actor, because I can play many roles convincingly.

At times I have wondered if my body was really my own.

I have occasionally had the silly feeling that a TV or radio broadcaster knew I was listening to him.

Having close friends is not as important as some people say.

In many ways, I prefer the company of pets to the company of people.

I have never had the desire to take off my shoes and walk through a puddle barefoot.

I have felt that my body and another person's body were one and the same.

In the past I have heard the voice of God speaking to me.

I'm much too independent to really get involved with other people.

Ordinary colours sometimes seem much too bright to me (without taking drugs).

I often prefer to be alone rather than being together with other people.
I have often felt uncomfortable when my friends touch me.

I have often felt that there were messages for me in the way things were arranged, like in a store window.

When I move to a new city, I feel a strong need to make new friends.

People sometimes think I am shy when I really just want to be left alone.

Some people can make me aware of them just by thinking about me.

I feel pleased and gratified as I learn more and more about the emotional life of my friends.

There are things that are more important to me than privacy.

I attach very little importance to having close friends.

I prefer watching television to going out with other people.

Flowers are not as beautiful as many people claim.

It has always made me feel good when someone I care about reaches out to touch me.

I think I could learn to read other's minds if I wanted to.

I am so good at controlling others that it sometimes scares me.

I have felt that something outside my body was part of my body.

I can't imagine that anyone would ever write a book about my life.

For several days at a time I have had such a heightened awareness of sights and sounds that I cannot shut them out.

When with groups of people, I usually prefer to let someone else be the centre of attention.

I have harmed people because I am unclean or evil.

At social gatherings, I am usually the 'life of the party'.

I have noticed sounds on my records that are not there at other times.

I have always enjoyed looking at photographs of friends.

I have sometimes had the passing thought that strangers are in love with me.

I have thought that I was being followed for a special reason.

Good luck charms don't work.

I consider myself to be pretty much an average kind of person.
I have usually finished my bath or shower as quickly as possible just to get it over with.

I have not lived the right kind of life.

In my daydreams I can hear the sound of a tune almost as clearly as if I were actually listening to it.

I have worried that people on other planets may be influencing what happens on earth.

There are few things more tiring than having along, personal discussion with someone.

I have sometimes danced by myself just to feel my body move with the music.

I have usually found love making to be intensely pleasurable.

I have felt that have been sent to save the world.

The bright lights of a city are exciting to look at.

On seeing a soft, thick, carpet, I have sometimes had the impulse to take off my shoes and walk barefoot on it.

I like to make long distance phone calls to relatives and friends.

Sometimes I have had the feeling that a part of my body is larger than it usually is.

Occasionally it has seemed as if my body has taken on the appearance of another person's body.

I am usually in an average sort of mood, not too high and not too low.

I sometimes have to touch myself to make sure that I'm still there.

When I'm feeling a little sad, singing has often made me happier.

I have very little desire to buy new kinds of foods.

I have been troubled by hearing voices in my head.

A car ride is much more enjoyable if someone is with me.

I am sure I am being talked about.

I have often enjoyed receiving a firm, warm handshake.

The government refuses to tell us the truth about flying saucers.

I like playing with and petting soft little kittens or puppies.
There have been people trying to poison me or do me great harm.

It's fun to sing with other people.

Knowing I have friends who care about me gives me a sense of security.

I often hesitate when I am going to say something in a group of people that I know more or less.

My relationships with other people never get very intense.

I have never wanted to go on any of the rides in an amusement park.

I feel that I must tell the whole world of my brilliant ideas.

I have never found a thunderstorm exhilarating.

After a busy day, a slow walk has often felt relaxing.

I do most of my best work during brief periods of intense inspiration.

People often behave so strangely that one wonders if they are part of an experiment.

Someone has had evil designs against me.

I have often been so excited about an involving project that I didn't care about eating or sleeping.

I've never cared much about the texture of food.

I prefer hobbies and leisure activities that do not involve other people.

I have felt that I have been interfered with sexually or electrically.

I sometimes have had the feeling that some parts of my body are not attached to the same person.

I am considered to be kind of a 'hyper' person.

I think I would make a good night club comedian.

Trying new foods is something I have always enjoyed.

When things are bothering me, I like to talk to other people about it.

I have never had the passing feeling that my arms or legs had become longer than usual.

It is not possible to harm other merely by thinking bad thoughts about them.

If reincarnation were true, it would explain some unusual experiences I have had.
I have felt that I am the vilest, most wicked person alive.

It is hard for me to make decisions.

I have seldom enjoyed any kind of sexual experience.

I have such a wide range of interests that I often don't know what to do next.

The sounds I hear in my daydreams are usually clear and distinct.

I have often enjoyed the feel of silk, velvet or fur.

I have had very little fun from physical activities like walking, swimming or sports.

Standing on a high place and looking out over the view is very exciting.

Numbers like 13 or 7 have no special powers.

Sometimes I have felt that I could not distinguish my body from other objects around me.

When I feel very excited and happy, I almost always known the reason why.

Now and they when I look in the mirror, my face seems quite different than usual.

I have felt that there was a special meaning in one side of my body being different from the other.

It has seemed at times as if my body was melting into my surroundings.

Many people consider me to be amusing but kind of eccentric.

I am usually content just to sit alone, thinking and daydreaming.

I have never cared to sunbathe; it just makes me hot.

I have felt that I have committed the unforgivable sin.

I have sometimes had the feeling that one of my arms or legs is disconnected from the rest of my body.

On occasions I have seen a person's face in front of me when no one was in fact there.

The beauty of sunsets is greatly overrated.

Playing with children is a real chore.

I have had the momentary feeling that I might not be human.
I have sometimes been fearful of stepping on sidewalk cracks.
The sounds of a parade have never excited me.
I have thought that I am the richest person in the world.
There are just not many things that I have ever really enjoyed doing.
Horoscopes are right too often for it to be a coincidence.
I have had the momentary feeling that someone's place has been taken by a lookalike.
It would make me nervous to play the clown in front of other people.
People have been trying to drive me insane.
My hearing is so sensitive that ordinary sounds become uncomfortable.
I frequently get into moods where I feel very speeded up and irritable.
I have often felt happy and irritable at the same time.
Parts of my body occasionally seem dead or unreal.
The people in my daydreams seem so true to life that I sometimes think they are.
I frequently write down the thoughts and insights that come to me when I am thinking especially creatively.
I am very nervous if I am going to speak in front of a group of strangers.
Sex is the most intensely enjoyable thing in life.
I am more sensitive than most other people.
It made me sad to see all my school friends go their separate ways when school was over.
I often change between positive and negative feelings towards the same person.
I have seen visions of strange things which no one else could see.
I frequently find that my thoughts are racing.
I usually work things out for myself rather than get someone to show me how.
I have never doubted that my dreams are the products of my own mind.
Sometimes part of my body has seemed smaller than it usually is.
The sound of rain falling on the roof has made me feel snug and secure.
257. (dG) I have felt that I am a very much greater person than most people think.
258. (HoP) When I feel an emotion, I usually feel it with extreme intensity.
259. (HoP) I would really enjoy being a politician and hitting the campaign trail.
260. (MgI) I have sometimes sensed an evil presence around me, although I could not see it.
261. (MgI) I have wondered whether the spirits of the dead can influence the living.
262. (SoAn) I sometimes become deeply attached to people I spend a lot of time with.
263. (dG) I have felt that I have a mission to carry out of great importance to the world.
264. (NPS) I easily lose my courage when criticised or failing in something.
265. (SoAn) People are usually better off if they stay aloof from emotional involvements with others.
266. (dC) I have felt that my insides are all rotten.
267. (MgI) I have felt that I mighty cause something to happen just by thinking too much about it.
268. (PAb) My hands or feet have never seemed far away.
269. (HoP) I can usually slow myself down when I want to.
270. (HoP) I like to have others think of me as a normal kind of person.
271. (PAb) I have sometimes felt confused as to whether my body was really my own.
272. (dC) People have been talking about me because of my wicked deeds.
273. (PhAn) A good soap lather when I'm bathing has sometimes soothed and refreshed me.
274. (PhAn) I have often found walks to be relaxing and enjoyable.
275. (GMS) I enjoy many different kinds of play and recreation.
APPENDIX 7.2

THE RUST INVENTORY OF SCHIZOID COGNITIONS

All questions are answered on a five-point scale from "STRONGLY DISAGREE" TO "STRONGLY AGREE".

1. Sometimes I feel ugly, and at other times that I am attractive.

2. I have never embarrassed myself by expressing unreasonable jealousy.

3. I consider no person or country to be my enemy.

4. I sometimes tell people too much about myself and almost regret it.

5. I have never seen anything that looked like a ghost.

6. Sometimes my thoughts seem so loud I can almost hear them.

7. I am almost always consistent in what I say and believe.

8. Most people are too stupid to realise which things in life are important.

9. In pitch dark I never see any visual images.

10. I have never 'come out in a cold sweat' upon realising what I have told someone about myself.

11. There are some people whom I trust completely.

12. I have, on occasions, tried to reach the very essence of an object with my mind.
13. When I try to help people they often misunderstand my motives.

14. I have occasionally had to put my sudden sniffing of a smell down to imagination.

15. I never use a lucky charm.

16. Secret organisations have no real power or influence on our lives.

17. I am sometimes unsure whether I have said something aloud or not.

18. Sometimes I suspect that the real world is nothing like what it seems.

19. I would not be in the least concerned if a person who believed in magic tried to put a spell on me.

20. It has never occurred to me that the world may be a figment of my imagination.

21. I am not a superstitious person.

22. I don't really understand why I say some of the things I do.

23. Sometimes I get a weird feeling that I'm not really here.

24. I have never suspected that people I am fond of may be secretly working against me.

25. Sometimes people or objects seem to me to glow with an inner light.

26. Things sometimes go so well for me that I suspect I may be receiving help from an outside agency.
APPENDIX 7.3

FORM OF CONSENT SIGNED BY ALL SUBJECTS PRIOR TO TESTING

DATE : ______________________

NAME : ______________________

ADDRESS : ______________________

________________________________ 
________________________________ 
________________________________ 

CODE : ______________________

CONSENT FORM

I consent to participate in the research study carried out by Mr S. Georgiades, a Psychologist at the Institute of Psychiatry, in which the different ways of thinking in the general population are studied.

I understand that all information obtained will be strictly confidential and will only be used for research purposes.

________________________________
Signature

________________________________
in the presence of
Stelios Georgiades
Psychologist